Sixth Edition

Michlovitz's MODALITIES FOR THERAPEUTIC INTERVENTION

James W. Bellew
Susan L. Michlovitz
Thomas P. Nolan, Jr.

Contemporary Perspectives in Rehabilitation

MODALITIES FOR THERAPEUTIC INTERVENTION

Sixth Edition

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Sixth Edition

Previously titled *Thermal Agents in Rehabilitation*, editions 1, 2, and 3

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This edition is dedicated to my wife, Mary Helen,
and daughters, Kate and Caroline,
who have amazed me with their joy, love, and support
throughout my career and life,
and to all the students past and present who inspire me every day.

— Jim Bellew

I would like to dedicate this edition to the
students, clinicians, and faculty who have
supported and had faith in this textbook since 1986.

— Sue Michlovitz

This edition is dedicated to the physical therapy faculty at
Stockton University and to the many Stockton physical therapy
students who have contributed to this textbook.

— Tom Nolan

Foreword

Pertain: be appropriate, related, or applicable.

synonyms: concern, related to, be connected with, be relevant to, regard, apply to, be pertinent to, refer to have, affect, involve, touch on

Sustain: strengthen or support physically or mentally

synonyms: comfort, help, assist, encourage, succor, support, give strength to, buoy up, carry, cheer up, hearten

The American Physical Therapy Association defines physical therapists as professional health care providers who . . .

". . . will be responsible for evaluating and managing an individual's movement system across the lifespan to promote optimal development; diagnose impairments, activity limitations, and participation restrictions; and provide interventions targeted at preventing or ameliorating activity limitations and participation restrictions. The movement system is the core of physical therapist practice, education, and research." (http://www.apta.org/Vision/)

There is little doubt that embedded within this definition is the recognition that physical therapists are outstanding authorities on movement pathology and, as such, are responsible for the implementation of new procedures and technologies, irrespective of the patient population for which each possesses the greatest treatment skills. While the advent and assimilation of novel manual skills and assistive technologies are undeniable, throughout our distinguished history a common thread has weaved its way through our professional fabric: our use of modalities as either primary sources of treatment or as adjuncts to our manual skills and the concurrent discourse with our patients. One might say that modalities pertain to much of what we do . . . often to relax or excite tissues or structures in preparation for enhanced function. More often than not, such applications bring comfort to our patients and foster compliance with the totality of a therapeutic plan. We could even believe the tools that we call modalities sustain us because so often there is unequivocal evidence of the immediacy to which our patients respond to their physiological impact. Such positive responsiveness infuses confidence in us by our patients and reaffirms that we are on the right path toward improving an existing pathology.

While this perspective appears encouraging and may validate the belief that we are truly helping our patients, we are equally justified in our concern that perhaps we have come to take for granted the myriad of modalities and the conditions that they can positively influence. Without a reference that is continuously updated and to which any clinician can turn with unabated confidence, perhaps we might lose sight of advances in these agents or in our ability to maintain our position as the foremost authorities in their use. *Modalities for Therapeutic Interventions,* originally called *Thermal Agents in Rehabilitation* in its first iteration as the very first volume within the *Contemporary Perspectives in Rehabilitation (CPR)* series exactly 30 years ago, is now experiencing its sixth edition. The fact that this text has truly withstood the test of time is testimony to how well it has evolved and become beloved as the "go to" textbook on modality use in rehabilitation. This edition has been brilliantly conceived by Drs. Bellew, Michlovitz, and Nolan and now—for the first time—is even more vibrant owing to the four-color format and color photos that pervade its content. All chapters have been updated, and the tradition that "binds" all volumes of the CPR series—challenging case histories and clinical decision-making formatting—is pervasive, as is the infusion of Key Points distributed throughout each chapter. These points stand out in blue print as beacons from which students can extract essential information within subject material.

Jim Bellew provides a new and exciting introductory chapter that reminds students and clinicians about the importance and use of modalities. New chapters on ultrasound (Chapter 4, David Lake), mechanical compression (Chapter 8, Robert Marsico), electrical stimulation for pain control (Chapter 11, Richard Liebano), and modalities for improving range of motion (Chapter 12, Andrew Starsky) and new content on laser therapy within the chapter on Therapeutic Modalities for Tissue

Healing (Chapter 15, Ed Mahoney) are contributions that contain information not previously addressed in the fifth edition. Throughout the text, attention is directed not only to evidence supporting the circumstances for optimal use of a modality (a concept gathering greater appreciation as we struggle to support additional treatment for our patients) but also—equally as significant and so often overlooked—the identification of situations and circumstances where evidence is lacking.

In a time when modalities might be less appreciated, we must not lose sight of the fact that our treatment approaches have become far more dynamic and interactive. If we choose a perspective that advocates for modality application as a vehicle to foster functionally based activity either in conjunction with its use or as an immediate consequence, we begin to see these steadfast stalwarts as our faithful partners, who have always been there for our use but whose appeal can be viewed in a more contemporary mode. For over 70 years they have been a part of our armamentarium. Indeed, they do pertain to the totality of our treatment, and their very presence has always been there to sustain us.

Our collective hope is that this philosophical bent will be conveyed to the next generation of students and clinicians, who will view this text as the friend it has become to past generations.

Steven L. Wolf, PhD, PT, FAPTA, FAHA
Editor-in-Chief, *Contemporary Perspectives in Rehabilitation* Series
Atlanta, Georgia
February 2016

Preface to the Sixth Edition

Circa 1982, I met Dr. Steve Wolf at a Pan American Rheumatology Meeting in Washington, DC. He had recently published a book on electrotherapy that I was using in my course at Hahnemann University. I told him I was using his book but needed one for my first course, Thermal Agents. I met F. A. Davis acquisitions editor, Bob Martone, shortly thereafter, and bingo, the second book in the Contemporary Perspectives in Rehabilitation was birthed. In this sixth edition, I have turned over the reins to Dr. Jim Bellew. He continues to team with Dr. Tom Nolan and our many authors to produce a high-quality textbook.

Over the decades of my career as a physical therapist, I have seen modalities used or not used in a similar manner as the action potential of a nerve—that is, "all or nothing." On one end of the spectrum we would frown upon "fake and bake" clinics. At the other end of the spectrum there are therapists and documents that profess the lack of need or that discourage use of any modalities for a patient. Somewhere between lies good clinical reasoning.

To instructors, please do not use the material in this book in isolation of other courses you teach. Combine the information into the curriculum related to musculoskeletal, neuromuscular, and integumentary problems. Foster rationale and logical uses of modalities in the patient-centered care model. Teach your students how to appropriately assess the need for a modality within a treatment paradigm and how to appropriately measure the outcome.

Over the last five editions, we have worked and reworked sections and chapters. You can read through the table of contents and peruse the book to appreciate the variety of topics covered by expert clinician authors. Aspects of rational clinical decision-making are threaded throughout the chapters. We want our patients to have the best chance to work toward functional mobility and improve their body structure and function, activities, and participation. The judicious use of modalities is a good place to begin.

To all young faculty and students who aspire to work on projects, be careful what you ask for! I met Steve Wolf in 1982, had a brief discussion, and was on the road to a textbook that is now in its sixth edition.

Enjoy this textbook and please do give us feedback.

Sue Michlovitz

Preface to the First Edition

Thermal agents are used in physical therapy and rehabilitation to reduce pain, to enhance healing, and to improve motion. The physical therapist should have a solid foundation in the normal physiologic control of the cardiovascular and neuromuscular systems prior to using an agent that can alter the function of these structures. In addition, a background in the physiology of healing mechanisms and of pain serves as a basis for the rationale of using thermal agents.

Often, the decision to include a thermal agent in a therapy plan or to have the thermal agent be the sole treatment rendered (as in the case of the frequently used "hot packs and ultrasound combination" for back pain) is based on empirical evidence. The purpose of this book is to provide the reader with the underlying rationale for selection of an agent to be included in a therapy program, based on (1) the known physiologic and physical effects of that agent; (2) the safety and use of the heat/cold agent, given the conditions and limitations of the patient's dysfunction; and (3) the therapeutic goals for that particular patient. The authors have been asked to review critically the literature available that documents the efficacy and effectiveness of each thermal agent. A problem-solving approach to the use of thermal agents is stressed throughout the text.

The primary audience for this text is the physical therapist. The student will gain a solid foundation in thermal agents, the clinician will strengthen his or her perspective of thermal agents, and the researcher is given information that will provide ideas for clinical studies on thermal agents. Athletic trainers and other professionals who use thermal agents in their practice should find this text of value.

The text is in three parts. Part I, Foundations for the Use of Thermal Agents, includes information from basic and medical sciences that can serve as a framework for the choice to include thermal agents in a rehabilitation program. A discussion of the proposed mechanisms by which heat and cold can alter inflammation, healing, and pain is included in these chapters.

Part II of the text, Instrumentation: Methods and Application, incorporates concepts of equipment selection, operation and maintenance, and clinical application. The leading chapter in this part is on instrumentation principles and serves to introduce concepts of equipment circuitry and safety as applied to equipment used for thermal therapy. Physical therapists have become responsible for product purchase and making recommendations about products through the expansion of consultation services, private practices, sports medicine clinics, extended care facilities, and home health care. Therefore, we must be prepared to engage in dialogue with manufacturers, product distributors, and other colleagues about the safety and quality of these products. To this end, some practical suggestions are provided in Chapter 3 to assist with purchase decisions.

Chapters 4 through 8 discuss the operation and application of heat and cold agents. Numerous principles of clinical decision-making are included within each chapter. There are certain principles inherent to all agent applications: (1) The patient must be evaluated and treatment goals established; (2) contraindications to treatment must be known; and (3) the safe and effective use of equipment must be understood.

Chapter 9, on low-power laser, deviates somewhat from the overall theme of thermal agents. Low-power laser is not expected to produce an increase in tissue temperature, so its effects could not be attributed to thermal mechanisms. Therefore, this cannot be categorized as a thermal agent. However, I believe this topic is worthy of inclusion in this text because (1) the indications for its use overlap those of thermal agents; (2) laser is a form of non-ionizing radiation, as are diathermy and ultrasound, which are used for pain reduction and tissue healing; and (3) laser would most likely be included in a physical therapy student curriculum in the coursework that includes thermal agents. At the time of this writing (summer 1985), low-power laser is still considered by the U.S. Food and Drug Administration as an investigational device. Only carefully designed clinical studies

will help determine the laser's clinical efficacy—perhaps contributing to the body of knowledge needed to change the laser's status from an investigational to an accepted therapeutic product.

Part III, Clinical Decision Making, is designed to assist the student and clinician in integrating basic concepts that have been presented throughout the entire book, emphasizing problem solving and evaluation.

Much information has been published in the medical literature on the effects or clinical results of heat and cold application. Oftentimes, the therapist is called upon to justify the use of a certain modality. A careful review of the research literature may be necessary to provide an explanation for treatment.

There are many areas that require further investigation. For example, contrast baths (alternating heat and cold) are often used in sports medicine clinics. But a careful review of the literature reveals that only scanty information supports the use of contrast baths for any patient population. It is important for the clinician to be able to interpret accurately and to apply the methods and results that are presented in the literature. The inclusion of a chapter (Chapter 10) on techniques for reviewing the literature and establishing a paradigm for clinical studies of thermal agents provides the clinician with such a background on which to build.

Chapters 11 and 12 are devoted to specific patient populations in which thermal agents are commonly used. The chapter on sports medicine is representative of a population with a known cause of injury and predictable course of recovery. The majority of these patients are otherwise healthy. On the other hand, the chapter on rheumatic disease presents a model for a patient population that can be expected to have chronic recurrent—sometimes progressive—dysfunction associated with systemic manifestations.

An appendix is included: temperature conversion scales (this text uses the centigrade scale).

Susan L. Michlovitz, PhD, PT, CHT

Acknowledgments

To continue into this sixth edition would not be possible without the continued support of our loyal users. Thank you to the faculty, students, and clinicians who have continued to use this text throughout its history. Many thanks are due to the special people at F. A. Davis who continue to support this text: Melissa Duffield, George Lang, and Margaret Biblis. A very special thank-you goes to the developmental editor, Susan Williams, of the Williams Company, for all the guidance and experience in completing this edition. Thank you to Jason Torres of J. Torres Photography for the outstanding photography included in this first full-color edition. Thank you to Drs. Joe McCulloch and Ed Mahoney of the School of Allied Health Professions at the Louisiana State University Health Sciences Center–Shreveport for their contribution of several key images throughout this text. Thank you to Dr. Rick Proctor and Dave Walters of DJO Global for supplying equipment for the photo shoot. And finally, but never last, a huge thank-you to the students who participated as models in this edition: Daniel Batteiger, Brooke Versteeg, Allison Colligan, and Austin Biefnes from the University of Indianapolis, and Jamie Umstetter, Brandon Dooley, Kavita Patel, and Matthew Romen from Richard Stockton University.

Biographies

James W. Bellew

James W. Bellew, PT, EdD, is Professor of Physical Therapy in the Krannert School of Physical Therapy at the University of Indianapolis. Dr. Bellew received his entry-level bachelor of science degree in physical therapy from Marquette University. After several years of clinical practice in Milwaukee, he received a master of science degree in physical therapy and doctor of education degree in exercise physiology from the University of Kentucky. His research encompasses the use of electrotherapeutic waveforms and muscle physiology. Dr. Bellew has published more than 50 peer-reviewed scientific manuscripts and abstracts in the areas of electrotherapy, exercise training, balance, and muscle physiology. He teaches in the areas of clinical medicine, therapeutic modalities, and human physiology. He is a regular presenter and speaker at the American Physical Therapy Association's (APTA) Combined Sections Meetings and is routinely sought nationally and internationally for consultation regarding clinical applications of electrotherapeutic agents. In 2013, he was named conference president for an international meeting on electrophysical agents in Amparo, Brazil. He is a member of APTA and Academy of Clinical Electrophysiology and Wound Management. Dr. Bellew resides with his family in Indianapolis and maintains a regular clinical practice at St. Francis Hospital Rehabilitation Services.

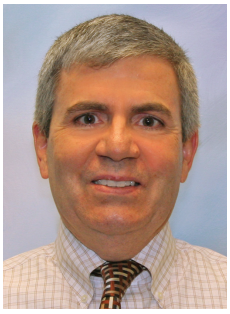
Susan Michlovitz

Susan Michlovitz, PT, PhD, CHT, is a hand therapist and physical therapist. Her clinical interests include arthritis, trauma, and disorders affecting the hand, wrist, and elbow. Dr. Michlovitz is also an adjunct associate professor of rehabilitation medicine at Columbia University, where she teaches in the Doctorate of Physical Therapy Program. In 2005, she was a professor in the Department of Physical Therapy at Temple University, Philadelphia. Her published research has been in determining the effectiveness of therapy interventions and in reliability and validity of examination techniques, mostly related to hand and upper extremity conditions.

Dr. Michlovitz has extensive experience in teaching therapists at the APTA Combined Sections Meetings, the American Society of Hand Therapists (ASHT), the American Association for Hand Surgery (AAHS) Annual Meetings, and the International Federation for Societies of Hand Therapists. She is an associate editor for case reports in the *Journal of Hand Therapy*. Her volunteer outreach work is spent with Guatemala Healing Hands Foundation for teaching and patient care in Guatemala City. She lives in Ithaca, New York, with her husband, Paul Velleman, their basset

hound/beagle, Mr. Baxter, their beagle Freddy, and a somewhat calico cat named Shayna. Sue is a wannabe photojournalist.

Thomas P. Nolan Jr.



Thomas Patrick Nolan Jr., PT, MS, DPT, OCS, is associate professor of physical therapy at Stockton University. Dr. Nolan received his bachelor of science in physical therapy from New York University and his master of science and doctor of physical therapy in physical therapy from Temple University. He is a certified orthopedic specialist (OCS) through the American Board of Physical Therapy Specialties. Dr. Nolan teaches physical modalities, electrotherapy, kinesiology of the spine, musculoskeletal physical therapy, and pharmacology at Stockton University, where he is also the coordinator of physical therapy continuing education courses. He is a per diem physical therapist for Virtua in Motion outpatient offices located in southern New Jersey. He is a member of APTA and the APTA New Jersey Chapter, the APTA Academy of Clinical Electrophysiology and Wound Management, and the APTA Orthopaedic Section. Tom lives in Marlton, New Jersey, where he enjoys spending time with his family at home and summers in Ocean City, New Jersey.

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Contents

How to Use This Book

THERAPEUTIC MODALITIES AS A CURRICULAR THREAD

Traditional classroom and lab-based education in the principles and administration of therapeutic modalities has remained a cornerstone in educational programs within the rehabilitation sciences. The history and evolution of the clinical rehabilitation sciences have shown that certain areas of practice, such as electrical stimulation for denervated muscle or ultraviolet treatment for psoriasis, have waned, whereas other areas of clinical practice, such as integumentary or wound care and oncology, have grown immensely over the past few decades. Consequently, curricular content has undergone continual change and updating. This flux of curricular content reflects the advancement of scientific discovery and application and the mounting rise of literature to bolster evidence-based practice. The fact that curricular content given to principles and application of therapeutic modalities has remained pervasive in educational programs within the rehabilitation sciences substantiates the continued contribution of this area of practice to the more encompassing patient management model.

Although principles and applications of therapeutic modalities remain foundational content in most programs in the rehabilitative sciences, this content is far too often insular or taught apart from other curricular content, such as orthopedics, neurological rehabilitation, integumentary care, patient management, and other areas. This is wholly ironic because therapeutic modalities represent a group of interventions used to augment or supplement interventions taught in these course areas. Many areas of rehabilitative science, such as orthopedics or neurological rehabilitation, are taught with strategic course sequencing with content increasing accordingly in more advanced courses. However, content in therapeutic modalities often exists in a single "how to" course or, worse yet, a smaller part of a single course. Few educational programs sequence curricular content in therapeutic modalities in a progressive manner. Rather, therapeutic modalities are often taught separate from the interventions they complement. For example, orthopedic or musculoskeletal courses include instruction in rehabilitation following surgical repair of the anterior cruciate ligament. Incorporation of therapeutic modalities, such as neuromuscular electrical stimulation, biofeedback, or cryotherapy, reflects the reality of clinical care and better represents the complete patient management model than teaching these elements in a separated or disengaged manner. Because therapeutic modalities are too frequently taught in isolation, students receive a limited "one-time" exposure. It is our intention that this book be used not only in the primary therapeutic modalities course but also in courses where therapeutic modalities supplement or complement the interventions taught in those content-specific course areas, such as orthopedics, neurological rehabilitation, and so on.

At risk is clinical competency when therapeutic modalities are taught in isolation with little to no carry-through in the curriculum to relate or connect therapeutic modalities to those conditions or impairments for which they are advocated. It is our suggestion that the content of this book be used throughout the curriculum where therapeutic modalities offer adjunctive interventions. By maintaining continuity throughout the curriculum between therapeutic modalities and the specific

clinical areas of their supported application, a curricular thread is created, thereby improving clinical decision-making skills and competency.

The following table represents specific chapter content in this text and the potential curricular areas where use of therapeutic modalities are part of common clinical practice. It is our belief that the content of this text may be threaded or cross-referenced across the curriculum to reinforce the supplementary role that is offered by therapeutic modalities.

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Chapter Content and Related Curricular Areas

This table shows how therapeutic modalities may be strategically threaded throughout a curriculum to complement the primary content of each section area. Doing so reinforces the role and use of therapeutic modalities as therapies aimed at augmenting or complementing additional areas that are part of the complete patient care plan.

Therapeutic modalities for:	Cross-referenced courses:
Flexibility/ROM Thermotherapy (Chapter 3) Ultrasound (Chapter 4) Hydrotherapy (Chapter 5) Traction (Chapter 7) Electrotherapy (Chapter 12)	Orthopedics, Neurological Rehabilitation, Therapeutic Exercise, Kinesiology, Integumentary for US and Hydrotherapy
Strengthening NMES (Chapters 9, 10, 13, 14) Biofeedback (Chapter 13)	Orthopedics, Therapeutic Exercise, Exercise Science/Physiology, Neurological Rehabilitation
Neuromuscular Reeducation FES (Chapters 9, 10, 14) Biofeedback (Chapters 13, 14)	Neurological Rehabilitation, Orthopedics
Pain Modulation Cryotherapy and Thermotherapy (Chapters 2, 3) Ultrasound (Chapter 4) Hydrotherapy (Chapter 5) LASER and Diathermy (Chapter 6) Traction (Chapter 7) Electrotherapy (Chapters 9, 10, 11) Alternative Modalities (Chapter 16)	Orthopedics, Integumentary
Tissue Healing Electrotherapy (Chapters 10, 15) Hydrotherapy (Chapter 5) Cryotherapy and Thermotherapy (Chapters 2, 3) Ultrasound (Chapter 4) Compression (Chapter 8) Alternative Modalities (Chapter 16)	Orthopedics, Neurological Rehabilitation, Integumentary, Pharmacology
Neurodiagnostics EMG and NCV (Chapter 17)	Orthopedics, Neurological Rehabilitation

SECTION I

Introduction to Therapeutic Modalities

CHAPTER 1

Therapeutic Modalities Past, Present, and Future

Their Role in the Patient Care Management Model

CHAPTER 1

THERAPEUTIC MODALITIES PAST, PRESENT, AND FUTURE

Their Role in the Patient Care Management Model

James W. Bellew, PT, EdD

THERAPEUTIC MODALITIES: ROLES IN REHABILITATION

Therapeutic modalities represent the administration of thermal, mechanical, electromagnetic, and light energies for a specific therapeutic effect; for example, to decrease pain, increase range of motion (ROM), improve tissue healing, or improve muscle activation. The terms *therapeutic modalities* and *physical agents* are often used interchangeably to describe a wide array of treatments and interventions that provide a variety of therapeutic benefits. The term *physical agents* reflects the use of physical energies—such as thermal, mechanical, electromagnetic, or light—but fails to include the purpose or intention of their application. The term *therapeutic modalities,* as used throughout this text, more appropriately reflects the ability of these interventions to provide therapeutic benefits.

Therapeutic modalities have long been, presently are, and will continue to be a part of rehabilitation and are used to complement other elements of the more comprehensive patient care plan, such as therapeutic exercise (e.g., strengthening, stretching, neuromuscular reeducation, balance), manual therapy (e.g., joint and tissue mobilization, manipulation), and patient education (e.g., body mechanics, postural retraining, home exercise program, risk reduction). Cold therapy and compression may be used in the early phases of rehabilitation to limit swelling and pain that a patient may experience following acute injury or surgery. Continuous ultrasound or other heat therapy may be applied to improve elasticity of ligaments or joint capsular structures before beginning ROM activities in a patient who has deficient ROM. Electrical stimulation may be used to increase

activation and facilitate volitional recruitment of skeletal muscle until the patient can effectively contract the muscle and begin additional activities. These examples reflect the complementary use of modalities to achieve clinical goals. Because the effectiveness of these treatments may vary from patient to patient, the practitioner is challenged to identify those patients who are more likely to respond to a specific intervention. In this manner, the practitioner must consider or judge the probability or likelihood that a given intervention will help a particular patient. These decisions and others represent the basis of *clinical decision-making*. Competency with clinical decision-making is the basis for effective patient outcomes and attainment of goals.

Therefore, clinical decision-making can be thought of as the process of using information, experience, and judgments to decide which clinical interventions will most likely improve the problems identified in the examination. The bottom line is this: When identifying and establishing an intervention plan, the focus should be on selecting interventions that will most likely achieve positive results or outcomes—both quantitative and qualitative. When judiciously selected and applied, therapeutic modalities may play a significant role in successful patient care.

Key Point! In 2014, the American Physical Therapy Association began recommending use of the term "biophysical agents" to collectively refer to physical agents and modalities. We, the editors of this text, support this recommendation and recognize the advancements of our profession in better delineating and understanding the role of biophysical agents in rehabilitation. To maintain consistency with the title of the previous five editions of this text, the term "modalities" will be used interchangeably with biophysical agents throughout this edition. As the transition to the term biophysical agents progresses, future editions of this text will integrate such use.

Clinical decision-making—regarding the best modality to use, when to use it, and which patients are most likely to respond—remains relevant, but more critical and incumbent upon the practitioner is the challenge to use current best evidence to better define the therapeutic dose for a given treatment. This point was well articulated by Meryl Gersh, a professor of therapeutic modalities, who stated, "We would not expect a subclinical dose of antibiotics to successfully treat an infection. So why do we continue to apply TENS at sensory thresholds or a strong, comfortable level of sensation when the evidence suggests that stronger intensities applied for longer durations result in significant analgesia?"

Key Point! The current challenge when using therapeutic modalities is to identify and establish consensus for optimal doses and treatment procedures.

As practitioners, we are often challenged by patients who have multiple impairments and dysfunctions. Our role as experts in rehabilitation is to identify and skillfully provide interventions to address these impairments, thus providing optimal recovery of function. Even when facing a seemingly uncomplicated patient case whose therapy plan is clear, the emergence of confounding variables often impacts the execution of the initial plan of care. Imagine this happening during therapy: Your patient, who has decreased ROM and strength, is unable to complete the appropriate therapeutic activities to address ROM and strength because of underlying pain, or your patient has significant swelling of the knee and is unable to effectively contract the quadriceps secondary to effusion inhibition. Although increasing ROM and strength or volitional muscle recruitment are obvious goals in the plan of care, attention may first need to be given to decreasing the pain or reducing the effusion to help the patient continue with the therapy plan.

In their assessment of how therapeutic modalities affect muscle inhibition following knee joint effusion, Hopkins et al[1] reported that effusion-induced inhibition of the quadriceps was temporarily suspended with application of cold or transcutaneous electrical nerve stimulation (TENS), noting a near complete reversal of quadriceps inhibition. This finding provides a rationale for using and considering therapeutic modalities as complements to the therapy plan.

Modalities as Part of the Comprehensive Plan

Therapeutic modalities have long been used in rehabilitation, and history of their use is well documented.

Although the use of therapeutic modalities has varied over the years, their application remains pervasive in many areas of clinical practice across several professions. In early 2014, the Centers for Medicare and Medicaid Services (CMS) released data on Medicare payments for services provided in 2012. Electrical stimulation (unattended) and ultrasound ranked sixth and eighth, respectively, among the top 10 procedures in total Medicare payments to providers of physical medicine and rehabilitation services in 2012 (available at www.healthdata.gov). With this sustained usage has come greater clinical interest, more research, and evidence of the effects of modalities, yet much regarding their use remains poorly agreed upon, ill-communicated, and even less accepted by some.

With advancing technology and scientific discovery has come the evolution and emergence of newer modalities that add to the spectrum of interventional strategies and that enhance their role in rehabilitation. Use of therapeutic modalities has been and will remain a cornerstone of rehabilitation for joint and soft tissue injury, acute and chronic pain, and impaired muscle function. Whether used only during specific phases of rehabilitation or throughout the entire rehab program, therapeutic modalities represent a group of interventions that are adjunctive components of a more comprehensive therapy plan. Figure 1-1 depicts the complementary role therapeutic modalities play in the complete intervention plan.

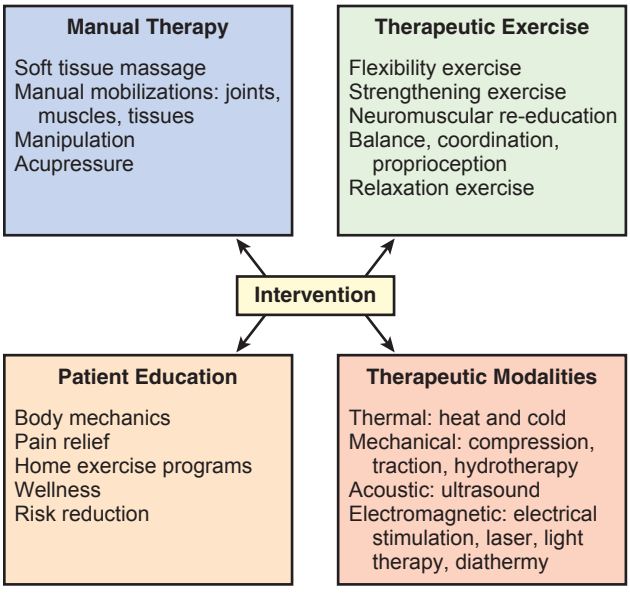

Fig 1 ■ 1 Therapeutic modalities represent a diverse group of interventions that add to and complement other therapies that are part of the comprehensive rehabilitation plan.

Clinicians should review current evidence when therapeutic modalities are considered as adjuncts for an intervention plan. Many techniques in common use have not been studied, which has led to scientific inquiry addressing the efficacy of many therapeutic modalities. However, it should be noted that many studies examining the effectiveness of therapeutic modalities often assess their efficacy when used alone or in isolation—separate from and counter to the supplementary role that the editors of this text and the American Physical Therapy Association (APTA) advocate.

Key Point! In 2014, the APTA Choosing Wisely campaign specifically addressed the use of therapeutic modalities, stating that clinicians should not "employ passive physical agents except when necessary to facilitate participation in an active treatment program." This followed their earlier position statement that "without documentation that justifies the necessity of the exclusive use of physical agents, the use of physical agents, in the absence of other skilled therapeutic or educational intervention, should not be considered physical therapy."[2] These statements reflect the standpoint from which we, the authors of this text, attempt to present the use and administration of and evidence for therapeutic modalities as complementary, not stand-alone, therapies used as part of the complete patient care plan.

TYPES OF THERAPEUTIC MODALITIES

Therapeutic modalities are generally categorized as *thermal* (heat and cold), *electromagnetic* (electrotherapy, diathermy, ultraviolet, and infrared light), or *mechanical* (traction and compression). These modalities are used to increase the probability of a specific therapeutic effect (e.g., decreased pain, increased ROM, tissue healing, or improved muscle recruitment). Therapeutic modalities may be procedural, in-clinic interventions, such as ultrasound, or they may be home-based interventions, such as ice packs or continuous, low-level heat wraps, and even electrical stimulation; these serve to enhance additional therapeutic interventions identified in the more extensive plan of care, such as ROM or muscle strengthening.

Key Point! The term *therapeutic modality* can imply a *type* of energy used by the modality, a specific *range* of that energy, or the *method* of application of that modality.

Remember the impairments you found in your evaluation of the patient with a suspected knee injury: decreased ROM, decreased strength, pain, and swelling? These are just a few of the many problems for which therapeutic modalities may be used in conjunction with other interventions. In this manner, therapeutic modalities are used to increase the probability that certain clinical outcomes are realized.

The term *therapeutic modality* can have several meanings that vary based on the context in which it is used. For example, ultrasound represents both a *form* of energy (i.e., sound energy) and a specific *range* of energy (i.e., greater than 20,000 Hz). By convention, ultrasound has come to represent a *method* or means of delivering a therapeutic modality. It is prudent to be as specific as possible regarding the administration of a modality. When applying ultrasound, for example, it is recommended that the specific frequency used (i.e., 1 MHz or 3.3 MHz) be documented in addition to documenting the form of energy applied (i.e., ultrasound). Human hearing can detect sound frequencies ranging from approximately 15,000 to 20,000 Hz. Thus, *ultrasound* is named for the frequency range above human hearing. Ultrasound derives its name because the sound frequency used with therapeutic ultrasound is in the megahertz range, well beyond the 15,000 to 20,000 Hz range the human ear can detect.

Thermal Modalities: Cold and Heat

Cryotherapy

Cryotherapy (i.e., cold therapy) is the use of cold to induce the therapeutic and physiological responses that result from a decrease in tissue temperature. Therapeutic application of cold will result in reduced blood flow and tissue metabolism—physiological responses that decrease bleeding and acute inflammation following injury or tissue disruption. The application of cold also reduces pain, as the threshold for pain perception is elevated, thereby desensitizing peripheral afferent nociceptors.[3] Collectively reducing swelling and pain may permit patients to complete the other components of the therapy plan, again reinforcing the supplementary role of modalities.

Therapeutic cold can be applied using ice, cold water, cold gel-filled packs, or vapocoolant sprays. Cold packs and ice packs are the most common and familiar applications of therapeutic cold (Fig. 1-2). Ice packs can easily be made at home and used as part of the patient's home program. Commercially made cold packs often contain a gel-like substance that allows the cold pack to mold to the affected body part. Cold water may provide therapeutic benefit and may be applied as cool whirlpool, cold water baths, or added to ice packs to create a slushy ice-water mixture that can be molded to the body part. In addition, larger pieces of ice held in the hand may be used to provide an ice massage (Fig. 1-3) or may be used as an "ice pop" (Fig. 1-4). Also used to reduce tissue temperature are topical, or vapocoolant, sprays (such as Spray and Stretch) that result in rapid, superficial, and short-lived tissue cooling by means of evaporation.

Whichever application of therapeutic cold is most appropriate and most effective will depend on several

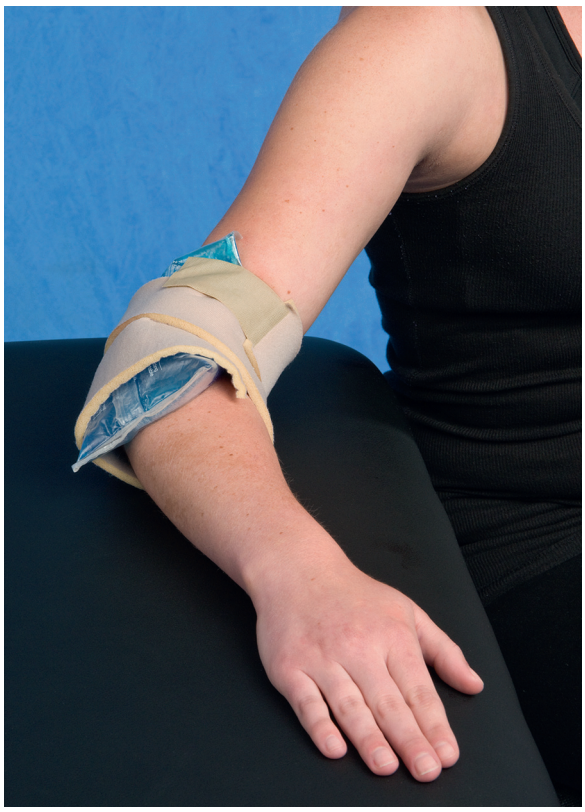

Fig 1 ■2 Cold therapy can be applied by use of gel or ice packs.

Fig 1 ■3 Handheld ice cups provide cold therapy during an ice massage.

factors, including the size of the affected area, the depth of the tissues to be treated, the patient's tolerance to cold, and whether the application will occur in the clinic or at home. More extensive descriptions of cryotherapy and therapeutic use of cold are found in Chapter 2.

Thermotherapy

The therapeutic application of heat provides a variety of benefits that augment the comprehensive therapy plan. Heat may facilitate tissue healing, relax skeletal muscles and decrease spasms, decrease pain, promote an increase in blood flow, and prepare joints, capsular structures, muscles, and other soft tissues for stretching, mobilization, and exercise.[4–7]

Heat can be applied in many forms and through various mediums. Warm water as used in a bath or whirlpool has long been used in rehabilitation and can easily be used at home. Use of heat packs, both in-clinic and at home, have led to the commercial production of single-use heat wraps that can be placed on various body regions (Fig. 1-5). Heat may also be delivered through the use of light, sound, and electromagnetic energies.

Fig 1 ■4 Use of handheld "ice pops" offers quick and efficient cold therapy to many areas where cold packs may be less effective.

Fig 1 ■5 Heat wraps are an easy and convenient source of heat therapy.

The warmth of the sun's rays is a well-known example of heat transfer via ultraviolet energy. Shortwave diathermy (SWD) can provide therapeutic heat through the use of electromagnetic energy, and acoustical or sound energy from ultrasound can be used to increase tissue temperature. Warm water and hot packs are used to raise tissue temperature in the skin and the superficial subcutaneous tissues, whereas continuous-wave ultrasound and SWD are better suited to raising temperature in deeper tissues (up to 5 cm). Selection of the appropriate form of therapeutic heat will depend on several factors, including the area to be treated, the depth of the tissues to be heated, the patient's tolerance to heat, the patient's medical history, and the interventions to be used that are complemented by therapeutic heat. More extensive detail on therapeutic heat and its application are presented in Chapter 3.

Electromagnetic Modalities

Electrotherapy

Electrical currents are used for a wide variety of therapeutic benefits and for an equally wide variety of needs. General therapeutic benefits of electrotherapy may include strengthening and relaxing skeletal muscle, decreasing pain, facilitating neuromuscular reeducation, augmenting ROM, attenuating disuse atrophy, promoting tissue and wound healing, reducing edema, increasing local blood flow, and delivering medicinal ions transdermally. The robust and wide-ranging therapeutic benefits of electrotherapy are derived from the selection of specific parameters of electrical currents such as amplitude, duration, and frequency.

Fundamental to most applications of electrical stimulation is the depolarization, or activation, of peripheral nerves. Use of TENS to decrease perception of pain is one of the most widely recognized applications of electrotherapy, and its clinical effects have been extensively researched.[8–10] Activation of skeletal muscle is used for increasing strength (known as *neuromuscular electrical stimulation,* or NMES) or for restoring or improving use of skeletal muscle during functional activities such as walking (known as *functional electrical stimulation,* or FES; Fig. 1-6). Research continues to delineate the benefits of electrotherapy for actuation of skeletal muscle.[11,12]

Use of certain electrotherapeutic currents have also demonstrated specific and unique effects on cell populations found in wounds and healing tissues.[13–16] Iontophoresis is the use of electrical current to facilitate the delivery of specific drugs and ions to reduce tissue inflammation, decrease local pain, reduce calcium deposits, and reduce scar restrictions. More extensive descriptions of the principles and applications of electrotherapy are presented in Chapters 9 and 10.

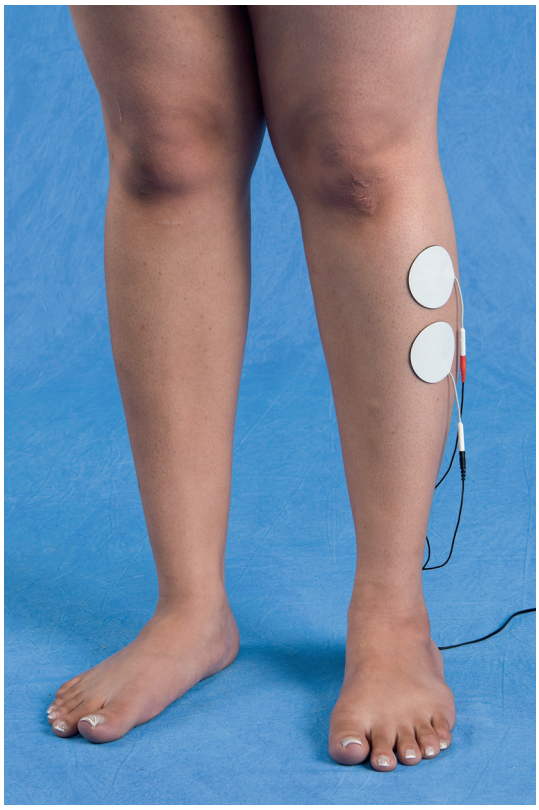

Fig 1 ■6 Electrical stimulation can be used to facilitate functional activities. Stimulation of the anterior tibialis in patients with impaired activation can assist in dorsiflexion of the ankle during gait.

Electromagnetic radiation is used for a variety of therapeutic benefits, both thermal and nonthermal. Classified according to the specific frequency of the electromagnetic wave, therapeutic electromagnetic radiation includes SWD, infrared radiation (IR), and ultraviolet (UVA and UVB) radiation. Continuous SWD and infrared are used to increase tissue temperature. IR increases temperature more in superficial tissues; SWD heats both superficial and deep tissues (Fig. 1-7). The therapeutic benefits of tissue heating complement soft tissue and joint mobilization,[17,18] muscle activation,[19] flexibility,[17] tissue healing,[20] and pain modulation.[19,21]

SWD has primarily been used as a thermal modality. Nonthermal benefits of therapeutic electromagnetic

Fig 1 ■7 Diathermy provides heating of deep tissues and may precede stretching or other range of motion activities.

radiation (e.g., UVA and UVB and pulsed diathermy) remain somewhat unclear but are thought to affect activity at the cellular level, perhaps by altering permeability of the semipermeable phospholipid bilayer, enhancing metabolic activity of the cell and production of adenosine triphosphate (ATP), or altering the activity of membrane-bound cell proteins.[20] More detailed descriptions of the therapeutic benefits and applications of electromagnetic energy are provided in Chapter 6.

Mechanical Modalities

Compression

Force, either of a compressive or distractive nature, may be used for therapeutic benefit during rehabilitation. Compressive force may come from application of wraps, stockings, or garments. It may also come from compression pumps and even from water via the hydrostatic pressure created when a body part is submerged in water. Compression techniques are applied to prevent, attenuate, or reverse swelling that may follow soft tissue injury or compromise the circulatory system, or they may be applied to alter formation of scar tissue during the proliferation and maturation phase of scarring.

The principal mechanism underlying the use of compression to manage edema is applying external compression on the body or body part to increase hydrostatic pressure in the interstitial space. This directs counterpressure at the outflow of fluid from the compromised vessels, thereby reducing the accumulation of fluid in the interstitial space. Compression may also be used during the formation and modeling of scar tissue (e.g., following burn injury) to minimize scar formation and reduce hypertrophic scarring. Unlike collagen synthesis, which requires oxygen, collagen lysis does not require oxygen; therefore, compression can be used to limit scar formation while not affecting scar lysis[22] (Fig. 1-8).

Traction

Mechanical or manual traction is the application of distractive forces to lessen or reduce compression on a structure and is most commonly associated with spinal traction (Fig. 1-9). By separating or reducing compression of adjacent segments, such as joints, or reducing pressure on anatomical structures, such as nerves, blood vessels, and joint capsules, traction may be used to decrease pain, increase ROM, improve functional ability, increase blood flow, and reduce muscle guarding. Manual therapy, exercises for muscle strengthening and

Fig 1 ■8 Compression can be used to limit or reduce swelling that often follows soft tissue damage.

Fig 1 ■9 Manual or mechanical traction is used to reduce the compression on a structure such as a joint, nerve, or tissue. Both clinical and home-based forms of traction are used for therapeutic benefit.

retraining, and neural mobilization are often incorporated in conjunction with traction as part of a patient's care plan.[23–26] Devices are available that allow the patient to perform traction at home as part of a comprehensive rehabilitation program. Chapter 7 details the therapeutic benefits and clinical application of traction, along with the controversies of proposed effects of these techniques.

CLINICAL APPLICATIONS OF THERAPEUTIC MODALITIES

Modulation of Pain

Pain may be the most common symptom that leads patients to seek medical intervention. The unique experiences of pain among individuals make this a challenging alteration in body function to manage. The neurobiophysiology of pain generation, transmission, and perception is well described in other sources, and all academic programs in rehabilitation sciences contain curricular content that describes pain.[27,28]

Early discussions of pain modulation were centered on interrupting the ascending pathways of pain (i.e., blocking the transmission of pain along the nerve pathways to more central centers). Widely recognized as the "gate control" theory of pain, this theory described a relationship between painful sensory input carried by small myelinated A-delta and unmyelinated C fibers versus larger diameter and myelinated A-beta nerve fibers.[29] Noxious stimuli carried by A-delta and C fibers are blocked by sensory input along A-beta fibers. Logically, then, efforts to treat pain are often directed at stimulating the large A-beta fibers through various means. Electrical stimulation targeting large afferent nerve fibers is common in rehabilitation, as is the use of ultrasound, cold, heat, diathermy, and other treatments to decrease or modulate noxious sensory input.

Understanding the neurobiophysiology of pain generation, transmission, and perception has grown immensely since the origination of the gate control theory. Chapter 11 addresses the use of electrical stimulation for pain modulation. For a more detailed description of the neurobiophysiology of pain, the reader is directed to other resources.[28] Attention given to the gate control theory of pain in the late 1960s and early 1970s spurred tremendous growth and development of handheld electrical stimulators designed to provide electrical stimuli to A-beta fibers. This period is considered the birth of TENS and other devices to deliver such currents.

Modulating pain is undoubtedly a central focus of rehabilitation, both in the initial stages and throughout the therapy plan. Because pain may limit or even preclude rehabilitative efforts to restore or increase function, attempts to decrease pain often coincide with or even precede efforts toward restoring function. Following the initial injury, for example, soft tissue insult may result in a cascade of inflammatory and reparative physiological events manifesting in pain. Swelling secondary to vascular damage may compress nearby structures, and chemical irritants associated with injury (e.g., bradykinin, PGE1, PGE2) may be released; both of these result in the generation and transmission of pain. Use of therapeutic modalities, such as cold and compression in the initial stages following injury, can reduce swelling and limit production and accumulation of pain-associated chemicals, thereby reducing the patient's perception of pain. This initial reduction of pain can then allow the patient to initiate activities as part of the larger therapy plan (Fig. 1-10).

In the later stages of rehabilitation, therapeutic modalities such as ultrasound may be used to facilitate formation and organization of collagen when administered right before soft tissue mobilization and flexibility exercises. Likewise, muscle weakness and lack of neuromuscular coordination have been associated with dysfunctional movement patterns, and practitioners commonly acknowledge that pain may result from these dysfunctional movement patterns. Electrical stimulation and

Fig 1 ■ 10 Cold therapy is often used during the initial stages of injury to decrease swelling and pain. The reduction of pain and swelling may allow the completion of other activities of the rehab plan.

electromyographic biofeedback can be used to increase muscular strength and coordination, thus addressing the underlying factors related to the movement dysfunction.

To reduce pain, therapeutic modalities may act locally at the site of injury and inflammation to limit the local chemical irritants; this positively impacts the perception of pain by reducing or attenuating the initial creation and generation of pain. (The local effects of therapeutic modalities on tissue's response to injury are addressed in chapters throughout this text and are a strong focus of Chapter 11.) The activity and direction of the migration of specific cells associated with the healing response of tissues, such as neutrophils and macrophages, can be influenced by applying therapeutic modalities such as electrical stimulation. This is further evidence of the enhanced effect on healing that can be harnessed with the use of therapeutic modalities.[15,16,30]

Key Point! Modalities are used to improve or ameliorate alterations in body function such as loss of ROM, pain, and tissue damage.

Alteration of Skeletal Muscle Performance: Facilitation and Inhibition

Therapeutic modalities can be used both directly and indirectly to influence the activity and performance of skeletal muscle to increase or decrease levels of muscle activation for therapeutic benefit. Direct applications of therapeutic modalities to facilitate skeletal muscle performance may occur, for example, by using electrical stimulation to depolarize peripheral nerves to recruit more motor units. A patient with decreased ability to contract the quadriceps after knee surgery may demonstrate increased muscle recruitment following application of electrical stimulation.

NMES and FES are used to increase strength, endurance, and functional use of skeletal muscle for a variety of therapeutic purposes. More recent evidence shows that NMES directly increases the volume or total number of motor units recruited and the duration those motor units are activated; these are both fundamental to the positive adaptations underlying gains in strength seen with NMES.[31] Facilitation of skeletal muscle in patients with compromised ability to activate specific muscles or muscle groups can be used to assist in functional activities such as retraining gait, increasing function of the upper extremity and hand, improving ROM, decreasing spasticity, and exercising to prevent muscle atrophy, cardiorespiratory decline, and bone degradation[32–34] (Fig. 1-11).

Modalities such as heat, cold, or electrical stimulation may also be used to directly inhibit or decrease skeletal muscle activity. By decreasing motor nerve conduction velocity and sympathetic activity in the injured muscles, modalities can play a large role in rehabilitating skeletal muscle. For example, a patient with hyperactivity of skeletal muscle following acute trauma from a whiplash injury may benefit from application of electrical stimulation to decrease muscular activity in the involved muscles, thus permitting ROM activities (Fig. 1-12).

By decreasing pain, therapeutic modalities may act indirectly on muscle and result in increased muscle performance. For example, a patient with subacute lumbar radiculopathy (e.g., low back injury with radiating pain

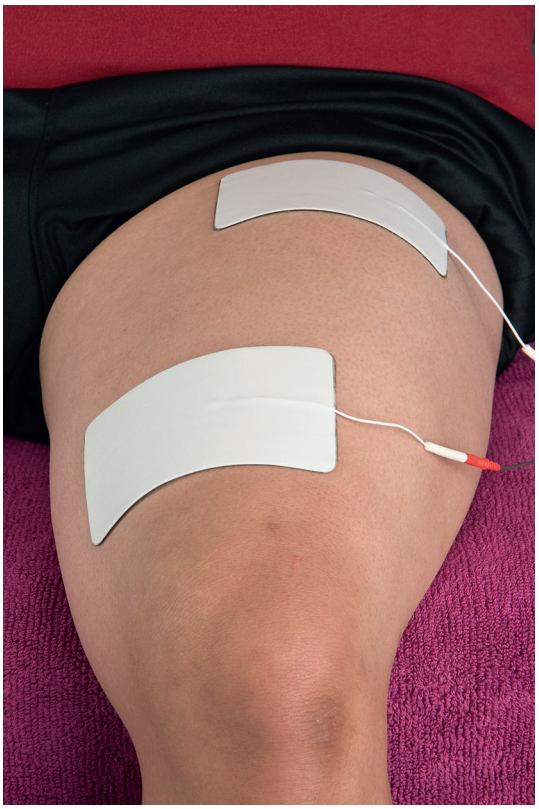

Fig 1 ■ 11 Neuromuscular electrical stimulation is used to increase strength, prevent or limit atrophy, and reeducate muscles. Electrical stimulation alters the manner in which muscle is activated, providing a stimulus for positive adaptation.

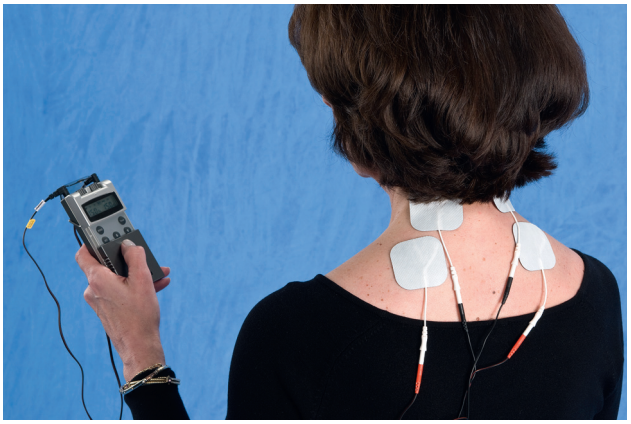

Fig 1 ■ 12 Electrical stimulation can be used to decrease the excitability or hyperactivity of skeletal muscle following injury.

due to nerve irritation) resulting from a lifting injury may report decreased pain after administration of cold. This may allow the completion of stabilization exercises that the patient was otherwise unable to perform because of pain. Cold application may also act indirectly on muscle activity by decreasing the synaptic activity of peripheral sensory nerves. This, in turn, may elevate the pain threshold, potentially allowing improved skeletal muscle activation secondary to decreased pain. Likewise, altering either blood flow to the muscle or cell membrane transport in muscle tissues via ultrasound, diathermy, or cold or heat modalities may indirectly facilitate improved performance of skeletal muscle.

Key Point! If you cannot explain the physiological and clinical reasoning for using the therapeutic modality you select, then perhaps you should not be using the technique!

Decreasing Inflammation and Facilitating Tissue Healing

Use of therapeutic modalities often is recommended following acute injury and tissue damage. The primary goals at this point are to minimize inflammation and promote the most expedient and effective healing process. Although it is critical to keep in mind that the *inflammatory stage* is the beginning of the process of tissue healing, use of therapeutic modalities can facilitate and augment progression through the stages of healing so as to provide expedient yet effective healing (Table 1-1).

TABLE 1–1. Normal Stages, Phases, and Events of Inflammation and Repair

Stage	Phase	Physiological Events
I	Inflammatory	• Vasoconstriction • Vasodilation • Hemostasis/clot formation • Cell-mediated phagocytosis
II	Proliferative	• Epithelialization • Collagen production • Closure/contraction • Revascularization
III	Maturation	• Collagen balance: synthesis/lysis • Collagen remodeling

Cold therapy has long been a standard treatment for the inflammation that occurs in the first several days following acute injury. This can decrease local blood flow and metabolic activity in the involved tissues, which provides support for using cold modalities in the period following injury when vascular increases in permeability and the resultant swelling are likely. The analgesic effect of cold also offers palliative benefit to the patient after injury. Use of electrical stimulation or compression to minimize leakage of large blood proteins from damaged vessels and to limit agglutination of proteins in the interstitial space can minimize duration and residual effects of the inflammatory phase.

The *proliferation stage* follows the onset of the acute inflammatory stage and is characterized by the production, organization, and infiltration of collagen at the site of tissue damage. Collagen serves to repair damaged tissue and represents the first stages in the formation of new tissue. Cells involved in the healing process, such as macrophages and neutrophils, demonstrate unique and specific behaviors as they migrate to the site of tissue repair.[13,16] Blood-borne proteins, such as fibrinogen and fibronectin, aggregate in the involved area, acting to reinforce collagen in the injured tissue. Modalities, such as superficial heat, ultrasound, and diathermy, can facilitate and enhance local blood flow and cellular activity, thereby promoting the proliferation or repair of the damaged tissue.

The third and final stage of tissue healing is the *maturation stage*. This stage is characterized by the modeling, remodeling, organization, and maturation of collagen into new tissue and may last from several days to years. Therapeutic modalities, such as ultrasound, are commonly used to influence the maturation and organization of collagen. Heating collagen tissue by applying ultrasound complements stretching and mobilization of newly formed, maturing collagen. This heat-and-stretch aids in restoring functional integrity to the newly formed and repaired tissue.

Increasing Tissue Extensibility: Flexibility and Range of Motion

Efficiency of functional movement depends on flexibility, and because disuse, immobilization, and detraining can negatively impact flexibility, rehabilitation often focuses on maintaining and restoring flexibility. Flexibility in tissue is largely related to the amount, organization, and extensibility of collagen—the primary protein imparting integrity to connective tissue.[35] Decreased extensibility and organization of collagen can lead to decreased flexibility and can therefore impair function. This decreased extensibility may persist and perhaps worsen unless the tissues can be exercised through full ROM activities.

Intervention aimed at improving or increasing flexibility must address the viscoelastic and remodeling properties of collagen. These properties are enhanced by elevating tissue temperature; therefore, heating of collagen facilitates elongation and deformation of collagen fibers to result in sustained or lasting gains in flexibility. These changes support the rationale for heat as an effective and appropriate modality. Heating delivered to connective tissues by modalities such as hot packs, continuous wave ultrasound, and continuous SWD complement stretching, mobilization, and other techniques and remodel connective tissue (Fig. 1-13). Further description of the use and effect of heat is presented in Chapter 3. Interventions for loss of motion are presented in Chapter 12.

The reparative processes of tissue healing are dependent on the production, organization, and maturation of collagen. Collagen is the most abundant protein in the body and has a tensile strength approaching that of steel. It is collagen that imparts strength to the newly formed tissue.[36,37]

Fig 1 ■ 13 Continuous wave ultrasound and other heat modalities can be used to increase tissue temperature. Decreased pain, increased tissue extensibility, and increased blood flow follow tissue heating and provide therapeutic benefit.

ASSESSING CLINICAL EFFECTIVENESS OF MODALITIES

Use of therapeutic modalities augments other interventions, increasing the probability that the collective effect of the therapies will result in the desired outcomes. Consequently, use of therapeutic modalities has remained a key element of rehabilitation.[38] Of late, however, therapeutic modalities have been scrutinized in regard to outcome measures, the most common being modulation or alleviation of pain.[10,20,39] Although scrutiny and examination of efficacy are warranted for all elements of the intervention plan, much of the scrutiny applied to therapeutic modalities has failed to assess them in their role as complementary interventions. This point is reflected in a 2009 Cochrane Review by Walsh et al[39] that examined and ultimately criticized the efficacy of TENS for treating acute pain. Randomized controlled trials of adults with acute pain (injuries that were less than 12 weeks old) were included only if they examined TENS given as the sole treatment. To assess therapeutic modalities separate from the other interventions they complement is contrary to the position of the American Physical Therapy Association and these authors.

Studies, data, and recent statements have also imposed a negative viewpoint regarding the efficacy of TENS in alleviating pain. These studies and statements have often assessed effectiveness using more quantitative methods, such as pain scales, and have overlooked qualitative measures of improvement in quality of life and functional ability, reduction in the use of pharmacological

agents, or simply patient satisfaction. Examining or assessing the efficacy of therapeutic modalities outside the context of their complementary role assumes that they have the inherent ability to induce the desired effect when used in isolation, which is inconsistent with the fundamental use of therapeutic modalities.

Recent Cochrane Reviews have concluded that studies examining the effectiveness of TENS are often plagued by heterogeneity in design, outcomes, chronic pain conditions, and methodological quality. Reporting of methods and results for analgesic outcomes were largely inconsistent across studies and were generally poor, making meta-analysis infeasible.[9,10]

Examinations of this nature have not demonstrated so much that therapeutic modalities are ineffective but more that research examining the effects may be ineffective and problematic. Effectiveness of therapeutic modalities must be considered in the context of their intended use—as adjuncts to other elements of the therapy plan of care. To examine the efficacy of modalities when used separately from the interventions they supplement is unfair. It also trivializes the adjunctive skill of application and coordination with other therapeutic interventions that skilled practitioners use when selecting and applying therapeutic modalities. Use of therapeutic modalities in unskilled, inexperienced hands and, more importantly, in isolation from other elements of rehabilitation can be compared with placing a scalpel in the hands of a novice versus the hands of a skilled surgeon—the probability of a successful outcome is inherently reduced.

Study of the efficacy of therapeutic modalities has not so much shown lack of efficacy for therapeutic modalities as much as it has shown a lack of quality research performed on therapeutic modalities.

USING THE RIGHT OUTCOME MEASURES

So what can we use as appropriate measures to assess the effectiveness of therapeutic modalities? This can greatly determine the attitudes and beliefs associated with clinical use of these therapies. If we use inappropriate measurements or match techniques with the wrong diagnoses or stages of healing, we are more likely to conclude that the modality is ineffective. If we do use appropriate measures, we are more apt to expand our understanding of when and for whom the modality is most appropriate.

There must also be consideration of whether a specific measure of effectiveness is appropriate for all patients or whether the effectiveness of a modality be measured differently for different patients based on the specific clinical presentation. For example, consider the following two patients: The first patient has cervical pain while sitting at work and the second has pain in the knee following surgery. As noted previously, the amount of pain and the location is often measured and used to assess efficacy of therapeutic modalities. But pain may be the sole dysfunction for one patient whereas for another patient it may be an anticipated consequence of some additional factor such as surgery. For these examples, it must be considered whether measurement of pain in and of itself is the best indicator of effectiveness for a given therapeutic modality. Perhaps the answer is yes for the first patient, where pain is the primary clinical complaint, and no for the second patient, where pain is an expected consequence and part of the rehabilitation process. In other words, the measure used to assess a modality should consider the role, relevance, or significance of the variable to be evaluated.

Academic preparation and experience are fundamental factors related to successful patient management. However, individuality and differences between patients will always influence the probability that a specific intervention will yield effective outcomes. This simply reflects nature and the natural differences among people. The extent to which patient individuality can influence our clinical decision-making is questionable but must be recognized.

The enigmatic nature of pain makes measurement precarious. The effectiveness of interventions to address pain is equally precarious. Pain assessment scales and other pain rating tools are used, and each practitioner likely has a preferred method of assessing pain. However, pain is often assessed in a quantitative manner, such as a 1-to-10 scale or a 10-cm line, and while necessary, these fail to consider qualitative matters such as functional ability with pain and quality of life.

Perhaps then, assessment of clinical effectiveness of therapeutic modalities should be considered in the larger picture of the patient's overall outcome. For example, pain is an expected part of the clinical course for many patients. Simply assessing the effectiveness of modalities for pain during periods when pain is expected to be present or elevated (i.e., in the acute stages of injury) may

yield less favorable attitudes toward modalities than if pain is assessed in terms of the functional improvements that were made when pain was attenuated or decreased, permitting other aspects of the therapy plan.

Practitioners are encouraged to assess the efficacy of therapeutic modalities when used with other components of the more comprehensive therapy plan. Many variables or methods of assessing effectiveness of therapeutic modalities are available. The astute practitioner will select measures that clearly reflect the expected or anticipated physiological effect of a therapeutic modality. Table 1-2 presents a variety of outcome measures associated with the use of modalities and the physiological rationale for their use.

OVERVIEW OF CONTRAINDICATIONS AND PRECAUTIONS

If a technique can have a positive effect, there is also potential for it to cause harm. For example, the correct dosage of aspirin may relieve a headache, but too much may cause gastric bleeding. The same principles of dosage and treatment selection apply to modalities. The practitioner must judge and determine whether to use specific techniques and must consider the probability that the modality will result in a favorable response. In addition, the practitioner must decide if the patient's history or current status presents any factors or risks that may render a specific

TABLE 1–2. Measures Used to Assess Effectiveness of Therapeutic Modalities

Measurement	Clinical Presentation	Therapeutic Modality	Rationale
Girth, circumference, and volumetrics	Swelling	Cryotherapy	Cold can reduce swelling and inflammation.
		Compression	Compression can decrease edema and swelling.
Goniometric measures	Decreased ROM, flexibility	Thermotherapy	Superficial heating of tissue before stretching can increase ROM.
		Diathermy	Continuous wave diathermy can increase tissue temperature to allow for increased elasticity of tissue.
		Ultrasound	Thermal ultrasound can increase tissue temperature to allow for increased elasticity of tissue.
Strength tests (manual muscle testing, dynamometry)	Decreased strength	Neuromuscular electrical stimulation	Electrical stimulation can enhance volitional muscle activation.
		Biofeedback	Biofeedback can augment volitional activation of muscle.
Tests of function (balance, jump height)	Decreased functional ability	Variable	Therapeutic modalities can help increase function.
Tissue healing (closure time, wound depth)	Compromised integumentary	Electrical stimulation	Monophasic current can increase rate of healing.
Pain (visual analog scale)	Pain	Cryotherapy	Application of cold can reduce pain.
		Thermotherapy	Application of heat can reduce pain.
		Ultrasound	Acoustic energy can decrease pain.
		Electrotherapy	Electrical stimulation can attenuate pain.

therapeutic modality harmful or disadvantageous to the patient's well-being.

Contraindications are specific situations in which a drug, procedure, or surgery should not be used because it may be harmful to the patient.[40] More specific to therapeutic modalities, contraindications have been defined as conditions or factors in which application of a modality over a specific location or region of the body could be harmful and thus the modality should not be used at this location/region.[40] Contraindications are against (or *contra-*) the usual indication to use a specific therapeutic modality. This may be due to an increased risk of an adverse effect or undesired outcome such as use of mechanical traction to the cervical spine in a patient with spinal instability or the use of cryotherapy in a patient with compromised circulation in the area to be treated.

Precautions present a somewhat different aspect to clinical decision-making. Although not outright contraindications, precautions reflect situations in which a patient is at some risk of experiencing an adverse event. In this case, treatment may proceed with caution and proactive measures should be taken to mitigate the risk of potential harm. This may include adjustment of treatment parameters such as intensity or frequency of treatment and closer monitoring of patient response to the treatment.[40]

The following list details the most common contraindications or precautions that practitioners are likely to encounter:

- *Active deep vein thrombosis or thrombophlebitis:* Physical energies applied to local areas of thrombosis or thrombophlebitis may dislodge or disrupt a thrombus, leading to blockage or occlusion to vital tissues; thus, most therapeutic modalities are considered a contraindication.
- *Hemorrhagic conditions:* Application of physical energies may result in disruption of platelet plug formation and uncontrolled bleeding; thus, hemorrhagic conditions are considered a contraindication.
- *Compromised, impaired, or diminished sensation:* Safe administration of most therapeutic modalities requires that the patient have the ability to feel the treatment so that proper adjustments in temperature, intensity, time, position, and so on, may be made to allow for optimal therapeutic benefit while minimizing risk for tissue injury. In most cases, compromised or impaired sensation presents a precaution but may become a contraindication in certain patients.
- *Compromised, impaired, or diminished cognition or communication:* Proper and safe administration of therapeutic modalities requires communication and feedback between the patient and the practitioner. An impaired ability to recognize or communicate the associated sensation of many modalities makes diminished cognition or impaired communication a precaution.
- *Electronic implants—pacemakers, cardioverter defibrillators, phrenic nerve stimulators, and pain pumps:* Administration of therapeutic modalities that deliver electrical or electromagnetic energy (i.e., ultrasound, electrical stimulation, and diathermy) near implanted or external electrical devices worn by the patient requires special consideration. The presence of these devices is typically considered a contraindication by most practitioners. This is mainly due to the potential for the energy emitted by the modality to interfere with the functioning of the electronic device. Application of other modalities, such as hot and cold packs, compression, and traction, may be used in patients with electronic implants but should at least be considered precautions.
- *Pregnancy:* Pregnancy is widely considered a contraindication to the use of modalities if the energies delivered may reach the low back, abdominal, and pelvic areas. This is largely due to the potential and unknown effect on fetal development.
- *Presence of malignancy:* Malignancy in the local area of modality application is considered a contraindication primarily if the therapeutic energy delivered has the potential to alter metabolic activity or blood flow in or around the area of the malignant tissue. These factors are associated with accelerated cell growth and are to be avoided because of the risk of proliferating the malignant cells. Although electrical stimulation has been used to manage cancer-related pain, it has generally been used during palliative care in late-stage cancer and evidence is equivocal.[8,40–42]

Clinical Controversy

There continues to be a lack of consensus as to whether a history of cancer versus the presence of cancer (i.e., active malignancy) remains a contraindication for some modality applications. Malignant tissues can metastasize and go undetected for periods of time, so there is no clear answer. Application within the local area remains a contraindication. Thus, there is no point at which a history of cancer should not be considered at least a precaution, if not a contraindication.

CLOSING COMMENTS

This text is written for and directed at those practitioners who recognize the proper role of therapeutic modalities and understand and embrace their role in the larger continuum of the intervention plan. The authors of this text encourage the use and application of therapeutic modalities within the context of their biophysical properties. In this textbook, we address each modality in this manner. It is imperative and mandatory that clinicians recognize how therapeutic modalities can complement their skills in the interventions used in comprehensive patient management. Scrutiny and further examination of therapeutic applications are warranted and encouraged but only when done so in a manner that is consistent with actual clinical use and in a way that considers and incorporates qualitative measures of improvement or effectiveness.

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SECTION II

Types of Modalities

Continued

—cont'd

CHAPTER 9
Foundations of Clinical Electrotherapy

CHAPTER 10
Clinical Electrical Stimulation

CHAPTER 2

COLD THERAPY MODALITIES

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The use of cold as a therapeutic agent has a long history, beginning in Egypt around 2500 BC.[1] However, the use of cold for injury management and rehabilitation did not become prevalent until the 1950s and 1960s.[2–4] Although many technological advances have been made in the realm of therapeutic modalities in the past century, the use of cold (water, ice, or gel) remains one of the most effective and least expensive modes of acute injury and pain management.

Cryotherapy, defined as the use of cold modalities for therapeutic purposes, is used as a first-aid measure after trauma and as an adjunctive tool in the rehabilitation of musculoskeletal and neuromuscular dysfunctions. The basis for cryotherapy is grounded in the physiological responses that occur when tissue temperature is lowered. Cold decreases blood flow and tissue metabolism, thus decreasing bleeding and acute inflammation immediately or soon after injury or surgery. Muscle spasms and tightness from myofascial trigger points can be diminished, allowing for greater ease of motion. Cold can elevate a patient's pain threshold, facilitating ease of exercises with less discomfort. Muscle force production can also be temporarily altered with tissue cooling.

Cold can be easily applied through a variety of means, including cold packs, ice massage, cool baths, cold compression devices, hypercooled air, or vapocoolant sprays. Caution should be taken, however, to avoid undue exposure to cold in persons with cold hypersensitivity, impaired circulation, diminished sensation, or hypertension. This chapter includes discussions on the physical principles, biophysical responses, and clinical applications of cold therapy modalities.

PHYSICAL PRINCIPLES

Cooling is accomplished by removing or abstracting heat from an object rather than by *adding* cold. Therefore, when a therapeutic cooling agent is applied, the temperature of the skin and underlying tissues is lowered by abstracting heat from the body (Fig. 2-1). The principal modes of energy transfer used for therapeutic cooling include conduction, convection, and evaporation (Table 2-1).

Conduction

Conduction is the transfer of heat by direct interaction of the molecules in the warmer area with those in the cooler area.[5] Warmer, rapidly moving particles give up, or transfer, heat to nearby cooler, slower-moving particles. The most common conductive methods of cooling in rehabilitation are placing ice or cold packs over an affected area, immersing a distal extremity in cool or cold water, or applying a cuff filled with ice water (with manual or automatic recirculation of the water) around the affected area. With these cooling agents, the body part comes in direct contact with the cold source, thus making conduction the form of energy transfer by which cryotherapy works.

The magnitude of the temperature change and secondary biophysical alterations depend on several factors

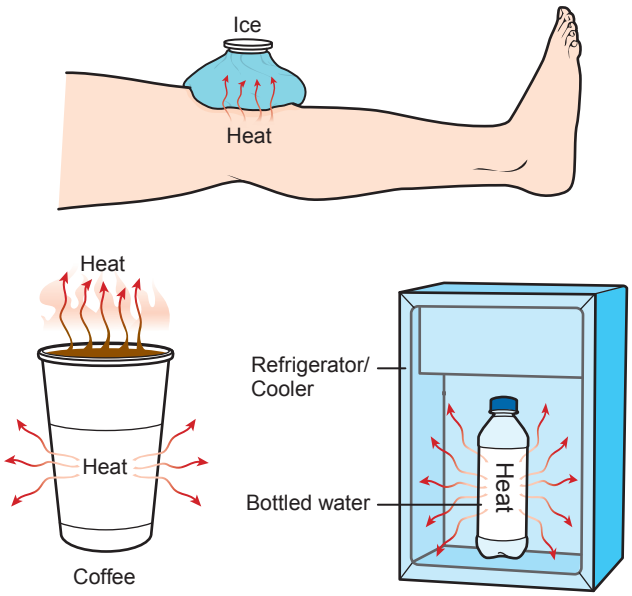

Fig 2 ■ 1 Heat abstraction. All cooling occurs via heat leaving one material and going into another. Cold is never added to something to reduce its temperature.

TABLE 2–1. Methods of Energy Transfer With Cold Modalities

	Conduction	Convection	Evaporation
Cold or ice packs	✓		
Ice massage	✓		
Vapocoolant sprays			✓
Controlled-cold units	✓		
Cool or cold immersion	✓	✓ (with agitation of water via turbines or motion of immersed body part)	

(Box 2-1). The following equation summarizes the rate of heat transfer by conduction:

$$D = \text{Area} \times k \times (T_1 - T_2)/\text{thickness of tissue}$$

D is the rate of heat loss (calories/second). *Area* is the extent of body surface cooled or heated (cm^2), and k is the thermal conductivity of tissues (calories/second/cm^2 × °C/cm^2) (Table 2-2), and T_1 and T_2 are the temperatures of the warm and cool surfaces (°C).

The greater the temperature gradient between the skin and the cooling source, the greater the resulting tissue temperature change may be. For example, after a 15-minute immersion of the forearm in a water bath of 34°F (1°C), subcutaneous tissue temperature dropped by 43°F (24°C).[6] With the same duration and area of immersion at 63°F (17°C), the decrease in temperature

Box 2 ■ 1 Factors Influencing Response to Cold Therapy

- Temperature difference between cold object and soft tissue
- Time of exposure
- Thermal conductivity of area being cooled
- Type and size of cooling agent
- Total body surface area cooled
- Activity level (increased activity → increased circulation → faster rewarming)
- Ability of cooling agent to maintain its temperature

TABLE 2–2. Thermal Conductivities (cal/s/cm² × °C/cm²)

Material/ Tissue	Thermal Conductivity (k)	
Silver	1.01	Good conductor
Aluminum	0.50	
Titanium	0.016	
Ice	0.005	
Water at 69°F (20°C)	0.0014	
Bone	0.0011	
Muscle	0.0011	
Fat	0.0005	
Air at 32°F (0°C)	0.000057	Poor conductor

in the subcutaneous tissue was only 11°F (6°C).[7] (See Box 2-2 for temperature conversion from Celsius to Fahrenheit and from Fahrenheit to Celsius.) Table 2-3 provides some common temperature conversion values for ease of reference.

Thermal conductivity (see Table 2-2) is a measure of the efficiency of a material or tissue to conduct heat. For example, metals are better heat conductors than nonmetals. Tissues with high water content, such as muscle, have better thermal conductivity than adipose tissues. Adipose acts as an insulator, providing resistance to heat transfer (gain or loss), and muscle usually underlies varying depths of adipose tissue. Therefore, the presence of adipose tissue can affect the rate of intramuscular cooling and rewarming.[8–10] Although it takes only 10 minutes to produce a 12.5°F (7°C) decrease in temperature in muscle underlying 1 cm (0.4 in.) of adipose tissue, 60 minutes are required to achieve the same temperature decrease in the presence of 3 to 4 cm (1.2–1.6 in.) of adipose tissue.[11]

Box 2 ▪ 2 Temperature Conversion

F = Fahrenheit temperature
C = Celsius temperature
$F = (9/5 \times C) + 32$
$C = 5/9\ (F - 32)$
Δ in C temperature = Δ in F temperature × 0.556
Δ in F temperature = Δ in C temperature/0.556

Key Point! Thermal conductivity should be considered when cooling areas where cutaneous or subcutaneous metal (such as body piercings, shrapnel, or joint replacements) is present, and also over scars that have altered circulatory response.

In addition to consideration of thermal conductivity, it is important to understand the effects of cold on blood flow to an injured area and why a cooled area can take such a long time to return to the precooled temperature. In fact, it can take a cooled area longer than a heated area to return to resting values. Arterial blood coming from the body core is warmer than the venous blood returning from the periphery. Arteries and veins course through the body in juxtaposition to each other. Normally, as warm blood flows toward the periphery, it passes by the cooler blood in veins that lie right next to the arteries. There is a countercurrent heat exchange between the warmer arterial blood and the cooler venous blood.

After a body area is heated, arteriole vasodilation allows cooler blood to rush into the area and carry away the heat. Cold causes the opposite effect—a vasoconstriction of arterioles—resulting in a decrease in the amount of warm blood flowing into the area. Thus, countercurrent heat exchange is reduced, and the area may not rapidly rewarm (Fig. 2-2). For example, when ice packs were applied around a dog's knee for 1 hour, it took more than 60 minutes after removal of the cold source before tissue temperature returned to resting values.[12]

When hot packs were applied for the same duration, temperatures rose and peaked within 15 minutes and then began to decline. The elevated temperature from

TABLE 2–3. Values for Temperature Conversion*

°C	−18	0	5	10	15	20	25	30	35	37	40
°F	0	32	41	50	59	69	77	86	95	98.6	104

*Some values are rounded off to the nearest whole integer.

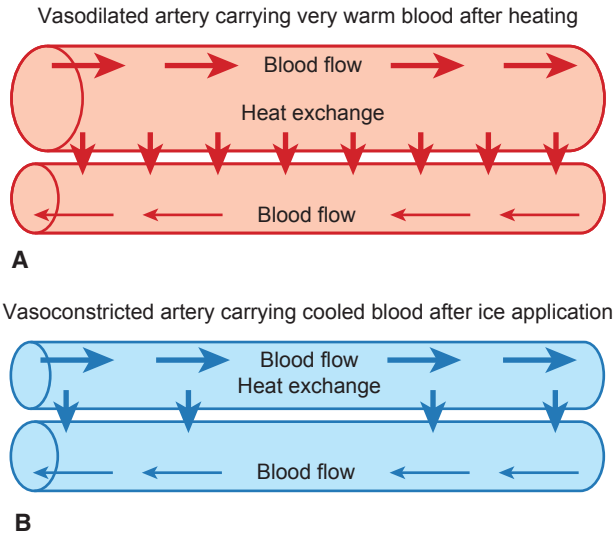

Fig 2 ■2 Heat exchange between an artery and a vein with (A) a vasodilated artery carrying very warm blood following a period of heating and (B) a vasoconstricted artery carrying cooler blood following a period of ice application.

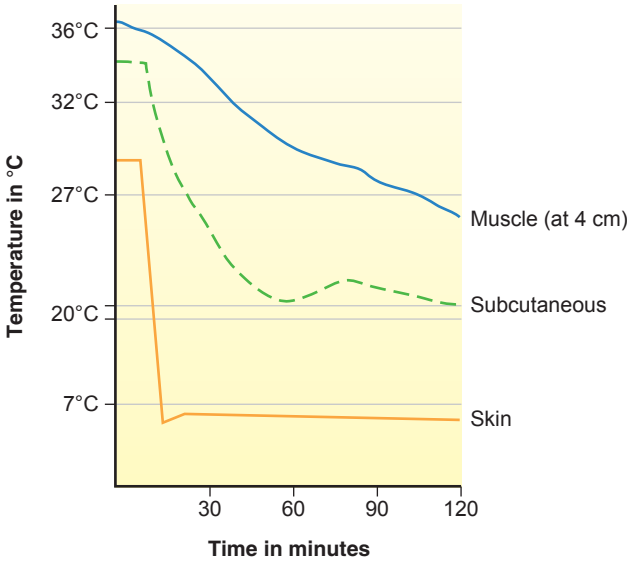

Fig 2 ■3 Temperature changes during ice-pack application to the calf. *(Adapted from Bierman W, Friedlander M. The penetrative effect of cold.* Arch Phys Ther. *1940;21:585.)*

hot pack application caused vasodilation, allowing cooler blood to flow into the area, dissipating heat. As long as the heat added was greater than that being carried away, the temperature remained elevated. After the heat source was removed, heat was rapidly lost by convection and radiation. Clinically, this implies that any examination or intervention that challenges a patient's muscle force generation should be avoided for a longer period of time after the application of cold versus an application of heat for a similar duration.

In another study, intramuscular temperature of the gastrocnemius muscle remained lowered for at least 3 hours after 20 minutes of cold baths at 50°F (10°C), for at least 4 hours after a 30-minute cold bath at the same temperature,[13] and for at least 1.75 hours after 20 minutes of ice packs.[14] The patient's level of activity can also influence the return of temperature to precooling levels. Exercise performed after cooling will increase blood flow to the area, resulting in a faster rate of rewarming.[15]

Cryotherapy is used to lower the temperature of subcutaneous tissues, muscles, and joints for pain and edema control. Because cold modalities can lower temperatures in these tissues, the time of exposure and the presence of dressings or bandages are factors to consider. Changes in skin temperature will occur very rapidly (typically within 1 minute) upon exposure to cold. Deeper tissues require longer duration of cold application to lower the temperature (Fig. 2-3). Muscle temperature at a depth of 4 cm (1.6 in.) can be lowered by an average of 2.2°F (1.2°C) within 5 minutes when cooled with an agent at 50°F (10°C).[10] However, when using ice packs, it can take as long as 30 minutes to lower muscle temperature by 6.3°F (3.5°C) at a depth of 4 cm.[16]

Key Point! Lowering skin surface temperature to 56.5°F (13.6°C) is sufficient to produce local analgesia.[17,18] A temperature of 50°F (10°C) can produce a 33% reduction in nerve conduction velocity.[19]

It is important to keep in mind that many of the studies done on tissue temperature change have been done on subjects who did not have an injury or pathology.[11,20] Tissue response to cold in the presence of new epithelialization, granulation, or capillary budding as well as scar formation or impaired circulation can only be estimated. One study demonstrated that 1 hour of cooling with an ice-water/compression device after arthroscopic knee surgery led to an intra-articular temperature that was 12.6°F (7°C) lower than that of a control group that had no cooling.[21] Compared to presurgical baseline intra-articular temperatures, the control group had a mean increase of 9°F (5°C) while the cooled group had a mean decrease of 3.6°F (2°C). This indicates that, at minimum, adding a cold modality after surgery can prevent a rise in intra-articular temperature, which often leads to increased swelling and pain.

Patient comfort should always be considered, regardless of the treatment technique selected. Warren et al[22] demonstrated greater intra-articular temperature reductions at 60 and 90 minutes of ice-bag application to the knee (23°F [12.8°C] and 27°F [15.2°C], respectively) compared to a commercial cold compression device (13°F [7.1°C] and 17.5°F [9.7°C], respectively). However, the ice-bag application was more painful than the cold compression device treatment. Similarly, when a continuous-flow cold device was compared with two intermittent-flow devices (one manually circulated and one automatically circulated), the continuous-flow unit was able to produce lower skin surface temperatures but its use was more painful than that of the intermittent-flow devices.[23] Although a greater cooling effect may be achieved by certain cold modalities, patients may be less apt to use applications that are painful.

The form in which cold therapy is applied (e.g., ice packs, ice massage, cold-water baths, or continuous-flow cold devices) can contribute to the degree of cooling. The magnitude of temperature change will be greater when ice packs are used compared with cold-water baths or frozen gel packs. Greater internal energy is required to melt the ice, which occurs as the solid bonding forces of the ice molecules are broken apart. Energy is first used to change the ice to water before raising the surrounding temperature.[5] Four common cryotherapeutic agents were compared during a 20-minute application to the gastrocnemius muscle. Skin surface temperatures were lowered by 35°F (19.6°C) with crushed ice, 30.6°F (17°C) with ice-water immersion, 26°F (14.6°C) with a bag of frozen peas, and 23.5°F (13°C) with a cold gel pack.[24] In another study, the use of ice massage was compared with that of cubed-ice bags during a 15-minute application. The ice massage reduced the intramuscular temperature by 7.7°F (4.3°C), while the ice bags reduced the temperature by 4.1°F (2.3°C).[25]

Key Point! Although ice massage may be able to cool a muscle at a faster rate than an ice bag, the practicality of a prolonged ice massage may not be feasible because it may take several ice-cup applications to cool the same area that an ice bag can cover. Sustaining pressure and motion of an ice cup for more than 10 minutes may be fatiguing; thus, an extended ice-bag application may be more appropriate.

Convection

Heat abstraction by convection occurs when there is direct contact between the skin and moving fluid particles. The principal method of using convection in cryotherapy is a cold whirlpool whereby water moves over the patient's skin via turbines that circulate the water or by the patient moving the body part within the water. Heat abstraction occurs at a faster rate with convection versus conduction given the same medium (water) at the same initial temperature. This is because new (cooler) molecules are continually introduced to the skin surface when movement is occurring; when no motion occurs, molecules remain in contact with the skin surface and are warmed via conduction. Thus, when a body part is immersed in stationary cold water, the molecules in contact with the skin begin to warm and form a shield around the immersed limb.

Despite the ability of cold whirlpools to effectively cool body areas, they are usually practical only for distal extremities (e.g., elbow, wrist, or ankle) and can be uncomfortable. Cold whirlpools are not recommended in the acute phase of healing because they put the limb in a dependent position, encouraging edema in the distal extremity. However, in later stages of healing, when edema formation is less of a concern, cold whirlpools may be used to reduce pain before performing exercises that improve range of motion (ROM) and activities that increase weight-bearing tolerance.

Key Point! The specific heat of water is several thousand times that of air, and heat loss is 25 times greater in water versus air at a given temperature.[26] The rate of heat loss through convection is evident to most people after spending time in a swimming pool, a lake, or the ocean. Immersion and movement in a cold pool (about 78°F [25.5°C]) causes heat loss much faster than occurs during time spent in the same air temperature. Remember your blue lips and chattering teeth when you did not want to get out of the pool as a child?

Evaporation

Vapocoolant sprays are used for temporary pain relief before stretching muscles with active trigger points or muscles with local spasm. These sprays use evaporation as a

means of energy transfer. Vapocoolant sprays, such as Spray and Stretch and Instant Ice (both a blend of 1,1,1, 3,3-pentafluoropropane and 1,1,1,2-tetrafluoroethane), are nonflammable, liquid aerosol skin refrigerants that are bottled under pressure and are emitted in fine sprays (Spray and Stretch requires a physician's prescription but Instant Ice does not) (Fig. 2-4).

Unlike former types of vapocoolant sprays that contained fluoromethane, both Spray and Stretch and Instant Ice are nonozone depleting. As the liquid leaves the pressurized canister, it begins to evaporate. When this transition occurs, the steam cools and extracts heat upon contact with the skin. The vapocoolant spray feels colder than room-temperature water sprayed on the skin because, like alcohol, it evaporates more quickly than water. This cold sensation serves as a counterirritant stimulus to the thermal afferents that overlie the target muscle, causing a reflexive reduction in motor neuron activity and allowing stretch to occur more easily.[27] The spray is applied with only a few sweeps across the skin. The temperature of the spray upon skin contact is 16°F to –4°F (–9°C to –20°C), depending on the distance of the nozzle from the skin.[28]

Fig 2 ■ 4 Vapocoolant spray. Gebauer's Spray and Stretch. *(Courtesy Gebauer Company, Cleveland, OH.)*

Although skin temperature can drop to about 59°F (15°C) for a few seconds, changes in subcutaneous tissue and muscle temperatures are negligible.[29] Although few studies have been published examining the effects of vapocoolant spray, two studies demonstrated that its use on the posterior aspect of the thigh could improve passive and active ROM (AROM) of the hip with the knee extended. These improvements in ROM, however, were quite small (less than 2°)[30,31] and would not facilitate functional change. Well-designed studies using this technique to reduce pain from trigger points are warranted.

BIOPHYSICAL PRINCIPLES OF TISSUE COOLING

Many of the clinical uses of cold are predicated on the physiological changes resulting from reducing tissue temperature. Cold is used in the management of acute trauma because:

1. The resulting arteriolar vasoconstriction reduces bleeding.
2. The decrease in metabolism and vasoactive agents (e.g., histamine and kinins) reduces inflammation and outward fluid filtration.
3. The pain threshold is elevated.

A reduction in skeletal muscle spasm can be postulated as an interplay of factors, which include a decrease in pain and muscle-spindle afferent sensitivity to discharge. Muscle performance may be temporarily enhanced following short-duration cold, although this effect may reverse when cold is applied for longer durations. Pain and perhaps joint inflammation in certain inflammatory rheumatic diseases can be decreased. However, there may also be an increase in joint stiffness secondary to the effect of cold, which increases tissue viscosity and decreases tissue elasticity. When tissue viscosity is increased and elasticity is decreased, resistance to motion increases.

Key Point! Performing muscle strength assessment after different durations of cold may lead to inaccurate findings: After a cold application of less than 5 minutes, a muscle may produce more force than in its noncooled state,[32] while a longer cold application may reduce a muscle's ability to generate force compared with its noncooled state.[33]

Hemodynamic Effects

When cold is applied, the immediate response is vasoconstriction of the cutaneous blood vessels and reduction in blood flow. The amount of blood flow to an area is inversely proportional to the resistance factors that impede flow. Vessel diameter is the most significant factor relating to blood flow. Any influence that causes vascular smooth muscle to contract will reduce vessel diameter (vasoconstriction). Conversely, when smooth muscle tone decreases, as it does with heating, vessel diameter increases (vasodilation).

Exposure to cold for a short time (15 minutes or less) generally results in vasoconstriction of arterioles and venules. The mechanism of action causing vasoconstriction involves a number of factors, including the direct action of cold on smooth muscle,[34] a reduction in vasodilating neurotransmitters,[35] and a reflex cutaneous vasoconstriction. The viscosity of blood determines, in part, resistance to blood flow. If viscosity increases, so does resistance to blood flow. The increase in blood viscosity resulting from cold exposure contributes to the decrease in blood flow. Figure 2-5 summarizes the effects of cold on microcirculation.

Blood flow to the skin is primarily under neural control and plays an important role in thermoregulation. Vasoconstriction of cutaneous vessels occurs as part of the heat-retention mechanisms of the body.[36] When skin temperature is lowered, cold thermal sensors (free nerve endings) in the skin are stimulated, causing a reflex excitation of sympathetic adrenergic fibers. Increased activity of these fibers causes vasoconstriction. This reflex vasoconstriction can also result in a generalized cutaneous vasoconstriction that, to a lesser extent, may also occur in the contralateral extremity.[37,38] The decrease in cutaneous blood flow is greatest in the area that is directly cooled. For example, cutaneous blood flow in a hand that is cooled in ice water slowed from a resting value of 16 mL/100 mL/min to 2 mL/100 mL/min (Fig. 2-6).[37]

As cooled blood returns to the general circulation, it stimulates the heat conservation area in the preoptic region of the anterior hypothalamus. Stimulation of this area will result in further reflex cutaneous vasoconstriction. If a large area of the body is cooled, shivering will occur as a heat-retaining mechanism.

Decreases in articular blood flow have also been demonstrated after cold application.[39] Ice packs at 32°F (0°C) applied to the knee joints of dogs for 10 minutes resulted in a 56% average decrease in resting intra-articular blood flow. The flow returned to precooled values approximately 25 minutes after the cold was removed.

When tissue temperatures are reduced below 50°F (10°C) for a period of time, a cold-induced vasodilation may follow the initial period of vasoconstriction. This was first discovered in 1930 when Lewis[40] found that, when fingers were immersed in an ice bath, skin temperature decreased dramatically during the first 15 minutes and then cyclically increased and decreased (due to vasodilation and vasoconstriction, respectively). When skin temperature fell below 50°F, it was hypothesized that a neurotransmitter—termed *substance H* (similar in action to histamine)—was released, resulting in arteriolar vasodilation. As warm blood came into the

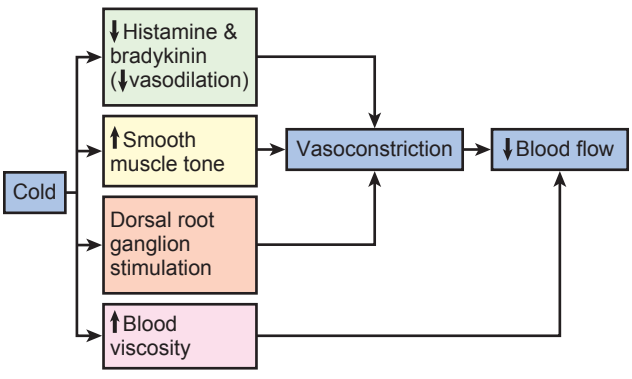

Fig 2 ■ 5 Effects of cold stimulus on local blood flow.

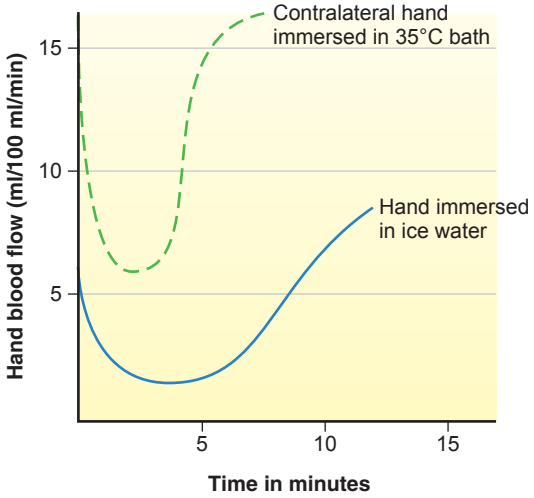

Fig 2 ■ 6 Blood flow to the hands following ice-water immersion of one hand. *(Adapted from Folkow B, et al. Studies on the reaction of the cutaneous vessels to cold exposure.* Acta Physiol Scand. *1963;58:342.)*

area and elevated skin temperature above 50°F, the ice bath was again effective in causing vasoconstriction. This response occurs predominantly in apical areas where arteriovenous anastomoses are located in the skin.[40,41]

Recently, the occurrence of cold-induced vasodilation has been both reproduced and challenged. Immersion of the middle finger for 30 minutes in water at 39°F (4°C) produced a rapid drop in finger pad and nailbed blood flow and temperature followed by an increase in both measures (that remained well below preimmersion values) and then a cycling of small increases and decreases (Fig. 2-7).[42] The response for cold-water immersion appears to be stronger in the fingers than in the toes[43] and does not acclimate with repeated cold immersions.[44] One of the conclusions from Lewis's original work was that cold-induced vasodilation resulted in an increase in temperature, at times up to a sixfold increase, which led clinicians to use cold as a means to increase blood flow. This theory has since been discounted, however, as the sixfold increase was measured from the lowest immersion skin temperature of 35.6°F (2°C) to the highest of 53.6°F (12°C) while the preimmersion skin temperature was 88°F (31°C).[45] Therefore, although relatively small oscillations in blood flow and temperature do seem to occur in response to immersion in water that is 50°F (10°C) or cooler, the overriding clinical effect is a dramatic reduction in both of these values during the time of immersion.

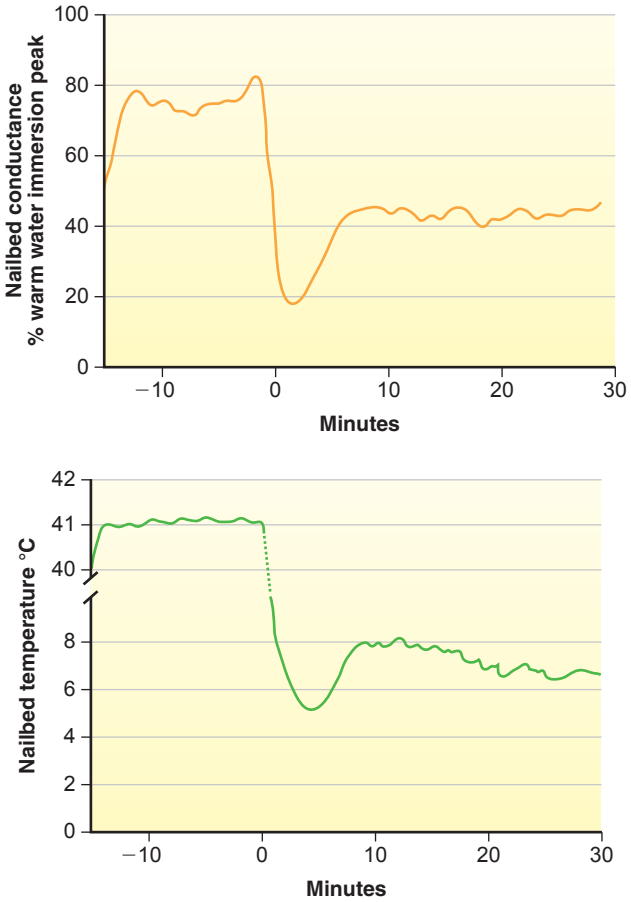

Fig 2 ■ 7 The effects of cold-induced vasodilation on (top) blood flow and (bottom) temperature of the nailbed during 30 minutes of immersion in 30°F (4°C) water. Blood flow was measured as a percentage of maximal blood flow during warm-water immersion. Note the fluctuations (vasodilation and vasoconstriction) in blood flow and temperature that never approach preimmersion values. *(Adapted with permission from O'Brien C. Reproducibility of the cold-induced vasodilation response.* J Appl Physiol. *2005;98:1334.)*

Posttraumatic Edema and Inflammation

For the first 24 to 48 hours after an injury, cold is usually the modality of choice for the following reasons:

1. Fluid filtration into the interstitial tissue may be reduced with cold because of vasoconstriction and prevention of dramatic increases in microvascular permeability.[46]
2. Inflammation and pain may be reduced.
3. Local metabolism is decreased, leading to a reduction in cellular energy demands and, thus, a decrease in secondary hypoxic tissue injury.[45]

The choice of cold has largely been based on empirical evidence. The duration and temperature of the cold exposure can have significant effects on tissue swelling. Some laboratory animal studies are discussed in this section; clinical reports will be presented in the "Clinical Indications for Cold Therapy" section that follows.

In most experiments using animals, trauma was induced through some type of crushing force, resulting in soft tissue damage or fracture. Cold was then applied for varying lengths of time. A summary of findings from studies that measured postinjury edema formation after the use of cold can be found in Table 2-4.[47–49] Figures 2-8 and 2-9 provide a graphic representation of some of these findings. These studies have generally shown a decrease or minimization of swelling when less intense cold was used or when intense cold was applied for several hours or less. Prolonged intense cold may result in increased edema formation.[47,49–51] The increase in edema is likely due to reperfusion of superficial vessels damaged by cold-induced

TABLE 2–4. Results of Selected Studies Using Varying Degrees of Postinjury Cold

Author	Injury Type	Type of Cold Application	Length of Intervention	Temperature/ Other Variables	Results	Clinical Relevance
Matsen et al[47]	Tibial fracture (rabbits)	Cold-water bags	24 hours cold	41°F–59°F (5°C–15°C)	↑ edema vs. controls	Exposure to intense cold for a prolonged period of time immediately postinjury may result in greater edema formation compared with no cold, cold for a shorter period of time, or less-intense cold.
			24 hours cold	68°F–77°F (20°C–25°C)	No difference from controls	
			6 hours cold	50°F (10°C)	No difference from controls	
			24 hours	Room temperature water (controls)	—	
Dolan et al[48] (Fig. 2-8)	Fractured limb (rats)	Cold-water immersion (with or without high-volt electrical stimulation)	3 hours cold	55°F (12.8°C)	↓ edema vs. controls	Application of cold immediately postinjury can reduce swelling compared with no treatment.
			3 hours electrical stimulation		↓ edema vs. controls	
			1 hour cold, then 2 hours electrical stimulation	55°F (12.8°C)	↓ edema vs. controls	
			No treatment (controls)		—	
McMaster & Liddle[49] (Fig. 2-9)	Fractured limb (rabbits)	Cold-water immersion	1 hour	86°F (30°C)	5% ↑ edema	Postinjury application of intense cold, as well as cycling of cold, may lead to greater edema formation.
			1 hour	68°F (20°C)	12% ↑ edema	
			1 hour in/ 1 hour out/ 1 hour in	86°F (30°C)	11% ↑ edema	
			1 hour in/ 1 hour out/ 1 hour in	68°F (20°C)	14% ↑ edema	

ischemia, which has also been shown to occur in noninjured tissues.[52]

Although the presence of edema is of concern in rehabilitation, it is also important to consider how cryotherapy can affect changes in inflammatory exudates. Farry and colleagues[52] used cold to treat experimentally induced radiocarpal ligament sprains in pigs. Crushed-ice packs were applied for 20 minutes followed by a 1-hour rest period, which was followed by another 20 minutes of cold—a protocol similar to typical postinjury methods used by laypersons. Although the cold produced an increase in subcutaneous swelling, there was histological evidence of decreased inflammation. Only 1 of the 20 treated limbs had signs of a pronounced inflammatory response (i.e., numerous polymorphonuclear leukocytes, such as polymorphs, plasma cells, lymphocytes, and fibrinous exudate). All others had either no inflammatory cells or minimal to moderate amounts of polymorphs and lymphocytes.

Alterations in microcirculation in response to cryotherapy were studied by Schaser et al.[35] Continuous cooling at 50°F (10°C) for 6 hours was compared with

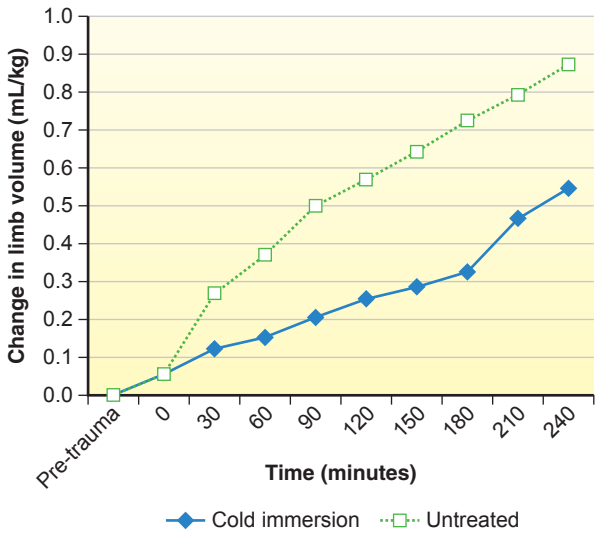

Fig 2 ■8 Changes in volume following trauma in treated (immersion in 55°F [12.8°C]) versus untreated limbs. *(Adapted with permission from Dolan MG, et al. Effects of cool-water immersion and high-voltage electric stimulation for 3 continuous hours on acute edema in rats.* J Athl Train. *2003;38:327.)*

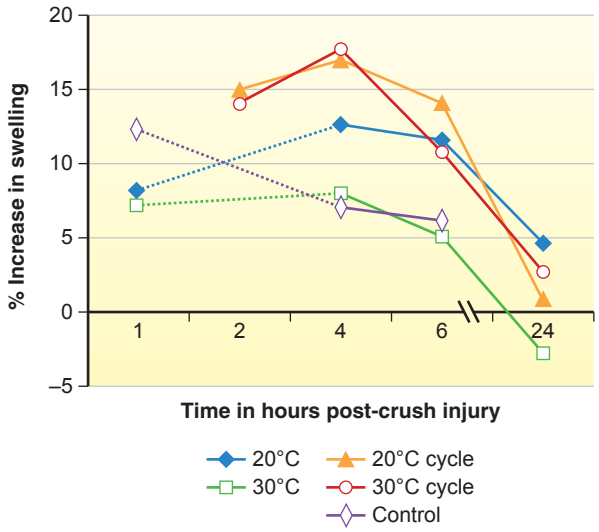

Fig 2 ■9 The effects of cold immersion on postcrush injury swelling in rabbits. Cycling consisted of 1-hour immersion/1 hour no immersion/ 1-hour immersion. Dashed lines indicate areas of missing data points. *(Adapted from McMaster WC, Liddle S. Cryotherapy influence on post-traumatic limb edema.* Clin Orthop Rel Res. *1980;150:283.)*

no cooling immediately after a closed soft tissue injury in experimental rats. Measurements taken 24 hours postinjury showed that the rats receiving the cold treatment demonstrated increased capillary density; narrowed venules with a corresponding increase in erythrocyte velocity; a reduction in adhering leukocytes, macrophages, and neutrophilic granulocytes; and a decrease in intramuscular pressure compared with the control group. Constant cooling for prolonged periods of time, as performed in this study, mimics the use of continuous cooling units that are becoming more prevalent in hospital and clinical settings.

Similar results were found by Lee et al[53] when the effects of 10 minutes of intense cooling (37.4°F [3°C]), moderate cooling (80.6°F [27°C]), and room temperature control (98.6°F [37°C]) on microcirculatory factors were studied in rats immediately following a crush injury. Both the intense and moderate cooling produced a reduction in venule diameter and in leukocyte adhesions, with the intense cooling producing the more dramatic responses. The intense cooling group showed a significant reduction in erythrocyte velocity, whereas the moderate cooling group showed a significant increase in erythrocyte velocity.

The combined results of these two studies[35,53] suggest that immediate cooling of injured tissue can reduce secondary tissue damage by decreasing capillary dysfunction, decreasing venule diameter, and reducing the accumulation of leukocytes. The reduction of intramuscular pressure may also reduce the risk for compartment syndrome.

Key Point! Cold reduces the overall metabolism and oxygen demand of living tissues. When an adult tooth is knocked loose or if an extremity is accidentally amputated, emergency instructions include placing the tooth or lost body part in a container with ice (however, the ice cannot touch the vascular tissues). Cooling allows for longer survival of these tissues during the time when they are separated from their natural blood supply because of the decrease in metabolism and oxygen requirements.

In summary, although postinjury cold application has not been shown to eliminate edema formation, moderate cooling for several hours or less may limit the extent of postinjury swelling. Intense cold, cycled cold, or cold applied for long periods of time may lead to greater edema formation. However, the positive factors of immediate cold application—including a lowered oxygen demand and metabolism,[45,54] reduced leukocyte and macrophage adherence, and decreased intramuscular pressure—outweigh the negative presence of increased edema, especially considering that edema can be

better managed with the addition of compression and elevation.

Peripheral Nerve Effects

Cold can alter the conduction velocity and synaptic activity of peripheral nerves (Box 2-3). If the temperature of a nerve is decreased, there will be a corresponding decrease in sensory and motor conduction velocities or even a failure of the nerve to conduct impulses. Synaptic transmissions can also be impeded or blocked. These factors, in turn, can raise pain tolerance and pain threshold. The quantity of the velocity change elicited depends on the duration and the degree of the temperature alteration.

Isolated cat nerves of various diameters and degrees of myelination were found to have different thresholds or sensitivities to cold stimuli.[55] In the saphenous nerve (afferent), reductions in nerve conduction velocity were observed first in small-diameter myelinated fibers. The fibers least sensitive to cold were small-diameter unmyelinated fibers. Cooling to 53.6°F (12°C) blocked conduction in A fibers, while considerably lower temperatures were required to block C-fiber conduction. Further examination showed that conduction in smaller-diameter A fibers (A-delta fibers) was affected first, and conduction in nerves with the largest diameter (A-alpha fibers) was affected last. When the motor fibers of the sciatic nerve were isolated and cooled, conduction was eliminated in the gamma fibers before the alpha fibers.

Cold applied locally[56,57] or via whole-body immersion[58] can also decrease nerve conduction velocity in humans. When ice packs were applied over the ulnar nerve for 5 minutes, motor conduction velocity decreased by 6% followed by a return to precooling conduction within 15 minutes after ice removal.[57] In a similar study, ice packs were applied over the ulnar nerve for a longer period of time (20 minutes) with a resulting 29.4% decrease in motor conduction velocity. Thirty minutes after the ice packs were removed, the conduction velocity was still 8.3% lower than precooled values.[56] These studies indicate that longer applications of cold can progressively decrease motor nerve conduction velocity. The longer the cold is applied, the longer it takes the nerve to return to precooled conduction velocity.

Ice-pack application to the tibial nerve also resulted in decreased motor conduction velocity. Algafly et al[19] cooled the tibial nerve of healthy subjects to 59°F (15°C) and then to 50°F (10°C) before rewarming back to 59°F. Significant decreases in conduction velocities were found at both temperatures compared to the contralateral tibial nerve, which served as a control. At 59°F, motor conduction velocity had decreased by 17%, and at 50°F the decrease was 33% (Fig. 2-10, top). Pain tolerance and pain threshold were also measured in this study. Both significantly increased at 59°F and 50°F compared to the

Box 2 ■ 3 Effects of Cold on Peripheral Nerves

- Increases threshold for depolarization
- Slows nerve conduction velocity
- Extreme cold can block nerve conduction

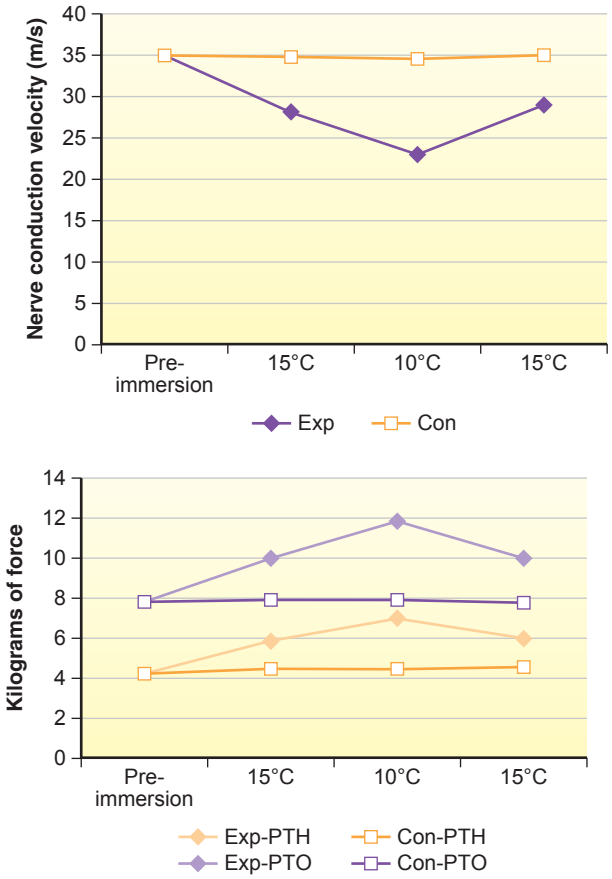

Fig 2 ■ 10 Changes in (top) nerve conduction velocity and (bottom) pain threshold and pain tolerance at skin temperatures of 59°F (15°C) and 50°F (10°C) compared with controls with no cooling. *Exp* = Experimental; *Con* = Control. *(Adapted with permission from Algafly AA, George KP. The effect of cryotherapy on nerve conduction velocity, pain threshold and pain tolerance.* Br J Sports Med. *2007;41:366.)*

control site. Again, findings at 50°F significantly differed from 59°F (Fig. 2-10, bottom). Results of this study indicate that application of cold at temperatures between 50°F and 59°F is effective in slowing motor nerve conduction and increasing the sensory stimulus needed to perceive pain.

Key Point! Although the effects of decreased or blocked nerve conduction velocity in sensory afferent nerves is desirable for assistance with pain reduction, sensory nerves cannot be cooled selectively. Reduced motor ability should be considered if your patient will be engaging in activity after the application of cold.

Caution should be observed when cold is applied in the area of neural tissue because of the risk of nerve injury or damage. One report described four cases of neurapraxia and one of axonotmesis in young athletes after cryotherapy.[59] In each case, ice packs were applied over a major nerve branch that was superficially located (e.g., over the peroneal nerve at the lateral border of the knee) or around the thigh for up to 2 hours (in the case of axonotmesis). On two occasions, 1 hour of cryotherapy around the knee of a patient who had sustained a hamstring strain was reported to cause axonotmesis of the peroneal nerve.[60]

Muscle Performance Effects

Thermal agents can affect the ability of a muscle to generate tension. The effects of cold applied before muscle contraction or functional activities and cold applied after muscle fatigue have been examined and are discussed in this section.

Isometric force generation of the quadriceps was measured before and after 5 minutes of ice massage to the entire anterior thigh of healthy subjects.[32] After icing, the subjects demonstrated a 2-kg (4.4-lb) increase in isometric force generation compared with precooling values. Because muscle temperature was not expected to be lowered with such a short period of ice massage over the large muscle mass, the increase in force generation may have resulted from increased blood flow to the quadriceps via sympathetic nerve activity changes as well as heightened psychological motivation to perform better posttest. Another explanation for the observed increase could have been the effect of short-duration cold on motor nerve excitability.[61] Facilitation of a single motor unit was seen after 1 to 2 minutes of icing over the biceps brachii muscle of healthy human subjects.[62] Isometric endurance was shown to increase in the elbow flexors after 10 minutes of cooling.[63]

When the duration of cold exposure is lengthened, muscle temperature will decrease. After cold immersion of healthy legs for 30 minutes at 50°F to 53.6°F (10°C to 12°C), Oliver et al[33] found that muscle temperature and plantarflexion strength decreased. This decrease could have been the result of reduced muscle blood flow at these lowered temperatures or from an increase in the viscous properties of the muscle. At 45 minutes postimmersion, plantarflexion strength began to increase over pretreatment values and continued to do so for the next 3 hours (Fig. 2-11).

Although findings from these studies may be helpful in understanding the effects of cold on muscle force generation, the ability of individuals to perform functional

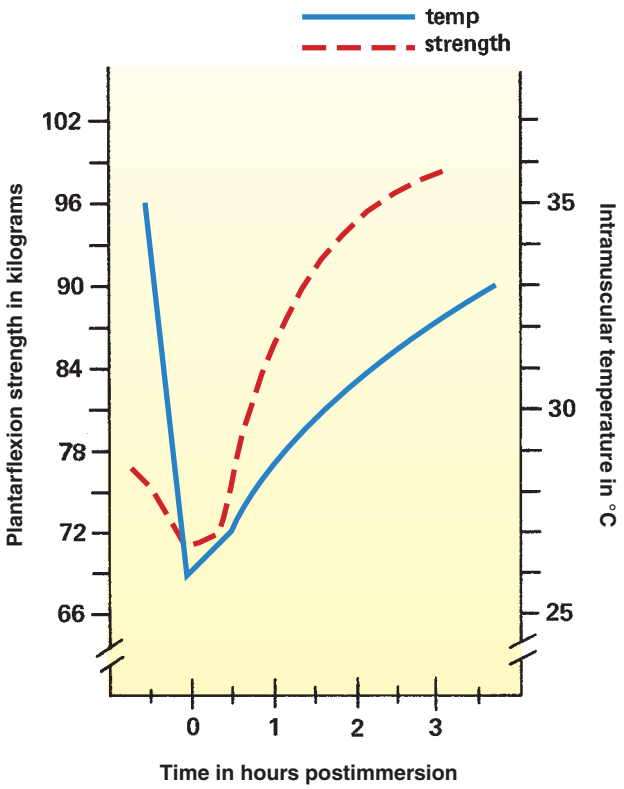

Fig 2 ■ 11 Plantarflexion strength and intramuscular temperature measurements postcold immersion at 50°F (10°C). *(Adapted from Oliver RA, et al. Isometric muscle contraction response during recovery from reduced intramuscular temperature.* Arch Phys Med Rehabil. *1979;60:126.)*

activities after cold application is more clinically relevant. Fischer et al[64] applied ice bags for 3 minutes and 10 minutes to the hamstrings of the dominant leg of various subjects. The 3-minute cooling had no effect on the subjects' shuttle run time, vertical jump distance, or a co-contraction test that involved a timed, resisted lateral stepping activity. The 10-minute cooling, however, resulted in decreased vertical jump distance and slower shuttle run and co-contraction test times. These changes were also seen 20 minutes postcooling.

A similar study was conducted by Patterson et al,[65] who immersed both lower legs of male and female subjects for 20 minutes in a 50°F (10°C) whirlpool. Pre- and posttesting consisted of a vertical jump, a 40-yard dash, and an agility test. Posttesting occurred at 5-minute intervals beginning 2 minutes after cooling and ending 32 minutes after cooling. Vertical jump height was significantly decreased at all posttesting intervals, and both the 40-yard dash and agility test times were significantly slower for at least several posttest intervals. No measure returned to precooling levels by the end of the 32-minute posttest period.

The results of these studies are clinically important in that some form of cold is often applied before exercise to reduce pain. Because muscle performance is negatively affected with cooling for 10 minutes or longer, caution should be taken if strenuous exercise or athletic activities are to be performed after muscular cooling.[66]

Key Point! Because cold can alter muscle force generation, strength assessments on patients during initial and follow-up examinations should be performed either before or several hours after a cold modality has been used.

Cryotherapy may affect proprioception by altering the somatosensory input necessary for reflex integration of neuromuscular control. Therefore, the ability to safely and efficiently perform functional activities can be hindered by lack of joint position sense. Wassinger et al[67] measured active joint position replication, path of joint motion replication, and throwing accuracy before and after a 20-minute ice-bag application to the subjects' dominant shoulder. Although active joint position replication was not affected after the cold application, which supported findings of previous studies,[68,69] both the path of joint replication and throwing accuracy were significantly decreased.

In another study, medial-lateral postural sway increased significantly after recently sprained ankles were immersed in ice water (39°F [4°C]) for 20 minutes.[70] Medial-lateral sway was greater in involved versus uninvolved legs before immersion. This difference increased after immersion. In the affected leg, significant pre- to postimmersion differences were found immediately after immersion and continued to be significantly different up to 20 minutes after the ankle was removed from the cold. Similar results have been found in other studies.[71–73] Douglas et al[71] found that 15 minutes of ice-water immersion had a significantly negative effect on dynamic medial-lateral balance indices compared with a control group.

Cryotherapy applied to the knee joint has been shown to have similar deleterious effects to joint position sense. Oliveira et al[74] demonstrated that a 20-minute ice-bag application to either the quadriceps or the knee joint significantly decreased knee joint position sense in healthy individuals. Surenkok et al[72] found similar results when measuring balance after a 30-minute cold gel pack application to the knee. Single-leg balance was significantly decreased immediately after removing the gel pack. However, 5 minutes after the cold was removed, balance returned to and then surpassed normal levels.

Although the aforementioned studies indicate that proprioception may be hindered for short periods of time after cryotherapy, Berg et al[75] found that application of an ice pack to the lateral ankle for 20 minutes did not alter peroneal reaction time when a sudden inversion force was introduced. Peroneal reaction time and peroneal muscle activity did not differ between cold and control sessions at 0, 15, and 30 minutes, postcold application. Similar results were found in a study conducted by Cordova et al[76] in which 30 minutes of ice-bag application did not significantly affect the stretch reflex and latency amplitude of the peroneus longus during an inversion perturbation. These studies used healthy volunteers, so it is not known if individuals with acute or chronic ankle instability would demonstrate similar results.

The type of cold used in the previous studies varied, so it is difficult to compare findings. When ice bags or cold packs are used, cooling is relatively local and structures that are not cooled likely remain unaffected. In addition, as will be discussed later in this chapter, the degree to which ice bags and cold gel packs reduce tissue

temperature differs. When limbs are immersed in cold water, more tissues are affected, which may lead to more widespread changes in nerve conduction, force production, and joint position sense. Some studies suggest that proprioception is reduced immediately after application of cold; therefore, patients should be educated to use caution when ambulating or exercising immediately after cold application to the lower extremities.[66] This is particularly important when working with patients who may have underlying balance deficits.

Key Point! Any examination or intervention that challenges balance, proprioception, accuracy, or agility should be avoided for a period of time after the application of a cold modality due to the reduction in somatosensory input that these tasks require.

Neuromuscular Effects

Conditions that result from central nervous system dysfunction, such as multiple sclerosis, cerebral palsy, and cerebrovascular accident, typically limit a person's ability to perform functional activities due to the presence of spasticity, muscle weakness, and inefficient movement patterns that lead to early fatigue. Spasticity often limits a person's ability to carry out purposeful movements at the variable speeds required to perform activities of daily living. It is associated with an increased resistance to passive stretch, increased deep-tendon reflexes (DTRs), and clonus. Many therapeutic interventions are used to reduce spasticity, including positioning, modalities, and pharmacological interventions.

In some patients with spasticity, cold application can temporarily decrease the amplitude of DTRs[77] and the frequency of clonus,[78–80] which may improve the patient's ability to participate in therapy programs. Cold facilitates alpha-motoneuron activity and decreases gamma-motoneuron firing. For spasticity to be reduced, the reduction in gamma activity should be proportionally greater than the increase in alpha activity. Spasticity reduction with cold may occur through at least two mechanisms:

1. A reflex decrease in gamma-motoneuron activity through stimulation of cutaneous afferents[78]
2. A decrease in afferent spindle discharge by direct cooling of the muscle[79,80]

Although the mechanisms responsible for the changes seen have not been fully elucidated, animal and human studies have been designed to clarify and provide a rationale for these observed responses. Some earlier studies are referenced for the interested reader but will not be discussed in this chapter.[77,79–84]

Knutsson[85] found that all patients with spasticity who were treated with 15 to 20 minutes of cold demonstrated a decrease in the frequency, duration, and threshold of clonus. Two-thirds also demonstrated a decrease in resistance to passive motion and an increase in AROM. A short-duration application of cold has also shown effectiveness in reducing spasticity of the masseter muscle in patients with cerebral palsy.[86] Significant increases in interincisal distance were found after a 1-minute icing procedure, which indirectly implies muscle relaxation.

The use of cooling vests, a more generalized form of cryotherapy, has been examined in a few patient populations. One study examined the effects of a cooling garment on timed walking tests, spasticity, balance, and dexterity in individuals with multiple sclerosis.[87] Subjects who wore a cooled vest (stored in a freezer at –4°F [–20°C]) for 45 minutes before performing the study tests showed significant improvements in the timed walking tests and several balance measures compared with those who wore a room-temperature vest. Subjectively, wearing the cooled vest led to less fatigue, spasticity, weakness, and balance and gait difficulties, and these improvements were reported to last 2 to 8 hours after the vest was removed. In a similar study,[88] individuals with myasthenia gravis demonstrated improvements in muscle strength, respiratory measures, and fatigue after wearing a cooling vest.

Key Point! If additional studies are able to demonstrate consistent benefits from cooling vests for managing spasticity, these devices may become just as common and available for purchase as ice packs and cold packs.

Grahn et al[89] studied how cooling one hand can affect exercise tolerance in 12 individuals with heat-sensitive multiple sclerosis. When subjects rested one hand on a curved metal heat-extraction device

maintained at 64.4°F to 71.6°F (18°C to 22°C), treadmill walking tolerance increased from 33 minutes to 44 minutes.

These studies indicate that local or regional cooling may be beneficial to individuals with certain neurological conditions. The presence of spasticity, which can impede normal movement patterns, may be temporarily reduced. This may encourage increased use of the affected limb during functional activities. Also, if cooling can improve endurance, strength, and walking efficiency, individuals may be encouraged to increase overall activity and may feel more confident participating in recreational and social activities. However, it should be noted that many individuals with spasticity report a worsening of symptoms when they become cold. Therefore, the use of cold is not appropriate for all individuals with a neurological condition.

CLINICAL INDICATIONS FOR COLD THERAPY

Most clinicians will agree that in the acute phase (24 to 48 hours) following trauma, cold should be the modality of choice and should be administered as soon as possible after injury. Even though cold may be uncomfortable for the patient during the first few minutes, pain will ultimately be reduced, and edema, inflammation, and muscle spasm will most likely be lessened (Box 2-4).

Beyond the acute phase of injury, heat may be the agent of choice for intervention. But in many cases, cold has been a successful part of a therapeutic regimen to facilitate muscle contractions, reduce joint pain caused by arthritis, and lessen muscle spasm. One study demonstrated that the use of repeated cold applications in individuals with chronic lateral epicondylitis was as effective as exercise or exercise plus cold in reducing pain and improving function.[90]

Box 2 ■ 4 Primary Goals With Use of Cold Therapy

- Limit edema formation.
- Reduce pain.
- Facilitate muscle relaxation.
- Limit secondary hypoxic tissue injury.

Acute Musculoskeletal Trauma

The most common applications of cryotherapy are for acute musculoskeletal trauma or postsurgical swelling and pain. One of the earliest clinical reports supporting cold therapy for edema control and pain management appeared in 1946.[91] A comparison was made between two groups of patients who had undergone a variety of orthopedic surgical procedures. One group (n = 479) had no cooling, while the other (n = 345) had ice bags over their soft casts for a 48-hour period; these were replaced every 4 hours. The group treated with ice packs required fewer swelling-related splitting of casts (5.31%) compared with the non-iced group (41.3%). The ice-treated group also had less inflammation as evidenced by a lower white blood cell count and fewer fevers above 101°F (38.3°C). No subject in the iced group had apparent hematomas or hemarthrosis compared with 16 of those in the group that received no ice. Also, fewer narcotics were administered to those subjects who were treated with ice, indicating that their pain was less. Lowered pain levels and a reduction of analgesic intake after cold application has also been reported by others.[92–95]

A recent systematic review of the effects of cryotherapy after total knee arthroplasty (TKA) found no substantial effect on the variables of transfusion rate, pain on postoperative days 1 and 3, use of analgesics, and length of hospital stay. The addition of cryotherapy did, however, have a significant effect on postoperative blood loss, pain on postoperative day 2, and knee range of motion at discharge.[96]

Although intuition might lead us to believe that cold rather than heat during the early phases postinjury would lead to a faster recovery, this comparison was not reported until 1982 by Hocutt et al.[97] Patients with severe ankle sprain were placed in one of three groups: (1) superficial heating one to three times per day for 15 minutes plus elastic bandaging initiated early after injury, (2) cold whirlpool one to three times per day at 40°F to 50°F (7°C to 10°C) or ice packs for 15 to 20 minutes plus elastic bandaging initiated early after injury, or (3) cold treatment initiated 36 hours after injury. Treatment was continued for a minimum of 3 days for all patients. The patients treated with early cold (within the first 36 hours) returned to full activity (running and jumping without pain) an average of

8 days sooner than those treated with heat or delayed cold. Therefore, the time at which cryotherapy is initiated following trauma can influence the time course for functional outcome.

Compression and Elevation

Cold (ice) is most often used in conjunction with rest, compression, and elevation in managing acute trauma; this is represented by the acronym RICE (rest, ice, compression, and elevation). Several acronyms have been developed since the original RICE, including PRICE (*P* for "protection") (Table 2-5)[98] and RICES (*S* for "stabilization").[45] These added components have the common theme of preventing further injury by avoiding harmful activity or undue stresses accompanied by the use of braces, wraps, or splints for support. Regardless of the acronym used, the goals of cold therapy are to reduce swelling and inflammation, prevent further injury, and return to functional activities as soon as possible.

In many circumstances, edema may be lessened with immediate application of cold, but results are inconsistent when cold is intense or applied for long durations. In addition, the use of cold does not appear to be effective in reducing edema after it has already formed, so it is imperative to apply cold as soon as possible after an injury occurs.[45] None of the animal studies reported earlier incorporated the use of compression or elevation. Compression can help prevent and reduce edema by increasing external pressure on tissues, which limits fluid loss from vessels. In addition, compression increases the rate and degree of cooling compared with cold without compression[99,100] (Fig. 2-12). Elevation takes advantage of gravitational forces and decreases capillary hydrostatic pressure. When this pressure is reduced, less fluid is forced out of vessels, limiting edema formation.

Two groups of researchers investigated cold and compression for the postoperative management of patients after TKA.[101,102] Levy and Marmar[102] measured pain relief, swelling, blood loss, and ROM in 80 patients who underwent unilateral or bilateral TKA. The patients received either compression alone or cold combined with compression, provided by a commercially manufactured inflatable cuff filled with ice water. Those patients who received cold plus compression had less pain, less swelling, and a lesser

TABLE 2–5. PRICE (Protection, Rest, Ice, Compression, and Elevation and Stabilization)

Intervention	Technique	Rationale
Protection	• Avoid activity that may cause additional harm • Splints or braces for immobilization or relative immobilization	• Prevention of further injury or harmful stresses on inflamed tissues • Avoid unwanted motion of injured area
Rest	• Immobilization, limited weight bearing • Limited-range active motion	• Limit irritation of inflamed tissues • Provides opportunity to ice, compress, and elevate
Ice	• Ice packs • Ice baths • Controlled-cold devices	• Reduce bleeding • Control pain • Reduce microvascular permeability • Reduce metabolism to limit secondary hypoxic injury • Limit edema
Compression	• Light compressive bandages • Cold compression devices	• Limit edema • Maintain gains in edema reduction
Elevation	• Extremity positioned above heart level	• Reduce hydrostatic pressure to limit edema formation
Stabilization	• Use of splints, braces, wraps, or casts	• Provide support to allow surrounding musculature to relax • Prevent unwanted or unexpected motion

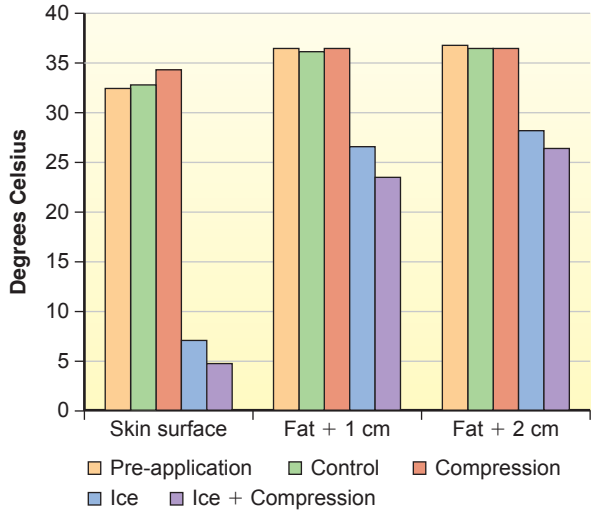

Fig 2 ■ 12 Temperature reductions at various depths with ice alone, compression alone, ice plus compression, and no treatment. *(Adapted with permission from Merrick MA, et al. The effects of ice and compression wraps on intramuscular temperature at various depths.* J Athl Train. *1993;28:241.)*

degree of blood loss while demonstrating a greater increase in ROM.

Healy et al[101] compared types of cold compressive dressings applied to the knees of 105 patients after TKA. A commercially available cuff consisting of an inflatable bladder filled with ice water was used for one group of subjects; the control group was treated with elastic bandage wraps and ice packs. Knee ROM, swelling, narcotic requirements, and wound drainage were measured. There were no significant differences between the groups for any of the variables. Both studies[101,102] advocated the use of cold compressive dressings for the postoperative treatment of TKA, but the type of cold compressive dressing did not appear to make any significant difference.

Adding compression to cold therapy can also improve microcirculation and tissue oxygenation.[54] When a series of three 10-minute sessions of cryotherapy plus compression at the ankle was compared with the same protocol with cryotherapy alone, superficial and deep capillary blood flow was greater in the combined group during the recovery phases (between the 10-minute applications). Tendon oxygenation was also greater in the combined group during the recovery phases. Postcapillary venous filling pressures were decreased in both groups during the treatment and the recovery phases.

Key Point! Although prolonged elevation of an extremity is difficult to achieve while maintaining function, continual compression can often be provided in the form of braces, wraps, or cold compression devices. This can help to counteract increases in hydrostatic pressures and reduced venous return from an extremity being in the dependent position.

The duration of cold and the extent of the resultant temperature drop are important factors to consider regarding effects on soft tissues. To lessen risk of thermal damage or an increase in limb volume from excessive cold exposure during acute or postoperative phases, the nature of the cold treatment must be taken into consideration. Less-intense cold applied for durations of 20 to 30 minutes several times a day in conjunction with elevation and compression appear to be a logical choice.

Cold Application Over Casts and Bandages

The presence of casts or bandages should not preclude the use of cold for edema or pain control, although the time to reach a minimum skin temperature is longer than when cold is applied directly to the skin. Okcu and Yercan[103] compared skin surface temperatures under two types of bandages and two types of casts in both injured (acute Grade III inversion ankle sprain) and uninjured individuals. Ice bags were applied over the casts or bandages. In all cases, skin surface temperature increased slightly immediately after applying the dressing or cast but then dropped 13°F to 29°F (7.3°C to 16.2°C) below baseline. In both the injured and uninjured groups, the greatest temperature changes were reached with cooling over a plaster cast (average change 27°F [15°C]), and the smallest change was seen with the use of a Robert Jones bandage (alternating layers of cotton padding and gauze) with the average change being 13.3°F (7.4°C). The time to reach the minimum recorded temperature was also shortest with the plaster cast (average 29 minutes) and longest with the Robert Jones bandage (average 47 minutes).

In a study by Ibrahim et al,[104] the type of dressing was also shown to affect cooling. Cold was applied to the knees of healthy volunteers with a continuous cooling unit over no dressing, over a thin adhesive dressing (Tegaderm), and over a dressing of wool and

crepe. The authors found that the wool and crepe dressing prevented the skin from reaching an effective cooling temperature of less than 68°F (20°C), whereas the skin temperatures with Tegaderm and no dressing both reached a temperature of 55.4°F (13°C). Similar results were found by Shibuya et al,[23] where effective cooling was achieved with a continuous cooling unit placed over standard postsurgical dressings with and without one layer of Robert Jones bandaging; effective cooling was not achieved when two layers of the bandaging were used. Table 2-6 demonstrates the progression of most to least effective cooling over various dressing and casting materials based on several studies.[23,103–105]

Key Point! Although studies have shown that cooling can occur with the application of cold over casts, it should be noted that it takes longer to achieve the same temperature under a fiberglass cast than it does under a plaster cast, and the presence of postsurgical dressings may prevent adequate cooling.

One of the underlying reasons for using cold after an acute musculoskeletal injury is to allow a more rapid return to function. However, few studies have looked at the effects of cryotherapy in conjunction with exercise or in relation to resumption of functional activities. Two systematic reviews point to the need for well-controlled studies in these areas.[106,107]

Pain and Muscle Spasm

As sequela to trauma, muscle spasm and pain often limit mobility and function. Many therapeutic techniques, including thermal agents, electrical stimulation, and manual therapy, are used with the common goals of reducing pain and muscle spasm, thus facilitating a more expedient recovery to normal function. In addition, many of these interventions may be used in lieu of pain medication. Patients can be easily instructed in the use of cold packs or ice massage at home for control of pain and muscle spasm.

TABLE 2–6. Cooling Under Casts and Bandages

Thin adhesive dressings	↑ Better cooling
Plaster casts	
Fiberglass casts	
Standard elastic bandages	
Robert Jones dressing (layered padding* and elastic bandages)	↓ Poorer cooling

*Typically a blend of cotton, crepe, rayon, or wool

Cooling the skin can elevate an individual's pain threshold[19,108] and reduce pain.[109] One theory for this process is that cold acts as a counterirritant,[99] and stimulation of thermal receptors in the skin (A-delta nerve fibers) may override pain signals from C fibers.[110] Another theory involves a decrease in afferent input via reduced nerve conduction velocity.[19] Following acute injury or inflammation, severe pain can limit motion and ultimately lead to joint stiffness in the absence of intervention. Therapeutic interventions are aimed at reducing pain and inflammation and maintaining or increasing ROM.

Cryokinetics (cold plus exercise) was a technique popularized in the 1960s by Hayden[3] and Grant.[2] Hayden wrote about a group of 1,000 military patients who sustained sprains, strains, and contusions during training. Ice massage or ice-water immersion was used to provide analgesia before ROM exercises. All but three of the patients returned to active duty within 2 days, and most required only one physical therapy treatment. In Grant's study of 7,000 patients with musculoskeletal injury from an army school, 90% had no more than three formal treatments with ice massage. Both Grant and Hayden cite that ice massage and exercise have the additional advantage of allowing patients to be more easily independent in self-treatment. The goal behind cryokinetics is that the patient be able to perform pain-free, graded, progressive exercise after a period of tissue cooling (via cold immersion, ice massage, or ice bags).[45] However, exercise must not be performed to the extent that reinjury or increased inflammation occurs.

Cold in combination with static stretch or hold-relax techniques (cryostretch) has been recommended for reducing muscle spasm or decreasing exercise-induced muscle soreness, thus increasing ROM. Cold is applied over the painful muscle using ice massage, ice packs, or cold packs. Either

during or immediately following the cold application, the stretch is performed with the idea of returning the muscle to its normal resting length. Knight and Draper[45] suggest that when cold is used in conjunction with stretching, the area should first be cooled until numbness is achieved, followed by cycles of passive stretch or hold-relax technique (a proprioceptive neuromuscular facilitation [PNF] technique).[111] This cycle of numbing and stretching is then repeated several times.

Delayed-Onset Muscle Soreness

Despite the positive effects of cold on pain and spasm, there is little research to support its use in reducing delayed-onset muscle soreness (DOMS). Prentice[112] induced muscle soreness in normal subjects through fatiguing concentric and eccentric contraction of the hamstrings. The following day, electromyographic (EMG) activity of the exercised muscle was increased from the pre-exercise measurement. EMG activity was measured as an indicator of muscle pain and spasm.[113] After 20 minutes of cold packs and either static stretch or PNF slow-reversal-hold stretch to the hamstrings, EMG activity was reduced, suggesting a decrease in muscle soreness and spasm. These techniques were compared with an untreated control group and two groups that were given hot packs and static stretch or PNF. Those who received cold had less measured EMG activity.

Crystal et al[114] studied the effects of cold-water immersion following a 40-minute downhill run. Immediately after the run, the experimental group stood quietly for 20 minutes in cold water (41°F [5°C]) while the control group stood on land for the same amount of time. No significant differences were found between these two groups for the variables of muscle contraction force, swelling, or pain measured at 1, 6, 24, 48, and 72 hours postimmersion.

Although a recent systematic review[115] found some evidence to support using cold-water immersion to lessen DOMS at 24, 48, 72, and 96 hours postexercise, most studies have shown no significant clinical benefit for using cold[116,117] to reduce DOMS, either with ice-water immersion,[114,118–120] whole-body cold-air exposure,[121,122] local cold-air exposure,[123] ice massage,[124] or contrast bath.[125] It remains questionable whether this technique is valuable.

Myofascial Pain Syndrome

Myofascial pain syndrome is defined as "the sensory, motor, and autonomic symptoms caused by myofascial trigger points."[27] There are often accompanying ROM deficits in the area of the myofascial trigger point or area of referred pain. A trigger point in muscle may result from muscular strain or postural imbalance and may be associated with sensitized nerves, increased metabolism, and decreased circulation. Trigger points are also thought to be present in skin, ligaments, and fascia.

The pioneering work in trigger point localization and therapy was done by Janet Travell, MD.[29] According to Travell, active points are associated with a decrease in ROM and moderate to severe pain that is relatively constant. Latent trigger points may also lead to restricted ROM, but pain is present only on palpation. Both active and latent trigger points can be located by digital pressure. Trigger points can be treated using a variety of techniques, including spray and stretch, ice sweeps, ice massage, sustained deep pressure, ultrasound, electrical stimulation, and low-power laser.[126,127] Common areas of trigger points can be around the cervical spine, shoulder girdle, lower back, and pelvis.[126] Trauma, poor body mechanics, and faulty posture are likely contributing forces.

A recent literature review highlights the need for well-controlled clinical studies regarding effective interventions for trigger points and myofascial pain syndrome.[127] It seems that the choice of treatment is largely based upon empiricism.[128] Interestingly, in spite of claims that vapocoolant spray and stretch techniques are widely used,[31] very few studies exist supporting its benefit in treating myofascial trigger points. A depiction of a spray and stretch treatment for a trigger point in the upper trapezius is shown in Figure 2-13A, and the spray and stretch technique is outlined in Case Study 2-4.

Ice massage has shown promise in reducing pain threshold sensitivity of trigger points.[129] When compared to sham ultrasound, 15 minutes of ice massage produced a significant decrease in trigger point pressure threshold and overall pain levels. Ice massage has also been shown to reduce pain pressure sensitivity of trigger points when compared to a menthol-based analgesic balm or placebo.[130]

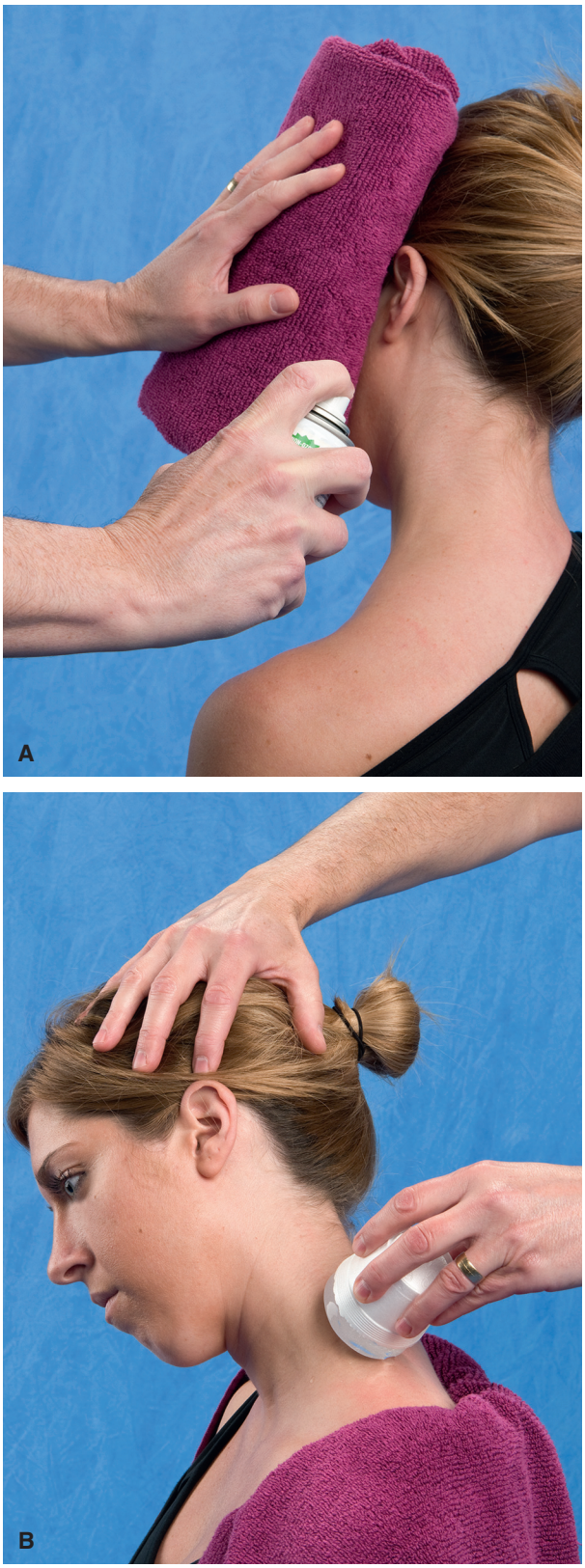

Fig 2 ■ 13 Options for myofascial trigger point treatment: (A) vapocoolant spray (and stretch) procedure for the upper trapezius; (B) ice cup plus stretch procedure for the levator scapula.

A technique for using ice massage and stretch for a trigger point in the levator scapula is shown in Figure 2-13B. Ice is applied in a small area over the levator scapula trigger point in circular motions for 5 to 7 minutes. The duration of the ice application should be sufficient to produce analgesia and allow for deep-pressure massage over the trigger point and stretching of the target muscles. Clinical trials comparing this technique with others in the management of trigger points would be beneficial.

GUIDELINES FOR CRYOTHERAPY

Cold can be administered by a variety of methods. Those discussed in this section include ice packs, flexible frozen gel packs, ice massage, cold baths or whirlpools, vapocoolant sprays, and manual and mechanical controlled-cold and cold compression units. The choice of agent depends on accessibility, body part to be treated, whether simultaneous compression and elevation are required, and size of the area to be cooled. For example, the shoulder may be most effectively cooled using a cold pack wrapped around the joint (Fig. 2-14A), whereas the foot and ankle may be best covered by a cold immersion bath (Fig. 2-14B).

When considering cryotherapy, the practitioner must be familiar with the patient's medical status and how long it has been since the injury occurred. (Precautions for cryotherapy are discussed in a later section.) Before initiating treatment, a small area of skin should be tested for hypersensitivity.[131] If hypersensitivity is apparent, this should be documented and the cold treatment discontinued. Signs of hypersensitivity include hives and wheals, the latter of which are raised, red, sometimes irregularly shaped areas that often burn or itch and can remain for 24 to 48 hours after exposure to cold (Fig. 2-15).

Following cryotherapy in any form, patients should avoid for 1 to 2 hours stresses that could potentially reinjure or aggravate the injury for which they were treated. The analgesia produced by the cold could mask exercise-induced pain, giving patients a false sense of security. Lowering of joint temperature can increase stiffness,[132] thereby decreasing reaction time and velocity of motion.[133] This, in combination with analgesia, may predispose patients to further injury.

When cold is applied to the skin, the area will become red. This occurs for two reasons. First, oxygen

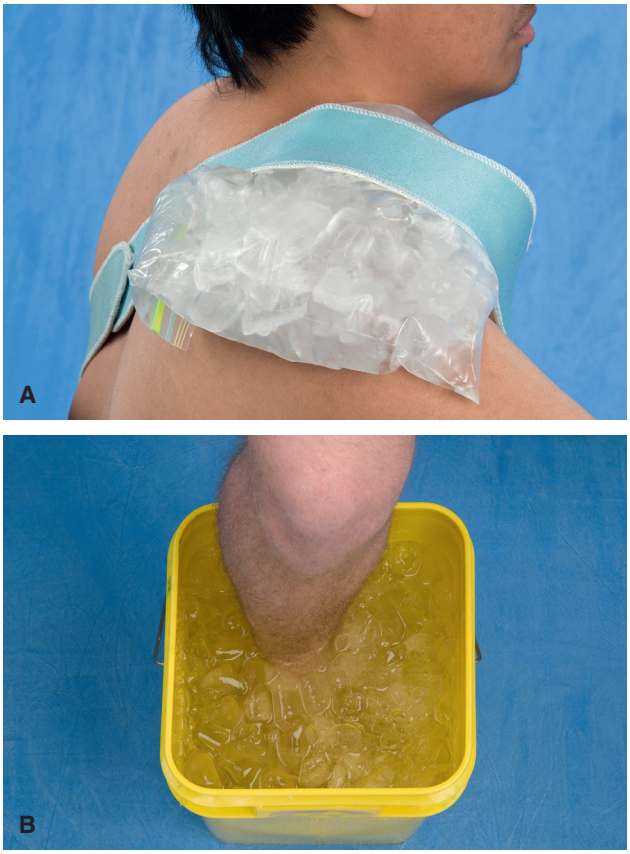

Fig 2 ■ 14 Cryotherapy options for various body regions: (A) ice bag for the shoulder (compression is added with sequential wrapping of the shoulder); (B) cold-bath immersion for the ankle (use of a toe cap will make immersion more comfortable yet allow cooling of the entire ankle).

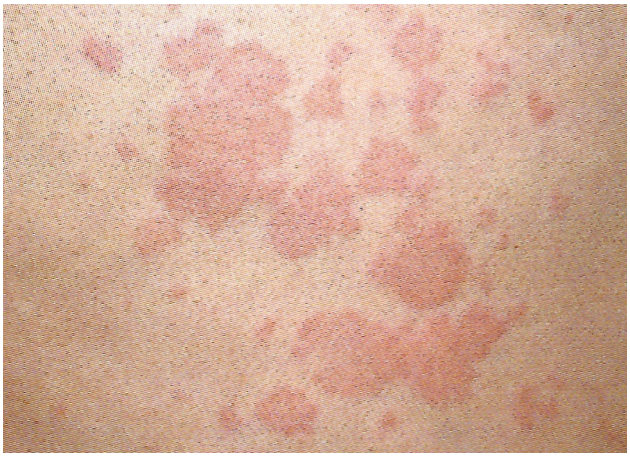

Fig 2 ■ 15 Example of wheals induced by exposure to cold.

does not dissociate as freely from hemoglobin at lowered temperatures; therefore, the blood passing through the venous system is highly oxygenated, giving a red color to the skin. Second, after a 10- to 15-minute period of cooling or upon removal of the cold stimulus, a reactive hyperemia may occur, bringing a greater amount of blood to the area.

Patients should be instructed about what to expect with application of a cold modality. When cold is to be applied for longer than several minutes, patients should be informed that they may initially feel intense cold, followed by a burning sensation, then aching, and finally numbness. Streator et al[134] found that providing patients with information about the sensations they would likely experience resulted in a lower level of reported pain than when no information was provided.

The temperature of the cooling agent may influence the level of discomfort the patient can expect. Galvan et al[135] found that pain ratings during immersion in ice water at 34°F (1°C) were 43% higher than immersion in water at 50°F (10°C) and 70% higher than immersion in water at 59°F (15°C). The authors also found that pain ratings across days and at the same temperature decreased, meaning that individuals somewhat adapted to the cold sensations. This may also be valuable information for patients, especially those who are reluctant to use cold after an initial uncomfortable experience.

Key Point! Informing patients that they will likely experience uncomfortable sensations (cold, burning, aching) before numbness with cold application and that these sensations often decrease in intensity with repeated applications may be beneficial in putting them at ease and gaining their trust.

Generally speaking, conductive cooling is administered for 20 to 30 minutes, with longer time periods recommended for areas covered by significant adipose tissue.[8] Intermittent cold applications (e.g., 20 minutes on, followed by two cycles of 10 minutes off and 10 minutes on) are more effective in reducing tissue temperature, blood flow,[98,136] and pain with activity[137] than just one 20-minute application. This cycling of cold, rather than repeated prolonged use of cold packs or other cooling agents, is suggested to avoid or reduce the occurrence of any adverse responses to nerves or blood vessels. In addition, if a patient remains inactive over several hours, repeated

applications of cold for 30 minutes every 2 hours may produce a progressive cooling effect.[138]

Planned activity before, during, or immediately after cryotherapy should be considered in conjunction with the goals of using this modality. Cardiovascular exercise performed immediately before ice-bag application will allow a more rapid cooling of muscular tissue due to increased blood flow that aids in the removal of heat from the cooled area.[139] When the goal is to achieve cooling at the level of muscular tissue, patients should refrain from activity while the cold modality is applied. Bender et al[140] demonstrated that, despite evidence of skin surface cooling, no intramuscular cooling occurred while subjects walked on a treadmill with an ice pack secured to the calf (Fig. 2-16).

Based on this finding, although it may seem time efficient, wrapping an ice pack over a target area and then allowing the patient to carry on with functional activities will not likely provide the desired therapeutic effect. Finally, if the goal of cryotherapy is to reduce deep tissue temperature for 30 minutes to several hours, activity should be avoided after the cold application. Exercise after removing ice packs from the gastrocnemius muscle increased subjects' intramuscular temperature to precooling levels within 10 minutes, while the intramuscular temperature of the subjects who remained at rest continued to decline in this same 10-minute period.[15]

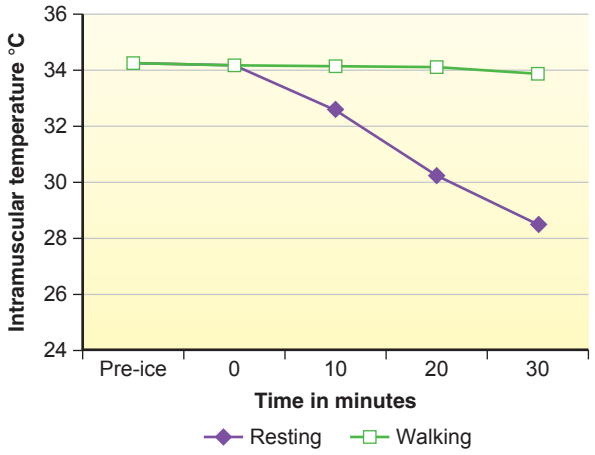

Fig 2 ■ 16 Intramuscular temperature of the gastrocnemius at rest and while walking on a treadmill with an ice bag secured to the calf. *(Adapted with permission from Bender AL, et al. Local ice-bag application and triceps surae muscle temperature during treadmill walking.* J Athl Train. *2005;40:271.)*

SELECTING A COOLING AGENT

When selecting a cooling agent, the clinician should consider which body area and how much of the body surface is to be cooled. For small areas (such as over a tendon, bursa, or small muscle belly), ice massage may effectively produce the cooling desired. If a distal extremity is to be cooled, as mentioned previously, a cool bath will most efficiently cover all surfaces. If there is concern about edema in distal extremities, a cold compression device may be most appropriate.

When cooling around a joint (such as the knee, elbow, or shoulder) or a larger muscle mass (such as lumbar or cervical paravertebral muscles), an ice pack secured with an elastic bandage or weighted to provide compression may be the best choice. Most studies show that ice or ice plus water in a plastic bag applied directly to the skin provides superior cooling compared with commercial gel packs or bags of frozen peas, and any agent that requires toweling will be less effective than one that does not.[14,24,141] Comparisons of various types of cooling agents are presented in a later section.

Sometimes the decision as to whether to use heat or cold for pain control is not always clear. Cold is always the appropriate choice in the acute phase of injury, while muscle spasms may respond to both heat (muscle relaxation) and cold (interruption of the pain/reflexive guarding cycle). The benefits of cold or heat with chronic inflammatory conditions are varied and may be patient dependent. In this case, a trial of either heat or cold is reasonable.

CONTRAINDICATIONS AND PRECAUTIONS FOR CRYOTHERAPY

Contraindications

Cryotherapy should not be used when treating patients who have specific cold-sensitivity symptoms. These conditions include, but are not limited to, cold urticaria, cryoglobulinemia, Raynaud's phenomenon, and paroxysmal cold hemoglobinuria.[142] Cold urticaria can include both local and systemic reactions. In response to local cold application, patients develop wheals characterized by erythematous, raised borders and blanched centers. Mast cell degranulation causes histamine to be released

into the area, markedly increasing capillary permeability and leading to redness, swelling, and wheal formation. In severe cases, patients develop generalized swelling involving mucous membranes and viscera. Systemic reactions include flushing of the face, a sharp drop in blood pressure, increased heart rate, and syncope.[143]

Cryoglobulinemia is a disorder characterized by the presence of cryoglobulins, abnormal blood proteins that precipitate and form a gel when exposed to low temperatures (e.g., below body temperature). This precipitation of cryoglobulins results in the aggregation of serum proteins, which can lead to ischemia or gangrene. Cryoglobulinemia is associated with multiple myeloma; certain types of viral and bacterial infections, including hepatitis C; chronic liver disease; systemic lupus erythematosus (SLE); and other rheumatic diseases.[144]

Raynaud's phenomenon is a vasospastic disorder and can be either idiopathic or associated with other disorders, such as systemic scleroderma, SLE, thoracic outlet syndrome, and trauma. Smoking and caffeine can also worsen the frequency and intensity of the symptoms. Cycles of pallor, cyanosis, rubor, and normal color of the digits may be accompanied by numbness, tingling, or burning. Attacks are precipitated by exposure to cold or by emotional stress.[145]

Paroxysmal cold hemoglobinuria can occur following local or general exposure to cold. Hemoglobin, which is normally found within red blood cells, is released from lysed red cells and appears in the urine.

Cold should not be applied over areas of nerve regeneration or compromised circulation. For patients with peripheral vascular disease that affects arterial circulation, the vasoconstrictive effects of cold could potentially compromise an already nutritionally deprived area (Table 2-7).

TABLE 2–7. Contraindications and Precautions for Cryotherapy

Contraindications	Precautions
• Cold urticaria	• Hypertension
• Cold intolerance	• Thermoregulatory disorders
• Paroxysmal cold hemoglobinuria	• Over a superficial peripheral nerve
• Cryoglobulinemia	• Over an open wound
• Raynaud's disease or phenomenon	• Over an area of poor sensation
• Over a regenerating peripheral nerve	• With individuals with poor cognition
• Over an area of circulatory compromise	• In the very young or very old
• Over an area of peripheral vascular disease	• Persons with aversion to cold

Precautions

Because cold can cause a transient increase in systolic and diastolic blood pressures,[146,147] careful monitoring should take place when cryotherapy is used with hypertensive patients. Blood pressure should be monitored before and throughout treatment. Discontinue treatment if blood pressure rises.

Cold should be applied cautiously on individuals with hypersensitivity to cold, impaired circulation, and thermoregulatory disorders.[148] If, after careful consideration, cryotherapy is the treatment of choice for these patients, it is necessary to closely monitor the patient's response to treatment and make any adjustments to the treatment parameters. Decreasing the duration and adjusting the intensity of the cold application may produce the desired effects without eliciting adverse reactions.

Because cold temperatures may impede wound healing, precaution must be taken when applying cold in the area of a wound. Lundgren et al[149] demonstrated a 20% reduction in wound tensile strength in rabbits kept at environmental temperatures of 53.6°F (12°C) compared with those kept at 68°F (20°C). Only innervated animals showed this impaired healing response, suggesting a reflex cutaneous vasoconstriction (and thus a reduction in blood flow) with cold application. Until demonstrated otherwise, it is probably prudent to avoid vigorous cold application directly over a wound during the initial 2- to 3-week period of healing.

As mentioned earlier, prolonged cold application from 1 to more than 2 hours over an area containing a superficial peripheral nerve (e.g., around the medial epicondyle of the elbow or fibular head) can lead to neurapraxia or axonotmesis.[59,60] If cold is to be applied in an area of a superficial nerve, a small pad should be used between the cold source and the nerve for protection.

A survey of athletic trainers conducted by Nadler et al[150] found that the most common complications from cryotherapy were from allergic reaction. Other complications were much less frequent and included intolerance/pain, burns, frostbite, and skin rash.

In addition to certain physiological factors contraindicating the use of cryotherapy, the patient's psychological response to this form of treatment should be taken into account. Some people have an aversion to cold and will not tolerate cryotherapy. This consideration is particularly important if cold is being used to decrease pain and promote skeletal-muscle relaxation.

METHODS OF PROVIDING CRYOTHERAPY

A variety of methods can be used to provide effective cryotherapy. This section describes the most common clinical applications of cold. All of these can be used by patients at home with proper instruction. A case study is presented for each type of cold modality to give the reader a clinical perspective. Table 2-8 provides information about the materials needed and the cost of different modes of therapeutic cold.

Cold Packs

Cold packs can be inexpensively purchased or easily made. They are typically composed of ice, ice plus water, water plus alcohol, gel, or chemicals (Fig. 2-17). To prevent skin damage, the use of toweling over the skin is recommended for any agent that has the ability to reach temperatures below 30°F (–1°C) . Any use of toweling, however, will decrease the effectiveness of the cooling agent.[151] When toweling is required, damp towels are superior to dry towels in facilitating energy

TABLE 2–8. Materials Needed and Average Cost of Typical Cryotherapy Agents

Method	Materials Required	Average Cost
Frozen peas	• Purchased bag of peas, frozen for several hours • Thin wetted or dry towel for skin protection	$1–$3
Homemade ice bag	• Medium to large plastic bag (doubled for protection against leaking) • Ice cubes or crushed ice • Tap water (optional)	<$1
Homemade cold pack (with alcohol)	• Medium to large plastic bag (doubled for protection against leaking) • 1 cup isopropyl alcohol • 2–4 cups tap water • Thin wetted or dry towel for skin protection • Place in freezer several hours prior to use	$1–$3
Ice/cold bath	• Bucket or basin large enough to immerse the limb • Tap water • Ice cubes	$4–$10
Commercial cold packs (gel)	• Purchased gel pack • Thin wetted or dry towel for skin protection	$7–$50
Cold compression units	• Appropriate sleeve for area to be cooled • Insulated cooler with hose	$60–$150
Continuous cold compression units	• Appropriate sleeve for area to be cooled • Insulated motorized cooler with hose	$140–$280
Controlled cold compression units	• Appropriate sleeve for area to be cooled • Insulated motorized cooler and compression pump with hose	$2,400–$2,700

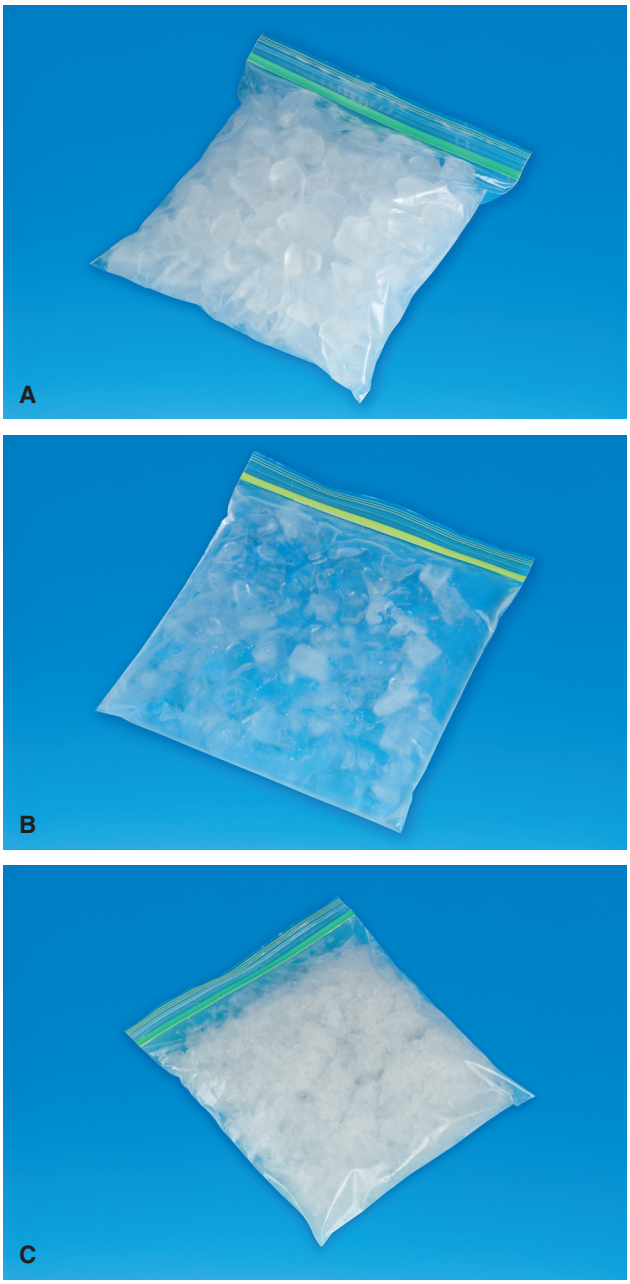

Fig 2 ■ 17 Various types of cold packs: (A) ice cubes, (B) ice cubes plus water, and (C) crushed ice.

transfer for thermal conduction.[45] If the towel is wet with room-temperature or lukewarm water, the initial contact with the skin will be more comfortable for the patient.

Crushed or cubed ice is typically placed in a plastic bag to create an ice pack. Ice is the most effective type of cold because it must undergo a phase change from solid to liquid, which causes greater heat extraction than when no phase change is required. Also, the temperature of the ice pack upon contact with the skin is typically just under 32°F (0°C), making it safe to apply directly to the skin. Water can also be added to the ice in the bag. Dykstra et al[14] found this method reduced skin and intramuscular temperature to a greater extent than cubed or crushed ice with no water (Fig. 2-18).

A common recommendation for home cold packs is the use of bags of frozen vegetables (peas tend to be preferred because they are small and round and conform well to body parts). Although using a bag of frozen peas may be a convenient, reusable, and low-cost alternative to ice packs, two studies have shown that frozen peas are not as effective in reducing skin surface temperatures compared with ice packs or a mixture of water and alcohol.[24,141] Kennet et al[24] also showed that the temperature of a bag of frozen peas taken from a freezer (14°F [–10°C]) was too cold for direct application to the skin and would require a layer of toweling. This toweling limits cooling by creating a barrier between the skin and the cold agent.

Commercial gel packs usually contain a silica gel and are available in a variety of sizes and shapes to contour to the area to be treated (Fig. 2-19A).

The flexible gel packs stored in a standard freezer will typically reach a temperature of –17°C (–1°F), which

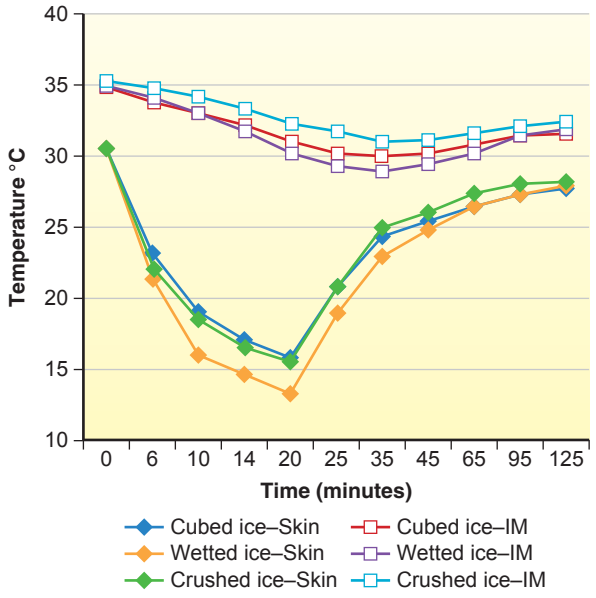

Fig 2 ■ 18 Changes in skin and intramuscular temperature with three different forms of ice packs during 20 minutes of application to the calf. *(Adapted with permission from Dykstra JH, et al. Comparisons of cubed ice, crushed ice, and wetted ice on intramuscular and surface temperature changes.* J Athl Train. *2009;44:136.)*

CASE STUDY 2-1 Ice Pack

A 42-year-old male is referred with a diagnosis of acute low back pain. Upon examination, he demonstrates pain and spasm in the right lumbar paraspinals. Hypertonicity of the musculature is observable and palpable, and the patient is limited in forward flexion, left lateral flexion, and right rotation due to pain and tightness.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of cryotherapy?
   ANSWER: Yes, muscle spasm and pain can be reduced with cryotherapy, allowing for performance of muscle stretching.
2. Is the patient appropriate for cryotherapy (i.e., do any of the general precautions or contraindications to cryotherapy apply to the patient or are there any specific considerations regarding application of cryotherapy to this patient)?
   ANSWER: The patient should be asked about the presence of any contraindications or precautions for the use of cold. A small nearby area of skin should be tested with the cold modality to determine if hypersensitivity (wheals, hives) is present.
3. What are the specific goals to be achieved with the use of cryotherapy?
   ANSWER: Reduction of muscle spasm and pain to allow for stretching of tight musculature.
4. What specific form of cryotherapy would be appropriate for the patient?
   ANSWER: Ice packs are most appropriate because of the surface area to be treated and the desire to cool tissues at the muscular level. Commercial cold gel packs are also appropriate (but not as effective).
5. What specific parameters of cryotherapy are appropriate for the patient?
   ANSWER:
   Type of ice pack: Plastic bag filled with crushed ice, cubed ice, or ice with water; a commercial gel pack is less effective but also appropriate (will use identical application technique except requires a layer of toweling between the cold pack and the patient's skin to prevent frostbite).
   Duration: 20 minutes or until the area is numb (additional time is required if there is significant adipose tissue overlying the target tissue). If muscle stretching is to be performed, this can be done at the end of the 20-minute period and then re-icing should occur for 5 to 10 minutes after the stretching period (this cycle can be repeated several times).
6. What are the appropriate and safe application procedures for ice packs?
   ANSWER:
   Instruct the patient: Inform the patient of the expected sensations of cold, burning, aching, and then numbness (analgesia).
   Preinspection: Inspect the area to be treated for skin compromise, and assess for intact sensation over the area to be treated.
   Equipment needed: Bag of ice, towels
   Patient position: Prone or sitting. If sitting, a wrap will be necessary to secure the ice pack to the affected area (this will also add a compressive force).
   Procedure: Place the ice pack directly on the patient's skin over the target area. A small weight can be placed on top of the ice pack to add a compressive force, which will allow for more effective cooling.
   Postinspection: Remove the ice pack and inspect the patient's skin. Redness will be present and may remain for 20 minutes or longer, depending on the patient's activity level or exercises performed. If stretching or light resistance is to be performed, it should be initiated as soon as possible after the ice pack is removed (a second or third application of cooling may be necessary to renumb the area through an exercise session).

could cause frostbite if applied directly to the skin. Therefore, a layer of damp toweling is required. Gel packs are not as effective at cooling skin or intramuscular tissues when compared with ice packs or ice-water packs,[24,141] because the gel does not go through a phase change; therefore, less energy is required for heat transfer. The requirement of toweling may also reduce the cooling effectiveness of this modality.

A mixture of water and isopropyl alcohol (2:1 to 4:1 ratio) can also be used as a homemade cold pack (Fig. 2-19B). The alcohol acts as antifreeze, which prevents the water from forming a solid and allows the pack

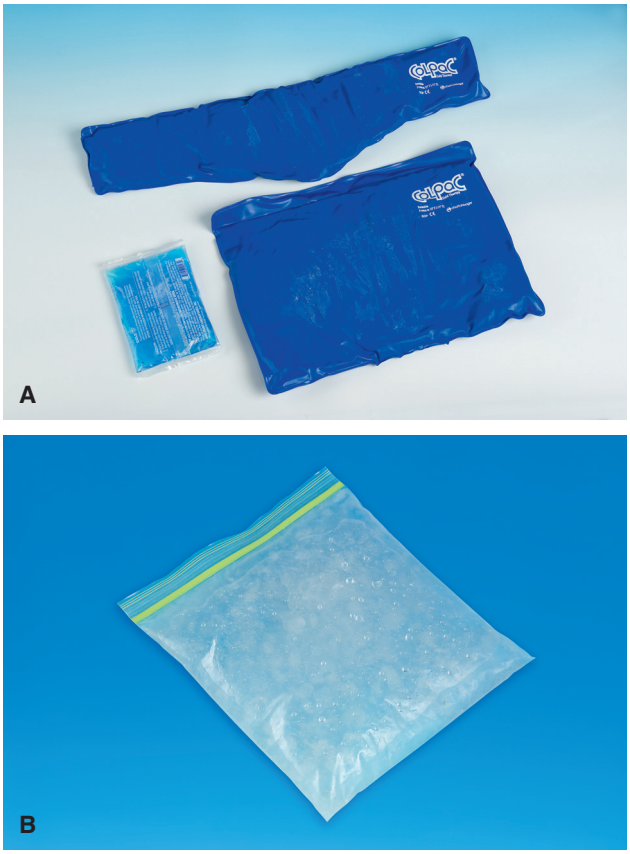

Fig 2 ■ 19 Examples of (A) commercial gel packs and (B) a homemade cold pack using a mixture of tap water and isopropyl alcohol (3 parts water to 1 part alcohol).

to remain pliable, similar to the commercial gel packs. This mixture should be stored in the freezer for several hours before application. Because the alcohol/water mixture can reach temperatures colder than ice, a thin layer of damp toweling should be placed between the skin and the pack. This type of cold pack has been shown to cool as effectively as a crushed ice pack over a 20-minute period (to a temperature of 50°F [10°C]).[141]

There are some cold packs that are chemically activated by squeezing or hitting them against a hard surface. These packs are usually marketed for first aid and designed for one-time use only. The chemical reaction inside some of the packs is at an alkaline pH and can cause skin burns if the package splits open and the contents spill out. Therefore, these packs are not recommended for general use.

As mentioned in the "Acute Musculoskeletal Trauma" section, compression and elevation are often used to control edema and improve the cooling ability of the cold modality. Ice bags are usually the most readily available form of cold when acute injuries occur, and elastic bandages can provide adequate compression over the cold packs on most body regions.

Ice Massage

Ice massage is usually performed over a small area (i.e., over a muscle belly, tendon, or bursa) or over trigger points before deep pressure release or massage. The technique is simple and can be taught to patients who can reliably follow directions for home use.

Water is frozen in paper or Styrofoam cups to make it easier for the practitioner to handle the ice. Just before use, the top of the cup is peeled back to expose the ice. Further peeling can occur as the ice melts. As an alternative, ice "lollipops" can be made by putting a wooden tongue depressor in the cup with the water (Fig. 2-20). The ice pop can be taken out of the cup and held by the tongue depressor for application. A 10 cm by 15 cm area (4 in. by 6 in.) can be covered in 5 to 10 minutes.[152] The ice is slowly rubbed over the skin using small overlapping circles or strokes.

Key Point! Styrofoam cups are often used for ice cups because they allow for more comfortable handling of the cup due to the insulating properties of the Styrofoam material (i.e., the hand holding the ice cup will not become cold). However, Styrofoam is not biodegradable and therefore not environmentally friendly. Wrapping a small washcloth around the base of the ice cup will prevent the hand from becoming too cold and is an environmentally friendly alternative to the handling problem.

During ice massage, the patient will probably experience the four common sensations of intense cold, burning, aching, and then analgesia. However, the stages of burning and aching should each pass rapidly within about 1 to 2 minutes. A prolonged phase of aching or burning may result if the area covered is too large or if a hypersensitive response is imminent (see "Contraindications and Precautions for Cryotherapy"). Skin temperature will usually not drop below 59°F (15°C) when the ice is continually moved over the skin;

CASE STUDY 2-2 RICE

A 17-year-old male is referred with a diagnosis of acute Grade II right lateral ankle sprain that occurred 12 hours prior. Upon examination, he demonstrates notable effusion throughout the right ankle (lateral greater than medial), decreased ankle AROM in all directions, and pain that increases with active or passive ankle motion and weight bearing.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of cryotherapy?

ANSWER: Yes; pain can be reduced with cryotherapy, allowing for performance of ROM within the patient's tolerance.

2. Is the patient appropriate for cryotherapy (i.e., do any of the general precautions or contraindications to cryotherapy apply to the patient, or are there any specific considerations regarding application of cryotherapy to this patient)?

ANSWER: The patient should be asked about the presence of any contraindications or precautions for the use of cold. A small nearby area of skin should be tested with the cold modality to determine if hypersensitivity (wheals, hives) is present.

3. What are the specific goals to be achieved with the use of cryotherapy (specifically RICE)?

ANSWER: The use of ice will reduce pain, allowing for initiation of gentle ROM exercises and at least partial weight bearing. Rest from typical or stressful activity will allow healing to proceed. Compression will help control effusion and edema and will increase the cooling ability of the ice. Elevation will reduce joint effusion and edema.

4. What specific form of cryotherapy would be appropriate for the patient?

ANSWER: Ice packs are most appropriate because these will conform well to the area.

5. What specific parameters of cryotherapy are appropriate for the patient?

ANSWER:

Type of ice pack: Plastic bag filled with crushed ice, cubed ice, or ice with water.

Type of compression: Elastic bandages (4 in. to 6 in. wide)

Duration: 20 minutes or until the area is numb. If ROM exercises or weight bearing is to be performed, this can be done at the end of the 20-minute period, and then re-icing (with compression and elevation) may occur for 5 to 10 minutes following the stretching period (this cycle can be repeated several times).

6. What are the proper application procedures for RICE?

ANSWER:

Instruct the patient: Inform the patient of the expected sensations of cold, burning, aching, and then numbness (analgesia).

Preinspection: Inspect the area to be treated for skin compromise, and assess for intact sensation over the area to be treated.

Equipment needed: Bag of ice or ice plus water, elastic bandage long enough to fully cover the ice bag and ankle, stack of towels or other materials to elevate the patient's leg.

Patient position: Supine.

Procedure: Place the ice pack directly on the patient's skin over the target area. Begin wrapping the elastic bandage around the ice pack and ankle and continue in a distal to proximal direction, stretching the elastic bandage to approximately 75% of its capacity.[45] Elevate the patient's lower extremity to a level above the heart and ensure that the leg is fully supported.

Postinspection: Remove the elastic bandage and ice pack and inspect the patient's skin. Redness will be present and may remain for 20 minutes or longer, depending on the patient's activity level or exercises performed. If ROM or weight bearing is to be performed, it should be initiated as soon as possible after the ice pack is removed (a second or third application of cooling, compression, and elevation may be necessary to renumb the area and reduce any accumulated fluid caused by dependent positioning).

Fig 2 ■20 Ice cups and ice lollipops. (A) Preparation for freezing, (B) after freezing, (C) using the ice cup on the wrist, and (D) using the ice lollipop on the wrist.

therefore, the risk of damaging tissue and producing frostbite is minimal.

Vapocoolant Spray

Vapocoolant sprays are used to treat trigger points and to induce relaxation of tight muscles before stretching (the physiological basis is described in the "Evaporation" section). When treating trigger points, the patient is positioned comfortably and the muscle containing the trigger point is placed on passive stretch. Spraying is done in unidirectional sweeps along the muscle over the trigger point areas and over the areas of referred pain while maintaining and gently increasing the passive stretch. The vapocoolant canister is held 12 to 18 inches away from the skin during spray applications. When using the spray to increase muscle length without identified trigger points, the muscle is sprayed along its length from the proximal to the distal attachments. Repeated treatments during the same session are done

CASE STUDY 2-3 Ice Massage

A 28-year-old female is referred with a diagnosis of right lateral epicondylitis that began after she spent 9 hours painting fence posts 2 days ago. Upon examination, there is exquisite tenderness in the area surrounding the right lateral epicondyle, pain and limitation with right wrist extension, and intense pulling in the right proximal forearm with passive wrist flexion.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of cryotherapy?
   ANSWER: Yes; cryotherapy can be effective in reducing pain and inflammation.
2. Is the patient appropriate for cryotherapy (i.e., do any of the general precautions or contraindications to cryotherapy apply to the patient, or are there any specific considerations regarding application of cryotherapy to this patient)?
   ANSWER: The patient should be asked about the presence of any contraindications or precautions for the use of cold. A small nearby area of skin should be tested with the cold modality to determine if hypersensitivity (wheals, hives) is present.
3. What are the specific goals to be achieved with the use of cryotherapy?
   ANSWER: Pain reduction and to allow the patient to perform ROM and stretching with less discomfort.
4. What specific form of cryotherapy would be appropriate for the patient?
   ANSWER: Ice massage (ice packs are also appropriate but would take longer and would not isolate the area as well).
5. What specific parameters of ice massage would be appropriate for the patient?
   ANSWER: Rubbing the ice directly on the skin over the affected area for 5 to 7 minutes or until analgesia (numbness) is achieved.
6. What are the proper application procedures for ice massage?
   ANSWER:
   Instruct the patient: Inform the patient of the expected sensations of cold, burning, aching, and then numbness in the treated area.
   Preinspection: Inspect the area for skin compromise, and assess for intact sensation over the area to be treated.
   Equipment needed: Ice cup or ice Popsicle, 3 small towels
   Patient position: Sitting with the right arm in a comfortable position that allows easy access to the affected area. After the patient is positioned, two towels should be placed on either side of the proximal forearm to catch water drips.
   Procedure: If using the ice-cup method, remove enough of the cup's top to expose at least 1 inch of ice, making sure to leave enough of the cup to hold on to (wrap a small towel around the cup's bottom to provide insulation for the hand holding the cup). After rubbing the ice briefly to make the top smooth (melting off any ice spurs or ridges), place the ice on the patient's skin over the affected area and begin moving the cup in small, slow circles. Continue for 5 to 7 minutes or until numbness is achieved. If the patient is to perform any stretching or ROM exercise, this should be initiated as soon as the ice massage is completed. Renumbing may be required after several minutes of exercise, but the time required to achieve numbness again should be less than the first application. This cycle of numbing and exercise may be repeated several times.
   Postinspection: Remove the ice cup and discard. Dry the skin and inspect for any adverse effects. Redness will be present and may remain for 20 minutes or longer, depending on the patient's activity level or exercises performed.

only after the skin has been rewarmed to avoid frosting the skin.

Manual and Electric Cold Compression Units

Cold compression devices (Fig. 2-21) allow for manual circulation of cold water through a cuff that is applied over an extremity. A variety of sizes and shapes of cuffs and sleeves are available to conform to any joint or extremity. To fill or recirculate cold water into the cuff, the insulated ice-water-filled container is connected to the cuff by a hose and then elevated, allowing gravity to pull water into the cuff. Manual recirculation is recommended every 1 to 2 hours to maintain the cooling effect. However, specific temperature monitoring and

CASE STUDY 2-4 Vapocoolant Spray

A 56-year-old female is referred with a diagnosis of headaches and right neck pain. Upon examination, several active myofascial trigger points are found in the right upper trapezius that reproduce the patient's lateral neck pain and headaches upon compression. She also reports tightness and discomfort with cervical flexion and left lateral flexion.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of cryotherapy?
   ANSWER: Yes; active myofascial trigger points may be treated with vapocoolant spray to reflexively inhibit the agonist muscle, followed by stretch that is intended to normalize the affected muscle's length.
2. Is the patient appropriate for cryotherapy (i.e., do any of the general precautions or contraindications to cryotherapy apply to the patient or are there any specific considerations regarding application of cryotherapy to this patient)?
   ANSWER: The patient should be asked about the presence of any contraindications or precautions for the use of cold. A small nearby area of skin should be tested with the vapocoolant spray to determine if hypersensitivity (wheals, hives) is present.
3. What are the specific goals to be achieved with the use of cryotherapy?
   ANSWER: Reflexive inhibition of active myofascial trigger points.
4. What specific form of cryotherapy would be appropriate for the patient?
   ANSWER: Vapocoolant spray.
5. What specific parameters of vapocoolant spray would be appropriate for the patient?
   ANSWER: Three to five slow (10 cm/sec [4 in./sec]), unidirectional sweeps of the spray in the direction of the pain pattern, avoiding direct overlap of each spray.[126]
6. What are the proper application procedures for vapocoolant spray (and stretch)?
   ANSWER:
   Instruct the patient: Inform the patient of the expected sensations of cold in the area to be sprayed.
   Preinspection: Inspect the area to be treated for skin compromise, and assess for intact sensation over the area to be treated.
   Equipment needed: Vapocoolant spray, small towel to protect the patient's eyes.
   Patient position: Sitting with the right arm holding the edge of a chair, right upper trapezius on slight stretch (cervical flexion, left lateral flexion, right rotation). Because the patient's head is rotated toward the spray, a small towel should be held over her eyes for protection from the spray.
   Procedure: Place the patient's right upper trapezius on slight stretch (cervical flexion, left lateral flexion, right rotation), which should be maintained throughout the procedure, taking up any slack as the muscle relaxes. Begin spraying in the direction of the patient's pain pattern, with the canister approximately 30 cm (12 in.) away and at a 30° angle to the skin. The first sweep should go directly over the primary myofascial trigger point. The remaining two to four sweeps should be along the referral pattern in the same direction as the first sweep but not directly over it. When the sweeps are completed, apply additional but gentle stretch to the upper trapezius, holding 20 to 30 seconds before the patient returns to normal alignment.
   Postinspection: The patient's skin may be slightly red over the treated area. Observe for any abnormal skin reaction to the spray. Because the spray evaporates quickly, there is little, if any, rewarming time required.

adjustment are not possible with these units. Compression is achieved by pressure exerted from the filled cuff that is wrapped around the joint or extremity and secured with Velcro. One study demonstrated that the Aircast Cryo/Cuff device was capable of maintaining a skin surface temperature between 68.7°F and 82.4°F (20.4°C and 28°C) when applied over a standard postsurgical dressing. It should be noted that the authors of this study recirculated the cold water in the sleeve every 15 minutes versus every hour as recommended by the manufacturer.[23]

Continuous cold compression units (Fig. 2-22) are similar to the devices described previously except that they use an electric pump to circulate cold water at intervals or rates that can be set by the user, depending on the level of cooling desired. Water temperatures can be

Fig 2 ■21 Cold compression units. (A) DonJoy ArcticFlow, (B) shoulder cuff, and (C) Aircast Cryo/Cuff. *(Courtesy DJO Inc., Vista, CA.)*

adjusted from 32°F to 104°F (0°C to 40°C), and the set temperature is maintained as long as the unit is on. Several studies[153–156] have investigated the differences in postoperative outcomes comparing continuous cold compression units and traditional icing protocols. Although outcome variables differed between each study, pain, edema, range of motion, and medication use were improved to a greater extent in patients who used continuous cold compression versus ice packs. Because cooling temperatures can be set to levels that are unsafe for prolonged skin exposure,[23] patient education about appropriate temperature settings is imperative. Khajavi et al[157] describe a case of severe compartment syndrome that resulted from improper use of a continuous cold compression unit set at 33°F (0.5°C) and used continuously for 5 days following an arthroscopic procedure of the knee.

Controlled-cold compression units add the effect of adjustable levels of compression (typically 5 to 75 mm Hg) at variable intervals. Thus, these units allow the treated

Fig 2 ■22 Controlled-cold compression units. (A) Bledsoe Cold Control, (B) shoulder wrap, (C) DonJoy IceMan, and (D) ankle wrap. *(Courtesy Bledsoe Brace Systems, Grand Prairie, TX; and DJO Inc., Vista, CA.)*

area to receive a constant cold temperature along with intermittent periods of compression. These units are most commonly found in sports medicine facilities and can cost several thousand dollars.

Cold Baths

When cooling the distal extremities, immersion of these parts in a cold bath is most practical (unless simultaneous elevation is desired, particularly in the presence of edema). This approach ensures circumferential contact of the cooling agent. Water temperatures for immersion vary from 50°F to 64.4°F (10°C to 18°C). The lower the temperature range, the shorter the duration of immersion required for cooling. In a study by Galvan et al,[135] it was found that pain ratings were significantly higher during immersion in 34°F (1°C) and 50°F (10°C) temperatures versus immersion in 59°F (15°C) water. However, pain ratings improved during both the 34°F and 50°F after the first several minutes of immersion, and average ratings never exceeded 4.5 on a 0 to 10 scale (0 = no pain; 10 = very, very strong pain) for any immersion temperature. As discussed in the "Convection" section, movement of the water particles over the skin, either with water turbulence or with movement of the body part, will increase the speed of cooling. A basin of water or a small whirlpool filled with water and crushed ice can be used (see Fig. 2-14B). The use of a toe cap will reduce the pain sensation in the toes, which tend to be the most painful area during immersion.[158] Cold-bath immersions can be used by patients at home with proper instruction.

Cold Gel

Commercially available gels or creams give the *perceived* sensation of cold. Most of these gels use chemicals, such

CASE STUDY 2-5 Cold Compression Device

A 32-year-old female is referred with a diagnosis of acute left knee sprain with suspicion of a torn anterior cruciate ligament. Upon examination, she demonstrates global knee pain and notable effusion. The patient's AROM is limited to 10° to 85°, and she ambulates with bilateral axillary crutches at 50% weight bearing on the left.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of cryotherapy?

ANSWER: Yes; pain can be reduced with cryotherapy, allowing for performance of range of motion within the patient's tolerance.

2. Is the patient appropriate for cryotherapy (i.e., do any of the general precautions or contraindications to cryotherapy apply to the patient or are there any specific considerations regarding application of cryotherapy to this patient)?

ANSWER: The patient should be asked about the presence of any contraindications or precautions for the use of cold. A small nearby area of skin should be tested with the cold modality to determine if hypersensitivity (wheals, hives) is present.

3. What are the specific goals to be achieved with the use of cryotherapy (specifically RICE)?

ANSWER: The use of cold will reduce pain, allowing for performance of ROM exercises and increased weight bearing. Compression will help control effusion and will increase the ice water's cooling ability. Elevation may also be used to help with reduction of effusion.

4. What specific form of cryotherapy would be appropriate for the patient?

ANSWER: A commercial cold compression unit.

5. What specific parameters of cryotherapy are appropriate for the patient?

ANSWER:

Type of cold: Ice water in the cold compression unit's container that is manually circulated through a wraparound cuff by raising and lowering the container.

Duration: 20 to 30 minutes (with elevation to maximize effusion reduction) or until the area is numb and swelling has reduced; if ROM exercises are to be performed, this can be done at the end of the initial period. This cycle of cooling/compression and exercise can be continued several times (cooling/compression time can be reduced after the first cycle unless greater than 30 minutes has passed since the cold was removed).

6. What are the proper application procedures for a cold compression device?

ANSWER:

Instruct the patient: Inform the patient of the expected sensations of cold, burning, aching, and then numbness (analgesia).

Preinspection: Inspect the area to be treated for skin compromise, and assess for intact sensation over the area to be treated.

Equipment needed: Cold compression device; ice; water; if elevation is used, a stack of towels or other material to elevate the patient's leg.

Patient position: Supine.

Procedure: Fill the insulated cold compression container with ice and then water to the levels indicated on the device. Place the cuff directly on the patient's skin surrounding the knee, and secure with the Velcro on the cuff. Position the patient (supporting the leg in elevation as desired). Attach the hose from the container to the cuff and fill the cuff with ice water according to the manufacturer's instructions. When the cuff is full, release the hose. If continual cooling is desired, recirculate the water in the cuff after 15 minutes.

Postinspection: Remove the cuff from the patient's knee and inspect the skin. Redness will be present and may remain for 20 minutes or longer, depending on the patient's activity level or exercises performed. If exercise is to be performed, it should be initiated as soon as possible after the cold compression device is removed (a second or third application of cold compression and elevation may be necessary to renumb the area and reduce any accumulated fluid caused by dependent positioning or exercise).

CASE STUDY 2-6 Cold Immersion

The 17-year-old patient discussed in Case Study 2-2 is now 10 days postinjury. His swelling is significantly reduced although still present with prolonged standing. He is having difficulty performing some resistive and weight-bearing exercises without pain. Upon examination, he demonstrates 25% AROM limitations in all ankle motions, mild to moderate pain with resistance in the directions of eversion and dorsiflexion, and difficulty completing a normal gait cycle (diminished passive ankle dorsiflexion in the terminal stance phase). The anterior and posterior talofibular ligaments, the calcaneofibular ligament, and the peroneal tendons are tender upon palpation.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of cryotherapy?

ANSWER: Yes; pain can be reduced with cryotherapy, allowing for performance of ROM, resistive exercise, and increased weight-bearing tolerance to complete a normal gait cycle.

2. Is the patient appropriate for cryotherapy (i.e., do any of the general precautions or contraindications to cryotherapy apply to the patient or are there any specific considerations regarding application of cryotherapy to this patient)?

ANSWER: Because the patient has received cryotherapy in the form of ice packs during the acute phase of his injury, he should be safe for other forms of cryotherapy.

3. What are the specific goals to be achieved with the use of cryotherapy?

ANSWER: The use of cold-bath immersion will reduce pain, allowing for performance of AROM, light to moderate resistive exercises, and walking with a normalized gait pattern.

4. What specific form of cryotherapy would be appropriate for the patient?

ANSWER: Cold-bath immersion will allow for more uniform cooling of the affected ligaments and tendons.

5. What specific parameters of cryotherapy are appropriate for the patient?

ANSWER:

Type of immersion: Basin filled with lukewarm to cool water and ice cubes.

Duration: 20 minutes or until the area is numb.

6. What are the proper application procedures for cold immersion?

ANSWER:

Instruct the patient: Inform the patient of the expected sensations of cold, burning, aching, and then numbness (analgesia).

Preinspection: Inspect the area to be treated for skin compromise, and assess for intact sensation over the area to be treated.

Equipment needed: Basin deep enough to allow water coverage of the patient's distal tibia, enough water to fill the basin to the stated level, ice cubes.

Patient position: Sitting in a chair so the feet touch the floor.

Procedure: Fill the basin with water and ice cubes to the required level. Placing a neoprene toe cap over the patient's forefoot will provide some insulation to the forefoot and will decrease the patient's discomfort.[158] Instruct the patient to immerse the ankle into the basin and ensure that the malleoli are below the water surface. The area will cool more effectively if the patient moves the limb within the water (convection). After the area is numb, the patient may then remove the limb from the water, dry the area, and perform the desired exercises (ROM, resistance, and gait). When the numbness has worn off or when the patient begins to feel discomfort, reimmersion can occur for 5 to 10 minutes until numbness has again been achieved. This cycle of numbing and exercise can be repeated several times.

Postinspection: The immersed area will be red for 20 minutes or more following the cooling period. Observe the skin for any adverse reactions to cold.

as ethanol and menthol, as the active cold-forming agents. They are applied to the skin overlying the area of injury and pain. One study demonstrated a decrease in pain and a decrease in perceived disability of higher magnitudes when cold gel was compared with placebo gel for soft tissue injuries of the hand, knee, leg, or ankle.[159] The gel was applied on the skin four times daily for 2 weeks. Another study demonstrated a significant reduction in low back pain ratings in a group of patients who received chiropractic adjustments that included Biofreeze application versus adjustments alone.[160] The appeal of these gels is their ease of application, ease of portability, and lack of need for refrigeration. However, actual cooling of the skin or subcutaneous tissues does not occur.

ASSESSMENT OF EFFECTIVENESS AND EXPECTED OUTCOMES

As with any therapeutic technique, the decision to use cold modalities in a therapy program should be based on the treatment's goals. The goals of cold application are determined by the patient and the practitioner after examinations, including the history of the present problem and subjective and objective measures of impairments and current functional status. The following outcome measures can be used in assessing effectiveness with cold:

- Edema—girth measures, volumetrics
- Pain—quantification via pain scales or questionnaires
- Range of motion—goniometric measures
- Functional movements—observation of gait quality or ease of AROM
- Muscle guarding—reflected in joint ROM and muscle flexibility measures

Of course, the decision of which of these measures to use will depend on the body limitation being addressed by the treatment.

Documentation

The treatment parameters, changes in patient response to treatment during and between sessions, and any modifications of the goals or treatment program should be accurately documented. Specific parameters for cold applications include the type of cold agent, treatment duration, site of application, patient position, and use of concurrent compression or elevation. Further descriptions of changes in skin temperature or appearance, quality of edema, and sensation are also documented. Clinical notes include periodic reassessments of the patient's overall functional level, especially in relation to the impairments directly affected by intervention.

Documentation Tips

- Type of cold agent
- Treatment duration
- Site of cold application
- Position of patient for cold application
- Use of concurrent compression or elevation
- Change in skin appearance
- Patient response (i.e., change in pain, edema, ROM)
- Adverse responses

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CHAPTER 3

THERAPEUTIC HEAT

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The use of therapeutic heat (thermotherapy) in the treatment of various conditions has been around for thousands of years. Understanding the physiological and biomechanical principles of therapeutic heat is one of the elements of successful patient treatment. The clinician must be well versed with the various conditions for which these biophysical agents are used and must be aware of the contraindications to the use of therapeutic heat. This text provides the clinician with the tools necessary to have a safe and effective patient outcome.

Warmth is associated with tranquility and relaxation. Heating of injured tissue has been used for centuries for pain relief and reduction of muscle spasm. In physical therapy, locally applied heating modalities are used to promote relaxation, provide pain relief, increase blood flow, facilitate tissue healing, decrease muscle spasm, decrease tissue tightness and joint stiffness, and prepare stiff joints and tight muscles for exercise.[1–30] Several studies[31–36] have examined the frequency of thermal agent use as an intervention in physical therapy treatments. These studies from Australia,[31,32] Canada,[33,34] and England[35,36] indicated that the percentage of daily use of therapeutic heating agents, such as hot packs and paraffin wax, ranged from 36.5% to 95% in various practice settings.

The physiological effects that occur from tissue temperature elevation are included in the rationale for selecting these modalities as part of a therapy paradigm. Elevation of collagen tissue temperature, for example, can alter viscoelastic properties, thus enhancing the effects of passive stretch for increasing range of motion (ROM).[9–11,14–16,23,26–28]

Many thermal modalities are available for tissue heating (Box 3-1). Some heating modalities primarily cause an increase in skin and superficial subcutaneous tissue temperature. Thermotherapy modalities, such as moist heat packs, paraffin wax, and fluidized therapy (Fluidotherapy), are used to:

1. Heat superficial joints, such as the hand, which has little soft tissue covering
2. Cause a heating effect in deeper structures, such as muscle, through reflex mechanisms
3. Heat soft tissue (muscle, tendon, superficial joint capsule) in order to increase its extensibility.

If the goal of intervention is to increase the temperature of deeper tissues, such as the knee joint capsule or

Box 3 ▪ 1 Thermal Modality Options

To ↑ tissue temperature within 1 to 3 cm depth

- Moist heat packs (e.g., hot packs)
- Paraffin wax bath
- Fluidotherapy
- Warm whirlpool
- Microwavable gel packs
- Air-activated heat wraps
- Electric heating pads

To ↑ tissue temperature within 1 to 5 cm depth

- Continuous ultrasound
- Continuous shortwave diathermy

the muscle belly of the quadriceps muscle, then another heating modality is logically selected.

Heating modalities, including continuous shortwave diathermy and continuous-wave ultrasound, can increase tissue temperature at depths ranging from 3 to 5 cm without overheating the skin and subcutaneous tissues. Therapeutic ultrasound is discussed in Chapter 4 and shortwave diathermy is covered in Chapter 6.

BIOPHYSICAL EFFECTS OF TEMPERATURE ELEVATION

Many sequelae can occur as a result of increasing the temperature of body tissues. The occurrence and magnitude of these physiological changes are dependent upon several factors:[1]

1. Extent of the temperature increase
2. Rate at which energy is being added to the tissue
3. Volume of tissue exposed
4. Composition of the absorbing tissue
5. Capacity of the tissue to dissipate heat (largely a factor of blood supply)

To meet therapeutic levels of vigorous heating, study results[1,9,14] indicate that tissue temperature must be elevated to between 104°F and 113°F (40°C and 45°C). Within these temperatures, hyperemia, which is indicative of increased blood flow, will occur. Above this range, there is potential for tissue damage. Below 104°F (40°C), heating is considered to be only mild.[1,9,12–14,37] Behavioral regulation, or how the body responds, and subjective responses associated with surface temperatures are illustrated in Table 3-1.

TABLE 3–1. Behavioral Regulation and Subjective Responses Associated With Surface Temperatures

Temperature °F (°C)	Body and Environmental Temperatures	Subjective Feeling Associated With Surface Temperatures
140 (60)		
131 (55)		
122 (50)		Tissue damage, burning pain
113 (45)		Very hot
104 (40) 95 (35)	Normal range of resting temperature of body 97.3–99.1°F (36.3–37.3°C)	Hot
86 (30)		Warm
77 (25)		Neutral
68 (20)	Region of thermal environmental comfort	Cool
59 (15)		Cold
50 (10)		Very cold
41 (5) 32 (0)	Behavioral regulation	

Key Point! To provide a therapeutic effect, tissue temperature should be elevated to 104°F to 113°F (40°C to 45°C).

The rate of temperature rise in response to the addition of thermal energy can influence physiological responses. Temperature elevation increases local blood flow[2,3,9,12,13,38–46]; thus, cooler blood comes into the area and acts to remove some of the heat produced. If the rate of temperature increase is very slow, the amount of heat added could be balanced out by the convective effect of cooler blood so that therapeutically effective heating levels may not be obtained. On the other hand, if temperature rises faster than excess heat can be dissipated, heat may build up to a point that stimulates pain receptors and may also cause tissue damage. The goal of heating is to achieve a therapeutic level of temperature elevation without causing adverse responses.

Physiological alterations can occur at the site of local temperature rise and in areas remote from the area of heat absorption. Usually, the larger the tissue volume affected by the addition of thermal energy, the greater the

likelihood for reflex, or consensual, changes in other areas and for systematic alterations. An increase in forearm temperature as a result of hot pack application could be expected to cause an increase in local blood flow, with minimal or no alterations in overall peripheral vascular resistance. On the other hand, immersion of a person in a water bath of 104°F (40°C) could result in systemic changes, such as a decrease in mean blood pressure, an increase in heart rate, and an increase in pulmonary minute ventilation.[1]

Several physiological responses to temperature elevation are important to understand when considering a heating modality for therapeutic purposes. The most relevant changes to address include alterations in metabolic activity, hemodynamic function, neural response, skeletal muscle activity, and collagen tissue physical properties. These changes, in part, serve as a foundation for the use of heat as an effective therapeutic modality. An understanding of the adverse reactions to the addition of thermal energy is also imperative for the delivery of a safe intervention.

Metabolic Reactions

Chemical reactions in cells of the body are influenced by temperature. Generally speaking, chemical activity in cells and metabolic rate will increase two- to threefold for each 50°F (10°C) rise in temperature.[43,47] Therefore, energy expenditure will increase with increasing temperature. With even mild increases in tissue temperature, the oxygen-hemoglobin dissociation curve shifts to the right, making more oxygen available for tissue repair.[4] However, as temperature rises past a certain point, usually 113°F to 122°F (45°C to 50°C), human tissues will burn because the metabolic activity required to repair tissue is not capable of keeping up with thermally induced protein denaturation.

Key Point! Metabolic reactions to heat include:

- ↑ Cell activity and metabolic rate (two- to threefold for each 50°F [10°C] ↑ in temperature)
- ↑ Oxygen uptake by tissues

An increase in chemical reaction rate can also have positive effects on human function. Oxygen uptake by tissues will increase.[39] Theoretically, therefore, more nutrients will be available to promote tissue healing.[4,5]

Vascular Effects

Increasing tissue temperature is usually associated with vasodilation, and thus with an increase in blood flow to the area.[1,2,5,8,12,13,37,38–49,50,52] But this general statement can be misleading. It is important to know which regions have increased blood flow. The control mechanisms are different for blood flow to different structures—for example, skin compared to skeletal muscle. Therefore, responses to temperature change will not always be the same, or if a response is in the same direction, it may not be of the same magnitude.

Skin blood flow has an important role both in nutrition and in the maintenance of constant core body temperature of 98.6°F (37°C) and is primarily under the control of sympathetic adrenergic nerves.[37,40,51,53] Vasodilation of resistance vessels of the skin will occur as a means of losing heat through local or reflex mechanisms. The skin is unique in that it has specialized vessels called *arteriovenous (AV) anastomoses,* which have an important role in heat loss.[53] These shunt vessels go from arterioles to venules to venous plexuses, thus bypassing the capillary bed. The blood flow through these anastomoses is under neural control. Activation occurs in response to reflex activation of temperature receptors or stimulation of heat loss mechanisms triggered in part by the circulation of warmed blood through the preoptic region of the anterior hypothalamus. These AV shunt vessels are found in the hands (palms and fingertips), feet (toes and soles), and face (ears, nose, and lips).

Blood flow changes in the skin can be caused by local[1,37,40–42,51,54] or reflex[37,40,41,50] mechanisms. Vasodilation of the heat-exposed skin can occur as a result of three factors:

1. An axon reflex
2. Release of chemical mediators secondary to temperature elevation
3. Local spinal cord reflexes

Heat applied to the skin stimulates cutaneous thermoreceptors. These sensory afferents carry impulses to the spinal cord. Some of these afferent impulses are carried through branches antidromically toward skin blood vessels, and a vasoactive mediator is released. This results in vasodilation through an axon reflex (Fig. 3-1). Petrofsky et al[40,41] also found that moist heat caused significantly higher skin blood flow than dry heat.

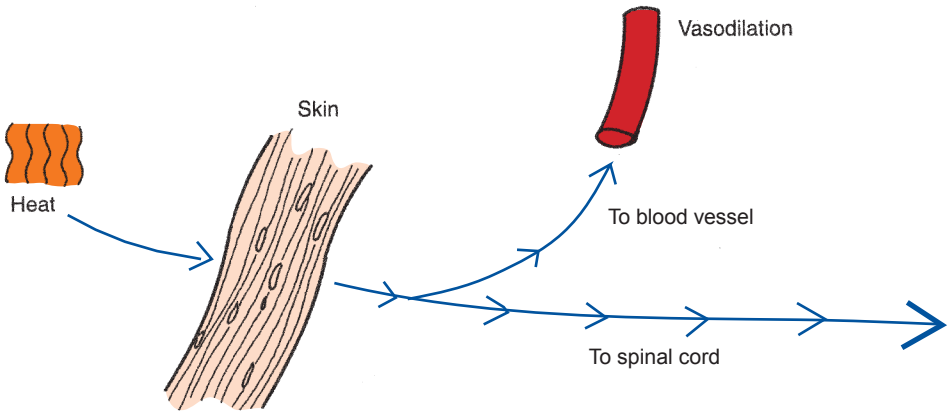

Fig 3 ■ 1 Schematic diagram of an axon reflex.

Heat produces a mild inflammatory reaction. Chemical mediators of inflammation, including histamine and prostaglandins, are released in the area and act on resistance vessels to cause vasodilation (Fig. 3-2). In addition, temperature elevation causes sweat secretion, and the enzyme kallikrein is released from sweat glands. This enzyme acts on a globulin, kininogen, to release bradykinin.[55] Vasodilation of resistance vessels (i.e., small arteries and arterioles) and an increase in capillary and postcapillary venule permeability occur because of the action of these chemical mediators on smooth-muscle tone and endothelial cell contractility, respectively. Because of an increase in capillary hydrostatic pressure and permeability, outward fluid filtration from vascular to extravascular space is favored. Therefore, heat within the therapeutic range can potentially increase interstitial fluid and cause mild inflammation.

A local spinal cord reflex is elicited through heat-activated cutaneous afferent stimulation. This reflex results in a decrease in postganglionic sympathetic adrenergic nerve activity to the smooth muscles of blood vessels.[56] A schematic of the reflex is diagrammed in Figure 3-3.

Vasodilatory effects of this reflex response are not limited to the area heated; rather, there will be a consensual (reflex) response in areas remote from the site of application. When one area of the body (e.g., the lower back) is heated, increases in skin blood flow occur in distal extremities that are not directly heated.[57–59] This principle of reflex vasodilation is considered to be safe to use in patients with peripheral vascular disease (PVD).[57] For example, cutaneous blood flow to the feet could be increased by applying heat to the lower back.

Key Point! Consensual heating to improve circulation in persons with PVD is not a usual rationale for using heat in clinical practice. In this instance, physiological response does not equate with clinical utility.

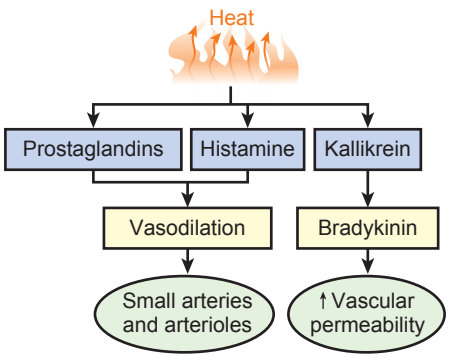

Fig 3 ■ 2 Metabolic effects of heat.

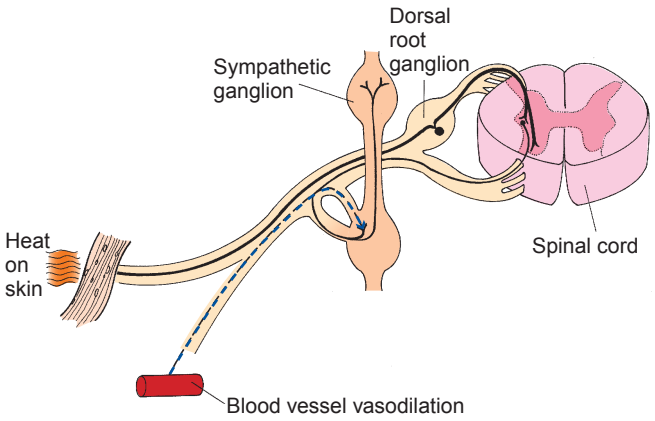

Fig 3 ■ 3 Heat applied to the skin leads to vasodilation. The change in activity of postganglionic sympathetic adrenergic fibers secondary to local heating is diagrammed.

Skeletal muscle blood flow is primarily under metabolic regulation and demonstrates the greatest response to increases or decreases in levels of exercise. When superficial heating modalities are given, minimal change in skeletal muscle blood flow is expected. This notion is supported by two reports on heat lamp (infrared) application. Crockford and Hellon[42] measured venous oxygen content following 20- to 30-minute exposures of the forearm. Superficial venous oxygen content increased, but there was no change in muscle blood flow. Wyper and McNiven[60] reported no change in muscle blood flow following heat (infrared heat lamp) treatment. Similarly, the finding by Robertson et al[14] of the lesser effect of superficial heat compared with deep heat in examining tissue extensibility suggests that the contribution of any skin response or any reflex vasodilation from heating the skin is minimal. Draper and Hopkins[7] measured intramuscular (vastus medialis oblique) and intracapsular (suprapatellar pouch) temperatures of 11 healthy human subjects following 2 hours of application of an air-activated heat wrap for continuous low-level heat therapy. They found significant differences in skin, intramuscular, and intracapsular temperatures compared with control subjects. Okada et al[52] and Hawkes et al[61] both found that muscle blood flow and temperature increased significantly following a 20-minute or 30-minute application of moist hot packs, respectively.

Heat is often used before exercise. Both heat and exercise can increase blood flow. Greenberg[45] compared moist heat packs alone, exercise alone, and moist heat packs plus exercise. Heat was applied for 20 minutes. Exercise consisted of squeezing a rubber ball once per second for 1 minute. The increase in blood flow from exercise was greater than from heat; however, the effects of moist heat packs plus exercise in combination were additive and greater than either modality used alone.

Kauranen and Vanharanta[62] compared the effects of hot and cold packs on motor performance of normal hands. The forearm was placed between two hot packs from elbow to fingers for 20 minutes on 3 successive days. Functional testing followed each heat treatment. The same procedure occurred the next week with cold packs except that the application was for 15 minutes. Reaction time, movement speed, tapping speed, and coordination for upper extremity motor control were measured. Results demonstrated that cold decreased all fine motor tasks. Heat decreased simple reaction time; however, finger-tapping speed was increased. The authors suggest that patients completing fine motor tasks may find it more difficult if cold is applied before executing the tasks compared with heat.[62]

Mayer et al[22] examined the use of continuous low-level heat wrap therapy and exercise in treatment of low back pain. Four treatment groups (heat wrap alone, heat wrap plus exercise, exercise alone, and back pain booklet) were examined daily for 5 days. The heat-plus-exercise group had the heat wrap applied for 1 hour before commencing the directional preference-based exercise program. Results indicated that the combination of low-level heat wrap therapy and directional preference-based exercise during the treatment of low back pain significantly improved functional outcomes and pain relief compared with either intervention alone for control.[22] Johnson and Park[63] found that the onset of vigorous exercise will lead to cutaneous vasoconstriction, the body's method of shunting blood to the muscles. This means that during vigorous exercise, the patient's skin may appear cool to the touch because the blood is being shunted to the skeletal muscles. In general, cold application appears to limit or decrease functional ability, whereas heat may improve or augment physical performance or ability.

Huang et al[64] found that a 20-minute application of hot packs to the knees of patients with knee osteoarthritis (OA) who were undergoing various forms of muscle-strengthening exercises provided significant pain relief, disability reduction, and walking speed after treatment and at follow-up when compared with nonexercised controls. Önes et al[65] demonstrated a decrease in pain, stiffness, and physical function in knee OA patients following 20-minute hot pack and 5-minute continuous-wave ultrasound treatment to the knees prior to five different quadriceps, hamstrings, and stepping exercises compared with exercise-only controls.

In contrast, Warner et al[66] found that a 15-minute application of hot packs to the knee joint did not influence quadriceps function in patients with arthrogenic muscle inhibition.

Neuromuscular Effects

Heat is used therapeutically to provide analgesia[5,8,12,25,50,51,67–69] and to assist in the resolution of pain and muscle-guarding spasms.[1,8,70–74] Although the

mechanisms of action are not totally understood, the underlying basis for use may relate to the ability of heat to elevate pain threshold,[50,75] alter nerve conduction velocity,[76–80] and change muscle spindle firing rates.[5,8,12,71,81] The increased firing rate of thermoreceptors in cutaneous tissue may block input from the primary nociceptive afferents to the dorsal horn (the "thermal gate theory").[5,8,12,71] Kim et al[25] found that temporal changes in pain scale and sensory threshold measurements were changed after 30-minute hot pack treatments in geriatric patients. Their theory is that sensory threshold is expected to be higher if pain is decreased. Sensory threshold was measured using an electrostimulator before and 0, 1, and 24 hours after hot pack application. The total sensory thresholds of the body regions were significantly increased just after hot pack application. Sensory thresholds returned to baseline levels after 24 hours. The authors suggest that reduction of shoulder, low back, and knee pain in these geriatric patients following 30-minute heat application with moist hot packs was due to a combination of self-reported pain scales and increases in sensory threshold.[25] An interesting depiction of the mechanisms of pain relief brought about by heat is provided in Figure 3-4. In addition, temperature elevation of skeletal muscle can temporarily change the ability to build tension and sustain prolonged activity.[83,84]

Raising subcutaneous tissue temperature using a variety of heating modalities has been demonstrated to alter sensory nerve conduction velocity.[77–80] The most pronounced changes appear to occur during the first 2.7°F to 3.6°F (1.5°C to 2°C) temperature increase.[77] However, the relevance of these findings to therapeutic use is not readily apparent. Heating over the area of a peripheral nerve can elevate the pain threshold. Fifteen minutes of high-intensity heat lamp (infrared radiation) was administered over the medial aspect of

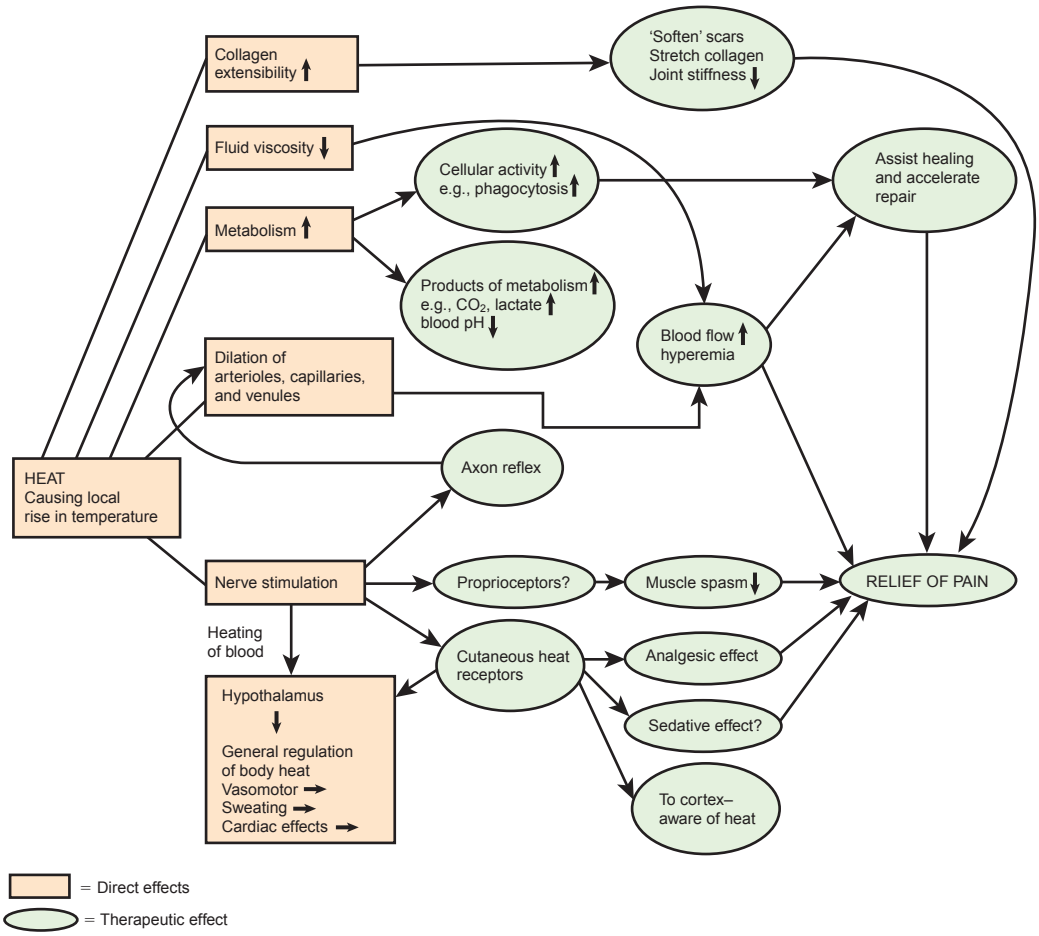

Fig 3 ■ 4 Mechanisms of pain relief due to heat. *(From Wells PW, Frampton V, Bowsher D.* Pain Management in Physical Therapy. *2nd ed. Oxford: Butterworth-Heinemann; 1994, p 154, with permission.)*

the elbow—that is, over the ulnar nerve. Pain threshold measurements distal to the site of application, over the tip of the little finger, revealed analgesia.[75] Direct heating over the area where pain was measured also produced analgesia. Kelly et al[79] found that fluidized therapy (Fluidotherapy) decreased sensory nerve latency of the superficial radial nerve after 20 minutes of application, suggesting analgesic effects at the site of application and at sites distal to the application. The clinical correlate is that heat can be a useful adjunct to reduce pain before stretching exercises, joint mobilization techniques, or active exercise.[1–3,4–6,10,11,15,16,23,26–28,37,51,83–88] Following proper instruction, including written instructions, heat can easily be used on a home basis prior to exercise.

Nadler et al[87] and Davis et al[88] suggested that some of the benefits of topical heat therapy may be mediated directly in the brain. Functional brain imaging has shown central effects of non-noxious warming of the skin with increased activity of the thalamus and posterior insula of the brain, supporting the beneficial psychosomatic effects of heat.

Muscle-guarding spasms can result from the overuse of a muscle during exercise or from activation of a protective mechanism to guard against movement of painful joints. Pain can be the event triggering a reflexive, tonic muscle contraction, thus beginning the pain-spasm-pain cycle.[1,4,5,12,13,69,89,90] The muscle spindle afferents that alter their rate of firing, primarily in response to tonic or static stretch, are the type II afferents. Elevation of muscle temperature to about 107.6°F (42°C) will decrease the firing rate of the type II afferents and increase the firing of the type Ib fibers from Golgi tendon organs (GTOs).[1,5,12,13,81] Therefore, with decreased firing of the type II afferents and increased GTO activity, we could predict a decreased firing of the alpha motoneuron and thus a reduction of tonic extrafusal fiber activity.

Heating modalities, such as moist heat packs, likely will not elevate muscle temperature to the degree necessary to alter type II or type Ib activity. Therefore, another mechanism must be postulated to account for the reduction in muscle spasm when the skin overlying the muscle is heated. Heating the skin has been demonstrated to produce a decrease in gamma (γ) efferent activity and sensory nerve action potential latencies.[44,79] With a decrease in γ activity, the stretch on the muscle spindle would be less, thus reducing afferent firing from the spindle. This indirect method ultimately results in decreased alpha (α) motoneuron firing, and thus less muscle spasm. Another theory for the mechanism reducing muscle tension comes from Kettenmann et al.[69] They used objective electroencephalogram measurements in patients with acute low back pain and found an acute therapeutic relaxation after wearing a back heat wrap for a minimum of 4 hours. The authors believe this reduction of pain and muscle tension was due to a decrease in nociceptive information load.

Heating modalities have also been used to help prevent or decrease the effects of delayed-onset muscle soreness (DOMS) after exercise.[22,29,70] Sumida et al[70] found the pain of eccentric-induced DOMS was reduced 30 minutes after hot pack application to the skin overlying the painful muscle compared to cold pack and control groups. They suggested the reason for the decrease in the visual analogue scale pain rating was because heat is soothing and produces feelings of comfort.

Mayer et al[22] examined the use of a wearable heat wrap on the low back region after eccentric exercise induced DOMS, using a prevention group, a treatment group, and control groups. For the prevention group, they found that 4 hours of wearing the heat wrap before exercise decreased pain intensity, disability, and self-reported physical function by 47%, 52%, and 45%, respectively. In the treatment group, pain relief was 138% greater at 24 hours, postexercise compared with the cold pack group. Mayer et al[22] suggested that the efficacy of heat wraps in preventing and decreasing low back DOMS is due to a combination of the heat wrap's thermal effect on muscle tissue, the analgesic properties of topical heat, and the fact that the subjects can remain active while wearing the heat wrap.

Petrofsky et al[29] used three different superficial heating agents to treat DOMS in patients with type 2 diabetes. The study included 120 subjects (young, older, and with type 2 diabetes) who completed a P90X exercise video for core fitness. Three heating modalities—heat wrap, hydrocollator pack, and chemical moist heat wrap—were assessed on how well they could reduce muscle soreness. Results indicated that the people with diabetes were more sore than the age-matched controls after the exercise bout. The greatest reduction in soreness after applying the modalities was moist heat, both immediately after application and up to 2 days, postexercise. All groups had decreased abdominal muscle soreness after superficial heat was applied.[29]

Elevating muscle temperature can also alter strength and endurance. In a study of normal volunteers, Chastain[82] used a deep-heating modality (continuous shortwave diathermy) over the quadriceps. During the first 30 minutes after terminating the heat, isometric strength was decreased, followed by an increase for the next 2 hours of measurement. Strength and endurance decreases following heating have also been reported in other studies on humans.[83,90] Immediately after immersion in whirlpools ranging in temperature from 104°F to 109.4°F (40°C to 43°C), quadriceps strength and endurance were reduced.[90] Edwards et al[83] found similar results following immersion of the lower extremity in a water bath warmed to 111.2°F (44°C) for 45 minutes. The muscle temperature after the 45-minute immersion had reached 101.3°F (38.6°C) from a normal mean of 95.2°F (35.1°C). In contrast, Warner et al[66] found a lack of effect of superficial heat to the knee on quadriceps function in individuals with arthrogenic muscle inhibition.

Key Point! The practitioner should be aware of the changes in muscle performance following heat (and cold) application, particularly when planning strengthening programs or performing valid assessments of performance such as manual muscle testing.

Connective Tissue Effects

Temperature elevation in combination with a stretch can alter the viscoelastic properties of connective tissues.[7,9–11,13–16,44,91–93,95,96] The viscous properties of connective tissue permit a residual elongation of connective tissue after the stretch is applied and then released. This is referred to as *plastic deformation*, or *elongation*.[97] An elastic structure will stretch under tension but will return to its original length when the load is removed. The elastic properties of connective tissue result in recoverable deformation.[96–98]

Key Point! The effects of heat on connective tissue include:

- ↑ Elasticity
- ↓ Viscosity
- ↓ Joint stiffness
- ↑ Muscle flexibility

Connective tissue will progressively shorten and joint contractures will develop following injury if full ROM exercises are not performed.[99] Adhesions, or the loss of ability of tissue layers to glide past one another, will also develop. Lacerations and crush and burn injuries result in scar tissue and often limit mobility.

Heat and stretch of connective tissue will cause plastic elongation.[96,97,100,101] Two factors must be considered in determining effective intervention strategies:

1. Temperature elevation—site, time, and amount
2. Stretch—duration, amount, and velocity

Greater residual length changes will occur when a stretch is applied during the time the tissue temperature is elevated at therapeutic levels (between 104°F and 113°F [40°C and 45°C]).[10,11,14,15,23,37] For in vivo experiments on rat tail tendon, temperature was elevated in a water bath of 113°F (45°C) for 10 minutes, and then elongation was performed and maintained until cooling to resting values occurred.[100] This was compared with stretch in a water bath of 77°F (25°C). Length increases were greater in the 113°F (45°C) bath, with less evidence of tissue damage.[101]

Bleakley and Costello[23] and Nakano et al[27] completed systematic reviews on the effects of thermal agents on range of movement and mechanical properties in soft tissues. They concluded that heat provides an added benefit on stretch-related gains of ROM and is an effective adjunct to developmental and therapeutic stretching techniques. Leung and Cheing[10] compared 20-minute superficial (hot pack) and deep (shortwave diathermy) treatments prior to stretching exercises for patients with frozen shoulder. They found a significant improvement in all groups in all outcome measures except for shoulder flexion range. The improvement in the shoulder score index (activities of daily living) and ROM was significantly better in the deep-heating group than in the superficial-heating group. Similarly, Knight et al[96] compared the effects of superficial heat (hot packs for 15 minutes), deep heat (continuous ultrasound, 1.5 W/cm^2 for 7 minutes), and stretch alone (control) on the extensibility of the plantar flexors. They found heat prior to stretch increased both active ROM (AROM) and passive ROM (PROM) compared with control, and the deep-heat group obtained the greatest increases in AROM and PROM. Lee and Ng[26] found that sustained stretch of the hamstring muscles for 30 seconds following a 20-minute hot pack application

led to greater relaxation of the hamstring muscles than stretching alone in severely mentally challenged children with accompanying spastic and/or dystonic hypertonia and hamstring tightness.

Three techniques are reported to provide for permanent elongation of collagen tissue:

1. Constant load of enough magnitude to overcome tissue elasticity
2. Rapid stretch followed by a period of holding in that position
3. Constant rate of stretching using a slow, steady stretch[37,96,97,102]

Lower loads of longer duration result in less tissue damage[38,97,98,102] and greater increases in joint ROM.[38,94,97–99,101] Lentell et al[38] demonstrated an increase in long-term improvement in shoulder flexibility when their subjects were treated with moist heat packs prior to low-load, prolonged stretch. Two groups (ice and stretch and heat and stretch) showed an improvement in shoulder flexibility compared with controls. However, only the heat-and-stretch group showed significant gains in shoulder flexibility compared with those who received stretching alone.[38]

Joint stiffness is a common complaint among patients with rheumatoid arthritis (RA) and OA (degenerative joint disease). Joint stiffness has the physical components of elasticity, viscosity, inertia, plasticity, and friction. Joint stiffness in normal subjects and in patients with RA is mainly attributable to the elastic properties of joint capsular structures.[6,103] Following immersion of the hands in a water bath of 109.4°F (43°C) for 10 minutes, there was a slight decrease in finger joint stiffness.[93] Heating of the hand to 113°F (45°C) with a heat lamp (infrared) resulted in a 20% reduction in metacarpophalangeal stiffness compared with heating to a temperature of 91.4°F (33°C).[96]

A controlled pilot study involving patients with RA was conducted to compare and assess the effects of ice versus heat on shoulder pain and limited mobility.[86] The heat-treated group showed a greater increase in shoulder abduction and flexion than the ice-treated group, although this difference was not statistically significant. In a systematic database search, Ayling and Marks[6] found paraffin wax applications resulted in significant improvements in rheumatoid arthritic hand function when followed by exercise. Similarly, Robinson et al[21] concluded that thermotherapy can be used as a palliative therapy or as an adjunct therapy combined with exercises for patients with RA and that wax baths appear especially helpful in the treatment of arthritic hands. Dilek et al[24] found that paraffin bath therapy seemed to be effective in reducing pain and tenderness and maintaining muscle strength in hand OA. Clearly, heating can result in decreased joint stiffness and increased tissue extensibility, thus facilitating ease of motion and gains in ROM.

PHYSICAL PRINCIPLES OF HEAT

Heat flow through matter (tissues) varies with the nature of the material (type of tissue) and is called *thermal conductivity*.[51] Changes in surface tissue temperature from heating modalities depend on the intensity of the heat applied, the time of heat exposure, and the thermal medium (product of thermal conductivity, density, and specific heat) for surface heat.[92] The greatest degree of temperature elevation with heating modalities occurs in the skin and the subcutaneous tissues within 0.5 to 2 cm of the skin surface.[104] In areas of adequate blood supply, temperature will increase to a maximum within 6 to 8 minutes of exposure.[39,47,75] Muscle temperature at depths of 1 to 2 cm will increase to a lesser degree and will require a longer duration of exposure (15 to 30 minutes) to reach peak values.[39,49,75,104] At a depth of 3 cm, using clinically tolerable intensities, muscle temperature elevation can be expected to be about 1.8°F (1°C) or less.[13,15,39,104] Recently Hawkes et al[126] found a 30-minute application of a moist hot pack to the calf muscle increased muscle temperature by 5.07°F (2.82°C) at a depth of 1 cm as measured by an implantable thermocouple.

In joints of the hand and wrist or foot and ankle, with relatively little soft tissue covering, heating modalities can raise intra-articular temperatures.[3,105–107] In fact, a 20-minute exposure of the foot to dry heat Fluidotherapy at 118°F (47.8°C) was shown to increase joint capsule temperature in the foot.[3] Even though there can be a reflex vasodilatory response on the unheated opposite extremity, no reflex temperature changes would be expected to occur.[107]

After the peak temperature is reached, there is a plateau effect or a slight decrease in skin temperature over the remainder of the heat exposure.[39,45,49,75,126] In contrast, a study by Kelly et al[79] using Fluidotherapy found that skin temperature remained significantly above pretreatment levels 20 minutes after completion of the treatment. Typical temperature responses of areas with intact circulation are shown in Table 3-1.

Fat provides insulation against heat; it has a low thermal conductivity (see Table 3-1). Therefore, tissues under adipose tissue are likely to be minimally affected by heating modalities. In a series of experiments, Petrofsky and Laymon[127] and Petrofsky et al[128] determined that in subjects with high body fat (exceeding 25% of their weight), heat transfer to muscle using superficial heat (moist hot packs) was significantly impaired. In individuals who are overweight, heat transfer from the hydrocollator pack accumulates more in the skin (due to the increased amount of adipose tissue), causing a much greater increase in skin temperature and a lower increase in muscle temperature.

To elevate deep tissues to therapeutically desired levels without burning the skin and subcutaneous tissue, a heating modality such as continuous ultrasound or shortwave diathermy should be selected.

Heat Transfer

The primary methods of heat transfer for heating modalities are conduction, convection, and radiation.[1,38,104] *Conduction* is a method of heat transfer where the kinetic motion of atoms and molecules of one object is passed on to another object. This kinetic motion, often described as "atoms jostling one another,"[51,104] is increased when one object is heated more than another and occurs more effectively if the objects are solids.[51,104] More details on principles of conduction are presented in Chapter 2.

Convection is the bulk movement of moving molecules, either in liquid or gaseous form, such that heat is transferred from one place to another.[51,104] The fluid movement can be pumped, such as blood within the body is pumped by the heart and warms all the parts to which it travels, or movement may occur because a heated liquid or gas, being less dense, floats upward.[51,104]

Radiation is the conversion of heat energy to electromagnetic radiation.[51,104] All objects at temperatures above absolute zero (–460°F [–273°C]) both emit and absorb radiant energy. Any heated object or element, such as an infrared heat lamp, gives off radiant heat. If an object or body part is brought close enough to the radiant energy source, heat will be absorbed. Radiant heat application using infrared lamps is rarely, if ever, used today in rehabilitation as a form of heating and therefore will not be discussed further.

Conductive Heat Modalities

Moist heat packs and paraffin wax baths are commonly used heating modalities for clinical use. For home use, many patients use electric heating pads, microwavable heat packs, air-activated heat wraps, and paraffin wax baths. These modalities transfer heat to the body via conduction because they are in contact with the skin and are at a much higher temperature than the skin surface to which they are applied. Therefore, thermal energy is lost from the modality and gained by the tissues. The quantity of heat gained and the subsequent physiological responses to that heat gain are dependent upon several factors, including but not limited to:

1. Thermal conductivity of the tissues
2. Body volume exposed
3. Time of exposure

Moist Heat Packs or Hot Packs

Moist heat packs provide a moist heat. Commercial moist heat packs consist of canvas or nylon cases filled with a hydrophilic silicate or some other hydrophilic substance or sand (Fig. 3-5). Moist heat packs are stored in a thermostatically controlled cabinet in water at a temperature between 158°F and 167°F (70°C and 75°C).[1,49,51,104] Moist heat packs come in a variety of shapes and sizes and should be chosen on the basis of the size and contour of the body part(s) to be treated.

The hot pack should be of the appropriate size to totally cover the intervention area and should be secured in place (Figs. 3-6 and 3-7). The pack should not be secured so tightly that the patient cannot remove it if it becomes too hot. The pack should be covered with layers of terry cloth toweling or commercial hot pack covers.

Fig 3 ■ 5 Two commercially available hot packs. These are heated in a thermostatically controlled unit.

Although there appears to be no definitive number of layers of toweling for wrapping moist heat packs, the consensus is about six to eight layers, depending on towel thickness. Commercial hot pack covers often need another layer or two of toweling to ensure adequate insulation from the hot pack.

Key Point! The quality of the towels used with a heat pack should be considered. Because air acts as an insulator, newer fluffier towels hold more air to insulate, thus retaining heat. Worn, thin towels will hold less heat and thus dissipate heat sooner.

As with all forms of heating modalities, the patient should feel only a mild to moderate sensation of heat during application; the old adage "the hotter the better" could result in skin burns. A significant early change in skin color may suggest overheating. Fair-skinned individuals may turn bright pink-red[40] or blotchy red and white; however, darker-skinned individuals may exhibit areas of darker and lighter color. Fyfe[49] suggests frequent monitoring of the patient until about the 9- to 10-minute mark after warmth is first perceived when the maximum heating begins to dissipate. If the pack feels too hot to the patient or the therapist detects distinct skin color change, more toweling should be added or the hot pack should be removed.

Fig 3 ■ 6 Hot pack application to the neck prior to exercise.

Fig 3 ■ 7 Hot pack application to the lower back before soft tissue mobilization and exercise. Note that the patient is in the prone position. If indicated for patient comfort, a pillow may be added under the abdomen.

Key Point! The practitioner should monitor the patient during hot pack application. After about 5 minutes, it is advisable to observe the skin color under the hot pack and obtain the patient's subjective feelings about the amount of perceived heat.

Patients should be advised not to lie with their full body weight directly on top of moist heat packs, particularly when the intended intervention area is on the trunk. Body weight will squeeze water from the pack and may accelerate the rate of heat transfer. In addition, local circulation could be reduced through compression of vessels, thus reducing circulatory convective cooling (dissipation of the heat). Both factors could cause overheating of the skin. Along this same line, the use of weights, such as sandbags, to hold the packs in position can potentially create hot spots in the patient's tissues.

If moist heat packs are recommended to be used at home by caregivers or reliable patients, adequate instructions should be provided. There are a variety of methods for using moist heat at home by patients or caregivers, including commercial moist heat packs (hydrophilic silicate) and small water tanks for storage; sand packs, which can be heated either in water or in a microwave oven; and gel packs, which can also be heated in water or in a microwave oven. A common rubber hot-water bottle covered with moist toweling will also suffice.

All forms of moist heat packs should be inspected regularly for leaks and should be discarded if leaking occurs. When hydrophilic silicate moist heat packs become old and worn, they leak the paste-like material within them, and they should be discarded as should gel packs that leak.

Clinically, moist heat packs appear to be used most often to help reduce pain and muscle spasm and to help improve tissue extensibility.[1,4,5,8,10,15,18,37,50,51,72,86,96,108,129] The moist heat they provide appears to rate quite highly among patients relative to their comfort, heating ability, and effectiveness.

CASE STUDY 3-1 Therapeutic Heat for Low Back Pain

Your patient is a 48-year-old male with chronic intermittent low back pain caused by unloading boxes from the back of his truck 22 months ago. He is the owner of a local courier company and spends his day driving a 2-ton delivery truck and loading and unloading materials. He usually wakes up stiff in the morning and does mobilizing and stretching exercises at home before going to work. He finds by the end of the day that he is stiff and sore both from lifting and from sitting while driving. His most recent diagnostic imaging from 6 months ago indicates early degenerative changes in the lumbar spine (L2 through L5) but with no central canal or foraminal stenosis. He has come to physical therapy with an aim to decrease his pain and improve his limited motion. Examination reveals approximately 30% restriction of lumbar motion in flexion and side-bending directions when standing, limited by pain and a sense of tightness in the low back muscles. Sitting ROM is similar to standing. He has moderate muscle spasm throughout his lumbar region and some tenderness on deep palpation. He exhibits some pain and limited motion with joint play testing of the lumbar vertebra. Neurological screen is negative. This patient is somewhat overweight, with a BMI of 28. He has a large waist with a noticeable anterior "paunch."

CLINICAL DECISION-MAKING

1. Does the patient have an impairment, activity limitation, or problem that can be improved or alleviated with the use of therapeutic heat?

ANSWER: The patient demonstrates decreased AROM, decreased passive intervertebral joint play, and increased pain with palpation. Application of therapeutic heat is indicated for all of these signs and symptoms and can be an effective and complementary adjunct to the complete rehabilitation plan.

2. Is the patient appropriate for therapeutic heat? Do any of the general precautions or contraindications to therapeutic heat apply to the patient, or are there specific considerations regarding the application of therapeutic heat to this patient?

ANSWER: The patient's history, signs, and symptoms are appropriate for the use of therapeutic heat. Contraindications in this situation include treatment over areas of recent or potential hemorrhage, malignancy, acute inflammation, infection, poor thermal sensitivity, or where heat rubs have recently been applied. However, given this patient's weight and waist circumference, caution should be used when applying heat to areas with increased adipose tissue because fatty tissue is not a good conductor of heat and a burn could result. Extra caution should be taken with this patient.

3. What are the specific goals to be achieved with the use of therapeutic heat?

ANSWER: Goals of treatment include decreasing pain, decreasing muscle spasm, increasing ROM, and increasing function without pain.

4. What specific aspects of therapeutic heat would be appropriate for this patient?

ANSWER: Options for therapeutic heat include moist heat pack, warm whirlpool, electric heating pad, and low-level continuous heat wrap. Moist heat pack is easy to use and may deliver more heat to deeper tissues than the electric heating pad. The heat wrap can deliver heat over a long duration and may allow a person to be more active during the treatment. Paraffin wax and Fluidotherapy are not practical options for treatment of the lumbar spine.

5. What are the proper application procedures for therapeutic heat?

ANSWER: Any position with the spine unloaded and in a neutral position should be fine. Supine with hips/knees flexed and resting on a bolster is a good option for most people (it is comfortable, with decreased stress on the low back, but it requires more frequent skin checks because the body weight can compress the vessels in the back that would otherwise dissipate the excess heat). Lying prone may put the patient's spine into too much extension; however, this can be corrected with pillows under the abdomen. The size selected of the moist heat pack, electric heating pad, or continuous heat wrap should be appropriate for the size of the area to be treated. A moist heat pack should be wrapped in at least six to eight layers of toweling to provide safe and adequate heating. More layers of toweling are required if the patient is to lie supine on the hot pack. Instruct the patient to contact you if the heat reaches an uncomfortable level. Return to check on the patient after 5 minutes. If the pack is too hot to the patient or the therapist detects distinct changes in skin color, more toweling should be added or the hot pack should be removed. Following treatment, always inspect the skin. Application time: between 10 and 30 minutes for moist heat pack or electric heating pad and up to 8 hours for continuous heat wrap.

Compared with other heating modalities or other modalities used to treat pain, moist heat packs fare well but not significantly better. Several studies[10,15,21,86,93,94] indicated that, although the moist heat packs decreased pain and muscle spasm and improved ROM, they were not significantly better than other modalities. Williams et al[86] found that moist heat packs did improve ROM at the shoulder more than ice, although not significantly. Interestingly, however, most of the patients in the ice intervention group asked if they could be treated with heat instead of ice after the study was completed. Leung and Cheing[10] found that hot packs did increase shoulder function, ROM, and pain relief; however, the shortwave diathermy group fared better in all three measurements. Similarly, Minton[15] found that both hot packs and ice packs improved straight-leg hip ROM following application to the hamstring muscles, with no significant difference between them. However, the increase in ROM following hot pack application was deemed to be due to the increase in tissue temperature and increased extensibility of collagen as well as a relaxing psychological response. It was felt that the cryotherapy increased ROM because of its inhibitory effect on muscle spasm and pain, yet these were all normal subjects with no known pathology.[15] Cetin et al[108] found that moist hot packs combined with transcutaneous electrical nerve stimulation (TENS) reduced pain, increased exercise performance, and improved function in women with knee OA. Many practitioners use the combination of superficial heat and TENS to help modulate pain. (See Box 3-2 for advantages and disadvantages of using moist heat packs.)

Box 3 ■ 2 Advantages and Disadvantages of Using Moist Heat Packs

Advantages

1. Ease of preparation and application
2. Variety of shapes and sizes available
3. Moist, comfortable heat
4. Relatively inexpensive to purchase and replace (assuming a tank is already owned)

Disadvantages

1. No method of temperature control once applied to patient
2. Does not readily conform to all body parts
3. Sometimes awkward to secure in place on a patient
4. Does not retain heat for longer than about 20 minutes
5. A passive intervention; patient exercise cannot be performed simultaneously
6. May leak and then must be discarded (hydrophilic or gel packs)

Paraffin Wax

Paraffin wax has several physical characteristics that make it an efficient source of heat. First, it has a low melting point of about 129°F (54°C). This can be lowered further by adding more paraffin oil or mineral oil so that the wax remains molten at temperatures between about 113°F and 129°F (45°C and 54°C). This molten state allows for a more even distribution of the wax around the part to be treated (usually distal extremities). Second, paraffin has a low specific heat, which means that it does not feel as hot as water of the same temperature; therefore, there is much less risk of a burn. Third, it conducts heat more slowly than water at the same temperature, thus allowing the tissues to heat up more slowly while decreasing the risk of a burn. This is particularly important when treating patients with sensitive skin or diminished skin sensation—for example, following burns.[84,107]

Paraffin is most commonly used for the distal extremities, including the fingers, hand, wrist, and perhaps elbow in the upper limb and the toes, foot, and ankle in the lower limb. The mixture of a paraffin wax (six or seven parts) and oil (one part) is commercially available and is melted and stored for use in thermostatically controlled stainless steel or plastic containers. These wax baths come in a variety of sizes; the smaller ones are ideal for patient use at home (Fig. 3-8).

There are two principal techniques of application:

1. Dip and wrap
2. Dip and reimmerse

Dip-and-wrap is the more practical of the two options. For both methods, the extremity to be treated should be washed and dried and all jewelry should be removed from the part. If a ring(s) cannot be removed, it should be covered with a piece of adhesive or surgical

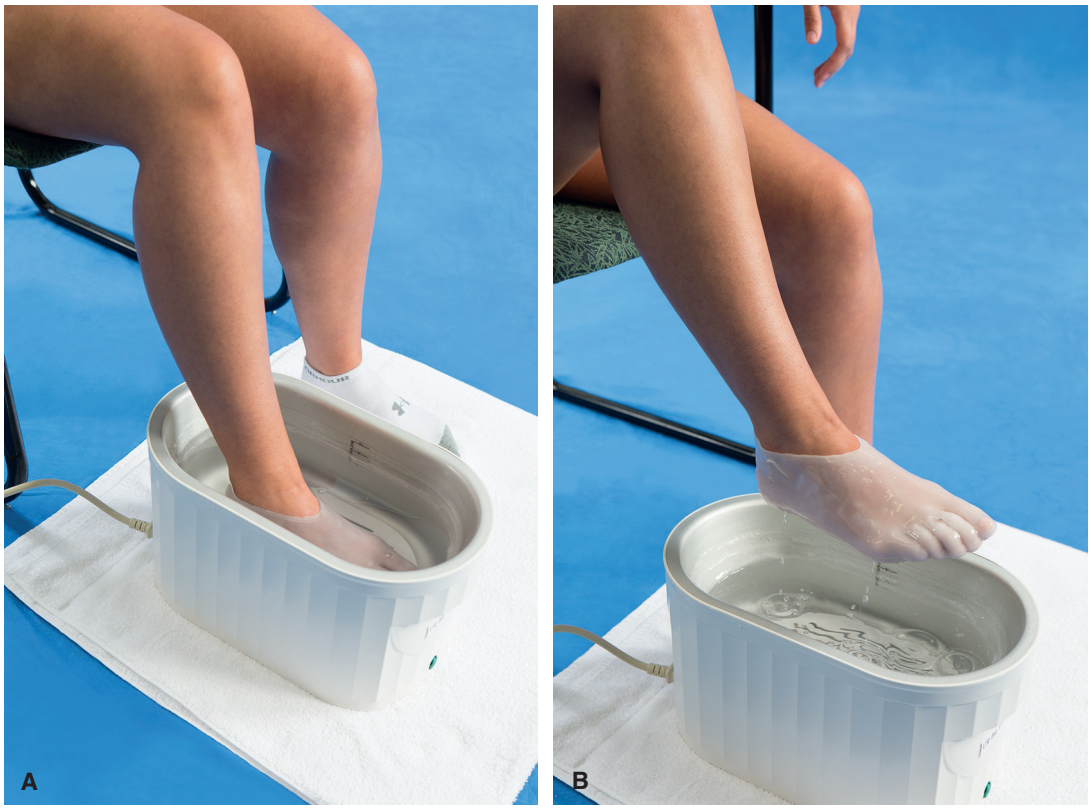

Fig 3 ■ 8 (A) Application of paraffin to the foot and (B) after dipping.

tape to prevent the wax from getting trapped in its crevices. When treating the hand and wrist, for example, the fingers should be slightly spread apart, the wrist relaxed, and the hand and wrist dipped into the wax to a few centimeters above the wrist joint. The hand is then removed from the wax and held above the bath until the wax has stopped dripping and the wax becomes opaque; then the hand is dipped again (Fig. 3-9).

The patient should be reminded not to move the hand and fingers so as not to break the seal of the glove being formed. This procedure is repeated about 8 to 10 times until a solid wax glove has formed around the fingers, hand, and wrist. The hand is then placed in a plastic bag and wrapped with a towel to help retain the heat. The end of the wrapped towel should be folded over to close the end of the extremity. Otherwise, the wrapped towel essentially makes a chimney and heat can escape. If there is a potential for edema to increase secondary to the heat, the part should be elevated above the level of the heart until the intervention time is over. Treatment time is usually 15 to 30 minutes.[24,104,124,125]

With the dip and reimmerse technique, after the wax glove has formed, the area covered by the glove is put back into the wax bath and kept there for the duration of the heat intervention (about 10 to 20 minutes). The most vigorous responses with respect to temperature elevation and blood flow changes will occur with the dip-and-reimmerse technique.[104,110-112,130] This technique is not well suited for most patients who are predisposed to edema or who cannot sit comfortably in the position required for intervention. This technique also precludes other patients from using the wax bath during that time period. If there are potential intervention areas that are not amenable to either of these methods, the wax can be applied with a paintbrush, using up to 10 coats of wax.

When using paraffin to improve skin pliability over healed burn areas, a temperature of 116.6°F (47°C) has been suggested.[84] Paraffin also lubricates and conditions the skin because of the mineral oil content.[113] This can be particularly useful when treating a scarred skin area. Using wax and stretching, Head and Helms[109] demonstrated a maintainable average increase in ROM

Fig 3 ■ 9 Application of paraffin to the hand. (A) Dipping; (B) with wax glove after multiple dips; (C) after wax removal, the patient can do light grip exercises before discarding wax.

of 7° to 10° in the joints of patients with burn scars. If wax is applied over a skin-grafted area, the graft should be stable and nonfragile, and the application should occur at least 10 days, postgraft.[109] Intervention is daily for 2 to 3 weeks.

Paraffin baths are often used as part of an intervention program in patients with RA. Wax is used in the nonflare phases to decrease pain and increase tissue extensibility. Dellhag et al[114] found wax baths to be an effective intervention for this population. Although they discovered no significant therapeutic effects with wax bath interventions alone, there was a significant improvement in stiffness, ROM, and grip function when the wax intervention was followed by active exercise. In a Cochrane review, Robinson et al[21] suggested paraffin wax baths combined with exercises can provide beneficial short-term effects for rheumatoid arthritic hands. Myrer et al[130] found that adding a topical analgesic (e.g., Joint-Ritis; 20% by weight) to a paraffin wax bath (dip-and-reimmerse method for 15 minutes) produced significant pain relief at rest and during movement in patients with hand OA.

Paraffin baths have been used in patients with systemic sclerosis (scleroderma). Sandqvist et al[115] found that after 1 month of daily treatment, paraffin hand baths significantly improved finger flexion and extension, thumb abduction, wrist flexion, and perceived stiffness and skin elasticity over the baseline values of the nontreated (control) hand.

Relative to other heat modalities, paraffin wax may not be significantly better at decreasing pain or increasing joint ROM. Hoyrup and Kjorvel[85] compared whirlpool and wax interventions for hand therapy. They measured hand volume, ROM, and level of pain immediately before and following 3 weeks of intervention. Although all subjects showed significant improvements in ROM as well as decreased pain levels, no significant differences were found between the modalities.

Paraffin should not be applied over open wounds because of the risk of burning the tissues. Patients with infected skin lesions should not use wax for fear of exacerbating the lesion. When contagious skin conditions or warts are present, the area is covered with a bandage or some form of plastic skin film prior to immersion in the wax bath; otherwise, the wax bath could become contaminated. See Box 3-3 for advantages and disadvantages of paraffin wax.

Electric Heating Pads

A method of applying a low-level heat over a long period of time (e.g., hours) has been available for years in the form of electric heating pads. The disadvantages of electric heating pads are that the patient must be near an electrical outlet, and some of these pads may heat up enough to produce superficial burns.

Electric heating pads are primarily used at home for temporary pain relief. These pads are usually square but also may be shaped like a cervical moist heat pack. The pads may have an adjustable intensity control. Electric heating pads should not be used during sleep in case the pad is inadvertently left on, risking excess tissue heating and skin burn. See Box 3-4 for advantages and disadvantages of electric heating pads.

Box 3 ▪ 3 Advantages and Disadvantages of Paraffin Wax

Advantages

1. Low specific heat allows for application at a higher temperature than water without the risk of a burn.
2. Low thermal conductivity allows for heating of tissues to occur more slowly, thus reducing the risk of overheating the tissues.
3. Molten state allows for even distribution of heat to areas like fingers and toes.
4. First dip traps air and moisture to create more even heat distribution.
5. Oils used in the wax add moisture to the skin.
6. Wax remains malleable after removal, allowing for use as an exercise tool.
7. Paraffin provides a comfortable, moist heat.
8. Replacing the wax is relatively inexpensive (assuming bath is already owned).

Disadvantages

1. Paraffin wax is effective only for distal extremities in terms of ease of application.
2. The most effective method of application is the bath method, which limits accessibility for other body parts to be treated effectively.
3. There is no method of temperature control once applied.
4. The heating lasts only about 20 minutes.
5. It is a passive intervention; patient exercise cannot be performed simultaneously.

Box 3 ■ 4 Advantages and Disadvantages of Electric Heating Pads

Advantages

1. Readily available for purchase at a reasonable cost for long-term use
2. A convenient method of heat application at home to be used prior to exercise
3. Provide a comfortable dry heat sensation

Disadvantages

1. Can cause skin and subcutaneous tissue burns if patient inadvertently falls asleep with the pad turned on
2. Patient must be near an electrical outlet during use
3. A passive intervention; patient exercise cannot be performed simultaneously

Air-Activated, Wearable Heat Wraps

Commercially available wearable heat wraps that are air activated can be worn for up to 8 hours at a time. These heat wraps are made of cloth embedded with multiple discs made of iron powder, activated charcoal, sodium chloride, and water. The discs are spaced throughout the cloth's application surface; when the wrap is removed from its sealed pouch and exposed to oxygen, the discs oxidize, producing an exothermic reaction and thus heat.[13] These wearable heat wraps maintain a temperature of about 104°F (40°C), elevate tissue temperature, and can be worn during activities of daily living and work and during sleep. The wraps are available in different sizes and shapes to accommodate body size and contour (Fig. 3-10).

Improved trunk flexibility, reduced pain, and less muscle stiffness in patients with lower back pain were reported by Nadler et al[12] in a trial of continuous low-level heat (e.g., 8 hours of continuous wear) using air-activated heat wraps (Fig. 3-11). This study showed that the effects were greater with the heat wrap usage than with administration of oral placebo medication.[12] Another study showed similar effects when the heat wrap therapy was compared with ibuprofen and acetaminophen.[116] This study was performed on patients with acute lower back pain; therefore, it is unlikely that loss of motion was due to adaptive shortening of connective tissue. The positive results could be attributed in part to

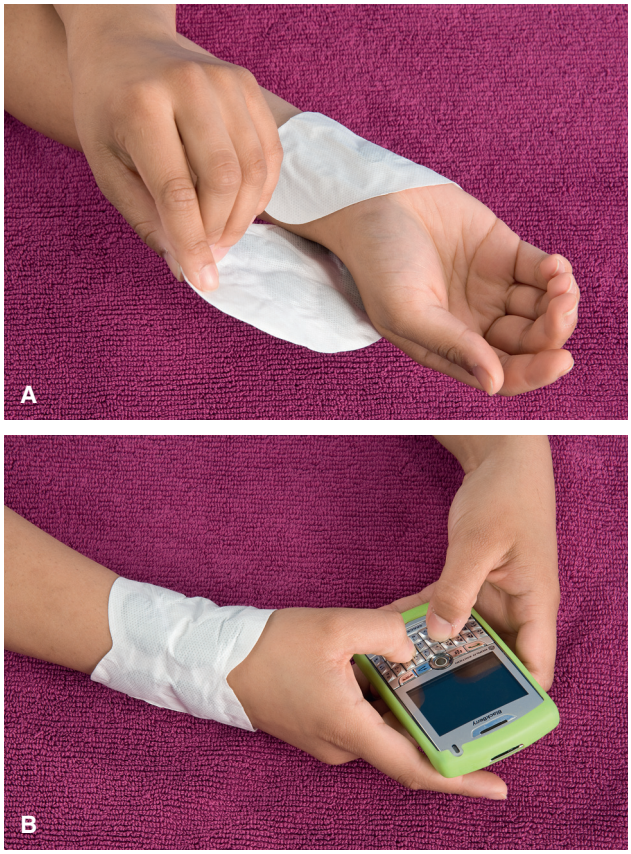

Fig 3 ■ 10 Air-activated heat wrap. (A) Wrap being applied to the wrist. Note the heat cells on the inner layer of the wrap. (B) Using the hand with wrap in place.

pain reduction and perhaps a reduction in muscle-guarding spasms.[116] These heat wraps have also been effective in low pain control when used during sleep.[117]

Kettenmann et al[69] examined the impact of low-level heat wrap therapy on acute low back pain by taking objective electroencephalogram measures in addition to other psychophysical measurements. They found the heat wrap group had reduced low back pain, a better night's sleep, a decreased number of daytime naps, and everyday situations being less stressful compared with the control group. Mayer et al[19] found the combination of low-level heat wrap and directional preference-based exercise during the treatment of acute low back pain significantly improved functional outcomes compared with either intervention alone or the control. Patients with wrist pain due to sprains/strains and arthritis had relief with use of a wrist wrap as compared with placebo medication.[118]

Another study by Mayer et al[22] found low-level heat wrap therapy was of significant benefit in the prevention

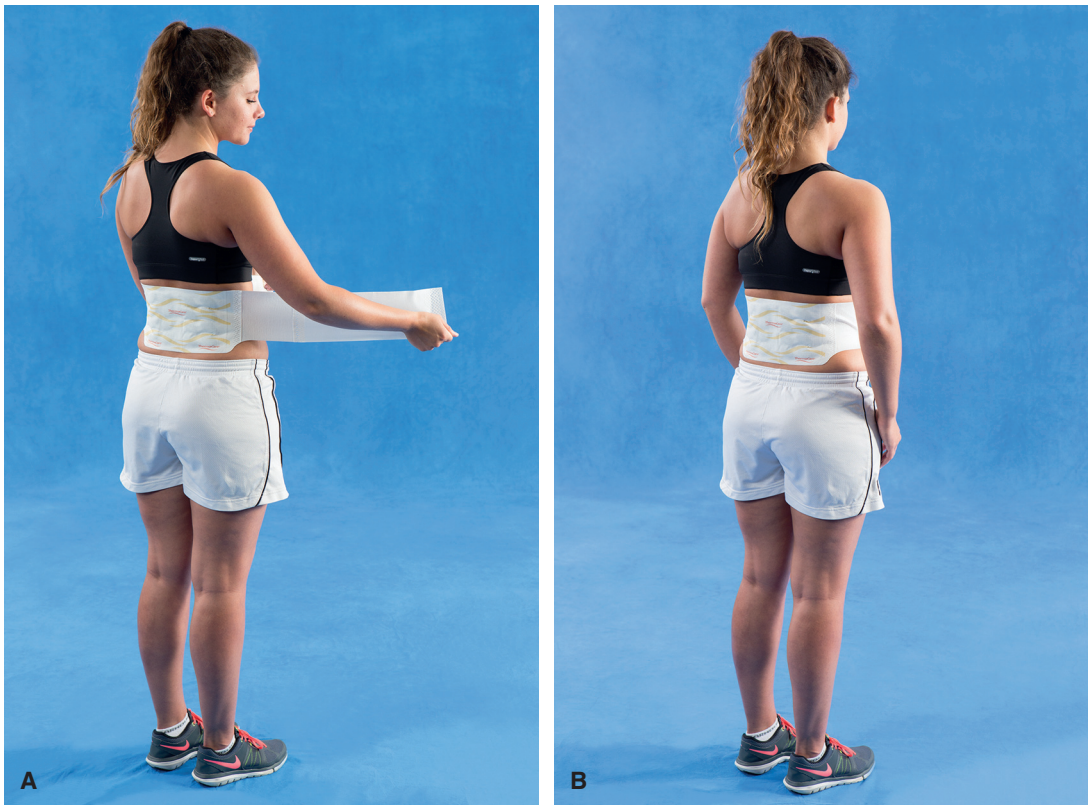

Fig 3 ■ 11 (A) Air-activated heat wrap being applied to lower back. (B) Patient can remain mobile while receiving heat.

and the early phase treatment of DOMS of the low back. It appears from these studies that the impact provided by low-level air-activated heat wraps is based on the length of application, which varied from 4 to 8 hours, compared with the 15- to 30-minute application of hot packs or paraffin wax.[22] Interestingly, Lewis et al[131] found that wearing of a superficial lumbar heat wrap for only 2 hours in patients with chronic low back pain was associated with a decrease in muscle activity and short-term aspects of well-being. See Box 3-5 for advantages and disadvantages of air-activated, wearable heat wraps.

Box 3 ■ 5 Advantages and Disadvantages of Air-Activated Heat Wraps

Advantages

1. Dry heat prevents clothing from getting wet.
2. Wraps provide a comfortable and low-profile heat source.
3. Wraps can be worn during activity and sleep.
4. Patients can easily be instructed in safe application.
5. Wraps are relatively inexpensive for short-term use.
6. Active exercises may be performed during wear.
7. When used as directed, wraps do not heat up to more than 104°F (40°C).

Disadvantages

1. Wraps can be used only one time.
2. If needed for extended periods of time, the expense must be considered.

Convective Heating: Fluidized Therapy (Fluidotherapy)

Fluidotherapy is a dry-heat modality that transfers heat energy by forced convection. Borrell and associates[3] suggest that, for heating, it is irrelevant whether the modality is providing wet or dry heat as long as the skin temperature is raised to the same temperature by both modalities. The Fluidotherapy system uses air-fluidized solids as the heat transfer medium. Warm air is uniformly circulated through the bottom of a bed of finely divided cellulose particles (finely ground corn cob called *cellex*) in a container. The solid particles become suspended when the stream of air is forced through them, making the fluidized bed behave and demonstrate

properties similar to those of liquids.[3] The viscosity of the air-fluidized system is low, allowing a patient to submerge body parts into the fluidized bed and suspend these parts similarly to a fluid bath, thus permitting exercise with relative ease.[119,120] The heat transfer characteristics within the fluidized bed and to parts submerged in it are similar to those of a mildly agitated liquid.[119] The combination of air flowing around the high surface area of the finely divided particles and the bulk movement of solids produces high heat fluxes and uniform temperatures throughout, thus providing a strong massaging action, sensory stimulation, and levitation.[120]

Fluidotherapy units come in a variety of sizes and are best used for treating the distal extremities. For joints and distal body parts, the patient places the body part to be treated through the entrance sleeve of the Fluidotherapy unit (Fig. 3-12). The sleeve is then secured to keep the cellulose particles from escaping. As the air stream is blown in, the particles become suspended and the treated body part feels as though it is immersed in a moving liquid bath such as a whirlpool.

Both temperature and the amount of particle agitation can be varied. Temperatures for intervention typically range from 102°F to 118°F (38.8°C to 47.8°C). The lower ranges are recommended for patients who have a greater predisposition for edema formation or who are in beginning programs for desensitization as they may not be able to tolerate higher temperatures. Agitation can be controlled for patient comfort. In addition, varying degrees of agitation can be used in a program of desensitization for hypersensitive areas.

Figure 3-12 Fluidized therapy (fluidotherapy) to the hand and wrist.

Patients can carry out exercises while the affected body part is within the cabinet. This is particularly effective for the distal extremities, such as the wrist, hand, and fingers and ankle, foot, and toes. If heat and stretch are desired, dynamic splinting can be used during the time of heat intervention to provide a gentle stretch, or stretching techniques can be used immediately following immersion in Fluidotherapy.

If it is desirable to treat a body part that also has an open wound, the wound can be protected with a plastic barrier or bag to prevent any fine cellulose particles from becoming embedded in the wound and to minimize the risk of cross-contamination.

The effects of Fluidotherapy on nerve conduction velocity and skin temperature in normal subjects was examined by Kelly et al.[79] They found that Fluidotherapy (heat plus tactile stimulation) significantly elevated superficial skin temperature compared with tactile stimulation alone and no treatment (control group). In addition, they found a concomitant decrease in distal sensory latency of the superficial radial nerve action potential as the skin temperature increased. The authors suggest that the higher skin temperatures obtained with Fluidotherapy may be of benefit clinically when the aim of treatment is to increase soft tissue extensibility or reduce joint stiffness with active or passive movements.[79]

The effectiveness of Fluidotherapy as a heating modality was compared with paraffin wax and hydrotherapy by in vivo temperature measurements.[3] Joint capsule and muscle temperatures in the hands and feet were measured at various depths with indication that the Fluidotherapy produced the greatest increase in tissue temperatures in all areas. The authors' conclusion was that the dry whirlpool (Fluidotherapy) delivered more heat than paraffin wax or hydrotherapy because higher temperatures can be tolerated in a dry environment.[3] This conclusion is questioned, particularly because paraffin wax allows tissues to be immersed in a bath with an operating temperature of 113°F to 129°F (45°C to 54°C) compared with the Fluidotherapy range of 102.2°F to 118.4°F (39°C to 48°C).

Alcorn and coworkers[121] used Fluidotherapy and exercise in the management of patients with sickle cell anemia. They demonstrated a marked reduction in the length of hospitalization (compared with the length of hospitalization by the same patients during previous episodes), a major reduction in the dosage of analgesics

previously administered, and a marked improvement in spine, trunk, and extremity ROM and gait. See Box 3-6 for advantages and disadvantages of Fluidotherapy.

CLINICAL APPLICATION: PRINCIPLES AND INDICATIONS

Heating modalities are used in therapeutic programs to assist in the reduction of pain and stiffness, to alleviate muscle spasm, to increase ROM, and to improve tissue healing by increasing blood flow and nutrients to an area. When heat is applied to the trunk, shoulders, hips, or knees, it is usually considered a mild heat. The site of dysfunction is often well below the surface, and the heat will produce desired responses through reflex mechanisms by stimulation of cutaneous afferents. Mild heating usually elevates temperature at the site of pathology to less than 104°F (40°C) and may be thought of as having a soothing, counterirritant effect.[37]

When a higher temperature—for example, between 104°F and 113°F (40°C and 45°C)—is desired at the involved structure, the appropriate modality for the intervention must be chosen. Paraffin, for example, may be a vigorous heater of the finger joints but only a mild heater of the shoulder. The principles of mild versus vigorous heating are summarized in Table 3-2.

Intervention time with all heating modalities usually varies from 15 to 30 minutes. This duration will allow time for maximal tolerable increases in tissue temperature and blood flow. However, with certain heat modalities, such as air-activated heat wraps, a much longer time of application is used. (See the previous section on air-activated heat wraps.)

Despite the widespread clinical use of heating modalities, there is a limited, albeit growing, number of well-designed clinical studies that address the efficacy of these modalities in a therapeutic regimen. There are several systematic reviews that examine the efficacy of superficial heating agents.[18,21,23,27] The remainder of this chapter will discuss safety issues and application techniques based on purported physiological rationales and the clinical experience of the authors. Details regarding specific studies related to the use of thermotherapy for pain control, reduction of muscle-guarding spasm, and increasing ROM are discussed in Chapter 12.

Box 3 ■ 6 Advantages and Disadvantages of Fluidotherapy

Advantages

1. Fluidotherapy is convenient and easy to administer.
2. Temperature of application can be controlled.
3. Agitation of dry particles can be controlled for comfort.
4. Variety of unit sizes allows for most body areas to be treated.
5. Fluidotherapy allows for some active exercise to be carried out during intervention.
6. Fluidotherapy provides a dry, comfortable heat.
7. Fluidotherapy can be used for desensitization of hypersensitive hands/fingers or feet/toes.

Disadvantages

1. Fluidotherapy is a relatively expensive modality to purchase.
2. Some patients are intolerant to the enclosed container (claustrophobic feeling).
3. Some patients are intolerant to the dry materials used.

CONTRAINDICATIONS AND PRECAUTIONS TO THERMOTHERAPY

Before deciding to use heat in therapeutic intervention, the therapist should determine the status of the patient's circulation and sensitivity to temperature and pain. The skin overlying the intervention area should be tested for thermal sensation using hot and cold water in test tubes or with other hot and cold objects such as spoons. Pain sensation can be determined by using the pin prick versus the blunt test. This information is necessary because determining the safe level of heat requires that the patient be able to perceive when the pain threshold has been reached. Response of thermal receptors in the skin is illustrated in Figure 3-13.

The following is a list of contraindications to the use of heating modalities:

- Application over areas with a lack of intact thermal sensation (risk of burn if patient cannot distinguish between hot and cold)
- Application over areas of vascular insufficiency or vascular disease (risk of burn if circulation is inadequate to dissipate heat in tissues)

CASE STUDY 3-2 Therapeutic Heat for Low Back Pain

Your patient is a 38-year-old construction worker who had his right hand crushed under a heavy weight at work 3 months ago. He suffered fractures to his second, third, and fourth proximal phalanges and his second and third metacarpals, and he dislocated the metacarpal-phalangeal joint of his thumb. His hand was immobilized in a splint for 6 weeks. He is unable to return to work because of the significant loss of function of his right hand. Your examination reveals decreased ROM of flexion, extension, and opposition of the thumb; decreased ROM of flexion and extension of the second, third, and fourth metacarpal-phalangeal joints; weakness of the hand intrinsic muscles; and pain with closing his hand and grasping objects.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of therapeutic heat?

ANSWER: The patient has significant dysfunction and limitations, including pain and decreased ROM and strength, which presently keep the patient from returning to work. Therapeutic heat can be used as part of the rehabilitation plan to allow the patient to complete ROM and hand-strengthening exercises that will lead to further functional retraining.

2. Is the patient appropriate for therapeutic heat? Do any of the general precautions or contraindications to therapeutic heat apply to the patient or are there any specific considerations regarding the application of therapeutic heat to this patient?

ANSWER: The patient's dysfunctions and limitations are appropriate for use of therapeutic heat. Contraindications in this situation include treatment over areas of recent or potential hemorrhage, malignancy, acute inflammation, infection, poor thermal sensation, or peripheral vascular compromise or where heat rubs have been recently applied.

3. What are the specific goals to be achieved with the use of therapeutic heat?

ANSWER: The primary goals are to decrease pain and to increase extensibility of soft tissue in order to gain more ROM in the restricted joints. Job-specific functional goals will be incorporated as appropriate.

4. What specific aspects of therapeutic heat would be appropriate for this patient?

ANSWER: Options for heating as a choice for this case include warm whirlpool, paraffin wax bath, and Fluidotherapy. Whirlpool and Fluidotherapy would allow the patient to perform active exercises during treatment; paraffin wax is a passive treatment. Moist heat packs or low-level heat wrap therapy would not work well with the hand, wrist, and fingers because the flat surfaces of the heat packs or wraps would not conform to the curvatures of the hand.

5. What are the proper application procedures for therapeutic heat?

ANSWER: Several therapeutic heating agents may be used for this patient, including warm whirlpool (100°F [38°C]) for 15 to 20 minutes, Fluidotherapy (104°F [40°C]) for 15 to 20 minutes, and paraffin wax using the dip-and-wrap method for 20 minutes. Because restoring hand mobility is a primary interest, warm whirlpool and Fluidotherapy are preferred, as AROM activities can be performed while the heat is applied. If restrictions from scarring are present, paraffin wax may assist in softening and mobilizing the restricted scar.

TABLE 3–2. Comparison of Mild and Vigorous Heating[1]

	Mild	Vigorous
Temperature elevation site of pathology	Low	High
Degree of temperature increase	Warmth—up to 104°F (40°C) comfortable sensation of warmth	Near tolerance levels up to 113°F (45°C)
Rate of rise of temperature	Slow	Rapid
Clinical examples	To cervical area for reducing muscle spasm upper trapezius	Fluidotherapy at 113°F (45°C) to the hand to increase tissue extensibility before ROM exercises

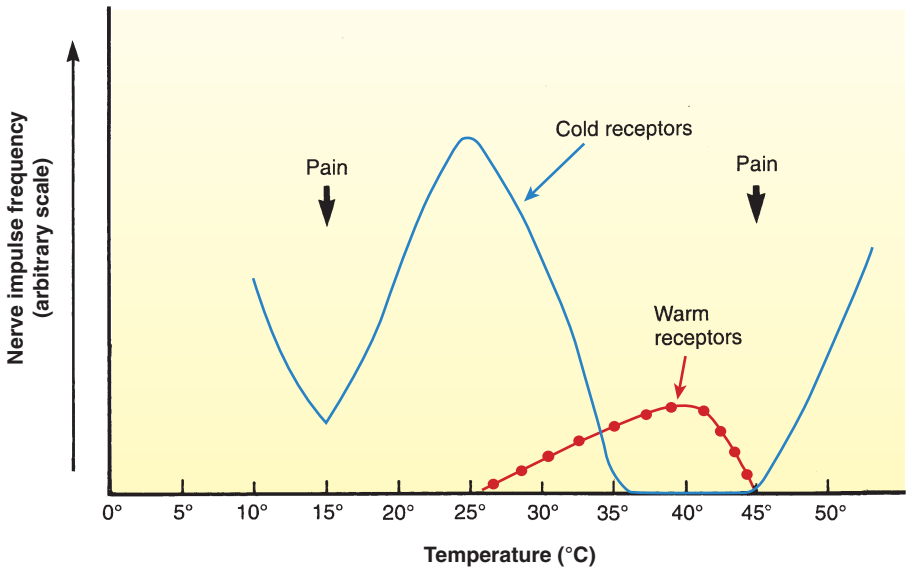

Fig 3 ■ 13 Responses of thermal receptors in the skin. *(From Low J, Reed A. Electrotherapy Explained: Principles and Practices. Oxford: Butterworth Heinemann; 1990, p 177, with permission.)*

- Application over areas of recent hemorrhage or potential hemorrhage (heat will increase bleeding)
- Application over areas of known malignancy (although the exact nature of what may happen when superficial heat is applied over a known malignancy is unknown, heat may increase activation and movement of malignant cells)
- Application over areas of acute inflammation (heat will aggravate and potentially increase inflammatory response)
- Application over infected areas where infection may spread or cross-contamination may occur (heat may cause infection to spread to other areas)
- Application over areas where liniments or heat rubs have recently been applied (heat will increase risk of burn because vessels are already dilated from the presence of liniment; the vessels cannot dilate further to dissipate more heat)
- Application in any situation deemed unreliable by the practitioner (e.g., language difficulties, which puts patients at risk because they may not understand the therapist's instructions).

CLINICAL DECISION-MAKING

The decision to use a thermal modality as part of a total intervention program should be based on a combination of factors, including the patient's diagnosis and medical status and the objective findings of the physical therapy examination. It is not until this information is gathered that the intervention goals are established. The plan of intervention to obtain these goals can include a thermal modality when indicated. It should be noted that, in most situations, it is only appropriate to apply one type of heating modality. To include two, such as paraffin and whirlpool, to the hand in one session would most likely be redundant. In certain situations, combining two electrophysical agents may be complementary to the treatment goals, such as the use of TENS with a hot pack for the treatment of pain.[80,108] Intervention is executed and follow-up completed. The procedures for clinical decision-making and carrying out the intervention are outlined in Box 3-7.

Box 3 ■ 7 Procedure for Clinical Decision-Making and Execution of Intervention With a Thermal Modality

1. Assess patient.
2. Establish intervention goals based on results of patient assessment and patient consultation.
3. Select intervention plan, including thermal modality when applicable, to meet these goals.
4. Choose thermal modality.
5. Select position for intervention.
6. Apply thermal modality (followed by exercise or other appropriate techniques).
7. Reassess and determine if intervention should continue or be modified or discontinued.
8. Establish a home program (which can include a thermal modality).

Heat Versus Cold

There are clinical situations when either heat or cold may be selected to meet intervention objectives or when one is clearly preferred over the other. Often the choice between heat and cold is empirical, but before the decision is made, certain factors should be considered:

1. Stage of injury or disease
2. Area of body treated
3. Medical status
4. Patient preference, which may be determined by cold or heat hypersensitivity
5. Decision to use thermal modalities as part of a home program.

Cold is the preferred modality during the acute stages of inflammation; heat at this stage may further aggravate inflammation. For patients who can tolerate it, cryotherapy may be chosen for reduction of muscle spasm (see Chapter 2). Cold can also reduce pain around joints before ROM exercises and may be easier for the patient to apply at home. On the other hand, heat may be better tolerated psychologically by persons with pain or muscle spasm, resulting in increased patient compliance with intervention programs. Temperature elevation will decrease joint stiffness and increase connective tissue extensibility, so if either of these is the intervention goal, heat is the modality of choice. In addition, heat to distal extremities seems to be tolerated better than cold. The advantages and disadvantages of using heat or cold are briefly outlined in Figure 3-14.

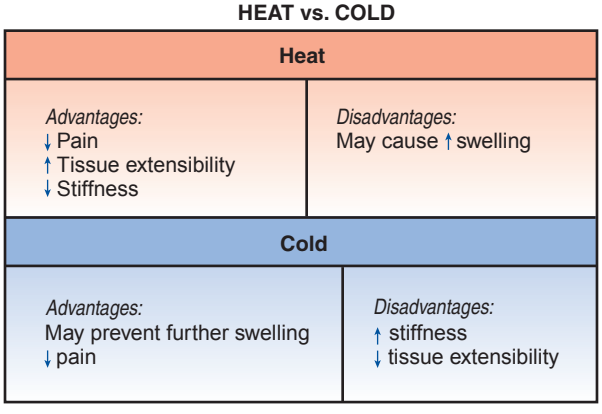

Fig 3 ■ 14 Intervention choice: heat versus cold.

Factors to Consider for Therapeutic Heat Techniques

The decision of which heat modality to select primarily depends on the location of the involved structure, the pathophysiology, and the degree of temperature elevation desired (Fig. 3-15). Generally speaking, deep heat is usually selected during the remodeling phase of an injury or a disease when tissue contractures persist or when heat is required deep into a joint.

When treating a patient with pain, muscle spasm, or both, the patient's position must be carefully selected and should be the most comfortable possible, particularly considering that the patient may need to remain in that position for up to 30 minutes. If muscle spasm is present, the muscle should not be in a position of "undue stretch" until some pain relief has occurred. If the patient has joint pain, the joint should be positioned in an open-packed position, with the ligaments and joint capsule slackened. In this position, intra-articular pressure[122] and stress on joint structures will be lessened.

As has been previously described,[38,96] the use of heat combined with low-load prolonged stretch may be amenable to elongating tissues secondary to contracture as heat increases extensibility of collagen tissue.

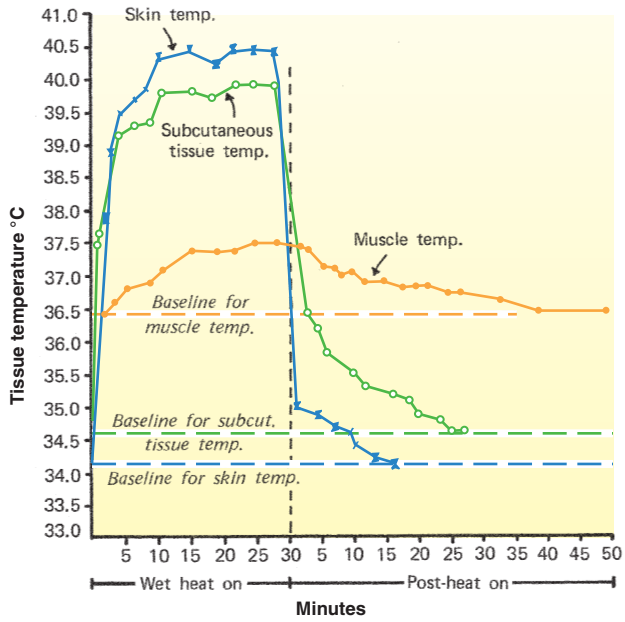

Fig 3 ■ 15 Curves representing changes in skin, subcutaneous tissue, and muscle temperatures obtained during and after 30 minutes of wet heat topically applied to the forearm. *(From Abramson et al. Changes in blood flow, oxygen uptake and tissue temperatures produced by the topical application of wet heat.* Arch Phys Med Rehabil. *1961;42:305, with permission.)*

Wet Versus Dry Heat

Many patients say that they have heard that moist heat is more penetrating and thus more effective than dry heat. Few clinical studies that examine the efficacy of one versus the other in obtaining functional intervention goals are available. Cosgray et al[123] compared moist hot packs with the Pneumatherm, a device that delivers pulsed, dry hot air to the skin at temperatures of approximately 120°F (49°C) on hamstring length. They found that the dry heat produced greater increases in hamstring length than moist heat or controls. It has been determined that dry heat can elevate surface temperatures to a greater degree but that moist heat can elevate temperature to a slightly deeper level.[124]

Home Application of Therapeutic Heat Modalities

In most cases, it is desirable to provide a patient with a means for pain control and for reducing stiffness before exercise when they are not within the confines of a supervised clinical situation. Gel packs, sand packs, and bead packs are commercially available and can be heated in either a water container or a microwave oven for short-duration heat applications (e.g., up to 20 or 30 minutes). Both paraffin wax baths and Fluidotherapy units are available in home-use sizes, which are generally for the hands, wrists, and feet. Electric heating pads also provide heat, and they are available in various sizes. A rubber hot water bottle filled with hot tap water and covered with a moist towel will also provide mild, comfortable heat. Adequate, clear, and concise instructions should be written down for the home patient, including the intervention time, method, frequency of application, and any special instructions and precautions to prevent inadvertent burns from excess heat.

Safety of Heat Application

The adverse responses that can occur with heat application primarily include the chance of increasing swelling in the case of acute edema and the chance of causing superficial tissue burns. Barillo et al[125] reviewed 4,510 records of patients admitted to one burn center between January 1978 and July 1997. Eleven admissions were due to burns caused by therapeutic application of heat.

ASSESSMENT OF EFFECTIVENESS AND EXPECTED OUTCOMES

The use of heat in a therapy program is based on the goals of the treatment. The goals are determined by the patient and the therapist after a thorough evaluation of the patient, which includes the history of the present problem and subjective and objective measures of impairments and current functional status. The following outcomes and measures can be used for assessing effectiveness with heat:

- Pain—quantification via scales (see Chapter 11)
- ROM—goniometric measures
- Muscle guarding—reflected in joint ROM and muscle flexibility measures.

Of course, the decision as to which of these measures to use will depend on the impairment being assessed.

Documentation

The astute clinician realizes the importance of documenting the specifics of intervention techniques and patient response to therapeutic intervention. Information included in the intervention record is outlined in the Documentation Tips that follow. Without such information, it is often difficult to replicate techniques and to adjust the plan of care as needed. A copy of home instructions should be kept with the patient's file.

Documentation Tips

- Thermal modality used
- Method of application, including duration
- Body area treated
- Patient position for intervention
- Special precautions or application concerns

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CHAPTER 4

THERAPEUTIC ULTRASOUND

David Lake PT, PhD

Therapeutic ultrasound (US) is one of the most commonly used, and misused, biophysical agents in physical therapy and other rehabilitative professions. Wong et al[1] reported that certified orthopedic clinical specialists use therapeutic US for decreasing soft tissue inflammation and pain and for increasing tissue extensibility, scar tissue remodeling, and healing acute soft tissue injuries. The most common diagnoses for which US is used by rehabilitation professionals include back, shoulder, knee, and neck pain and difficulty in walking and other gait abnormalities.[2] US represents more than 5.8% of all line items submitted to the Centers for Medicare and Medicaid Services (CMS) for payment.[2] A recent study[3] reported that 82.4% of physical therapists use US—and 36.4% use it daily. Most US use occurs in private practice settings (58.4%) and in the musculoskeletal area of practice (74.8%). Therapists use it on an average of 40% of their patients. Although quite high for a given modalities treatment, this rate of use is a considerable decline from the 94% that reported daily US use two decades ago.[4]

Despite its use, the evidence for the effectiveness of therapeutic US has not been well documented in the rehabilitation literature. In fact, most studies have shown it not to be effective when compared to placebo controls. A critical analysis of the literature shows, however, that this level of negative evidence may be more related to problems in study design and to the variability of patient responsiveness to US rather than to the ineffectiveness of US. Interestingly, Amijo-Olivo et al[3] report that only 13% of therapists using US make their clinical decisions using research evidence while 40% use their own clinical experiences.

PHYSICAL PRINCIPLES OF ULTRASOUND

US is high-frequency mechanical waves delivered using acoustic energy. Sound waves exert their mechanical action by pressing the initially vibrating molecules into adjacent molecules, which in turn causes them to vibrate. These newly vibrated molecules will then transmit their energy into vibrating molecules adjacent to them. When no molecules are present, such as in a vacuum, there would be no transmission of sound energy. Conversely, in substances (or tissues) with higher molecular densities, transmission of US would be more efficient because there are more molecules per given volume. For example, human speech at normal volumes can be heard only a few meters in air while whale vocalizations can be heard over many kilometers in water because water is a denser medium than air. Conversely, ultrasonic energy can be dissipated (attenuated) more quickly in denser substances because denser substances offer more resistance to molecular motion (i.e., acoustic impedance). Because of the poor transmission of US waves through air, a coupling agent is needed. Generally an aqueous gel is used between the US applicator and the skin to enhance the efficient transmission of ultrasonic energy to body tissues from the US applicator.

Sound waves are transmitted in a medium longitudinally (along the direction of the sound wave) by alternative compressions and rarefactions of the molecules in the medium. Compressions are areas of increased density of the molecules. Rarefactions are areas of decreased density (Fig. 4-1). The degree of compression and rarefaction is dependent upon the magnitude of the wave energy. The duration of these compressions and rarefactions is determined by the frequency at which the wave was generated. This can be illustrated using a Slinky. Move one end of the Slinky forward and backward very quickly, and you can see the metal rings moving together (compression) and apart (rarefaction) as the movement progresses along the length of the Slinky. These longitudinal waves move away from the emitter in all different directions (called *dispersion*). The sound energy can be focused or collimated to give a greater directionality, such as in the shape of the bell of a wind instrument or the shape of an acoustic speaker. In therapeutic US, a specifically designed applicator is used to collimate the beam.

When acoustic waves reach a point of a change in tissue density, the waves can be reflected, refracted,

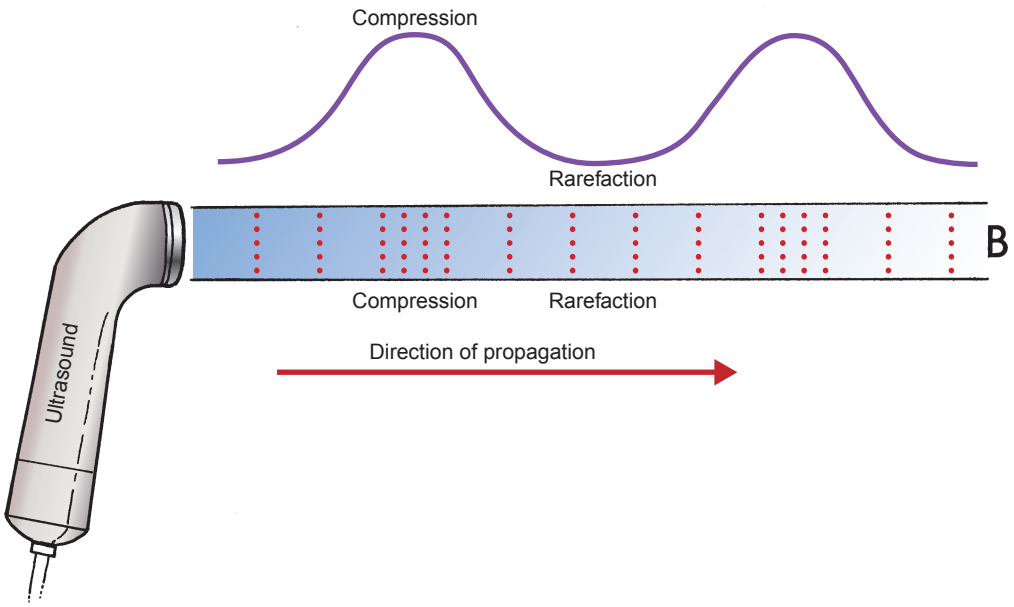


Fig 4 ■ 1 Illustration of a collimated beam of ultrasound energy. The corresponding pressure wave is shown; compression results in areas of increased molecular concentration, whereas rarefaction results in areas of decreased molecular concentration.

or absorbed. When the wave is absorbed, the kinetic energy of movement is transformed into thermal energy. Those waves that pass through the denser tissue will be refracted or bent and will no longer follow their original path. Finally, waves can be reflected. The angle of reflection is dependent upon the angle on incidence at the interface between tissues of differing densities. Reflected waves can interact with the incident waves by enhancing the wave intensity if the waves interact in synchrony (forming what is called a *standing wave*) or by diminishing wave intensity if the waves interact asynchronously.

You can apply these principles to the application of ultrasonic energy to human tissues. Most body tissues behave as liquids of varying densities. Bone is an exception, as it acts as a solid. Thus, although longitudinal waves predominate in most tissues, both longitudinal and transverse transmission of ultrasonic waves occur in bone.

In the body, ultrasonic energy is more rapidly attenuated and converted from acoustic energy to thermal energy in dense tissues, such as ligaments, tendons, and other connective tissues, than in the less dense muscle or even less dense adipose tissue (Table 4-1). Consequently, higher tissue temperatures will result when therapeutic US is applied to connective tissues than when applied to muscle and other less dense tissues. Additionally, when sonicating an area with high-density tissues, acoustic wave reflection can potentially produce standing waves, which may increase the intensity of the acoustic wave at the junction between the high-density and lower-density tissues. In this way, applying US over bone may dramatically increase the temperature of the periosteum and result in discomfort.

TABLE 4–1. Attenuation of a 1-MHz Ultrasound Beam in Various Tissues

Tissue	Attenuation (%/cm)
Blood	3
Fat	13
Muscle	24
Blood vessel	32
Skin	39
Tendon	59
Cartilage	68
Bone	96

Key Point! More dense connective tissues, such as ligaments and tendons, absorb US better than less dense tissues such as muscle and fat. Thus, US is more effective at heating more dense tissues than less dense tissues.

PRODUCTION OF ULTRASOUND WAVES

The two main components of the US device are the generator and the applicator. The generator is in the "big box" of the US device. Its key component is an electrical oscillator that generates the high-frequency alternating current that matches the intensity and frequency parameters of the applicator's piezoelectric crystal.

The US applicator is composed of two key elements: the piezoelectric crystal and the sound head (Fig. 4-2). The piezoelectric crystal is composed of a thin sheet (2 to 3 mm thick) of lead zirconate or titanate ceramic. The piezoelectric crystal is transversely compressed and expanded by sending an alternating electrical current through it, referred to as the *reverse piezoelectric effect* (Fig. 4-3). When no current is sent through the crystal, it maintains its normal shape (Fig. 4-3A). When current

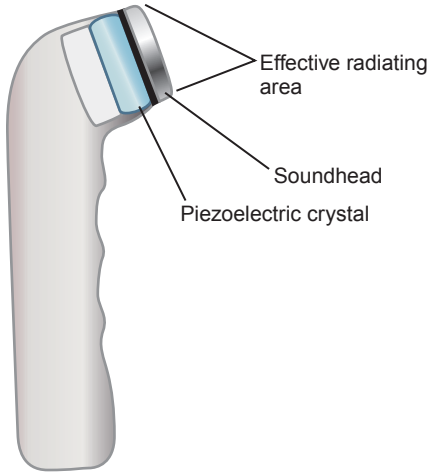


Fig 4 ■ 2 Schematic of a typical ultrasound applicator. The piezoelectric crystal transduces electrical energy into acoustic energy that is transmitted by the sound head.

of one polarity is sent through the crystal, a compression (concavity) of the crystal occurs (Fig. 4-3B). When the polarity is reversed in the alternating current, an expansion (convexity) of the crystal occurs (Fig. 4-3C). This rapid transverse compression and expansion produces the acoustic wave, which is then transmitted through the sound head connected to the piezoelectric crystal. This alternation of compression and expansion occurs between 1 million and 3 million times per second (i.e., 1 and 3 megahertz frequency). The structure and size of the crystal produces a collimated beam of acoustic waves. The beam is slightly smaller than the size of the crystal because the crystal's ends do not expand or contract much. The area of the crystal that moves is the effective radiating area (ERA).The crystal is not perfectly uniform in shape, nor does it expand and contract uniformly across its surface. This leads to a nonuniformity of the acoustic energy at different parts of the collimated beam, which will be described later in the "Beam Nonuniformity Ratio (BNR)" section of this chapter.

The sound head of the US device is often aluminum, stainless steel, or ceramic. It covers the irradiating surface and is connected to the piezoelectric crystal. The stainless steel sound head is preferred over the aluminum and ceramic sound heads because of its enhanced durability. Acoustic energy from the crystal is conducted to the sound head and then through a conductive gel into the skin of the treatment area. It is important to understand that the ERA is smaller than the actual size of the sound head.

Key Point! To ensure appropriate delivery of the US energy, clinicians must take into account the fact that the ERA of the US energy is actually smaller than the sound head applicator.

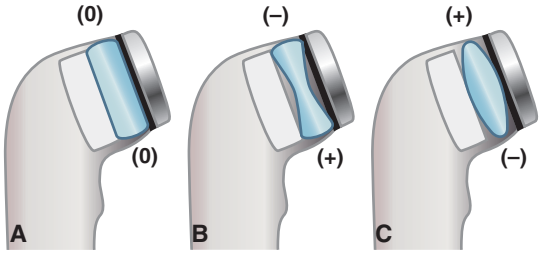


Fig 4 ■ 3 Schematic of the conformational shape change of the piezoelectric crystal under the influence of an applied alternating current. (A) With no current flow, the crystal shape remains unchanged. A concave (B) or convex (C) change in shape occurs as the direction of current flow across the crystal alternates.

CHARACTERISTICS OF THE ULTRASOUND WAVE AND TREATMENT PARAMETERS

When using therapeutic US, the rehabilitation professional needs to be aware of the characteristics of the US wave. An understanding of these characteristics is critical in the proper dosing and administration of therapeutic US.

Frequency

Frequency of the US wave is the number of waves per second delivered to the patient. In most US units, frequency ranges from 0.75 to 3.3 MHz (millions of cycles per second). Most therapeutic US units designed and manufactured after 1990 have dual-frequency applicators designed to deliver frequencies of both 1 MHz and 3 or 3.3 MHz. Lesser frequencies penetrate deeper. For example, think of a car with the radio on and windows closed. From the outside, you hear the lower frequency bass beat, not the higher frequencies. Studies have shown effective treatment depths with 1 MHz of up to 6 cm deep and 3 MHz being effective up to 2.5 cm deep.[5] A common misconception is that increasing the intensity of the ultrasonic energy will produce a deeper penetration. Increasing the power of the US only increases the energy at the depths of penetration produced by changing the frequency.[6,7] Tissues absorb 3-MHz US at a rate three times faster than 1-MHz US.[7,8] Because the rate of tissue heating is also related to the rate of absorption, US at 3 MHz will heat tissues three times faster than 1 MHz (Table 4-2). In practice, US delivered at 3 MHz is used for more superficial structures, such as exposed tendons and ligaments, while 1 MHz is used to treat deeper structures such as most muscles and fascia. However, the rehabilitation professional can use frequency differences to change the rate of heating of the tissue being sonicated.

TABLE 4–2. Comparison of the Rate of Heating (°/min) of Muscle Tissue Using 1-MHz and 3-MHz Ultrasound

	Frequency	
Intensity (W/cm²)	1 MHz	3 MHz
0.5	0.07°F (0.04°C)	0.54°F (0.3°C)
1	0.36°F (0.2°C)	1.08°F (0.6°C)
1.5	0.54°F (0.3°C)	1.61°F (0.9°C)
2	0.72°F (0.4°C)	2.52°F (1.4°C)

Key Point! Depth of US penetration is dependent on frequency, not intensity, and depth is inversely proportional to the US frequency, such that 1 MHz will penetrate deeper than 3 MHz.

Intensity

Power of the ultrasonic energy is the product of wave phase duration and wave amplitude (intensity). Because wave duration is not changed at a fixed frequency, amplitude or intensity is adjusted to change the power or magnitude of the acoustic energy. Power is measured in watts, but in therapeutic US application, power is most commonly expressed as the spatial average intensity (SAI) measured in watts/cm^2. SAI is calculated by dividing the power in watts by the ERA.

$$\text{Spatial Average Intensity (SAI)} = \frac{\text{Power (watts)}}{\text{ERA (cm}^2\text{)}}$$

Clinical Controversy

SAI is commonly and incorrectly described as the power divided by the sound head area; however, as previously shown, the actual sound head area is greater than the ERA, which is determined by the crystal's size and mobility. This difference varies widely among US units from different manufacturers, so clinicians should refer to specifications in the product's user guide.

No definitive guidelines are available that state an optimal intensity for therapeutic US despite the widely held perception that US should be delivered at an intensity of 1.5 watts/cm^2. It is generally recommended that practitioners use the lowest intensity possible to achieve the desired therapeutic effect.[9]

The variance in ERA in US applicators from different manufacturers can impact the effect of the treatment. Merrick et al[10] compared three different US units and found that the highest ERA heated faster and was able to reach the target of approximately 7°F (4°C) rise in temperature in muscle in 6 minutes while the other units could not reach that level of temperature change even after 10 minutes of sonication.

Mode

Most, if not all, US devices can deliver both continuous and pulsed US. In continuous US, the US is delivered at a constant energy level throughout the treatment. In pulsed US, there is a periodic cessation of the energy flow, so no US is delivered for a period of time (Fig. 4-4). As a result, the total energy level delivered is reduced. The term *temporal (or time) average intensity* (TAI) is used to describe this lesser level of energy. The time that the energy is flowing is termed the *pulse duration,* and the combined time of energy flow and lack of flow is termed the *pulse period*. The degree to which the total energy level is reduced is dependent upon the *duty cycle,* which is defined as pulse duration (on-time) divided by the pulse period (on-time + off-time) times 100 to result in a percentage of on-time to total-time of the pulse period. In Figure 4-4, the pulse on-time is 2 milliseconds, off-time is 8 milliseconds, and the pulse period is 10 milliseconds, which yields a duty

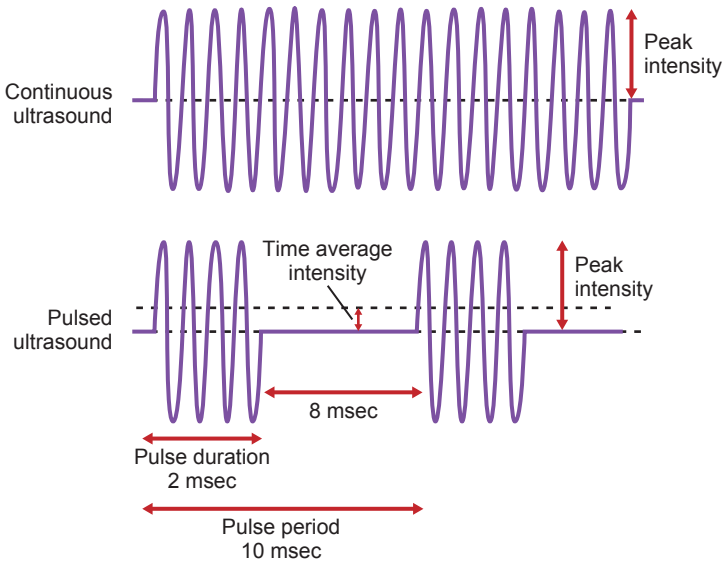


Fig 4 ■ 4 Illustration of continuous US and pulsed US. For pulsed US, the pulse duration and pulse period are illustrated. Note that temporal peak intensity (TPI) is the same for both continuous and pulsed US, but the time (or temporal) average intensity (TAI) for pulsed US is less due to the periodic cessation of energy flow. For the pulsed US, the pulse duration is 2 msec on-time, 8 msec off-time, and pulse period (total time) is 10 msec, which yields a duty cycle of 20%. Notice that the TAI is 20% of the TPI.

cycle of 20%. Thus, the TAI is 20% of the temporal peak intensity (TPI).

Most US machines will be equipped to deliver at least three levels of duty cycle: 20%, 50%, and 100%. Some will offer more variation, which is really an unneeded option. Continuous US is used when heating is needed, and pulsed US is used when the energy level needs to be reduced to produce the so-called nonthermal US.

OTHER PRINCIPLES OF THERAPEUTIC ULTRASOUND

Beam Nonuniformity Ratio

Discussed previously, the acoustic energy generating crystal is not perfectly uniform in shape, nor does it expand and contract uniformly across its surface. This leads to a nonuniformity of the acoustic energy at different parts of the collimated beam. This nonuniformity is measured with a hydrophone by the manufacturer (Fig. 4-5). The beam nonuniformity ratio (BNR) is how this variation is assessed. BNR is the ratio between the spatial peak intensity (I_{SP}) measured anywhere within the ERA and the spatial average intensity (I_{SA}) averaged over the entire ERA (Fig. 4-6). Better (and more expensive) US units have a lower BNR indicating a more uniformly shaped beam. Many US units have BNRs between 5 and 6, although better units have BNRs of about 2 to 3.

$$\text{BNR} = \frac{\text{spatial peak intensity } (I_{SP})}{\text{spatial average intensity } (I_{SA})}$$

These areas of peak intensity can form "hot spots." When set at 1.5 watts/cm^2, an US applicator with a 6:1 BNR has peak intensities of up to 9 watts/cm^2 (1.5 W/cm^2 × spatial peak intensity of 6), which can easily cause tissue damage or discomfort if an area is sonicated for too long. To equally distribute these hot spots around the treatment area and avoid burns or discomfort, the US head must be continuously moved in a pattern over the treatment area throughout the period of application. Units with higher BNR are more likely to cause discomfort due to hot spots.

Key Point! To minimize the chance of patient discomfort from hot spots, look for devices with a low BNR (i.e., 3 or less). However, these units are typically more expensive than units with a higher BNR.

However, two different US units with the same BNR may differ in discomfort level when the acoustic energy is applied under identical circumstances. This has led to the concept of peak area of maximal BNR (PAMBNR),[11] which is an assessment of how much of the ERA has the higher peak intensities. US applicators with a higher PAMBNR (because of the larger area of "hot spot") will be less comfortable than those with an identical BNR with a lower PAMBNR (Fig. 4-7). Although the Food and Drug Administration requires that the BNR be prominently posted on the US applicator, applicators do not come with any indication of PAMBNR.

Treatment Area

The appropriate size of a treatment area is likely the most problematic issue surrounding the use of therapeutic US. Evidence shows that to get significant heating effect, US must be applied in an area preferably two times the ERA[12,13] (and no greater than four times), particularly when using 1 MHz, which heats less intensively (Fig. 4-8). This is a very small area compared to

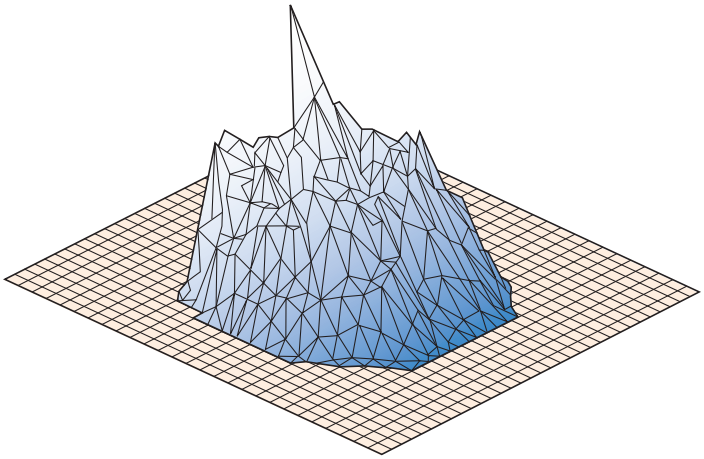


Fig 4 ■ 5 Graphic of the relative intensities of acoustic energy in a therapeutic US beam measured with a hydrophone.

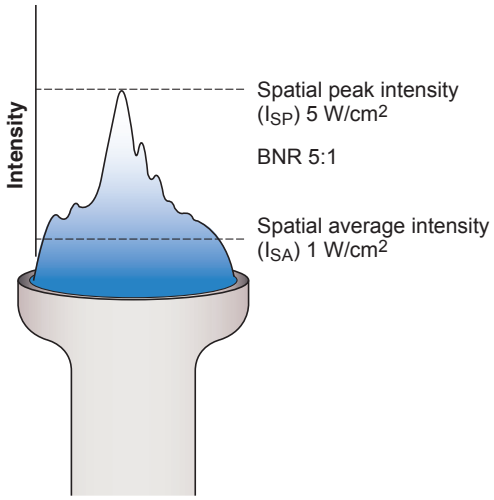


Fig 4 ■ 6 Illustration of relationship of spatial peak intensity (I_{SP}) to spatial average intensity (I_{SA}) in an US applicator with a beam nonuniformity ratio (BNR) of 5.

the areas generally treated by clinicians. Remember that the ERA is smaller (and in some cases much smaller) than the sound head of the applicator.

Key Point! Applying US over too large of an area is the biggest mistake made by clinicians when using US. The area treated with US should not be larger than four times the ERA. More often than not, the ERA is less than the size of the sound head.

When treating an area larger than four times the ERA, the "dose" of the treatment is consequently and significantly lessened. For example, doubling the treatment area halves the dose of therapeutic US. This will dramatically limit the use of US as a heating modality. US can be used to heat most exposed tendons and ligaments and small muscles, such as paraspinal muscles of a given spinal level (i.e., single segment). However, a common use seen in many clinics is sonicating long areas of muscles (e.g., paraspinal muscles over multiple levels), which will not significantly heat any area of the tissue. Heating large muscles—such as the quadratus lumborum, quadriceps femoris, hamstrings, and other similarly large muscles—is simply out of the question with US. Such large areas can be deep-heated only using diathermy.

Clinical Principles in Practice: Observing Ultrasound Parameters

With a little creativity, acoustic streaming, ERA, and some of the key parameters of US can be observed. First, take some tape, preferably at least 1 inch wide, and wrap it around the sound head's applicator, leaving approximately ½ to ¾ of an inch extending from the sound head's surface to make a small well or cup. Then, while holding the sound head so the well or cup is upright, pour a small amount of water into the well up to or near the tape's margin. Now turn on the US at 100% continuous. When the intensity is increased, the water will begin to move in response to the US waves moving through the water. When the US mode is changed to pulsed, the movement of the water will show a pulsed pattern. This movement is acoustic streaming. Try this for all of the duty cycle settings the device has and observe the differences in the water's movement. Additionally, observe the surface of the sound head and note how the region where the sound waves emanate is less than the actual surface area of the sound head. This difference represents the ERA of the sound head. In devices with a high BNR, it may be possible to observe a more centralized region of acoustic streaming that is less than in the immediately surrounding area. This "peak" area of water movement reflects the BNR of the device and the region where hot spots are more likely to develop with thermal US.

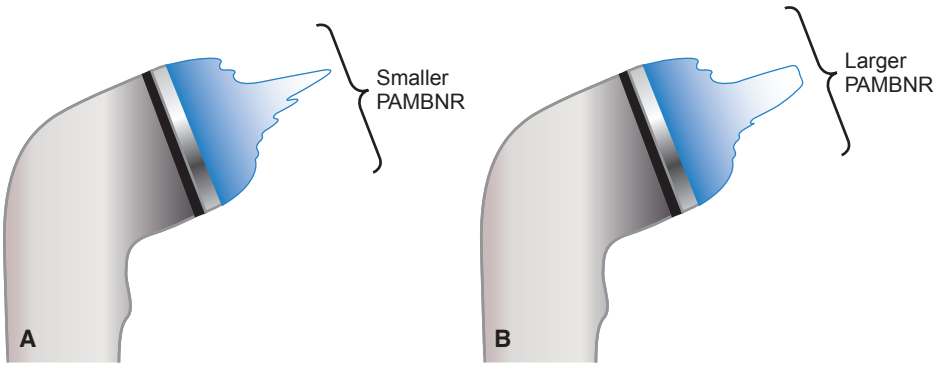


Fig 4 ■ 7 Illustration of two US applicators with identical BNR. Applicator A has a smaller peak area of maximal BNR (PAMBNR) than Applicator B. Notice the much smaller area of the peak intensity "hot spot" in Applicator A.

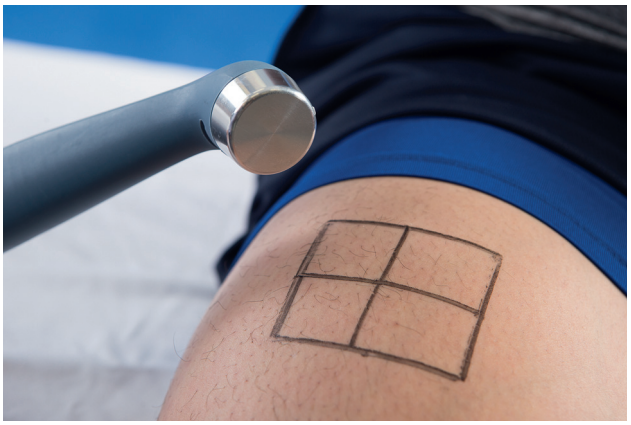


Fig 4 ■ 8 An appropriate treatment area is not larger than four times the effective radiating area (ERA). The treatment area shown is divided into four squares, each equal to 1× the ERA of the sound head.

Duration of Treatment and Number and Frequency of Treatments

Vigorous heating is defined as an increase of approximately 7°F (4°C) in the tissue because this has been shown to increase the distensibility of connective tissues in vitro.[14] Many rehabilitation professionals limit their US treatments to 5 to 10 minutes, and many insurance companies limit treatments to 8 minutes. However, using 1-MHz US (1.5 watts/cm^2, 2 × ERA), it takes 11 minutes to heat skeletal muscle to 6°F (3.5°C) and even longer to heat to vigorous heating of 7°F (4°C). More superficial muscles can be heated to 9.5°F (5.3°C) in 6 minutes using 3 MHz (1 W/cm^2, 2 × ERA).[8]

There remains no predetermined duration of treatment that has been found to be effective. Rather, each application of US is unique. The treatment's duration is dependent upon the size of the area being treated, the settings of the US unit (intensity, frequency, and mode), and the specific condition being treated. The duration must be adjusted to meet the needs of each individual patient.

Likewise, there remains no universal agreement regarding the number and frequency of US treatments. Treatments are often administered two to three times per week for 10 to 15 treatments. Some insurance companies have even specified a standard treatment of three to four times per week for a month,[15] which would be 12 to 16 treatments. When pairing thermal US with stretching techniques, the US would be applied immediately before (and perhaps during) the application of the stretching. The number and frequency of paired US/stretching treatments would be the same as if stretching were used alone. The number of treatments seen in the literature range from a single treatment[16] to daily (five times per week) treatments for 12 weeks.[17]

Variation in Ultrasound Units

US units can vary in the ERA of their applicators, which can have an impact on treatment effectiveness. However, treatment effectiveness can vary beyond what can be predicted by differences in ERA.

Merrick et al[10] reported considerable variation in heating of skeletal muscle when using three different US devices (Omnisound 3000, Dynatron 950, and Excel Ultra III) at the same 3 MHz, 1.5 W/cm^2 sonication over an area twice the size of the transducer head for 6 minutes. The heating using the Omnisound unit was 50% greater (11°F/6°C) than that seen with either the Dynatron or Excel units (less than 7°F [4°C]). Because of the differences in ERA and proportion of the sound head size to ERA, the three treatment areas, when compared to ERA, were 2.8, 3.2, and 5 for the Omnisound, Excel, and Dynatron units, respectively. So the Dynatron and Excel units were treating an area approximately 75% and 15%, respectively, greater than that treated by the Omnisound. So it is not surprising that there was a greater rise in muscle temperature when treated with the Omnisound unit.

When the rise in temperature with the Omnisound is normalized for the areas treated with the Dynatron and Excel units, one would expect to see a 6°F (3.4°C) and 10°F (5.4°C) rise, respectively. So the slightly less than 7°F rise in temperature seen with the Dynatron unit is close to its estimate, and the Excel unit's approximately 7°F rise is considerably less than would be expected. So other factors besides the comparable areas treated must explain the relatively poor performance of the Excel unit. These results confirmed a previous study that also showed differences in heating capacity in two different US devices when using the same treatment parameters.[18] The factors that make these US units different cannot be predicted by the clinician when using a US unit.

Key Point! US units usually differ in the ERA of their applicators and may differ in other ways that affect efficiency of treatment. Thus, no two US units can be considered to deliver the same treatment efficiency. The clinician must readjust the treatment protocols based upon the specific unit and applicator being used. In this manner, each application of US represents a unique or individualized event.

Variation in Tissue Response to Therapeutic Ultrasound

Ultrasonic energy is more rapidly attenuated and converted from acoustic energy to thermal energy in dense tissues, such as ligaments, tendons, and other connective tissues, than in the less dense muscle. So it is not surprising that US applied to tendons will produce a significantly greater and faster rise in temperature than when applied to skeletal muscle (Table 4-3).

As shown in Table 4-1, the rate of attenuation of the US waves in connective tissues, such as in ligaments and tendons, is 2.5 times that of skeletal muscle. The heating rate and temperature rise seen in tendons is between 2.5 and 3 times that seen in skeletal muscle under similar sonication conditions. Thus, it is critical for the clinician to recognize this and reduce the duration or intensity of the US or increase the area being sonicated or all three when applying US to ligaments and tendons.

Cooling of Tissues After Ultrasound Application

When US is used as an adjunct to other treatments and to heat tissue before stretching it, it is important that the clinician realize how fast a tissue cools after treatment.[19] Immediately following the end of sonication, a rapid phase of cooling occurs, which lasts for about 5 minutes. It is followed by a slower rate of cooling that lasts until the temperature of the tissue returns to normal. The rate of cooling depends upon how warm the tissues are heated and does not seem to differ between tissue types, with similar results seen in tendon[19,20] and muscle[21,22] (Table 4-4).

As shown in these data, there is a very narrow therapeutic window. For example, after sonication ends, clinicians have only 4 to 5 minutes to administer any cotreatments they wish to do even if they heat the patient to double the 7°F (4°C) rise of temperature required for vigorous heating effect.

TABLE 4–3. Rise in Temperature in 4 Minutes and Rate of Temperature Rise With Ultrasound Delivered at 3 MHz, 1 W/cm² and an Area of 2 × ERA

Tissue	Temperature Rise	Rate of Temperature Rise
Muscle[21]	5.4°F (3°C)	1.35°F (0.75°C)/min
Patellar tendon[20]	14.9°F (8.3°C)	3.8°F (2.1°C)/min
Achilles tendon[23]	14.2°F (7.9°C)	3.6°F (2°C)/min

Variability of Patient Response: Responders and Nonresponders

One of the problems with the use of therapeutic US is the wide variability in heating when identical US treatments are given to different patients. In one study, Burns et al[23] noted that with the same application of US (3 MHz, 1 watt/cm², 4 minutes to the Achilles tendon), some subjects responded with less than a 2°F (1°C) rise in temperature while others had greater than a 30°F (17°C) rise. Similar variability was seen in other studies in both tendon and muscle (Table 4-5).

Why such variability of response to US exists is unclear. Many factors could contribute to this, including percent body fat, tissue hydration, blood flow to the tissues, variable change in tissue metabolism, and the protein content of the tissues. The relative influence of each of these and other factors has not been studied. However, even if all factors contributing to this variability were known, there is probably no way to know which patients have any or all of these factors. Consequently, it is not likely that the responsiveness of patients to US could be determined—other than to try US for a few treatments to see how they respond. If patients do not report a warming with a relatively high US dose, it is likely they are "nonresponders."

These nonresponders may be the reason that so many studies on the use of US show no significant difference between the intervention and control groups. Included in these studies are probably many subjects who do not

TABLE 4–4. Rate of Cooling of Skeletal Muscle and Tendon After the Cessation of 3-MHz Sonication

Tissue	Heated Temperature (°)	Rate of Cooling (°/min)	Time to Cool to Below 7°F (4°C) Rise
Skeletal muscle[21]	9.5°F (5.3°C)	0.94°F (0.52°C)	2 min
Patellar tendon[20]	9°F (5°C)	0.94°F (0.52°C)	1.9 min
	14.9°F (8.3°C)	1.66°F (0.92°C)	4.7 min
Achilles tendon[19]	7.2°F (4°C)	1.01°F (0.56°C)	Immediate
	14.2°F (7.9°C)	1.64°F (0.91°C)	4.3 min

respond to US. If there are a number of these nonresponders in the study, these subjects will lessen any difference that may exist between those treated with US and those who are not (controls). On the other hand, it also appears that some patients respond robustly to heating with US. With these patients, the clinician must be careful not to overheat them. Again there is no way to identify these patients without an initial trial of US. So the best way to approach patients with US is an initial trial where the intensity is slowly increased to determine the response.

Variability in Application Medium

Three important principles should guide the application of US:

- The radiating waves of US must be kept perpendicular to the skin surface; thus, the applicator must remain in contact with the skin surface.
- A coupling medium must be used between the US applicator and the skin.
- The applicator faceplate must be continually moved during the treatment.

It is important to keep the radiating waves of US entering the skin at a 90° angle (perpendicular) to the skin to avoid excessive reflection of the waves. When US waves transition from one medium to another, there will be a certain amount of wave reflection, while the rest of the waves will penetrate (Fig. 4-9). However, when the incident angle of the US waves is greater than 15° off of perpendicular, there can be almost complete reflection of the waves.

US waves are not transmitted through air, so a coupling medium is needed between the applicator and the skin.[24] Coupling media take three common forms: viscous aqueous gel, water immersion, and gel pads. US gel is generally a combination of propylene glycol, glycerin, phenoxyethanol, and a colorant. It serves as a lubricant for easy movement of the US applicator over the skin and as a medium for almost 100% transmission of the ultrasonic energy.[25–27] Other gels, such as K-Y Jelly, also have a high transmissivity.[27] In general, most creams and ointments have a relatively low transmissivity, both alone and when mixed with US gel.[24,25] Recent studies have suggested that low concentrations (25%) of topical analgesics such as Flexall[28,29] and even higher concentrations (50%) of Biofreeze or Nature's Chemist[30] in mixture with US gel can be effective in allowing the transmission of ultrasonic energy. However, there is no evidence that adding these agents to the US gel enhances the clinical effectiveness of US.

TABLE 4–5. Variability of Heating Response to Ultrasound Based Upon Mean Value ± Three Standard Deviations

		Range (Mean ± 3 SD)	
Tissue	Heated Temperature	Low	High
Skeletal muscle[22]	7° ± 2°F (4° ± 1.1°C)	1.3°F (0.7°C)	13°F (7.3°C)
Patellar tendon[20]	15° ± 3°F (8.3° ± 1.7°C)	5.7°F (3.2°C)	24°F (13.4°C)
Achilles tendon[19]	14° ± 3.6°F (7.9° ± 2°C)	3.4°F (1.9°C)	25°F (13.9°C)

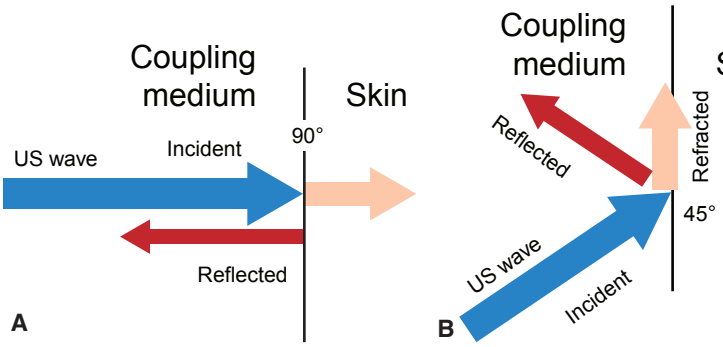


Fig 4 ■ 9 Relative reflection of US waves comparing a (A) 90° incident angle with a (B) 45° incident angle. Note that when directed perpendicular to the skin, US waves are absorbed by the tissue. However, at incident angles greater than 15 degrees, there may be complete reflection of the US waves.

Clinical Controversy

Some applications of US involve the mixing of additives, such as creams or ointments, into the US gel. Because these additives are poor mediums for US, their addition is more likely to diminish the therapeutic effect of US than to enhance it.

Water-immersion techniques are used when sonicating over irregular surfaces where good contact between the US applicator and skin cannot be easily maintained or if the area to be sonicated is smaller than the faceplate of the US applicator. In some studies where degassed water was used,[25,26] there was little or no difference in the heating effect of US between degassed water-immersion or gel when sonicating over the irregular surface.[31] With the water immersion technique, the US head should be within 1 cm of the area being sonicated.[32] Even though several studies have reported equivalent transmissivity when comparing water and ultrasonic gel,[25,27] other studies have shown that to get equivalent heating, the intensity has to be increased by as much as 50% when using 1-MHz US[33] and doubled or more when using 3-MHz US.[32]

US gel pads typically have a 9-cm diameter and a 2-cm thick transparent disk of US gel with a plastic coating (Fig. 4-10). Gel pads are used when sonicating over irregular surfaces when good contact between the US applicator and skin cannot be easily maintained or if the area to be sonicated is smaller than the faceplate of the applicator. Studies have shown that when using US at 1 MHz, gel pads have an equivalent transmissivity[34] and similar heating effect as when using US gel at 3-cm depth in muscle.[35]

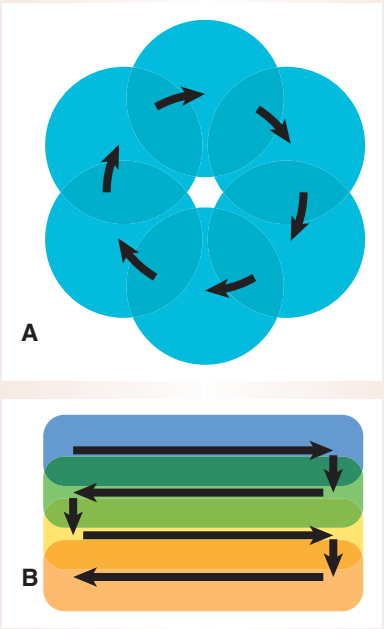
Clinical Principles in Practice: Velocity of Ultrasound Head Movement

It is commonly thought that the velocity or speed at which an US head is moved will affect the efficacy of treatment. Often recommended is a slow movement velocity of about 3 to 4 cm/s. However, Weaver et al[36] studied this using 1-MHz US and found no significant differences in tissue heating between movement of the US applicator at 2 to 3 cm/s, 4 to 5 cm/s, and 7 to 8 cm/s. This was confirmed in an unpublished thesis by Liceralde,[37] who reported no differences in tissue heating between movement velocity of the US applicator at 2 cm/s, 4cm/s, and 6 cm/s, using 1-MHz US. When properly applied in an area no larger than two to four times the ERA, it is not likely that the sound head would be moved at speeds faster than examined in these studies making velocity of movement inconsequential. However, when (incorrectly) administering US in too large an area, it becomes very easy to move the sound head too quickly, making velocity a concern and further minimizing the likelihood that tissue temperature will be affected.

Fig 4 ■ 10 An ultrasonic gel pad can be used to administer US over areas where the surface contour is irregular such as bony areas of the hand or foot.

Clinical Principles in Practice: Patterns for Moving the Sound Head

US is applied using a slow stroking pattern, but clinicians differ on the pattern of movement. The most common pattern is overlapping circles, either clockwise or counterclockwise, overlapping the previous circle by approximately half the ERA of the sound head. Alternatively, stroking may be in parallel strokes, either horizontally or vertically, but again overlapping the previous stroke by approximately half the ERA of the sound head. The area being treated may dictate the pattern of movement used, but regardless of the pattern, the sound head should remain perpendicular and in contact with the skin.



(A) Overlapping circles (clockwise or counterclockwise) and (B) overlapping parallel strokes. In both movements, the overlap should be no more than one-half of the ERA.

A summary of the considerations for application of US are provided in Box 4-1. Good clinical use of US will follow these suggestions.

THERMAL EFFECTS OF ULTRASOUND

US is a series of high-frequency sound waves. It is thought that when these high-frequency sound waves are absorbed by a tissue, the sound waves' mechanical energy is converted into thermal energy due to vibrating the molecules within the tissue. Greater levels of power (expressed usually as power density [W/cm^2]) and duration (in minutes) and reduced treatment area (generally expressed in multiples of ERA) increase this local effect and increase the heating of the tissue.[8] Additionally, the use of a higher frequency of US (i.e., 3 MHz) heats the tissue to a greater extent than using lower frequencies (i.e., 1 MHz). It is thought that with the lack of penetration of the US with higher frequencies, all of the ultrasonic energy is confined to a smaller volume of tissue, thus increasing both the energy density and the temperature of the tissue.[8] The amount of heating may also be related to tissue density with tissues such as tendons heating faster and to a greater degree than less dense tissues such as muscle.[8,20]

Box 4 ■ 1 A Checklist for Proper Application of Ultrasound[7]

- Treat the correct size area.
- Select the appropriate treatment duration.
- Adjust the intensity for the desired effect.
- Use the correct frequency.
- Don't treat all tissues with the same parameters.
- Move the sound head at an appropriate speed.
- If combined with stretching, stretch during the last few minutes of the US treatment and/or immediately after heating.

Clinical Principles in Practice: Thermal Ultrasound

The concept of heating is the most misunderstood aspect of US. When applying US for a thermal effect, some clinicians incorrectly believe patients may or may not feel warmth. When in truth, if the patient does not feel warmth, the dose is inadequate to produce thermal effects. If the tissue is being heated appropriately, the patient will feel warmth. The most common reason for not administering a proper heating dose is covering too large an area, but other parameters of US must also be considered. At any given application of US, parameters may need to be adjusted to achieve a thermal effect and sensation of warming. This emphasizes the previously stated notion that each application of US is a unique and individualized encounter and must be approached as such.

NONTHERMAL EFFECTS

In addition to the thermal effects of US, nonthermal effects have also been described. These nonthermal effects have been attributed to two processes produced by US: cavitation and acoustic streaming. Both are thought to affect cell membrane properties.

The cycles of compressions and rarefactions of US exert forces on the cell membranes and tissue fluids, resulting in the formation and accumulation of thousands of gas bubbles under the influence of Bjerknes forces. This formation of gas bubbles is termed *cavitation*. Cavitation is often described as stable or unstable. Stable cavitation is the cyclical expansion and contraction of gas bubbles in response to the acoustic cycle of compressions and rarefactions. Unstable cavitation may occur from an accumulation of a large volume of gas bubbles that collapse. This large-scale collapse is thought to be a cause for cell and tissue damage, although it has never been substantiated in vivo. O'Brien stated that there is "no experimental evidence to suggest that cavitation events occur in mammalian tissue from exposure-like conditions employed with clinical ultrasound."[38]

Cavitation has been reported in vitro[39–41] at clinically relevant levels, but this research has been criticized.[42] Leighton et al[43] failed to demonstrate cavitation in vivo in human cheek tissue. It has been speculated that soft tissues have large amounts of viscoelastic material, in contrast to a pure liquid, and recent theoretical predictions suggest that cavitation thresholds are higher for soft tissues than liquids.[44]

Acoustic streaming is the forward movement of fluid by US waves.[45] However, the bulk movement of fluid lacks force to affect tissue membranes or intercellular activity of cells; therefore, it has been suggested to have no physiological effects.[45] It has also been suggested that cavitation sets up eddy currents in the fluid surrounding the vibrating bubbles and that these eddy currents in turn exert a twisting and rotational motion on nearby cells.[46] It is postulated that these twisting and rotational forces can produce changes in cellular permeability and the release of substances such as histamine and other vasoactive agents.[46] However, this form of microstreaming requires cavitation, and the existence of cavitation in vivo has been challenged.[42]

Low-intensity US has been shown to alter cellular permeability[47,48] and to produce mast cell degranulation,[46] microshearing effects,[49] and free radical formation.[38] All of these effects have been demonstrated in vitro but not in vivo.[38,42]

TREATMENT EFFECTIVENESS OF THERAPEUTIC ULTRASOUND

Wong et al[1] reported that certified orthopedic clinical specialists use US for decreasing soft tissue inflammation, tissue swelling, and pain; increasing tissue extensibility and scar tissue remodeling; and healing acute soft tissue injuries (Table 4-6). Amijo-Olivo et al[3] reported similar use patterns among practicing physical therapists. Most interesting is that 37.3% of therapists reported that they used therapeutic US to "meet patient expectations" and 18.2% used it for its placebo effect.

Defining Therapeutic Effectiveness

Evidence for therapeutic applications in this chapter is based solely upon human clinical studies relying on common evidence-based-practice, strength-of-evidence pyramids, which shows that animal studies ranked much lower in terms of their application to clinical

TABLE 4–6. Common Uses of Therapeutic Ultrasound by APTA Certified Orthopedic Clinical Specialists[1] and Practicing Physical Therapists[3]

Impairment	Orthopedic Clinical Specialists	Physical Therapists
Soft tissue inflammation	83.6%	73.5% (acute) 61% (chronic)
Tissue extensibility	70.9%	65.1%
Scar tissue remodeling	68.8%	74.2%
Tissue healing	52.5%	58.9%
Pain	49.3%	37.1% (acute) 24.4% (chronic)
Soft tissue swelling	35.1%	NR
Meeting patient expectations	NR	37.3%
Placebo effect	NR	18.2%

NR = not reported.

decision-making. Studies are rated on a four-class scale, based upon their research design:

Class 1—represents randomized controlled trials (RCTs) with an active intervention, appropriate control groups, and subjects randomly assigned to each group.
Class 2—represents studies with an active intervention and control group without randomization to groups.
Class 3—represents studies with an active intervention but without a control group; these are often single group designs.
Class 4—represents all other studies, including single subject "experimental" designs and observational studies that involve case control, cohort, and case studies.

In this chapter, a control group is defined as any comparison group that receives no treatment or receives a sham (or placebo) treatment. When studies compared two treatments or when the other treatment is simply a variation of the treatment under investigation, this second group is not considered a control group because no untreated comparison measurement can be established to compare the effect of experimental intervention.

Clinical outcomes will be based upon whether they show a statistically significant improvement in the measured outcome variables, indicated by "yes," or whether they do not show a statistically significant improvement, indicated by "no." In studies with multiple related outcome variables, a yes or no will be indicated for each outcome measure. In a randomized controlled trial (class 1 study), there needs to be a statistically significant difference when the change in the experimental group is compared to the change in the control group. In a study without a proper control (class 2 or 3 studies), there needs to be only a significant difference in the pre-post intervention change values. In some observational studies (class 4 study), such as case studies, and in many older studies, statistical significance testing is not done. In this circumstance, the yes or no designation is made where there is a substantial effect of the therapeutic US on the outcome variable.

Therapeutic effectiveness will be classified using the proportion of studies showing improvement in clinical outcomes (modified from the approach of Belanger[50]), as follows:

- Substantiated: when the number of studies reporting improvement is greater than or equal to 60%
- Conflicting: when the number of studies reporting improvement is less than 60% but greater than or equal to 40%
- Unsubstantiated: when the number of studies reporting improvement is less than 40%
- Lack of evidence: when there are fewer than five studies of any type

Strength of evidence will be based upon the proportion of studies in each rating class, as follows:

- Strong evidence: when 50% or more of the studies are in class 1
- Moderate evidence: when greater than 50% of the studies are in classes 1, 2, and 3
- Weak evidence: when greater than 50% of the studies are in class 4
- Lack of evidence: when there are fewer than five studies reporting on the outcomes under investigation

However, an appropriate comparison of articles must be based upon a consistent use of the modality. For example, doses of US application need to be somewhat consistent for articles to be compared. When necessary, studies with continuous and pulsed US will be reviewed separately.

There are many studies where US is part of a number of interventions. When this is the case and US effects cannot be isolated from the other interventions, the study will either not be included in comparisons or the additional interventions will be noted in the table.

For example, if there are five studies on the use of US for a particular condition, conclusions can be reached. If four of the five studies show improvement in the condition being treated, US would be classified as having a substantiated effect on this condition. However, if two of these studies are RCTs and three involve case studies, US would be classified as having weak evidence for this substantiated effect.

As already described, US dose is a parameter that must be evaluated when reviewing the US literature. Application of US may be lacking proper intensity,

duration, or area of application. Problems with any of these components of dose would lead to an insufficient treatment to obtain the desired effect. In many cases, the dose is probably inadequate to produce the heating necessary to achieve the desired effect. For nonconnective tissues, a heating dose using 1 MHz should be 1.5 W/cm^2 or greater, 10 minutes or greater in an area two times the effective radiating area of the sound head. For 3 MHz, those values could be reduced by about 40% but can be used only for more superficial structures. For the tables in this chapter, area treated is not included because this part of the dose was inconsistently reported or reported in a vague manner, such as "over the lateral aspect of the shoulder." So when reviewing the results of studies, the critical reader should pay attention to dose, particularly in those studies with negative results.

Key Point! A critical evaluation of the literature on US must consider the treatment dosage and size of the area treated, if even described. Far too often, these parameters are poorly described or not described at all. This makes a critical analysis of the literature challenging.

Ultrasound for Painful Conditions

Pain is a common symptom that is treated by US. Much of the pain is secondary to inflammatory conditions, tissue swelling, or lack of tissue extensibility. Pain under these circumstances will be discussed with those conditions. The use of US has been extensively studied for three common painful conditions: myofascial pain syndrome (specifically myofascial trigger points), back pain, and nonspecific shoulder dysfunction. Tendinitis, bursitis, and adhesive capsulitis will be discussed later.

Myofascial Pain

Studies that have investigated the effect of US treatments on myofascial pain syndrome, specifically myofascial trigger points, are shown in Table 4-7. Eight of the 11 studies investigated the effects of continuous US on myofascial trigger points, and seven of these studies were RCTs. Three major outcomes were investigated by most of these studies: pain, pressure-pain threshold to applied pressure, and cervical range of motion (ROM). With the majority of studies for each of these outcome measures being RCTs, there is strong evidence for all effects studied. Of the five studies that measured pain levels, 60% reported a decrease in pain. Of the seven studies that measured pressure-pain threshold, 57% reported an increase in this threshold. Of the six studies that measured cervical ROM, 33% reported an increase. When looking at all of the studies, there is substantiated effect for decrease in pain, conflicting evidence for increase in the pressure-pain threshold, and unsubstantiated effect for an increase in cervical ROM.

Examining the literature more deeply, Manca et al[51] observed that US application had a significant effect when compared to an untreated control but not when

TABLE 4–7. Effect of Ultrasound Treatments on Myofascial Pain Syndrome (Specifically Myofascial Trigger Points)

Reference	Rating	US Intervention	Outcomes	Improvement
Lee et al, 1997[93]	1	Single treatment, 6 min, 1 MHz, continuous at 0.5 W/cm^2	1. ROM cervical lateral flexion 2. Pain intensity—VAS 3. MTrP pressure-pain threshold—pressure algometer	1. No 2. No 3. No
Esenyel et al, 2000[52]	1	5/wk, 6 min, 1 MHz, continuous at 1.5 W/cm^2 for 2 wks	1. Pain—VAS 2. MTrP pressure-pain threshold—pressure algometer 3. Lateral bending ROM to opposite side	1. Yes 2. Yes 3. Yes

Continued

TABLE 4–7. Effect of Ultrasound Treatments on Myofascial Pain Syndrome (Specifically Myofascial Trigger Points)—cont'd

Reference	Rating	US Intervention	Outcomes	Improvement
Srbely et al, 2008[94]	1	Single treatment, 5 min, 1 MHz, continuous at 1 W/cm^2	MTrP pressure-pain threshold—pressure algometer	Yes
Ay et al, 2011[95]	1	5/wk, 10 min, 1 MHz, continuous at 1.5 W/cm^2 for 3 wks	1. Pain—VAS 2. # trigger points 3. MTrP pressure-pain threshold—pressure algometer 4. Cervical ROM—flexion-extension, lateral flexion and rotation 5. Disability—neck pain disability scale	1. Yes 2. Yes 3. Yes 4. Yes 5. Yes
Aguilera et al, 2009[96]	1	Single treatment, 2 min, 1 MHz, continuous at 1 W/cm^2	1. Pain threshold—VAS with pressure on MTrP 2. Basal EMG activity 3. Active cervical rotation ROM	1. Yes 2. Yes 3. No
Draper et al, 2010[97]	1	Single treatment, 5 min, 3 MHz, continuous at 1.4 W/cm^2	Decreased muscle stiffness—pressure algometer	Yes
Kannan, 2012[98]	3	5/wk, 5 min, 1 MHz, continuous at 1.5 W/cm^2 for 3 wks	1. Pain—VAS 2. Provocative pain test—0–4 scale 3. Active cervical lateral flexion	1. Yes 2. No 3. No
Manca et al, 2014[51]	1	5/wk, 12 min, 3 MHz, continuous at 1.5W/cm^2 for 2 wks	1. Pain—VAS 2. Cervical ROM—lateral flexion 3. MTrP pressure-pain threshold—pressure	1. No 2. No 3. No
Gam et al, 1998[99]	1	2/wk, 3 min, 1 MHz, 20% pulsed at 3 W/cm^2 for 4 wks	1. Pain at rest—VAS 2. Pain with normal daily activity—VAS 3. Daily analgesic usage—type and # tablets/day 4. Number, size, and tenderness of MTrP 5. Global preference—treatment satisfying	1. No 2. No 3. No 4. No 5. No
Srbely et al, 2008[94]	1	Single treatment, 10 min, 1 MHz, 50% pulsed at 0.52 W/cm^2	MTrP pressure-pain threshold—pressure algometer	Yes
Sarrafzadeh et al, 2012[100]	3	4/wk, 10 min, 1 MHz, 20% pulsed at 1.2 W/cm^2 for 1 wk	1. Pain—VAS 2. Cervical ROM—lateral flexion 3. MTrP pressure-pain threshold—pressure	1. Yes 2. Yes 3. Yes

VAS: visual analog scale; MTrP: myofascial trigger point.

compared to a placebo control. With this in mind, one of the studies that showed uniformly positive effects of US[52] did not use a placebo control. If this study is eliminated from consideration, both cervical ROM and pain become unsubstantiated, and there are conflicting results for pressure-pain threshold. So there is clearly a strong unsubstantiated effect for the treatment of myofascial trigger points with US to improve cervical ROM. It could be argued that both pain and pain-pressure threshold show conflicting results. Thus, it would be difficult to argue that US has any effect in the treatment of myofascial pain syndrome and myofascial trigger points. However, if a therapist wishes to use US to treat pain in these patients using current best evidence, the recommended intervention approach would be 10-minute treatments four to

CASE STUDY 4-1 Treatment of Trigger Point Pain With Ultrasound

Mr. Roberts is a 25-year-old professional football defensive lineman. He reports point pain in his upper scapular area with pain limiting his cervical ROM, particularly side bending to the left. Upon examination, two active trigger points are palpated in the right middle scalene muscle, one near the vertebral attachment and one near the proximal attachment.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of US?

ANSWER: US may be effective in reducing the pain, and there is some evidence suggesting improved range of motion in patients with myofascial trigger points particularly when combined with other interventions such as stretching.

2. Is the patient appropriate for therapeutic US—that is, do any of the general precautions or contraindications to the use of US apply to the patient, or are there any specific considerations regarding application of US to this patient?

ANSWER: There are no specific contraindications or precautions noted. The area should be inspected for trauma and acute inflammation, considering the patient's occupation. In addition, precaution should be made to keep the applicator lateral on the neck and not to sonicate the anterior neck.

3. What are the specific goals to be achieved with the use of US?

ANSWER: Reduced pain is a goal that is supported by the literature. There is some controversy as to whether there is reduced pain to palpation and increased cervical ROM associated with the treatment of trigger points with US. So whether to include these as therapeutic goals would be up to the clinician's reading of the literature.

4. What are the specific parameters of US that are appropriate for the patient?

ANSWER: From evidence in the literature, the clinician should consider treating the patient daily on each trigger point for 10 minutes, with each treatment using 1-MHz, continuous US set at 1 to 1.5 W/cm^2 over the trigger point. Using a 5-cm^2 US head, the clinician would be able to treat only one of the trigger points because they are quite far apart—too far to include them both in the appropriate treatment area (two to four times the ERA).

5. What is the expected prognosis for this patient?

ANSWER: There should be a reduction in pain and improvement in reported function. Expected duration of treatment is 2 to 3 weeks or 10 to 15 treatments to achieve the desired outcome. However, there is some evidence that improvement in pain can be seen with a single treatment, so observing the patient's response to the first few treatments might be a good indication of the eventual success of this intervention approach.

6. What are the proper application procedures for US with this patient?

ANSWER: Instruct the patient as to the purpose of the US and procedure to be used. Be sure to explain that he should feel a gentle warming sensation but should not feel any stinging or burning discomfort. Inspect the neck for trauma and acute inflammation. Clean the neck of any lotions or ointments that might have been applied. Apply a little bit of stretch to the scalene muscle to be treated with a slight, nonpainful lateral bending to the left. Apply gel to the US head. Place the US to the single trigger (two to four times the ERA), seeking continuous feedback from the patient and applying some additional stretch as the treatment continues. Repeat the treatment at the next trigger point.

five times per week using 1 or 3 MHz applied continuous at 1 to 1.5 W/cm^2 for 2 to 3 weeks.

Back Pain

Studies that have investigated the effect of US treatments on back pain are shown in Table 4-8. Of the six articles that investigated the effect of continuous US on back pain and function, only five specifically addressed pain. Of these five, three are RCTs and two are uncontrolled studies; also, three reported a decrease in pain, one reported no change in pain, and one had mixed results. Thus, in the use of US to treat back pain, there is strong evidence for a substantiated

TABLE 4–8. Effect of Ultrasound Treatments on Back Pain Syndrome

Reference	Rating	US Intervention	Outcomes	Improvement
Aldes and Grabin, 1958[101]	3	5/wk, 10 min, 1 MHz, continuous at 0.3–0.8 W/cm^2 for 3 wks	Pain	Yes*
Nwuga, 1983[102]	1	3/wk, 10 min, 1 MHz, continuous at 1–2 W/cm^2 for 4 wks	1. Spine ROM—1 degree 2. Pain—on 1–4 scale	1. Yes 2. Yes
Ansari et al, 2006[103]	1	3/wk, 8 min, 1 MHz, continuous at 1.5 W/cm^2 for 3 wks	1. Functional rating index 2. Lumbar ROM 3. H-reflex—latency and Hmax/Mmax ratio	1. Yes 2. Yes 3. No
Mohseni-Bandpei et al, 2006[104]	3	1–2/wk, 5–10 min, 1 MHz, continuous at 1.5–2.5 W/cm^2 for 3–5 wks	1. Pain—VAS 2. Disability—Modified Oswestry LBP Disability Questionnaire and Pain Disability Index 3. Lumbar ROM 4. Median frequency and slope back muscle EMG activity	1. Yes 2. Yes 3. Yes 4. No
Durmus et al, 2010[105]	1	5/wk, 10 min, 1 MHz, continuous at 1 W/cm^2 for 3 wks	1. Pain at rest—VAS 2. Pain with activity—VAS 3. Disability—Modified Oswestry LBP Disability Questionnaire and Pain Disability Index 4. Walking—6-min walk 5. Quality of life—SF-36 and Beck Depression Inventory	1. Yes 2. No 3. No 4. Yes 5. Yes—SF-36 physical functioning/no others
Ebadi et al, 2012[53]	1	3/wk, 8 min, 1 MHz, continuous at 1.5 W/cm^2 for 4 wks	1. Functional disability—Functional Rating Index 2. Global pain—VAS 3. Back ROM 4. Endurance time—median frequency EMG decline rate during a Biering-Sorensen test	1. No 2. No 3. No 4. No

*No statistical analysis.

effect. However, if only the RCTs are considered, one reports a decrease in pain, one reports no change, and one has mixed results. This would perhaps suggest conflicting results, but no definite conclusions can be reached from only these three studies.

In addition, five of these studies showed an improvement in back function—either an increase in range of motion, an improved score of a functional rating index, or a decline in a disability index. Of these five, four are RCTs and one is an uncontrolled study; also, three reported an improvement in function and two reported no change. Thus, in the use of US to treat back dysfunction, there is strong evidence for a substantiated effect. However, if only the RCTs are considered, two report an improvement in function and two report no change. This would perhaps suggest conflicting results, but no definite conclusions can be reached from only these four studies. The effect of US on back pain and dysfunction was recently examined in a single blind placebo controlled study by Ebadi et al suggesting that the addition of US to an exercise program significantly improved function, lumbar ROM, and endurance time.[53]

However, if a therapist chooses to use US to treat back pain and dysfunction based on current best evidence, the optimal treatment would appear to be US administered three times per week for 10 minutes per treatment, using 1MHz, continuous US at 1 to 2 W/cm^2 for a 4-week course of treatment.

Ultrasound for Shoulder Conditions

US has also been used to treat a variety of shoulder conditions. Studies that have investigated the effect of US treatments on these nonspecific shoulder conditions are shown in Table 4-9.

In the use of continuous US on nonspecific shoulder conditions, there are six total studies; of these, two are RCTs that reported on shoulder dysfunction. Only three of these studies reported explicitly on pain. Because the four uncontrolled studies show a positive effect, there would be a moderate substantiated effect of US on shoulder

TABLE 4–9. Effect of Ultrasound Treatments on Nonspecific Shoulder Conditions

Reference	Rating	US Intervention	Outcomes	Improvement
Echternach, 1965[106]	3	3–5/wk, 5–8 min, 1 MHz, continuous at 1.4–1.8 W/cm^2, 2–3 wks	ROM and function	Yes
Inaba and Piorkowski, 1972[107]	1	5/wk, 10 min, 1 MHz, continuous at 0.5–2 W/cm^2 for 3 wks	Shoulder ROM without protective reactions or complaints of pain	No
Herrera-Lasso et al, 1993[108]	3	3/wk, 10 min, 1 MHz, continuous at 0.5–1 W/cm^2 for 4 wks followed by stretching and exercise	1. Shoulder flexion and abduction ROM—degrees 2. Pain—VAS	1. Yes flex; no abduction 2. Yes
Shehab and Adham, 2000[109]	3	3–5/wk, 10 min, 1 MHz, continuous at 0.5–2 W/cm^2 for 3–4 wks followed by stretching and exercise	1. Shoulder flexion and abduction ROM—degrees 2. Pain—VAS	1. Yes 2. Yes

Continued

TABLE 4–9. Effect of Ultrasound Treatments on Nonspecific Shoulder Conditions—cont'd

Reference	Rating	US Intervention	Outcomes	Improvement
Kurtais-Gürsel et al, 2004[110]	1	5/wk, 10 min, 1 MHz, continuous at 0.42 W/cm^2 for 3 wks	1. Pain—on 0–3 scale 2. Shoulder active and passive ROM—degrees 3. Health Assessment Questionnaire (HAQ)—2 to 3 questions in 8 sections, each scored 0–3 4. Shoulder Disability Questionnaire (SDQ)—16 items scored yes/no	1. No 2. No 3. No 4. No
Johansson et al, 2005[111]	3	2/wk, 10 min, 1 MHz, continuous at 1 W/cm^2 for 5 wks combined with home exercise program	Combination of three measures (Constant-Murley shoulder assessment; Adolfsson-Lysholm, shoulder score; and UCLA shoulder scoring scale)	Yes
Nykänen, 1995[112]	1	3/wk, 10–12 min, 1 MHz, 20% pulsed at 1 W/cm^2 for 3–4 wks	1. Shoulder abduction ROM—degrees 2. Pain-free shoulder abduction ROM—degrees 3. Supraspinatus pain score (0–3) 4. Pain index—on scale of 4–20 5. Function (ADL) index—on scale of 3 to–14	1. No 2. No 3. No 4. No 5. No
van der Heijden et al, 1999[113]	1	2/wk, 2 min/cm^2 of area, 1 MHz, 20% pulsed at 1.5 W/cm^2 for 6 wks	1. Overall improvement—7-point Likert scale 2. Shoulder Disability Questionnaire (SDQ) 3. Therapist rated symptom severity—VAS	1. No 2. No 3. No
Ainsworth et al, 2007[114]	1	US dose varied; 3–4/wk, 4.5 min average (range 3–7 min), 1 or 3 MHz (46%, 39%, 15% not specified), 20% pulsed at average 0.5 W/cm^2 (0.2–1 range)	1. Shoulder Disability Questionnaire (SDQ-UK)—23 items scored yes/no 2. Global assessment of change shoulder problem—5-point scale of "complete recovery" to "much worse" 3. Average pain during the previous 24 h during the day and during the night—two VAS scales 4. Perception of functional deficit—VAS 5. EuroQol EQ-5D and the EuroQol health thermometer scales—both 0–100 6. Active and passive shoulder ROM—degrees	1. No 2. No 3. No 4. No 5. No 6. No

dysfunction related to nonspecific shoulder conditions. However, with both RCTs showing no effect, it would be difficult to endorse the use of continuous US for this condition. There is insufficient evidence on the effect of continuous US on pain associated with nonspecific shoulder conditions. But if the therapist is still considering using continuous US, current best evidence would suggest that administering treatment three to five times per week, using 1 MHz applied in a continuous mode at 0.5 to 2 W/cm^2 for 10 minutes for 3 to 4 weeks may be beneficial, particularly when accompanied by stretching and exercise.

Evidence is much clearer for pulsed US on nonspecific shoulder conditions. Even with only three studies, there is insufficient evidence to reach a conclusion. All three studies are RCTs and all show no effect. So it is clear that pulsed US should not be recommended for the treatment of nonspecific shoulder pain and dysfunction.

Ultrasound for Inflammation

There have been quite a few studies on a variety of inflammatory conditions, including lateral epicondylitis, carpal tunnel syndrome, calcific tendinitis, bursitis, and arthritis. Studies that have investigated the effect of US treatments on lateral epicondylitis are shown in Table 4-10.

TABLE 4–10. Effect of Ultrasound Treatments on Lateral Epicondylitis

Reference	Rating	US Intervention	Outcomes	Improvement
Binder et al, 1985[115]	1	2–3/wk, 5–10 min, 1 MHz, 25% pulsed at 1–2 W/cm^2 for 4–6 wks	1. Pain—VAS 2. Pain—wrist extended 3. Pain—lifting weight 4. Grip strength—hand dynamometer 5. Overall outcome	1. Yes 2. Yes 3. Yes 4. Yes 5. Yes
Haker and Lundeberg, 1991[116]	1	2–3/wk, 10 min, 1 MHz, 25% pulsed at 1 W/cm^2 for 4–5 wks	1. Pain—wrist extended 2. Pain—change from pre-treat 3. Pain—lifting weight 4. Grip strength—hand dynamometer	1. No 2. No 3. No 4. No
Pienimaki et al, 1996[54]	3	2–3/wk, 10–15 min, 1 MHz US, 20% pulsed at 0.3–0.7 W/cm^2 for 8 wks	1. Pain at rest—VAS 2. Pain under stress—VAS 3. Functional limitations—various measures 4. Grip strength—hand dynamometer 5. Wrist/forearm muscle isokinetic performance	1. No 2. No 3. No 4. No 5. No
Pienimaki et al, 1998[55]	3	2–3/wk, 10–15 min, 1 MHz US, 20% pulsed at 0.3-0.7 W/cm^2 for 8 wks	1. Physiotherapy visits in 3 years post–US treatment 2. Medical consultations in 3 years post–US treatment 3. Treatments in 3 years post–US treatment 4. Sick-leave days in 3 years post-treatment	1. No 2. No 3. No 4. No
Davidson et al, 2001[117]	3	2–3/wk, 10 min, 1 MHz, 20% pulsed at 1 W/cm^2 for 3 wks	1. Pain level 2. Pain-free grip strength 3. DASH disability questionnaire scores	1. Yes 2. Yes 3. Yes

Continued

TABLE 4–10. Effect of Ultrasound Treatments on Lateral Epicondylitis—cont'd

Reference	Rating	US Intervention	Outcomes	Improvement
D'Vaz et al, 2006[17]	1	Daily, 20 min, 1.5 MHz, pulsed at average 30 mW/cm^2 for 12 wks	1. Pain—VAS 2. Grip strength—hand dynamometer 3. Patient-Rated Forearm Evaluation Questionnaire	1. No 2. No 3. No
Aldes, 1956[56]	3	3/wk, 5 min, 1 MHz, continuous at 0.5–1 W/cm^2 for wks	1. Pain upon palpation 2. Pain with "doorknob test"	1. Yes* 2. Yes*
Lundeberg et al, 1988[57]	1	2–3/wk, 10 min, 1 MHz, continuous at 1 W/cm^2 for 5–6 wks	1. VAS pain 2. Resisted wrist extension 3. Lifting test 4. Grip strength 5. Patient satisfaction	1. No 2. No 3. No 4. No 5. No
Akin et al, 2010[58]	1	Daily, 5 min, 1 MHz, continuous at 1.5 W/cm^2 for 3 wks	1. Pain while relaxed—VAS 2. Pain with movement—VAS 3. Grip strength—hand dynamometer 4. SF-36 5. Disabilities of the arm, shoulder, and hand (DASH)—score 1–5 on each of 30 items 6. Satisfaction with treatment—score 1–5	1. No 2. Yes 3. No 4. No 5. Yes 6. Yes

*No statistical analysis.

Lateral Epicondylitis

For the use of US in the treatment of lateral epicondylitis, the evidence is not strong. Using pulsed US, 40% of the five articles show a positive response, placing the results at the low end of conflicting evidence. With only two RCTs, the strength of evidence is classified as moderate. D'Vaz et al[17] were not included in this analysis because, with the US at a very low dose, it is hard to distinguish the intervention from a placebo control. Even when the two studies that show the short-term[54] and long-term results[55] from the same treated group of patients are combined, the evidence is still conflicting. The effect of US on lateral epicondylitis is not clarified when considering the effect of continuous US. The only study showing consistent effects on both function and pain is uncontrolled.[56] One RCT[57] shows no effect, and one RCT[58] shows very mixed effects on both pain and function. So it would be difficult to recommend the use of US to treat lateral epicondylitis. However, if a therapist chooses to use US to treat lateral epicondylitis, the current best evidence would suggest trying two to three times per week, for 5 to 10 minutes per treatment using 1 MHz, 25% pulsed at 1 to 2 W/cm^2 over a period of 3 to 6 weeks.

Carpal Tunnel Syndrome

With seven total studies (four being RCTs) and 71% positive results, there is strong substantiated evidence for the use of continuous US to treat carpal tunnel syndrome and expect positive functional outcomes and a reduction of pain (Table 4-11). However, an effect on median nerve electrophysiological changes is unsubstantiated. With only four studies, use of pulsed US is lacking sufficient evidence to reach a conclusion. However, with 75% of the four existing studies reporting a positive effect of US, this suggests that there may be a beneficial effect of pulsed US on carpal tunnel syndrome. Using the literature as a

Text continues on page 114

TABLE 4–11. Effect of Ultrasound Treatments on Carpal Tunnel Syndrome

Reference	Rating	US Intervention	Outcomes	Improvement
Ebenbichler et al, 1998[118]	1	5/wk for 2 wks and 2/wk for 5 wks, 15 min, 1 MHz, 20% pulsed at 1 W/cm^2 for 7 wks	1. Median motor and sensory nerve conduction 2. Present and Worst Pain—VAS 3. Hand grip and finger pinch strength—dynamometer 4. Overall change at the end of the treatment—on 5-point scale	1. Yes 2. Yes 3. Yes 4. Yes
Bakhtiary, 2004[119]	3	5/wk, 15 min, 1 MHz, 20% pulsed at 1 W/cm^2 for 3 wks	1. Pain—VAS 2. Pinch and hand grip strength—dynamometer 3. Nerve conduction	1. Yes 2. Yes 3. Yes
Baysal et al, 2006[120]	3	5/wk, 10 min, 1 MHz, 20% pulsed at 1 W/cm^2 for 3 wks	1. Pain—VAS 2. Tinel's and Phalen's sign 3. Two-point discrimination—radial 3 digits 4. Carpal tunnel symptoms—11 items on 1–5 scale 5. Hand function—8 items on 1–5 scale 6. Pinch and hand grip strength—dynamometer 7. Median nerve conduction 8. Satisfaction—on 4-point scale	1. Yes 2. Yes 3. No 4. Yes 5. Yes 6. Yes 7. Yes 8. No
Yildiz, 2011[121]	1	5/wk, 15 min, 1 MHz, pulsed at 1 W/cm^2 for 2 wks	1. Carpal tunnel symptoms—11 items on 1–5 scale 2. Pain—VAS 3. Hand function—8 items on 1–5 scale 4. Median nerve conduction	1. No 2. No 3. No 4. No
Oztas et al, 1998[122]	1	5/wk, 5 min, 3 MHz, continuous at 1.5 W/cm^2 and 0.8 W/cm^2 and 0.0 W/cm^2 for 2 wks	1. Present pain—VAS 2. Pain—amount day, night, and awakened with pain—on 4-point scale 3. Median motor and sensory nerve conduction	1. No 2. No 3. No
Koyuncu, 1995[123]	3	5/wk, 5 min, 1 MHz, continuous at 1 W/cm^2 for 4 wks *or* 5/wk, 5 min, 3 MHz, continuous at 1 W/cm^2 for 4 wks	1. Pain—on 0–3 scale 2. Paraesthesia—on 0–3 scale 3. Superficial touch sensation—normal/decreased 4. Median nerve motor conduction 5. Median nerve sensory conduction	1. Yes 2. Yes 3. Yes 4. Yes—3 MHz only 5. Yes

Continued

TABLE 4–11. Effect of Ultrasound Treatments on Carpal Tunnel Syndrome—cont'd

Reference	Rating	US Intervention	Outcomes	Improvement
Piravej, 2004[124]	3	5/wk, 10 min, 1 MHz, continuous at 0.5 W/cm^2 for 4 wks	1. Pain—VAS 2. Presence of pain and/or paraesthesia symptoms at night or day—on 4-point scale 3. Frequency of awakening—on 4-point scale 4. Median nerve SNAP amplitude 5. Median nerve conduction velocity 6. Median nerve—CMAP	1. Yes 2. Yes 3. Yes 4. Yes 5. No 6. No
Ekim, 2008[125]	1	5/wk, 5 min, 3 MHz, continuous at 1.5 W/cm^2 for 2 wks	1. Carpal tunnel symptoms—11 items on 1–5 scale 2. Pain—VAS 3. Hand function—8 items on 1–5 scale 4. Grip strength—dynamometer 5. Tinel's and Phalen's tests 6. Median nerve conduction	1. Yes 2. Yes 3. Yes 4. No 5. No 6. No
Dincer et al, 2009[126]	1	5/wk, 3 min, 3 MHz, continuous at 1 W/cm^2 for 2 wks	1. Boston Questionnaire—11 questions on symptom severity and 8 questions on functional status 2. Patient satisfaction—on 5-point scale 3. Pain 0–10 VAS 4. Median motor and sensory nerve conduction	1. Yes 2. Yes 3. Yes 4. Yes
Bilgici, 2010[127]	3	5/wk, 5 min, 3 MHz continuous at 1.5 W/cm^2 for 4 wks	1. Carpal tunnel symptoms—11 items on 1–5 scale 2. Pain—VAS 3. Hand function—8 items on 1–5 scale 4. Grip strength—dynamometer 5. Two-point discrimination—radial 3 digits 6. Median nerve conduction	1. Yes 2. Yes 3. No 4. No 5. Yes 6. No
Duymaz et al, 2012[128]	1	5/wk, 5 min, 1 MHz, continuous at 0.8 W/cm^2 for 3 wks	1. Flex-Ext ROM 2. Pain at rest, during movement, or at night—VAS 3. Two-point discrimination 4. Sensory thresholds—Semmes-Weinstein monofilament 5. Tinel's and Phalen's tests 6. Carpal tunnel symptoms—11 items on 1–5 scale 7. Hand function—8 items on 1–5 scale	1. No 2. No 3. No 4. No 5. Yes 6. No 7. No

TABLE 4–11. Effect of Ultrasound Treatments on Carpal Tunnel Syndrome—cont'd

Reference	Rating	US Intervention	Outcomes	Improvement
			8. Hand grip and pinch strength—dynamometer	8. No
			9. General health assessment	9. No
			10. Median nerve conduction	10. No

SNAP = Sensory nerve action potential; CMAP = compound motor action potential.

CASE STUDY 4-2 Treatment of Carpal Tunnel Syndrome With Ultrasound

Mr. Hope is a 45-year-old male with bilateral carpal tunnel syndrome who reports wrist and hand pain and loss of fine movement in his left hand. The patient is obese and has type 2 diabetes.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of US?

ANSWER: US has been shown to be effective in reducing the pain and patient-reported disability associated with carpal tunnel syndrome. However, there is no evidence of an improvement in grip strength or electrophysiological measures of median nerve function. The patient's type 2 diabetes needs to be addressed. The question is whether the symptoms being exhibited are from the carpal tunnel syndrome or from the diabetes. The clinician needs to confirm that the symptoms are reproduced by extreme flexion or extension of the wrist.

2. Is the patient appropriate for therapeutic US—that is, do any of the general precautions or contraindications to the use of US apply to the patient, or are there any specific considerations regarding application of US to this patient?

ANSWER: The diabetes could be a contraindication for the US treatment if there is loss of blood flow and sensation in the wrist, so this needs to be checked. There is no indication of problems with vascular supply or innervation to the wrist, but these should be tested before initiating the use of US.

3. What are the specific goals to be achieved with the use of US?

ANSWER: Reduced pain and improved function of the left hand. Reduction of pain and improved function can be expected with the US treatment.

4. What are the specific parameters of US that are appropriate for the patient?

ANSWER: From evidence in the literature, the clinician should consider treating the patient daily for 5 minutes per treatment using 1MHz, continuous US set at 1 to 1.5 W/cm^2 over the ventral wrist. Using a 5-cm^2 US head, the clinician should be able to treat much of the wrist area (two to four times the ERA).

5. What is the expected prognosis for this patient?

ANSWER: There should be a reduction in pain and improvement in reported function. Expected duration of treatment is 4 weeks, or 20 treatments, to achieve the desired outcome. However, there is some evidence that improvement can be seen within 2 weeks of treatment.

6. What are the proper application procedures for US with this patient?

ANSWER: Instruct the patient as to the purpose of the US and procedure to be used. Be sure to explain that he should feel a gentle warming sensation but should not feel any stinging or burning discomfort. Inspect the wrist for proper blood flow and sensation. Clean the wrist of any lotions or ointments that might have been applied. Place the forearm in a relaxed supinated position on a flat surface for easy access to the wrist. Place gel on the US head. Apply the US to the limited treatment area (two to four times the ERA), seeking continuous feedback from the patient.

guide for practice, it would be recommended that US be used at least five times per week for 5 to 10 minutes with either 1- or 3-MHz continuous US at 0.5 to 1.5 W/cm^2. Treatments should continue for 4 weeks, but at least one study demonstrated sufficient improvement within 2 weeks. Alternatively, if the therapist chooses to use pulsed US, it is suggested that they treat the patient five times per week for 15 minutes using 1 MHz set at 20% pulsed and 1 W/cm^2 for 3 weeks.

Calcific Tendinitis

In studies on calcific tendinitis, the dosage used was quite varied, with five studies using continuous and two studies using pulsed US (Table 4-12). Considering the

TABLE 4–12. Effect of Ultrasound Treatments on Calcific Tendinitis

Reference	Rating	US Intervention	Outcomes	Improvement
Bundt, 1958[129]	4	5/wk, 5 min, 1 MHz, continuous at 1.5 W/cm^2 for 2 wks	1. Shoulder pain 2. Radiographic analysis	1. Yes 2. Yes
Flax, 1964[130]	2	5/wk, 10 min, 1 MHz, continuous at 2 W/cm^2 for 2–6 wks	1. Shoulder ROM 2. Radiographic analysis	1. No* 2. No*
Echternach, 1965[106]	3	3–5/wk, 5–8 min, 1 MHz, continuous at 1.4–1.8 W/cm2 for 2–3 wks	ROM and Function	Yes
Perron and Malouin, 1997[131]	1	3/wk, 5 min, 1 MHz, continuous at 0.8 W/cm^2 for 3 wks after nine treatments of acetic acid iontophoresis	1. Area and density of the calcium deposits (CD) 2. Passive shoulder abduction ROM 3. Pain intensity during passive abduction—on 0–5 scale	1. No 2. No 3. No
Shomoto et al, 2002[132]	1	3/wk, 5 min/ERA, 3 MHz, continuous at 1–2 W/cm^2 for 9–13 wks	1. Shoulder pain with movement 2. Radiographic analysis	1. Yes 2. Yes
Ebenbichler et al, 1997[133]	4	4–5/wk, 10 min, 1 MHz, 20% pulsed at 2 W/cm^2 for 4–8 wks	1. Shoulder pain and ROM 2. Radiographic analysis	1. Yes* 2. Yes*
Ebenbichler et al, 1999[134]	1	5/wk, 10 min, 1 MHz, 20% pulsed at 2.5 W/cm^2 for 3 wks, then 3/wk for additional 3 wks	1. Radiographic analysis 2. 100-point Constant Score 3. Pain index 4. QOL—VAS	1. Yes 2. Yes 3. Yes 4. Yes

* No statistical analysis.
ERA = effective radiating area; QOL = quality of life.

five studies using continuous US, 60% of the studies report a positive effect on pain or function, with 80% of the studies in the class 1–3 categories. This indicates a moderate confirmed effect of continuous US on pain or dysfunction associated with calcific tendinitis. A positive effect in both studies using pulsed US suggests a beneficial effect of pulsed US on calcific tendinitis of the shoulder. However, these two studies were from the same research group, and their work has not been confirmed. Using the literature as a guide for practice, it is recommended that the clinician treat at least three times per week for 10 minutes at 1- or 3-MHz continuous US at 1 to 2 W/cm^2. Treatments should continue for 4 to 8 weeks but may need to continue longer if sufficient improvement is not seen.

Bursitis

Six of nine studies addressing bursitis were RCT, two were controlled studies without randomization to groups, and three were group studies lacking controls (Table 4-13). The remaining three studies were case studies. Two of the studies[59–60] used the same data set, so these studies were merged into a single result. Seven of the eight results reported decreases in pain, increases in ROM or function, or both. Using the standardized criteria, these results would suggest that there is moderate evidence for a substantiated effect of continuous US on bursitis.

However, on closer inspection, there were only four studies that reported on pain reduction, four studies that reported on increased ROM, and three studies that reported on functional improvement. So on each of these measures there is insufficient evidence to support the use of US. Seven of the eight studies did not use statistical analyses to reach their conclusions. In addition, the single RCT demonstrated no statistically significant effect of US on pain, ROM, or five functional measures, and one controlled study without randomization to groups reported no improvement in ROM. Therefore, it could easily be argued that there was no substantiated effect. So even though the use of US to treat bursitis may be recommended, there are valid concerns about its effectiveness.

With all of these considerations, if the clinician chooses to use US on bursitis, the best evidence would suggest treating the patient three times per week for 5 to 10 minutes per treatment using 1 MHz, continuous at 1 W/cm^2 over a period of 3 to 4 weeks.

Arthritis

In reviewing the literature on the effect of continuous US on the treatment of patients with arthritis, US seems to have a moderate, substantiated effect, with 46% of the studies being RCTs and 67% of the studies having positive outcomes for pain reduction, increased ROM, and/or functional improvements (Table 4-14).

However, on closer inspection, there were 11 studies that reported on pain reduction, nine studies that reported on increased ROM, and eight studies that reported on functional improvement. Of the studies on pain reduction, 55% were RCTs and 73% reported a positive outcome, which suggests a strong, substantiated effect of US on reducing pain associated with arthritis. However, when considering only the six RCTs, half of these reported pain reduction and half did not, which would suggest a conflicting effect. Of the studies on increased ROM, only 33% were RCTs and 56% reported a positive outcome, which would suggest a moderate conflicting effect of US on increasing the limited ROM associated with arthritis. With only three RCTs, no conclusions can be reached by analyzing the results of only RCTs on the effect of US on limited ROM. Of the studies on improving functional performance, 63% were RCTs and 50% reported a positive outcome, which suggests a strong, conflicting effect of US on improving limited function associated with arthritis. When considering only RCTs, the positive results fall to 40%, which is the lowest limit before becoming unsubstantiated. Thus, the strongest effect of continuous US is on pain reduction and the least effect is on improving functional limitations in patients with arthritis.

There is an insufficient number of studies using pulsed US to form any conclusions, particularly because the studies come from the same investigators. However, these studies do raise an interesting possibility. They suggest that even though US may be effective in reducing pain, increasing ROM, and improving functional limitations in patients with arthritis,[61] pulsed US does not improve any of these symptoms when added to other treatments[62] (e.g., exercise).

The effect of US on the symptoms of arthritis is not clarified by dividing the studies into higher-dose or lower-dose US. Among the nine higher-dose studies (1 MHz, continuous with power equal to or greater than 1 watt/cm^2), five were positive, three

TABLE 4–13. Effect of Ultrasound Treatments on Bursitis

Reference	Rating	US Intervention	Outcomes	Improvement
Roden, 1951[135]	2	5/wk, 2–15 min, 1 MHz, continuous at 1.1 W/cm^2 for 4 wks	ROM shoulder abduction—degrees	Yes*
Bearzy, 1953[136]	4	3–5/wk, 5 min, 1 MHz, continuous at 2–4 W/cm^2 for 1–2 wks	Pain—patient report	Yes*
Aldes et al, 1954a[59†]	3	3/wk, 5–10 min, 1 MHz, continuous at 0.4–1.5 W/cm^2 for 3–4 wks (calcareous deposits up to 9–12 wks)	1. Improvement—on 0–3 scale 2. Reduction of calcareous deposits (when present)	1. Yes* 2. Yes*
Aldes and Klaras, 1954b[60†]	3	3/wk, 5–10 min, 1 MHz, continuous at 0.4–1.5 W/cm^2 for 3–4 wk (calcareous deposits up to 9–12 wks)	1. Improvement—on 0–3 scale 2. Reduction of calcareous deposits (when present)	1. Yes* 2. Yes*
Grynbaum, 1954[137]	2	5/wk, 7–10 min, 1 MHz, continuous at 0.5–2.5 (1.5 average) W/cm^2 for 3 wks	1. ROM shoulder—degrees 2. Pain	1. No* 2. Yes*
Cline, 1963[138]	4	5/wk, 1 MHz, continuous at 2 W/cm^2 for 2 wks	Reduction of calcareous deposits	1. Yes*
Echternach, 1965[106]	3	3–5/wk, 5–8 min, 1 MHz, continuous at 1.4–1.8 W/cm^2 for 2–3 wks	ROM and function	Yes
Gorkiewicz, 1964[139]	4	3–4/wk, 8 min, 1 MHz, continuous at 1.5 W/cm^2 for 4 wks	Pain during shoulder ROM	Yes*
Downing and Weinstein, 1986[140]	1	3/wk, 6 min, 1 MHz, continuous at 1.2 W/cm^2 for 4 wks	1. ROM—degrees 2. Pain—on 0–3 scale 3. Five functional tasks—performance difficulty on 0–10 scale	1. No 2. No 3. No

* No statistical analysis.

† Two studies used the same data set.

TABLE 4–14. Effect of Ultrasound Treatments on Arthritis

Reference	Rating	US Intervention	Outcomes	Improvement
Aldes and Jadeson, 1952[141]	3	3/wk, 3–15 min, 1 MHz, continuous at 1–2.5 W/cm^2 for 2–3 wks	Pain—degree of perceived improvement	Yes*
De Preux, 1952[142]	4	2/wk, 10 min for each of 3 areas, 1 MHz continuous at 1.5–2 watts/cm^2 for 5–15 wks	1. Pain—nocturnal and when walking 2. ROM—degrees	1. Yes* 2. Yes*
Lehmann et al, 1954[143]	3	5/wk, 5–10 min, 1 MHz, continuous at 0.5–2 W/cm^2 for 2 wks	Shoulder ROM	Yes†
Schwartz, 1953[144]	3	3/wk, 5–10 min, 1 MHz, continuous at up to 2 W/cm^2 for 1–8 wks	Pain—scale not indicated	Yes*
Hawkes et al, 1986[145]	3	5/wk, 6 min, 3 MHz, continuous at 0.25 W/cm^2 for 3 wks	1. Pain—VAS 2. Grip strength—mm Hg 3. Joint circumference 4. Articular index—0–3 joint tenderness to pressure 5. ROM 6. Timed task—dial 6 digits on rotary phone 7. Checklist activities	1. Yes 2. Yes 3. Yes 4. Yes 5. Yes 6. Yes 7. Yes
Mueller et al, 1954[146]	1	5/wk, 5 min, 1 MHz, continuous at 2 W/cm^2 for 2 wks	1. Pain—score of pain with movement, at rest, awakened by pain 2. Physician overall improvement assessment	1. No 2. No
Svarcová et al, 1988[147]	4	3/wk, unclear, 3 wks	Pain—VAS	Yes
Falconer et al, 1992[148]	1	5/wk, 5 min over each of 4 areas, 1 MHz, continuous at 2 W/cm^2 for 2 wks	1. Pain—VAS 2. ROM	1. No 2. No
Konrad, 1994[149]	1	3/wk, 10 min, 1 MHz, continuous at 0.05W/cm^2 for 3 wks	1. Number of painful articulations 2. Number of swollen articulations 3. Circumference of the PIP joints (mm) 4. Morning stiffness (min) 5. Dorsal flexion of the wrist 6. Gripping strength (mm Hg)	1. Yes 2. Yes 3. No 4. Yes 5. Yes 6. Yes

Continued

TABLE 4–14. Effect of Ultrasound Treatments on Arthritis—cont'd

Reference	Rating	US Intervention	Outcomes	Improvement
Ozgönenel et al, 2009[150]	1	5/wk, 5 min, 1 MHz, continuous at 1 W/cm^2 for 2 wks	1. Pain during movement—VAS 2. WOMAC osteoarthritis index 3. Time to walk 50 meters	1. Yes 2. Yes 3. Yes
Kozanoglu et al, 2003[151]	4	5/wk, 5 min, 1 MHz, continuous at 1 W/cm^2 for 2 wks	1. WOMAC osteoarthritis index—30% change 2. Pain during movement—VAS 3. ROM 4. Global Improvement Score of 0–4	1. Yes 2. Yes 3. Yes 4. Yes
Huang et al, 2005a[61]	1	3/wk, 5 min each over 3 areas, 1 MHz, continuous at 1.5 W/cm^2 for 8 wks	1. ROM 2. Pain—VAS 3. Lequesne's functional index—pain, distance walked, ADLs 4. Isokinetic peak torque 5. Time to walk 50 meters	1. No 2. No 3. No 4. Yes 5. Yes
Ulus et al, 2012[152]	1	5/wk, 10 min, 1 MHz, continuous at 1 W/cm^2 for 3 wks	1. Pain—VAS 2. WOMAC osteoarthritis index 3. Lequesne's functional index—pain, distance walked, ADLs 4. Hospital Anxiety and Depression Scale (HADS) 5. Time to walk 50 meters	1. No 2. No 3. No 4. No 5. No
Huang et al, 2005a[61]	1	3/wk, 5 min each over 3 areas, 1 MHz, 25% pulsed at 2.5 W/cm^2 for 8 wks	1. ROM 2. Pain—VAS 3. Lequesne's functional index—pain, distance walked, ADLs 4. Isokinetic peak torque 5. Time to walk 50 meters	1. Yes 2. Yes 3. Yes 4. Yes 5. Yes
Huang et al, 2005b[62]	1	3/wk, 5 min each over 3 areas, 1 MHz, 25% pulsed at 2.5 W/cm^2 for 8 wks	1. ROM 2. Pain—VAS 3. Lequesne's functional index—pain, distance walked, ADLs 4. Isokinetic peak torque 5. Time to walk 50 meters	1. Yes/No‡ 2. Yes/No‡ 3. Yes/No‡ 4. Yes/No‡ 5. Yes/Yes‡

* No statistical analysis.

† Ultrasound combined with massage and exercise.

‡ Ultrasound combined with exercise compared to control with exercise alone.

PIP = proximal interphalangeal; WOMAC = Western Ontario and McMaster Universities.

were negative, and one had mixed results. Among the four lower-dose studies (pulsed US or continuous US at a very low power of 0.05 watts/cm^2), two were positive and two were either negative or mixed results. One study did not include sufficient description of the dose to determine whether it was a high or low dose.

So even with these conflicting results, if a clinician is considering using US to treat arthritis, it would be recommended to treat the patient three times per week for

CASE STUDY 4-3 Treatment of Rheumatoid Arthritis With Ultrasound

Ms. Simon is a 58-year-old female with rheumatoid arthritis who reports pain in the joints of her hands and limited mobility with inability to grasp objects and hold them as long as she would like. The patient also has a cardiac condition and has an implanted pacemaker.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of US?
   ANSWER: The patient reports pain and disability associated with her rheumatoid arthritis. Evidence from the literature suggests that US could be effective in treating her hand pain. Furthermore, by reducing her pain, there is the possibility of helping with the reported disability associated with her disease process. However, direct evidence for helping with the "stiffness'" and disability is limited.

2. Is the patient appropriate for therapeutic US—that is, do any of the general precautions or contraindications to the use of US apply to the patient, or are there any specific considerations regarding application of US to this patient?
   ANSWER: Because the clinician will be treating in a bony area with very little soft tissue mass, care must be taken to avoid overheating the periosteum. There is no indication of problems with vascular supply or innervation, but these should be tested before initiating the use of US. The patient has a pacemaker, but the treatment area is not near the pacemaker site.

3. What are the specific goals to be achieved with the use of US?
   ANSWER: Reduced pain and improved grasping of objects. Reduction of pain can be expected with the US treatment, and the therapist is hoping that the reduction in pain can benefit the dysfunction with grasping and holding of objects.

4. What are the specific parameters of US that are appropriate for the patient?
   ANSWER: From evidence, the clinician should consider treating the patient three times per week for 5 to 10 minutes per treatment using 1MHz, continuous US set at 1 to 2 W/cm^2 over the painful joints. Because of the amount of bone in the area and the irregularity of the surface when treating the hands, it is recommended that the US be applied using a gel pad. Also, using a 5-cm^2 US head will allow the clinician to treat only a couple of joints at a time (area two to four times the ERA). The clinician should select the most restricted and painful joints or expect to apply multiple treatments, pausing to do ROM exercises between each treatment with the treated joints.

5. What is the expected prognosis for this patient?
   ANSWER: There should be a reduction in pain and perhaps improvement in function. The latter may be due to the decrease in pain. Expected duration of treatment is 2 to 3 weeks or six to nine treatments to achieve the desired outcome.

6. What are the proper application procedures for US with this patient?
   ANSWER: Instruct the patient as to the purpose of the US and procedure to be used. Explain that she should feel a gentle warming sensation as a result of the US application but should not feel any stinging or burning. Inspect the hand for proper blood flow and sensation. Clean the hands of any lotions or ointments that might have been applied. Place the hand to be treated on a flat surface. Place the gel pad between the applicator and the treatment area and apply gel to the US head. Apply the US to the limited treatment area (two to four times the ERA), seeking continuous feedback from the patient. Have the patient perform ROM exercises immediately after treatment. Inspect the hand after treatment for abnormal reddening.

5 to 10 minutes per treatment using 1MHz, continuous US set at 1 to 2 W/cm^2 for 2 to 3 weeks.

Ultrasound for Soft Tissue Healing

Healing of dermal wounds has been the major form of soft tissue healing for which US has been used. Of the 10 studies on the use of pulsed US in wound healing, nine are RCTs (Table 4-15). Four of the studies, including three of the RCTs, have a positive outcome. Four of the RCTs have negative outcomes, and two RCTs have mixed outcomes. Using the standardized criteria, this evidence strongly suggests a conflicting effect of pulsed US on wound healing.

TABLE 4–15. Effect of Ultrasound Treatments on Dermal Wounds

Reference	Rating	US Intervention	Outcomes	Improvement
Paul et al, 1960[153]	3	3/wk, 2–4 min, 3 MHz, continuous at 0.5–1 W/cm^2 for 2 wks	Wound healing	Yes
Callam et al, 1987[154]	1	1/wk, 1 min/probe head area covered, 1 MHz, continuous at 0.5 W/cm^2 for 12 wks or until healed	1. Wound area 2. Cumulative % healed in relation to time	1. Yes 2. Yes
Ericksson et al, 1991[155]	1	2/wk, 10 min, 1 MHz, continuous at 1 W/cm^2 for 8 wks or until healed	1. Wound area 2. Cumulative % healed in relation to time	1. No 2. No
Dyson et al, 1976[156]	1	3/wk, 5–10 min, 3 MHz, 20% pulsed at 1 W/cm^2 for 4 wks	Wound area	Yes
Roche and West, 1984[157]	1	3/wk, 5–10 min, 3 MHz, 20% pulsed at 1 W/cm^2 for 4 wks	Wound area	Yes
Ferguson, 1981[158]	4	5/wk, 3 min, 1 MHz, 20% pulsed at 0.5 W/cm^2 for 2–4 days	1. Pain—patient report 2. Hematoma	1. Yes 2. Yes
McDiarmid et al, 1985[64]	1	3/wk, 5–10 min, 3 MHz, 20% pulsed at 0.8 W/cm^2 for 4 wks	1. Cumulative % healed in relation to time clean wounds 2. Cumulative % healed in relation to time infected wounds 3. Median healing time	1. No 2. Yes 3. No
Lundeberg et al, 1990[159]	1	3/wk, 10 min, 1 MHz, 10% pulsed at 0.5 W/cm^2 for 4 weeks, then 2/wk for 4 wks and 1/wk for 4 wks	1. Wound area 2. Cumulative % healed in relation to time	1. No 2. No
Nusbaum et al, 1994[160]	1	3/wk, 5 min/5 cm^2 wound area, 3 MHz, 20% pulsed at 0.2 W/cm^2 for 6 wks combined with UV-C	Mean percentage of change per week in ulcer size	No, with trend toward US/UV-C

TABLE 4–15. Effect of Ultrasound Treatments on Dermal Wounds—cont'd

Reference	Rating	US Intervention	Outcomes	Improvement
ter Riet et al, 1996[65]	1	5/wk, 4–8 min (based upon size), 3 MHz, 20% pulsed at 0.1 W/cm^2 for 12 wks	1. Mean surface reduction (cm^2) 2. Mean surface reduction (%) 3. Mean healing rate (cm) 4. Mean volume reduction (mL) 5. Mean volume reduction (%) 6. Mean clinical improvement in report mark 7. Mean clinical improvement (%) 8. Subgroups based upon wound size 9. Subgroups based upon wound infection	1. No 2. No 3. No 4. No 5. No 6. No 7. No 8. Slight effect small 9. No
Franek et al, 2004[63]	1	7/wk, 5–20 min (5+1 min/cm^2 above 5 cm^2), 1 MHz, 20% pulsed at either 1 or 0.5 W/cm^2 for 3 wks	1. Average weekly rate of ulcer area reduction 2. Average weekly rate of ulcer volume reduction 3. Average weekly rate of ulcer suppurate area reduction 4. Relative ulcer suppurate area after treatment	1. Yes for 0.5 W/cm^2 2. Yes for 0.5 W/cm^2 3. Yes for 0.5 W/cm^2 4. Yes for 0.5 W/cm^2
Taradaj et al, 2008[161]	1	6/wk, 5–20 min (5+1 min/cm^2 above 5 cm^2), 1 MHz, 20% pulsed at 0.5 W/cm^2 for 7 wks	1. % total surface area change 2. % volume change with compression and drug therapy compared to compression and drug therapy alone 3. % total surface area change 4. % volume change with same as above after surgical intervention	1. Yes 2. Yes 3. No 4. No
Watson et al, 2011a,[162] 2011b[163]	1	1/wk, 5–10 min (based upon size), 1 MHz, 20% pulsed at 0.5 W/cm^2 for 12 wks	1. Time to healing of the largest eligible leg ulcer 2. Proportion of patients healed by 12 months 3. % change in ulcer size 4. Absolute change in ulcer size 5. Proportion of time participants were ulcer-free 6. Health-related quality of life 7. Incidence of adverse events	1. No 2. No 3. No 4. No 5. No 6. No 7. No

UV-C = ultraviolet C.

However, interpretation changes when subpopulations of studies are reviewed. Franek et al[63] have suggested that lower doses of pulsed US, specifically intensities of 0.5 W/cm^2, have an effect, although doses of 1 W/cm^2 and above do not. When reviewing RCTs using pulsed US doses of 0.5 W/cm^2 or below, four of the six studies (67%) show negative results for US when compared to standard treatment or sham controls. When reviewing RCTs using pulsed US doses of 1 W/cm^2, two of three studies (67%) show a positive

result. This contradicts the result of Franek et al[63] and suggests that there is a strong, unsubstantiated effect of lower-dose US on wound healing and that there may be a substantiated effect of higher-dose US. McDiarmid et al[64] suggested that US is more effective in patients whose wounds are infected, but this is contradicted by the work of ter Riet et al.[65]

There are only three studies investigating the effects of continuous US, so there is insufficient evidence to reach a conclusion. These studies were included in a recent Cochrane Collaborative review that found that "there is no reliable evidence that US hastens healing of venous ulcers."[66]

If a clinician chooses to use US despite the conflicting evidence for its effectiveness in treating wounds, the recommended protocol would be to apply 1MHz, 20% pulsed US at an intensity of 1 W/cm^2 for a duration of 5 minutes plus 1 minute for each square centimeter above 5 cm^2 of wound area. It is recommended that treatment be done three to five times per week. However, with this conflicting evidence, clinicians should consider other wound care approaches such as electrical stimulation, Pulsavac, or even MIST Therapy (discussed later in this chapter).

Ultrasound for Improving Tissue Extensibility

There are an insufficient number of studies in each area where US has been used to increase tissue extensibility. However, there are seven studies on the effect of continuous US on tissue extensibility, and they focused on adhesive capsulitis, knee extensibility, limited dorsiflexion of the ankle, hip contracture, and Dupuytren's contracture (Table 4-16). These seven studies include four RCTs (57%) and three studies that support the use of US to treat limited tissue extensibility (43%,), suggesting strong but conflicting evidence for an effect. However, with the four RCTs, three report no improvement with US when compared to a control group

TABLE 4–16. Effect of Ultrasound Treatments on Tissue Extensibility

Reference	Rating	US Intervention	Outcomes	Improvement
Lehmann et al, 1961[164]	3	5/wk, 5 min to each of 3 areas, 1 MHz, continuous at 1–2.5 W/cm^2 for 3 wks, in addition to exercise, massage, gait training	Hip and knee ROM—degrees	Yes
Hamer and Kirk, 1976[165]	3	2–7/wk, 5–8 min, 1 MHz, continuous at 0.5 W/cm^2 for 2 wks	1. Shoulder ROM—degrees 2. Pain—on scale of 1–5	1. Yes* 2. Yes*
Markham and Wood, 1980[166]	3	1/wk, 4–10 min, 1 or 3 MHz, continuous at 0.25–0.75 W/cm^2 for 3–12 wks	1. MP, DIP, PIP ROM—degrees 2. Span—cm 3. Grip strength—lb	1. Yes * 2. Yes* 3. Yes*
Draper et al, 1998[167]	1	Twice daily, 7 min, 3 MHz, continuous at 1.5 W/cm^2 for 5 days	1. Daily change in ankle ROM—degrees 2. Residual change in ankle ROM over 5 days—degrees	1. Yes 2. No

TABLE 4–16. Effect of Ultrasound Treatments on Tissue Extensibility—cont'd

Reference	Rating	US Intervention	Outcomes	Improvement
Reed et al, 2000[168]	1	2–7/wk, 3 MHz, continuous at 1.5 W/cm^2, single treatment	1. Knee ROM—degrees 2. Pain—on scale of 1–5	1. No 2. No
Knight et al, 2001[169]	1	3/wk, 7 min, 1 MHz, continuous at 1.5 W/cm^2 for 6 wks	Active and passive ankle ROM	No
Dogru et al, 2008[170]	1	5/wk, 10 min, 1 MHz, continuous at 1.5 W/cm^2 for 2 wks	1. Shoulder ROM—degrees 2. Pain—VAS 3. SPADI —13 pain and function items; each 0–100 4. SF-36	1. No 2. No 3. No 4. No

* No statistical analysis.
DIP = distal interphalangeal.

of stretching alone or with sham US, and one has mixed results. As a result from the evidence, it is difficult to recommend the use of continuous US for limited tissue extensibility. However, a careful examination of many of these studies suggest that either too little power was used or that it was applied over too large an area to achieve sufficient heating to enhance tissue extensibility.[14] Even considering this lack of evidence to support its use, if the clinician chooses to use continuous US to increase tissue extensibility, it should be applied three to five times per week at either 1 or 3 MHz at a level that would produce substantial heating to the tissue to be stretched. This would mean applying the US to an area not greater than two to four times the ERA and at a high enough power to produce noticeable heating, which would probably be 2 to 3 W/cm^2 in most patients.

Clinical Controversy

Use of continuous US for increasing tissue extensibility is not well supported by the literature. However, many of the studies suggesting no effect used insufficient US power, or as is so common, the US was applied over too large of an area. These clinical errors make interpretation of the literature precarious at best.

Ultrasound for Remodeling Scar Tissue

There are only four studies on the effects of continuous US on scar tissue remodeling, which represents insufficient evidence to make conclusions related to the effectiveness of this use of therapeutic US (Table 4-17). It should be noted that the sole RCT reports no effect.

Ultrasound for Tissue Swelling

There are only four studies on the effects of continuous US on tissue swelling, specifically ankle edema following acute sprain, which represents insufficient evidence to make conclusions related to the effectiveness of this use of therapeutic US (Table 4-18). However, it should be noted that all four studies are RCTs, and none of the studies demonstrate an effect of US on swelling, suggesting a strong unsubstantiated effect of US on tissue swelling.

REVIEW OF THE EVIDENCE

Table 4-19 shows a review of the evidence for the major suggested uses of continuous US in clinical practice. There is considerable evidence for US's effectiveness in treating a number of conditions. For example, there is

TABLE 4–17. Effect of Ultrasound Treatments on Scar Tissue Remodeling

Reference	Rating	US Intervention	Outcomes	Improvement
Bierman, 1954[171]	4	1–2/wk, 6–8 min, 1 MHz, continuous at 1–2 W/cm^2		
		Case 1: 3 wks	Case 1: Distance—fingertip to palm in inches and functional activities	Case 1: Yes*
		Case 2: 8 wks	Case 2: Pain and finger ROM	Case 2: Yes*
		Case 3: 36 wks	Case 3: Finger ROM	Case 3: Yes*
Markham and Wood, 1980[166]	3	1/wk, 4–10 min, 1 or 3 MHz, continuous at 0.25–0.75 W/cm^2 for 3–12 wks	1. MP, DIP, PIP ROM—degrees 2. Span—cm 3. Grip strength—lb	1. Yes * 2. Yes* 3. Yes*
Walker, 1983[172]	4	4 min, 1 MHz, continuous at 1 W/cm^2; frequency and duration not stated	Reduction of keloid scar	Yes
Ward et al, 1994[173]	1	3/wk, 10 min, 1 MHz, continuous at 1 W/cm^2 for 2 wks	1. ROM—degrees 2. Pain during stretching—VAS	1. No* 2. No*

* No statistical analysis.

TABLE 4–18. Effect of Ultrasound Treatments on Tissue Swelling

Reference	Rating	US Intervention	Outcomes	Improvement
Van Lelieveld, 1979[174]	1	5/wk, 5–10 min, 1 MHz, continuous at 0.5 W/cm^2 for 2 wks	Ankle swelling (joint circumference in cm)	No
Williamson et al, 1986[175]	1	3/wk, treatment parameters not described	Ankle swelling	No
Nyanzi et al, 1999[176]	1	Daily, 2 min, 3 MHz, 20% pulsed at 0.25 W/cm^2 for 3 days	Ankle swelling (joint circumference in cm)	No
Zammit and Herrington, 2005[177]	1	3/wk, 6 min, 3 MHz, 20% pulsed at 0.50 W/cm^2 for 2 wks	Ankle swelling (figure-8 in cm)	No

TABLE 4–19. Summary of Evidence for the Commonly Stated Uses of Continuous Therapeutic Ultrasound

Commonly Stated Use	Strength of Evidence	Therapeutic Effectiveness
Pain		
Myofascial pain, MTrPs—pain	Strong	Substantiated
Myofascial pain, MTrPs—pressure-pain threshold	Strong	Conflicting
Myofascial pain, MTrPs—cervical ROM	Strong	Unsubstantiated
Back pain	Strong	Substantiated
Back dysfunction	Strong	Substantiated
Nonspecific shoulder conditions—pain	Lacking evidence	Lacking evidence
Nonspecific shoulder conditions—dysfunction	Moderate	Substantiated
Inflammation		
Lateral epicondylitis	Lacking evidence	Lacking evidence
Carpal tunnel syndrome—pain	Strong	Substantiated
Carpal tunnel syndrome—dysfunction	Strong	Substantiated
Carpal tunnel syndrome—electrophysiological results	Lacking evidence	Lacking evidence
Calcific tendinitis—pain or function	Moderate	Substantiated
Bursitis—pain or function	Moderate	Substantiated
Arthritis—pain	Strong	Substantiated
Arthritis—function	Moderate	Conflicting
Arthritis—ROM	Strong	Conflicting
Soft Tissue Healing		
Dermal wounds	Strong	Conflicting
Tissue Extensibility		
Combined adhesive capsulitis, knee extensibility, limited ankle dorsiflexion, hip contracture and Dupuytren's contracture—ROM	Strong	Conflicting
Scar Tissue Remodeling		
Combined burn scars, keloid scars, lacerations, Dupuytren's contracture—ROM	Lacking evidence	Lacking evidence
Tissue Swelling		
Ankle edema following acute sprain	Lacking evidence	Lacking evidence

Conclusions are drawn from all studies on specific use of ultrasound.

strong substantiated evidence for the use of continuous US to treat:

- The pain of myofascial pain syndrome and trigger points
- Pain and dysfunction associated with painful back conditions
- Pain and dysfunction of carpal tunnel syndrome
- Pain associated with arthritis

There is moderate substantiated evidence for the use of continuous US to treat:

- Dysfunction associated with nonspecific shoulder conditions
- Calcific tendinitis
- Bursitis

Table 4-20 shows the recommended continuous US protocols for each of the areas that show a substantiated effect from review of the evidence for each application. A consistency between the US protocols is noted. It is unclear whether this consistency is the result of a uniformity of the most effective approach to treating each of these differing conditions or if it reflects a uniformity of choice of protocols of the investigators and that a variety of other approaches might be valuable but have not been investigated.

A review of the evidence for the major suggested uses of pulsed US in clinical practice is shown in Table 4-21.

TABLE 4–20. Recommended Continuous Ultrasound Protocols for Each of the Conditions That Show a Substantiated Effect From Review of the Evidence for the Application of Ultrasound to Each Condition

Condition	Strength of Evidence	Recommended Intervention Protocol
Pain		
Myofascial pain, MTrPs—pain	Strong	Three times per week for 10 minutes each treatment, using 1 MHz at 1–2 W/cm^2 for a 4-week course of treatment
Back pain	Strong	Three times per week for 10 minutes each treatment, using 1 MHz at 1–2 W/cm^2 for a 4-week course of treatment
Back dysfunction	Strong	
Nonspecific shoulder conditions—dysfunction	Moderate	Three to five times per week for 10 minutes each treatment, using 1 MHz at 0.5–2 W/cm^2 for 3–4 weeks may be beneficial, particularly when accompanied by stretching and exercise
Inflammation		
Carpal tunnel syndrome—pain	Strong	Five times per week for 5–10 minutes with either 1 or 3 MHz at 0.5–1.5 W/cm^2 with treatments continuing for 2–4 weeks
Carpal tunnel syndrome—dysfunction	Strong	
Calcific tendinitis—pain or function	Moderate	Three times per week for 10 minutes with either 1 or 3 MHz at 1–2 W/cm^2 with treatments continuing for 4–8 weeks but may have to last longer if sufficient improvement is not seen
Bursitis—pain or function	Moderate	Three times per week for 5–10 minutes using 1 MHz at 1 W/cm^2 over a period of 3–4 weeks
Arthritis—pain	Strong	Three times per week for 5–10 minutes using 1 MHz at 1–2 W/cm^2 for 2–3 weeks

TABLE 4–21. Summary of Evidence for the Commonly Stated Uses of Pulsed Therapeutic Ultrasound

Commonly Stated Use	Strength of Evidence	Therapeutic Effectiveness
Pain		
Myofascial pain, MTrPs—pain	Lacking evidence	Lacking evidence
Myofascial pain, MTrPs—pressure-pain threshold	Lacking evidence	Lacking evidence
Myofascial pain, MTrPs—cervical ROM	Lacking evidence	Lacking evidence
Nonspecific shoulder conditions—pain	Lacking evidence	Lacking evidence
Nonspecific shoulder conditions—dysfunction	Lacking evidence	Lacking evidence
Inflammation		
Lateral epicondylitis	Moderate	Conflicting
Carpal tunnel syndrome	Lacking evidence	Lacking evidence
Calcific tendinitis	Lacking evidence	Lacking evidence
Arthritis	Lacking evidence	Lacking evidence
Soft Tissue Healing		
Dermal wounds	Lacking evidence	Lacking evidence
Tissue Swelling		
Ankle edema following acute sprain	Lacking evidence	Lacking evidence

Conclusions are drawn from all studies on specific use of ultrasound.

Studies have been done in several areas, but in most areas, there have been an insufficient number of studies to conclude that pulsed US has any effect. In the one area where there is sufficient evidence, the evidence is conflicting. As can be seen, there is no compelling evidence for any use of pulsed US.

The most interesting finding from this review of the literature is that US seems to be most effective in treating painful conditions by reducing the pain. Improvements in function are not as robust and are likely the result of the reduced pain rather than any other factor. Additionally, there seems to be no effectiveness of pulsed US in the treatment of inflammatory conditions, which is a major application of US. It has always been stated that the cellular effects of cavitation and microstreaming reduce the inflammatory processes. However, evidence does not seem to support this. Most likely any effect on the symptoms of inflammatory conditions is related to pain relief, which is a heating effect and, therefore, more likely produced by continuous US. Of course, any heating of an area with an active inflammatory process raises concerns and may suggest that relief of pain and subsequent improvement of function is best managed with other interventions.

CONTRAINDICATIONS AND PRECAUTIONS

In a special issue of *Physiotherapy Canada,* Houghton et al[67] reviewed the evidence basis of the contraindications and precautions commonly listed for US.

Evidence is moderate to strong for the contraindication of the use of continuous US in the following areas and conditions:

- Pregnancy—over the abdomen, low back
- Active bone growth at the epiphysis
- Cancer—over a known or suspected area of malignancy
- Tuberculosis infection—infected tissue, particularly that under tension (swelling/abscesses)
- Hemorrhagic conditions—over an area of active bleeding; can be used in area of hematoma and in persons with bleeding disorders (hemophilia)
- Impaired circulation—recommendation to use pulsed US, particularly for wounds in these areas (but note the lack of evidence for pulsed US for this use)

In the following areas and conditions, there is poor or no evidence for the contraindication of continuous US, but the consensus opinion is to still consider these as contraindications:

- Myositis ossificans—over an area with this condition
- Deep vein thrombosis or thrombophlebitis—over an area with this condition
- Acute injury—signs or suspicion of inflammation
- Recently irradiated tissue—skin areas where radiation therapy has been done
- Impaired sensation—nerve damage, so no sensory feedback about excessive warmth or burning
- Impaired cognition or communication—no feedback about excessive warmth or burning
- Skin disease—including damaged and at-risk skin
- Implanted cardiac pacemaker or other implanted electronics—unsure if damage to device could occur because of either heating or vibration
- Reproductive organs—particularly the thermally sensitive testes
- Eyes
- Anterior neck—particularly over the carotid arteries, carotid sinus, and vagus and phrenic nerves

The following areas and conditions are considered as precautions to the use of continuous US:

- Plastic or cemented implants—moderate evidence for damage to these
- Spinal cord and superficial or regenerating nerves—poor evidence for this but still considered a precaution

Applications over metal implants or over the chest, heart, or head are often listed as contraindications or precautions to the use of continuous US. However, it is consensus opinion that these areas should not be judged contraindications for the use of continuous US.

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The following areas and conditions are considered as precautions to the use of pulsed US:

- Active bone growth at the epiphysis
- Areas of infection—infected tissue, particularly that under tension (swelling/abscess)
- Acute injury—signs or assumption of inflammation
- Impaired sensation—nerve damage, so no sensory feedback about excessive warmth or burning
- Impaired cognition or communication—no feedback about excessive warmth or burning
- Impaired circulation
- Skin disease, including damaged and at-risk skin
- Plastic or cemented implants—moderate evidence for damage to these
- Spinal cord and superficial or regenerating nerves—poor evidence for this but still considered a precaution

As with continuous US, it is commonly seen that application over metal implants or the chest, heart, and head are listed as contraindications or precautions to the use of pulsed US. However, it is consensus opinion that these areas should not be judged contraindications to the use of pulsed US.

OTHER USES OF THERAPEUTIC ULTRASOUND

There are three other clinical uses of therapeutic US: phonophoresis, low-intensity pulsed US, and noncontact low-frequency US.

Phonophoresis

Phonophoresis is the application of US to enhance the absorption of topical agents through the skin. This is generally done by applying US directly to the topical agent or adding the topical agent to the US gel. The effects of US to enhance the entry through skin of a topical agent has been attributed to both physical "pushing" of the agent through the skin and to an increase in the permeability of the dermal layer.[9] However, neither mechanism is likely. US waves lack the coherence to produce substantial "pushing" of molecules, and evidence for increased cellular permeability is either lacking in in vivo studies[38,42] or occurs with US frequencies greater than the standard 3 MHz used in clinical practice.[68] The most likely cause is the thermal effect produced by US.[69]

Application of phonophoresis is done with either pulsed or continuous US. There is no clear indication which method should be preferred. Strapp et al[70] reported a greater disposition to hydrocortisone into tissues with the use of continuous US. However, Ebrahimi et al[71] reported greater effects of lidocaine on skin sensation when applied with pulsed versus continuous US. Continuous US raises the risk of heating the tissue, which may be contraindicated in an acutely inflamed tissue. However, Byl[69] suggests that thermal effects produced by US may be the mechanism of enhanced diffusion of topical agents into the skin.

Most commonly, hydrocortisone and analgesics (such as salicylates and lidocaine) are administered with phonophoresis. Results of studies involving the phonophoretic application of hydrocortisone are conflicting, with some studies showing improvement in various conditions[72–74] while others show little or no effect.[75–79] Likewise, there are conflicting results for phonophoretically applied salicylates and lidocaine, with some studies showing lessening of pain or increased skin analgesia[71,80–82] and others showing no change in pain or skin sensitivity.[83–85] So from the evidence, phonophoresis cannot be recommended when many other approaches are available to treat pain and inflammatory conditions.

Low-Intensity Pulsed Ultrasound

Low-intensity pulsed ultrasound (LIPUS) is the delivery of pulsed, medium-frequency (1.5 MHz) US at a much

lower intensity (0.03 W/cm^2) than conventionally delivered US. It is delivered via a stationary applicator that contacts the skin through the regular ultrasonic gel. Its primary clinical use is in bone fractures that show difficulty in healing[86–89] and in speeding the repair at tendon-bone junctions.[90] LIPUS produces fracture healing by increasing bone angiogenesis and stimulating osteogenesis by the stimulation of bone cell differentiation (via stimulation of multipotent stem cells), enhanced osteoblastic growth, and calcified matrix production.[90] These effects are probably the result of both direct mechanical and piezoelectric stimulation of bone cells.

Application is standardized with the delivery of 0.03 W/cm^2 pulsed with a 20% duty cycle for 20 minutes over the bone fracture site. This does not have to be set but is programmed into the applicator. The treatment is applied daily until bone healing has occurred.

Low-Intensity Therapeutic Ultrasound (LITUS)

Low-intensity therapeutic ultrasound (LITUS) has been applied to bone healing under the term *low-intensity pulsed ultrasound* (LIPUS) for several years. Although there is some evidence to support its use, the general consensus is that there is insufficient evidence to support its routine use in fracture healing. Recently, LITUS has been applied to soft tissue healing and is being promoted as sustained acoustic medicine (SAM). This technique uses a very-low-intensity pulsed US (0.132 W/cm^2, 20% pulsed) administered over a 1- to 4-hour period using one or two stationary transducers (called *SAM ultrasound applicators*), depending upon the size of the area to be treated, for at least 4 days per week (Fig. 4-11).

It has been recommended for use in both acute and chronic tendinopathy for periods of 2 to 12 weeks, depending upon the diagnosis. The SAM US applicator is applied using a coupling bandage. Despite the applicator having a relatively high BNR (reported as less than 5.1), there has been no evidence of tissue damage following a 4-hour treatment. Presently, there is little research using this device, but it has been suggested that LITUS may be effective in reducing pain associated with chronic myofascial conditions. Several studies are presently being conducted.

Fig 4 ■ 11 Sustained acoustic medicine (SAM) is a new technology used to provide controlled-release, long-duration US treatment for up to 4 hours daily. *(Courtesy of ZetrOZ, Inc., Trumball, CT.)*

Noncontact Low-Frequency Ultrasound

Noncontact low-frequency ultrasound (NCLFUS) involves the aerosolization of saline water with the use of low-frequency US to cleanse and debride devitalized tissue covering and surrounding the wound. This approach is commonly known by its commercial name of MIST Therapy. Evidence suggests that the NCLFUS stimulates wound healing by atraumatic selective tissue debridement, wound stimulatory effects, and antibacterial activity.[91] This approach is done by applying a continuous 40 kHz, 0.5 W/cm^2 US through the transducer into the applicator unit, which then dispenses a fine mist of sterilized saline or water to the wound. The duration of treatment varies from 3 to 20 minutes, depending upon wound size, and is set by entering the wound size into the applicator console.[92]

Documentation Tips

Appropriate documentation of US application should include the following:

- US parameters
  - Intensity (W/cm^2), frequency (typically 1 or 3 or 3.3 MHz), duty cycle (typically 20%, 50%, or 100%)
- Treatment duration (in minutes)
- Sound head size
- Treatment area
- Coupling agent (e.g., ultrasonic gel, gel pad, underwater)
- Patient position
- Patient response

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CHAPTER 5

HYDROTHERAPY

The Use of Water as a Therapeutic Agent

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Hydrotherapy is the use of water to facilitate healing and exercise and to enable patients to achieve therapy goals. The properties of water provide treatment options that might otherwise be difficult or impossible to provide with land-based interventions. For example, in a pool, a patient can be placed in non-weight-bearing positions—supine, prone, or sitting—with the use of buoyant devices. Movement and exercise of the upper and lower extremities can be facilitated in these positions by the effects of buoyancy. Water in the pool can serve as resistance to exercise to facilitate muscle strengthening.

Historically, hydrotherapy was delivered in metal tubs of water agitated by an attached electric motor (commonly

known as *whirlpools*) or by immersion in larger pools of water (e.g., swimming pools). Today the use of whirlpools, particularly for treating wounds, has decreased because of infection concerns and the resources and expense required for their use. Newer techniques that deliver a pressurized stream of water to wounds and incorporate suction, such as pulsed lavage with suction (PLWS) devices, have mostly replaced whirlpools for wound care. Pool therapy, or aquatic therapy, has become increasingly popular in rehabilitation programs. Other methods of hydrotherapy, such as contrast baths, have limited support in the research literature for effectiveness.

PHYSICAL PROPERTIES OF WATER

An understanding of water's static and dynamic properties as they apply to immersion and exercise is important to appropriately and effectively use hydrotherapy for therapeutic interventions. This section will discuss the physical properties of water, including buoyancy, drag forces viscosity, hydrostatic pressure, hydrodynamics, thermodynamics, physiological effects, and mechanics.

Buoyancy

One of the most important properties of water is buoyancy. Archimedes' principle states that "the buoyant force on a body immersed in a fluid is equal to the weight of the fluid displaced by that object."[1] A body or body part immersed in water will experience this buoyant force, which reduces the force of gravity on the body and, thus, decreases weight-bearing on the lower part of the body. A person immersed in water up to the neck will have about 10% of the body weight bearing on the lower body; immersion up to the xiphoid process will bear about 33% body weight on the lower body; and immersion up to the anterior superior iliac spines will bear about 50% body weight on the lower extremities.[2] Exercise of the extremities can be assisted by buoyancy. A person standing in water up to the neck can raise an extremity with the assistance of buoyancy. Buoyancy can also resist movement, such as an extremity moving downward against the force of buoyancy (Fig. 5-1). Resistance exercises for strengthening can be performed against the force of buoyancy. For example, a patient recovering from surgical repair of the rotator cuff muscles of the shoulder can use the buoyant force of water to assist in raising the extremity from the side of the body to 90° of flexion or abduction while standing in neck-deep water. When returning the extremity to the side, buoyancy can be used as a resistance to this movement.

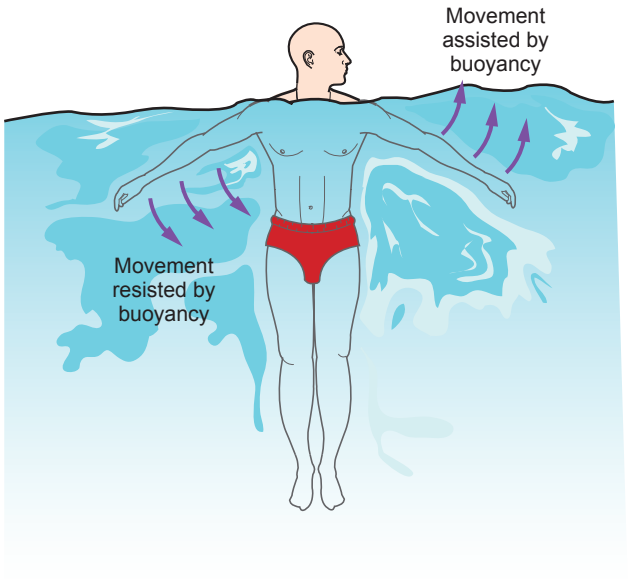

Fig 5 ■ 1 Exercising with buoyancy assists exercise; exercising against buoyancy resists exercise.

When a body is partially immersed in water, it is subjected to two opposing forces: buoyancy and gravity. The reference point for analyzing the effects of gravity on the body is the center of gravity, whereas the reference point for analyzing the effects of buoyancy of the part of the body that is immersed in water is the center of buoyancy. The body will remain balanced, and no movement will take place if these forces are equal and opposite to each other. When these forces are unequal and unaligned, the body or body part will tend to move or rotate. This motion may cause a person standing or sitting in a pool of water to become unstable and potentially unbalanced. Therapists must be aware of the combined effects of gravity and buoyancy of a person immersed in water and use appropriate guarding and/or flotation devices to prevent loss of balance.

The buoyancy of an object in water depends on its density (mass per unit volume). Objects that are denser than water will have less buoyancy and will tend to sink. Objects that are less dense than water will experience more buoyancy and will tend to float. The densities of various substances can be described by a number value known as *specific gravity*. The specific gravity of an object is defined as the ratio of the object's density to the

water's density at 39.2°F (4°C). The specific gravity value assigned to water is 1, whereas the specific gravity of the body with air in the lungs is 0.96[3] (Table 5-1). Objects with a specific gravity less than 1 will displace a proportional amount of water and will float. The body will displace about 90% of water when immersed and about 2.6% will float. This enables a person lying supine in water to keep the face out of the water while most of the body is immersed slightly below the surface (Fig. 5-2).

Buoyancy of the body will be affected by the amount of air in the lungs. Fully inflated lungs will increase buoyancy, and deflated lungs will decrease buoyancy. Buoyancy is also dependent upon body composition. Obese individuals will have increased buoyancy because fat tissue has a lower specific gravity than most other tissues. Females have greater buoyancy than males because females have a higher body fat percentage. This provides an advantage for females for swimming, resulting in an approximate 30% lower energy expenditure than males.[4]

TABLE 5–1. Specific-Gravity Values

Substance	Specific Gravity
Water	1
Ice	0.917
Air	1.21×10^{-3}
Average human body with air in lungs	0.96
Average human body without air in lungs	1.1
Subcutaneous fat	0.85
Cortical bone (femur)	1.85
Cancellous bone (vertebral body)	0.47

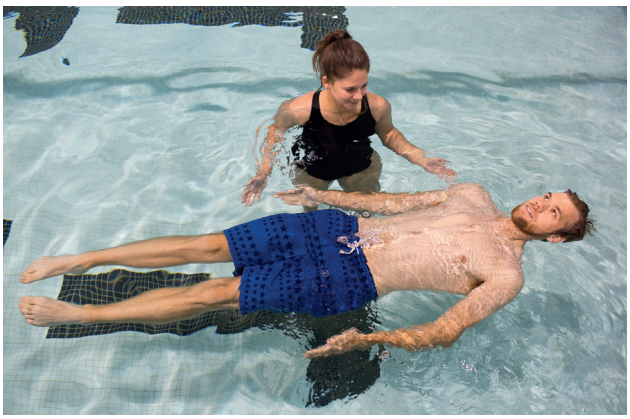

Fig 5 ■ 2 Floating in a pool demonstrates the effects of buoyancy.

Viscosity, Drag Forces, and Hydrostatic Pressure

In addition to buoyancy, three other mechanical properties of water can have an effect on the body or body part when immersed in water. Viscosity is the internal friction present in liquids secondary to the cohesive forces between the molecules. When an arm or a leg is moved through water, the water's viscosity will resist this movement. The faster the limb is moved, the greater the resistance encountered secondary to water viscosity. If a patient experiences pain during exercise and stops pushing against the water, the force of the water against the body part drops instantly.[5] This can serve as a protective effect during exercise in water.

Drag force resists the movement of an object or body part in water. This force is parallel to the direction of movement but in the opposite direction. Doubling the velocity of movement of an object or body part in water will quadruple the magnitude of the drag force. If the object or body part moves downward in the water, then the resistance encountered is the sum of both the drag force and the buoyancy.[4] The amount of the object or body part's surface that is aligned perpendicular to the water during movement against the water will also affect the amount of drag force encountered. A paddle moved flat against the water will encounter more drag force than a paddle turned sideways.[4]

Hydrostatic pressure is the force that water exerts on the immersed body or body part. This force impacts the body equally from all directions at a given depth of immersion. The amount of hydrostatic pressure will vary, depending on the depth of immersion of the body part. A person standing in a pool of water up to the neck will have a greater amount of hydrostatic pressure against the feet than against the trunk or shoulders (Fig. 5-3). Therefore, when exercising near the water's surface, the patient will encounter less resistance than when exercising at a greater depth. This increased pressure may encourage venous return in a proximal direction from the lower extremities. Jamison[6] advocated this therapeutic effect of aquatic therapy, combined with the effect of viscosity, for increasing lymph flow and reducing edema in patients with lymphedema. However, the dependent position of the body part may cancel this effect. McCulloch and Boyd[7] found that the combination of heat and the dependent position of an extremity in a whirlpool have the potential to encourage lower extremity swelling.

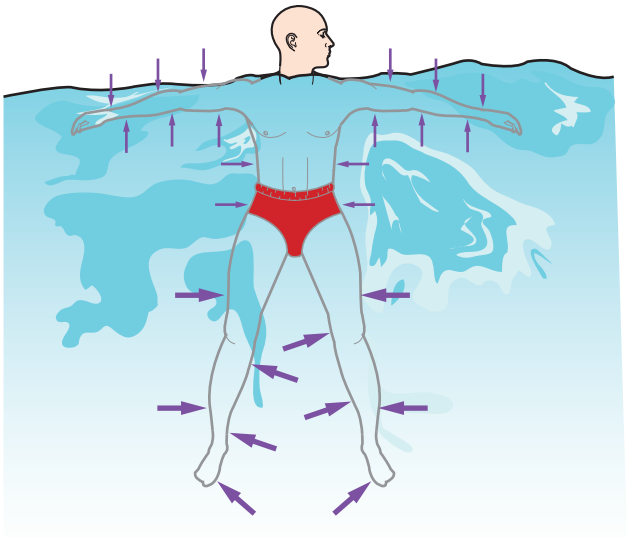

Fig 5 ■ 3 The amount of hydrostatic pressure against the body depends upon the depth of submersion of the body part. Smaller arrows represent lower hydrostatic pressure; larger arrows represent greater pressure.

Key Point! Hydrostatic pressure is present against any body part immersed in water. The deeper the body part is immersed, the greater will be the pressure.

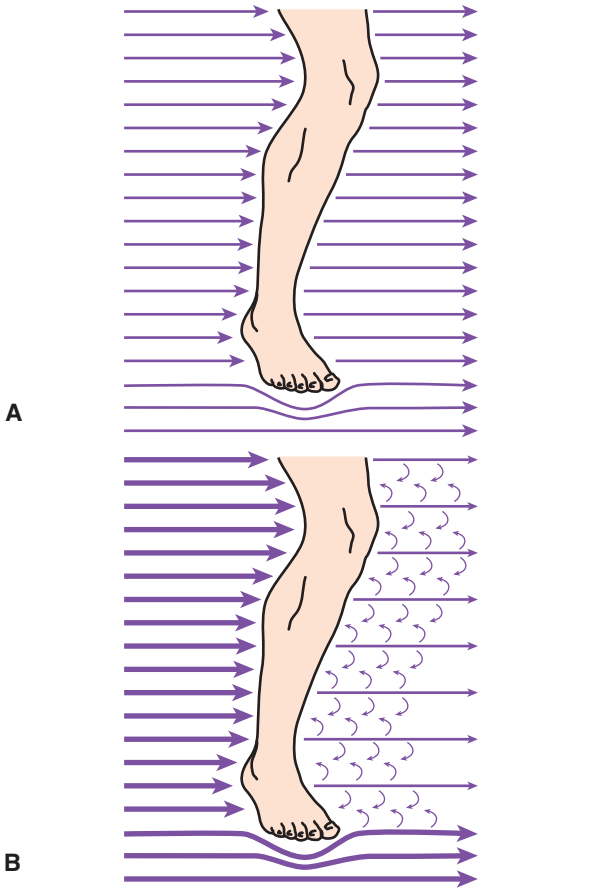

Fig 5 ■ 4 (A) Laminar (streamline) flow of water around the lower extremity and (B) turbulent flow of water around the lower extremity.

Hydrodynamics

Water in motion has physical properties known as *hydrodynamics.* The study of hydrodynamics includes two types of fluid dynamics: streamline (or laminar flow) and turbulent flow. Streamline flow occurs when each particle of the fluid follows a smooth path without crossover of paths (Fig. 5-4). Turbulent flow is the flow of fluids in erratic, small, whirlpool-like circles called *eddy currents* or *eddies.*[1] Viscosity of water is much greater during turbulent water flow, resulting in more resistance to movement. Movement of a body part in water at rest will encounter minimal turbulence, although the movement itself will create some turbulence. Movement against turbulent water will encounter more resistance, as experienced while moving a body part in a whirlpool when the water is agitated.

Thermodynamics: Heat Transfer

The physiological effects of hydrotherapy primarily depend upon the temperature of the water, how much of the body is immersed in the water, and whether the person or body part in the water is at rest or moving. If the water's temperature is greater than the temperature of the body or body part in the water, then the temperature of the body or body part will increase. If the water's temperature is cooler than the temperature of the body or body part, then the temperature of the body or body part will decrease. The rate of heat loss in body tissues to water at moderate temperatures is 25 times greater than the rate of heat loss to air at the same temperature.[4] When the body or body part and the water are not moving, some heat will be transferred by conduction. However, the layer of water immediately adjacent to the skin may act as a "sealer," which tends to inhibit conductive heat transfer. Agitation of the water in a whirlpool will limit this sealer effect. Heat will be transferred primarily by convection when the patient is moving in the water or when the water moves across the skin's surface.

The body can also lose heat by radiation and evaporation, depending on the amount of the body that is

immersed and the temperature and humidity of the environment. Radiation is the exchange of electromagnetic energy between the warmer surface of the body and the cooler surrounding air. Evaporation is the loss of body fluids to the environment by sweating. These processes do not occur when the body surface is immersed in water. Care must be taken to expose a sufficient amount of body surface to the environment outside the hydrotherapy tank or pool to allow for radiational cooling and evaporation. The ability of the body to lose heat to help maintain a constant core temperature is limited when the hydrotherapy environment is too warm or too humid. It is best to maintain the hydrotherapy environment at an air temperature between 65°F and 80°F (18.3°C and 26.7°C) and humidity between 50% and 65%.[8,9]

Another property of water related to heat transfer is specific heat. This is a measure of the amount of energy (heat) stored in a material and the amount of energy required to heat the material. The amount of heat required to change the temperature of a given material is proportional to the mass of the material and to the temperature change. This can be expressed by the following equation:

$$Q = mc\,\Delta T$$

where Q is the amount of heat, m is the mass of the material, c is the specific heat of the material, and ΔT is the change in temperature.[1] Water has one of the highest specific heats of all substances (1 kcal/kg·C° at 15°C, read kilocalories per kilogram degree Celsius).[1] This is evident in the difference in heat transfer between water and paraffin. Paraffin is usually applied to the skin safely at a temperature between 113°F and 129.1°F (45°C and 54°C), whereas water at these temperatures would feel uncomfortably hot and can burn the skin.

PHYSIOLOGICAL EFFECTS OF WATER

Hemodynamics

The hemodynamic effects of hydrotherapy include local changes in circulation and systemic effects on cardiac function. The water's temperature will either increase localized circulation secondary to vasodilation or decrease localized circulation secondary to vasoconstriction. Generally, warmwater temperatures of about 95.9°F (35.5°C) will result in vasodilation. Cold water temperatures below 80.6°F (27°C) will cause vasoconstriction. Table 5-2 summarizes the physiological effects of commonly used water temperatures for hydrotherapy.

Immersion of the entire body except the head or immersion of only the face will result in a set of cardiovascular effects known as the *dive reflex*.[10] These effects include bradycardia, peripheral vasoconstriction, and preferential shunting of blood to vital organs. However, these effects are dependent on the water's temperature. Immersion in warm or hot water increases heart rate. Immersion in water at body temperature has a neutral effect on heart rate.[5]

Immersion in water may affect blood pressure. Enhanced venous return secondary to a rise in central venous pressure with immersion increases cardiac volume, which causes an increase in right atrial pressure and increased cardiac output (assuming normal cardiac pump mechanics). However, the effect on blood pressure may be blunted secondary to the effects of the dive reflex, resulting in bradycardia.

TABLE 5–2. Water Temperature Classifications and Expected Physiological Effects for Hydrotherapy

Water Temperature Classifications	
Very cold	33°F to 55°F (1°C to 13°C)
Cold	56°F to 65°F (14°C to 18°C)
Cool	66°F to 80°F (19°C to 26°C)
Tepid	81°F to 92°F (27°C to 32°C)
Neutral	93°F to 96°F (33°C to 35°C)
Warm	97°F to 99°F (36°C to 37°C)
Hot	100°F to 104°F (38°C to 40°C)
Very hot	105°F to 110°F (41°C to 43°C)
Expected Physiological Effects	
Very cold, cold, cool	Vasoconstriction, analgesia, possible anesthesia
Tepid and neutral	Likely no loss of body heat or change in core temperature or limb size (best temperature range for pool exercise)
Warm and hot	Vasodilation, analgesia, relaxation
Very hot	Same as warm and hot temperatures but may cause rapid fatigue and overheating

The effects on the cardiovascular system (sudden vasoconstriction, decrease in heart rate, and increase in heart volume from increased venous return) may not be tolerated by some, particularly those with cardiovascular deficits. To limit the effects on the cardiovascular system, it is best to enter the water slowly, wetting the face and hands first, and avoid full-body immersions in cold water.[11] When exercising in water, monitoring heart rate may not be an accurate reflection of cardiovascular stress. Therefore, monitoring perceived exertion is preferred over monitoring of heart rate.

Key Point! When exercising in water, monitoring heart rate may not be an accurate reflection of cardiovascular stress. Therefore, monitoring perceived exertion is preferred rather than monitoring heart rate.

Effects of Water on the Respiratory System

Immersing the body in water may affect the ability to breathe. Hydrostatic pressure against the chest will tend to inhibit lung expansion. Also, increased circulation to the center of the body during immersion will increase circulation in the chest cavity, further inhibiting lung expansion. Maximal oxygen uptake is lower during most forms of water exercise than during exercise on land.[10] These effects result in an increase in the total work of breathing. At a tidal volume of 1 liter, the total work of breathing increases 60% during immersion of the body up to the neck.[11]

Effects of Water on Renal Function

Water immersion can affect renal function, resulting in increased urine output (diuresis), increased sodium excretion, and increased potassium excretion. These effects can be enhanced when the individual is immersed in cold water. Patients should empty their bladders before entering a therapeutic pool.

Effects of Water on the Neurological System

The effects of water immersion on the neurological system are primarily temperature dependent. (The effects of cold and heat on the neurological system are discussed in Chapters 2 and 3.) Warm water tends to be relaxing, whereas cold water tends to be invigorating or stimulating. Whether these effects are secondary to direct effects on the nervous system or are examples of psychological responses is debatable and requires further study.

Effects of Water on the Muscular System

Immersion of muscles in water has been shown to significantly increase oxygen delivery to the muscles and increase the removal of metabolic waste products. This effect is believed to be secondary to reflex changes in blood vessel tone and the compressive effect of hydrostatic pressure.[11] Other effects on the muscular system, such as increased muscular blood flow caused by immersing muscle in warm water, are probably secondary to the temperature of the water. Clinical use of hydrotherapy for relaxation of muscle spasm may in part be a result of increased blood flow caused by immersion in warm water. Agitation of the water, as in a whirlpool bath, may also help relax muscle spasm by breaking the "pain/spasm" cycle by increasing sensory stimulation of the water against the skin overlying the spastic muscle.

In addition, muscle strength may be increased with exercise in water, especially when exercising against turbulence. There is no evidence that muscles at rest will be strengthened by simply placing the limb in still or agitated water; however, the relaxing, stimulating, or analgesic effects of a whirlpool may be helpful preparation for an exercise program. For example, a patient with low back pain may benefit from whirlpool agitation against the back before starting an exercise program on land or in a pool.

MECHANICAL EFFECTS OF WATER

The mechanical effects of water are primarily caused by agitation of water in a whirlpool or application of a high-force stream of water to the body. The force of the water can help debride loose necrotic tissue in a wound and cleanse it of dirt and other contaminants. However, the use of whirlpools for wound debridement is no longer recommended because of the softening effect of water on tissues, which can damage new granulation tissue and cause maceration of intact skin.[12] The mechanical effects

of a high-force stream of water for debridement of wounds can be applied via PLWS devices, which are discussed later in this chapter.

AQUATIC (POOL) THERAPY

Aquatic therapy has wide therapeutic applications for pediatric, geriatric, cardiopulmonary, neurological, and orthopedic patients.[5,13–15] Aquatic therapy can be used to promote relaxation, improve circulation, restore mobility, strengthen muscles, provide gait training with less stress on weight-bearing joints, stimulate the vestibular system, facilitate sleep, increase one's capacity for stress, and improve psychological and emotional outlook.[13,14,16] The temperature and buoyancy of water combined with decreased body weight and joint compression and the psychological effects of participating in an enjoyable activity can reduce pain and muscle spasms and increase range of motion (ROM).[5] A recent systematic review on the effectiveness of therapeutic pools in the management of rheumatoid arthritis concluded that there is a short-term reduction in pain and improvement in the health status of patients compared to no or other interventions.[15] However, the long-term benefits require further studies.

Another systematic review with meta-analysis demonstrated improved function and no increased risk of wound-related adverse events in adults after orthopedic surgery. Early aquatic therapy was as effective as land-based therapy in improving function in adults in the early postoperative period without increasing risk of wound-related adverse effects.[17] A recent systematic review with meta-analysis examined the effects of therapeutic aquatic exercises on symptoms and functions in individuals with lower limb osteoarthritis. The meta-analysis demonstrated a significant effect on pain, self-reported function, physical functioning, stiffness, and quality of life. This review concluded that therapeutic aquatic exercise is effective in managing symptoms associated with lower limb osteoarthritis.[18]

Hydrotherapy is often combined with movement (exercise) for therapeutic effects. Slow rhythmical movements combined with rotary-type motions may decrease spasticity of muscles. For example, a 45-year-old male with cerebral palsy attended aquatic therapy for gait dysfunction secondary to an adverse reaction to anti-anxiety medication. This patient performed slow rhythmic and rotary movements in the pool. He had improvement in the tone in his lower extremities, resulting in less spasticity and a more normal gait pattern. Aquatic therapy was chosen for him to increase endurance, improve static and dynamic posture, and increase independence in ambulation with a walker.[19] A recent quasi-experimental study demonstrated a significant and large effect on gross motor function scores and significantly higher physical activity enjoyment scores in pediatric patients with cerebral palsy compared to a control group. This study concluded that pediatric aquatic therapy can be an effective therapy for children with cerebral palsy.[20]

CASE STUDY 5-1 Selecting Pool Therapy for a Patient With Cerebral Palsy

A 45-year-old male with cerebral palsy is referred for balance and gait training secondary to severe balance and endurance problems caused by an adverse reaction to an anti-anxiety medication. Before this, the patient ambulated independently, played sports, and was active in his community. Upon testing, the patient demonstrated decreased balance and endurance and inability to ambulate without a walker.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of pool therapy?

ANSWER: Yes, pool therapy can be used to improve endurance, balance, and gait.

2. Is the patient appropriate for pool therapy? Do any of the general precautions or contraindications to pool therapy apply to the patient, or are there any specific considerations regarding the application of pool therapy to this patient?

ANSWER: None of the precautions or contraindications apply to this patient. He is able to enter the pool using a ramp and handrails with the assistance of the physical therapist. Pool therapy is an appropriate intervention for this patient.

3. What are the specific goals to be achieved with the use of pool therapy?

ANSWER: Improved endurance and balance and decreased use of assistive device for ambulation.

Continued

CASE STUDY 5-1 Selecting Pool Therapy for a Patient With Cerebral Palsy—cont'd

4. What specific aspects of pool therapy would be appropriate for this patient?

ANSWER: The force of buoyancy can be used to resist movement and improve endurance. The slow rhythmical movements combined with rotary-type motions promote improved tone in the lower extremities and a more normal gait. Seated bicycling and ambulating back and forth and sideways in different depths of water will promote improved endurance and balance and promote a more normal gait.

5. What are the proper application procedures for pool therapy?

ANSWER:

Instruct the patient: Inform the patient of the purpose and procedure, including the schedule of pool therapy sessions and the need for appropriate swimwear. Assess the patient's previous swimming experiences and his expectations for the sessions.

Presession: Determine the techniques to be used during the session (i.e., seated activities versus standing activities, depth of water to assist or challenge balance, types and speeds of movements to achieve goals of session).

Pool session: Monitor vital signs to ensure patient safety. Use safest method for entry into pool. Proceed with activities chosen for session and modify as needed.

Postsession: Assess balance and gait as patient exits pool, including trying a different method of exit from that used for entry. Assess balance and gait on land and monitor vital signs. Determine progression of next pool therapy session.

Hydrotherapy can facilitate exercises designed to strengthen muscles. Repetitive exercises in water and swimming strokes can improve coordination, and the warmth of the water can increase blood supply to the musculoskeletal tissues.[4] Kicking exercises in water can stimulate venous return, and the hydrostatic pressure provided by deeper water can help move fluids upward—all of which can assist with improving circulation insufficiency.[4]

Aquatic therapy can be included as part of a postoperative rehabilitation program (Case Study 5-2). The use of water can provide buoyancy to assist, support, and resist exercises. Turbulence of the water and the speed of movement can facilitate exercises used to increase ROM and strength of the involved extremity and can help to maintain ROM and strength of noninvolved extremities.[21,22]

CASE STUDY 5-2 Selecting Pool Therapy for a Patient Following Shoulder Surgery

A 23-year-old male is referred for increased ROM and strengthening of the right shoulder secondary to rotator cuff and labrum repair. Upon testing, the patient was limited to 90° of flexion and abduction of the shoulder and weakness of the rotator cuff muscles and the anterior and middle deltoid.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of pool therapy?

ANSWER: Yes, pool therapy can be used to improve ROM and increase the strength of the rotator cuff and anterior and middle deltoid muscles.

2. Is the patient appropriate for pool therapy? Do any of the general precautions or contraindications to pool therapy apply to the patient or are there any specific considerations regarding the application of pool therapy to this patient?

ANSWER: None of the precautions or contraindications apply to this patient. The surgical site is healed and there is no opening or drainage that would be of concern. Pool therapy is an appropriate intervention for this patient.

3. What are the specific goals to be achieved with the use of pool therapy?

ANSWER: Increased ROM and strength of the rotator cuff and anterior and middle deltoid muscles.

4. What specific aspects of pool therapy would be appropriate for this patient?

ANSWER: The force of buoyancy can be used to assist the motions of the involved muscles and can

CASE STUDY 5-2 Selecting Pool Therapy for a Patient Following Shoulder Surgery—cont'd

be used to resist movement and improve strength of these muscles while maintaining ROM and strength of the elbow and wrist joints of the involved side.

5. What are the proper application procedures for pool therapy?

ANSWER:

Instruct the patient: Inform the patient of the purpose and procedure, including the schedule of pool therapy sessions and the need for appropriate swimwear. Assess the patient's previous swimming experiences and his expectations for the sessions.

Presession: Determine the techniques to be used during the session (i.e., seated, standing, prone, or supine activities; depth of water to assist motions or challenge muscle; types and speeds of movements to achieve goals of session).

Pool session: Monitor vital signs to ensure patient safety. Use safest method for entry into pool. Proceed with activities chosen for session and modify as needed.

Postsession: Assess ROM and strength of involved muscles. Determine progression of next pool therapy session.

Hydrotherapy can facilitate exercise programs for patients who cannot tolerate exercise on land. Patients with chronic low back pain have benefited from aquatic exercise programs (Case Study 5-3).[16,23] Aquatic exercise programs also can be beneficial for patients following total joint replacements. A 55-year-old female with restricted weight-bearing after total hip arthroplasty secondary to osteoarthritic changes will experience decreased body weight and joint compression during exercises in a pool, resulting in improved ROM and increased strength of the involved musculature.[24,25]

CASE STUDY 5-3 Selecting Pool Therapy for a Patient With Low Back Pain

A 64-year-old female is referred for strengthening of the back musculature secondary to low back pain and inability to tolerate land-based exercises. Upon testing, the patient was able to walk limited distances and was unable to move the trunk freely during any activities.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of pool therapy?

ANSWER: Yes, pool therapy can be used to decrease pain, improve ROM, and increase the strength of the trunk musculature.

2. Is the patient appropriate for pool therapy? Do any of the general precautions or contraindications to pool therapy apply to the patient, or are there any specific considerations regarding the application of pool therapy to this patient?

ANSWER: None of the precautions or contraindications apply to this patient. Pool therapy is an appropriate intervention for this patient.

3. What are the specific goals to be achieved with the use of pool therapy?

ANSWER: Decreased pain, increased ROM, increased strength of the trunk musculature, and increased endurance.

4. What specific aspects of pool therapy would be appropriate for this patient?

ANSWER: The buoyancy can be used to support the body while the muscles can be worked. The buoyancy can assist the motions of the involved muscles and can be used to resist movement and increase strength of these muscles while improving endurance.

5. What are the proper application procedures for pool therapy?

ANSWER:

Instruct the patient: Inform the patient of the purpose and procedure, including the schedule of pool therapy sessions and the need for appropriate swimwear. Assess the patient's previous swimming experiences and her expectations for the sessions.

Continued

CASE STUDY 5-3 Selecting Pool Therapy for a Patient With Low Back Pain—cont'd

Presession: Determine the techniques to be used during the session (i.e., seated, standing, prone, or supine activities; depth of water to assist motions or challenge muscle; types and speeds of movements to achieve goals of session).
Pool session: Monitor vital signs to ensure patient safety. Use safest method for entry into pool. Proceed with activities chosen for session and modify as needed.
Postsession: Assess ROM and strength of involved muscles and endurance for ambulation on dry land. Determine progression of next pool therapy session and eventual progression to land-based exercises.

Patients who have inadequate orofacial control can benefit from hydrotherapy by working on lip closure and bubble blowing, provided there are no problems with their swallowing mechanisms. In addition, holding one's breath in water, gradually increasing the time in the pool, and swimming can improve endurance and/or respiratory function for patients with pulmonary dysfunction.[26–29] Impairments and functional deficits, including inadequate sitting and standing balance and ambulatory dysfunction, can be addressed with pool therapy.[20,28] Patients with balance problems can benefit from aquatic therapy because hydrostatic pressure and buoyancy help to support the body. Balance in chest-deep water is easier, and practiced recovery from disturbed balance can facilitate head righting and trunk control.

Aquatic therapy can facilitate arousal because of the changed environment when entering a pool. Also splashing, kicking, and turbulence of water can improve arousal. Patients with hypersensitivity disorders can benefit from aquatic therapy by gradually immersing one extremity at a time into the pool along with performance of enjoyable activities in the pool to help decrease hypersensitivity. Aquatic therapy can be beneficial for patients with cognitive impairments, including those with perceptual-spatial problems such as neglect of distance or depth, poor eye-hand coordination, poor concept of midline, decreased sequencing abilities, and poor socialization skills.[30] Psychological impairments[31,32] such as depression, lack of confidence, and/or decreased motivation can be addressed with aquatic therapy. Athletes can benefit from aquatic therapy because water becomes an isophysiological environment that requires the same functional physiology used in competitive sports. Sport-specific movement patterns can be performed in water, and their intensity can be increased over time, depending on the athlete's functional improvements on land.[33,34]

Indications, Precautions, and Contraindications

Most patients will be able to participate in aquatic therapy. However, precautions should be considered before proceeding with pool therapy (Table 5-3). Patients who are fearful of water may experience increased symptoms, such as muscle guarding or improper technique, during exercises. Some patients may be leery of losing their balance or sinking to the bottom. In instances like these,

TABLE 5–3. Aquatic Therapy Precautions

Precaution	Explanation
Aspiration risk	Follow precautions if patient has a history of aspiration.
Catheters	Patients with indwelling catheters require proper clamping and fixation.
Cognitive impairments	Concerns with safety when entering and leaving pool. Must be monitored closely while in pool.
Tetraplegia or high paraplegia	Difficulty with thermoregulation. Monitor patient and temperature of water and air to prevent heat prostration and hypothermia.
Open wounds	Must be covered with waterproof dressing to prevent infection and maceration of periwound tissue.
Dry skin or skin rashes or rash	Time in pool may exacerbate dryness.

a gradual orientation to the water, including the use of flotation devices or starting slowly in shallow water to build confidence, may be needed.[33]

Patients with neurological involvement, such as those with multiple sclerosis, may fatigue quickly when exercising in water greater than 91.4°F (33°C).[35–37] Patients with controlled epilepsy may need to be monitored for medication compliance before treatment and responses during treatment. Anyone with problems affecting the cardiovascular system, such as high or low blood pressure or history of angina, heart disease, or compromised pump mechanics, will require close monitoring during treatment.[38–40] Meyer and Leblanc[41] reviewed selected publications on patients with left ventricular dysfunction or stable congestive heart failure and made the following suggestions for rehabilitation and secondary prevention:

1. Temporary abnormal hemodynamic responses may be elicited by immersion up to the neck.
2. Water therapy is absolutely contraindicated in patients with decompensated congestive heart failure.
3. Feeling good in water does not equate with left ventricular toleration of increased volume loading caused by immersion.
4. If patients with previous severe myocardial infarctions and/or congestive heart failure can sleep supine, they may be able to tolerate bathing in a half-sitting position, provided immersion does not exceed the xiphoid process.
5. Patients with Q-wave myocardial infarctions older than 6 weeks may exercise in a pool for orthopedic reasons if they do so in an upright position and immersion does exceed the xiphoid process.

An algorithm to support clinical decision-making for prescribing swimming for patients with left ventricular dysfunction and/or stable congestive heart failure can be found in the article by Meyer and Leblanc.[41]

Patients with open wounds may participate in pool therapy if the wound is covered and secured with a waterproof dressing. Those with catheters, colostomies, intravenous lines, or other open lines may participate in pool therapy with proper clamping and fixation. Similar precautions should be followed for patients who have G-tubes and suprapubic appliances. These patients should be observed for any adverse reactions to pool therapy. Patients with medically controlled seizure disorders or fear of water should be monitored for any adverse reactions to the treatment.

Several factors must be considered when deciding whether pool therapy is appropriate for a patient. Any situation that creates the potential for contaminating the water is a contraindication for aquatic therapy, such as open wounds without occlusive dressings, incontinence of bowel or bladder, skin infections, menstruation without internal protection, and patients with isolation precautions. A general rule to follow is patients should be free of bowel and bladder accidents for at least 5 days before pool therapy. Patients with a history of uncontrolled seizures during the last year, severe cardiac precautions, acute fever, upper respiratory infection, severe mental disorders, and severe pulmonary conditions (especially with vital capacity less than 1,500 milliliters) are considered poor candidates for aquatic therapy because these conditions may be exacerbated by pool therapy. Patients with halo vests are not candidates for pool therapy because the location of the halo pins in the skull is considered an open wound. See Table 5-3 for a list of precautions and Table 5-4 for a list of contraindications for aquatic (pool) therapy.

TABLE 5–4. Aquatic Therapy Contraindications

Contraindication	Explanation
Bowel or bladder incontinence	If this is uncontrolled, patient cannot enter pool because of danger of cross-contamination of other tissues and other people in the pool.
Tracheostomy	Contraindication because of respiratory concerns.
Infectious diseases	Contraindication for aquatic therapy because of the danger of spreading infection to others in the pool.
Cardiovascular problems	Contraindicated in patients with decompensated congestive heart failure. Monitor vital signs of patients with other cardiovascular problems, such as uncontrolled blood pressure.
Uncontrolled seizures	Contraindication because of danger of having a seizure while in the pool.
Respiratory compromise, vital capacity less than 1 L	Contraindication if immersion is likely to exacerbate symptoms.

Clinical Controversy

Patients with arthritis of the knees, hips, and spine are often prescribed aquatic therapy by their physicians. Exercise in a pool offers many benefits; however, do these benefits justify the cost of resources required for aquatic therapy? Can patients with arthritis benefit equally as well with therapeutic exercise programs in rehabilitation clinics ("land" therapy)? What is the optimal number of exercise sessions per week for aquatic therapy? And how many sessions of aquatic therapy are needed for improved function? When can patients be discharged from aquatic therapy programs to pool exercise programs that they can perform independently? Does the chronic nature of arthritis justify long-term therapy programs? These are some of the questions that practitioners must consider when initiating aquatic therapy programs for patients with arthritis.

Pools and Pool Area

Therapeutic pools vary in size and shape, depending on the patient populations served and space allocations at a given facility. Traditional swimming pools, specially designed therapeutic pools, and self-contained exercise units can be used for aquatic therapy. Traditional swimming pools are at least 100 feet long and 25 feet wide with a sloping bottom that begins at a depth of 3 to 4 feet and gradually increases to a depth of 8 to 10 feet. This type of pool can accommodate several groups of patients and the therapists who are conducting the therapy session (Fig. 5-5). Smaller self-contained exercise pools are designed for individual patient use (Fig. 5-6). These units do not allow for the therapist to be in the pool with the patient; therefore, they are not for patients with low functional levels who require assistance in the pool.

Access to the traditional pool is by ramp, stairs, ladders, or mechanical overhead lift. Some specially designed pools have floors with adjustable heights to permit easier access. Therapeutic pools have a built-in filtration and chlorination system. The room in which the pool is housed needs to be adequately ventilated to avoid condensation accumulating on walls, windows, and floors. A private area for changing clothes, showering, and cooling down should be available to the patient after therapeutic exercise sessions.

Access to the smaller self-contained exercise pools is either via a door and steps located on one side of the unit or from a stair or two inside the unit. These units usually have their own filtration system and may include built-in exercise stations, varying water depths, and integrated treadmills. Some pools produce adjustable currents that increase exercise resistance and simulate wave action.

Fig 5 ■5 Patient exercising in a therapeutic pool with therapist assistance. (*Courtesy of Louisiana State University Health Sciences Center-Shreveport.*)

Many types of equipment can be used in pool therapy sessions. These include floats, resistive paddles, paddleboards, swim fins, flotation vests, parallel bars, treadmills, handrails, weighted stools and chairs, and weighted walkers and other assistive devices. Examples of patients exercising with equipment can be seen in Figures 5-7, 5-8, and 5-9. The type of equipment used during a particular session is based on the patient's current functional level and the goals for the aquatic therapy session.

Pool Care and Safety Precautions

Regular care and cleaning of therapeutic pools is essential to avoid the buildup of *Pseudomonas aeruginosa,*[42–44] an infection that causes folliculitis. Organic contaminants in a pool reduce the effectiveness of chlorine when used as a bactericidal agent. Frequent use of pools increases the total organic carbon as well as ammonia and organic nitrogen found in the pool. Regular care of therapeutic pools should include at least weekly cleaning and twice-daily chlorine and pH level tests. Walking surfaces leading up to and around the pool should be slip-resistant and kept free of barriers.

Safety rules and regulations and emergency procedures should be established. Rules and regulations should be posted and observed by both patients and

Fig 5 ■ 6 (A) Therapeutic hydrotherapy units. (B) Therapeutic hydrotherapy unit with body weight support harness. *(Photos courtesy of Ferno-Washington, Inc.)*

staff. An obvious danger whenever a patient is immersed in water is drowning. Patients who are immersed in large whirlpools or therapeutic pools should be monitored continuously, especially if they have decreased mental function or drowsiness caused by medication. Children must always be closely supervised. Life preservers should be available for all therapeutic pools. At least one staff member who is CPR certified should be present at all times in case of an emergency.

Fig 5 ■ 7 Patient exercising using fins on feet for added resistance in a therapeutic pool.

Fig 5 ■ 8 Patient exercising upper extremities with therapist using paddles in a therapeutic pool.

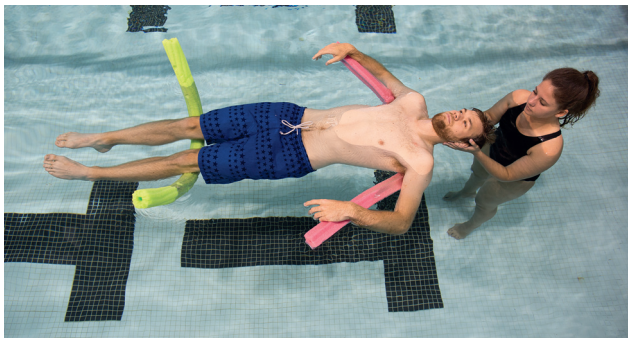

Fig 5 ■ 9 Therapist stretching cervical spine while patient uses foam noodles for buoyancy in a therapeutic pool.

Key Point! Patients who are immersed in large whirlpools or therapeutic pools should be monitored continuously, especially if they have decreased mental function or drowsiness caused by medication.

Clinical Applications of Therapeutic Pools

Practitioners should meet with patients before exercise in a therapeutic pool to discuss the treatment schedule and the procedures to be used. The patient's previous swimming experiences should be addressed, including any fear of water and expectations for the session. Other topics to be discussed with patients before entering a therapeutic pool are any bowel and bladder problems, use of any assistive or adaptive devices, type of clothing to be worn while in the pool and after leaving the pool, and medications.

Key Point! Practitioners should have a complete list of the patient's current medications and discuss with the patient any effect these medications may have while exercising in the pool. Medications that cause drowsiness or sedation, such as muscle relaxers, require close monitoring of the patient while in the pool.

The practitioner should be aware of the increased demand placed on the patient's cardiovascular and pulmonary systems. Core temperature can be increased by the water temperature, and muscular contraction places a greater demand on heat dissipation, the respiratory system, and the exposed integument. General considerations should include a maximum immersion time of 20 minutes for noncompromised cardiopulmonary patients and less time for elderly, hypertensive, and cardiopulmonary patients. It may be advisable to initiate treatment for 10 minutes and increase as tolerated.[45,46]

Vital signs need to be monitored to ensure patient safety. Water temperatures vary, and therapeutic pool temperatures cannot be quickly adjusted. In general, water temperatures between 96.2°F and 98.2°F (36°C and 37°C) are considered high and between 86°F and 94°F (30°C and 34.5°C) are considered low. High temperatures may be recommended for patients with disease processes, such as rheumatoid arthritis (except in the acute stage), although low temperatures may be recommended for patients with spasticity or for those whose immersion time may last 20 to 45 minutes. A patient's fatigue factor should also be considered when choosing therapeutic pool temperatures.[37,40]

A complete review of specific types of exercises and exercise programs is beyond the scope of this text. See Box 5-1 for a list of references for more information on aquatic exercise programs and Box 5-2 for websites related to aquatic therapy. See Table 5-5 for pertinent studies on aquatic therapy.

Box 5 ■ 1 References for Aquatic Therapy Exercise

Adams HP, Norton CO, Tilden HM. *Aquatic Exercise Toolbox*. Updated ed. Champaign, IL: Human Kinetics; 2006.

Brody LT, Geigle PR. *Aquatic Exercise for Rehabilitation and Training*. Champaign, IL: Human Kinetics; 2009.

Kisner C, Colby LA. *Therapeutic Exercise, Foundations and Techniques*. 6th ed. Philadelphia: F.A. Davis; 2012.

Lepore M, Gayle GW, Stevens FS. *Adapted Aquatics Programming—a Professional Guide.* 2nd ed. Champaign, IL: Human Kinetics; 2007.

Norton CO, Jamison LJ. *A Team Approach to the Aquatic Continuum of Care*. St. Louis, MO: Elsevier Health Sciences; 2000.

Rosenstein AA. *Water Exercises for Parkinson's: Maintaining Balance, Strength, Endurance, and Flexibility.* Rev ed. Enumclaw, WA: Idyll Arbor; 2008.

Sova R. *Essential Principles of Aquatic Therapy and Rehabilitation.* Port Washington, WI: DSL; 2003.

Vargas LG. *Aquatic Therapy: Interventions and Applications*. Enumclaw, WA: Idyll Arbor; 2004.

Box 5 ■ 2 Websites Related to Aquatic Therapy

www.ncpad.org/
This website is maintained by the National Center on Physical Activity and Disability. It has articles on aquatic therapy under the exercise and fitness category.

www.aeawave.com
This website provides standards and guidelines for aquatic fitness programming from the Aquatic Exercise Association.

www.mayoclinic.com/health/aquatic-exercise/SM00055&slide=1
This website provides basic instruction on a few simple aquatic exercises through a short series of slides with accompanying information.

www.humankinetics.com
This website provides information on books related to aquatic therapy.

TABLE 5–5. Pertinent Literature on Aquatic Therapy

Condition	# of Studies	Variables	Conclusions	Reference/Type of Study
Rheumatoid arthritis	6	• Pain • Health status	Effective in reducing pain and improving health status compared with no or other interventions in short-term. Long-term benefit is unknown.	Al-Qubaeissy et al, 2013[15] (SR)
Chronic low back pain	N/A	• Strength • Aerobic capacity • Flexibility • Back pain and disability • Quality of life	Short duration (2 months) and high frequency (5 times/week) improve strength, aerobic capacity, flexibility; decrease back pain and disability; increase quality of life	Baena-Beato et al, 2013(CCT)[23]
Fibromyalgia	27	• Quality of life • Physical function • Stiffness	Effective in improving quality of life, physical function and stiffness compared to no treatment; insufficient evidence to support benefits in all patients with fibromyalgia.	Lima et al, 2013[13] (SR & MA)
Postoperative orthopedics	8	• Function • Pain • Edema • Strength • ROM	Effective in improving function; does not increase the risk of wound-related adverse events; as effective as land-based therapy in reducing pain and edema and increasing strength and ROM.	Villalta & Peiris, 2013[17] (SR & MA)
Osteoarthritis (OA)	11	• Pain • Self-reported function • Physical functioning • Stiffness • Quality of life	Effective in managing symptoms associated with lower limb OA.	Waller et al, 2014 (SR & MA)[18]
Cerebral palsy (children)	N/A	• GMFM • PAES	Effective for children with cerebral palsy with poor Gross Motor Function Classification System level.	Lai et al, 2014[20] (QEPS)

CCT = controlled clinical trial; GMFM = gross motor function measure; MA = meta-analysis; N/A = not applicable; PAES = Physical Activity Enjoyment Scale; QEPS = quasi-experimental study without randomized assignment; RCT = randomized control trial; SBCT = single-blind controlled trial; SR = systematic review.

A Word About Hot Tubs and Jacuzzis

Hot tubs and Jacuzzis are popular in many health clubs and homes. These types of units are not intended for aquatic pool sessions because of their small size and high water temperature. Water temperatures generally range from 102°F to 104.5°F (38.9°C to 40.5°C), which places a greater demand on the cardiovascular and pulmonary systems. Regular care and cleaning of hot tubs and Jacuzzis is required to avoid the buildup of *P. aeruginosa,*[42–44] which can be accelerated by high temperatures and turbulence of the water, resulting in an increased potential for the presence of pathogens. A good filtration and chlorination system, similar to those found in therapeutic pools, is an essential requirement for all hot tubs and Jacuzzis.

WHIRLPOOLS

Whirlpools are enclosed stainless-steel or acrylic tanks of various sizes that are used clinically to provide therapeutic effects. These tanks have an attached motor, called a *turbine,* which agitates the water in the tank to create

the whirlpool effect. Smaller tanks can be portable and filled with a hose; larger tanks are typically stationary and have attachments to faucets to provide water to the tank. Whirlpools are intended to be used as single-patient treatments and must be drained and cleaned after each use.

Whirlpools have historically been used for treating a variety of musculoskeletal conditions (Box 5-3). Cold whirlpools have been used to help control pain and swelling of acute sprains and strains. Warm whirlpools are often used to facilitate motion and exercise for subacute and chronic stages of sprains and strains. Whirlpools may be applied following orthopedic surgery (after the surgical skin wounds are fully healed) to help soften scar tissue, reduce pain, and promote restoration of motion. The fluidity and pressure properties of whirlpools assist in taking off dry, scaly skin after casts have been removed, and the thermal and buoyant properties of a warm whirlpool assist in increasing mobility.[47]

Patients with osteoarthritis or rheumatoid arthritis may benefit from whirlpool therapy. The water's buoyant effects will help support the affected joints, decrease pain, and increase mobility. The thermal effects of a warm whirlpool may help decrease the pain of arthritis.[48–52] The relaxing effect of warmwater whirlpools is well known. Anxious patients may benefit from partial or full immersion (up to the neck) in a whirlpool to facilitate cooperation with rehabilitation procedures.[31] The patient may use earplugs or headphones to blunt the noise of the turbine, which promotes relaxation while in the whirlpool.

Previously, whirlpools have been used for the treatment of open wounds. Unfortunately, whirlpools increase the risk of infection, can damage fragile tissue from water agitation, and can cause complications of swelling in limbs treated in the dependent position in warm water. Therefore, their use for open wounds is no longer recommended.[12,53,54]

Box 5 ■ 3 Musculoskeletal Conditions Treated by Whirlpool

- Sprains and strains
- Contractures
- Postsurgical repair of joints and soft tissues
- Healing fractures of bones
- Osteoarthritis
- Rheumatoid arthritis

Types of Whirlpools

Whirlpools are available in various sizes and shapes to accommodate either a body part or full-body (up to the neck) immersion. Small whirlpools are designed to treat distal parts of the upper or lower extremities. Many are portable, with attached wheels for ease of movement (Fig. 5-10). Larger whirlpools can accommodate the entire upper or lower extremity or partial or full immersion of the body. "Low-boy" tanks have low walls that allow for ease of transfer in and out of the tank (Fig. 5-11). Patients in a low-boy are usually in a long-sitting position during treatment. "High-boy" tanks, sometimes referred to as *hip tanks,* require a chair or lift to transfer in and out of the tank (Fig. 5-12). Patients in a high-boy tank are usually sitting on a removable seat attached to rungs on the side of the tank, or they may sit on a high chair outside the tank with their lower extremity

Fig 5 ■ 10 Small portable (ankle/foot/hand) whirlpool.

Fig 5 ■ 11 Low-boy whirlpool.

Fig 5 ■ 12 High-boy whirlpool.

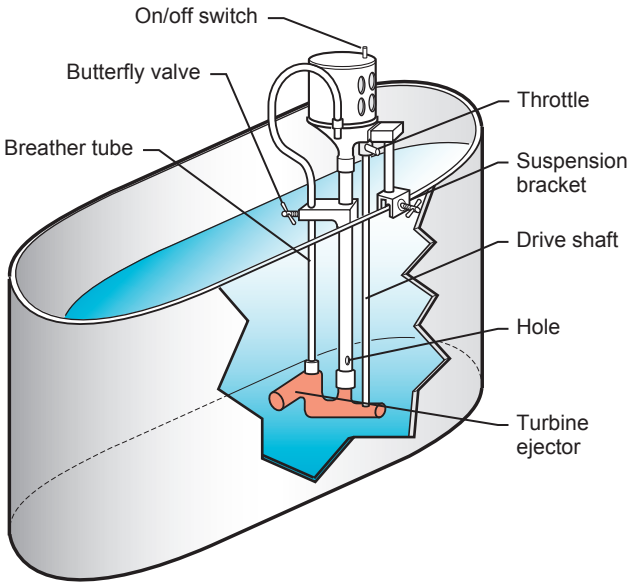

Fig 5 ■ 13 Turbine attached to a whirlpool.

dangling in the tank. Some tanks are specially designed for treating patients with burns. They are usually larger than extremity tanks and require a lift for patient transfer into and out of the tank. Some of these tanks are designed without seams; this helps prevent infections by limiting sites in the tank that are more difficult to clean and disinfect.

Turbine

The turbine is the electrical motor pump that creates the agitation of the water in a whirlpool (Fig. 5-13). A switch atop the turbine turns the motor on and off. Attached to the turbine are two or three tubular metal shafts. One of these tubes is the drive shaft, which contains an impeller housed in a casing at the bottom of the tube. The amount of water ejected at the base of the drive shaft determines the force of the ejected water. Adjusting the throttle near the top of the shaft can control the amount of water ejected. The other tube is called the *breather tube.* The amount of air mixing with the ejected water (aeration) at the base of the breather tube can be adjusted by turning the butterfly valve near the shaft's top. Agitation of the water in the whirlpool tank can be regulated by adjusting the force of the ejected water and the amount of aeration. The entire turbine assembly is usually mounted on a spring-loaded adjustable pole on the side of the whirlpool tank. This allows for height and side-to-side adjustments of the turbine, which enables the clinician to direct the ejected water toward or away from the body or body part in the whirlpool tank.

Some turbines have a small hole near the bottom of the turbine's middle shaft. This hole must be under the water whenever the turbine motor is turned on. Care must also be taken to ensure that this hole and the turbine ejector at the bottom of the turbine shaft are not blocked by bandages, wound packing, or fingers or toes.

Electrical Safety

Electrical safety is an essential concern for all whirlpools. Ground-fault circuit interrupters (GFCIs) are required for all electrical outlets where whirlpools are located. Proper function of the GFCIs should be checked and the condition of the turbine and attached wire should be inspected for damage before performing any whirlpool treatments. The attachment of the turbine on the adjustable pole should also be inspected to be certain it is tightly secured. An unsecured turbine could come loose during position adjustments and fall into the whirlpool tank, potentially causing a fatal electrical shock. Patients should be instructed to *never* touch the turbine, including the on/off switch. Only the healthcare practitioner should operate the on/off switch during whirlpool treatments.

CLINICAL APPLICATIONS FOR WHIRLPOOLS

Preparatory Considerations

Whirlpools should be inspected and properly cleaned before use. The room where the whirlpool is located should have a comfortable temperature to prevent chilling of the patient, and the humidity level should allow for efficient heat exchange from the patient to the environment. The floor adjacent to the whirlpool should be dry, preferably with a nonskid surface. After the therapeutic goals of the treatment are determined, the appropriate temperature of the water should be selected. Commonly used temperatures and their corresponding generic names are listed in Table 5-2. In general, temperatures between 97°F and 104°F (36°C and 40°C) are best when the heating effects of water are desired. Temperatures between 56°F to 80°F (14°C and 26°C) are best when the cooling effects of water are desired.

Caution must be used when choosing a water temperature for a patient who has peripheral vascular disease or sensory loss. Choose tepid or neutral water temperatures for these patients and for full-body-immersion (up to the neck) whirlpools. When choosing a safe water temperature for patients with peripheral vascular disease, make sure the water temperature is not greater than 1°F or 1°C above the skin temperature of the extremity to be immersed in the water. Water temperature should not exceed 100°F (38°C) for patients who have cardiovascular or pulmonary disease.

The next consideration before immersing the patient in the whirlpool is the condition of the body part to be treated. The skin should be examined for temperature, presence of edema, open lesions, color, sensation, and tenderness, and the results of this examination should be documented. Clothes should be removed from the body part to be immersed. Patients who will have their pelvis immersed in water should wear a bathing suit or suitable covering to protect privacy. The treatment procedure and safe operation of the whirlpool should be explained to the patient to reduce anxiety and promote safety. Therapists should assess the need for assistance to safely enter and exit the whirlpool. Mechanical lift devices may be necessary to assist patients who cannot stand or transfer to and from the whirlpool.

The patient should be positioned comfortably in the whirlpool using padding or pillows as needed to limit pressure on body parts that may be against the side of the tank. The position of the turbine should be adjusted before activating the agitation, depending on the desired direction of the water jet. Do not direct the agitation directly toward any body area that may be damaged or experience increased pain. The force of the water jet should also be adjusted before activation, depending on whether the goal is maximal or minimal mechanical effects on the body part. After the patient is placed in the whirlpool, the therapist can turn on the turbine and adjust the direction and force of the water jet based upon patient tolerance for the agitation.

Patients should be provided with a bell or alarm they can activate if the therapist's assistance is needed during the whirlpool treatment. Children, elderly patients, and patients with cognitive deficits should not be left unattended during the treatment session. Patients on a mechanical lift device or stretcher should be secured by straps when receiving treatment. Patients sitting in an elevated chair with a lower extremity dangling in a whirlpool should wear a seat belt to hold them securely in the chair.

Whirlpool Duration

The length of time for treatment in a whirlpool varies depending upon the patient's diagnosis, goals of the treatment, the patient's physical and mental condition, and patient tolerance. The usual treatment duration is 20 minutes when the goal is heating of the body part. Borrell et al[55] demonstrated that 20 minutes was enough time to increase skin, muscle, and joint capsule temperature in the hand and foot. Abramson et al[56] demonstrated that a 20-minute application of moist heat increased blood flow, and further exposure of up to 2 hours had no real effect on increasing the peak response obtained at the 20-minute mark.

Whirlpools used for cryotherapy can be limited to 10 to 15 minutes, depending on patient tolerance for cold water. A duration of 10 to 30 minutes is recommended for patients who are exercising in the whirlpool, depending on the patient's medical status.

Cleaning and Disinfecting Whirlpools

No chemical additives are necessary in whirlpool water when the skin is intact. Whirlpool tanks must be drained, cleaned, and disinfected after each use. Practitioners

should wear gloves and goggles to protect the hands and eyes when doing this. The inside surface of the tank, turbine tubes, and drains should first be manually scrubbed with a cleanser, then thoroughly rinsed with clean water. Next, a disinfectant should be applied to the inside of the tank and turbine tubes (following label-recommended dilutions) for no more than 10 minutes. The tank is then thoroughly rinsed and dried. Sterilization of whirlpool tanks is not practical or necessary.

Upper and Lower Extremity Techniques

Whirlpools for the upper extremity require a high-boy tank or a raised small tank. The patient should be seated comfortably on a chair or stool next to the tank and positioned so that the back is supported and the arm is placed in the tank without bending the trunk forward. A towel or other form of padding should be placed under the arm and against the tank edge to avoid constricting the circulatory and lymphatic system and compressing the nerves (Fig. 5-14).

Lower-extremity immersion or immersion to the mid-thoracic level can be achieved by using either a high-boy or a low-boy whirlpool tank. The high-boy tank requires that the patient be able to flex the hip and knee. The length of this tub does not allow for full extension of the average adult's lower extremity and limits the amount of range-of-motion exercises a person can perform while in the whirlpool. However, its depth allows a greater body surface area to be submerged safely and comfortably, including submersion up to the mid-thoracic region. The low-boy tank is not as deep but has greater length than the high-boy, and it affords the patient the ability to fully extend the lower extremities and perform full-motion exercises for the knees. When only the distal portion of the lower extremity has to be immersed, a small whirlpool tank is best. The high-boy and low-boy afford greater body-surface immersion than a small-extremity tank; however, the low-boy may allow for greater lower-extremity extension than the high-boy. If the patient is unable to safely transfer into one of these tubs, a hydraulic chair lift or mechanical lift device can be used.

Key Point! Caution must be taken when using whirlpools to treat the distal upper or lower extremity because the dependent position of the extremity in the tank may promote edema, especially when higher temperatures are used.

Fig 5 ■ 14 Whirlpool treatment to the hand and forearm. Caution should be used to avoid a totally dependent position of the hand. If possible, active ROM exercises should be performed during the treatment to encourage venous and lymphatic return, thus minimizing edema formation.

Magness et al[57] studied the effect of whirlpool on volume in the upper extremity. They measured upper-extremity volume in 20 normal male and female volunteers before and after the immersion of the same extremity for 20 minutes in a whirlpool bath at temperatures ranging from 92.3°F to 111.9°F (33.5°C to 44.4°C). In addition, 20 patients with various upper-extremity disorders were treated by whirlpool in the same manner at temperatures ranging from 100°F to 104°F (37.8°C to 40°C) for 20 minutes. The results revealed a significant increase in volume for the normal subjects directly related to the higher water temperatures. There was also a significant increase in volume of the patient's extremity, and the rise in volume was greater than in the normal subjects at the specific temperatures treated.

Hoyrup and Kjorvel[58] studied the effect of whirlpool and paraffin dips on hand volume, ROM, and pain in patients with traumatic hand injuries. The patients received whirlpool treatments at 109.4°F (43°C) and paraffin dips at 122°F (50°C). One-half of each group performed exercises; the other half did not. A significant

reduction in pain and increase in motion were found. There was no significant change in hand volume during a 3-week period. However, daily increases in hand volume were significant, and the increased volumes were significantly greater in the whirlpool group. These studies suggest whirlpool treatments may not be the best choice for patients who have edema in their distal upper or lower extremities.

Clinical Controversy

Whirlpools for acute sprains allow the practitioner to apply cold water to the entire circumference of a joint and allow for active ROM during the treatment. The goal is to decrease edema using the effects of cold and assist active movement of the joint to help maintain mobility. However, the dependent position of the extremity in the whirlpool tank inhibits venous return, which may limit edema reduction and possibly exacerbate edema in the joint. Therefore, practitioners must consider whether other cryotherapy modalities would be a better choice for acute sprains when the goal is to limit or decrease edema.

Full-Body Immersion Technique

Some larger whirlpools can allow immersion of the body in water up to the neck (full-body immersion). Water temperature should be in the neutral to warm range to prevent chilling or overheating the patient. Patients must be screened carefully for tolerance to this technique, including assessment of vital signs. Some cardiovascular and pulmonary conditions and cognitive deficits preclude full-body immersion. Patients may experience some light-headedness after full-body immersion, especially if the water was very warm. This may be avoided by having the patient rest in a lying or sitting position for 5 to 10 minutes before standing after a full-immersion whirlpool.

INDICATIONS FOR WHIRLPOOL TREATMENTS

Musculoskeletal Conditions

Historically, whirlpools have been used to treat musculoskeletal conditions such as sprains and strains. Cool or cold whirlpools are typically used to help control pain and swelling of acute sprains and strains, and warm or hot whirlpools are often used to facilitate motion and exercise for subacute and chronic stages of sprains and strains. ROM of stiff or painful joints may be facilitated by whirlpool treatment. Stretching of soft tissue contractures may be aided by the simultaneous or immediate preapplication of a warm whirlpool. Whirlpools may be applied after orthopedic surgery (after the surgical skin wounds are fully healed) to help soften scar tissue, reduce pain, and promote restoration of motion. Patients with healing fractures may benefit from whirlpool when clinical union has been achieved. The fluidity and pressure properties of whirlpool assist in taking off dry, scaly skin after casts have been removed, and the thermal and buoyant properties of a warm whirlpool may assist in increasing joint and limb mobility.[47]

Patients with weakness of the extremities may be able to move their extremities in the buoyancy of water and may be unable to move them otherwise. Patients with osteoarthritis or rheumatoid arthritis may benefit from whirlpool. The buoyant effects of the water will decrease weight-bearing compression forces on the affected joints and can decrease joint pain and facilitate exercise. The thermal effects of a warm or hot whirlpool may help decrease the pain of arthritis.[48-52]

Circulatory Conditions

The effect of a warm or hot water whirlpool on blood flow was studied by Cohen et al.[59] They reported a 21% increase in extremity blood flow after a 101.5°F (38.6°C) whirlpool and a 50% increase in extremity blood flow after a 108.5°F (42.5°C) whirlpool. There was no significant difference in extremity blood flow between extremities immersed in whirlpools without agitation and those with agitation. Therefore, the effect on blood flow is more likely dependent upon the water's temperature rather than any mechanical effects of whirlpool agitation. Extremities with edema should not be placed in warm or hot water whirlpools because heat will increase tissue temperature and intravascular pressure,[60] thereby increasing inflammation and peripheral arterial blood flow, which will likely increase edema.[57,58,61]

Cool or cold-water whirlpools have been used for acute or chronic conditions for reduction of edema. The hydrostatic pressure of water and its effects on

circulation and renal function may help to reduce postoperative peripheral edema.[62] The use of a cool or cold-water whirlpool will cause vasoconstriction and reduce vascular permeability. This may also help to reduce edema. However, the dependent position of an extremity required for using a whirlpool may inhibit edema reduction. Although Dolan et al[63] found that immersing the hind limbs of rats in the dependent position in cold water effectively curbed edema formation, McCulloch and Boyd[7] found that placing the lower extremities of humans in the dependent position in an empty whirlpool tank for 20 minutes increased limb volume. Therefore, caution must be used when placing an extremity with edema in a whirlpool. Active movement of the limb in the whirlpool may help inhibit increased edema. Limb circumferences must be measured before and after treatment to closely monitor the effect of the whirlpool and the dependent position of the limb on edema.

Psychological Conditions

The relaxing effects of warmwater whirlpools are well known. Anxious patients may benefit from partial or full immersion in a whirlpool to facilitate cooperation with rehabilitation procedures.[30,31] Partial immersion in a cool or cold whirlpool may stimulate drowsy or stuporous patients to facilitate participation in rehabilitation. See Table 5-6 for precautions and Table 5-7 for contraindications for clinical use of whirlpools.

CONTRAST BATH

Repeated immersion of an extremity into a tub or tank of hot water and alternating with a tub or tank of cold water has been proposed to increase blood flow in the immersed body part without causing or increasing edema. Typically, a fixed time ratio of 3 or 4 minutes in the hot bath to 1 minute in the cold bath is performed for 4 to 5 cycles. Alternating between hot and cold water is thought to trigger a vascular pumping action caused by vasodilation (hot water) and vasoconstriction (cold water). Hypothetically, this will stimulate local circulation in the treated extremity and, to a lesser extent, increase circulation in the contralateral untreated extremity. However, several research studies[64–67] and systematic reviews[68,69] found little evidence supporting the effectiveness of this technique. Fiscus et al[70] studied the effects of a 4:1 (hot:cold) contrast bath on circulation in the lower

TABLE 5–6. Whirlpool Precautions

Precaution	Explanation
Malignancies	Danger of metastasis due to increased blood flow with warm or hot water temperatures.
Sensory impairments	Danger of burns if water is too hot.
Hypersensitivity to cold	Cold urticaria may occur.
Pulmonary disease	Poor ability to resist hydrostatic pressures with full-body immersion; monitor patient for signs of respiratory distress.
Cardiac insufficiency	May have difficulty adapting to maintain thermal homeostasis; limit maximum immersion time to 10 minutes initially and monitor vital signs during treatment.
Unstable blood pressure	Blood pressure problem may be exacerbated with full immersion in hot or cold water.
Impaired circulation	Patients with peripheral vascular disease (diabetes, arteriosclerosis): danger of burns; do not place in water temperatures greater than 95°F (35°C).
Edema	May increase edema if warm or hot water is used.
Acute febrile episode	May exacerbate fever if warm or hot water is used.
Acute inflammation	May aggravate inflammation if warm or hot water is used.
Thermal regulation impairment	Danger of thermal shock if fully immersed in warm or hot water because convection and sweating may be impaired.
Rheumatic arthritis (RA)	Warm or hot water may not be tolerated by patients in the acute stage of RA.
Multiple sclerosis	May increase fatigue and weakness in water temperatures above 88°F (31°C).
Confusion or impaired cognition	May not be able to report problems or discomfort.
Fear of water	May refuse full-body immersion or induce panic.

TABLE 5–7. Whirlpool Contraindications

Contraindication	Explanation
Incontinent bladder or bowel	May contaminate water and cause infections.
Alcohol or drug ingestion	Reduced judgment; may enhance a hypotensive response; inquire if patient has ingested alcohol or drugs before using the whirlpool.
Uncontrolled epilepsy	Full-body immersion contraindicated because of increased risk of drowning during an attack.
Danger of hemorrhaging	Agitation of water may increase bleeding if warm or hot water is used.
Post-op surgical incisions with dehiscence, sutures, or staples	Contraindicated because whirlpool can prolong or prevent wound closure.
Skin ulcers caused by venous insufficiency, edema, or lymphedema	Contraindicated because whirlpool can prolong or prevent wound closure.
Skin conditions such as atopic eczema, ichthyosis, and senile pruritus	May exacerbate condition due to removal of natural skin moisture.
Tissue flaps or recent skin grafts	May not tolerate the agitation or may not be able to compensate for extremes of heat or cold.
Skin infection	May cause cross-contamination of the other body parts immersed in the water.
Thrombophlebitic areas	Use of warm or hot water whirlpool is contraindicated because of the danger of dislodging the clot with enhanced circulation in the area.

CASE STUDY 5-4 Selecting Whirlpool for a Patient With Musculoskeletal Dysfunction

A 72-year-old female who has a history of osteoporosis fell while walking in her yard 6 weeks ago, sustaining a Colles fracture of her right wrist. She had a closed reduction and cast application to the right forearm, wrist, and hand. The cast was removed 3 days ago. Today, she has mild swelling of the wrist and significant loss of ROM. She has poor tolerance to passive stretching and joint mobilization of the wrist.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of hydrotherapy?
ANSWER: Yes, loss of ROM secondary to stiffness, contracture, and pain of a joint is an indication for the use of hydrotherapy to help decrease pain and facilitate active and passive ROM.

2. Is the patient appropriate for application of hydrotherapy (i.e., do any of the general precautions or contraindications to hydrotherapy apply, or are there any specific considerations regarding application of hydrotherapy to this patient)?
ANSWER: As long as this patient has intact sensation and can sit with her arm in a whirlpool, she should be able to safely receive a hydrotherapy treatment for her right lower arm. The mild edema in her wrist will need to be monitored because any increase in edema following whirlpool will require discontinuation of this treatment.

3. What are the specific goals to be achieved with the use of hydrotherapy?
ANSWER: Decreased pain in wrist, increased tolerance for passive ROM, stretching and joint mobilization, and increased mobility of the wrist and forearm.

4. Do you have the specific type of hydrotherapy that is appropriate for this patient?
ANSWER: A small whirlpool on a stand or pedestal would be best; a high-boy tank can also be used so that the patient can sit on a chair with her arm comfortably inside the tank (see Fig. 5-10).

5. What specific hydrotherapy parameters would be appropriate for this patient?
ANSWER: To decrease pain and facilitate improved mobility of the wrist and forearm, the following parameters would be appropriate:
Water temperature: Warm (97°F–99°F [36.1°C–37.2°C]) or cool (66°F–80°F [18.9°C–26.7°C]),

CASE STUDY 5-4 Selecting Whirlpool for a Patient With Musculoskeletal Dysfunction—cont'd

depending on the patient's comfort with water temperature.
Agitation: Begin with low setting, with the force directed at the wrist and forearm if tolerated, and gradually increase agitation to maximally tolerated level.
Duration: 15 minutes.
Frequency: Either daily or every other day at the beginning of the therapy session until pain and stiffness are reduced and no longer limiting tolerance to stretching, joint mobilization, and exercise.

6. What are the proper (e.g., effective and safe) application procedures for hydrotherapy related to this case example?

ANSWER:

Instruct the patient: Inform the patient of the purpose and procedure. Be sure to explain the anticipated sensation and effect of the whirlpool and to immediately report any discomfort to the practitioner.
Preinspection: Inspect the area to be treated for skin compromise and sensation. Measure the circumference of the wrist to determine amount of edema present.
Patient position: Patient should be able to sit comfortably in a chair with her arm in the whirlpool tank and a towel for padding under her axilla.
Postinspection: Remove the arm from the water, dry the skin with a towel, and inspect the skin for any signs of irritation or adverse effects. Measure the circumference of the wrist to determine any effect of the whirlpool on edema.

legs of 24 healthy men. Fluctuations in blood flow occurred during the 20-minute contrast bath; however, the authors believed that these changes were likely secondary to changes in cutaneous circulation and not intramuscular circulation. A study by Cote et al[71] found that contrast baths *increased* edema in postacute sprained ankles. A recent study by Shih et al[72] compared the effect of the time ratio of hot to cold baths on brachial artery mean blood velocity. They found that the addition of a 10-minute hot bath after an initial 3-minute hot and 1-minute cold bath during the second cycle of contrast baths resulted in increased brachial artery blood flow velocity.

More studies are needed to determine the effect of this technique on edema and blood flow after subsequent contrast bath cycles. Contrast baths have been recommended for pain relief and desensitization. Kuligowski et al[73] found decreased pain perception and improved elbow flexion after contrast baths and eccentric contractions of the elbow flexors. Vaile et al[74] studied the effect of contrast baths on delayed-onset muscle soreness (DOMS). Subjects who received contrast baths after lower-extremity exercise had a reduction in the physiological and functional deficits associated with DOMS, including improved recovery of isometric force and dynamic power and a reduction in localized edema.

Clinical Controversy

There is insufficient evidence to support the use of contrast bath for the purpose of reducing edema in distal extremities.

Patients who have any contraindications for the use of heat, cold, or water should not receive contrast baths. Patients with small-vessel vascular disease secondary to diabetes, arteriosclerotic endarteritis, or Buerger's disease may not be able to tolerate the rapid change in temperatures used in contrast baths, depending on the severity of the disease. Circulation in these patients must be monitored carefully.

NONIMMERSION IRRIGATION OF WOUNDS

Pulsed Lavage With Suction

PLWS is a hydrotherapy modality for the irrigation and debridement of open wounds. Pulsed lavage provides a controlled pulsating pressurized stream of irrigants to the wound. The pressure is maintained between 4 and 15 pounds per square inch (psi), depending on the amount of eschar and necrotic tissue in the wound and the patient's tolerance. Concurrent suction applied to

the wound creates a negative pressure in the wound bed during treatment to effectively remove irrigants and facilitate elimination of pathogens.[75] The most common irrigants used during PLWS are saline and tap water. A systematic review by Moore and Cowman[76] found no difference in wound healing when using either saline or water.

PLWS is indicated for the cleansing and debridement of wounds caused by arterial insufficiency, venous insufficiency, neuropathic ulcers, pressure ulcers, small burns, postsurgical wounds, infected wounds, fasciotomies, partial-take split-thickness skin grafts, and trauma.[77,78] PLWS can help decrease bacteria and infection in wounds and promote granulation and epithelialization.[79] Ho et al[80] applied daily low-pressure (11 psi) pulsatile lavage treatments with 1 L of normal saline to subjects with spinal cord injury and stage III and IV pelvic pressure ulcers. A control group received only sham treatment and dressing changes. Subjects in the treatment group had an enhanced ulcer healing rate compared to the control group. Strong evidence exists that PLWS decreases bacterial load within wounds,[81,82] which may be the primary mechanism through which it enhances wound healing rates.

PLWS has several clinical advantages over whirlpool treatments for wound care. It is portable and can be performed at bedside or in the home. Morgan and Hoelscher[83] performed a retrospective cohort study that found treating wounds at home using pulsed lavage effectively removed necrotic tissue, promoted wound healing, and was more cost-effective than in-hospital whirlpool treatments or surgical debridement. PLWS requires shorter treatment times than whirlpool treatments and can be used to treat a small area rather than placing an entire extremity in a whirlpool. A major advantage of PLWS over whirlpool is that the limb can be properly elevated and the lavage site can be site-specific, particularly for patients with edematous limbs or multiple venous insufficient ulcers.[78] Cross-contamination is less likely because PLWS is delivered through a single-patient device with a disposable tip. The pressure delivered to a wound with PLWS (unlike a whirlpool) can be precisely set in pounds per square inch psi, allowing for safer delivery of nondamaging pressure to the wound. PLWS can deliver the therapeutic benefits of a whirlpool without its potential adverse effects.[84] Possible disadvantages of PLWS include the cost associated with single-use attachments and the inability to treat large surface wounds such as extensive burns.

Key Point! PLWS is preferred over whirlpool for cleansing and debriding wounds because of decreased risk of damage to tissue and cross-contamination. Also, treatment can be performed with the limb in a nondependent position.

CASE STUDY 5-5 Selecting PLWS for a Patient With Spinal Cord Injury and Stage III Pressure Ulcer

A 36-year-old male with a 10-year history of quadriplegia was admitted to the hospital for treatment of stage III sacrococcygeal pressure ulcers. His wounds had minimal necrotic tissue, mild foul odor, and minimal serosaguinous exudate, with no evidence of cellulitis, tunneling, or fistulas. The ulcers measured 6 × 8 cm and 4 × 9 cm with an average depth of 1 to 2 cm. He received standard wound care with regular dressing changes to provide an optimal moist healing environment.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction or impairment that can be improved with the use of PLWS?
ANSWER: Yes, daily PLWS treatment of pressure ulcers has been shown to promote healing of stage III and IV pressure ulcers.

2. Why would PLWS be a consideration for this patient?
ANSWER: This patient is appropriate for daily treatment of his wounds using PLWS for thorough wound cleansing and promotion of healing. At this time, he does not have purulent drainage, greater than 50% necrotic tissue, and sepsis, so daily treatment is sufficient. The area of his wounds is insensate, so this is a precaution that must be considered during PLWS treatment.

3. What specific outcome measures can be used to assess the effectiveness of PLWS?
ANSWER: Decreased bacteria present in the wound, promotion of healing that can be assessed by wound appearance (clean with little or no necrotic tissue and exudate), measurement of wound size, and presence of granulation tissue.

CASE STUDY 5-5 Selecting PLWS for a Patient With Spinal Cord Injury and Stage III Pressure Ulcer—cont'd

4. Are there any precautions or contraindications to consider for this patient?
ANSWER: This patient has quadriplegia, so his tissues are insensate in the area of his wounds. This is a precaution when performing PLWS because the patient cannot report any discomfort that would be a warning sign of tissue damage during treatment.

5. What specific parameters would be appropriate for this patient?
ANSWER: PLWS parameters for this patient are to set irrigation pressure at 4 to 15 psi, set suction at 60 to 100 mm Hg, and choose a soft splash shield tip. Perform treatment for 5 to 15 minutes, depending on condition of wounds and patient tolerance.

6. What are the proper steps for using PLWS for this patient?
ANSWER: Proper infection control procedures must be followed. The therapist must wear a protective mask, gown, and gloves, and the patient must wear a mask. Dressings must be removed from the wounds. The patient must be positioned properly to allow access to the wounds and maintain patient comfort during the treatment. The wound must be inspected and its condition documented. Specific PLWS parameters are set, and the procedure performed until treatment session goals are met (usually about 10 minutes). After treatment, the wound is again inspected and its condition documented. Dressings are reapplied to the wounds. Single-use items and infection control attire are discarded properly.

Clinical Application

PLWS has replaced whirlpools for cleansing and debriding infected, necrotic, or granulating wounds. PLWS devices have specially designed applicator tips for irrigating surface wounds and tracts, tunnels, and areas of undermining.[78] Several PLWS devices are available (Fig. 5-15). Consult the manufacturer's recommendations for proper operation of the device before use.

Clinical application of PLWS (Fig. 5-16) requires the following steps:[78]

1. In addition to the PLWS device, the facility where the treatment is performed must provide the following:
   - Wall or portable suction
   - Suction regulator
   - One or two bags of normal saline (0.9% sodium chloride)
   - Collection canister
   - A private room with walls and doors that close
   - Infection-control garments: face shield, mask, hair covering that also covers the ears, fluid-proof gown with long sleeves, fluid-resistant shoe covers, and gloves
2. Wash hands before treatment.
3. Don infection-control garments, including a surgical mask for the patient. The U.S. Department of Labor Occupational Safety and Health Administration guidelines for infection control (available at www.osha.gov) should be followed during PLWS treatments.
4. Cover all exposed items in the treatment field with a towel, including any exposed tubes, ports, or other wounds not being treated.

Fig 5 ■ 15 Pulsed lavage with suction (PLWS) for treatment of an open wound. *(Courtesy of DAVOL Company, Warwick, Rhode Island.)*

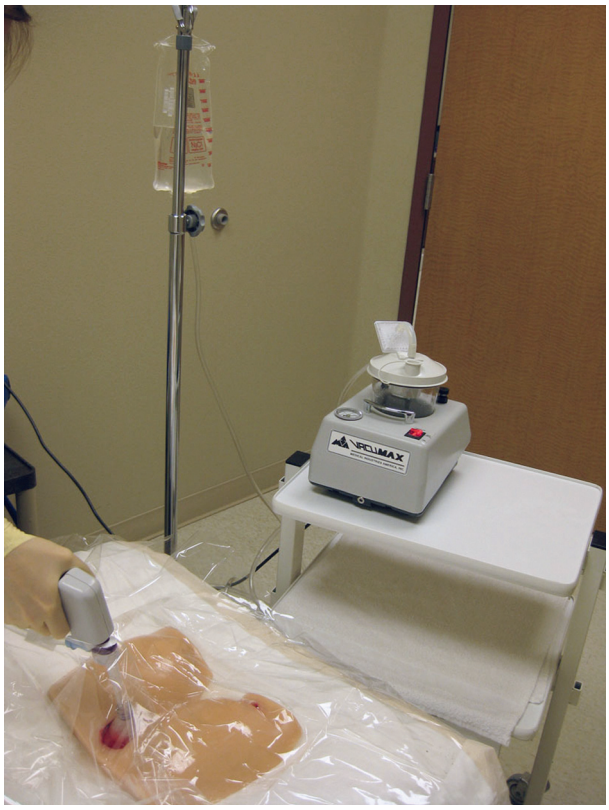

Fig 5 ■ 16 PLWS set-up for treatment of a simulated pressure ulcer.

5. Warm saline bags to 102.2°F to 104°F (39°C to 40°C).
6. Set suction regulator between 60 and 100 mm Hg (Table 5-8).
7. Choose appropriate tip for applicator (see Table 5-8).
8. Set irrigation pressure (see Table 5-8).
9. Position patient comfortably, and remove dressings from wounds.
10. Begin irrigation and carefully monitor patient's response.
11. Maintain total contact with the wound bed if using a splash shield tip.
12. Perform PLWS for 10 to 20 minutes, depending on patient response and wound response to treatment. (See Table 5-9 for recommended frequency of treatments.)
13. When treatment is completed, all disposable parts of the device, including the suction canister, must be discarded in a white plastic bag with a biohazard label. The suction canister should be emptied in a commode before being discarded.
14. Inspect wound and document any changes in appearance, drainage, or size.
15. Reapply appropriate wound dressings.
16. Discard any linen exposed to aerosolized blood or body fluid into soiled linen receptacles, and clean all surfaces and stretchers, wheelchairs, and carts with disinfectant.
18. Remove all infection-control garments and dispose properly.
19. Wash hands.

Some precautions need to be followed when performing PLWS (Table 5-10). No known absolute contraindications currently exist for PLWS; however, only experienced clinicians should use PLWS for complex wounds with fistulas, exposed cavity linings, or long wound tunnels into body cavities.[7]

Clinical Controversy

Debridement of wounds has historically been performed in whirlpools. PLWS has been shown to be more effective than whirlpool for debridement of wounds with less risk of tissue damage and cross-contamination.

TABLE 5–8. Parameters for PLWS Treatment

Suction	Continuous mode at 60 to 100 mm Hg; low range if applied near a vessel, in a tunnel, an undermined area, or near a cavity lining; decrease if bleeding occurs or pain increases.
Irrigation tip	Soft splash shield for tip for surface wounds; long flexible tip for tracts and undermining.
Irrigation pressure	Adjustable between 4 and 15 psi lower range for tunnels: 4 to 6 psi; higher range for reducing bacterial levels in infected wounds and for wounds with large amounts of exudate or necrosis: 12 to 15 psi.
Duration of treatment	5 to 15 minutes, depending on size of wound, presence of exudate and necrosis, and patient tolerance.
Frequency of treatment	See Table 5-9.

TABLE 5–9. Frequency and Discontinuation Recommendations for PLWS

Frequency	Daily	Twice Daily	3 Times Per Week	Discontinue
Most wounds	X			
Greater than 50% necrotic		X		
Purulent drainage		X		
Sepsis		X		
Full granulation base			X	
NPWT being used			X	
No increased granulation for 1 week				X
No decreased necrotic tissue for 1 week				X
Wound closed				X

NPWT = negative pressure wound therapy.

Adapted with permission from Loehne HB, PT. Management of Chronic Wounds, Wound Healing Treatment Interventions. Presented at American Physical Therapy Association Annual Conference in Baltimore, MD; June 13, 2009.

TABLE 5–10. Precautions for Pulsed Lavage With Suction[7]

Precaution	Explanation
Over insensate areas	Patient may not be able to report pain that could lead to tissue damage.
Patients taking anticoagulant medications	Higher risk of bleeding from procedure.
Wounds with tunnels, tracts, or undermining	Risk of tissue damage to areas that cannot be seen by practitioner.
Near major vessels	Risk of damage to vessels, causing bleeding.
Near cavity linings such as pericardium of peritoneum	Proximity to the heart and other vital structures.
Recent bypass, graft sites, or flaps	Risk of damage to bypass, graft sites, or flaps.
Exposed bone, tendons, or vessels	Risk of damage to bone, tendon, or blood vessels from procedure.
Facial wounds	Proximity to eyes and other vital structures.

There are no known absolute contraindications to PLWS.[7]

ASSESSMENT OF EFFECTIVENESS AND EXPECTED OUTCOMES FOR HYDROTHERAPY

Clinical Decision-Making

The decision to include hydrotherapy in an intervention plan should be based upon the evidence for effectiveness. Practitioners must regularly consult the latest published literature to match their knowledge and expertise with the needs of the patient. Searches of databases for hydrotherapy enable the practitioner to base clinical decisions on available evidence of effectiveness. Key words or medical subject headings are helpful when searching databases for information on the effectiveness of hydrotherapy. See Table 5-11 for pertinent studies on whirlpool, contrast bath, and PLWS.

Goals and Outcomes

Goals of hydrotherapy should be clearly documented in the initial examination of the patient. The effectiveness of a hydrotherapy intervention can be assessed based upon accomplishment of these goals and on response of the patient to each treatment. Outcomes can be determined by measuring the effect on impairments, which can include pain, joint ROM, strength, wound condition, edema, balance and coordination, and the assessment of function (see Table 5-12 for suggested potential outcome measures

TABLE 5–11. Pertinent Literature on Whirlpools, Contrast Bath, and PLWS

Condition	Variables	Type of Hydrotherapy	Conclusions	Reference/Type of Study
People with "chronic stroke"	Pre- and post-WOMAC pain and stiffness indexes	Whirlpool, 104°F (40°C) for 40 minutes, 5x/week for 8 weeks	WOMAC pain score and stiffness indexes significantly lower ($P < 0.05$) for group that received whirlpool	Lim et al, 2013[85] (RCT)
People with chronic myofascial pain syndrome in upper trapezius without depression	Pain (visual analog scale), anxiety (Korean versions of Beck Anxiety Inventory and WHO QoL Assessment-Brief form)	Whirlpool, 93°F (34°C) to 96.8°F (36°C) for 30 minutes, 3x/week for 2 weeks	Significantly greater decrease in pain ($P = 0.002$) and anxiety ($P = 0.010$) in group that received whirlpool	Im et al, 2013[86] (RCT)
Normals, RA, diabetes, ankle/foot injuries	Blood flow, intramuscular and subcutaneous temperature	Contrast bath	May increase superficial blood flow and skin temperature; evidence on edema conflicting; no relationship between physiological effects and outcomes has been established	Breger Stanton et al, 2009[87] (SR)
Athletes and sedentary individuals, DOMS, edema, sprains	Multiple physiological and functional parameters	Contrast "therapy," including contrast bath	Insufficient evidence that contrast therapy aids in the treatment of injury or recovery from exercise	Hing et al, 2008[68] (SR)
Pelvic pressure ulcers	Linear and volume measurements of ulcer dimensions	PLWS	Enhanced stage III and IV ulcer healing rates	Ho et al, 2012[80] (RCT)
Pressure ulcers	Measurement of length and width of wounds	Wound cleansing techniques, including whirlpool	No statistically significant change in healing for ulcers with or without a whirlpool	Moore & Cowman, 2008[76] (SR)

WOMAC = Western Ontario and McMaster Universities Arthritis Index; RCT = randomized control trial; SR = systematic review.

TABLE 5–12. Hydrotherapy Outcome Measures

Aquatic Therapy	Whirlpool	PLWS
Pain scales (e.g., visual analog scale)	Joint ROM	Wound size and depth
Range of motion	Pain level assessment	Presence of exudate and necrotic tissue in wound
Strength	Tolerance to exercise	Presence of granulation tissue in wound
Edema	Joint stiffness	Odor
Balance (e.g., Tinetti scale)	Muscle strength tests	Pain level assessment
Coordination		
Function (FIM, SF-36)		

for aquatic therapy, whirlpool, and PLWS). Function may be a longer-term determination of the effectiveness of hydrotherapy interventions and an important concern for third-party payers. Functional inventories or questionnaires, such as the Functional Independence Measure (FIM), SF-36, and the Oswestry Low Back Pain Disability Questionnaire, may be helpful in assessing hydrotherapy effectiveness.

Documentation Tips

Documentation of hydrotherapy interventions should include the type of hydrotherapy performed, the parameters of the treatment, and the patient's response to the treatment. Specific documentation for each type of hydrotherapy should include:

- **Aquatic therapy**
  - Water temperature
  - Duration of the treatment
  - Goals of pool therapy
  - Movement or exercise techniques performed during therapy
  - Assistive equipment used during therapy
  - Means of entry and exit
  - The patient's tolerance to the treatment, including vital signs
  - Any adverse reactions
  - Post-pool therapy changes in identified impairments or functional limitations
- **Whirlpool**
  - Type of whirlpool
  - Body part immersed in water (partial or full immersion)
  - Temperature of the water
  - Agitation (mild, moderate, or full) and whether directed at or away from the body part
  - Duration of treatment
  - Passive (no movement during whirlpool) or active (movement or exercise during whirlpool)
  - Patient response or tolerance for the treatment
  - Post-whirlpool changes in identified impairments or functional limitations
- **PLWS**
  - Condition of wound, including location, size, and characteristics
  - Irrigation pressure in pounds psi
  - Patient response to treatment (tolerance to irrigation, pain)
  - Wound response to treatment (any bleeding, debridement of necrotic tissues, and exudate)
  - Duration of treatment

Additional documentation of patient response to hydrotherapy may include any evidence of adverse reactions, including marked temperature changes of the patient's skin; changes in skin color such as erythema, blanching, or cyanosis; or changes in skin appearance such as excessive softening or wrinkling of skin.

In addition, any change in the patient's pain, muscle spasm, ROM, strength, joint appearance, edema, coordination, orofacial control, endurance, functional level, and psychological state should be noted. Practitioners should be mindful that warm or hot water temperatures and full-body immersion tend to cause transient weakness during or immediately after hydrotherapy interventions. Vital signs should be monitored and recorded during and immediately after hydrotherapy for any patient with a history of cardiac or pulmonary disease to determine tolerance to the treatment.

Hydrotherapy in all its forms can be physically and psychologically therapeutic. However, practitioners must avoid indiscriminate use of this modality. Careful application of the principles of physics of water, knowledge of the physiological effects of water at various temperatures, and evidence of this modality's effectiveness for a given patient is essential for effective treatment. The advantages and disadvantages of hydrotherapy, including the cost of treatment, must be compared to other therapeutic interventions. Practitioners must be prepared to justify the choice of hydrotherapy as the best cost-effective intervention indicated for their patient.

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CHAPTER 6

ELECTROMAGNETIC WAVES—LASER, DIATHERMY, AND PULSED ELECTROMAGNETIC FIELDS

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The understanding of electromagnetic theory in the 19th century led to the development of the wireless telegraph and eventually to the development of radio, television, and cell phones. A whole new world of communication evolved that has profoundly changed the world we live in. The use of electromagnetic radiation, or waves, for therapeutic purposes began in the early 20th century when physicians began using high-frequency currents to heat muscles and joints.[1] The penetration of electromagnetic waves through body tissues enabled deeper heating than superficial heating devices (such as hot packs applied to the skin). Eventually, the development of devices that produce electromagnetic waves popularized what became known as *diathermy*. Originally diathermy included "ultrasonic" therapy (ultrasound);[2] however, the therapeutic effect of ultrasound is produced by mechanical vibrations, not electromagnetic waves (see Chapter 4). Today diathermy includes only therapeutic devices that produce electromagnetic waves from the microwave and radio frequency sections of the electromagnetic spectrum.

The use of electromagnetic waves from the ultraviolet part of the electromagnetic spectrum to treat skin problems became popular in the 20th century. During the latter part of the century, the development of lasers enabled the use of visible light (which is also an electromagnetic wave) for surgery and therapeutic purposes.

This chapter will include the use of light from the visible radiation part of the electromagnetic spectrum

(lasers and light-emitting diodes), radio frequency and microwave frequency waves (diathermy), and pulsed electromagnetic fields (PEMFs). The use of ultraviolet radiation for therapeutic purposes is covered in Chapter 15.

ELECTROMAGNETIC WAVES

Electromagnetic waves are waves of energy that are propagated through space. Accelerating electric charges produce electromagnetic waves of moving electric and magnetic fields. The electric and magnetic field strengths in an electromagnetic wave are illustrated in Figure 6-1. Notice that the electric and magnetic fields at any point of the electromagnetic wave are perpendicular to each other and to the direction of the wave's motion. These waves travel through space at the speed of light.[3]

Electromagnetic waves are grouped according to their quantum energy, frequency, and wavelength, known as the *electromagnetic spectrum* (Fig. 6-2). Lower-frequency waves are known as *radio waves*. These frequencies are used to transmit radio and television signals. Cellular phones, radar, and microwave ovens use higher frequencies in the "microwave" frequency range. Diathermy devices produce electromagnetic waves from either the shortwave or microwave frequency ranges. Lasers produce electromagnetic waves from the visible light frequency range. Much higher frequencies are used to produce ultraviolet light and x-rays for diagnostic purposes.

Electromagnetic waves of all wavelengths possess certain unique properties. They transport electrical and magnetic energy through space. Unlike sound waves, electromagnetic waves do not require a medium through which to travel and can travel unimpeded through a vacuum. Electromagnetic waves are composed of pure energy; therefore, they do not contain matter but do have an effect on the matter through which they travel. This occurs because matter contains electrical charges that interact with and are influenced by electromagnetic waves as they pass through the matter. The direction of propagation of radiant energy is normally a straight line. However, these waves of energy can be reflected, deflected, and absorbed by the media through which they travel.

Key Point! Regardless of the type of wave propagation, there is a fundamental relationship between frequency and wavelength, which is given by the equation $c = \lambda f$, where c is the speed of light, λ is the wavelength, and f is the frequency of an electromagnetic wave.[3]

All electromagnetic waves listed on the electromagnetic spectrum travel through space at the speed of light (3×10^8 m/s).[3]

The human body cannot detect most electromagnetic waves. Skin molecules can detect infrared waves because they resonate at infrared frequencies, resulting in absorption of energy and warming of the skin. The warming of the skin by the sun's rays is attributed to the infrared radiation produced by the sun. The eyes can detect wavelengths between 4 and 7×10^{-7} m, which is the visible light range of the electromagnetic spectrum.[3] Electromagnetic waves with frequencies higher than visible light, such as ultraviolet and x-rays, are not detected by the body and may be harmful to human tissues. A common example is sunburn, caused by damage to the skin from prolonged exposure to ultraviolet radiation from the sun.

Key Point! The human body cannot detect most electromagnetic waves. However, these waves can cause physiological effects as they travel through the body's tissues.

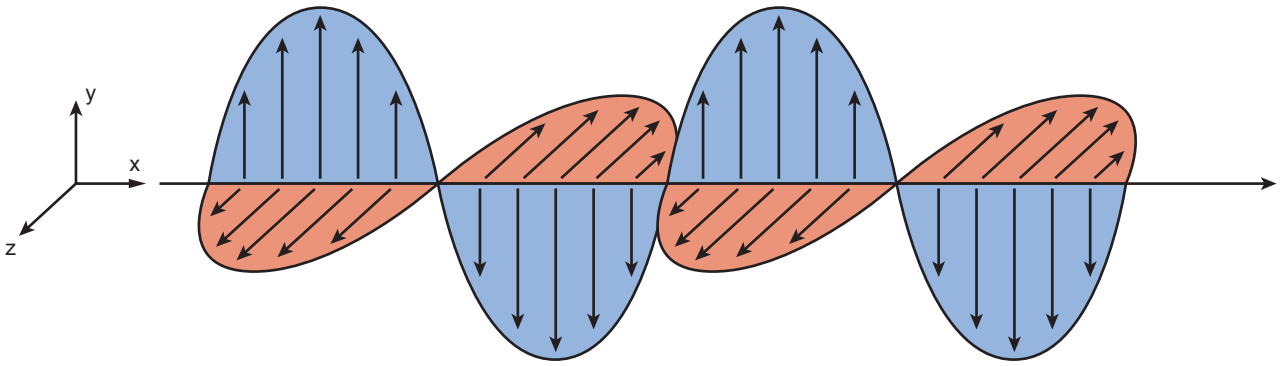

Fig 6 ■ 1 Alternating electric and magnetic fields surrounding an electromagnetic wave. Note that the electric and magnetic fields are perpendicular to each other and to the direction of the wave. *(Art concept by Sara Monath.)*

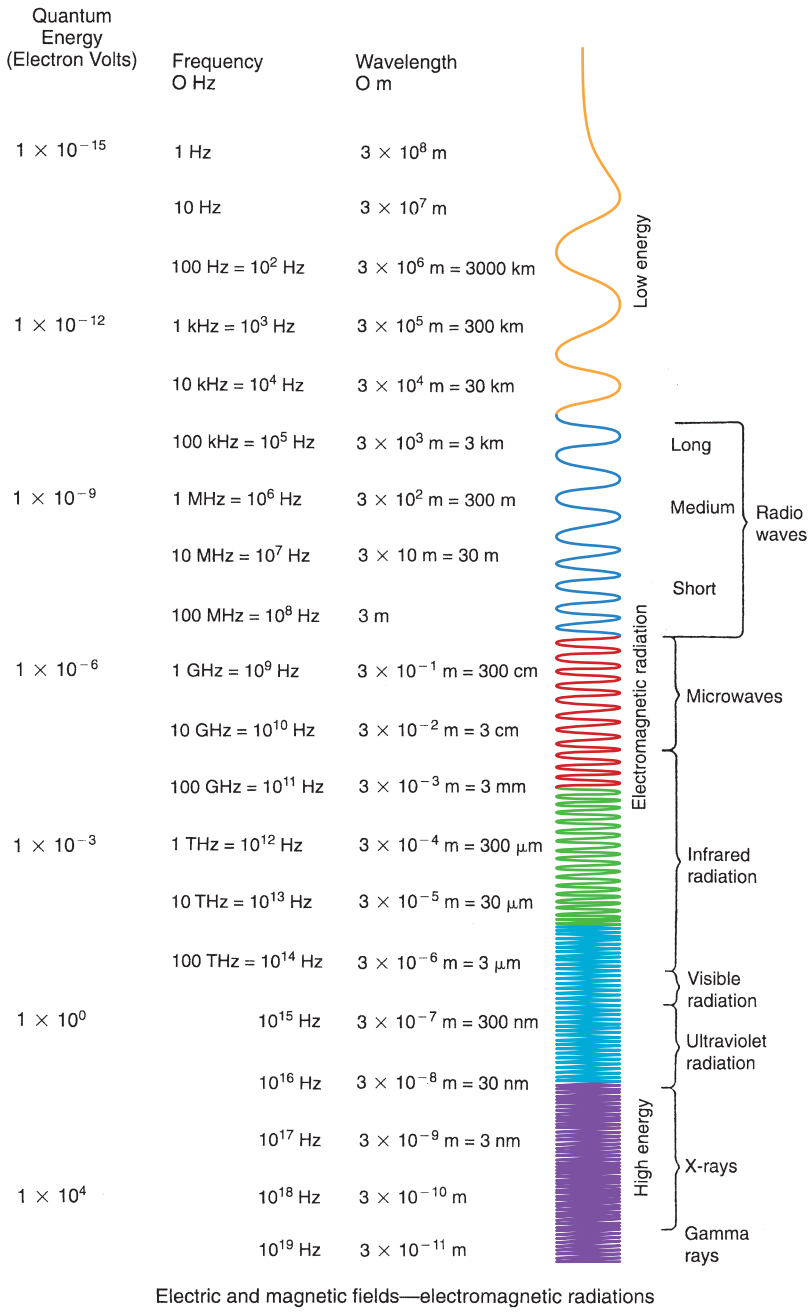

Fig 6 ■ 2 Electromagnetic spectrum of radiations organized by quantum energy, wavelength, and frequency. *(Adapted from Low J, Reed A, eds.* Electrotherapy Explained: Principles and Practice*. Oxford: Butterworth-Heinemann; 1992, p 230.)*

LIGHT THERAPY

The contemporary use of light in rehabilitation includes ultraviolet (UV) light, light-emitting diode (LED), supraluminous light-emitting diode (SLED), and laser. Types of lasers used by rehabilitation professionals include both class 3b low-power (500 milliwatts or less) and class 4 high-intensity lasers (greater than 500 milliwatts).

Ultraviolet light is one of several biophysical agent modalities available to clinicians, including physical therapists, to facilitate the healing of chronic skin wounds. In particular, the ultraviolet C (UVC) band of UV light has bactericidal effects in addition to other photobiostimulatory properties that may facilitate skin wound healing.

Key Point! Photobiostimulation is the application of light energy to biological structures, including tissues, cells, and subcellular components, to produce changes in their structure or function via nonthermal effects of light.

Laser

Laser is an acronym for "light amplification by stimulated emission of radiation." Theoretical foundations for the technology and use of laser light began in the early 1900s. Physicists and engineers developed the technology and instrumentation throughout the 20th century as applied use of laser evolved in various fields of engineering, manufacturing, and medicine.[4] As early as the 1970s, laser was being used by health-care providers, including physical therapists, in the United States, Europe, and Asia. The applications of laser in medicine vary widely—from surgical use of high-intensity thermal lasers for cutting and cauterizing tissues to low-intensity nonthermal lasers used by physical therapists for healing wounds and treating painful and inflammatory neuromusculoskeletal conditions.[5,6]

State of Events

The history of laser in rehabilitation as practiced in the United States is variable in its popularity and use. In the early 2000s, the U.S. Federal Drug Administration (FDA) cleared low-level infrared laser devices for the treatment of carpal tunnel syndrome.[7] This action stimulated a renewed interest in laser therapy, a proliferation of laser devices, and a wider incorporation of laser as a biophysical agent modality into physical therapy practice. Presently, low-level (low power) or "cold" laser devices most commonly available to physical therapists include helium-neon (HeNe) and infrared lasers, such as gallium-aluminum (GaAl) and gallium-aluminum-arsenide (GaAlAs) lasers. These lasers are generally considered nonthermal (or nonheat) producing, although a newer generation of high-intensity laser therapy devices has recently become available to physical therapists. These devices are suggested to have some thermal properties associated with their application.[8]

History

Following Albert Einstein's early theories of stimulated light emission to create concentrated light, American physicist Theodore Maiman produced the first laser device in 1960 using a ruby crystal. Ali Javan, also an American physicist, soon followed with the development of a helium-neon gas laser device.[4] A wide variety of lasing media, power outputs, and types of devices, including the development of semiconductor laser diodes, quickly evolved. Laser technology was refined for use in various industries, including manufacturing and medicine. The higher-power output lasers were effective for precise cutting of materials. Surgical lasers were likewise found to be effective alternatives to bladed scalpels, with additional benefits of simultaneous cauterization of bleeding tissues during laser surgery.[4] In the late 1960s, Hungarian physician Endre Mester discovered that low-power laser application had demonstrable effects on biological tissues that were solely due to the biostimulatory effects of the unique light properties of laser. In the early 1970s, Mester went on to show that low-power laser was beneficial in the treatment of chronic skin wounds.[9,10]

Low-power output or low-level laser therapy (LLLT) has been in widespread use in Europe and Asia since the 1970s. In the United States, the use of low-level laser in physical therapy and rehabilitation was sporadic and limited until 2002, when the FDA cleared low-level laser use in the treatment of carpal tunnel syndrome. Low-level laser and recently marketed high-intensity lasers are currently used by physical therapists to treat a wide variety of painful and inflammatory neuromusculoskeletal conditions. Figure 6-3 illustrates the use of LLLT for the treatment of carpal tunnel syndrome and the cervical spine.

Physical Properties of Lasers

Natural sunlight and white light from incandescent lightbulbs have wavelengths and frequencies that represent a broad range of the electromagnetic spectrum, including visible, infrared, and ultraviolet light. Natural and white light also widely scatter and attenuate when released from its source. Conversely, the technology and processes used to create laser light result in light production that has specific and unique properties that differentiate laser light from other light sources, notably a concentrated beam of light energy (Fig. 6-4).

The most common laser devices use semiconductor laser diodes (Fig. 6-5). A laser diode has various materials (lasing media) in its construction that give the produced

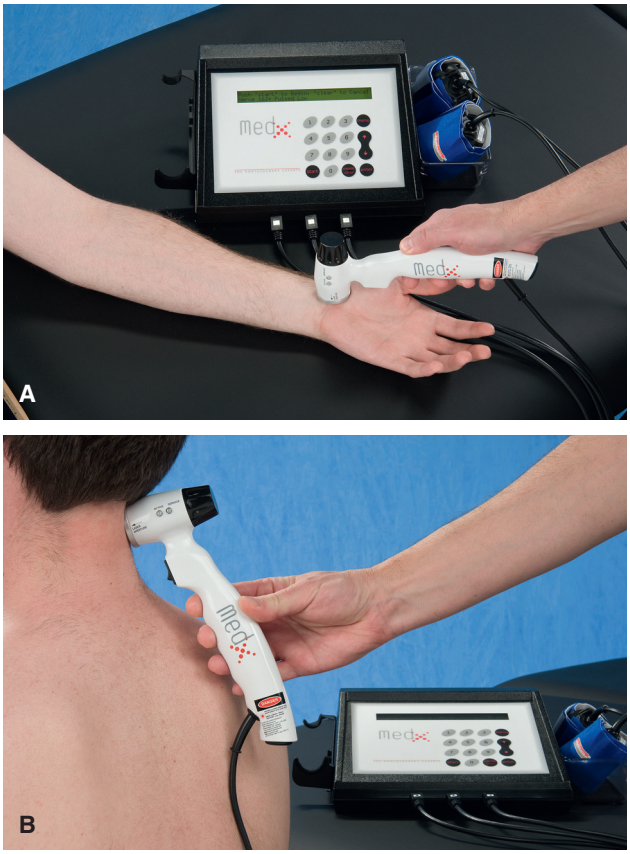

Fig 6 ■ 3 (A) Use of LLLT for treating carpal tunnel syndrome using a handheld wand. (B) Use of LLLT for treating cervical spine osteoarthritis using a handheld wand.

light unique wavelength and other output characteristics. The lasing media may include helium and neon (HeNe lasers), gallium and arsenide (GaAs lasers), and often other materials, including carbon dioxide (Co_2 lasers) and neodymium, yttrium, aluminum, and garnet (Nd-YAG lasers).[11,12]

Considering the acronym LASER, stimulated emission occurs within the laser diode, eventually leading to light amplification. This occurs when electrical energy is applied to the resting atoms of the laser media, causing the atoms' electrons to achieve a heightened state (Fig. 6-6). When this heightened state of the electrons decays, the electrons emit photons of light energy that

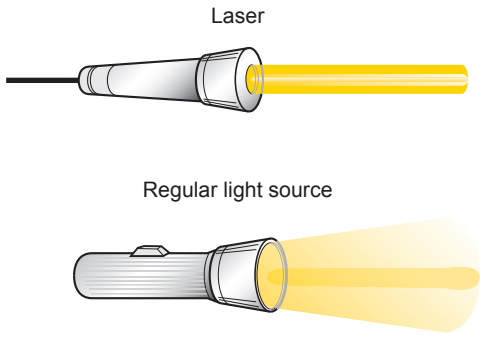

Fig 6 ■ 4 Schematic comparison of laser light to ordinary light. Laser is coherent while regular light is scattered.

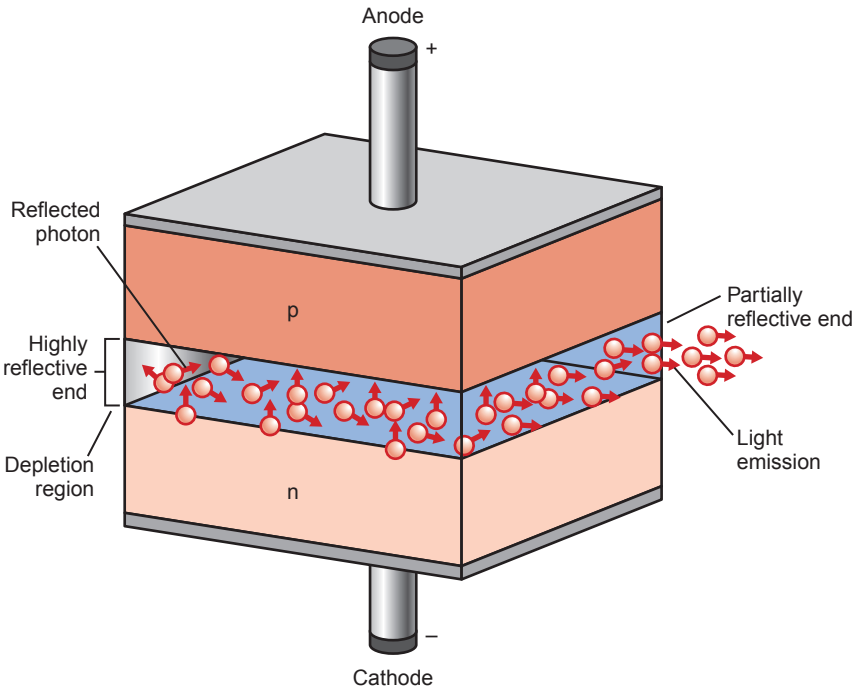

Fig 6 ■ 5 Laser semiconductor diode. Anode and cathode are components of the electrical source that stimulates electrons originating from lasing materials in the *n* region of the diode. Stimulated photons of light energy are amplified in the middle region between the highly and partially reflective ends of the diode and then released as emitted light into the environment. Note: *n* refers to negatively charged semiconductor material, and *p* refers to positively charged semiconductor material.

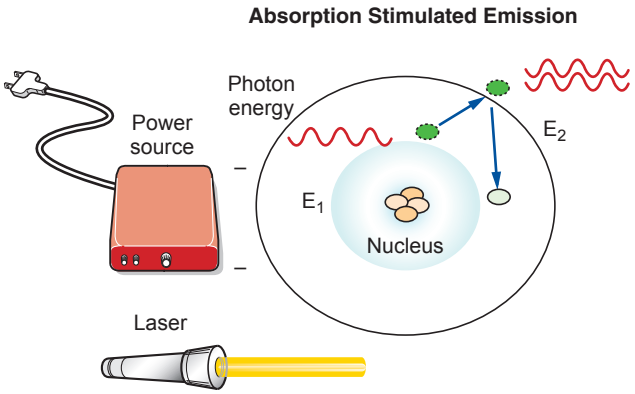

Fig 6 ■ 6 Stimulated emission. If a photon strikes an electron in an upper quantum, a second photon of equal wavelength as well as temporal and spatial orientation is produced. Subsequent collisions of photons with excited electrons amplify the photons, yielding laser light.

strike other stimulated atoms and combine with the photons released from those other decaying electrons. The chain reaction continues, leading to multiple photons traveling in phase with each other. The presence of reflective and semireflective mirrors within the diode facilitates the resulting amplification of light energy. This concentrated light energy may then be released into the environment or applied to biological tissues.

This process of laser light production leads to the properties of coherence and monochromaticity. Coherence is a property that is unique to laser light. *Monochromaticity* means that the light produced is a single wavelength, expressed in nanometers (nm) that may be within the infrared (invisible) region of the electromagnetic spectrum or may be visible as a specific color. Examples are the GaAs 904-nm infrared laser that produces invisible light and the HeNe 632.8-nm laser that produces red light. The particular lasing media used in the semiconductor diodes is related to the specific wavelength of the laser light that is produced. Therefore, each laser diode produces light of a single wavelength positioned within the electromagnetic spectrum (Fig. 6-7).

Coherence has two components: temporal coherence and spatial coherence (see Fig. 6-6). In temporal coherence, stimulated emission of electrons of a lasing medium leads to photons of light energy that are in phase with each other as they travel in a wavelike manner. Spatial coherence results when the laser light is subjected to a combination of reflecting mirrors and focusing lenses within the laser device that allows the light to be further amplified and concentrated into a beam. Therefore, the property of coherence requires that the light be monochromatic because coherence occurs when the dimensions of the phases of the many photons of light are the same.

The properties of monochromaticity and coherence of laser light allow the light energy to be concentrated, giving the laser light the appearance of a beam of light (collimation). This gives the laser light directionality—that is, in general, laser light beams can travel greater distances in a single direction with less divergence than sunlight or incandescent light (see Fig. 6-5). When applied to biological tissues, the amount of laser light energy absorption, and therefore depth of penetration through the skin without absorption, will vary, depending on the wavelength of the light. For example, HeNe (632.8 nm) red laser light will have most of the light energy absorbed within the first 2 to 3 millimeters of skin depth compared with GaAs (830 nm) infrared laser light that may penetrate up to 2 to 4 centimeters with less absorption.[11]

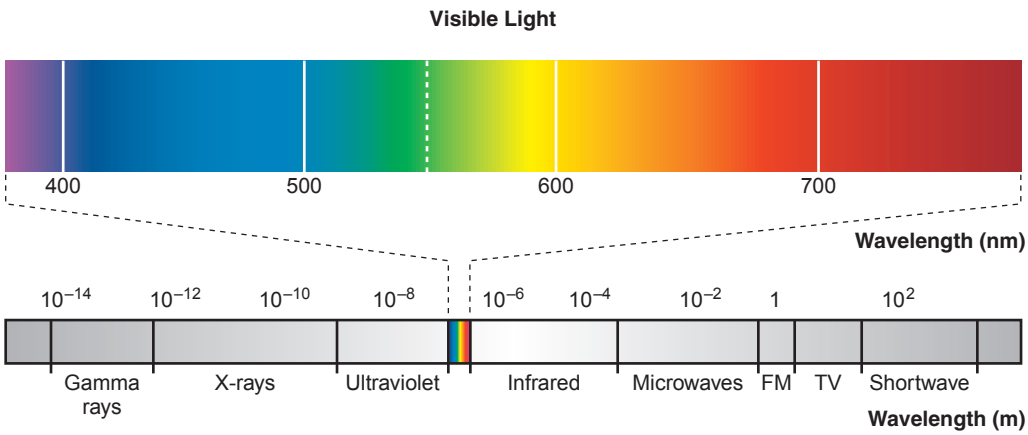

Fig 6 ■ 7 Electromagnetic spectrum. Comparison of position and ranges of visible light and infrared laser wavelengths.

Key Point! The amount of laser light energy absorbed by biological tissues depends on the wavelength of the light.

The power output of a laser device is measured in watts (W) or milliwatts (mW), which is 10^{-3} watts. A watt describes the amount of energy released by the laser per second, expressed as joules per second (J/sec). Conversely, a joule is the product of the power output (W) and the time of laser irradiation (seconds). Therefore, J = W × time (sec). Laser energy density may also be determined by knowing the surface area of the applicator probe tip and would be measured in J/cm^2. Laser dosimetry is often measured as total joules per treatment session or in joules per treatment point.

Physiological Effects of Lasers

The photobiostimulatory effects of laser are associated with the wavelength of the produced light because of specific cellular components that absorb certain wavelengths of light energy.[13] The amount of light energy absorbed by cells will also help determine the photobiostimulatory effects. In general, the result is the alteration, facilitation, or inhibition of chemical reactions that are not the effect of thermal stimulation.[13,14]

Chromophores are photosensitive molecules within the membranes of cells and organelles that are sensitive to specific wavelengths of light. For example, infrared wavelengths between 700 and 1,200 nm are absorbed by mitochondrial cytochromes. Wavelengths between 400 and 700 nm, notably visible red light, are absorbed by myoglobin and hemoglobin.[15,16]

Tissue Repair

Mitochondrial chromophore stimulation has been shown to increase respiratory chain activity, enhance adenosine triphosphate (ATP) synthesis, contribute to cellular repair and reproduction, and increase production of messenger RNA (mRNA) that codes for pro-collagen.[15] Increased human osteoblastic in vitro proliferation with HeNe laser has been observed.[17] Animal studies have shown increased neuronal sprouting and migration in cultured rat brain with infrared laser and increased fibroblastic growth in chicken culture (in vitro) in the presence of high glucose levels with green laser light.[18]

Inflammation

Infrared laser was reported to increase vasodilation due to increased histamine and nitric oxide levels, resulting in decreased tissue ischemia and increased perfusion, transport of nutrients, and removal of cellular debris.[19] Infrared laser was also found to decrease prostaglandin E_2 (PGE_2) in human subjects with Achilles tendonitis.[20] PGE_2 is a chemical mediator that facilitates the inflammatory process. Animal studies found that HeNe laser also reduced PGE_2 synthesis following induced acute inflammation in rats.[21] Laser light in the visible range with a wavelength of 650 nm decreased inflammatory cell (neutrophil) migration in induced mouse pleurisy.[22]

Pain

Low-level laser has been shown to modulate various chemical mediators of pain, including pain associated with inflammation. Infrared laser has been reported to increase levels of beta-endorphin—an endogenous opiate. Infrared laser was also observed to decrease plasma levels of bradykinin and kallikrein.[23] Bradykinin and kallikrein are proinflammatory mediators that also facilitate pain transmission. Suppression of depolarization of C-fiber afferent pain nerves and decreased motor and sensory nerve conduction and amplitude were observed following infrared laser irradiation.[24]

Instrumentation and Clinical Application of Lasers

Laser devices currently used in rehabilitation settings include low-level lasers and high-intensity lasers (HILT). Low-level lasers are defined as those producing a maximum peak power output equal to or less than 500 mW. High-intensity lasers produce a peak power greater than 500 mW. In addition, lasers are classified according to the risks from exposure to laser light that are associated with power outputs and wavelengths. The FDA has accepted a system of laser device labeling that is based on the dangers of retinal and tissue injury from laser light exposure (Table 6-1). For example, a class 3a laser is limited to 5 mW of maximum power output, and direct eye exposure to this laser for more than 2 minutes can cause serious retinal damage. Class 2 and 3a lasers are used in laser pointers. Low-level lasers used in rehabilitation are commonly class 3b lasers. Class 4 lasers include

TABLE 6–1. Laser Classification System

Class	Power	Standard Qualifications
1	≤ 0.5 mW	Safe for any exposure time with intended use of device
2	≤ 1 mW	Limited to visible light (laser pointers); momentary viewing within limits of blink reflex
3a	1–5 mW	Momentary viewing without eye damage (laser pointers)
3b	5–500 mW	Hazardous to the eye if viewed directly
4	> 500 mW	Can cause skin burns and permanent eye injury from direct and indirect viewing

high-intensity thermal lasers with power outputs exceeding 500 mW. These lasers are used for cutting and cauterizing tissues, with power outputs in the hundreds and thousands of watts. Most laser devices, including low-level and high-intensity lasers, have continuous and pulsed modes of light application. Laser instrumentation may include single or multiple (cluster) laser diode devices. Diode clusters may incorporate diodes of the same wavelength or multiple wavelengths.

Key Point! Low-level lasers used for therapeutic treatments are classified by the FDA as class 3b lasers. They require the patient and the clinician to wear eye protection to prevent retinal damage.

HeNe lasers used in rehabilitation have a wavelength of 632.8 nm. Typically most of the light energy generated by these lasers will be absorbed superficially within 0.5 cm of the skin surface point of application. HeNe lasers may be more appropriate for the treatment of chronic skin wounds. Infrared lasers with longer wavelengths—for example, 830 to 904 nm—have a deeper penetration, with the majority of light energy absorbed within 2 to 4 cm of the skin surface.

Low-level laser therapy relies on mechanisms of nonthermal photobiostimulation for the desired clinical effects.[9,10,12] Analysis of clinical research suggests the effectiveness of low-level lasers may depend on specific ranges of dosages applied to injured or inflamed tissues and structures. Currently evolving clinical guidelines recommend dosages for effective laser use for several types of painful inflammatory conditions. For example, the World Association for Laser Therapy provides dosing recommendations for low-level laser wavelengths based on its analysis of clinical laser research trials.[25]

Laser dosimetry may be calculated in joules per treatment point, total joules per treatment session if there are multiple treatment points, and total joules per course of treatment. For continuous mode laser application, dosage in total joules is calculated by multiplying the power output of the device (W) by the time (sec) of irradiation. For example, using a 30-mW continuous peak power output device applied to a treatment point for 30 seconds, the dosage is 0.9 joules (0.03 W × 30 sec = 0.9 J). If the device has three identical diodes in a cluster applicator probe applied to a treatment site, the total dosage is 2.7 J (0.9 J × 3 = 2.7 J).

The applicator probe has an aperture for each diode that allows the release of laser light into the environment. When in contact with the skin, the area of each diode aperture will equal the area of irradiation (spot size), which will determine the energy density (fluence) delivered by that diode. Therefore, the dose measured in joules/point will be known. When in continuous mode of application, energy density (J/area) is calculated by dividing the joules (W × sec) by the area of laser irradiation. For example, 0.9 J applied in direct skin contact with a 1-cm^2 diode aperture tip represents an energy density of 0.9 J/point. If the tip area is 0.5 cm^2, the energy density is 1.8 J/point. Therefore, although the energy density of a device may be reported as J/cm^2, the actual diode tip area should be known to accurately determine the energy density when the probe is in direct skin contact. The total joules per treatment session will be the sum of joules for all irradiation points.

When laser is applied in the pulsed mode, the total energy applied is determined by first calculating the mean power (P_m) output of the laser diode. This is found by multiplying peak power output (W) by the pulse frequency (Hz) by the pulse duration (sec). The total energy equals the mean power multiplied by the time of irradiation in seconds (Total energy = P_m × sec). The energy density is the total energy divided by the area of irradiation. For example, for a single diode laser that provides a peak power of 30 mW with a pulse rate of 1,000 Hz and a pulse duration of 300 microseconds, the mean power is 9 mW (i.e., 0.03 W × 1,000 Hz × 0.0003 sec). For a treatment time of 30 seconds, the total energy is

0.27 J (i.e., 0.009 W × 30 sec). If the area of the probe tip is 0.5 cm^2, the energy density with direct skin contact application is 0.27 J/point.

Key Point! Calculation of the total energy delivered to a patient by pulsed low-level laser is the mean power (peak power output × pulse frequency × pulse duration) multiplied by the time of irradiation of the laser to the skin.

After a dosage has been determined to treat a particular condition or structure, the laser application parameters may need to be calculated based on the power output of the device, whether a continuous or pulse mode will be used, and the number of diodes to be activated. Some laser devices simply require entering the desired dosage, and the laser delivery parameters will preset. There is insufficient clinical research evidence at this time to know whether using pulsed or continuous modes of laser application to arrive at the desired dosage provides better outcomes.

Techniques of laser application include direct skin contact over the structures to be treated. For larger areas, a grid may be used to ensure uniform treatment requiring multiple points of irradiation. When low-level laser therapy is used to treat chronic skin wounds, a grid may be used over a transparent protective barrier to avoid direct contact with an open wound bed. Alternatively, the applicator probe may be kept off but within 1 cm of the wound surface using either a stationary grid technique or slowly moving the probe in a waving manner.

In contrast with low-level laser devices with peak power outputs below 500 mW that typically do not heat tissues or have thermal effects, high-intensity class 4 lasers used by physical therapists with peak power outputs of up to 10 to 15 W have been described as likely causing thermal effects.[8] The limited clinical research studies using class 4 HILT devices report dosages in joules that far exceed the recommended dosing ranges based on clinical trials reported in the low-level laser therapy research literature.

Techniques for applying HILT consider the inherently higher-energy density due to high power output; therefore, the applicator must be moved continuously over a larger area of skin surface, similar to the application of ultrasound (Fig. 6-8).

Fig 6 ■ 8 Technique for HILT to the posterior shoulder.

Indications for the Use of Lasers

Based on the preponderance of clinical research, the two major categories for laser treatment are pain and inflammation associated with musculoskeletal conditions and chronic skin wounds. The great majority of clinical trials evaluate the effects of LLLT, with relatively few studies testing the clinical effects of HILT. The following descriptions of laser applications are based on a review of relevant randomized controlled clinical trials and systematic literature reviews (Tables 6-2 and 6-3).

Fibromyalgia and Myofascial Pain Syndrome

The effects of infrared laser therapy on fibromyalgia and myofascial trigger points were reported in several studies often with conflicting findings. In three separate trials, patients diagnosed with fibromyalgia received treatment

TABLE 6–2. Effective Applications of High-Intensity Laser

Condition	Outcome Variables	Reference/Type of Study
Temporomandibular dysfunction	Pain, mandibular AROM/PROM	Marini et al, 2010[68] (RCT)
Spine (low back)	Pain, functional measure	Fiore et al, 2011[70] (RCT)
Shoulder pain/ dysfunction	Pain, ROM, functional measure	Santamato et al, 2009[8] (RCT)

AROM = active range of motion; PROM = passive range of motion; RCT = randomized controlled trial.

TABLE 6–3. Ineffective Applications of Low Level Laser

Condition	Outcome Variables	Reference/Type of Study
Fibromyalgia/ myofascial pain syndrome	Pain, functional measure, AROM	Dundar et al, 2007[29] (RCT)
Spine (lumbar)	Pain, ROM, modified	Ay et al, 2010[33] (RCT)
TMJ	Pain, mandibular ROM	Melis et al, 2012[34] (SR) Petrucci et al, 2011[35] (SR) Tengrungsun et al, 2012[36] (SR)
Shoulder pain/ dysfunction	Pain, AROM	Bal et al, 2009[44] (RCT) Dogan et al, 2010[45] (RCT) Vecchio et al, 1993[46] (RCT) Yeldan et al, 2009[47] (RCT)
Carpal tunnel syndrome	Pain, strength, functional measure NCV, nerve cross sectional area	Tascioglu et al, 2012[52] (RCT)
Achilles tendonitis	Pain, functional measure	Tumulty et al, 2012[54] (RCT)
Degenerative joint disease (knee)	Pain, functional measures, ROM	Toscioglu et al, 2004[58] (RCT) Bjordal et al, 2003[59] (SR)
Degenerative joint disease (hand)	Pain, functional measure, ROM	Meireles et al, 2010[60] (RCT)
Wounds	Wound size, pain	Lucas et al, 2000[64] (SR) Leclere et al, 2010[67] (RCT)

RCT= randomized controlled trial; SR = systematic review.

to tender points with 20-mW, 904-nm infrared GaAs laser and a 50-mW, 830-nm GaAlAs laser.[26–28] The patients received dosages of 2 J/point, 5 days per week for 2 weeks. The authors reported significant improvement for the laser-treated subjects in pain, movement, and quality-of-life measures. Two studies reported contradictory findings when using infrared laser to treat active trigger points associated with myofascial pain syndrome. Dundar et al[29] found no significant differences in pain and mobility after treatment with 830-nm GaAlAs laser 5 times per week for 3 weeks with patients receiving dosages of 7 J/point and a total of 42 J/session. Conversely, Gur et al[26] reported improvement in pain and mobility using a 20-mW, 904-nm GaAs laser 5 times per week for 2 weeks with dosages of 2 J/point applied to cervical and shoulder trigger points. Therefore, it appears that infrared laser is most effective in treating trigger point and tender point pathology of the cervical region with dosages of 2 J/point, 5 days a week for 2 weeks.

Painful Conditions of the Spine

Studies by Konstantinovic et al[30] and Chow et al[31] concluded that infrared laser provides significant improvement in pain, mobility, and functional measures for individuals with acute and chronic cervical spine pain. Konstantinovic et al used a 905-nm, 12-mW laser to painful areas with dosages of 2 J/cm^2 per point, totaling 12 J/session, 5 times per week for 3 weeks. In two studies, Chow et al[31,32] treated painful cervical spine areas with an 830-nm, 300-mW laser with dosages of 9 J/point, 2 times per week for 7 weeks. Conversely, Ay et al[33] used an 850-nm laser with dosages of 40 J/cm^2, 5 times per week for 3 weeks and found no significant benefits from laser use in treating acute and chronic low back pain. These findings are consistent with the positive outcomes described above regarding the benefits of laser in the treatment of cervical pain associated with fibromyalgia and myofascial pain syndrome. Similarly, lower dosages of 2 J/cm^2 and 9 J/point consistently provide better outcomes compared with higher dosages of 40 J/cm^2.

Temporomandibular Dysfunction and Pain

Systematic reviews of the literature provided by Melis et al,[34] Petrucci et al,[35] and Tengrungsun et al[36] concluded that LLLT for temporomandibular dysfunction and pain either provides no benefits or that there is insufficient evidence to recommend LLLT for this condition. Two recent trials provided equivocal results supporting the conclusions of the systematic reviews. Da Silva et al[37] reported that use of a 70-mW, 780-nm infrared laser

with dosages of 52.5 J/cm^2 and 105 J/cm^2 resulted in significant improvement in pain relief, mandibular protrusion, and lateral movement but no improvement in pain with opening of the jaw. Ahrari et al[38] found no improvement in pain and jaw movement in patients treated with 80-mW, 810-nm pulsed infrared laser with dosages of 3.4 J/cm^2.

Shoulder Pain and Dysfunction

Clinical trials done to determine the effects of LLLT on conditions of the shoulder suggest that there is insufficient evidence to support the use of LLLT. This appraisal is consistent with systematic literature reviews reported by Michener et al[39] and Thornton et al.[40]

Abrisham et al[41] reported significant improvement in pain and mobility in patients diagnosed with subacromial syndrome when treated with an 890-nm laser, 2 to 4 J/cm^2, 5 times a week for 2 weeks. Nonspecific shoulder pain and decreased mobility significantly improved as reported by Bingöl et al,[42] who used a 50-mW, 904-nm GaAs laser with dosages of 1.61 J/cm^2 per point, 5 times per week for 2 weeks, and by Stergioulas,[43] who used an 810-mW GaAlAs laser with a dosage of 1.8 J/cm^2 2 times per week for 4 weeks and then once a week for 3 weeks. Conversely, no significant changes were found in pain and mobility measures following LLLT for rotator cuff tendonitis, subacromial impingement, and nonspecific shoulder pain by numerous authors when using infrared lasers with dosages ranging from 1.6 to 7 J/cm^2 with similar treatment protocols of 2 to 5 times per week for 2 to 8 weeks.[44–47]

Lateral Humeral Epicondylitis

In a systematic literature review and meta-analysis, Bjordal et al[48] concluded that LLLT with 904-nm wavelength provided short-term improvement of tennis elbow with dosages of 0.25 to 1.2 J/point, treating 2 to 6 points at the painful tendon origin. In a clinical trial, Emanet et al[49] reported significant improvement in pain, grip strength, and functional measures in subjects treated with a 100-mW, 905-nm laser with a dosage of 1 J/cm^2 5 times per week for 3 weeks.

Carpal Tunnel Syndrome

Findings from clinical trials evaluating the use of LLLT to treat carpal tunnel syndrome (CTS) are equivocal. Studies with positive outcomes concluded that those with uncomplicated mild to moderate CTS were effectively treated with LLLT with infrared wavelengths from 780 to 904 nm and with dosages ranging from 6 to 13.5 J/point, 2 to 5 points per session directly over the carpal tunnel, 5 times per week for 2 weeks. Ekim et al[50] reported reduced pain and improved functional ability posttreatment and at 3 months, follow-up for patients treated with a 50-mW, 780-nm laser, receiving 6 J/point at 5 points over the carpal tunnel, 5 times a week for 2 weeks. Chang et al[51] used a 30-mW, 830-nm laser to deliver 9.7 J/cm^2 per point and reported improved pain, grip strength, and function at a 2-week follow-up. Tascioglu et al[52] reported no significant improvement in pain and function in patients treated with 50-mW, 830-nm wavelength laser, receiving 1.2 J/point at 5 points over the carpal tunnel, 5 times per week for 3 weeks. The significant difference in dosage ranges between trials with positive outcomes (6 to 13.5 J/point) and those with negative outcomes (0.9 to 1.2 J/point) may account for the differences, supporting previous observations that LLLT effectiveness may be related to applied dosages for a given diagnosis or structure.

Achilles Tendonitis

There is limited and conflicting evidence from studies that tested the effects of LLLT in the treatment of Achilles tendonitis. Stergioulas et al[53] reported on the combined effects of LLLT and exercise and found that the use of a 60-mW, 820-nm laser applied to the Achilles tendon, at 0.9 J/cm^2, 5.4 J/session, significantly reduced ankle pain. Conversely, Tumulty et al[54] found no significant improvement in pain and functional measures when combining exercise with a 100-mW, 810-nm laser at 3 J/cm^2, 18 J/session, 3 times per week for 4 weeks.

Degenerative Joint Disease

The majority of clinical trials evaluating the effects of LLLT on degenerative joint disease (DJD) involved patients with knee pathology. The findings and conclusions of clinical trials are often contradictory. In separate studies, Alfredo et al,[55] Hegedus et al,[56] and Gur et al,[57] reported that LLLT application ranging from 20 to 50 mW with wavelengths ranging from 830 to 904 nm was effective in improving pain, mobility, and function

in patients with knee osteoarthritis. Dosages ranged from 2 to 6 J/cm^2 and 20 to 48 J/session, with treatment frequencies ranging from 2 to 5 times per week for 2 to 4 weeks.

Conversely, Toscioglu et al[58] reported no improvement in outcome measures in patients with knee DJD due to osteoarthritis when treated with a 50-mW, 830-nm laser at both 1.5 J/cm^2 and 3 J/cm^2, 5 times per week for 2 weeks. In a related systematic review and meta-analysis, Bjordal et al[59] concluded that there are conflicting results regarding the use of LLLT for treatment of knee osteoarthritis. Meireles et al[60] reported on the effects of LLLT in the treatment of DJD of the hand due to rheumatoid arthritis. The authors found that application of a 70-mW, 785-nm laser, at 3 J/cm^2 per point, 2 times per week for 8 weeks did not improve pain, mobility, or function.

Wound Healing

As early as 1971, initial clinical applications of LLLT were proposed by Mester[61] to treat open skin wounds.[9] Since then, numerous human and animal studies, both in vivo and in vitro, were conducted to test the effectiveness of various wavelengths of LLLT on physiological processes associated with wound healing. A systematic review of basic science literature examined proposed physiological mechanisms and the effectiveness of LLLT relative to these mechanisms that would account for improved wound healing. The authors concluded that the mechanisms that would be affected by LLLT were not verified, and the outcomes of LLLT application in both in vivo and in vitro models were inconsistent.[62] A later review of related basic science research that employed animal models concluded that LLLT within the visible red and infrared wavelength spectrum improved rates of wound healing with mean dosages of 4.2 J/cm^2.[63]

The results of clinical research trials on humans are also variable. A systematic review of research conducted by Lucas et al[64] concluded that infrared LLLT was not effective in the treatment of decubitus ulcers, venous leg ulcers, and chronic wounds. More recent systematic reviews and meta-analyses by Enwemeka et al[65] and Woodruff et al[66] reported that LLLT may be effective in assisting closure of skin wounds. Based on reviews of recommended treatment protocols, LLLT wavelengths in the visible red and infrared range at energy densities between 4 and 12 J/cm^2 may be most effective for wound management. This is consistent with the overarching observation that LLLT effectiveness in general is dependent on specific dosage ranges, with dosages that are too low or high being ineffective and higher dosages possibly being harmful. For example, Leclere et al[67] reported that application of a 15-W infrared laser at a dosage of 90 J/cm^2 in patients with venous leg ulcers was no more effective than placebo treatment.

Clinical Controversy

The clinical effectiveness of LLLT has not been well established through adequate randomized controlled clinical trials (RCTs). Although numerous RCTs appear in the published literature, experimental design, procedure issues, and inconsistencies across trials often lead to conflicting research findings and conclusions.

High-Intensity Lasers

By definition, high-intensity lasers currently used by physical therapists are class 4 lasers with peak power outputs above 500 mW. Class 4 "hot" lasers used for surgical procedures that require cutting or cauterization may have peak power outputs in excess of 300 W. In contrast, class 4 lasers used by physical therapists range from 10 to 15 W. In addition to expected photostimulatory effects consistent with LLLT, application of greater HILT dosages may also provide thermal effects.[8] Treatment protocols for HILT describe total dosages that are far in excess of recommended dosages for LLLT, suggesting that the photostimulatory mechanisms that require specific dosage ranges may not apply to HILT. Therefore, the thermal effects of these class 4 devices may account for the positive clinical outcomes reported in the relatively few RCTs available that used HILT.

Key Point! Unlike LLLT, positive clinical outcomes of HILT are likely caused by the thermal effects of these lasers.

Marini et al[68] reported improved pain and mobility in patients with temporomandibular joint (TMJ) dysfunction using a pulsed 45-W, 910-nm infrared laser that delivered a total of approximately 480 J/session to

the TMJ region. Panton et al[69] used a 10-W, 980-nm infrared laser with a dosage of 600 J/point for a total dosage of 4,200 J/session to active tender points in patients with fibromyalgia, resulting in significantly improved pain and mobility. Fiore et al[70] effectively treated patients with acute low back pain with an Nd-YAG pulsed laser with a mean power of 6 W, delivering a dosage of 2,600 J/session. Finally, Santamato et al[8] used the same laser device to treat patients with subacromial impingement syndrome, delivering 2,050 J/session and reporting significant improvement in pain and mobility. Possible thermal effects were suggested in this study.

Contraindications and Precautions for Lasers

Laser device manufacturers publish precautions and contraindications for their products. Clinicians must familiarize themselves with the technical information about these devices because of the wide variability among these products, including multiple diode clusters, different power outputs (including high-intensity devices), and units that combine biophysical agents such as laser with electrical stimulation. Tables 6-4 and 6-5 list precautions and contraindications for laser use.

A chief contraindication is direct exposure to the eye without protective eyeglasses. Laser classification systems are based on different laser light exposure times that could cause eye damage. For this reason, class 3b and 4 lasers typically require that appropriate goggles (often provided by device manufacturers) be worn by both the clinician and the patient.

TABLE 6–4. Precautions for Laser and LED/SLED

Precaution	Explanation
Impaired sensation	High-intensity lasers and SLED arrays may generate cutaneous or subcutaneous heat and require intact sensation sufficient for the patient to be aware of excessive heating.
Indirect eye exposure	Due to risk of retinal damage with laser, appropriate protective eyeglasses for the therapist and patient are required.

TABLE 6–5. Contraindications for Laser and LED/SLED

Contraindication	Explanation
Direct eye exposure	Retinal damage due to concentrated laser light energy
Pregnancy	Avoid treatment of the low back and pelvic/abdominal region during pregnancy because the effects of any light therapy during pregnancy are unknown.
Active malignancy	Avoid treatment over areas of active malignancy because known molecular, cellular, metabolic, and circulatory effects of laser and LED/SLED application may exacerbate malignancies.
Active hemorrhage	Avoid treatment over areas of abnormal bleeding because increased circulatory response and vasodilation with laser and LED/SLED application may increase hemorrhage.
Open growth plates	Avoid use of laser and LED/SLED over open growth plates because exposure may interfere with active growth plates in children due to the general effect of increased metabolic and circulatory activity.
Endocrine system	Avoid exposure of endocrine glands, notably the thyroid gland in the anterior region of the neck, because changes in circulating levels of related hormones may occur.

Observed and documented adverse effects of many treatments are often not known when establishing general contraindications. However, conservative rationales to protect the safety of potentially vulnerable patients are the norm. Contraindications for laser include treatment over the low back, pelvic, and abdominal regions of pregnant women; diagnosis of malignancy; and treatment near or over the thyroid gland, over a hemorrhagic region, and over open growth plates in children. Precautions and contraindications for the application of superficial and deep heat should also be considered when using class 4 lasers due to the possible photothermal effects.

Light-Emitting Diodes

The reemergence of low-level laser technology and clinical applications in physical therapy in the early 2000s paralleled the development and availability of LED devices, whose proposed benefits are comparable to those of LLLT. Specific FDA clearance for the new LED devices, particularly infrared LEDs, was not required because of previous approvals of infrared lamps used by physical therapists for decades as a superficial heat modality. Infrared heat lamps have been largely replaced by moist heating pads and hot packs because of the increased risk of burns from the heat lamps.

Physical Properties of LED

LEDs can produce light that is monochromatic or polychromatic and includes visible and infrared light wavelengths. SLEDs are similar to LEDs but produce higher-intensity light energy. Laser diodes produce light that is monochromatic, coherent, and directional. In contrast, LEDs and SLEDs may be monochromatic but with limited coherence and directionality.[71] Therefore, the classification systems for laser devices do not apply to LEDs and SLEDs. The power output can range from 5 to 40 mW for LEDs and up to 90 mW for SLEDs. Typically, LEDs and SLEDs are arrayed in clusters or pads with as many as 20 or 30 diodes. Because treatment time is often longer, the total amount of light energy with clusters of LEDs can far exceed the dosages used during LLLT applications. The amount of light energy provided by LED and SLED light sources can produce superficial heating of the skin.[72]

Key Point! LEDs are similar to lasers because the light produced by both is monochromatic. However, the light emitted by LEDs has limited coherence and directionality.

Proposed Physiological Effects of LED

Studies have reported that the effects of light on cellular processes are the same for both laser and nonlaser light sources.[73,74] The effects of light on cells appear to be wavelength-dependent, assuming the necessary amount of light energy reaches and is absorbed by biological tissues.[73–75] Numerous in vivo and in vitro animal and human studies have demonstrated that LEDs affect cellular processes similarly to laser.[75–77]

The physiological effects of infrared light on biological tissue are believed to occur primarily by photochemical reactions rather than thermal effects. Leonard et al[78] found that infrared light treatments were more effective in improving sensation in patients with peripheral neuropathy than in patients who received placebo pads that emitted a comparable thermal effect. Various researchers reported that infrared LEDs decreased conduction velocities and evoked potentials in normal nerves, although others found that LEDs had no effect on peripheral nerves or that observed increases in conduction velocity may be due to superficial heating of the skin over the sensory nerve tested. Horwitz et al[79] reported that application of infrared light to the skin for 30 minutes increases plasma nitric oxide (NO). Infrared light releases NO from red blood cells and causes vasodilation and increased circulation in the treated tissues. NO increases vascular perfusion by dilating arterioles, resulting in enhanced tissue oxygenation, nutrient delivery, and removal of waste products of metabolism. Human blood lymphocytes irradiated with infrared light had an increased level of ATP in cells. These effects may explain the enhancement of wound healing in patients treated with infrared light. Promotion of circulation with resultant oxygenation in tissues treated with infrared light may stimulate nerve growth in patients with peripheral neuropathy.

Clinical Controversy

The light produced by laser technology and LED/SLED technology is similar in its characteristics, including a lack of coherence and collimation in nonlaser light. This has led to ongoing discussion about whether various sources of light produce the same clinical effects and proposed benefits. The majority of clinical research on light therapy in physical therapy and rehabilitation is limited to LLLT.

Indications for LED Therapy

Several studies on the effects of LED applications to open skin wounds reported generally favorable outcomes, including the treatment of chronic venous ulcers,

diabetic ulcers and wounds, and ulcers associated with scleroderma.[75–77,79] In contrast with the LLLT literature, there are relatively few RCTs that have evaluated the effects of LED therapy on painful, inflammatory musculoskeletal conditions (Tables 6-6 and 6-7).

Montes-Molina et al[80] reported significant improvement in pain and function in patients with nonspecific shoulder tendinopathy after treatment with a cluster of 7 LEDs (600 nm) and 12 SLEDs (950 nm) with a dosage of 10 J/cm^2 and a total of 168 J/session for 10 sessions. Neupane et al[81] observed decreased inflammation of arthritic joints treated with methotrexate and an LED array compared with methotrexate alone. Hsieh et al[82] used a 6.24-W, 830-nm LED cluster providing 41.6 J/cm^2 per session to patients with knee osteoarthritis and found no significant improvement in pain and function compared with placebo.

Research reports on the effects of LED therapy on peripheral neuropathy are conflicting in their findings. Several studies have reported improved sensation in patients with peripheral neuropathy who were treated with nonthermal infrared light.[83–86] However, Clifft et al[87] found that 30 minutes of infrared light applied 3 days per week for 4 weeks was no more effective than placebo in increasing sensation in subjects with diabetic peripheral neuropathy. A related study found that applying an array of LED/SLED to the radial nerve had no effect on nerve conduction characteristics.[88] The evidence from RCTs appears insufficient to support the use of LEDs for neurological and musculoskeletal conditions, although further clinical research is warranted.

Contraindications and Precautions for LEDs

Considering that the physiological effects of LEDs may be similar to those of laser diodes, the precautions and contraindications for LEDs would be consistent with those for laser described previously. Also, because the total wattage of LED and SLED cluster pads may cause heating of the skin and potential burns, precautions and contraindications associated with superficial heating modalities should also be followed (see Chapter 3).

Assessment of Effectiveness of Light Therapy

When establishing a clinical guideline, comprehensive reviews of the laser literature should be limited to

TABLE 6–6. Effective Applications of LED/SLED

Condition	Outcome Variables	References/Type of Study
Wounds	Wound size, pain	Fulop et al, 2009[75] (SR); Minatel et al, 2009[77] (RCT); Horwitz et al, 1999[79] (RCT)
Shoulder pain/dysfunction	Pain, ROM	Montes-Molina et al, 2012[80] (RCT)
Degenerative joint disease (RA)	Measurement of activated inflammatory lymphocytes	Neupane et al, 2010[81] (ICT)
Peripheral neuropathy	Sensation measure; sensation self-report	Prendergast et al, 2004[84] (WSD); DeLellis et al, 2005[85] (RRS); Powell et al, 2004[86] (QS)

ICT = in vitro comparison trial; QS = questionnaire survey; RA = rheumatoid arthritis; RCT = randomized controlled trial; ROM: range of motion; RRS = retrospective record survey; SR = systematic review; WSD = within subject design.

TABLE 6–7. Ineffective Applications of LED/SLED

Condition	Outcome Variables	References/Type of Study
Degenerative joint disease (knee)	Pain, functional measure	Hsieh et al, 2012[82] (RCT)
Peripheral neuropathy	Sensation measure	Clifft et al, 2005[87] (RCT)

RCT = randomized controlled trial

reports that provide the strongest evidence to support or refute the use of laser, namely RCTs and systematic reviews of these studies. Many randomized controlled trials have significant methodological flaws or the research reports provide insufficient information to judge the quality of the studies. Also, the wide variety of protocols to test the effects of laser makes comparison across trials difficult. These factors account for the conflicting conclusions often found in this literature. Systematic reviews and meta-analyses also may use different criteria for including and excluding trials. The World Association for Laser Therapy (WALT) recommends guidelines for conducting randomized controlled trials and systematic reviews to promote valid and comparable laser research data.[89] These or similar guidelines may assist in producing improved laser research designs that could be consistently applied. WALT also provides recommended dosage ranges for different LLLT wavelengths that may provide a basis for standardizing research protocols so that comparisons may be made between trials.[25] Examples of these dosing recommendations are found in Table 6-8.

Critical appraisal summaries of current literature suggest that for many painful, inflammatory conditions, there appears to be insufficient evidence to support the use of laser. However, there appears to be moderate confidence in evidence for the use of infrared LLLT to treat certain conditions (Table 6-9). Cervical tender points and trigger points associated with fibromyalgia and myofascial pain syndrome were effectively treated with infrared LLLT at dosages of 2 J/point. Also, there is moderate confidence in the evidence for using infrared LLLT at dosages of 1 J/point, at 4 to 6 points, to treat lateral elbow epicondylitis. Although the evidence is contradictory, patients with mild to moderate CTS without prior surgical intervention may be effectively treated with LLLT at dosages ranging from 6 to 13.5 J/point compared with lower point dosages. Reviews of evidence, notably the previously referenced

TABLE 6–8. Selected Dosage Guidelines (World Association for Laser Therapy)*

Condition	Number of Points or Areas (cm^2) of Irradiation	Total Joules Per Session	Notes
Tendinopathies			
Carpal tunnel syndrome	2–3	8	min 4 J/point
Lateral epicondylitis	1–2	4	max 100 mW/cm^2
Supraspinatus	2–3	8	min 4 J/point
Achilles tendon	2–3	8	max 100 mW/cm^2
Arthritis			
Temporomandibular joint	1–2	4	min 4 J/point
Cervical spine	4–12	16	min 4 J/point
Lumbar spine	4–8	16	min 4 J/point
Knee (medial)	3–6	12	min 4J/point

*Laser class 3b, 780–860 nm, GaAlAs lasers, continuous or pulsed, mean output: 5–500 mW; World Association for Laser Therapy (WALT), www.walt.nu.

TABLE 6–9. Treatment Guidelines for LLLT

Condition	Dosage	Frequency
Cervical tender/trigger points associated with fibromyalgia/myofascial pain syndrome	2 J/cm^2; 11–18 points	5 times/week; 2 weeks
Carpal tunnel syndrome	6–13 J/point; 2–5 points	5 times/week; 2 weeks
Lateral epicondylitis	1 J/point; 4–6 points	5 times/week; 3 weeks

systematic reviews, suggest that chronic skin wounds may benefit from LLLT at 4 to 12 J/cm^2. There is insufficient evidence for the use of HILT to treat musculoskeletal conditions. Yet, the findings from the limited numbers of clinical trials are encouraging and suggest that HILT may be beneficial, possibly due to photothermal and photobiostimulatory effects.

Additional RCTs are necessary to demonstrate the effectiveness of HILT. Laser literature is often cited in support of LED and SLED use. This is because of the relative lack of research trials, especially clinical research using LEDs and SLEDs. Conclusions that both laser light and LED light have the same or similar physiological effects contribute to this practice. Because reported dosages in the LED literature are often higher than those recommended based on LLLT trials, future clinical trials may be more meaningful in comparing LLLT and LED outcomes if dosages are in alignment.

Clinical Application of Laser

Preparation of the Patient and Device

The patient is examined to determine if laser treatment is an appropriate part of the plan of care based on the diagnosis. This requires knowledge of the physiological and desired clinical effects of laser as well as the precautions and contraindications. The clinician should then explain the treatment, procedure, and expected benefits to the patient, giving the patient the opportunity to decline. The steps for patient preparation and administration of laser treatment are described in Box 6-1.

Documentation

Proper documentation is essential for meaningful communication to others about patient management, including laser treatment. Recording the details of laser administration is necessary so that treatment parameters can be assessed for effectiveness and modified as necessary, particularly in the absence of strong clinical guidelines on laser application. In addition to documenting the details of laser application, it is necessary to document desired clinical outcomes/goals for the patient that are measureable and meaningful and the actual outcomes as the result of laser treatment (Box 6-2).

Box 6 ▪ 1 Preparation of Patient and Device for Laser Treatment

1. Select the appropriate infrared low-level laser device.
2. Describe the desired benefits to the patient, including how lasers work, any possible adverse effects, and what the patient can expect to experience during the treatment.
3. Position the patient appropriately, shielding the treatment area from others in the clinic.
4. Expose the skin of the area to be treated.
5. Provide protective eyewear to the patient.
6. Inspect the skin over the treatment area for open wounds, other lesions, and sensation.
7. Select the parameters on the laser device to provide the desired dosage to the intended points of application.
8. Don protective eyewear.
9. Position the laser applicator so it is in contact with the skin.
10. Activate the device.
11. Instruct the patient to inform the clinician if the treatment cannot be tolerated for any reason.

Box 6 ▪ 2 Clinical Outcome Measures for Laser Effectiveness

1. Active and passive range of motion
2. Manual muscle testing
3. Grip dynamometry
4. Pain (e.g., VAS, pain questionnaires)
5. Pressure algometry
6. Sensory testing
7. Nerve conduction and evoked response studies
8. Functional measures (e.g., Oswestry, Waddell disability indices, FIM, SF-36, DASH)

DIATHERMY

The term *diathermy* literally means to "heat through."[1] Historically, the term has been applied to electromagnetic or ultrasonic modalities that can heat subcutaneous deep tissues such as muscles and joints.[2] Today, diathermy refers to the use of electromagnetic waves from the radio frequency or microwave frequency ranges

CASE STUDY 6-1 Selecting LLLT for Myofascial Trigger-Point Pathology

A 45-year-old male presents with left upper trapezius pain of 3 weeks' duration. He states that he awoke one morning with neck and left upper shoulder pain that continues to be bothersome. He reports left upper trapezius pain to be 5/10 at rest, increasing to 8/10 upon palpation. Specific point tenderness is present in one location that elicits referred pain into the left scapula. Cervical active range of motion (AROM) is 45 degrees, rotation to the left and 80 degrees rotation to the right.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction or limitation that can be improved with the use of LLLT?
   ANSWER: Yes, signs and symptoms suggest left upper trapezius myofascial pain syndrome with trigger-point pathology.
2. Why would LLLT be considered for this patient?
   ANSWER: LLLT is indicated for the treatment of myofascial trigger-point pathology and is supported by moderately strong evidence for its use.
3. What specific outcome measures can be used to assess the effectiveness of LLLT?
   ANSWER: The pain rating scale (0–10 scale) can be used to measure pain at rest and with palpation and measurement of active cervical rotation and to assess improvement and the effectiveness of laser.
4. Are there any precautions or contraindications to consider with this patient?
   ANSWER: The patient's history did not include conditions such as active cancer or hemorrhage that would contraindicate LLLT. The treatment area is the left upper trapezius and is not in proximity to any endocrine glands. Both the patient and therapist will wear protective eyewear to prevent retinal damage caused by eye exposure.
5. What specific parameters are recommended for this patient?
   ANSWER: The best evidence for LLLT in treating upper trapezius myofasciitis suggests the use of infrared laser (830–904 nm) at 2 J/cm^2, 11 to 18 points of irradiation, 5 times a week for 2 weeks.
6. What are the proper steps for using LLLT with this patient?
   ANSWER:
   1. Inform the patient and describe the laser device in lay terms, noting that it is low-level laser and does not create heat.
   2. Position the patient in a comfortable sitting position with the left cervical and upper trapezius muscle area exposed.
   3. Calculate or enter the dosage parameters in the device.
   4. Shield the treatment area from others in the clinic.
   5. Don protective eyewear and provide eyewear to the patient.
   6. Apply the laser applicator to the skin over the tender/painful areas.
   7. Activate the laser for each treatment point.

CASE STUDY 6-2 Selecting LLLT for Carpal Tunnel Syndrome (CTS)

A 30-year-old female is referred to you with a diagnosis of carpal tunnel syndrome of the right hand. She works as a data input technician and reports that she has had hand pain, intermittent numbness, and slight grip weakness for about 4 weeks. She states that she is 6 weeks, pregnant and that her physician told her that the CTS is more likely associated with work-related repetitive use and not the pregnancy. Examination findings include general handache at rest that is rated at 7.5 on a visual analog scale (VAS); grip strength of 11 lb, measured with a grip dynamometer (right grip is 16 lb); and active wrist extension of 30 degrees limited by wrist pain.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction or limitation that can be improved with the use of LLLT?
   ANSWER: Yes, mild to moderate CTS with no history of prior surgery or recent steroid injection.

CASE STUDY 6-2 Selecting LLLT for Carpal Tunnel Syndrome (CTS)—cont'd

2. Why would LLLT be considered for this patient?
ANSWER: LLLT is indicated for mild to moderate CTS and is supported by moderately strong evidence for its use.

3. What specific outcome measures can be used to assess the effectiveness of LLLT?
ANSWER: The effectiveness of LLLT can be measured by improved range of motion (ROM) of the wrist; pain measurement scales and questionnaires; grip strength testing, including dynamometry; sensory testing; and functional use tools.

4. Are there any precautions or contraindications to consider with this patient?
ANSWER: Treatment of the low back or pelvic region during pregnancy is a contraindication, but treatment of the wrist during pregnancy is not. There is no history of active cancer or hemorrhage. Protective eyewear will be worn as a precaution to prevent eye exposure. Treatment of the wrist will not risk exposure of endocrine glands.

5. What specific parameters are recommended for this patient?
ANSWER: The best evidence recommends infrared LLLT, 6 to 13 J/point, 2 to 5 points over the carpal tunnel, 5 times a week for 2 weeks.

6. What are the proper steps to using LLLT for this patient?
ANSWER:
1. Inform the patient and describe the laser device in lay terms, noting that it is low-level laser and does not create heat.
2. Situate the patient in a comfortable sitting position with the right hand and wrist in supination, supported on a table, and with the skin exposed over the area to be treated.
3. Calculate or enter the dosage parameters in the device.
4. Shield the treatment area from others in the clinic.
5. Don protective eyewear and provide eyewear to the patient.
6. Apply the laser applicator to the skin on the points over the carpal tunnel.
7. Activate the laser for each treatment point.

of the electromagnetic spectrum. Biophysical agents that deliver ultrasonic waves are known as *ultrasound,* which is covered in Chapter 4.

Physical Principles of Diathermy

Diathermy devices deliver electromagnetic waves to the tissues of the body. The most common diathermy device in use today delivers 27.12 MHz frequency waves from the short wavelength radio wave section of the electromagnetic spectrum and is commonly referred to as *shortwave diathermy* (SWD). Devices that deliver electromagnetic waves from the microwave range of the electromagnetic spectrum are known as *microwave diathermy* (MWD). The frequency most commonly used for microwave diathermy is 2,450 MHz. In the United States, the Federal Communications Commission (FCC) has carefully regulated electromagnetic frequencies for television and radio transmission, radar, and medical applications. FCC-approved frequencies for diathermy are listed in Table 6-10.

Diathermy can be delivered continuously or through regular pulses or bursts of radio frequency energy. Pulsed

TABLE 6–10. Radio Frequencies Approved by the FCC for SWD/MWD and PEMF

Frequency (MHz)	Wavelength	Type of EM Radiation
13.56	22 m	SWD/PEMF
27.12*	11 m	SWD/PEMF
40.68	7.5 m	SWD/PEMF
915.00	33 cm	MWD
2450.00	12 cm	MWD

*Most widely used frequency for SWD and PEMF.
EM = electromagnetic.

shortwave diathermy (PSWD) uses a timing circuit to electronically interrupt the delivery of electromagnetic waves to the patient, resulting in bursts of pulse trains containing a series of high-frequency sine wave oscillations emitted from the treatment applicator. Each pulse train has a preset duration or "on time" and is separated from successive pulse trains by an "off time" that is determined by the pulse repetition rate or frequency (Fig. 6-9). Pulse frequency varies from 1 to 7,000 pulses per second (pps), depending on the device. Some devices allow the clinician to adjust the pulse frequency; in others, the frequency is set by the manufacturer. Devices that deliver radio frequency waves to the patient at a very low intensity are known as *pulsed electromagnetic fields* (PEMF) or *pulsed radio frequency energy*. The intensity of the waves delivered by these devices is too low to create heat in body tissues; thus, these devices are classified as nonthermal modalities.

The radio or microwave frequency waves produced by diathermy devices pass through the tissues of the body and initially are not detected. Gradually, absorption of energy by the tissues may increase tissue temperature, depending on the intensity of the waves and whether the waves are continuous or pulsed. Patients may perceive warmth as the tissue temperature rises. Generally, the closer the diathermy applicator is to the patient's skin, the greater the effect on the tissues.

Diathermy can affect deeper tissues of the body than superficial modalities, such as hot packs, because radio frequency waves can travel through superficial tissues. There is minimal reflection of waves at tissue interfaces and on bone, so there is little accumulation of energy at these interfaces as would occur with ultrasound (Box 6-3).

Box 6 ■ 3 Clinical Outcome Measures for Diathermy Effectiveness

1. Pain assessment (e.g., VAS, pain questionnaires)
2. Active and passive range of motion of joints
3. Strength testing of muscles (manual muscle testing, handheld dynamometers, isokinetic dynamometers)
4. Flexibility of muscles, tendons, and fascia
5. Functional measures (e.g., Oswestry, Waddell disability indices, FIM, SF-36, DASH)
6. Posture analysis
7. Relaxation scales
8. Depression scales (e.g., Beck Depression Inventory)
9. Analgesic intake

Therapeutic Diathermy Devices: Delivery of Electromagnetic Waves to the Patient

Shortwave Diathermy

Shortwave diathermy can be delivered to body tissues either continuously (no interruption of electromagnetic waves coming from the device) or by pulse (regular brief interruption of waves from the device). There are two types of SWD: electric field (capacitive) method and magnetic field (inductive) method.

Capacitive Method

The capacitive method of SWD (also known as the *electric field method*) uses an applicator system that requires

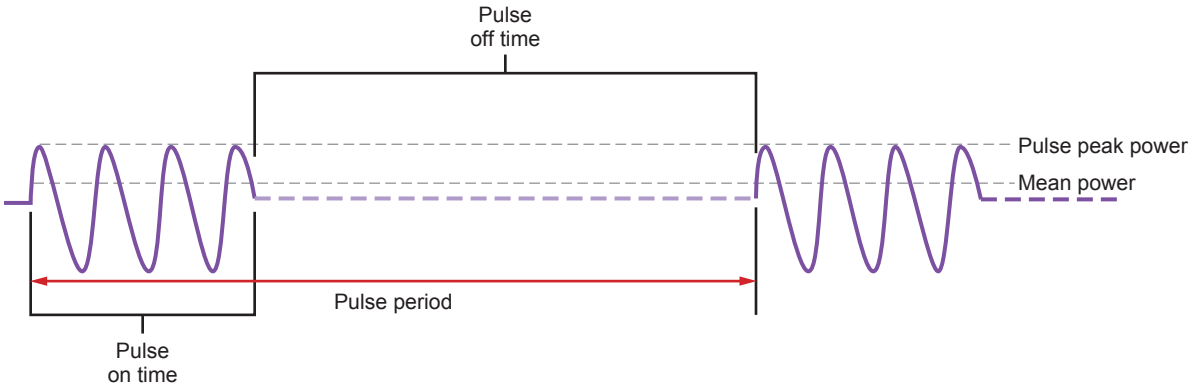

Fig 6 ■ 9 Pulsed shortwave diathermy (PSWD). The 27.12 MHz frequency wave is interrupted to create bursts (pulses) of waves, separated by a period of no bursts ("off-time"). The amount of energy delivered to the tissues depends on the *mean power,* which is determined by the pulse peak power, pulse frequency, and pulse duration. *(Art concept by Sara Monath.)*

that the patient's tissues become part of a capacitor (Fig. 6-10). A capacitor is a device that can store electrical charge and consists of two conducting objects placed near each other and usually separated by a dielectric. A dielectric is an insulating sheet placed between two electrically charged plates, causing an increase in capacitance.[3] By placing a body part between the capacitive electrodes of the SWD device, the body part serves as a dielectric. The capacitive electrodes cause an oscillating electric current to flow through the body tissues between the electrodes. Resistance to this current flow in the tissues results in tissue heating.

Leitgeb et al[90] found the specific absorption rate of radio frequency waves for capacitive SWD to be higher for fat tissue than for muscle, because fat tissue has a greater resistance to current flow than muscle. The result is greater heating of fat tissue and less heating of muscle.

Capacitive SWD devices have adjustable arms with metal plates (electrodes) at their ends that deliver electromagnetic radio waves to the patient. Most capacitive SWD plates have a glass or plastic guard surrounding each metal plate to prevent contact between the electrode and the patient's skin. A severe electrical burn may occur if either the therapist's or the patient's skin contacts the bare metal plate of the diathermy device. A single layer of terrycloth toweling should be placed between the plate guards and the patient's skin to prevent concentration of the electric field on perspiration that may accumulate on the skin. Most plates can be adjusted through a distance of about 3 cm within the guard. The plastic plate guard should be located about 2 to 10 cm (1 to 3 inches) from the skin for optimal heating, and the metal plate should be as far from the skin as the guard allows (at least 2.5 cm). Proper positioning of the plate and guard in relation to the skin provides effective absorption of thermal energy and safe heating of tissues. The closer the metal plates (electrodes) are to the skin, the greater the heat sensation. As the plate-to-skin distance increases, heat perception decreases. Adjustment of plate-to-skin distance allows for variation in the amount of heating of body tissues. The capacitive plates should always be positioned so the distance between any part of the two plates is at least as great as the plate's diameter. Capacitive plates should not be placed over areas of thick subcutaneous fat because fat tissue may be heated considerably more than muscle when both tissues are exposed to the capacitive SWD electric field.

Capacitive SWD plates can be positioned in two possible arrangements: contraplanar or coplanar. In the contraplanar arrangement, the plates are placed on each side of the body part so that the body part creates a biological capacitor, as previously described. A coplanar arrangement requires that the plates be positioned parallel on the same body surface rather than placing the body part between the plates (Fig. 6-11). In this arrangement, both superficial and deep tissues are parallel to the two applicator plates. The contraplanar arrangement is best for applying diathermy to extremities, whereas the coplanar method is best for treating the neck, back, or pelvis.

Inductive Method

The inductive method of SWD (also known as the *magnetic field method*) requires an inductive applicator that

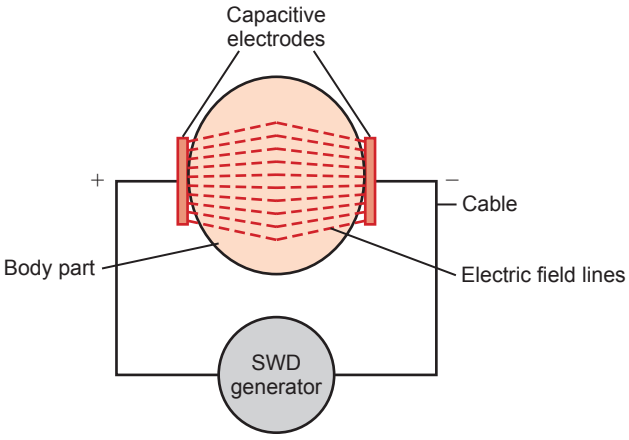

Fig 6 ■ 10 Capacitive method of diathermy. The body part is placed between two electrodes and becomes part of the electric field. The relative field density is greater closer to the electrodes. *(Illustration by Sara Monath.)*

Fig 6 ■ 11 Coplanar arrangement of capacitive diathermy electrodes applied to a patient's back.

creates an oscillating magnetic field that induces "eddy" currents in body tissues (Fig. 6-12). These eddy currents flow along pathways of higher conductivity, causing heating in these tissues. The greatest density of eddy current activity—and therefore the greatest amount of heating—occurs in low-impedance (high-conductivity) tissues containing the highest electrolyte content, such as skeletal muscle and blood. Less heating occurs in high-impedance tissues such as skin and fat.

Two types of inductive coil applicators can be used to deliver the magnetic field energy to the patient: drums or sleeves. Drum applicators contain a coil-shaped cable that is contained within a rigid plastic insulator housing. The treatment surface of the plastic applicator serves the same function as the plate guard of the air-spaced plate applicator: to keep the inductive coil away from the skin. There are two types of drum applicators: a monode and a diplode. A monode is a drum used to treat a single body surface and requires a single layer of terrycloth toweling to create additional spacing and moisture absorption from the skin (Fig. 6-13). The diplode is a hinged drum that allows one or more body part surfaces to be treated simultaneously. The diplode consists of a rectangular induction coil contained within an insulator housing that also serves to space the coil away from the patient's skin. Approximately 1 cm of terrycloth toweling is recommended to separate the diplode applicator from the patient's skin because the coil is closer to the treatment surface within the diplode. The towel will help prevent excessive surface heating of the skin and will absorb perspiration.

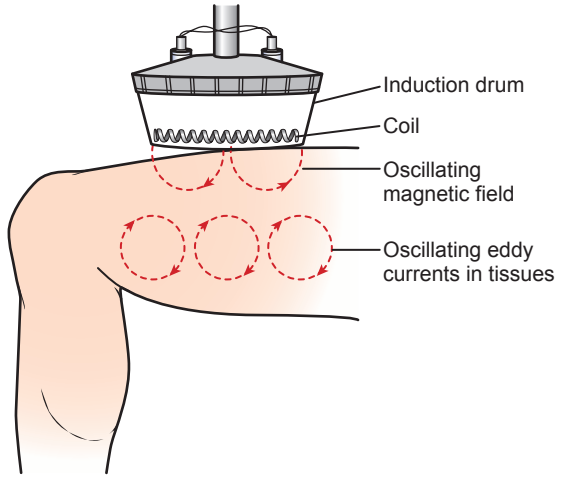

Fig 6 ■ 12 Inductive diathermy with monode drum placement over tissues creates a magnetic field that induces oscillating eddy currents in the tissues. *(Art concept by Sara Monath.)*

Fig 6 ■ 13 Application of induction drum (monode) to a patient's low back. A single layer of towel is placed between the skin and the surface of the drum.

An induction sleeve is a new method of delivering diathermy to a patient (Fig. 6-14). Sleeves are designed to fit around a body part, such as the elbow and forearm, providing a circumferential treatment effect. Advantages of diathermy sleeves include their portability and ease of application. A disadvantage is their heating ability is limited to moderate tissue temperature increases.[91]

Some diathermy devices have external cables that connect the applicator to the console and deliver the electromagnetic energy from the high-frequency oscillating circuit to the applicator (see Fig. 6-11). Cables on older devices may not be adequately shielded and may emit radiation in all directions when the applicators are energized. In this case, care must be taken to prevent the cables from coming into direct contact with the patient or any metal or synthetic materials.

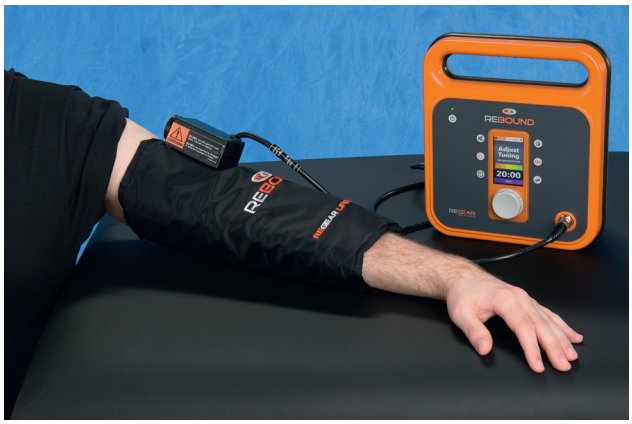

Fig 6 ■ 14 Induction diathermy delivered by a coil in a sleeve shaped to fit the body part. *(Rebound System courtesy of Game Ready, 1201 Marina Village Parkway, Suite 200, Alameda, CA 94501.)*

Key Point! Capacitive (electric field) diathermy will cause greater heating in skin and subcutaneous fat tissue than muscle. Inductive (magnetic field) diathermy will more effectively heat muscle than capacitive diathermy with less heating of skin and subcutaneous tissue.

Pulsed Shortwave Diathermy (PSWD)

Today, most diathermy devices are either pulsed shortwave diathermy or have both continuous and pulsed modes. Most PSWD devices use the inductive method with a drum electrode. Bursts of electromagnetic waves are created by interrupting the flow of continuous waves generated by the device. The burst (also known as *pulse*) duration is preset by the device manufacturer in a range usually from 40 to 400 microseconds (μsec). The clinician can adjust the burst or pulse frequency, which usually ranges from 1 to 1,000 bursts per second (also called *pulses per second,* or pps). The peak pulse power (the power in watts delivered during a pulse) ranges between 100 and 1,000 W in most devices that provide PSWD.

However, in clinical practice, the heating effect on the patient's tissues depends on the *average* power of the device. The average power can be determined by the following equation:

peak power (W) × pulse duration (s) × pulse frequency = average power

For example, if the peak power of a PSWD device is 250 W, the pulse duration is .0004 second, and the pulse frequency is set at 145 Hz, the average power is 14.5 W.[92] Generally, with most PSWD devices, the highest mean power output that can be delivered is lower than the power delivered during most continuous SWD treatments (80 to 120 W). However, PSWD devices can vigorously heat deep tissues. Draper et al[91] demonstrated that a PSWD device with an average power of 48 W can increase muscle tissue temperature at a depth of 3 cm up to 8.3°F (4.6°C).

Microwave Diathermy

Generation of electromagnetic waves in the microwave frequency range requires a device called a *magnetron*. A magnetron generates alternating current at a high power level, which is transmitted to an antenna housed inside an emitter (drum or applicator) that directs the electromagnetic field to the body part to be treated. Microwave ovens used for heating and cooking food use a magnetron similar to those used for diathermy. Frequencies allotted for MWD in the United States are listed in Table 6-11. In Europe, Australia, and New Zealand, 434 MHz is also approved for medical use.

Application of MWD requires proper alignment of the emitter in relationship to the body part to be treated. Placing the emitter at a right angle to the body part ensures that microwaves will be applied perpendicular to the skin's surface and will limit reflection of waves. Some emitters are placed at a small distance from the skin's surface with an air gap between the emitter and the skin. Other emitters are designed to be placed in contact with the skin.

MWD at higher frequencies (915 or 2,450 MHz) can be used to treat a more focused area of the body than SWD can provide. However, the thermal effects will not penetrate as deep as SWD or ultrasound.[93] This is because microwaves are partially reflected from tissue

TABLE 6–11. Dosages for Diathermy Treatments[152]

Dosage Level	Response*	Tissue Temperature Rise	Average Watts (PSWD)	Suggested Clinical Use
Dose I	Nonthermal	None	N/A	Acute injuries (sprains, strains, etc.), edema reduction, cell repair
Dose II	Mild heat sensation	1.8°F (1°C)	12 W	Subacute injuries and inflammation
Dose III	Moderate heat sensation	3.6°F (2°C)	24 W	Pain, muscle spasm, chronic inflammation
Dose IV	Vigorous heating	7.2°F (4°C)	48 W	Increase blood flow, heating of collagen tissues for stretching of soft tissues

*Based upon patient's report of perception of tissue heating.

layers, resulting in standing waves that concentrate the energy absorption in superficial tissues. There is an increased risk of damage to superficial tissues because of this greater absorption of energy. Microwaves have a higher frequency than shortwaves; therefore, a higher intensity of energy is delivered to the tissues.

The use of MWD has declined significantly since the 1980s, particularly in the United States. SWD is preferred over MWD because of greater depth of heating and a more predictable heat distribution pattern. Recently, especially in Europe, MWD devices with a carrier frequency of 434 MHz have become available. These devices have a greater depth of heating because of the lower carrier frequency. Indications and contraindications for MWD are similar to SWD.

The effectiveness of MWD was investigated by Ortega et al.[93] Patients with chronic neck pain were distributed into three groups that received the following: Group 1 received continuous MWD at a power of 80 W (thermal response), Group 2 received pulsed MWD at an average power of 5 W (athermal response), and Group 3 had an unplugged MWD device (placebo). No difference in outcomes was found among the three groups.

Fig 6 ■ 15 Portable PEMF device for promotion of bone healing. *(Courtesy of Orthofix Orthopedics North America, 1720 Bray Central Drive, McKinney, Texas.)*

Pulsed Electromagnetic Fields

There is some confusion in the literature about the use of the term *pulsed electromagnetic fields,* or PEMF. Some publications use this term to indicate application of pulsed shortwave diathermy at an intensity that may cause heating of tissues. Other publications use PEMF to describe electromagnetic waves delivered at a very low intensity that do not cause heating of tissues. Bassett[94] limited PEMFs to devices that deliver low-energy magnetic fields that cause nonthermal effects on tissues, such as promotion of bone healing. In this chapter, PEMF will be limited to those devices that deliver very low-intensity electromagnetic waves that do not heat tissues. The use of magnetic fields for therapeutic purposes is discussed in Chapter 16.

PEMF devices have been in use since the 1950s, when the FCC approved the Diapulse device for therapeutic purposes.[95] These devices generate PEMFs by an induction coil aerial that is placed on the skin's surface. Average power is typically limited to 1 W or less. Portable battery-powered PEMF devices are available that can be worn by the patient (Fig. 6-15). These devices are primarily used to promote tissue healing, presumably by nonthermal facilitation of cellular metabolism and ionic fluctuation.

Physiological Effects of Diathermy

The frequencies of electromagnetic waves used for diathermy applications are *not* capable of depolarizing motor nerves or eliciting a contractile response from innervated or denervated skeletal muscle. Electrical excitation of a nerve or muscle requires a pulse duration of about 0.1 ms (millisecond) or longer. The pulse duration of an electromagnetic wave at a frequency of 27.12 MHz is only 36 nanoseconds ($36{,}310^{29}$ sec); therefore, excitation and depolarization of these tissues does not occur because the wavelength (duration) of each cycle of the high-frequency alternating current does not last sufficiently long to cause migration of ions through cellular membranes of nerve or muscle. Electromagnetic waves in the radio frequency range are nonionizing radiation, which means that there is insufficient energy concentration to

dislodge orbiting electrons from atoms. Radio frequency energy delivers only a fraction of the energy level required to produce ionization in tissue. For example, the energy at a frequency of 100 MHz (in the FM radio frequency band) is approximately 300 million times too weak to produce ionization.[96] Therefore, radio frequency electromagnetic waves will not induce mutations or the uncoupling of DNA single strands.

Key Point! The electromagnetic waves produced by diathermy devices will not cause depolarization of nerves or muscles. They are nonionizing and will not cause cell mutations.

Thermal Effects of Diathermy

Electromagnetic energy from the radio or microwave frequency part of the spectrum may or may not be perceived as the waves travel through biological tissues. Regardless of whether it is perceived by the body, thermal effects can occur. The goal of most diathermy treatments is to increase the temperature of body tissues. The amount of energy delivered to the patient will depend on the type of diathermy (continuous or pulsed); the proximity of the electrode, drum, or emitter to the patient's skin; and the duration of the treatment. The ability of a diathermy device to raise tissue temperature depends on the device's power output, measured in watts (W). The power output range for most SWD devices is 55 to 500 W, which is sufficient to raise the temperature of most tissues to a range of 99.5°F to 111.2°F (37.5°C to 44°C). The range of peak (instantaneous) pulse power for most pulsed shortwave devices is 100 to 1,000 W. However, the potential for producing a heating effect with these devices depends on the *average* power delivered to the tissues with successive bursts of pulse trains. The highest average power that can be delivered with PSWD devices is lower than the usual power output delivered during continuous SWD treatments. The thermal effects of diathermy on tissues will result in similar physiological responses to superficial heating modalities. This is discussed in Chapter 3.

One key difference is the greater depth of heating that occurs with diathermy compared to superficial thermal modalities. How is heat produced in body tissues when radio or microwave frequency electromagnetic waves travel through them? Body tissues contain large numbers of ions. When these ions are exposed to a high-frequency oscillating electromagnetic field, they are accelerated first in one direction and then the other. A result of this increased ionic motion is an increase in collisions with nearby molecules. These collisions increase the random motion of ions, which in turn leads to increased internal kinetic energy and heat generation in the tissues (Fig. 6-16A). Additional significant heating occurs because many tissues, especially muscle and blood, are primarily composed of water. Water molecules are electrically neutral; however, they are also polar or have polarity because one end of the molecule is

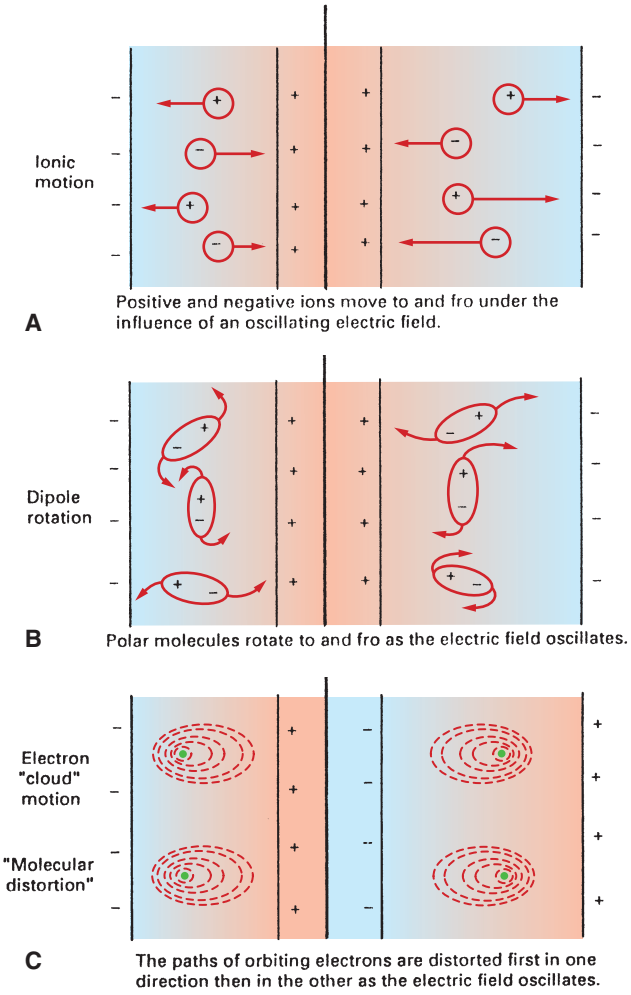

Fig 6 ■ 16 Effects of electromagnetic waves on ions and molecules in tissues of the body. (A) Back-and-forth motion (oscillations) of ions results in collisions that produce heat. (B) Rotation of dipoles (water molecules) results in increased random motion of adjacent molecules and produces heat. (C) Distortion of atoms and molecules that are not charged results in increased random motion of adjacent molecules, which may cause some heat. *(Modified from Robertson, Ward, Low, Reed.* Electrotherapy Explained, Principles and Practice*. 4th ed. Oxford, UK: Butterworth Heinemann Elsevier; 2006.)*

positively charged while the other end is negatively charged. Molecules with this type of polarity are known as *dipoles*. When exposed to a high-frequency oscillating electromagnetic field, the polar water molecules undergo dipole rotation. In the process of spinning, they collide with other molecules, increasing random motion and heat generation (Fig. 6-16B). Other atoms and molecules that are not electrically charged (nonpolar) may have the paths of their orbiting electrons shifted by the oscillating electromagnetic field, which results in a back-and-forth oscillation of their electron cloud. This distortion of the electron cloud allows for only minimal friction and movement between adjacent molecules that, in turn, results in minor heating[97] (Fig. 6-16C). Ward[98] believed increased ionic motion is the most efficient mechanism for converting high-frequency current into heat.

Key Point! Diathermy can heat tissues at a greater depth than superficial heat modalities. Heating by diathermy is caused by the effect of electromagnetic waves on ions, water molecules (dipoles), and electrons in the body's tissues.

Inductive SWD and PSWD will more effectively elevate the temperature of deep tissues than capacitive SWD.[1,99] Inductive diathermy is particularly effective for heating tissues with high conductivities and high electrolyte content, particularly those well perfused with blood such as muscle. High temperatures are also produced in areas surrounding joints. Inductive SWD and PSWD cause less heating in fat, bone, and collagen tissues compared to capacitive diathermy. The effectiveness of SWD to heat muscle tissue has been demonstrated.[100–103] They showed that induction PSWD (800 bursts per second [bps], 400 μsec burst duration, 48 W average root mean square [RMS] power) effectively increased muscle temperature as much as 39.2°F (4°C) after 15 minutes of treatment. Mitchell[104] used the same parameters for PSWD and demonstrated an increase in muscle temperature up to 35.6°F (2°C) at a depth of 2.5 cm.

Heating of tissues can be demonstrated by monitoring changes in circulation. Millard[105] studied the effects of SWD heating on skin blood flow and showed that clearance of radioactive sodium increased nearly 150% after exposure, resulting in an average temperature rise of 41.5°F (5.3°C). Muscle clearance rates increased by 36%, with a muscle temperature rise of 41.3°F (5.2°C). Mayrovitz and Larsen[106] demonstrated increased microvascular blood perfusion in a group of subjects who received 40 minutes of PSWD (600 pps, 65 μsec pulse duration, 35 W average power) to their forearms. Harris[107] demonstrated a 100% increase in circulation in knee joints after SWD application based upon radio-sodium clearance from the joints.

Capacitive SWD requires placing the patient's tissues between the electrodes (plates) of the device. The plates, intervening air, and body part form a capacitor that can store an electrical charge (see Fig. 6-10). The charge of the high-frequency electric field oscillates from one plate to the other, resulting in tissue heating. Increased muscle blood flow after 10 minutes of capacitive SWD (80 W) was demonstrated by Karasuno et al.[108] They found blood flow increased greater with SWD than another group that received electric hot packs. However, the rate of fat tissue heating is greater than muscle with capacitive SWD because the field intensity is highest close to the electrodes and fat tissue is closer to the electrodes than muscle. Figure 6-17 shows a comparison of the heating effects of capacitive and inductive diathermy.

Treatment of Pain

Reduction of pain by diathermy can most likely be explained by the well-known analgesic effects of heat

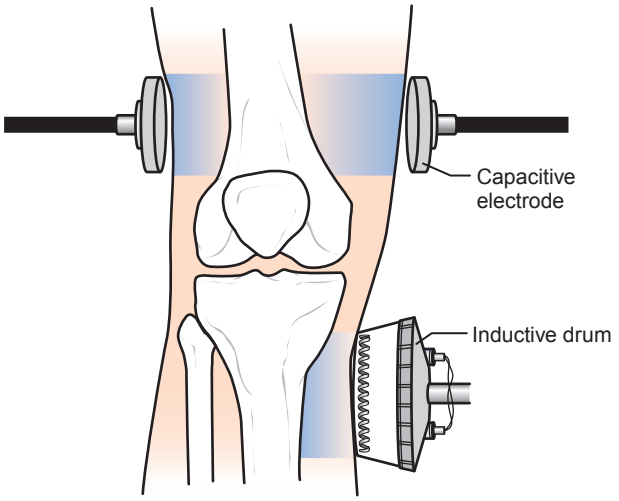

Fig 6 ■ 17 Comparison of capacitive and induction diathermy on heating of tissues. Note that capacitive tends to heat superficial fat tissue greater than inductive, and inductive heats deeper muscle tissue greater than capacitive. *(Art concept by Sara Monath.)*

(see Chapter 3). McCray and Patton[109] compared the effects of inductive SWD to moist hot packs on the sensitivity of trigger points in the neck, shoulders, and back. SWD was more effective at relieving pain at trigger points than moist heat. In a study by Cetin et al,[110] women with osteoarthritis of the knees had pain relief after a combination of capacitive SWD, hot packs, and isokinetic exercise. Literature reviews of SWD for treatment of knee osteoarthritis have found significant relief of pain[111,112] and function.[113] However, Laufer et al[114] were unable to demonstrate any effect on pain in patients with osteoarthritis in the knees who received either PSWD at 300 pps (300 μsec pulse duration, mean power 18 W), PSWD at 110 pps (82 μsec pulse duration, mean power 1.8 W), or sham diathermy. Akyol et al[115] found no significant improvement in outcome measures for females with bilateral knee osteoarthritis treated with SWD.

There is insufficient evidence to justify the use of diathermy for treatment of chronic neck and back pain. Durmus et al[116] found no significant improvement in females with chronic back pain after microwave diathermy. Ortega et al[93] found no improvement in pain and outcome scores for patients with chronic neck pain after either continuous or pulsed MWD. Kim et al[117] reported improvements in outcome measures for subjects with chronic lower back pain who received "electromagnetic diathermy" at 50 W combined with nerve mobilization.

Diathermy has been shown to relieve pain after foot surgery,[118] in mild to moderate idiopathic carpal tunnel syndrome,[119] and in athletes with supraspinatus tendinopathy.[120] Abdominal pain caused by chronic pelvic inflammatory disease can be relieved by SWD.[121,122]

Some pain relief may be secondary to nonthermal mechanisms. Wagstaff et al[123] compared the effects of inductive SWD, PSWD at 82 pps (mean power 23.2 W), and PSWD at 200 pps (mean power 23.4 W) applied to three groups of subjects with low back pain. All three groups had significant pain relief. The groups that received PSWD had either no perceptible heating or very low (barely perceptible) heating of tissues, because the mean power levels were lower than the usual mean power needed to cause perceptible heating (believed to be about 38 W PEMF). Nonthermal application of PSWD does not cause heating of tissues but has been shown to help control pain in patients with idiopathic trigeminal neuralgia[124] and to decrease pain and increase mobility of the TMJ in patients with TMJ arthralgia.[125]

Effects on Tissue Flexibility

The combination of heat and prolonged low-load stretch (PLLS) has been shown to effectively increase tissue elasticity and facilitate connective tissue flexibility and joint ROM. Peres et al[126] applied PSWD (800 pps, 400 μsec pulse duration, 48 W average power) to the musculotendinous junction of the triceps surae for 20 minutes, with PLLS added for the last 5 minutes of the treatment. An ice bag was applied to the musculotendinous junction for 5 minutes after the 20-minute diathermy treatment. Subjects who received PSWD, PLLS, and ice had greater immediate and net ROM increases than subjects who received PLLS alone.

Draper et al[127] found that subjects with tight hamstrings who received PSWD (800 pps, 400 μsec pulse duration, 48 W average RMS power) to their hamstrings for 5 minutes during stretching and 10 minutes after stretching of the hamstrings had greater flexibility (increased knee extension) than subjects who received sham diathermy and stretching. However, Martinez-Rodriguez et al[128] did not find improvement in hamstring flexibility after PSWD for 15 minutes to the posterior thigh.

Diathermy may help increase tissue flexibility without stretching. Robertson et al[129] applied SWD for 15 minutes to the lower leg by placing two capacitive pads parallel to the leg and close to the skin and perpendicular to the top of a plinth. Subjects did not perform stretching during or after the treatment. Ankle dorsiflexion ROM increased 5.2% after SWD compared to 2% for subjects who received hot packs. No change was observed for a control group that received no heat.

Effects on Nerve Conduction Velocity and Muscle Function

Applying diathermy to muscle tissue may affect muscle contraction. Currier and Nelson[130] compared the effects of exercise (riding an ergometer for 5 minutes) and SWD (applied to the low back for 20 minutes) on motor nerve conduction velocity of the peroneal nerve. Motor nerve conduction velocities increased for both conditions;

however, exercise caused a greater increase in conduction rate than diathermy. Chastain[131] used an isokinetic dynamometer to measure the isometric strength of the quadriceps in subjects who received inductive SWD for 20 minutes. Initially, isometric strength decreased an average of 2.15 newton meters (Nm). After 30 minutes, strength increased an average of 14.1 Nm; after 90 minutes, the strength levels for each subject fluctuated. Clinicians need to consider possible changes in muscle strength after diathermy treatments and adjust the timing of exercise programs and quantification of muscle strength. The application of diathermy immediately preceding exercise may augment exercise performance. Cetin et al[110] found that applying physical modalities, including capacitive SWD, before isokinetic exercise in patients with symptomatic osteoarthritis of the knees led to improved exercise performance and function.

Promotion of Tissue Healing

Diathermy has been shown to promote healing of soft tissues and bone.[132–135] Increases in tissue temperature that occur with thermal-level SWD and MWD can increase circulation, which explains improved tissue healing rates. Effects on tissue regeneration were demonstrated by Hill et al[136] using PSWD at a mean power of 48 W for 10 minutes. This resulted in increased fibroblast proliferation. PSWD at a mean power of 6 W for 10 minutes increased chondrocyte proliferation. In a study by Mayrovitz and Larsen,[137] evidence for nonthermal effects on healing was demonstrated using PSWD, which increased local microvascular perfusion around the perimeter of ulcers in patients with diabetes. PEMFs using a Diapulse device have been shown to accelerate healing of pressure ulcers.[138,139] Studies by Bassett[140] and Sharrad[132] demonstrated effective promotion of fracture healing by PEMFs. Portable battery-operated "bone stimulators" are in common use today for promoting healing of nonunion or delayed-union fractures and arthrodeses.

Clinical Application of Diathermy

The decision whether to use diathermy requires an understanding of the physiological effects of this modality and its indications, precautions, and contraindications based on published evidence. Advantages and disadvantages of this modality compared to other devices with similar effects must also be considered. The clinician should thoroughly explain the effects of diathermy to the patient, including any possible negative effects, and allow the patient to choose whether to receive the treatment.

Dosage

Dosage is a measure of the amount of energy delivered to the body that is absorbed and converted into heat. Dosimetry is the measurement of this energy transfer in calories per unit of time. Dosimetry measurements require small temperature probes to be inserted into or through the skin to measure the heat produced by the diathermy treatment. Temperature probes cannot be inserted into a patient's tissues in clinical practice; therefore, it is not possible to measure the amount of electromagnetic energy transferred from a diathermy device to the patient's tissues. Clinicians must closely monitor the patient's report of heat sensation on their skin and deeper tissues and use this as a guide for dosage during diathermy treatments.

Patients with a documented or a potential sensory deficit must be tested for pain and temperature sensation before beginning treatment to determine if they are capable of reporting changes in cutaneous heat sensation. Based on Joule's law, the maximum tissue temperature reached regardless of the heat modality depends on the square of the intensity, the tissue impedance, and the duration of the heat treatment. The amount of tissue temperature elevation depends on several factors, including the adequacy of blood perfusion through the tissues. Poor blood perfusion, such as in arterial insufficiency of the lower extremities, may not allow for adequate heat dissipation when heat from any source is applied directly to the poorly perfused tissues. When tissues are adequately perfused, the body's thermoregulatory mechanism increases heat removal from the local tissue until a new, higher temperature steady state is reached, thus avoiding tissue damage from excessive heat. It is essential to frequently monitor the patient during the first 10 minutes of tissue heating with diathermy to determine the amount of heating that is occurring in the tissues and the patient's tolerance of it.

The clinician needs to estimate the intensity and treatment duration (dosage) required to increase tissue temperature to the desired therapeutic range (between 99.5°F and 111.2°F [37.5°C and 44°C]). See Table 6-11 for

suggested dosages for diathermy treatments. Choice of dosage depends on several factors such as the stage of tissue healing (acute, subacute, or chronic), the location of the target tissues, the approximate thickness of tissues overlying the target tissue, and the patient's tolerance. The clinician then chooses the intensity and treatment duration that will most likely achieve effective tissue heating. If the chosen dosage is nonthermal, then a PSWD or PEMF device is set at an average power less than 38 W so that the patient will not perceive any heat sensation. Some diathermy devices display intensity on a linear scale of arbitrary units such as 0 to 10. The correlation of this scale with power (watts) may be described in the device manual. The amount of power delivered to the patient will be an estimate based on the intensity value displayed by the device and the patient's response.

Key Point! Therapists can estimate the dosage of diathermy delivered to the patient based on the device's power output (average power for PSWD), the duration of treatment, and the intensity of the heat perceived by the patient during the treatment.

Duration of diathermy treatments typically are 15 to 30 minutes. Draper and colleagues[100,102] applied PSWD at 800 Hz and an average power output of 48 W for 20 minutes to the triceps surae. Peak muscle temperature increases at 3 cm below the skin occurred at 15 minutes and slightly declined at 20 minutes of treatment. Duration of treatments with either nonthermal PSWD or PEMF vary widely in the literature, from 30 minutes[141,142] to 3 hours.[143] Some of these treatments were described as "magnetotherapy," with intensity measured in gauss or micro-teslas (see Chapter 16).

The frequency and number of diathermy treatments are based on the goals of treatment. Diathermy can be applied daily or several times a day for promotion of tissue healing and pain relief. Facilitation of tissue stretching requires diathermy to be administered immediately prior to stretching and is typically performed daily or several times a week. There is no known limit on the number of diathermy treatments that can be administered to a patient. However, diathermy treatments should be discontinued when treatment goals are achieved or the treatment is not effective. It is difficult to judge the number of treatments required to achieve treatment goals; more research is needed to establish these guidelines.

Steps for Clinical Application of Diathermy

- Explain the purpose and effects of diathermy to the patient.
- Inspect the skin area where the diathermy will be applied for any precautions or contraindications. Record in the patient's therapy record any discoloration, scars, or other skin blemishes found on the skin prior to treatment.
- Position the patient comfortably on a nonmetallic table or chair with the body part to be treated exposed and positioned for proper placement of the diathermy applicator.
- Remove all metal objects from the patient (e.g., jewelry) and the treatment area.
- Place one layer of terrycloth or cotton towel over the skin where the diathermy will be applied (not necessary for induction sleeves and low-power treatments below about 38 W when heating of tissues is minimal or zero).
- Position applicator appropriately in relation to patient's skin:
  - Capacitive plates: both must be placed an equal distance from the skin surface, about 1 to 3 inches (2 to 10 cm) from the skin, parallel to skin surface.
  - Inductive drum: place directly over tissues to be treated, almost touching towel on skin.
  - Microwave drum: place parallel to patient's skin, about 1 to 3 inches (2 to 10 cm) away.
- Instruct the patient not to move the body part during treatment and not to touch the diathermy applicator or device during treatment.
- Instruct the patient to focus on any heat sensation produced during treatment and to report the level of heat perceived (mild, moderate, or strong) and any discomfort associated with this heat sensation.
- Choose a dosage for the patient based on the patient's diagnosis and the goals of the treatment (see Table 6-11).
- Some PSWD devices allow adjustment of pulse frequency and pulse duration. Consult Table 6-11 for suggested parameters.
- Set the timer on the device for 15 to 30 minutes, depending on treatment goals.

- Slowly increase the intensity until the patient reports heat sensation. Adjust the intensity based upon the chosen dosage and patient's tolerance.
- Monitor the patient during the treatment, and adjust the intensity based on the patient's report of the amount of heating.
- At the conclusion of the treatment, turn off the device and remove the applicator from the patient. Remove any towels from the skin and inspect the area. A mild erythema of the skin is normal after diathermy.
- Assess effectiveness of the treatment and document in the patient's record.

Indications for Diathermy

SWD, PSWD, and MWD

Most of the indications for thermotherapy described in Chapter 3 also apply to SWD, MWD, and PSWD. There are two primary advantages of using diathermy for thermotherapy: It provides a greater depth of heating than superficial thermal modalities and it can heat a larger area (greater amount of tissue) than ultrasound. These advantages make diathermy the best choice for heating large joints, such as the knee or shoulder, or large muscles, such as the hamstrings. Also the risk of soft tissue burns is less with diathermy than with conductive heating modalities such as hot packs.

The research literature includes many studies that support the use of diathermy for therapeutic interventions (Table 6-12). There are also studies that found diathermy was ineffective (Table 6-13). Literature reviews published since 2000 are listed in Table 6-14. Generally, these reviews support the use of SWD for treatment of some musculoskeletal conditions, such as osteoarthritis of the knees. The reviews also support the belief that PSWD may be beneficial for treating ankle sprains.

TABLE 6–12. Effective Applications of Diathermy

Condition	Outcome Measures	Type of Diathermy	Reference/Type of Study
OA (knees)	VAS, WOMAC, 15 min walking time	SWD	Boyaci et al, 2013[172] (RANC)
OA (knees)	Pain scale Goniometry	SWD and PSWD	Teslim et al, 2013[173] (RANC)
Chronic lower back pain	Oswestry, location of symptom scale, isokinetic knee muscle strength	"Electromagnetic diathermy" at 50 W	Kim et al, 2012[117] (RCT)
OA (knees)	VAS, time to walk 15 m, ROM, WOMAC, Health Profile, Paracetamol intake	SWD	Atamaz et al, 2012[174] (RCT)
Chronic pelvic inflammatory disease	VAS, presence of pus cells	SWD	Lamina et al, 2011[122] (RCT)
OA (knees)	Numerical pain rating scale, knee OA outcome score (KOOS)	PSWD (average power 14.5 W)	Fukuda et al, 2011[92] (RCT)
Decreased elbow ROM after fracture/dislocation ORIF	Elbow ROM	PSWD	Draper, 2011[175] (CSR)
Adhesive capsulitis of the shoulder, S/P RC surgery and manipulation	Shoulder ROM	PSWD	Draper & Gage, 2010[176] (CS)
Decreased knee ROM after ACL repair	Knee ROM	SWD knee sleeve (35 W)	Draper & Van Patten, 2010[177] (CS)
OA (knees)	Pain disability index scores	SWD	Cetin et al, 2008[110] (RCT)
OA (knees)	Synovial sac thickness	SWD	Jan et al, 2006[178] (RCT)

TABLE 6–12. Effective Applications of Diathermy—cont'd

Condition	Outcome Measures	Type of Diathermy	Reference/Type of Study
Calcific tendinopathy	Pain, ROM, disability index scores, presence of calcium deposits on x-ray	MWD	Di Cesare et al, 2008[133] (CS)
Joint ROM	Ankle dorsiflexion	PSWD	Seiger & Draper, 2006[151] (CSR)
Joint ROM	Ankle dorsiflexion	SWD	Robertson et al, 2005[129] (RCT)
Joint ROM	Ankle dorsiflexion	PSWD	Peres et al, 2002[126] (RCT)
Hamstring flexibility	ROM of knee with hip at 90°	PSWD and long-duration stretching	Draper et al, 2004[127] (RCT)
RA	Pain scale joint stiffness	MWD	Usichenko, 2003[179] (RCT)
Acute muscle injury	Pain (VAS) ultrasonography	MWD	Giombini et al, 2001[180] (RANC)

CS = case study; CSR = case series; RANC = randomized allocation of subjects, no control; RCT = randomized controlled trial; VAS = visual analog scale; WOMAC = Western Ontario and McMaster Universities arthritis index.

TABLE 6–13. Ineffective Applications of Diathermy

Condition	Variables	Type of Diathermy	Reference/Type of Study
Tight hamstrings	Active and passive ROM, VAS	PSWD	Martinez-Rodriguez et al, 2014[128] (RCT)
Chronic low back pain	VAS, Oswestry, PDI, 6-minute walk, trunk muscle strength, spine endurance tests, SF-36, Beck depression inventory	MWD	Durmus et al, 2014[116] (RCT)
Chronic neck pain	VAS, NDI, SF-36	Continuous and pulsed MWD	Ortega et al, 2014[93] (RCT)
Subacromial impingement syndrome	VAS, goniometry, SPADI, shoulder disability questionnaire, shoulder isokinetic muscle strength, hand grip strength, SF-36, Beck depression index	MWD	Akyol et al, 2012[119] (RCT)
OA (knees)	VAS, WOMAC, 6-minute walk, isokinetic knee strength, SF-36, Beck depression inventory	SWD	Akyol et al, 2010[115] (RCT)
OA (knees)	Thai WOMAC index, 100 m walk speed, stair ascent/descent time, patient satisfaction, adverse events	SWD	Rattanachaiyanont & Kuptniratsaikul, 2008[181] (RCT)
Neck disorders	Pain questionnaire	PSWD	Dziedzic et al, 2005[182] (RCT)
Joint ROM	Ankle dorsiflexion (retention of gains in ROM after stretching)	PSWD	Brucker et al, 2005[183] (FRM)
OA (knees)	WOMAC Index	PSWD	Laufer et al, 2005[114] (RCT)
OA (knees)	Joint inflammation knee ROM	PSWD	Callaghan et al, 2004[184] (RCT)
Hamstring flexibility	Sit and reach ROM	PSWD and short-duration stretching	Draper et al, 2002[185] (RCT)

FRM = 2×4 factorial design with repeated measures; RCT = randomized controlled trial; NDI = neck disability index; PDI = pain and disability index; SPADI = shoulder pain and disability index.

TABLE 6–14. Published Literature Reviews: Diathermy Applications

Type of Diathermy	# of Studies Reviewed	Conclusions	Reference
SWD & PEMF	14	Significant relief of pain at 4 and 8 weeks after initiation of treatment in subjects with knee OA. Also improved function after 8 weeks of treatment.	We et al, 2013[112]
SWD	7	Small significant effects on pain and muscle performance for subjects with knee OA; effects primarily immediately posttreatment only.	Laufer & Dar, 2012[111]
PSWD & PEMF	9	Improved clinical scores and function for subjects with knee OA; no improvement in pain compared to control groups.	Vavken et al, 2009[113]
SWD	3	Less effective than spinal manipulation for LBP; no difference: SWD vs. sham SWD.	Chou and Huffman, 2007[186]
MWD	NS	Effective for short-term care of musculoskeletal injuries.	Giombini et al, 2007[134]
SWD and PSWD	11	Equivocal findings; studies had poor methodological quality.	Marks et al, 1999[187]
PSWD	9	Effective for ankle sprains only at higher peak and mean powers and longer treatment durations.	Low, 1995[188]

LBP = low back pain; NS = not specified.

Clinical Controversy

Clinicians often have a range of thermal modalities to choose for their patients. Superficial thermal modalities are usually readily available and generally easy to apply; however, their depth of heating is limited to superficial tissues except in areas of the body that have a thin soft tissue covering, such as fingers, hands, toes, and feet. Effective heating of deeper tissues, such as muscles and joints in most areas of the body, requires either ultrasound or diathermy. Diathermy is more effective than ultrasound for heating large muscles and joints; however, the cost of diathermy devices and clinician bias or lack of knowledge of the research literature may preclude its clinical usage.

Continuous SWD may be used for selective heating of pelvic organs for women with chronic inflammatory pelvic diseases.[121,122] Heating of the pelvic cavity produces a significant increase in vascularity and blood flow, which causes increased cardiac output. Improved pelvic blood flow will enhance delivery of antibiotics to inflamed tissues.

The use of mild heat (dose level II) SWD has been found to help relieve pain associated with herpes zoster (shingles).[144,145] SWD treatments should begin immediately or within a few days of the onset of the herpes zoster rash. Daily 20-minute applications at the level of the involved dorsal root ganglia are recommended until pain is decreased or the blisters dry up.

Key Point! Diathermy is indicated whenever heating of deep tissues (up to 3 to 5 cm) is desired. Diathermy is particularly effective at heating large areas of body tissues, such as large muscles (e.g., hamstrings) and large joints (e.g., hips).

PEMF

The research literature supports the use of nonthermal pulsed electromagnetic fields for promoting superficial wound and bone healing. Research support for other applications of PEMF is limited. A systematic review published by McCarthy et al[146] analyzed five randomized controlled trials of PEMF (defined as "electromagnetic fields with on-and-off effect of pulsing to produce athermal effects") and reported that PEMF does not significantly decrease the pain of knee osteoarthritis. Thamsborg et al[147] reported that PEMF did not

significantly relieve pain in patients with knee osteoarthritis; however, there was a significant beneficial effect on knee stiffness in subjects younger than age 65. Aktas et al[148] found no convincing evidence that PEMF was beneficial for subjects with subacromial impingement syndrome. More studies are needed to determine if the nonthermal effects of PEMF are effective for other clinical problems.

Precautions for Diathermy

A thorough history and patient examination must be performed before diathermy treatment to determine if any precautions must be followed. A precaution requires the practitioner to modify the application of diathermy and/or closely monitor the patient during treatment. Precautions for diathermy are listed in Table 6-15.

Contraindications for Diathermy

The patient history and exam is also important to determine any contraindications for diathermy. A contraindication precludes the use of diathermy for that patient. See Table 6-16 for a list of contraindications for diathermy. The thermal effects of diathermy are

CASE STUDY 6-3 Selecting Diathermy for an Athlete With Tight Hamstrings

A 21-year-old female field hockey player has a history of recurrent right leg hamstring strains that have been limiting her ability to run and play field hockey. Measurement of the right knee extension ROM with the right hip flexed at 90° ("90/90" test for hamstring tightness) finds 30° limitation from full extension compared to the left knee, where knee extension ROM with the hip flexed at 90° is 15° from full extension. She has been performing frequent stretching exercises for the right hamstrings with minimal change in flexibility.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction or impairment that can be improved or lessened with the use of diathermy?
ANSWER: Yes, she has a movement dysfunction caused by persistent tightness of her right hamstrings.

2. Why would diathermy be a consideration for this patient?
ANSWER: The thermal effects of diathermy can help relax the hamstrings and soften tropocollagen bonds to facilitate stretching of the musculotendinous complex.

3. What specific outcome measures can be used to assess the effectiveness of this modality?
ANSWER: Hamstring tightness measured by her ability to passively extend the right knee with the hip at 90° of flexion. Another outcome measure is her ability to return to running and playing field hockey without any pain or stiffness in her right hamstrings.

4. Are there any precautions or contraindications to consider with this patient?
ANSWER: This patient has one contraindication: metal jewelry piercing of the umbilicus that she will be able to remove prior to treatment. She also has one precaution: She is currently menstruating. The therapist informs her that there is a slight chance that PSWD could increase her menstrual flow because of the proximity of the pelvic cavity to the treatment area.

5. What specific parameters are recommended for this patient?
ANSWER: PSWD parameters: pulse frequency 800 pps, pulse duration 400 μsec, and an average power of 48 W. Dosage level IV will be required (vigorous heating). Duration of treatment will be 15 minutes.

6. What are the proper steps to using PSWD for this patient?
ANSWER: She will be positioned comfortably prone and one layer of cotton or terrycloth towel will be placed over the right posterior thigh. The PSWD induction drum will be placed over her right posterior hamstrings, almost touching the towel. The intensity of the device will be gradually increased until she reports a strong (vigorous) heating sensation in her right hamstrings. Duration of treatment will be 15 minutes. Immediately after PSWD treatment, she will perform aggressive hamstring stretching.

CASE STUDY 6-4 Selecting Diathermy for a Patient With Osteoarthritis of the Knees

A 62-year-old male has a history of pain and stiffness of his knees because of degenerative arthritis (osteoarthritis). He has been referred to physical therapy by his general practitioner for bilateral knee exercises to help strengthen his knees and decrease his pain. The physical therapist's examination finds ROM of both knees is close to normal; however, there is stiffness during motion and moderate discomfort at the end ranges of motion. Manual muscle testing finds 4/5 strength for his quadriceps and hamstrings. He has difficulty walking long distances and walking up and down stairs.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction or impairment that can be improved or lessened with the use of diathermy?

ANSWER: Yes, he has a movement dysfunction caused by pain, stiffness, and weakness of his knees. He can be classified as having a Musculoskeletal Practice Pattern E: impaired joint mobility, motor function, muscle performance, and ROM associated with localized inflammation (osteoarthritis of the knees).

2. Why would diathermy be a consideration for this patient?

ANSWER: The deep heating effects of diathermy can decrease pain and stiffness of his knees, which may enable him to perform exercises and improve his ability to walk.

3. What specific outcome measures can be used to assess the effectiveness of this modality?

ANSWER:

- VAS to assess pain level in his knees
- ROM measurements of his knees to assess stiffness and pain at end ranges
- Strength testing of his knees (manual muscle testing or dynamometry)
- Six-minute walking distance test or 15-meter time to walk test and ability to ascend and descend stairs
- Knee OA Outcome Score

4. Are there any precautions or contraindications to consider with this patient?

ANSWER: The clinician must ask this patient if he has any metal imbedded in his legs. If he has a history of a fracture with surgical fixation with metal screws or plates, this would be a precaution for PSWD and a contraindication for SWD or MWD. Circulation problems in the legs could also be a precaution or contraindication. A history of cancer, such as melanoma, would be a contraindication for diathermy over or near cancerous tissues.

5. What specific parameters are recommended for this patient?

ANSWER: Either induction SWD or PSWD are appropriate for this patient. Appropriate parameters for SWD for this patient are level III dosage (moderate heat sensation) for 15 minutes to each knee. Appropriate parameters for PSWD are frequency of 800 bps, burst (pulse) duration of 400 μsec, peak pulse power of 150 W, and average power of 48 W, for 15 minutes to each knee.

6. What are the proper steps for using diathermy for this patient?

ANSWER: The patient will need to be comfortably positioned supine on a nonmetallic table or sitting in a nonmetallic chair. One layer of a cotton or terrycloth towel will be draped over the knee. The SWD or PSWD inductive drum will be positioned close to the skin of the patient's knee or the knee will be placed in an inductive sleeve. The patient will be instructed to focus on the heating sensation in his knee during the treatment and to report the level of heating perceived to the clinician. The clinician will slowly increase the intensity until the patient reports a sensation of heat. The intensity will then be adjusted until he feels a "moderate" level of heat. The duration of treatment for each knee will be 15 minutes.

Note: The patient examination determined that he had normal sensation in his knees, so it is unnecessary to test for hot and cold sensation prior to treatment.

TABLE 6–15. Precautions for Diathermy

Precaution	Explanation
Small pieces of metal or metal fixation devices (screws, plates, rods) imbedded in the body	Precaution for PSWD because studies have not found any harmful effects of administering PSWD over these items.
Intrauterine contraceptive devices with copper	Diathermy may cause slight heating of device but not a contraindication.
Decreased sensation	Precaution because patient may not be able to recognize overheating of tissues; use lower intensities initially to assess ability to feel heat.
Impaired circulation	Precaution because tissues may not be able to respond to increased metabolic demands caused by heating of tissues.
Over growing epiphyses in children	Some authors are concerned about the effect of electromagnetic waves on cell growth of epiphysis; consider it a precaution.
Obese patients	Precaution or contraindication, depending on amount of subcutaneous adipose tissue that may overheat during diathermy, particularly capacitive diathermy.
Use of diathermy near other medical electrical devices, especially near patients who have electrodes attached to their skin	Electromagnetic waves from diathermy devices may interfere with the function of these devices and may exacerbate the intensity of stimulation under electrodes.
Over the low back or pelvis in women who are menstruating	A precaution because diathermy may increase menstrual flow.
Atrophic, infected, or damaged skin	Use caution when applying thermal diathermy because of possible sensation and circulatory impairments of skin, intolerance to heat, or spread of infection. Severe conditions are a contraindication.

TABLE 6–16. Contraindications for Diathermy

Contraindication	Explanation
Any metallic object on the patient or within the electromagnetic field	Contraindicated because metals can overheat and burn tissue.
Any metals imbedded in the body	Contraindicated for continuous SWD and MWD because of potential overheating of metal, resulting in tissue burns.
Metal loops imbedded in the body	Contraindicated for all forms of diathermy; can act as an antenna and overheat, burning tissues.
Cancerous tissues	Contraindicated because heating of tissues may promote growth and spread of cancer (malignancy).
Hemorrhage or increased tendency of hemorrhage	Contraindicated because heating of tissues may exacerbate or cause hemorrhage.
Cardiac pacemakers, implanted stimulators, including any device with lead wires attached or unattached	Electromagnetic waves from diathermy can interfere with the function of these devices and cause malfunction and damage to tissues.
Acute injury or inflammation	Contraindicated for thermal diathermy because heating of tissues may exacerbate swelling and inflammation and damage tissue.
Pregnancy	Contraindicated because the effects on the fetus are unknown and may be harmful.
Joints or organs containing high levels of fluid, including the eyes or testes	Contraindicated because thermal diathermy can rapidly heat fluids in these structures, possibly causing injury.
Synthetic materials such as clothing, pillows, and bed coverings	Contraindicated because thermal diathermy may overheat these materials, which can burn tissues.

Continued

TABLE 6–16. Contraindications for Diathermy—cont'd

Contraindication	Explanation
Head, face, or TMJ for patients wearing contact lenses	Contraindicated because thermal diathermy may cause overheating of perforations in lens, which may damage the eye.
Unconscious patients or patients who are mentally confused	Contraindicated because patient is unable to determine amount of heating occurring in tissues; confused patients may not be able to maintain position during treatment.
Over moist clothes, dressings, or accumulations of perspiration on the skin	Thermal diathermy will heat fluids faster, which may cause burns.
Loss of sensation in area of body to be treated	Patient will not be able to perceive the heating effects that could cause tissue damage.
Patients with severe circulatory impairment (peripheral vascular disease, etc.)	Ability of the circulatory system to adjust to increased tissue temperature will be compromised, increasing the risk of burns.

contraindicated whenever heating of tissues may cause harm (see Chapter 3 for precautions and contraindications for heating of tissues). Any significant tissue heating should be avoided if acute inflammation is present, if there is a tendency for hemorrhage, if perfusion is restricted by vascular compromise, or if the patient is febrile or has insensate skin that prevents accurate reporting of heat sensation.

A unique concern for diathermy is the presence of metal on the patient or in the treatment field area. Metals are highly conductive. Electromagnetic waves will take the path of least resistance and pass through metal more readily than body tissues, resulting in faster heating of the metal, which can burn the skin. Patients receiving diathermy should lie on nonmetallic tables or sit on nonmetallic chairs. Metal objects within the immediate treatment area should be removed. This includes metal jewelry or piercings on the patient; clothing that contains metal zippers, hooks, buttons, or fasteners; or braces or devices that contain metal. The presence of imbedded metal in a patient has long been considered a contraindication for diathermy.

Scott[149] explained how metal acts as a "shunt" of the electric field and produces little or no heating in the metal but causes great dissipation of the heat in the tissues at the ends of the shunt. Therefore, the metal itself is unlikely to cause burning of tissues; however, the tissues at the end of the shunt may be burned. The longer the metal, the greater the shunting effect and the greater the chance of tissue damage. Small pieces of metal imbedded in body tissues are unlikely to cause tissue burning. Draper et al[150] performed PSWD (27.12 MHz, 400 μsec, 800 pps, 48 W for 20 minutes) using a monode induction coil applicator over the elbow of a 39-year-old woman who had an internal fixation with metal screws and a titanium plate in the joint. The patient reported a gentle, warm feeling and no discomfort. The physician who later removed the hardware reported no evidence that the metal implants had overheated. Seiger and Draper[151] reported on a case series of patients with surgical metal implants in ankles that received PSWD (27.12 MHz, 400 μsec, 800 pps, 48 W for 20 minutes). All the patients reported a mild vibration and no pain or burning, and all had improvement in ankle ROM without any evidence of harmful effects attributable to PSWD. The authors advise caution when applying PSWD over metal implants in peripheral joints. Draper[152] states that PSWD can be safely performed over implanted metal fixation devices such as rods, plates, and screws. However, application over imbedded metal looped wires is contraindicated because these act as an antenna and will become overheated by electromagnetic waves and can burn surrounding tissues.

No studies have shown whether continuous SWD and MWD are safe over metal implants, so these forms of diathermy should still be considered a contraindication. Copper-bearing intrauterine contraceptive devices contain a small amount of metal that does not overheat when exposed to diathermy.[153–155] Applying diathermy to the pelvis or low back of patients who have these devices is *not* contraindicated.

Key Point! All external metals, including jewelry and metal tables and chairs, must be removed from the area of diathermy exposure prior to treatment. Metal surgical fixation devices imbedded in a patient, such as screws and plates, are unlikely to be a problem when PSWD is administered over these devices. All forms of diathermy, including PSWD, must not be administered over metal loops of wire imbedded in the body.

No form of diathermy should be used on patients who have implanted electronic devices such as pacemakers, defibrillators, or neurostimulators. Wires attached to these devices can act as antennas and cause significant tissue damage. Wires are sometimes left in the body after these devices are removed, so clinicians must be certain that no wires remain before using diathermy on these patients.[156]

Organs and tissues with high fluid volume have a higher conductivity and will heat more quickly than surrounding tissues when exposed to electromagnetic fields from diathermy devices. This may result in overheating and tissue damage, so it is best to avoid exposing the eyes, testes or ovaries, fluid-filled joints, and internal organs to diathermy.[156] Some clinicians[157] recommend not applying diathermy over epiphyseal tissues because of the possibility of adversely affecting the growth plate; however, this has not been confirmed by research using human subjects. Diathermy over cancerous tissues is contraindicated because of the danger of increasing blood flow to the cancer cells and promoting malignancy.[156,157]

Clinicians must use caution when exposing any synthetic materials (such as nylon, foam rubber, or plastics) on or in the patient to electromagnetic fields from diathermy devices. Synthetic clothing should be removed from the treatment area. Any pillows and table coverings made from these materials should also be removed. Internal synthetic devices may overheat and cause tissue damage. Scott[158] warned that electromagnetic fields can overheat contact lenses in the eyes. Riambau et al[159] reported a case in which a patient who had a Vanguard endograft repair of an abdominal aortic aneurysm had a thrombosis of the left limb of the endograft after an MWD treatment for lumbar pain.

Clinician Exposure to Electromagnetic Fields

There has been concern about the electromagnetic fields created by diathermy devices and their effect on clinicians and others who may be in proximity of the device during treatment. Many factors determine the amount of electromagnetic field exposure to clinicians during diathermy treatments, including type of diathermy, size and placement of electrodes on the patient, output intensity, and the clinician's location in relationship to the active diathermy device.[160,161] The use of diathermy is not believed to be harmful to clinicians if they follow safety guidelines for limiting exposure to electromagnetic fields.[162–165] These guidelines include maintaining a distance of 1 to 2 m (3.28 to 6.56 ft) from active SWD devices and 0.5 to 1 m (1.64 to 3.28 ft) from active PSWD devices.[161,166]

Female clinicians who are pregnant must consider the effects of exposure to electromagnetic fields on the fetus. Some studies have found weak correlations between clinician exposure to electromagnetic fields from diathermy treatments and spontaneous abortion and congenital malformations.[167–170] Ouellet-Hellstrom and Stewart[171] found a correlation between MWD and miscarriages but no correlation between SWD and miscarriages. At this time, because of the uncertainty of the research, female health-care practitioners who are or may be pregnant can set up diathermy devices for patient treatments but should not remain in the treatment area when the diathermy device is on. Draper[152] considers PSWD a precaution for pregnant clinicians, so remaining in the same room when PSWD is in use is acceptable.

Key Point! Clinicians should limit their exposure to electromagnetic waves from diathermy devices by maintaining a safe distance from the devices during treatments (1 to 2 m from SWD devices, 0.5 to 1 m from PSWD devices). Pregnant clinicians should not remain in the treatment area during SWD and MWD administration. However, this precaution is probably not necessary for PSWD.

Clinical Decision-Making: When Is Diathermy the Treatment of Choice?

A patient presents with a condition that is an indication for diathermy. How does a clinician determine which type of diathermy is best? Or would ultrasound be a better choice? Is diathermy a cost-effective alternative to other interventions for this condition? Conditions that may benefit from deep heating of tissues will require either ultrasound or diathermy, because superficial thermal modalities will not significantly heat tissues that are more than 1 to 3 cm below the skin's surface. (See Table 6-17 for a comparison of ultrasound and diathermy.)

Ultrasound primarily heats dense collagen tissue such as tendons, ligaments, and joint capsules. The area of tissue heating is limited to a small area about twice the size of the sound head. Diathermy heats tissues with high fluid content, such as muscle, and will heat a much larger area than ultrasound. The type of diathermy will depend on the treatment's goal. Vigorous heating of a large area of the body may require SWD (or MWD if available). Inductive diathermy is more effective in heating muscle and will heat fat tissue less than capacitive diathermy. If mild or moderate heating is desired, then PSWD is a good choice. Acute conditions may benefit from PSWD with a mean power intensity set at a less than thermal range or PEMF, which will not heat tissues.

Today's diathermy devices are safer, technologically superior, and easier to use than devices used during the 20th century. However, diathermy use in the United States remains uncommon. Its use is more common in other countries such as Ireland, the United Kingdom, Australia, and Canada.[165] The higher cost of diathermy devices (compared to ultrasound devices and most superficial thermal modalities) may affect clinicians' decision to purchase this modality. Clinicians need to consider the advantages of diathermy compared to other modalities, which include a greater depth of heating, larger area treated, longer duration of thermal effect, and the clinician does not need to remain present during the treatment. Multiple patients can receive diathermy treatment while the clinician is attending to other patients. Consideration of these advantages, weighed against the disadvantages of diathermy discussed previously, will enable the clinician to make a wise decision regarding the use of this modality for patient treatment.

Clinical Controversy

Some clinicians have expressed concern about the safety of administering diathermy to patients and about the safety of the clinicians who are administering the treatments. The radio or microwave frequency waves administered by diathermy devices do not cause cell mutations that could lead to cancer. No harm will likely occur to patients when precautions and contraindications for diathermy are followed. Clinicians should practice "prudent avoidance" to limit frequent direct exposure to electromagnetic waves. Pregnant clinicians should follow these same recommendations. The risks and benefits of all treatments must be considered by health-care clinicians. Considering the benefits of the deep-heating effects of diathermy and the low risk of harm, diathermy can be safely and effectively administered to patients.

TABLE 6–17. Comparison: Diathermy and Ultrasound

Clinical Aspects	SWD/PSWD	Ultrasound
Type of energy	Electromagnetic	Mechanical
Depth of tissue heating	3–5 cm	2–3 cm
Type of tissue heated	Muscle, fat, skin	Mostly dense, high-collagen tissues (tendons, tissue interfaces)
Rate of tissue heating	Constant	Fluctuates with movement of sound head
Area affected	About 200 cm^2 (using induction drum)	About 2–15 cm (depending on size of sound head)
Heat retention	Moderate	Minimal
Treatment time	15–30 minutes	5–15 minutes
Clinician attendance during treatment	Unattended	Attended
Average cost of device*	$8,000–$10,000	$1,500–$3,000

*Based on quotes from three biomedical companies in U.S. dollars.

Documentation

Consistent and accurate documentation of treatment parameters and the patient's response to treatment is important for justification of continued diathermy treatments, insurance reimbursement, and outcomes assessment. Clinicians are encouraged to collect and analyze outcomes data and publicize effective and ineffective responses to diathermy interventions.

Documentation Tips

- Type of diathermy: SWD, MWD, PSWD, or PEMF
- Method: capacitive (electric field), induction drum, or sleeve (magnetic field)
- Body part exposed to the electromagnetic waves
- Patient's position during treatment
- Duration of treatment
- Peak power in watts (W) for SWD and MWD
- Pulse/burst frequency and duration (if adjustable) and average power for PSWD and PEMF
- Dosage (I through IV) or type (nonthermal, mild, moderate, or vigorous heating)
- Other modalities or procedures performed
- Patient response (positive or negative comments, effect on pain, skin color changes, sweating, or other responses to treatment)
- Goals of treatment and outcomes

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CHAPTER 7

SPINAL TRACTION

Charles Hazle, PT, PhD

FOUNDATIONS OF TRACTION

The practice of using traction—applying tensile forces to the long axis of the spine—to treat patients with spinal-mediated pain has been advocated for centuries. Modern support for traction stemmed largely from the British physician James Cyriax, who in the 1940s recommended using traction to treat patients with suspected disc lesions.[1] Practitioners from Cyriax's time to those who use more recent treatment approaches, including those developed by Australian physiotherapist Geoffrey Maitland, also proposed traction to be of value in treating patients with spinal disorders.[2,3] The rationale for this intervention in patient care may have evolved, but the fundamental concept of its usage has remained remarkably consistent over the years.

In the current evidence-guided era, the use of traction has been more closely examined for effectiveness in patient care. Many practitioners continue to cite traction as an essential clinical modality, often based on patterns observed in patient care experiences, although objective evidence of its value remains limited. In this chapter, the physiological and biomechanical effects of spinal traction will be described along with the clinical trials employing this modality. Additionally, the conventional traction methods often used by clinicians along with variations on traditional uses will be covered from a practical perspective.

BIOMECHANICAL AND PHYSIOLOGICAL EFFECTS OF TRACTION

Cervical Spine

Among the purported effects of traction is increasing the space between the vertebrae. The theorized value of intervertebral separation is for normalizing morphology—more specifically the disc's position and increasing the dimensions of the intervertebral foramen containing the spinal nerve root. Imaging studies in vivo and with cadaveric specimens have investigated the theoretical effects of traction on the cervical spine motion segment.

One study using fresh cadaveric human specimens[4] and another using live subjects[5] yielded nearly identical results. The dimensions of the intervertebral foramina

were measured with computed tomography (CT) and radiography, respectively. In both studies, traction with the cervical spine in a neutral position significantly increased foraminal size. Combined cervical flexion and traction did not increase foraminal size greater than either flexion or traction alone. Other studies have documented a decrease in pressure within the intervertebral foramen[6] and an increase in the dimensions[7] of the intervertebral foramen with flexion of the cervical spine; these findings likely serve as the basis for including flexion when applying cervical traction.

In live humans, cervical intervertebral disc spaces were observed to increase with traction of almost 30 pounds while positioned in neutral and in flexion. Similar changes in the intervertebral disc spaces were not observed when traction was administered in extension of the cervical spine. Separation of the zygapophyseal joints was achieved only with traction in extension. In this study, however, the investigators reported that traction in this position was intolerable for many subjects, thereby limiting its clinical utility.[7]

The effect of traction on the disc has been of primary interest in other investigations. For example, CT was used to assess cervical disc herniations in 13 subjects before and immediately after 20 minutes of traction.[8] After traction, the mean area of the disc herniation was reduced and disc space was increased. Further, the spinal canal area and vertical dimension of the cervical spinal column were increased. Notably, the duration of these effects was not measured.

Multiple studies have examined the effect of traction on the musculature surrounding the cervical spine, with remarkably varied results. Interestingly, elevated,[9,10] diminished,[11,12] and no detectable changes[13] in muscle activity have all been observed as a result of cervical traction.

The effect of cervical traction on autonomic function has also been studied in two recent investigations. Significant changes were observed at higher magnitudes of traction (30% of body weight) but not at lower levels typically used in routine clinical practice. The changes in autonomic function noted in the subjects may have been associated with discomfort occurring from inordinately high tension levels of the experimental traction.[14,15]

In two studies employing animal models and, thus, not specific to a region of the human spine, histological changes subsequent to the application of traction were evaluated. Using a porcine model, traction was observed to encourage fluid exchange and nutrient transport through the anulus fibrosus. The investigators suggested that molecular convection and cell viability of the degraded discs increase with traction treatment.[16] In a rodent model, traction reportedly provided significant beneficial effect in maintaining disc height of degenerated discs, potentially retarding the process of degeneration.[17]

Lumbar Spine

The effects of traction on anatomic spatial relationships of the lumbar spine and the level of trunk muscle activity have also been evaluated by several investigators. In one study,[18] trunk muscle activity in 29 asymptomatic subjects increased initially with the application of traction but quickly subsided to prior levels. Whether traction was continuous or intermittent yielded no difference in the observations of muscle activity. In assessing for other potential responses, another investigation observed an increase in blood flow in the musculature commensurate with lessening pain associated with the use of traction.[19]

More than 60 years ago, Cyriax proposed that negative pressure created by traction draws in a protruding disc, reducing the extension of disc tissue beyond the vertebral body margin.[1] Intradiscal pressures have been observed to increase during active lumbar traction (distractive force by the subject's effort).[20] This is consistent with observations that intradiscal pressure increases with trunk muscle activation even with concurrent distractive force applied to the lumbar spine.[21]

Of relevance to possibly decreasing nerve root compression in radicular disorders arising from disc herniations, a reduction of disc material beyond the borders of the vertebral bodies was noted in 21 of 30 subjects as assessed by CT. The reduction effect was greatest in patients with median herniations and lowest among those with lateral herniations.[22] These measurements were completed before and during the application of lumbar traction. The investigators did not attempt to measure the persistence of these changes after traction.

In another trial,[23] the disc dimensions of 24 subjects with confirmed lumbar disc herniations who received traction for treatment were compared by CT with a control group of 22 subjects who had disc herniations but did not receive traction. Otherwise, both groups received the same treatment with physical therapy modalities and

medications over a course of 15 sessions. Those subjects who received traction demonstrated a substantially greater reduction of the total area of herniated disc material as measured by CT.

In another study using young asymptomatic adults, the effects of lumbar traction on stature and postural alignment were assessed. At 50% of body weight, changes were noted immediately after the conclusion of traction, but the observed effect was short-term and absent within a few minutes.[24]

Although initially noteworthy, the lasting effect of the changed anatomical relationships caused by traction remains in question. Prior to use of these sophisticated imaging analyses, a cadaveric study of traction[25] determined the elongation induced during traction did not continue beyond 30 minutes after traction removal. Similarly, another study[26] demonstrated that a return to pretraction relationships occurred only 10 minutes after the release of traction.

Pain occurring distal to the knee with straight leg raising was measured before and after traction in a group of subjects with positive straight leg raising below 45° of hip flexion.[27] An increase in the angle of hip flexion prior to lower-extremity pain provocation during straight leg raising was observed immediately after lumbar traction at magnitudes of 30% and 60% of body weight. The straight leg raise angle in these subjects was compared to subjects receiving no traction and to subjects receiving traction at 10% of body weight. Duration of these changes was not measured (Box 7-1).

Box 7 ■ 1 Biomechanical and Physiological Effects of Traction: Synopsis of Literature

Cervical Spine

- Fluid exchange and nutrient transport within the disc may be enhanced. The duration of any such effect is unknown.
- There is evidence that intervertebral foramina dimensions increase during traction application. Whether this can be further influenced by positioning in flexion or lateral flexion has not been established.
- Limited evidence suggests that disc herniation extension tends to be reduced when measured immediately after traction.
- Evidence is conflicting as to the effects of traction on the activity of cervical spine musculature.
- The duration of any observed biomechanical or physiological effect is not known.

Lumbar Spine

- Fluid exchange and nutrient transport within the disc may be enhanced. The duration of any such effect is unknown.
- During passive traction, intradiscal pressures can be reduced or become negative. Traction from patient-generated forces may increase intradiscal pressures. These pressures are thought to rapidly return to their prior state when traction ceases.
- The expanse of herniated disc material is suggested to reduce in some subjects during traction. Most single-observation studies suggest the effect is temporary. A cumulative effect with repeated traction sessions may occur, but evidence of such is very limited.

BASIC APPLICATIONS OF CLINICAL TRACTION

Several methods of traction have been used to treat patients with mechanical neck and back pain; the method used was often based on tradition rather than evidence of specific clinical value. Individual practitioner and patient care experiences often serve as the stimulus for the development of these general practices, which are subsequently propagated by convention. The lack of substantial data to support particular utilization guidelines has allowed for numerous permutations in the elemental practice of applying tension to the spine for symptom relief. To adequately understand traction and its uses, this chapter provides details on the basic features routinely available on modern traction devices. A comprehensive description is not possible due to the many uses of this modality.

Components of the Traction Table

Traction tables allow patients to receive treatment of the cervical and lumbar spine regions in relative comfort (Fig. 7-1). The table is adjustable in height by a hand or foot control switch. At one end of the table is the mechanical traction device, consisting of an electric motor and a control panel. The traction unit usually produces the tensile force via a cable that extends from the electric motor. The cable is attachable to the traction harnesses, which directly contact the patient. With the evolution of computer technology, the control panels of traction units have become increasingly sophisticated. Many modern models have touchscreen features and the

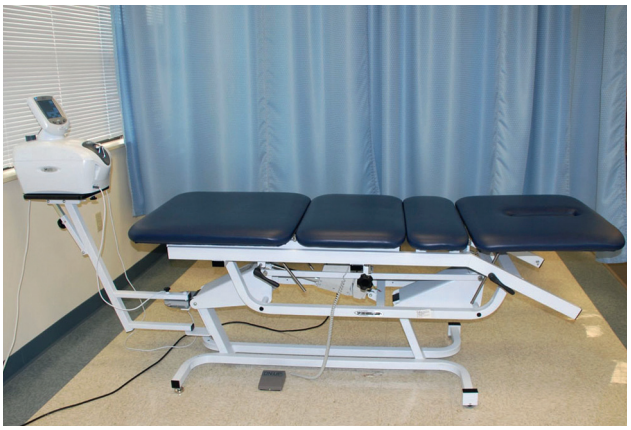

Fig 7 ■ 1 Traction table.

capacity to adjust numerous variables in the delivery of traction (Fig. 7-2). Treatment duration, cycle times, tension levels, and progressive or regressive steps in tension can be programmed into a treatment session with these controls.

Most traction tables also have a split top. The table surface, with its multiple segments, can be separated to minimize the friction from the patient's body on the table surface when applying traction to the lumbar spine. The section of the table nearest the motor can be unlocked so it glides within tracks that are set on rollers on the table's frame (Fig. 7-3). This allows the patient's pelvis and lower extremities to be moved while the remainder of the trunk is stabilized on the nongliding sections of the table. Thus, traction can be more accurately administered to the lumbar spine by significantly reducing the friction provided by the patient's superincumbent body weight. The nongliding segments of the

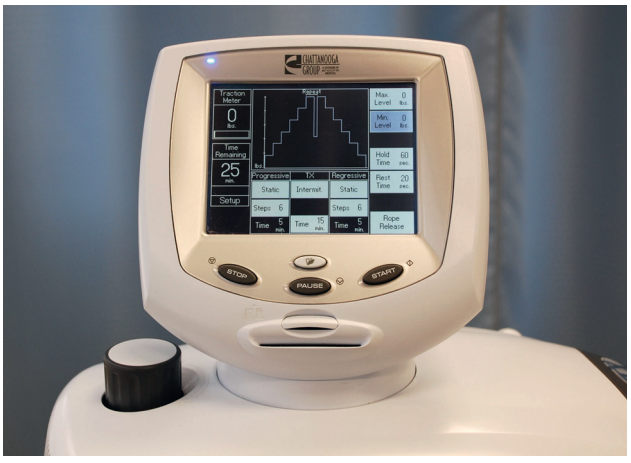

Fig 7 ■ 2 Traction control panel.

Fig 7 ■ 3 Separable traction table surface.

table often have adjustable tilting features to create more options for traction or to facilitate patient comfort with positioning.

Most commercially available traction tables now routinely have a removable harness unit that allows traction to be applied to the cervical spine (Fig. 7-4). These

Fig 7 ■ 4 Occipital harness for cervical traction.

harnesses usually snap-fit or pressure-fit into a latch mechanism on the motorized portion of the table. The occipital harnesses have mobile and stationary segments. Adjustable padded wedges on the harness directly contact the patient's head and neck along with a flat padded area for the occiput. The wedges fit snugly against the posterolateral aspects of the patient's cranium and are often capable of being approximated toward the patient's midline with a screw knob adjustment to prevent slipping during traction. The padded wedges are affixed to the movable segment of the harness, and the harness slides along the stationary portion via the tension produced by the traction motor. Occipital harnesses usually have a strap that can be tightened over the patient's forehead to secure the head in the harness (Fig. 7-5). The need to secure the patient's head firmly is greater with higher amounts of tension because the potential for the harness to slip increases. At lower levels of tension, practitioners often avoid using the strap for patient comfort.

Another standard feature of traction tables is a patient-controlled safety switch (Fig. 7-6). Patients receiving traction usually have intervals of indirect supervision. Thus, their safety is enhanced with a manually activated switch that allows them to immediately release the traction while sounding an alarm for assistance. Patients who experience anxiety with an unfamiliar intervention are often comforted by knowing that they are in control of the traction unit. Similarly, if patients experience discomfort while receiving traction, the switch allows them to release the tension and notify the clinician.

Traction tables represent significant capital equipment investments for most clinical settings and are often used for many years. At the time of this publication, the price for traction tables with the accompanying motor unit ranges from approximately $4,000 to $19,000, depending on the features and options included.

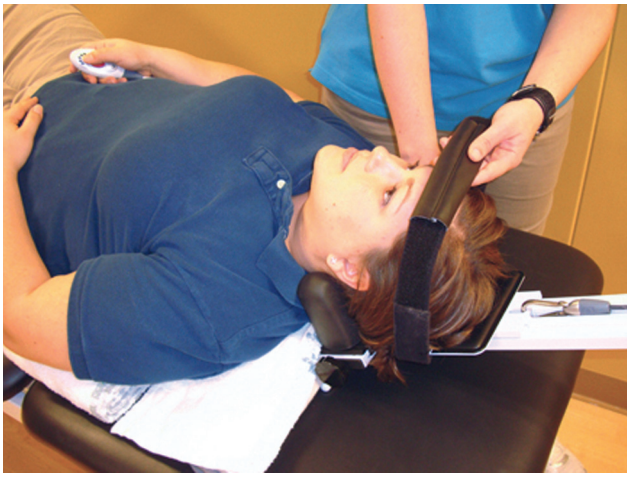

Fig 7 ■ 5 Securing strap of cervical harness.

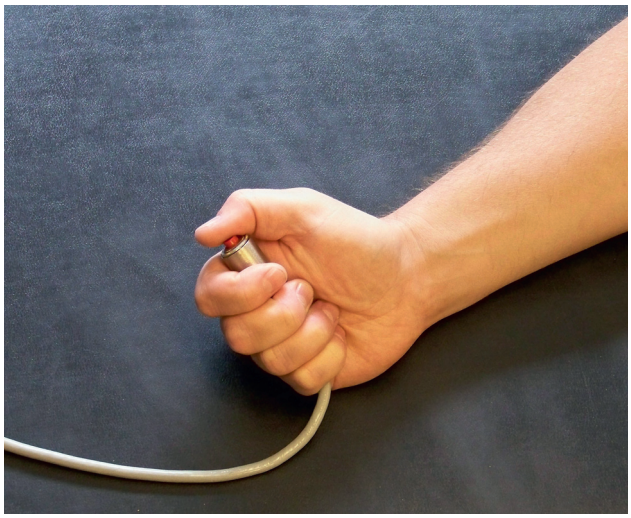

Fig 7 ■ 6 Patient-controlled safety switch.

Cervical Spine Traction: Procedures and Practice

Essential Elements

When describing the physical arrangement of cervical traction, *harness* may not be an ideal descriptor because current clinical apparatuses are considerably different from older devices. Before 1990, the portion of the equipment directly contacting the patient usually consisted of straps that encircled the patient's occiput and chin in order to deliver a superiorly oriented force. Practitioners grew concerned, however, about the force being applied to the mandible and through the temporomandibular joints with these harnesses. As a result, occipital-contact-only harnesses were developed and have now become universally adopted. These devices are generally accepted as more comfortable for all patients in addition to avoiding compressive force through the temporomandibular joints.

The typical preparatory steps for a cervical traction treatment session include the following:

- Step 1: The immobile portion of the cervical harness is first attached to the motorized unit housing or frame to provide for a stable base on which the mobile portion will operate (Fig. 7-7).

Fig 7 ■ 7 Step 1: Attachment of cervical harness to motor.

- Step 2: The cable is slackened from the motor, usually by a release lever or an electronic switch, and then linked to the mobile portion of the harness (Fig. 7-8).
- Step 3: Any slack in the cable is then removed without changing the position of the mobile portion of the harness (Fig.7-9).
- Patient comfort and relaxation during cervical traction are promoted as much as possible. For this reason, practitioners will often place small, well-insulated hydrocollator packs against the inferior edge of the harness upon which the patient will lie (Fig. 7-10).

Fig 7 ■ 8 Step 2: Slackening of cable to allow attachment of mobile portion of harness.

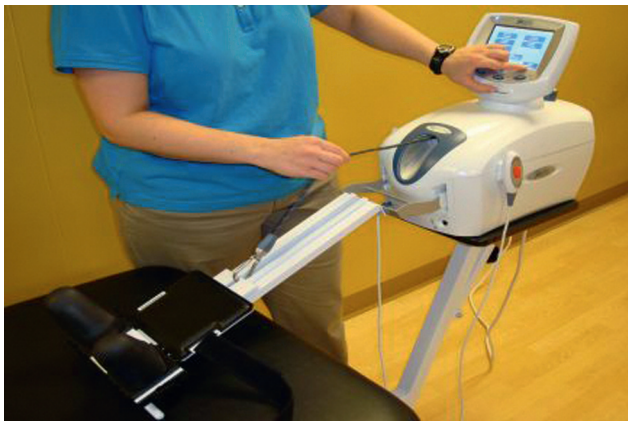

Fig 7 ■ 9 Step 3: Removal of cable slack.

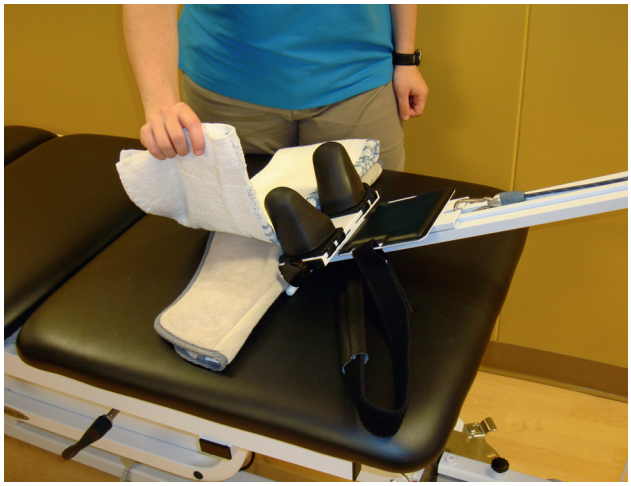

Fig 7 ■ 10 Placement of optional small hydrocollator pack.

Most patients find the warming sensation from the hydrocollator packs across the upper thoracic and scapular regions relaxing, although the packs probably have negligible therapeutic value. Caution must be taken to prevent the hydrocollator packs from overheating the patient's upper back. This is particularly a concern in older patients who perhaps have less ability to dissipate heat.

- Step 4: The harness is opened and the patient is asked to lie supine on the table with the head placed on the flat padded area of the harness (Fig. 7-11).
- Step 5: Proper positioning of the patient in the harness is important for comfort (Fig. 7-12).
- Step 6: If the padded wedges are positioned too high relative to the patient's mastoid processes, slippage may occur during the traction session. In addition, patients

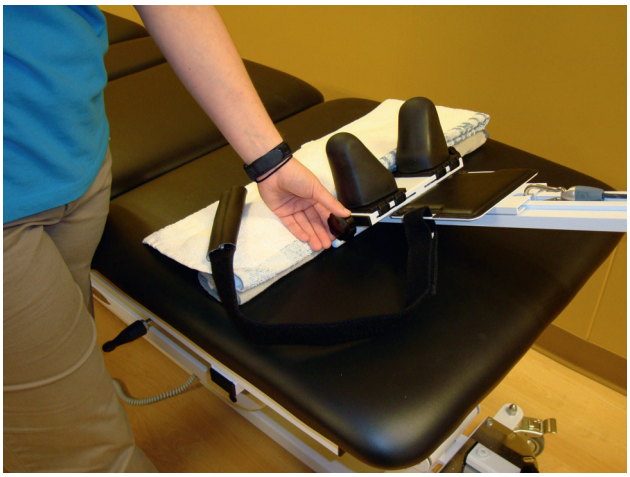

Fig 7 ■ 11 Step 4: Opening of the harness to accommodate the patient.

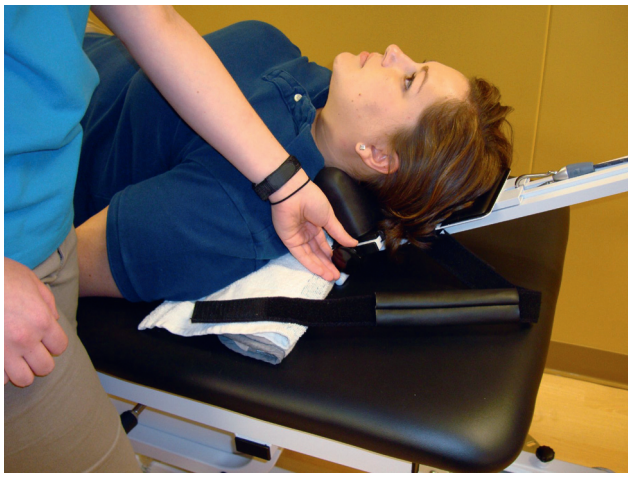

Fig 7 ■ 13 Step 6: Adjustment of harness and padded wedges to patient.

Fig 7 ■ 12 Step 5: Positioning of the patient in the cervical harness.

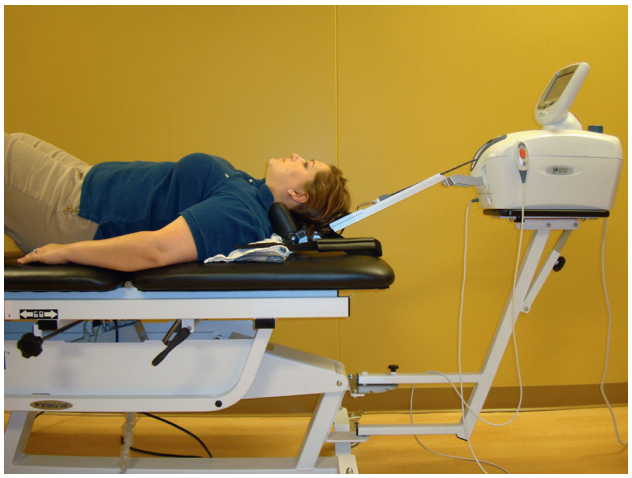

Fig 7 ■ 14 Step 7: Angle of cervical traction.

often report discomfort if the wedges are positioned too high, compressing the posterior cranium. Careful positioning of the harness and padded wedges usually allows the wedges to contact more along the inferior aspect of the cranium. The adjustable wedges are then approximated toward the midline to securely fit the harness to the patient's occiput (Fig. 7-13). Caution must be used to not overtighten this adjustment, which would cause an uncomfortable compressive sensation on the patient's neck.

- Step 7: The angle of the traction harness is adjusted, if necessary, for the relative amount of cervical flexion and any desired lateral flexion. Sagittal plane positioning is usually accomplished by adjusting the table height relative to the fixed height motor and control unit, whereas any lateral flexion occurs from relocation of the mechanized traction unit to either side (Fig. 7-14).
- Step 8: Positioning with one to two pillows or perhaps a bolster placed under the patient's knees can help promote comfort. Some practitioners place support under the arms proximally with small rolled towels. In one study, patient subjects preferred support under the upper extremities.[28] Such positioning and support enhances patient relaxation and may be particularly important in those with excessive thoracic kyphoses or remarkably protracted scapulae (Fig. 7-15). Practitioners will discuss with the patient the sensory experiences and responses they should expect while receiving traction. The patient should be instructed to notify the clinician immediately if pain increases or if the patient becomes uncomfortable in any way during the session. To ensure a mutual understanding of this communication, ask patients to verbalize their comprehension of these instructions.

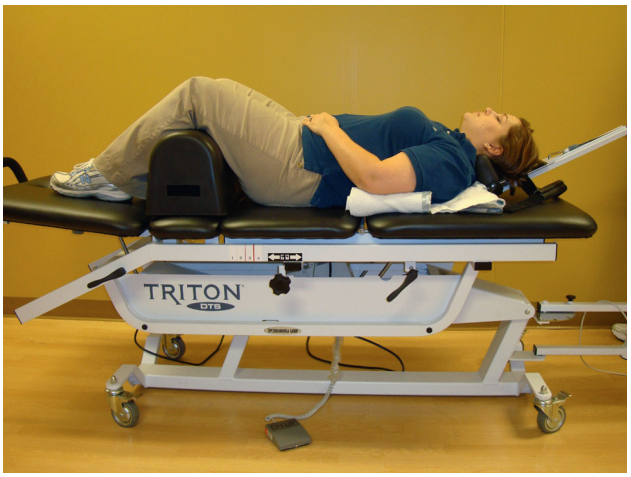

Fig 7 ■ 15 Step 8: Positioning the patient's upper and lower extremities for comfort in cervical traction.

- Step 9: The patient is provided with an aid to call for assistance or is shown the safety switch (Fig. 7-16).
- The chosen settings for the duration and dosage of the traction unit are programmed into the control panel and traction is initiated.
- The prudent clinician will directly observe the traction unit mechanics and the patient response when the session begins. A minimum of 2 to 3 minutes of observation is recommended. This provides further opportunity to ensure the patient's comfort, assess an immediate response, and reinforce expected and unintended responses during the treatment session.

Mechanical Preparation

In addition to patient comfort, the actual arrangements of the table and harness angle are also variables that affect cervical traction, particularly in reference to the angle of cervical spine flexion as measured by the harness relative to the horizontal table surface (see Fig.7-14). Many practitioners, however, have methods of choosing the angle of application or the line of pull for the cervical traction harness, such as selecting the angle depending on the level of the cervical spine that is most symptomatic. For example, the lower cervical spine levels are often treated with greater amounts of flexion while the middle and upper levels may be treated with considerably less flexion. As described earlier, the selection of traction angle based on presumed changes of anatomic relationships during traction has not been validated.

Other practitioners choose a more simplistic approach of asking the patient to report the angle of greatest comfort and adjust the table height accordingly. In addition to the sagittal plane angle, some practitioners will use a lateral flexion angle, presumably to have greater distractive effect on the side of the cervical spine with greater involvement, in the presence of asymmetrical or unilateral symptoms (Fig. 7-17). Most traction tables now have the option of setting the motor and control unit at angles in the patient's coronal plane to achieve traction in cervical lateral flexion. Whether a particular arrangement of the cervical harness results in

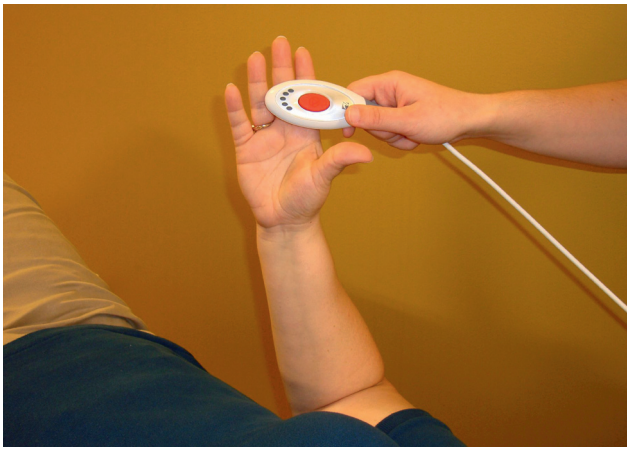

Fig 7 ■ 16 Step 9: Providing the patient with the safety switch or another aid to call for assistance.

Fig 7 ■ 17 Positioning of cervical traction with right-side bias.

better patient outcomes than other harness setups has not been validated and remains a convention. Decision-making in individual patient care situations based on examination findings and positions of relief may suggest a particular method; however, generalizations about this aspect of traction decision-making must be made with reservation because of a lack of evidentiary support.

Traction Dosage and Decision-Making

In addition to the angle of traction, several other variables are at the discretion of the practitioner, such as the amount of tension, the duration of treatment, and the timing of the cycle with intermittent traction. These variables may collectively be considered traction dosage. Again, the use of accepted, if not substantiated, practices are generally seen in reference to these parameters. Practitioners often view traction dosage as analogous to the grading of manually applied passive movements to the spine (Box 7-2).

Comfort is a foremost consideration with patients receiving traction. As a rule, the amount of tension applied to the cervical spine should not cause or increase symptoms. In the presence of peripheral symptoms, possibly of nerve-root origin, practitioners generally agree that distal symptoms warrant particular observation for changes during and immediately after traction. If the patient reports any worsening of upper-extremity symptoms during a treatment session, whether pain or paresthesia, traction should immediately cease. Similarly, if symptoms worsen after traction, the practitioner should reconsider the traction dosage or determine if traction is an appropriate treatment option at that time.

Box 7 ▪ 2 Variables at Practitioner's Discretion in Delivery of Cervical Traction

- Static or intermittent
- Angle of application
- Dosage
  - Amount of tension
  - Duration of traction
  - Cycle
    - Total cycle duration
    - Proportion of time of maximum vs. minimum tension
    - Inclusion of ascending or descending steps at initiation or conclusion

The practitioner's assessment of the acuity and irritability of the patient's condition is part of this reasoning process. Mechanical pain syndromes characterized by pain that is quickly and easily increased with minimal movement or provocation generally suggest conservative traction dosages initially. Symptoms not highly irritable or acute may allow tolerance of greater dosages. Thus, the practitioner is challenged to find a threshold that has a beneficial effect without applying excessive force or provoking symptoms.

Key Point! After deciding to use traction for an individual patient, most practitioners choose a conservative traction dosage for the initial application. This will establish a favorable, or at least neutral, patient response to the trial treatment.

Within the context of the patient having been recently evaluated, perhaps with provocative maneuvers, assessing the patient's response during and subsequent to the first traction session requires caution. A common occurrence is for the patient to report a modest pulling or stretching sensation within or immediately adjacent to the spine during treatment. This is not an indication to stop or alter the traction. Indeed, frequently, patients will describe this stretching or pulling sensation favorably, with the sensation often diminishing over the course of the traction session. Practitioners often hypothesize that this sensation may indicate an improvement of soft tissue extensibility. Thus, such a response is generally viewed as favorable.

Tension amounts typically range between 10 and 25 pounds for the upper limit of intermittent cervical traction.[29–32] Although continuous and intermittent traction are possible on most tables, the majority of practitioners will use intermittent traction, cycling between two levels of tension for the duration of the session. For the lower level of tension, practitioners often use approximately one-half the level of tension as the upper level for intermittent traction. If continuous traction is chosen, less tension and total duration than the upper level of intermittent traction are usually selected.

With cervical traction applied in the supine position, as opposed to the older practice of seated traction with a vertical pull, the weight of the head is less of a factor in the net amount of tension applied to the cervical spine. The angle of the traction harness, however, may

be a factor in the net tension applied. A greater angle of cervical flexion will increase the vertical force component of the head's weight, whereas the vertical component will be negligible with a low flexion angle. Thus, to achieve a comparable net traction on the cervical spine, the amount of tension programmed into the traction unit may need to be relatively larger to compensate for a greater flexion angle. The practitioner may elect to increase the tension setting by an increment of 2 or 3 pounds at the greater flexion angle and then reassess the patient's response for comfort and symptom reduction.

The treatment session usually lasts 10 to 20 minutes, as determined by the overall dosage and the acuity and irritability of the patient's condition. In the absence of well-established criteria, this is arbitrary, as is the timing of the cycles with intermittent traction. A cycle proportion of 30 seconds at the greater tension to 10 seconds at the lower tension of intermittent traction is common, but wide variation of this occurs due to therapist preference. With this cycle proportion and total time being substantial variables at the practitioner's discretion, caution should be used when selecting these amounts based on the individual patient characteristics.

Patients receiving traction will also occasionally experience a rebound effect, characterized by a reduction of symptoms during traction followed by an increase and persistence of symptoms for minutes or hours after the session ends. When a rebound effect occurs, detailed communication between the practitioner and patient is required for accurate interpretation. A brief, transient increase of centrally perceived symptoms after traction may be a one-time localized tissue response or may be an indication for a lower traction dosage while continuing with the original plan of care. Conversely, an increase of symptoms lasting several hours, particularly those including distal symptoms, demands that the practitioner reconsider using traction. When interpreting this feedback from the patient, the practitioner must also consider concurrent interventions being administered, along with the patient's activities and behaviors after the first traction session. Thus, practitioner–patient communication is paramount in appropriate decision-making for traction, particularly after the initial trial or significant changes in the dosage.

After a traction session ends, common practice is to release the tension from the harness, loosen the harness from the neck, and allow the patient to rest for approximately 5 minutes before rising. The patient should also be allowed to rest briefly in a sitting position after arising from supine to minimize any positional hypotension. A patient reporting dizziness or feeling faint during or immediately after traction or upon returning to sitting demands close observation and a check of vital signs. The patient should not rise from the table until such a response subsides. A prolonged response may warrant a medical consultation for underlying conditions, and the practitioner should reconsider using traction with this patient.

Before deciding to use traction in subsequent sessions, the practitioner should carefully interpret the patient's response to the prior session. If the patient reports temporary or lasting symptom relief (particularly with peripheral pain or paresthesia) and greater function, this suggests a possible benefit. Other objective evidence indicating favorable responses to cervical traction can include an increase in cervical range of motion (ROM), increased ability to complete specific daily activities (especially involving the upper extremities), improved upper-extremity reflexes, increased upper-extremity strength (e.g., grip), or normalization of previously observed sensory losses.

Key Point! Among those practitioners who use cervical traction, there is consensus that it cannot be a sole intervention for patients with cervical spine mechanical pain syndromes. Use of therapeutic exercise, manual therapy techniques, and neural mobilizations are clearly supported by evidence[33] for patients with cervical spine dysfunction and are often used in concert with cervical traction.

Lumbar Spine Traction: Procedures and Practice

Essential Elements

Traction has a longer historical use to treat patients with mechanical dysfunction of the lumbar spine than it has for those with cervical spine syndromes. There is a similar, if not greater, lack of substantive evidence as to its value and for the methodology of optimal application, but lumbar traction remains a common intervention employed by many clinicians.

After the decision to use traction, the first variable at the practitioner's discretion is whether to apply it while the patient is prone or supine. No well-developed criteria exist to guide this decision. Some practitioners prefer to use one position exclusively, unless patient comfort or poor response suggests an alternative. Other practitioners will place their patients in the position of greatest comfort as the starting point. For example, if the patient's symptoms are less severe when lying prone, then the practitioner may use prone positioning for traction. Alternately, supine traction may be chosen if the patient achieves greatest relief from lying supine with hips and knees flexed. Yet another factor may be patient age. Older patients, perhaps more likely to have mechanical pain associated with degenerative changes of the posterior elements of the lumbar spine, are thought to typically respond more favorably to a flexed, supine position.

Mechanical Preparation

After the patient's position is determined, the traction table and harnesses can be set with these considerations:

- During supine positioning, well-insulated hydrocollator packs are often used under the patient's back (Fig. 7-18). There is no known direct clinical benefit from the heat application; however, such a measure can promote patient comfort and usually encourages relaxation during the treatment. Because of the superincumbent body weight and limited flexibility for modification after traction has begun, particular caution must be employed to avoid burning or overheating the patient. Older individuals with less ability to dissipate heat may require additional layers of insulation and more frequent monitoring during treatment.
- Step 1: After the hydrocollator packs are placed on the table surface, the pelvic and thoracic harnesses are positioned with consideration for the location of the separation of the table segments and subsequent patient positioning (Fig. 7-19).
- Step 2: As the patient assumes position on the table, the patient's lumbosacral junction or targeted area of the lumbar spine is positioned over the separation between mobile and stationary segments of the table (Fig. 7-20). This allows the distraction to be more

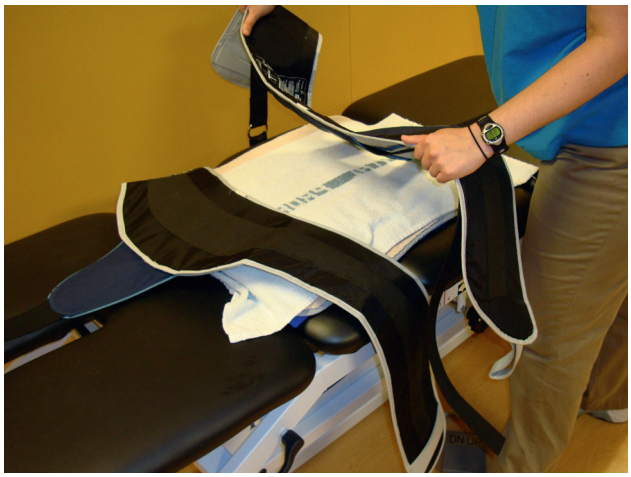

Fig 7 ■ 19 Step 1: Placement of the pelvic and thoracic harnesses for lumbar traction.

Fig 7 ■ 18 Placement of optional large hydrocollator pack.

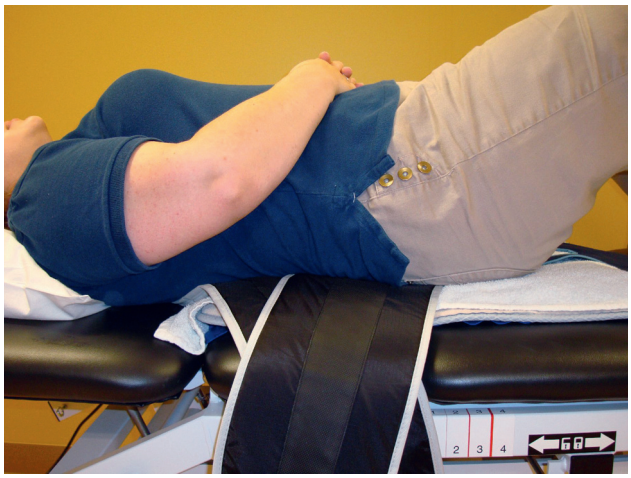

Fig 7 ■ 20 Step 2: Positioning of the target area of traction over the table separation.

localized to the spinal segments of interest when the gliding portion of the table is released.

- Step 3: After the patient is asked to lie supine on the harnesses and the lower extremities are positioned for comfort with the hips and knees partially flexed, support is placed under the knees. Many practitioners prefer a "90-90 position" in which a stool is placed under the calves of the supine patient, resulting in the hips and knees each being positioned at approximately 90 degrees of flexion (Fig. 7-21).
- Step 3 alternative: Other options include use of a bolster under the flexed knees to achieve a comfortable position for the patient (Fig. 7-22).
- Step 4: Precise placement of the harnesses is important for patient comfort and to achieve greatest traction effect. The traditional pelvic harness with two securing straps requires the upper of the two straps securing the harness to be placed superior to the patient's iliac crests. The lower strap will ideally be inferior to the iliac crests but superior to the greater trochanters. Thus, each strap on the pelvic harness will be seated against a bony prominence when traction is applied, minimizing the likelihood of slipping (Fig. 7-23). The thoracic harness is best placed inferior to the widest lateral dimension of the rib cage. Thus, when traction is applied, the thoracic harness will also seat against the bony prominences at the flare of the rib cage. Some newer lumbar traction harnesses have only one securing strap, which may require more attention to detail with fitting the harness (Fig. 7-24).

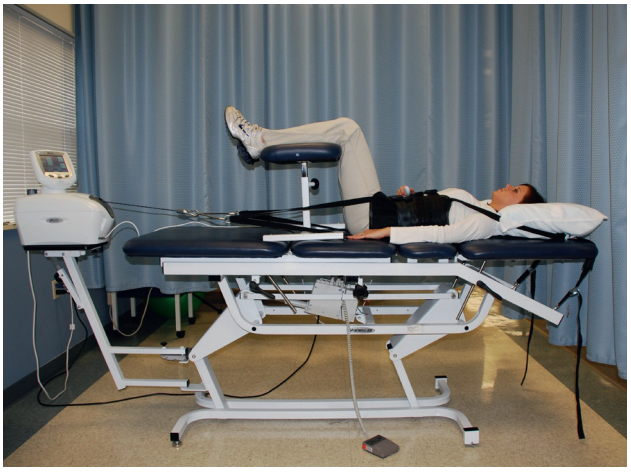

Fig 7 ■21 Step 3: Positioning for "90-90" lumbar traction.

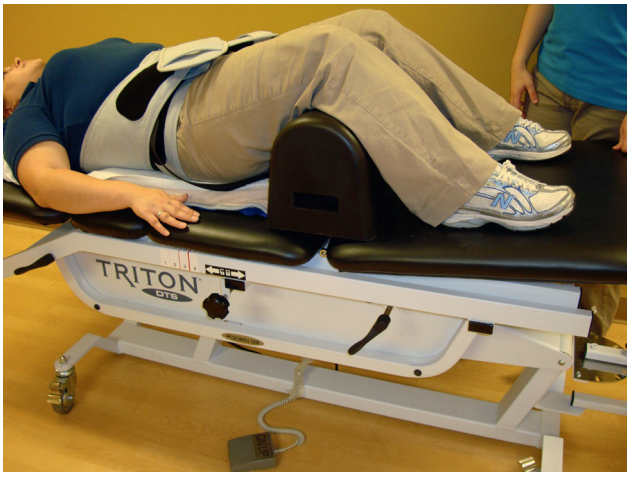

Fig 7 ■22 Step 3, alternative: Positioning for traction in supine with a bolster under the knees.

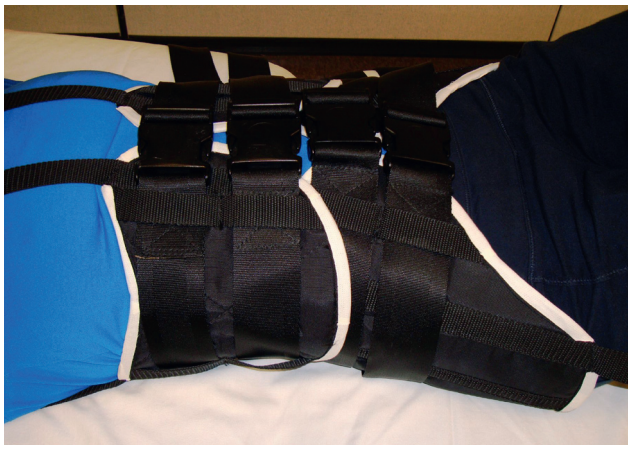

Fig 7 ■23 Step 4: Overlap of thoracic and pelvic harnesses with both seating against bony prominences.

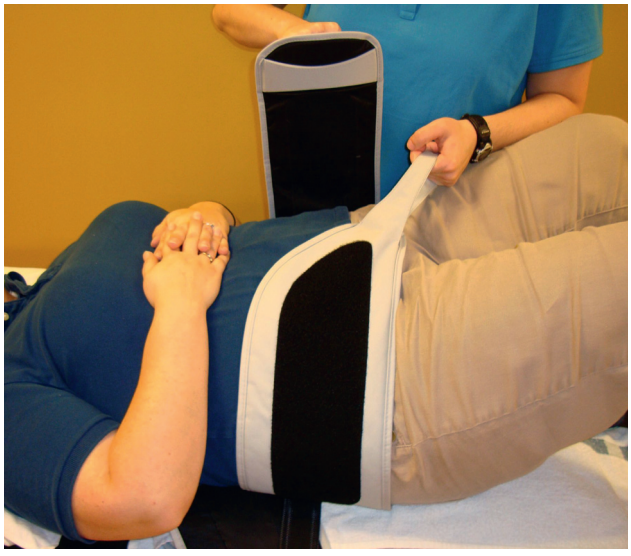

Fig 7 ■24 Traction harness with a single strap.

The straps on both harnesses are clasped and tightened (Fig. 7-25). The extent of tightening is dependent on the amount of traction tension to be used. Larger amounts of tension require the straps to be firmly secured, whereas more modest traction tension does not require the straps to be as tight. The goal is patient comfort while preventing the harnesses from slipping from their optimal placements on the patient's body. For female patients, a rolled towel placed vertically between the breasts before closing the thoracic harness may help prevent discomfort. Clothing can occasionally interfere with the function of the thoracic harness. Multiple layers of clothing, particularly with synthetic materials offering minimal friction or bulky garments, can compromise the thoracic harness's position when tension is applied. If the patient can maintain modesty and remove excess layers of upper-body clothing, a more secure and sustained positioning with the thoracic harness is often achieved.

- Steps 5 and 6: After the harnesses are secured on the patient, slack in the straps anchoring the thoracic harness to the fixed portion of the table is removed to minimize any upper-body movement when tension is applied. The cable is released from the motorized unit (Fig. 7-26) and attached to the pelvic harness (Fig. 7-27). The sequence of some steps may vary, depending on the traction unit and harnesses being used. In all cases, the harnesses are secured first around the patient before slack is removed from the other harness attachments.
- Step 7: After the patient is positioned and the harnesses are secured, the slack in the cable between the harness and motor is removed (Fig. 7-28).
- Step 8: Next, the table surface lock is released, allowing the table segments underlying the lumbar spine and pelvis to separate when the traction tension increases (Fig. 7-29).
- The traction parameters are programmed into the control panel and traction is initiated.
- The prudent practitioner will closely observe the patient and apparatus over a minimum of two to three cycles of intermittent traction, ensuring that the harnesses are secured and do not slip. Particular attention is warranted to avoid allowing the thoracic

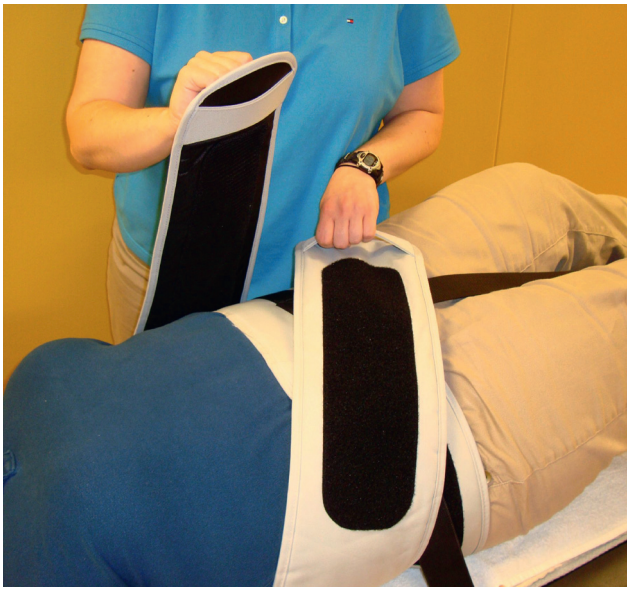

Fig 7 ■25 Tightening of harness in preparation for lumbar traction.

Fig 7 ■26 Step 5: Slackening of cable to allow attachment to the harness.

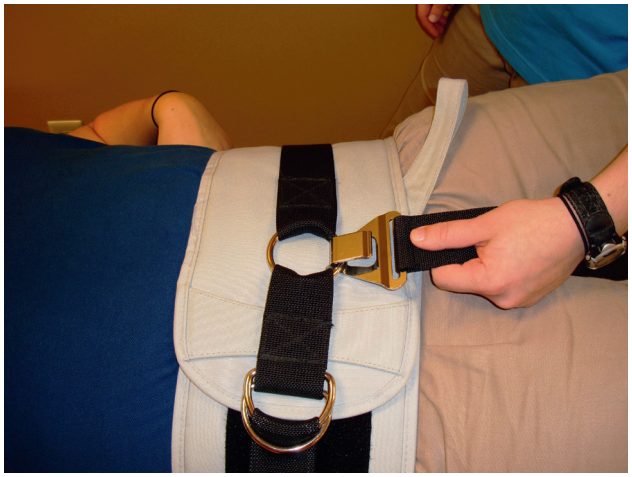

Fig 7 ■27 Step 6: Attachment of cable to pelvic harness.

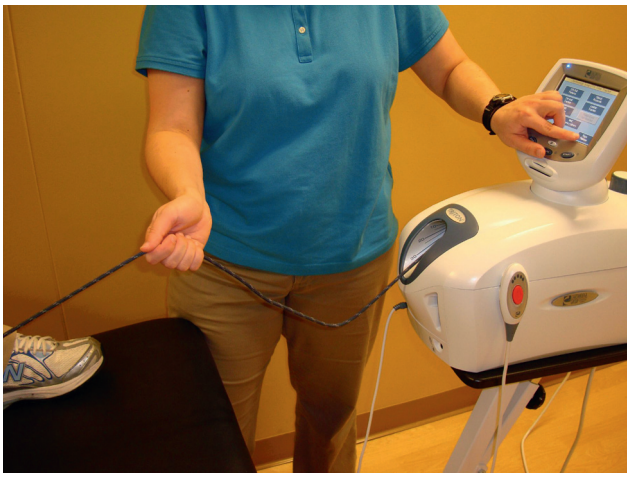

Fig 7 ■28 Step 7: Removal of cable slack.

Fig 7 ■29 Step 8: Release of table lock.

harness to slip superiorly on the patient and into the axillae. Some patients may have body types that make it challenging to adequately secure the harnesses to avoid slippage during traction.

With prone positioning, the harnesses are similarly placed on the table such that the lower lumbar segments or the lumbosacral junction align with the splits of the tabletop's segments (Fig. 7-30). Usually, this allows the patient's face to fit comfortably in the opening of the table's head segment. The tilt of this segment is often adjusted slightly down for greater patient comfort. The inability to communicate face-to-face with the patient during prone traction requires diligence on the practitioner's part to ensure the patient is not having an unfavorable response.

Some practitioners prefer to apply manual mobilizations immediately to the lumbar spine or encourage the patient to complete extension exercises immediately after traction and before weight-bearing through the spine. This may be accomplished more easily with prone traction because only the belts being loosened and the table surfaces being secured are necessary to proceed. If those interventions are chosen to follow supine traction, a nearby table can be brought next to the traction table, allowing the patient to roll prone onto the second table. As described in the section on cervical traction, a rest period after the release of traction is usually warranted before proceeding.

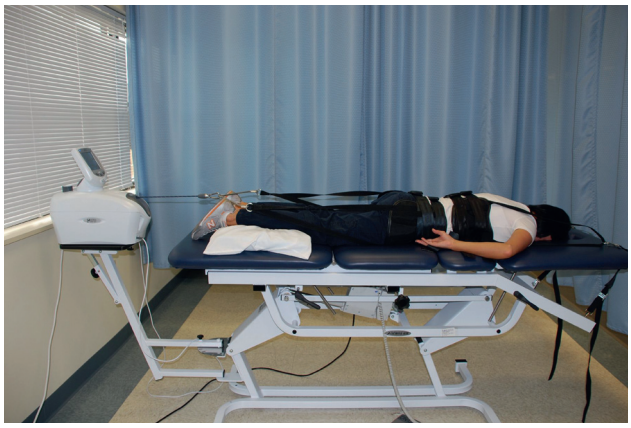

Fig 7 ■30 Positioning for prone lumbar traction.

With prone or supine positioning, the table height and the angle of the traction unit relative to the table can be adjusted, but these adjustments may not exert the same effect as in traction of the cervical spine. Without the stationary arm of the cervical traction harness providing for greater angulation of the tension, only minor variation of the traction tension toward lateral flexion are possible with lumbar traction arrangements.

Traction Dosage and Decision-Making

The variables in lumbar traction tension are similar to those described previously for the cervical spine. Additionally, the practitioner must determine whether supine or prone positioning is preferable. Positioning of the lower extremities may also be a consideration because this influences the alignment of the lumbar spine during traction. Collectively, total duration, cycle time, and amount of tension comprise the traction dosage. Values are usually set conservatively to establish patient tolerance, particularly with conditions that are highly irritable or acute. Use of tension up to approximately one-half of

the patient's body weight is common practice, although this may not be selected for the initial treatment session. Similar to cervical traction, a cycle of approximately 30 seconds at the maximum tension to 10 seconds at the lower tension is typical, although this is subject to practitioner preference. Manipulation of these variables can significantly increase or decrease total traction dosage (Box 7-3).

Communication between the practitioner and patient before the traction session and immediately afterward about expectations and responses is a necessity. Further communication during the next visit is essential to assess the patient's response before doing another treatment. A reduction in pain, particularly with lower-extremity symptoms, along with increased ambulatory or sitting tolerances indicate a positive response to lumbar traction. More objective evidence such as increasing the angle of hip flexion during straight leg raising before symptom provocation along with improvement in deficits in reflexes or sensation also suggest benefit. Assessment of the patient's response to traction must always be made within the context of concurrent interventions and the patient's other activities or behaviors.

Patient Safety

Generally, patients will not be supervised during the entire treatment period. Therefore, patients must have a call system they can use if they need assistance during the traction session. Even though the patient safety switch is available, some practitioners prefer a bell or similar audible call method that patients can use if they merely need to make minor adjustments or if they have a question and do not need to immediately terminate the traction treatment.

Box 7 ▪ 3 Variables at Practitioner's Discretion in Delivery of Lumbar Traction

- Supine or prone position
- Positioning of lower extremities to influence lumbar spine alignment and comfort
- Static or intermittent
- Angle of application
- Dosage
  - Amount of tension
  - Duration of traction
  - Cycle
    - Total cycle duration
    - Proportion of time of maximum vs. minimum tension
    - Inclusion of ascending or descending steps at initiation or conclusion

Key Point! The patient and practitioner must communicate about expectations and anticipated responses from traction to ensure that no harm is done and that the experience for the patient is favorable.

Patients must understand that they should alert the practitioner if they experience any undesirable responses during treatment. Questions should be asked and answered before a patient's first traction experience. The practitioner must ensure that the patient understands that traction should be a comfortable experience, rather than one to be endured for later benefit. If the patient's symptoms worsen, particularly if they are referred or radicular in nature, the patient should notify the practitioner or other clinical staff immediately. Similarly, the practitioner or other clinical staff must frequently assess the patient's comfort and response during the traction session. Patients with highly acute or irritable conditions require meticulous observation to ensure that they are not having an adverse response to the traction.

Safety check: With lumbar traction, a mandatory procedure is to secure the lock for the tabletop segments immediately after releasing the tension at the end of the session. Failure to do so may result in a sudden shift of the mobile portion of the table while the patient is on it or when rising from it, resulting in injury or irritation of symptoms, essentially negating the benefit of the traction or worsening the patient's condition.

Little scientific rationale exists to support the use of heat concurrently with traction, but if done, caution should be taken to avoid overheating or burning a patient lying on the hydrocollator packs. Older patients or those with other thermoregulatory issues require special care with heat application. Frequent communication with the patient as to the comfort level with the heat is advisable. The practitioner must bear in mind that the heat is to promote patient relaxation rather than to bring about a specific therapeutic value. Thus, its use is not essential.

Adverse effects have been reported from the use of traction, albeit infrequently. The majority of clinical trials fail to mention adverse events. Existing reports are often lacking in detail but typically consist of symptom exacerbation, either localized to the neck or back or including

peripheral or radicular symptoms. Headaches, nausea, and fainting have also been reported.[34,35] Although apparently rare, these occurrences establish the need for cautious decision-making before using traction, regular monitoring of patients while receiving traction, and communication with patients as to their responses to the intervention.

Indications for Traction

Clearly established indications for using traction to treat the cervical or lumbar spine for mechanical pain syndromes remain elusive. Historically, the trend has been to use traction more prominently in the overall treatment plan when patients present with signs and symptoms consistent with radicular symptoms or other clinical criteria suggestive of radiculopathies. As classification schemes for patients with cervical or lumbar spine syndromes have evolved, some have included a subgroup of patients for whom traction is a preferred or optional intervention. Validation of these subgroups has not yet been accomplished.

The initial proposal of a treatment-based classification for low back pain contained a subgroup of patients for whom traction was suggested as the preferred intervention.[36] This categorization was largely based on patients experiencing distal symptoms when they performed trunk movement in any direction. Subsequent descriptions of the Treatment-Based Classification System[37,38] did not as clearly delineate a traction subgroup. The presence of symptoms distal to the knee, particularly when worsened with extension movements along with a crossed straight-leg raise and neurological deficits, were cited in one study as possible indicators for traction improving the probability of a favorable patient outcome.[39] Many clinicians will consider traction for patients with lumbar spine syndromes when peripheral symptoms or overt radicular signs are not reduced by movement or position testing in a manner similar to that initially proposed by Delitto et al.[36] The more recently published and contextually different Impairment-Based Classification System for Low Back Pain presents traction as a complementary intervention and does not specifically identify a patient subgroup best matched for mechanical traction treatment.[40] More simplistically, the reduction of symptoms with manually applied traction may be another criterion used in decision-making, although this has also yet to be validated (Fig. 7-31).

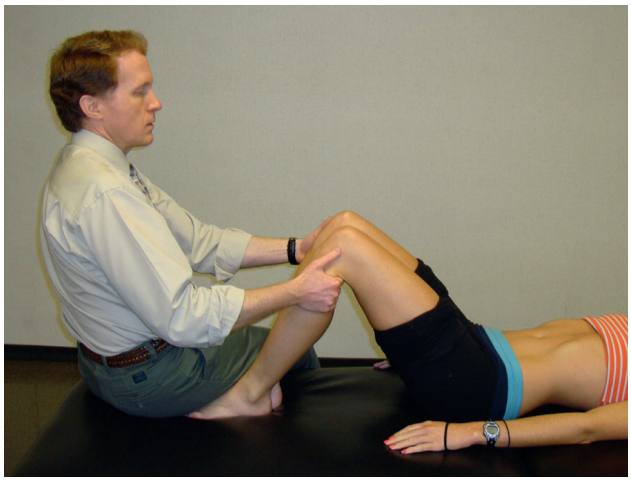

Fig 7 ■ 31 Application of manual lumbar traction during examination.

The Impairment-Based Classification for Neck Pain and accompanying clinical practice guideline[33] cites traction as a preferred intervention for those patients presenting with radiating upper-extremity symptoms. Evidence for this designation includes provocation of upper-extremity symptoms with foraminal compression testing, such as Spurling's maneuver (Fig. 7-32); reduction of those symptoms with manually applied traction (Fig. 7-33); and possible accompaniment of neurological involvement with upper-extremity sensory, motor, and reflex deficits. Notably, a clinical prediction rule for identifying patients most likely to benefit from cervical traction has been proposed.[41] The predictive variables

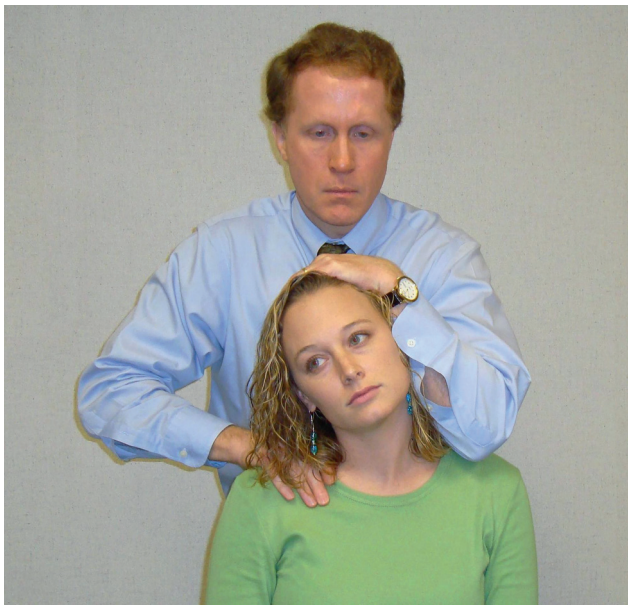

Fig 7 ■ 32 Spurling's maneuver during examination.

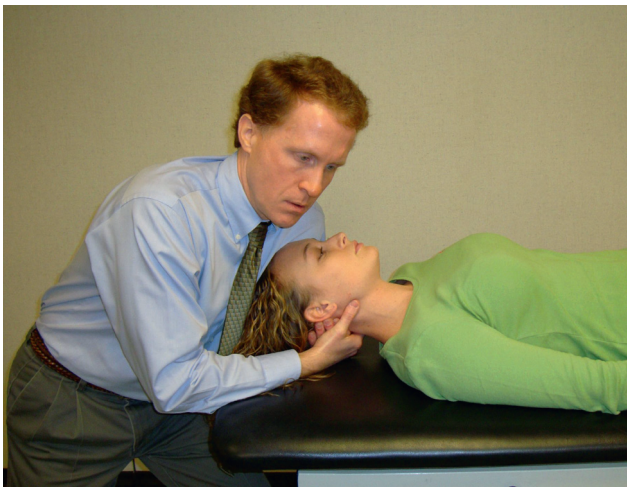

Fig 7 ■ 33 Application of manual cervical traction during examination.

included peripheralization of pain with lower cervical mobility testing, a positive shoulder abduction test, age of 55 or older, reduction of symptoms with manual distraction, and a positive upper-limb tension test. The reported statistical support for the derivation of this clinical prediction rule was robust; however, the study has been criticized for its methodology,[42] and validation of the clinical prediction rule has yet to be completed. Similarly, other investigators have proposed a clinical prediction rule for identifying those patients who might specifically benefit from home cervical traction.[43] The predictor variables in this study were pain at a level of 7 out of 10 or greater, a score on the Fear Avoidance Beliefs Questionnaire Work Subscale of less than 13, relief with manually applied traction, and pain perceived distal to the shoulder. Validation of this clinical prediction rule also has yet to occur.

Key Point! Clearly and definitively delineating the criteria for which patients might respond most favorably to traction has yet to occur. In the absence of clear practice guidelines, practitioner discretion is the greatest variable in traction usage. The evolution of evidence supporting manual therapy and exercise suggests these interventions be considered as a concurrent first option rather than traction for many patients with cervical and lumbar spine clinical syndromes.[33,40,44,45] For patients who have success with these interventions, traction should be considered a second-line or complementary intervention.

Contraindications for Traction

Before using traction on the spine, practitioners must screen patients for conditions that may cause adverse responses or for which definitive contraindications exist (Box 7-4). Cervical traction is contraindicated in patients with acute cervical spine trauma, particularly recent whiplash-associated disorders. Connective tissue diseases or rheumatologic disorders that can result in tissue laxity or joint hypermobility/instability are specific contraindications for the cervical spine. Foremost among these is rheumatoid arthritis, which is often characterized by asymptomatic subluxations of the upper cervical spine.[46–50] Ankylosing spondylitis, although characterized by rigidity and ossification in the lumbar and thoracic spine regions, may lead to upper cervical instability and is also a specific contraindication.[46,47,51–53]

Other diseases and disorders known to affect bone integrity, such as osteoporosis and osteopenia, are also contraindications for traction. Similarly, traction is contraindicated in patients with histories of steroid use or those who use medications that weaken or demineralize bone. Localized hypermobility or instability in the region of interest is also a contraindication.

Traction is contraindicated in patients who have received surgical stabilization or decompression of the spine or in patients with spine implants or prosthetic discs. The structural integrity of these devices or the bone-implant material interface may be compromised by the traction force.

Patients who have undergone cervical spine discectomies often will have also received interbody fusions at

Text continues on page 230

Box 7 ■ 4 Contraindications for Mechanical Traction

- Acute cervical trauma, including whiplash-associated disorders
- Osteoporosis or osteopenia
- Use of steroids or other medications that tend to compromise bone integrity
- Rheumatologic disorders affecting connective tissue, including rheumatoid arthritis and ankylosing spondylitis
- Joint hypermobility/instability
- Pregnancy
- Prior surgical stabilization or decompression
- Spinal implants/prosthetic discs
- Nonmechanical pain

CASE STUDY 7-1 Cervical Traction

A 38-year-old male presents for physical therapy with primary complaints of left neck, periscapular, and arm pain. He also describes paresthesia distal to the left elbow, extending into the first and second digits. He is unable to identify a specific event precipitating these symptoms but noted them gradually increasing the day after moving a household appliance 1 week ago. Upon questioning, he specifically denies any trauma to his cervical spine. His occupation is manager of several multiunit apartment buildings. He reports an inability to complete both the more physically demanding aspects of his job and the desk and computer-based responsibilities because of his symptoms.

Upon clinical examination, cervical active ROM is observed to be severely pain-limited in all planes, particularly with extension, left rotation, and left lateral flexion. His upper extremity reflexes are at 2+, except for the left biceps brachii, which is at 1+. Muscle testing across the upper extremities yields grades of 5/5 on the right. Left upper-extremity muscle tests are all pain-limited to 4+/5, except for the left biceps brachii at 4/5. Grip strength on his dominant right side is at 105 pounds and the left is at 27 pounds. Light touch sensory ability is diminished at the pad of the left second digit. Spurling's maneuver on the right is negative but on the left elicits an increase of distal paresthesia. Manually applied traction reduces the left periscapular and proximal arm pain. Left upper-limb tension testing is highly provocative of symptoms in the arm and forearm, with full positioning limited to 30° deficient of full elbow extension during the test.

CLINICAL DECISION-MAKING

1. Does the patient have a problem that can be improved with the use of traction?

ANSWER: Although specific indications for traction remain unclear, the examination findings are noteworthy. Given that manual traction reduced his symptoms, adding traction to his treatment regimen may be warranted, particularly since no other position or measure has offered pain relief over the past week.

2. Is the patient appropriate for application of traction? Do any of the general precautions or contraindications to traction apply, or are there any specific considerations regarding application of traction to this patient?

ANSWER: The patient denies any medical history that would contraindicate the use of traction. The examination findings and his history since the onset suggest his condition to be highly acute and irritable. The presence of multiple suggestions of peripheral neurological deficit requires close observation and careful decision-making, as further deterioration would suggest worsening of his condition.

3. What are the specific goals to be achieved with the use of traction?

ANSWER: The immediate goal for this patient would be to reduce the pain, which is theorized to be of nerve-root origin. Pain relief will allow the patient to resume restorative sleep and perhaps return to a portion of his job duties. A reduction of pain will also enhance his ability to complete the therapeutic exercise and other active interventions planned for home and in subsequent clinical visits.

4. What specific type of traction is appropriate for the patient?

ANSWER: The evidence is limited, but intermittent traction at the angle of cervical flexion offering the lowest level of peripheral symptoms is a reasonable starting point from which to begin a trial of traction. This may be adjusted during the course of traction and after the first treatment based on his responses.

5. What specific parameters of traction would be appropriate for the patient?

ANSWER: The apparent level of acuity and irritability of his condition demands caution with the initial trial of mechanical traction. Traction dosage on the first day should be modest. As such, a reasonable trial with intermittent traction may be cycling between 12 and 6 pounds for 30 and 10 seconds, respectively. The initial total treatment time may be as little as 10 minutes. These variables, however, are subject to adjustment during the course of the traction session. Given his level of pain and inability to participate in daily demands, a follow-up visit the next day may be warranted.

6. What are the effective and safe application procedures for traction related to this patient?

ANSWER: A dramatic improvement with one session of traction is an unrealistic expectation. Perhaps the first objective is to establish his tolerance to the chosen intervention. Assessing the patient's status during and immediately after the first session of traction is imperative. Any increase of symptoms peripherally is to be specifically avoided. If this occurs during the traction, the treatment should stop immediately. His response for the next several hours after traction will be, in part, the basis for determining the direction of his care at his next visit. A cumulative effect of pain reduction with subsequent applications is expected. Failure for this to occur indicates the need for medical consultation and diagnostic imaging, particularly if the neurological deficits persist or worsen.

CASE STUDY 7-2 Lumbar Traction

A 46-year-old male arrives at physical therapy with primary complaints of right low back, buttock, thigh, and calf pain of 2 weeks' duration. He describes the onset of these symptoms subsequent to lifting a spool of wire into a truck at his construction job. He reports difficulty maintaining any body position for more than 30 minutes, with sitting being the most provocative of his symptoms. He denies any change in bowel or bladder function since the onset of these symptoms.

Upon examination, pain grossly limited lumbar ranges of motion in all planes, and provocation of distal symptoms at the end of the available range in each direction is observed. Passive side-gliding of his lumbar spine is severely limited with distal symptoms in the left and a firm barrier to the right. He is intolerant of testing the S1 motor distribution when standing on the right because of pain. In supine, muscle testing across the lower extremities is particularly remarkable for ankle eversion on the right at 4/5 with no other deficits observed. Muscle stretch reflexes are at 2+, except for the right ankle at 1+. Straight leg raising on the right causes an increase of distal symptoms at 30° and is negative on the left. Decreased light touch sensation is noted at the great toe of his right foot. Attempts at repeated movement testing of the lumbar spine for centralization and eliminating the block to right side-gliding result only in an increase of the lower-extremity symptoms. Upon the application of manual lumbar traction during the examination, he reports a modest reduction of his back and lower-extremity symptoms.

CLINICAL DECISION-MAKING

1. Does the patient have a problem that can be improved with the use of traction?

ANSWER: At the time of this examination, manually applied traction is the only procedure that reduces the patient's symptoms, particularly those in his lower extremity. All of the other attempts to centralize or reduce his pain worsen his symptoms. His outcome is difficult to project given the acuity and apparent irritability of his condition and the accompanying neurological deficits that suggest radiculopathy. Based on the examination findings, however, a trial of traction to assess his response is the best option to start his recovery process.

2. Is the patient appropriate for traction? Do any of the general precautions or contraindications to traction apply, or are there any specific considerations regarding application of traction to this patient?

ANSWER: Upon review of his history and overall health status, there are no indications that traction is inappropriate to use in his treatment. Given the severity of his condition and apparent nerve-root involvement, caution is required. The specific traction dosage on the first attempt will be modest to assess his tolerance, and he will require close supervision during traction. Additionally, he will require close observation in the period immediately after traction and before he leaves the clinic to assess his response for future treatments.

3. What are the specific goals to be achieved with the use of traction?

ANSWER: With conditions suggesting radicular pain, a reduction of peripheral symptoms is usually an early objective. If these symptoms can be decreased, he will become a candidate for other interventions not currently tolerated, which may accelerate his progress. These would include manual therapy, self-mobilization, motor control exercise, and higher-demand exercise consistent with his functional demands at work.

4. What specific type of traction is appropriate for the patient?

ANSWER: With his most comfortable position being supine with his hips and knees flexed, the initial trial would logically incorporate this positioning. He gained relief with the manually applied traction during the examination as his hips and knees were flexed while his feet were resting on the table. Thus, replicating that position for the first attempt would be logical and could be accomplished with a bolster under his knees. There is no evidence to support either static or intermittent traction being superior overall or with subgroups of particular clinical presentations. With any sustained mechanical stresses apparently being poorly tolerated, the preferred initial arrangement is intermittent traction.

5. What specific parameters of traction would be appropriate for the patient?

ANSWER: Given the acuity and apparent irritability of his condition, the initial trial of traction will require caution. The initial treatment to assess his response and attempt to establish a tolerance level will be at

Continued

CASE STUDY 7-2 Lumbar Traction—cont'd

25% of his body weight and for a shorter than usual period of 10 minutes. With his body weight at 200 pounds, the traction unit will be set at 50 pounds at the maximum tension and 25 pounds for the minimum tension. The time settings will be programmed for 10 total minutes with 30 seconds at maximum tension and 10 seconds at the minimum tension. Because of his condition's acuity and apparent irritability, two progressive and two regressive steps are also being programmed into the traction control unit.

6. What are the effective and safe application procedures for traction related to this case example?

ANSWER: Using traction with this patient is only the first step in what will evolve into a multimodal treatment approach. Provided he responds favorably to the traction on the first visit, the traction parameters will be progressed at the time of his second visit and perhaps subsequent visits to greater amounts of tension and longer duration. If symptoms are reduced, his response to repeated movements will be reassessed with the objective of advancing his self-treatment regimen to include self-mobilization procedures, most likely in a lumbar extension orientation. Exercises to activate the musculature supporting the lumbopelvic complex will also be incorporated early in his rehabilitation and progressed according to his tolerance and the quality of his recruitment patterns. He may also become a candidate to receive manual therapy at some point in his treatment. As his distal symptoms diminish, traction will likely be discontinued. The overarching concept is that traction in this patient's care is an early treatment option to reduce pain and facilitate an increased tolerance to other interventions established to be effective.

the involved segment and thus should not receive traction. This modality may not be strictly contraindicated in a patient with a remote history of a simple lumbar discectomy on a single level, but the practitioner should be careful when deciding which treatment option is most appropriate and the intended effect. Given the altered anatomy following discectomy, the practitioner must contemplate the proposed benefit from traction as opposed to other therapeutic options.

Because of multiple unknowns and the potential risks, lumbar traction during pregnancy is to be avoided. Use of cervical traction during pregnancy may not be absolutely contraindicated, but consideration must be given to hormonal influences potentially affecting tissue laxity.

If the patient reports pain in or around the spine that cannot be determined in the initial examination to be of mechanical origin, the practitioner should suspect potentially serious pathology. The inability to find movements or positions to relieve pain may be indicative of a serious health condition beyond the scope of physical therapy. As such, further medical consultation or diagnostic testing may be warranted rather than defaulting to traction as a mechanical treatment. Similarly, patients who report histories of cancer should have a thorough diagnostic evaluation, including imaging, before receiving traction because of the potential for disease recurrence or metastases.

Precautions

Individuals with claustrophobia may not be well suited to receive mechanical traction, particularly for the lumbar spine. The perception of being enclosed in the harnesses and confined on the table may precipitate anxiety that will preclude the patient from participating in traction. Similarly, if the traction table is located in a small room in the clinical setting, the effect may be magnified. For this reason, many practitioners prefer to place their traction tables in a curtained area within a more spacious area of the clinic. With some patients, the curtain partition, perhaps even partly opened, allows enough relaxation for the treatment to be well tolerated.

Patients with chronic obstructive pulmonary disease or other respiratory disorders may also find the harnesses required for lumbar traction uncomfortable and may be compromised by the supine position. Being able to secure the harnesses adequately while permitting the patient to relax and breathe comfortably may be difficult to achieve. If patients are distressed by the mechanical traction positioning and apparatus, treatment options other than traction may be better.

HOME TRACTION

An outgrowth of the benefit and sometimes transient symptom relief of clinical traction is the availability of a variety of home traction units. These units are often available over the counter or from some retail outlets. Other more sophisticated traction devices are available only through health-care providers or by prescription from a physician.

Cervical Spine

Recently, portable equipment closely replicating clinical models has become available for home use (Fig. 7-34). These usually consist of an occipital harness capable of gliding on a small, stable frame. Traction is provided by a manually operated pneumatic pump that includes a gauge for quantifying tension. The patient lies supine and is positioned in the harness similar to the larger clinical models. Some home models have adjustable harnesses, allowing the patient to adjust the pads by simply turning a knob. Other models have self-adjusting pads that seat against the occiput when traction is initiated. The patient achieves the traction effect by activating the manual pump, causing the harness to glide along the frame away from the base. An intermittent mechanical traction effect can be achieved by cycling the pressure up to a designated amount (usually replicating the amount found beneficial during clinical treatment), sustaining that pressure for a time interval (e.g., 1 minute), then releasing the pressure by a valve on the pump. Multiple cycles of this sequence can be completed. Manipulating the variables for home traction on one of these models can closely simulate that achieved on clinical traction tables.

Perhaps the simplest and least expensive apparatus, improvised many decades ago, is still available for cervical spine traction. A water-weighted bag attached to a cord coursing over a pulley that provides tension to a harness worn on the head can provide a modest traction effect for the cervical spine. These devices, used while sitting, must first overcome the weight of the head in order to traction the neck. Additionally, there is the risk that pressure through the mandible can cause or exacerbate temporomandibular joint dysfunction. Achieving intermittent traction with one of these models is not easily accomplished, and static traction may be more pragmatically completed, particularly if the patient is unassisted during the home traction session.

Lumbar Spine

Home lumbar traction units have been largely impractical and of questionable value until recently, when the design features of successful in-home cervical spine traction units were incorporated into lumbar spine units. A similar pneumatic pumping device is used to separate two surfaces on which the patient can lie (Fig. 7-35). The traction tension is usually considerably less than that produced by clinical models, but a sufficient approximation to that achieved clinically may have a modest effect or complement clinical traction. These units are usually compact and are self-contained in a carrying case. Patients with high levels of pain may have difficulty administering home lumbar traction without assistance. Positioning on the floor is required; thus, the associated mobility demands and the need to self-tighten the harnesses may prove difficult for someone with significant pain-limiting impairments. Lifting and carrying the

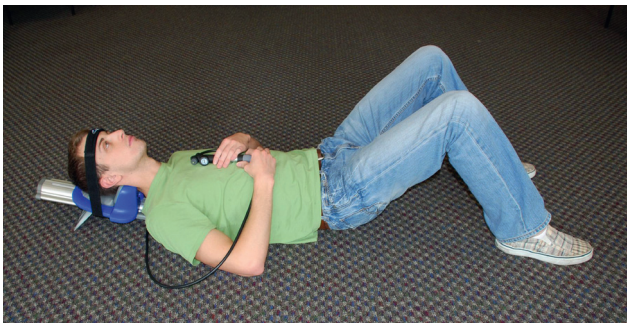

Fig 7 ■ 34 Home cervical traction unit.

Fig 7 ■ 35 Home lumbar traction unit.

home lumbar traction unit may present a similar challenge. Most of the pneumatic traction units, whether for the cervical or lumbar spine, have safety valves to prevent potentially injurious tension levels.

The usual intent of providing patients with home traction units is to enhance the effect achieved clinically and empower patients in their recovery. After a beneficial dosage is identified in the clinical setting, tension amounts and durations for home treatment may be replicated with home equipment. Alternately, individuals may attempt self-treatment at their own discretion or possibly by their physician's choice without clinical supervision.

The practitioner should guide the patient carefully through the process of home traction, completing the steps with the home unit while still under clinical supervision. Patient education to avoid causing injury with a home unit is important. Printed or written instructions detailing traction dosage and recommended frequency should be provided to the patient. In addition, patients are to be cautioned to take particular safety measures such as using a timer with an alarm to avoid falling asleep in the traction unit. Prolonged single-session use from patients falling asleep during home traction has been known to significantly exacerbate pain. To minimize the risk of pain worsening from home traction unit use, practitioners must require patients to verbalize their understanding and demonstrate appropriate use of home traction before they initiate self-treatment. Similarly, specific questioning of the response to home traction and any related necessary problem-solving are essential in subsequent clinical visits.

Home units are usually purchased, although some medical equipment providers will allow rental. The simple water bag home cervical traction units are available for as little as $20. Currently, the more sophisticated pneumatic versions emulating clinical models are approximately $400 to $500. The pneumatic lumbar home traction models are approximately $450 to $600. Because of this cost, home traction should not be recommended for patients unless clear benefit is demonstrated from clinical use and several sessions are anticipated for maximum benefit.

In part related to home devices, the effects of traction in water in patients with low back pain and signs of nerve-root compression have also been studied. Subjects accomplished low-magnitude lumbar traction by buoyant devices under the axillae while strap weights were attached to their ankles. Outcomes were also measured in a comparison group using only land-based interventions. The hydrotraction group reportedly had greater pain relief and centralization of symptoms compared to the land-based group. Obviously, great caution is warranted with any attempt of achieving similar traction results using ankle weights in water.[54]

PATIENT OUTCOME EVIDENCE

Despite traction having been a widely used intervention for neck and back pain for decades, strong evidentiary support for its use is lacking. Many studies evaluating traction are methodologically deficient and may not offer meaningful results. Additionally, many of the studies possessing better research designs do not correlate well to routine clinical practice. Thus, the ability to generalize the results in patient care scenarios may be limited.

Multiple studies have used simulated traction or traction at presumably ineffective levels of tension for comparisons against traction at greater and theoretically therapeutic levels of tension.[30,55–59] Although such methods allow allocation concealment to a greater degree, the actual differences in interventions between the groups may be minimized.

Other studies have used traction as the sole intervention,[31] which does not replicate the generally accepted standard of care. In routine clinical practice, patients receiving traction will frequently have complementary interventions such as manual therapy, exercises for key muscle recruitment and strengthening, postural correction, and neural mobilization.[29,33,60–62] Using multiple interventions, however, complicates or precludes isolating any treatment benefit as a result of a single intervention, including traction. The conflict between ideal clinical research and offering best clinical care according to professional judgment may not be easily reconciled in trials. To achieve strong evidentiary support in a study, the preferred study design is to isolate the independent variable of interest. The interaction between interventions or synergistic effects of various treatments, however, may not be appreciated in the results of such study designs.

One of the most frequent methodological issues is the heterogeneity of groups receiving traction in clinical studies. Practitioners have long sought to delineate particular

patient characteristics that would predict those responding best to traction. Despite robust design otherwise, multiple studies combine patients with various clinical presentations in the groups being compared.[30,55–58,63–65] Thus, potential benefit in subsets of patients with neck or back pain syndromes who are receiving traction may not be easily recognized. As described earlier, limited evidence remains toward identifying patient characteristics that predict improved responses with traction. Efforts to identify those patients who are most likely to benefit from traction continue in recent and ongoing research.[39,66]

Auto-traction or patient-powered traction was equated to passive traction and has been used as the variable of interest.[56,65] Given the well-documented activation of spinal-supporting musculature with simple limb movements and more complex motor patterns,[67–75] self-traction may be completely ineffective in achieving distraction of spinal structures. In a previously mentioned study,[20] activation of the patient's musculature when performing self-traction easily precluded any distractive effects on the spine. Further, intradiscal pressures have increased during self-traction attempts.[20,21] Thus, considering auto-traction to be equivalent to passively administered traction also gives rise to methodological concerns of whether the interventions are comparable.

A small number of clinical trials, representative of clinical practice, yield conflicting results.[58,76] Case series have been published suggesting value in traction, particularly in patients presenting with signs and symptoms consistent with radiculopathies.[29,60,61] The absence of control or comparison groups, however, limit the ability to assess the effect of traction as the key variable. Additionally, the outcomes of patients receiving traction cannot be easily differentiated from those patients receiving alternative interventions or from the natural history of the disorders.

Recently, two studies have evaluated what the investigators described as traction for the lumbar spine; however, analysis of their mechanical apparatus and forces imparted on the patients reveals a nontraditional delivery of traction. Rather than longitudinal forces applied to the lumbar spine, these investigators applied external forces that lifted the trunk in supine so as to increase the lordotic curve, sometimes with a modest concurrent longitudinal force. The design and outcomes of these studies to date preclude any meaningful interpretation of the results.[77,78]

A noteworthy randomized trial assessed the value for cervical traction by comparing three patient groups of 13 subjects each. All had signs and symptoms of cervical radiculopathy with corresponding imaging results. All three groups received multimodal intervention approaches, including biophysical agents, exercises, and manual therapy. One group additionally received manually applied traction, and another group received mechanical traction. Both traction groups had superior outcomes with less severe neck and peripheral symptoms and less analgesic consumption compared to the nontraction group. The outcome differences were sustained across all assessment points, including at a 6-month follow-up.[79]

Similarly, in a relatively large, recent clinical trial of patients presenting with cervical radiculopathy, the addition of clinical mechanical traction was found to improve outcomes over those completing exercise only or exercise plus simple over-door home traction. The group receiving mechanical traction sustained better outcomes at multiple time points, including 12 months after enrollment.[80]

Recently published systematic reviews and suggested medical practice guidelines[34,35,81,82] have concluded there is minimal evidence for the use of traction to treat patients with neck and back pain. The majority of studies included in the systematic reviews had small numbers of subjects and contained moderate to high risk of bias, limiting their value. However, the conclusions regarding the absence of substantive evidence must be considered within the context of the inadequacy of the research and are generalized for populations, not particular individuals who may respond favorably to traction. Physical therapy clinical practice guidelines describe a modest but limited role for traction in treatment of patients with neck and back pain as part of multimodal intervention approaches.[33,40]

Key Point! Sound clinical reasoning and problem-solving based on individual patient factors remain incumbent on the practitioner in the application and assessment of mechanical traction in a multimodal treatment approach of spinal pain syndromes.

The cumulative evidence for benefit from cervical spine traction in mechanical neck pain syndromes is

modestly greater than that for the lumbar spine. Although evidence-based practice and clinical guidelines can assist practitioners in the clinical reasoning processes of patient care, such information does not exclude or minimize the importance of making decisions of care based on the individual patient.

Documentation Tips

For documentation of traction treatment, describing the variables listed in Boxes 7-2 and 7-3 is appropriate. Additionally, describing the patient's responses to traction during the treatment session and immediately afterward while still under observation is recommended.

Clinical Controversy: Spinal Decompression

Spinal decompression has been marketed intensely in recent years as a new method of addressing back and neck pain while yielding remarkable results. Popular media advertising through the radio, newspapers, and the Internet has often been used to promote decompression as a "breakthrough" in the treatment of spinal disorders. Spinal decompression equipment usually imparts cyclical longitudinal force on the spine with the patient lying supine on a tablelike device with a motorized unit (Fig. 7-36). According to the U.S. Food and Drug Administration (FDA), spinal decompression is described differently from traction largely because of a technicality. In the application process for equipment approval, manufacturers must label the equipment according to its presumed effects. Spinal decompression is labeled separately from traction on 510(k) applications to the FDA for marketing medical devices. Also, decompression has been granted a Current Procedural Terminology code apart from traction. These technical differences enhance the ability for marketers to distinguish decompression from traction in media campaigns.

Advocates of decompression claim physiological and biomechanical effects greater than traction, particularly on the intervertebral disc. Decreased or negative levels of intradiscal pressure have been measured in vivo during decompression.[83] Three case series suggested noteworthy improvement levels in patients receiving decompression and are frequently the sources of statistics used in marketing campaigns.[84–86] A systematic review of the clinical trials using decompression reveals that six of the seven studies report no difference in outcomes with spinal decompression; one investigation reported less pain but no change in disability of the subjects.[87] Additionally, the preponderance of the studies evaluating spinal decompression has been of relatively poor methodological quality. This is exemplified in a recently published retrospective case series with no long-term follow-up.[88]

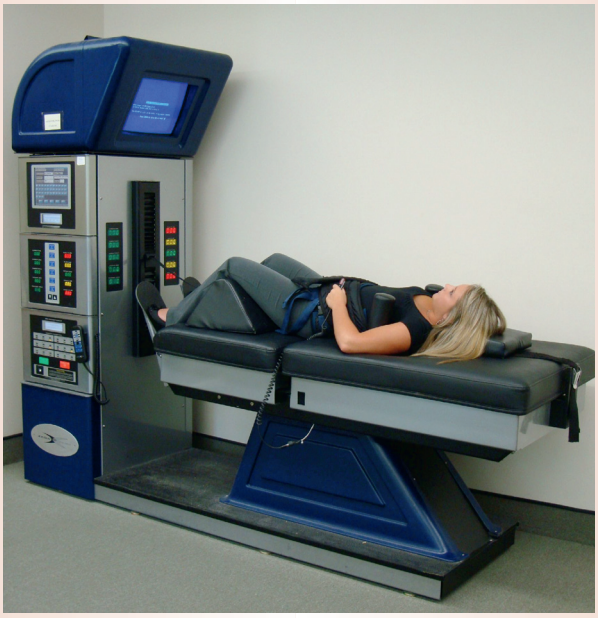

Fig 7 ■ 36 Lumbar spinal decompression unit.

Manufacturers and some professional groups openly promote decompression services as a means of increasing practice revenue at minimal effort. Assistant staff members, typically without substantive training in treating spinal disorders, are often used to administer the decompression to patients over a course of approximately 20 visits without requiring the time and effort of the supervising practitioner. As discussed among some practitioners, decompression's primary benefit to the practitioner is the increase in cash-paying patients. Strategies emphasizing the aesthetic qualities of decompression devices are often used in marketing to the public without regard to evidence of efficacy.[89] The lack of evidence in the current literature has led to the conclusion that spinal decompression has not been validated to be a superior intervention to traction.[82,90]

Inversion

One method of providing a distractive effect on the low back that does not require a clinical setting is the use of inversion devices, which allow gravity-facilitated traction. Home inversion equipment is available without prescription or assignment by practitioners. With many of these devices, the individual sits in a frame and then secures the lower extremities and pelvis with straps. The device can be unlocked and the person can invert body position such that the upper trunk weight is distracted from the secured pelvis and lower extremities. Inversion is remarkably simple and documented to increase lumbar intervertebral space immediately after the procedure.[91]

The inverted position, however, can have other unintended effects. Major increases in intraocular pressure leading to optic nerve dysfunction have been observed.[92,93] In addition, significant alterations in blood pressure has also been observed.[94–96] Anxiety while inverted is also common.[97] Inversion would not be an ideal choice for persons with histories of dizziness or vertigo. The physical demands to safely invert one's own body and then carefully return to an upright position are also a necessity for this process and may not be easily accomplished by some persons, particularly those in significant pain. Thus, safety and adverse effects are an issue, particularly when these devices are used without assistance or supervision.

A recent pilot study compared two groups of patients with single-level lumbar disc herniations. One group received physical therapy and the experimental group received physical therapy in addition to using a home inversion unit. A noteworthy reduction in surgeries was reportedly achieved in the inversion group compared to the physical-therapy-only group. On detailed review of the study results, however, none of the pain scales, functional outcome measures, or imaging results differed in the two groups. Thus, the implication of clearly superior outcomes in the inversion group (as stated in some promotional materials) must be interpreted cautiously.[98]

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CHAPTER 8

INTERMITTENT PNEUMATIC COMPRESSION

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HISTORY AND THEORY OF APPLICATION

The use of compression of vasculature for therapeutic purposes has been around since the 1800s, when physicians experimented with the application of external compression to improve circulation. A paper published in 1917 in the *Journal of the American Medical Association* described the use of compression for thromboangiitis obliterans.[1] Today, compression devices use a mechanical pump and a sleeve to deliver intermittent pneumatic compression (IPC), also known as *vasopneumatic compression.* Rehabilitation practitioners use IPC devices as an intervention for patients with circulatory conditions and with a variety of outcome measures (Box 8-1) (Figs. 8-1 and 8-2).

IPC is often used for edema control. Compression of tissue can elevate the pressure of fluid in interstitial spaces higher than that of blood and lymph vessels. The change in pressure gradient may facilitate the flow of fluid out of the interstitial space back into the venous and lymphatic vessels for drainage. IPC has also been used for a variety of clinical conditions, including improving venous circulation to prevent blood clots, healing stasis ulcers, and reducing lymphedema.[2,3] Recent research has focused on the treatment of arterial insufficiency with IPC[4] and for treatment of sensory impairment after a cerebrovascular accident (CVA).[5]

Although the exact mechanism of how IPC affects venous blood flow remains elusive, a proposed theory is that, when compression is applied, it results in forward propulsion of blood flow. This accelerated blood flow helps prevent venous stasis and can cause distention of the lumen, increasing the peak flow velocity by as much as 200%,[6,7] which may aid in the clearance of the valve sinuses.

INDICATIONS FOR INTERMITTENT PNEUMATIC COMPRESSION

Edema

Edema in a limb or joint can delay patient healing and interfere with rehabilitation. This condition occurs when an abnormal amount of fluid collects in the interstitial space. Edema can be localized due to increased vascular permeability, a lymphatic blockage, or venous obstruction. It is also associated with the acute phase of inflammation, reduction of plasma proteins, and electrolyte or fluid

Box 8 ■ 1 Outcome Measures of Intermittent Pneumatic Compression

- DVT prevention
- Edema reduction
- Improved sensory function after CVA
- Faster venous stasis wound healing
- Increased blood flow in PAD
- Reduction in limb volume in lymphedema

imbalances and when limbs are in the dependent position. Systemic edema can occur due to pathology of the cardiac, pulmonary, or renal systems.

Edema can slow down the rehabilitation process in many patient populations. The fluid can occupy a joint space, leading to pain and limitation of range of motion. The collection of fluid in the lower extremities can increase the metabolic demand of ambulation and decrease functional capacity. If the lymph system is impaired, it can increase susceptibility to infection. Reducing edema should be part of the plan of care. IPC is indicated as an appropriate intervention for edema reduction; however, acute edema following trauma should not be treated with IPC because it may increase bleeding.

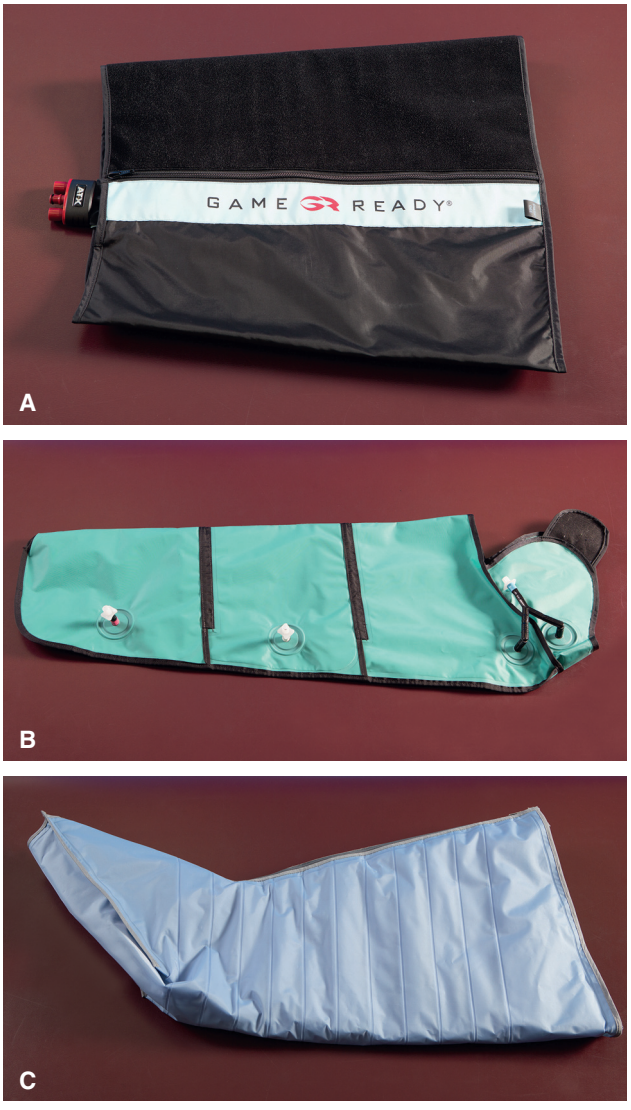

Fig 8 ■ 1 (A) Single and (B and C) multichamber sleeves for application of IPC.

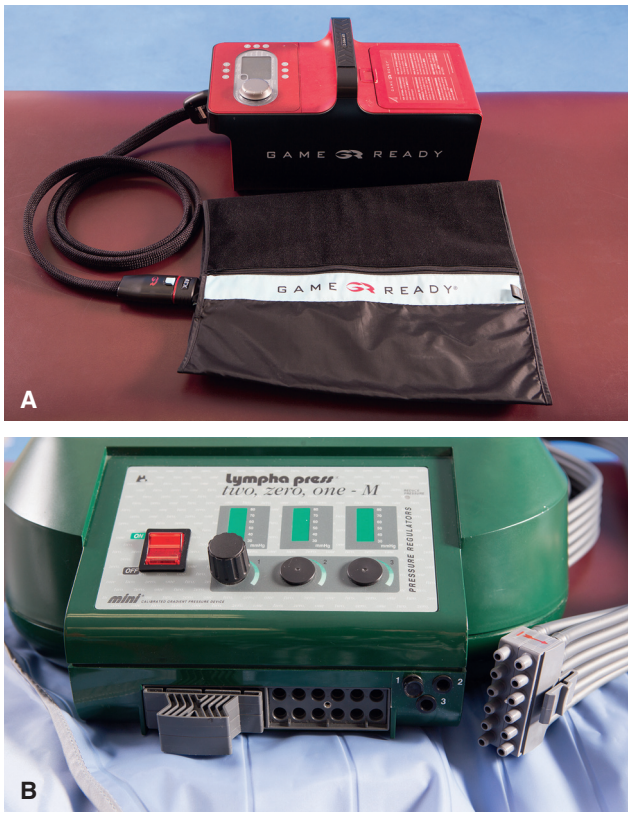

Fig 8 ■ 2 (A) Single and (B) multichamber IPC mechanical pumps.

Key Point! When edema is present, it is very important to determine its cause. A thorough history must be obtained and a careful patient examination performed.

Prevention of Venous Thromboembolism

It has been estimated that by the year 2030, there will be 572,000 total hip replacements and 3.48 million total knee replacements in the United States.[8] It is widely known that orthopedic surgery increases the risk for

venous thromboembolism (VTE). It is further estimated that as many as 80% of patients undergoing orthopedic surgeries will develop VTE without prophylactic interventions. Even with prevention strategies that include pharmaceutical intervention and mechanical compression, VTE is the most common cause for emergency readmission after lower-extremity joint replacements.[9] The Agency for Healthcare and Research Quality guidelines include IPC for VTE prevention.[10] One advantage of using IPC for VTE prevention over anticoagulant therapies is that IPC does not increase the risk of bleeding. A portable IPC device that delivers a higher peak venous velocity through a rapid inflation sleeve was found to be as effective as low-molecular-weight heparin at preventing VTE, but IPC had significantly less risk of bleeding events in patients who underwent a total hip replacement.[11]

In patients with neurological injuries, IPC has been shown to significantly reduce the risk of developing VTE.[12] The American College of Chest Physicians published "Evidence-Based Clinical Guidelines for the Prevention of VTE" in 2012. These guidelines recommend using IPC for patients following acute CVA, for patients with intracranial hemorrhage, for acutely ill patients, and for patients who have had abdominopelvic surgery.[13]

Key Point! In addition to IPC, the most important preventive technique for VTE is early mobilization. Mounting data suggest that mobilization as soon as a patient is hemodynamically stable—even for patients in the ICU setting—is important to prevent VTE and improves clinical outcomes.

Peripheral Artery Disease

Peripheral artery disease (PAD) affects 12% to 14% of the general population.[14] Individuals with PAD typically have intermittent claudication and low functional capacity. If PAD worsens over time, it may lead to critical limb ischemia and possibly limb loss. By applying sequential IPC from distal to proximal to the ischemic limb, IPC empties the capacitance vessels by expelling blood proximally. The arteriovenous pressure gradient is increased and arterial inflow is augmented. Sultan et al[15] sequentially applied IPC by utilizing the ArtAssist sequential compression biomechanical device to individuals with severe PAD (Rutherford category ≥ 4). The treatment protocol consisted of 6 to 8 hours of IPC each day for 12 to 24 weeks. Subjects who underwent treatment had significant improvements in increased mean toe blood pressure, increased mean popliteal flow, and resolution of rest pain.

Physical function declines as PAD progresses, and individuals with PAD often complain of intermittent claudication that can lead to decreased quality of life. A recent study evaluated the effects of IPC on quality of life measures (SF-36 health status questionnaire) and ambulation tolerance. The experimental group received IPC for 3 hours per day for 3 months. Significant improvements were found in six out of eight domain scores of the SF-36 and 6-minute walk distance of the experimental group.[16]

Key Point! Weight-bearing exercise is recommended for individuals with PAD. The American College of Sports Medicine recommendations for individuals with PAD include weight-bearing aerobic exercise 3 to 5 days per week and resistive exercise 2 days per week.[17]

Venous Stasis Ulcers

Venous insufficiency can lead to the formation of venous stasis ulcers. The underlying cause of venous stasis ulcers is venous hypertension resulting from valvular incompetence and/or obstruction of the vein. Risks of developing venous stasis ulcers include prolonged immobility, obesity, calf muscle pump dysfunction, and pregnancy. Additionally, there may be a genetic predisposition in some patients toward venous stasis ulcers. Epidemiological estimates of venous insufficiency in the Western world vary. A study in 2014 estimated that chronic venous disease affected 25 million adults in the United States.[18] Prevalence estimates range from 1% to 40% in women and from 1% to 17% in men.[19]

IPC mimics the action of the gastrocnemius/soleus muscle pump complex. A one- or multichambered sleeve worn around the foot or leg is connected to a pneumatic pump that pumps air into the sleeve. Devices with more than one chamber can inflate sequentially to promote an ascending pattern (i.e., distal to proximal) of compression in the limb. IPC applied to limbs with venous stasis can

decrease venous pressure and interstitial edema. IPC will collapse the superficial venous system, forcing blood into the deep system. The increase in subcutaneous pressure will aid in the prevention of leaking blood, fibrin, and protein from the skin capillaries. Research using an animal model[20] attributes shear stress on tissues that occurs during mechanical compression with vasodilatory, profibrinolytic, and antithrombotic effects.

IPC for patients with venous stasis ulcers was found to accelerate wound closure when applied 1 hour per day in the following manner: IPC increased to 70 mm Hg over 20 seconds, compression sustained at 70 mm Hg for 20 seconds, and then deflated for 20 seconds. This method had a mean time of ulcer closure of 20 days compared to 60 days using traditional methods alone. However, the study did not use statistical measures to compare the groups.[21]

A systematic review[22] of IPC for treating venous stasis ulcers was published in 2003. The authors concluded that subjects were generally happy with the results of IPC, and they had higher compliance with IPC compared to other compression methods such as stockings. The effectiveness of IPC on venous ulcer healing was equivocal. However, the conclusions stated that IPC may be an alternative to other compression therapies for patients who refuse, are unable to comply with, or failed other methods of compression. A 2011 intervention review in the Cochrane Collaboration found IPC contributes to the faster healing of venous ulcers when compared to no compression.[23] However, the reviewers could not determine how effective IPC is when compression stockings/bandages are already in use.

The speed at which IPC is applied may have an impact on outcomes. A study found that a fast sequential IPC regimen was more effective at healing venous ulcers than a slow regimen.[24] The sequential compression pressures were the same for two protocols, but the speed of IPC was varied. Subjects with venous ulcers were randomized to either a fast regimen group (0.5-sec compression rise time and 6-sec inflation/12-sec deflation ratio) or a slow regimen group (60-sec compression rise time and 30-sec inflation/90-sec deflation ratio). Both regimens were applied for 1 hour daily. The mean rate of venous stasis ulcer healing was significantly faster in the fast regimen. A review article on the effects of IPC on venous stasis ulcers suggested that rapid inflation, which cycles two to three times per minute, is superior to slow inflation.[25]

Key Point! Other biophysical agents such as electrical stimulation, can be used to treat venous stasis ulcers. In addition, compression bandages and dressings are also utilized to aid in ulcer healing. Effectiveness of interventions can be monitored by recording serial objective measures of the wound either by measuring the wound depth, width, and diameter or by tracing the wound or using photography.

Lymphedema

It has been estimated that 10% of women with gynecological cancer will develop lymphedema in the lower extremity.[26] The incidence of lymphedema of the upper extremity in individuals with breast cancer after 5 years varies from 5% to 42%.[27,28] IPC has been used to treat lymphedema. A proposed mechanism is that IPC decreases capillary filtration, which decreases lymph formation.[29]

The effectiveness of IPC for treating lymphedema has been studied. The results are inconclusive. In 2007, a systematic review of common conservative therapies for lymphedema of the upper extremity after breast cancer was performed.[30] Five studies were included in this review. The review concluded that IPC was likely to provide a larger percentage volume reduction compared to other interventions. Uzkeser and Karatay[31] investigated the effect of adding IPC to a lymphedema protocol. Two groups received manual lymphatic drainage, compression garments, and exercises. The experimental group also received IPC with a pressure of 40 mm Hg for 45 minutes, 5 times per week for 3 weeks. Both groups had a significant reduction in their volumetric measurements; however, the addition of IPC did not improve outcomes. A study by Shao et al[32] did not find IPC to be effective in postoperative breast cancer lymphedema. The lack of effectiveness of IPC in these studies may be related to the pressure settings applied to the extremity during treatment. A study published in 2013 suggested that, in order to create proximal flow, tissue fluid head pressure must reach greater than 30 mm Hg.[33] IPC was applied to limbs with lymphedema at various pressures. IPC applied at 50 to 120 mm Hg was not able to achieve tissue fluid head pressure of 30 mm Hg in the entire lower extremity. Inflating and deflating the cuff

around the limb for 50 seconds on and 50 seconds off had the most favorable effect on achieving tissue fluid head pressure of 30 mm Hg. Olszeweski et al[34] used treatment pressures of 50 to 120 mm Hg to show movement of fluid. A follow-up study found this protocol was effective for patients with stage II to IV leg lymphedema.[35] This is in contrast to recommended pressures of 30 to 60 mm Hg for treating lymphedema or venous insufficiency.[36,37] One of the challenges in establishing compression protocols for IPC is that there are no noninvasive means of measuring accurate changes in lymphatic flow.[38]

Clinical Controversy

The traditional IPC treatment approach for lymphedema has not included compression of the abdomen or trunk. Considering the treatment philosophy of manual lymphatic drainage, it is necessary to create an empty reservoir for fluid to move to from the extremity.[39] However, very few clinical trials have compared manual lymphatic drainage with and without decongestion. Despite this, some authors have concluded that truncal treatment is a necessary component for effective treatment of lymphedema.[40] A study that compared truncal and arm IPC to arm IPC alone did not find that it significantly improved outcomes.[41]

A comparison of a traditional IPC device with a newer model that included trunk compression was published in 2011.[42] The authors concluded that the advanced IPC device, which had multiple chambers in sleeves that were applied to the arm and trunk, was more effective than a traditional application without trunk compression. However, the traditional IPC was applied at 30 mm Hg and the advanced IPC was applied at 45 to 75 mm Hg. The on:off times of compression varied considerably between studies. Based upon the inconsistent pressures and different on:off times used in these studies, it is difficult to recommend one approach over the other.

Key Point! Lymphedema can worsen with trauma to the involved limb. When performing IPC for lymphedema, it is imperative that all health-care providers avoid assessing blood pressure or performing vein/arterial puncture of a limb that is either at risk for lymphedema or has known lymphedema. Hospital and rehabilitative centers usually place limb ID bracelets on these patients to alert health-care providers to avoid the use of the limb.

CLINICAL APPLICATION OF IPC

Preparation for Treatment

The clinician should place the IPC compression unit on a stable surface in close proximity to the limb to be treated. The compression sleeve should be checked to be sure it is clean and dry before applying it to the patient. Compression sleeves come in a variety of sizes and shapes. When selecting a sleeve for a patient, use the smallest one that will provide adequate coverage to the treatment area. Consult the manufacturer's guidelines for suggested parameters for the IPC device. (Tables 8-1 and 8-2 provide examples of manufacturer parameters.)

Many facilities have policies and procedures that address guidelines for application of IPC. Inflation pressure is the maximum pressure reached during the period of inflation. Arterial capillary pressure is generally about 30 mm Hg, so any pressure above that amount should be useful in assisting an arteriovenous pressure gradient and encouraging the absorption of edema. On:off time sequence refers to the ratio of time that the machine is

TABLE 8–1. Arterial IPC and Biocompression Sequential Circulator SC-3008: Recommended Parameters for IPC

	Arterial–IC-BAP	BioCompression SC-3008
Pressure	• 120 mm Hg • Applied usually to foot, calf only	• 40–50 mm Hg • Applied to entire lower limb
Cycle Time	• Multiple chambers • Inflation: 4 sec × 3 cycles per minute • Calf inflate delay 1 sec/50 sec off	• Multiple chambers • Inflation time: 40 sec • Deflation time: 5 sec

TABLE 8–2. Lympha Press: Recommended Parameters for IPC

Compression Level	Pressure	Appropriate for:	Frequency/Duration
Venous Ulcer/Venous Stasis			
Low compression	< 40 mm Hg	Patients with wounds, insensate, or fragile tissue (can increase with monitoring if no adverse response seen)	60 min once or twice per day. Shorter, more frequent sessions may be helpful for patients who can comply. Evening or afternoon sessions will provide the most response.
Moderate compression	40-60 mmHg	Most patients	
High compression	>60 mmHg	Extreme fibrotic changes; lipodermatosclerosis	
Lower-Extremity Lymphedema			
Low to moderate compression	30–60 mm Hg	Low to normal body mass; stage 1 or stage 2 lymphedema; patients with wounds or with insensate or fragile tissue (can increase with monitoring if no adverse response seen)	60 min once or twice per day. Shorter, more frequent sessions may be helpful for patients who can comply. Evening or afternoon sessions will provide the most response.
Moderate to high compression	> 60 mm Hg	Stage 3 lymphedema, heavy fibrosis, and/or high body mass. Use < 40 mm Hg compression over abdomen for comfort	
Upper-Extremity/Postmastectomy Lymphedema			
Low to moderate compression	30–50 mm Hg	Low to normal body mass; stage 1 or stage 2 lymphedema; patients with wounds or with insensate or fragile tissue (can increase with monitoring if no adverse response seen)	60 min once or twice per day
Moderate to high compression	40–60 mm Hg	Stage 2 or stage 3 lymphedema, heavy fibrosis, or high body mass. Use < 40 mm Hg compression over torso for comfort.	

inflated and the time the machine is deflated. In some machines, this parameter is preset. Recommendations in the literature for on:off times vary significantly; however, patient comfort is the most important determining factor. Total treatment time is the length of time (duration) the treatment device is applied. In clinical practice, this is frequently a convenience-based time determined by availability of equipment and space. Total treatment time is usually between 45 minutes and 1 hour. Recommendations in the literature vary from 45 minutes up to 4 hours. See Table 8-3 for a summary of recent studies on the effects of IPC.

Clinical Controversy

It has generally been accepted that inflation pressure should not exceed diastolic blood pressure minus 10 mm Hg. However, this recommendation is not based on consensus of many investigations. A paper by Morris[38] found that the standard pressures applied for deep vein thrombosis (DVT) prophylaxis were based on previous practice. Patients with different diagnoses—chronic venous ulcer, lymphedema, and peripheral artery disease—may need different pressures for the treatment to be successful.

TABLE 8–3. Summary of Recent Pertinent Literature on IPC

Disorder	Author/Year	No. of Subjects	Time On/Off	mm Hg of Compression	Total Treatment Time	Days per Week	Duration of Treatment	Results
Chronic venous ulcers	Vanscheidt, 2009[36]	28	Not given	Sustained pneumatic compression (SPC)—20, 30, and 40 mm Hg at the calf, ankle, and foot	SPC for 6 hours	SPC every 2 days	N/A	SPC greatest reduction of edema at 40 mm Hg
				Intermittent pneumatic compression (IPC) with 40, 50, and 60 mm Hg at the calf, ankle, and foot	IPC for 2 hours	IPC with 46 hours in between		IPC greatest reduction of edema at 60 mm Hg
Chronic venous ulcers	Nikolovska et al, 2005[24]	104	Rapid inflation: 0.5-sec inflation, 6-sec sustained compression, 12-sec deflation Slow inflation: 60-sec inflation, 30-sec sustained compression, 90-sec deflation	45 mm Hg ankle, 35 mm Hg calf, 30 mm Hg thigh in both groups	1 hour per day	Daily	6 months	Significantly better healing of ulcers in the rapid-inflation group

Continued

TABLE 8–3. Summary of Recent Pertinent Literature on IPC—cont'd

Disorder	Author/Year	No. of Subjects	Time On/Off	mm Hg of Compression	Total Treatment Time	Days per Week	Duration of Treatment	Results
DVT	Colwell et al, 2012[11]	410	8-sec compression 36- to 56-sec decompression	50 mm Hg	Continuous	Daily	10 days	When compared with low-molecular-weight heparin, use of the mobile compression device for prophylaxis against venous thromboembolic events following total hip arthroplasty resulted in a significant decrease in major bleeding events.
DVT	Dennis et al, 2013[12]	2876	11 sec on and 20–60 sec off	45 mm Hg lower chamber 40 mm Hg middle chamber 30 mm Hg upper chamber	Continuous	Daily	30 days	IPC is an effective method of reducing the risk of DVT and possibly improving survival in a wide variety of patients who are immobile after a CVA.
Lymphedema	Zaleska et al, 2014[35]	18	50 sec on in each of the 8 chambers (400 seconds for entire sleeve) and 50 sec off	120–50 mm Hg pressure decreased in the proximal chambers	45 min	Daily	3 years	Permanent decrease of limb circumference and increased elasticity of tissues
Lymphedema	Uzkeser and Karatay, 2013[31]	31	Not given	40 mm Hg	45 min	5 days	3 weeks	The pneumatic compression pump did not contribute to the reduction of lymphedema.

Peripheral artery disease	Manfredini et al, 2014[4]	12	Gradient pump (GP) = 20 sec on, 40 sec off for 5 minutes. Rest 5 minutes and repeat. Sequential foot-calf device (SFC) = 3 sec on foot/calf and 17 sec off	GP = systolic BP − 20 mm Hg with max being 120 mm Hg 120 mm Hg	GP for 35 min SFC for 2 hours	2 days	2 days with 48-hour interval in between	The gradient compression device increased foot oxygenation in severe PAD and had high compliance to treatment, unlike the sequential foot-calf device.
Infra-popliteal diffuse peripheral obstructive disease	Chang et al, 2012[16]	31	Rise time: 1 sec Delay: 4 sec Deflation time: 15 sec	120–140 mm Hg	3 hours	Daily	3 months	Improved walking ability, transcutaneous oxygen tension of the target limb, and quality of life
Peripheral vascular disease	Sultan et al, 2011[15]	171	Inflation: 4 sec Deflation: 16 sec	120 mm Hg	6–8 hours	Daily	12 weeks	Provided increased popliteal flow, ameliorated amputation-free survival while providing relief of rest pain
Peripheral artery disease	van Bemmelen et al, 2001[43]	14	Sequential foot-calf device: 3 sec on foot/calf and 17 sec off	120 mm Hg	1–2.4 hours	Daily	12 weeks	Nine limbs had increased blood flow and were salvaged for up to 2.5 years. Longer treatment time correlated with limb salvage

Treatment Application Guidelines

1. Review the patient's history and check for any condition that would contraindicate compression therapy. See Boxes 8-2 and 8-3 for precautions and contraindications to IPC, respectively.
2. All jewelry and clothing in the treatment area should be removed.
3. The entire limb should be exposed to allow for a complete examination of the limb before applying the compression sleeve.

Box 8 ■ 2 Precautions to Intermittent Pneumatic Compression

Recent skin graft	Application of IPC could interfere with adherence of graft.
Acute local dermatologic infections	Contact with the stocking or the IPC sleeve as well as perspiration can cause spread of infection.
Impaired sensation or mentation	Injury could occur.

Box 8 ■ 3 Contraindications to Intermittent Pneumatic Compression

Acute pulmonary edema	Application of IPC can return interstitial edema back to the venous circulation—increasing the stress on the heart and lungs, which are already compromised.
Congestive heart failure	As with acute pulmonary edema, application of IPC can increase stress on the heart and lungs.
Recent or acute deep vein thrombosis	Application of IPC to an area with DVT can cause the thrombus to dislodge from a vein wall, travel to the heart or lung, and block an artery.
Acute fracture	In cases of acute and unstable bone fracture, changes in pressure could cause movement and delay healing.
Uncontrolled hypertension	Compression may elevate blood pressure and increase vascular load to the heart.

4. Measure and record the patient's blood pressure, heart rate, and respiratory rate to create a baseline for comparison during and after treatment and to establish a maximum pressure guideline.
5. Circumferential measurements of the limb must be determined and recorded before and after each treatment session (Fig. 8-3). These are most easily documented and compared if recorded in a chart or table format. Select landmarks that can be easily located and will not change as edema decreases. References to bony landmarks are generally most easily reproducible. Measurements are most accurate if performed by the same examiner using the same measurement technique each treatment session. Measurements are generally taken every 10 cm from the landmark. The same landmarks should be used before and after treatment and in subsequent treatments to allow for accurate assessment of changes. Volumetric measurements may also be used.
6. Patients will need to remain in one position for the duration of the treatment, so they must be positioned comfortably with maximum access to their environment. Positioning to allow reading, viewing television, or even computer access will occupy patients during treatment and help alleviate boredom.
7. Patients should empty the bladder before initiating treatment. Fluid returning to the circulation is filtered through the kidneys; thus, urinary urgency is not uncommon. Trips to the restroom can result in delay or interruption of the treatment.

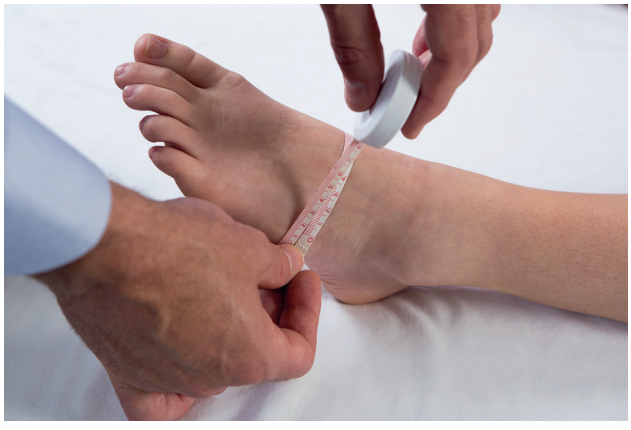

Fig 8 ■ 3 Measurement of foot circumference prior to treatment with IPC.

8. A treatment table with appropriate height and size to accommodate the area being treated is essential. The patient should not have the feeling that any body part is unsupported or will slide off the edge of the table with minimal movement or position shifting. Pillows, wedges, or other supports should be used to keep the area being treated in a comfortable position while elevated above the level of the heart. An elevated position can encourage venous return.
9. If there is an open wound in the treatment area, it should be covered with a dressing, such as a sterile gauze pad or Telfa pad that is secured in place.
10. The limb to be treated should be clean and dry.
11. The limb's skin sensation and the patient's mentation must also be assessed, because patients with impaired sensation or decreased awareness may not realize when there is too much pressure over a nerve or bony area or be able to notify staff if there is a problem.
12. Before applying the sleeve to the limb, a stockinette should be placed over the limb to be treated (Fig. 8-4). Wrinkles in the stockinette should be smoothed out because these can cause restriction and discomfort.
13. After the sleeve is applied over the stockinette, the hose to the pump and the sleeve should be connected. In units with sequential compression, there will be multiple hoses. It is important to make sure that the hoses are inserted in the proper location (i.e., order) to ensure the proper inflation sequence is administered. The hoses and receptacles are frequently color-coded to avoid confusion.
14. After the hoses are attached, set the appropriate parameters on the pump's dials. Most IPC devices are supplied with manufacturer's guidelines that usually contain a wide range of options for safe application. (See Tables 8-1 and 8-2.)
15. The patient must be given a bell or call button to alert the practitioner during treatment if any discomfort or unusual sensations occur.
16. Monitor the patient's vital signs during treatment. Modify or end treatment if there is a significant change.
17. Treatment should be terminated while the limb is still in an elevated position (Figs. 8-5 and 8-6). To terminate treatment, turn the pressure dial to the off or "0" position and turn the power off. Disconnect the tubing from the machine and treatment sleeve and then remove the treatment sleeve from the patient. Remove the stockinette and assess the skin for any pressure or reddened areas.

Fig 8 ■ 4 Application of stockinette to limb prior to treatment with IPC.

Fig 8 ■ 5 IPC for lower extremity with leg in elevated position.

Fig 8 ■ 6 IPC for upper extremity with arm in elevated position.

18. After treatment, provide assistance as needed to help the patient to a standing position. Slight dizziness or unsteadiness is not uncommon because of the circulatory changes that may occur with prolonged immobilization. If these symptoms are prolonged or severe, another form of treatment may need to be considered.
19. If indicated, range of motion or other exercises may be performed with the extremity elevated, if possible. After appropriate exercise, application of a compression garment or elastic compression wrap will help maintain the effects of the treatment. Also instruct the patient in an appropriate home treatment program.
20. The intervention session should be clearly documented, including the posttreatment circumferential measurements.

Possible Complications of Treatment

- Swelling in other areas may occur as a result of fluid returning to the circulation. This is often controlled with elevation and gentle active exercise.
- Stiffness of joints within the treatment area sometimes occurs as a result of prolonged immobilization in one position. This generally resolves quickly and is not a cause for concern unless it lasts longer than about 15 to 30 minutes. If stiffness persists, try an alternative, better-supported position.
- Shortness of breath may indicate fluid overload in the lungs or pulmonary embolism. Monitor shortness of breath closely and contact the physician if this persists.
- Numbness or tingling in the distal extremity may indicate DVT or nerve irritation or nerve damage. Stop treatment and assess immediately if your patient complains of numbness or tingling.

Key Point! Pharmacologically controlled hypertension should be considered a precaution to IPC therapy. Monitoring blood pressure before and after IPC treatments is recommended to monitor the hemodynamic response to compression.

CASE STUDY 8-1 IPC for Lower-Extremity Ulcer

Joan is a 55-year-old African American female who has come to the clinic for treatment of a right lower-extremity ulcer. The ulcer is located 2 cm proximal to her medial malleolus. Surrounding her wound is hemosiderin staining of her right and left leg. Her legs swell as the day progresses. She is 5 feet, 5 inches tall and weighs 250 pounds, with a BMI of 41.6. She is a cashier at a grocery store and stands for most of her 6-hour shift. She leads a sedentary lifestyle and has a medical history of diabetes mellitus and high cholesterol. Medications include Glucophage, Lipitor (statin), and 81 mg of aspirin.

CLINICAL DECISION-MAKING

1. What is the most likely cause of her lower-extremity wound?
ANSWER: Joan most likely has venous insufficiency. Her wound is on the medial aspect of her leg (venous ulcers are usually on the medial lower extremity). Swelling of the leg increases as the day progresses and is consistent with venous stasis. Brown staining of the legs is consistent with hemosiderin-staining red blood cells that collect in the pooled blood and are lysed by the body, causing hemosiderin to stain the skin.

2. Is Joan a candidate for using IPC?
ANSWER: Yes, IPC is indicated to aid in healing her venous stasis ulcer, using the following parameters: rapid inflation, 6 seconds on/12 seconds off, pressure set at 35 to 45 mm Hg at the calf and ankle, for 1 hour daily.

3. How would you objectively assess the wound?
ANSWER: Measure the wound length, width, and depth; trace the wound; or use digital photography. Note any necrotic versus granulation tissue.

4. What are the expected goals associated with IPC?
ANSWER: IPC should improve circulation in her leg, decrease edema, and increase the healing rate of her wound, thereby improving her tolerance for standing and walking and improving her quality of life.

5. Should Joan perform exercise?
ANSWER: Yes. While the evidence for exercise being able to improve venous function and healing of ulcers is lacking,[43] aerobic exercise as recommended by the American College of Sports Medicine (ACSM)[17] will address her risk factors for coronary artery disease and peripheral artery disease. She has three out of six risk factors for PAD.[44] For individuals

CASE STUDY 8-1 IPC For Lower-Extremity Ulcer—cont'd

with PAD, the ACSM recommend adherence to the FITT (frequency, intensity, time, and type) principles:

- Frequency: Weight-bearing aerobic exercise 3 to 5 days per week; resistance exercise at least 2 days per week.
- Intensity: Moderate intensity (i.e., 40% to 60% VO_2-reserve) that allows the patient to walk until a pain score of 3 (i.e., intense pain) on the 4-point pain scale is reached.
- Time: 30 to 60 minutes; initially, some patients may need to start with 5- to 10-minute bouts and exercise intermittently to accumulate a total of 30 to 60 minutes.
- Type: Weight-bearing aerobic exercise, such as walking, and non-weight-bearing exercise, such as arm and leg ergometry. Cycling may be used as a warm-up but should not be the primary type of activity. Resistance training is recommended to enhance and maintain muscular strength and endurance.

Documentation Tips

The following should be documented with application of IPC, preferably in a table format for ease of comparison of response to each treatment session:

- Vital signs taken before, during, and after each treatment session
- Circumferential measurements before and after treatment
- Area treated
- Size and description of wound (if present)
- Position of patient during treatment
- Inflation pressure
- On:off ratio
- Total treatment time
- Response to treatment

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CHAPTER 9

FOUNDATIONS OF CLINICAL ELECTROTHERAPY

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OVERVIEW OF ELECTROTHERAPY

Electrotherapy Is Not as Challenging as It May Seem

Clinical electrotherapy is sometimes considered challenging and difficult to comprehend. This is far from reality if one possesses some basic understanding of the fundamentals of electricity and charge, anatomy and physiology, and clinical application. This chapter will set the foundations to make clinical electrotherapy understandable and, more importantly, clinically useful and beneficial to patients. Despite the varied terminology and uses of electrotherapeutics across the many areas of rehabilitation, there is one common purpose: application of stimulation to elicit or facilitate some desired therapeutic response. Whether electrical stimulation (ES) is used to activate skeletal muscle for strengthening or improving volitional movement, relaxing skeletal muscle to facilitate functional activity, decreasing pain, improving circulation, or facilitating tissue healing all are based on the stimulation of tissues from applied electric currents.

This chapter will delineate the steps to learning ES in a way that will be user-friendly, trying to minimize confusion as you develop new terminology and understanding. This can be likened to learning to drive a car; if you are taught to drive in one brand of car, you are likely to be successful if you use those same skills, knowledge, and

experience when driving a comparable vehicle; therefore, effectiveness is based on a competent knowledge and application of fundamental basics.

The overall purpose of this chapter is to address the fundamental principles of electricity and electrical charge that underlie the therapeutic effects for which electrotherapy is used. This chapter and the following will present not only the "how," but also the "when," "why," and "what" of electrotherapy. Chapters 11, 13, and 14 will present more specific applications of clinical electrotherapy for patients with pain (Chapter 11), musculoskeletal impairment (Chapter 13), and neurological impairment (Chapter 14).

PRINCIPLES OF ELECTRICITY: MAKING THE PHYSICS MAKE SENSE

To discuss clinical applications and use of electrotherapy without first addressing the fundamentals of electricity is like asking someone to play a game without telling them how. Without some prior knowledge and understanding of the basic rules and strategy of the game, it may appear as if someone is truly playing the game, but eventually the outcome will reveal the lack of knowledge. So it is with clinical electrotherapy—without an understanding of the fundamentals, clinical effectiveness is less likely. A functional and useful understanding of electrotherapy must include and begin with the basics.

Charge

Charge is the fundamental underlying property of electromagnetic force and serves as the mechanism by which living cells communicate with one another. Measured in coulombs (C) or microcoulombs (μC), charge is obtained by the addition or removal of electrons and occurs when atoms of elements are acted upon by external physical forces such as friction, heat, and chemical or electrical sources. Although atoms are composed of positive protons and negative electrons, the concept of charge is specific to the net gain or loss of electrons (Fig. 9-1). A net gain of electrons results in a negative charge, whereas a net loss of electrons results in a positive charge.[1] An object that becomes positively charged does so by the loss of electrons, not by the addition of protons. A positively charged object has lost electrons,

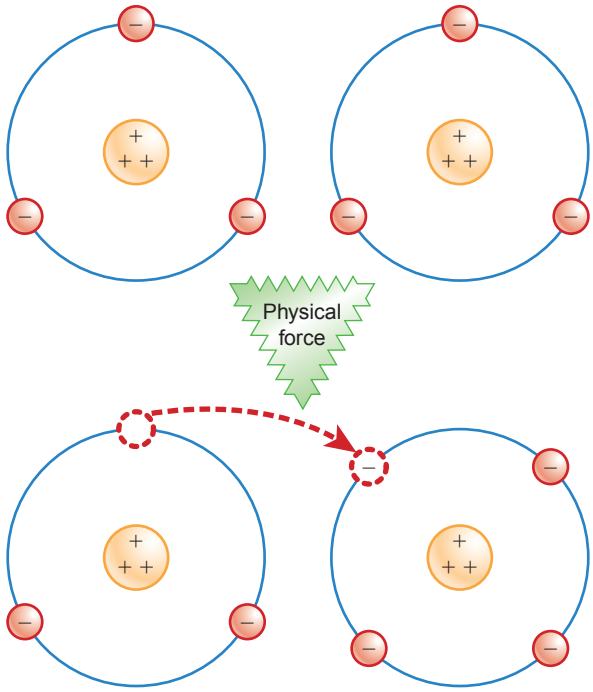


Fig 9 ■ 1 "Charge" is conferred by transfer of electrons. Addition of electrons results in net negative charge, whereas loss or removal results in net positive charge.

whereas a negatively charged object has gained electrons. An atom or molecule that has gained or lost an electron is termed an *ion,* and the process by which an atom or molecule acquires a negative or positive charge is termed *ionization*.[2] Thus, the bottom line is, it's all about the electrons.

Four fundamental properties of electrical charge explain how charge is used for therapeutic purposes:[2]

1. There are two types of charge—positive and negative.
2. Like charges repel while opposites attract.
3. Charge is neither created nor destroyed.
4. Charge can be transferred from one object to another.

The fundamental concepts of charge and ions underlie the clinical uses of electrotherapy from the more obvious iontophoresis, named for the administration of select medicinal ions, to all other forms of electrotherapeutics.

Polarity and Creation of Electric Force Fields

Charge is further described by its polarity, with *polarity* referring to the net charge of an object—being either negative or positive. In a simple circuit, such as one created when electrodes are applied to a patient, one

electrode is the positive pole and one is the negative pole. The pole or electrode with net negativity is termed the *cathode,* and the pole or electrode with net positivity is the *anode*.[1] A common household battery is a simple example of charge and polarity (Fig. 9-2). One pole has a concentration of electrons and the other has a deficiency.

Key Point! Use of the terms *anode* and *cathode* can be confusing, but clarity depends on the type of electrical system being discussed. From a chemistry perspective, the type of reaction is used to define anode and cathode. In this model, the cathode is the electrode where reduction of a chemical species is occurring, while the anode is the electrode where oxidation of a chemical species is occurring. For a common household battery, which discharges electrical energy until depletion, the anode is the part that gives up electrons. While discharging energy, the anode is the negative electrode. When recharging a battery, the anode becomes the positive electrode. The cathode is the part of the battery that absorbs electrons. While discharging energy, the cathode is the positive electrode. When recharging a cell, the cathode becomes the negative electrode. In a biological system, such as our neurons, which continually depolarize (i.e., discharge) and repolarize (i.e., recharge), the anode is considered the region, or side of the cell membrane, with a deficiency of electrons and the cathode is the region, or side of the membrane, with excess electrons.

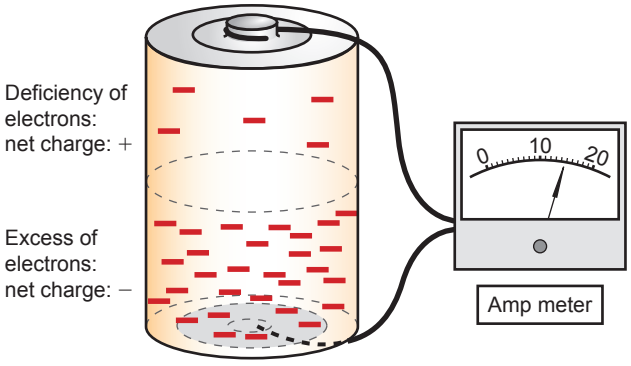


Fig 9 ■ 2 A common household battery. The separation of charge creates a concentration gradient. When connected, current flows between the poles.

Keeping in mind the second fundamental concept of electricity—that like charges repel and opposite charges attract—the force created by the separation of charge may be one of attraction or repulsion and represents the *electrical field*. Magnets and magnetic fields offer a good analogy of the force of an electrical field. If like poles of two magnets are slowly brought near each other, the magnets begin to repel each other. If opposite poles of the magnets are approximated, the magnets are "pulled" toward each other (Fig. 9-3). The larger the magnets, the larger and stronger the force field.

Fundamentals in Clinical Practice: Principles of Charge, Ions, and Electrical Fields

The clinical relevance and applicability of electrical charge, ions, and electrical fields can be seen during the use of iontophoresis. For example, a clinician chooses to treat a patient with dexamethasone sodium phosphate, a negatively charged anti-inflammatory drug, or medicinal ion, which is delivered transcutaneously via electrical current. Knowing that like charges repel, the practitioner attaches the negative electrode of a circuit to the electrode containing the like-charged drug. The force of the electrical force field created by charge will "push" or repel the negatively charged medicine away from the negative electrode and into the tissues requiring treatment.

Voltage

The force of attraction or repulsion created by an electric field represents potential energy. The greater the force, the greater the potential energy. This force is termed *voltage* and represents the driving force that moves electrons.[1] The unit of electrical force is the volt (or millivolt).

A voltage force is best explained when considering the interaction of two magnets or two charged bodies as one approaches the other. When a larger magnet or charged body with a greater polarity or charge approaches another stationary magnet or charged body with the same but smaller polarity or charge, the force field exerted by the larger body on the smaller body increases as the distance between the two decreases. At some point, the repulsive force overcomes the inertia acting on the smaller body, and the smaller body is repelled away from the larger. The force of the electrical field that caused the smaller

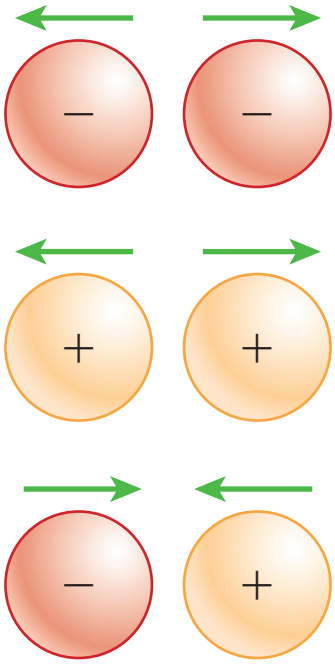


Fig 9 ■ 3 Like charges are repelled away from each other. Opposite charges are attracted to move closer to each other.

body to move is the voltage force. Voltage may also be referred to as the *electromotive force* or *electrical potential energy*.[1] The term *electromotive force* refers to the property of the force inducing or causing movement and, in this case, movement of electrons. Electricity produced by a power plant is moved, or delivered, to homes miles away. This requires a considerable amount of work. To deliver electricity over this distance, a large amount of voltage is required. This is why you often see warning signs reading "High Voltage."

The change in electrical potential energy and the distance charges are moved represents the effects of a voltage force. Voltage can be thought of as the force that pushes charge. Keeping with the fundamental principle of electrical charge, voltages are created whenever oppositely charged particles are separated or when like charges are brought closer together. In terms of conventional electricity for purposes of power, these charged particles are the electrons that are moved within a powered machine and wires. In our biological system, however, voltage forces are created by the uneven distribution of charged particles, resulting in regions that are more or less negative or positive to adjacent regions. In tissues, these charged particles are ions, such as sodium (Na^+), potassium (K^+), and chloride (Cl^-).[3] In conventional circuits and electrical generators used to deliver power to our homes and electrotherapeutic devices, the charged, flowing particles are electrons. This text focuses on the electrophysiological and therapeutic effects of electrical stimulation on the human body; thus the terms *ions* and *electrons* will be used in place of *charged particles* (Box 9-1).

Key Point! In our bodies, the separation of charges (i.e., ions) across the cell membrane creates a concentration, or electrochemical, gradient. The greater concentration of sodium outside versus inside the cell and the greater concentration of chloride (Cl^-) inside versus outside the cell reflects an electrochemical gradient. It is this separation of charge across the cell membrane, and thus electrical potential, that establishes a voltage force allowing the cell to depolarize and initiate or transmit electrical signals via movement of ions.[3]

Conductors and Insulators

For ions or electrons to move freely, they require materials that permit such movement. Materials in which ions or electrons move freely are termed *conductors*. Metals and water are examples of conductors. In the human body, tissues such as muscle, nerve, and bodily fluid serve as conductors (Fig. 9-4). In part, this reflects the high water content and presence of ions in these tissues.

Box 9 ■ 1 Hands-On Learning

To better understand the role ions have in the creation of electrical current, place the lead wires of an electrotherapy device into a small bowl, making sure the leads are separated and do not touch. (Do not submerse the device in the water!) A device with a visible amp meter or output for monitoring current flow is preferred. (Note: A voltmeter is not the same as an amp meter and may not work in this example.) First, pour distilled water into the bowl. After turning on the device, increase the amplitude. What happens? Nothing! Note that the meter does not move, indicating that no current is flowing. If your device lacks a meter, you may hear a warning beep indicating the lack of a complete circuit and no current flow. This occurs because distilled water has had all ions removed, so there are no charged particles to move in the bowl. Now add salt to the water and watch the amp meter register flow of current. This occurred because the salt (NaCl) dissociated into sodium (Na^+) and chloride (Cl^-), allowing charged particles to move. The movement of ions represents flow of current.

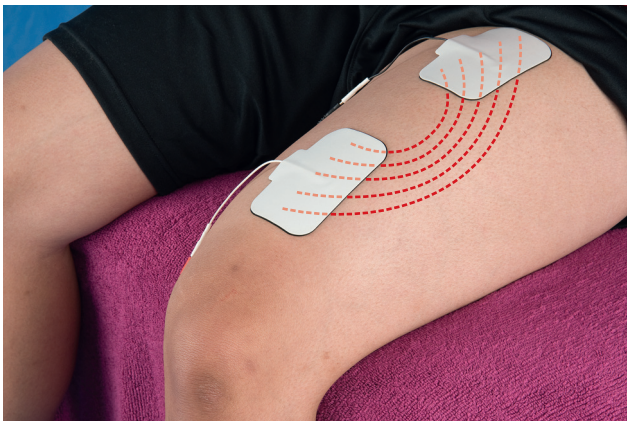


Fig 9 ■ 4 Current flows between electrodes by passing through biological tissues, which serve as conductors. This allows current to reach the peripheral nerves and muscles.

Materials in which charged particles are not free to move or do not move easily are termed *insulators.* Rubber and plastic are typical materials considered to be insulators. Often conductors and insulators exist together both in everyday life and in biological examples. Take, for instance, an extension cord used in our homes: The metal wire inside conducts current to our appliances while the plastic coating insulates the current, preventing it from entering our body and shocking us. In the human body, fat is an insulator and does not allow ions or electrons to move as freely as do nerves.

Key Point! Myelin, a lipid-rich material surrounding the axons of nerves, serves as an insulator, allowing rapid transmission of electrical signals along the nerve. When myelin is damaged, insulation is compromised and the speed of conduction is slowed.

Current

The movement of ions or electrons in a conductor in response to a voltage force is termed *current.* The flow of current is directly proportional to the magnitude of the driving force (i.e., the voltage). Current is the quantity or amount of ions or electrons flowing at a given time and is designated by the universal symbol I.[1] The international unit for current is the ampere (amp or A), but most therapeutic applications of current use milliamperes (mA, or thousandths of an ampere). Of note, microcurrent is conventionally defined as current less than 1 mA, as in the unit microamperes (μA or millionths of an ampere) (Box 9-2).

Box 9 ■ 2 What Direction Does Current Flow?

By convention, flow of current is designated as flowing from positive to negative. However, the astute student may notice that this is inconsistent with the laws of mass action and concentration gradients. If current is the movement of electrical charge and electrical charge is defined by gain or loss of electrons, the law of mass action tells us that movement should occur from high concentration (areas of more electrons or negativity) to low concentration (areas of less electrons or more positivity) (Fig. 9-5). Then the flow from a greater concentration of electrons to one of lesser electrons must be from negative to positive. But this is opposite. Indeed, this is how electrons move, but it remains that current is designated as flowing from positive to negative despite being contrary to scientific law. The details underlying this inconsistency are varied, but scientific lore has it that scientists studying electrical charge noted movement of "electric fluid" but were uncertain as to what exactly was moving, how, and in what direction. Out of attempts to describe their observations, they were forced to designate a direction of the movement and to arbitrarily designate the place from which flow seemed to originate as "positive" and the place to which flow appeared to go as "negative." This designation of the direction of current flow remains despite counterexplanation.

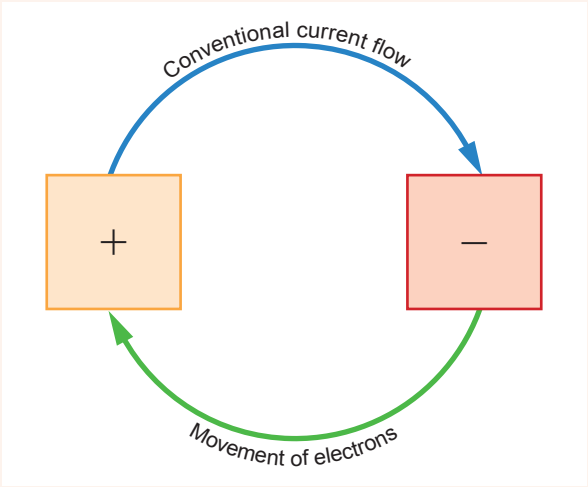


Fig 9 ■ 5 Flow of current versus flow of electrons.

Fundamentals in Clinical Practice: Human Tissue as Conductors and Insulators

While treating a patient with low back pain, a clinician chooses electrical stimulation to help activate the lumbar muscles in order to lessen the pain. The patient is obese with a large amount of lumbosacral adipose tissue that does not conduct current well. For current to penetrate deep enough to stimulate the nerves of the lumbar muscles, the clinician

increases the intensity of the current. Because adipose tissue is a better insulator than a conductor, it resists the current and results in an uncomfortable stinging sensation at the electrodes as the high current activates nociceptors.

Ohm's Law: Resistance, Capacitance, and Impedance

So far, we have described current as the free or unresisted flow of ions or electrons in a conductor in response to an applied voltage. The magnitude of current flow is directly proportional to the voltage force and quantity of charge moving. Rarely, if ever, does current flow in biological tissues without some kind of resistance. *Resistance* is opposition to the flow of current and comes in many forms in the body (Box 9-3).[1]

The relationship between resistance and the flow of current is given in Ohm's law: I = V/R, where current (I in amperes) is directly proportional to the voltage force (V) pushing the current and inversely proportional to resistance (R) to the voltage force.[2] The standard international unit of resistance is the *ohm*. From a more clinical view, Ohm's law tells us that the more resistance there is to the flow of current, the less current there will be. Being aware of factors that affect biological resistance is important when applying ES for therapeutic use. Calloused or very dry skin, for example, presents high resistance, so the flow of current through an area such as the heel or foot may be greatly reduced. This knowledge can impact applications, such as iontophoresis, when trying to move ions across the skin into the plantar soft tissues.

Box 9 ■ 3 Resistance to Current Flow

Resistance to the flow of current varies but comes from many biological sources:

- Skin
- Hair
- Fascia
- Ligament
- Callus
- Fat
- Bone
- Tendon
- Scar

Capacitance and impedance are properties of current flow and are related to resistance. Capacitance is the degree to which electrical charge is stored in a system containing conductors and insulators, such as the human body.[1] Capacitance arises from the storage of charge in an insulator that is within a field of current. Because current does not pass freely across an insulator, the electrical potential across the insulator increases the electrical potential energy of the molecules of the insulator. This storage of electrical energy in the insulator reflects capacitance. When flow of current ceases, the energy stored in the insulator flows back through the conductors (Box 9-4). The clinical significance of capacitance is seen in some devices that have a capacitor. Most modern stimulating devices linearly decrease the flow of current when the application has ended. This gradual decrease in current allows the capacitor to discharge stored

Box 9 ■ 4 Static Shock: Why Does It Happen?

When acted upon by external forces, such as heat, friction, chemical, electrical, or other physical forces, the number of electrons may be altered. Rubbing your feet on the carpet results in a transfer of electrons from the floor to your body via friction. These electrons are temporarily stored in your body, which acts as a capacitor. When you touch a metal object, it acts as a conductor, creating a circuit, and the stored electrons flow freely from you into a metal object (Fig. 9-6). The physical effect is a surprising shock!

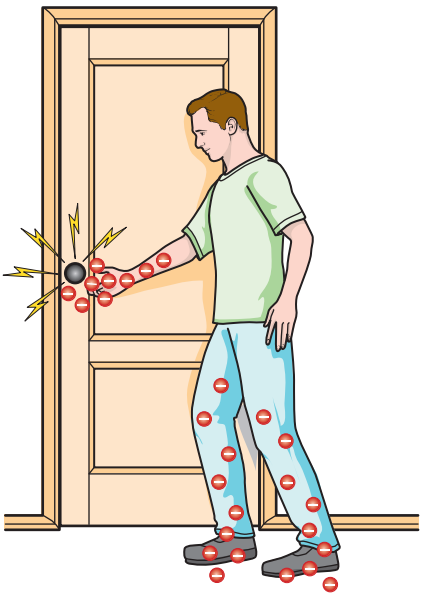


Fig 9 ■ 6 Electrons are transferred, resulting in accumulation of charge and flow when a circuit is completed.

charge. If the clinician abruptly terminates the application by unplugging or turning off the device before the current has decreased, the current stored in the capacitor can freely flow through the electrodes. This is often perceived as a very brief and uncomfortable surge in the stimulation's intensity.

Impedance is a form of resistance to the flow of current but is frequency-dependent. Impedance more accurately reflects the voltage-to-current ratio because it incorporates both the properties of resistance and capacitance. In simplest terms, impedance is the resistance to the flow of alternating current, whereas resistance is specific to direct current. Clinically, gels and adhesive conductive agents on electrodes serve to decrease impedance and increase conductivity between the electrode and skin. This emphasizes the importance of appropriate and adequate use of coupling agents, such as electroconductive gel, and the importance of regularly inspecting the integrity of adhesive electrodes.

CURRENTS AND WAVEFORMS

Terminology used to describe electrotherapy can seem confusing; however, most of this is due to inconsistency in the terms and language used to describe electrotherapy and procedures. Greater understanding and increased comfort with the language of electrotherapy is readily achieved if some basic fundamental terms are used. In addition, a clinically competent understanding of the terminology of currents and waveforms is easily obtained if you keep in mind that these currents and waveforms are merely modifications of the two most fundamental electrical currents: direct current (DC) and alternating current (AC).

From the standpoint of electrophysics (a branch of physics from which electrotherapy comes), there are only two types of electrical current: direct current and alternating current. Although these currents can be used for therapeutic purposes, the most common form of current used in electrotherapeutics is pulsed current, or pulsatile current. This is the terminology we will use in this text. In regard to applied science, pulsed current is the third major class of electrical current. (Terminology and descriptions used in this text are consistent with the American Physical Therapy Association's *Electrotherapeutic Terminology in Physical Therapy* and the Academy of Clinical Electrophysiology and Wound Management.[1])

The Basic Currents

Understanding electrical currents is easy when visualizing their specific parameters. Consider a graphical depiction of each current and the particular ways in which each would be described or drawn. When you begin to use terms to explain the shape, magnitude, and duration of currents, you are describing the current *waveform*. The waveform is simply a depiction of the characteristics that represent a given current. A good example of a waveform is shown on the Etch A Sketch (Fig. 9-7).

All currents have parameters in the vertical (*y* coordinate) and horizontal (*x* coordinate) directions (Fig. 9-8). Parameters in the horizontal axis are used to describe and quantify time or duration characteristics of current

Fig 9 ■ 7 An Etch A Sketch shows the basic *x-y* components of waveforms, representing time and amplitude of current.

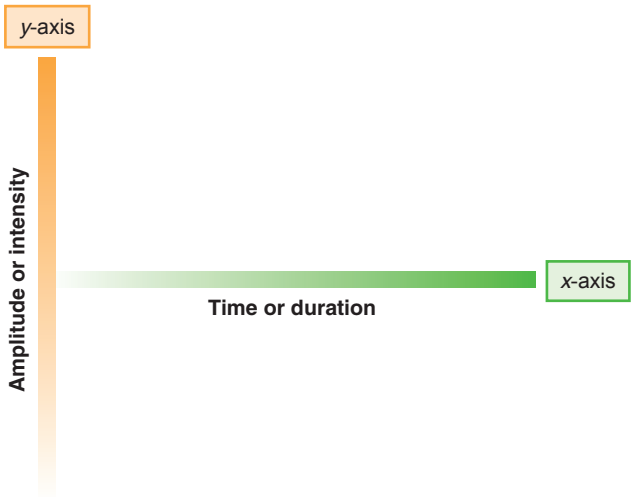


Fig 9 ■ 8 Basic *x-y* axes for time and amplitude. Time may be in seconds (sec), milliseconds (msec, 10^{-3}), or microseconds (μsec, 10^{-6}). Intensity may be in volts (V), millivolts (10^{-3}, mV), microvolts (10^{-6}, μV), amperes (amp), milliamperes (10^{-3}, mA), or microamperes (10^{-6}, μA).

(in milli- or microseconds), whereas parameters in the vertical axis are used to describe or quantify magnitude or intensity (in milli- or microamps or milli- or microvolts).

Direct Current

Direct current is the continuous unidirectional flow of ions or electrons for at least 1 second[1] (Fig. 9-9). Here, the term *direction* implies flow from positive to negative or negative to positive. By convention, deviation from the isoelectric baseline in the upward direction implies current flow in the positive direction; conversely, deviation in the downward direction implies flow in the negative direction. (Ironically, this designation is reversed for electromyography, as discussed in Chapter 17.) The most common or familiar source of DC is the household battery. When the poles are connected in a circuit, such as when putting batteries in a device, current will flow. Variations of DC exist, but to accurately be called DC, they must remain unidirectional and uninterrupted for a period of time.[1] Other forms of DC include *interrupted DC,* where the direction of flow ceases after 1 second before resuming in the same direction for at least 1 second; *reversed DC,* where the flow ceases after 1 second before resuming in the opposite direction for at least 1 second; and *interrupted reversed DC,* which is a combination of both.

When using DC, one of the electrodes will be the anode (positive) and one will be the cathode (negative). This will remain so unless the direction of current reverses, as in reversed DC. The most common clinical uses of DC are for iontophoresis (Fig. 9-10) and wound care.

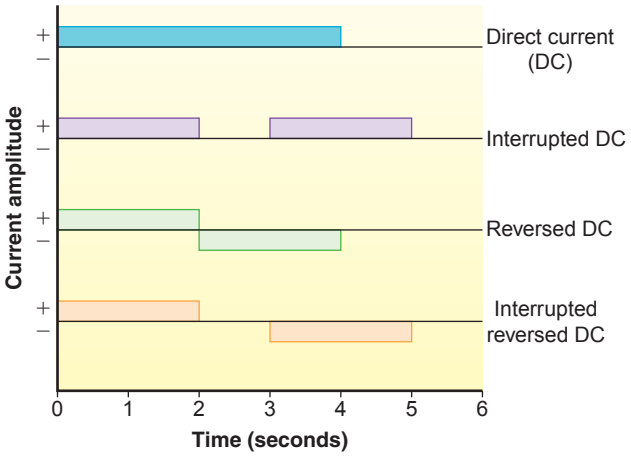


Fig 9 ■ 9 Direct current (DC) comes in many forms, conventional DC (top) being the most commonly used.

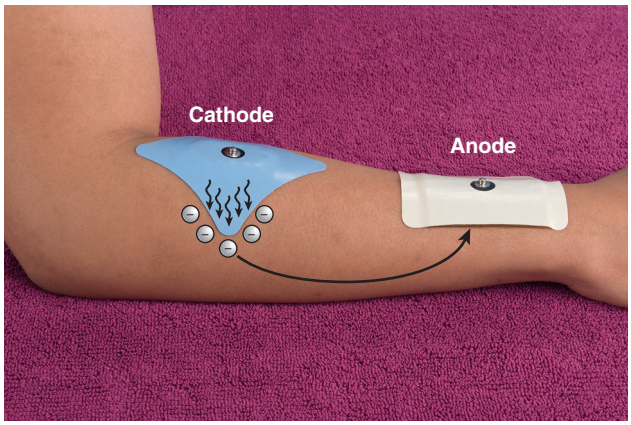


Fig 9 ■ 10 Iontophoresis uses direct current to move ions. Negatively charged ions placed under the cathode will be "pushed" or repelled into the tissue.

Alternating Current

In contrast to DC, alternating current (AC) is the uninterrupted bidirectional flow of ions or electrons and must change direction at least one time per second[1,4] (Fig. 9-11). The rate at which AC switches direction is termed *frequency* and is described with the international unit *hertz* (Hz) or in the unit *cycles per second*. The most common or familiar source of AC is the electricity coming from the wall outlets in our homes, supplying electricity to most appliances (e.g., your cell phone charger uses an AC source). Clinical use of pure sinusoidal AC is not common; however, modulated forms of AC, such as burst-modulated AC (i.e., Russian current) and amplitude-modulated AC (i.e., interferential current), are commonly used and will be discussed later this chapter.

Key Point! Think about it: AC must change direction at least one time per second. If it does not, then what type of current would it be? DC.

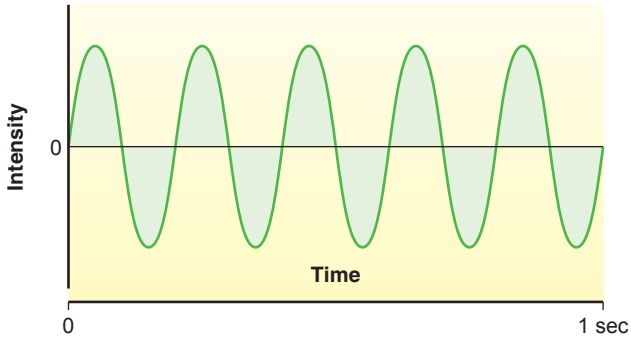


Fig 9 ■ 11 Alternating current (AC) as a sinusoidal waveform.

Pulsed Current

Because the electrophysiological effects of DC or AC are not well suited for most electrotherapeutic applications, a third category of current has been designated: pulsed current (PC). Pulsed current, sometimes termed *pulsatile current,* is the uni- or bidirectional flow of ions or electrons that periodically ceases for a period of time before the next electrical event.[1]

The definition of *PC* reflects flow of current that ceases before the next "event." This event is a *pulse*—an isolated electrical event separated from the next by a period of time termed the *interpulse interval*. Referring back to the *x-y* coordinate system, the duration (*x* coordinate) and amplitude (*y* coordinate) of the pulse give the time and magnitude of the voltage or current of the pulse, respectively. Descriptions of PC refer to the shape or configuration of the pulses (Box 9-5). Common forms of pulsed current include square, rectangular, and triangular pulses (Fig. 9-12). Waveform shape (i.e., the amplitude and duration of a pulse) has been shown to impact tolerance and the magnitude of effect when stimulating skeletal muscle.[4–6] This is discussed further in Chapter 13.

The generation of two or more consecutive pulses separated from the next series of consecutive pulses is termed a *burst,* and the time between bursts is the *interburst interval*[1] (Fig. 9-13). The frequency at which bursts are generated is the *burst frequency,* while the frequency of the underlying waveform in the burst is termed the *carrier frequency.*[1,7,8] In some cases, the uninterrupted generation of pulses at a fixed frequency is used. This is termed a *train* of pulses and is different from bursts in that there is no interruption of the pulses at a set frequency (i.e., bursts). The practical use of burst and carrier frequency is presented later in this chapter.

Box 9 ■ 5 PC Versus DC: Effects on Muscle Stimulation

PC is used for stimulating skeletal muscle for strengthening and activity. Because PC is a series of pulses, muscle fibers can be stimulated frequently, resulting in tetanic contraction. But what will happen if using DC? Won't this result in a tetanic contraction because DC flows continuously? No! DC will depolarize the muscle and cause a single twitch, but only one. To get a tetanic contraction, the muscle must depolarize and repolarize before depolarizing again. The time between successive pulses of pulsed current allows the muscle fibers to repolarize so they can be depolarized again. DC results in a sustained state of depolarization. The muscle cannot repolarize until the DC temporarily ceases.

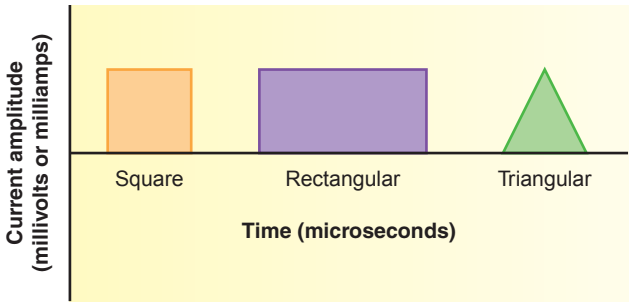


Fig 9 ■ 12 Pulsed current comes in many shapes, including square, rectangular, and triangular waveforms.

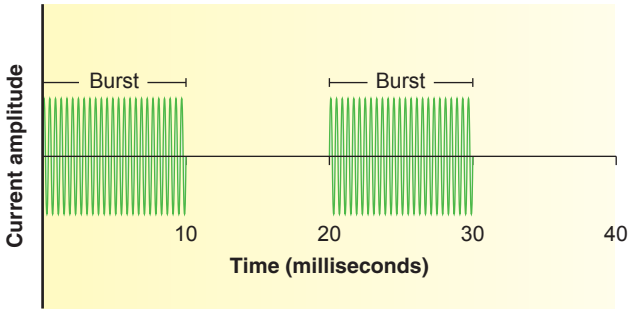


Fig 9 ■ 13 Burst and carrier frequency: In this figure, a 2,500 Hz AC carrier frequency is delivered in bursts. The figure shows 10-msec bursts with 25 cycles per burst and 10 msec between bursts. Twenty-five cycles of AC in 10 msec equates to 400 μsec per cycle and a carrier frequency of 2,500 Hz. Two bursts in 40 msec equate to a burst frequency of 50 bursts per second (bps).

Key Point! Think about it: What is the longest duration a pulse can have and still be termed a pulse? Less than 1 second.

Pulsed current may be monophasic or biphasic, with a *phase* being the flow of current in one direction for a short period of time[1] (Fig. 9-14). A *monophasic* pulse deviates from the isoelectric line in only one direction, depending on what direction the current initially flows before ceasing (i.e., returning to the isoelectric line). With monophasic pulses, ions or electrons move briefly

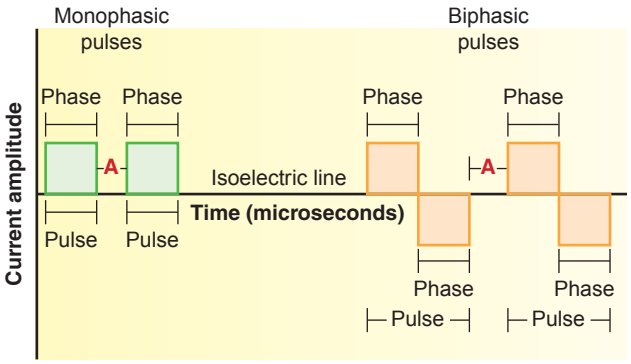
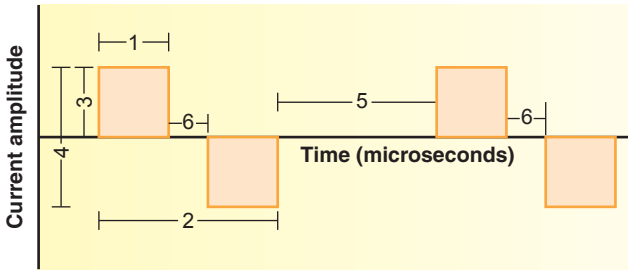


Fig 9 ■ 14 Mono- and biphasic current. For monophasic pulses, *phase* and *pulse* are synonymous. Biphasic pulses have phases that deviate from the isoelectric line in different directions. (A represents the interpulse interval.)



1. Phase duration
2. Pulse duration
3. Peak amplitude
4. Peak-to-peak amplitude
5. Inter-pulse interval
6. Intra-pulse interval

Fig 9 ■ 15 Amplitude and time (duration) characteristics of pulsed current.

in only one direction before stopping. *Monophasic pulsed current* is the delivery of repeated monophasic pulses separated from each other by an *interpulse interval;* it is produced by intermittently interrupting a DC current source. Some sources commonly use the term *pulsed DC.* However, this term is not preferred and is used to mean a monophasic pulsed current.

In contrast to a monophasic pulse, a *biphasic* pulse is one that deviates from the isoelectric line first in one direction, then in the other direction. *Biphasic pulsed current,* therefore, is the delivery of repeated biphasic pulses separated from the next pulse by an interpulse interval. By definition, with monophasic pulsed current, a *pulse* and a *phase* are synonymous.

When further describing and differentiating pulsed waveforms, similarity in amplitude and duration of each phase must be considered. The amplitude-dependent characteristics used to describe waveforms reflect the *y* coordinate when plotting waveforms (Fig. 9-15). *Amplitude,* often referred to as *intensity,* is the magnitude of current or voltage with respect to the isoelectric or baseline on the *x-y* current-time plot. Amplitude is reported in units of current (amps, milliamps, or microamps) or voltage (volts, millivolts, or microvolts) and can be described in terms of a single phase or both phases. Most uses of ES use milliampere amplitude. The highest current or voltage reached in a phase of a monophasic pulse or in any one phase of a biphasic waveform is termed the *peak amplitude.* The highest value measured from the peak of the first phase to the peak of the second phase of a biphasic waveform is termed the *peak-to-peak amplitude.* For monophasic waves, there is no peak-to-peak value.

Pulse duration is the total time elapsed from the beginning to the end of a single pulse, including the interphase (intrapulse) interval. If the phase durations of the biphasic pulse are 150 microseconds (μsec) each and the interphase interval is 50 μsec, the total pulse duration is 350 μsec.

The time-dependent characteristics used to describe waveforms reflect the *x* coordinate when plotting waveforms (see Fig. 9-15). *Phase duration* is the time from the beginning of one phase to its end. *Pulse duration* is the time from the beginning to the end of all phases plus the interphase interval within one pulse. The *interphase interval* (or intrapulse interval) is the time between phases of a single pulse, whereas the *interpulse interval* is the time between successive pulses.[1] Phase and pulse duration are most commonly reported in milliseconds (msec) or microseconds (μsec).

Pulse duration is often labeled *pulse width* on many devices. This terminology is not preferred because pulse duration is a unit of time, whereas *width* implies a unit of linear measure.

Modulation of Pulsed Current

Modulation of pulsed current is widely used in electrotherapeutics to impart a variety of different effects. The duration for which a series of pulses or bursts is delivered is termed the *on-time,* and the duration or time between a series of pulses or bursts is the *off-time*. The percentage of the on-time to the total time (on-time plus off-time) multiplied by 100% is termed the *duty cycle*.[1] Duty cycle is a commonly reported parameter of pulsed currents, but some confusion can arise from the use of

this term. Because duty cycle is a relative measure of the "on" to "total" time, a variety of combinations can result in the same duty cycle. For example, a clinical application for muscle strengthening may use an on-time of 10 seconds with an off-time of 40 seconds. The duty cycle of this application would be 10 seconds (on-time) divided by 50 seconds (total-time) multiplied by 100%, or 20%. However, the same duty cycle also accurately describes any on-time and off-time ratio of 1 on to 4 off. Thus, applications of 1 second on and 4 seconds off, 15 seconds on and 60 seconds off yield the same duty cycle of 20%. The significance here is that the use of duty cycle does not always accurately reflect the specific on- and off-times. This can lead to errors when using ES in follow-up visits. This can be avoided if actual on- and off-times are individually documented in absolute seconds instead of expressed as duty cycle.

Amplitude Modulation

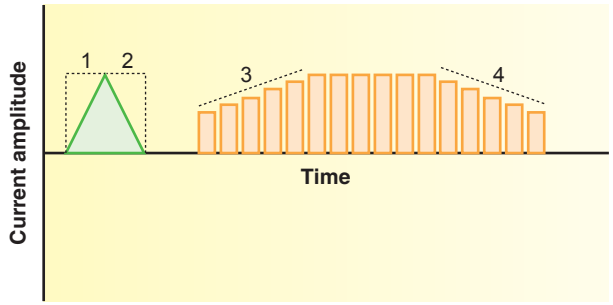
Modulation of the amplitude characteristics of pulsed current is used for differing effects. Often it is necessary to gradually or progressively increase the amplitude of a current to the desired intensity. Take, for example, stimulating muscle. A gradual increase in current amplitude and muscle activation may be more tolerable and ultimately beneficial than a rapid increase and abrupt muscle contraction. For instance, a progressive rise in amplitude, and thus muscle activation, may be used to stimulate the tibialis anterior when assistance with ankle dorsiflexion is desired for toe clearance during the swing phase of gait.

Ramp refers to the progressive increase or decrease in amplitude (Fig. 9-16). When the amplitude is progressively increased, it is termed *ramp-up,* and when amplitude is decreased, it is *ramp-down*. Use of stimulation to facilitate a functional hand grasp–release may incorporate ramp-up and ramp-down to produce a controlled and functional activity.

The terms *rise time* and *fall time* are used to describe the time required for the leading edge of a single phase to reach peak amplitude and the time required for the trailing edge of a single phase to return to the isoelectric line, respectively. Ramps are specific to the increase or decrease in the amplitude of a series of pulses.

It is important to understand how the use of ramp times affects the total current delivered to the patient. Because current is still being delivered to the patient during both the ramp-up and ramp-down times, they should be considered part of the on-time. Long ramp times can greatly reduce the total amount of current delivered to the patient and thus greatly impact the overall therapeutic effect. For example, Figure 9-17 shows two series of pulses, each with an on-time of 10 msec. Series A has a ramp-up time of 1 second and no ramp-down. Series A reaches peak amplitude in 1 second and maintains that amplitude for the duration of the on-time—9 seconds in this example. In contrast, series B has a ramp-up of 4 seconds and a ramp-down of 2 seconds. Thus, series B is at peak amplitude for only 4 of the 10 seconds as opposed to 9 seconds in series A. Because series A is at peak amplitude longer than B, the total amount of current delivered to the patient is greater in series A.

It is important to read the user's manual for electrotherapeutic devices so the clinician understands how ramps are incorporated into the total on- and off-times. For example, some manufacturers include ramp-down time in the off-time. However, this is problematic because current is still being delivered during the ramp-down and should be considered on-time (Fig. 9-18).



1. Rise time (microseconds)
2. Fall time (microseconds)
3. Ramp up (seconds)
4. Ramp down (seconds)

Fig 9 ■ 16 Amplitude modulation of pulsed current: ramps, rise time, and fall time.

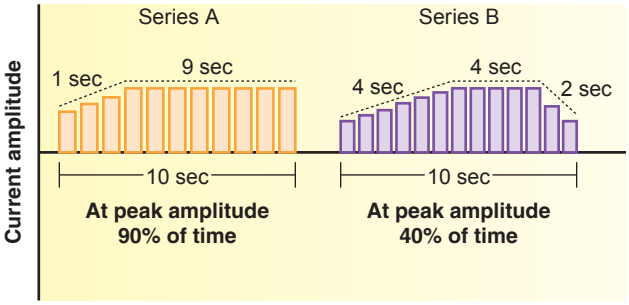


Fig 9 ■ 17 Effect of ramps on total current delivered.

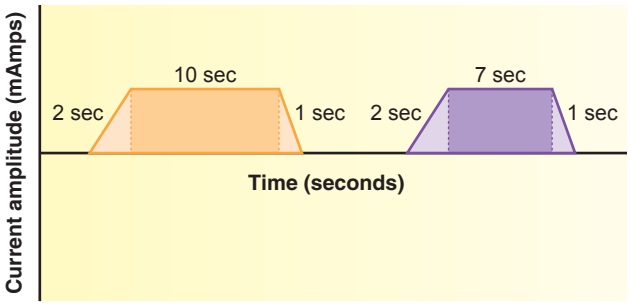


Fig 9 ■ 18 How ramp times are incorporated with the total on-time varies by devices. For example, suppose a 10-second on-time is desired with a 2-second ramp-up and a 1-second ramp-down. The image on the left indicates that the ramps are separate from the 10-second on-time. This equates to 13 total seconds of on-time, 30% longer than initially selected. In contrast, the image on the right shows that the ramps are included as part of the total on-time. This then means the current is at peak amplitude for only 70% of the total on-time.

In this case, the off-time may not accurately reflect a period when there is no current. Other manufacturers extend the total on-time to include the ramp-up. Thus, as an example, a 3-second ramp-up may be added to a selected on-time of 10 seconds, resulting in a total on-time of 13 seconds. If the clinician is not aware of these variations and nuances in the manufacturer specifications, clinical outcomes following treatment may vary greatly.

Phase and Pulse Charge

When the amplitude and time-dependent characteristics are considered together, waveforms can then be viewed in terms of the "area under the curve," or the integrated sum of current amplitude and duration. This represents the total charge delivered in each phase or pulse (Fig. 9-19). *Phase charge* is the charge within one phase of a pulse, and *pulse charge* is the cumulative charge of all phases within a single pulse.[1] Evidence has shown that phase charge is a critical determinant of the magnitude of the physiological effect when using stimulation to activate skeletal muscle.[9] For monophasic pulses, phase charge and pulse charge are synonymous. For biphasic waveforms, determining average current delivered is not as easy and requires further consideration. Note that the area under the curve can be increased by increasing either the pulse amplitude or duration, or both. Ultimately, the magnitude of the electrophysiological response is largely influenced by the total amount of current delivered to the patient—that is, the phase or pulse charge.

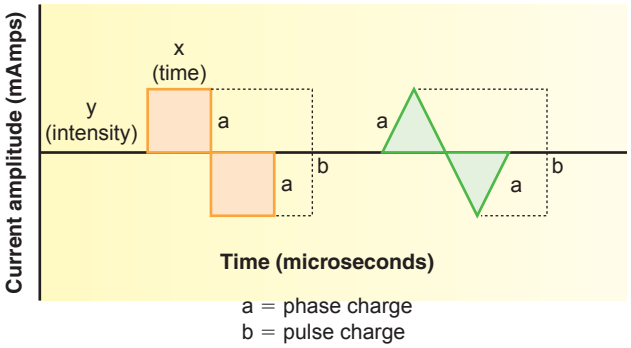


Fig 9 ■ 19 Phase and pulse charge.

Fundamentals in Clinical Practice: Pulse Charge

Using a handheld transcutaneous electrical nerve stimulator (TENS) unit with controls for pulse duration and amplitude, set the pulse duration and frequency as low as possible. Apply the electrodes to the extensor or flexor muscles of your forearm. Then begin by increasing the intensity of the device until you notice small but easily observed muscle twitches. Now leave the intensity alone. Slowly increase the pulse duration. What effect is observed? The increase in the stimulus intensity is the result of increasing the total pulse charge (area under the curve) by increasing pulse duration, not amplitude.

For biphasic waveforms, if the sum of current amplitude and duration of the first phase (i.e., phase charge) are identical to the second, the phases are termed *symmetrical*. On a current-time plot, the phases will look identical in shape. If the amplitude and duration characteristics between the two phases of the biphasic waveform differ in any manner, the phases are termed *asymmetrical* and the graphical depiction on a current-time plot will not be identical—that is, the phases will not be the same shape.[1] If the phases of a biphasic waveform are not symmetrical, then it is necessary to determine if the integrated sums of the phases are equal (i.e., have equal phase charges). Despite not being symmetrical in shape, the area under the curve (the phase charges) can be equal or unequal. If the area under the curve of the first phase is equal to that of the second phase, the phases are termed *balanced*. In this case, the average charge is zero because equal amounts of current flow in both directions of the

biphasic wave. If the phases are not of equal area, they are termed *unbalanced*. In this case, the average charge is not zero and the total pulse charge will have the polarity of the phase with greater charge. Thus, one electrode will maintain a net negative charge and the other a net positive charge reflected by the magnitude of the difference in phase charges.

The significance of symmetry, asymmetry, and balance lies in the potential for delivering a current with a net charge or polarity. This can be thought of again as using area under the curve. If the phases of a biphasic waveform are symmetrical or asymmetrical but balanced, the net charge of the current is zero.[1] This means that there is no sustained polarity, or no sustained positive or negative pole, because equal amounts of current flow in each direction and the direction is constantly changing. If, however, the phase charges of a biphasic waveform are asymmetrical and unbalanced, more current is generated in one direction than the other. This results in a net charge. The pole with the greater area under the curve determines the current's net charge. In this case, there will not be an average of zero charge but rather a net accumulation of charge with a polarity of the phase with greater quantity.

In Figure 9-20, three biphasic currents are shown: one with phase charges of +15 μC and –15 μC, one with phase charges of +15 μC and –6 μC, and a third with phase charges of +6 μC and –15 μC. The first results in a current with zero net charge, as the +15 and –15 balance each other out. However, the asymmetrical phases of the middle example will result in a pulse with a net charge of +9 μC while the third example will result in a net charge of –9 μC. In this case, one electrode will remain more negative than the other while one remains more positive as long as the current flows. When the current has zero net charge, there is no sustained polarity and each electrode alternates equally from being the anode or cathode according to the frequency. With unbalanced waveforms, one electrode will maintain a great negativity and the other positivity; in this case, there is a true anode and cathode, depending on the net charge under each.

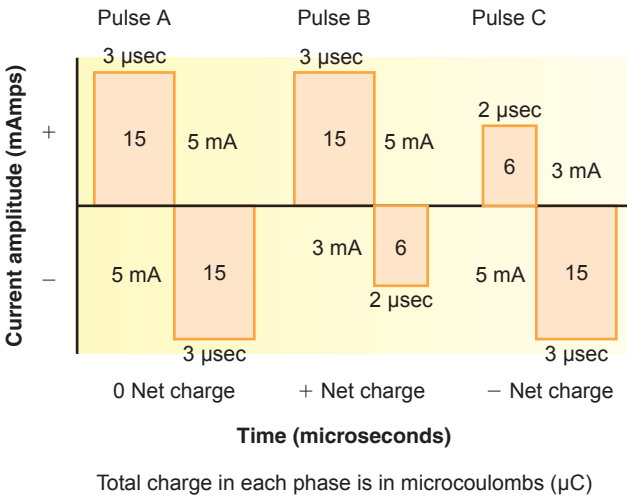


Fig 9 ■ 20 The combination of amplitude and duration creates an "area under the curve" that represents the total charge delivered. Pulse A is symmetrical and balanced with equal amounts of charge in each phase, resulting in zero net charge. Pulses B and C are asymmetrical and unbalanced, resulting in net charge.

There are only three commonly used currents that do not result in zero net charge and thus can result in the accumulation of charge with a specific polarity: DC, monophasic pulsed current, and asymmetrical unbalanced biphasic current. The significance of not having zero net charge is that biological tissues respond differently when anodal and cathodal stimulation are sustained at an electrode. These reactions are presented in the "Electrochemical Effects" section of this chapter.

Most therapeutic uses of monophasic pulsed current and asymmetrical unbalanced waveforms incorporate interphase and interpulse intervals long enough to minimize any adverse polar effects while still allowing for therapeutic benefit. Monophasic pulsed current is commonly used in wound care specifically for the effects observed at each pole (see Chapter 15 for treatment details). In contrast, DC does not have such intervals. Thus, charge continues to accumulate at each electrode until flow of DC ends. Without attention and knowledge of the electrochemical effects of sustained polarity, the polar effect of DC can be quite dramatic and potentially harmful. In fact, reactions ranging from itching to skin burns can occur. However, electrotherapeutic devices that use these three currents typically have lower peak amplitudes, lower frequencies, and long interphase and interpulse intervals, essentially allowing the charge to dissipate without damaging tissue.

Many clinical electrical devices offering symmetrical biphasic or asymmetrical balanced waveforms have lead wires with a black and red or white lead. To those unfamiliar with principles of electrical waveforms, this would seem to indicate a cathode and anode, respectively. However, neither of these waveforms result in a net charge or polarity, so the designation of a cathode and anode is incorrect.

Besides the amplitude and time characteristics used to describe pulsed current, other descriptors aid in describing and differentiating waveforms and, thus, therapeutic effects (Box 9-6 and Fig. 9-21). *Frequency* is the term used to describe the number of pulses occurring in 1 second and is reported as pulses per second. Earlier it was stated that frequency, in hertz (Hz), is used to describe the number of times AC switches direction in 1 second. However, hertz and pulses per second are both used to describe the frequency of pulsed current. A lesser used derivative of frequency (but still relevant to the discussion of electrotherapy) is *period*. Period is the inverse of frequency and is calculated as $1/f$. In other words, the period is the time from one point on a waveform to the identical part on the next pulse. For example, a pulsed current occurring 1,000 times per second (1,000 Hz) has a period of 1/1,000, or 0.001. Thus, a new pulse occurs every 1 msec.

Oftentimes, an electrotherapeutic device or current used to control pain or edema or to enhance muscle performance will be described by its frequency as high, medium, or low. There remains no universally accepted definition of these terms; therefore, their use is not recommended.

Box 9 ■ 6 Describing Pulsed Current: The Bottom Line

When describing pulsed current, three *basic* characteristics need to be specified:

- The waveform type and shape (e.g., symmetrical biphasic square)
- The pulse frequency (e.g., 50 Hz)
- The pulse duration (e.g., 400 μsec)

Figure 9-21 shows a flow diagram of the various waveforms and the key parameters to be considered with each.

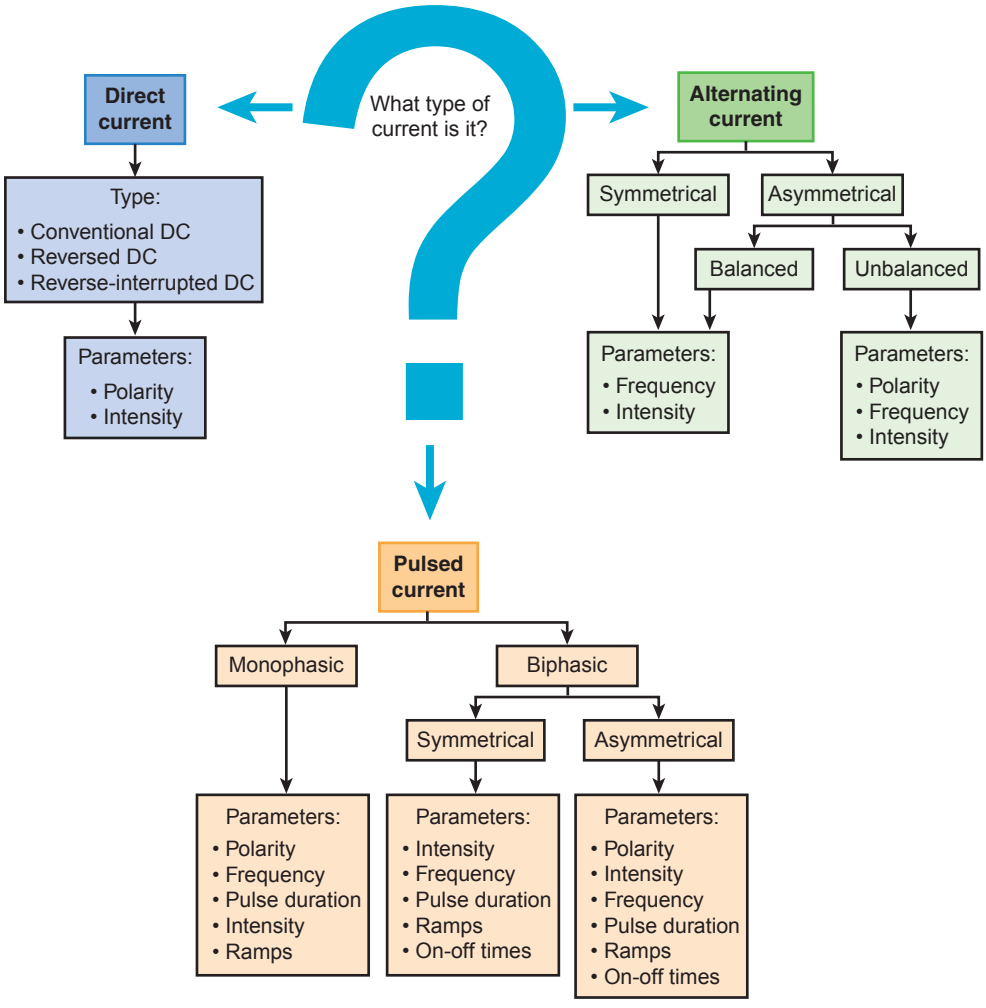


Fig 9 ■ 21 Classification of waveforms and key parameters.

Fundamentals in Clinical Practice: Frequency

Delivering pulses more or less frequently can have a dramatic effect. Using a TENS unit, place the electrodes over the wrist extensor muscles and set the pulse duration and frequency as low as possible. Increase the intensity until a small but visible muscle twitch occurs. Now slowly increase the frequency. What happens? Delivery of more frequent pulses increases the total amount of charge delivered per unit time, and the response of the muscle is greater. The increased muscle response is directly related to the increased frequency of pulses delivered.

PHYSIOLOGICAL RESPONSE TO ELECTRICAL CURRENT

The effects of ES are variable and are based on the specific type of current applied, the parameters of these currents, and the dosage. There are three general electrophysiological effects that occur with the delivery of electrical current: chemical, thermal, and physical.

Electrochemical Effects

The flow or movement of electrical current through the body is due to the movement of ions in the fluid component of bodily tissues. Excluding blood, our body's extracellular fluid environment is largely salt water due to the concentrations of sodium and chloride (sodium chloride; NaCl). In response to electrical stimulation, NaCl is split into Na^+ and Cl^- ions. The Na^+ and Cl^- ions then move according to the principles of charge or electricity. Thus, the positively charged Na^+ will migrate toward the cathode and the negatively charged Cl^- will move toward the anode (Table 9-1).

TABLE 9–1. Localized Effects of Sustained Charge[7]

	Anode (+)	Cathode (–)
Attracts	Cl^-	Na^+
Forms	HCl	NaOH
Process	Sclerotic	Sclerolytic
Effect	Skin hardens	Skin softens*

* Increases risk of electrical burn and tissue damage to DC.

At the cathode, Na^+ joins with water to form sodium hydroxide, an alkaline reaction. This results in a localized area of pH greater than the pH of 7.4 of extracellular fluid. At the anode, Cl^- joins with water in an acidic reaction to form hydrochloric acid (HCl), thereby creating a localized acidic environment with pH less than 7.4. The localized accumulation of Na^+ at the cathode secondary to an applied electrical field causes water to move with Na^+ via ionohydrokinesis.[10] The local increase in water at the cathode results in a decrease in the protein density of the local tissue, thereby softening the tissues and imparting a sclerolytic effect. In contrast, the localized accumulation of Cl^- at the anode results in an acidic effect, a decrease in local water content with subsequent increase in protein density, and a hardening, or sclerotic, effect.[10] The effects of cathodal stimulation are sometimes used to soften restrictive scars through the sclerolytic effects observed under the cathode electrode.

To remember the local effects of charge, it may be helpful to remember the following: If your car breaks down and you don't have AAA, you might have to take a CAB (*a*node *a*ttracts *a*cid and *c*athode *a*ttracts *b*ase).

If the current is DC or monophasic pulsed current, the Na^+ and Cl^- ions will continue to accumulate at the cathode and anode, respectively, as long as the current continues to flow. If the current is alternating or biphasic and changing directions at a specific frequency, then Na^+ and Cl^- moves back and forth between the electrodes. Recall that with AC and biphasic currents, each electrode alternates being the cathode or anode as the direction of current flow changes. Adverse effects of ES resulting from acidic or alkaline reactions at the electrode-skin interface are more common with DC current and, to a lesser extent, monophasic pulsed currents.

Most monophasic pulsed currents (e.g., high-volt pulsed current [HVPC]) have such short pulse durations and long interpulse intervals that any charge accumulated at the electrode-skin interface dissipates before causing lasting adverse polar effects.

Electrothermal Effects

The laws of thermodynamics tell us that energy is neither created nor destroyed but rather exchanged, and during this exchange, some energy will be lost in the form of heat. As charged particles move through a conductor, the friction encountered by the particles results in microvibration

of the conductor's elements.[2] This friction and vibration reflect kinetic energy created by moving parts, and heat is a product of this energy. The amount of heat produced in biological tissues depends on the amount of current flowing, the resistance to the current, and the duration the current flows.

Because the skin resists the transcutaneous delivery of current, accumulation of heat at the skin is a concern. Superficial hair, calloused skin, dried skin caused by alcohol cleansers, and lotions and oils increase skin resistance; shaving hair and removing lotions and oils reduces skin resistance. The redness noted under electrodes following stimulation is the most common result of the thermal or electrochemical effects of current and will usually dissipate within hours.

Electrophysical Effects

The ability to depolarize and propagate electrical signals is what allows nerve and muscle cells to be excitable and to communicate with each other. An understanding of the electrophysical effects of current must begin at the cell. The electrophysical responses underlying the therapeutic effects of electrical stimulation are based on the stimulation, or depolarization, of cells. The specific types of cells stimulated will differ based on several factors, including electrode location, current type and amplitude, and the integrity of the patient's neuromuscular system.

At rest, living cells maintain a separation of charge across the cell membrane such that a net negativity exists in the intracellular environment. This separation of charge represents an electrical potential, or, in other words, a voltage potential difference. The separation of charge is maintained by the selective permeability of the cell membrane to specific ions within the intra- and extracellular environments—chiefly sodium (Na^+) and potassium (K^+).[3] The resting membrane is relatively, but not completely, impermeable to Na^+, which is held in greater concentration outside the cell. In contrast, the resting membrane is significantly more permeable to K^+, which is held in greater concentration inside the cell. However, driven by concentration gradients, small amounts of Na^+ leak into the cell and K^+ leaks out. Adenosine triphosphate–driven Na-K^+ pumps help to maintain the separation of ions by transporting 3 Na^+ out of the cell to every 2 K^+ back into the cell. The resultant effect of 3 Na^+ out to 2 K^+ in is a net negativity on the inside of the cell and the creation of an electrical potential. This potential across the cell membrane is termed the *resting membrane potential* (RMP) and varies for different types of cells based on the relative permeability to Na^+ and K^+. For neuronal cells, the RMP of the membrane (the neurolemma) is roughly –70 millivolt (mV), whereas the RMP of skeletal muscle membrane (the sarcolemma) is closer to –90 mV and that of cardiac myocytes is –85 mV.[3]

The separation of charge across the cell membrane by the Na-K^+ pump results in the cell's polarization. So what is the significance of being polarized? In order to be depolarized, the cell membrane must first be polarized. In the presence of a chemical, thermal, physical, or electrical stimulus in our case, the permeability of the cell membrane to Na^+ is increased, resulting in a reduction in the RMP. The process of depolarization reflects a reduction of the RMP and movement of ions across the cell membrane; the fundamental physiological effect of most applications of electrical stimulation is depolarization of the cell membrane.[3]

When applying this information to the clinical use of ES, it should be noted that electrodes are used to deliver therapeutic currents. As current flows from positive to negative between electrodes, the concentration of negatively charged ions or electrons at the cathode induces the depolarization of the cell membrane, which, when a critical threshold of depolarization is reached, will depolarize the cell; this precipitates the process of depolarization along the cell membrane. Because the RMP of the neurolemma has a threshold for activation at –70 mV, the application of electrical stimulation elicits its electrophysical effects by first depolarizing the nerve. This is a key point when discussing the differences between using ES to activate innervated muscle versus using ES to activate denervated muscle. More details of using ES to stimulate innervated and denervated muscle are presented in Chapter 13.

Clinical Controversy

It is a common misconception that electrical stimulation to contract muscle works by directly stimulating the muscle fibers. This is not accurate, assuming the muscle maintains normal innervations. The nerve, with an RMP of –70 mV, will depolarize before the muscle cell with an RMP of –90 mV. Activation of innervated skeletal muscle occurs by first depolarizing the nerve and then propagating the stimulus along the motor axon, across the neuromuscular junction, and the across the sarcolemma.

Response of Excitable Tissues to Stimulation

For an electrical stimulus to depolarize the cell and elicit an action potential, it must be of sufficient strength and duration. If strength (i.e., current amplitude) and duration (i.e., pulse duration) are insufficient, the cell is not depolarized and the generation and propagation of action potentials is not initiated. The "stimulus" is an electrical pulse or, more commonly, a series of pulses. The relationship between stimuli of sufficient amplitude and duration to successfully depolarize the cell is such that stimuli of increasingly shorter duration require a nonlinearly increasing amplitude. For nerve and muscle cells, there is not a single strength and duration but rather a range of stimulus combinations with varying strength and duration capable of depolarizing the cell and thus generating action potentials. If these combinations were determined and plotted for a given nerve, the graphic depiction would yield a strength-duration curve (S-D curve) (Fig. 9-22). The line of the curve reflects stimuli of minimally sufficient strength or amplitude and duration to stimulate the tissue.

The various combinations of strength and duration in Figure 9-22 reflect the assortment of parameter options that can be used to stimulate the given nerve or muscle. Both stimulus A and B are capable of exciting the cell, but stimulus B, with much lower amplitude, may be more comfortable to the patient. Furthermore, as can be seen from the S-D curve, stimuli of very short duration cannot bring the cell to depolarization despite the amplitude (see stimulus C). Stimuli exceeding the minimal strength and duration capable of depolarizing the cell are termed *suprathreshold stimuli.* In contrast, stimuli with amplitude and duration not capable of depolarizing the cell are termed *subthreshold stimuli.* Note that a pulse duration may be so short that stimulation is not achieved despite increasing the stimulus amplitude (point C, Fig. 9-22).

Earlier in this chapter, the amplitude and duration of a pulse were used to draw a visual representation of the waveform. The area under the curve, termed the *phase charge,* was shown to increase by increasing the amplitude or the duration of the stimulus. The S-D curve is the physical manifestation of this principle, as each point on the S-D curve represents a different combination of stimulus (pulse) strength and duration that can stimulate tissue. Consider Figure 9-22 again: Moving from left to right on the curve, as the intensity of the stimulus is decreased, the tissue may still be excited by increasing the duration. In contrast, as the duration of a pulse is decreased, the amplitude must increase to excite the tissue.

Testing of all the possible combinations of strength and duration capable of exciting a tissue is not feasible because most clinical devices have a limited pulse duration. The S-D curve reflects the graphical plot of each nerve or muscle tested during strength duration testing. From the S-D curve, two objective pieces of information are derived. *Rheobase* is the minimum strength (mA) of a stimulus of very long duration that is capable of eliciting a minimally detectable motor response (Fig. 9-23). After rheobase is determined, it is possible to determine chronaxie—the duration (μsec or msec) of a stimulus two times the rheobase strength capable of eliciting a minimally detectable motor response. Chronaxie can be used to assess the integrity of the tissue because healthy innervated tissue should have a chronaxie less than 1 msec. Prolonged chronaxie, often 10- to 20-fold longer, is indicative of denervation or other pathology involving

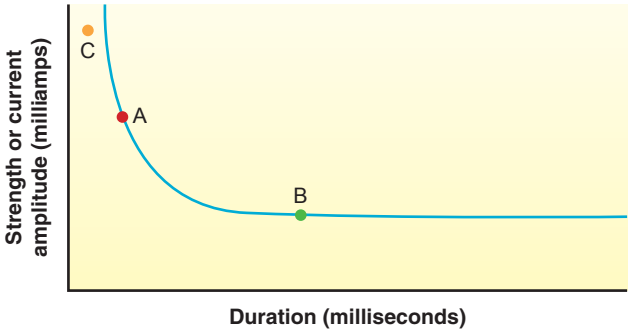


Fig 9 ■ 22 Strength-duration curve. Points A and B represent two combinations of stimulus amplitude and duration capable of eliciting a motor response. The stimulus point C is incapable of eliciting a response, as the pulse duration is too short despite the greater intensity.

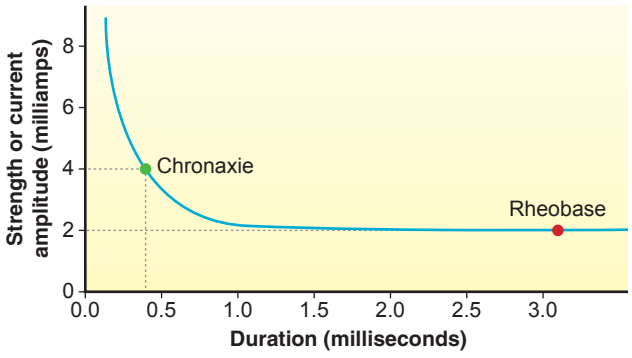


Fig 9 ■ 23 Chronaxie and rheobase.

the excitability of the tissue.[4] Technological advances in neurodiagnostics have rendered S-D testing obsolete; nevertheless, observing a tissue's response to stimuli of varying strength and duration is valuable in understanding tissue responses to electrical stimulation (Box 9-7).

Using a stimulus with an amplitude or duration greater than that minimally capable of eliciting an action potential will not alter or change the action potential. Action potentials are generated in an "all-or-nothing" manner, so a stimulus with an amplitude or duration greater than minimally required will not generate a larger or bigger action potential, and no action potential will be generated if the minimally required amplitude and duration are not reached. When failing to elicit a motor response, a common misconception is that the patient is denervated when in fact the device used probably does not offer a combination of strength and duration capable of eliciting a motor response. Further neurodiagnostic testing may be needed for patients who show abnormal or absent responses to stimulation.

The S-D curve is based on the stimulus parameters capable of eliciting a minimally detectable motor response, but different nerve types have their own combinations of strength and duration needed for excitation. When a nerve is excited, the excitatory response is dependent on the stimulus parameters but also on two other key factors: nerve size and location of the electrodes. Most applications of clinical electrical stimulation involve stimulation of a peripheral nerve. Peripheral nerves are commonly termed *mixed peripheral nerves* to reflect the various nerve subtypes contained within a peripheral nerve. Nerve fibers within a peripheral nerve differ in diameter and in their resistance to excitation.[11] In the presence of a stimulus of sufficient strength and duration, nerve fibers with the greatest diameter and lowest resistance will depolarize first. The largest of the nerve fibers within a mixed peripheral nerve are the A-alpha (A-á) carrying motor and proprioceptive signals; thus, these are first to be depolarized (Table 9-2). To excite the smaller-diameter A-â fibers (touch and pressure sensation) and even smaller-diameter A-ä (pain and temperature) and C fibers (pain), stimuli of progressively greater amplitude and duration are required. In comparison, to directly depolarize the muscle membrane of a denervated muscle, stimuli of significantly greater amplitude and duration are required.

Although larger-diameter nerve fibers are more easily excited, the location of the fibers to the electrodes will affect the order of recruitment. The fibers closest to the electrode will be excited before those fibers farther away (Fig. 9-24). But among those fibers that are stimulated, the largest will still be excited before the smaller. Fibers deeper or farther from the stimulating current can be stimulated by increasing the amplitude of the stimulus. Thus, A-β sensory nerves of the superficial dermis are activated before the larger-diameter A-α, which lie deeper. This largely explains why you feel the stimulus before a motor response is noted.

The strength-duration relationship describes the amplitude and duration characteristics of a single stimulus (i.e., pulse) capable of depolarizing the cell and eliciting a single

Box 9 ■ 7 Using a TENS Unit for a Modified Strength-Duration Curve

If you have a TENS unit with manual controls for pulse duration and intensity, you can perform a modified strength-duration curve. (Most clinical stimulators do not have a pulse duration long enough to complete a full S-D curve.) Place two small electrodes of one channel of the TENS unit on your wrist extensors of one arm. Set the pulse duration as high as possible and set the frequency at 3 to 5 Hz. Then slowly increase the intensity, looking for a small but visible motor response (i.e., twitches). Note the intensity required to elicit this response. Now slowly decrease the pulse duration approximately 20% and note what happens. The motor response will diminish. To return the motor response to the prior level, you must increase the intensity. Repeat this process of decreasing the pulse duration by 20% and noting the increase in intensity required to maintain the motor response as a small but visible motor twitch. If you plot these combinations of pulse duration and intensity, you will have performed a modified S-D test and experienced firsthand the relationship between stimulus strength and duration.

TABLE 9–2. Differentiation of Mixed Peripheral Nerve

	Function	Diameter
Type A		
Alpha α	Proprioception, motor	12–20 μm
Beta β	Touch, pressure	5–12 μm
Delta δ	Pain, temperature	2–5 μm
Type C		
Dorsal root	Pain	0.4–1.2 μm

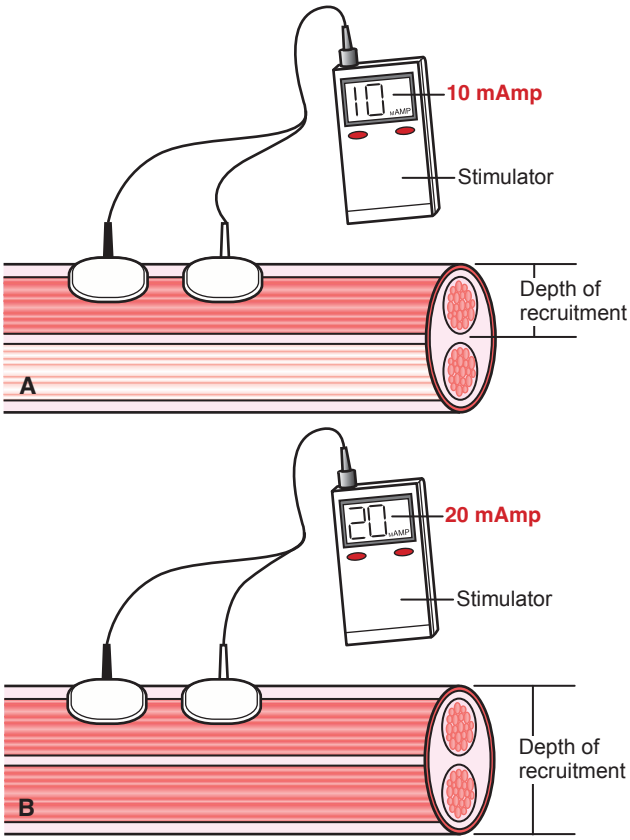


Fig 9 ■ 24 (A) With stimulation to a mixed peripheral nerve, large-diameter fibers will be recruited before smaller-diameter fibers. (B) Increasing the stimulus amplitude will recruit more fibers, as the electrical stimulus is able to reach and depolarize a greater number of nerve fibers.

action potential. However, single action potentials do not lead to purposeful muscle activity. The tension created in a muscle fiber twitch from a single action potential will completely decrease unless followed by a subsequent action potential. If the frequency of action potentials is increased, the subsequent muscle fiber twitches will occur before the previous one has decreased, resulting in increased tension. *Twitch summation* refers to the addition, or summation, of tension in individual muscle fibers, thereby resulting in greater whole muscle tension. This occurs because the duration of a fiber twitch may be less than 100 to a few 100 msec, whereas an action potential can be generated every 1 to 5 msec, allowing action potentials to come before the muscle fibers have completely relaxed. Longer duration and greater muscle tension occurs with increasing frequency of stimulation, so even though a single action potential in a muscle fiber generates twitch tension, greater tension can be realized by increasing the frequency of stimulation.[3] The relationship between stimulation frequency and muscle force is called the *force-frequency relationship.* When the muscle fibers are stimulated so frequently that the tension generated in the fibers does not have time to decrease between action potentials, the tension is sustained; this is termed *tetany*.

Levels of Response to Electrical Stimulation

There are three levels of response to therapeutic electrical stimulation: sensory, motor, and noxious (although, most of the time, we are not trying to produce noxious stimulation). With stimuli of relatively low amplitudes that can still excite the nerve, the first perception of an electrical stimulus is often described as pins and needles or tapping secondary to excitation of A-β fibers in the superficial dermis. If the amplitude and duration of the stimulus is increased, excitation of the A-α fibers (alpha motor neurons) will occur and a motor response will be elicited. The magnitude of the motor response is proportional to the intensity of the stimulus (up to a point); thus, the initial motor response may appear as fasciculations or small twitches in the muscle. With increasing amplitude, the motor response will increase to a more full and robust contraction. Further description of the effect of muscle fiber recruitment and therapeutic effects of ES appears in Chapter 13.

With further increases in amplitude or duration beyond that capable of eliciting sensory and motor responses, the A-δ and C fibers are excited, eliciting the perception of pain. This is the third level of response to electrical stimulation. Eliciting a noxious-level response is sometimes used for therapeutic purposes, as will be described in Chapter 11.

Fundamentals in Clinical Practice: Electrode Placement

When you attempt to elicit a muscle contraction, your patient reports the onset of strong tingling and then pain, but you do not see a motor response. What could be the cause of this? If the electrodes are not placed over areas of A-α nerves (i.e., motor tissue), the stimulus is incapable of eliciting a motor response because there is little to no motor tissue in the area stimulated. Relocation of the electrodes to regions where the alpha motor neurons exist or to areas of more depolarizable muscle tissue will yield the desired motor response.

The first time most students and patients use therapeutic electrical stimulation, it is common to see a low tolerance level to stimulation. Upon subsequent applications, tolerance usually improves, allowing use of stimulus parameters suitable for the clinical goals to be achieved. If a patient is unable to tolerate the stimulus parameters required to elicit the necessary therapeutic effect, the efficacy of this treatment is greatly compromised. For example, when using stimulation to improve the strength of skeletal muscle, the patient must be able to tolerate an intensity that is sufficient to activate the muscle at a level consistent with eliciting adaptation. If the patient is unable to tolerate the stimulus needed to elicit an appropriate motor response, an alternative approach to facilitate muscle activation should be selected. The first attempt at using ES can be considered a familiarization session, allowing the patient an opportunity to experience the stimulus while you fully explain the anticipated sensations and effects. In addition, to completely educate your patient regarding their experience, you must undergo therapeutic ES yourself.

THERAPEUTIC CURRENTS BY NAME: VARIATIONS OF THE BASIC CURRENTS

When you choose to include ES in the intervention plan, you must then determine the stimulus characteristics, including the type of waveform, to elicit the desired response, whether that is motor activation (e.g., muscle contraction), sensory stimulation (e.g., a "buzzing sensation" with no muscle contraction), or something else. Most units in clinical settings today are multiwaveform devices offering more than one type of current waveform and thus more than one type of potential response or effect. Even though all waveforms are derived from DC, AC, or pulsed current, variations in the specific parameters of these three currents are what differentiate the many therapeutic currents used today. By modulating previously described basic parameters of current, a myriad of waveforms and subsequent electrobiophysical effects are possible. Therefore, it is critical to understand currents and waveforms in order to maximize the potential of electrotherapeutics.

Much confusion is created by the various and inconsistent use of waveform names, in part due to the industry's enthusiasm in marketing equipment and trying to claim the "leading edge" with a new current or feature. When discussing electrotherapeutics, inconsistency in terminology leads to confusion and, more often, miscommunication and misunderstanding. Therefore, it is recommended that waveforms be described by the specific parameters that constitute the waveform rather than terms or names perpetuated by clinical lingo. Often times, these clinical names are fleeting, coming and going like fashion trends. To help the clinician decide which waveform to use and how to differentiate one from another, the more popular or common waveforms will be described here using their popularized names with some description as to their clinical use. Greater explanation of their clinical use and evidence for use will be presented in later chapters addressing specific therapeutic applications.

Russian Current

Perhaps the most widely recognized waveform in clinical use today is Russian current, named after Dr. Yakov Kots, a Russian exercise physiologist credited for popularizing this waveform in the 1970s. Touting strength gains up to 40% in elite Russian Olympic athletes, Kots's claims were significant because they represented gains in healthy individuals, something previously unrealized. Significant gains in muscular strength and power beyond that accompanying training were attributed to use of the Russian current, thus increasing its popularity.[12] Russian current is simply a variation of alternating current. Conventional Russian current as described by Kots is a 2,500 Hz alternating sinusoidal current that is interrupted and delivered in short bursts. This is termed *burst modulation* and is a defining characteristic of Russian current. The bursts are delivered at 50 bursts per second with a burst duration of 10 msec and an interburst interval of 10 msec (Fig. 9-25). The frequency of the sine wave that is interrupted into bursts is called the *carrier frequency* (usually 2,500 Hz). Within each 10-msec burst, there are 25 complete cycles of AC. This results in a waveform with a therapeutic, or treatment, frequency of 50 Hz and 400 μsec cycle duration (i.e., 1/2,500 cycles per second), which is well suited for activating skeletal muscle.

The original Russian 10/50/10 protocol calls for 10-second contraction time and 50-second off-time for 10 repetitions.[12] Because the burst duration is equal to the interburst interval, Russian current is

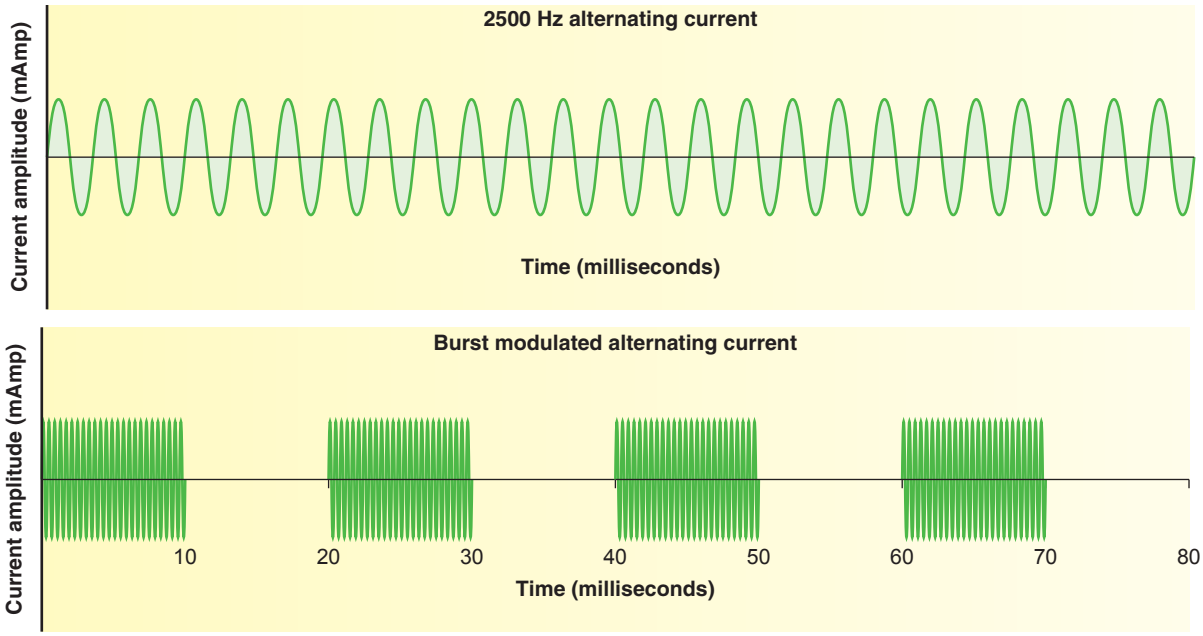


Fig 9 ■ 25 Russian current: 2,500 Hz AC is burst modulated at 50 bps using 10-msec bursts.

often described as having a 50% duty cycle. This duty cycle is different from the one defined earlier, which was on-time divided by total-time × 100%. The 50% duty cycle of Russian current is a "relative" duty cycle, sometimes referred to as *Russian duty cycle,* while the actual duty cycle of a 10-second on/50-second off protocol would be 16.7% (10/60 × 100%). The 50% relative duty cycle and 400-μsec pulse duration are two of the proposed benefits of burst-modulated AC for muscle strengthening. Simply stated, when Russian current is "on," it is on for a long time. However, the evidence for burst-modulated AC (i.e., Russian current) versus other waveforms for muscle strengthening remains inconclusive.[12–15] In 2009, Ward[16] reported that the evidence for Russian current ranges from a single case study that showed increased strength[17] to evidence of no increase in strength.[18] Recent studies have examined whether Russian current is the optimal current for increasing strength. Two studies found that a 1,000-Hz carrier frequency elicited greater torque than 2,500 Hz. Data also indicated that burst durations of 2 to 5 msec may be better suited for muscle stimulation in comparison to the 10-msec burst duration of conventional Russian current.[19,20] Most recently, Bellew et al (2012 and 2013)[21,22] supported the findings of Ward, suggesting lesser carrier frequency and duty cycle elicited significantly greater muscle force than Russian current and with forces approaching 100% voluntary efforts—a level not previously obtained with stimulation.[22]

A variation on Russian current recently made available in the United States is "Aussie" current—a 1,000-Hz burst-modulated AC current delivered in 4-msec bursts. Greater torque production and decreased rate of muscle fatigue have been reported with the Aussie current compared to the conventional 2,500-Hz, 10-msec burst waveform of Russian current.[8,23]

Fundamentals in Clinical Practice: Using Best Evidence in the Clinic

A patient with decreased volitional activation and 4– strength of the right quadriceps is referred for strengthening following right total knee arthroplasty. Using best evidence, the clinician chooses Aussie[19,20] or burst modulated biphasic pulsed current[21,22] with carrier frequency close to 1,000-Hz and burst duration less than 10-msec. The quadriceps are stimulated for 10-second contractions with 50-second rest periods between the 10 training contractions.

High-Volt Pulsed Current

High-voltage currents gained popularity in the 1970s with interest in using currents of high amplitude. High-volt pulsed current, also known as *high volt* or *high-volt*

pulsed galvanic, is a twin-peaked monophasic pulsed current waveform with peak voltage typically reaching 150 to 500 V, a short pulse duration typically lasting 50 to 100 μsec, and a frequency of 1 to 120 Hz[24] (Fig. 9-26). However, the term *galvanic,* traditionally given to interrupted DC, is no longer preferred because it does not accurately reflect the waveform characteristics of HVPC.

Because use of high-voltage pulses necessitates very short pulse durations in order to avoid tissue damage, HVPC uses a twin-peak pulse with an almost instantaneous rise time and immediate exponential fall-time—thus the name "twin-peak" monophasic.[24] The short pulse duration, typically up to 100 μsec, and long interpulse interval result in a low relative duty cycle, often 1% or less.[25–27] This long interpulse interval permits use of high-peak voltages yet results in a much lower average current over the entire duty cycle.[24] The product is a high-peak voltage but overall low average treatment voltage.

To understand the high-intensity voltage and short pulse duration of HVPC, think about touching a very hot stove. The high heat of the stove can be very dangerous, but if you touch it very quickly and infrequently, there is little to no risk of burn. However, if you touch the stove for a longer time and more frequently, you may burn yourself.

HVPC is a monophasic waveform, which means one electrode will accumulate negative charge (cathode) and the other will accumulate positive charge (anode). One electrode is placed over the treatment area; this is the active, or treatment, electrode. The other electrode is placed at some site away from the treatment site; this is the reference, or dispersive, electrode. Most devices on the market offering HVPC will allow you to select the polarity of the active, or treatment, electrode as either positive or negative. The dispersive, or reference, electrode consequently becomes the other polarity. Some modern and many older versions of HVPC devices use a dispersive electrode that is much larger in relation to the active electrodes (Fig. 9-27). This and the ability to choose a polarity of the current are classic telltale signs that the device generates a monophasic pulsed current and is likely an HVPC unit, but the best way to tell is by reading the machine's user manual. Clinical uses and what specific effects are attributed to the anode or cathode of a monophasic waveform are presented in Chapter 15.

HVPC is typically administered using one of three modes: continuous, reciprocating, or surge. *Continuous mode* refers to the continuous and uninterrupted flow of pulsatile current for the entire treatment session. This is not to be confused with direct current, as HVPC is the flow of monophasic pulsed current, not DC. Continuous mode does not have on- and off-times, as would be used in activating skeletal muscle, but rather remains on until the current is turned off. With continuous mode, the amplitude of each pulse remains constant throughout the duration of the session.

Reciprocating mode delivers current first to one of the active electrodes and then to the other active electrode (assuming more than two active electrodes) in an alternating or reciprocating manner. This mode does not alter the flow of current to the dispersive pad but simply reciprocates the flow between the two active electrodes. If only one active electrode is used, reciprocating mode is not chosen. Clinicians select the time that current flows at each electrode before switching. With reciprocating

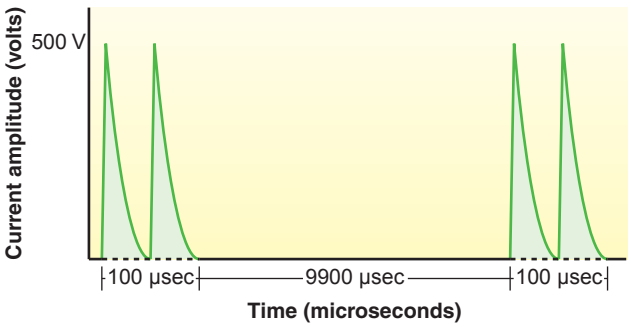


Fig 9 ■26 High-volt pulsed current is characterized by a high-peak intensity and short pulse duration (shown at a frequency of 100 Hz).

Fig 9 ■27 An identifying characteristic of many high-volt pulsed current devices is a large dispersive electrode.

mode, the amplitude of each pulse remains constant throughout the duration of the session unless the clinician adjusts it; this is called *balancing*. Balancing allows the peak intensity of the current at one or both active electrodes to be decreased by a selected percentage. For example, the current in one active electrode may be dampened by 25% to accommodate a sensitive placement site.

Finally, *surge mode* modulates the amplitude of the pulse train so that the amplitude of each successive pulse progressively increases to a peak over the duration of the on-time. This is similar to the ramp-up function of other pulsatile currents.

The specific waveform characteristics of HVPC (amplitude, frequency, and pulse duration) make it possible to stimulate both sensory and motor nerves, so HVPC is used for a variety of clinical purposes. Pain modulation, activation of skeletal muscle, and tissue healing are the most common uses. Because HVPC has a true anode and cathode, it has become a widely used and evidenced waveform for tissue repair and wound healing.[28,29] Tissue repair and wound healing are undoubtedly the areas of use that have the greatest degree of support for HVPC. (More information on use of electrical stimulation for tissue healing appears in Chapter 15.) Modifying the pulse duration and frequency of HVPC can yield a waveform similar to conventional TENS for electroanalgesia, making HVPC useful in managing pain.

Because HVPC can stimulate skeletal muscle, it has also been used for muscle reeducation. Keeping in mind the strength-duration relationship of a stimulus, the very short pulse duration of HVPC necessarily requires a high-intensity stimulus, and this is often perceived as painful by the patient. In contrast to the 400-μsec pulse duration and 50% relative duty cycle of Russian current, the 100-μsec duration and 1% relative duty cycle of HVPC is considerably less. To date, there is no sufficient evidence to support using HVPC to strengthen muscle despite its ability to stimulate skeletal muscle.

The bottom line for using HVPC to strengthen muscle is this: Simply because HVPC can elicit a motor response does not make it suitable—or, more importantly, effective—for that purpose. Other waveforms more suitable to muscle activation and strengthening are available and are addressed in Chapter 13.

Occasionally, therapeutic currents will be referred to as *high volt* or *low volt*—terms that refer to the magnitude of the voltage used to drive the current. Conventional use of these terms has led to the designation that *high volt* refers to a current with a voltage in excess of 150 V and typically up to 500 V. In contrast, the term *low volt* is given to a current with a voltage less than 150 V.

Interferential Current

In the early to mid-2000s, interferential current (IFC) was reported to be the most popular and commonly used form of electrotherapy in Europe and Australia.[25] Although no such data is available for use in the United States, IFC remains a popular waveform. IFC is derived from the interference or superimposition of two symmetrical but asynchronous, kilohertz frequency, alternating currents resulting in a single treatment, or interference, current with properties uniquely different than the two original currents.[31] Traditionally, sinusoidal alternating current has been used for IFC, but some devices now use square, rectangular, or even triangular alternating current. The premise of IFC is that when two asynchronous kilohertz (KHz) frequency (1 to 10 KHz) currents are directed to intersect or interfere, the waves are periodically in synch or in phase with each other, and the amplitudes of the two currents will sum together[32,33] (Fig. 9-28). This is termed *constructive interference*. Equally periodic, the currents will be out of phase, resulting in destructive interference, and the amplitudes will negate each other.[31] As the two currents go in and out of synch, the amplitude of the interference current gradually increases and decreases. Because of the modulation of amplitude, IFC is referred to as *amplitude modulated AC.*

The currents are maximally in or out of phase at a rate equal to the difference between frequencies of the currents interfered. For example, interference of a 4,000-Hz and a 4,100-Hz current, the lesser of which is termed the *carrier frequency,* results in an interference current of 100 Hz. This frequency is termed the *beat frequency* and typically ranges from 1 to 200 Hz. The amplitude of the IFC will peak and fall at a frequency equal to the beat frequency. The beat frequency reflects the therapeutic frequency—that is, the frequency that elicits the therapeutic effect. Most devices on the market today allow the clinician to select a specific beat frequency. A specific beat frequency can be obtained from several possible interference currents as long as the difference between the currents is the same. The physiological

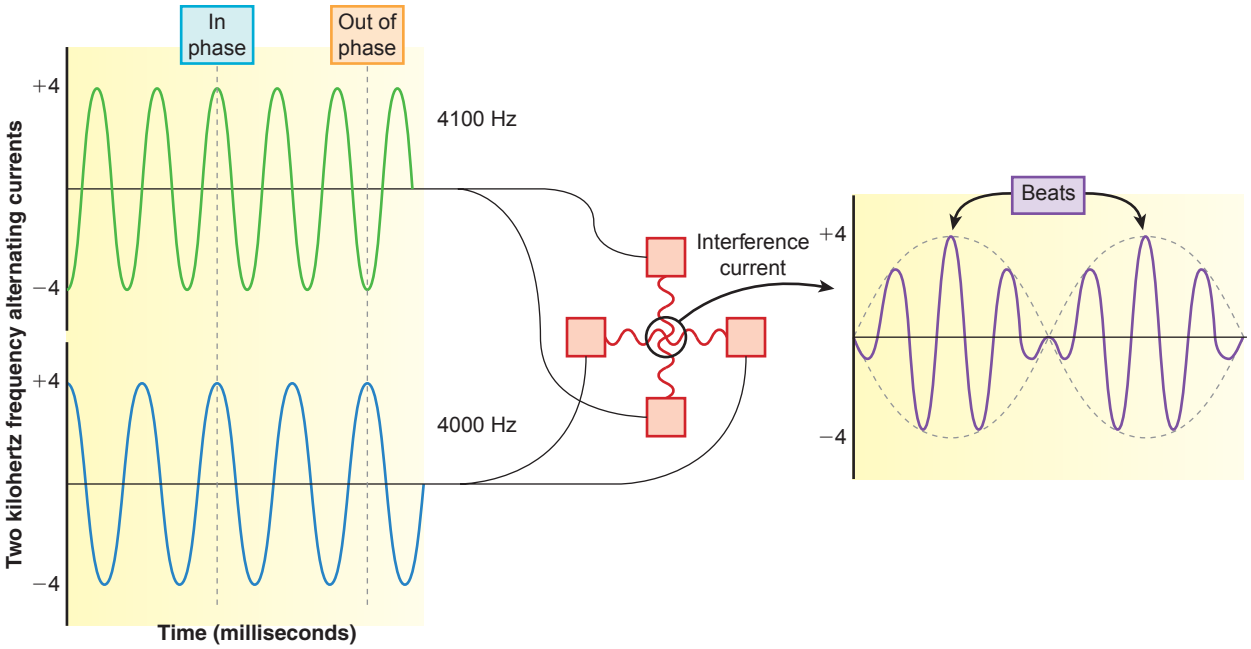


Fig 9 ■ 28 Interferential current is the interference of two asynchronous kilohertz frequency currents to form a current with amplitude modulation.

effects elicited by IFC are based on the beat frequency, not the frequencies of the input currents or current shape (i.e., sinusoidal or other). For example, to elicit a tetanic muscle contraction, a beat frequency consistent with a tetanic response is used (e.g., 50 Hz). On most contemporary devices, the clinician selects the beat frequency while the carrier frequency is prefixed at 4,000 Hz. Still other devices permit the clinician to adjust the carrier frequency within 1,000 to 5,000 Hz. The effect of a lesser carrier frequency is a longer pulse duration; however, there remains no evidence to support the selection of one carrier frequency over another.

A 100-Hz beat frequency is obtained from each of these input currents: 4,000 and 4,100 Hz; 2,500 and 2,600 Hz; 4,900 and 5,000 Hz; or any combination differing by 100 Hz.

Some IFC devices allow the frequencies of the input currents to be modulated, resulting in a modulated beat frequency (Fig. 9-29). This effect is termed *sweep* but is not available on all devices. Sweep is either a linear or peak-to-peak sweep. With linear sweep, the beat frequency is modulated continuously from a maximum to a minimum frequency, whereas a peak-to-peak sweep results in the alteration of the beat frequency between only the maximum and minimum[34] (Table 9-3). *Swing* is used to denote the temporal characteristics of the sweep pattern with "^" used to denote a linear or continuous sweep and

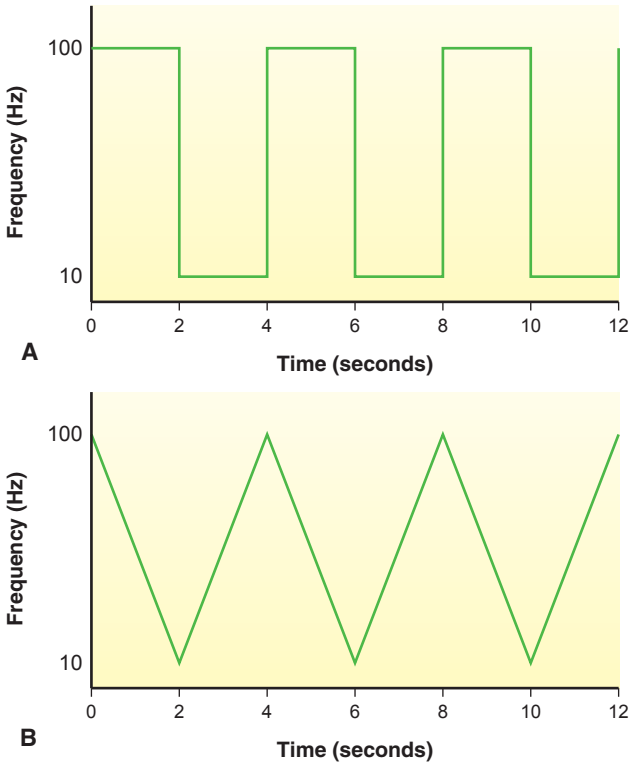


Fig 9 ■ 29 Sweep is modulation of the beat frequency of IFC and is either peak-to-peak or continuous. Swing represents the temporal characteristics of the sweep or the timing of the sweep. (A) A peak-to-peak sweep, denoted by "∫," alternates the beat frequency between only the lowest and highest beat frequency selected. (B) A continuous sweep, denoted by "^," results in a beat frequency that progressively increases and decreases between the lowest to highest beat frequency selected.

TABLE 9–3. Language of Interferential Current

Parameter	Definition	Effect	Use
Frequency			
Carrier frequency	Lesser frequency of the two currents interfered		
Beat frequency	Frequency the interfered currents are maximally in and out of synch	Resultant frequency from interference of two KHz currents	The beat frequency determines the physiological effect
Sweep	Modulation of the beat frequency	Varies the beat frequency of the interference wave	To provide low- and high-frequency stimulation within the same treatment
Swing			
"∫"	Temporal characteristics of sweep	Alters the beat frequency from minimum to maximum	To use only the minimum and maximum beat frequencies
"^"		Linear and continuous sweep between the minimum and maximum beat frequency	To use a continuously changing beat frequency from minimum to maximum
Vector scan	Amplitude modulation of the input currents	Increases the area of the interference pattern	To provide stimulation to a larger tissue area

"∫" used to denote peak-to-peak sweep;[34] for example, a 2^2 sweep from 100 to 10 Hz will decrease continuously from 100 Hz to 10 Hz over 2 seconds and then increase continuously and linearly over the next 2 seconds. In contrast, a 2∫2 sweep from 100 to 10 Hz denotes that the beat frequency remained at 100 Hz for 2 seconds before switching to 10 Hz for 2 seconds. The proposed benefits of sweep are a modulated rather than fixed beat frequency, thus avoiding accommodation, and the ability to use both high- and low-frequency stimulation within the same treatment, although these have not been substantiated.

The increase and decrease in amplitude created by asynchronous currents going in and out of phase is similar to the effect of jumping on a trampoline with another person. When two jumpers are in synch, the height of the jump is increased, but when out of synch, the jumpers can cancel out each other's bounce.

IFC is administered using four electrodes positioned so that the two currents intersect each other perpendicularly within the tissues of the patient. This is known as *quadripolar IFC* (Fig. 9-30). The point of intersection and site of the new interference current is assumed to be in the geometric center of the electrodes. However, this cannot be accurately determined because of nonhomogenous tissue resistances. Current will take the path of least resistance, so the path of one or both currents will probably be altered, resulting in interference at some point other than the one assumed.

The perpendicular interference of two currents results in a pattern that looks like a clover-leaf electrical field (Fig. 9-31). To increase the area of the interference and stimulate greater tissue area, modulation to the interfered currents can be used. *Vector scan,* offered on some devices, is the modulation of the amplitude of one or both of the input currents, resulting in a rhythmic change in position of the interference pattern.[31] This is depicted as an oscillating clover-leaf shape, much like

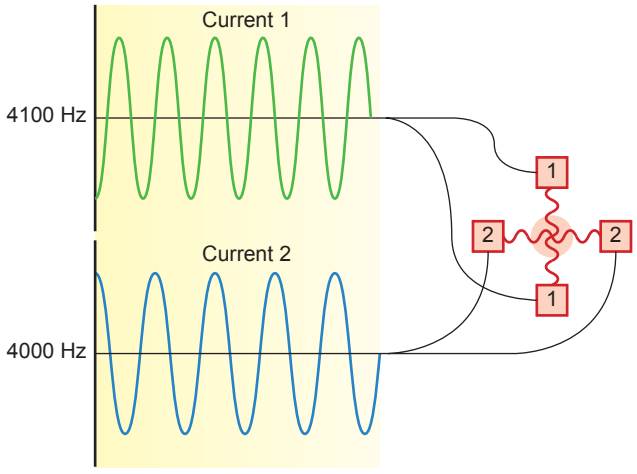


Fig 9 ■ 30 Quadripolar interferential current.

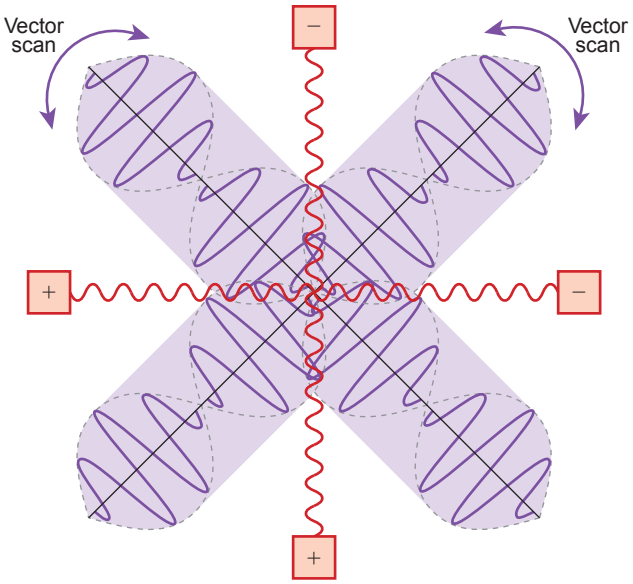


Fig 9 ■ 31 With the perpendicular interference of currents, a cloverleaf-shaped field of current is created. Vector scan is amplitude modulation resulting in expansion of the cloverleaf field.

the motion of the agitator of a washing machine rotating back and forth. Vector scan is thought to increase the effective treatment area of the stimulation.

A common variation of IFC available on many stimulators is *premodulated IFC*. Premodulated IFC is created by the interference of two alternating currents within the stimulator device, not by interference within the tissues, as with quadripolar IFC. Thus, only a single current is applied to the patient. Premodulated IFC is delivered using two electrodes instead of four, and is thus sometimes called *bipolar IFC*.

There are three proposed and theoretical advantages of IFC compared to other waveforms: (1) alternating sinusoidal currents with frequencies exceeding 1 KHz are hypothesized to more easily penetrate the skin due to reduced skin impedance; (2) the amplitudes of the two currents summate in the deeper tissues, thereby passing the more superficial sensory afferent nerves; and (3) a beat frequency of the interference current can be selected that is similar to other waveforms used for muscle stimulation and pain modulation.[7,30–32]

More recently, however, the proposed benefits of IFC have been questioned.[16] The suggested decrease in skin impedance noted with KHz frequency current is not specific to the frequency but rather to the short pulse duration necessarily associated with KHz frequencies. Skin impedance is directly related to pulse duration, not frequency, so that at very short pulse durations, impedance is reduced. If a pulsed current has a similar pulse duration as the KHz frequency current, the impedance across the skin is similarly low.[16] Furthermore, the interference pattern that characterizes IFC is not simply in the region geometrically predicted by electrode applications; rather, it is much more diffuse based on the nonhomogeneity of resistance in various tissues in the path of the current (Box 9-8).

Although IFC is most commonly used for electropain modulation, its similarities to conventional Russian current have led to the study of its effectiveness in eliciting muscle contraction. A seminal study from 1989[14] reported that IFC was inferior to conventional Russian current for eliciting muscle torque. However, the phase durations of the two currents were not equal. More recently, Bellew et al[21,22] compared IFC and conventional Russian current but with equal phase durations and reported significantly greater force production with IFC.

Fundamentals in Clinical Practice: Use of Interferential Current for Pain Modulation

A patient is referred for acute low back pain secondary to a lifting injury. Pain is local to the lumbar paraspinals, and active contraction of the muscles increases the pain. The clinician chooses interferential current to provide electroanalgesia. A treatment, or beat, frequency of 100 Hz is desired, so the clinician selects a carrier frequency of 4,000 Hz from one channel and 4,100 Hz for the second channel. The four electrodes of the two currents are applied paraspinally, such that the currents are directed to intersect perpendicularly. The stimulus intensity is increased until the patient reports a sensory-level sensation.

Low-Intensity Direct Current (Microcurrent)

Technically speaking, microcurrent is any current with an amplitude less than 1 mA (10^{-3}A).[1] The microcurrent waveforms offered on electrotherapy devices are either DC or monophasic pulsed current. Although *microcurrent* is the name by which this waveform is offered on many multiwaveform devices, it has also been called *low-volt pulsed current, microelectrical neuromuscular stimulator,* or *microelectrical stimulation*. A current of microamperage amplitude is insufficient to excite sensory or motor nerves. Thus, names implying stimulation of nerve or muscle are deceiving. Any notion of therapeutic benefit

Box 9 ■ 8 Premodulated Current Versus Interferential Current

Premodulated current is often confused with IFC. However, significant differences exist.[13] With premodulated current, the two currents are interfered within the device before delivery to the patient (see Fig. 9-32). With premodulated IFC, the currents do not interfere within the patient tissues nor is the amplitude summed within the patient. Premodulated current is often referred to as *exogenous* or *bipolar IFC* because it is created outside the patient and because two electrodes versus four are used with quadripolar IFC.

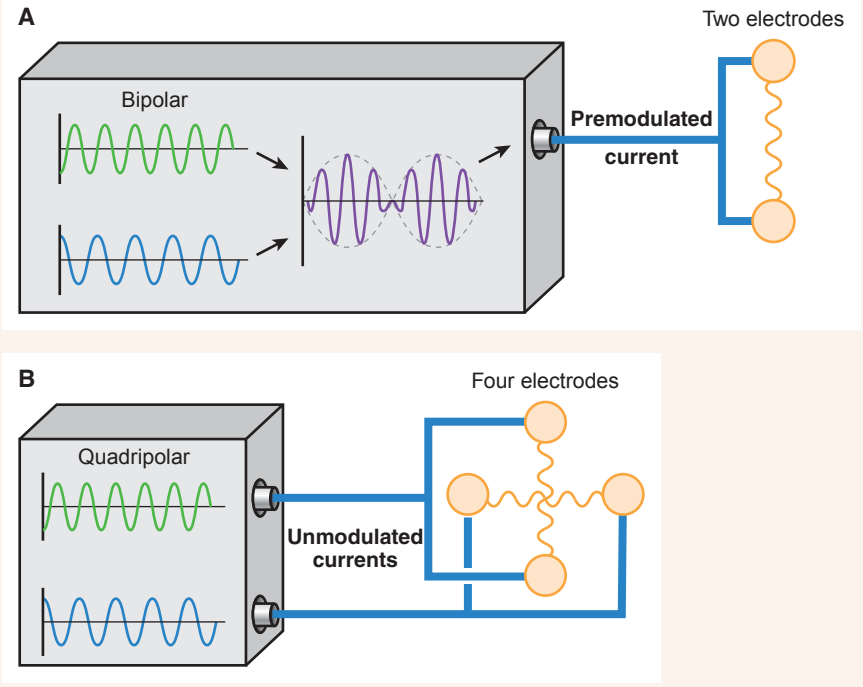


Fig 9 ■ 32 (A) Premodulated (bipolar) IFC is created in the stimulator, resulting in a single current delivered by two electrodes. (B) Quadripolar IFC is created by two currents crossing in the tissues and is delivered by four electrodes.

derived from activation of sensory or motor nerves is physiologically implausible.[34] The APTA recommends using the term *low-intensity direct current* (LIDC) for this microamperage current.[1]

LIDC is typically DC or monophasic pulsed current with a peak current amplitude in the microamperage range. If the waveform is monophasic pulsed current, the pulse duration is dependent on the frequency. A typical pulse duration is 500 msec (0.5 sec)—much longer than most other pulsed currents. Frequency on most LIDC stimulators varies from 1 to 1,000 Hz. Because LIDC is either DC or monophasic, one electrode will remain the anode and the other the cathode. Unique behaviors or characteristics demonstrated by particular cell populations in response to anodal or cathodal stimulation have been reported.[35,36] A more specific and modern discussion of cell and tissue responses to electrical stimulation appears in Chapter 15.

LIDC originated after observations of microamperage DC flowing out of injured tissue.[29,36,37] The outward flow of current indicated the presence of an electrical potential across the skin—termed the *transepithelial potential* (TEP). Different from the resting membrane potential of single cells, the TEP is created by the separation of ions across sheets of epithelial cells (i.e., skin) (Fig. 9-33). This separation of ions across the skin leaves the external skin surface with a net negative charge, known as the *skin battery.*[29,37] When the skin is injured, a pathway is created that allows flow of positively charged ions from the deeper tissues to the skin surface.[37] As positive charges leave the injured tissue, the wound loses it positivity and becomes negative relative

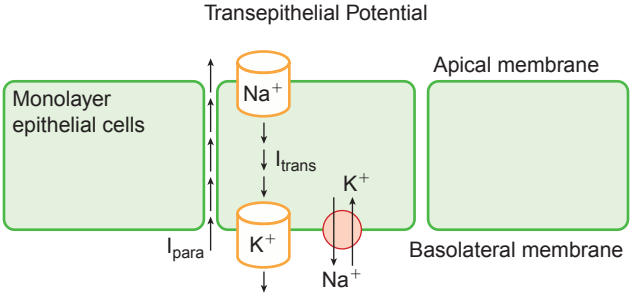


Fig 9 ■ 33 The transepithelial potential underlies the "current of injury."

to the flow of positive ions to the surface. Because of this, the wound becomes the cathode of the current. In tissues lateral to the site of injury where the TEP is maintained, the concentration of positive ions now flows toward the wound cathode, creating what is termed *lateral currents of injury*. The outward flow of current and the lateral currents have been termed the *currents of injury.* Clinical use of LIDC is based on these endogenously produced currents and is intended to augment the current of injury.

At low pulse durations and frequency, HVPC can result in current with microamperage intensity, despite its name. If the frequency or pulse duration of HVPC is increased, the total current amplitude delivered is likely to exceed microamperage levels, resulting in milliamperage intensity.[25]

Fundamentals in Clinical Practice: Clinical Use of Microcurrent and Best Evidence

A patient is referred for treatment of a skin wound that has failed to close following an open reduction and internal fixation of the left distal radius 4 weeks ago. The clinician wants to facilitate tissue healing and chooses to use LIDC to stimulate epithelialization of the tissues. A device offering LIDC is chosen, and using best current evidence, the cathode of the current is placed over the wound site with the anode placed adjacent to the wound.

Symmetrical and Asymmetrical Biphasic Pulsed Currents

Symmetrical and asymmetrical biphasic pulsed currents represent a group of waveforms widely used for muscle stimulation and pain modulation. The pulses are commonly square, rectangular, or triangular and vary in duration and amplitude based on the physiological response desired (Table 9-4). Asymmetrical pulsed currents are often included along with symmetrical biphasic pulsed currents on many clinical and handheld stimulators, yet there is little data to support or refute use of asymmetrical pulsed current over symmetrical for purposes of activating muscle or modulating pain.

Symmetrical or asymmetrical biphasic pulsed currents are not specifically given to any particular physiological or treatment effect. These are both simply waveform shapes. The specific uses and effects that can be derived from their application is more a matter of what or how the clinician can control or select the waveform parameters. For example, a stimulator offering these currents may not offer or allow the clinician to select the parameters specific to muscle activation or strengthening such as on- and off-time or ramp-up or ramp-down. In this case, the symmetrical or asymmetrical biphasic waveform may not be of use for muscle activation but may be better suited to pain modulation.

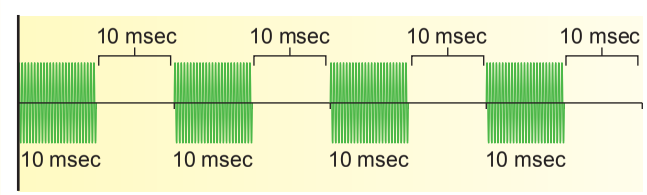
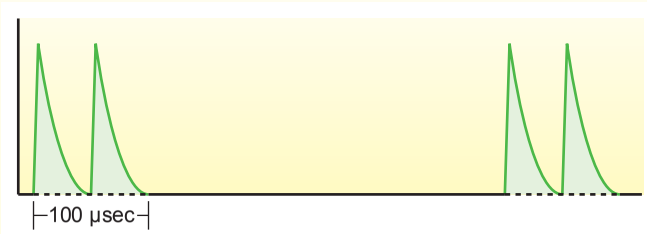
Traditionally, symmetrical biphasic pulsed current has been one of the most commonly used waveforms for activating skeletal muscle along with the burst-modulated AC (i.e., Russian). Many early studies of the effects of electrical stimulation on skeletal muscle have used symmetrical biphasic pulsed or Russian current. Examination of the ability of each current to generate muscular torque has suggested that either there is little to no difference in the peak torque elicited[11] or that torque was greater using the biphasic pulsed current.[13]

A form of symmetrical biphasic pulsed current that incorporates a fixed interphase (or intrapulse) interval is available for muscle activation on some commercial stimulators (VMS: Variable Muscle Stimulation). Use of the interval was shown to elicit similar muscle torques with less current amplitude when compared to a symmetrical biphasic waveform without the interval, although this has not been widely substantiated.[38]

A variation of symmetrical biphasic pulsed current is a burst-modulated form in which three symmetrical square or rectangular biphasic pulses are delivered consecutively in each burst. It may be easier to think of this waveform as a burst-modulated polyphasic waveform containing six phases with 100-μsec interphase intervals per burst (Fig. 9-34). The VMS Burst waveform is similar to burst-modulated AC (i.e., Russian or Aussie currents) in that a KHz frequency carrier current can be delivered in a burst format with burst durations in the millisecond range. The burst duration is dependent on the phase duration selected. With typical phase durations of a few hundred microseconds, as is common for activating muscle, the burst duration and effective carrier frequency of this waveform are consistent with those examined by Ward et al,[16,20] who suggested greater torque is elicited with burst durations of 2 to 5 msec and carrier frequencies less than 2,500 Hz.

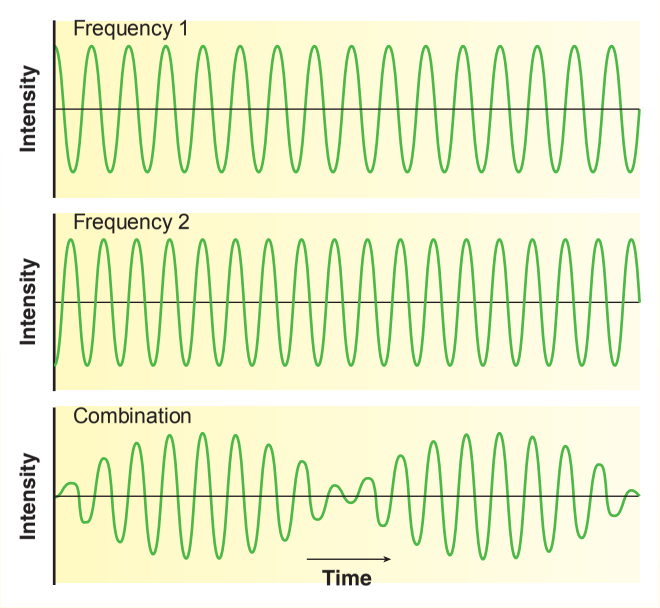
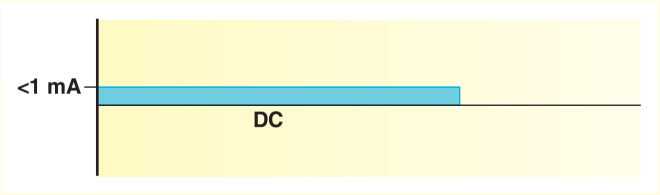
Evidence has shown that this burst-modulated biphasic square pulsed current is more optimal than Russian for activating skeletal muscle.[21] Using equal burst frequency,

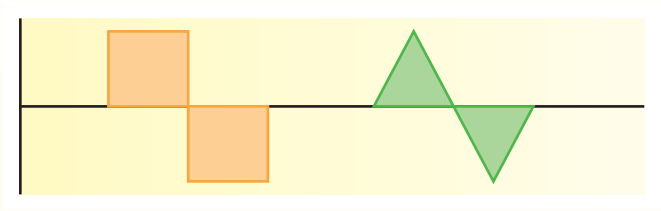
TABLE 9–4. Waveforms, Uses, and Common Parameters

Type	Use	Waveform	Parameters	Settings
Russian 	Activation of skeletal muscle for strengthening and endurance (NMES)	Burst-modulated AC	Pulse duration: Frequency: Amplitude: Treatment time: Ramp-up: Ramp-down:	200–800 μsec 30–100 Hz To maximal contraction for strengthening/ endurance (NMES) 10 sec on/50 sec off × ≥ 10 reps 1–2 sec 1–2 sec
High volt 	Pain modulation Wound/tissue healing	Twin-peak monophasic pulsed	Pulse duration: Frequency: Amplitude: Duration: Amplitude: Duration: Pulse duration: Frequency: Amplitude: Duration:	50–100 μsec 1–100 Hz Sensory level: to perceivable level: 10–30 min Motor level: to visible motor response: 1–30 min 50–100 μsec 100 Hz Below sensory threshold Several minutes

Continued

TABLE 9–4. Waveforms, Uses, and Common Parameters—cont'd

Type	Use	Waveform	Parameters	Settings
Interferential or Premodulated 	Pain modulation	Amplitude modulated AC	Pulse duration: Frequency: Amplitude: Duration:	200–400 μsec Fixed-beat frequency: 100 Hz Full-spectrum sweep: 10–150 Hz Sensory level 10–30 min
Microcurrent 	Wound/tissue healing	Low-intensity DC (microamperage current)	Pulse duration: Frequency: Amplitude: Duration:	0.5 msec, or DC 1 Hz, or DC Less than 1 mA Minutes to hours

Symmetrical biphasic: square or triangular 	Activation of skeletal muscle for strengthening and endurance (NMES), or functional electrical stimulation (FES)	Symmetrical biphasic: square or triangular	Pulse duration: Frequency: Amplitude: Treatment time: Ramp-up: Ramp-down	200–800 μsec 20–60 Hz NMES: to maximal contraction for strengthening/endurance FES: to level for functional use NMES: 10-sec on/50-sec off × ≥ 10 reps FES: activity dependent NMES: 1–2 sec FES: activity dependent NMES: 1–2 sec FES: activity dependent

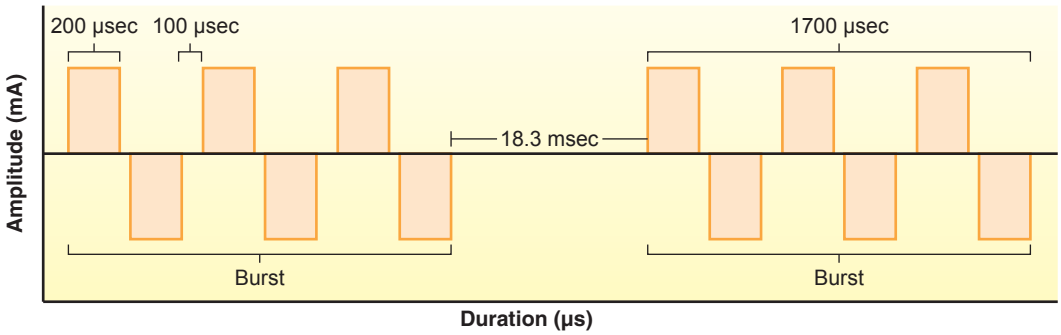


Fig 9 ■ 34 Burst-modulated pulsed current. In this example, with six phase durations of 200 µsec and five fixed interphase intervals of 100 µsec, the burst duration is 1.7 msec. Within the burst, the period of each biphasic pulse is 600 µsec equating to a carrier frequency of 1,667 Hz (i.e., 1/period). At a burst frequency of 50 bps, the interburst interval is 18.3 msec, equating to a relative duty cycle of 9%.

phase duration, and current amplitude but a lesser carrier frequency and burst duration as supported by Ward et al,[8] Bellew et al[21] reported significantly greater muscle force when using the symmetrical biphasic pulsed current. Further delineation of waveform characteristics suggested that the biphasic wave (VMS Burst) elicited greater phase charge than the sinusoidal wave (Russian). This was quantified by Laufer et al, who reported one-third less phase charge for a sine wave than a square wave of like duration and amplitude.[13]

THE BOTTOM LINE FOR ELECTROTHERAPY

Competent, safe, and effective use of electrical stimulation requires that the clinician understand the basics of electrotherapeutic currents and waveforms. Use of electrotherapy offers a variety of physiological effects that can be used to provide substantial therapeutic benefits to patients. While many variations of electrical waveforms have emerged over time, the electrophysiological effects remain relatively well understood. Recognizing the role of clinical electrotherapy in patient management and its potential offerings is a starting point.

Documentation Tips

Appropriate documentation of the application of electrical stimulation should include the following:

- Waveform
  - Symmetrical biphasic square, twin-peak monophasic, Russian, interferential, etc.
- Waveform parameters (these will depend on the waveform used)
  - Pulse duration and frequency, amplitude, on- and off-time, ramp-up and ramp-down, burst duration, beat frequency, sweep, scan, swing
- Electrode
  - Type, shape, and size
  - Placement location
  - Integrity of skin before and after treatment
- Patient position
- Dosage
  - For neuromuscular electrical stimulation (NMES): the amplitude required to achieve the desired response
  - For iontophoresis: the product of current amplitude × treatment duration (e.g., 80 mA × min)
- Treatment duration

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CHAPTER 10

CLINICAL ELECTRICAL STIMULATION

Application and Techniques

James W. Bellew, PT, EdD

Considering the many waveform parameters presented in Chapter 9, it should not be surprising that many clinicians feel intimidated or confused by clinical electrotherapy. However, a competent and clinically effective knowledge of electrotherapeutics can easily be obtained by understanding how parameters create the many effects produced with electrotherapy. Unfortunately, intimidation and confusion are perpetuated by commercial hype, poorly informed clinicians, complex-looking instrumentation, and sometimes misleading user's manuals. Waveforms vary in so many ways, and these variations are even more obvious when looking at the variety of dials, knobs, switches, and lights that are found on electrotherapeutic devices.

INSTRUMENTATION FOR ELECTROTHERAPY

Classifying Electrotherapeutic Devices

Electrotherapeutic devices can be described by the type of current generated and the power sources. Common clinical names used to describe devices include *Russian, high-volt, microcurrent,* and *interferential*. Although these names are common, use of more

specific descriptors of the individual waveforms is encouraged. When classifying by power source, devices are either line-powered or battery-powered. Line-powered devices are powered by wall current (110 volt, 60 hertz [Hz] in North America) and are plugged into a wall outlet for use. These devices are sometimes referred to as *clinical devices* because their portability is limited. Battery-powered devices obtain their power from a variety of different battery sources, including the common AA, AAA, and 9-volt batteries as well as rechargeable batteries. The major advantage of battery-powered devices is their portability, allowing them to be used outside the clinic and while the patient is engaged in activity.

Line-powered devices have traditionally been capable of delivering greater current intensities than battery-powered devices. This was particularly true with stimulators delivering large-amplitude currents for muscle strengthening. For many years, evidence suggested that battery-powered stimulators used for muscle strengthening could not sufficiently activate skeletal muscle when compared to line-powered devices. However, evidence has altered this opinion. In studies of healthy, strong adults, Laufer et al[1] and Lyons et al[2] reported evidence that newer battery-powered devices can stimulate skeletal muscle to levels once considered achievable only by line-powered devices.

Clinical Controversy

A common misconception is that, because batteries provide direct current (DC), battery-powered devices must deliver DC to the patient. This is for the most part inaccurate, the exception being devices used for iontophoresis. Although DC is provided by a battery, electrotherapeutic devices may provide a variety of other waveforms, none or only one of which may be DC. Likewise, line-powered devices receiving alternating current (AC) from the wall outlet do not deliver true 60 Hz AC to the patient. Rather, the alteration of battery-provided DC and household AC to other therapeutic waveforms occurs through the use of rectifiers, transformers, filters, and regulators within electrical devices. These take an input current and modify the current into a waveform to be used for therapeutic purposes.

Therapeutic electrical devices generally consist of two primary components—a signal generator and modulator circuits for modulating the source current into the output current. For the majority of applications, this rectified current is pulsed. For most line-powered electrotherapeutic devices in clinical use today, several waveform options are available, whereas battery-powered devices tend to have only one type of current or a limited few (Fig. 10-1). Because electrotherapeutic devices generate an output current, they are sometimes referred to as generators, although this is not as common as it once was.

Control of Electrical Stimulation: The Dials and Buttons

Various waveform parameters, such as amplitude, frequency, pulse duration, and ramp, are often at the practitioner's control. The many dials, buttons, and switches on most devices are controlled by some form of oscillator circuitry. An output amplifier helps modify the intensity of the current delivered to the patient by controlling either the voltage or the current. Some devices available

Fig 10 ■ 1 Most line-powered units offer a variety of electrotherapy waveforms.

today allow the clinician to choose between a current output with constant voltage (CV) or constant current (CC). Voltage and current are directly proportional. Thus, a change in one necessitates a change in the other based on the resistance of the circuit. With CV devices, the voltage force driving the current will remain constant. As a result of the varying biological resistances encountered by current as it passes through the body, such as bone and fat, the current will increase or decrease while the voltage remains constant. If a patient sweats while receiving stimulation from a CV device, resistance decreases and current may increase to higher levels. This should always be considered when using heating packs with stimulation.

A CC device maintains a constant flow of current, despite varying biological resistances, by adjusting the voltage force pushing the current. Regarding which is better for clinical use, it is helpful to keep in mind that without the flow of current, none of the biophysical responses elicited by electrotherapy would be possible. With constant voltage, the current may decrease to an ineffective intensity, whereas with CC, the intensity is maintained closer to the level required for the desired therapeutic effect (Fig. 10-2).

Controls for pulse duration and frequency are also common to many devices. Pulse duration may often be labeled *pulse width,* although this is not preferred terminology, because *pulse duration* implies a chronological unit (i.e., milliseconds), whereas *width* implies linear measurement. Pulse frequency is often labeled as *rate, pps* (pulses per second), or *frequency,* and the clinician can select the number of pulses per second of pulsatile current. For waveforms using bursts (e.g., Russian) or beats (e.g., interferential current), the frequency is either fixed or controlled by an altogether different mechanism. Keep in mind that devices offering control for pulse width generate pulsatile current because neither DC nor AC has pulse duration. Pulse duration is the total time elapsed from beginning to end of all phases, including the interphase interval within a pulse.

To understand what a device can be used for, simply look at what parameters the dials and buttons control. For example, if a device has controls for on-time and off-time as well as ramp-up and ramp-down, it is probably designed to activate skeletal muscle for strengthening or functional activity. If a device offers control of frequency and pulse duration but lacks on- and off-time controls, it is probably used for electroanalgesia and not strengthening (Fig. 10-3).

It cannot be assumed with accuracy that a numerical change on the control, whether the display is analog or digital, will result in the same change in parameter output. For example, many analog or digital controls allow linear increments from 0 to 10, or something similar. One cannot assume that with a 0-to-10 range, the increase from 1 to 2 will result in a 10% change in parameter output, nor can one assume that a single unit increase from 5 to 6 or from 7 to 8 and so on will result in equal change. In addition, it cannot be assumed that a half turn of a dial will result in a 50% change in that

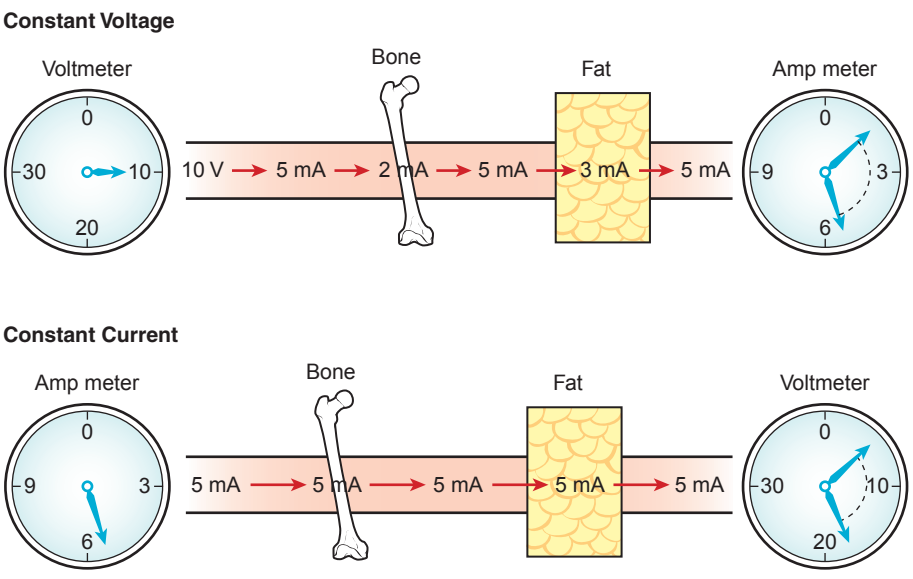

Fig 10 ■ 2 Constant voltage devices maintain the voltage, but current flow will vary through tissues with different resistance. Constant current devices maintain current flow by modulating the voltage through tissues with varying resistance.

Fig 10 ■ 3 A look at the controls will likely indicate what a device can do. A muscle stimulator allows control of the on- and off-time, ramp, and frequency. TENS units often have controls for pulse duration, frequency, and modulation.

parameter. Although most handheld and many line-powered units have amplitude dials that go from zero to maximum amplitude in one 360-degree turn, some line-powered devices have rotary dials that are not fixed to one complete turn and must be rotated several times to increase or decrease the current output.

To understand the linearity of the output controls, clinicians should familiarize themselves with each device and observe the electrophysiological effect on tissue when changing parameters. It is prudent to never deliver to a patient a current that you have not first tried yourself. Only through personal experience and familiarity can one truly understand and describe the feeling of the current to the patient.

Two noticeable changes in electrotherapeutic devices have evolved due to advances in technology. The first is the transition from analog controls (often dials) to digital controls (often buttons). Although the progression from analog to digital control may seem like technological progression, there are limitations to the use of digitally controlled parameters. For example, some devices used to activate skeletal muscle only allow the practitioner the option of selecting 30, 50, or 100 pps. These may be suitable for many applications, but the use of any other frequencies is not possible.

The second noticeable technological change is the advent and inclusion of preprogrammed clinical treatment protocols. Although these are designed to assist the practitioner in clinical decision-making and parameter selection, choosing parameters based on patient-specific needs and desired electrotherapeutic effects is encouraged. One of the main reasons modern electrotherapeutic devices are made with less manual control is because clinical competency in using these parameters has declined. A well-educated clinician should strive to select specific waveform parameters rather than opt for preprogrammed settings.

Electrodes: Types and Choices

An *electrode* is defined as the device that serves as the interface relaying current between an electrical stimulation (ES) device and the patient. Often, little consideration is given to the choice of electrodes, preparation for electrode placement, placement sites, and care of electrodes. This is ironic because electrodes can be considered the most important link between the device and the patient. Successful use of electrotherapy depends on correct selection of electrodes so the appropriate tissues are stimulated.

There are two classes of electrodes: (1) surface or transcutaneous electrodes and (2) invasive or indwelling electrodes. Most clinical applications of electrotherapy use surface electrodes to deliver current transcutaneously from the device to the patient. Surface electrodes can be used in reverse fashion to relay electrical signals from the patient to a device during electromyographic biofeedback. The most common use of invasive electrodes is for recording the electrical activity of subjects during electrodiagnostic examinations. Invasive or indwelling electrodes may also be used to deliver therapeutic stimulation, but this is much less common and typically used for kinesiologic examination where fine-wired electrodes are inserted into muscle.

Electrodes vary in many ways, such as shape, dimensions, flexibility, method of adherence to the skin, cost, and material (Fig. 10-4). Electrodes must conduct current well, be flexible enough to conform to varying body surfaces, and be durable. Metal electrodes are usually made of tin, steel, or aluminum and require a wet sponge as an interface between the metal and the tissue. Electrodes made from metal are excellent conductors and are usually durable, but they often lack the flexibility to conform over body contours. Use of metal electrodes is associated with an increased risk of electrical burns and has largely given way to commercially manufactured disposable electrodes. The most common use of metal electrodes is seen in handheld applicators and stimulating probes.

Flexible electrodes are usually made of carbonized silicon rubber, having the advantage of pliability while

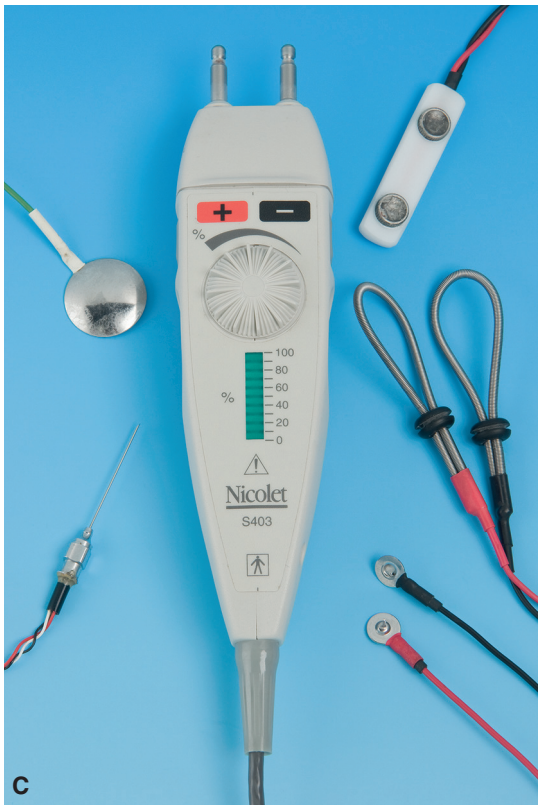

Fig 10 ■ 4 Electrodes come in many shapes and sizes, indicating a variety of uses, including (A) carbon and sponge reusable electrodes; (B) adhesive disposable electrodes; and (C) needle, bar, disk, ring, and probe electrodes used for diagnostic studies.

maintaining good conductivity. Flexible electrodes may be designed for long-term repeated use, short-term use (several days to weeks), or one-time use. Short-term or one-time-use electrodes are intended for single-patient use and are disposable. They are often coated with a conductive coupling agent, such as karaya gum (an organic extract of sterculia trees), whereas reusable clinical electrodes may not have a conductive agent and therefore require the addition of electroconductive gel before use. It is recommended that the patient be asked about any known allergies to adhesives or derivatives of such, as allergic reactions to electrodes are rare but not unseen (Fig. 10-5).

Disposable electrodes come in many different sizes and shapes, including those designed for small sites, such as the fingers, to much larger sites, such as the lumbosacral region. One disadvantage of disposable electrodes is that they need to be replaced often, yet the number of times that reusable electrodes can be safely and effectively used varies greatly. Disposable electrodes used for strengthening typically have a life of 18 to 20 applications before patients notice a change in tolerance to the stimulus or a greater-intensity stimulus is needed to achieve the same effect.[3] The number

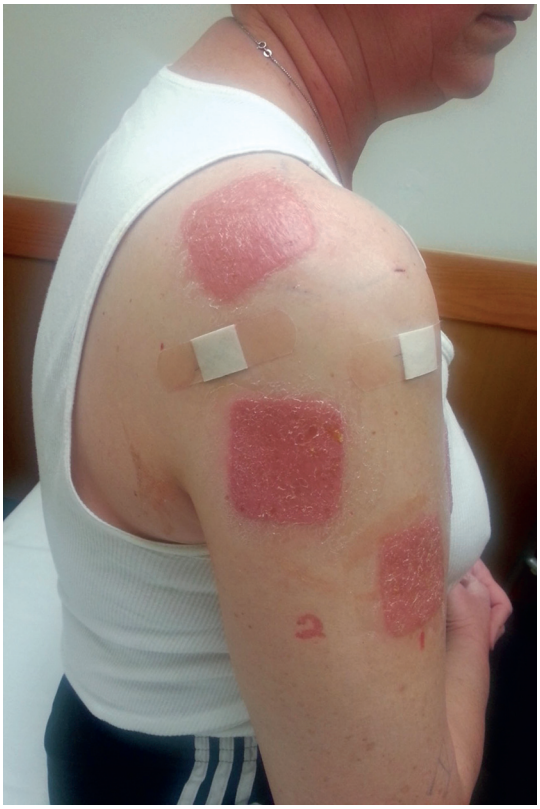

Fig 10 ■ 5 Patient with allergic reaction to adhesive electrodes.

of uses of each electrode should be recorded. For sanitary reasons, each patient should be issued a set of electrodes that are not to be shared with others; these can be kept in the patient's chart or a different location.

The interface where the pin lead from the electrical stimulation device joins the electrode should be examined. The pin may enter directly into the electrode or may connect to a small lead wire connected to the electrode. This junction is clinically referred to as a *pigtail*. This pigtail junction is a common site of breakdown in the machine-to-electrode interface. A compromise of this interface can lead to current leakage and the potential for electrical burn. The conductive surface of the electrodes should be checked before and after use. The surface should appear uniform and without pitting, pocking, tearing, or other areas of compromised integrity (Fig. 10-6). All of these increase the risk for adverse events and, at the very least, reduce the effectiveness of the application (e.g., current delivered to the patient).

Key Point! Signs that electrodes need to be replaced can be both subjective and objective. Subjective patient reports may include a change in the perceived quality of the stimulus, as in a stinging sensation or a decreased feeling of current (assuming the parameters are the same as in previous applications) or the acute onset of such sensations that were not present on previous applications. Objective signs can include skin reddening more than in previous applications; changes in the physical appearance or integrity of the conductive surface, pigtail connector, or pin interface; or a decrease in the physiological response (e.g., less muscle contraction), again assuming the parameters remain the same.

Applying Electrodes

Before applying electrodes, the skin surface must be evaluated and prepared to ensure optimal conductance and to limit impedance. A thorough cleansing of the skin with soap and water is recommended to remove oils, dirt, and lotions, which resist current. Preparing the skin with alcohol-based cleaners is common, but this should be used with caution, as the drying and desiccating effect on the skin can lead to increased resistance to current. It is

Fig 10 ■ 6 Visual inspection of electrodes prior to and after use is recommended. Breakdown of the connector and conductive surface is common after several uses.

recommended to shave hair at the electrode placement site to improve adherence of the electrodes, but shaving should occur the day prior to application of electrotherapy, because scraping of the skin acts to acutely denude the stratum corneum, reducing overall skin impedance; this can result in uncomfortable stimulation. More aggressive preparation of the skin by abrasive scrubbing, often with alcohol wipes, is not necessary, again considering the acute effect of reducing skin impedance to current and exposing the more sensitive layers of the dermis.

Electrodes using sponges for interfaces must be wet with a conductive liquid. Tap water is commonly used because it is a good conductor; distilled water will not conduct as well because the natural impurities that act as conductive ions have been removed. Saline is a good conductor and is often used, but it is more costly than tap water. Flexible carbonized silicon rubber electrodes that are not manufactured with a coupling agent require the addition of a hypoallergenic aqueous gel ("electrode" or ultrasonic gel). Most flexible adhesive electrodes are manufactured with a self-adhesive conductive polymer that serves as both the conductive medium and the adhesive. Electrode adhesiveness can be improved by applying a drop or two of water. This is especially true after repeated applications, but with too many uses, conductivity will be reduced. Following treatment, sponges must be sanitized with soap or bleach solutions between patient use. It may be prudent for each patient to have sponges issued that are not shared with others. Even flexible carbonized silicon rubber electrodes deteriorate over repeated use and need to be replaced. If the patient reports a decrease in the tolerance to the current or noticeably more current

is required to elicit the same effect, the electrodes likely need to be replaced.

Short-use disposable electrodes also begin to deteriorate, with erosion of the coupling agent occurring after repeated use (see Fig. 10-6). Often the patient will report a burning sensation under an electrode. These "hot spots" are often due to a compromise or erosion of the conductive medium. To prolong the life of short-use disposable electrodes, care should be given to keep the conductive surface from drying out. Electrodes should be stored on the plastic sheet from the package. Most of these plastic sheets are marked "NO" on one side and "ON" on the other side. The "ON" side is covered with a special polymer to prolong the life of the electrode, so electrodes should be placed on the "ON" side.

The choice of electrode size should be made with two things in mind: the goal of the treatment and the size of the area to be stimulated. Usually, the larger the area to be stimulated, the larger the electrodes need to be. For example, electrodes used to stimulate the quadriceps muscles should be larger than those for the forearm muscles. Electrode size and the amount of current it conducts determine current density. *Current density* is the current per unit area of the electrode (mA/cm^2 or mm^2). This assumes that there is uniform conduction of current across the electroconductive surface of the electrode, that the electrode surface is in full contact with the skin, and that no hot spots are present. Current density is inversely proportional to electrode size. For the same amount of current, the current density in a smaller electrode will be greater than that in a larger electrode (Fig. 10-7).

High levels of stimulation are generally not used with small electrodes, because the greater current density may result in uncomfortable burning sensations. If electrodes of different shapes are used at the same time, the current density across the smaller electrode will be greater, resulting in the perception of a stronger stimulus under the smaller electrode. Some electrode configurations will take advantage of this, as we will see shortly. It is more common that the electrode size is equal for most clinical applications. Electrode size should reflect the size of the area that is to be stimulated. Although larger electrodes will have less current density than smaller electrodes (assuming like current amplitude) and will be more comfortable for the patient, use of larger than necessary electrodes can lead to stimulation of tissues that are not necessary and even counter to the purpose of the stimulation. Take, for example, using electrical stimulation to activate the anterior tibialis with the goal of dorsiflexing the ankle to clear the toes during the swing phase of gait. Electrodes that are larger than necessary may likely stimulate the peroneus longus and introduce a plantarflexion and eversion component, which opposes the desired effect of the anterior tibialis.

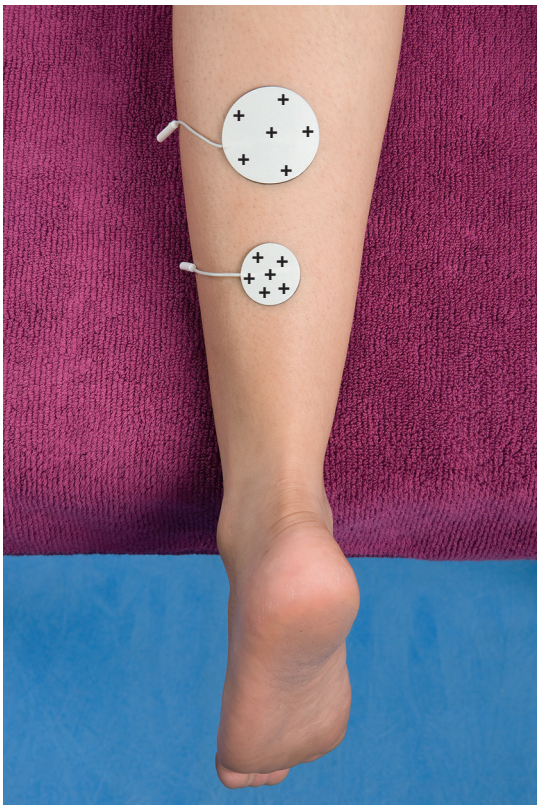

Fig 10 ■ 7 With the same amount of current delivered to electrodes of differing size, the current density will be greater under the smaller electrode.

Attaching electrodes to the skin also requires some consideration. Although self-adhesive electrodes usually require no additional adhesive or means of fixing the electrodes to the skin, other electrodes will. When attaching electrodes, the primary consideration is to make sure the conductive surface is over the tissue to be treated and that the full surface remains in contact with the skin. Use of adhesive tapes and elastic bands are an effective way to keep electrodes in place. Sometimes, small weights are placed atop the electrode to keep them in position, but this method and the use of tape or elastic bands must be used with attention. It is not uncommon to see flexible electrodes bend or curl under the pressure of weights or excessively tight tape or elastic bands (Fig. 10-8). This can result in a lack of contact of the outer edges of some

Fig 10 ■ 8 When using elastic or tape to place electrodes, avoid curling or bending the electrode. Curling will reduce the conductive area of the electrode, increase the charge density, and increase the risk of burn.

electrodes and increased current density, impedance, discomfort, and risk of electrical burn under the compressed area of the electrode.

Insecure or loose attachment of electrodes can result in their movement and in changes of the electrode-to-skin contact. Loose contact is usually an early sign that self-adhesive electrodes need to be replaced. Special attention needs to be given to electrodes specifically designed for iontophoresis, which use DC current. These should not be attached with additional tape, bands, or weights because these may greatly increase the current density with DC and result in skin irritation, discomfort, or electrical burn. Sleeping with active electrodes can also be dangerous because the electrode may peel and lose full contact with the skin, increasing the current density to a dangerous level.

Key Point! To date, there is not substantial evidence from quality clinical studies to support or refute the use of adhesive reusable electrodes over reusable gelled carbon electrodes. At this time, selection is more a matter of clinician preference but consideration should be given to choosing the electrodes that offer the best clinical effect.[4]

Placement of Electrodes

Where electrodes are placed is determined by what electrotherapy is trying to accomplish and what tissues need to be stimulated to achieve that goal. Dermatomal, sclerotomal, or myotomal sensory distributions can be used. If, for example, stimulation of sensory nerves is desired, electrodes need to be placed over the relevant sensory nerves. If motor stimulation is desired, the stimulus needs to be placed over the motor nerve innervating the muscle to be activated. In normally innervated patients, the neurolemma (nerve membrane) of a motor nerve has a lower threshold for activation than the sarcolemma (muscle membrane); therefore, ES will depolarize the motor nerve, not the muscle directly. Motor nerves are traditionally stimulated over the motor points, which are the regions where the motor nerve is most easily excited and thus accessible to stimulation. They are typically found in the muscle belly or proximal third of the muscle. In electrophysiological terms, motor points are the locations where the greatest motor response is found for a given amount of stimulus—the place where resistance to the current is least. Although motor point charts have traditionally been used and are still widely available and distributed by manufacturers as clinical guides to electrode placement, variability does exist between individuals.

Key Point! Because tremendous inter- and intra-patient variability exists in motor point locations, as shown in classic motor point maps, the clinician should identify or locate "functional" motor points.[5] Functional motor points, determined by clinical assessment, are the areas where the best or most robust motor response is noted.

Clinical Application

It is relatively easy to find the functional motor points of a muscle in most patients. Using a stimulator capable of eliciting a motor response, place two small, gelled electrodes (versus adhesive) on the muscle to be stimulated. Set the parameters to produce a motor response and increase the amplitude. Slowly move or glide the electrodes on the surface of the muscle belly while observing for the greatest muscle response. The areas demonstrating the greatest response are considered the functional motor points and the areas where electrodes should be placed during muscle stimulation (Fig. 10-9).

Another technique used to identify the location for electrode placement uses direct palpation by the clinician.[99,100] To do this, the clinician applies

Fig 10 ■ 9 Moving or gliding small, gelled electrodes over the muscle belly while providing stimulation will assist in locating the functional motor points. The areas with the greatest motor response to the stimulation are the functional motor points.

one electrode to his or her forearm, palm, or dorsum of the hand while placing the other electrode on the patient (Fig. 10-10).

The clinician then applies conductive gel to three or four fingers of the hand used to palpate and places the fingers on the patient in the intended area of stimulation. (The current density through the therapist's fingers will be higher if only one or two fingers are used, so use three or four fingers.) Finger contact with the patient creates a circuit between the patient and the subject. In this manner, the clinician's fingers become an electrode, which will be used to explore and locate the region of least resistance. After increasing the intensity of the stimulus, the clinician simply moves the fingers around the intended area of stimulation, looking for the site that elicits the greatest motor response. Electrodes are then applied to this area for stimulation in the customary manner.

Fig 10 ■ 10 Creating a circuit between the patient and clinician allows for a moving palpation to identify the functional motor points. One electrode is in the hand of the clinician and the other is on the patient. Finger contact with the patient completes the circuit.

When placing electrodes for motor stimulation, a very common mistake occurs when one electrode is placed over a motor point and the other over a more distal site. The problem with this can be twofold: (1) the distal electrode is often placed away from the region of the motor nerve where the optimal response is obtained, rendering the stimulus less effective, and (2) the distal electrode is often placed in a region where there is significantly less or no depolarizable muscle tissue (Fig. 10-11). Thus, when the clinician increases the intensity, the patient is more likely to report a strong sensation of the stimulus but with little to no motor activity. This problem seems to be very common in motor stimulation of the muscles of the forearm and lower leg, where the majority of the muscle mass lies in the upper one-half of the limb segment. An electrode is commonly placed over the proximal muscle mass and one placed distally near the wrist or ankle. Although the proximal one is likely near a motor point, the distal electrode is not over the muscle group but rather a large tendon area. Besides not achieving the motor response desired, stimulation of nonmotor tissues with motor-level stimulation can cause discomfort and will likely require a decrease in the stimulus amplitude, thus diminishing the effect of the treatment.

Clinical Application

Recall from Chapter 9 that the normal order of response to ES is a sensory followed by a motor and then a noxious (e.g., painful) response. During stimulation of muscle, if the patient's sensation goes straight

from a sensory to a noxious response, it is probably because one or more of the electrodes is not over an area of muscle tissue. A relocation of electrodes closer to a motor point will result in the appropriate order of response.

The distance between electrodes is termed *interelectrode distance*. Considering that current will take the path of least resistance, the clinician must ensure that the stimulus current has the best chance of reaching the desired tissue. Electrodes should not be so close together that they may come in contact with each other. This may result in increased current density in the superficial tissue between the electrodes and increase the chance of burning the patient. Making sure the stimulus current reaches deeper tissues where motor nerves, muscles, and bone lie requires appropriate spacing of electrodes; then the current is less likely to travel in the superficial tissues between electrodes. Instead, current travels in the deeper tissues, thus increasing the likelihood that the desired tissues will be stimulated (Fig. 10-12). With wider spacing, greater current amplitude can often be used to reach the deeper tissues. Wider spacing can also lead to more motor units being activated (for a neuromuscular electrical stimulation [NMES] application) or more sensory fibers activated (in a transcutaneous electrical nerve stimulation [TENS] mode).

Electrode Configurations

The placement, or configuration, of electrodes will greatly impact the effect of ES. The majority of electrotherapy techniques consist of a single circuit and two electrodes placed over the target tissue. The most common electrode configuration is a *bipolar electrode configuration,* in which all the electrodes of a single circuit are placed over the treatment area (Fig. 10-13B). A bipolar configuration is most commonly used with biphasic

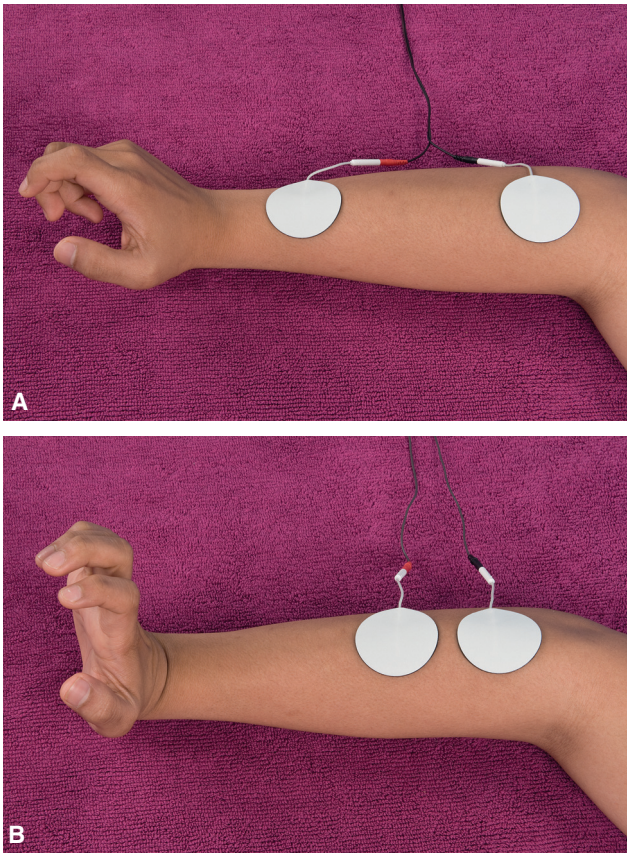

Fig 10 ■ 11 Electrodes should be positioned over the muscle tissue to be stimulated. Placement of electrodes distal to the muscle fibers (A) results in stimulation over areas without muscle tissue and may be uncomfortable or result in lesser response from the muscle. Placement over more muscular tissue (B) results in a more robust response.

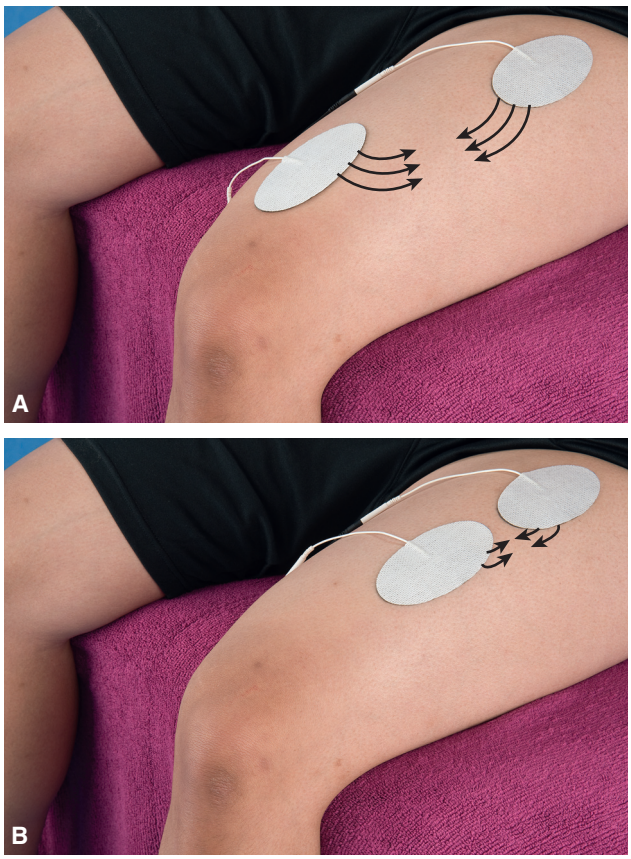

Fig 10 ■ 12 The interelectrode distance will affect the depth of current penetration. Wider placements (A) will result in greater depth of penetration than narrower (B).

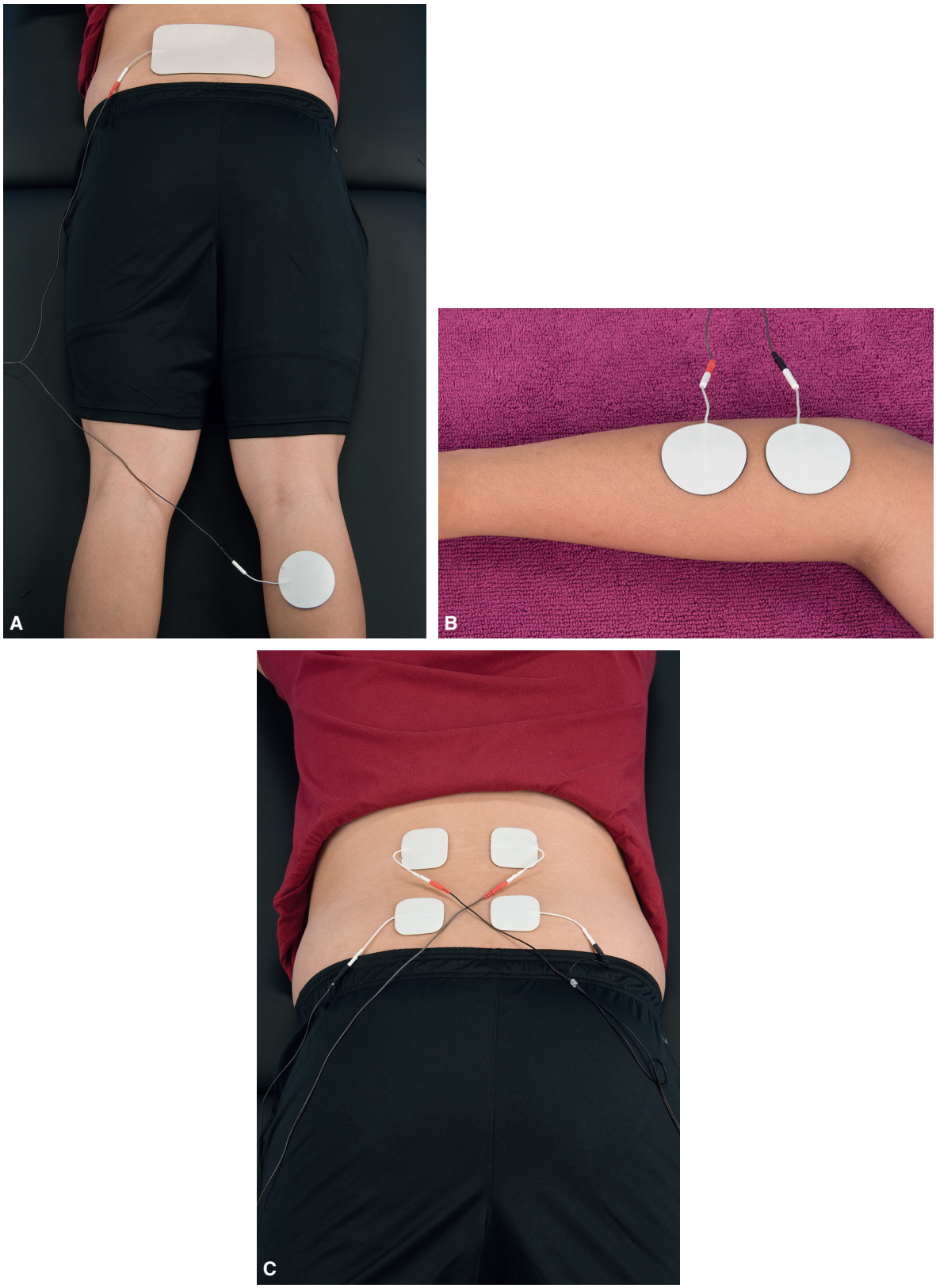

Fig 10 ■ 13 (A) Monopolar, (B) bipolar, and (C) quadripolar electrode placements.

pulsed currents, burst-modulated AC (e.g., Russian), and premodulated current during stimulation of muscle (i.e., NMES and functional electrical stimulation [FES]) and electroanalgesia (i.e., TENS). The electrodes are usually the same size, so the current density through each electrode is equal. If the two electrodes differ in size, the smaller of the two will necessarily have a greater current density and impedance than the larger electrode. All the electrodes are considered treatment or active electrodes with a bipolar electrode orientation.

Key Point! Bipolar electrode placement for muscle stimulation may be either parallel to the fiber direction of the muscle or perpendicular. To date, there is no substantial evidence to support selection of one over the other. The most appropriate placement is the one that elicits the most desirable motor response.

Sometimes it is desirable to have stimulation only under a single electrode. In this case, an electrode of a single circuit may be positioned over the treatment area while the other electrode is placed at a site where a therapeutic effect is not intended. This configuration is termed a *monopolar arrangement* (Fig. 10-13A). Monopolar electrode placement is most commonly used with DC and monophasic pulsed currents during iontophoresis and many applications for wound or tissue healing. It is important to recognize that the lead wires from a device generating a single circuit can be split, or bifurcated, allowing two (or even more) electrodes to be used at the treatment site. If a lead wire is bifurcated to allow more than one electrode at the treatment area with a third placed over a nontreatment area, it is still considered a monopolar arrangement. With a monopolar electrode orientation, the electrode(s) over the target tissue is the treatment or active electrode, whereas the other electrode is termed the *reference* or *inactive* electrode.

Fundamentals in Clinical Practice: Monopolar Electrode Configuration With One or Two Active Electrodes and a Single Inactive Electrode

The clinician treating a stage IV pressure ulcer with twin-peak monophasic current may choose to place an active electrode over the wound to deliver cathodal (negative) stimulation while placing the inactive or reference electrode adjacent to the wound. In this application, there are only two electrodes of a single circuit—one treatment and one reference. If treating acute swelling of the lateral and medial ankle secondary to sprain, the clinician may choose to bifurcate the lead wire of the active electrodes to deliver cathodal (negative) stimulation to both sides of the joint while placing the inactive electrode on the leg. In this application, there are three electrodes—two over the treatment area and one away yet still maintaining a single circuit.

As with bipolar orientation, if electrodes differ in size, the smaller electrode will have a greater current density. Because of the electrochemical effects of DC and monophasic pulsed current, asymmetrical electrode size with these currents must be considered so as to avoid adverse effects. More often than not, if one electrode of an electrotherapy device uses a much larger electrode (2 in. × 4 in. and ranging up to 8 in. × 10 in.), then the device likely provides DC, monophasic pulsed current, or unbalanced asymmetrical pulsed current. In these currents, one electrode will remain the anode and the other the cathode.

Bifurcation of lead wires can allow a greater area to be treated but decreases the current density at each individual electrode because the current is now shared between the electrodes. For example, if a monopolar electrode arrangement with a single active electrode placed over the gastrocnemius with current amplitude of 50 mA is then bifurcated to allow two electrodes over the gastrocnemius, the current density under the two electrodes is now 25 mA (assuming both electrodes are of equal size and impedance and total current output remains 50 mA). The overall effect of the change in stimulus amplitude must be considered when choosing to use more than one active electrode.

A third electrode configuration exists, which involves the application of two or more electrodes from two separate circuits. The electrodes are placed so that the currents are intentionally crossed, or interfered, as in interferential current. This configuration, using at least four electrodes, is termed a *quadripolar configuration* (Fig. 10-13C). True quadripolar interferential current is the most common application using a quadripolar electrode configuration. Table 10-1 summarizes these configurations.

Simply using four electrodes does not constitute a quadripolar orientation. It is not uncommon to see four

TABLE 10–1. Basic Electrode Configurations and Clinical Use

Name	Electrode Location	Common Use
Monopolar	Active electrode of single circuit over target area; inactive over nearby nontreatment area	Pain modulation, iontophoresis, tissue healing
Bipolar	Both or all electrodes of single circuit over target tissues	Muscle activation, pain modulation
Quadripolar	Four electrodes of two circuits over target tissues	Pain modulation

electrodes used for muscle activation or pain modulation over large areas. These uses are typically two separate circuits with bipolar electrode arrangement applied to treat the larger area. To have true interferential current, there must be two currents of differing frequencies configured to cross or interfere.

The name *monopolar* is misleading because there are always two poles—an anode and a cathode—required to make a complete circuit. The electrode with the greater concentration of negative ions or electrons is the cathode, whereas the anode has a lesser concentration of negative ions or electrons. The greater concentration of negative ions near the cathode reduces the resting membrane potential across the cell, thereby depolarizing the nerve. In contrast, the lesser concentration of negative ions at the anode results in hyperpolarization of the cell and decreased responsiveness to stimulation. Because the cathode is the site of depolarization and the anode is the site of hyperpolarization, the cathode is termed the *active electrode* and the anode the *inactive electrode*. However, with AC and biphasic currents that regularly alternate or change direction of current flow, the electrodes also alternate from being the cathode or anode (Box 10-1).

Box 10 ▪ 1 Which Color Wire Should I Use Where?

The lead wires of many electrotherapy devices are manufactured with red and black ends. Many clinicians assume this indicates the positive and negative leads, respectively. However, with currents that continually change direction, such as AC and biphasic currents, the polarity is continually changing, so the red or black lead is neither the anode nor the cathode for more than the duration of a single phase of a biphasic pulse (i.e., microseconds). Only when using DC, monophasic pulsed current, or unbalanced asymmetrical pulsed current would the red and black truly indicate an anode and cathode.

Whether an electrode elicits the effect of a specific pole (i.e., has a polar effect) is a matter of whether polarity is sustained under that electrode. For example, AC, symmetrical biphasic pulsed current, and balanced asymmetrical pulsed current result in no net charge (sometimes termed *zero net DC*); thus, the electrodes do not remain a true anode or cathode for longer than a few hundred microseconds because the current is regularly "alternating" between positive and negative, depending on the frequency. Consider the polarity of each electrode to be constantly changing from positive to negative, never maintaining or accumulating enough charge to induce a polar effect (i.e., a cathodal or anodal effect). In contrast, three waveforms—DC, monophasic pulsed current, and unbalanced asymmetrical biphasic pulsed current—can cause true polar effects, because current either flows in only one direction (DC) or flows in one direction more than the other. This results in greater-than-zero net charge at the electrodes. A sustained anode and cathode with polar effects are present only with the latter three waveforms (Fig. 10-14). It should be noted that the pictures of waveforms in a manual depict what is happening only at one electrode. What is happening at the other wire is a mirror image of this, reflected about the isoelectric line.

ELECTROTHERAPY APPLICATION AND TECHNIQUES: WHY USE ELECTROTHERAPY?

Deciphering the "Electro Lingo Code"

Clinical applications of ES are most commonly used for the following purposes: to activate skeletal muscle for improving muscle performance or strengthening, to attenuate or alleviate pain, to improve blood flow, to decrease or control edema, or to facilitate tissue healing. For various reasons, ES for these purposes has come to

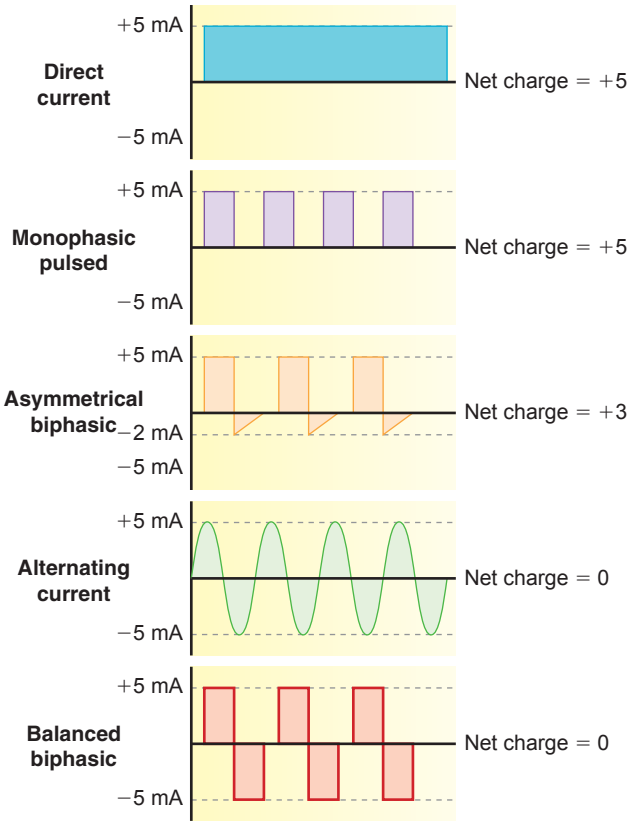

Fig 10 ■ 14 Direct current (DC), monophasic pulsed, and unbalanced asymmetrical biphasic pulsed currents will develop a net charge and thus have a sustained anode and cathode because current flows in only one direction or one direction more than the other. No net charge is accumulated with alternating current (AC), symmetrical biphasic pulsed current, and balanced biphasic currents. Note that net charge would be negative if current flowed only in the negative direction or more current flowed in the negative direction than positive.

be known or recognized by a variety of acronyms, abbreviations, or clinical lingo. Activation of skeletal muscle for strengthening is referred to as *neuromuscular electrical stimulation* (NMES). Activation of skeletal muscle for reeducation or movement training for functional use is referred to as *functional electrical stimulation* (FES). ES for modulation of pain is called *transcutaneous electrical nerve stimulation* (TENS), despite nearly all forms of clinical electrotherapy being transcutaneous.

Use of ES to skeletal muscle below the threshold for muscle contraction has become known as *therapeutic* or *threshold electrical stimulation* (TES).[6–8] ES for scoliosis—*scoliotic electrical stimulation* (SES)—is not as common as it once was; nevertheless, it represents another use of an acronym to describe a type of application or ES.[9–10]

Other forms of ES are often recognized and referred to by the type of current used—for example, interferential current, high-volt pulsed current, Russian current, microcurrent (low-intensity direct current), and others not listed in this chapter. Because of all the confusion in terminology, documentation of electrotherapy should more appropriately specify the type of waveform used and any pertinent parameters, particularly if the treatment is to be replicated in subsequent sessions (Box 10-2).

Electrotherapy for Activation of Skeletal Muscle: Strengthening and Reeducation

Clinical electrotherapy for muscle strengthening and reeducation is often directed toward treating or preventing muscular atrophy following disuse, immobilization, or detraining; increasing or maintaining range of motion (ROM); and reeducating, retraining, or

Box 10 ■ 2 Clinical Decision-Making With ES

Regardless of the purpose, when deciding to use ES, the steps in the clinical decision-making process remain the same. Questions to ask when considering use of electrotherapy include the following:

1. What is the clinical goal?
2. Is the patient appropriate for an electrotherapeutic agent?
3. Is there a type of electrotherapeutic agent that can assist in achieving this goal?

If there is an identifiable goal and the answers to the latter two questions are yes, then use of electrotherapy is indicated. The clinician must then continue with the following questions:

1. Is equipment with the appropriate waveform available?
2. What are the specific parameters of the selected waveform?
3. What electrodes and electrode configuration should be used?
4. What factors would necessitate a change in the treatment plan?

The clinician should always explain the procedures to the patient and interpret the effectiveness of the treatment, making appropriate accommodations as necessary. It is prudent for the clinician to apply to patients only an electrotherapeutic agent that they themselves have tried. In this manner, the clinician can offer a more factual description of the sensation and effect. This can only serve to enhance the patient–clinician relationship and trust.

facilitating muscle for movement and posture. NMES has generally come to imply use of ES for purposes of increasing strength in innervated muscle, whereas *electrical muscle stimulation* (EMS) implies the stimulation of denervated muscle. The fundamental difference between NMES and EMS is the tissue that is actually stimulated and depolarized to elicit the therapeutic effect. With NMES in innervated muscle, the intact peripheral nerve, which has a lower threshold for depolarization, depolarizes first and initiates contraction of the skeletal muscle—thus the name *neuromuscular electrical stimulation.* In contrast, ES in denervated muscle does not depolarize the peripheral nerve but rather the muscle itself—thus the name *electrical muscle stimulation.* The term *functional electrical stimulation* has come to describe ES of innervated or denervated skeletal muscle for the purposes of facilitating or enhancing functional movement (e.g., to assist in dorsiflexion of the ankle or wrist extension in the paretic anterior tibial muscles or wrist extensors, respectively).

Strengthening: Neuromuscular Electrical Stimulation

The effect of ES for activating skeletal muscle, whether for strengthening or for reeducation, has been studied much more than some other common physical therapy techniques. ES for strengthening in healthy, nonweak subjects has shown that (1) use of electrical stimulation for strengthening yields better results than no exercise at all;[11,12] (2) there is no difference between the strength gains resulting from voluntary exercise or the use of electrical stimulation alone, assuming the training intensity is similar;[11,13] and (3) there appears to be no benefit to the use of ES combined with exercise versus using each separately.[14,15]

Using ES for strengthening in weak but normally innervated subjects has shown that in the initial periods of rehabilitation when voluntary exercise may be difficult, ES results in greater gains in strength than voluntary exercise or no exercise at all. In the postrehabilitation stages, there is often little to no difference in strength between subjects who used ES and those who did not. Also, there appears to be a significant positive relationship between the intensity of the ES and the strength gained.[16,17]

Key Point! Many earlier studies claiming NMES to be inferior to voluntary exercise for strengthening failed to use similar training intensities—that is, the intensity of the voluntary exercise was greater than that of the NMES. The fundamental principles of strength training would tell us that dissimilar training intensities will not result in similar advancements in strength.

Historically, the most commonly used waveforms for strengthening have been burst-modulated AC (e.g., Russian) and symmetrical biphasic waves of square, rectangular, or triangular shape (Table 10-2). Earlier evidence suggested that the most effective waveform for eliciting muscle force varies between patients, but more

TABLE 10–2. Parameters for NMES and FES

Indication	Type	Waveform	Pulse Frequency	Pulse Duration	Amplitude	Duration
Muscle strengthening	NMES	Biphasic PC or burst-modulated AC (Russian or Aussie)	50–80 pps or bursts per sec	200–800 μsec	As high as tolerated with a goal of reaching more than 60%–70% maximal voluntary contraction (MVC)	10–20 strong contractions
Muscle contraction for functional use	FES	Biphasic PC or burst-modulated AC (Russian or Aussie)	20–60 pps or bursts per sec	200–800 μsec	To level commensurate with functional activity	Task specific

recent evidence has suggested that Aussie current (a form of burst-modulated AC) and burst-modulated biphasic pulsed current (both described in Chapter 9) may be more effective for generating muscle force.[18–22] The typical pulse duration used to activate muscle for strengthening is 200 to 800 microseconds (μsec), with frequencies ranging from 20 to 100 pps. An optimal force-frequency range of 50 to 80 Hz to elicit muscle force has been reported.[23] Interestingly, abdominal stimulators commonly advertised on television do not offer pulse duration and frequency suitable for contracting skeletal muscle; these devices are merely gimmicks.

A bipolar electrode configuration is used with most applications of NMES, with the electrodes placed directly over the muscle to be stimulated. Keep in mind that in healthy, innervated muscle, depolarization of the peripheral nerve innervating the muscle results in contraction, so the electrode placement should be consistent with the anatomic location of the peripheral nerve to the muscle(s) being treated (i.e., over functional motor points). Typical treatment sessions should consider the number of contractions (i.e., repetitions) rather than the duration of the treatment (e.g., 15 minutes) in the same manner a strength-training program would count repetitions. A more detailed discussion of using NMES for muscle strengthening and musculoskeletal applications appears in Chapter 13 (Box 10-3).

Reeducation and Retraining: Functional Electrical Stimulation

ES is often used in patients with paralysis or in those who have an inability to volitionally activate skeletal muscle secondary to stroke, cerebrovascular insult, head injury, spinal cord injury, peripheral nerve injury, or other neurological disorders. ES is used to facilitate and improve purposeful movement, assist with ambulation, reverse cardiopulmonary deconditioning, attenuate bone demineralization following spinal cord injury, and improve circulation. By virtue of its use for functional purposeful activity, FES is used for this application of ES and is differentiated from NMES in that the primary goal is not increased strength (Fig. 10-15).

Box 10 ■ 3 How Long Is a Treatment of NMES?

Many applications of ES and other biophysical agents are applied for a fixed duration. But ES for muscle strengthening should be considered differently. Use of defined sets and repetitions are the cornerstone of strength and conditioning programs and should likewise be used when using ES. For example, figure out the number of contractions, or repetitions, a patient should do and calculate the duration of the treatment from the contraction (i.e., on-time) and rest time (i.e., off-time) of the ES. If using 10-second contraction time with 50 seconds between contractions, the subject would complete one contraction with a rest period every minute. To complete 20 contractions would require a treatment duration of 20 minutes.

Key Point! The interpretation of "functional" should address specific activities functional to the patient, and improvements should be measureable (e.g., a functional grasp or reach measured by accuracy or time to complete a specific task).[24]

Use of ES as a substitute for the intact nervous system was popularized in the 1960s by Liberson et al,[25] who reported using ES as a means of improving gait in patients with hemiparesis and common peroneal nerve palsy. This example of nervous system plasticity underlies much of the continuing clinical and investigative efforts given to better understand how stimulation can be used to enhance purposeful and functional movement in patients with compromised nervous systems or who have difficulty voluntarily activating their muscles.

More recently, electromyographic (EMG) FES was used to trigger muscle stimulation in paretic muscles (primarily wrist and finger extensors) following neurological compromise. EMG-triggered muscle stimulation requires the patient to generate a threshold level of muscle activation from either the paretic muscle or other muscle of the same extremity in order to elicit or trigger an electrical stimulus to the paretic muscles, often the wrist and finger extensors.[26–28] This technique is held to link motor intention of the extremity with motor response.[27]

The use of contralateral controlled functional electrical stimulation (CCFES) has emerged from the EMG-triggered stimulation and uses sensors placed on the contralateral hand. Motions performed in the contralateral hand are detected and used to trigger or stimulate muscles in the contralateral side paretic hand.[27] The stimulated hand can then perform functional tasks or complete exercise retraining activities. Knutson and

Fig 10 ■ 15 (A) Neuromuscular electrical stimulation (NMES) and (B) functional electrical stimulation (FES) are used in a variety of ways. *(Courtesy of Bioness, Inc., Valencia, California.)*

colleagues[27,29] have shown promising results using CCFES in patients with loss of hand function secondary to hemiplegia.

ES to facilitate functional movement is based on activation of the skeletal muscle. Thus, the stimulus parameters used will be similar to those used to strengthen muscle; however, some parameters differ, reflecting the difference in the clinical goals of each type of stimulation. Whereas ES use for strengthening requires intensities at or near those maximally tolerated, the intensity for FES should only be enough to meet the demands of the specific functional task. For example, to assist in dorsiflexion of the ankle to clear the toes during the swing phase of gait, the intensity need only be enough to move the ankle through enough ROM so the functional task of clearing the toes is accomplished. The pulse rate or frequency of the stimulus should be considered in light of fatigue. A higher frequency will result in fatigue sooner than a lesser frequency; thus, lower frequencies are desirable with FES to avoid or delay fatigue of the activated muscle. However, the parameters chosen must still be capable of activating the muscle to accomplish the task.

The most common waveforms used for FES are symmetrical biphasic square and triangular waves and burst-modulated AC (e.g., Russian; see Table 10-2). The pulse duration must be capable of activating the muscle and typically will range between 200 and 800 μsec. When stimulating muscle for functional activity, pulse frequency typically ranges from 20 to 60 pps—less than NMES. Ultimately, the determination of specific stimulus parameters depends on whether the muscle activation accomplishes the functional task. In this regard, slight variations will exist within and between patients. Further description of FES for functional activity and specific treatment techniques appears in Chapter 14.

Electrical Stimulation of Denervated Muscle

The majority of evidence regarding ES for increasing strength has involved healthy, nonweak or healthy, weak subjects, but all of the subjects remained innervated. FES has likewise focused primarily on patients with intact peripheral nervous systems (i.e., intact lower motor neurons). Thus, for NMES and the majority of applications for FES, ES works via an intact peripheral nervous system supplying the muscle(s) to be activated.

The process of activating denervated muscle by ES is uniquely different from stimulating innervated muscle. In innervated muscle, the electrical stimulus depolarizes the peripheral nerve, which results in depolarization and activation of the skeletal muscle. In the case of denervation, the peripheral nerve can no longer be depolarized. Thus, activation of the muscle requires depolarization of the muscle membrane itself—the sarcolemma. This requires a stimulus of significantly greater amplitude and

CASE STUDY 10-1 NMES in the Lower Extremity

A patient is referred for strengthening of the quadriceps following a total knee arthroplasty 5 days prior. The patient demonstrates a decreased ability to volitionally activate the quadriceps in a manner suitable for performing his home and clinical exercise program. NMES is recommended for the quadriceps to retard atrophy and promote strengthening until the patient can volitionally contract the muscle enough to meet the rehab goals.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of electrotherapy?
   ANSWER: Yes, an impaired ability to volitionally activate the quadriceps during the postoperative period can result in atrophy, loss of strength, decreased function, and delayed attainment of rehab goals.

2. Is the patient appropriate for electrotherapy (i.e., do any of the general precautions or contraindications to electrotherapy apply, or are there any specific considerations regarding application of electrotherapy to this patient)?
   ANSWER: The patient is appropriate for NMES.

3. What are the specific goals to be achieved with the use of electrotherapy?
   ANSWER: To activate the contractile mechanics of the quadriceps muscle. The goal is to use NMES to provide a therapeutic stress to the muscle to promote strengthening and retard atrophy until the patient can generate acceptable force with volitional activation.

4. Do you have the specific type of electrotherapy that is appropriate for the patient?
   ANSWER: Yes, a clinical NMES device will be used.

5. Can you select the specific parameters of electrotherapy that are appropriate for the patient?
   ANSWER:
   Waveform: Burst-modulated AC (e.g., Russian or Aussie), biphasic PC (e.g., VMS), or burst-modulated biphasic current (e.g., VMS-Burst) may be used.
   Pulse duration: 200 to 800 μsec
   Pulse frequency: 50–80 pps is optimal for NMES
   On:off time: 10 sec on (contraction) with 30 to 50 seconds off (rest). If fatigue is an issue, then bias toward the longer rest interval.
   Ramps: Ramp-up 1 to 2 sec; ramp-down 1 to 2 sec
   Intensity: Maximally tolerated contractions with a goal of at least 50% of the maximal isometric contraction of the uninvolved side. (See the "Intensity or Dosage" section in Chapter 13 for further description.)
   Duration: 10 to 20 strong contractions or repetitions

6. What are the proper application procedures for electrotherapy?
   ANSWER:
   Instruct the patient: Inform the patient of the purpose and procedure. Be sure to explain the anticipated sensation and effect of the stimulation. In this case, a strong sensation should be noted in the quadriceps that will lead to robust contraction of the muscle.
   Preinspection: Prepare the electrode site by gently washing with soap and water, noting any compromise of the skin. Removal of excessive hair may be required to ensure good electrode contact.
   Electrode placement: Select electrodes that appropriately cover the majority of the muscle belly. If using carbon gelled electrodes, affix the electrodes with elastic wraps, being sure not to bend or deform the electrodes.
   Patient position: Position the patient with the knee slightly flexed over a bolster or up to 90 degrees to place the quadriceps in a slightly lengthened position; a muscle in a shortened position is not at optimal length for developing tension. Fixate the lower leg to allow for an isometric contraction of the quadriceps.
   Postinspection: Inspect the skin for any signs of adverse effects of stimulation such as redness or burns. Redness is not uncommon but should resolve within 24 hours.

CASE STUDY 10-2 NMES in the Upper Extremity

A patient is referred for strengthening of the right wrist extensors following distal radius fracture. Upon examination, the patient demonstrates weakness of the primary wrist extensors and excessive substitution of the extensor digitorum communis (EDC), resulting in hyperextension of the metacarpal phalangeal joints.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of electrotherapy?

ANSWER: Yes, the patient has weakness of the extensor carpi radialis brevis (ECRB), extensor carpi radialis longus (ECRL), and extensor carpi ulnaris (ECU) and is demonstrating a substitution pattern using the EDC.

2. Is the patient appropriate for electrotherapy (i.e., do any of the general precautions or contraindications to electrotherapy apply, or are there any specific considerations regarding application of electrotherapy to this patient)?

ANSWER: The patient does not present with any contraindications or precautions.

3. What are the specific goals to be achieved with the use of electrotherapy?

ANSWER: To increase strength of the ECRB, ECRL, and ECU to reduce the undesired substitution pattern.

4. Do you have the specific type of electrotherapy that is appropriate for the patient?

ANSWER: NMES can be used to strengthen the ECRB, ECRL, and ECU.

5. Can you select the specific parameters of electrotherapy that are appropriate for the patient?

ANSWER:

Waveform: Burst-modulated AC (e.g., Russian or Aussie), biphasic PC, or burst-modulated biphasic (e.g., VMS-Burst) may be used.
Pulse duration: 200 to 800 μsec
Pulse frequency: 35 pps
On:off time: 10 sec on (contraction) with 30 to 50 seconds off (rest)
Ramps: Ramp-up 1 to 2 sec; ramp-down 1 to 2 sec
Intensity: Maximally tolerated contractions with a goal of at least 50% of the maximal isometric contraction of the uninvolved side and a motor response correcting for the substitution pattern.
Duration: 10 to 20 strong contractions

6. What are the proper application procedures for electrotherapy?

ANSWER:

Instruct the patient: Inform the patient of the purpose and procedure. Be sure to explain the anticipated sensation and effect of the stimulation. In this case, a strong sensation of stimulation should be noted in the muscles of the extensor forearm that will lead to contraction of the muscles.
Preinspection: Clean the area to be treated and check for skin compromise. Identify any surface abnormalities that may prevent optimal placement of electrodes.
Electrode placement: Identify the functional motor points for optimal electrode placement over the ECRB, ECRL, and ECU. Because of the small size of the extensor muscle group, a monopolar electrode configuration may be desired with the stimulating electrode over the muscle belly, but a biphasic configuration may be used if both electrodes can be placed over muscular tissue, avoiding placement of the distal electrode over a more tendinous area. Electrodes may be adhesive-disposable or carbon-gelled but should be proportionate to the size of the area to be stimulated. Too-large electrodes may result in cross-stimulation of additional muscles, and too-small electrodes will increase current density and likely result in an uncomfortable sensation.
Patient position: The arm may be rested on the treatment table with the patient seated or recumbent. The patient should not be positioned so that the electrodes are compressed by body weight. The patient may be encouraged to hold on to an object such as a dowel rod or similar cylinder shape during stimulation, as the sustained MCP flexion during grasp reduces substitution from the EDC.
Postinspection: Remove the electrodes and inspect the skin for any signs of skin irritation or adverse effects. If redness appears, explain that this is not uncommon and should disappear in less than 24 hours.

CASE STUDY 10-3 FES for Dorsiflexion During Gait

A patient suffered a fracture of the fibular head, resulting in damage to the deep peroneal nerve, and remains unable to actively dorsiflex the ankle against gravity. The patient is referred for electrical stimulation for orthotic substitution during gait. Manual muscle testing reveals trace volitional contraction of the anterior tibialis.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of electrotherapy?
   ANSWER: Yes, the patient has decreased ability to volitionally contract the anterior tibialis and has gait impairment with decreased dorsiflexion secondary to recent nerve injury.

2. Is the patient appropriate for electrotherapy (i.e., do any of the general precautions or contraindications to electrotherapy apply, or are there any specific considerations regarding application of electrotherapy to this patient)?
   ANSWER: The patient does not present with any contraindications or precautions.

3. What are the specific goals to be achieved with the use of electrotherapy?
   ANSWER: To facilitate recruitment of the anterior tibialis to provide dorsiflexion during gait.

4. Do you have the specific type of electrotherapy that is appropriate for the patient?
   ANSWER: FES can be used to recruit the anterior tibialis.

5. Can you select the specific parameters of electrotherapy that are appropriate for the patient?
   ANSWER:
   Waveform: Biphasic PC or burst-modulated AC (e.g., Russian) may be used
   Pulse duration: 200 to 300 μsec
   Pulse frequency: 20 to 40 pps (less than NMES so as to prevent fatigue)
   On:off time: To be set based on patient's cadence or use of heel-switch to trigger stimulation to muscle
   Ramps: Ramp-up 1 sec; ramp-down 1 sec; modified to the patient's gait pattern.
   Intensity: In contrast to NMES, where maximal contractions are desired, contraction intensity for FES is to a level commensurate with the desired effect. In this case, the functional goal is dorsiflexion that permits normalized gait. Therefore, the intensity should be increased to elicit contractions of this magnitude.

6. What are the proper application procedures for electrotherapy?
   ANSWER:
   Instruct the patient: Inform the patient of the purpose and procedure. Be sure to explain the anticipated sensation and effect of the stimulation. In this case, stimulation should be felt in the anterior tibialis with muscle contraction upon unloading and clearing the foot from the floor during gait.
   Preinspection: Because this patient will use FES often and for prolonged periods of time, it is critical to properly clean the area to be treated and check for skin compromise and to instruct the patient in these procedures for future applications.
   Electrode placement: Identify the motor points for optimal electrode placement over the anterior tibialis. Both electrodes should be placed over muscular tissue, avoiding placement of the distal electrode over more tendinous areas. Electrodes may be adhesive-disposable or carbon-gelled but should be proportionate to the size of the area to be stimulated. Electrodes that are too large may result in cross-stimulation of the peroneus longus and result in unwanted ankle eversion. Electrodes that are too small will increase current density and likely result in an uncomfortable sensation.
   Postinspection: Remove the electrodes and inspect the skin for any signs of skin irritation or adverse effects. If redness appears, explain that this is not uncommon and should disappear in less than 24 hours. It is prudent to reiterate that the patient should inspect the skin if getting frequent FES treatments.

duration than is required for depolarizing intact peripheral nerve and thus innervated muscle (see Chapter 9). Pulse durations of 10 milliseconds or greater may be required, and the majority of electrical stimulators on the market do not offer a pulse duration of this magnitude. DC has been used with the clinician applying the electrode to the muscle for several seconds. Modern devices offering DC cannot deliver sufficient amplitude to activate denervated muscle. If the muscle has not been activated for an extensive period (e.g., several months),

either by stimulation or volition, or if denervation occurred some time ago, the contractile mechanisms of the muscle may have fibrosed and will no longer be able to operate.[30]

The major premise underlying use of ES in denervated muscle is that ES will maintain contractile properties of the muscle while awaiting reinnervation, but the literature remains controversial. Supporters of using stimulation to denervated muscle contend that atrophy and fibrosis can be attenuated and delayed while improving recovery;[31–33] however, opponents assert that stimulation may interfere with or interrupt endogenous mechanisms for reinnervation and collateral nerve sprouting[34–39] (Box 10-4).

Electrotherapy for Modulating Pain

Using ES to modify or modulate pain may be the most common utilization of electrotherapy. Interest in the use of ES for pain modulation or electroanalgesia greatly heightened in the 1960s with Melzack and Wall's gate theory of pain control. Their theory asserted that selective stimulation of the large-diameter afferent A-beta sensory fibers can result in a gating, or blocking, of noxious afferent input from smaller-diameter unmyelinated nociceptive C fibers and small myelinated A-delta fibers at the level of the spinal cord.[33] Melzack and Wall's gating theory led to an increase in the production and use of handheld, battery-operated, TENS devices to treat pain.[40] To this day, the term *transcutaneous electrical nerve stimulation* implies the use of ES for purposes of attenuating or alleviating pain.

TENS has been used for modulating pain and has been reported in a wide variety of patient populations. Traditionally, use of TENS has been divided into applications for acute or chronic pain, although more recent evidence has shown this delineation to be inadequate.[41–43] (Use of ES for pain modulation is addressed in greater detail in Chapter 11.) The majority of studies examining the effect of TENS on pain have used portable stimulators, most of which are handheld and battery-powered despite similar waveform options on line-powered clinical units.[44] The majority of clinical applications of electrotherapy are based on pain modulation through stimulation of sensory and motor nerves. To a lesser degree, ES is used at subsensory levels.

Sensory-level stimulation, traditionally termed *conventional, sensory*, or *high-frequency TENS,* is characterized by a pulse frequency of at least 50 pps and a pulse duration of 50 to 100 μsec (Table 10-3). These parameters appear well suited for selective stimulation of the large A-beta fibers for gating pain.[45] Electrodes are placed over or adjacent to the site of pain or along the dermatome or myotome, usually in a bipolar arrangement. With high-frequency TENS, the patient will likely describe a sensation of tingling or buzzing. The gate control theory of pain is thought to underlie sensory-level TENS and acts as an ascending method of pain modulation. Sensory-level TENS is often used during activities and for prolonged periods of time, making it the most common form of TENS used.

Motor-level stimulation for electroanalgesia is based on stimulation of muscle and is commonly referred to as *low-frequency* or *acupuncture TENS*. The pulse frequency is typically 1 to 10 pps, and because a motor response is elicited, a longer pulse duration of 150 μsec or greater is common (see Table 10-3). The intensity should elicit a strong visible motor response, which is often seen as robust twitches corresponding to the frequency. Electrodes are placed over the affected area or in areas related to the pain, such as dermatomes or acupuncture points—thus the name *acupuncture TENS.* A bipolar electrode arrangement is most common. The patient's sensation should be of obvious muscle twitching and may be associated with prickling and stinging.

Box 10 ■ 4 Stimulation of Denervated Muscle: Continued Controversy

Use of ES for denervation of the muscles innervated by the facial, or seventh cranial, nerve (i.e., Bell's palsy) was once common yet controversial. Despite recent reports of modest improvements in voluntary control of facial muscles following use of high-volt pulsed current (e.g., twin-peak monophasic) up to 6 hours per day,[111] evidence for ES of denervated muscle remains sparse and with questionable design and validity. Collectively, these factors have resulted in a declining use of ES for denervated muscle over what was once seen years ago.

Bottom line: Electrical stimulation can be used to effectively activate innervated skeletal muscle for purposes of strengthening and for participation in functional activity. Although the type of stimulus parameters used for both are similar (pulse frequency, duration, waveform, etc.), the specific determination of each parameter (i.e., frequency or intensity) can vary greatly, depending on the goals. ES for denervated muscle remains controversial and unsupported.

TABLE 10–3. Parameters for Pain Modulation (TENS)*

Type	Waveform	Pulse Frequency	Pulse Duration	Amplitude	Duration
High-frequency stimulation	Mono- or biphasic pulsed current	At least 50 pps, typically 80–110 pps	50–100 μsec	mA to maximum comfortable perception (or paresthesia). No or barely visible motor activity	20–30 min (longer if used during activity)
Low-frequency stimulation	Mono- or biphasic pulsed current	Short: less than 10 pps	High: greater than 150 μsec	mA to visible muscle twitches	20–45 min
Brief intense stimulation	Mono- or biphasic pulsed current	High: approximately 100 pps	High: greater than 150 μsec	mA to visible strong muscle twitches	Less than 15 min
Hyperstimulation (noxious point stimulation)	DC or monophasic	High: 100 pps Low: 1–5 pps	Long: greater than 250 μsec, up to 1 second	mA to highest tolerated painful stimulus	30–60 sec to each area

*These parameters are not specific to traditional handheld TENS stimulators and can be repeated on many line-powered clinical stimulators.

In contrast to high-frequency TENS, low-frequency TENS is thought to act via descending methods of pain modulation by the release of endogenous opioids (e.g., endorphins and enkephalins).[46–47]

The effects of low-frequency stimulation appear to last several hours, longer than high-frequency stimulation. Because of the stronger stimulus intensity and activation of muscles, use of low-frequency TENS during activity or work is not recommended. Low-frequency stimulation is often used periodically throughout the day in 15- to 30-minute applications instead of continuously. High-frequency TENS is often used in the acute stages of recovery when motor-level stimulation with low-frequency TENS may not be indicated or tolerated. In later phases of rehabilitation, low-frequency stimulation may be used. In this manner, a clinician may use both forms of TENS on the same patient.

A method of ES combining both high- and low-frequency TENS is known as *brief-intense TENS*. The stimulus is increased to a strong sensation, resulting in marked fasciculations or tetanic nonrhythmic muscle contraction. Brief-intense TENS is applied similarly to low-frequency TENS in periodic and brief applications but usually not greater than 15 minutes (see Table 10-3). This type of stimulation can be uncomfortable for the patient and is a form of noxious-level stimulation thought to elicit the release of endogenous opioids.

Hyperstimulation, another form of noxious-level stimulation, is usually applied locally via probe electrodes or small electrodes and uses monophasic currents with long pulse durations approaching 1 second or direct current. Either a low pulse frequency (1 to 5 pps) or high pulse frequency (100 pps) is used. Hyperstimulation is often applied over acupuncture points or dermatomal distributions of a peripheral nerve but not over areas of motor nerve where a strong motor response would be elicited.[44] Hyperstimulation is thought to lessen pain through descending methods via release of endogenous opioids. Collectively, acupuncture (low-frequency TENS), brief-intense, and hyperstimulation TENS are considered to work via descending opiate-mediated electroanalgesia.

Current waveforms used for pain modulation differ but are generally characterized by variations in two basic parameters—pulse frequency and duration. Pulsed currents, including monophasic waveforms such as twin-peaked monophasic (e.g., high volt), symmetrical and asymmetrical, balanced and unbalanced, and biphasic currents as well as amplitude-modulated AC (e.g., interferential current) are commonly used. With TENS, there are no on-times or off-times or ramp-up or ramp-down controls, as with NMES or FES. The parameters manipulated for TENS are not specific to handheld units and are found on many line-powered clinical units, but, by convention, many clinicians would not consider these TENS units. Traditionally, a TENS stimulator could be identified by analog controls or dials for pulse duration and frequency with amplitude dials for each of two channels. The dial for pulse duration often ranged

from 50 to 250 μsec while frequency ranged from less than 10 pps up to 200 pps. The last two decades have shown an increase in digital units with preprogrammed settings and clinical protocols designated for pain of a particular type or region (e.g., neck, shoulder, knee). Some newer handheld TENS units no longer allow the clinician to manipulate the specific parameters apart from selecting a preprogrammed protocol and the treatment intensity.

Key Point! The term *TENS* has often been inaccurately designated to imply use of a handheld stimulator for pain modulation. Many line-powered stimulators offer the same stimulus parameters and waveforms as handheld devices and thus are well suited for TENS. These units are just not as portable as the handheld stimulators.

Common to many TENS units are options to select burst and modulated TENS (Fig. 10-16). The burst function of most TENS units delivers the selected current in brief, intermittent periods of stimulation or packages of pulses (i.e., bursts). Modulated TENS varies per manufacturer, but in general, *modulation* implies that the specific waveform parameters (usually pulse duration and frequency) are periodically altered or modulated by the internal circuitry of the device. Modulation is used to prevent or lessen the chances of developing accommodation to the electrical stimulus, thus rendering the stimulation less effective. A patient experiencing a modulated waveform is likely to describe the sensation as changing, moving, waving, or increasing and decreasing.

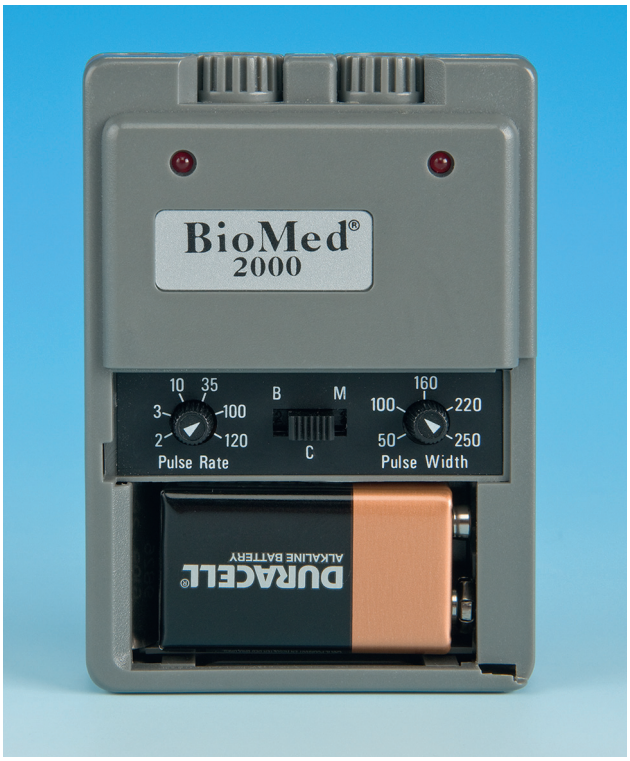

Fig 10 ■ 16 A typical handheld TENS unit with controls for pulse duration, frequency, and options for modulating the current output.

Electrotherapy for Preventing or Reducing Edema

Edema, or swelling, from soft tissue injury or trauma can hinder tissue repair and can lead to pain, reduced mobility, and delayed return to maximum possible function.[48–50] Edema at the knee and ankle has been clearly associated with motor inhibition and decreased excitability, which further reduces function and prolongs rehabilitation.[37,51] Increased capillary permeability and leakage of plasma proteins, leukocytes, and water into the interstitial space following soft tissue injury results in localized swelling.[48] The effects of ES on vascular permeability in rats have suggested that cathodal high-voltage pulsed current (i.e., twin-peaked monophasic current) at 120 pps frequency and 10% below the motor threshold may be best at limiting edema formation in the acute posttraumatic period when vessel permeability is increased.[52–60] Evidence suggests that although four 30-minute applications 30 to 60 minutes apart resulted in significant edema reduction,[56,58] continuous applications of 3 to 4 hours are more effective.[52,54,55,57,60]

What appears clear is that cathodal high-voltage electrical stimulation is an effective means of limiting the onset of swelling but only during the acute period, when vascular permeability is increased. After leakage has occurred and localized swelling has been produced, management of swelling becomes more challenging. This is frequently the case, as patients are often not seen for treatment for days or weeks after their injury.

Once edema has accumulated and become chronic, management must focus on clearing the swelling, rather than preventing it. Evidence for management of chronic edema is limited to a few studies, mostly given to chronic hand edema, and all used electrically stimulated muscle contractions (e.g., NMES). It is thought that repetitive contractions of the muscles compress venous

and lymphatic vessels, helping to reduce swelling while increasing venous and lymphatic return.[61,62]

The waveform most supported for edema management in the acute phase is high-volt monophasic pulsed current (e.g., twin-peak monophasic), but when using muscle activation, other waveforms suitable to motor activation can be used (Table 10-4). High-volt pulsed current is often applied with the active or treatment electrodes placed in the immediate area of injury. In many cases, this may be directly over a peripheral joint or soft tissue region, with the electrodes bracketing the joint and the dispersive pad placed nearby (a monopolar arrangement; Fig. 10-17). The size of the electrodes should reflect the size of the joint or area. Thus, larger electrodes are used at the knee than at the wrist. The clinician should designate the active electrodes placed over the swollen area as the cathode. In the acute stages, where motor activation is not desired, a pulse frequency of 100 to 125 pps is common, with an intensity eliciting a perceptible sensory response but below the motor threshold. If using high-volt pulsed current (e.g., twin-peak monophasic), the twin-phase pulse duration is most likely fixed at 2 to 100 μsec.

There has remained some clinical opinion that changing the polarity of the active or treatment electrodes halfway through a treatment session from negative (cathodal) to positive (anodal) provides some additional benefit over continual negative stimulation. This practice has never been supported by the literature; thus, continuous use of negative stimulation over the acutely swollen area remains recommended.

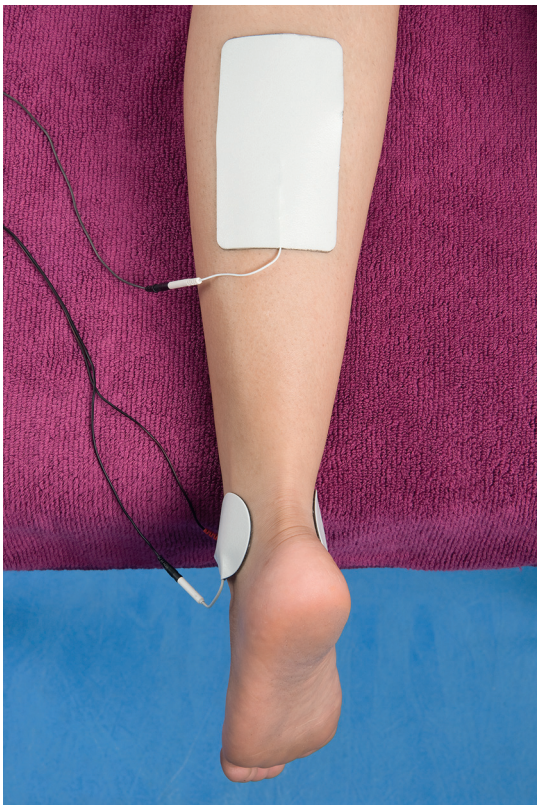

Fig 10 ■ 17 Use of electrical stimulation to reduce swelling. Cathodal stimulation with high-volt pulsed current is used in the acute stages. Muscle stimulation for pumping is used after the acute stage.

Treatment duration in the acute phase can range from several minutes to hours, but additional precaution should be taken if applying stimulation consistently

TABLE 10–4. Parameters for Edema Management

Indication	Type	Waveform	Pulse Frequency	Pulse Duration	Amplitude	Duration
Acute (within 24–72 hours)	Sensory-level stimulation	Monophasic pulsed (e.g., twin peak)	100–125 pps	2–100 μsec	mA to comfortable sensory perception (approximately 10% below motor threshold)	20–45 min to several hours
Existing edema (subacute or chronic)	Motor-level stimulation (e.g., NMES)	Biphasic PC or burst-modulated AC (e.g., Russian)	20–80 pps or bursts per sec	100–600 μsec if PC	mA to tetanic contraction	1:1 on:off ratio (e.g., 10–20 min of rhythmic contractions at 3 sec on, 3 sec off)

CASE STUDY 10-4 TENS

A patient involved in a motor vehicle accident 2 days ago presents with soft tissue injury to the posterior cervical spine and the upper thoracic region on the right side. Active and resisted ROM are painful and limited. Palpation is more revealing of tenderness in the right upper trapezius.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of electrotherapy?
   ANSWER: Yes, the patient demonstrates decreased ROM, pain, and tenderness to palpation of the upper trapezius following a motor vehicle accident.

2. Is the patient appropriate for electrotherapy (i.e., do any of the general precautions or contraindications to electrotherapy apply, or are there any specific considerations regarding application of electrotherapy to this patient)?
   ANSWER: Given that there are no contraindications or precautions, this patient is appropriate for the use of electrical stimulation.

3. What are the specific goals to be achieved with the use of electrotherapy?
   ANSWER: To decrease the acute pain associated with soft tissue injury stemming from the accident.

4. Do you have the specific type of electrotherapy that is appropriate for the patient?
   ANSWER: TENS can be used to address acute musculoskeletal pain. Because the patient demonstrates increased pain with resisted testing of the paracervical muscles, high-frequency TENS is indicated.

5. Can you select the specific parameters of electrotherapy that are appropriate for the patient?
   ANSWER:
   Waveform: Mono- or biphasic pulsed current may be used.
   Pulse duration: 50–100 μsec secondary to acute pain and pain with resisted ROM
   Pulse frequency: 50–110 pps (high frequency) for acute musculoskeletal pain
   Intensity: To maximal but still comfortable perception of tingling without tetanic contraction. Notice of a weak motor or muscle response is okay with high-frequency TENS.
   Duration: 20 to 30 min or longer as needed

6. What are the proper application procedures for electrotherapy?
   ANSWER:
   Instruct the patient: Inform the patient of the purpose and procedure. Be sure to explain the anticipated sensation and effect of the stimulation. In this case, a perceivable yet comfortable sensation of stimulation should be noted in the muscles of the paracervical spine.
   Preinspection: Clean the area to be treated and check for skin compromise. If excessive hair is present in the area, shaving it off may enhance the delivery of the stimulation.
   Electrode placement: Two electrodes in a bipolar configuration are placed over the right upper trapezius.
   Patient position: The patient may be supine or prone with head resting on pillows or may be sitting upright.
   Postinspection: Remove the electrodes and inspect the skin for any signs of skin irritation or adverse effects. If redness appears, explain that this is not uncommon and should disappear in less than 24 hours.

Six weeks later, the patient demonstrates the ability to perform active and resisted ROM but remains symptomatic in the right upper trapezius. The clinician now opts to use low-frequency or acupuncture TENS. The same waveform and device may be used, but now the pulse duration is increased to 250 μsec while the pulse frequency is reduced to 10 pps. The electrodes are placed over the muscle tissue of the right upper trapezius, and the intensity is increased to elicit visible muscle contraction of the muscle up to patient tolerance.

for several hours secondary to the electrochemical effects that can occur at the anode and cathode when using monophasic current. When using stimulated muscle activation for reducing edema in the postacute and chronic stages, the waveform selected must be suitable for stimulating muscle. Symmetrical or asymmetrical biphasic pulsed currents (e.g., square, rectangular, or triangular waveforms) and burst-modulated AC (e.g., Russian) are commonly used. Because muscle activation is desired, electrodes must be placed over muscle tissue of the swollen joint. The bracketing of joints used in the acute stage would not allow for stimulation of muscle, because little muscle exists directly over joints. The specific parameters should allow for rhythmic contraction

and relaxation of the muscle (e.g., 10-second contraction followed by 10-second rest). Pulse frequency may range from 20 to 80 pps and pulse duration from 100 to 600 μsec with an intensity that is capable of eliciting rhythmic tetanic contractions. Typical treatment sessions of muscle pumping last from 10 to 20 minutes. Stimulation beyond 10 to 20 minutes may increase blood flow to the area and provoke further swelling.

In the acute and, particularly subacute periods, following injury, the local vasculature may remain weakened and prone to further leakage. It is possible that repeated muscle contractions via NMES may result in increased local blood flow and may overload weakened local vasculature. Therefore, use of NMES for managing edema is recommended only when swelling has stabilized or does not increase following use of motor-level stimulation.

CASE STUDY 10-5 Using Electrical Stimulation for Management of Edema

A patient presents to the clinic the morning after sustaining an ankle sprain during a lacrosse match. Acute swelling of the left lateral ankle and laxity in ligaments of the lateral ankle are noted. ROM is decreased secondary to discomfort and swelling.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of electrotherapy?
   ANSWER: Yes, the patient has acute swelling, pain, and decreased ROM following musculoskeletal injury to the ankle.

2. Is the patient appropriate for electrotherapy (i.e., do any of the general precautions or contraindications to electrotherapy apply, or are there any specific considerations regarding application of electrotherapy to this patient)?
   ANSWER: The patient does not present with any contraindications or precautions.

3. What are the specific goals to be achieved with the use of electrotherapy?
   ANSWER: Reduction of acute swelling to reduce pain and permit ROM exercises as indicated in the therapy plan.

4. Do you have the specific type of electrotherapy that is appropriate for the patient?
   ANSWER: High-volt pulsed current (i.e., twin-peak monophasic) is selected, as it may assist in reducing and preventing further swelling.

5. Can you select the specific parameters of electrotherapy that are appropriate for the patient?
   ANSWER:
   Pulse duration: 100 μsec
   Pulse frequency: Approximately 100 pps
   Intensity: Increase intensity until a small but visible motor response is elicited and then reduce the intensity to a level approximately 10% below the motor threshold.
   Duration: 15 to 30 minutes.

6. What are the proper application procedures for electrotherapy?
   ANSWER:
   Instruct the patient: Inform the patient of the purpose and procedure. Be sure to explain the anticipated sensation and effect of the stimulation. In this case, a sensation of buzzing or strong tingling should be noted at the ankle. Depending on the amount of swelling at each malleoli, the patient may have more or less sensation of the electrical stimulus.
   Preinspection: Clean the area to be treated and check for skin compromise. Observe any bony areas where appropriate electrode contact may be a challenge.
   Electrode placement: Electrodes are placed on the lateral and medial aspect of the ankle bracketing the joint. A dispersive pad is placed on the posterior thigh to complete the circuit. The treatment pads are designated as the cathode and the reference as the anode.
   Patient position: Because the intervention is addressing swelling, the patient should be positioned with the ankle elevated above the torso. Supine positioning with the ankle elevated by pillows or a bolster is recommended.
   Postinspection: Remove the electrodes and inspect the skin for any signs of skin irritation or adverse effects. If redness appears, explain that this is not uncommon and should disappear in less than 24 hours.

Electrotherapy for Increasing Circulation

ES is often used to increase blood flow in superficial and deep tissues. This can be accomplished using various types of ES, but all generally fall into either sensory or motor-level stimulation (Table 10-5). Local increase in skin blood flow occurs by vasodilation of cutaneous vessels;[63,64] low-frequency TENS at a sensory level (e.g., 4 pps at 250 μsec) appears to be more effective than high-frequency TENS (e.g., 100 pps at 100 μsec).[65] Deeper arterial blood flow does not appear to increase with sensory-level stimulation, but it does with motor-level stimulation using rhythmic muscle contractions.[66–69] Miller et al[66] reported that the increased blood flow following motor-level electrical stimulation resulted in a longer-lasting increase in flow than was measured following voluntary muscular contraction alone.

To promote superficial local blood flow via cutaneous vasodilation, sensory-level stimulation is used with electrodes placed paraspinally, over peripheral nerves, acupuncture points, dermatomes, myotomes, or other tissue areas where increased blood flow is desired. Because low-frequency TENS appears most effective, a stimulus with a pulse frequency between 4 and 100 pps and a pulse duration of 4 to 600 μsec at a sensory-level intensity is recommended. To promote deeper blood flow via arterial or venous flow, ES capable of eliciting a motor-level response at least 10% of the maximal voluntary contraction is required. Stimulation at a frequency of 20 to 80 pps, a pulse duration of 100 to 600 μsec, and 5- to 10-second on-time and 5- to 10-second off-time is recommended. Electrodes should be placed over muscle of the area where increased blood flow is desired. Recommended treatment time for both sensory- and motor-level stimulation is 15 to 30 minutes. Because of the resistance to blood flow created by muscle contraction, sustained contractions without rest intervals are not recommended. The rhythmic effect of muscular contraction with intermittent relaxation may assist in venous blood flow and prevention of deep-vein thrombosis in the lower extremities during period of immobilization, when voluntary motor activity is impaired, or following surgery[70] (see Table 10-5).

Electrotherapy for Promoting Tissue Healing

Use of ES for healing of chronic wounds is well supported in the peer-reviewed literature. In a meta-analysis of ES helping to heal chronic wounds, Gardner et al[71] concluded that wounds treated with ES showed a 144% greater healing rate when compared to the normal rate of healing; the greatest effect was noted in pressure ulcers. Use of ES for wound healing is based on the transepithelial potential (TEP) and injury current, which was presented in Chapter 9. Wounds that fail to heal properly appear to have lost the injury current and are unable to restore the TEP. The application of exogenous or clinical ES is thought to replace or augment the natural or endogenous current associated with healing.

TABLE 10–5. Parameters for Increasing Peripheral Blood Flow

Indication	Type	Waveform	Pulse Frequency	Pulse Duration	Amplitude	Duration
Vasospastic disorders	Sensory-level stimulation	Mono- or biphasic pulsed current	4–100 pps	4–600 μsec	mA to comfortable sensory perception	15–30 min
	Motor-level stimulation	Mono- or biphasic pulsed current	1–5 pps	200–600 μsec	mA to visible muscle twitches	15–30 min
Diminished arterial blood flow	Motor-level stimulation (e.g., NMES)	Biphasic pulsed current or burst-modulated AC (e.g., Russian)	20–80 pps or bursts per sec	100–600 μsec	mA to robust tetanic muscle contractions	1:1 on:off ratio (e.g., 10–15 min of rhythmic contractions)

The benefit of ES for chronic wounds appears greatest in recalcitrant stage III and IV ulcers. Reimbursement standards support use in these conditions. The Centers for Medicare and Medicaid Services recognize ES for treating wounds only if the wound is a chronic stage III or IV ulcer and there have been no measurable signs of healing for at least 30 days of treatment with standard wound care. Therefore, documentation of the stage of the ulcer, duration of standard care, and any responses to treatment (e.g., wound size, presence of granulation tissue, and epithelialization) is essential for reimbursement of services.

There are a variety of cellular and tissue responses to ES. It appears that cells associated with tissue healing, such as neutrophils, macrophages, and lymphocytes, are charged and are attracted to migrate toward the opposite charge when exposed to an electrical field.[72,73] This migration of cells to a specific pole of an electrical field is termed *galvanotaxis*. Intracellular influx of calcium increases when cells are exposed to electrical fields, increasing activity of cytoskeletal elements, such as actin microfilaments, that underlie the galvanotaxic properties exhibited by some cells.[72,73]

CASE STUDY 10-6 Increasing Blood Flow

A patient with diabetic neuropathy and decreased sensation in the plantar aspect of both feet is referred for exercise and intervention. The clinician wants to increase arterial circulation in the feet to decrease the risk of ulceration.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of electrotherapy?

ANSWER: Yes, the patient has diabetes-related peripheral neuropathy with decreased sensation of the plantar aspects of both feet.

2. Is the patient appropriate for electrotherapy (i.e., do any of the general precautions or contraindications to electrotherapy apply, or are there any specific considerations regarding application of electrotherapy to this patient)?

ANSWER: The patient has decreased sensation in the intended treatment area. Decreased sensation is a precaution for electrotherapy, so the clinician must carefully monitor the treatment both prior to and after stimulation.

3. What are the specific goals to be achieved with the use of electrotherapy?

ANSWER: To increase local blood flow in the plantar aspect of the feet to facilitate improved sensation.

4. Do you have the specific type of electrotherapy that is appropriate for the patient?

ANSWER: Motor-level stimulation is indicated to increase local blood flow. Waveforms and parameters used for eliciting motor-level stimulation may be used. The stimulation should produce rhythmic muscle twitches in the plantar intrinsic muscles.

5. Can you select the specific parameters of electrotherapy that are appropriate for the patient?

ANSWER:

Waveform: Mono- or biphasic pulsed current may be used.

Pulse duration: 300 μsec—suitable for motor-level stimulation

Pulse frequency: 5 pps for low-frequency motor stimulation

Intensity: Until small but visible muscle twitches are produced in the plantar muscle region

Duration: 15 to 30 min

6. What are the proper application procedures for electrotherapy?

ANSWER:

Instruct the patient: Inform the patient of the purpose and procedure. Be sure to explain the anticipated sensation and effect of the stimulation. Decreased sensation in the treatment area may limit the patient's perception of the stimulation. Careful monitoring by the clinician is required to ensure proper administration of the stimulation.

Preinspection: Clean the area to be treated and check for skin compromise and possible ulceration. Excessive callus formation on the plantar areas may increase resistance to the stimulation.

Electrode placement: Two electrodes in a bipolar configuration are placed over the plantar aspect of each foot.

Patient position: The patient will be placed supine on a treatment table.

Postinspection: Remove the electrodes and inspect the skin for any signs of skin irritation or adverse effects.

Other specific cell activities are noted in response to ES. The large negatively charged surface membrane proteins of the fluid-mosaic phospholipid cell wall show movement to the side of the cell facing the anode. Certain cells, such as endothelial cells, elongate and orient themselves to the field lines created by ES. Cell adhesion molecules, such as fibronectin, show increased binding of cells within the extracellular matrix. Intracellular second messenger systems, such as cyclic AMP and G-proteins, show increased activity. Additionally, protein kinase activity is increased, resulting in increased expression of epithelial and fibroblastic growth factors.[72,73]

An understanding of the effects of ES on tissue healing has come from studies using calcium-channel antagonists (e.g., lanthanum) or blockers (e.g., nitrendipine or verapamil). These drugs can slow or even stop increased cell activity observed in response to an electrical field.[72,73]

High-volt pulsed current (e.g., twin-peak monophasic) remains the most commonly used and supported current for tissue healing. The current may be applied directly into the wound or in the region around it. Prior to application, the specific polarity to be used must be determined and is termed the *active* or *treatment electrode.* In general, the cathode is used first in the early period of wound inflammation and infection. The anode is then used for debridement (e.g., phagocytosis and autolysis) and for promotion of epithelialization during the later proliferation stage of healing. The other electrode (dispersive or inactive electrode), and thus pole, is placed nearby on the skin (approximately 15 to 30 cm from the wound) in a monopolar arrangement. If stimulation is to be introduced directly into the wound, the active electrode should be placed in a saline-moistened sterile gauze and placed into or onto the wound. (Keep in mind that distilled water will not conduct, so saline is best.) If the stimulus is to be delivered near the wound, the electrodes are placed on either side of it so current will travel through the wound (Fig. 10-18).

A pulse frequency of approximately 100 to 125 pps at a sensory-level intensity is recommended. Pulse duration is usually maintained on most high-volt pulsed devices at 2 to 100 μsec. Low-intensity direct current (e.g., microcurrent) is also used and by definition is below the sensory threshold. Like high-volt pulsed monophasic current, the anode and cathode of DC are used for their specific properties. In patients with compromised sensation in the wound area, a trial application of the stimulus over an area of intact sensation can be used to gauge patient comfort and tolerance. Daily treatments of 45 to 60 minutes are recommended (Table 10-6). Further discussion of ES for tissue healing is presented in Chapter 15. Because current flow will be greatest directly between the electrodes, the electrode placement sites should be rotated around the wound edge approximately 45 to 90 degrees on subsequent applications to ensure the wound is fully exposed to the stimulation. Failure to do so may result in the wound closing on only one side.

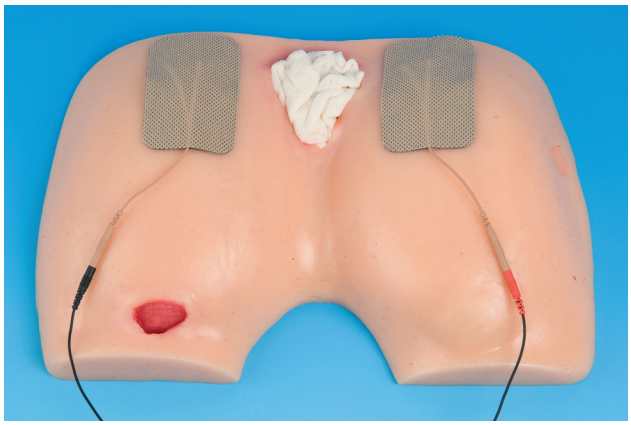

Fig 10 ■ 18 Use of electrical stimulation for wound healing. Electrodes may be placed on either side of the wound. The clinician should rotate the dispersive electrode or the pair of electrodes 30 to 90 degrees on subsequent treatments to fully expose all edges of the wound to the current.

Biofeedback

EMG biofeedback differs from the previously described uses of ES in that no current is actually delivered to the patient. In contrast, EMG biofeedback involves the assessment and recording of skeletal muscle activity so that the practitioner and patient can use the information to alter future muscle activity, whether that be to increase or decrease movement.[74] Improvement in function and decrease in pain are still the primary goals with EMG biofeedback, despite the differences with other forms of electrotherapy. Oftentimes, EMG biofeedback is not included in the same category as other electrotherapeutic applications where current is applied to the patient, but this can be used to enhance volitional muscle activation, thereby providing therapeutic benefit. More details and clinical applications of EMG biofeedback are presented in Chapter 13.

TABLE 10–6. Parameters for Enhancing Tissue Healing

Indication	Type	Waveform	Pulse Frequency	Pulse Duration	Amplitude	Duration
Epithelialization, autolysis, and reactivation of inflammatory process	Sensory-level stimulation; anode placed at wound	Monophasic pulsed current (e.g., twin-peak monophasic)	100 pps	4–100 μsec	mA to comfortable sensory response	60 min, 5–7 days per week
Promotion of granulation of wound	Sensory-level stimulation; cathode placed at wound	Monophasic pulsed current (e.g., twin-peak monophasic)	100 pps	4–100 μsec	mA to comfortable sensory response	60 min, 5–7 days per week
Bactericidal effect for infected wound	Sensory-level stimulation; cathode or cathode followed by anode placed at wound	Monophasic pulsed current (e.g., twin-peak monophasic)	100 pps	4–100 μsec	mA to comfortable sensory response	30–60 min of cathodal stimulation or 20 min of cathodal stimulation followed by 40 min of anodal stimulation

CASE STUDY 10-7 Tissue Healing

A 49-year-old man with diabetes is referred with a stage IV recalcitrant pressure ulceration at the lateral lower leg. There have been no observable signs of tissue healing for the last 30 days of standard wound care. The clinician wishes to facilitate granulation in the wound area.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of electrotherapy?
ANSWER: Yes, the patient has a stage IV recalcitrant pressure ulceration at the posterior lower leg.

2. Is the patient appropriate for electrotherapy (i.e., do any of the general precautions or contraindications to electrotherapy apply, or are there any specific considerations regarding application of electrotherapy to this patient)?
ANSWER: Diabetes may be associated with decreased sensation in the treatment area. Assessment of sensation is recommended prior to stimulation, and close monitoring of treatment is warranted if compromised sensation is noted.

3. What are the specific goals to be achieved with the use of electrotherapy?
ANSWER: To promote and encourage granulation of the wound bed.

4. Do you have the specific type of electrotherapy that is appropriate for the patient?
ANSWER: High-volt pulsed current (i.e., twin-peak monophasic current) may be used, as this monophasic waveform can deliver cathodal and anodal stimulation.

5. Can you select the specific parameters of electrotherapy that are appropriate for the patient?
ANSWER:
Waveform: Monophasic (high-volt) pulsed current
Pulse duration: 100 μsec
Pulse frequency: 100 pps
Intensity: To produce a mild sensation
Duration: 60 min
Frequency: 5–7 days per week

CASE STUDY 10-7 Tissue Healing—cont'd

6. What are the proper application procedures for electrotherapy?

ANSWER:

Instruct the patient: Inform the patient of the purpose and procedure. Be sure to explain the anticipated sensation and effect of the stimulation. Decreased sensation in the treatment area may limit the patient's perception of the stimulation.

Preinspection: The ulceration represents an area of decreased resistance to current and should be inspected prior to stimulation.

Electrode placement: The treatment electrode is placed within a saline-moistened sterile gauze pad placed directly within the ulceration, and the reference or inactive electrode is placed adjacent to the ulceration. The treatment electrode is designated as the cathode and the reference the anode.

Patient position: The patient will be placed supine or side-lying on the opposite side.

Postinspection: Remove the electrodes and inspect the ulceration and adjacent skin for any signs of adverse effect.

IONTOPHORESIS

Iontophoresis is a technique in which current is used to induce the transcutaneous movement of ions across the skin into target tissues. Clinical use of iontophoresis is based on the fundamental concept that like charges repel and opposites attract. An ion is an atom that has lost or gained electrons and thus becomes positively or negatively charged. To mobilize or deliver negatively charged ions into the treatment area, an electrode containing the negative ions attached to the cathode of an electrical circuit is placed over the treatment area. For a positively charged ion, the anode is used. For the majority of clinical applications, iontophoresis implies the delivery of a medicinal ion for therapeutic benefit. The electrode containing the medicinal ion is termed the *treatment, active,* or *delivery electrode,* and the other electrode is termed the *inactive, reference,* or *dispersive electrode.* Note that either the anode or cathode can be used as the treatment electrode, depending on the polarity of the ions being delivered.

Current is delivered to the electrodes via a device. Most devices used for iontophoresis use DC and are small and portable and battery-operated. The DC provides a unidirectional electric current to induce ion movement. There are some devices that use alternating current, although evidence for their use is limited.[75,76] It has been proposed that the alternating nature of AC may increase the permeability of the tissue, facilitating the passage of the ions.[77] Still, some units using AC for iontophoresis do so using AC with a "DC offset" that essentially mimics the properties of DC that induce ion movement.[78]

Iontophoresis has been used for many conditions, including soft tissue inflammatory conditions,[79,80] neuralgia,[81] edema,[82] ischemic skin ulcers,[83] hyperhidrosis,[84,85] plantar warts,[86] gouty arthritis,[87] calcific tendonitis,[88] scar tissue,[89] Peyronie's disease,[89–91] and other disorders of connective tissue. For most of these uses, the evidence is generally favorable.[92] The most studied use of iontophoresis is of corticosteroids, such as dexamethasone, which is used to reduce inflammation of local soft tissues.

Physiology of Iontophoresis

Underlying the process of iontophoresis is the electrical repulsion of ions. Using ES to move charged ions into the target tissues has long been the cornerstone explanation for iontophoresis and is termed *electromigration*. However, more recent explanations of the physiological mechanisms underlying iontophoresis have been described.[93]

Electroporation is an increase in the porosity of the superficial skin in response to ES and may facilitate movement of ions into the tissues.[93–96] Skin is naturally hydrophobic, so it presents a barrier to the transcutaneous movement of ions in a solution.[97] Following ES, a temporary increase in the skin's porosity allows ions to more easily penetrate the tissues. The exact mechanisms by which electroporation occurs are not known, but it is clear that cell organization and function of skin are altered in response to ES.[94–96]

Another mechanism that is used to explain the migration of ions into the tissues is based on "volume flow," or the bulk movement of solute in response to an electric field. Not to be confused with simple diffusion, this movement is in response to an applied electrical field and not concentration gradients.[93] When electrodes are applied to the skin, ions of positive charge within the

extracellular fluid—that is, sodium (Na^+)—are attracted to the cathode, whereas negatively charged ions such as chloride (Cl^-) are attracted to the anode. The bulk movement of solute such as Na^+ and Cl^- in response to the electrical field is thought to provide a mechanism by which ions are moved into the tissues. This process is termed *electroosmosis,* or *ionohydrokinesis*[93,98] (Fig. 10-19).

Electroosmotic flow occurs in the same direction as flow of counterions (ions of charge opposite the skin). Because human skin maintains a net negative charge, the direction of electroosmotic flow is from the anode to the cathode.[93] For positively charged ions delivered from the anode, electroosmotic flow may assist in transdermal delivery, but in contrast, electroosmotic flow may hinder delivery of negatively charged ions from the cathode.[93] This opposition to delivery of negative ions from the cathode is increased only when using larger current amplitudes, as the increased force of the electrical field will necessarily increase the ionic movement.

When considering the collective effects of electromigration, electroporation, and electroosmosis, there is reason to consider using smaller-amplitude current to deliver negative ions to minimize the counterflowing electroosmosis. The "wear-home" iontophoresis systems with self-contained electrocircuitry use this principle by delivering a smaller-amplitude current over a longer period of time versus in-clinic systems. Likewise, the effect of electroosmosis and counterion flow makes anodal delivery of negatively charged ions or neutral drug molecules such as glucose a future consideration. This use of anodal delivery for negatively charged ions has been termed *wrong-way iontophoresis* but reflects the idea that delivery of larger, negatively charged ions may be more effective when delivered secondary to properties of electroporation and electroosmosis.[93]

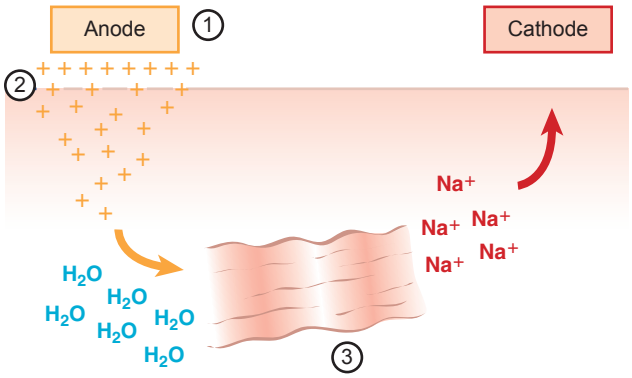

Fig 10 ■ 19 Movement of ions into tissue by three mechanisms: (1) electrical repulsion of like charges; (2) electroporation, increasing openings in the skin surface for ions; and (3) electroosmosis or movement of water and sodium (Na^+) toward the cathode, creating a stream by which ions are moved.

Application of Iontophoresis

There are two important aspects of iontophoresis: (1) knowing the polarity of the ion or drug to be used and (2) having good conductivity so there is not a chemical burn under the electrodes. If the incorrect pole is used during the procedure, there is little to no chance the ions will make it into the desired tissue.

Selecting an ion with a known polarity may seem obvious, but at one time there was considerable controversy over the polarity of dexamethasone, the most commonly used drug for iontophoresis. Dexamethasone was once considered to be positively charged, and many textbooks and guidelines for iontophoresis instructed in using the anode to deliver dexamethasone. It was not until 1992 that dexamethasone was shown to be best phoresed from the cathode. This is now the standard procedure for using dexamethasone.[101]

Selecting an Ion

The primary determinant in selecting an ion or drug for iontophoresis is whether the ion has a therapeutic effect on the condition being treated. For example, if treating an inflamed tendon with the goal of reducing inflammation and pain, an ion with anti-inflammatory capability is recommended. Because iontophoresis is a procedure in which ions are transferred across the skin, it is critical to specify what ion or medicine is to be phoresed. Several pharmacological and nonpharmacological ions are used in therapeutic iontophoresis (Table 10-7). The most common of these is dexamethasone sodium phosphate. Because of the widespread use of this drug, the term *iontophoresis* has conventionally come to imply the use of dexamethasone, although this does not accurately reflect the procedure of iontophoresis. Therefore, it is recommended that use of the term *iontophoresis* be clarified by specifying what type of ion or medication was used. In summary, when performing iontophoresis, you should always answer the question, "Phoresis of what?"

Effective iontophoresis is predicated on several factors, one of which is the concentration of the ion or drug used. It is advised that drugs for iontophoresis be

TABLE 10–7. Indications and Drugs Commonly Used With Iontophoresis

Indication	Drug	Solution	Delivery Electrode Polarity	Effect
Inflammation	Dexamethasone	4 mg/mL in aqueous solution	Negative	Anti-inflammatory
Calcific tendonitis, myositis ossificans	Acetic acid	2%–5% aqueous solution	Negative	Believed to increase solubility of calcium deposits
Adhesive capsulitis and other soft tissue adhesions	Iodine	5%–10% solution or ointment	Negative	Sclerolytic effects
Soft tissue pain and inflammation	Lidocaine	4%–5% solution or ointment	Positive	Local anesthetic effects
Muscle and joint pain	Salicylates	10% trolamine salicylate ointment or 2%–3% sodium salicylate solution	Negative	Analgesic and anti-inflammatory
Local subacute or chronic edema	Hyaluronidase	Reconstitute with 0.9% sodium chloride to provide a 150-mg/ml solution	Positive	Dispersion of local edema
Skeletal muscle spasms	Calcium chloride	2% aqueous solution	Positive	Decreased excitability of peripheral nerves and skeletal muscle
Skeletal muscle spasms, myositis	Magnesium sulfate	2% aqueous solution or ointment	Positive	Muscle relaxant
Skin ulcers	Zinc oxide	20% ointment	Positive	Acts as a general antiseptic and may increase tissue healing
Hyperhidrosis	Tap water	N/A	Alternating polarity	Decreased sweating of palms, feet, or axillae

Source: Ciccone, CD. *Pharmacology in Rehabilitation.* 5th ed. Philadelphia: FA Davis; 2016, p 658.

obtained from a licensed pharmacist to ensure proper concentrations and storage.

Electrode Selection and Placement

Current density is the amount of current per unit of the electrode's conductive surface area and is calculated as:

$$\text{Current density (CD)} = \frac{\text{current amplitude (mA)}}{\text{conductive surface area of the electrode (cm}^2\text{)}}$$

It is critical to note that an electrode's size and its conductive surface area are not the same. Most commercially manufactured electrodes for iontophoresis have a region of adhesive material extending beyond the edges of the conductive surface area (Fig. 10-20). This renders the conductive surface area smaller than the apparent electrode size. A maximal current density of 0.5 mA/cm^2 (3.3 mA/in.^{2}) at the cathode and 1 mA/cm^2 (6.6 mA/in.^{2}) at the anode have been recommended as safe.[98,102,103] Caustic damage to the tissue at the cathode caused by formation of sodium hydroxide is of great concern. Therefore, it is recommended that the cathode's electroconductive surface area exceed that of the anode, even up to twice the area.

When using cathodal stimulation to the smaller active electrode, it is recommended that the current amplitude be monitored to minimize risk of tissue damage. Most

Fig 10 ■ 20 The electroconductive surface of most electrodes used for iontophoresis is less than the apparent size of the electrode.

commercially manufactured iontophoresis electrodes have a conductive surface area that allows a current density within acceptable ranges for most iontophoresis devices, with a maximal current output of 4 to 5 mA. Many of the commercially prepared electrodes include chemical buffers within the electrode to minimize the chance of electrical burn. Although the process of iontophoresis can theoretically be administered using any type of electrode placed over a porous material containing the ions (e.g., a sponge or towel), for reasons of safety, commercially prepared electrodes should be used.

The placement of electrodes for iontophoresis is directly dependent on the site being treated. The active electrode delivering the therapeutic ion is placed over the desired treatment area. In many cases, palpation to identify the most painful area will reveal the local area best suited for application. Prior to application, inspect the skin for color and integrity and record this in the patient's treatment record. Repeat this upon removing the electrodes to determine the skin's tolerance of the current. The inactive electrode is placed at a site distant from the treatment electrode, but some consideration should be given to the distance between electrodes (i.e., the interelectrode distance). The closer the electrodes, the more superficial will be the current and potential movement of ions. A greater interelectrode distance will facilitate greater depth of current penetration and potentially ion delivery.

Dosage and the Iontophoretic Equation

Because iontophoresis usually involves delivery of a drug, it is necessary to specify the dose that is delivered. The iontophoretic equation is used to calculate the dose and is based on the current amplitude of the DC current and the duration the current is delivered:

$$\text{Dosage (mA min)} = \text{Current (mA)} \times \text{Duration (min)}$$

Typical clinical dosages range from 20 to 80 mA min with maximal dosages reaching 160 mA min. As of yet, there has been no clearly defined dosage that is more effective than another and, in many cases, is decided upon by patient response and tolerance.[75] Current amplitude with clinical iontophoresis at dosages of 20 to 80 mA min typically range from 0.1 to 4 mA, depending on patient tolerance and the duration current flows to obtain the desired dose. Duration is variable and has been a source of discussion. There remains disagreement as to the optimal dose and specific parameters used to obtain it. Some evidence has suggested that longer duration with lower amplitude facilitates greater depth of delivery.[104]

To date, there remains no universal agreement as to whether the process of iontophoresis is specific to the administration of a medicinal ion or simply implies the use of current to mobilize ions. Thus, it is suggested that when performing iontophoresis, the clinician should clearly state what ion was "phoresed."

Adverse Effects: Current Not Drugs

There is a greater risk of skin irritation and redness when using DC than with other currents. This is why it is critical to inspect the skin both before and after treatment. The tingling, itching, and redness that can occur with iontophoresis have inaccurately been attributed to the drug(s) used when actually it is the DC current that causes these effects. Cations such as Na^+ that form sodium hydroxide (NaOH) are attracted to the cathode. The formation of NaOH creates an alkaline reaction, and the movement of water with sodium via osmosis results in a decrease in protein density (sclerolysis) in the tissues under the cathode, which softens the tissue. This increases the risk of tissue breakdown or electrical burn. At the anode, an acidic reaction is created as anions such as Cl^- are attracted to it, reducing the local pH in the tissues near the anode. Likewise, as water moves away from the anode with Na^+, protein density increases (sclerosis) and tissue hardens. These factors must be considered even after a single treatment but certainly when

iontophoresis is applied multiple times per week. In some patients showing skin compromise, it may be necessary to skip a treatment to allow the skin to heal. A summary of these reactions is presented in Table 10-8.

Recent Advances in Iontophoresis

If using peak current amplitudes of 4 mA, typical clinical treatments of 20 to 80 mA min require a 5- to 20-minute duration. At peak amplitude, patients may report stinging, tingling, or itching and mild reversible irritation.[105] Because maximal amplitude can be uncomfortable and patients are required to remain in the clinic, manufacturers designed wireless, disposable, battery-operated electrodes. These electrodes can be worn home by the patient, eliminating the need to remain in the clinic for the treatment, and they can deliver a low amplitude current for a longer duration, even up to 24 hours.[106] Newer iontophoresis electrodes consist of a single adhesive "patch" containing a complete microcircuit. A small button-sized battery delivers DC from 0.1 to 4 mA to an embedded anode and cathode to which the ionized drug and saline have been added (Fig. 10-21).

TABLE 10–8. Associated Reactions at Each Electrode During Iontophoresis

	Anode	Cathode
Attracts	Cl^-	Na^+
Forms	HCl	NaOH
Process	Sclerotic	Sclerolytic
Effect	Skin hardens	Skin softens*

* Increases risk of electrical burn and tissue damage to DC.

Fig 10 ■ 21 Wireless disposable electrode systems for iontophoresis.

These newer iontophoresis electrodes may be initially charged while the patient is in the clinic. This charging provides an initial voltage that overcomes the skin's initial resistance to the current. After a few minutes of charging and a reduction in skin resistance, the wear-home disposable electrode then delivers a lower-intensity current to phorese the drug. The electroconductive surface area is generally 6 to 9 cm^2, keeping the current density well within a safe range. Protocols for these electrode systems provide dosages similar to in-clinic procedures but seem to be gaining popularity for their ability to provide the same dose but at a lower (i.e., more comfortable) amplitude.

At the time of this writing, the literature is void of studies examining efficacy for these new wireless iontophoresis systems, but studies are under way. Positive evidence does seem to exist for the iontophoretic delivery of fentanyl, a rapid-acting opioid, for management of acute pain.[107,108]

Iontophoresis Versus Tap Water Galvanism

Although iontophoresis is the movement of ions in response to ES, conventional clinical use of the term has led the process to be associated with delivery of a pharmacological ion. Tap water galvanism (TWG) is the use of DC in tap water to create a constant, unidirectional electrostatic field.[85] The naturally occurring impurities found in tap water, such as magnesium, calcium, and chloride ions, are sufficient enough to conduct electrical current. TWG has long been shown to help reduce the symptoms of hyperhidrosis, a condition of excessive sweat production from the eccrine sweat glands, typically of the hands and feet.[85,109] The hands or feet are placed within water, becoming part of the electrical field as a low-amplitude DC current is applied (Fig. 10-22).

The mechanism of action of TWG remains unclear, but two mechanisms have been proposed: (1) DC current encourages hyperkeratinization in which waxy, keratinous plugs form and act to obstruct the sweat glands, and (2) DC may induce a functional disturbance that interferes with the stimulus-secretion coupling, impairing the electrochemical gradient of sweat output and thus decreasing sweat production.[110] Evidence for the use of TWG in the management of hyperhidrosis has existed for decades,[85,109] with the most recent evidence showing therapeutic benefit using a commercial iontophoresis device delivering DC current[84] (Box 10-5).

Fig 10 ■ 22 Most devices used for iontophoresis deliver direct current, which can be used to treat hyperhidrosis. Placement of the hands in a water bath with an electrical field created by direct current can be used to address palmar hyperhidrosis.

Box 10 ■ 5 Guidelines for Use of Iontophoresis

1. Select an ion appropriate for the pathology or condition.
2. Identify the correct polarity of the ion to be used.
3. Determine if the patient is allergic to the ion or drug.
4. Select an iontophoresis device providing DC current.
5. Select electrodes with appropriate size to maintain current density in recommended range.
6. Inspect and prepare area to be treated before application.
7. Place active electrode over target tissue(s) and inactive electrode away at a distance not less than twice the diameter of the cathode.
8. Choose an effective dosage by selecting an appropriate duration and amplitude.
9. Slowly increase the amplitude to peak.

After treatment, inspect the skin integrity for signs of adverse effect to current.

PRECAUTIONS AND CONTRAINDICATIONS

Effective use of ES is founded on two primary factors: (1) careful differentiation of patients who stand to benefit from the intervention from those for whom electrotherapy is not appropriate and (2) careful, knowledgeable, and safe application of the intervention. Thus, contraindications and precautions to application of electrotherapy must be considered during treatment selection (Box 10-6 and Box 10-7, respectively).

Box 10 ■ 6 Contraindications

Electrodes should not be placed over:

- The trunk or heart region in patients with demand-type pacemakers or implantable cardioverter defibrillators (ICDs)
  - In patients where use of electrotherapy is desired but contraindicated for pacemakers or ICDs, electrocardiographic examination by a cardiologist during a trial application of stimulation can determine the potential for interference with these devices.
- The pelvic, abdominal, lumbar, or hip region in pregnant women
  - Although the effects of electrical stimulation on the fetus or uterus are not clearly known, caution is recommended if using stimulation near these areas. For this reason, it is advised to ask all premenopausal women about the possibility of being pregnant prior to administering stimulation in these areas.
  - Pregnant women with known history of miscarriage may not be suitable to receive ES, regardless of the area of application. The unknown effects of ES on fetal health should be disclosed to the patient.
- Carotid bodies
  - Located on the anterolateral neck between the sternocleidomastoid and trachea; stimulation of these bodies may induce abnormal heart function. For this reason, proper electrode placement when treating posterior cervical musculature must ensure that carry-over of stimulation to the anterolateral neck does not occur.
- Phrenic nerve or urinary bladder stimulators
  - Interference from electrical stimulators may alter or compromise the function of these stimulators
- Areas of known peripheral vascular disease, including arterial or venous thrombosis or thrombophlebitis
  - Because stimulation may increase blood flow to the treatment area, avoid stimulating areas of compromised blood flow
- The phrenic nerve, eyes, or gonads
- Areas of active osteomyelitis
- Areas of hemorrhage

Safety With Electrotherapeutics: "Primum Non Nocere"

Latin for "first, do no harm," the phrase *primum non nocere* should always be considered when preparing to use an electrotherapeutic agent. Using electrical current by nature brings with it inherent risk, danger, and potential for adverse events. However, clinicians aware of safety measures and safety practices can greatly minimize these risks and safely and effectively deliver therapeutic electrical stimulation. The primary concern with the use of electrical current is shock. Although the

Box 10 ■ 7 Precautions

1. Electrotherapy should be used with caution in patients:
   - Without intact sensation, as they may be unable to sense or detect and recognize abnormal sensations. An exception to this is when using low-amplitude current for wound healing.
   - Who are unable to communicate, as they may be incapable of accurately providing feedback regarding the stimulation.
   - With compromised mental ability or lack of cognition, as they may be unable to understand directions.
   - With cardiac dysfunction, including uncontrolled hypertension or hypotension, or irregular heart rate or rhythm.
   - With epilepsy or other seizure disorders.
2. Over neoplasms (active or previous):
   - Use of electrotherapy in patients with active cancer should include consultation with the patient's physicians and documentation of patient consent.
   - Use of electrotherapy over sites of inactive neoplasm or elsewhere in patients with history of cancer should be done only after thorough explanation of the anticipated risks and benefits.
   - Written documentation of patient and physician consent is recommended.
3. Electrodes should not be placed over:
   - Compromised skin (except if treating for wound/tissue repair).
   - Tissues vulnerable to hemorrhage or hematoma.
   - Cervical (i.e., neck) or craniofacial regions in patients who have a history of cerebrovascular accident or seizures.
4. Do not use ES devices within approximately 5 yards of diathermy units or other source of electromagnetic radiation.

CASE STUDY 10-8 Iontophoresis

The patient is a 28-year-old female recreational tennis player who developed right lateral elbow pain 6 weeks ago. She presents to the clinic with a chief complaint of right elbow pain during tennis and when grasping with her hand. Palpation reveals tenderness and elicits pain over the lateral epicondyle and common extensor tendon of the right elbow. Wishing to address the acute soft tissue inflammation of the extensor tendon, the practitioner chooses to use iontophoresis and selects dexamethasone for its anti-inflammatory properties. After choosing an electrode with a 3 × 3 cm conductive surface area, the dexamethasone is applied to the electrode, and the electrode is placed over the lateral epicondyle and extensor soft tissue. Because dexamethasone is a negatively charged ion, the cathode of the DC current will be delivered to the electrode containing the dexamethasone. To complete a circuit, saline is placed on the inactive electrode, which is placed at a distance not less than twice the diameter of the smallest electrode. The amplitude of the current is increased to 4 mA, creating a current density of 0.44 mA/cm^2 (4 mA/9 cm^2). A 40-mA min dosage is selected, so at 4 mA, the duration of the treatment will be 10 minutes. To complete the treatment, the clinician removed the electrodes and inspected the tissue for signs of adverse effect of the current.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of electrotherapy?
ANSWER: Yes, the patient exam reveals relatively acute and localized tenderness to the extensor tendon of the lateral epicondyle.

2. Is the patient appropriate for electrotherapy (i.e., do any of the general precautions or contraindications to electrotherapy apply, or are there any specific considerations regarding application of electrotherapy to this patient)?
ANSWER: The patient is a 28-year-old female and of reproductive age. Pregnancy is contraindicated with iontophoresis, so the practitioner should explain this concern and ask the patient if she is pregnant.

3. What are the specific goals to be achieved with the use of electrotherapy?
ANSWER: To decrease local tissue inflammation and damage.

4. Do you have the specific type of electrotherapy that is appropriate for the patient?
ANSWER: Iontophoresis offers a method for delivering dexamethasone sodium phosphate transcutaneously to a localized soft tissue area.

Continued

CASE STUDY 10-8 Iontophoresis—cont'd

5. Can you select the specific parameters of electrotherapy that are appropriate for the patient?

ANSWER:

Waveform: DC
Dosage: 40 to 80 mA/min
Intensity: Up to 4 to 5 mA
Duration: 10 to 20 min, depending on dosage and intensity

6. What are the proper application procedures for electrotherapy?

ANSWER:

Instruct the patient: Inform the patient of the purpose and procedure. Be sure to explain the anticipated sensation and effect of the stimulation.
Preinspection: Inspect the skin in the area to be treated as well as the area where the reference, or inactive, electrode will be placed.
Electrode placement: The appropriate volume of dexamethasone is applied to a commercially manufactured electrode and placed over the lateral epicondyle. The volume of medication is dependent on the electrode size and is often marked on the electrode. The reference electrode is placed on the triceps of the same arm away from the treatment electrode. Because dexamethasone is a negatively charged ion, the treatment electrode is designated as the cathode and the reference electrode as the anode.
Patient position: The patient may be placed sitting with the arm resting on the treatment table.
Postinspection: Remove the electrodes and inspect the skin beneath the treatment and reference electrode for any signs of adverse effect. The practitioner must be aware that frequent treatments may result in tissue breakdown at the lateral epicondyle (cathode) and hardening at the triceps (anode).

intensity of the current flowing from the therapeutic device to the patient is often well below harmful levels, the danger of electrical shock lies in the current flowing between the electrotherapeutic device and the wall current (household current). So although battery-powered devices may present less risk than line-powered ones, attention must nevertheless be given to ensure the safest environment for the patient and clinician. Any excess or short circuit must be terminated before it reaches a patient or the clinician.

Perhaps the most common and effective safety measures regarding electrical current from line-powered devices are the hospital-grade, three-prong plugs that have a safety ground in direct connection to the earth ground and the ground-fault circuit interrupters (GFCI). True ground returns current to the ground (i.e., earth) and does not include passage through the patient. GFCIs detect any loss or leak of current in the circuit created between the patient and the device. Most GFCIs are activated when a differential in current out versus current returned is greater than 3 to 5 mA. If a leak of this level is detected, the GFCI immediately stops flow of current within approximately 25 msec.

Although grounded plugs and GFCIs are essential safety measures, routine inspection is still important. Prior to every use, the clinician should take the time to inspect the integrity of the three-prong plug, the GFCI wall outlet, the power cord to the device, and the insulated coating on the lead wires. Lead wires are often run over by carts holding the devices and the leads can get caught on objects while moving the device, stretching and stressing the leads. This can damage the protective rubber or plastic coating and present a potential pathway for current to leak; this creates a risk to the patient or clinician. Likewise, the power cord from the wall to the device can be run over, pinched, or pulled loose. Thus, careful inspection of these components is critical.

More extensive inspections performed by trained personnel are also recommended and required by many standards. Although there is presently no stipulation as to the frequency of formal inspection, an annual inspection should be done at the minimum, and more frequent inspections should be directly related to the frequency the device is used. Careful attention should also be given to the position of the patient and the placement of the electrodes so that electrodes do not come in contact with conductive surfaces that may lead to current leak. Some examples include metals from treatment tables and chairs, including bed rails and armrests, and other equipment that may be nearby such as orthotic equipment or

a variety of hospital equipment containing metal. Likewise, lead wires can easily become caught or snagged on beds, tables, and chairs, so a watchful eye for these potential situations can lead to greater safety with electrotherapeutic stimulators.

Documentation Tips

Follow the documentation guidelines explained in Chapter 9.

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SECTION III

Clinical Applications of Modalities

CHAPTER 11

MECHANISMS OF PAIN AND USE OF THERAPEUTIC MODALITIES

Richard E. Liebano, PT, PhD

The International Association for the Study of Pain defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (www.iasp-pain.org).[1] Pain is the most common complaint and the most prevalent symptom that requires intervention among patients in rehabilitation programs. Perception of pain is influenced by a variety of factors such as cultural differences, motivation, emotional states, and past experiences with pain.[2] Although clinicians often treat patients with pain with the goal of reducing or eliminating pain, it is important to note that pain is an important protective sensation. In this manner, pain can serve as an indicator or pathology, physical stress, or injury. Therefore, when pain is present, treatment should be targeted to solve the underlying cause.

TYPES OF PAIN

Pain can be described as acute, chronic, or referred. Pain can also be described according to the origin, or the relationship between the symptoms and the underlying pathology. To best understand the mechanisms of electro-pain modulation and improve clinical management of pain, the types of pain must be differentiated.

Acute Pain

Acute pain is a symptom that results from injury and/or disease that causes or can cause tissue damage through infection, trauma, the progression of a metabolic disorder, or a degenerative disease. Acute pain is generally but not universally described as pain lasting less than 12 weeks (i.e., 3 months). Acute pain is typically well located and defined, depending on the type of tissue involved. Superficial (e.g., skin) pain is typically sharp and easy to locate. On the other hand, acute deep-tissue pain from muscles, joints, or viscera can be diffuse and difficult to locate.[1] Acute pain serves to protect against further tissue damage, and when tissue injury is present, pain may be maintained in order to allow time for proper tissue healing. Therefore, the symptoms can reflect the underlying pathology.[3] The clinical treatment of acute pain can be pharmacological or nonpharmacological, involve rehabilitation or surgery, or other procedures aimed at addressing the peripheral tissue damage.[1]

The stimulus responsible for acute pain has a short latency and is associated with increased muscle tone, heart rate, blood pressure, skin impedance, and other manifestations related to the increase of activity of the sympathetic nervous system. Autonomic, psychological, and behavior responses persist while the stimulus is present.

Key Point! Acute pain is associated with an actual physiological event (e.g., tissue damage, infection, trauma, metabolic disorder, etc.). Because acute pain is often associated with changes in heart rate, blood pressure, and even respiratory rate, measurement of vital signs is warranted.

Chronic Pain

Chronic pain is commonly defined as persistent or recurrent pain existing for 3 to 6 months or pain that persists beyond the normal time expected for healing of injured tissue. Chronic pain follows acute pain and is also associated with structural and functional changes in the central nervous system that require multiple therapeutic approaches. Central sensitization, or the amplification of neural signaling within the central nervous system that underlies pain hypersensitivity, is a characteristic of chronic pain. The persistence of chronic pain associated with injury or disease, such as diabetes or arthritis, may result in changes in the physiological properties of the peripheral nervous system.

Chronic pain is no longer considered a symptom, is not protective, and may even be considered a disease itself.[4] Generally, chronic pain is associated with physical, emotional, social, and financial disability.[5] Patients with chronic pain often report physical inactivity due to the long period of immobility, resulting in decreased muscle strength and functional capabilities, and may also exhibit signs of depression. Furthermore, patients with chronic pain can pose significant financial and public health considerations as noted in rising costs of pharmacological management and challenges to medical care models.[6,7] More succinctly, chronic pain is difficult to manage. Clinicians must rely on a multidisciplinary approach and should involve more than one therapeutic modality.[4]

Key Point! Chronic pain is pain that was once acute but has persisted beyond the normal time expected for healing and no longer serves as a protective mechanism.

Referred Pain

Referred pain is defined as pain that occurs at a site remote from the source of the disease or injury, usually a visceral or muscle source.[8–10] It is generally believed that referred pain occurs due to convergence of cutaneous, visceral, and skeletal muscle nociceptors on the common nerve root of the spinal cord.[9] The brain interprets the afferent input as arising from cutaneous structures because of the higher proportion of cutaneous afferents converging on second-order transmission neurons.[10] A

common example is referred pain that radiates to the left shoulder, arm, jaw, or chest during angina or myocardial infarction.

Key Point! Treating pain of unknown or unidentifiable origin is considered a contraindication to TENS. Masking undiagnosed pain with TENS can postpone proper treatment and lead to worsening of the underlying condition.

PAIN PATHWAYS

Most textbooks that include the use of TENS for pain modulation do not address the underlying physiology of pain. Perhaps not considered requisite to the use of TENS by some, a fundamental understanding of the mechanisms of pain can enhance the clinician's administration of TENS and allow the clinician to better explain the rationale for it. With this in mind, this chapter will briefly address the current conceptual understanding of the physiological mechanisms of pain before we cover clinical applications of TENS.

Peripheral Pain Pathways

Nociceptors (also called *pain receptors*) are free, noncorpuscular nerve endings of A-delta (δ) and C fibers that conduct nerve impulses toward the central nervous system. They are found in the skin, muscle, joints, bone, and viscera and have a high threshold for activation. This threshold is lowered by release of chemical substances after tissue injury and inflammation. These chemical substances include prostaglandins, bradykinin, histamine, acetylcholine, potassium ions, substance P, calcitonin gene-related peptide, serotonin (5-HT), nerve growth factor, thromboxane, acidic environment, leukotriene, and adenosine triphosphate. In other words, these substances sensitize the nerve endings (Fig. 11-1).

A-δ or group III fibers are thin myelinated fibers (1–5 μm) that respond to high-intensity mechanical and thermal stimuli. They have a conduction velocity of 5 to 30 m/s and therefore are responsible for fast-conducting peripheral pain signals. The function of A-δ fibers is associated with the first pain sensation, precise location of noxious stimuli on the body, and generation of withdraw reflexes. The sensations associated with A-δ fiber activation are usually described as sharp, stabbing, or pricking.

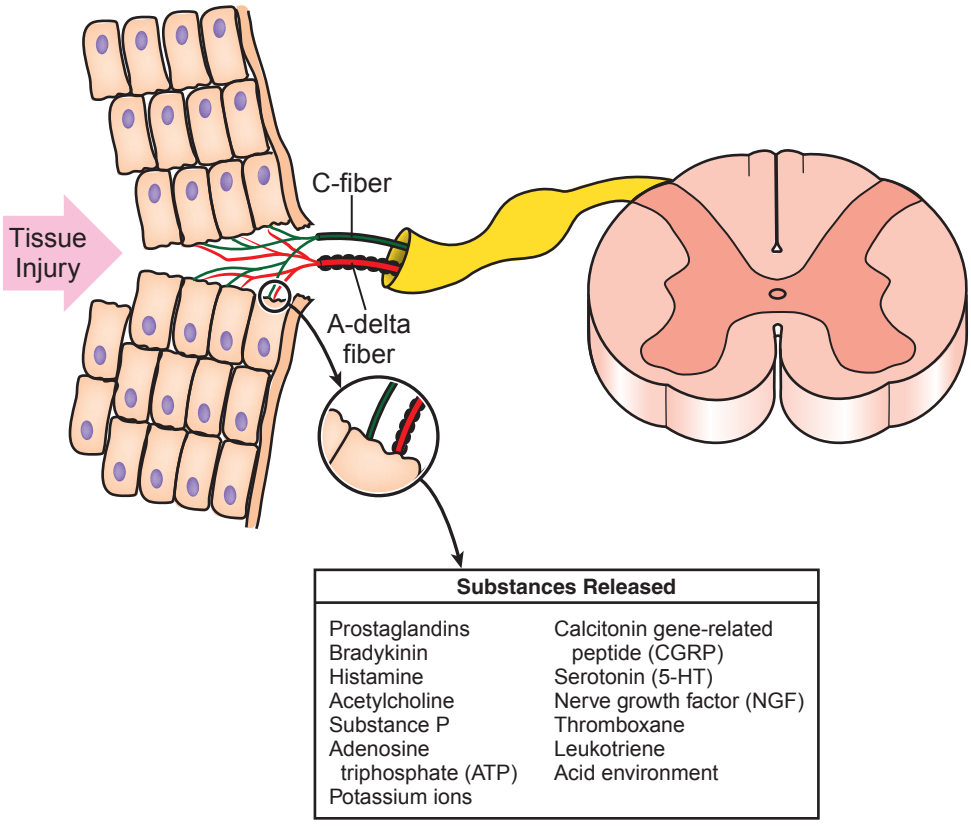

Fig 11 ■ 1 Peripheral sensitization of pain fibers.

C or group IV fibers are unmyelinated and thin fibers (0.1 to 1 μm) that respond to a broad range of painful stimuli, including mechanical, thermal, or chemical. They are also called polymodal fibers, meaning that they respond to a variety of painful stimuli and have a conduction velocity of 0.5 to 2 m/s. The pain produced by activation of C fibers is considered second pain and characterized as slow, dull, aching, burning, and long lasting. C fibers generate second-pain sensation and are important to prevent further tissue damage (Table 11-1).

Fundamentals in Clinical Practice

To illustrate the differing characteristics of C and A-δ pain in a real-life example, think about the pain felt when striking your shin on something. The initial sharp stabbing pain is mediated by the A-δ pain fibers and the residual dull throbbing pain that persists well after the incident is mediated by C pain fibers.

Central Pain Pathways

A-δ and C fibers (first-order neurons) make direct or indirect synapses through interneurons, with second-order neurons in the dorsal horn of the spinal cord (Fig. 11-2). These second-order neurons are sometimes called *central nociceptive transmission neurons* because they transfer the nociceptive impulses from the spinal cord and brain stem to the higher centers of the brain. Two types of second-order neurons have been identified: (1) a nociceptive-specific second-order neuron, also known as a *high threshold neuron,* that receives input from peripheral nociceptors only and (2) a wide-dynamic range (WDR) neuron that receives input from both nociceptive and non-nociceptive primary afferent fibers. After tissue injury, both high-threshold neurons and WDR neurons become sensitized. This phenomenon is termed *central sensitization* and results in an increase of receptive field size, increased responsiveness to non-noxious or noxious stimuli, and/or decreased threshold to non-noxious or noxious stimuli.[1]

Although several ascending pain pathways have been identified, the spinothalamic tract (STT) is generally considered the main pathway responsible for transmitting nociceptive input from somatic and visceral tissue to higher centers. Most neurons of the STT cross the midline at the spinal cord level via the anterior white commissure to ascend to the ventroposterior lateral thalamic nucleus and medial thalamic nuclei. STT cells carry discriminative features of type and location of pain and respond to noxious thermal and mechanical stimuli.[9] The spinomesencephalic tract ascends to the midbrain and terminates in periaqueductal gray (PAG). Spinomesencephalic cells terminating in the PAG may activate a descending inhibitory system that promotes analgesia due to the release of serotonin. The spinoreticular tract is constituted by cells that ascend to the medullary reticular formation and to the pontine reticular formation. Neurons of the spinoreticular tract allow for suppression or facilitation of pain and are involved in the motivational, emotional, and unpleasant components of pain.[9]

TABLE 11–1. Physiological Characteristics of Nociceptive Afferents

A-δ Fiber	C-Fiber
High threshold	High threshold
Myelinated	Unmyelinated
1–5 μm diameter	0.1–1 μm diameter
Fast conducting (5–30 m/sec)	Slow conducting (0.5–2 m/sec)
Responds to high-intensity heat, cold, and mechanical stimuli	Responds to high-intensity heat, cold, mechanical, and chemical stimuli (polymodal)
Generates fast/first pain with withdraw reflexes and sensations	Generates slow/second pain sensations
Associated with the precise location of noxious stimuli on the body	Associated with the prevention of further tissue damage

ELECTRICAL STIMULATION FOR PAIN CONTROL

History of Electrical Stimulation for Pain Modulation

The first records of use of electricity as a therapeutic modality predate the discovery of electricity. Stone carvings in tombs from the Egyptian Fifth Dynasty (about 2500 BC) depict the use of a species of electric catfish (*Malapterurus electricus*) found in the River Nile and

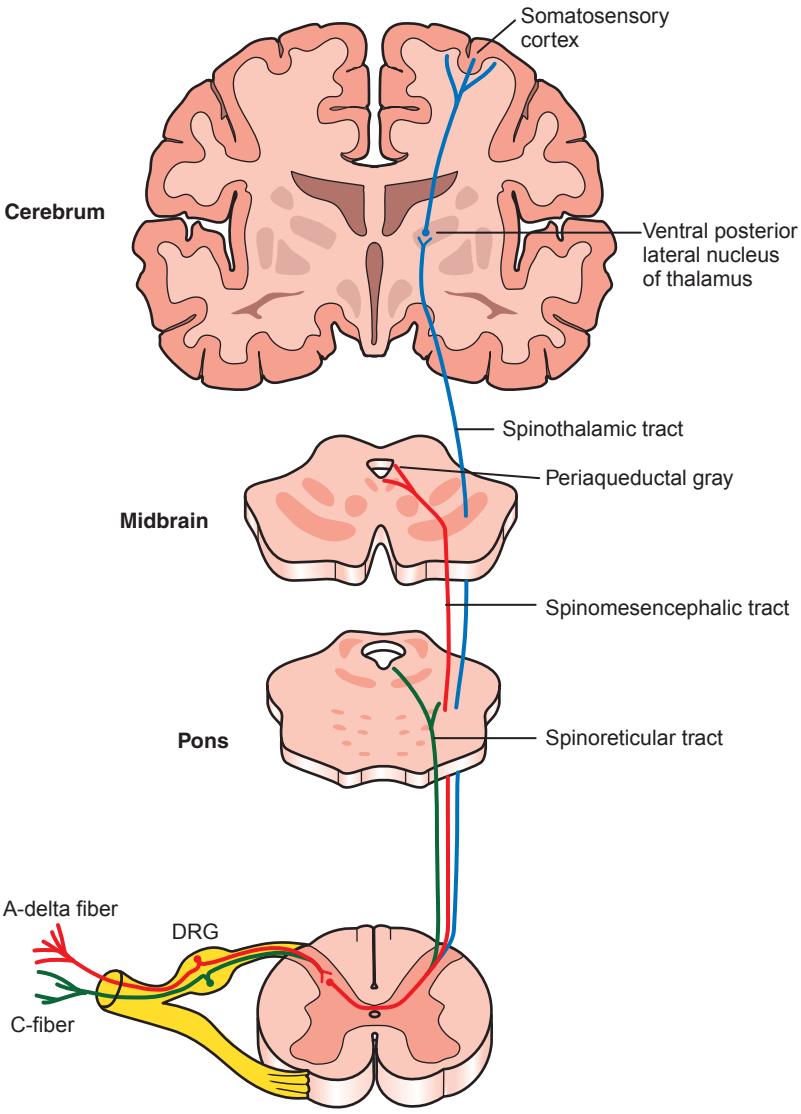

Fig 11 ■2 Central pain pathways.

electric rays (*Torpedo marmorata*) to treat painful conditions. Hippocrates (400 BC) referred to the use of electric fish to treat arthritis and headache, although Scribonius Largus (AD 46), one of the first Roman physicians, wrote *Compositiones Medicae*, which is believed to be the earliest documentation of using black torpedo fish as electrotherapy to treat gout and headaches.[2,10–13] Although electric fish have long disappeared from medical use, and nowadays patients with headaches are most frequently treated with pharmacological agents, the use of transcutaneous electrical stimulation of the trigeminal nerve to treat headache syndromes has resurged with the use of modern devices (http://www.cefaly.ca/site/studies). Today, most transcutaneous electrical stimulation devices generate low-voltage pulsed currents and are widely used by heath-care professionals to treat different types of acute and chronic pain.

Transcutaneous Electrical Nerve Stimulation

Transcutaneous electrical nerve stimulation (TENS) involves the application of electrical stimulation to the skin via surface electrodes to stimulate nerves fibers to produce different physiological effects.[14–19] This is a widely used nonpharmacological, noninvasive, safe, easy-to-use, and low-cost technique to control pain.[14,16]

The first TENS units were developed and became popular after publication of the gate theory of pain by Melzack and Wall in 1965.[20] According to this theory, stimulation of large-diameter afferents inhibits

nociceptive fiber-evoked responses in the dorsal horn. Since then, TENS became known worldwide. TENS is currently one of the most frequently employed electrophysical agents used by health-care professionals to relieve acute and chronic pain.[14,16,21] The main mechanism of action of TENS is the activation of peripheral opioid receptors[22] and opioid receptors within the central nervous system.[23–25]

Physical Principles of TENS

TENS is not a specific type of equipment or electric current; rather, it is a method of activating nerve fibers by delivering electrical impulses through the skin using surface electrodes. Thus, the use of any type of electrical current that activates nerve fibers without disruption of the skin can be called TENS and can be completed using a variety of different stimulators.[26] Although TENS does not refer to any specific type of current, the most common and regularly used TENS units traditionally emit two basic types of electric current: a rectangular biphasic symmetrical pulsed current or a biphasic asymmetrical balanced pulsed current (Fig. 11-3). Both types of electric currents are considered nonpolarized, meaning that they have no sustained polarity and can be used for long periods of time without presenting the risk of chemical burns to the skin.

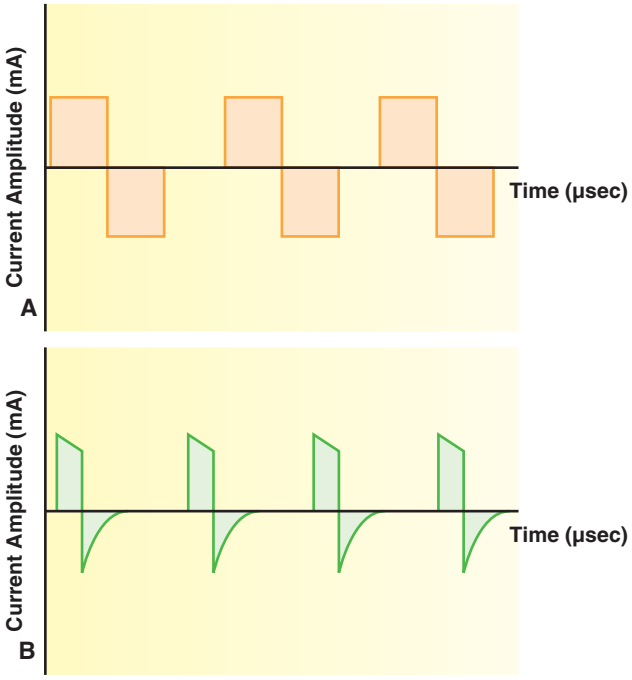

Fig 11 ■3 The two most common waveforms for TENS: (A) biphasic symmetrical pulsed current; (B) asymmetrical biphasic balanced pulsed current.

Clinical Controversy

TENS is a method of nerve stimulation used to provide pain modulation, not a specific type of device or stimulator. A variety of waveforms other than symmetrical and asymmetrical biphasic pulsed currents can be used for TENS. Thus, high-volt pulsed current, burst-modulated alternating current, interferential currents, and others can be used for TENS (i.e., electro-pain modulation) if they can provide the appropriate stimulus parameters of pulse frequency, pulse duration, and amplitude (described later in this chapter).

Key Point! Because the waveforms most commonly used for TENS are symmetrical or balanced asymmetrical, there is no net DC charge accumulated at either electrode. For these waveforms, there is no evidence to support the specific placement of the red and black lead colors common to many TENS stimulators.

Fundamental Parameters of TENS

When TENS is administered, the electrical stimulation can be varied in terms of the three fundamental parameters of electro-pain modulation: frequency, amplitude, and pulse duration. The frequency is a time-dependent characteristic that refers to the number of pulses delivered per second; this is measured in hertz (Hz). The amplitude (also called *intensity*) refers to the magnitude of current or voltage applied by the unit; this is often measured in milliamps (mA) or volts (V) or millivolts (mV). The pulse duration or pulse width is the length of time one pulse lasts; this is measured in microseconds (μsec) or milliseconds (msec). The term *pulse duration* is preferable to *pulse width* because duration is a time-based measurement rather than a linear size.

Most TENS units offer the clinician the ability to manipulate or control the variables of amplitude, pulse duration, and frequency with either digital or analog adjustments. However, clinicians should note that for many analog control dials and even some digital, the change in the stimulus parameters may not

coincide with the magnitude of change to the dial. For example, an increase in a variable (e.g., amplitude) from 2 to 3 on a dial ranging from 1 to 10 may not correspond to a 10% increase in that specific variable (e.g., amplitude). In order to best appreciate and understand the actual magnitude of change in a variable, clinicians should feel the change through firsthand experience.

In addition to control of the fundamental parameters of pulse frequency, duration, and amplitude, another control or switch may be available that will allow the user to choose between continuous pattern of pulses (often labeled "C" or "normal" on some TENS units), burst pattern (high-frequency trains of pulse delivered at low frequency, often labeled "B"), or modulated pattern (variation in either pulse duration, frequency, or amplitude parameters in a cyclic fashion, often labeled "M") (Fig. 11-4). The choice of modulated pattern has been included by manufacturers to overcome sensory habituation and to provide more comfort to the patient; however, there is a lack of studies confirming these claims.

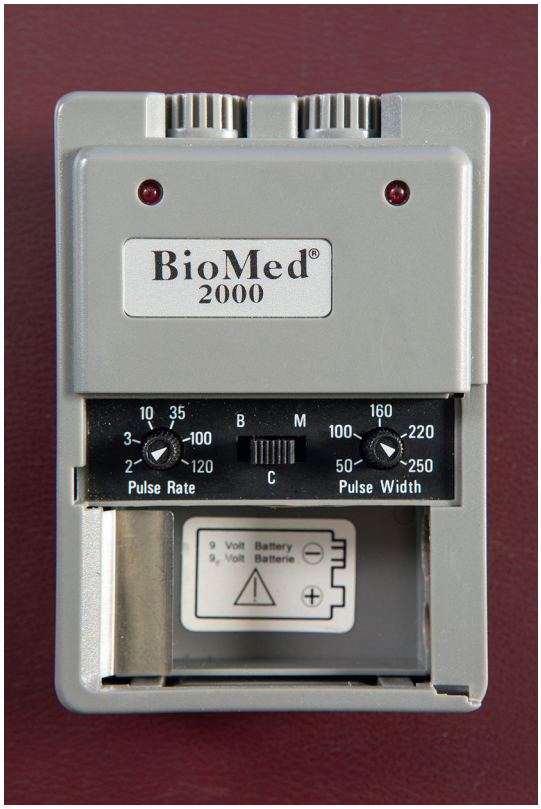

Fig 11 ■ 4 A TENS unit with a slide-switch control for selecting burst (B), continuous (C), or modulated (M) pattern TENS.

Key Point! The fundamental stimulation parameters of TENS are frequency, pulse duration, and pulse amplitude. To date, the specific waveform shape has not been shown to be a significant parameter for electro-pain modulation.

MODES OF TENS

Traditionally, four modes of TENS are most commonly used and reported: conventional TENS, acupuncture-like TENS, burst train TENS, and brief intense TENS (Fig. 11-5).

Conventional TENS

Conventional TENS, often called *sensory* or *high-frequency/low-intensity TENS,* is the most commonly used mode of TENS. The stimulation parameters have a low intensity, a high frequency (typically 80–110 Hz), and a short pulse duration (50–100 μsec). This combination of parameters preferentially activates the Group II (Aβ) afferent nerve fibers, producing a sensation of comfortable paresthesia (i.e., a sensory-only effect, without a motor or muscular response).

Acupuncture-Like TENS

Acupuncture-like TENS, or low-frequency/high-intensity TENS, is stimulation performed with intensity high enough to evoke visible muscle contractions (i.e., a motor response) at myotomes related to the origin of the pain using a frequency below 10 Hz (usually 1–4 Hz) and a long pulse duration (~200 μsec). With this mode of TENS, in addition to activation of group II fibers (Aβ), there is activation of group I (Aα) fibers, leading to rhythmic muscle twitches. It is generally assumed that these muscle contractions would activate the fibers of group III (Aδ) arising from muscle ergoreceptors. However, recordings of spinal cord dorsum potentials in animals showed that only large-diameter primary afferent fibers from deep tissue are activated by both low- and high-frequency TENS at sensory intensities up to and including motor thresholds.[27] Increasing intensity to twice the motor threshold was necessary to recruit Aδ afferent fibers. One human study demonstrated that low-frequency TENS (4 Hz) applied at a maximal tolerable

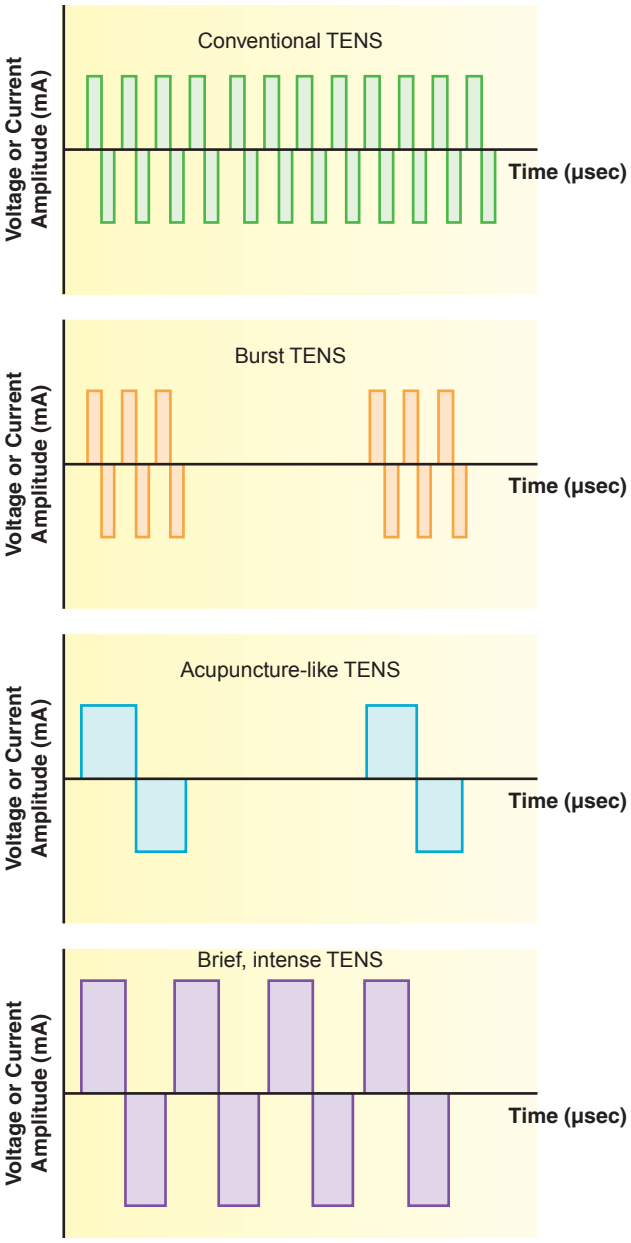

Fig 11 ■5 Most common waveform types for TENS.

intensity activates only Aβ afferent fibers, whereas Aδ activation only occurs at intensities above maximal tolerable intensities. The authors thus concluded that conventional and acupuncture-like TENS activates similar peripheral afferent fibers, predominately Aβ.[28]

Key Point! By convention, TENS has come to imply use of a handheld battery-powered device used for pain modulation. This is erroneous because most TENS modes can also be found or created on the majority of clinical multi-waveform line-powered stimulators.

Burst Train TENS

Burst train TENS is a combination of conventional and acupuncture-like TENS and has high-frequency trains of pulses (around 100 Hz) delivered at a low frequency (1–4 Hz) and long pulse duration (~200 μsec) at motor level. Some authors refer to burst train mode of TENS as acupuncture-like TENS.[2,28] Some patients prefer this mode to acupuncture-like TENS because the pulse trains produce a more comfortable muscle contraction.[2,10,29]

Brief Intense TENS

Brief intense TENS uses a high-frequency (100–150 Hz), a long pulse duration (150–250 μsec), and an amplitude at the patient's highest tolerable intensity for a short period of time (generally less than 15 minutes). Brief intense TENS has been successfully used to provide short local hypoalgesia during minor painful procedures, including wound dressing changes, skin debridement, suture removal, and venipuncture.[30] Due to its inherent discomfort, this mode of TENS is rarely used clinically as a first option. Brief intense TENS is usually used when other TENS modes have not resulted in sufficient pain modulation. The commonly used parameters of each TENS mode are described in Table 11-2.

ANALGESIC MECHANISMS OF TENS

The gate control theory of pain is the most commonly used theory to explain the inhibition of pain by TENS.[31] This theory proposes that stimulation of large-diameter afferent fibers (Aβ) activates local inhibitory circuits in the dorsal horn of the spinal cord and prevents nociceptive impulses carried by small-diameter fibers (C and Aδ) from reaching higher brain centers. The gray matter of the spinal cord is divided into 10 layers (Rexed laminae), which basically progress from posterior to anterior (as laminae I–X). The incoming pain fibers (Aδ and C) synapse in laminae I–V, whereas Aβ fibers from cutaneous mechanoreceptors synapse in laminae III–VI (Fig. 11-6). The Aα fibers arising from neuromuscular spindles and Golgi tendon organs terminate in laminae VI, VII, and IX.[32] The substantia gelatinosa (laminae II and III)[33] consists of small, densely packed interneurons that act as a gate.[20,34] These interneurons have an inhibitory effect on the terminals of pain fibers that synapse with the T cell. According to the original gate

TABLE 11–2. Parameters of TENS Modes Commonly Used for Pain Control

Modes	Parameters		
	Frequency	*Pulse Duration*	*Amplitude/Intensity*
Conventional	10–250 Hz	≤ 100 μsec	Sensory level
Acupuncture-like	< 10 Hz (1–4 Hz)	150–200 μsec	Motor level
Burst train	100 Hz, delivered at 2 Hz	150–200 μsec	Motor level
Brief, intense	100–150 Hz	150–200 μsec	Noxious level

control theory of pain, when Aβ fibers are activated, they send excitatory stimuli through a collateral branch that activates the substantia gelatinosa (SG) interneurons that inhibit the pain fiber terminals and T cell activity. Therefore, SG interneurons close the gate to nociceptive traffic and reduce pain. On the other hand, Aδ and C fibers send an inhibitory collateral branch that reduces the inhibitory effect of SG upon the pain fiber terminals, increasing the firing of the T cell. In this case, the gate is open and nociceptive traffic can proceed to upper centers, resulting in pain. The gate control theory of pain also recognizes the presence of descending influences from a central control that could help modulate pain.

The gate control theory of pain was published more than 40 years ago and has been expanded and revised several times since its original publication in 1965. The original gate control model suggests that pain fibers directly inhibit the SG cells. However, pain fibers are excitatory and activate an interneuron that inhibits SG cells. Moreover, the original theory only suggests a presynaptic inhibition. Further studies have shown the occurrence of postsynaptic inhibition as well[1,35,36] and that the inhibitory neurotransmitter GABA may play a role in the presynaptic and postsynaptic inhibition.[37] Despite these new findings, the gate control theory of pain can still be viewed as a valuable contribution to neuroscience, as it is still used to explain the analgesic mechanisms of several therapy interventions.

Analgesic Mechanisms of Low-Frequency TENS

Animal studies support that low-frequency TENS (less than 10 Hz) produces antihyperalgesia by triggering descending inhibitory pathways that include the ventrolateral PAG that sends projections to the rostral ventromedial medulla (RVM), which then projects to the spinal cord (Table 11-3). Blockade of these areas in

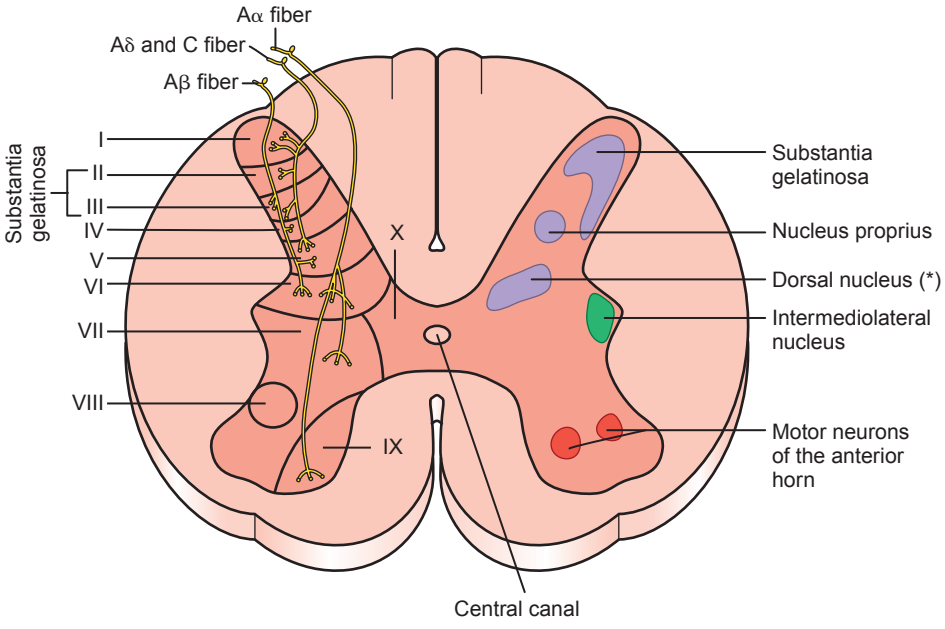

Fig 11 ■ 6 Sensory input onto Rexed laminae.

TABLE 11–3. Summary of Analgesic Mechanisms for Low- and High-Frequency TENS

Low-Frequency TENS	High-Frequency TENS
Activates PAG, RVM, and SC descending pathway	Activates PAG, RVM, and SC descending pathway
Activates μ-opioids receptors in RVM, SC, and in periphery	Activates δ-opioids receptors in RVM and SC
Uses GABA and activates $GABA_A$ receptors in SC	Uses GABA and activates $GABA_A$ receptors in SC
Uses serotonin and activates 5-HT_2 and 5-HT_3 receptors in SC	
	Reduces glutamate and aspartate release
	Increases concentration of β-endorphins and methionine encephalin in CSF
Uses acetylcholine and activates muscarinic receptors (M1 and M3) in SC	Uses acetylcholine and activates muscarinic receptors (M1 and M3) in SC
Activates α_{2A}-adrenergic receptors in periphery	Activates α_{2A}-adrenergic receptors in periphery
	Reduces substance P in SC and periphery

CSF = cerebrospinal fluid; 5-HT = 5-hydroxytryptophan; GABA = gamma-aminobutyric acid; PAG = periaqueductal gray; RVM = rostral ventromedial medulla; SC = spinal cord; TENS = transcutaneous electrical nerve stimulation.

the central nervous system inhibits the analgesic effects of TENS. There is evidence that low-frequency TENS activates μ-opioid (mu) receptors in the RVM and in the spinal cord.[23–24] In the spinal cord, low-frequency TENS also activates serotonin 5-HT_{2A}, 5-HT_3,[38] $GABA_A$,[39] and muscarinic M1 and M3 receptors[40] and is associated with increased release of serotonin.[41–43]

Peripheral μ-opioid receptors are also involved in the analgesia induced by low-frequency TENS. Sabino et al[22] showed that blockade of peripheral opioid receptors by naloxone (Narcan) at the site of application prevents the reduction in hyperalgesia produced by low-frequency TENS but not high-frequency TENS in an animal model of inflammatory pain. Peripheral α_{2A}-adrenergic receptors are also involved in the antihyperalgesic effects of low-frequency TENS.[44]

Analgesic Mechanisms of High-Frequency TENS

High-frequency TENS increases the concentration of β-endorphins in the bloodstream and cerebrospinal fluid and methionine encephalin in the cerebrospinal fluid in human subjects[45–47] (see Table 11-3). In animals with joint inflammation, high-frequency TENS reduces glutamate and aspartate concentrations in the spinal cord dorsal horn by activation of δ-opioid (delta) receptors.[48] Blockade of δ-opioid receptors in the RVM or spinal cord or blockage of synaptic transmission in the ventrolateral PAG prevents the reversal of hyperalgesia produced by high-frequency TENS.[23–24] High-frequency TENS also enhances release of the neurotransmitter GABA in the spinal cord dorsal horn and activation of $GABA_A$[39] and muscarinic receptors (M1 and M3)[40] in the spinal cord.

Peripherally, high-frequency TENS reduces substance P in dorsal root ganglion neurons[49] and activates α_{2A}-adrenergic receptors.[44]

Analgesic Tolerance and TENS

Tolerance may be defined as a decrease in analgesic effectiveness with repeated use of a given therapeutic method.[50] Repeated stimulation of opioid receptors by repeated administration of opioid drugs can lead to an analgesic tolerance. The repeated application of either low- or high-frequency TENS can also result in analgesic tolerance as shown in rats[25] and human[19] studies. Tolerance induced by low-frequency TENS results in cross-tolerance at μ-opioid receptors in the spinal cord, whereas the analgesic tolerance induced by high-frequency TENS results in cross-tolerance at δ-opioid receptors in the spinal cord in animals.[25] Solomon et al[51] showed that people who had been using opioid pharmacological agents long enough to develop tolerance before surgery also did not respond to TENS when used postoperatively.

Because most commercially available opioid drugs activate μ-opioid receptors, the development of tolerance in these receptors will make opioid users nonresponders to low-frequency TENS. A recently published study confirmed data from experimental animal studies showing that chronic pain patients treated with opioids responded favorably to high-frequency TENS (conventional TENS) but did not respond to low-frequency TENS (acupuncture-like TENS). In the patients who were not treated with opioids, both low- and high-frequency TENS promoted analgesia.[52] Therefore, when deciding which frequency of TENS to use, clinicians should consider the patient's pharmacological history.

In patients with no history of opioid use, both low- and high-frequency TENS modes can be used. In general, high-frequency TENS is tested as a first option and clinicians should observe the patient's response. If the response is poor, low-frequency TENS should be tried or the placement of the electrodes should be changed. Law and Cheing[53] compared three different frequencies of TENS (2 Hz, 100 Hz, and alternating frequency 2 Hz/100 Hz) in patients with knee osteoarthritis. In the three groups receiving TENS, pain intensity was lower than in the placebo group but no difference was observed between the groups receiving active TENS, suggesting that both low- and high-frequency TENS successfully lessen pain.

Key Point! Patients concurrently or recently using opioid medications are less likely to respond to low-frequency TENS due to cross-tolerance developed from stimulation of μ-opioid receptors. These patients are more likely to respond to high-frequency TENS. Patients without concurrent or recent use of opioids are considered "good responders" to both low- and high-frequency TENS.

Importance of Stimulus Intensity

Regardless of the frequency that has been selected, it is imperative that the adjustment of the amplitude (intensity) of current be performed properly. The amplitude should be increased until the patient reports the strongest comfortable paresthesia. The amplitude adjustment has been a determining factor in the success of TENS because only stronger intensities promote a significant hypoalgesia.[54–58] Systematic reviews of the literature performed by Bjordal and colleagues[59,60] evidenced that TENS is more effective when applied at adequate intensities to patients with knee osteoarthritis and to manage postoperative pain. When given at inadequate intensities, it is ineffective.

Furthermore, during TENS administration, the patient must often be questioned about the occurrence of sensory habituation. If present, the amplitude must be increased again until the patient feels a strong but comfortable paresthesia.[61] Moreover, Sato et al[62] showed in rats with knee joint inflammation that increasing pulse amplitude by 10% per day of both low- and high-frequency TENS delayed the onset of analgesic tolerance. They also showed that TENS applied with low intensity (e.g., 50% of motor threshold) had no analgesic effect when compared with sham TENS, while TENS applied with high intensity (e.g., 90% of motor threshold) reversed hyperalgesia, confirming that appropriate stimulus intensity is critical for successful TENS application. These results suggest that, in clinical practice, patients should be encouraged to increase TENS amplitude to a strong but comfortable intensity just below pain threshold and continue to increase intensity as tolerated every day.[63]

Caffeine Consumption and TENS

TENS analgesia may be mediated through activation of adenosine A1 receptors in the spinal cord.[64,65] Thus, caffeine, a competitive adenosine receptor antagonist, may interfere with TENS effectiveness. In 1995, Marchand and Charest[66] demonstrated in a placebo-controlled study that caffeine (i.e., a 200-mg pill) could block the analgesic effect of high-frequency TENS in healthy pain-free participants exposed to experimentally induced thermal pain. Nevertheless, a further study investigated the effect of a strong cup of coffee (i.e., 100 mg of caffeine) on response to TENS in healthy human participants experiencing experimentally induced pain.[67] The authors concluded that a single cup of coffee has no effect on hypoalgesia. However, because caffeine is also found in tea, chocolate, energy drinks, soft drinks, and some over-the-counter drugs, monitoring caffeine intake before treatment is essential to maximize the analgesic effect of TENS.

CLINICAL ADMINISTRATION OF TENS

Electrodes

An electrode is a conductive material applied as the interface between a TENS stimulator and the patient's skin. The function of electrodes is to provide an electrical medium to allow passage of therapeutic currents through body tissues.[10] Two common types of electrodes are used in TENS systems: electrodes made of carbon-loaded rubber and self-adhesive electrodes made of woven cloth designed to be of short-term use and disposable. Carbon-loaded rubber electrodes are used with conductive wet gel and are typically held in place with adhesive tape or wraps. Reusable self-adhesive electrodes are designed for use on a single patient and are not used with conductive gel.

More recently, electrode garments have become available. Conduction garments are available as gloves, socks, and sleeves for legs (knee) and arms (elbow).[10] Cowan et al[68] investigated the hypoalgesic effects of high-frequency TENS delivered via a glove electrode compared with standard self-adhesive electrodes. Both electrodes showed similar hypoalgesic effects, giving clinicians another option of electrode type that can be used.

Key Point! The American Physical Therapy Association 2014 Choosing Wisely Campaign addressed the use of biophysical agents, stating that clinicians should not "employ passive physical agents except when necessary to facilitate participation in an active treatment program." In accordance, TENS is often used to overcome or attenuate pain that may otherwise limit or prevent a patient from participating in activities of daily living, occupational tasks, or additional therapeutic activities of the complete patient care plan. In this manner, TENS can effectively be used as part of an "active treatment program."

Size of Electrodes

Electrode size is important because it influences the current density (i.e., current per unit area). Current density is calculated by dividing the amplitude of current by electrode area. Therefore, larger electrodes will result in lower current densities and in a more comfortable stimulation, whereas small electrodes have a higher current density and are more likely to cause discomfort.[69,70] Obviously, the area of a patient's body to be treated must also be considered when choosing electrode size for TENS application. Generally, small electrodes (0.8×0.8 cm) are more comfortable for stimulating superficial nerves (0.1-cm depth) and thin fat layers (0.25-cm depth); larger electrodes (4.1×4.1 cm) are more comfortable for thicker fat layers (2-cm depth) and deeper nerves (1.1-cm depth).[71] Small electrodes are better for selectively activating discrete points such as acupuncture or trigger points, whereas larger electrodes are more useful when larger areas need to be stimulated (e.g., spine pain).[10,72]

Electrode Configurations

To obtain and optimize the desired effects from TENS, appropriate positioning of the electrode is necessary. Electrodes may be positioned in a variety of different configurations, depending on the patient's complaint. The most commonly used form is the positioning of electrodes directly in the painful area—in which the electrodes are placed around or over the painful area. The electrodes may be parallel or crossed (if using two channels). For example, when treating diffuse knee pain, electrodes from two channels can be placed in a crossed manner of the anterior knee. The electrodes can also be positioned over the course of the peripheral nerve responsible for innervating the painful area. Another possibility is to place the electrodes parallel to the spine on the intervertebral foramen to promote the stimulation of nerve roots of the spinal nerves innervating the painful dermatomes of the involved region. Electrodes can also be placed over acupuncture points, motor points on the myotomes related to the area of pain, and trigger points. Ideally, attempts are made to find the best method of electrode placement for each patient. Several examples of electrode positions to treat a variety of pain conditions are shown in Figure 11-7.

Clinical Controversy

Electrodes may be positioned across the spine using one or two channels. The goal in many cases is to cover as much of the painful area as possible (e.g., multiple nerve root levels or facet joints). When attempting to stimulate unilateral nerve roots at multiple levels, the electrodes should be place parallel, not perpendicular, to the spine.

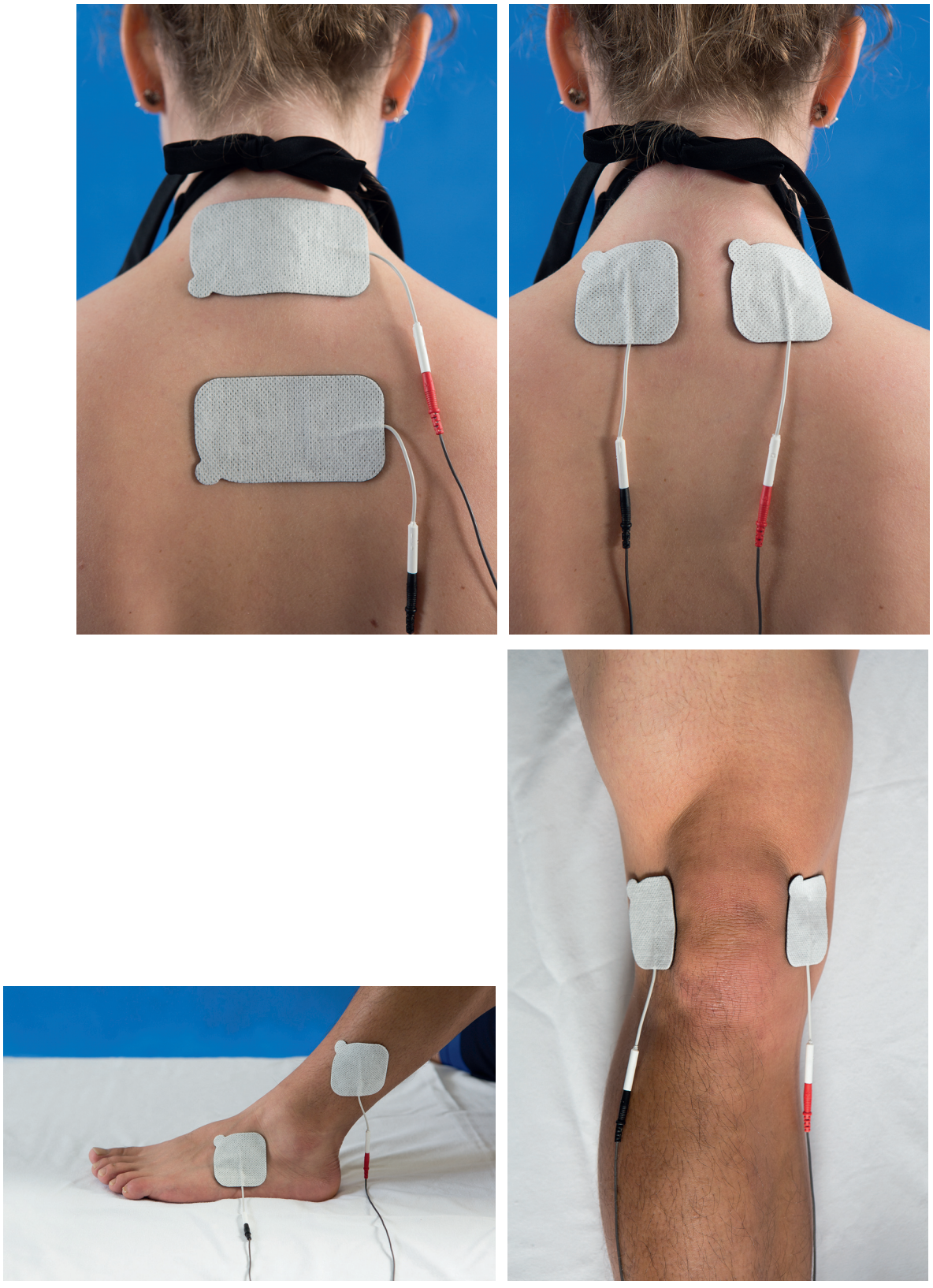

Fig 11 ■ 7 Common electrode configurations for TENS.

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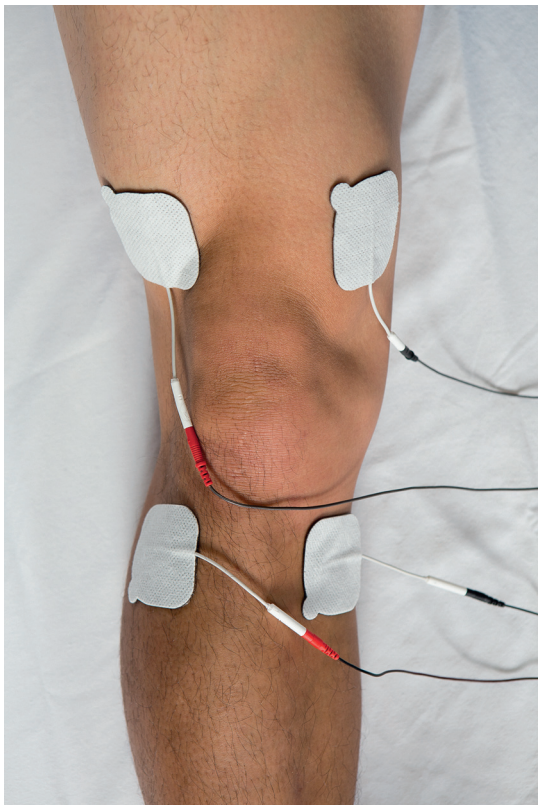

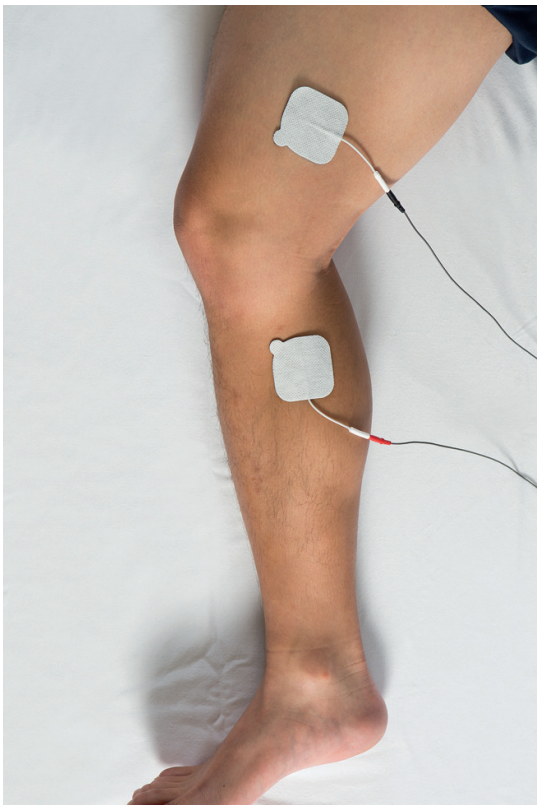

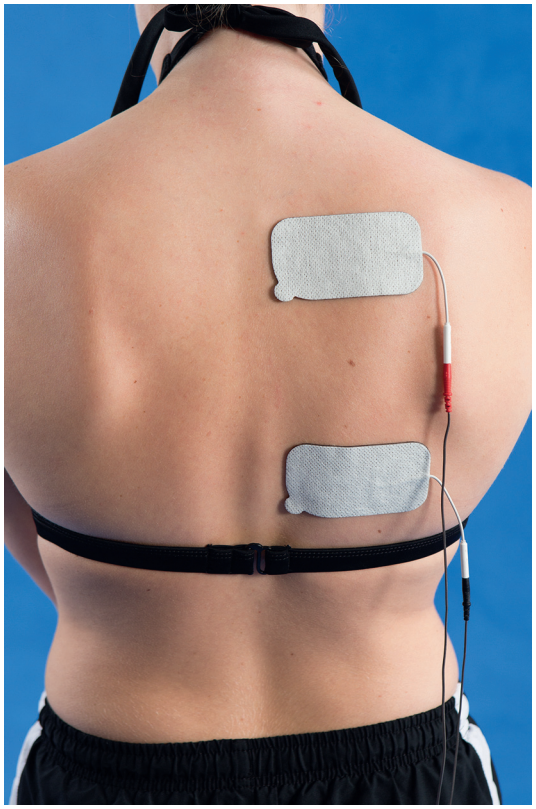

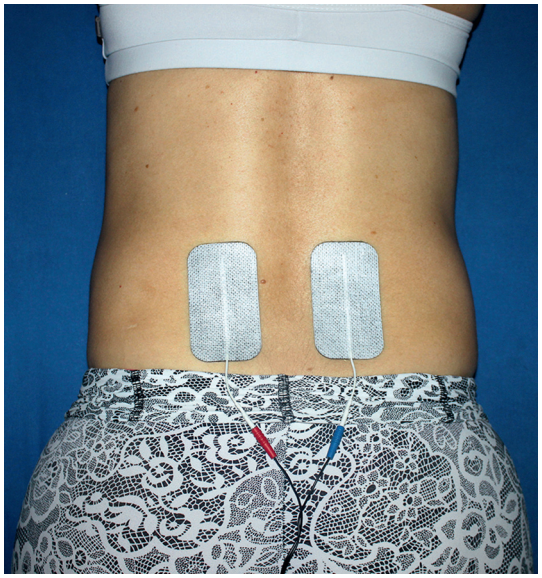

Fig 11 ■ 7—cont'd

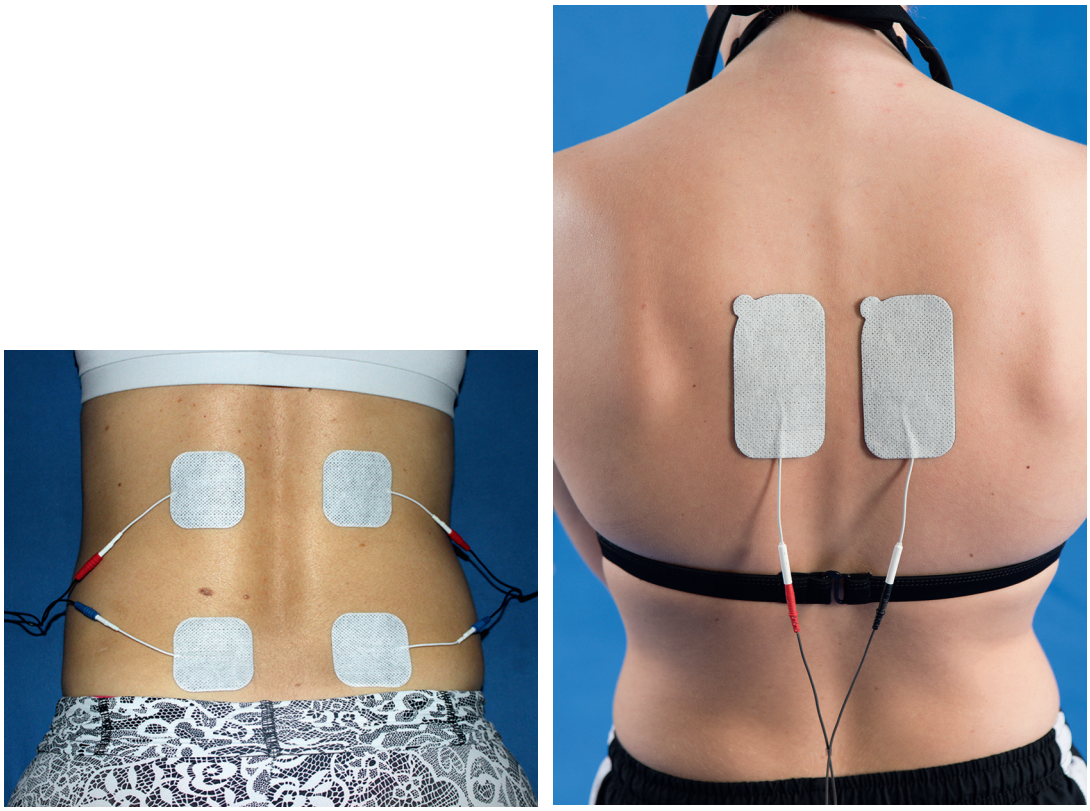

Fig 11 ■ 7—cont'd

Treatment Time

The first TENS treatment should be kept relatively short (i.e., 30 minutes or less) to allow the patient to get used to the sensation and to allow the clinician to monitor the patient's analgesic response and any adverse reactions. After the initial treatment, TENS can be applied for up to an hour at a time.[2] If TENS is being used at home, the patient should be advised to use it as often as required but only for 1 hour at a time to avoid or minimize skin irritation. Taking 30-minute breaks between applications will reduce the likelihood of skin irritation.[2] Cheing et al[73] found that the optimal application time for analgesia in patients with knee osteoarthritis is 40 minutes. The need for relatively long times for effectiveness of TENS has limited its use in rehabilitation clinics. In many countries, patients who respond well to TENS are advised to purchase portable, battery-powered units for home use, thus enabling them to use extended treatment durations. When patients are issued a TENS device for home use, clinicians must instruct the patient in proper skin inspection and protection.

Sequence for Application of TENS

1. If necessary, clean the skin in the region to be stimulated. Alcohol may be used, but for repeated applications, recall that alcohol is a skin desiccant. A mild soap and water will serve the same purpose. Because hair is a poor conductor, removal of excessive hair is recommended to enhance conductivity.
2. Connect the lead wires to the TENS stimulator and then to the electrodes.
3. When using silicon-carbon electrodes, electroconductive gel should be liberally applied across the entire surface of the electrodes that will contact the patient's skin, and the electrodes should fit evenly to the region to be stimulated. If tape or adhesive wraps are used to position the electrodes, the clinician must make sure that the entire surface area of the electrode remains in contact with the skin and is not bowed due to pressure from the tape or wraps. When using adhesive electrodes, the use of gel and tape or wraps is not needed.
4. Adjust the parameters accordingly: pulse frequency, pulse duration, and duration of treatment.

5. Turn the TENS unit on, making sure that the current amplitude is at the minimum value when initiating treatment.
6. Slowly increase the current amplitude until the patient reports the initial onset of a tingling sensation (paresthesia). Continue to increase the current amplitude until the patient reports a strong but comfortable sensation. In the event of sensory habituation (i.e., if the patient reports a decrease in the awareness of the stimulation), adjust the current amplitude back to a strong but comfortable sensation. Adjust the current amplitude to the desired effect (i.e., sensory or motor) based on the desired TENS mode previously described.
7. When treatment time has completed, reduce the amplitude to the minimum and turn off the unit. Remove the electrodes carefully and evaluate the status of the skin. Slight pinkness or pale redness of the skin under the electrodes is common due to the stimulation but should resolve within a few hours. Signs of skin compromise, such as blistering or bright redness, are signs of an adverse event. In this situation, TENS should not be used again until the skin has healed, and thereafter, stimulation parameters should be adjusted to reduce the amount of stimulation delivered to the patient.

ELECTRICAL CURRENTS FOR PAIN MODULATION

Interferential Current

Interferential current (IFC) was developed in the 1950s by Dr. Hans Nemec in Vienna and became increasingly popular in the United Kingdom during the 1970s.[74] IFC is a simple method of nonpharmacological and noninvasive treatment used mainly to induce analgesia,[75–80] produce muscle contraction,[81–83] and reduce edema.[84,85] Currently, IFC is one of the most commonly used electrotherapeutic currents used in rehabilitation.[86] In the United States, IFC is almost exclusively associated with pain modulation despite IFC's ability to be used for other therapeutic purposes.

IFC consists of the transcutaneous application of two medium-frequency (i.e., kilohertz [kHz]), alternating currents to yield a single treatment, interference, or therapeutic current with amplitude modulated frequency typically ranging from 0 to 250 Hz (additional descriptions of IFC are in Chapter 9). The two main advantages historically attributed to IFC are reducing the impedance of the skin, resulting in less discomfort than is usually experienced in other traditional low-frequency currents, and reaching deeper tissues.[85,87] However, there is no evidence to support these claims.[26,88,89]

Adjustable Parameters

Carrier Frequency

Some IFC devices allow the carrier frequency to be adjusted from 1 to 10 kHz. A carrier frequency of 2 kHz is generally used to induce muscle contractions, whereas 4 kHz is used to reduce pain. This is because when a frequency of 2 kHz is used, the phase duration of the waveform is 250 μsec—a phase duration suited to skeletal muscle activation. With a carrier frequency of 4 kHz, the phase duration is 125 μsec—a phase duration less suited to activation of skeletal muscle. Thus, considering the strength–duration relationship,[90] the longer phase duration when using 2 kHz would be more suitable for activating motor nerve fibers, whereas the lesser phase duration when using a carrier frequency of 4 kHz would be more selective for sensory nerve fibers.

However, this recommendation comes from textbooks and equipment manuals. Although recent literature shows greater muscle force production when using greater phase durations,[83,91] only one published study has investigated the analgesic effect when using differing carrier frequencies and IFC. In this study, IFC with a carrier frequency of 1 kHz promoted a greater hypoalgesic response in healthy subjects during and after stimulation than 8 kHz and 10 kHz. However, carrier frequencies of 1 kHz and 2 kHz are perceived as more uncomfortable than 4 kHz, 8 kHz, and 10 kHz.[80]

Amplitude-Modulated Frequency

Historically, amplitude-modulated frequency (AMF), also called *beat frequency,* was claimed to be the main analgesic component of IFC.[74,85,87,92,93] Some authors have advocated that an AMF of 100 Hz is optimal to induce analgesia,[77,94,95] and therefore, this frequency has been frequently used in studies assessing hypoalgesic effects of IFC.[75,77,96–98] However, Johnson and Tabasam[76] reported no difference in the magnitude of change in pain threshold for a range of AMFs on

cold-induced pain in healthy subjects. Gundog et al[99] compared three different AMFs (40 Hz, 100 Hz, and 180 Hz) in patients with knee osteoarthritis and concluded that the effectiveness of different AMFs of IFC was not superior when compared with each other. Another study assessed the influence of AMF on pain induced by pressure in healthy subjects. The authors compared the absence (0 Hz) or presence (100 Hz) of AMF and concluded that the addition of an AMF parameter to interferential therapy did not influence mechanical pain sensitivity in healthy subjects.[86] Therefore, there is insufficient evidence to date to support that AMF has any role in determining the physiological hypoalgesic effects of IFC.

Clinical Controversy

The default setting for the AMF of IFC on most commercially manufactured IFC stimulators is 100 Hz, despite lack of evidence to support or refute the selection of any specific frequency.

Sweep Frequency

On some IFC stimulators, the AMF can be modulated in two basic ways: constant beat frequency mode (static) or modulated beat frequency mode. Modulation of the beat frequency is termed *sweep* (see Chapter 9 for more details on sweep). In the sweep beat frequency mode, the AMF (base treatment frequency) is automatically and rhythmically increased and decreased within a range (spectrum). The primary clinical purpose of sweep frequency is to prevent or decrease sensory habituation; however, there is a lack of studies that investigate the importance of using a sweep frequency for pain control.

Sweep Pattern, Swing Pattern, or Sweep Mode

The temporal characteristics or pattern of the AMF sweep is termed the *swing*. Most machines offer several sweep patterns that can be adjusted by the therapist. A 6∫6 sweep pattern oscillates a base AMF to a peak AMF over a 6-second period. In a 1^5^1 swing pattern, the base AMF is retained for 5 seconds; then all the frequencies in the set spectrum are traversed in 1 second up to the highest frequency, which is then maintained for 5 seconds. Thereafter, the AMF returns in 1 second to the lowest set value. A 1∫1 sweep pattern delivers the base and peak AMFs for 1 second (see Chapter 9 for further description of the sweep and swing modulation of IFC).

Evidence for the selection of one specific sweep (swing) pattern over another is not available. Johnson and Tabasam[75] compared the analgesic effects of different swing patterns of IFC (1∫1, 6∫6, 6^6, or burst) on cold-induced pain in healthy subjects and found no differences in the hypoalgesic effects of using different swing patterns.

Treatment Time

The recommended treatment time for IFC remains a matter of discussion. Debate likely stems from inconsistent durations used in previous studies. IFC is commonly administered for 10 to 20 minutes.[100,101] A recent systematic review of IFC for musculoskeletal pain found that the most common protocols used IFC ranging from 10 to 20 minutes for 2 to 4 weeks for 12 sessions.[102] Nevertheless, more recent studies have applied IFC over 30 minutes.[103,104] Despite these studies, there is no consensus regarding optimal treatment duration for IFC.

Key Point! Despite the unique and sometimes confusing characteristics of IFC, it is simply another waveform that can be used for electro-pain modulation. By adjusting the parameters accordingly, IFC can be a form of TENS. A fixed or static beat frequency can be used to deliver low- or high-frequency TENS. Or, if a sweep pattern from low to high frequency is used, both low- and high-frequency TENS can be administered within the same treatment.

Analgesic Mechanisms of IFC

The most cited analgesic mechanism of IFC is the gate control theory of pain, previously described.[20,85] Other mechanisms—such as increased blood flow, activation of descending analgesic pathways, physiological blockade of nerve conductions, and placebo effect—have also been suggested.[105] However, these mechanisms have not yet been confirmed by experimental and clinical studies and are therefore speculative.[1]

Burst-Modulated Alternating Currents

Alternating currents (ACs) used clinically are normally kilohertz-frequency AC (1–10 kHz), delivered in low-frequency bursts (up to approximately 100 Hz). Therefore, they are called *burst-modulated alternating currents* (BMACs). Russian current (the most widely recognized form of BMAC) and IFC (an amplitude and burst-modulated form of AC) are the most common types of BMAC used clinically. BMAC stimulation has been claimed to be more comfortable than low-frequency pulsed currents. Because skin impedance is inversely proportional to the frequency of AC, claims have been made that BMACs have the advantage of diminishing the impedance of the skin and subcutaneous tissues and therefore can penetrate into the deeper tissues when compared with low-frequency pulsed currents. Although this theory has been widely reported in the literature,[96,106,107] this claim has been recently questioned by some authors[26,88] because skin impedance to pulsed current depends on the phase duration, not the pulse frequency. Thus, if the pulsed current has the same phase duration as the BMAC, the skin impedance would be the same even if the pulse frequency is lower.[61,88] Although the clinical efficacy of BMACs for pain modulation is still under debate, the use of BMACs for pain has increased over time.

A novel type of BMAC, commercially known as *Aussie current,* has been proposed by Professor Alex Ward from Australia. Aussie current uses a carrier frequency of 1 kHz for torque production or 4 kHz for hypoalgesia (Fig. 11-8). A carrier frequency of 1 kHz produces higher torque than 4 kHz, but 4 kHz is more comfortable than 1 kHz and therefore is more often used for pain control. Although conventional IFC typically uses a sinusoidal carrier waveform, Aussie current uses rectangular bursts similar to Russian current (i.e., the amplitude of the burst is constant, not modulated like IFC) (Fig. 11-9).The rationale for using rectangular burst is that wave amplitude is the same throughout the burst duration, while in sinusoidal bursts, some parts of the burst are below sensory threshold while other parts are above. Thus, the effective burst duration for any given nerve fiber is uncertain.[88]

Burst durations of 1 to 4 milliseconds (msec) have been reported to be optimal for both sensory and motor stimulation in humans.[108,109] Burst duration of IFC will vary depending on the AMF chosen. When AMF is set to 100 Hz, burst duration will be 10 msec (i.e., longer than optimal). With this concern in mind, Aussie current uses shorter burst duration (2 msec for torque production or 4 msec for hypoalgesia).

Fig 11 ■8 The Neurodyne Aussie Sport A, an Aussie current stimulator *(Courtesy of Ibramed, Amparo, S.P. Brazil.)*

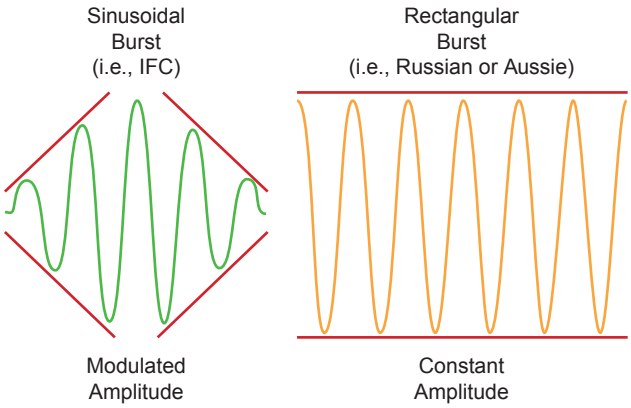

Fig 11 ■9 Variations in burst patterns: sinusoidal versus rectangular.

Ward and Oliver[110] compared monophasic pulsed current with a frequency of 50 Hz and a phase duration of 500 μsec with BMAC (1-kHz alternating current, burst modulated at a frequency of 50 Hz with a 20% duty cycle) to establish whether there were differences in hypoalgesic efficacy as assessed by cold pain threshold measurements. The authors found that both were equally effective at elevating the cold pain threshold.

Ward et al[111] compared BMAC (4-kHz AC applied in 4-msec bursts at 50 Hz) and TENS (125-μsec phase duration applied at a frequency of 50 Hz) efficacy for experimental cold pain threshold. Both generated

significant changes, though they were not significantly different from each other. The authors suggested, however, that as the BMAC stimulation generated less discomfort than the TENS in previous studies, there may be a clinical advantage to its use, and it should be considered as a clinical option.

There are no studies to date investigating the mechanisms of action of BMACs for pain control, but BMACs may work in the same manner as low-frequency pulsed currents delivered by standard TENS devices.

Clinical Controversy

Microcurrent is defined as current with an amplitude below 1 mAmp, thus in the micro-amperage range. This waveform continues to be advertised for electro-pain modulation despite any evidence that supports its use for pain.

CONTRAINDICATIONS AND PRECAUTIONS FOR TENS

Most contraindications to TENS or other forms of electro-pain modulation are hypothetical and based on what should be considered fundamental clinical knowledge. The reality is that relatively little consensus about contraindications and precautions exists in many electrotherapy texts and guidelines proposed by different associations. Furthermore, there is much disagreement in the designation of what is considered a precaution or a true contraindication. For example, cancer routinely appears as a precaution if not a contraindication, despite some evidence for the use of TENS in the palliative care of cancer-related pain (Table 11-4). Greater delineation of the patient-specific case should guide the clinician in determining the appropriateness of TENS.

Perhaps the most comprehensive resource identifying precautions and contraindications to TENS was compiled by Houghton et al[112] in 2010. According to these authors, precautions are situations in which a patient is at some risk of experiencing an adverse event with administration of TENS, whereas contraindications are those specific situations in which TENS should not be used because it may be harmful to the patient. They further classified some situations as local contraindications, indicating scenarios in which the application of TENS over a specific location or region of the body could be harmful and thus TENS should not be used at this location/region.

The following are recognized precautions (P) or contraindications (C) to the use of electro-pain modulation or TENS:

- Undiagnosed pain (P): Masking undiagnosed pain with TENS can postpone proper treatment and lead to worsening of the underlying condition.
- Pacemaker or other active implants (C, local): TENS has been shown to interfere with the functioning of certain types of cardiac pacemakers and active electric

TABLE 11–4. Clinical Studies on TENS for Cancer-Related Pain

Reference	Type of Cancer Pain	n	Results
Avellanosa & West, 1982[115]	Mixed types of cancer	60	28.3% = excellent response 36.2% = fair response 35% = no relief
Robb et al, 2007[116]	Chronic pain following breast cancer treatment	41	TENS was no better than placebo.
Searle et al, 2009[117]	Cancer bone pain	1	Pain at rest decreased from 4 to 1 and pain during movement decreased from 7 to 2 measured by a 0–10 numerical rating scale.
Bennett et al, 2010[118]	Cancer bone pain	24	TENS may have benefits on movement-related pain.
Loh & Gulati, 2013[119]	Sarcoma cancer pain	8	Seven out of 8 patients had a qualitative or quantitative reduction in their sarcoma-related pain.
Loh & Gulati, 2015[120]	Mixed types of cancer	84	TENS provided positive benefits in cancer patients.

n = number of subjects.

implants. Therefore, its use should be avoided over or close to these areas. A study by Rasmussen et al[113] suggested that most patients with a permanent cardiac pacemaker could safely use TENS providing it is not applied over the chest. In this particular study, TENS was applied to the lumbar area, cervical spine, left leg, and lower arm area ipsilateral to the pacemaker in 51 patients. No adverse effects were found in any of the 20 models of pacemaker involved in the study. Chen et al[114] described two case studies where pacemaker function was assessed by electrocardiogram (ECG) and Holter monitor. TENS was applied to the upper back/lumbar area, right side posterior neck, and right shoulder. In both cases, ECGs during the TENS trial did not reveal cardiac pacemaker dysfunction, but extended cardiac monitoring with the Holter monitor showed interference with pacemaker function; however, neither patient reported any cardiac symptoms during these episodes. The abnormalities did not recur after decreasing the sensitivity of the pacemakers. Therefore, it would be advisable to discuss the situation with a cardiologist and perform an initial trial with ECG/Holter monitoring when a therapist is considering administering TENS treatment to a patient with cardiac pacemaker.

- Pregnancy (C, local): The effects of passing an electrical current through the uterus of a pregnant woman have not been determined. Because there is no evidence showing that TENS application is safe during pregnancy, electrodes should not be placed on the abdomen, pelvis, low back, or hips, unless TENS is used for labor pain. The major risk may be during the first trimester of pregnancy because TENS could induce labor or harm the developing fetus.
- Carotid sinus (C): The carotid sinus is located at the origin of the internal carotid arteries, which contain baroreceptors that detect changes in blood pressure. Placement of electrodes over the anterior neck should be avoided because stimulation of the carotid sinus may cause a drop in heart rate and blood pressure. Moreover, TENS administration in this area can stimulate the vagus nerve and phrenic nerve and cause a laryngeal spasm.
- Damaged skin (C, local): The lower skin impedance of damaged skin will result in a high current flow through a small area, causing discomfort or pain and possible further damage.
- Lack of normal skin sensation (C, local): Placing electrodes over skin that has deficient sensation can result in the use of excessively high stimulus intensities, which may cause skin irritation and even an electrical burn. Moreover, treatment will be ineffective if the appropriate afferent nerves are not stimulated. If sensation is absent in a specific area, the electrodes may be placed proximal in an area with intact sensation.
- Patients with impaired cognition or who do not comprehend the clinician's instructions (C): These patients should not be treated with TENS. If patients are required to operate a TENS unit themselves, they should be cognitively competent at doing so safely.
- Thrombosis or thrombophlebitis (C): Electrodes should not be placed on a limb affected by a venous or arterial thrombosis or thrombophlebitis because TENS may increase circulation, increasing the risk of dislodging a thrombus and leading to embolism.
- Hemorrhage (C): Patients with current or recent hemorrhage should not be treated with TENS because it may increase blood flow and cause further bleeding.
- Malignant tumors (C, local): TENS should not be administered over malignant tumors because risk of affecting cancer cell growth or metastasis is unknown.
- Active epiphysis (P): TENS and electrical stimulation should be avoided over areas of active epiphysis in children. This is considered a precaution because there are no studies showing the effects of TENS in this area.
- Eyes, internally, and on reproductive organs (C): TENS electrodes should never be placed over the eyes, internally (e.g., in the mouth), or on reproductive organs because the effects of electrical current on these areas are unknown.

CASE STUDY 11-1 TENS With Chronic LBP and Use of Opioids

ADH is a 35-year-old female housekeeper who developed a posterolateral disc herniation at the L4/L5 level. Over the past 3 years, she has had moderate low back pain that radiates down the back and through the buttock to the left leg. Her pain increases during spinal flexion or when she remains seated for long periods of time. She has been taking opioids to control pain during the past 4 months. However, she is experiencing unwanted opioid-related side effects and the efficacy of medication has been decreased over the last month. The clinician's main goal is to decrease pain and reduce opioid dosage. In addition to manual therapy techniques and exercises, the clinician recommends home use of TENS.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of TENS?
ANSWER: Yes, the patient presents with radiating low back pain into the buttock and leg.

2. Is the patient appropriate for TENS (i.e., do any of the general precautions or contraindications to TENS apply to the patient or are there any specific considerations regarding application of TENS to this patient)?
ANSWER: Although there are not specific contraindications to the use of TENS, the patient's history notes use of opioids with signs of analgesic tolerance. As most opioid drugs are μ-receptor agonists, this type of receptor would have developed tolerance, which can be observed by the decrease of drug effectiveness. Consideration for type of TENS used should be noted in light of concurrent opioid use.

3. What are the specific goals to be achieved with the use of TENS?
ANSWER: TENS may be used to alleviate this patient's moderate radiating pain and to assist in weaning from opioid use.

4. What specific form of TENS would be appropriate for the patient?
ANSWER: A portable handheld battery-operated TENS device is preferred so the patient may continue use of TENS independently at home as part of her complete rehabilitation plan.

5. What specific parameters of TENS are appropriate for the patient?
ANSWER: An appropriate plan of care would be to initiate TENS using the following parameters:
- TENS mode: continuous (high frequency)
- Pulse duration: 100–200 μsec
- Pulse frequency: 80–100 Hz
- Current amplitude: strongest comfortable intensity
- Electrode type: self-adhesive, reusable electrodes
- Electrode number: four (two channels of stimulation)
- Electrode size: 50 × 50 mm
- Electrode setup: placed over areas of pain in left paravertebral region and over the course of the sciatic nerve.
- Treatment time: 60 minutes
- Treatment sessions per day: as often as needed
  - *Rationale:* High-frequency TENS should always be tested as a first option. In this case, the patient has been taking opioids for 4 months and presents with signs of analgesic tolerance. Because most opioid drugs are μ-receptor agonists, this type of receptor would have developed tolerance, which can be observed by the decrease of drug effectiveness. Low-frequency TENS activates μ-opioid receptor and will not be effective. On the other hand, high-frequency TENS activates δ-opioid receptors that have not developed tolerance.

6. What are the appropriate and safe application procedures for home TENS?
ANSWER: The clinician should instruct the patient in proper use of TENS during a supervised clinical session. Instruction should address manipulation of the stimulator and all parameters. The patient should also be instructed in proper electrode placement and care to extend their use, and the patient should be advised to inspect the skin under the electrodes before and after each application.

CASE STUDY 11-2 Interferential Current for Cervical Pain

YRF is a 50-year-old male who was driving his car 2 days ago when he was hit from behind. He suffered a whiplash injury to the neck, resulting in neck pain 8/10 with referred pain to the shoulders and severe muscle-guarding spasm. X-rays showed no signs of vertebral fracture. The clinician's main goal is to decrease pain and muscle spasm and restore the cervical spine range of motion (ROM). The clinician decided to use IFC to alleviate pain and spasm so that the patient may engage in the additional components of the complete rehabilitation plan.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of electro-pain modulation?

ANSWER: Yes, the patient presents with acute soft tissue injury to the cervical region with peripheral radiation into the shoulders.

2. Is the patient appropriate for electro-pain modulation (i.e., do any of the general precautions or contraindications to electro-pain modulation apply to the patient or are there any specific considerations regarding application of electro-pain modulation to this patient)?

ANSWER: The patient presents with musculoskeletal symptoms that may be attenuated with the use of electro-pain modulation. Because this patient is not concurrently using opioid medication, the concern for cross-tolerance is not present.

3. What are the specific goals to be achieved with the use of IFC?

ANSWER: IFC may be used to alleviate this patient's cervical and radiating shoulder pain so that he may complete additional components of his rehabilitation plan such as ROM exercises.

4. What specific parameters of IFC are appropriate for the patient?

ANSWER: An appropriate plan of care would be to initiate IFC using the following parameters:

- Carrier frequency: 4 KHz
- AMF: 120 Hz
- Sweep frequency: 60 Hz
- Sweep pattern: 6∫6 seconds
- Vector: scan or dynamic
- Current amplitude: strongest comfortable intensity
- Electrode type: auto-adhesive
- Electrode technique: quadripolar (two channels of stimulation) with electrodes placed bilaterally over the mid-cervical spine and upper thoracic spine in a square or rectangular manner so that the involved tissues are within the area of the electrodes. The two channels should be crossed so that the interference current is targeted within the boundaries of the four electrodes.
- Electrode size: 50 × 50 mm
- Treatment time: 20 to 30 minutes
- Treatment sessions per day: one, during clinic visits. IFC should be applied before manual therapy and/or exercises so that these treatment modalities can be performed with less pain or discomfort to patient.
  - *Rationale:* Carrier frequency of 4 kHz is usually used for analgesia because it is more comfortable than lower carrier frequencies. Despite the lack of evidence supporting specific AMF values for each injury phase, it has been suggested by manufacturers to set high AMF values (approximately 120 to 150 Hz) for acute pain. Sweep frequency is activated to minimize sensory habituation. A sweep pattern of 6∫6 is often used for acute injuries because it changes the AMF values slowly, providing a subtle stimulus variation. Dynamic vector or vector scan is activated to cover a larger treatment area. IFC is usually applied for shorter periods of time (about 20 min) when compared to traditional TENS due to time constraints in the clinical setting. Although presently there are some portable IFC units available on the market, most units are large and expensive, restricting their use to outpatient departments.

5. What are the appropriate and safe application procedures for IFC?

ANSWER: The clinician should also inspect the skin under the electrodes before and after each application. Before administration, the clinician should educate the patient on the expected sensation of the IFC.

Documentation Tips

Appropriate documentation of TENS application should include the following:

- Mode of TENS
  - Conventional, acupuncture, burst train, brief intense
- Waveform type
  - Biphasic (symmetrical or asymmetrical and balanced or unbalanced), monophasic, BMAC, etc.
- Waveform parameters
  - Pulse duration and frequency
- If interferential current
  - Sweep and scan
  - Carrier frequency and beat frequency
- Level of stimulation intensity
  - Sensory, motor, or noxious
- Electrode
  - Type, shape, size, and number
  - Placement or location
  - Integrity of skin before and after treatment
- Patient position
- Treatment duration
  - Number of contractions or duration of treatment

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CHAPTER 12

THERAPEUTIC MODALITIES FOR IMPROVING RANGE OF MOTION

Andrew Starsky, PT, PhD

Loss of range of motion (ROM) can occur from a variety of causes, including but not limited to pain, immobilization, edema, spasticity, and decreased muscle strength. Although many treatment strategies may be used to restore or improve ROM, therapeutic modalities represent one such strategy. The use of biophysical agents, such as cold, heat, and ultrasound, is appropriate in many cases for addressing limited ROM. Likewise, electrical stimulation in the form of transcutaneous electrical nerve stimulation (TENS) for pain relief or neuromuscular electrical stimulation (NMES) for decreasing spasticity and activating skeletal muscle may be useful for improving ROM. While the physiological mechanisms and clinical administration of each of the biophysical agents have been presented in greater detail in earlier chapters of this text, this chapter presents the use of modalities as part of the active treatment plan for increasing ROM due to limited joint mobility. Therefore, the intent of this chapter is to show the clinician how to effectively use therapeutic modalities to increase ROM.

CLINICAL REASONING

Limited ROM can affect the efficiency of many tasks, including gait and activities of daily living. For example, lack of full knee extension can decrease step length in gait, making ambulation less efficient. Lack of elbow extension may make reaching difficult. Limited wrist mobility can affect upper extremity activities. This limited motion can be a result of injury, immobilization, or arthritis. A thorough assessment of motion will enhance

the development of an appropriate plan of care, which will include the judicious and careful use of modalities. Which modality to use is based on the examination findings as well as an understanding of the course of injury recovery.

SOURCES OF LOSS OF MOBILITY AND RANGE OF MOTION

Many musculoskeletal and neuromuscular disorders result in limited active and passive ROM. The main contributors to limited ROM include pain, edema, arthritic joint stiffness, and joint contracture. Neurologically, spasticity can also be an impairment that may lead to limited ROM. These will be examined in detail, and the appropriate interventions for each will be discussed.

Joint Pain as a Limiter of ROM

Pain has been discussed extensively in this text. Pain with joint movement can cause the patient to avoid movements that cause pain, thus not taking the joint through its entire ROM, either actively or passively. This will lead to limited ROM, typically within a few days. Pain can also trigger muscle spasm, which serves to further minimize joint movement. An example of this is adhesive capsulitis of the shoulder, which may have started due to tissue injury and the pain-spasm-limited-ROM cycle. Any modality—such as TENS, cryotherapy, and deep or superficial heat—that decreases pain can be useful for these situations (Fig. 12-1). It is optimal to perform these modalities before or concurrent with active interventions to gain the most ROM. Greater details regarding physiological mechanisms and clinical applications of TENS, cryotherapy, and deep or superficial heat are found in the respective chapters for each modality in Section II of this text.

Key Point! When using biophysical agents to address ROM that is limited by pain, it is recommended that ROM activities occur during or immediately after application of the modality when the pain is decreased.

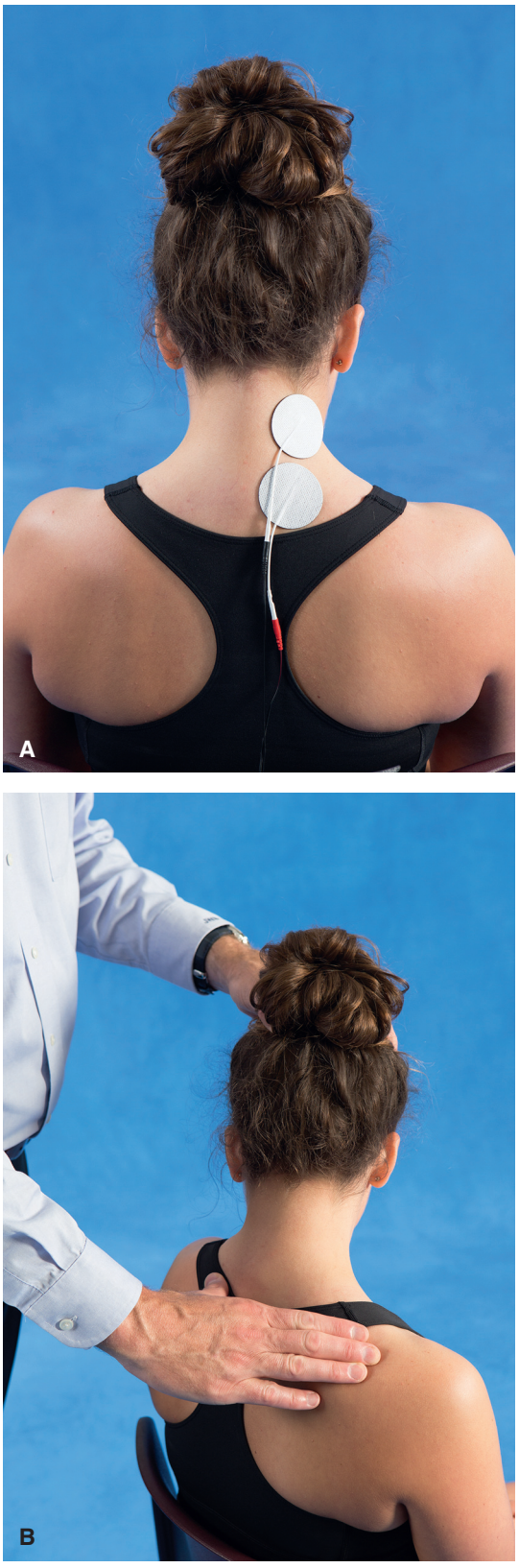


Fig 12 ■ 1 The use of TENS, among other biophysical agents, may be used to (A) reduce pain prior to (B) stretching and ROM activities.

Edema After an Injury

Fluid leaking into the interstitium can increase the fluid volume of a limb. This fluid could also leak into the joint space, which would likely activate nociceptors around the joint. All of these could limit motion. Chronic edema can lead to fibrosis of the tissue, which could also limit ROM. Ideally, however, edema would be addressed before it reaches the chronic state to minimize the risk of fibrosis. Edema can be addressed acutely with cryotherapy and compression and can be addressed chronically with either electrically stimulated muscle contractions that create a pumping action or with manual therapy. Greater details regarding physiological mechanisms and clinical applications of cryotherapy, compression, and electrical stimulation for edema are found in the respective chapters for each modality.

Joint Stiffness Associated With Arthritis

Both osteoarthritis and rheumatoid arthritis can lead to breakdown of joint surfaces and shortening of periarticular structures of the joint. Osteoarthritis (OA) of the knee, for instance, is present in approximately 13% of women and 10% of men over age 60.[1] This type of arthritis can result from normal joint stresses or from trauma. Patients with osteoarthritis will display decreased joint space and osteophyte infiltration, both of which can decrease joint motion. Because the process of OA is not generally associated with inflammation, heating modalities, such as continuous ultrasound or short-wave diathermy, may be used to warm restricted tissues before or during active stretching or ROM interventions. Loss of knee extensor strength because of decreased activity in a patient with OA of the knees may be addressed through the use of neuromuscular electrical stimulation to the quadriceps.

Rheumatoid arthritis (RA) affects approximately 1.3 million people in the United States.[1] RA is a systemic autoimmune disorder that is characterized by inflammation of the synovium of the joint capsule and the lining of tendon sheaths. Modalities may be used to treat the loss of ROM due to arthritis if there is an intact and stable joint; however, thermal modalities must be used with caution to avoid overheating already inflamed tissues. For RA, cold modalities and nonthermal ultrasound may be used to facilitate improved ROM and active functional ability. Greater details regarding physiological mechanisms and clinical applications of cryotherapy and thermotherapy, diathermy, and electrical stimulation are found in the respective chapters for each modality. Box 12-1 shows intervention goals for patients with OA or RA.

Joint Contracture as a Result of Injury and/or Immobilization

Numerous injuries and conditions can result in a loss of ROM secondary to joint contracture and soft tissue shortening/adhesions (Box 12-2). After almost any orthopedic surgery, such as knee replacement, rotator cuff repair, Achilles tendon repair, or amputation, a loss of joint motion and joint contracture is possible. This is partially attributable to the inflammatory and proliferative response from the surgery but can also be attributed to the period of relative immobilization after the surgery. There can be structural changes in the length of collagen fibers, a disorganization of these fibers, and a reduction

Box 12 ▪ 1 Potential Intervention Goals for Patient With Arthritis

- Decrease joint swelling.
- Decrease pain.
- Decrease joint stiffness.
- Increase functional mobility.
- Increase muscle strength.
- Increase ROM.
- Promote weight loss with increased physical activity.

Box 12 ▪ 2 Conditions Associated With Joint Contracture and Soft Tissue Shortening

- Burns
- Fracture and associated immobilization
- Osteoarthritis
- Rheumatoid arthritis
- Prolonged inactivity and/or immobilization
- Scar tissue formation following injury
- Spasticity
- Muscle spasm
- Tendon injury and associated immobilization

in the water content of the glycosaminoglycans in the tissues.[2] It is usually the clinician's goal to prevent this contracture entirely, but if it is not possible, modality interventions may be applied in conjunction with stretch to restore motion. Modalities that might be appropriate for this condition typically include superficial or deep heat either before or in conjunction with stretching.

Key Point! When decreased ROM is due to joint contracture, it is recommended that stretching of the involved tissues occur during and immediately after application of the thermal agent.

Loss of ROM Secondary to Spasticity

Spasticity is a common impairment that results from a central nervous system insult such as a stroke, spinal cord injury, cerebral palsy, or multiple sclerosis. It has been defined as "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes … resulting from hyperexcitability of the stretch reflex."[3] Muscle spasticity is attributed to a lack of inhibition to either the alpha motor neurons of the spastic muscle or lack of inhibition to the gamma neurons that control its stretch sensitivity. The prevalence of spasticity in the first year post-stroke is 17% to 39%.[4] Spasticity of a muscle makes it difficult to stretch that muscle, sometimes resulting in muscle and tendon shortening. This could then be treated as any loss of ROM noted previously. Typical treatment of spasticity includes stretching, splinting, and serial casting. Spasticity can also be treated medically, with botulinum toxin (Botox) being a common agent to temporarily and partially paralyze the spastic muscle. Botox works by preventing the release of acetylcholine from presynaptic neurons of motor nerve endings at the neuromuscular junction.[5] NMES used in conjunction with Botox or alone has been shown to be helpful in reducing spasticity.

Much discussion and disagreement persists on the use and efficacy of therapeutic modalities for spasticity. Common treatments continue to include applying cold agents (i.e., icing) for a prolonged period directly to spastic muscles to reduce muscle temperature prior to ROM activities, stretching, and electrical stimulation. Three clinical methods of using electrical stimulation have been proposed for treating spasticity. The first method is administration of motor-level electrical stimulation over the antagonistic muscle to elicit contractions, a mechanism known as *reciprocal inhibition.* A second method is applying motor-level stimulation directly to the spastic agonist muscle to induce fatigue. The third method is applying sensory-only stimulation to the spastic muscle, a method used to induce sensory habituation and then decrease spasticity (see Chapter 14 for more details).

ASSESSMENT OF LOSS OF MOTION

Assessing the loss of motion secondary to tissue and structural changes can be very basic. As part of a comprehensive evaluation, assessment of active and passive ROM is essential.[6,7] This assessment of motion will be the framework for deciding if a therapeutic agent can or should be used to improve ROM and which one would be most appropriate.

For example, a patient who has undergone immobilization following an ankle fracture is likely to demonstrate limited ROM in dorsiflexion. However, further evaluation is necessary to differentiate the source of restriction and thus guide clinical decision-making regarding use of biophysical agents. Goniometric measurements of both active and passive ROM can quantify the magnitude of ROM loss. Manual assessment can be used to identify if the loss of motion is more the result of soft tissue restrictions or changes in articular alignment following the fracture. Loss of ROM due to restrictions of soft tissues, such as the Achilles tendon, can be effectively addressed with therapeutic modalities, whereas compromised ROM due to articular configuration may require other intervention strategies. Other suggested clinical tests and measures for motion are outlined in Table 12-1.

INDICATIONS AND EVIDENCE FOR USE OF THERAPEUTIC MODALITIES TO INCREASE ROM

Heat or Cold

Appropriate use of typical heating modalities to restore ROM is well documented in the research. Superficial (i.e., hot packs) and deep (i.e., continuous ultrasound and short-wave diathermy) heat are commonly used either before or during stretching to increase joint motion. These modalities can also be appropriate to heat up a joint or other tissue before joint or soft tissue mobilization.

Applying heat to these tissues alters its viscoelasticity, allowing it to be stretched with less applied force. This heat

TABLE 12–1. Assessment Tools for Motion: Differentiation of Tissues Causing Limited Motion

Motion-Limiting Factor	Technique for Assessment
Scar tissue	• Describe external scar (keloid, hypertrophic) • Scar self-assessment
Edema	• Limb girth measurement • Figure-eight measurement (for foot/ankle and hand/wrist) • Volumetric water displacement
Muscle length	• Examining joint motion with the muscle taut or slackened
Active insufficiency (muscle weakness or loss of tendon excursion)	• Comparing active and passive ROM
Passive insufficiency (joint contracture)	• Comparing active and passive ROM • Assessing joint accessory motions
Spasticity	• Ashworth score • Instrumented torque assessment

also increases the extensibility of the tissue, allowing it to be more easily elongated and plastically deformed. *Plastic deformation* refers to a permanent change in tissue length that lingers even when the stretching force is removed (typically it is desired to make the tissue longer). Early literature that examined these changes in frog and rat tissues shows that the tissue temperature must be increased to 104°F (40°C) or greater to facilitate these gains.[8–11]

A clinician may use superficial heat in the form of hot packs, paraffin, and Fluidotherapy. Hot packs, whether applied in the clinic or at home, remain popular because they are easy to apply and economical. Studies have shown that application of superficial heat in conjunction or with static stretch improves the ROM at the hip, ankle, and shoulder.[12–15]

One study found that the use of ice (specifically ice bags) to the hamstrings while applying static stretch yielded a larger increase in hamstring flexibility compared to stretching alone or stretching combined with superficial heat (hot packs).[16] It was hypothesized that a decrease in pain or depression of the stretch reflex resulted in improved muscle stretch. Perhaps this may be an appropriate intervention when a patient is unable to tolerate heating modalities.

Deep-heat modalities, such as continuous ultrasound and short-wave diathermy, may be more appropriate at heating tissues that contribute to loss of motion.[17] These modalities can raise muscle and tendon temperature at least 39°F (4°C), the threshold for eliciting plastic deformation.[18] Ultrasound has been shown to decrease the tissue stiffness of trigger points in the upper trapezius as measured by pressure algometry.[19] This may have been due to tissue changes or decreased pain.

After the tissue is heated, it is deemed most appropriate to apply a stretch immediately.[20] The idea of a "stretch window" has been established, demonstrating that muscle temperature stays above 39°F (4°C) for about 3 minutes after application of 3 MHz ultrasound (US).[21] It is hypothesized that the application of 1MHz US to deeper tissues may increase the duration of this stretch window because the superficial tissues may serve as an insulator to decrease the rate of heat loss.

Consideration must also be given to the magnitude and duration of the stretch that is applied with adjunctive heat modalities. Although permanent tissue plastic deformation can likely be obtained with a very long duration of stretch (hours or days) without concurrent use of thermal modalities, studies have shown that heat in conjunction with a prolonged stretch can induce changes in clinically measured flexibility.[22] Similar results have been found for increasing flexibility in the plantar flexors.[23] These studies and others support the use of thermal modalities for improving tissue extensibility in contrast to use of no thermal agent at all.

Other therapeutic interventions besides passive stretch, when combined with heat modalities, may be appropriate as well. Moderate evidence exists in the literature that supports techniques such as joint mobilization, exercise, and splinting to help improve ROM. Box 12-3 details other strategies to improve tissue length.[24,25]

Box 12 ▪ 3 Management of Joint Contractures

- Heat concurrent with or followed by stretch
- Joint mobilization
- Active and passive ROM exercises
- Casting at end range
- Low-load, prolonged stretching with dynamic splinting device
- Static progressive stretching with device
- Surgical release/lengthening of tissues

CASE STUDY 12-1 Treatment of Adhesive Capsulitis

The patient is a 21-year-old female diagnosed with adhesive capsulitis of the right shoulder. The patient presents with limitations in flexion, abduction, and external rotation. Palpation also reveals tightness in the muscles and joint capsule of the anterior shoulder, both of which are likely contributors to the decrease in ROM.

CLINICAL DECISION-MAKING

1. Does the patient have dysfunction or impairment that may be improved or lessened by the use of ultrasound?
ANSWER: Yes, tightness in the joint capsule and anterior shoulder musculature might benefit from the use of this modality.

2. Why would ultrasound be a consideration for this patient?
ANSWER: The thermal effects of ultrasound, either coupled with or followed by passive stretch, would be appropriate to increase tissue elasticity and allow for plastic deformation of these anterior shoulder tissues.

3. What specific outcome measures can be used to assess the effectiveness of the agent?
ANSWER: Active and passive ROM should be measured pre- and posttreatment. A shoulder-specific or patient-specific outcomes measure could be used as well.

4. Are there any precautions or contraindications to consider with this patient?
ANSWER: Please refer to Chapter 4.

5. What specific parameters are recommended for this patient?
ANSWER: Because these tissues are superficial, 3 MHz would be the frequency. Thermal effects are desired; therefore, a 100% duty cycle would be appropriate. A treatment time of 8 to 10 minutes at an intensity that produces gentle heat would be optimal. Performing the ultrasound with the patient in a position of end-range abduction and external rotation would be optimal as well (Fig. 12-2A). The ultrasound treatment would be followed by manual stretching (Fig. 12-2B).

Fig 12 ■2 Thermal ultrasound (A) applied with the shoulder followed by (B) manual stretching into external rotation.

6. What are the proper steps to using ultrasound for this patient?
ANSWER: First, assess the presence of any contraindications or precautions. Inspect the skin and position the patient appropriately. Perform the ultrasound with the parameters noted earlier, moving the limb into even more abduction and external rotation during the treatment. Finish by inspecting the skin and performing passive stretch. Remeasure ROM after treatment.

Electrical Stimulation

Electric stimulation may also be an appropriate modality to improve ROM. If ROM is limited by pain, TENS before or during stretch can be useful.[26] If ROM is limited by spasticity, then NMES may be helpful in temporarily decreasing the spasticity, thus allowing better passive stretch and gains in ROM.[27,28] If ROM is limited by other mechanisms, NMES set up in an alternating pattern to the limits of range may be useful.

Stevens-Lapsley et al[29] performed a randomized controlled trial on subjects after a total knee arthroplasty. Out of 66 subjects, half received standard rehabilitation plus NMES to the quadriceps muscle. The subjects performed 15 strong yet tolerable contractions, twice per day for 6 weeks, initiated 48 hours after surgery. Parameters for this protocol are listed in Table 12-2. Subjects would apply the stimulation with the knee in approximately 60° of flexion. At 3.5 weeks, there was significantly greater active knee extension in the NMES group as compared to the control group. Interestingly, the subjects never applied stimulation in a fully extended position, yet were able to actively achieve greater extension.

A similar protocol has been used in the treatment of spasticity. Wright and Granat[30] utilized NMES with parameters similar to those used by Lapsley et al[29] in a single subject with hemiplegic cerebral palsy (CP). After only 2 weeks of 30 minutes daily home NMES, the subject gained almost 50° of active wrist extension. Similar protocols have been used with spastic subjects post-Botox as well.

For ROM that may be limited in both flexion and extension, NMES on both muscles may be applied. For example, in a subject with limited wrist flexion and extension, one channel of NMES electrodes may be placed on the wrist extensors with the other channel on the wrist flexors. The stimulation would then be set with parameters noted in Table 12-3.

Stimulation would begin with the wrist in a neutral position. Then the extensors would be stimulated to achieve maximum tolerable extension (Fig. 12-3A). The second channel is then set to achieve maximum flexion (Fig. 12-3B). This protocol then will take the joint through full ROM. The subject is instructed to activate the muscles volitionally as tolerated, and fatigue is carefully monitored. A similar protocol can be used for the elbow that displays limited ROM. One channel of NMES would be applied to the triceps and set to achieve the maximum tolerable elbow extension, while the other channel would be set to achieve the maximum tolerable flexion (Fig. 12-4A and 12-4B). As pictured, an exercise skate (Patterson Medical, Warrenville, IL) on a firm surface will assist in the movement of the joint. The subject is encouraged to assist with the motion as tolerated.

Another option for joint ROM is to utilize TENS. In a randomized, placebo-controlled trial by Blum et al[26] involving 22 subjects, half the subjects utilized conventional TENS after rotator cuff reconstruction. Subjects were instructed to utilize the device for 1 hour, twice per day, for 90 days beginning on the day of surgery. Two channels were used: One pair of electrodes was applied at the superior angle of the scapula and the medial deltoid; the other pair was applied at the start and end of the surgical incision. Forty-five days postoperatively, the subjects who

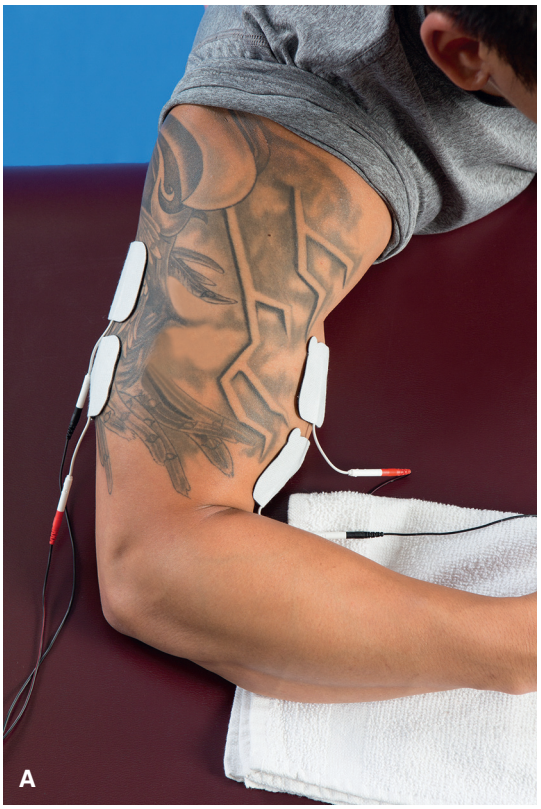
TABLE 12–2. Parameters for Stimulating a Single Muscle to Improve ROM[29]

Parameter	Setting
On-time	15 sec (includes 3-sec ramp-up)
Off-time	45 sec
Pulse duration	200–600 μsec
Frequency	50 pulses per sec
Waveform	Russian, Aussie, balanced symmetric biphasic
Treatment time	30 to 60 min at least every other day

TABLE 12–3. Parameters for Stimulating Two Muscles to Improve ROM

Parameter	Setting
On-time (each channel)	10 sec (includes 3-sec ramp-up)
Off-time (each channel)	10 sec
Pulse duration	200–600 μsec
Frequency	35 to 50 pulses per sec
Waveform	Russian, Aussie, balanced symmetric biphasic
Mode	Alternating (only one channel is active at a time)
Treatment time	30 to 60 min at least every other day

Fig 12 ■ 3 Elicited muscle contractions of (A) the forearm wrist flexors to assist in restoring wrist flexion ROM and (B) the forearm wrist extensors to assist in restoring wrist extension ROM.



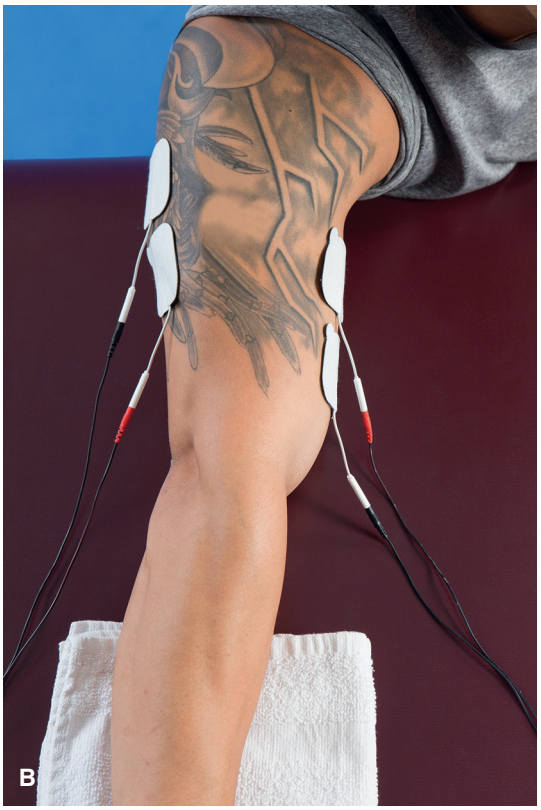


Fig 12 ■ 4 Elicited muscle contractions of (A) the biceps to improve elbow flexion and (B) the triceps to improve elbow extension.

used TENS showed significantly improved active and passive ROM. Again, the subjects did not use the TENS during stretch, yet still made these improvements.

Key Point! Use of NMES for improving ROM is not as much for improving strength as it is for eliciting robust muscle contractions that can facilitate active ROM.

Extracorporeal Shock Wave Therapy

Extracorporeal shock wave therapy (ESWT) is the delivery of acoustic shock waves with a high peak pressure (100 MPa) and a short duration (10 msec). These shock waves are created either by electrohydraulic, electromagnetic, or piezoelectric conversion. The therapeutic effect of these waves penetrates a few centimeters into the tissues and is thought to trigger an inflammatory response in the targeted tissues and change their viscoelasticity.[31] ESWT is used to treat a variety of musculoskeletal impairments and has only recently been used in the treatment of spasticity and subsequent ROM deficits.

In a study by Troncati et al,[32] 12 patients with chronic stroke and spasticity in the upper limb were treated with an electromagnetic lithotripter. The subjects received two treatments of ESWT to the flexor muscles of the forearm 1 week apart. Sixteen hundred pulses were applied with an energy density of 0.08 mj/mm^2 at the interosseous muscles of the hand and 0.105 mj/mm^2 to the muscles in the forearm. Passive ROM and upper limb motor control were improved both after treatment and at 3 and 6 months posttreatment.

Similar effects have been found in patients with spasticity secondary to other neurological insults. In a randomized, placebo-controlled trial, Vidal et al[33] examined the spasticity and ROM of 15 subjects with cerebral palsy after they were treated with either ESWT or placebo to their spastic upper and lower extremity muscles. Subjects received three treatments separated by a week. Patients treated with the ESWT demonstrated decreased spasticity and increased ROM, both immediately after treatment and 2 months later. Although the exact mechanism of how ESWT changes spasticity and improves ROM has not been fully described, it remains a plausible option for treatment of limited ROM due to neurological insult.

SPECIAL CONSIDERATIONS FOR USE OF THERAPEUTIC MODALITIES FOR CONTRACTURES

Contractures From Burn Scars

It may seem odd to apply heat to contractures that resulted from a burn; however, the literature contains two reports about this combination. Burns and Conin[34] used paraffin on the contractured hands of a patient who had scarring from burns in conjunction with a position of finger stretch that was held by a mechanical restraint, typically a small hand weight. The authors recommended leaving the paraffin on for 15 to 20 minutes and applying the wax to an area larger than usual to lubricate the surrounding skin and ease the discomfort from the tight skin that typically results post-burn.

Head and Helms[35] discussed a similar intervention. They made the adjustment of lowering the paraffin temperature to 91.4°F (33°C) to deliver less thermal energy to the newly healed skin. Treatment was applied for 20 to 30 minutes concurrent with gentle stretch and joint ROM improved by an average of 8°. Patients also noted less pain and improved pliability of the skin after treatment.

Contractures Postcancer and Necrotizing Fasciitis

A case report by Johnson and Draper[36] examines the treatment of a patient who was 3 years post–necrotizing fasciitis that developed in the course of her chemotherapy treatment for breast cancer. Debridement of the fascia resulted in contracture to both hips. Pulsed shortwave diathermy was applied to the upper thigh, hip, and buttock region for 20 minutes at 48 watts. This was followed by manual therapy and muscle energy techniques to the hips. The subject demonstrated improved ROM in the hips after 6 weeks of treatment.

FURTHER CLINICAL CONSIDERATIONS

A thorough patient history and comprehensive examination will identify the presence of precautions and/or contraindications that may influence modality choice. Impaired circulation or sensation might limit the use of

heat or cold modalities because of decreased ability to thermoregulate these tissues. Earlier chapters examine the specific contraindications and precautions for the modalities mentioned here.

Expectations for Improvements in ROM

When therapeutic modalities are used as a complementary part of the comprehensive active treatment plan to increase ROM, clinicians should consider the amount of improvement necessary to meet clinical goals. How much ROM can improve will vary depending on several factors, including the cause of the limited ROM and the normal physiological ROM at a specific joint. Furthermore, the clinical significance of increased ROM can vary, depending on the specific patient case. For example, a 5° improvement in ankle dorsiflexion ROM will likely have a greater clinical and functional significance than a 5° increase in shoulder flexion. In this regard, clinicians must rely upon additional knowledge of anatomy and biomechanics.

Documenting Improvement in ROM

The clinical decision to use therapeutic modalities should be based on findings of limited ROM obtained in the initial examination. Once the plan of care has been implemented, clinicians should reassess ROM to identify if the rehabilitation goals for ROM are being met. There is no universally agreed upon frequency for reassessment. However, reassessment is recommended both within a treatment session and following a series of treatments. For example, ROM can be measured on a patient with limited shoulder external rotation before and after administration of a thermal agent combined with stretching/ROM exercises to assess change in ROM within a treatment session (Fig. 12-5); a change in ROM can be assessed after six treatment sessions of the thermal agent with stretching/ ROM exercises.

Home Versus Clinic Use of Modalities for Loss of Motion

It is likely and recommended that the initial trial of a modality will be done in a clinical setting under supervision of an appropriately trained clinician. Based on known properties and effects, ultrasound or short-wave diathermy might be a reasonable choice to heat deep tissues. However, these devices are typically not available and not appropriate for independent home use.

Commercial or homemade heat and cold modalities are easily available at low cost. Clinicians need to be very diligent in their instructions on the use of these modalities at home to decrease the risk of injury. Box 12-4 identifies some items for educating patients regarding home use of modalities. It is recommended that the

Fig 12 ■ 5 (A) Application of short-wave diathermy to the shoulder prior to (B) manual stretching.

Box 12 ■ 4 Items to Include in Instructions for Home Use of Modalities

- Duration of application
- Frequency of application
- Skin protection (wet towel for cold, dry towels for heat)
- Timing of application, pre- or postexercise/stretching
- Observation for adverse reactions
- Contact number for practitioner

patient be exposed to a similar heat or cold modality at the clinic so the practitioner can monitor closely for any adverse reaction to the modality.

Home use of electrotherapy requires that the stimulation unit be either loaned or rented by the patient or caregiver. The patient or caregiver must display knowledge of electrode placement and device operation and safety before the device is issued.

DECISION-MAKING FOR SELECTION OF THERAPEUTIC MODALITY

The practitioner should consider the physiological rationale, the mechanism of action (e.g., heat the tissues, relieve pain) desired, the best research evidence, the patient's goals, and precautions or contraindications for each modality to select the optimal intervention. Selection may be limited by clinical availability or home environment. Typically, deeper tissues may respond better to modalities such as ultrasound or short-wave diathermy, if available. More superficial tissues may respond better to hot packs, paraffin, or cryotherapy. Finally, if a patient wishes to use modalities and stretching at home, a commercial hot or cold pack is most appropriate.

Clinical Application for Thermal Modalities

The application of a modality is a process, not an event. Using a modality to assist in gaining ROM is a process as well. The following steps offer a guideline for this process:

1. Isolate the tissues to be treated. Is the limited ROM due to muscle, tendon, or joint tightness?
2. Check for contraindications and/or precautions.
3. Inspect the skin of the area to be treated.
4. Check for any hypersensitivity to the type of energy being delivered.
5. Move the joint or tissue into a position of gentle stretch.
6. Apply the modality at the appropriate parameters (i.e., duration, intensity). Continue to stretch as tolerated during the application of the modality.
7. Inspect the skin after the application of the modality.
8. Continue with stretching and/or other therapeutic interventions.

Clinical Application for Electrical Modalities

The application of an electrical modality is also a process, not an event. The following steps offer a guideline for this process:

1. Isolate the tissues to be treated. Is the limited ROM due to muscle, tendon, or joint tightness?
2. Check for contraindications and/or precautions.
3. Inspect the skin of the area to be treated.
4. Check for any hypersensitivity to the type of energy being delivered.
5. If using NMES, either stimulate the antagonist to the spastic muscle or stimulate both flexors and extensors into an appropriate ROM. If using TENS, apply and activate as you passively take the joint through its ROM. Have the patient adjust the intensity as needed.
6. Apply the modality at the appropriate parameters (duration, intensity). Continue to stretch as tolerated during the application of the modality.
7. Inspect the skin after the application of the modality.
8. Continue with stretching and/or other therapeutic interventions.

CASE STUDY 12-2 Hot Packs for Limited Knee ROM

The patient is a 65-year-old female who had left total-knee arthroplasty 1 month ago. The patient presents with limitations in extension of the left knee. Palpation also reveals tightness in the distal hamstrings, proximal gastrocnemius, and posterior joint capsule of the knee, all of which are likely contributors to the decrease in ROM.

CLINICAL DECISION-MAKING

1. Does the patient have dysfunction or impairment that may be improved or lessened by the use of hot packs?
ANSWER: Yes, tightness in the posterior soft tissue structures of the left knee might benefit from the use of hot packs followed by stretching.

2. Why would hot packs be a consideration for this patient?
ANSWER: The thermal effects of the superficial hot pack, either coupled with or followed by passive stretch, would be appropriate to increase tissue elasticity and allow for plastic deformation of the posterior tissues of the knee. Because this is a fairly large area and short-wave diathermy is not available, moist hot packs would be appropriate.

3. What specific outcome measures can be used to assess the effectiveness of the agent?
ANSWER: Active and passive ROM should be measured pre- and posttreatment. A knee-specific or patient-specific outcomes measure could be used as well.

4. Are there any precautions or contraindications to consider with this patient?
ANSWER: Please refer to Chapter 3.

5. What specific parameters are recommended for this patient?
ANSWER: Because these tissues are superficial, commercial or homemade hot packs would work well. The patient should insulate the skin with 8 to 10 layers of dry towels. A treatment time of 20 to 30 minutes where the patient feels only gentle heat would be optimal. Applying the hot pack with the patient in a position of end-range extension would be optimal as well (Fig. 12-6A). The hot pack treatment would be followed by manual stretching (Fig. 12-6B).

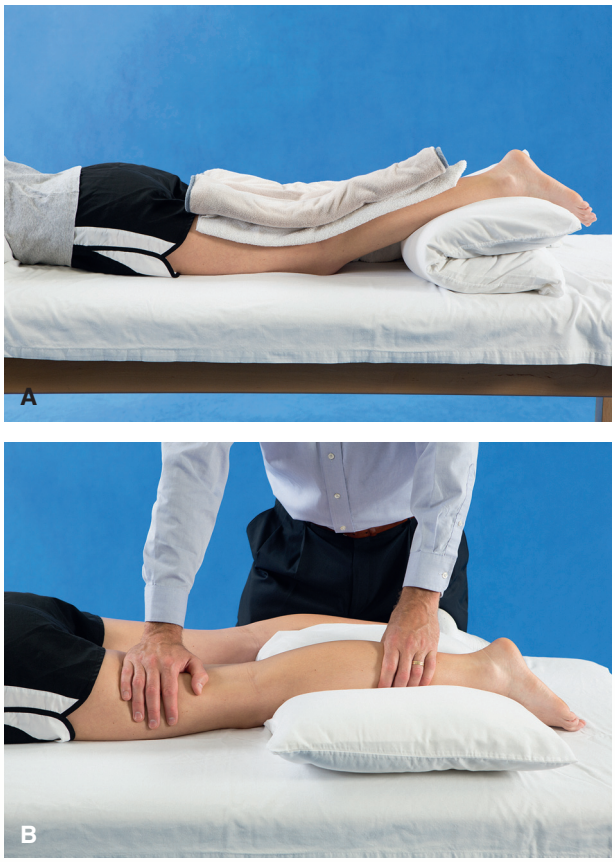


Fig 12 ■ 6 (A) Application of hot pack to the posterior knee for knee flexion contracture followed by (B) manual stretching of knee into extension.

6. What are the proper steps to using hot packs for this patient?
ANSWER: First, assess the presence of any contraindications or precautions. Inspect the skin and position the patient appropriately. Apply the hot pack with parameters previously noted, moving the limb into even more extension during the treatment. Finish by inspecting the skin and performing passive stretch. Remeasure ROM after treatment.

CASE STUDY 12-3 Treatment of Achilles Tendon With Limited Dorsiflexion

The patient is an 18-year-old male with limited ankle dorsiflexion ROM following immobilization for 6 weeks. The patient presents with 3° of passive ankle dorsiflexion determined to be the result of decreased flexibility in the Achilles tendon.

CLINICAL DECISION-MAKING

1. Does the patient have dysfunction or impairment that may be improved or lessened by the use of ultrasound?

ANSWER: Yes, tightness in the Achilles tendon might benefit from the use of this modality.

2. Why would ultrasound be a consideration for this patient?

ANSWER: The thermal effects of ultrasound, coupled with sustained passive stretch into dorsiflexion, would be appropriate to increase tissue elasticity and allow for plastic deformation of the tendon.

3. What specific outcome measures can be used to assess the effectiveness of the agent?

ANSWER: Active and passive dorsiflexion ROM should be measured pre- and posttreatment. A shoulder-specific or patient-specific outcomes measure could be used as well.

4. Are there any precautions or contraindications to consider with this patient?

ANSWER: The patient does not present with any considerations to avoid use of continuous ultrasound.

5. What specific parameters are recommended for this patient?

ANSWER: Because these tissues are superficial, 3 MHz would be the recommended frequency. Thermal effects are desired; therefore, a 100% duty cycle would be appropriate. A treatment time of 10 to 12 minutes at an intensity that produces gentle but noticeable heat would be optimal. Performing the ultrasound with the patient in a prone position with manual stretch into dorsiflexion would be ideal (Fig. 12-7).

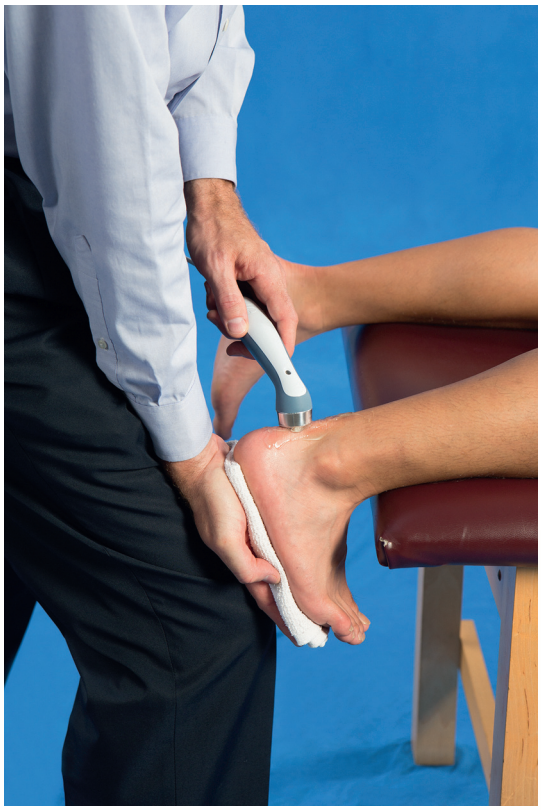


Fig 12 ■ 7 Application of thermal ultrasound to the Achilles tendon while manual stretching into dorsiflexion is applied.

6. What are the proper steps to using ultrasound for this patient?

ANSWER: First, assess the presence of any contraindications or precautions. Inspect the skin and position the patient appropriately. Perform the ultrasound with the parameters noted earlier, moving the limb into even more abduction and external rotation during the treatment. Finish by inspecting the skin and performing passive stretch. Remeasure ROM after treatment.

Documentation Tips

When applying modalities to improve ROM, documentation of the following should be included:

1. ROM/clinical measures of flexibility
   - Before and after application of the modality and other interventions
2. Specific parameters of the modality (refer to specific chapters on each modality)
3. Patient position
4. Tissue stretch mechanism (i.e., manual, device, or weight)
5. Tissue stretch duration
6. Patient response to treatment (e.g., inspect skin for adverse effects)
7. Patient education regarding potential home use of physical modalities

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CHAPTER 13

ELECTROTHERAPY FOR MUSCULOSKELETAL DISORDERS

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Neuromuscular electrical stimulation (NMES) is commonly used in a variety of clinical settings to evoke contractions in an effort to enhance the rehabilitation of human skeletal muscles.[1–4] This chapter will (1) review early training studies that incorporated NMES training of skeletal muscle, (2) provide data to support the idea that properly designed NMES training protocols can result in improved neuromuscular performance, (3) provide guidelines for the implementation of NMES training for individuals with musculoskeletal disorders or other conditions, (4) provide an overview of NMES-induced muscle recruitment, and (5) outline some of the drawbacks associated with NMES training. This information is intended to help practitioners provide evidence-based treatment when utilizing NMES to improve muscle size and strength.

RATIONALE FOR NEUROMUSCULAR ELECTRICAL STIMULATION

The goal of using NMES is to induce contraction of skeletal muscle to obtain the physiological improvements that result from exercise training. NMES is a widely accepted modality that is used to treat atrophic muscle after injury or disease, although a consensus on specific programs and parameters to improve neuromuscular performance has yet to be reached. Early studies targeting increases in muscle mass were not consistently successful. However, recent studies have demonstrated the potential of NMES training protocols to elicit a hypertrophic response in skeletal muscle, suggesting that this training

modality has the potential to be a valuable tool for rehabilitation when utilized appropriately.

The neuromuscular system is perhaps the most highly plastic system in the human body, showing dramatic adaptations in response to changes in activity. Skeletal muscle can adapt by increasing or decreasing the amount of contractile proteins, by changing its fiber type composition, or by altering its metabolic profile to sustain force production.[5,6] Adaptations resulting from activity are not limited to peripheral skeletal muscle, with concurrent adaptations occurring in neural systems in response to activity.[7,8] Specifically, enhanced recruitment of motor units (an alpha motor neuron and all the muscle fibers innervated by it) is accomplished through improved central control of firing frequency or synchronization of motor units—that is, by activating motor units more frequently and in unison, muscle force can be increased. These factors underlie the use of NMES.

NMES FOR MUSCLE STRENGTHENING

NMES is used clinically as a tool to strengthen weakened muscle in persons with musculoskeletal disorders and is supported by evidence in the scientific literature. Muscle strengthening is thought to result from two primary mechanisms: increased muscle size or improved motor unit recruitment (nonmuscle mass adaptations).[9] Increasing muscle mass usually takes several weeks to occur, while nonmuscle mass adaptations can occur more rapidly. Nonmuscle mass adaptations are typically due to increased motor unit recruitment, which is caused by increasing (1) the number of motor units recruited, (2) the frequency that motor units are recruited (Fig. 13-1), or (3) recruitment motor units in a more synchronized manner (i.e., at the same time).[7,10] The manner in which NMES-induced motor unit recruitment increases muscle force is similar to the game of tug-of-war. If the individual members of a team pull more frequently and at the same time, the result is a more forceful pull or tug.

Recently a model was created from patients progressing to total knee arthroplasty that delineated the two major causative factors underlying the precipitous loss of strength after surgery; however, this model lends itself well to other populations demonstrating postoperative or postinjury loss of strength.[11] These causative factors were defined as intrinsic (i.e., muscle atrophy and fiber loss) and extrinsic or neural (i.e., impaired motor unit recruitment in regard to size, number, frequency, and synchronization). In the initial weeks postinjury or procedure, the decline in strength is predominantly due to neural impairment, but over time, this neural influence is surpassed by intrinsic loss (e.g., decreases in muscle mass) due to prolonged disuse or detraining. This suggests that prolonged or sustained impairment in neural recruitment begets further intrinsic loss. It is during this initial period, when motor unit recruitment is most impaired, that the benefits of NMES are most evident.

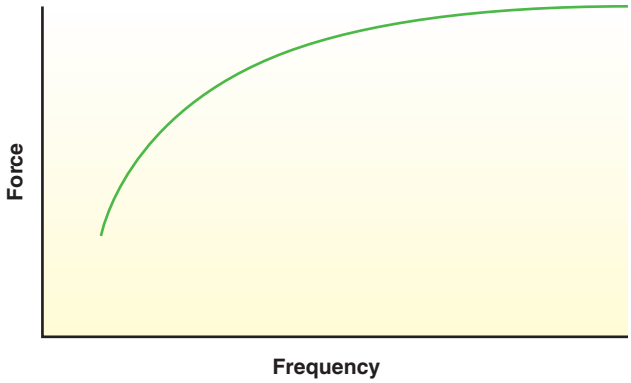

Fig 13 ■ 1 Force-frequency relationship. Note that as frequency of activation is increased, force is progressively increased until tetany is reached (the point where further increases in frequency do not produce increases in force production). *(Adapted with permission from Gregory CM, Dixon W, Bickel CS. Impact of varying pulse frequency and duration on muscle torque production and fatigue.* Muscle Nerve. *2007; 35(4):504-509.)*

Key Point! Improved muscle size is an adaptation that occurs with repeated muscle overload and is consistent with the principles of voluntary muscle strengthening. Increases in muscle mass typically take several weeks to occur; this may not be a reasonable expectation during rehabilitation programs in the current health-care environment, where shorter supervised training and reduced visits are typical. Increases in strength observed in the initial weeks of training are typically due to changes in motor unit recruitment.

Previous studies have investigated the effects of NMES training on muscle strength in participants without disability. These studies often compared the effects of NMES, voluntary isometric exercise, and no exercise on strength of the quadriceps femoris.[12–15] Results generally

showed that both the NMES and exercise groups displayed increased strength, but there were no differences between these two groups. As expected, strength in the no-exercise control group was unchanged. One criticism of these studies is that NMES and voluntary exercise may not have produced equivalent forces during treatment (e.g., voluntary exercise often used maximal contractions), leading to inequality in training doses. Many early NMES studies did not in fact try to maximize the amount of stimulation delivered nor attempt to have equal training loads. Therefore, activated motor units were not necessarily being exercised to the same degree in both the voluntary and NMES groups, which likely impacted results. Despite these limitations, some studies have shown gains in strength after NMES training to be similar to voluntary exercise.

Key Point! Similar gains in strength have been observed in studies comparing voluntary exercise and NMES. However, voluntary exercise often used maximal contractions, whereas less-than-maximal contractions were used with NMES.

A few studies have investigated changes in muscle size after NMES training in healthy individuals. The results have provided a foundational basis for understanding the effects of NMES on muscle. These studies have reported increases in voluntary strength after NMES training; however, this finding alone does not imply a resultant hypertrophy, because factors other than increases in muscle size can account for improved voluntary force generation after training (i.e., nonmuscle mass adaptations). For example, in a single subject design, Delitto et al[16] reported impressive strength gains despite average fiber size decreasing by approximately 16% after NMES training. Although a reduction in fiber size is surprising, strength gains were realized without remarkable muscle fiber hypertrophy. These findings confirm that improvements in strength and size after NMES are not necessarily related.

There is evidence that NMES can prevent atrophy and induce hypertrophy in individuals with weakness caused from neurological disorders[17,18] or from orthopedic injury,[4,19] but clear, foundational data on the effects of NMES on muscle size are limited. However, some studies demonstrate that NMES can elicit muscle hypertrophy in skeletal muscle when utilized appropriately (i.e., suitable mode, dosage). For example, the triceps brachii muscle in primates was trained using NMES, and skeletal muscle biopsies were obtained pre- and post-training.[20] NMES was used to cause contraction of the elbow extensors (60 Hz, 5 sec on/10 sec off for 60 min) 5 days per week for 3 weeks at the maximum stimulation intensity tolerated "without causing discomfort to the animals." The results showed that the average size of all muscle fiber types increased between 13% and 45%. It needs to be noted that these gains were realized over an extremely short training period. In another study, the triceps surae was stimulated for 21 consecutive days with 15 to 20 contractions per session at an amplitude of 40 to 45 mA. Skeletal muscle biopsy analyses revealed that muscle fiber size increased approximately 16% after 21 days of stimulation.[21] The fact that such increases in fiber size can be realized in such short training programs illustrates the potential impact of NMES.

Additional data examining the effects of NMES training[22,23] have incorporated the use of dynamic contractions with high training loads (about 70% to 80% of maximal voluntary isometric contraction) in an effort to mimic voluntary training studies that are known to result in muscle hypertrophy (i.e., progressive resistance training). Ruther et al[22] studied subjects who trained 2 days per week for 9 weeks with either voluntary or NMES-induced isokinetic exercise. Stimulation intensity was set to elicit 70% of maximal voluntary isometric torque. The results indicated a 10% and 4% increase in quadriceps femoris muscle size in NMES and in voluntary training groups, respectively, suggesting that resistance training with NMES may be superior to voluntary training—at least for causing muscle hypertrophy during the early phase of a resistance-training program. These findings are consistent with data showing that early strength gains during voluntary resistance-training programs are not typically due to muscle fiber hypertrophy.[24] Stevenson and Dudley[23] used a similar protocol in subjects who reported active participation in a voluntary resistance-training program at least 2 days per week. One leg received NMES training at approximately 70% of maximal voluntary isometric torque. Stimulation intensity was progressively increased. By the end of 8 weeks of training, muscle size had increased by about 10% in the NMES-trained leg. Table 13-1 summarizes the results of these studies and others in which NMES was used for muscle strengthening or hypertrophy.

TABLE 13–1. NMES for Muscle Strengthening or Hypertrophy

Author	Training	NMES Parameters	Outcome
Cabric et al, 1987[21]	Isometric 15–20 contractions 1 time/day 21 days	PC Group 1: 50 Hz Group 2: 2,000 Hz	Increased type I muscle fiber area
Caggiano et al, 1994[12]	Isometric 10 contractions 3 days/week 4 weeks	PC: biphasic symmetrical 200 μsec 25 pps 36% MVIC	Increased strength
Currier and Mann, 1983[13]	Isometric 10 contractions of 15 sec with 15 sec of rest 3 days/week 5 weeks	Burst-modulated AC (Russian) 2,500 Hz 50 bps > 60% MVIC	Increased strength
Delitto et al, 1989[16]	Isometric 10 contractions/ session 3 days/week 4 weeks	Burst-modulated AC (Russian) 2,500 Hz 75 bps Max tolerable current (95%–126% MVIC)	Increased strength Increased type I fiber area Reduced type II fiber area
Iwasaki et al, 2006[74]	Isokinetic 10 sets of 10 3 days/week 6 weeks	Burst-modulated AC (Russian) 5,000 Hz 20 bps Max tolerable current	Increased strength
Laughman et al, 1983[14]	Isometric 10 reps 5 days/week 5 weeks	Burst-modulated AC (Russian) 2,500Hz 50 bps 33% MVIC	Increased strength
McMiken et al, 1983[15]	Isometric 10 contractions 4 days/week 3 weeks	PC 100 μsec 75 pps Max tolerable current	Increased strength
Mohr et al, 1985[75]	Isometric 10 contractions 3 days/week 5 weeks	PC: twin-peak monophasic pulses 45 μsec 50 pps Max tolerable	Increased strength in voluntary exercise only
Ruther et al, 1995[22]	Isokinetic 3 to 5 sets of 10 2 days/week 9 weeks	PC: symmetrical biphasic pulses 500 μsec 50 pps 70% MVC	Increased muscle cross-sectional area with NMES (10%)
Stevenson et al, 2001[23]	Isokinetic 3 to 5 sets of 10 2 days/week 8 weeks	PC: symmetrical biphasic pulses 450 μsec 70 pps 70% MVC	Increased muscle cross-sectional area (11%)

MVC = maximal voluntary contraction; MVIC = maximal voluntary isometric contraction; PC = pulsed current.

For several decades, NMES has been used after various surgical procedures for the knee. This probably stems from the fact that quadriceps force is reduced with knee effusion due to neural inhibition (i.e., arthrogenic muscle inhibition).[25,26] Because the quadriceps femoris is an important muscle to focus on during rehabilitation of the knee, practitioners will often turn to NMES for assistance if the patient has difficulty volitionally activating the quadriceps. Studies using NMES after knee surgery have shown positive changes in muscle performance. Several reports in the literature address the use of NMES in patients after anterior cruciate ligament (ACL) reconstruction and total joint arthroplasty surgeries (Table 13-2).

Several studies have examined the effect of NMES on the quadriceps femoris muscles after ACL reconstruction.[2,27–31] Some suggest that early intervention with intense NMES enhances recovery of strength and function.[29,30] Early studies compared the use of NMES-evoked contractions to voluntary exercise during the early phase of rehabilitation (first 6 weeks)[27] or compared the addition of NMES to the rehabilitation programs.[30] These studies utilized burst-modulated alternating current (AC; Russian current) at intensities that were "maximally tolerated." A greater increase in quadriceps muscle strength was seen in those subjects in the NMES groups.[27,30] One study randomly assigned participants who were 1 week post–ACL reconstruction into four isometric strengthening treatment groups: high-intensity NMES, high-intensity voluntary exercise, low-intensity NMES, and a combination of high- and low-intensity NMES.[29] The groups that trained with high-intensity NMES and the combination of low- and high-intensity NMES showed significant improvements in strength and knee flexion/extension during gait when compared to the other two groups.[29] The intensity of stimulation used was the "maximum tolerated" by each participant, suggesting that the training was sufficiently intense to evoke neuromuscular adaptations. It should be noted that not all studies that have utilized NMES in the treatment of patients after ACL reconstruction have shown significantly better results with NMES.[28]

TABLE 13–2. NMES for Anterior Cruciate Ligament Reconstruction

Author	Training	NMES Parameters	Outcome
Delitto et al, 1988[27]	Isometric 15 contractions 5 days/week 3 weeks	Burst-modulated AC (Russian) 2,500 Hz 50 bps Max tolerable current	Increased strength
Fitzgerald et al, 2003[2]	Isometric 10 contractions 2 days/week 11 weeks	Burst-modulated AC (Russian) 2,500 Hz 75 bps Max tolerable current	Increased strength
Paternostro-Sluga et al, 1999[28]	Isometric 48 5-sec and 24 10-sec contractions 7 days/week 6 weeks	PC: biphasic symmetrical 220 μsec 100 pps Tolerable, strong visible contraction	Increased strength (no difference between groups)
Snyder-Mackler et al, 1995[29]	Isometric 15 contractions 3 days/week 6 weeks	Burst-modulated AC (Russian) 2,500 Hz 75 bps Max tolerable current	Increased strength
Snyder-Mackler et al, 1991[30]	Isometric 15 contractions 3 days/week 3 weeks	Burst-modulated AC (Russian) 2,500 Hz 75 bps Max tolerable current	Increased strength Improved gait pattern

NMES is also commonly used in patients after total knee arthroplasty (TKA; Table 13-3). These individuals have considerable knee effusion, pain, and impaired ability to voluntarily activate their quadriceps femoris similar to that seen after ACL reconstruction.[32] Data have shown that 1 year after TKA, muscle atrophy, weakness, and functional limitations persist.[33] Case studies done on patients post-TKA surgery found promising results using burst-modulated AC (Russian current) delivered to evoke 10-second contractions for a total of 10 repetitions at "maximally tolerated" intensity.[3,34] The case studies suggested that after 6 weeks of training, some subjects showed greater strength in the surgical leg at 6 months post-TKA than the limb that was not surgically repaired. A randomized controlled trial has now been published that investigated how the addition of NMES for 6 weeks after TKA impacted strength and function. Remarkably, participants that received the NMES along with a standard rehabilitation program had better outcomes for up to 1 year when compared to a control group that only received the standard rehabilitation.[4]

Another randomized controlled trial compared a progressive strengthening program with or without NMES-influenced function after TKA with a group that received conventional rehabilitation with perhaps a less intense training protocol.[35] The results indicated that as long as the individuals were participating in a structured progressive strengthening program (with or without NMES), they made significantly greater improvements than those who were undergoing standard rehabilitation. This study highlights the importance of training intensity, and in this case, function was apparently achieved regardless of whether NMES was used. In some cases, NMES may be required to achieve sufficient intensity during rehabilitation due to impairments with voluntary muscle activation. There is data to support that high-intensity NMES can improve the clinical outcomes of these patients.

A unique patient population where the use of NMES-induced muscle contractions has shown positive results is in critically ill patients who have spent time in the intensive care unit (ICU; Table 13-4).[36–39] Patients with prolonged stays in the ICU often experience large functional limitations, muscle weakness, and fatigue—otherwise known as ICU-acquired weakness. Interventions, such as mobility training or resistance exercise, are often not feasible or practical to complete in this clinical setting. In an effort to address these issues, therapists have begun

TABLE 13–3. NMES for Total Knee Arthroplasty

Author	Training	NMES Parameters	Outcome
Lewek et al, 2001[34]	Isometric 10 contractions 11 sessions	Burst-modulated AC (Russian) 2,500 Hz 40–75 bps Max tolerable current (35%–50%)	Single case study, patient showed improvements in strength and met goals
Petterson et al, 2009[35]	Isometric 10 contractions 2–3 days/week 6 weeks	Burst-modulated AC (Russian) 2,500 Hz 50 bps Max tolerable current (min level = 30% MVIC)	Improvements in strength and function (no differences between groups that did not receive NMES)
Stevens et al, 2004[3]	Isometric 10 contractions 3 days/week 6 weeks	Burst-modulated AC (Russian) 2,500 Hz 50 bps Max tolerable current	Increased strength
Stevens-Lapsley et al, 2012[4]	Isometric 15 contractions 2 sessions/day 6 weeks	Symmetrical biphasic 250 μsec 50 pps Max tolerable current	Improved strength and function for up to 1 year compared to control group

TABLE 13–4. NMES for Critically Ill Patients

Author	Training	NMES Parameters	Outcome
Abdellaoui et al, 2011[36]	Isometric 1 hour/day 5 days/week 6 weeks	Symmetrical biphasic 400 μsec 35 pps Max tolerable current	Improved strength and function
Vieira et al, 2014[39]	Isometric 2, 1-hour sessions 5 days/week 8 weeks	Symmetrical biphasic 300–400 μsec 50 pps Max tolerable current	Improved muscle mass and function
Sillen et al, 2014[37]	Isometric 2, 1-hour sessions 5 days/week 8 weeks	Symmetrical biphasic 15 or 75 pps Max tolerable current	Improved function with either 15 or 75 pps but increased strength with 75 pps only

utilizing NMES of major muscle groups with promising results. The use of NMES in the critically ill patient is not isolated to patients who have prolonged stays in the ICU, but there have been several clinical studies conducted in patients with conditions such as chronic obstructive pulmonary disease or chronic heart failure that make typical exercise and rehabilitation programs challenging. Protocols are used to evoke visible muscle contractions in large muscle groups. For example, in a small randomized controlled trial, investigators stimulated both the quadriceps and hamstrings with a symmetrical biphasic waveform of 400 μsec and a pulse frequency of 35 Hz for 1 hour per day, 5 days/week for 6 weeks.[36] They found that the group that received NMES was stronger and walked farther during a 6-minute walk test. Others have shown similar results, providing good evidence that NMES is a promising tool for intervention in critically ill patients.

More recently, systemic reviews have been published reporting the benefit of NMES in patients undergoing a variety of surgical procedures to the knee, including ligament and meniscal repairs and total joint arthroplasty.[11,40–42] Collectively, these reviews suggest that the evidence for NMES is most robust when NMES is applied early in the rehabilitation period, with optimal benefit observed when NMES is applied in the first 1 to 4 weeks after surgery. Furthermore, evidence from these reviews suggested that patients who received NMES reached markers of subjective and functional improvement sooner than subjects who did not receive NMES. That NMES was shown to result in sooner and greater restoration of volitional muscle strength when applied early in the rehabilitation period lends support to Kittelson's model, which suggests that early impairment in motor unit recruitment largely underlies loss of strength and evidences the need to administer NMES sooner rather than later after surgical intervention or injury.

Examination, Evaluation, and Prognosis

The initial examination should evaluate the patient's medical history, paying specific attention to possible precautions and contraindications for the use of NMES. Individuals with musculoskeletal disorders will typically have some degree of voluntary muscle activation, but they may still benefit from NMES. The practitioner should assess the muscle innervation status of patients who have a possible neurological injury, such as peripheral nerve injuries (e.g., nerve compression, crush, or laceration), or who have a history of neurological disorders (e.g., multiple sclerosis, stroke, or spinal cord injury). It is important to note whether the patient has an upper or lower motor neuron injury because lower motor neuron injuries do not respond to NMES. (See "Electrical Muscle Stimulation (EMS) Applied to Denervated Muscle" later in this chapter.)

If innervation status is unknown, the practitioner can test for this by applying NMES to the involved muscle and observing its response. Electrodes should be applied so that the motor nerves of the weak muscles are within the area where current will flow. Ask the patient to report the first sensation of electrical stimulation (usually a tingling feeling), noting the milliamps when this occurs and understanding that, if sensation is compromised, the patient may not feel anything. Continue to increase the current until you see a motor response. If the patient reports sensation and there is an observed motor response, then the muscle is innervated and able to respond to NMES. Should there be no sensation of electrical stimulation (ES) and a lack of motor response, further neurophysiological testing may be indicated (see Chapter 17). If this is the case, the practitioner may need to refer the patient to another health-care provider who can conduct electromyography tests to evaluate the degree of injury. Additional tests for strength, range of motion (ROM), pain, and functional status are indicated to evaluate the appropriateness of NMES compared to other interventions (Table 13-5).

Key Point! Stimulation of denervated muscle requires long pulse durations (greater than 1 millisecond), which most stimulators are unable to deliver.

Intervention

Patients with decreased muscle strength caused by musculoskeletal dysfunction, especially those with muscle atrophy, can benefit from NMES. However, the *intensity of training* is a key factor in the efficacy of NMES strengthening protocols. Most studies that have shown effectiveness have used intensities or dosages that were "maximally tolerated," with some studies using intensities as high as 70% of maximum voluntary contractions (see Tables 13-1 through 13-3). It is important to realize that prescription of NMES exercise should be done in a similar fashion as voluntary training programs. Thus, the sets and reps performed as well as the load (i.e., % maximal voluntary contraction [MVC]) used should be carefully prescribed and monitored in NMES training programs.

Practitioners need to consider several factors when applying NMES: electrode placement, on- and off-times, number of repetitions, frequency, and duration of treatment. It is important to choose the appropriate muscle stimulator based on the desired stimulation parameters to achieve the neuromuscular response. NMES is utilized in an effort to facilitate the contraction of skeletal muscle when there is sufficient need to do so. Understanding the differences between voluntary and artificial activation of skeletal muscle is necessary to determine whether NMES is indicated (Box 13-1).

TABLE 13–5. Examination for NMES for Strengthening

Tests of	Question	Reason
Muscle innervation	Has innervation status been compromised?	NMES for strengthening typically requires innervation of the muscle.
Strength	What is current voluntary strength (manual muscle test or muscle dynamometry)?	To determine effect of treatment.
Range of motion	Are there any ROM limitations present?	Decreased ROM may impact functional outcomes.
Sensation	Is sensation present, absent, or diminished?	More frequent monitoring of skin integrity is needed with reduced sensation.
Pain	Is there pain at rest or with activity? How severe?	Pain could be a limiting factor for the strength-training program.
Spasticity	Is spasticity present?	Antagonist spasticity may affect ability to accurately assess strength.
Function	What functional limitations are present?	To determine effect of treatment on function.
Cognitive status	Is cognitive status sufficient to provide feedback?	To ensure safety of use.

Box 13 ■ 1 Factors to Consider When Applying NMES

- Line or battery-powered stimulator
- Stimulation parameters
- Electrode placement
- On- and off-times
- Dosage (intensity)
- Number of repetitions/sets
- Frequency of application

Voluntary Versus NMES Exercise: Differences in Muscle Recruitment

Recruitment of skeletal muscle during *voluntary* contractions (i.e., without NMES) follows a predictable and orderly pattern—starting with smaller and progressing to larger motor units as increasing forces are required. It should also be noted that the number of motor units and the firing frequency can be altered during voluntary activities. In contrast, *artificial* activation (e.g., during NMES) does not follow these same principles and results in a more random pattern of activation of motor units (e.g., small and large motor units will be recruited even when low forces are produced). Additionally, alterations in the number of motor units or firing frequency are typically not available with clinical stimulators without stopping and starting the treatment multiple times. Consequently, all activated motor units have the same firing rate and will produce relatively similar forces, which could be 100% of their force-producing capacity at higher frequencies of stimulation. When the decision is made to utilize NMES, the practitioner should apply the basic principles of muscle conditioning to the design of the protocol (i.e., current intensity, repetitions, sets, and frequency of treatment) while considering the differences in motor unit activation. It is also important to monitor the patient's initial response to treatment and all subsequent contractions.

Selecting a Stimulator

The ES device should be able to deliver parameters that are capable of maximizing force development of muscle and activating a large percentage of the muscle fibers in an effort to provide muscle overload. Studies have examined the use of small portable stimulators versus larger line-powered devices with mixed results (Fig. 13-2). Snyder-Mackler et al[19] initially reported that significantly

Fig 13 ■ 2 Examples of (A) portable and (B) clinical plug-in stimulators.

greater forces were created using a line-powered stimulator compared to a portable device in participants who had had ACL reconstruction. However, the authors of this study also acknowledged that there might be other portable stimulators available that may create more force.

More recently, Laufer et al[43] compared two portable stimulators and one line-powered device and reported that, when stimulation parameters were kept constant across stimulators, the portable stimulators created more force. Other reports have also indicated that portable stimulators can evoke similar torque outputs as line-powered stimulators.[44] Based upon these studies, practitioners should realize the importance of understanding the capabilities of their available stimulators and choose one that is appropriate for the intervention desired (Table 13-6).

Clinical Controversy

Most battery-powered, handheld NMES stimulators are incapable of delivering current intensities sufficient to elicit muscle contractions at levels capable of improving strength. For this reason, line-powered stimulators are recommended for NMES to optimize outcomes.

Stimulation Parameters

When selecting the parameters, the practitioner can choose from several options to achieve the desired response. If the goal of NMES is to enhance muscle strength, the highest pulse duration and amplitude tolerated by the patient should be used.[45] The frequency should be adjusted to achieve a smooth forceful contraction—generally achieved with greater than 30 pulses per second (pps) (see Fig. 13-1). If one chooses to use burst-modulated AC (BMAC), a burst frequency of 50 bursts per second is recommended. Although Russian current may be the most recognized form of BMAC, other variations are available that may be more effective at eliciting muscle torque.[46] Therefore, when using BMAC for muscle strengthening, the following parameters are recommended: a burst duration of 2 to 10 milliseconds (msec), a relative duty cycle of 10% to 50%, a carrier frequency of 1,000 to 2,500 Hz, and the highest amplitude tolerated.[46]

It is important to understand that higher stimulation frequencies (pps) will also lead to greater muscle fatigue. Therefore, choose an on-time/off-time ratio that allows for sufficient muscle recovery between repetitions. Support for this is provided by a study that investigated the effects of duty cycle on metabolic changes within the calf musculature; there was a significantly greater force decline during contraction and a significantly decreased muscle pH with a 10-sec on/10-sec off on:off time ratio compared to 10 sec on/50 sec off.[47] If muscle fatigue is an ongoing problem, the practitioner can reduce frequency and increase the rest time between contractions so that each stimulated contraction is evoking maximum force.

TABLE 13–6. NMES Parameters Typically Used for Muscle Strengthening[2-4,22,23,29,34]

Waveform	Symmetrical or asymmetrical biphasic pulsed current, burst-modulated alternating current (e.g., Russian current)
Pulse duration	200–600 μsec
Frequency	20–100 pps (bursts per second)
Amplitude	To obtain strong muscle contraction (maximum tolerated or current necessary to achieve ≥ 50% of MVC)
Ramp-up time	1–5 sec
Ramp-down time	1–2 sec
Duty cycle	1:3 to 1:5 with on-time up to 10 sec and off-time up to 50 sec
Treatment time and duration	At least 10 contractions or up to 1 hour/day 3–5 times/week 4–8 weeks
Carrier frequency*	1,000–2,500 Hz
Burst duration*	2–10 msec
Relative duty cycle*	10%–50%

*Specific to burst-modulated AC.

Key Point! To decrease the effect of fatigue and ensure that NMES is eliciting as much muscle force as possible, it may be necessary to increase the rest period between maximally tolerated contractions.

Ramp time is often used for patient comfort during ES, allowing time for the current to increase and decrease slowly rather than abruptly. However, a longer ramp-up time is not always the most comfortable for each patient and will reduce the amount of time that all motor units are activated. Shorter ramp times are recommended so that the muscle can be activated for sufficient periods of time. Be mindful that both the patient's feedback and muscle response are critical in guiding these decisions.

Electrode Placement

Electrode placements for NMES applications depend on the size and location of the targeted muscle (Box 13-2). If the goal is to optimize muscle strength by facilitating the contractions, the electrode placement should be arranged to recruit as many motor units as possible. Electrode configurations may be monopolar, bipolar, or quadripolar, depending on the size of electrodes and the size of the target muscle. Figure 13-3 shows examples of different electrode configurations. The practitioner should recognize that current density is inversely proportional to electrode area and should select electrodes of an appropriate size for the targeted muscle. A photo image of electrode placement can be taken to record treatment and make it easier to reproduce a successful stimulation session.

Intensity or Dosage

The appropriate intensity or amplitude of the ES is perhaps the most important parameter of NMES and the one most underused by practitioners. The "overload" principles of strength training and prior studies of NMES clearly show that contractions near maximal tolerance are required for increasing strength. Contractions near 70% of the MVC of the opposite limb are common in studies reporting increased strength following NMES. Many practitioners do not deliver an appropriate intensity to elicit contractions of this level; thus, patients are less likely to realize the benefits of NMES.

Determining or quantifying the appropriate stimulus intensity for NMES is easy and should be performed before the first session. To ascertain an appropriate treatment intensity or dosage, the maximal isometric strength of the patient's uninvolved side should be measured first. This can be completed with an isokinetic dynamometer, a standard pulley-style weight stack, or a handheld dynamometer. The patient is then positioned on the same device to test the involved side. After appropriate setup of the NMES, the intensity of the ES is increased as muscle force is measured. The stimulus intensity required to reach a specific submaximal percentage of the opposite side is recorded and used as the dosage for subsequent applications of NMES. The patient can then be moved to a treatment table or area using the newly quantified intensity or dosage. Typical target percentages for NMES training sessions may be 50% or greater, although initial applications may need to be closer to 30%. This process of quantifying the dosage of stimulus intensity should be repeated at regular intervals throughout rehabilitation as strength improves so as to optimize the effects of the treatment (see Fig. 13-3 and Fig. 13-4).

Key Point! The amplitude required to elicit a specific level of muscle force can be measured and is termed *dosage*. A dosage eliciting greater than 50% of the MVC is recommended for strengthening.

Monitoring Treatment

Similar to voluntary exercise, treatment with NMES may lead to delayed-onset muscle soreness (DOMS). Consequently, the practitioner needs to educate the patient regarding the likely effects of muscle soreness as would be done following volitional exercise. It is normal for the skin under the electrodes to feel warm and appear pink after treatment due to the electrothermal effects of the stimulus.

Box 13 ▪ 2 Tips for Electrode Placement

- If electrodes are too small for the targeted muscle, increased current density may lead to discomfort.
- If large electrodes are not available, splitting or bifurcating the two electrodes into four or using two channels will increase the target area.
- Several attempts may be needed to optimize the electrode placement to get the desired response.

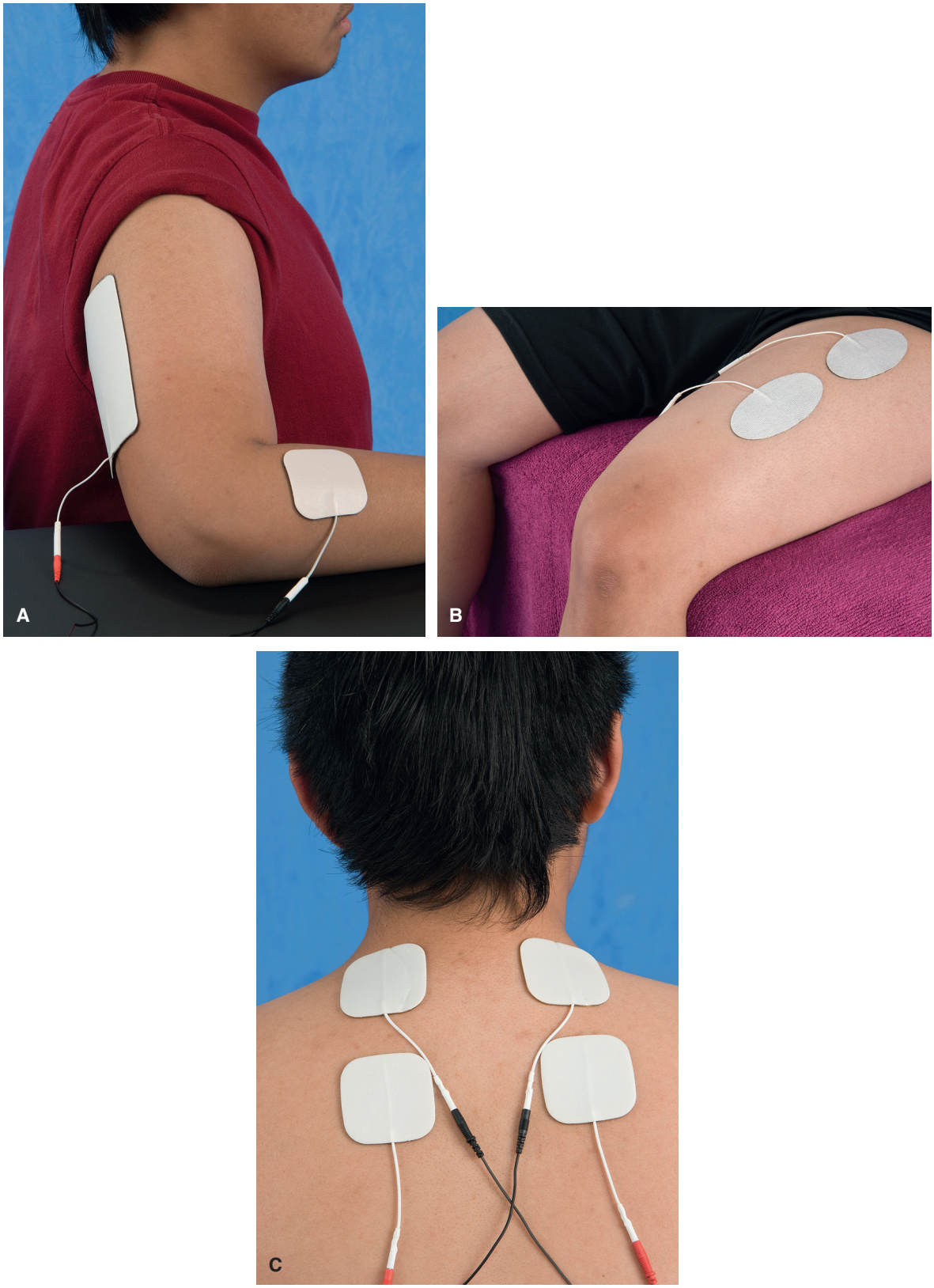

Fig 13 ■3 (A) A monopolar arrangement involves placing one electrode over the target muscle(s), such as the wrist extensors pictured here, and a larger electrode in another area where muscle activation is not desired, such as the triceps. Two different sizes of electrodes are used because the current density needs to be greater over the targeted muscle. (B) A bipolar arrangement places two electrodes over the targeted muscle(s) pictured here for the quadriceps femoris. (C) Another example of a bipolar arrangement splits or bifurcates the lead wires from the single circuit to use four electrodes over the target area. Two separate circuits in bipolar arrangement can also be used.

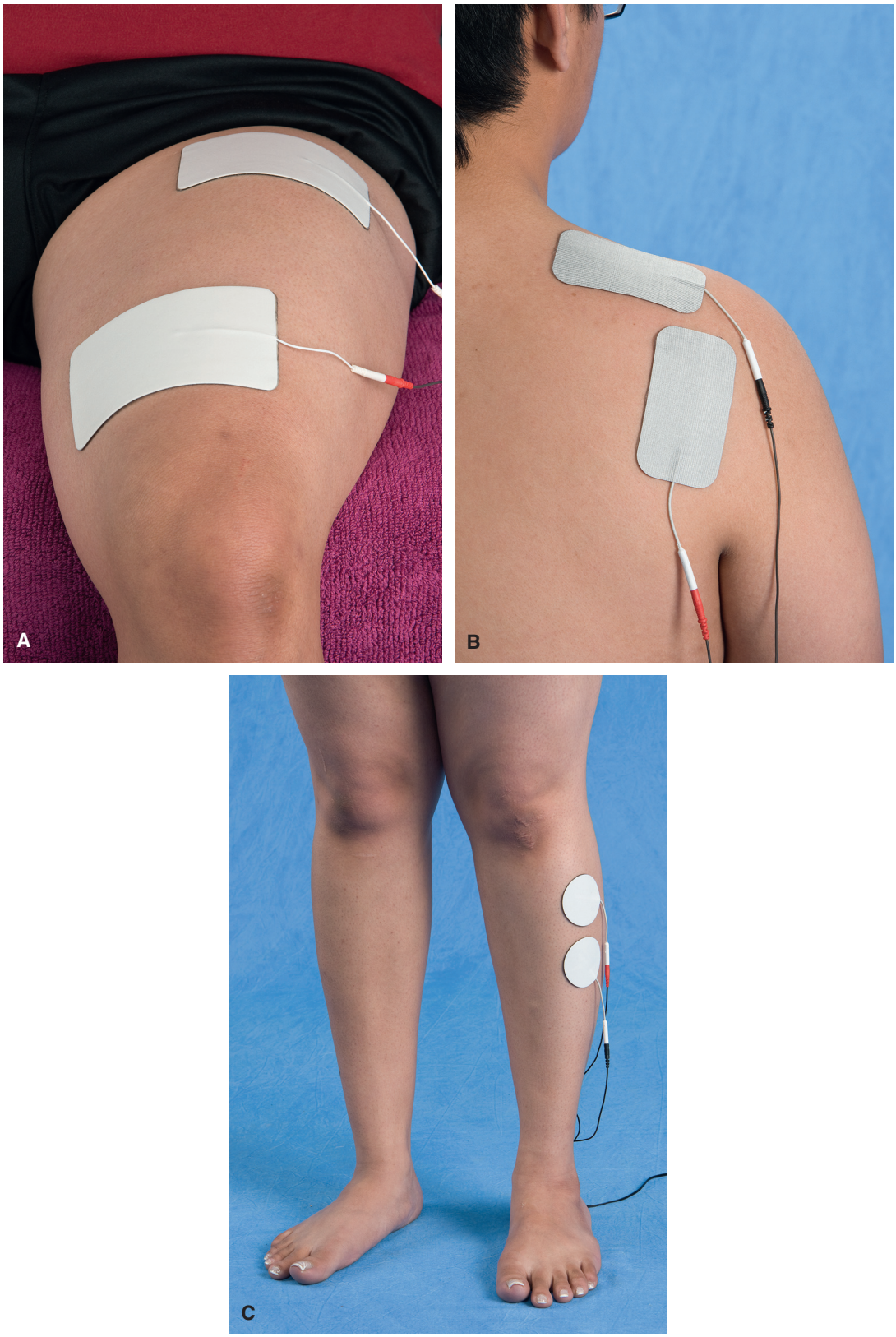

Fig 13 ■ 4 (A) This picture demonstrates one possible placement of electrodes to stimulate the quadriceps femoris muscle. To cause this large muscle group to contract, the practitioner should use appropriately sized electrodes, which may vary between patients. (B) Electrodes to posterior rotator cuff muscles; the exact location to optimize the response will vary among patients. The goal is to move the humerus superiorly into the glenoid fossa without creating abduction. (C) This picture demonstrates one possible placement of electrodes to obtain balanced dorsiflexion. This placement will vary among patients, and the practitioner may need to try several different placements before obtaining the desired response.

However, skin should be checked for any excessive redness or irritation. As with any exercise program, adjustments may need to be made to the NMES regimen based upon individual patient responses. For example, it may not be possible to achieve the targeted intensities on the first day of treatment because the patient may be apprehensive with the new program. Treatments from day to day may change. Each time that ES is utilized, practitioners should proceed with caution and be aware of the patient's response (both verbal and nonverbal).

CASE STUDY 13-1 NMES for Muscle Strengthening

A 42-year-old police officer presents with a chief complaint of decreased strength of the knee extensors after left ACL reconstruction 4 weeks ago. The ACL rupture occurred 6 months prior while playing basketball.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of NMES?

ANSWER: Yes, decreased strength secondary to orthopedic injury can be improved with NMES.

2. Is the patient appropriate for NMES (i.e., do any of the general precautions or contraindications to NMES apply to the patient, or are there any specific considerations regarding application of NMES to this patient)?

ANSWER: If the patient has been screened to ensure general precautions or contraindications do not apply, then it is appropriate to use NMES.

3. What are the specific goals to be achieved with the use of NMES?

ANSWER: Increase strength of the quadriceps femoris muscle by using NMES to evoke strong contractions in accordance with principles of muscle strengthening. A functional goal would be a return to full duty at work.

4. What specific form of ES would be appropriate for the patient?

ANSWER: A waveform that evokes muscle contractions. This would be either biphasic pulsed current or burst-modulated AC (i.e., Russian or Aussie).

5. What specific parameters of NMES are appropriate for the patient?

ANSWER:

Current: Biphasic pulsed current (or burst-modulated AC)
Pulse duration: 400 to 800 μsec
Frequency: 50 Hz (or 50 bursts per second)
Amplitude: A dosage set to obtain as strong of a contraction as tolerated or according to specific percentage of MVC
Ramp time: 1 to 2 sec
Duration: Select 1:5 ratio of 10 sec on, 50 sec off. Perform at least 10 contractions, 3 days per week.

6. What are the proper (i.e., effective and safe) application procedures for NMES related to this case example?

ANSWER:

Instruct the patient: Inform the patient of the purpose and procedure and explain the anticipated sensation and effect of the stimulation. The goal of treatment is a motor response, so explain to the patient how much contraction is expected and required for the treatment to be effective. Initially, it may be necessary to offer the patient a familiarization session to become accustomed to the sensation of ES.
Preinspection: Generally observe the area to be treated for skin integrity.
Electrode application: Two electrodes in a bipolar arrangement using a single channel, one proximally and laterally over the vastus lateralis and rectus femoris and one distally and medially over the vastus medialis. Application of the electrodes may require several attempts to optimize the response.
Patient position: Patient is positioned on an isokinetic dynamometer or table with the knee flexed 45° to 90°. The distal extremity should be securely fixed to ensure isometric contraction.
Treatment monitoring: Observe the muscle response and the reaction of the patient to NMES. If tolerated, the amplitude may need to be increased during the session to achieve the effect.
Postinspection: Remove the electrodes and inspect the skin for any signs of skin irritation or adverse effects. If redness occurs, explain to the patient that this is not uncommon and should disappear in less than 24 hours.

CASE STUDY 13-2 NMES for Decreasing Shoulder Subluxation

A 16-year-old female gymnast has chronic posterolateral glenohumeral joint instability that increases toward the end of practice sessions.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of NMES?
   ANSWER: Yes, assuming there are no structural problems that require surgery, it appears that muscles involved in shoulder stability are susceptible to muscle fatigue, and NMES-induced exercise can improve neuromuscular performance.
2. Is the patient appropriate for NMES (i.e., do any of the general precautions or contraindications to NMES apply to the patient, or are there any specific considerations regarding application of NMES to this patient)?
   ANSWER: If the patient has been screened to ensure general precautions or contraindications do not apply, then it is appropriate to use NMES.
3. What are the specific goals to be achieved with the use of NMES?
   ANSWER: Improve neuromuscular performance of specific muscles that contribute to stabilizing the shoulder during activity. A functional goal would be a return to full workouts without pain.
4. What specific form of ES would be appropriate for the patient?
   ANSWER: A waveform that is appropriate for evoking muscle contractions. This would be either biphasic pulsed current or burst-modulated AC (i.e., Russian).
5. What specific parameters of NMES are appropriate for the patient?
   ANSWER:
   Type of stimulator: A portable stimulator that can meet desired parameters is preferable so treatment can be done outside of the clinic.
   Current: Biphasic pulsed current (or burst-modulated AC)
   Pulse duration: 400 to 800 μsec
   Frequency: 50 Hz (or 50 bursts per second)
   Amplitude: A dosage set to obtain as strong of a contraction as tolerated
   Ramp time: 1 to 2 sec
   Duration: Select 1:2 ratio of 10 sec on, 20 sec off. Perform at least 15 contractions, 3 to 5 days per week. As treatment progresses, gradually decrease the off-time and increase the on-time to improve fatigue resistance and increase the number of contractions.
6. What are the proper (i.e., effective and safe) application procedures for NMES related to this case example?
   ANSWER:
   Preinspection: Generally observe the area to be treated for skin integrity.
   Electrode application: Two electrodes in a bipolar arrangement—one over the supraspinatus and one over the posterior deltoid. Additional electrodes may be added to scapular stabilizers, if necessary. Application of the electrodes may require several attempts to optimize the response.
   Patient position: Patient is seated in a chair.
   Treatment monitoring: Observe the muscle response and the reaction of the patient to NMES. The number of repetitions as well as the on:off ratio may need to be increased as treatment progresses to achieve the desired effects.
   Postinspection: Remove the electrodes and inspect the skin for any signs of skin irritation or adverse effects. If redness occurs, explain to the patient that this is not uncommon and should disappear in less than 24 hours.

NMES AND MOTOR UNIT RECRUITMENT

The ability to discriminately select and properly implement NMES requires an awareness of the physiological and functional differences that exist between electrically elicited and voluntary contractions. Specifically, it is important to understand potential differences with respect to temporal (i.e., firing pattern of recruited motor units/fibers) and spatial characteristics (i.e., location of recruited fibers within the muscle) of motor unit recruitment as well as the amount of fatigue realized when using NMES versus voluntary contractions. These issues are the subject of some debate,[48] and their impact on skeletal muscle function

must be considered in the design of appropriate training paradigms when using this biophysical agent.

The Henneman size principle of voluntary motor unit recruitment describes the progressive recruitment of alpha motor neuron cell bodies in order of increasing size from small to large.[49] Although this principle describes the activation of motor units based on their size rather than their type, it is often associated with the progressive recruitment of slow, typically small, motor units followed by fast, typically large, motor units. Some have suggested that the use of NMES results in a reversal of the size principle, thereby recruiting fast motor units prior to slow.[50,51] This rationale is based on the fact that larger cell bodies are more easily depolarized due to less impedance to current flow, and their axons have faster conduction velocities than their smaller counterparts.[52,53] Although the premise of a reversal in the size principle may hold true during direct nerve stimulation, evidence surrounding transcutaneous application of NMES suggests that muscle fiber recruitment during ES occurs in a nonselective, spatially fixed, and temporally synchronous pattern rather than a reversal of the physiological voluntary recruitment order.[48,54–56]

Key Point! A fundamental difference exists between activating motor units voluntarily versus electrical stimulation–induced. Voluntary activation results in a predictable order of recruitment, resulting in activation of fast fatigable fibers typically at high relative intensities, whereas, with ES, there is no predictable order of recruitment—thus, the potential to activate both slow and fast fibers at lower contraction intensities.

In addition to recruitment order, differences exist in the amount of muscle fatigue when voluntary and NMES activities are compared. A functional consequence of NMES is an increased fatigability relative to voluntary activation, which is independent of stimulation intensity.[57,58] Reasons other than recruitment order (i.e., recruiting fast, fatigable fibers) can account for this outcome. One explanation for this phenomenon is that during voluntary actions, asynchronous motor unit recruitment patterns allow for additional motor units to be activated when muscle fibers that were initially recruited become fatigued.[59] As previously mentioned, NMES recruitment is not based on motor unit characteristics, such as size or fiber type, and is spatially fixed. Thus, NMES recruitment does not allow for alterations in recruitment between repeated contractions during treatment and therefore contributes to increased fatigability.

An additional mechanism that explains increased fatigability during NMES is that, during voluntary actions, muscle force can be maintained by modulating the firing frequency of active motor units. During NMES, however, firing frequency is fixed and based on the selected frequency of the current used.[59] Thus, the ability to counter fatigue (i.e., maintain external force production) in voluntary efforts can be accomplished by one or both of the following: recruiting additional motor units as those initially recruited become fatigued (i.e., asynchronous recruitment) or activating more motor units at lower (i.e., subtetanic) firing frequencies. Neither of these recruitment strategies is available during NMES-induced muscle contractions. Therefore, recruitment of muscle fibers using NMES is spatially and temporally fixed and results in a subsequent drop in force whenever any of the fibers being activated become fatigued. This increased fatigability is a fundamental problem when using NMES for functional activities and could potentially limit training responses.

A common misconception associated with motor unit recruitment through NMES is that only motor units near the skin's surface can be activated. Patterns of activation after NMES of the quadriceps femoris have been mapped using magnetic resonance imaging (MRI).[57] MRI is a valid and reliable method of quantifying the amount of muscle utilized during both voluntary and electrically stimulated activities.[60] Images reveal that even during lower levels of stimulation intensity (about 25% of maximum voluntary isometric contraction), skeletal muscle fibers near the femur are activated. Even if the electrical current activates only the most superficial nerves, the muscle fibers innervated by these nerves are seemingly spread throughout the muscle. This results in the recruitment of muscle fibers located deep within the muscle (Fig. 13-5).

Key Point! Because of differences in the way motor units are activated during voluntary activity versus during ES, repeated contractions with ES will result in greater and earlier muscle fatigue than with voluntary actions.

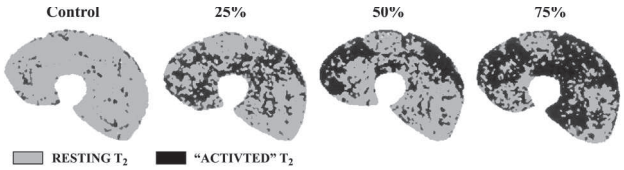

Fig 13 ■ 5 Representative single-slice T2 maps of the quadriceps femoris from one subject at rest (control) and after NMES at a level that elicited an initial torque equal to 25%, 50%, or 75% of MVIT. Dark regions represent "activated" skeletal muscle. *(Adapted with permission from Adams GR, Harris RT, Woodard D, Dudley GA. Mapping of electrical muscle stimulation using MRI.* J Appl Physiol. *1993;74(2):532-537.)*

Limitations of NMES

Although there are some distinct advantages for using NMES-evoked contractions to activate skeletal muscles, these same factors can be used to suggest limitations of its use. For example, consideration should be given to the fact that synchronous or fixed recruitment of motor units may not be advantageous from a metabolic perspective. Needle electromyography (EMG) has been utilized to measure the frequency of activation of human skeletal muscles during voluntary activation and has shown that slow and fast skeletal muscles have in vivo firing frequencies of approximately 10 and 30 Hz, respectively, during MVC.[58] These frequencies are lower than what is typically applied during clinical NMES. Oftentimes, practitioners utilize frequencies of 50 Hz or more to ensure tetanic contractions that produce great amounts of force.

Key Point! The indiscriminate activation of fast and slow motor units at firing frequencies that are higher than typically achieved voluntarily may contribute to increased fatigue and may potentially limit the amount of training that can be completed. This can be addressed to some degree by providing sufficient rest periods between contractions or by lowering the activation frequency. Previous studies have had success using 10-second contractions with 50-second rest intervals (i.e., 1:5 ratio).

Clinical Controversy

Because commonly used stimulation frequencies of NMES can exceed in vivo firing frequencies of some motor units, fatigue may happen sooner and may limit the extent of training that can be completed with NMES. Longer rest periods between contractions are recommended to limit the potential for this fatigue.

CASE STUDY 13-3 NMES for Endurance

A 38-year-old postal carrier is being treated for chronic mechanical low back pain aggravated by prolonged walking while carrying his mailbag. He has decreased endurance of the lumbar paraspinals.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of NMES?
   ANSWER: Yes, conditioning of the lumbar paraspinals is a key component to many rehabilitation programs for low back pain. ES can be used to address both gross strength and endurance for his occupational demands.
2. Is the patient appropriate for NMES (i.e., do any of the general precautions or contraindications to NMES apply to the patient or are there any specific considerations regarding application of NMES to this patient)?
   ANSWER: Yes, the patient is appropriate with no precautions or contraindications noted.
3. What are the specific goals to be achieved with the use of NMES?
   ANSWER: To increase the endurance of the patient's lumbar paraspinals with consideration of his prolonged walking while carrying the mailbag.
4. What specific form of ES would be appropriate for the patient?
   ANSWER: NMES with emphasis on parameters for increasing endurance. Biphasic pulsed current or burst-modulated AC (i.e., Russian or Aussie) can be used.
5. What specific parameters of NMES are appropriate for the patient?
   ANSWER:
   Pulse duration: 400 to 800 μsec
   Frequency: 60 to 80 pps; higher frequency will train the muscle for endurance
   Amplitude: Maximally tolerated contractions are not necessary because we are addressing endurance of the muscles.

Continued

CASE STUDY 13-3 NMES for Endurance—cont'd

Ramp time: 1- to 2-sec ramp-up
Duration: At least 10-sec contractions and up to 20 sec with progressively decreasing off-time

6. What are the proper (i.e., effective and safe) application procedures for NMES related to this case example?

ANSWER:

Electrode application: Bipolar electrode placement using two channels, one for each of the left and right lumbar paraspinals. One electrode placed paraspinally just proximal to L5 with the other on the ipsilateral side placed just above.

Preinspection: Generally observe the area to be treated for skin integrity.
Patient position: Patient is positioned comfortably, preferably in prone or side-lying position.
Treatment monitoring: Observe the muscle response and the reaction of the patient to NMES. Progressively increasing on:off over time may facilitate achieving goals of treatment.
Postinspection: Remove the electrodes and inspect the skin for any signs of skin irritation or adverse effects. If redness occurs, explain to the patient that this is not uncommon and should disappear in less than 24 hours.

Contraction-induced muscle injury and the resultant DOMS is typically associated with eccentric muscle actions.[61,62] Although this phenomenon is common to most resistance-training paradigms, the magnitude of this response may be exaggerated using NMES and could potentially limit training volume and subsequent adaptations. For example, in a traditional resistance-training program, eccentric and concentric forces are dictated by the external load applied. In addition, the amount of muscle recruited to accomplish these actions is less during the eccentric phase of the contraction. However, programs that incorporate electrically evoked actions may be performed on an isokinetic dynamometer where external loads are not predetermined or limited. The spatially fixed recruitment strategies previously discussed keep the amount of activation constant during NMES, thus resulting in eccentric forces well above those measured concentrically.[63] Thus, the use of high-intensity eccentric contractions during NMES could potentially induce greater muscle damage than a voluntary training program. These are limiting factors because practitioners often see the need to reduce eccentric loading with ES. However, it has been reported that eccentric contractions are necessary to obtain sufficient degrees of skeletal muscle hypertrophy.[64]

A final limitation to NMES training is the limited number of muscle groups that can be trained. As previously mentioned, high-intensity NMES is facilitated by the use of an isokinetic dynamometer or a similar piece of equipment that can safely secure the limb and control joint motion during training. Although traditional resistance-training programs favor large-muscle-group, multijoint exercises (i.e., dead lift, squat, bench press), it is difficult to incorporate NMES with these activities. NMES training is limited to muscle groups using isolated joints and within a given plane of motion. Examples of potential muscle groups to be trained include those that elicit flexion and extension about the ankle (triceps surae and tibialis anterior), knee (quadriceps femoris and hamstrings), and elbow (biceps and triceps brachii). Thus, although the benefits of NMES training are potentially great, significant limitations do exist to its use and resultant application to training.

Clinical Controversy

NMES is often criticized because it does not involve muscle activation during multijoint activities as traditional resistance-training programs can. However, NMES can be used during rehabilitation to retard loss of and increase strength when multijoint activities cannot be performed.

ELECTRICAL MUSCLE STIMULATION APPLIED TO DENERVATED MUSCLE

The efficacy of ES with muscles that have been denervated is controversial and has been questioned in the scientific literature.[65] It should be noted that when referring to the

use of ES with denervated muscle, the term *NMES* is no longer appropriate because this assumes the peripheral nervous system is intact and the muscle is being activated through the stimulation of the peripheral nerve. When using EMS for denervated muscle, the practitioner is attempting to directly activate the muscle through depolarization of the sarcolemma. This act requires different stimulation parameters from standard NMES. Differences include utilizing longer pulse durations, greater amplitude of stimulation, and often a different waveform (direct current). Many commercially available stimulators may not allow modulation of these parameters to accomplish direct stimulation of muscle. In addition, using direct current increases the risk of electrode-related burns, especially when using higher amplitudes of stimulation.

The literature on stimulating denervated muscle generally reports mixed results regarding its effect on preserving muscle and its potential effect on reinnervation.[65–69] Previously published studies have utilized a variety of stimulation parameters because most practitioners and researchers do not agree on the optimal method to stimulate denervated muscle. Some have suggested that a low-frequency current (2 to 4 pps) better preserves denervated muscle tissue; however, others argue that a higher-frequency current (20 to 40 pps) is optimal. Most would agree that the pulse duration should be greater than or equal to 1 msec. In fact, many studies have greatly exceeded this value.[65] There is some controversy as to whether nerve growth is suppressed when ES is used or if reinnervation is suppressed due to the activity at the motor end plate with ES. A review of relevant studies using ES for denervated muscle suggests several conflicting conclusions.[65]

Clinically, ES for denervated muscles has most commonly been applied to facial muscles after facial nerve palsy (i.e., Bell's palsy) to preserve the muscle while reinnervation occurs. Some studies suggest that using stimulation could be beneficial[66] and some claim it could be harmful.[70] In cases of lower motor neuron injury at the cauda equina, it has been reported that the affected muscle can be trained with a long pulse duration (30 to 50 msec), moderate frequency (16 to 25 pps), and high amplitude (250 mA).[67] However, it took these researchers 1 to 4 years to induce significant muscle hypertrophy. More research is needed to determine whether EMS for denervated muscle shows any future promise as a clinical intervention. There is a renewed interest in this topic for using functional electrical stimulation to provide function or to preserve muscle to allow for future regenerative techniques. However, this requires specialized equipment and results have been inconclusive thus far.[67,71,72]

Examination, Evaluation, and Prognosis

If ES for denervated muscles is to be considered, tests of strength, ROM, sensation, and function are important—just as with applying NMES for innervated muscle. A review of the patient's medical history should include the cause and length of time of denervation. Electromyography tests should be conducted by a trained electromyographer to evaluate the degree of denervation (see Chapter 17). Patient prognosis for improvement with EMS appears to depend upon length of time since denervation and the number of remaining motor units. Overall, the prognosis for strengthening denervated muscle with EMS is not as good as it is for innervated muscle.

Intervention

Because of the high variability among studies, parameters are more difficult to select in trying to stimulate denervated muscle. Table 13-7 provides the range of parameters used by the majority of studies reviewed. As shown in the table, very long pulse durations (msec as opposed to μsec) are required to stimulate denervated muscle. One potential risk associated with this is burning the patient. Careful attention must be given to the skin during treatment, especially because these patients are also likely to have impairments in sensation.

Electrodes used for denervated muscles tend to be either very large to cover the entire bulk of the muscle or very small to attempt to isolate the motor end plate. A probe type of electrode can also be used for stimulating small muscles (Fig. 13-6).

Key Point! The stimulation of denervated muscles is not widely endorsed but is often done despite questionable efficacy. We suggest that practitioners proceed with caution.

TABLE 13–7. EMS Parameters Typically Used for Stimulating Denervated Muscle[65,67]

Waveform	Monophasic or DC
Pulse duration	1–450 msec (long)
Frequency	1–500 pps
Amplitude	To obtain contraction but low to prevent burns
Ramp-up time	Not identified
Ramp-down time	Not identified
Duty cycle	Highly variable 30 minutes, 8 hours per day
Treatment time and duration	5–7 days per week 4 days to 4 years

Fig 13 ■6 An example of a stimulator designed to stimulate denervated muscle. The small tip is placed over the muscle. With denervation, the practitioner needs to move the point of the stimulator along the muscle to attempt to activate any intact motor units. The long pulse duration with this stimulator allows muscle fibers to be stimulated directly.

CASE STUDY 13-4 Electrical Stimulation for Denervated Muscle

A 43-year-old female awoke 10 days ago with insidious onset of right-sided facial paralysis with inability to fully close her lips and eye on the right side after a recent sinus infection. Examination shows only trace contraction of the right-sided facial muscles.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of NMES?
ANSWER: Yes, the patient has loss of volitional muscle control of the right-sided facial muscles. ES may be used to activate these muscles.

2. Is the patient appropriate for NMES (i.e., do any of the general precautions or contraindications to NMES apply to the patient or are there any specific considerations regarding application of NMES to this patient)?
ANSWER: No specific contraindications or precautions are present.

3. What are the specific goals to be achieved with the use of ES?
ANSWER: To stimulate the involved muscles in order to facilitate restoration of volitional control of the right-sided facial muscles.

4. What specific form of ES would be appropriate for the patient?
ANSWER: Based on the strength-duration relationship discussed in Chapter 9, activation of muscles demonstrating denervation requires a pulse duration and amplitude greater than that required for innervated muscle.

5. What specific parameters of ES are appropriate for the patient?
ANSWER:
Pulse duration: At least 1 msec or greater. Because many clinical stimulators do not offer pulse durations greater than 1 msec, practitioners should use the longest pulse duration possible. Direct current (DC) or monophasic currents are often used with a probe electrode and manual trigger controls to administer stimulation (see Fig. 13-6).
Frequency: Low frequency (1 to 4 pps) or higher frequency (20 to 40 pps)
Amplitude: A dosage set to elicit as much muscle contraction as tolerated by the patient
Ramp time: 1 to 2 sec for comfort
Duration: Contractions should be sustained for several seconds as tolerated; on-times of 3 to 10 sec with off-times of 30 to 50 sec between contractions.

6. What are the proper (i.e., effective and safe) application procedures for NMES related to this case example?
ANSWER:
Electrode application: Because of the smaller facial muscles, electrodes should be appropriately small for the area to be stimulated. Practitioners should be

CASE STUDY 13-4 Electrical Stimulation for Denervated Muscle—cont'd

aware of the increase in current density when using smaller electrodes. A probe electrode should be appropriately small and allow for more localized stimulation of specific facial muscles. Practitioners should also be aware that there is an increased risk of burning the patient, so close monitoring of the skin is essential.
Preinspection: Generally observe the area to be treated for skin integrity.
Patient position: Patient is typically seated in a chair.
Treatment monitoring: Observe the muscle response and the reaction of the patient to NMES. The amplitude may need to be increased during the session to achieve the effect, if tolerated.
Postinspection: Remove the electrodes and inspect the skin for any signs of skin irritation or adverse effects. If redness occurs, explain to the patient that this is not uncommon and should disappear in less than 24 hours.

BIOFEEDBACK

Electromyographic (EMG) biofeedback is the monitoring, detection, or assessment of skeletal muscle activity so that the information gained can be used by the patient and clinician to influence future activity of skeletal muscle, whether for increasing or decreasing activity.[73] Biofeedback shares the same purpose as other forms of electrotherapy—to improve function and decrease pain. Where biofeedback differs is that none of the currents or waveforms previously described are actually delivered to the patient. Instead, the electrical activity generated by contraction of skeletal muscle is detected and used for therapeutic purposes. Because current never reaches the patient, biofeedback is not generally considered an electrotherapeutic agent; however, because the electrical activity is still part of the therapeutic process, biofeedback is discussed with other forms of electrotherapy used for therapeutic purpose.

Key Point! Biofeedback does not involve the delivery of electrical current. It can be offered as an option in initial therapy sessions when patients are apprehensive about ES or when the clinician wants to provide kinesthetic awareness of motion. After a trial or two of biofeedback, many patients feel more comfortable receiving ES for muscle activation.

Clinically, EMG biofeedback is generally used to either increase or decrease activity of skeletal muscle. Techniques in which increased volitional activity of muscle is desired are considered facilitatory; techniques in which decreased activity is desired are considered inhibitory. Examples of facilitatory and inhibitory EMG biofeedback are listed in Table 13-8.

Recording and Displaying the EMG Signal

EMG biofeedback requires specialized equipment to detect and record the electrical activity associated with muscle recruitment (Fig. 13-7). A device for detecting and transducing the electrical activity of muscle contraction into visual or audio feedback to the patient is required. Most biofeedback devices offer both visual and audio feedback and are portable.

TABLE 13–8. Clinical Examples of Facilitatory and Inhibitory Biofeedback

Facilitatory	• To increase muscle activity after surgery or injury when volitional recruitment is impaired • To normalize the balance of muscles acting at a joint where one muscle group may be insufficient • To improve volitional motor control following dysfunction of the central nervous system • To increase volitional control of pelvic floor muscles for rehabilitation of urinary incontinence
Inhibitory	• To help decrease activity in muscles demonstrating spasticity caused by dysfunction of the central nervous system • To help decrease activity in muscles demonstrating increased activity caused by postural stress or anxiety • To help decrease muscle activity associated with chronic pain

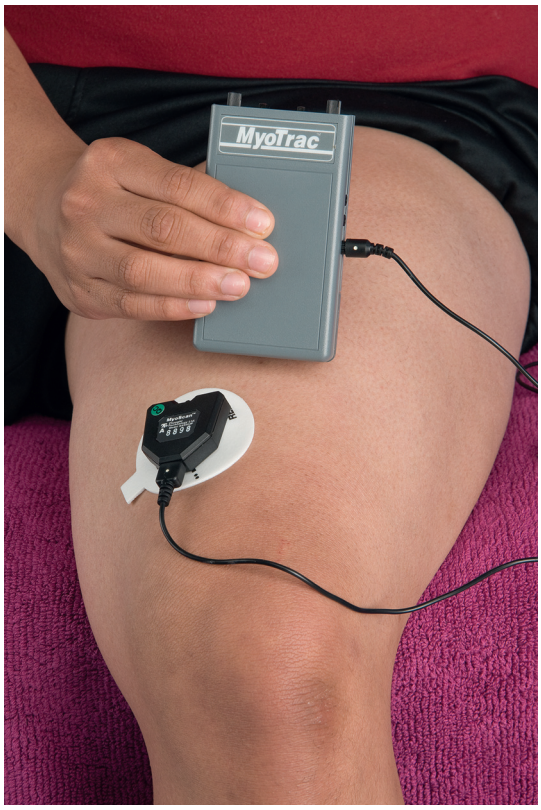

Fig 13 ■ 7 An example of biofeedback device and electrode placement.

Electrical activity associated with skeletal muscle depolarization is measured in units of microvolts (1,000,000 μV = 1 V) and is then amplified to millivolts for transduction to audio or visual feedback. The degree to which these electrical signals are amplified reflects *sensitivity,* which refers to the ability to detect an event. In terms of biofeedback, *sensitivity* refers to the ability to detect the electrical activity associated with muscle contraction. Typical devices used for EMG biofeedback offer sensitivity settings (sometimes called *gain*) of 1, 10, 100, or 1,000 microvolts (μV). This means the smallest level of muscle activity that can be detected is 1 μV. Sensitivity and gain are inversely related, although the terms are often used synonymously, such that at the highest sensitivity, the gain setting is lowest. For example, at the lowest gain setting of 1 μV, the sensitivity is greatest, capable of detecting as little as 1 μV of change in muscle activity. In contrast, a high gain setting of 1,000 μV is much less sensitive and only capable of detecting change of 1,000 μV. As the need for sensitivity decreases, the gain setting will be increased accordingly. The sensitivity of a biofeedback device is set depending on the need for amplification and the clinical goals. Less sensitivity is needed when muscle activity is very high. More sensitivity is needed when volitional muscle activity is low.

Electrode Type and Electrode Placement Considerations for EMG Biofeedback

The electrodes used for biofeedback are often specifically made for detecting electrical activity of muscle, but some devices that serve as electrical stimulators also offer EMG biofeedback and use conventional disposable adhesive electrodes similar to those used with other forms of electrotherapy. Placement of electrodes should consider the intent of the biofeedback (i.e., facilitatory or inhibitory) and whether the muscles are demonstrating high or low activity. The muscle fibers most likely to be detected are those closest to the electrodes, if these muscle fibers are activated. A wider electrode placement will detect electrical activity from a larger volume of muscle than will a narrower electrode placement. When trying to assess "very active" muscles, closer electrode placements are recommended. In contrast, when the volitional activity of muscle is decreased, there is a greater need to assess or monitor a larger muscle volume. In this case, a greater sensitivity and wider electrode placement is recommended to detect activity over more muscle volume. Electrode size does not increase the amplitude of the activity detected but rather simply detects more area of muscle fiber.

Patient Training Strategies With EMG Biofeedback

A requirement for the use of clinical EMG biofeedback is that the muscle be at least partially innervated and that the patient be able to somewhat activate the muscle. The level of muscle activity that the patient is able to reach, whether it is increased or decreased activity, is termed the *threshold*. If the clinical goal is to increase volitional activation of a muscle, then a facilitatory threshold is set. When the patient increases muscular activity to a threshold target, audio or video feedback is provided. If the clinical goal is to decrease volitional activity of muscle, then an inhibitory threshold may be set so that when the patient decreases the muscular activity to a threshold target, audio or video feedback is likewise provided.

As a patient is able to increase volitional activity, the sensitivity can be decreased and the electrode placement can be narrowed to focus on more specific areas that may

need additional facilitation. As a patient is able to relax or quiet a highly active muscle, the sensitivity may need to be increased and electrode placement widened to continue assessing muscle activity.

EMG biofeedback provides a clinically useful and indirect intervention to increase or decrease volitional muscle control by recording muscle activity and using this information to allow the patient to alter future muscle activity. Because no electrical stimulus is applied to the patient, there are no contraindications to using EMG biofeedback, assuming volitional activation of the muscles to be examined is appropriate (Box 13-3).

Box 13 ■ 3 Six Questions to Answer When Considering Use of Biofeedback

1. Is the patient appropriate for use of biofeedback?
2. Which muscles are to be monitored?
3. Is the intent to facilitate or inhibit muscle activity?
4. How sensitive must the assessment be?
5. Should the electrodes be placed close together or wider apart?
6. How will these factors change as the patient improves?

CASE STUDY 13-5 Biofeedback for Facilitating Muscle Activation

A 76-year-old female with right total knee arthroplasty completed 4 weeks ago is referred for strengthening of the knee extensors. She is unable to tolerate NMES at a dosage intensity sufficient for increasing strength. Biofeedback is chosen to assist her in activating her quadriceps.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction, limitation, or problem that can be improved with the use of NMES?
ANSWER: Yes, the patient has postoperative weakness and dysfunction with contracting her quadriceps.

2. Is the patient appropriate for NMES (i.e., do any of the general precautions or contraindications to NMES apply to the patient or are there any specific considerations regarding application of NMES to this patient)?
ANSWER: Yes, given that biofeedback only records electrical activity from the patient rather than delivering it, EMG biofeedback is appropriate.

3. What are the specific goals to be achieved with the use of NMES?
ANSWER: To enhance the patient's ability to volitionally recruit her muscle for purposes of increasing strength.

4. What specific form of ES would be appropriate for the patient?
ANSWER: EMG biofeedback for facilitation.

5. What specific parameters of EMG biofeedback are appropriate for the patient?
ANSWER: Initially, the clinician chooses a higher sensitivity setting (gain of 1 μV) and wider electrode placement over the proximal muscle belly and the distal vastus medialis because the patient demonstrates decreased volitional activity of the quadriceps. Two weeks later, the patient's right knee extensor strength is measured at 60% of the left. Because the patient is demonstrating increased ability to volitionally activate the quadriceps, the clinician chooses to decrease the sensitivity (gain increased to 100 μV) and place the electrodes closer together over the vastus medialis.

Documentation Tips

Appropriate documentation of NMES application should include the following:

- Waveform type
  - Russian, biphasic pulsed, etc.
- Waveform parameters
  - Pulse duration and frequency, contraction and rest time, ramp-up and ramp-down
- Electrode
  - Type, shape, and size
  - Placement or location
  - Integrity of skin before and after treatment
- Patient position
- Treatment duration
  - Number of contractions or duration of treatment

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CHAPTER 14

NMES AND FES IN PATIENTS WITH NEUROLOGICAL DIAGNOSES

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Patients with neurological conditions have impairments and functional limitations that may be addressed through the use of electrical stimulation. For example, a person who has sustained a stroke may have multiple impairments, such as decreased strength, motor control, and passive range of motion (ROM); compromised balance; and spasticity. These impairments contribute to functional limitations and disability. Mobility is compromised, so an important component of rehabilitation is to improve mobility to allow for greater independence. Neuromuscular electrical stimulation (NMES) and functional electrical stimulation (FES) may be used clinically to address some of these areas. For patients with neurological impairments, NMES is defined as the use of electrical stimulation (ES) to activate muscles through stimulation of intact peripheral motor nerves, and FES is the use of NMES to promote functional activities.[1]

EXAMINATION NEEDS

A thorough examination must be performed to determine the patient's appropriateness for receiving ES (Box 14-1). Table 14-1 identifies items that should be considered when determining if a patient with a neurological condition is suitable for ES. This table is not all-inclusive. Other pertinent examination items specific to the patient should be included.

One examination item is testing muscle innervation, which is critical to deciding appropriateness for ES. The patient must have an upper motor neuron injury to the targeted muscles to obtain a muscle response with standard clinical stimulation. A quick test for gross innervation is to apply electrical stimulation and observe if the muscle achieves a fused contraction. However, a more in-depth evaluation may be needed if sensation or spasticity prevents a visible response. An examination looking for signs of an upper motor neuron lesion (i.e., presence or absence of spasticity) should also be performed. If denervation is suspected, nerve conduction velocity or electromyography (EMG) tests can be conducted by a trained electroneuromyographer to evaluate the denervation and appropriateness for electrical stimulation.

Box 14 ■ 1 Concerns Specific to Diagnosis

Stroke

- Cognitive status
- Preexisting medical issues
- Spasticity
- Blood pressure

Spinal Cord Injury

- History of spontaneous fractures (osteoporosis)
- History of autonomic dysreflexia
- Sensation
- Orthopedic concerns
- Respiratory demands
- Spasticity
- Pressure sores
- Preexisting medical issues

Cerebral Palsy

- Orthopedic issues
- History of seizures
- Implanted devices
- Cognitive status

Multiple Sclerosis

- Spasticity
- Fatigue
- Cognitive status
- Preexisting medical issues

Pediatric Onset Conditions

- Scoliosis
- Hip subluxation
- Torsional deformities of bones
- Osteoporosis

TABLE 14–1. Examination and Rationale for the Use of Electrical Stimulation in Populations With Neurological Conditions

Tests of:	Question	Reason
Muscle innervation	If a neurological condition is present, are muscles capable of being stimulated?	Electrical stimulation applications typically require innervation of the muscle (an upper motor neuron injury).
Strength	What is current strength (manual muscle test or muscle torque)?	To determine muscles to treat and effect of treatment.
Range of motion	Are any ROM limitations present?	Decreased ROM may impact functional outcomes. Electrical stimulation may increase ROM.
Sensation	Is sensation present, absent, or diminished?	More frequent monitoring is needed with insensate skin.
Pain	Is pain present at rest or with activity? How severe?	To determine any positive or negative effects on pain.
Spasticity	Is spasticity present?	Spasticity can impact the choice of stimulation parameters. Spasticity may be positively or negatively impacted by electrical stimulation.
Function	Are any functional limitations present?	To determine effect of treatment on function.
Cognitive status	Is cognitive status sufficient to provide feedback?	Safety of use.
Caregiver assistance	Does patient require caregiver assistance to use electrical stimulation at home if needed?	Determine availability of assistance.
Other treatments	Are any other treatments being used or modified that may impact the intervention with electrical stimulation?	May impact ability to assess effects of intervention.

NEUROMUSCULAR ELECTRICAL STIMULATION

NMES for Muscle Strengthening

NMES has been applied to patients with neurological conditions to enhance strength in many muscle groups (Table 14-2). Primary diagnoses studied include stroke, cerebral palsy (CP), spinal cord injury (SCI), and multiple sclerosis (MS). Studies have examined NMES using a strengthening paradigm and have shown that patients who display decreased muscle strength due to a neurological problem may benefit from NMES, especially when muscle atrophy is present. However, a systematic

TABLE 14–2. Summary of Studies Using NMES for Strengthening

Authors	Population	Method	Results
Powell et al, 1999[4]	Acute stroke	Rehab + NMES to wrist extensors 3x/week for 8 weeks vs. rehab alone	Increased isometric strength and hand function
Kimberley et al, 2004[5]	Chronic stroke	NMES to wrist and finger extensors vs. sham treatment	Increased finger extensor strength, grasp and release ability, and cortical activity
Knutson et al, 2009[6]	Chronic stroke	NMES for hand opening (on-/off-times started in cycles of 5/20 seconds with on-times increasing and off-times decreasing over the 12-week intervention	Increased finger extension AROM, torque production, and upper extremity function
de Kroon and IJzerman, 2008[7]	Chronic stroke	EMG triggered NMES to the wrist extensors vs. cyclical NMES	Improvements in grip strength and function with no differences between groups
Boyaci et al, 2013[8]	Acute stroke	EMG triggered NMES to the wrist extensors vs. NMES	Increases in grip strength, spasticity, FIM scores, Fugl-Meyer scores, and Motor Activity Log.
Wright and Granat, 2000[9]	Hemiplegic CP	35-day program of NMES 30 min/day to wrist extensors	Increase wrist extension strength and hand function
Kamper et al, 2006[10]	Hemiplegic CP	NMES to wrist extensors and flexors (15 min, 6 days/week, for 3 months)	Improved wrist extension torque and muscle coactivation with movement
Vaz et al, 2008[11]	Hemiplegic CP	NMES to wrist extensors (3x/week for 8 weeks)	Increase in wrist strength without functional change
Ozer et al, 2006[13]	Hemiplegic CP	NMES to wrist extensors and dynamic bracing vs. dynamic bracing alone	Increased grip strength and ROM
Hazlewood et al, 1994[15]	Hemiplegic CP	NMES to dorsiflexors 1 hour a day for 35 consecutive days vs. matched controls	Increased dorsiflexion strength and PROM
Yıldızgören et al, 2014[12]	Hemiplegic CP	NMES to wrist extensors	Increased grip strength, hand function, decreased spasticity
van der Linden et al, 2003[16]	Diplegic, hemiplegic, quadriplegic CP	NMES to gluteus maximus for 1 hour/day, 6 days/week for 8 weeks	No effects on strength, gait, or PROM
Stackhouse et al, 2007[145]	Diplegic CP	Intensity of isometric quadriceps and plantar flexor stimulation based on ≥ 50% of maximal voluntary contraction (percutaneous electrodes)	Larger strength and walking speed gains compared to volitional exercise group
Bélanger et al, 2000[22]	SCI	NMES to quadriceps 5 days/week, for 24 weeks, one leg given resistance, the other no resistance	Greater rate of increase seen on the resisted side. Both sides increased distal femur and proximal tibia bone density.
Livesly, 1992[26]	MS	NMES to quadriceps and hamstrings, 12 min, 5 days/week, for 6 weeks vs. sham	No to small effect on strength.

FIM = functional independence measure.

review[2] concluded that NMES has modest support in the stroke population and that it is difficult to make conclusions for other neurological populations because of the small number of low-quality studies. A more recent review concluded that, although studies are limited, NMES is an effective biophysical agents in patients with central nervous system lesions.[3]

NMES improves strength and other impairments in people with neurological conditions, so the practitioner may have combined goals of enhancing strength, ROM, and functional outcomes in these patients. Overall, studies are difficult to compare due to differing outcome measures, stimulation parameters, and treatment durations. Table 14-3 identifies ranges of parameters used.

This section will focus on NMES applications. However, many studies have used FES to improve function yet reported a strengthening effect of the treated muscles. FES studies are described in the FES section of this chapter because these two techniques in theory use a different approach. It is important to keep in mind that traditional strengthening protocols require overload for strengthening to occur. Although this principle also applies to NMES for strengthening, many studies investigating NMES in those with neurological conditions do not overload the muscle to the extent used for treating musculoskeletal diagnoses without neurological injury.

Key Point! Use of NMES and FES in patients with neurological disorders is based on having intact peripheral nerve innervation to the target muscle groups (i.e., an upper motor neuron injury).

Stroke

Research has shown improvements in upper-extremity strength and function following an NMES strengthening program. Improvements have been reported in isometric strength of the wrist[4] and finger extensors,[5,6] grip strength,[7] hand function, [4,6] grasp and release ability,[5] and cortical activity (seen via functional MRI).[5] A randomized controlled trial (RCT) compared EMG-triggered NMES to NMES exercise post-acute stroke and reported no differences in functional outcomes between groups; however, both groups improved.[8]

Cerebral Palsy

Improvements have been reported following both upper- and lower-extremity NMES applications in children with CP. Upper-extremity changes have included improved wrist extension strength,[9–12] hand function,[9,12] muscle coactivation with movement,[10] grip strength,[13] ROM,[13] and spasticity.[12] A recent review concluded that NMES is effective for increased muscle strength, ROM, and function in children with CP despite the lack of RCTs.[14] NMES to the dorsiflexor muscles has led to gains in dorsiflexion strength and passive ROM,[15] while NMES to the gluteus maximus resulted in no gains.[16] Electrical stimulation was also found to have a medium effect on gait-related impairments in children with CP in a review; however, this review contained articles on NMES and FES, so it is difficult to separate out the effects.[17]

For children with CP, a popular technique in the 1990s and early 2000s was threshold electrical stimulation, which involves low-level (sensory only) stimulation

TABLE 14–3. Parameters Typically Seen for Muscle Strengthening[4,6–8,11,12,15,22,27,28,142,146–151]

Waveform	Symmetrical or asymmetrical biphasic pulsed current; burst-modulated AC (i.e., Russian current) or burst-modulated biphasic pulsed current
Pulse duration	100–800 μsec
Frequency	30–100 pps
Amplitude	To obtain strong contraction (should be related to a percentage of maximum voluntary isometric contraction)
Ramp-up time	1–5 seconds
Ramp-down time	1–2 seconds
Duty cycle	1:3 to 1:5 with on-times up to 10 seconds
Treatment time and duration	At least 10 contractions or up to 1 hour/day, three to five times per week, for 4 to 8 weeks

delivered overnight with the theory that muscle strength would result from an increase in blood flow. Even though some benefits were reported early,[18] more recent RCTs have shown it to have no impact on motor or walking function,[19] spasticity,[20] ROM,[20] or muscle growth.[20] Therefore, this technique is not recommended.

Spinal Cord Injury

For patients with complete SCI (no motor function below level of injury), electrical stimulation has been studied to increase muscle mass and stimulated muscle strength. However, most of these studies have used FES rather than NMES to achieve outcomes. (See the "Functional Electrical Stimulation" section later in this chapter.) Two studies have looked specifically at NMES for strength and reported increases in strength,[21,22] self-care,[22] and mobility.[22] Another application involves stimulation to the gluteal muscles to improve seated pressure for skin protection and prevention of pressure ulcers. Studies have reported decreased seating interface pressures,[23,24] decreased pain,[21] increased skin pliability,[25] and increased muscle bulk[25] using NMES.

Multiple Sclerosis

One study (NMES vs. sham treatment) examined NMES strengthening for people with MS[26] and showed little to no effects on quadriceps and hamstring muscle strength following a 12-minute, 5 times weekly, 6-week NMES program (3, 10, or 35 pps; 200 μsec). Another study[27] used NMES to augment strength training of the quadriceps and found no added effect of NMES (100 pps, 400 μsec, 3 sec on, 4 sec off). However, the NMES was set to achieve only 45 degrees of movement of knee extension so may not have been sufficient to augment strengthening.

Examination for Muscle Strengthening

Specific examination items for strengthening are listed in Table 14-4.

Intervention for Muscle Strengthening

The practitioner needs to decide on the optimal means of applying NMES. The appropriate muscle stimulator must be chosen based upon desired stimulation parameters to achieve a strong muscle response. Electrode placement, on- and off-times, number of repetitions, and frequency and duration of treatment are all important considerations. The practitioner should apply the basic principles of muscle strengthening in designing the intervention, always monitoring the patient's response to treatment. However, there are some differences between voluntary exercise and exercise with NMES in terms of muscle fiber type recruitment that should be kept in mind.

TABLE 14–4. Examination and Rationale for NMES for Strengthening

Tests of:	Question	Reason
Muscle innervation	Are muscles capable of being stimulated?	NMES for strengthening typically requires innervation of the muscle.
Strength	What is current strength (manual muscle test or muscle torque)?	To determine effect of treatment.
Range of motion	Are any ROM limitations present?	Decreased ROM may impact functional outcomes.
Sensation	Is sensation present, absent, or diminished?	More frequent monitoring needed with insensate skin.
Pain	Is pain present at rest or with activity? How severe?	To determine effect of strengthening on pain.
Spasticity	Is spasticity present?	Antagonist spasticity may affect ability to accurately assess strength.
Function	Are any functional limitations present?	To determine effect of treatment on function.
Cognitive status	Is cognitive status sufficient to provide feedback?	Safety of use.

Stimulation Parameters

See Table 14-3 for typical strengthening parameters. The practitioner does have options in selecting these parameters to achieve the desired response, and as with any intervention, patient response will guide the choice of stimulation parameters. If the goal of NMES is to enhance muscle strength, the highest pulse duration, amplitude, and frequency tolerated by the patient should be used.[28] It is important to realize, however, that maximizing stimulation parameters will lead to greater muscle fatigue. Therefore, it is critical to choose an appropriate duty cycle to allow for muscle recovery between repetitions (Box 14-2).

Key Point! Ramp-up and ramp-down times are often selected for patient comfort. However, it is important to remember that a longer ramp-up time will lead to a slower generation of force and is not always the most comfortable for the patient. The patient's feedback and muscle response will help guide decision-making. In patients with neurological conditions who have spasticity, a longer ramp-up time is often required when stimulating the antagonist muscle to avoid a quick stretch of the spastic muscle.

Electrode Placement

Electrode placement for NMES applications depends primarily on the size of the targeted muscle. Because the goal is to optimize muscle strength, the electrode should be of sufficient size to recruit as many motor units as possible.

Box 14 ▪ 2 Critical Considerations for Strengthening With NMES

- Overload of muscle is important.
- Longer ramp-up times may be uncomfortable when trying to get a maximal contraction.
- There is some evidence for the use of NMES in neurological populations, but stronger research is needed.
- A wide range of stimulation parameters are reported, making it difficult to determine optimal parameters and dosing.

Monitoring Treatment

Patients with neurological conditions have impairments that require close monitoring during electrical stimulation. For example, sensation may be decreased over the targeted area, hypersensitivity may be present, or spasticity may be triggered by NMES. As with any exercise program, adjustments may need to be made to the NMES program based on patient response. A patient should not be sent home with a home-based NMES program until the practitioner is confident that it can be properly carried out at home (Box 14-3).

NMES for Increasing Range of Motion

NMES has been applied as an alternate method to increase tissue extensibility or ROM. Chapter 12 discusses the principles behind the use of modalities to address decreased ROM. NMES is potentially advantageous for increasing ROM, as it can provide repetitive motion of the shortened musculotendinous

Box 14 ▪ 3 General Application and Monitoring Information for NMES and FES

- Instruct the patient: Inform the patient of the purpose and procedure. Be sure to explain the anticipated sensation and effect of the stimulation. Because you want a motor response, explain to the patient how strong of a contraction is expected and required for the treatment to be effective. Initially, it may be necessary to offer the patient a familiarization session to become accustomed to the sensation of electrical stimulation. With a child, make a game of it or distract the child with a video or other activity.
- Preinspection: Inspect the area to be treated for skin compromise and assess for intact sensation over the area to be treated.
- Treatment monitoring: Monitor the patient's response to treatment by observing the muscle response and his reaction to it. The amplitude may need to be increased during the session to achieve the effect if tolerated. If the patient has cognitive deficits, ask the patient for feedback about how the electrical stimulation feels. If the patient is unable to respond, stop the treatment after several minutes, remove the electrodes, and check the skin. If there are no skin issues, the treatment can resume.
- Postinspection: Remove the electrodes and inspect the skin for any signs of skin irritation or adverse effects. If redness occurs, explain to the patient that this is not uncommon and should disappear in less than 24 hours.

complex and surrounding tissues over a period of time by stimulating the antagonist muscle. NMES used in this manner may be an efficient way for a practitioner to increase ROM, as the patient or family can often be taught to perform this technique safely at home, increasing the amount of time that the tissues are exposed to stretch.

Studies have reported that NMES prevented wrist flexion contractures following acute stroke,[29] increased wrist extension passive ROM (PROM) following stroke,[30] increased wrist flexion PROM and improved ability to manipulate objects for children with CP,[9] and increased ankle dorsiflexion PROM for children with CP.[31] Other studies have reported increases in PROM when NMES has been applied for a variety of reasons,[14,32–34] including strengthening, reducing spasticity, and increasing function.

ROM improvements have been seen with NMES either used directly to increase ROM or as a result of NMES applied for another application. Due to these findings, more controlled research is needed to identify the best method to apply when using NMES to increase tissue extensibility and to predict anticipated outcomes.

Examination, Evaluation, and Prognosis

Table 14-5 indicates some important aspects of an examination when considering using NMES to increase tissue extensibility. The general precautions and contraindications for using NMES mentioned in Chapter 10 are still applicable and should be included in the examination.

Intervention

Electrodes should be selected based on the size of the antagonist muscle. Table 14-6 identifies commonly used stimulation parameters to improve tissue extensibility.

Key Point! A critical factor when using NMES for tissue extensibility is to minimize fatigue so that the patient can increase treatment time. Fatigue can be minimized by using the lowest frequency and highest amplitude that creates the needed force.[28]

Amplitude should be set to achieve a 3+/5 contraction that will stretch the tightened muscle. A contraction stronger than this may cause discomfort because the joint will be more forcefully moved to its limits in ROM. As maximal stimulation parameters aren't used, a portable stimulator may be sufficient for this application, making home treatment more feasible. The greatest variation in treatment recommendations involves the treatment times and duration. Therefore, the practitioner should set the treatment times first on patient

TABLE 14–5. Examination and Rationale for NMES for Tissue Extensibility

Tests of:	Question	Reason
History	Is there a history of bony injury or deformity?	NMES will address only soft tissue impairment.
Strength	Is voluntary movement present in agonist or antagonist?	Potential for strength improvement using NMES.
Range of motion	What is available ROM? How long has decreased ROM been present?	Significantly decreased ROM and chronic contracture have poorer prognosis.
Sensation	Is sensation present, absent, or diminished?	More frequent monitoring needed with insensate skin.
Pain	Is pain present at rest? With activity? How severe?	To determine effect of increasing ROM on pain.
Spasticity	Is spasticity present?	Strong spasticity may impact outcomes if not addressed.
Function	What are the functional limitations due to decreased ROM?	To determine effect of treatment on function.
Cognitive status	Is cognitive status sufficient to provide feedback?	Safety of use.
Caregiver assistance	Does patient require caregiver assistance to use NMES at home?	Determine availability of assistance.

TABLE 14–6. Parameters Typically Seen for Enhancing Tissue Extensibility[31–34,36,37,152]

Waveform	Symmetrical or asymmetrical biphasic
Pulse duration	200–300 μsec
Frequency	12–33 pps
Amplitude	To obtain 3+/5 contraction
Ramp-up time	3 seconds (for comfort)
Ramp-down time	1–2 seconds
Duty cycle	1:1 (typically 10 seconds on, 10 seconds off)
Treatment time	15 minutes to 6 hours per day, 1 to 4 times/day, and duration for 2 weeks to 6 months

tolerance (monitoring for muscle soreness) and then on clinical judgment based on the goals of the treatment and the patient's current status (i.e., acute versus chronic stroke). In starting a program using NMES, the practitioner may begin with a longer off-time and a shorter treatment time and advance quickly to a 1:1 duty cycle and longer treatment times in order for the patient to develop tolerance and muscle endurance for the length of the treatment (Box 14-4).

Box 14 ■ 4 Critical Considerations for Using NMES to Increase ROM

- Parameters should be set so that stretching principles are followed (low load, prolonged application).
- Stimulation parameters should be set to minimize fatigue (low frequency and amplitude), because goal is longer treatment time.
- A wide range of stimulation parameters are reported, making it difficult to determine optimal parameters and dosing.
- Stronger research is needed.

NMES for Decreasing Spasticity

Spasticity is defined as a velocity-dependent increase in tone that is frequently assessed by moving the limb quickly and observing the response.[35] Clinically, spasticity can interfere with function. Three theories have been proposed for using NMES to decrease spasticity. The first theory states that NMES applied to the antagonist muscle decreases spasticity through reciprocal inhibition of the spastic agonist muscle.[36] Reciprocal inhibition occurs through inhibitory interneurons within the spinal cord.[37] The second theory proposes that NMES applied to the spastic agonist muscle works

CASE STUDY 14-1 NMES to Improve Strength

An 8-year-old boy with mild spastic diplegic cerebral palsy is experiencing an increase in his crouched gait pattern (increased hip and knee flexion during stance) that you feel is due to decreased quadriceps muscle strength. He is having trouble keeping up with his peers while playing soccer.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction or impairment that may be improved or lessened by the use of NMES?
ANSWER: This patient may benefit from quadriceps strengthening with NMES.

2. Why would NMES be a consideration for this patient?
ANSWER: He may benefit from increased strength and may have trouble fully activating his muscles on his own. Goals include increased strength of the quadriceps muscles and decreased knee flexion during stance. A functional goal could be to have him play soccer with his friends.

3. What specific outcome measures can be used to assess the effectiveness of NMES?
ANSWER: Manual muscle testing (MMT) for strength, gait speed, and running speed, 6-minute walk test

4. Are there any precautions or contraindications to consider with this patient?
ANSWER: Considerations when designing the treatment include orthopedic concerns, cognitive status and ability to understand treatment, seizure history, ROM limitations, and spasticity.

5. What specific parameters of NMES would be appropriate for the patient?
ANSWER: Pulse duration: 200–800 μsec
Frequency: 50+ pps
Amplitude: To obtain a strong contraction as tolerated
Ramp time: 1–2 seconds
Duration: Start low to get him used to the stimulation (5 sec on, 25 sec off) and work up to 10 seconds on, 30 seconds off. Perform at least 10 contractions, 3 days per week.

CASE STUDY 14-1 NMES to Improve Strength—cont'd

6. What are the proper steps to using NMES for this patient?

ANSWER:

Electrode application: Two electrodes, one proximally and laterally over the vastus lateralis and rectus femoris and one distally and medially over the vastus medialis and the rectus femoris (Fig. 14-1).

Patient position: The exercise can be done a few different ways and may require some creativity on the physical therapist's part. The child can work with the stimulation, or you can have the child try to relax and let the stimulation turn on the muscle.

- The child can sit in a comfortable chair with the leg moving against gravity. Weights can be added if more effect is needed.
- The child can sit on a computerized dynamometer with the knee fixed at 60° of flexion. An isometric contraction can then be performed.
- The activity can be turned into a functional activity (as FES) by having the child do step-ups, squats, or other closed-chain quadriceps exercises. The physical therapist can trigger the FES through a remote switch to time the stimulation with the activity.

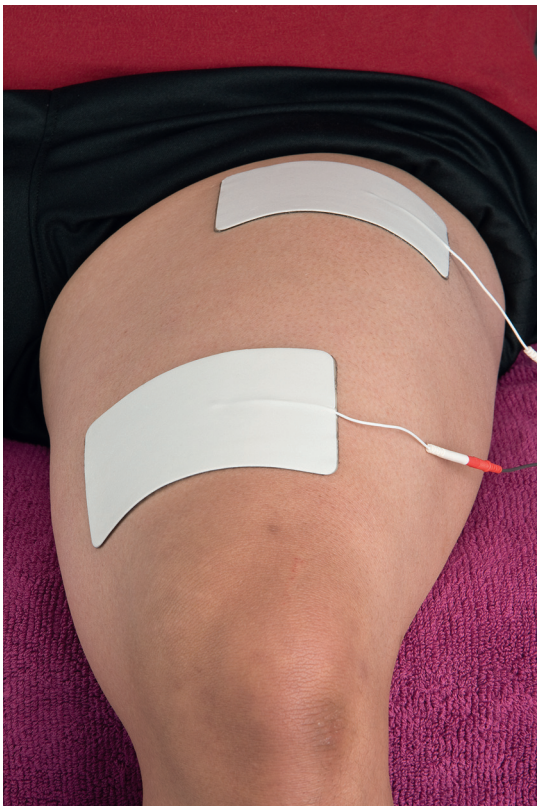

Fig 14 ■ 1 Electrode placement for stimulation of the quadriceps femoris muscle.

by fatiguing the muscle or by providing recurrent inhibition via Renshaw cells.[36] Stimulating the motor units of the spastic agonist muscle excites the Renshaw cells, which then inhibit the same spastic motor units.[37] The third theory states that electrical stimulation that delivers only sensory stimuli (doesn't create a muscle contraction) leads to sensory habituation that then leads to a decrease in spasticity.[36]

Clinically, NMES has been used to address spasticity in many patients who have neurological conditions with or without voluntary movement. Table 14-7 reviews studies that address spasticity in different populations. Several problems exist with much of the literature. Few studies include a control or comparison group, and inconsistencies exist in regard to treatment duration, length of time of effect, and how spasticity is measured. In addition, studies using NMES or FES for other purposes may also report a decrease in spasticity following intervention.[8,38]

Examination, Evaluation, and Prognosis

Table 14-8 provides some important aspects of the patient examination when considering using NMES to decrease spasticity. Based upon the literature, the treatment needs to be provided on an ongoing basis to maintain the effect, and a longer treatment time may be necessary.

Intervention

Table 14-9 identifies parameters reported in the literature for decreasing spasticity by creating a muscle contraction with NMES. One important parameter when treating patients with spasticity is the ramp-up time. When stimulating the antagonist to the spastic muscle, a short ramp-up time may create a spastic response of the agonist due to the quick stretch. The practitioner should monitor the patient for this response and adjust the ramp-up time accordingly.

TABLE 14–7. Literature on NMES for Decreasing Spasticity

Authors	Population	Method	Results
Carty et al, 2013[153]	SCI	NMES to quadriceps and hamstrings	Improvements in Spinal Cord Assessment Tool for Spastic Reflexes (SCATS) scores and visual analog scale.
Kamper et al, 2006[10]	CP	NMES to wrist flexors and extensors	No change in spasticity.
King, 1996[154]	Stroke	NMES to wrist extensors: passive stretch and NMES groups	NMES group had greater decrease in spasticity of wrist flexors as measured by torque meter.
Miller et al, 2007[155]	MS	TENS to quadriceps muscles	Decreased spasticity after 2 weeks for group using TENS 8 hours/day but not for group using 1 hour/day.
Potisk et al, 1995[156]	Stroke	Sensory stimulation over sural nerve	Decreased plantar flexor spasticity as measured by an electrohydraulic brace.
Robinson et al, 19888[157]	SCI	NMES to quadriceps femoris	Decreased quadriceps spasticity immediately after treatment only.
Sahin et al, 2012[158]	Stroke	NMES to wrist extensors	Decreased spasticity via Modified Ashworth Scale.
Scheker et al, 1999[159]	CP	NMES to wrist and finger extensors while wearing a wrist splint	Decreased wrist flexor spasticity as measured by a compliance scale. Increased upper extremity function.
Seib et al, 1994[160]	SCI, TBI	NMES to anterior tibialis: sham and treatment groups	Treatment group had subjective decrease in plantar flexor spasticity lasting 24 hours after treatment.
Wang et al, 1998[36]	Stroke	Sensory stimulation to T12 and L1 paravertebral areas to decrease plantar flexor spasticity	Decreased passive resistance of plantar flexors as measured by dynamometer.
Weingarden et al. 1998[33]	Stroke, TBI	NMES to wrist and finger extensors	Decreased wrist flexor spasticity.
Yıldızgören et al, 2014[12]	CP	NMES to wrist extensors	Decreased spasticity and increased function.

TABLE 14–8. Examination and Rationale for NMES for Spasticity Management

Tests of:	Question	Reason
Strength	Is voluntary movement present in agonist or antagonist?	Potential for strength improvement using NMES and for decreased antagonist spasticity to allow weak agonist to move.
Range of motion	Is a fixed contracture present?	A decrease in spasticity may have limited results if significant ROM deficits are present.
Sensation	Is sensation present, absent, or diminished?	More frequent monitoring needed with insensate skin.
Pain	Is pain present? How severe?	To determine effect of decreased spasticity on pain.
Spasticity	How significant is the spasticity?	To determine effect of intervention.
Function	Does spasticity affect functional abilities?	To determine effect of treatment on function.
Cognitive status	Is cognitive status sufficient to provide feedback?	Safety of use.
Caregiver assistance	Does patient require caregiver assistance to use NMES at home?	Determine availability of assistance.
Other treatments	Are antispasticity medications or other treatments being used or modified?	May impact ability to assess effects of intervention.

TABLE 14–9. Parameters Typically Seen for Decreasing Muscle Spasticity Using NMES[10,33,36,153–160]

Waveform	Symmetrical or asymmetrical biphasic, burst-modulated AC (i.e., Russian current)
Pulse duration	250–500 μsec
Frequency	20–100 pps
Amplitude	To obtain a contraction (at least a grade 3-/5)
Ramp-up time	0.5–3 seconds
Ramp-down time	0–3 seconds
Duty cycle	Variable (1:1, 3:4, 10:7) but typically larger ratio than for muscle strengthening
Treatment time and duration	10–60 minutes per day except one study treated for 8 hours. Treatment typically needs to continue for effect to remain, unless recovery of movement is occurring.

Studies utilizing sensory-level electrical stimulation use similar parameters except that the amplitude is kept low to avoid a muscle contraction. Another difference is that the stimulation typically is delivered continuously for the treatment time rather than using a duty cycle. A higher frequency (up to 100 pps) is often used.

As with other NMES applications, electrode size must be relative to the muscle size. A small, portable stimulator may be sufficient for using NMES to reduce spasticity because the goal is not to maximize force production. A portable stimulator also offers the advantage of being easily used at home after patient/caregiver training has occurred (Box 14-5).

NMES for Decreasing Urinary Incontinence

NMES has mainly been used to decrease incontinence for people without neurological conditions; however, some studies have addressed its use for patients with neurological conditions. An RCT with subjects with MS showed a greater reduction in incontinence episodes when intravaginal NMES was added to a program of pelvic floor training and biofeedback (85% reduction compared to 47% reduction with sham NMES).[39] A study with children with myelomeningocele showed

Box 14 ■ 5 Critical Considerations for Using NMES to Decrease Spasticity

- There is some evidence to support the use of NMES, but more research is needed.
- A wide range of stimulation parameters are reported, making it difficult to determine optimal parameters and dosing.
- NMES can be provided to the agonist or antagonist muscle.

CASE STUDY 14-2 NMES for Increasing Tissue Extensibility and Decreasing Spasticity

A 71-year-old woman presents with complaints of tightness and spasticity in her left hand and wrist and the inability to use the hand functionally. She holds her hand and wrist in flexion. Her past medical history reveals a right cerebral vascular accident 1 month prior, hypertension, and coronary artery disease. She currently lives with her daughter. Her goals are to become independent with several activities of daily living (ADLs).

CLINICAL DECISION-MAKING

1. Does the patient have dysfunction or impairment that may be improved or lessened by the use of NMES?
ANSWER: Yes, she has decreased ROM, spasticity, and likely has decreased strength.

2. Why would NMES be a consideration for this patient?
ANSWER: NMES may allow her to decrease her spasticity and increase independence with ADLs. Your immediate goals can be to increase ROM and decrease spasticity. If successful, later goals can focus on strength and functional outcomes. A functional goal could be for her to hold a fork in that hand or to use that hand as an assist for bimanual activities.

3. What specific outcome measures can be used to assess the effectiveness of NMES?
ANSWER: Modified Ashworth Scale, Functional Independence Measure, Wolf Motor Function test, 9-hole peg test, Arm Motor Ability Test, ROM

Continued

CASE STUDY 14-2 NMES for Increasing Tissue Extensibility and Decreasing Spasticity—cont'd

4. Are there any precautions or contraindications to consider with this patient?

ANSWER: This patient may have decreased cognition and sensory awareness that may impact the safety of using NMES. Her PROM needs to be assessed. If her contracture is significant (little motion), the issue may involve more than just soft tissue, and NMES would not be appropriate. However, because her stroke was 1 month ago, she likely has primarily a soft tissue limitation.

5. What specific parameters are recommended for this patient?

ANSWER:

Pulse duration: 200–300 μsec
Frequency: 20–30 pps
Amplitude: To obtain a 3+ contraction
Ramp-up time: Up to 3 seconds (watch for spastic response)
On-/off-time: Start with 10:30 and change to 10:10 as endurance improves
Duration: 15-minute sessions to start, working up to several hours

6. What are the proper steps to using NMES for this patient?

ANSWER:

Electrode application: Place electrodes on the wrist extensor muscles to provide a stretch to the wrist flexors (Fig. 14-2). Electrodes should be placed in a bipolar arrangement using a single channel to the wrist and finger extensor muscles on the forearm. The best response will allow her wrist and fingers to move through their full available PROM. Spasticity reduction will be targeted based on the principle of reciprocal inhibition using this approach. If your clinic has access to a NESS H200 (see the "Functional Electrical Stimulation" section), this device may be assessed with this patient to also meet her goals. The device would then allow the advancement to functional use when sufficient PROM is gained.

Patient position: With her seated comfortably with good posture, place her forearm on a towel or other soft surface on a table. Position her forearm in neutral pronation/supination to allow the wrist to extend in a gravity-eliminated position. If her hand quickly returns to a fully flexed position after each muscle contraction, place a soft roll in her hand to maintain a better position.

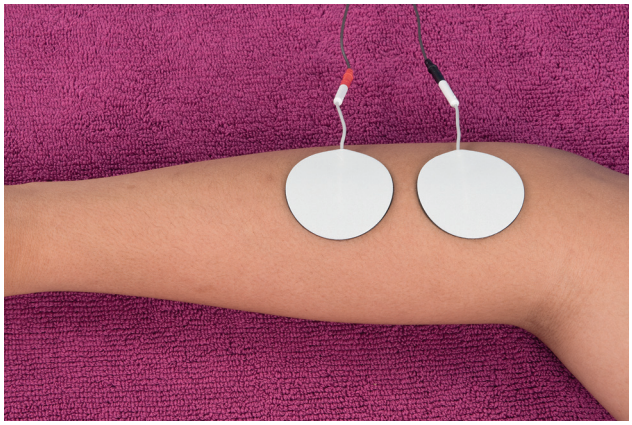

Fig 14 ■ 2 Electrode placement for stimulation of the wrist extensors.

decreased episodes of incontinence following transcutaneous stimulation compared to sham stimulation.[40]

Implanted sacral nerve stimulators have been approved by the FDA for patients with urinary retention or with urge incontinence who have failed all conservative therapies. The device stimulates the third or fourth sacral nerve root through a quadripolar electrode placed in the sacral foramen and an implanted stimulator placed in the upper buttock[41] (Fig. 14-3). Reduction in symptoms has been sufficient for Medicare to provide coverage for the device beginning June 2001.[42,43] In addition to reducing symptoms for patients without neurological conditions, success has been shown with patients with multiple sclerosis,[42,44] those with Parkinson's disease, spinal cord injury, and cerebral palsy and those who have had a stroke.[44]

Examination, Evaluation, and Prognosis

Precautions and contraindications for NMES for urinary incontinence include decreased sensation, pregnancy or plan to become pregnant, recurrent vaginal or urinary infections, vaginal lesions or fistulas, anal fissures, prolapsed uterus, atrophic vaginitis, recent pelvic surgery,

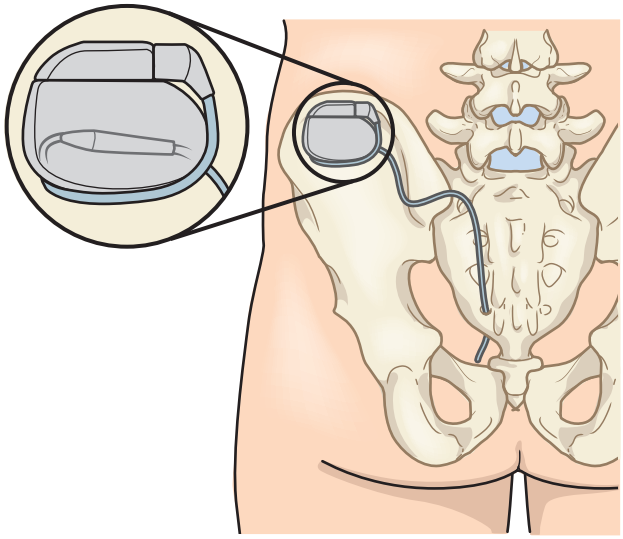

Fig 14 ■ 3 An electrode is surgically passed through the skin and placed immediately adjacent to the S3 nerve root within the sacral foramen. The implanted stimulator is placed subcutaneously in the buttock. This completely implanted system allows a signal to be delivered to the S3 nerve root to control incontinence.

pelvic irradiation, and neurological conditions.[45,46] Examination may include an assessment of strength of the pelvic floor muscles using an intravaginal probe that measures intravaginal pressure. A patient diary is recommended before and after treatment to document changes in incontinence (Box 14-6).

Box 14 ■ 6 NMES for Urinary Incontinence With Nonneurological Conditions

- Typically for stress incontinence (leakage with cough/exertion) or urge incontinence (sudden strong urge to empty bladder due to abnormal bladder contractions).[108]
- Recommended for patients who fail other more conservative treatments, including medication and pelvic floor exercise.[137,138]
- Common cause is damage to the type II fibers of pelvic floor muscles and possible partial denervation of the pudendal nerve during childbirth.[139]
- NMES can activate type II fibers to increase intravaginal pressures.[139]
- NMES mechanisms:
  - Facilitation of urine storage through reflex inhibition of the overactive detrusor muscle[138]
  - Increased urethral pressure through pudendal efferents to the striated urethral muscles[138]
- Electrodes are placed intravaginally, intrarectally, or on the skin surface.[44–46,140–142]
- Some studies show decreases in the episodes of incontinence,[40,44,51,137,140] but other studies report no effect.[140,143,144]
- Medicare approved for those who fail conservative treatment.[137]

Intervention

Intervention strategies have varied in terms of electrodes, waveforms, and parameters used. Table 14-10 provides the range of parameters typically used. The frequency is important based on different physiological mechanisms. Lower frequencies are used to create inhibition (primarily for urge incontinence), and the higher frequencies are used to activate motor units (primarily for stress incontinence). However, one study showed no differences between the waveforms in terms of outcomes for women with stress incontinence.[47] Typically a vaginal electrode is used for women (Fig. 14-4) and a rectal electrode for men. Studies have also used surface electrodes applied to the anus or pubic symphysis[48,49] or to the posterior

TABLE 14–10. Parameters Used for Decreasing Incontinence[33-35,37,39-41,115]

Waveform	Symmetrical or asymmetrical biphasic, amplitude-modulated AC (i.e., interferential current)
Pulse duration	250–1,000 μsec
Frequency	Urge incontinence: 5–20 pps (12 pps common) Stress incontinence: 20–50 pps (50 pps common)
Amplitude	To maximum tolerable levels (max 100 mA)
Ramp-up time	2 seconds
Ramp-down time	2 seconds
Duty cycle	1:3, 1:2, 1:1 (typically 5 sec on-time)
Treatment time and duration	15–30 minutes, 1–3 times/day, 4–12 weeks

Fig 14 ■ 4 Examples of incontinence electrodes. A vaginal electrode is typically inserted to stimulate the pelvic floor muscles to decrease incontinence. This figure shows a stimulator and two types of vaginal electrodes. *(Courtesy of Utah Medical Products, Inc., Midvale, UT.)*

tibial nerve.[50] Several companies manufacture stimulators specific for treating urinary incontinence, including the typically used parameters and vaginal/rectal electrodes. An RCT showed no differences in outcomes for women who used intravaginal stimulation vs. surface electrodes (suprapubic and ischial tuberosity).[51]

FUNCTIONAL ELECTRICAL STIMULATION

FES is traditionally considered the use of NMES within a functional activity. There are many activities in which FES can be incorporated, and the practitioner can be creative in how it is applied. There are some more common uses of FES, such as reducing shoulder subluxation, improving hand function, decreasing foot drop during gait, and exercise to maintain joint mobility, cardiovascular function, and lean mass, among other things. As mentioned earlier, patients with neurological conditions and spasticity usually require a longer ramp-up time when stimulating the antagonist muscle to avoid a quick stretch of the spastic muscle. However, with FES applications, ramp time needs to be considered in relation to the length of the contraction required for the activity. For example, dorsiflexion to clear the foot occurs quickly during gait. If using electrical stimulation to create dorsiflexion while walking, a long ramp time would be inappropriate and may delay the onset of the contraction or extend the length of the contraction beyond the needs of the activity (the muscle would be on too long).

Key Point! When using FES, it is recommended that the functional activity be meaningful to the patient and that the activity should have measurable goals so as to assess progress. (See "Suggested Outcome Measures" at the end of the chapter.)

FES for Shoulder Subluxation

Subluxation of the shoulder is a common problem after an acute stroke, occurring in up to 80% of patients within the first few weeks following stroke. This problem results from the lack of musculature support around the flaccid shoulder girdle, creating stress on the supporting ligaments and joint capsule as gravity creates a traction-type force. As a result, the humerus subluxes inferiorly[52] (Fig. 14-5). Subluxation is usually painful and can limit

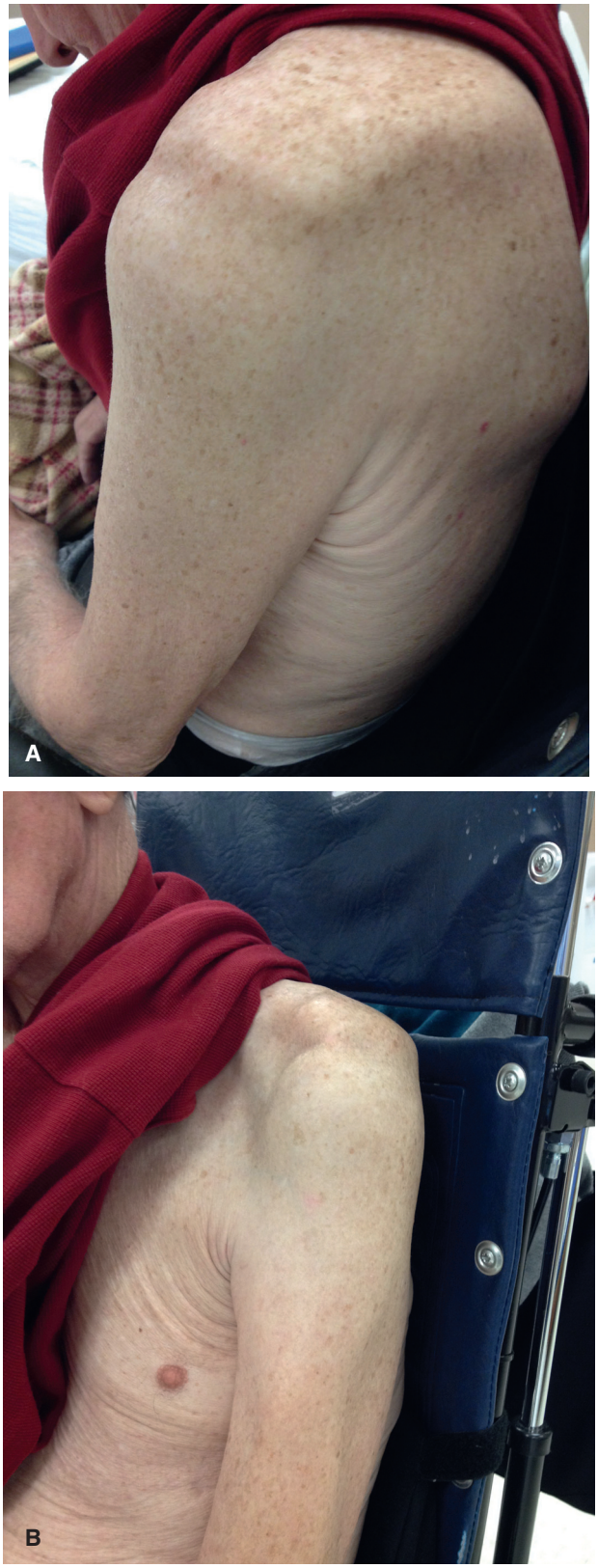

Fig 14 ■ 5 This patient, who sustained a stroke, presents with a resulting shoulder subluxation shown from (A) posterior-lateral and (B) anterior-lateral views.

the patient's ability to participate in rehabilitation of the upper extremity. It is typically treated with a supportive sling. However, the use of a sling does not address the flaccid muscles that caused the subluxation and can interfere with rehabilitation of the rest of the upper extremity.

FES using electrodes on the skin can decrease shoulder subluxation by stimulating muscles around the shoulder girdle, most commonly to the posterior deltoid and supraspinatus muscles. This orthotic assist can help to provide more normal glenohumeral alignment and create stability for distal movement during rehabilitation.[53]

Several studies, with a meta-analysis,[54] have reported on the benefits of FES for shoulder subluxation; findings indicated that FES was successful for subluxation in the acute stage following stroke but not the chronic stage (Table 14-11). Across the studies reviewed, a subluxation on average of 6.5 mm was reduced by an average of 1.9 mm with the use of FES as compared to conventional treatment alone. More recent studies have examined the use of combined approaches with NMES in conjunction with positioning,[55] Bobath,[56] or conventional therapy[57] with two studies reporting better gains with a combined approach[56,57] and one not showing change[55] in subluxation and other measures.

An alternative method for providing FES for shoulder subluxation is the use of percutaneous electrodes as opposed to electrodes applied to the skin surface. Percutaneous electrodes are thin wires implanted near the motor point of a muscle. The electrode exits the skin to allow connection of the cables that attach to a stimulator (Fig. 14-6). Some potential advantages of this type of electrode are decreased discomfort, ease of use because electrodes do not need to be reapplied for each treatment session, and repeatability of muscle responses.[58] Similar outcomes have been reported with the use of percutaneous electrodes as compared to electrodes applied to the skin surface.[34,59] Yu et al[59] reported that nine out of 10 patients perceived less discomfort with percutaneous electrodes.

Examination, Evaluation, and Prognosis

When determining if a patient is suitable for receiving FES for shoulder subluxation, the examination should include tests of upper-extremity strength, ROM, spasticity, the degree of shoulder subluxation, and function (Table 14-12). Ratings of pain should be obtained, and medical history should be reviewed to determine time since stroke and any medical contraindications for use of FES.

TABLE 14–11. Studies Using FES for Decreasing Shoulder Subluxation

Authors	Population	Method	Results
de Jong et al, 2013[55]	Acute stroke	FES + positioning for 8 weeks	No change in primary measures of PROM or pain
Faghri et al, 1994[52]	Acute stroke	FES for 6 weeks vs. conventional physical therapy	Decreased shoulder subluxation, spasticity, and pain.
Fil et al, 2011[56]	Acute stroke	FES + Bobath vs. Bobath alone for acute admission (about 12 days)	No subject in FES + Bobath group developed subluxation while 19% in Bobath-only group developed subluxation.
Koyuncu et al, 2010[57]	Acute stroke	FES/conventional therapy vs. therapy alone (4 weeks, 20 sessions total)	FES group had decreased subluxation.
Linn et al, 1999[161]	Acute stroke	FES vs. conventional physical therapy	Decreased shoulder subluxation and pain after 4 weeks. No difference between groups after 12 weeks.
Chantraine et al, 1999[162]	Acute stroke	FES vs. conventional physical therapy	Increased motor scores and decreased pain and subluxation in the FES group after 5 weeks.
Chae et al, 2005[163]	Acute stroke	Percutaneous FES vs. sling (6-week program)	Greater decreases in pain than subjects treated with a sling. Effects lasted for 12 months.

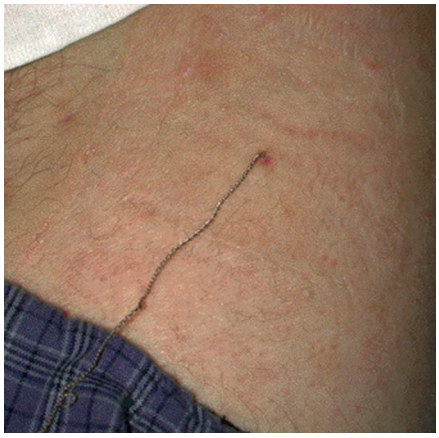

Fig 14 ■ 6 Percutaneous electrodes are implanted near the nerve or the motor point. In this picture, an electrode was implanted to the quadriceps femoris via the femoral nerve. To protect the electrode throughout the day, an occlusive bandage is placed over the electrode and connector block. Percutaneous electrodes can be tunneled underneath the skin to exit in any location. When more than one percutaneous electrode is implanted, they typically all exit in the same location.

Intervention

Electrodes are typically applied to the posterior deltoid and the supraspinatus muscles. The middle deltoid may be used if there is difficulty isolating the posterior deltoid. The practitioner may find a better response by placing a smaller electrode on the posterior deltoid to increase the current density and a larger one on the supraspinatus in order to create the feeling of the humerus being pulled superiorly without creating significant abduction of the upper extremity. Activation of the upper trapezius muscle should be avoided. Table 14-13 provides typical parameters used for FES for shoulder subluxation.

FES for Upper-Extremity Function

FES has been applied to improve hand and upper extremity function in patients following stroke, traumatic brain injury (TBI), CP, and SCI. Using commercial devices such as the NESS H200, FES has been more recently applied for hand function in patients who have had a stroke as well as for TBI. The NESS H200 is a forearm- and hand-molded orthosis with five electrodes to stimulate the extensor digitorum, the extensor pollicis brevis, the flexor digitorum superficialis, the flexor pollicis longus, and the thenar muscles. The wrist is held in approximately 10° to 20° of extension within the orthosis (Fig. 14-7). The H200 can provide both exercise and functional grasping abilities. Studies have reported effects on spasticity,[33,60] ROM,[33] motor function,[60,61] and functional abilities[61–63] with the use of the NESS H200 (and its earlier counterpart, the Handmaster) in patients following TBI and stroke. Two of these studies were RCTs[61,63] that compared traditional therapy and traditional therapy plus FES. Both studies included subjects with acute stroke, but Thrasher et al[61] also included those with chronic effects of stroke and reported that the effects were only significant when applied after an acute stroke.

TABLE 14–12. Examination and Rationale for FES for Shoulder Subluxation

Tests of:	Question	Reason
History	Onset of stroke?	Greatest effect seen with acute stroke
Strength	Is voluntary movement present in involved upper extremity?	Potential for improvement
Range of motion	What is upper extremity ROM?	Potential for improvement
Sensation	Is sensation present, absent, or diminished?	More frequent monitoring needed with insensate skin
Pain	Is pain present at rest or with activity? How severe?	To determine effect on pain
Spasticity	Is spasticity present?	Potential for improvement
Function	What are the functional limitations?	To determine effect of treatment on function
Pain	Is shoulder or upper extremity pain present?	Potential for improvement
Cognitive status	Is cognitive status sufficient to provide feedback?	Safety of use
Caregiver assistance	Does patient require caregiver assistance to use NMES at home?	Determine availability of assistance

TABLE 14–13. Parameters Typically Seen for FES for Shoulder Subluxation[52,56,57,161–164]

Waveform	Symmetrical or asymmetrical biphasic, or burst-modulated AC (i.e. Russian current)
Pulse duration	200–350 μsec
Frequency	30–60 pps
Amplitude	To achieve effect without abduction or shoulder elevation
Ramp-up time	3 seconds
Ramp-down time	3 seconds
Duty cycle	Range 1:1 to 15:1. Typically start with a 1:5, 1:3, or 1:1 but goal is to quickly increase on-time and decrease off-time as muscle endurance improves. On-times up to 30 seconds can be used if tolerated by muscles.
Treatment time	20 minutes to 6 hours (start low and increase as duration endurance improves), 5–7 days per week, for 4–8 weeks

Fig 14 ■ 7 Patient following a CVA using the NESS H200 for upper extremity functional activity. *(Courtesy © 2009 Bioness, Inc., Valencia, CA.)*

Other studies have used different devices to apply FES for hand function, using a variety of techniques, including EMG control[7,8,64,65] or intensity control using the other hand,[6] bilateral hand and other functional activities,[66,67] percutaneous electrodes,[64] and robotics.[68] These studies have reported similar effects on decreasing spasticity, increasing motor ability, and increasing function. However, one study that provided the intervention

CASE STUDY 14-3 FES for Decreasing Shoulder Subluxation

A 65-year-old man sustained a left cerebrovascular accident (CVA) 2 weeks ago. He has been complaining of right shoulder pain in the past week and is hesitant about attempting to use the right upper extremity for function. A radiograph showed the presence of a 7-mm inferior subluxation of the humerus from the glenoid fossa. He has been wearing a sling during the past week. His medical history reveals hypertension. His goal is to decrease the pain.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction or impairment that may be improved or lessened by the use of FES?
   ANSWER: Yes, FES can help to decrease the shoulder subluxation and pain.
2. Why would FES be a consideration for this patient?
   ANSWER: FES is appropriate for this patient due to subluxation and pain secondary to stroke. The goals are to decrease the degree of subluxation and reduce pain so that he can participate in functional upper-extremity training.
3. What specific outcome measures can be used to assess the effectiveness of FES?
   ANSWER: Visual Analog Scale, measurement of the subluxation (finger breadths), Arm Motor Ability Test, or other ADL/arm function tests, depending on deficits noted.
4. Are there any precautions or contraindications to consider with this patient?
   ANSWER: Special considerations include any possible cognitive and sensory deficits that may limit safety.
5. What specific parameters are recommended for this patient?
   ANSWER:
   Stimulator: Portable stimulator preferred so treatment can continue outside of therapy.

Continued

CASE STUDY 14-3 FES for Decreasing Shoulder Subluxation—cont'd

Pulse duration: 200–350 μsec
Frequency: 30–40 pps
Amplitude: Enough to cause visible reduction in subluxation (elevation of head of humerus). If shoulder abduction is seen, amplitude is too high.
Ramp time: 3 seconds
Treatment: You decide to start with on-times of 10 seconds and off-times of 30 seconds, providing treatment for 30 minutes, three times per day. If tolerated, increase on-times and total treatment time with goals of reaching up to 30-second on-times and 2-second off-times for up to 6 hours. Progression should be made as quickly as possible.

6. What are the proper steps to using FES for this patient?

ANSWER:

Electrode application: Two electrodes, one over the posterior deltoid and one over the supraspinatus (Fig. 14-8). Goal is to move the humerus superiorly into the glenoid fossa without creating abduction. If necessary, the middle deltoid can also be tried instead of the posterior deltoid.
Patient position: To start, the patient's arm needs to be supported on a table or with a sling. As subluxation and pain reduce, the support may be removed.
Treatment monitoring: Monitor his response to the treatment by observing the decrease in subluxation and asking about pain.

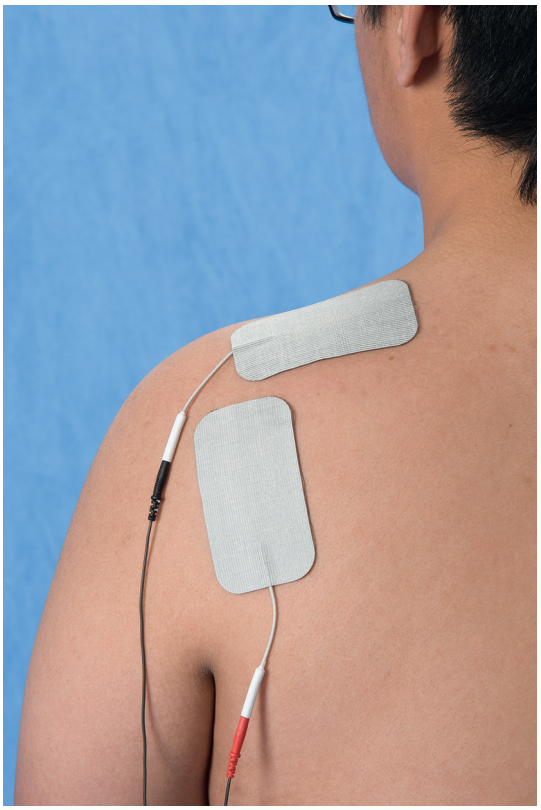

Fig 14 ■ 8 Electrodes to reduce a shoulder subluxation are typically placed over the posterior deltoid and the supraspinatus. The exact location to optimize the response will vary between patients. The goal is to move the humerus superiorly into the glenoid fossa without creating abduction.

for only 4 weeks concluded that more sessions would be needed to have a significant effect compared to a control group.[69] Knutson et al[67] reported improved Fugl-Meyer scores and Wolf Motor Function Test scores after 12 weeks in a sample of subjects.

More limited research has been done on FES for upper-extremity function in children with CP. Several case studies have looked at FES for hand function. Carmick[70] applied FES for finger and wrist extension and finger flexion in a child with hemiplegic CP. The child initially wore a splint to control wrist position while using FES. The therapist or the parents triggered the FES to create flexion or extension while the child attempted to use the hand functionally. After 9 months of treatment, the child was able to perform activities such as tying his shoes without the splint or FES. In another report, Carmick[71] indicated increased awareness of the involved upper extremity after treatment with FES in one child with hemiplegic CP and increased bilateral hand use in another child, also with hemiplegic CP. Although these are all case studies, they do suggest that FES can impact functional hand use in these populations.

More research effort has been directed toward providing hand function for patients with complete tetraplegia at the fifth and sixth cervical levels. Studies have reported improved function with the use of the Handmaster[69] and with implanted FES systems.[72,73] Numerous studies of implanted FES systems have reported increased functional independence using a device (the Freehand) that stimulates eight upper-extremity muscles and provides a control source that allows the

user to activate and control the grasp and release patterns. The Freehand, which was clinically available, is no longer on the market.

Examination, Evaluation, and Prognosis

In considering the use of FES for hand function, important tests include those of strength, ROM, sensation, function, and spasticity. The diagnosis and presence of voluntary movement will help the practitioner decide if functional benefits are expected only with the stimulation turned on or if functional benefits with it off are anticipated due to muscle reeducation. For example, a patient following stroke may gain enough muscle strength and motor control to be able to function without the FES. However, another patient may not make these same gains and thus want to use the device throughout the day to provide the ability to grasp. Another important consideration is that if FES is to be used for the hand or forearm only, the patient needs to have sufficient shoulder and elbow strength and ROM to operate the device functionally.

Intervention

In a clinical environment, FES for the upper extremity can be applied using a surface stimulator. If the practitioner wants to control the stimulation during functional activities, remote switches are available for some stimulators. The practitioner then needs to trigger the FES when appropriate for the activity. For example, grasp can be triggered during a functional reaching activity and then turned off when the patient is ready to release the item. It is important that the practitioner be creative in selecting activities that may be aided by FES in an attempt to provide a new functional movement or to enhance one already present (Box 14-7). Table 14-14 identifies typically used stimulation parameters.

FES for Ambulation

Much focus with FES has centered on ambulation—either in enhancing it for those with some voluntary muscle movement or in creating it for those with no voluntary lower extremity movement. It is common for practitioners to use FES as a dorsiflexion assist for patients who present with decreased foot clearance (foot drop) during the swing phase of gate (Fig. 14-9). This technique has been applied to patients with stroke, TBI, CP, and incomplete SCI but is not limited to these populations (Table 14-15).

Box 14 ■ 7 Key Points for Using FES to Increase Upper Extremity Function

- Patients with an acute stroke can increase their functional abilities following combined programs of traditional therapy and FES. The intervention may need to last longer than 4 weeks for an effect.
- FES can be delivered through commercially available systems specifically designed for the forearm/hand or other types of stimulators.
- More research is needed on FES for upper-extremity function in children with CP.
- Patients with tetraplegia at C5 or C6 levels can improve hand function with an implanted FES system.

TABLE 14–14. Parameters Typically Seen for Hand Function[8,12,33,60–64,67,69,71–73,165]

Waveform	Symmetrical or asymmetrical biphasic
Pulse duration	200–350 μsec
Frequency	12–50 pps
Amplitude	To achieve desired movement for function. Keep as low as is feasible.
Ramp-up time	Very short to achieve effect
Ramp-down time	Very short to achieve effect
Duty cycle	N/A; stimulation is timed with demand of functional activity
Treatment time	30–45 minutes once or twice per day, 3–6 times per week, for 6–16 weeks

Key Point! The most common clinical application of FES for ambulation involves using it as a dorsiflexion assist for decreased foot clearance during swing. FES applied in this manner may replace the use of an ankle foot orthosis, and functional ambulation has been shown to be comparable with an ankle foot orthosis versus FES for function following stroke.[74–76] However, patients more often prefer FES.[77]

Fig 14 ■ 9 A child with hemiplegic cerebral palsy using FES to assist with clearance during the swing phase of gait.

Timing of the stimulation is controlled with a remote switch or a foot switch placed in the shoe, typically underneath the patient's heel. When pressure is removed from the foot switch, as in the initiation of swing, stimulation will begin. It will end when pressure is again placed onto the foot switch. If a patient does not achieve heel contact during gait, alternate positions for the foot switch can be investigated. Other means for triggering FES for foot drop are incorporated into devices made specifically for this application. Two systems are currently on the market in the United States: the WalkAide and the Bioness NESS L300. Both systems involve placing a cuff containing the electrodes around the upper calf to deliver the stimulation (Fig. 14-10). The WalkAide uses a tilt sensor that detects the leg's position in order to trigger the FES. The NESS L300 uses a sensor mounted on the shoe and computer-based algorithms to control the timing of the simulation (Fig. 14-11). For both devices, the practitioner determines the specific needs of each patient during the initial training phase. The stimulation levels provided by the WalkAide are 50 to 250 μsec, 16.7 to 33 pps, and up to 200 mA. Those

TABLE 14–15. Key Studies That Examine FES During Walking

Authors	Population	Method	Results
Bethoux et al, 2014[76]	Stroke	Compared AFO to FES for foot drop	Increased 10-meter walk test scores (both groups). No difference between groups.
Daly et al, 2011[110]	Stroke	FES to many muscles with BWSTT vs. BWSTT alone	Improved gait coordination (both groups), maintained in FES group after 6 months.
Dunning et al, 2009[82]	Stroke	Two cases with FES for foot drop	One gained in TUG and both gained in 6 MWT scores.
Embrey et al, 2010[168]	Stroke	FES for dorsiflexion and plantar flexion	Gains in 6 MWT, Emory Functional Ambulatory Profile, Stroke Impact Scale.
Kesar et al, 2011[114]	Stroke	FES for dorsiflexion and plantar flexion on TM at fast vs. self-selected speed	Fast group had greater gains in ground reaction forces, trailing limb position, and knee flexion in swing.
Kluding et al, 2013[75]	Stroke	Compared AFO to FES for foot drop	Improvements in gait speed and other functional outcomes in both groups with no differences between groups. FES group had higher satisfaction.
Laufer et al, 2009[83]	Stroke	FES for foot drop	Patients continued to make gains in gait speed 1 year later with the FES on and off.
Mun et al, 2014[86]	Stroke	FES for foot drop with and without FES to gluteus medius or quadriceps	Greater gains in gait speed and step length with the added muscles.

TABLE 14–15. Key Studies That Examine FES During Walking—cont'd

Authors	Population	Method	Results
Paul et al, 2008[88]	MS	FES for foot drop versus walking without FES	Increased gait speed and decreased physiological cost of walking with FES.
Carmick, 1995[91]	CP	FES to plantar flexors	Improved gait, balance, posture, AROM, PROM, foot alignment.
Damiano et al, 2013[93]	CP	FES for foot drop	Increased muscle size, increased dorsiflexion in swing with FES on compared to off.
Orlin et al, 2005[92]	CP	FES for dorsiflexion and plantar flexors flexion	Increased dorsiflexion in swing with FES on compared to off.
Bajd et al, 1999[96]	Incomplete SCI	Peroneal FES for flexors withdrawal and FES for plantar flexion	Improvements in swing phase. Added FES to plantar flexors increased gait speed.
Field-Fote and Roach, 2011[109]	Incomplete SCI	Peroneal FES for flexors withdrawal plus over-ground walking or BWSTT*	Increased walking speed for both, walking distance for over-ground training.
Granat et al, 1993[97]	Incomplete SCI	FES to many muscles	Decreased quadriceps tone and physiological cost of gait, increased strength and stride length.
Hitzig et al, 2013[111]	Incomplete SCI	FES for knee extension/flexion and dorsiflexion and plantar flexion with BWSTT vs. non-FES aerobic/resistance training	Increased mobility scores for FES group (Spinal Cord Independence Measure). No other differences between groups.
Johnston et al, 2003[98]	Incomplete SCI	FES to many muscles	Improved gait speed, kinematics, energy cost of walking.
Postans et al, 2004[112]	Incomplete SCI	FES to many muscles with BWSTT	Increased walking speed and endurance.
Sharif et al, 2014[113]	Incomplete SCI	FES to many muscles with BWS	Increased 6 MWT and walking scores (WISCI). Improved quality of life (SF-36).

*This group had other groups not using FES. Refer to full article for details.

AFO = ankle foot orthoses; BWSTT = body weight supported treadmill training; MWT = minute walk test; TM = treadmill; TUG = times up and go; WISCI = walking index for spinal cord injury.

for the NESS L300 are 200 μsec, 30 pps, and approximately 30 to 35 mA. In January 2009, Medicare approved coverage for the WalkAide and the NESS L300 for people with incomplete SCI.

Stroke and Multiple Sclerosis

Early studies showed effects of FES for foot drop following stroke with improvements reported in dorsiflexion during swing,[74,78] spontaneous recovery,[79] recovery in muscle force three times greater than controls,[80] walking speed,[81] the Timed Up and Go,[82] gait symmetry,[82] and a 6-meter walk test.[82] Improvements have also been reported in function, social integration, and walking speed for subjects following stroke who had used the NESS L300 for 1 year.[83] Other studies have examined the outcomes with implanted electrodes for FES for foot drop. These have the advantage of being in a consistent place, so the muscle response should be more consistent. Reported findings include increased walking speed[81,84,85] and distance.[81] Another study[86] added FES to the quadriceps or the gluteus medius along with the anterior tibialis and reported greater gains in gait speed and step length compared to just using FES to the anterior tibialis. FES to the quadriceps or hamstrings can be added using a second cuff with the NESS L300. Improvements in walking speed and energy expenditure have been seen with FES applied to the peroneal nerve in patients with MS or following stroke.[87] However, another study that involved subjects with MS found increased walking

Fig 14 ■ 10 This figure shows, from left to right, the sensor that detects pressure increasing and decreasing beneath the heel, the cuff that contains the electrodes that is wrapped around the patient's proximal tibia/fibula to stimulate dorsiflexion during swing, and the FES controller. *(Courtesy © 2009 Bioness, Inc., Valencia, CA.)*

Fig 14 ■ 11 Patient using the NESS L300 following a CVA. *(Courtesy © 2009 Bioness, Inc., Valencia, CA.)*

speed and decreased physiological cost of walking when subjects walked with or without FES (which was applied for foot drop).[88]

Cerebral Palsy

Outcomes for children with CP have included increases in ROM and improvements during ambulation with the FES turned off.[89] Other studies have reported improved ambulation,[90,91] foot clearance,[92,93] range of motion,[94] motor control,[94] strength,[94] and function[90,91] with FES. Some of these studies studied the effect of adding FES to the gastrocnemius muscles as well.[90,92] Another study applied FES to the gastrocnemius during gait and reported an increase in force production during walking.[95] More recent work[93] has used the WalkAide and found that gait speed was similar with and without the FES on but that the size of the anterior tibialis muscle increased.

Spinal Cord Injury

FES for walking in those with SCI has received considerable focus. FES for dorsiflexion assist alone or in combination with FES to other muscles has augmented swing, increased walking speed, and increased strength for patients with incomplete SCI.[96–98]

Much focus has also been directed toward providing ambulation for patients with complete thoracic SCI using surface or percutaneous electrodes or implanted systems.[98–103] The Parastep is an FDA-approved device for ambulation that stimulates the peroneal nerve to create a flexor withdrawal response for stepping and stimulates the gluteal and quadriceps muscles for stance. It is controlled through push buttons on a walker (Fig. 14-12). The Parastep has been reported to improve lower-extremity blood flow,[104] cardiovascular status,[105] muscle size,[106] and lean tissue[106]; however, it had no effect on bone density.[107] In July 2002, Medicare approved the use of FES for standing and walking for individuals with SCI.[108]

FES Gait Combined With Treadmill Training

The combination of FES for gait and treadmill training has received more attention for people poststroke or SCI, and much of the recent research is in this area. These studies range from stimulating for dorsiflexion and stepping only[109] or for multiple muscles.[110–114] Studies are showing gains in walking speed on and off the treadmill,[109,112,114] lower-extremity motor scores (SCI),[109] quality of life,[111,113] walking measures (Walking Index for SCI,[113] 6-minute walk test[110,113]), function (SCI Independence Measures), ground reaction forces,[114] gait coordination,[110] and participation.[111]

Fig 14 ■ 12 A woman with a spinal cord injury uses the Parastep system to ambulate down a hallway at the Miami Project. The push buttons are located on the walker handles, enabling this woman to initiate steps as desired. *(Reprinted with the permission of Sigmedics, Inc.)*

Examination, Evaluation, and Prognosis

Clinically, the main application of FES for ambulation is to assist dorsiflexion. Because this technique is typically applied to those with neurological conditions, the practitioner must know if the targeted muscles are innervated. As with other applications, tests of strength, ROM, sensation, function, and spasticity are important. Walking speed is an additional test that may be helpful in evaluating results of the treatment and describing the gait pattern before treatment. The diagnosis and presence of voluntary movement will help the practitioner to decide if functional benefits are expected with the stimulation turned on or if functional benefits with it off are anticipated due to muscle reeducation.

Intervention

In applying FES for dorsiflexion assist, two electrodes are applied to create balanced dorsiflexion—meaning the foot is balanced between inversion and eversion. A small amount of eversion is acceptable. However, excessive inversion places the patient at risk of an inversion ankle sprain. Electrodes are typically applied to stimulate the anterior tibialis and the peroneal muscles. Some portable stimulators can be equipped with a remote switch that the therapist will control or a foot switch that will react to the patient's movement. A foot switch is placed into the shoe on the patient's affected side. When weight is removed from the switch to initiate swing, stimulation will be triggered. When weight is reapplied at initial contact, stimulation stops. This method is the ideal way to create a dorsiflexion assist in the clinic because the therapist can focus on the patient's response rather than on the timing with the remote switch. Again the practitioner should be creative in other ideas for enhancing ambulation using FES or using FES as a tool for muscle reeducation (Box 14-8). Table 14-16 identifies typically used stimulation parameters for dorsiflexion assist.

FES for Exercise

FES Cycling

FES has also been applied as a form of exercise, mainly for patients with SCI. One method for this is FES

Box 14 ■ 8 Key Points for Using FES to Increase Foot Clearance During Ambulation

- The patient's response to the device can be assessed using any portable stimulator that provides some type of trigger to time the FES to walking.
- Two devices (WalkAide and NESS L300) specific for foot drop are currently on the U.S. market. Each company can be contacted to try a device with a patient.
- Medicare has approved coverage for the WalkAide and NESS L300 for patients with incomplete SCI. Insurance coverage for other conditions varies and is dependent on the insurance company.
- The use of FES for foot drop can have a large impact on walking function.

TABLE 14–16. Parameters Typically Seen for Dorsiflexion Assist[83,87,89–91,96,97,166,167]

Waveform	Symmetrical or asymmetrical biphasic
Pulse duration	200–350 μsec
Frequency	30–40 pps
Amplitude	To achieve 3-/5 contraction
Ramp-up time	0–1 second
Ramp-down time	0–1 second
Duty cycle	N/A; stimulation is timed with demand of functional activity
Treatment time	Determined by muscle fatigue and duration

cycling. A specially designed bicycle that provides cyclical stimulation to the quadriceps, gluteal, and hamstring muscles is used. Treatment is typically provided for 30 to 60 minutes, three times per week. Studies of FES cycling have reported improvements in muscle volume and fiber size, fat-free tissue, bone density, cardiovascular fitness, and lower extremity circulation (Table 14-17). Numerous studies have examined the effects of FES cycling on the cardiovascular system of adults with SCI.[115] Studies involving cycling training 2 to 3 days per week for 12 to 16 weeks have shown increases in peak oxygen uptake,[116,117] cardiac output,[116,118] stroke volume,[118] and pulmonary ventilation.[116] Overall, these results indicate that FES cycling has a positive effect on individuals with SCI. People undergoing this exercise regimen were able to expend additional calories to improve overall fitness and showed a pronounced effect on cardiovascular health.

Another technique for FES cycling is to add volitional arm exercise at the same time. A study by Raymond et al[119] showed that subjects could obtain higher oxygen uptake and power output, which may lead to better cardiovascular benefits.

Although FES cycling has primarily been used by patients with complete SCI, this technique is now being

TABLE 14–17. Key Studies That Examine FES Cycling*

Authors	Population	Method	Results
Berry et al, 2008[169]	SCI	FES cycling	Increased maximal oxygen uptake during cycling
Bloomfield at al, 1996[170]	SCI	FES cycling	Increased bone density around knee if power > 18 watts
Chen et al, 2005[171]	SCI	FES cycling (quadriceps/hamstrings)	Increased bone density around knee but not spine
Chilibeck et al, 1999[172]	SCI	FES cycling	Increased muscle fiber size
Demchak et al, 2005[173]	SCI	FES cycling vs. no cycling acute SCI	Less muscle loss for FES cycling group
Dolbow et al, 2013[174]	SCI	FES cycling	Improved quality of life
Frotzler et al, 2008, 2009[175,176]	SCI	FES cycling	Increased bone density around knee. Not all lost when stopped.
Griffen et al, 2009[177]	SCI	FES cycling	No change in lipids
Lauer et al, 2011[178]; Johnston and Wainwright, 2011[127]; Johnston et al, 2011[179]	SCI	FES cycling vs. passive cycling vs. stimulated exercise (pediatric)	Improved bone density, oxygen uptake and muscle size in FES group. No change in lipids.
Lai et al, 2010[180]	SCI	FES cycling vs. no cycling acute SCI	Less bone loss for FES cycling group
Sadowsky et al, 2013[181]	SCI	FES cycling as part of activity-based program	Increased neurological and functional performance, increased muscle size/strength, decreased spasticity, and improved quality of life
Johnston and Wainwright, 2011[127]	CP	FES cycling (quadriceps, gluteals, gastrocnemius)	Improvements in strength, TUG, SF-36

TABLE 14–17. Key Studies That Examine FES Cycling*—cont'd

Authors	Population	Method	Results
Harrington et al, 2012[128]	CP	FES cycling (quadriceps only)	Increased power output and heart rate and decreased variability
Ralston et al, 2013[182]	Incomplete SCI	FES cycling	No effect on urine output, swelling, and spasticity
Ambrosini et al, 2012, 2011[183,184]	Stroke	FES cycling vs. non-FES cycling (added tibialis anterior)	Improved Motricity Index (MI), trunk control test, upright motor control test, gait speed, and muscle activation for those who received FES
Janssen et al, 2008[124]	Stroke	FES cycling	Increased maximal oxygen uptake and heart rate above resting while cycling
Lee et al, 2013[125]	Stroke	FES cycling	Improved 6 MWT, balance, Barthel and oxygen uptake
Lo et al, 2012[126]	Stroke	FES cycling (quadriceps and hamstrings)	Decreased spasticity
Fornusek et al 2014[122]	MS	FES cycling	Perceived benefits: improved transfer ability, circulation, spasticity, and strength
Ratchford et al, 2010[123]	MS	FES cycling	Improvements in 2-minute walk test, timed 25-foot walk, timed up and go

*Electrodes on quadriceps, hamstrings, and gluteal muscles unless otherwise stated.

used for patients following stroke and for those with MS, incomplete SCI, or cerebral palsy. In the past, these populations were typically excluded due to concerns with the inability to tolerate the sensation of FES. However, many patients can tolerate levels that are sufficient for making gains. Studies have shown a reduction in spasticity[120-122] improved cycling smoothness,[121] improved function,[122,123] strength,[122] and circulation[122] for people with MS. For patients who have had a stroke, a small RCT[124] with chronic stroke experienced gains in aerobic capacity in a 6-minute walk test and in the Berg Balance Scale after a 6-week program of FES cycling two times per week. Other studies have showed increased function and balance[125] and decreased spasticity.[126] Studies with people with CP have shown improved strength, quality of life, and function[127] as well as power output and cycling smoothness.[128]

FES cycling is usually done on a stationary cycle, either one that the user transfers onto or one that allows the user to stay in his or her wheelchair (Fig. 14-13). However, cycling with FES over ground is another option and is more widely used in Europe. Due to the power output required to cycle outdoors and muscle fatigue, cycling distance is limited. Nevertheless, one study added a motor to a cycle in order to provide power from the legs through FES and through the motor.[129]

Upper extremity cycling is another technique that has recently been used for people with tetraplegia. Using the RT300 FES cycle, an arm attachment can be added to

Fig 14 ■ 13 A young woman with a spinal cord injury using the RT300 FES cycle. *(Courtesy of Restorative Therapies, Inc., Baltimore, MD.)*

CASE STUDY 14-4 FES to Improve Foot Drop

A 16-year-old female with an acute C8 incomplete spinal cord injury is experiencing recovery in both lower extremities and has started gait training with a walker. However, recovery for her left ankle muscles is slower, and she is unable to clear her left foot while walking.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction or impairment that may be improved or lessened by the use of FES?
ANSWER: This patient may benefit from FES to achieve dorsiflexion during gait. Because her spinal cord injury was at C8, she should have an upper motor neuron lesion to her dorsiflexors. This can be tested by applying NMES and looking at the response.

2. Why would FES be a consideration for this patient?
ANSWER: FES would allow her own muscles to contract while she is walking, which may be of benefit as she is showing recovery. The immediate goal is to eliminate the foot drop so that she can obtain sufficient foot clearance during gait. The long-term goal is for her to recover enough to walk without the FES.

3. What specific outcome measures can be used to assess the effectiveness of FES?
ANSWER: 6-minute walk test, 10-m walk test, walking index for spinal cord injury

4. Are there any precautions or contraindications to consider with this patient?
ANSWER: Considerations for this patient include possible autonomic dysreflexia, decreased sensation (including proprioception), ROM of her ankle, and plantar flexor spasticity.

5. What specific parameters are recommended for this patient?
ANSWER: There are a couple of options for a stimulator. A portable stimulator with a foot switch can be used. The foot switch is placed in the shoe under the patient's heel. With the switch in place, the FES will be activated when pressure is removed from it during terminal stance/preswing. Another option is to use one of the commercially available devices designed specifically for foot drop (WalkAide or NESS L300).
Pulse duration: 200–350 μsec
Frequency: 30–40 pps
Amplitude: Enough to achieve sufficient dorsiflexion for foot clearance
Ramp time: None, because contraction needs to occur quickly.
Duration: Timed with gait. Monitor patient for fatigue of dorsiflexors.

6. What are the proper steps to using FES for this patient?
ANSWER: Because her spinal cord injury was at C8, she should have an upper motor neuron lesion to her dorsiflexors. This can be tested by applying NMES and looking at the response.
Electrode application: Two electrodes, one over the anterior tibialis and one over the peroneal nerve (or muscles; Fig. 14-14). The peroneal nerve electrode (for eversion) may be placed proximally near the fibular head or posterior to the lateral malleolus. The goal is to achieve dorsiflexion without inversion. Inversion is never acceptable due to the risk of an inversion sprain.
Patient position: If possible, place the knee in a position that mimics terminal stance, because that is when the FES needs to be activated. The orientation of the skin to the peroneal nerve can change with the position of the knee. You may need to readjust the electrode position after the patient is standing.
Treatment monitoring: Monitor response to the treatment by observing her gait.

Fig 14 ■ 14 Electrode placement to obtain dorsiflexion. Placement will vary between patients, and the clinician may need to try several different placements before obtaining the desired response.

provide unilateral FES to up to six muscles around the shoulder, elbow, and wrist, or it can provide bilateral FES to three muscles per upper extremity. There is no research with this specific device; however, one study[130] with a different device reported gains in oxygen uptake and power output for one subject with C6 tetraplegia and power output only for another subject also with C6 tetraplegia. In that study, only the bilateral biceps and triceps were stimulated.

When initiating an FES exercise program, several decisions need to be made regarding stimulation levels, which muscle to stimulate, and treatment dosage. Typically there is a preset limit on the current level, and the cycle will adjust the amplitude delivered based upon the cadence (speed) that the user is trying to maintain. For example, the patient may have a target cadence of 50 rpm and maximum amplitude of 140 mA. If the patient can cycle at 50 rpm with only 80 mA, the cycle won't increase the amplitude. However, if the legs start to slow down, the amplitude will increase to keep the legs cycling at 50 rpm (Box 14-9).

FES Rowing

A newer FES exercise technique is FES-assisted rowing for people with SCI. It is currently not available in the United States, but hopefully it will be in the future. This technique starts with a standard rowing ergometer and makes modifications to the seating to accommodate the needs of people with SCI. The user pulls back on the rowing handle and activates a button on the handle to turn on the stimulation to the quadriceps. The amount of pressure through the button determines the amount of stimulation delivered. The user then slowly releases the button to return to the flexed position. One study[131] showed gains in oxygen uptake and distance rowed after a program of FES rowing sessions performed for 30 minutes, three times per week for 12 weeks. The increases were reported to be comparable to those achieved with combined FES cycling/upper-extremity exercise and upper-extremity exercise alone.

Box 14 ■ 9 Critical Considerations for Using FES Cycling in the Clinic

- Set initial stimulation levels lower for patients with remaining sensation.
- Encourage volitional movement if present as long as it doesn't interfere with the cycling motion (i.e., if volitional movement causes significant spasticity during cycling).
- Typical muscles are quadriceps, hamstrings, and gluteal muscles, but others may be possible, depending on patient needs and the cycle being used.
- Cycling for 30 to 60 minutes, three times per week, is supported by the literature for patients with SCI. Cycling five times a week is reported but may be overly fatiguing.
- Typical cycling cadence (speed) is 40 to 50 rpm with resistance increasing as the patient is able.
- Read the precautions and contraindications for the FES cycle that is being used.

BIOFEEDBACK

Biofeedback (Fig. 14-15) is another option for improving function in patients with neurological conditions, and it can be combined with FES. For this technique, the patient can volitionally activate a muscle by trying to reach a target EMG amplitude. The FES can then complete any remainder of the motion or activity that

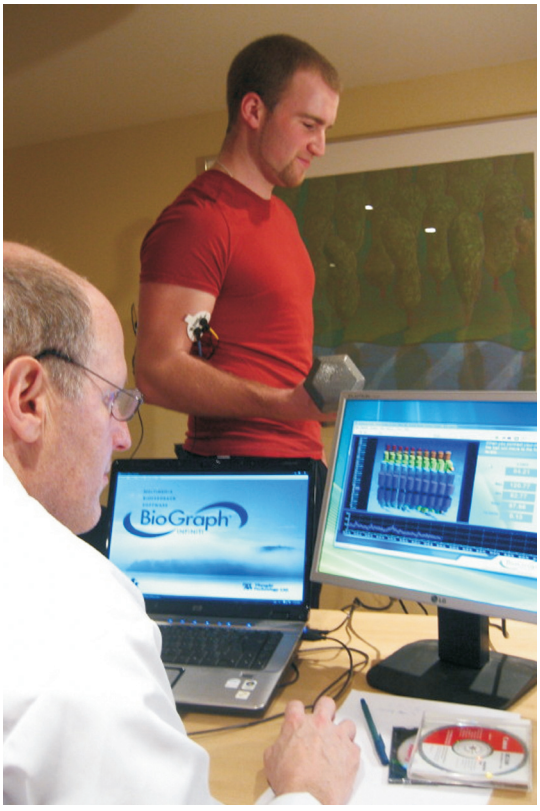

Fig 14 ■ 15 This figure displays biofeedback to the biceps and triceps muscles. This technique could be used to teach a patient to contract the biceps while relaxing the triceps. *(Courtesy © 2010 Thought Technology.com.)*

the patient is unable to complete. This combined technique of biofeedback with FES allows the patient to do as much as possible while obtaining feedback about their level of volitional activation but still have sufficient force generation from the FES to perform or complete the functional task.

Biofeedback has been used as a tool to aid recovery following stroke, to teach new movement patterns, and to decrease bladder dysfunction. Current evidence does not show support for the technique when used in patients who have had a stroke;[132] however, the studies to date are small, not well designed, and report varying outcomes, which makes comparisons difficult. A few studies have reported clinical changes after stroke patients followed a program of EMG biofeedback. One study[133] reported greater improvements in upper-extremity ROM and function for subjects at least 6 months after their stroke who had biofeedback

CASE STUDY 14-5 FES for Exercise

A 45-year-old man with a T8 complete spinal cord injury is concerned about his health and fitness. He has gained 10 pounds in the past year and feels that he has become more sedentary. He complains about increased fatigue with pushing his wheelchair. He is also worried about his bone density and his overall health.

CLINICAL DECISION-MAKING

1. Does the patient have a dysfunction or impairment that may be improved or lessened by the use of FES?
ANSWER: This patient may benefit from FES cycling to improve his fitness level, bone density, and muscle size. A functional goal could be to improve his endurance for functional tasks (i.e., wheelchair propulsion).

2. Why would FES cycling be a consideration for this patient?
ANSWER: Exercise options are limited for him due to his SCI. He also wants to improve his bone density and muscle size in his legs so that he is healthier.

3. What specific outcome measures can be used to assess the effectiveness of the agent?
ANSWER: VO_2, heart rate, 6-minute wheel test. You could work with his physician about imaging for his bone density.

4. Are there any precautions or contraindications to consider with this patient?
ANSWER: Considerations for FES cycling include osteoporosis, decreased sensation, spasticity, respiratory status, skin integrity, and any preexisting medical issues.

5. What specific parameters are recommended for this patient?
ANSWER:
Pulse duration: 150–400 μsec
Frequency: 30–40 pps
Amplitude: Enough to achieve the cycling motion. FES cycles usually have a limit that can be set, and the stimulation will adjust to keep the legs cycling at a preset speed (usually 40 to 50 revolutions per minute). A common maximum amplitude is 140 mA for each muscle.
Ramp time: None, because contraction needs to occur quickly.
Duration: Goal is to improve endurance to achieve 30 to 60 minutes of cycling, 3 days per week.

6. What are the proper steps to using FES cycling for this patient?
ANSWER:
Electrode application: Electrodes are placed bilaterally on the quadriceps, hamstrings, and gluteal muscles. Attach the stimulation cables correctly, as the cycle turns the proper muscles off and on through the cable. If connected incorrectly, the wrong pattern will occur.
Patient position: If the patient is using a cycle without a seat (e.g., RT300), he will remain seated in his wheelchair for cycling. It is recommended that his knees have a minimum flexion angle of 30° to 45° while cycling. Place his feet on the pedals and secure them with the straps over the foot and the calf.
Treatment monitoring: Monitor his response to the treatment by watching blood pressure and heart rate. Suspend treatment if he exceeds safe changes in these responses to the exercise and decrease the stimulation intensity.
Postinspection: Inspect the skin under the straps and any bony prominence that was in contact with the wheelchair while cycling (i.e., low back, ischial tuberosities). Extra care needs to be taken with his skin due to his lack of sensation.

added to a 1-year program of occupational therapy (OT) and FES. Subjects receiving only OT and FES also made gains, but not as much as those receiving OT, FES, and biofeedback.

Other studies have examined biofeedback for other populations. In ambulatory subjects with incomplete SCI, biofeedback training for the gluteus medius muscle during gait led to significant reductions in their Trendelenburg gait pattern. The biofeedback device in this study gave the subjects feedback if they were insufficiently activating the gluteus medius muscle while walking.[134] For children with CP, a program of biofeedback to increase activity (excitation) of the anterior tibialis muscles and to decrease the activity (inhibition) of the gastrocnemius muscles was added to a more traditional strengthening and functional program. Children who had the added biofeedback training showed reductions in gastrocnemius spasticity and increases in active ROM at the ankle. Biofeedback combined with traditional training did not provide greater improvements in gait over the traditional program alone.[135] However, biofeedback was not used during gait, suggesting specificity of treatment.

These few studies show that biofeedback may be a useful tool in the clinic with patients with neurological conditions. The same principles for use with any population can be applied, either for trying to facilitate desired motion, to inhibit unwanted motion, or to improve a functional task.

Documentation Tips

- Treatment type (NMES or FES) and purpose (e.g., spasticity reduction)
- Muscle(s) stimulated
- Electrode location, type, and size
- Stimulation parameters
- Treatment intensity and duration for NMES
- Type and duration of activity for FES
- Patient position
- Response to treatment (e.g., improved gait speed for FES)
- Any skin irritation post-treatment

Suggested Outcome Measures

See EDGE documents in the neurology section of the APTA website for information about outcome measures.[136] The outcome measures chosen will vary greatly depending on the application.

Strengthening

- Strength via MMT or dynamometry
- Improvement in a functional task that strength will impact

ROM

- Goniometry
- Improvement in a functional task that increased ROM will impact

Spasticity

- Modified Ashworth Scale
- Spasms frequency scale
- Spinal Cord Assessment Tool for Spastic Reflexes (SCATS)
- Improvement in a functional task that decreased spasticity will impact

Urinary Incontinence

- Bladder diaries
- Strength of pelvic floor muscles
- Quality of life measures: Continence Quality of Life Questionnaire or Overactive Bladder Questionnaire

Shoulder Subluxation

- Pain: visual analog scale
- Amount of subluxation: finger breadths or x-ray

Upper Extremity

- Strength via MMT or dynamometry and ROM
- Functional Independence Measure
- Action arm reach test
- 9-hole peg test
- Fugl-Meyer scores
- Arm motor ability test
- Box and blocks test
- Jebsen Taylor Arm Function Test
- Spinal cord impendence measure
- Wolf Motor Function Test
- Stroke Rehabilitation Assessment of Movement (STREAM)
- SF-36

Ambulation

- 6-minute walk (or wheel) test
- 10-m walk test
- Gait speed
- Motricity Index
- Stroke Rehabilitation Assessment of Movement (STREAM)
- 12-Item Multiple Sclerosis Walking Scale
- Emory Functional Ambulatory Profile
- Stroke Impact Scale
- Community Balance and Mobility Scale
- Walking Index for Spinal Cord Injury (WISCI)
- World Health Organization Quality of Life-BREF
- SF-36

Exercise

- VO_2 (oxygen uptake) and heart rate
- 6-minute walk test if ambulatory
- World Health Organization Quality of Life-BREF
- SF-36

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CHAPTER 15

THERAPEUTIC MODALITIES FOR TISSUE HEALING

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THE NORMAL HEALING PROCESS

Understanding the normal healing process will facilitate proper use of a given modality. There are three basic phases of wound healing: inflammation, proliferation, and remodeling. The inflammatory response is the body's nonspecific defense mechanism and begins almost immediately following injury. Inflammation can be triggered by a variety of causes, including trauma, disease, invading pathogens, or allergic reactions. Acute inflammation is characterized by varying degrees of redness, warmth, pain, swelling, and loss of function.

During the inflammatory phase, hemostasis occurs first. It begins almost immediately after injury and is responsible for stopping bleeding at the injury site. The most important cellular component is the platelet, which responds to the injured area, adheres to exposed collagen, and forms a clot that stops the bleeding. In addition, platelets play a role in later stages of healing because of the growth factors they produce. After the bleeding stops, edema continues to accumulate in the injured area due to extravasation, which is movement of fluid from the blood vessels into the extravascular space. This excess fluid results in swelling, redness, and elevated local temperature. In addition, distention of the local tissues and irritation of the free nerve endings in the injured area can result in pain. Although inflammation is usually perceived as being negative, a healthy inflammatory process is critical to successful healing. The tissue distention creates space for the influx of phagocytic cells and proteins that set the stage for later phases of healing. These cells, specifically neutrophils and macrophages, are responsible for cleaning the wounded area of nonviable material so the proliferative phase can begin.

The inflammatory phase leads to the proliferative phase, in which fibroblasts and keratinocytes predominate. The function of this phase is to repair the defect. It can last several weeks, depending on the injury. Fibroblasts are attracted to the wound by macrophages in the inflammatory phase and typically arrive in the area 48 to 72 hours after injury. Fibroblasts lay down collagen and elastin, which replace the tissue that was damaged in the initial injury or removed during the inflammatory process. If the injury resulted in a break

in the skin, keratinocytes will also be important to cover the wound with a new layer of epithelium.

The final stage is remodeling. During this phase, the newly formed collagen matrix is rearranged and continues to gain tensile strength. This stage is by far the longest and can last in excess of 1 year.

Tissue healing can become problematic at any phase. Tissues may remain chronically inflamed or may fail to regenerate as needed to heal the defect. In an effort to maximize healing, biophysical agents have been used in all phases of wound healing. The remainder of this chapter focuses on common physical modalities and their effects on tissue healing. A more encompassing discussion of the healing process is available elsewhere.[1]

CONVENTIONAL ULTRASOUND

The properties of conventional 1- and 3-MHz frequency ultrasound (US) as well as the physics describing generation and transmission of sound waves are discussed in Chapter 4. This chapter will focus on how the physical properties of US contribute to wound healing. When continuous-wave (100% duty cycle) US at a sufficient intensity is used, tissue temperatures are elevated as acoustic energy is transferred to the tissues in the form of heat. Although this thermal effect has been shown to reduce pain and muscle spasms in chronic injuries, it has not been shown to affect tissue healing directly. Most of the beneficial effects in regard to tissue healing stem from the proposed nonthermal effects of US, often delivered with a 20% duty cycle pulsed mode.

With pulsed US, the off-time of each pulse period allows the thermal energy to dissipate, so the net tissue temperature is not elevated. Instead of effecting change through temperature elevation, pulsed US causes gas bubbles in the tissues to expand and compress in response to the vibration caused by US waves. If the US intensity is too high, the bubbles will burst and damage the tissue. At therapeutic intensities, the bubbles may expand and contract to a smaller extent without bursting. This is referred to as *stable cavitation* and is not by itself clinically significant.

The stable vibration of the gas bubbles sets the stage for the remaining nonthermal effects, namely acoustic streaming and microstreaming. The vibration of gas bubbles leads to pressure differences within the tissue. This causes a circular flow of cellular fluids, known as *acoustic streaming*. Acoustic streaming is thought to be responsible for transporting materials within the US field, which can be used to alter cellular activities. The same general process is occurring at a cellular level and is called *microstreaming*. These microscopic currents help move ions that have accumulated next to the cell membrane, which will improve the cell's activity. To optimize the effectiveness of conventional US, the modality must be used properly and at the appropriate time. Through the nonthermal processes listed above, the use of US shortly after injury has been shown to accelerate the inflammatory process.[2] Because applying US during the inflammatory phase causes mast cells to release histamine, it is hypothesized that US may also cause the release of other chemical mediators that are stored in mast cells, which would lead to wound healing.[3]

Several studies have demonstrated that US can affect different aspects of cellular metabolism in vitro, including alterations in the amount of calcium uptake and growth factor production.[3–5] Many clinicians recommend US in the inflammatory phase because these factors can lead to a rapid onset of the proliferative phase of wound healing. In this phase, US increases collagen synthesis by human fibroblasts in vitro.[6] Another proposed mechanism by which US promotes tissue healing in the proliferative phase is by increasing the rate that capillary beds and blood vessels form; this has been demonstrated in ischemic muscle.[7] Animal studies examining the role of US in the remodeling phase of healing are largely inconclusive and appear to support the belief that US is most effective if begun within the week following injury. The challenge has been translating the physiological effects of US that are seen at a cellular level into clinically significant findings, such as faster wound closure, decreased rates of recurrence, or improved appearance.

Recent clinical studies have produced variable results when investigating the effects of US on wound healing. Due to factors such as studies without control groups or variable parameters, it is difficult to synthesize all the data pertaining to tissue healing to determine if US is effective. Even in studies with similar populations and methods, results are often inconclusive. A meta-analysis by Johannsen et al[8] reviewed the available literature pertaining to US in the treatment of chronic leg ulcers. Results showed that US significantly reduced wound size

at 4 weeks and 8 weeks compared to controls but did not demonstrate a statistical improvement in the percentage of ulcers that healed completely. Another meta-analysis reported evidence that US has a mild positive effect in healing venous ulcerations, but the authors point out that the studies used poor-quality research with limited sample sizes.[9]

Additionally, the effects of the local vascular status seem to play a role in the effectiveness of US treatment. Wound healing in full-thickness excisional wounds improved with US treatment if the wounds were well vascularized but was delayed for ischemic wounds.[10] This finding may be an important key to explaining why effects evident in controlled animal trials with surgically created wounds are often difficult to see in human wounds where ischemia is often an underlying cause.

For pressure ulcers, pooled results of randomly controlled trials do not support the use of conventional US for wound healing.[11] Despite the in vitro evidence supporting US for wound healing, the results in human wounds are equivocal at this point. In many cases, new studies only seem to raise more questions. For example, the recent VenUS III randomized controlled trial from the United Kingdom found the addition of 1-MHz US delivered at an intensity of 0.5 W/cm^2 and a 20% duty cycle to standard care was not cost-effective, nor did it affect healing rates.[12,13] In this study, US was only performed weekly at dressing changes. In a 2008 study from Poland, also involving venous leg ulcers, the same treatment parameters were used, except six treatments were performed per week instead of one.[14] Authors of this study assessed the effectiveness of US and surgical interventions on venous leg ulcers and concluded that US in conjunction with conservative care was more effective than conservative care alone. They also concluded that there was no added benefit for US in conjunction with a surgical intervention and that surgery was more effective than conservative management. Although both used the same parameters, the frequency of treatment may have been a factor in them reaching two differing conclusions about the effectiveness of US in venous leg ulcers. More well-designed, controlled trials with larger patient samples are required to determine the true effectiveness of therapeutic US. Table 15-1 lists contraindications and precautions to US.

TABLE 15–1. Contraindications to Therapeutic Ultrasound

Neoplasm
Pregnancy or using US over reproductive tissue
Circulatory impairment
Over eyes
Implants
Over epiphyseal plates in children
Sensory impairment
Bleeding/acute trauma
Over neural tissue
Infection

LOW-FREQUENCY ULTRASOUND

Over the past 10 years, the use of low-frequency ultrasound (LFUS) has increased in popularity to the point that it is becoming a common practice in the wound care setting. Although it shares many of the same principles as conventional US, LFUS requires a separate US machine with a frequency of 20 to 40 kHz (i.e., 20,000 to 40,000 Hz), compared to 1 to 3 MHz (1 million to 3 million Hz) for conventional US. Therefore, this form of therapy may not be a feasible option in a clinic that sees wound care clients only occasionally.

LFUS can be further described as low intensity or high intensity, which will have different effects on the wound. The low-intensity form uses a fine saline mist to transmit the 0.2 to 0.6 W/cm^2 of ultrasonic energy to the tissues at a frequency of 40 kHz[15] (Fig. 15-1). This is a noncontact, nonthermal therapy that is used to promote wound healing through the stimulation of cells and the removal of bacteria and other substances that delay healing. This form of low-intensity US was described as noncontact low frequency ultrasound (NCLFUS) in Chapter 4 and is known by its proprietary name MIST therapy.

Key Point! LFUS cannot be administered by conventional US devices more commonly used for musculoskeletal applications. A device specific to LFUS must be used.

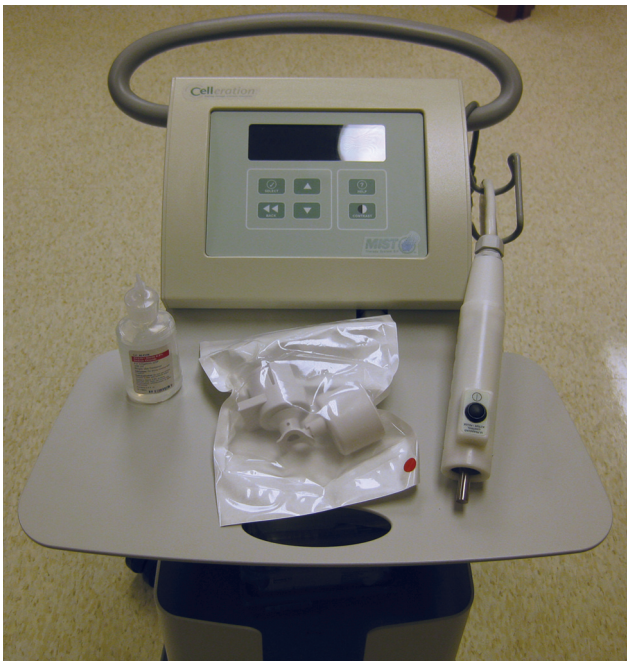


Fig 15 ■ 1 MIST low-frequency, low-intensity ultrasound device. *(Courtesy of Celleration, Inc., Eden Prairie, MN.)*

The number of peer-reviewed studies relating to the effectiveness of NCLFUS continues to expand, including one meta-analysis and four prospective double-blind, randomized controlled trials. Early studies investigated the effects of this form of US on diabetic foot ulcer healing and ischemic wound healing, respectively. Ennis et al[16] reported that 40.7% of diabetic foot ulcers healed within 12 weeks when treated with US compared to 14.3% that were treated with a sham device. In the study involving ischemic patients, Kavros et al[17] reported a 63% reduction in wound size compared to a 29% reduction in the control group.

One meta-analysis involved 444 patients from eight studies. Based on the pooled data, the authors concluded that an 85% reduction in wound area within 7 weeks, an 80% reduction in wound volume within 3 months, and a 79% reduction in pain would be expected with NCLFUS.[18] In addition to accelerating wound healing, a second utility for NCLFUS is reducing bacteria in wounds. In vitro studies have demonstrated effective bacterial reduction in as little as one treatment for certain bacteria, such as *Pseudomonas aeruginosa* and *Escherichia coli,* but no effect on *Staphylococcus aureus* was observed.[19] In vivo results also show a positive effect on the reduction of *P. aeruginosa* biofilms and other strains of bacteria that is similar to the effect of a topical silver dressing.[19,20]

The second form of LFUS uses a higher intensity and is delivered at a frequency of 22 to 35 kHz (depending on the machine being used) via a probe that is in direct contact with the tissue. Unlike NCLFUS, which works through cell stimulation and the removal of bacteria, this type is intended as a debridement tool capable of removing exudate, biofilm, and slough from the wound surface. Treatment time with this form of therapy is dependent upon the wound characteristics, primarily wound size and amount of nonviable tissue present. It is rare for a treatment to exceed 10 minutes in duration for most wounds. Due to the direct contact, tissue heating does occur and can be damaging if left stationary on the wound. This is the newest form of US on the market and is best described as *ultrasound-assisted wound debridement* (Fig. 15-2).

Evidence in the form of randomized, controlled studies for this form of LFUS is sparse, but numerous case studies and in vitro evidence demonstrate effectiveness in the removal of nonviable tissue and the destruction of bacteria. In one prospective, randomized, controlled trial involving 67 patients, ultrasound-assisted debridement

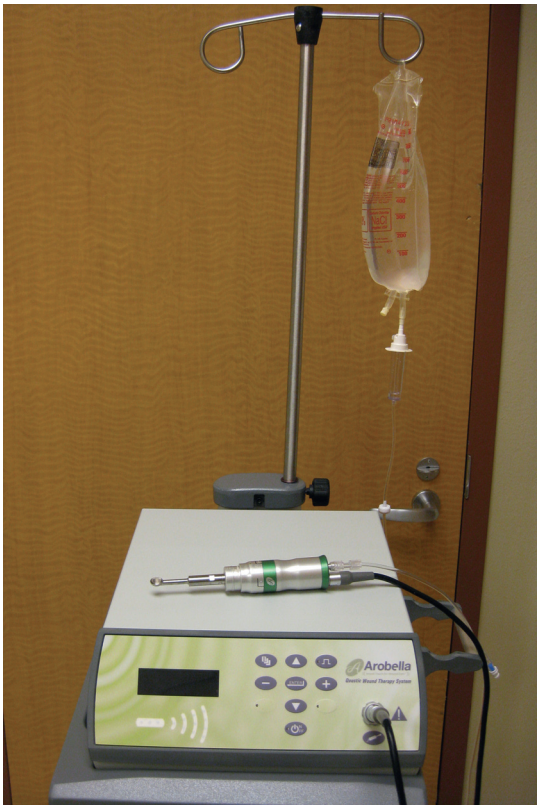


Fig 15 ■ 2 Arobella low-frequency, high-intensity device. *(Courtesy of Arobella, Inc., Minnetonka, MN.)*

was as efficacious and beneficial as traditional wound debridement.[21] In vitro, ultrasonic debridement reduced bacterial colonization by greater than 1 $\log_{10}$ compared to irrigation with saline[22] and was shown to increase the periwound skin perfusion pressure.[23] A recent in vitro investigation found a dose-dependent effect of 35-kHz LFUS on methicillin-resistant *Staphylococcus aureus* (MRSA), with significant reductions in bacteria levels and increased sizes of zones of inhibition with longer treatments.[24] These findings in support of both high- and low-intensity forms of LFUS as treatments for bacterial overload in a wound are promising and will benefit from experiments in chronic wounds to further elucidate the true level of effectiveness.

ULTRAVIOLET LIGHT

Ultraviolet (UV) radiation has a shorter wavelength (100 to 400 nm) than visible light and lies between the violet end of the visible spectrum and x-rays on the electromagnetic spectrum. UV is a form of light energy and can be further divided into ultraviolet light A (UVA), ultraviolet light B (UVB), and ultraviolet light C (UVC). All are produced naturally by the sun, but UVC is blocked by the ozone layer. As a result, the ambient sunlight we receive contains primarily UVA, small amounts of UVB, and no UVC.

Key Point! The ozone layer blocks UVC light; however, special lights can be used to deliver UVC treatments, which have been shown to be effective in treating localized infections.

In terms of wavelength and energy, UVA is closest to visible light and is the longest (i.e., wavelength) and least energetic; UVC is the shortest and most powerful, lying closest to x-rays on the light spectrum. Ultraviolet light has been shown to stimulate skin pigmentation, cell proliferation, and epidermal thickness; to enhance blood flow in cutaneous capillaries; to facilitate wound debridement; and to kill bacteria, which supports its use for healing.[25–30]

The most common use of ultraviolet light is for the treatment of skin disorders, including psoriasis, vitiligo, lichen planus, dermatitis, and more. Treatment for these conditions typically involves using ultraviolet light of varying wavelengths in one of three ranges: UVA (320–400 nm), broadband (BB) UVB (290–320 nm), or narrowband (NB) UVB (311–313 nm). The lower end of this range (between 290 and 310 nm) has been associated with nontherapeutic responses, such as burning and premature aging, because those wavelengths carry more energy than higher wavelengths do. As the longest wavelength, UVA will penetrate deeper into the skin, but it is the least powerful and is often used with a skin-sensitizing agent, such as psoralen. Due to the risks associated with BB-UVB and side effects of psoralen, NB-UVB has become the common treatment approach for most patients.

Because NB-UVB eliminates the lower wavelengths that are primarily responsible for producing erythema, positive results for the treatment of skin conditions have been achieved with fewer burning episodes.[31] Wavelengths longer than NB-UVB do not have an effect on psoriasis and vitiligo. Shorter wavelengths produce more burning, which could potentially be more carcinogenic.[32] NB-UVB was found to have comparable healing results with fewer burns and has longer duration of remission of psoriasis compared to those treated with broadband therapy.[33] Because of the lower energy emitted, larger starting doses are needed compared to broadband and more lamps are needed because of the reduced power.

The evidence for ultraviolet light as an effective modality for treating skin disorders is overwhelmingly positive. Two of the most common disorders treated are vitiligo and psoriasis. The application of NB-UVB or psoralen and UVA (PUVA) are considered to be the most important treatments for patients with vitiligo that affects more than 10% to 20% of the skin surface.[34] Several articles have demonstrated NB-UVB to be superior to PUVA treatment, as assessed by stability of the disease after treatment and color match of the repigmented skin for generalized vitiligo.[35–37]

It is proposed that repigmentation occurs when NB-UVB is applied to vitiligo because it stimulates proliferation and migration of melanocytes, which are the cells responsible for skin pigment.[38] A randomized controlled study involving 56 patients compared the effectiveness, measured as the percentage of repigmentation, of NB-UVB and PUVA for the treatment of vitiligo. Following 6 months of treatment, there were no significant differences in median repigmentation rates between NB-UVB and PUVA groups (45% vs. 40%, respectively). However,

when side effects are considered, the benefit of NB-UVB becomes apparent, as only 7.4% reported problems compared to 57.2% of the PUVA group.[39]

An alternative to ultraviolet light for the treatment of vitiligo is cultured autologous melanocyte transplantation, in which the patient's own melanocytes are transferred into the hypopigmented areas. The idea that repigmentation could be improved by combining the transplant with NB-UVB was supported by a 2014 study in which patients who underwent transplantation received either varying doses of NB-UVB or no UVB therapy. The group that received 20 sessions of NB-UVB before and 30 sessions after transplantation had significantly better results than other groups, with 81.3% of patients achieving repigmentation in more than 90% of the area and 94.8% of the patients experiencing at least a 50% improvement.[40]

Regarding psoriasis, several recent articles reviewed the evidence for ultraviolet light and came to varied conclusions.[41,42] Although PUVA was found to be more effective than NB-UVB in some studies and is still in use, many clinicians view it as a viable alternative for patients who do not respond favorably to NB-UVB rather than as a first choice of treatment. When compared to broadband therapy, narrowband has been found to be more effective in at least six different studies.[43]

In addition to its effectiveness, UVB has proved to be a safe treatment option. There is a low incidence of acute adverse events, and it can be used effectively at suberythemogenic doses, which eliminates the discomfort associated with burns and reduces the risk of cancer.[32,44] However, there is conflicting evidence regarding the cancer risk associated with UVB treatment. In animal studies, narrowband therapy has been implicated with DNA damage typically seen in cancer, and some human studies report a higher carcinogenesis of narrowband compared to broadband.[45] There are also studies reporting no evidence for increased skin cancer risk with UVB treatment.[46] Ultimately, a greater long-term risk is expected with higher doses. As a result, the smallest amount of UVB exposure necessary to achieve results should be used. The best frequency for treatment has yet to be definitively established. Treatment every other day was found to be as effective as daily therapy in one study, although NB-UVB therapy applied during two sessions per week took 50% longer for psoriasis to resolve compared to three weekly sessions in a randomized, observer-blinded trial.[47,48] Studies have also found no significant difference between NB-UVB administered twice per week versus four times per week or three times versus five times per week.[47,49] Currently, the most common recommendation is three treatments per week for at least 3 months.[43]

Clinical Controversy

Exposure to ultraviolet light is associated with certain cancers. However, it is an effective treatment for several skin diseases. Due to this conflict, it is recommended that the smallest effective dose be utilized to minimize the cumulative effects of ultraviolet light and that sunscreen be applied to the surrounding areas where treatment is not needed but exposure to UV is possible.

Unlike UVA and UVB, UVC is primarily used to reduce bacterial colonization in wounds. During the middle of the last century, its use fell out of favor as new antibiotics came into use and the effects of long-term radiation became apparent. However, as drug-resistant bacteria become more problematic, clinicians have reverted to previous methods, including UVC.

UVC radiation has wavelengths between 200 and 280 nm and is typically delivered from a lamp in the 250-nm wavelength range. At this wavelength, the erythemal effectiveness peaks but rarely causes burning as UVB and UVA do, even at high doses.[28] This is due to the poor penetration of UVC because it is almost completely absorbed by the epidermis. In addition, it is thought that UVC is less carcinogenic because any mutations it may cause will be sloughed off by the constantly changing epidermis.[50]

UVC has repeatedly exhibited effectiveness against bacteria, including those that are resistant to antibiotics. Both in vivo and in vitro studies have impressive outcomes. In vivo results have demonstrated 100% kill rates of MRSA at 180 seconds. In vitro studies have demonstrated effectiveness in reducing bacteria in artificially inoculated animal wounds.[51,52] The total exposure time to eradicate bacteria from chronic wounds is longer; this is due to several factors, including increased amounts of bacteria and bacteria that have invaded the tissue. Studies of a 3-minute exposure time of UVC to wounds of various etiologies showed greatly reduced numbers of bacteria,

but the bacteria were not completely eradicated from the wound (Fig. 15-3). Colonized superficial wounds respond well, but results are not as strong for heavily infected wounds or deep wounds.[53] There is also preliminary data that UVC may be a helpful adjunct in cancer therapy, as low doses of UVC killed most in vitro cancer cells while exposure to higher doses of UVA and UVB were less effective.[54]

For any ultraviolet treatment, the proper dose needs to be calculated. The power of the lamp, the distance the lamp is held from the skin, the patient's previous exposure to ultraviolet energy, and any medications that may sensitize the patient to UV light must all be recorded. The most important factor that needs to be determined is treatment duration, which ultimately depends on the patient's skin color. Individuals with light skin will burn faster than those with darker skin and must be treated with a smaller dose. As the amount of pigment increases in the skin, the amount of UV therapy applied will have to be raised to get the desired benefit. This is the same thing that occurs at the beach when fair-skinned people burn easily but those with a tan do not.

Dosing for UV therapy is determined by performing a minimal erythema dose (MED) test (Box 15-1). In this test, the smallest amount of time needed to produce mild sunburn is calculated (Fig. 15-4). When NB-UVB is used to treat psoriasis, the most common starting dose is between 0.5 and 1 MED and is increased incrementally, depending on the amount of erythema induced.[43] Dosage for UVC in wound care is different because there is no epidermis in the wound to assess for erythema. Instead, the dosage is usually given in terms of time, based on the characteristics of the wound tissue, including amount of necrosis and bacteria present. A typical dose for most wounds is no more than 180 seconds.[55]

Fig 15 ■ 3 DermaWand UVC applicator and control unit. Wand can be changed to provide UVB as well. *(Courtesy of National Biological Corporation, Twinsburg, OH.)*

Box 15 ■ 1 Performing a Minimal Erythema Dose (MED) Test

Equipment needed:

MED card, stopwatch, eye protection, sun protectant, drape for exposed skin

- Create a card with four 2 cm × 2 cm cutouts.
- Choose a test area of skin that is similar in pigmentation to the area to be treated, if possible.
- Cleanse the skin in the area to be tested to remove all oils, lotions, dry skin, etc.
- Apply a sun protectant (SPF 30 or greater) or a 2-mm layer of petrolatum and a paper towel to adjacent skin that may be exposed.
- Apply UV goggles to both the clinician and the patient.
- The card from step 1 is placed over the test area, and the 3 squares to the right are covered.
- The lamp is positioned and turned on. At 15 seconds, the second square is uncovered, followed by the third square at 30 seconds and the final square at 45 seconds.
- The lamp is turned off at 60 seconds. At this point, the square on the far left has received 60 seconds of UV light, followed by 45, 30, and 15 seconds in the other three squares.
- Twenty-four hours later, the skin is assessed for the area with barely perceptible redness. The time corresponding to that square is the MED.

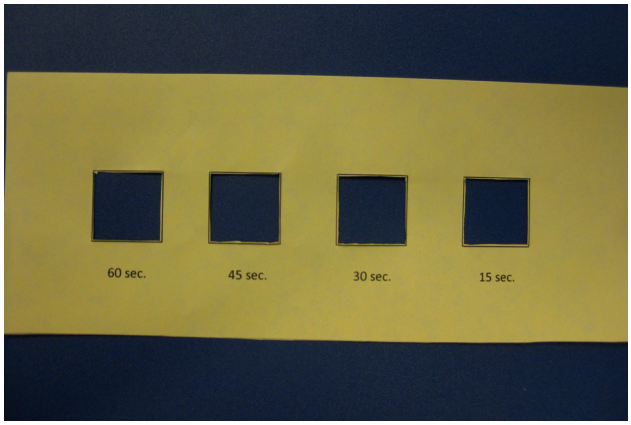


Fig 15 ■ 4 MED card for calculating a minimal erythema dose.

ELECTRICAL STIMULATION

A review of the general principles of electrotherapy is suggested before reading this section. For an in-depth discussion on the basics of electrotherapy, see Chapter 9.

Electrical stimulation (ES) for tissue healing is intended to accentuate the normal healing process that occurs when skin is injured. Foulds and Barker[56] identified a separation of charge between the interior and exterior of the human skin. Based on these findings, they were able to determine that the skin's exterior has a negative polarity relative to the skin's interior. This difference creates potential energy in the same way that a battery stores energy; thus, it is referred to as a *human skin battery.* A primary factor in the production of the negative external polarity is the movement of sodium (Na^+) along its concentration gradient from the external skin surface into the epithelial cells. When there is a break in the skin, the interior of the wound is positively charged compared to the periwound area, which creates a voltage difference at the wound margin. This difference in electrical charges drives the healing process—as cells and ions are drawn to areas of opposite polarity and repelled from areas with the same polarity (see Fig. 9-33 for additional explanation).

When ES is applied to the skin to promote tissue healing, it produces current flow in the tissues that mimics the natural skin battery. The two most commonly used waveforms to promote tissue healing are high-volt pulsed current (HVPC) and low-intensity direct current (LIDC, or *microcurrent*). (For a review of the specifics of these current types, see Chapter 9.) HVPC and LIDC are both monophasic currents that result in a net flow of charged particles, which in turn, yield the desired effects of wound healing. The process of attracting charged cells to an electric field of opposite polarity is known as *galvanotaxis*. When a positively charged electrode (anode) is placed over a wound, cells and ions that are negatively charged will be drawn toward the electrode. Cells and ions that are positively charged will be repelled.

Key Point! The skin should be inspected before treatment for any signs of irritation and reassessed after treatments. This is especially important in wound healing where the traditional currents are monophasic and can lead to an accumulation of charge under the electrodes. Extra caution should be used when electrodes are placed close together because there is less area for the charge to disperse.

Studies pertaining to the effects of galvanotaxis on different cell types have led to a general understanding of which electrode setup will be most effective during different stages of healing. When tissue is injured, there are certain endogenous cues that guide wound healing. These cues include substances released by cells in the wound, the combination of open space on one side of a cell adjacent to the wound combined with pressure from adjacent cells propelling cells into the wound, and mechanical stimulation caused by the injury.[57] When an electrical current is applied to tissues, the effects of these cues on cellular movement can be enhanced, stopped, or reversed, depending on the strength and polarity of the current applied. In other words, it appears that current is the overriding factor that determines which direction cells will migrate, not the endogenous factors.[57]

When a current is applied to actual wounds rather than isolated (or plated) cells or individual ions, the predicted response is not as straightforward. New data has shed light on this issue, with important clinical implications as explained below. Previous treatment recommendations were based solely on the galvanotactic effects. A frequently cited article published in 1985 reported that epidermal cells migrated toward the anode when direct current was used because they are negatively charged.[58] Based on this finding, most references that are more than a few years old recommend using the anode over the wound site to stimulate epithelialization. However, research showing the effects of an applied electrical field to a layer of corneal epithelial cells shows that normal rate of epithelial migration is enhanced when the wound is treated with cathodal stimulation, and migration away from the wound edge occurred when treated with the anode.[59] In response to this new information, the cathode is now used to promote re-epithelialization of a wound. (See Table 15-2 for an overview of the appropriate polarity for each aspect of wound healing.)

In addition to the effects of galvanotaxis on cell migration, ES has also been shown to increase cellular proliferation (the creation of new cells) in fibroblasts. An increase in DNA and protein synthesis has been identified in several studies involving the application of stimulation.[62–65] In one study by Bourguignon, an increase in calcium uptake and an upregulation of insulin receptors was reported.[66] These changes led to an increase in protein and DNA synthesis, which are important for the production of granulation tissue.

None of the cellular mechanisms that occur in response to ES would be of any significance if they did not

TABLE 15–2. Choosing the Appropriate Current Based on the Goal of Treatment[60,61]

Treatment Goal	Cells Recruited	Polarity of Current
Debridement	Macrophages	Anode
	Neutrophils	
Infection	Activated neutrophils	Anode placed over an ionic silver dressing to drive silver into wound or cathode to attract activated neutrophils
Granulation	Fibroblasts	Cathode
Wound contraction	Myofibroblasts	Alternate anode and cathode
Epithelialization	Keratinocytes	Cathode
	Epidermal cells	

translate into improved clinical outcomes. There is an abundance of clinical evidence supporting both the LIDC and high-voltage approaches to tissue healing. (See Table 15-3 for a brief summary of the effects of LIDC on tissue healing in humans.)

Clinical evidence for the use of high-voltage pulsed current is also strong and corroborates the findings in animal and in vitro models. In human studies, HVPC has led to improved blood flow,[75,76] faster healing rates,[77] and higher percentages of wounds that heal versus a control group.[77] A meta-analysis by Gardner et al[77] examined the effects of various forms of ES on chronic wound healing. Based on data from the 15 studies reviewed—consisting of 591 ulcers in the treatment group and 212 in the control group—the authors found a significant increase in the overall healing rates when

TABLE 15–3. Low-Intensity Direct Current and Tissue Healing in Humans

Author	Study Type and Population	Parameters	Polarity	Results
Junger et al, 1997[67]	15-patient case series; venous ulcers	630 μA at 128 pps with pulse duration of 140 μsec with for 30 min/day	(−) for 7–14 days, then (+) for 7–10 days, then (−)	Improved capillary density; ulcer size reduced 63%
Assimacopoulos, 1968[68]	Three case reports; venous ulcers	50–100 μA continuously	(−)	All healed within 42 days
Wolcott et al, 1969[69]	5 patients in a clinical trial; venous ulcers	200–800 μA for ≥ 2 hr/day	(−)	Higher rate of healing than control
Wolcott et al, 1969[69]	75 ulcers in a clinical trial; ischemic ulcers	200–800 μA for ≥ 2 hr/day	(−)	40% healed in treatment group; 0% of controls healed
Gault and Gates, 1976[70]	Controlled trial; 106 ulcers in 76 patients; ischemic ulcers	200–800 μA for 6 hr/day	(−) for 3 days or until 3 days after infection cleared, then (+)	48% healed and another 11% improved > 95%; no control ulcers healed
Wolcott et al, 1969[69]	6 patients in a clinical trial; multiple pressure ulcers	200–800 μA for ≥ 2 hr/day	(−)	5/6 of wounds treated with stimulation healed; 0/6 of controls healed
Barron et al, 1985[71]	Prospective study; 6 patients with nonhealing pressure ulcers (retrospective)	600 μA, 3x/wk for 3 wk	Biphasic	5/6 healed in 1 month; 95% average area of reduction
Wood et al, 1993[72]	Double-blind, controlled study; 74 patients with chronic stage II and III pressure ulcers	600 μA pulsed current 3x/wk for 8 wk	(−)	Treatment: 58% healed; 72.9% reduced > 80%; 0 increased in size Control: 3% healed; 12.9% reduced > 80%; 32/3% increased in size

Continued

TABLE 15–3. Low-Intensity Direct Current and Tissue Healing in Humans—cont'd

Author	Study Type and Population	Parameters	Polarity	Results
Huckfeldt et al, 2007[73]	RCT; 30 patients with full thickness burns	50–100 μA dependent upon wound resistance	(+)	36% faster healing in treatment group
Carley et al, 1985[74]	30-patient clinical trial with wound of various etiologies	300–700 μA depending on innervation of skin	(−) for first 3 days, then reversed	Treatment group healed 1 to 2.5x faster than paired controls after 3 wk of stimulation

RCT = randomized controlled trial.

ES was used compared to the control group. A statistical difference was not obtained when healing rates for different forms of ES were compared. Other forms of ES have been successfully used in wound care, but a discussion of them is beyond the scope of this chapter.

Optimal results with ES for wound healing involve more than selecting the correct polarity; as with neuromuscular electrical stimulation, dosage must also be considered. In the literature, a dosage of current that is between 250 to 500 μC/sec produced the most favorable results; this is true for both high-volt and low-volt stimulation.[61] Unfortunately, when a current is applied through surface electrodes, we cannot be sure of exactly how much current is being delivered to the tissue because of periwound resistance, conductivity, and other factors. To achieve a dose in the specified range using HVPC, voltage is set in the range of 75 to 150 V, which sensate patients will perceive as a strong tingling paresthesia around the wound. If patients are insensate, the voltage should be turned up until muscle twitching is visible, then turn the voltage down just enough to stop the twitching. For optimal results, treatment should take place every day or nearly every day for 45 to 60 minutes.

Other factors that will determine the amount of current being delivered are pulse frequency and pulse duration. On many machines, pulse duration is preset for HVPC, so the clinician is not able to select a duration. Pulse frequency is usually set to 100 pps. Because of the discrepancies between the data related to polarity, clinical use is determined by the evidence as well as best practice. Current best practice recommendations for using HVPC to treat noninfected wounds include starting with the cathode at the wound site. Continue with cathodal stimulation as long as wound progress is being made. When progress stops or the wound regresses, switch the polarity to positive and continue to treat the wound with the anode as long as progress is being made. If necessary, change the polarity back to negative if progress stops again. If this is the case, use the cathode for seven to 14 treatments as long as healing continues.[61] To reduce the risk of infection, sterile electrodes should be used on wounds. Electrodes should never be shared with other patients. (See Box 10-6 and Box 10-7 for a list of contraindications and precautions to ES.)

INFRARED ENERGY

Initially, infrared energy was used for its local heating (i.e., thermal) effect, but this is no longer a common practice for most clinicians. Currently, the primary use for infrared energy is to restore protective sensation, which is often lost in diseases that affect the peripheral nerves, most notably diabetes, and is a primary risk factor in the development of foot ulcers.[78] Many practitioners have used monochromatic infrared energy (MIRE) to restore sensation to affected limbs. The MIRE device delivers an 890 nm wavelength of infrared energy that is transmitted directly to the skin via pads containing 60 diodes each[79] (Figs. 15-5 and 15-6).

MIRE was initially approved by the Food and Drug Administration to increase circulation and reduce pain. The proposed mechanism of action is the stimulation of nitric oxide (NO) release from hemoglobin into the circulation when hemoglobin absorbs the infrared energy. Because NO is a strong vasodilator, increased amounts of it under the diodes will lead to improved circulation in that area.[79] This is supported by a recent investigation into the effects of MIRE on microcirculation.[80] In this

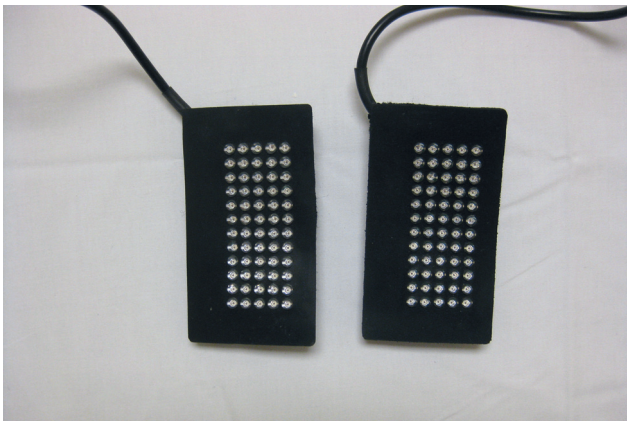


Fig 15 ■ 5 Anodyne pads with 60 diodes per pad used to deliver MIRE. *(Courtesy of Anodyne Therapy, LLC, Tampa, FL.)*

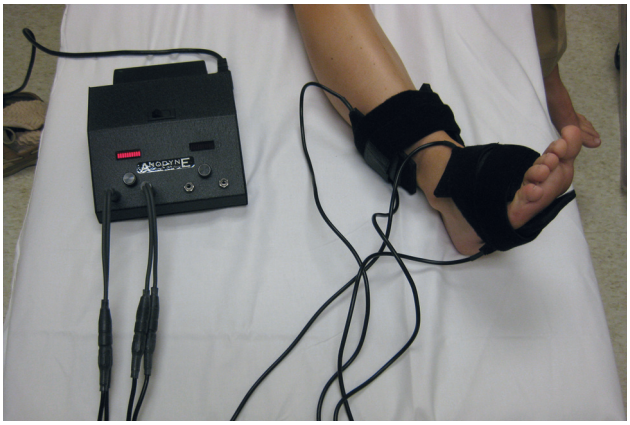


Fig 15 ■ 6 Pad placement for MIRE treatment of neuropathy: two pads on lower leg and one each on the plantar and dorsal aspects of the foot.

study, 30 healthy subjects were assigned to either an active MIRE group, a placebo MIRE group, or a control group that received warm packs on the feet. Following a 30-minute session, the active group had significantly higher capillary blood flow velocities and superficial skin blood flow compared to the other groups. The authors logically concluded that, because the group treated with warm packs did not have an increase in blood flow, the effects from MIRE must be related to the photo energy and not the warming effect. The fact that MIRE can cause vasodilation and improve microcirculation is the basis for its use in the treatment of peripheral neuropathy; it is hypothesized that the increased circulation will lead to healthier nerves and a return of sensation.

Experimentally, results from studies using MIRE to improve sensation are equivocal. A number of studies report that MIRE had a positive effect on loss of protective sensation, ranging from mild improvement to complete resolution.[81–85] Many of the early studies that found MIRE to be beneficial were noncontrolled and industry funded. Conversely, three independent, double-blind, placebo-controlled studies involving 117 neuropathic patients did not find MIRE to be beneficial for treating impaired sensation when compared to control groups.[86–88] The authors of many studies point out that small sample sizes may limit the power of the study, making it difficult to identify a difference between the control and treatment group. The most recent randomized, controlled study was a single-blinded study on 30 feet from 24 patients with diabetic neuropathy. No significant differences between the sham and treatment group were found when assessed 6 weeks and 12 weeks following 12 daily sessions of MIRE.[89]

Clinical Controversy

Although early evidence was favorable, recent blinded, independent studies have failed to show effectiveness of MIRE for the management of neuropathy. In addition to the controversy over MIRE's effectiveness, there is also disagreement pertaining to the duration of effect, if any, after the modality is discontinued.

At present, there are still many unexplored factors that may play a role in the effectiveness of MIRE therapy. Severity of neuropathy at the initiation of therapy appears to be related to MIRE's effectiveness.[83] In addition, there is no clearly defined frequency and duration of treatment in the literature that offers the best evidence for successful outcomes. From a functional standpoint, recent data also suggests MIRE is not an effective intervention for balance associated with diabetic neuropathy or quality-of-life issues associated with osteoarthritis of the knee.[90,91] The only way to truly determine the effectiveness of MIRE is for more well-designed, double-blind studies with larger sample sizes to account for small changes to be performed. There is currently no consistent body of evidence supporting MIRE as a modality for the treatment of sensory neuropathy.

INTERMITTENT PNEUMATIC COMPRESSION

Intermittent pneumatic compression (IPC) is used for a variety of conditions, as outlined in Chapter 8. Of

these conditions, the most common is venous thromboembolism prophylaxis. Several other indications have particular relevance to wound healing. These are for the management of venous, ischemic, and lymphatic complications. This section will focus on the physiological effects of compression therapy that promote wound healing for each of these three indications. Treatment guidelines and a discussion of the proper use of IPC in the clinic are covered in more detail in Chapter 8.

Despite the wide use of intermittent compression, no single theory has been proven regarding the exact mechanism of action. Proposed effects of IPC can be mechanical or chemical in nature. The mechanical effects, as identified in a study by Khanna et al,[92] include improved vascularity, cyclical loading, and a redirection of blood flow.When IPC is applied, it squeezes the limb, which compresses blood vessels and causes blood to move forward through the vessel (Fig. 15-7). The increased force leads to an increased peak flow velocity, which is thought to reduce the amount of blood that pools in the sinuses around the valves.[93] When the pooling of blood is reduced in the veins, pressure there will also be reduced. This creates a larger pressure gradient between the arterial system and the venous system, which leads to increased blood flow to the affected limb. With more oxygenated blood in the vessels, there is more blood available for tissue healing, including soft tissue and bone.[94,95] The theory that increased blood flow caused by compression will bring nutrients to tissues to assist with the healing process seems to be supported by the studies discussed in Chapter 8.

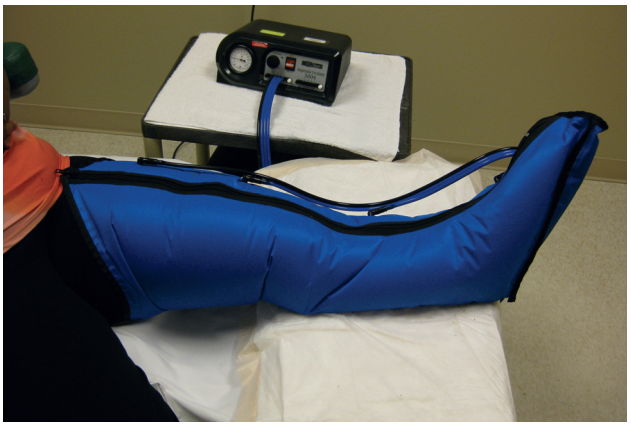


Fig 15 ■ 7 Intermittent pneumatic compression applied to treat an ulceration caused by venous insufficiency. *(Courtesy of Bio Compression Systems, Inc., Moonachie, NJ.)*

The second proposed mechanical effect pertains specifically to injured bone. Applying IPC to muscles proximal to a fracture site has been shown to increase cyclical loading in the bones of sheep. It is hypothesized that the same effect would be achieved in humans, leading to increased bone formation at the fracture site.[96] The third proposed mechanical effect is a redirection of blood flow that occurs when the venous system is compressed. When compression is applied to the leg, raising the pressure in the venous system, venous channels in the long bones are recruited to assist with venous circulation. Ultimately, adding IPC increases blood flow to the bone in the area being compressed, which is thought to assist with bone healing. Significant increases in blood flow to the periosteum following IPC have been demonstrated in rabbits.[94] In humans, adding IPC to one limb following the injection of a radiopharmaceutical agent led to significantly increased uptake by the long bones, indicating that more blood was being delivered to the bones under compression.[97]

One of the proposed chemical effects of IPC is an increased production of nitric oxide. This effect has been demonstrated experimentally in cultured endothelial cells by exposing them to a cyclic strain at 60 cycles per minute and in rats following 30 minutes of IPC.[98,99] It is thought that nitric oxide is produced in response to the shear stresses on the endothelium of the blood vessels created by compression.[100] Because nitric oxide is a vasodilator, it is believed that an increase in nitric oxide will lead to increased blood flow to the area.

A second chemical effect of pneumatic compression is an increase in certain proinflammatory agents. The proposed benefit of this relates to the secondary effect of these agents. In addition to their role in promoting inflammation, chemical mediators, such as substance P and calcitonin gene-related peptide, play a role in the early phase of tissue healing.[100,101] An investigation by Dahl et al[95] identified increased levels of these mediators, along with elevated sensory neuropeptides following daily compression therapy given to rats with imposed Achilles tendon injuries. The increased levels of mediators were accompanied by increased production of fibroblasts and a higher blood vessel density in the area of treatment.

In the past, IPC typically referred to a single- or multichamber pump like those used for the management of venous dysfunction. Compression was applied with a

prolonged on-time relative to the off-time to facilitate movement of fluid from the affected limb. As a general guideline, the pressure was set to a fairly low level, frequently in the 30- to 40-mm Hg range, but always below the patient's diastolic pressure to prevent arterial compromise. Now, with the development of pumps specifically designed for the management of lymphedema and arterial compromise, the category of IPC has expanded. Pneumatic compression devices that are designed specifically for lymphedema mimic the effects of manual lymphatic drainage, which is considered to be the gold standard for lymphedema management. These pumps, referred to as *advanced compression devices,* work at a lower pressure than venous pumps and produce a rhythmic stimulation designed to move fluid from the limb toward the trunk. Results from a 2013 study, funded by the device manufacturer and involving 196 patients with lower extremity lymphedema, found a significant reduction in limb volume along with improvements in level of pain and increased functional ability reported by patients.[102] This study did not include a control group or a comparison to traditional compression devices. In a randomized, controlled trial, pneumatic compression devices were compared to advanced compression devices for patients with lymphedema following breast cancer treatment. The 36 subjects completed 1 hour of compression daily for 12 weeks. The group treated with the advanced compression device had a 29% reduction in edema compared to a 16% increase in edema for the standard group.[103]

When IPC is used to treat venous insufficiency, one of the contraindications is significant ischemia. A prudent recommendation is to avoid this type of compression when the ankle-brachial index (ABI) is below 0.5 and to use it with caution when between 0.5 and 0.8. However, compression devices that deliver short-duration, high-pressure contractions are now available that are intended to treat limbs with arterial compromise (i.e., those with an ABI below 0.5).

There are several major differences between arterial compression pumps and venous pumps. The most obvious difference is the appearance, as shown in Figure 15-8. The arterial pumps cover only the foot and calf as opposed to the whole leg and have bladders under the sole of the foot and behind the calf. The second major difference is the amount of compression applied, with the arterial pumps applying a much higher pressure to the leg than venous pumps. For each of the devices pictured, the bladder at the foot will rapidly fill to a preset pressure of 120 mm Hg, followed by filling of the calf bladder after a 1-second delay. A third difference involves the sequencing of compression and relaxation. Although venous pumps may be applying compression as much as three times longer than the relaxation phase (90 sec on, 30 sec off), these devices provide compression for 3 seconds followed by a 17-second relaxation time. The combination of the forceful contraction to clear the venous system and the long relaxation time are intended to reperfuse the ischemic tissues. For best effect, this treatment is performed daily for several hours, so it is often used in the home setting.

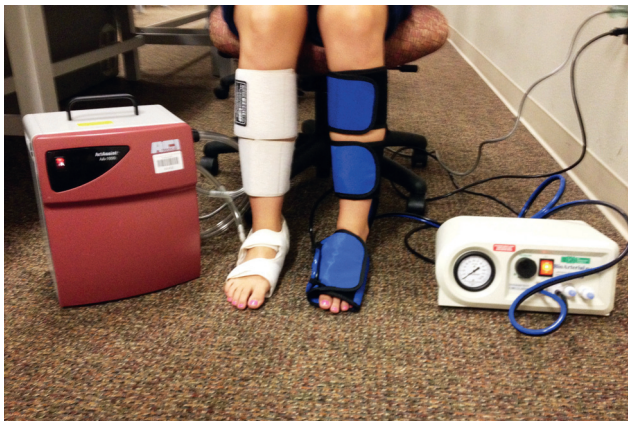


Fig 15 ■8 Arterial compression pumps. Left leg: BioArterial Plus System. Right leg: ArtAssist. *(Devices courtesy of Bio Compression Systems, Inc. Moonachie, NJ; ACI Medical, San Marco, CA)*

Evidence supporting the use of arterial compression for ischemic limbs is strong. Several prospective, randomized studies found IPC delivered with the arterial compression devices was beneficial for patients with intermittent claudication. Two separate studies compared IPC (2–2.5+ hours per day for 5–12 months) combined with unsupervised exercise and aspirin versus a control group that received aspirin and unsupervised exercise. Results of both studies indicate that compression therapy is effective for reducing claudication symptoms as evidenced by improvements in walking distance before the onset of pain and walking distance before the patient must stop.[104,105] When IPC is compared to supervised exercise and unsupervised exercise for claudication management, the compression and supervised exercise groups had comparable outcomes, and both were significantly better than the unsupervised group.[106] Additionally, improvements gained via IPC were preserved at 6 to 12 months following conclusion of treatment.[104,106]

The second subset of arterial patients who benefited from pneumatic foot and calf compression is people with critical limb ischemia (CLI). CLI is more severe than intermittent claudication and is associated with pain at rest or with ulcers and gangrene. Without revascularization, these limbs often require amputation. A controlled study involving 48 patients with nonhealing ischemic wounds following toe or trans-metatarsal amputation evaluated the effectiveness of IPC over an 18-month period. Of the 24 patients treated with IPC, 14 had complete wound healing and 10 required below-knee amputations (42%). Only four of the 24 patients in the control group had complete healing, while the remaining 20 (83%) underwent below-knee amputations.[107] Other positive outcomes have been shown with foot and ankle compression for CLI, including improved quality-of-life scores, improved amputation-free survival time, improved walking ability, and increased transcutaneous oxygen pressure (TcPO2) readings.[108,109]

Clinical Controversy

Pneumatic compression can be dangerous when used inappropriately to manage lymphedema and arterial compromise. Devices specifically designed to mimic manual lymphatic drainage for lymphedema and to provide high pressures over a short duration for arterial issues have demonstrated effectiveness in managing their intended conditions.[107]

SUPERFICIAL HEATING MODALITIES

The primary indications for superficial heating are pain reduction, promotion of tissue distensibility, and relief of muscle spasms. Superficial heat is not commonly used to promote tissue healing directly. However, heating injured tissues does have a theoretical benefit in regard to healing, so the proposed mechanisms for how this may occur with superficial heat will be addressed briefly.

The superficial heating modalities most commonly used are hot packs and paraffin wax. Hot packs can be divided into disposable and reusable varieties as well as moist and dry. The disposable hot packs are rarely used in clinical practice because of the excessive cost and waste associated with them. When a hot pack is heated in a microwave or hydrocollator or through an exothermic reaction (in the case of a disposable pack), the pack absorbs heat. The hot pack is then placed on the skin over a protective layer of towels to prevent burning. The heat will be transferred from the hot pack to the skin, with a 1-cm to 2-cm layer of towels acting as insulation to prevent the skin temperature from rising too high. This layer is critical because hydrocollator temperatures are typically set between 158°C and 176°F (70°C and 80°C), and the desired skin temperature achieved is approximately 104°F (40°C).

Key Point! Tissue temperature will increase faster when superficial heat is applied under compression therapy. If compression must be applied (e.g., with a patient lying on a hot pack), extra layers of towels should be placed under the hot pack to avoid tissue trauma. Special care must be taken when thermal modalities are used in areas with reduced vascularity because the ability to dissipate heat may be affected.

Similarly, paraffin wax transfers heat to the skin through conduction. To accomplish this, the affected extremity is repeatedly dipped in wax until a layer of wax 2 to 3 mm thick is produced. It is then wrapped to provide insulation. Wax can be comfortably applied at temperatures of 122°F (50°C) because the wax will cool and solidify when it comes in contact with the skin. The wax is typically left in place for 15 to 20 minutes, at which point it is adding a small amount of heat to the tissue and providing insulation that greatly reduces the amount of heat loss. This is a common superficial heating modality in the treatment of rheumatoid arthritis and burns in areas of potential contracture formation, most notably the hands.

Although studies using superficial heat to promote tissue healing are lacking, superficial heating is known to raise the local skin temperature and lead to vasodilation.[110,111] An elevated local metabolism and increased blood flow accompanying increases in temperature also assist tissue healing. A second proposed mechanism by which superficial heat could promote healing is the reduction of muscle spasms, which can lead to local ischemia. Wright and Sluka[112] describe how heat could reduce ischemia by increasing the firing of the type Ib Golgi tendon organ afferent fibers. This reduces the firing of the agonist muscles, which would reduce muscle spasms

and, in turn, reverse localized ischemia. Despite the documented local improvement in blood flow that could potentially contribute to tissue healing, there is a lack of evidence that supports superficial heat as a primary modality to promote tissue healing.

If superficial heating is used, either as a primary modality to increase blood flow or in conjunction with another modality, several concerns need to be addressed. The skin should always be inspected before initiating therapy with a hot pack. The clinician should assess for skin sensitivity to hot and cold and arterial perfusion and should perform a thorough history to identify any condition or medication that may make the tissue intolerant to heat. Because metal conducts heat faster than soft tissue, caution must be taken when applying heat to any area with implants. Superficial heating should be avoided in cases of poor vascularity because the lack of blood flow will impair heat dissipation via convection. Finally, because heating leads to vasodilation and increased blood flow to the area, it is contraindicated in the presence of cancer and is generally harmful in heavily draining wounds because drainage will increase. Even if there are no contraindications, the patient should still be frequently reassessed during the treatment and should be given some mechanism, such as a bell, to call for the therapist if the heat source gets too hot.

CRYOTHERAPY

Similar to superficial heat, cryotherapy is not a common modality in the management of open wounds. A major goal of wound healing is to promote a normothermic environment. When superficial heat or cryotherapy is applied to an area without intact skin, the effects can be amplified and can potentially cause damage. Many open wounds are complicated by reduced blood flow to the area. With the application of heat, this impairs the body's ability to dissipate that energy and may cause local damage. With ice, the effects of local vasoconstriction may be harmful to healing tissues. However, because of its role in reducing edema, cryotherapy, in the form of ice or cold packs, is a common treatment during the initial inflammatory process following soft tissue injuries in the presence of intact skin. As the cold pack is placed on the skin, energy is transferred between the ice pack and skin, leading to warming of the ice pack and cooling of the tissue. As tissues cool, blood vessels in the area constrict and microvascular permeability decreases.[113,114] Despite the wide use of cold therapy, few studies have been performed to determine the optimal parameters of treatment (i.e., duration and frequency of treatment and temperature of the cold pack at the beginning of treatment).

A study by Enwemeka et al[115] investigated the effects of cold pack therapy at different tissue depths. For superficial tissues (1 cm deep) in healthy individuals, a significant drop in temperature was observed following 8 minutes of cold pack therapy. Although there was a rapid change superficially, 20 minutes of cold pack therapy did not reduce the temperature of tissues deeper than 2 cm. Interestingly, the authors noted a temperature reduction in the deeper tissues after the cold pack was removed. One factor that may play a role in this is the shunting of blood from the superficial vessels to the deep vessels due to vasoconstriction followed by a reversed flow of blood from the deep to superficial vessels to reheat the tissue. The effective depth of penetration depends on the amount of circulation in the area as well as the type and amount of tissue that is present. Ice used to lower the temperature of deeper tissues will be impaired by adipose tissue because of its insulating qualities.

Key Point! With the exception of ice massage, ice should never be placed directly on the skin due to the risk of frostbite. It is critical to ensure that a patient has an adequate vascular supply before initiating cryotherapy.

Due to different quantities of adipose tissue between species, it is difficult to base recommendations for treatment duration and frequency of cryotherapy on animal studies, but the physiological changes may be the same. Closed tissue injuries in rats responded favorably to the application of cryotherapy. Improvements in functional capillary density, decreased intramuscular pressures, reduced number of granulocytes, and reduced tissue damage were all noted with cryotherapy.[116]

These findings support the use of ice in the inflammatory phase to reduce edema, which can speed up the healing process. Although ice is commonly used in all phases of rehabilitation to manage pain and muscle spasms, there is no evidence that it promotes healing beyond the inflammatory phase. A systematic review of randomized controlled trials showed ice to be more

effective than heat or alternating heat and cold to manage edema.[117] This review pointed out the lack of consistent protocols, making an evidence-based recommendation for treatment duration difficult. A general consensus is that 15 to 20 minutes is an acceptable time frame because it is long enough to decrease tissue temperature but is not likely to cause tissue damage.[115,117–119] The majority of studies involve superficial injuries to ankles and knees, so caution must be taken when attempting to extrapolate these findings to deeper injuries, such as a hamstring tear.

Investigations into the most effective temperature for cryotherapy are predominantly related to the acute treatment of burns. Although a specific temperature has not been identified as most effective, cold tap water (at 54° to 64°F [12° to 18°C]) has proven to be more effective than ice in reducing the amount of tissue damage following a burn.[120] In fact, the application of ice caused more tissue damage to burned tissue than no treatment at all.[120,121]

HYDROTHERAPY

Hydrotherapy, in the form of whirlpools, has been a mainstay in wound care for decades. Initially used in burn units to assist with removing adhered dressings, whirlpool use expanded in some clinics to cleaning not just burns but also the majority of wounds. Since that time, the use of whirlpools has been curtailed due to concerns over cross-contamination and the development of new methods to deliver hydrotherapy to wounds (Fig. 15-9). Even in burn units, the use of immersion whirlpool is not as common as it once was. A 2010 survey of burn centers found that 83% still used hydrotherapy but only 45% used immersion hydrotherapy either exclusively or in combination with shower cart hydrotherapy, compared to 81% of centers in 1990.[122] The most common reasons listed by survey respondents for using hydrotherapy in burn care were to wash off exudates, facilitate dressing changes, and to wash off topical creams. Only 43.6% of respondents stated wound debridement as the primary indication for hydrotherapy. A literature search using the terms *wound, whirlpool,* and *hydrotherapy* found no studies comparing the use of traditional whirlpool to another form of wound treatment published within the past 5 years.

Despite the reasons it is used in practice, as discussed above, hydrotherapy in wound healing is primarily used for mechanical debridement (Table 15-4). Loosely adhered necrotic tissue and slough, exudate, dirt, and contaminants in and around the wound are removed by the movement of the water.[123] Use of whirlpools for wound cleansing has several proposed benefits. First is the reduction in pain associated with dressing removal because it soaks the bandage and allows it to come off slowly. The warm water in a whirlpool can also improve blood flow to the immersed area, potentially improving healing. In the past, whirlpool use in addition to standard dressing changes showed improved wound healing in stage III and IV pressure ulcers compared to standard dressing changes alone.[124] However, when a RevMan analysis was performed on this data, no significant differences were found.[125] The mechanisms by which whirlpool therapy improves wound healing have not been analyzed in any form of a controlled study. Proposed methods

Fig 15 ■ 9 Whirlpool tank with agitator. *(Courtesy of Whitehall, City of Industry, CA.)*

TABLE 15–4. Hydrotherapy Indications

Whirlpool	Pulsed Lavage
• Stage III or IV pressure ulcers with heavy amounts of necrotic tissue • Burns • Removal of adherent dressings • Greater than 50% necrotic tissue	Variety of wound types, including: • Pressure ulcers • Diabetic foot ulcers • Venous insufficiency ulcers • Deep or tunneling wounds • Infected surgical sites • Heavily contaminated wounds • Burns • Multiple wounds

include reducing bacterial colonization and increasing the tissue temperature due to the warm water.

Despite the proposed benefits, there are also many possible side effects associated with whirlpool therapy. This modality has the potential to transmit bacteria from one patient to another as well as from an infected wound to a noninfected wound on the same patient if both wounds are immersed in water.[126] This risk can be reduced by cleansing the whirlpool effectively, which can be time-consuming, and by adding antimicrobial agents to the water, which can be hazardous to healthy tissues.[127–129] Prolonged immersion in water also has the potential to macerate the periwound skin and increase edema when positioned in a dependent position. This is particularly problematic for venous ulcers of the lower limb.[130] Another issue with whirlpools is that it is difficult to control the pressure of the water at the wound's surface. Whirlpool is typically used for 20 to 30 minutes, three to five times per week. Its use should be discontinued when the wound has a healthy bed of granulation.

In response to many of the problems inherent in whirlpool therapy, newer, more directed approaches to debridement have been developed. Pulsed lavage or pulsed lavage with suction (PLWS) has become increasingly popular as alternatives to whirlpool therapy for the treatment of open wounds (Box 15-2). Pulsed lavage involves irrigating the wound (typically with saline) at a set pressure (Fig. 15-10).

The recommended water pressure range is 4 to 15 psi. Pressures less than this have proved to be ineffective for removing debris. Pressures higher than this range are implicated in tissue damage and possibly a spread of bacteria.[131] Pulsed lavage has been shown to be effective for debridement, and it increases the rate of granulation tissue formation compared to whirlpool.[132] PLWS is not without risk of contamination because aerosolization of particles may occur, but this risk can be minimized by treating the patient in a private room and using a plastic shield to protect the area. Different attachments are also available to assist with the treatment of open tracts and tunnels. As with whirlpool, the use of pulsed lavage should be discontinued when a clean wound is achieved. Another form of hydrotherapy, referred to as *hydrosurgery,* involves a high-velocity water jet that rapidly debrides tissue and is often used in place of sharp debridement. Further discussion on that modality is beyond the scope of this chapter.

Box 15 ■ 2 Proper Use of Pulsed Lavage

- Coordinate therapy with patient's medication schedule to maximize comfort.
- Position the patient comfortably with treatment area exposed and easily accessible.
- Assemble sterile, single-patient pulsed lavage unit, including irrigants, suction canister, and lavage gun. *Note:* Warming irrigants under warm running water prior to therapy may increase comfort and reduce vasoconstriction in the area to be treated.
- Ensure that the patient is appropriately draped and that the therapist is wearing personal protective equipment (i.e., gloves, mask, gown) to avoid backsplash. The lavage gun should be covered with a plastic barrier to reduce aerosolization.
- Treat wound at 4 to 15 psi to remove nonviable material.
- Treatment duration will vary based on wound size, patient tolerance, and amount of nonviable tissue.
- At the completion of treatment, the unit should be disposed of in accordance with facility policy; in some cases, the device may be saved for subsequent use on the same patient.

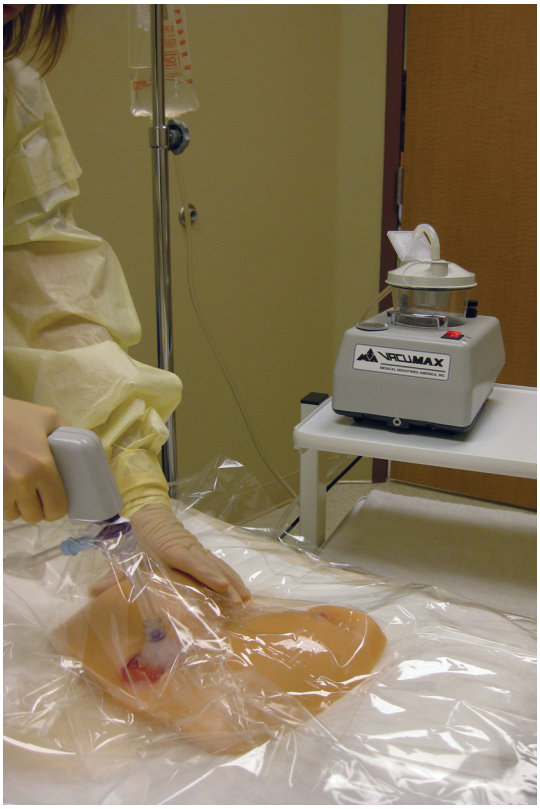


Fig 15 ■ 10 Simulated use of pulsed lavage with suction on a sacral ulcer. Note the plastic splash protector. *(Courtesy of Davol, Inc., Cranston, RI.)*

Clinical Controversy

Opinions on the use of whirlpool therapy are varied. Many wound care clinicians feel there are very rare instances in which a whirlpool is appropriate, given other methods to cleanse the wound, while other clinicians use whirlpool at the majority of patient visits (Box 15-3; Tables 15-5, 15-6, and 15-7).

Box 15 ■ 3 Proper Use of Whirlpool

- Assess patient and patient's record for any contraindications or concerns regarding whirlpool therapy.
- Coordinate therapy with patient's medication schedule to maximize comfort.
- Set the water temperature to 96°F to 102°F (35.5°C to 39°C) for most wound care applications. For total immersion, the temperature should be at the lower end of the range; higher temperatures are okay for local immersion.
- Remove clothing from the area to be treated and immerse patient in water.
- Turn on agitators after patient is safely positioned.
- Typical treatments last approximately 20 minutes.
- The whirlpool is cleansed and disinfected after each use.

TABLE 15–5. Contraindications/Precautions for Full-Body Immersion in Whirlpool Therapy

Contraindication	Rationale
Cardiac dysfunction	Inability to regulate temperature/dissipate heat if submerged in warm water
Bowel incontinence	Contamination of wound and whirlpool
Impaired consciousness	Risk of drowning
Severe peripheral vascular disease	Inability to dissipate heat may lead to tissue damage
Infectious conditions that can spread in water	Risk of contamination
Multiple sclerosis	Hot water contraindicated
Uncontrolled bleeding	Risk of hemorrhage
Pregnant women	Risk of injury to fetus with high temperatures
Granulating wounds	No need for debridement
Precautions	
Confusion	Risk of drowning
Urinary incontinence	Contamination
Hydrophobia	
Certain medications	Reduced tolerance to heat

TABLE 15–6. Contraindications/Precautions for Local Immersion in Whirlpool Therapy

Contraindication	Rationale
Uncontrolled bleeding	Increases blood flow to area; risk of hemorrhage
Maceration	Weakens skin; may increase risk of skin infection
Venous insufficiency ulcers	Prolonged dependent positions and warm environment will exacerbate edema
Granulating wounds	No need for debridement
Precautions	
Infections that can spread in water	Risk of contamination
Impaired cognition	Potential injury from falling; contact with turbine
Recent skin grafts	Graft destruction

TABLE 15–7. Contraindications/Precautions for Pulsed Lavage

Contraindication	Rationale
Uncontrolled bleeding; blood vessels in the wound	May exacerbate the problem
Granulating wounds*	No need for debridement
Precautions	
Recent skin grafts in area	Potential to disrupt graft
Areas that cannot be visualized	Unable to determine what is being debrided

*Note: Wounds that are colonized or contaminated despite a healthy appearance would be appropriate for pulsed lavage.

LASER THERAPY

The use of lasers in tissue healing remains controversial because of conflicting results. Factors that are thought to contribute to the discrepancy between studies with positive and negative findings include the duration and frequency of laser therapy, the wavelength of the laser, and the species of animal being studied. Attempting to compare the effectiveness of laser therapy when laser parameters are not standardized from one study to the next would be akin to assessing the effectiveness of exercise without standardizing the mode, duration, intensity, and frequency. Varying results in animal studies appear to be related to how loose the skin is on different types of animals, with looser-skinned animals such as cows

and mice responding better than tight-skinned animals such as pigs.[133]

On the positive side, a meta-analysis performed by Enwemeka et al[134] examined the effects of low-level laser therapy (LLLT) on in vivo tissue repair in animals and humans. Their results revealed a positive effect of laser therapy on tissue repair and revealed specific indices of tissue repair, including collagen formation, rate of healing, tensile stress, strength, and overall flap survival. Several other studies on diabetic rats, which were not included in Enwemeka's analysis, produced similar results. Wound collagen content was stimulated and a reduction in wound area was noted with laser therapy compared to control groups.[135,136] In the inflammatory stage of healing, reduced edema, reduced number of inflammatory cells, increased phagocytosis by white blood cells, and proliferation of inflammatory cells have all been attributed to laser therapy.[137–139] The effects continued to be positive in the proliferative phase with an increased number of myofibroblasts, collagen deposition, and epithelial cells observed.[139,140]

In addition to the multitude of studies that have demonstrated positive outcomes using LLLT, there are many studies that did not find an effect of laser therapy.[141–147] A literature review by Posten et al[148] pointed out many positive effects that have been observed with laser therapy but concluded that there is not an adequate body of literature to support widespread use of lasers in wound healing. Flemming and Cullum[149] performed a Cochrane review that examined laser's effects on venous ulcer healing and did not find any benefit; however, this review was withdrawn in July 2014 in order to produce an updated appraisal.

Currently, there appears to be a strong indication in the literature that laser may be effective for wound healing, but research has yet to determine the most beneficial parameters for laser therapy. Positive results have been demonstrated with different forms of laser, including helium neon (632.8 nm) and gallium arsenide (904 nm), with helium neon having the largest body of evidence.[150] Recommendations for parameters for the use of LLLT for a variety of conditions are presented in Chapter 6. Table 15-8 lists contraindications and precautions for LLLT.

TABLE 15–8. Contraindications to Low-Level Laser Therapy

- Exposure to eyes
- Over cancer
- Pregnancy

NEGATIVE-PRESSURE WOUND THERAPY

Negative-pressure wound therapy (NPWT) involves the application of subatmospheric pressure to wound tissues. The concept of using negative pressure is not new. It has been used in drains to remove body fluids for much of the last century.[151] Negative pressure has become a mainstay in the management of large and complex wounds since its introduction (Tables 15-9 and 15-10).

The wound care practitioner has multiple options when deciding which NPWT device to use. Each has three basic components: a power source, a wound dressing, and a mechanism to collect drainage. Line-powered

TABLE 15–9. Indications for Negative-Pressure Wound Therapy[157]

Acute Wounds	Chronic Wounds
• Traumatic and surgical wounds • Dehisced wounds • Skin grafts and graft substitutes • Fasciotomy • Partial thickness burns • Flap salvage	• Stage III and IV pressure ulcers • Diabetic foot ulcers • Arterial ulcers (post-revascularization) and venous ulcers

TABLE 15–10. Contraindications and Precautions for Negative-Pressure Wound Therapy[158]

Contraindications	Precautions
• Exposed vital organs • Inadequately debrided wounds • Untreated osteomyelitis or sepsis in the vicinity of the wound • Presence of untreated coagulopathy • Necrotic tissue with eschar • Malignancy in the wound • Non-enteric and unexplored fistulas • Allergy to any component of the device	• Active bleeding • Anticoagulant medications • Exposed blood vessels

devices designed for inpatient settings tend to be the largest and can contain the most drainage while battery-powered and mechanical spring devices have the advantage of increased portability but do not hold as much fluid. The power source, either battery or line power or the recoil from a spring, creates suction that pulls fluid from the wound into a disposable collection canister or, in some cases, into the absorbent dressing. The dressing consists of a contact layer that is in intimate contact with the wound surface and an occlusive dressing on top in order to create a vacuum seal. The contact layer is either a foam or gauze dressing, depending on which device is being used, and is covered with an occlusive film or hydrocolloid dressing, also dependent upon the device in question. Dressings are commonly left in place for several days, often longer following skin grafting, depending on the amount of drainage. Additionally, some of the devices allow for an intermittent setting (e.g., 5 minutes on and 2 minutes off), which has been shown to increase blood flow compared to continuous therapy but is not used in many cases because it can be difficult to maintain a vacuum seal.[152]

Clinical Controversy

A major difference between NPWT devices is the contact layer, which can be foam, gauze, or absorbent dressings. Controversy exists regarding the proposed risks and benefits associated with using different materials with negative pressure.

The first of the subatmospheric devices designed specifically for wound care was the VAC device (Kinetic Concepts, Inc,. San Antonio, TX). As a result, a large proportion of negative-pressure studies were performed with this device (Fig. 15-11). Based on the outcomes of VAC therapy, several proposed mechanisms of action have developed, including removal of wound exudate and bioburden, edema reduction, improved blood flow, and a promotion of fibroblasts in the wound.[153] Although all of these may contribute to wound healing, microdeformation is thought to be the primary means by which NPWT influences wound healing. Microdeformation is the mechanical deformation of cells in response to a stimulus—in this case, the application of subatmospheric pressure. This theory has been supported by studies that identified increased

Fig 15 ■ 11 The use of negative pressure wound therapy to manage a dehisced abdominal wound. *(VAC device shown courtesy of Kinetic Concepts, Inc., San Antonio, TX.)*

cellular proliferation and angiogenesis in response to mechanical stress on tissues.[154,155]

Negative-pressure wound therapy has changed the way wound care is performed in many facilities. An important benefit of negative pressure is the ability to leave the dressing in place for several days compared to twice-daily dressing changes that were typical of wet to moist dressings. The ability for a dressing to remain in place has several positive effects, including cost and time for health-care professionals to change the dressing and, most importantly, less pain for the patient due to the reduced frequency of dressing changes.[156]

Key Point! Regardless of which NPWT device is used, it is important to protect the periwound skin from irritation. This can be accomplished by treating the skin with a skin prep wipe, bordering the wound margins with a dressing, and always ensuring that the contact layer is cut to fit the wound rather than overlapping intact skin.

Evidence has demonstrated positive clinical outcomes with NPWT, including improved healing times, shorter hospital stays, reduced infection rates, and increased survival of flaps and grafts. NPWT is typically not intended to be used until complete wound closure; rather, it is used as a method to prepare the wound bed for definitive closure.[157] Improvements to the early forms of NPWT now allow for a wider range of interventions with this therapy. These improvements include

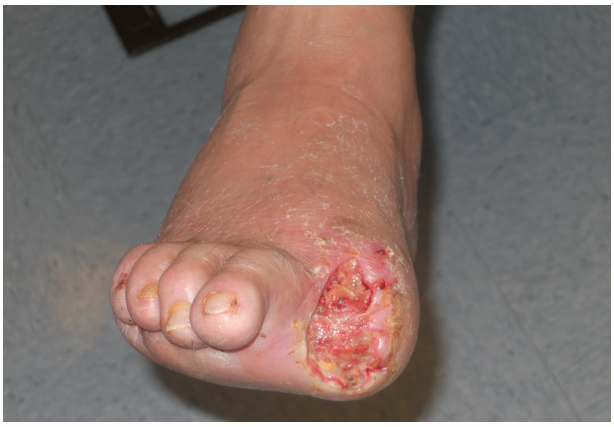
antimicrobial contact layers, disposable devices that are now available to assist with the management of closed incision sites, and machines that can infuse medications or solutions into the wound area. For a thorough review of expert guidelines pertaining to the use of negative pressure, refer to the guidelines document by Bollero et al.[157]

Clinical Controversy

Despite the moderate to strong evidence that NPWT promotes healing, the majority of the wounds treated have been acute/postsurgical, so controversy still exists concerning its effectiveness compared to other therapies in chronic wounds.

CASE STUDY 15-1 Neuropathic Ulceration

A 46-year-old patient with diabetes developed a neuropathic ulceration on the great toe, which ultimately required amputation due to infection. The ulcer presents with adherent fibrinous slough as shown in the photo. Posterior tibial and dorsalis pedis pulses are palpable, but standard care consisting of wound cleansing, moist wound bed preparation, and selective debridement have not been successful.



CLINICAL DECISION-MAKING

1. What are potential impediments to wound healing in this scenario? For each problem, are there therapeutic modalities that could improve the outcome?

ANSWER:

Impediment to Healing	Modality	Suggested Parameters
Bioburden/nonviable tissue present	Low-frequency ultrasound (LFUS)	High-intensity LFUS at 22.5–35 kHz, 100% intensity for debridement. Repeat at each visit until nonviable tissue is eradicated.
	Electrical stimulation (ES)	High-volt pulsed current (HVPC): Cathode on wound site, anode on calf; 75–150 V, 100 pps for 45–60 minutes, at least 5 days/week.
	Pulsed lavage with suction (PLWS)	Daily treatment at pressure between 4 and 15 psi. Lower end of range for initiation of treatment and higher for more bioburden/slough.
Residual infection	Ultraviolet light	UVC: Drape periwound tissue, cover eyes. Apply therapy 1 inch (2.5 cm) from wound for 30 seconds to initiate treatment. Perform at each visit until cultures are negative.
	LFUS	High-intensity LFUS: same parameters as above.

Continued

CASE STUDY 15-1 Neuropathic Ulceration—cont'd

Impediment to Healing	Modality	Suggested Parameters
		Low-intensity LFUS: 0.2–0.6 W/cm^2 delivered at 40 kHz (preprogrammed into machine); treatment duration is dependent upon wound size. Treatment frequency of at least 3 days/week.
	ES	HVPC: Anode on top of silver contact layer on wound, cathode on calf; 75–150 V, 100 pps for 45–60 minutes, at least 5 days/week.
Lack of granulation	Negative pressure wound therapy (NPWT)	Continuous therapy at 125 mm Hg to initiate therapy. Use of antimicrobial contact layer if bioburden present. Change dressing every 2–3 days.
	ES	HVPC: Cathode on wound site, anode on calf; 75–150 V, 100 pps for 45–60 minutes at least 5 days/week.

2. What assessments should be performed before initiating a therapeutic modality?

ANSWER: In addition to confirming the neuropathy and vascular status mentioned in this case, the clinician should document wound characteristics (e.g., location, size, depth, color, drainage amount and type, and odor) and periwound characteristics (e.g., callus, erythema, irritation, and tenderness).

3. What should be documented in the patient's medical record?

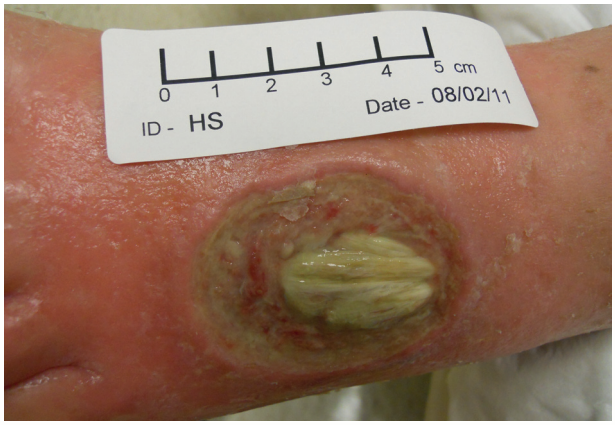
ANSWER: The specific modality and all applicable parameters, including treatment time, should be documented. Documentation should also include patient's tolerance to therapy as well as a pre- and posttreatment assessment of the wound and periwound skin.

4. What other factors will impact the choice of therapeutic modality?

ANSWER: A mainstay to the management of a neuropathic foot ulceration is offloading. Frequency of visits may be governed by the choice of offloading device to be used; for example, it would not be feasible to change a cast three times per week. Additionally, many advanced dressings are intended to be left in place for several days at a time and would not be cost-effective if removed for daily treatment. For modalities that need to be performed more often than dressings need to be changed, the clinician should feel the benefit of the modality outweighs any added cost or potential decreased effectiveness of changing to an alternate dressing or offloading device.

CASE STUDY 15-2 Vascular Insufficiency

A 61-year-old patient presents to the outpatient clinic with a chief complaint of constant pain in the left leg. She gets mild relief with pain medications and keeping the foot in a dependent position. Pain increases with leg elevation and walking. She is currently only able to walk 1 to 2 minutes with a walker before she needs to rest due to the leg pain. The wound, located on the dorsum of the foot, is approximately 15% red with a layer of yellow slough and exposed tendon in the wound bed. Wound margins are well defined. The periwound is shiny and edematous.



CASE STUDY 15-2 Vascular Insufficiency—cont'd

CLINICAL DECISION-MAKING

1. What are potential impediments to wound healing in this scenario? For each problem, are there therapeutic modalities that could improve the outcome?

ANSWER:

Impediment to Healing	Modality	Suggested Parameters
Bioburden/nonviable tissue present	Whirlpool	Not recommended in this instance. With impaired vascularity, there may be insufficient blood flow to dissipate heat, and the dependent position may exacerbate edema.
	Electrical stimulation (ES)	High-volt pulsed current (HVPC): Cathode on wound site, anode on calf; 75–150 V, 100 pps for 45–60 minutes, at least 5 days/week.
	Pulsed lavage with suction (PLWS)	Daily treatment at pressure between 4 and 15 psi. Lower end of range for initiation of treatment and higher for more bioburden/slough.
Lack of granulation	Negative-pressure wound therapy (NPWT)	Continuous therapy at 125 mm Hg to initiate therapy. Use of antimicrobial contact layer if bioburden present. Change dressing every 2–3 days.
	Low-frequency ultrasound (LFUS)	Low-intensity, LFUS at 40 kHz, 100% intensity.
Decreased vascularity	Pneumatic compression	Arterial pumps that deliver short duration (3 sec on followed by 20 sec off), high compression (120 mm Hg) therapy can be used to assist with arterial function. Compression will be applied to the foot and calf, typically for multiple hours daily. Traditional venous pumps would be contraindicated due to poor vascularity.
	ES	Sensory: Cathode on wound site, anode on calf; 75–150 V, 100 pps for 45–60 minutes, at least 5 days/week. Motor: Evidence suggests applying electrodes to lower leg, at an amplitude to elicit subtetanic contractions and a variety of pulses per second, can be effective, but this may be limited by patient tolerance.

2. What assessments should be performed prior to initiating a therapeutic modality?

ANSWER: The vascular status of this patient is of critical importance. With the amount of tissue loss depicted, this patient should undergo a complete vascular workup to determine if surgical interventions are appropriate. Clinical tests that can be performed in the clinic include ABI, pulse palpation, and capillary refill to assess the vascular status and active range of motion to assess tendon viability. Additional tests that can help determine a course of action include functional outcome measures and an assessment of claudication severity.

3. What should be documented in the patient's medical record?

ANSWER: A pre- and postintervention skin assessment should always be documented. In this case, it may be necessary to hold therapeutic modalities until further vascular testing can be performed. Results of any clinical vascular findings and functional limitations are imperative.

4. What other factors will impact the choice of therapeutic modality?

ANSWER: Failure to perform a thorough wound and vascular examination prior to initiating any therapeutic modality will unnecessarily increase the risk of complications. For example, if the underlying cause of the wound is not recognized as vascular and the clinician applies traditional pneumatic compression, it may further compromise blood flow to the area.

CASE STUDY 15-3 Tendon Injury

A 35-year-old recreational runner suffered a partial tear of his flexor hallucis longus tendon while competing in an adventure race 1 week ago. He had attempted to manage the pain and swelling with NSAIDs until seeking treatment today.

CLINICAL DECISION-MAKING

1. Based on the stage of tissue healing that the patient is currently in, which modalities may be most beneficial?
ANSWER: The patient is currently in the inflammatory phase and will likely remain there until stress on the injured tissue can be removed. In addition to immobilization of the tendon, anti-inflammatory modalities, such as cryotherapy, may be effective in reducing inflammation. Cryotherapy, whether as an ice pack, ice immersion, or ice massage, can be utilized. Because this is a superficial structure, the duration of treatment will not need to be as long as it would be for deep structures.

2. What are the important outcomes as well as adverse events that should be monitored?
ANSWER: Cold therapy is designed to reduce the edema and pain associated with inflammation. This can be effectively assessed by volumetric or girth measurements as well as visual analog scales or subjective ratings of pain for edema and pain, respectively. In this case, a baseline level of function in the form of a questionnaire may be more desirable than a physical test due to the acute nature of the injury. Most adverse events can be avoided by assessing for any vascular pathology such as Raynaud's phenomenon, cryofibrinogenemia, and cold urticaria. The skin should be inspected frequently to ensure tolerance to therapy.

3. As the inflammation subsides, are there modalities available that could continue to assist with the patient's healing process?
ANSWER: Numerous modalities could be incorporated into the treatment plan to assist with returning the patient to an optimal level of function. Thermal modalities, such as hot packs or conventional US, could be applied immediately before or during stretching activities to increase tissue extensibility. Conventional US and laser therapy may also be effective at promoting proliferation because the target area is sufficiently small and superficial to allow for adequate transfer of energy to the affected tissue.

4. What parameters would be most effective to achieve the desired effects?
ANSWER: Thermal modalities should be applied to achieve a tissue temperature above 104°F (40°C; roughly a 7.2°F [4°C] increase). Because it is often difficult to apply a hot pack to the plantar aspect of the foot, conventional US may be a good alternative in this case. As described in Chapter 4, the use of continuous wave US when used to treat an area equal to twice the effective radiating area (ERA) will take approximately 2.5 minutes to raise the tissue temperature 7.2°F (4°C) when applied at 3 MHz at 2 W/cm^2. Laser therapy has also demonstrated effectiveness in promoting healing in a variety of tissue types. For a full discussion on optimal parameters for laser therapy, refer to Chapter 6.

Documentation Tips

- Tissue area stimulated
- Waveform type (e.g., high volt, low-intensity direct current, etc.)
- Stimulation settings
  - Polarity, frequency, and pulse duration
  - Type, size, and placement of electrodes
- Purpose (e.g., epithelialization, debridement, etc.)
- Patient position
- Duration and frequency of stimulation
- Response to treatment (i.e., inspection of tissue area)
- Any skin irritation posttreatment.

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CHAPTER 16

ALTERNATIVE MODALITIES FOR PAIN AND TISSUE HEALING

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We have chosen to operationally define alternative modalities as "technologies that have become popularized for treatment of conditions involving chronic pain and delayed tissue healing." There are a number of thoughts about why these techniques may be of interest to the therapist and patient. Our patients want to get better. We want our patients to get better. For a variety of reasons, previous interventions may have been unsuccessful—thus, the search for another option. In many cases of chronic pain and delayed tissue healing, attempts may be made to maximize recovery of function at any cost. In this chapter, we discuss the use of magnet therapy, monochromatic infrared therapy (MIRE), hyperbaric oxygen therapy (HBOT), and extracorporeal shock wave therapy (ESWT). The popularity of these interventions may not parallel their published effectiveness. Magnet therapy, MIRE, and HBOT are within the scope of practice of nonphysician practitioners; ESWT is used by physicians and surgeons.

MAGNET THERAPY

Magnets have been used for therapeutic purposes for more than 2,000 years.[1,2] Greek healers in AD 200 used magnetic rings as a treatment for arthritis. During the emergence of complementary/alternative medicine (CAM) in the late 20th and early 21st centuries, interest has been increasing regarding the use of magnets for therapeutic benefit.[3] The use of pulsed electromagnetic fields (PEMF) for promotion of fracture healing and other orthopedic problems, such as osteoarthritis, has been commonplace during this time.[4]

Physical Principles of Magnets

Magnets are metals, such as iron, that exhibit an attractive or repulsive force. The force field around a magnet is represented in Figure 16-1. The number of lines per unit area representing the magnetic field is proportional to the magnitude of the field. The distance between two magnets will determine the amount of force exerted on each magnet. The shorter the distance between two magnets, the greater the force between them. The force one magnet exerts on another magnet can be described as the interaction between the magnetic fields of each magnet. This force can be either repulsive or attractive. When two north or two south poles are brought close together, the force is repulsive. When two opposite poles (one north and one south pole) are brought close together, the force is attractive. The direction of the magnetic field at a given point is defined as the direction that the north pole of a compass needle would point when placed at that point. The pole of a freely suspended magnet that points toward the north is the north pole. The other pole, which points toward the south, is called the south pole. The earth acts as a huge magnet with a magnetic field and north and south poles.[5] For thousands of years, voyagers have used compasses for navigation that are attracted to the earth's north pole by the earth's magnetic field.

Key Point! Magnets used for therapeutic effects are known as static or permanent magnets. The strength of the magnetic field produced by a permanent magnet is expressed in units known as teslas (T) or gauss (G). The relationship of tesla to gauss is 1 G = 10^{-4} T.

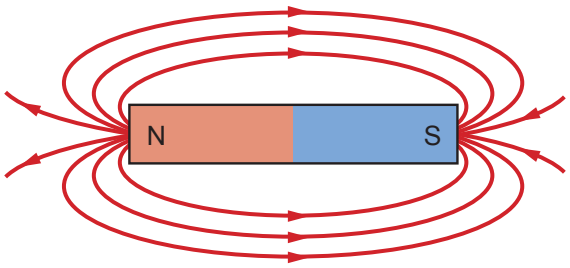

Fig 16 ■ 1 Magnetic field lines drawn around a bar magnet having north (N) and south (S) poles.

The magnetic field of the earth at its surface is about 0.5 G—or 0.5 × 10^{-4} T.[5] Most magnets marketed for therapeutic effects have an advertised strength of 500 to 1,000 G compared to the 15,000 G produced by magnetic resonance imaging (MRI) devices.[6] Blechman et al[7] used a Gaussmeter to determine the field flux density in gauss for magnets marketed for medical use. Field flux density contributes to the determination of the magnetic field's tissue penetration, which can be considered its "effective dosage." The gauss listed by the distributors or suppliers of four out of five magnets differed significantly from the measurements taken with the Gaussmeter.

Magnets applied to the skin (or in close proximity to the skin) are made from ferromagnetic metals, usually iron or a combination of iron and other metals.[5] These magnets are known as "static" magnets because the magnetic field created by these metals is continuous.

Key Point! All magnets have two poles. However, the application of magnets for therapeutic purposes can be unipolar or bipolar. Unipolar magnets are arranged so that only one pole is facing or touching the skin, usually the north pole. The south pole of the magnet is facing away from the skin. Bipolar magnets are arranged so that both the north and south poles are facing or in contact with the skin, usually using multiple magnets.[6,8]

Pulsed Magnetic Fields

Movement of charges in an electric current will produce a magnetic field. The magnetic field produced by a current in a straight wire is represented by lines in the form of circles with the wire at the center (Fig. 16-2).[5] Electrical devices available to practitioners today can create strong magnetic fields around a coil. The patient's body part is placed within the magnetic field. Other devices use weaker magnetic fields, and a flat coil is placed in contact with the body part. Therapeutic application of a PEMF is also known as *magnetotherapy*. The physiological effect of pulsed magnetic fields is likely secondary to induction of currents in the tissues exposed to the fields, resulting in movement of ions across cell membranes and stimulation of DNA transcription.[4,9] See Chapter 6 for more information about pulsed electromagnetic fields.

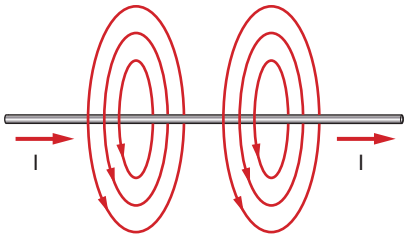

Fig 16 ■2 Magnetic fields generated around a live electric wire. Arrows represent the direction of current.

Proposed Physiological Effects of Static Magnets

Speculation about the physiological effects of static magnets has existed for many years. Most manufacturers' claims for the therapeutic effects of magnets have not been substantiated.[6,9–13] However, an abundance of experimental and clinical data demonstrates that magnetic fields may have a profound effect on biological tissues at the cellular level. Magnetic fields are capable of inducing selective changes in the microenvironment around and within the cell, including the cell membrane. Modifications of cellular activity may occur with exposure of the cell to magnetic fields, and these modifications may correct selected pathological states. These modifications appear to strongly depend on the parameters of the applied magnetic field. Practitioners need to consider that the known effects of magnetic fields on the cellular level may not translate into therapeutic effects at the clinical level.[14]

Review of the Literature on Static Magnet Therapy

Table 16-1 lists a sample of relevant literature on static magnet therapy. An analysis of these studies allows the practitioner to conclude that magnets may be beneficial for treatment of the following conditions:

- Reduction of pain associated with peripheral neuropathy[15]
- Reduction of pain in the knees for patients with rheumatoid arthritis[16]
- Relief of pain and stiffness in the neck and shoulders[17]
- Reduction of pain intensity and improved sleep in patients who have fibromyalgia.[18,19]

TABLE 16–1. Selected Literature Review: Magnet Therapy

Author and Year	Patient Population	Intervention(s)	Results	Conclusions
Wallis et al, 2012[12]	Older women with urinary incontinence	Subjects wore undergarments either incorporating 15 static magnets (800–1,200 G) or placebo inert discs for 12 hours per day for 3 months.	No statistically significant difference between groups for any outcome measures.	Static magnets are not beneficial for women with urinary incontinence.
Kanai et al, 2011[17]	Patients with neck and shoulder pain or stiffness	Patients wore either a strontium ferrite magnet around the neck (55 mT) or a nonmagnetic device for 7 days.	Significant relief of pain and increase in surface and deep body temperatures in the group that wore the magnet.	A magnetotherapeutic device worn around the neck for 7 days may help decrease neck and shoulder pain and increase tissue temperatures.
Curtis et al, 2011[79]	Sixty-five adults with moderate to severe migraines	Randomized to 8 weekly treatments of craniosacral therapy, low-strength static bipolar magnets (300–500 G), or inert magnets.	Subjects who received active or inert magnet therapy did not achieve comparable level of credibility and expectancy compared to craniosacral therapy.	Low-strength static magnet therapy is not an effective attention control intervention for patients with migraines.

Continued

TABLE 16–1. Selected Literature Review: Magnet Therapy—cont'd

Author and Year	Patient Population	Intervention(s)	Results	Conclusions
Richmond et al, 2009[11]	Patients with osteoarthritis (OA) ($n = 45$)	Subjects wore a wrist strap for 16 weeks containing one of the following: 2 neodymium magnets Attenuated magnets (placebo) Demagnetized discs (dummy) Copper bracelet (placebo)	Generally ineffective for managing pain, stiffness, and physical function in patients with OA. No adverse effects.	Therapeutic effects attributed to magnets are likely secondary to nonspecific placebo effects; however, magnets may provide patients with hope.
Colbert et al, 2008[80]	A literature review of magnets applied to acupuncture points as therapy	Forty-two studies reviewed.	Thirty-seven studies (88%) reported a therapeutic benefit. Only adverse reactions were exacerbation of hot flashes and skin irritation from adhesives.	Overall poor quality of controlled trials reviewed precludes any evidence-based treatment recommendations.
Pittler et al, 2007[10]	Systematic review and meta-analysis of randomized trials of studies on static magnets	Nine studies qualified for inclusion in this review.	Evidence does not support use of static magnets for pain relief.	Static magnets cannot be recommended as an effective treatment for pain, although evidence is insufficient to exclude a clinically important benefit for people with osteoarthritis.
Harlow et al, 2004[21]	OA of hip or knee ($n = 194$)	Wore standard strength, weak, or dummy magnetic bracelets for 12 weeks.	Pain ↓ more in subjects who wore standard-strength bracelets.	Uncertain whether results secondary to effects of magnets or placebo effect.
Weintraub et al, 2003[15]	Diabetic peripheral neuropathy ($n = 375$)	Wore magnetized insoles or sham continuously for 4 months.	Significant decrease in symptoms during months 3 and 4 in group that wore magnetized insoles.	Static magnetic fields penetrate up to 20 mm and analgesia is achieved over time.
Hinman et al, 2002[23]	Chronic knee pain ($n = 43$)	Wore pads with magnets or placebos on knees for 2 weeks.	Significant improvement in pain, physical function, and gait speed in magnet group compared to placebo group.	Suggests that application of static magnets over painful knee joints reduces pain and enhances functional movement.
Martel et al, 2002[22]	Young, healthy males ($n = 20$)	Wore static magnets or placebos on anterior surface of forearms for 30 minutes.	Average blood flow not significantly different between groups.	Static magnets do not result in significant alterations in resting blood flow.

TABLE 16–1. Selected Literature Review: Magnet Therapy—cont'd

Author and Year	Patient Population	Intervention(s)	Results	Conclusions
Hinman, 2002[23]	Adults without symptoms of cardiovascular disease or cardiac problems (n =75)	Subjects lay on mattress with 42 magnets or a placebo for 15 minutes.	No "clinically meaningful" changes in heart rate or blood pressure.	Results support the safe use of unipolar static magnetic fields < 1,000 G.
Alfano et al, 2001[18]	Patients with fibromyalgia	Subjects slept on magnetic sleep pads for 6 months, randomized into 3 groups: 1. Negative-polarity magnetic sleep pads 2. Variable polarity magnetic sleep pads 3. Sham (inactive magnets)	Significant difference among groups in pain intensity ($p = 0.03$) at 6 months; all other outcome measures not significant.	Static magnetic fields may help decrease pain intensity in patients with fibromyalgia. Note: Success of patient blinding was not assessed; therefore, patients may have been able to discern true from sham magnets.
Sweeney et al, 2001[24]	Healthy student volunteers (n =13)	Single 5 × 11 cm magnet or sham applied to anterior thigh for 60 minutes.	No difference in skin or intramuscular temperatures measured at 20, 40, and 60 minutes of application.	Flexible therapeutic magnets were not effective for increasing skin or deep temperatures.
Segal et al, 2001[16]	Patients with rheumatoid arthritis and persistent knee pain (n = 64)	Four quadripolar static magnet devices (MagnaBloc) or control devices with one magnet were attached around the knees and worn for 1 week.	Average changes in pain intensity were significant for both groups; not significant between groups for percentage pain reduction.	Both MagnaBloc and control devices were significantly efficacious in decreasing pain from baseline.
Collacott et al, 2000[20]	Chronic low back pain (n = 20), mean duration 19 years	Bipolar permanent magnets and sham applied to the low back for total of 18 hours for both groups (1 week with magnets and 1 week with shams).	No significant differences between real and sham magnets for pain and lumbosacral spine ROM.	Application of one variety of permanent magnet had no effect on a small group of subjects with chronic low back pain.
Tis et al, 2000[25]	Knee surgery or knee injury in past 6 months (n = 20)	Application of pad with seven bar magnets, placebo magnet, or no magnet to anterior thigh for 10 minutes before isokinetic exercise.	No significant difference in isokinetic force production among all three groups.	Application of magnetic pad to quadriceps muscle does not appear to increase isokinetic force production.

Evidence to support therapeutic effects of static magnets is insufficient to recommend their use for the following:

- Chronic low back pain[20]
- Osteoarthritis[10,11,21]
- Cardiovascular effects (e.g., heart rate, blood pressure, blood flow)[22,23]
- Skin or deep tissue temperature[24]
- Muscle force production[25]
- Urinary incontinence[12]

Clinical Applications of Magnet Therapy

Static magnets are readily available and relatively inexpensive. Advertisements promoting the therapeutic effect of magnets are ubiquitous. Magnets can be incorporated into garments and supports and worn throughout the day without the need for direct clinician supervision (Fig. 16-3). Indications for magnet therapy may include pain control for patients with post-polio syndrome[26] and for patients with pain associated with peripheral neuropathy.[15,27,28] A report of the Guideline Development Subcommittee of the American Academy of Neurology noted that magnetic therapy may help decrease fatigue for patients with multiple sclerosis; however, there is no beneficial effect on depression.[29] Patients with pain in the knees associated with rheumatoid arthritis may also benefit from magnet therapy.[16] However, the effectiveness of long-term use of magnets for these applications has not been determined. Spielholz[30] reviewed evidence for use of static magnets for heel pain, delayed-onset muscle soreness, back pain, knee pain, chronic pelvic pain, fibromyalgia, and wound healing, and concluded "there is little objective evidence to support the claims of pain relief that can be attributed specifically to using static magnets."

Precautions and contraindications for static magnets are not certain at this time; however, some possible precautions and contraindications are listed in Box 16-1. Adverse effects from exposure to static magnets are unlikely because of the relatively weak magnetic fields generated by these devices (usually less than 1,000 G).

Fig 16 ■3 Elastic wraps with magnets (A and B). (A) Note the pouches containing the magnets. The wrap on the right has had the pouches opened to show the magnets.

Box 16 ■ 1 Magnet Therapy: Precautions and Contraindications

- Do not use magnets near cardiac pacemakers, defibrillators, insulin pumps, or any other internal or external electronic devices because magnets may interfere with the control of these devices.
- Do not place magnets over the low back or pelvic region of a woman during pregnancy because the effect on the fetus is unknown.
- Avoid placing magnets over or near cancerous tissues to prevent the possibility of facilitating growth or spread of the cancer by magnetic fields.
- A protective barrier between the skin and the magnet may help to prevent skin irritation.
- There are some reports of dizziness, light-headedness, discomfort, and malaise after exposure to magnetic fields, so monitor patients closely during and after treatment.

MONOCHROMATIC INFRARED PHOTO ENERGY

The use of infrared radiation lamps for heating of biological tissues was a common clinical practice during the last century.[31] Today, the use of infrared lamps as superficial heating agents has largely been replaced by hot packs because of the increased risk of burns from the constant heating effect of these lamps. The emergence of devices that provide monochromatic infrared energy emitted from diodes has reintroduced the use of infrared radiation to clinical practice. These devices, known as *monochromatic infrared energy,* or *MIRE,* are thought to exert their clinical effect by influencing vasoactive mediators rather than by heating tissues.

Physical Principles of MIRE

MIRE devices produce energy waves from the near-infrared portion of the electromagnetic spectrum. This portion of the spectrum has wavelengths ranging from 780 to 1,500 nanometers (nm), which are the infrared wavelengths closest to visible light. Devices that produce near-infrared radiation are known as *luminous* because the light produced is visible.[6] MIRE devices produce monochromatic near-infrared photo energy at a wavelength of 890 nm from a series of 60 gallium aluminum arsenide diodes on a flexible pad.[32,33] Figure 16-4 shows a MIRE device. Horwitz et al[34] used a photodiode to measure the uniform average power over a MIRE pad

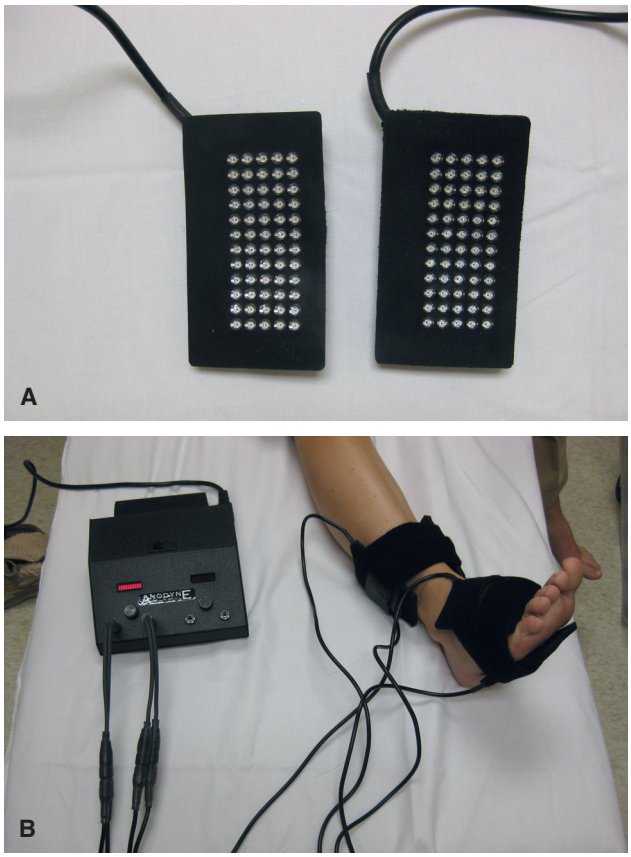

Fig 16 ■ 4 (A) Pad placement for MIRE device and (B) with diode arrays on flexible pads.

with a surface of 22.5 cm^2 and found that the diode array on the pad produced 9 mW/cm^2. Each pad had a total energy density of 43.2 J/cm^2 per 30-minute treatment.

Proposed Physiological Effects of MIRE

Infrared radiation can stimulate the production of nitric oxide (NO), which is a potent vasodilator, resulting in enhanced circulation, tissue oxygenation, nutrient delivery to tissues, removal of waste products, and relaxation of smooth muscle cells.[35] NO may play a role in pain reduction through its effects on the microenvironments of nerve and vascular pathways. The physiological effects of MIRE on biological tissue are believed to occur primarily by photochemical reactions, such as the stimulation of NO production, rather than by thermal effects.

Human blood lymphocytes irradiated with MIRE had an increased level of ATP in cells.[36] These physiological effects may explain the enhancement of wound healing in patients treated with MIRE.[34] Promotion of

circulation with resultant increased oxygenation of tissues treated by MIRE may result in promotion of nerve growth in patients with peripheral neuropathy. Several studies have reported improved sensation in patients with peripheral neuropathy who were treated with MIRE.[32,37–40] Leonard et al[33] found that MIRE treatments were more effective in improving sensation in patients with peripheral neuropathy than in patients who received placebo pads that emitted a comparable thermal effect. Horwitz et al[34] reported that application of MIRE to the skin for 30 minutes increased plasma NO. However, in a randomized, double-blind, placebo-controlled study, Clifft et al[41] found that 30 minutes of active MIRE applied 3 days per week for 4 weeks was no more effective than placebo MIRE in increasing sensation in 39 subjects with diabetic peripheral neuropathy. Lavery et al[42] randomly assigned 69 patients with diabetes to receive either MIRE or a sham device for 40 minutes every day for 90 days. They found no significant differences between groups in outcome variables that included nerve conduction velocity, vibration perception threshold, Semmes-Weinstein monofilament testing, Michigan Neuropathy Screening Instrument, visual analogue scale for pain, and a neuropathy-specific quality-of-life scale.

Review of the Literature on MIRE

Table 16-2 provides a review of the relevant literature on MIRE. An analysis of these studies allows the practitioner to conclude that MIRE *may* be beneficial for treatment of the following conditions:

- Peripheral neuropathy,[32,33,37–40,43,44] although two studies failed to show improvement[41,42]
- Chronic venous ulcers, diabetic ulcers and wounds, and ulcers associated with scleroderma[34,45]

MIRE has not been shown to be beneficial for patients with osteoarthritis of the knees.[35,46]

Key Point! Effective applications of MIRE for peripheral neuropathy have used wavelengths of 890 nm for daily 30- to 40-minute treatments.

TABLE 16–2. Selected Literature Review: Monochromatic Infrared Energy

Author and Year	Patient Population	Intervention(s)	Results	Conclusions
He et al, 2013[45]	15 diabetic rats	MIRE to skin wounds	MIRE appeared to promote collagen deposition in early stage of wound healing.	Overall wound healing not significantly different from control group.
Ammar, 2012[44]	35 patients with peripheral neuropathy randomly assigned to 2 groups	1. MIRE, exercise, and balance 2. Exercise, balance, no MIRE	Significant improvements in pain, sensation, and balance in MIRE group.	MIRE effective in improving sensation, balance, and decreasing pain in patients with peripheral neuropathy.
Hsieh et al, 2012[35]	73 subjects with knee osteoarthritis randomly assigned into 2 groups	1. MIRE for six 40-minute treatments for 2 weeks 2. Placebo MIRE (as above)	No significant differences between groups for 7 outcome questionnaires.	MIRE is not effective in improving outcomes for patients with knee osteoarthritis.
Hsieh et al, 2012[46]	71 subjects with knee osteoarthritis randomized into 2 groups	1. One session of MIRE for 40 minutes 2. One session of 40 minutes of placebo	No increase in knee arterial blood flow velocity, heart rate, or blood pressure between groups. No detrimental effects observed.	One 40-minute session of MIRE had no effect on blood flow, heart rate, or blood pressure in patients with knee osteoarthritis.

TABLE 16–2. Selected Literature Review: Monochromatic Infrared Energy—cont'd

Author and Year	Patient Population	Intervention(s)	Results	Conclusions
Lavery et al, 2008[42]	69 patients with diabetes randomized into 2 groups	1. Active MIRE every day for 40 minutes for 90 days 2. Sham MIRE (as above)	No significant difference between groups for the following variables: nerve conduction velocity, vibration perception, Semmes-Weinstein monofilaments, Michigan Neuropathy Screening Instrument, VAS, and neuropathy-specific.	There is a strong placebo effect associated with MIRE; both experimental and sham groups had improvement in outcomes.
Harkless et al, 2006[43]	Review of medical records of 2,239 of mostly Medicare-aged community-dwelling patients with peripheral neuropathy	Received MIRE as part of their medical intervention	The number of insensate sites on the feet improved by 66%; 53% of patients no longer had loss of protective sensation; pain reduced by 67%.	MIRE was associated with significant improvement in foot sensation and neuropathic pain.
Clifft et al, 2005[41]	Diabetic peripheral neuropathy ($n = 39$)	Active or placebo MIRE 3x/week for 4 weeks	No significant difference between subjects who received active or placebo MIRE.	MIRE may not be any more effective than placebo in improving plantar sensation.
DeLellis et al, 2005[39]	Medical records of 1,047 patients with peripheral neuropathy	Association between treatment with MIRE and increased foot sensitivity	71% decrease in insensate sites on both feet; only 43.9% of patients had persistent lack of protective sensation.	MIRE seems to be associated with significant clinical improvement in foot sensation.
Powell et al, 2004[40]	Diabetic peripheral neuropathy, over age 64 ($n = 68$)	Questionnaire to determine relationship between improved foot sensitivity following MIRE and incidence of new foot wounds	One out of 68 patients who completed the questionnaire developed a new foot wound.	Improved foot sensitivity following MIRE treatments appears to be associated with a lower incidence of new foot wounds in patients with diabetes.
Kochman, 2004[37]	Diabetic peripheral neuropathy ($n = 27$), polyneuropathy secondary to alcohol abuse ($n = 6$); decreased sensation secondary to peripheral vascular disease ($n = 5$)	MIRE daily for 30 to 40 minutes using two pads, one each placed medially and laterally on lower leg; no control group	All patients had restoration of protective sensation measured by SWME and significantly higher Tinetti scores, reduced Tinetti fall risk category by one level, and average falls per patient reduced by 93% for 3 months after treatment.	Comprehensive therapy intervention that includes MIRE has potential to improve sensation and balance and to decrease fall frequency.

Continued

TABLE 16–2. Selected Literature Review: Monochromatic Infrared Energy—cont'd

Author and Year	Patient Population	Intervention(s)	Results	Conclusions
Leonard et al, 2004[33]	Diabetic peripheral neuropathy ($n = 27$)	Subjects received 12 MIRE treatments to one leg and sham devices (created heat but no IR) to the other leg	Significant decrease in average sites insensitive to 5.07 SWM in subjects who had not progressed to profound sensory loss (almost 50% improvement in restoration of protective sensation); substantial improvement in self-reported balance after six treatments.	MIRE treatments improve sensation in the feet of subjects with diabetic peripheral neuropathy, improve balance, and reduce pain.
Kochman et al, 2002[32]	Diabetic peripheral neuropathy ($n = 49$)	MIRE pads on foot and lower leg; total of four pads with diode arrays, 30 minutes × 12 treatments; no control group	85% improvement in sensory perception documented by Semmes-Weinstein monofilaments.	Results suggest that in an outpatient setting MIRE consistently has the effect of improving neural function in patients with diabetes.
Horwitz et al, 1999[34]	Lower extremity venous ulcers ($n = 3$), diabetes and wound dehiscence ($n = 1$), scleroderma with an ulcer on the hand ($n = 1$)	MIRE at home for 30 minutes, frequency of treatment varied; one subject with ulcer treated in clinic once per week initially, then every other week; no control group	All subjects with ulcers had healing of ulcers; subject with wound dehiscence had complete closure of wound; subject with scleroderma had healing of ulcer.	Additional research is needed to show whether MIRE is independently responsible for wound healing in these patients.

IR = infrared; SWME = Semmes Weinstein monofilament examination; VAS = visual analog scale.

Clinical Applications of MIRE

The U.S. Food and Drug Administration (FDA) has approved the use of MIRE for enhancement of circulation and reduction of pain.[34] Currently, clinical devices are marketed for use by inpatient or outpatient facilities and portable devices for at-home use. The wavelength of these devices is set at 890 nm. The flexible pads containing the diode arrays are placed in contact with the skin. Placement of pads on the skin allows for hands-free application of treatment—an advantage over low-level laser, which requires hands-on treatments. When placing over an open wound, a transparent dressing, such as OPSITE, can be placed over the wound to prevent contamination.[34] Most of the success reported with MIRE has occurred with 30- to 40-minute treatments performed daily or several times a week. One study reported a topical burn on the dorsal aspect of the foot after a patient fell asleep for several hours with a MIRE device on the foot.[40] No other harmful effects of MIRE have been reported in the literature. See Box 16-2 for a list of precautions or contraindications for MIRE.

HYPERBARIC OXYGEN THERAPY

The use of hyperbaric oxygen chambers to treat divers who have decompression sickness (the "bends") has been a common practice for many years. Other uses of

Box 16 ■ 2 Monochromatic Infrared Energy: Precautions and Contraindications

- Do not perform MIRE over the low back or pelvic region of a woman during pregnancy because the effect on the fetus is unknown.
- Avoid placing MIRE pads over or near cancerous tissues to prevent the possibility of facilitating growth or spread of the cancer by the effects of the infrared radiation.
- Goggles are not required during treatment because the flexible pads containing the diode arrays block the infrared radiation from the eyes; however, avoid placing the pads over the eyes.

hyperbaric oxygen therapy (HBOT) approved by the Undersea and Hyperbaric Medical Society include air or gas embolism, carbon monoxide poisoning, enhancement of wound healing, refractory osteomyelitis, delayed radiation injury, compromised skin grafts and flaps, and thermal burns. Recently, interest has increased in the therapeutic effects of HBOT for musculoskeletal problems associated with chronic regional pain syndrome (CRPS) and fibromyalgia.[47]

Physical Principles of HBOT

HBOT involves inhalation of 100% oxygen in a pressurized hyperbaric chamber at a pressure greater than one absolute atmosphere (ATA), which is the atmospheric pressure at sea level (1 ATA = 760 mm Hg). Typically, HBOT is administered at 2 to 3 ATA for a duration of 30 to 120 minutes. HBOT can be administered to a single patient using a monoplace chamber in which oxygen pressure is raised to 100% or to more than one patient in a multiplace chamber, where each patient breathes oxygen through a mask.[48] Patients lie supine in monoplace chambers, which are typically 22 inches in diameter and constructed of Plexiglas with metal ends (Fig. 16-5). Multiplace chambers are typically found only at major medical centers and teaching institutions. These chambers can accommodate 12 or more patients who are either sitting in chairs or lying on gurneys in the chamber.[49] The cost of hyperbaric oxygen chambers can be a major barrier to patient accessibility. A monoplane chamber costs about $400,000 for purchase and installation; a multiplace chamber costs millions of dollars.[50]

Fig 16 ■ 5 A monoplace hyperbaric oxygen chamber. *(Courtesy of Perry Biomedical, Riviera Beach, FL)*

Proposed Physiological Effects of HBOT

HBOT increases the amount of physically dissolved oxygen in plasma, resulting in immediate hyperoxygenation and hyperoxia. At 2 ATA, there is a 10-fold increase in oxygen tension in blood.[51] The increase in dissolved oxygen in the plasma provides tissues with a readily available supply of oxygen so that they do not have to rely on hemoglobin-bound oxygen, which is less accessible to injured tissue secondary to sympathetically induced vasoconstriction caused by inflammation.[52] The beneficial effects of hyperoxia occur primarily through improved oxygen delivery to tissues. The result is preservation of tissue viability in ischemic areas, provided there is no occlusion of major arterial vessels. Hyperoxia promotes enhanced wound healing by increasing the diffusion distance of oxygen through tissues fluids, which improves oxygen delivery to ischemic and hypoxic tissues. Collagen production by fibroblasts is enhanced, resulting in increased collagen synthesis and angiogenesis, which promotes wound closure rates in hypoxic tissues.

Hyperoxia enhances oxygen-dependent intracellular killing mechanisms of leukocytes and prevents infection from anaerobic microorganisms through production of toxic oxygen radicals that have a direct lethal effect on these microorganisms.[51] Elevation of tissue oxygen enhances the "oxidative burst" that ultimately dispatches

ingested pathogens, helping to clear infections from tissues that are suboptimally supplied with oxygen. Hyperoxia will also enhance antibiotic uptake in infected tissues and improve the effectiveness of the antibiotic.[49] Hyperoxygenation has a direct vasoconstricting effect, resulting in decreased capillary transudation flow rates that inhibit edema formation and a reduction of vasogenic edema in patients with compartment syndrome. Hyperoxygenation also enhances microcirculation by reducing local interstitial pressure.[51]

Review of the Literature on HBOT

Table 16-3 provides a review of selected studies and reviews of the use of HBOT. An analysis of these studies and reviews allows the practitioner to conclude that HBOT may be effective for treatment of the following:

- Diabetic foot ulcers[50,53–57]
- Refractory osteomyelitis[50,53,54]
- Decrease amputations by healing wounds and ulcers[54]
- Chronic regional pain syndrome[48,58]

TABLE 16–3. Selected Literature Review: Hyperbaric Oxygen Therapy

Author(s) and Year	Patient Population	Intervention(s)	Results	Conclusions
Oliveira et al, 2014[57]	Retrospective observational study of patients with diabetic foot ulcers: 26 foot lesions, including 13-foot ulcers Wagner grade 2 or greater and 13 amputation stump ulcers	HBOT	23 completed treatments; complete epithelialization of primary lesion achieved in 15 (65%); mean healing period 16 weeks.	HBOT may be associated with ulcer healing in diabetic foot ulcers.
Kranke et al, 2012[56]	Cochrane database for systemic reviews: HBOT for chronic wounds (9 studies reviewed: 8 studies on diabetic foot ulcers, 1 study on venous ulcers)	HBOT	HBOT significantly improved ulcer healing in short term but not the long term.	HBOT can be an effective intervention for short-term healing of diabetic ulcers and venous ulcers.
Tiaka et al, 2012[50]	Patients with diabetic foot ulcers (review of the literature)	HBOT	HBOT improves tissue hypoxia; enhances perfusion; ameliorates edema; reduces inflammation; promotes fibroblast proliferation, collagen production, and angiogenesis. May help eradicate infections secondary to bacteriocidal activity and improved leukocyte and macrophage function.	HBOT may help heal persistent, ischemic, or neuroischemic infected diabetic ulcers, including recalcitrant osteomyelitis and progressive necrotizing infection.

TABLE 16–3. Selected Literature Review: Hyperbaric Oxygen Therapy—cont'd

Author(s) and Year	Patient Population	Intervention(s)	Results	Conclusions
Cristante et al, 2012[61]	Rats with spinal cord injury (SCI) randomized into 3 treatment groups	1. Placed in HBOT chamber 30 minutes after SCI 2. Placed in HBOT chamber 1 day after SCI 3. No intervention (control); Basso, Beattie, and Bresnahan scales used for functional evaluation 1 day following SCI and weekly for 1 month	No significant differences between groups on second day; on 7th, 21st, and 28th days, groups 1 and 2 performed significantly better than the control group.	HBOT beneficial in the functional recovery after SCI in rats if administered just after or within 24 hours of injury.
Londahl et al, 2011[81]	75 patients with diabetes and chronic foot ulcers present for > 3 months, randomized into 2 groups	1. HBOT in multiplace chamber at 2.5 ATA for 85 minutes, 5x/day for 8 weeks (total of 40 treatments) 2. Hyperbaric air (placebo) in multiplace chamber for 85 minutes 5x/week for 8 weeks (total of 40 treatments)	Improved quality-of-life measures.	HBOT improves long-term health-related quality of life in patients with chronic foot ulcers, possibly because of better ulcer healing.
Londahl et al, 2010[55]	94 patients with Wagner grade 2, 3, or 4 ulcers present > 3 months randomized into 2 groups	1. HBOT in a multiplace chamber, 2.5 ATA, for 95 minutes 5x/week for 8 weeks 2. Hyperbaric air (placebo) in multiplace chamber for 95 minutes 5x/week for 8 weeks	At 1-year follow-up, 37 patients achieved ulcer healing: 52% in HBOT group, 29% in placebo group.	HBOT enhances foot ulcer healing in selected patients with diabetes; at 1-year follow-up, HBOT doubled the number of healed ulcers compared with placebo.
Goldman, 2009[53]	Systematic review of 45 studies of at least 5 subjects	HBOT	High level of evidence for decreasing risk of amputation and healing of wounds. Moderate level of evidence for healing of arterial ulcers and refractory osteomyelitis. Low to moderate level	Safe technique for treatment of refractory osteomyelitis, wounds, and ulcers. Medicare-approved for diabetic foot ulcers (Wagner grades 3, 4, and 5).

Continued

TABLE 16–3. Selected Literature Review: Hyperbaric Oxygen Therapy—cont'd

Author(s) and Year	Patient Population	Intervention(s)	Results	Conclusions
			of evidence for healing of ablative or reconstructive surgery and salvage of flaps or grafts.	
Yildiz et al, 2006[48]	Literature review	HBOT	Studies reviewed found HBOT to be effective for fibromyalgia, chronic regional pain syndrome, myofascial pain syndrome, migraine and cluster headaches, and ischemic leg pain.	Evidence of effectiveness of HBOT for musculoskeletal conditions; optimal treatment protocols need to be established.
Bennett et al, 2005[59]	Cochrane Review of randomized controlled trials that compared effects of HBOT to no HBOT (sham or control) on healing of fractures	HBOT	No studies met inclusion criteria.	No evidence from randomized trials to support or refute use of HBOT for preventing poor healing or treating poorly healing broken bones.
Bennett et al, 2005[60]	Cochrane Review of randomized controlled trials of effects of HBOT on delayed-onset muscle soreness (DOMS) and closed soft tissue injury	HBOT	Nine small trials involving a total of 219 subjects were reviewed. No significant difference found between groups that received HBOT and control or sham HBOT for ankle and knee sprains and DOMS; no difference in groups for long-term pain scores, swelling, or muscle strength.	Insufficient evidence to establish the effects of HBOT on ankle and knee sprains and on experimentally induced DOMS. Some evidence that HBOT may increase interim pain in DOMS.
Kiralp et al, 2004[58]	Patients with chronic regional pain syndrome (CRPS), $n = 71$ average age 29.4	37 received HBOT, 34 normal air (control) during 15 90-minute sessions in hyperbaric chamber at 2.4 ATA (5 days a week, 1 session per day)	Significant difference in pain, wrist flexion, and wrist circumference for the HBOT group.	HBOT is an effective and well-tolerated treatment for reduction of pain and edema and increasing wrist motion in patients with CRPS.

TABLE 16–3. Selected Literature Review: Hyperbaric Oxygen Therapy—cont'd

Author(s) and Year	Patient Population	Intervention(s)	Results	Conclusions
Greensmith, 2004[49]	Literature review	Reviewed basic research and clinical studies of HBOT	Indications for HBOT include crush injury, compartment syndrome, acute traumatic peripheral ischemia, wound healing, refractory osteomyelitis, radiation injury, compromised skin grafts and flaps, thermal burns.	An expensive, technology-intensive therapy. More research is needed to determine optimal indications, timing, and dosing protocols.
Wang et al, 2002[54]	Literature review	HBOT	Indications for HBOT include refractory osteomyelitis, nonhealing wounds, non- or delayed union fractures, radiation-induced tissue injury, thermal burns, acute traumatic ischemia, compromised skin and bone grafts, and muscle flaps.	HBOT is a cost-effective modality that significantly reduces length of hospital stay, amputation rate, and wound care expenses.
Staples et al, 1999[52]	"Untrained" male university students, $n = 66$, ages 18–35	Exercise-induced DOMS treated with HBOT (100% O_2 1 hour/day at 2 ATA) or sham HBOT (21% O_2 1 hour/day at 1.2 ATA).	Significant difference in recovery of eccentric torque compared to sham group; no significant difference in pain scores.	Exposure to 1 hour of HBOT initiated within 20 minutes after exercise for 3–5 days enhances muscle torque recovery but does not significantly affect DOMS.

- Necrotizing infections[50]
- Radiation injuries[54]
- Compromised skin grafts and flaps[53,54]
- Thermal burns[54]
- Crush injuries and compartment syndrome[48–50,54]

More studies are needed to establish optimal treatment parameters, such as duration and frequency of treatment and dosing protocols. Insufficient evidence exists to support the use of HBOT for the following:

- Nonhealing fractures[54,59]
- Delayed-onset muscle soreness[52,60]
- Ankle and knee sprains.[60]

A promising study by Cristante et al[61] found that HBOT promoted functional recovery following spinal cord injury in rats.

Clinical Applications of HBOT

In the United States, HBOT is approved by Medicare for treatment of diabetic foot ulcers (Wagner grades 3, 4, and 5).[53] HBOT is considered a relatively safe treatment modality. However, Goldman[53] identified six possible side effects of treatment:

- Increased cardiovascular afterload because of the peripheral vasoconstrictive effect of oxygen

- Oxygen toxicity that can affect the central nervous system, pulmonary function, and ocular function
- Barotrauma to the inner ear, sinuses, dental, and pulmonary tissues
- Hypoglycemia
- Confinement anxiety

See Box 16-3 for a list of contraindications and precautions for HBOT.

Key Point! The use of HBOT is limited by the cost and availability of hyperbaric chambers. A used monoplace chamber may cost $400,000 and a multiplace chamber several million.[50]

EXTRACORPOREAL SHOCK WAVE THERAPY

The concept of using high-energy shock waves is not new to us. Most are familiar with the use of lithotripsy for kidney stones. Extracorporeal shock wave therapy (ESWT) is used to treat musculoskeletal conditions such as calcific tendonitis, lateral epicondylitis, and plantar fasciitis. This modality is within the practice realm of physicians and surgeons, but at the time of this writing, it is not a technique used by other health-care practitioners. It is important, though, for practitioners to be familiar with this novel application of sound waves because patients may ask about it as a treatment option.

Box 16 ■ 3 HBOT: Contraindications and Precautions

Contraindications	Precautions
Undrained hemothorax or untreated pneumothorax	COPD or asthma
Currently receiving chemotherapy or radiation	Seizure disorders
Pressure-sensitive implanted medical device	Claustrophobia
Patients taking certain antineoblastic antibiotics, such as bleomycin or doxorubicin, or the antineoplastic heavy metal compound cisplatin	Chronic sinusitis or upper respiratory infection
Recent ear or sinus surgery	Fever and/or dehydration
Pregnancy	History of spontaneous pneumothorax

Physical Principles of ESWT

Single-impulse, focused acoustical sound waves having a rapid rise in pressure are used for ESWT. The shock waves can be generated by a number of methods:

- Reverse piezoelectric (electrical to mechanical energy)
- Electromagnetic
- Electrohydraulic

The energy is transmitted from the device through coupling cushions that are capable of transmitting acoustical energy. The treatment region must be targeted via ultrasound or fluoroscopic x-ray imaging devices to ensure that the energy is focused to an accurate location. The duration of treatment may be up to 30 minutes. Energy for ESWT is described in joules per area (mJ/mm^2) and is characterized as high or low.[62]

Typical shock wave characteristics are very high peak-pressure amplitudes with rise times of less than 10 nanoseconds and a short lifecycle (10 milliseconds)[63] (Fig. 16-6). ESWT single pulses are at 1 to 4 per second (at a potential frequency of 1,000 to 4,000 shock wave pulses). The location of the maximum peak positive pressure is the focus of the shock wave. Successful therapy is dependent upon concentration of the shock wave's focus.[63] The total energy of a treatment ("energy flux density") includes the number of shock impulses and the energy density together—known as energy per square area (mJ/mm^2).

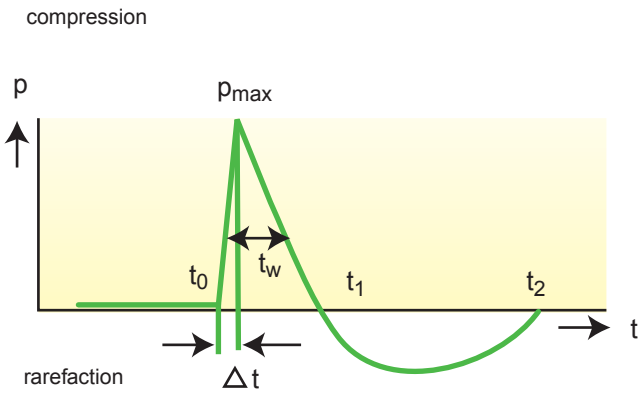

Fig 16 ■ 6 A single shock wave used for extracorporeal shock wave therapy. Note parameters of focused shock wave. T (change in time); P_{max}, pressure maximum; Δt pressure rise time; t_w, half width time; Pr, negative peak pressure. *(Diagram courtesy of Sonorex, Inc., Fayetteville, NC.)*

Proposed Physiological Effects of ESWT

Shock waves transmit through and are absorbed by soft tissue and reflected at interfaces of tissues with different densities. The biophysical and physiological effects proposed for this energy include disintegrating calcium deposits, hyperstimulation analgesia, neovascularization, and changes in cell permeability.[64] The latter two effects have been reported in studies on animal models. Needle-shaped hemorrhages (petechiae) on the skin may occur after shock wave therapy. These are attributed to the effect of shock waves on gas-filled hollow bodies ("cavitation bubbles") in fluids in the tissues. External pressure from shock waves on these bubbles can cause the bubbles to collapse, sending out a jet of water that causes petechiae to form in the skin.[63]

Review of the Literature on ESWT

Table 16-4 provides a review of selected articles on ESWT. In addition, an excellent systematic review of ESWT use for calcific and noncalcific rotator cuff tendonitis (RCT) has been published in the *Journal of Hand Therapy*.[64] An analysis of these studies allows the practitioner to conclude that ESWT may be effective for treating the following conditions:

- Chronic plantar fasciitis[65–67]
- Calcific tendonitis of the rotator cuff in the shoulder,[68,69] although disintegration of calcium deposits may not correlate with improvements in pain and function[68,70]
- Lateral epicondylitis ("tennis elbow")[71,72]
- Achilles tendinopathy[73,74]

TABLE 16–4. Selected Literature Review: Extracorporeal Shock Wave Therapy

Author(s) and Year	Patient Population	Intervention(s)	Results	Conclusions
Wang et al, 2014[76]	67 patients with a total of 72 chronic foot ulcers	ESWT to foot ulcers twice per week for 3 weeks; dosage dependent on ulcer size: number of impulses = area × 8, at least 500 shocks at 4 Hz, energy level 0.11 mJ/mm^2	At the 1-year follow-up, 55.6% of ulcers were healed and 57.4% at 5-year follow-up; blood flow increased significantly up to 1 year, decreased between 1 and 5 years; mortality rate 15%, rate of amputation 11%.	ESWT effective in diabetic and nondiabetic foot ulcers; however, effects decrease between 1 to 5 years following treatment.
Zhao et al, 2013[77]	Seventy patients with diagnosis of primary symptomatic osteoarthritis of the knees with pain for previous 3 months were randomly assigned into 2 groups	1. ESWT at 0.25 mJ/mm^2, 4,000 pulses at a frequency of 6 Hz, once per week for 4 weeks 2. Placebo ESWT	ESWT group had greater decrease in pain and greater improvements in function (WOMAC) than placebo group.	ESWT can be effective management of knee osteoarthritis; however, the role of ESWT in the treatment of knee osteoarthritis is unclear.
Al-Abbad et al, 2013[74]	Six studies on the effects of ESWT on chronic Achilles tendinopathy were reviewed systematically	Outcome measures were pain (VAS or numerical rating scale) and outcome questionnaires.	Four out of the 6 studies showed significant improvement in outcome measures.	Assigned a grade C (NHMRC recommendation) evidence for low-energy ESWT in the treatment of chronic insertional and noninsertional Achilles tendinopathy.

Continued

TABLE 16–4. Selected Literature Review: Extracorporeal Shock Wave Therapy—cont'd

Author(s) and Year	Patient Population	Intervention(s)	Results	Conclusions
Dizon et al, 2013[66]	Eleven studies on the effects of ESWT on chronic plantar fasciitis were reviewed; 8 of these included in a meta-analysis.	Outcome measures were morning pain, overall pain, and activity pain	Four out of 8 studies found ESWT was significantly more effective than placebo; high- and moderate-intensity ESWT was more effective than low intensity. Adverse effects included calcaneal pain and erythema, edema, paresthesia, and bruising.	High- and moderate-level ESWT may effectively decrease pain for patients with chronic plantar fasciitis.
Ioppolo et al, 2012[70]	68 patients with shoulder pain (for at least 4 to 6 months) and calcific deposits, randomly assigned into 2 groups	1. ESWT at an energy level of 0.20 mJ/mm^2 2. ESWT at an energy level of 0.10 mJ/mm^2 Both received 2,400 pulses once per week for 4 weeks	Significant improvement in pain (VAS) and function (Constant Murley Scale) for 0.20 group compared to 1.10 group; improvement was not related to disappearance or size of calcifications.	ESWT at an energy level of 0.20 mJ/mm^2 effectively reduced pain and improved function in patients with supraspinatus calcifying tendonitis.
Ibrahim et al, 2010[67]	Fifty patients with unilateral chronic plantar fasciitis randomly assigned into 2 groups	1. ESWT at an energy level of 0.16 mJ/mm^2, 2,000 impulses per session for 2 sessions 2. Placebo ESWT	ESWT group had a significant decrease in pain (VAS) and function (Roles and Maudsley score) at 4, 12, and 24 weeks' follow-up compared to placebo group.	Low-energy ESWT can effectively decrease pain and improve function for patients with chronic plantar fasciitis.
Furia, 2008[73]	Chronic noninsertional Achilles tendinopathy	Single dose of high-energy ESWT, 3,000 shocks at 0.21 mJ/mm^2 for a total energy flux density of 604 mJ/mm^2 or traditional forms of nonoperative therapy for 6 months	Statistically greater % of patients who had ESWT had improvement in Roles and Maudsley scores (patient assessment of pain and limitations of activity) compared to control group.	Single treatment of high-energy ESWT is a safe and effective procedure for chronic noninsertional Achilles tendinopathy.
Cacchio et al, 2006[69]	90 patients, random assignment into 2 groups	1. Low-intensity ESWT (0.10 mJ/mm^2, 2,500 impulses per session × 4 sessions	Pain and functional level significantly more improved for 2,500 impulse group compared to placebo group	Low-intensity ESWT at 2,500 impulses per session can significantly reduce pain and improve function for patients with calcific

TABLE 16–4. Selected Literature Review: Extracorporeal Shock Wave Therapy—cont'd

Author(s) and Year	Patient Population	Intervention(s)	Results	Conclusions
		2. Placebo (ESWT 25 impulses per session)	after treatment and at 6-month follow-up; calcifications disappeared in 86% of 2,500 impulse group compared to 8.8% in placebo group	tendonitis of the shoulder.
Wilner and Strash, 2004[65]	Chronic proximal plantar fasciopathy (plantar fasciitis)	ESWT using 1,800 shocks at 18 kilovolts and 4 Hz/sec (under general anesthesia)	More than 2 years after treatment, patients rated level of improvement: excellent/good 87%, fair 11%, no improvement 2%.	ESWT is an effective and safe noninvasive intervention for chronic plantar fasciitis.
Pleiner et al, 2004[68]	Symptomatic calcific tendonitis of the shoulder for greater than 6 months	ESWT 2 × 2000 impulses at 0.28 mJ/mm^2 at an interval of 2 weeks or < 0.07 mJ/mm^2 (control group)	Shoulder function significantly improved compared to control; calcifications completely resolved in 19% compared to 8% in control; significant decrease in pain at 1 week but not at 3 and 7 months.	ESWT can significantly improve shoulder function and may help disintegrate calcific deposits but may not result in reduction in pain.
Chung and Wiley, 2004[71]	Lateral epicondylitis of at least 3 weeks but less than 1-year duration	ESWT delivered one session per week for 3 weeks at 2,000 pulses and energy flux density 0.03–0.17 mJ/mm^2 or sham ESWT	"Success rates" based on level of pain, use of pain meds, quality of life (EuroQoL questionnaire), and grip strength was 39% for ESWT and 31% for sham.	No meaningful difference in pain scores, grip strength, or quality of life between patients who received ESWT and those who received sham ESWT.
Rompe et al, 2004[72]	78 tennis players with recalcitrant MRI-confirmed tennis elbow of at least 12 months duration, randomly assigned to 2 groups	1. Low-energy ESWT (EFD 0.09 mJ/mm^2, 3 × 2,000 pulses, repetition rate 4 Hz) weekly for 3 weeks 2. Placebo ESWT for 3 weeks	At 3 months: higher improvement in pain and LEFS with ESWT; 65% of patients in ESWT group achieved at least 50% decrease in pain, compared to placebo group (28%).	ESWT is more effective than placebo for reduction of pain and improvement of LEFS score for patients with lateral epicondylitis.

EFD = energy flow density; LEFS = lower extremity functional scale; WOMAC = Western Ontario and McMaster Universities Arthritis Index.

- Chronic foot ulcers, either diabetic or nondiabetic[75,76]
- Osteoarthritis of the knees[77]

Certainly the addition of new studies to the literature to support the use of this modality for management of chronic tendinopathy and fasciopathy would be welcome.

Clinical Applications of ESWT

The FDA has approved the use of certain devices for ESWT. The manufacturer of these devices will have evidence of FDA approval. Clinical application of ESWT intensity has been categorized into three levels:[66]

- Low intensity: < 0.1 mJ/mm^2
- Moderate intensity: 0.1–0.2 mJ/mm^2
- High intensity: > 0.2 mJ/mm^2

The most common applications for ESWT have included management of chronic plantar fasciitis (with or without a heel spur), tendonitis of the shoulder, and tendonitis of the elbow (Fig. 16-7). Another indication for use is in the management of nonunion fractures. Some side effects can occur after treatment, including hematoma, reddening, petechiae, local soft tissue swelling, and transient pain. Osteonecrosis of the humeral head associated with ESWT is reported in the literature.[78] Contraindications for ESWT are outlined in Box 16-4.

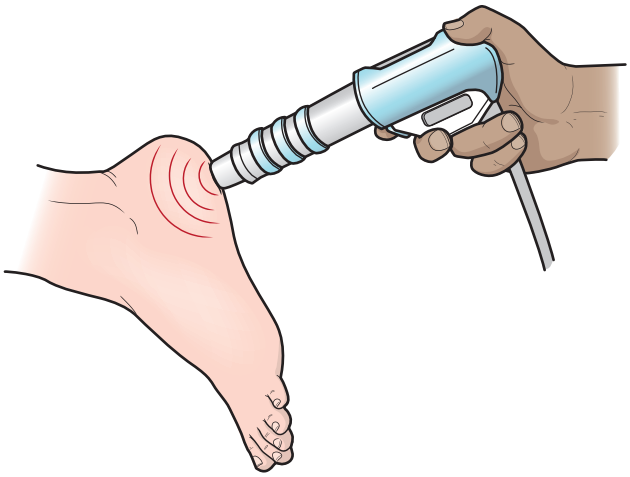

Fig 16 ■ 7 Extracorporeal shock wave therapy applied to the heel for plantarfasciitis.

Box 16 ■ 4 Extracorporeal Shock Wave Therapy: Contraindications

- Bleeding conditions
- Pacemakers
- Medications that prolong blood clotting
- Children
- Pregnancy
- Acute injuries

Documentation Tips

As with all biophysical agents, thorough documentation of the application is essential. Depending on the specific modality administered, the following are key elements to document:

- The specific type of modality used
- The body part treated
- Patient position for treatment
- Treatment parameters such as duration, intensity, power, wavelength (e.g., MIRE)
- Integrity of the skin in area treated, before and after

CASE STUDY 16-1 Extracorporeal Shock Wave Therapy for Lateral Epicondylitis

A 32-year-old female with a history of lateral elbow pain for 18 months presents for rehabilitation. She is a recreational golfer and has not been able to continue playing as regularly as she would like due to pain in the lateral elbow. Prior unsuccessful treatments have included rest, ice, NSAIDs, ultrasound, bracing, corticosteroid injection, and platelet-rich plasma therapy. Palpation reveals pain and tenderness localized to the right lateral epicondyle. Cozen's test for lateral epicondylitis is positive on the involved side.

CLINICAL DECISION-MAKING

1. Does the patient have an impairment, activity limitation, or problem that can be improved or alleviated with the use of ESWT?
   ANSWER: Yes, the patient has a history of and clinical confirmation of lateral epicondylitis that is limiting participation in golf. Prior conservative care and more aggressive therapies have been unsuccessful. What limited evidence is available suggests ESWT may be beneficial for lateral epicondylitis.

CASE STUDY 16-1 Extracorporeal Shock Wave Therapy for Lateral Epicondylitis—cont'd

2. Is the patient appropriate for ESWT? Do any of the general precautions or contraindications to ESWT apply to the patient, or are there specific considerations regarding the application of ESWT to this patient?
ANSWER: Prior interventions have failed and the patient does not present with any contraindications to ESWT.

3. What are the specific goals to be achieved with the use of ESWT?
ANSWER: ESWT will be used to stimulate collagen production and neovascularization in the tissues of the lateral epicondyle.

4. What are the proper application procedures for ESWT?
ANSWER:
a. Seat the patient beside a treatment table with her right arm resting comfortably on a towel with the elbow flexed approximately 90 degrees.
b. Manually locate the region of tenderness and associated soft tissues of the common extensor tendon and extensor muscles.
c. Apply ultrasonic gel to the treatment area.
d. Select the treatment parameters:
- Applicator head size: 12–15 mm should be sufficient
- Type of shocks: continuous
- Pressure: 2 bar
- Frequency: 10 Hz
- Number of shocks: 2,000

e. Number and frequency of applications: 3 to 6 sessions applied within 5 to 10 days of each other.

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CHAPTER 17

ELECTROPHYSIOLOGICAL TESTING OF NERVES AND MUSCLES

Arthur J. Nitz, PT, PhD; James W. Bellew, PT, EdD

WHAT IS ELECTRONEUROMYOGRAPHY?

Clinical electroneuromyography includes observing, analyzing, and interpreting the bioelectrical activity of muscles and nerves as they respond to electrical stimulation, needle provocation, and voluntary activation. Most often, the testing consists of a combination of nerve conduction studies (NCS) and electromyography (EMG), although additional tests may occasionally be appropriate. These may include somatosensory-evoked potentials, brain stem auditory-evoked potentials, visual-evoked potentials, intraoperative monitoring, and repetitive stimulation. Of these additional electrophysiological tests, somatosensory-evoked potentials and repetitive stimulation are more commonly accomplished on a routine basis. However, this overview focuses on clinical NCS and EMG studies.

ANATOMY AND PHYSIOLOGY REVIEW

Peripheral Nerve Structure

The anatomic unit of the nervous system is the *neuron,* with its various processes or nerve fibers.[1] By contrast, the functional unit of the neuromuscular system is the

motor unit, consisting of the anterior horn cell, the nerve root, the plexus, individual nerve fibers, the neuromuscular junction, and all the muscle fibers innervated by that axon (Fig. 17-1).

ENMG testing assesses various aspects of the neuron and the motor unit. EMG testing examines the delineated components of the motor unit, allowing the practitioner to determine the location of a neuromuscular impairment.

Results of ENMG testing cannot be used to identify the actual cause of a nerve or muscle impairment, although it is often assumed to do so. For instance, if testing reveals that a patient has slowed neural conduction in the median nerve across the carpal tunnel of the wrist and the opponens pollicis muscle demonstrates evidence of denervation with EMG examination, we would refer to this condition as carpal tunnel syndrome. However, the ENMG testing identifies only the location (carpal tunnel) and severity (mild, moderate, severe) of the condition, not its cause. The cause could be an abnormally thickened flexor tendon putting pressure on the nerve, a thickened transverse carpal ligament (flexor retinaculum), a bony impingement from advanced arthritis or from a fracture, or a temporary generalized edema accumulation such as can occur during the second and third trimesters of pregnancy. Imaging studies (radiograph, magnetic resonance imagine [MRI], etc.) may be able to identify the anatomical or structural cause of a condition, but ENMG is restricted to identifying the location of a neuromuscular impairment.

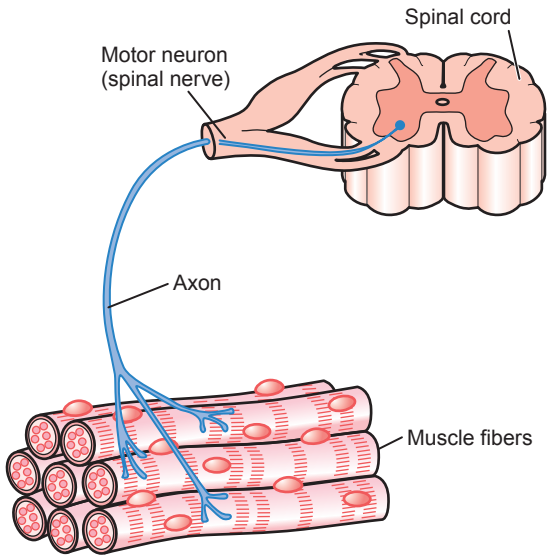


Fig 17 ■ 1 The functional unit of the neuromuscular system is the motor unit, which consists of the anterior horn cell, the nerve root, the brachial or lumbosacral plexus, individual nerve fibers, the neuromuscular junction, and all the muscle fibers innervated by that axon.

Peripheral nerves are composed of a variety of elements and cell types. One naturally thinks of the axon, which is composed of neurofilaments and microtubules along with mitochondria interspersed in the cytoplasm (Fig. 17-2). Myelinated nerve fibers are surrounded by layers of lipoprotein sheets (myelin) produced by the Schwann cells that lie on the outside of the axon. Periodic interruption of myelin occurs where longitudinally sequential Schwann cells meet, forming the node of Ranvier—an area of relatively decreased resistance to ionic exchange—and more easily permitting depolarization (Fig. 17-3). In large myelinated nerve fibers, this depolarization "jumps" from node to node, thereby accelerating the speed of conduction while simultaneously reducing the space needed for a nerve with this conduction speed. This diminishes the energy needs for depolarization because it is occurring only at the nodes of Ranvier and not along the entire course of the nerve.

Nerves are comprised of numerous unmyelinated fibers, mast cells, fibroblasts, blood vessels, and extensive connective tissue elements. In fact, as seen in Figure 17-4, connective tissue surrounds the entire nerve (epineurium), the fascicles (perineurium), and the individual axons (endoneurium).

Peripheral Nerve Function

As ENMG testing is primarily a functional assessment of the neuromuscular system, a discussion of a few key features of nerve physiology is appropriate. The principal basis for the production and conduction of electrical signals in biological tissue is the separation of charge across

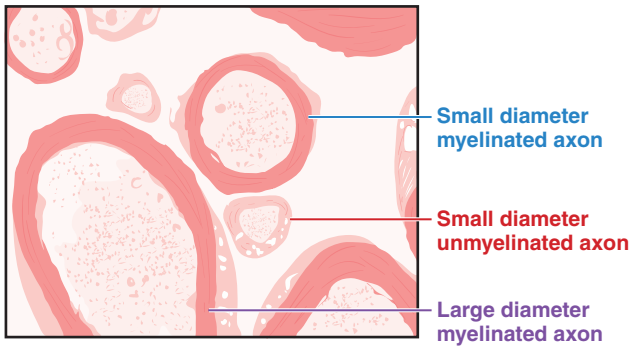


Fig 17 ■ 2 Electron micrograph cross-section of a peripheral nerve with axons of various diameter. Note that some axons are surrounded by myelin but others are unmyelinated. Axons typically consist of numerous subcellular organelles, such as microtubules, neurofilaments, connective tissue, and mitochondria, which reflect the complexity of nerves.

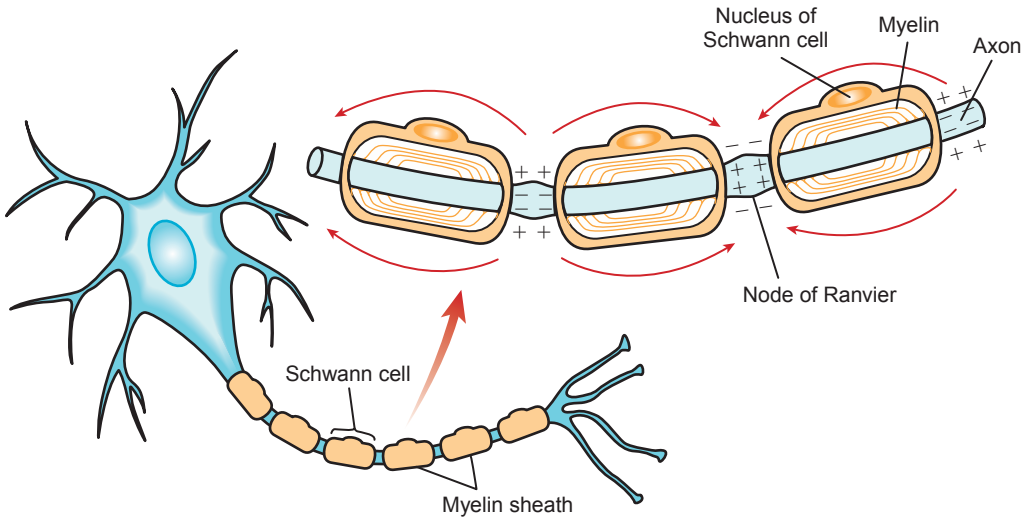


Fig 17 ■ 3 Node of Ranvier for a typical myelinated nerve fiber. Absence of myelin at the node facilitates the propagation of neural impulses via "saltatory" conduction. Saltatory conduction increases speed of conduction, decreases space needs, and reduces the amount of energy expenditure required for impulse propagation. For myelinated nerve fibers, depolarization occurs only at the node of Ranvier.

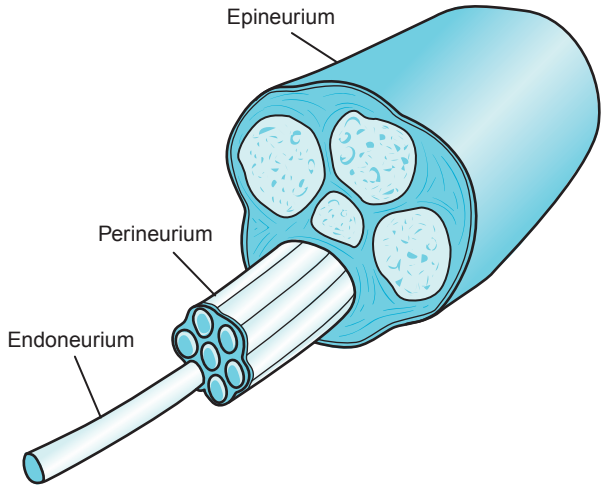


Fig 17 ■ 4 The connective tissue elements of a peripheral nerve are extensive and include the epineurium surrounding the entire nerve, the perineurium encircling the fascicles, and the endoneurium around each axon.

the cell membrane of the major ions—potassium (K^+), sodium (Na^+), and chloride (Cl^-). The cell membrane is designed to permit selective permeability of these ions so that at rest the inside of the nerve and muscle cells are electrically negative (assisted by the sodium–potassium pump). This electronegativity is referred to as the *resting membrane potential* and establishes the excitability of nerve cells.

When a stimulus of sufficient amplitude occurs, the voltage across the nerve cell membrane will exceed a threshold value, producing an "all-or-none," self-perpetuating, and nondecremental action potential (Fig. 17-5). The all-or-none feature of nerve physiology is especially important in nerve conduction testing because it helps the practitioner determine how much stimulus to apply to the nerve during the procedure. It is necessary to apply a strong enough stimulus to exceed the nerve's threshold, thereby achieving a self-propagating action potential. When this level of stimulus is reached, no amount of additional stimulus strength will increase the response. Either the nerve (or muscle fiber) depolarizes or it does not—that is, "all or none."

Finally, an important feature of excitable tissue related to electrophysiological testing is volume conduction. Nerve and muscle are electrically excitable, whereas skin, subcutaneous fat, connective tissue, bone, blood vessels, and other tissues are not. Because of this, electrical stimulation can be applied transcutaneously, and the practitioner can be assured that the stimulus will excite only the tissue laden with electrolytes—nerve and muscle. As a result, direct visualization and stimulation

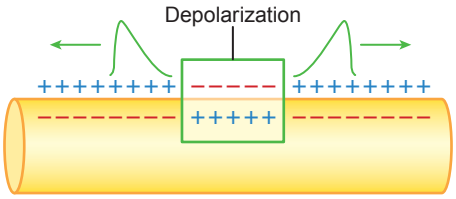


Fig 17 ■ 5 The wave of depolarization proceeds in two directions and is self-propagating when a threshold stimulus is reached. When a threshold stimulus occurs, the axon responds completely (the "all-or-none" feature).

of the muscle and nerve are not necessary and can be accomplished relatively comfortably.

A final important feature of neural conduction worth considering is referred to as *saltatory* (literally "jumping") *conduction* (see Fig. 17-3). This property of neural conduction results from the resistance to depolarization produced by the presence of myelin derived from the Schwann cells associated with many axons. Consequently, the "path of least resistance" becomes the nonmyelinated node of Ranvier. Therefore, depolarization along a myelinated axon "jumps" from node to node rather than being propagated along the entire length of the fiber in a sequential or contiguous fashion.

Saltatory conduction has at least three benefits for nerve conduction. First, the speed of saltatory conduction is substantially higher than nonsaltatory (i.e., contiguous) conduction. Second, the amount of energy needed to propagate a nerve response by saltatory conduction is considerably less because depolarization and the ATP-driven sodium–potassium pump are necessary only at the nodes of Ranvier where the "action" is occurring. Finally, the relative physical space needed for a nerve that can conduct at the speeds necessary to achieve physiological normalcy (velocities of 40 to 70 meters per second) are markedly less for myelinated nerves than for unmyelinated fibers.

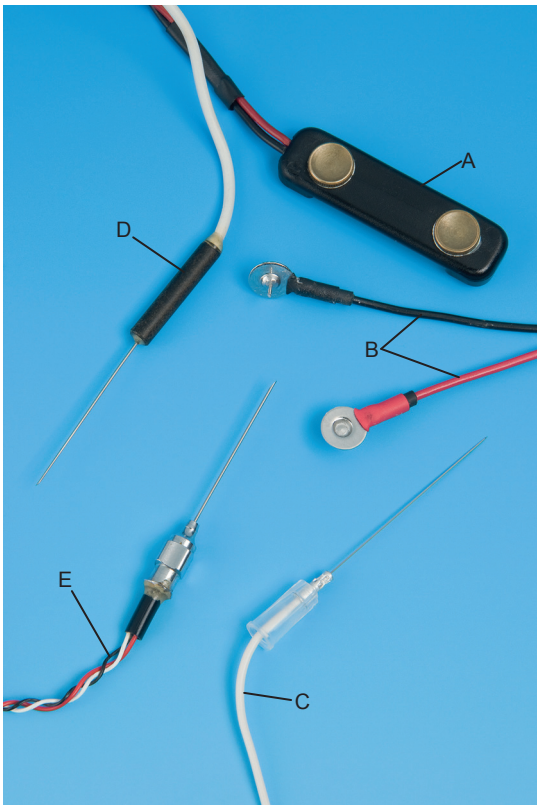


Fig 17 ■ 6 Standard EMG/NCS electrodes used in everyday practice. (A) Surface ("bar") electrode for NCS. (B) Surface disk electrodes for NCS and some EMG applications. (C) Monopolar needle electrode for EMG. (D) Concentric needle electrode for EMG. (E) Bipolar needle electrode for EMG.

EQUIPMENT TO CONDUCT ENMG

An EMG machine permits visualization and recording of human bioelectric signals so that nerves, muscles, and the neuromuscular junction can be objectively evaluated. However, the magnitude of these bioelectrical signals is ordinarily so small that they must be filtered and amplified in order to be processed, measured, and displayed on an oscilloscope or a computer screen. Simultaneously, the signal is sent to speakers that allow the examiner and the patient to "hear" the muscle or nerve response to activation, which serves as a form of biofeedback—especially for the patient.

Electrodes attached to the patient allow the equipment to record the signal. As seen in Figure 17-6, there are surface electrodes for nerve conduction studies ([NCS] and some aspects of EMG) and needle electrodes (for most EMG and the occasional application for NCS). Nerve conduction testing requires an adjustable stimulator to trigger a stimulus that depolarizes the nerve, whose response is captured on the oscilloscope (Fig. 17-7).

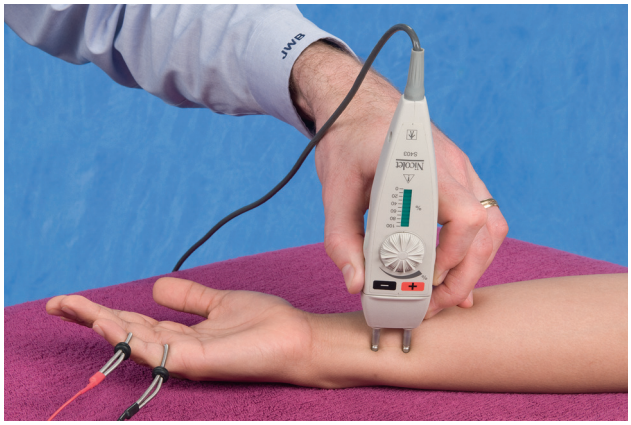


Fig 17 ■ 7 A stimulator and recording ring electrodes for nerve conduction velocity testing.

These responses can be physically manipulated with the dials or controlled with computer software to obtain a variety of measurement properties of the waveform, including latency, amplitude, and area under the curve. Nerve conduction velocity (NCV) can be calculated from these responses by dividing the latency difference between

two spatially separated waveforms into the distance between the two stimulation sites (Fig. 17-8). A more detailed description of this process is presented later in the chapter.

INDICATIONS: WHO NEEDS ENMG TESTING?

Patients with signs and symptoms of a neuromuscular disorder can usually benefit from the information obtained from ENMG testing. Although there are occasional exceptions, patients who complain of numbness, tingling, pain involving the sensory division of the peripheral nervous system (PNS), and weakness (implicating the motor division of the PNS) are usually referred for ENMG testing. Because complaints and objective findings of sensory and motor impairment may arise from central nervous system (CNS) disorders, one feature of electrophysiological testing is to delineate such complaints as deriving from either the PNS or the CNS.

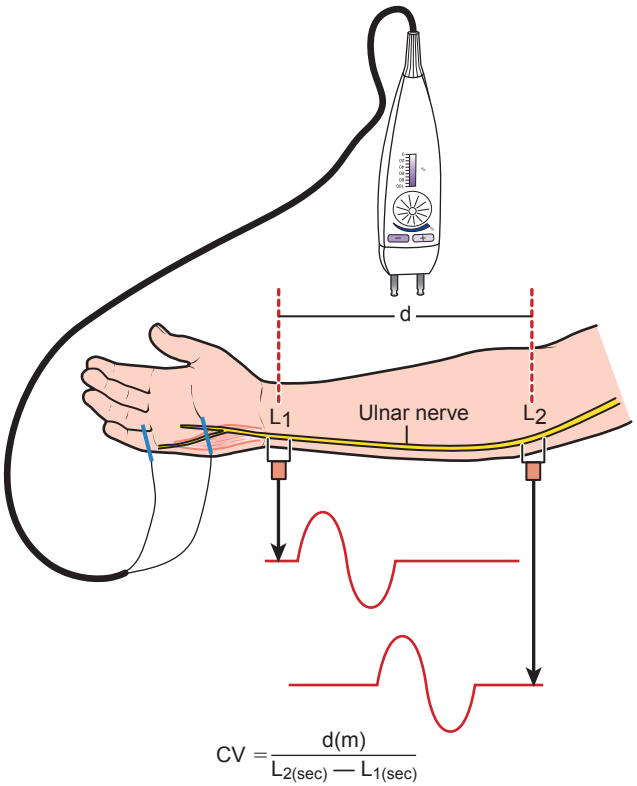


Fig 17 ■ 8 Motor nerve conduction for the ulnar nerve: Recording electrode is on the abductor digiti minimi muscle. Stimulation is applied just proximal to the wrist to obtain the distal motor latency (L_1). A proximal latency (L_2) is obtained by stimulating the ulnar nerve at the elbow, and the distance between the two sites of stimulation is recorded.

ENMG testing is a particularly sensitive procedure for directly evaluating PNS complaints. CNS disorders are primarily established by inference from relatively normal findings with this form of testing and, more commonly, from imaging studies such as MRI. One notable exception would be the patient who has a disease of the anterior horn cells—such as amyotrophic lateral sclerosis (ALS), also known as *Lou Gehrig's disease*—which are located in the spinal cord but are functionally part of the lower motor neuron. In the case of ALS, the peripheral nerve responses to electrical stimulation are altered during motor nerve conduction studies. Needle EMG examination of the muscles supplied by the axons deriving from the affected nerve cells will show evidence of an unstable membrane (denervation). Nevertheless, ENMG testing is usually conducted when PNS—not CNS—disorders are suspected.

There are two major types of nerve injury—segmental demyelination and axon degeneration—that electrophysiological testing seeks to delineate. The first type, *segmental demyelination,* represents a focal conduction abnormality along the course of an otherwise normal axon and is best detected by nerve conduction study.[2] Unlike the changes noted by needle EMG, conduction abnormalities can be present almost immediately after the disorder begins or after the onset of nerve injury. Usual causes of segmental demyelination include mild to moderate compression, such as that seen with carpal tunnel syndrome, or a primary demyelinating condition as observed after exposure to diphtheria toxin. Regardless of the cause, the effect on the nerve test is slowed conduction velocity.

The second major type of nerve abnormality is *axon degeneration* (axonopathy). It is best detected by needle EMG examination but usually takes about 21 days after injury to be demonstrated.[2] This is the general length of time necessary for Wallerian degeneration to proceed sufficiently to manifest itself as muscle membrane instability, which the needle EMG identifies. Causes of axon loss include severe nerve compression (e.g., nerve root compression from a herniated disc) or direct trauma, inflammation, and ischemia of the nerve. Most often, the speed of conduction is preserved (at least early on), but the amplitude of the compound muscle action potential or sensory nerve action potential is reduced. These NCS changes are usually preceded by EMG spontaneous potentials already mentioned.

Key Point! *Wallerian degeneration* is the degeneration of an axon that has been severed from its cell body. The myelin sheath also degenerates, but the neurilemma does not and forms a tube that will direct the growth of the regenerating axon.

Both axonopathy and demyelination seem to have similar clinical manifestations. Both can lead to weakness, and both often produce complaints of pain or other sensory manifestations. Even though there is great value in delineating a condition as primarily arising from one cause or the other, the reality is that many dysfunctions encountered in the PNS will have both segmental demyelination and axon degeneration characteristics. An example is the patient who has moderate to severe levels of compressive median neuropathy at the wrist (e.g., carpal tunnel syndrome). The NCS will invariably demonstrate clear slowing of conduction across the wrist (demyelination) as well as reduction in amplitude of action potentials—motor and sensory (usually associated with axon injury and loss). In addition, the EMG will often show signs of spontaneous potentials, another indication of axonopathy. Because of this common overlap in demyelination and axon degeneration associated with nerve disorders, it is recommended that ENMG testing include both nerve conduction study and needle EMG to thoroughly examine a patient with PNS complaints.

Clinical Examples of Diagnostic Dilemma for Which ENMG Testing Is Important

Example 1

A 45-year-old female office worker has had 6 weeks of neck pain and numbness in the left nondominant thumb, index, long, and portions of the ring finger. The numbness regularly awakens her at night, but the cervical spine pain makes it difficult to achieve a position of comfort to fall asleep. An MRI of the cervical spine demonstrates some degenerative disc changes at C6 and C7 with some slight compression of the nerve root in the foramen. No spinal stenosis is identified by the imaging study. Manual cervical spine distraction alleviates much of the neck pain and some, but not the majority, of the hand numbness. Rotation of the head and neck to the left aggravates the radicular symptoms. The patient has a diminished triceps deep-tendon reflex (DTR) and 3/5 muscle weakness of the left triceps muscle, forearm flexors, and extensors. She demonstrates a positive median nerve compression test and a positive Phelan test, both at the left wrist. Muscle testing of the intrinsic muscles of the hand reveals a 3+/5 abductor pollicis brevis voluntary muscle test. The patient's family physician has recently informed her that she has "borderline" type 2 diabetes, which is currently being monitored and managed by diet and exercise. The patient has been referred for physical therapy with the standard "evaluate and treat" prescription.

Reasonable goals for this patient include a reduction in C6–7 radicular signs and symptoms in the left upper extremity, improvement in cervical spine range of motion (ROM), and reduction in sensory symptoms in the median nerve distribution. Hopefully, this approach will result in less sleep disturbance for her. It seems appropriate to include cervical traction and neural glides[3–8] in the treatment procedures for this patient, but there is some uncertainty about this case that suggests that additional information would allow the practitioner to make more informed decisions about management. Specifically:

1. How much of this patient's complaint of numbness is the result of a median neuropathy at the wrist (e.g., carpal tunnel syndrome)?
2. How much of the hand numbness is attributed to the radiculopathy that her signs and symptoms suggest?
3. Do any of her complaints relate to the recent onset of type 2 diabetes and its tendency to adversely affect peripheral nerve function?
4. Is it likely that mechanical traction will effectively deal with the patient's radicular complaints? How severely injured is the nerve root?
5. Would this patient benefit from a resting night splint for the wrist along with neural glides, or would this be wasted effort?

Is ENMG testing able to answer these questions so that the intervention can be more focused and a more realistic prognosis can be offered? Here are some reasons that an ENMG examination should be conducted with this patient:

1. ENMG can establish whether she has a compressive neuropathic injury at the wrist and determine its severity.

2. ENMG can determine whether the apparent weakness in the upper extremity is the result of nerve root compression and establish whether the muscle response is still deteriorating or beginning to show signs of recovery.
3. ENMG can explore the motor and sensory responses of several nerves in both arms to see whether the type 2 diabetes has diffusely affected neural function.

The answers provided by ENMG testing will firmly establish and document the extent of neural injury and involvement in this case and give a much clearer prospect of improvement. Information such as this will allow the therapist to focus on therapies or interventions that are likely to result in the most success for the patient.[9] Oftentimes, this allows the patient to spend time on those aspects of the home program (e.g., home traction or neural glides) that are more likely to reinforce the therapy administered at the clinic and usually results in fewer clinic visits to achieve discharge goals.

Example 2

A 64-year-old male who sustained a proximal humeral fracture 6 weeks previously has now been sent to initiate physical therapy with a request to "concentrate on *active* arm elevation." The initial evaluation reveals 115° of passive flexion but virtually no active flexion or abduction. Furthermore, palpation of the deltoid muscle during the patient's efforts to abduct and flex the glenohumeral joint suggests essentially no discernable muscle activation. There is decreased sensory acuity to crude and light touch over the middle deltoid ("deltoid patch"). Two weeks later, the patient is noted to have 125° of passive flexion but still no active arm elevation. At this point, the therapist must ask whether this patient has a low threshold of pain tolerance, is poorly motivated, has profound "disuse atrophy," or has actually sustained a neural injury (axillary, in this case). A brief needle EMG demonstrates that the deltoid muscle is severely denervated, and the patient essentially is unable to volitionally activate a sufficient number of motor units to elevate the arm. The patient has sustained an axillary nerve injury.

With this information, the focus of therapy efforts, temporarily, must be to protect the limb until reinnervation occurs and concentrate on those muscles that have normal or near normal innervation (i.e., interscapular stabilizers and serratus anterior and upper trapezius muscles). In this way, the therapist minimizes the likelihood of harming the patient by directing therapeutic efforts to those structures that have the potential to benefit from exercise and stimulation.[10–12] Additional therapeutic activity for those muscles that are denervated should be withheld until the lengthy process of reinnervation nears completion, at which point more aggressive exercise can begin.

There are numerous similar clinical scenarios in which such questions arise during the delivery of care and the standard clinical examination does not sufficiently delineate the most appropriate treatment direction. Additional information about the status of the neuromuscular system is needed, and ENMG testing can often provide such data, which can then guide clinical decisions.

PRECAUTIONS

Patients with pacemakers represent a precaution for conducting ENMG testing because the nerve stimulator may interfere with the pacemaker signal. This concern is especially relevant for the NCS and less so for the EMG portion of the study. Individuals taking blood thinners (anticoagulants) are another group that needs to be monitored carefully during and after the needle EMG to be certain that excessive bleeding does not occur. In patients who have blood-transmittable diseases (e.g., HIV, hepatitis, Creutzfeldt-Jakob disease), all universal precautions should be taken (i.e., latex gloves, eye protection, gown) to protect the examiner from possible infection during the ENMG testing procedure.[13]

NERVE CONDUCTION STUDIES

Nerve conduction studies assess motor and sensory nerve function by recording the evoked response produced by electrical stimulation of the nerve. Several important clinical questions can be answered by NCS:[13]

1. Is the peripheral nervous system involved in the patient's condition?
2. What is the location of the peripheral nerve condition, and is more than one nerve involved?
3. Is the peripheral nerve condition mild, moderate, or severe (conduction block partial or complete)?
4. Does the condition appear to be focal or diffuse (systemic disorder)?

5. Does the condition primarily involve motor fibers or sensory fibers, or are they equally affected by the nerve condition?

With these questions in mind, an attempt to adhere to the following NCS principles should be made:[13]

1. Examine motor and sensory fibers whenever possible.
2. Test several segments of nerve suspected to be involved.
3. Be prepared to test upper and lower limbs if the preliminary findings warrant this approach.
4. Test when likely to obtain optimal "diagnostic yield." Because NCS findings can demonstrate abnormality almost immediately after the onset of a condition, there is no "bad time" to conduct this test. This is not true for the EMG examination, where it may take approximately 3 weeks for abnormalities to manifest.

General Influencing Factors

A variety of factors may significantly impact NCS results. The examiner must remain alert to their influence on these testing procedures:[14,15]

1. Upper versus lower limb study
2. Age
3. Limb length (height)
4. Limb temperature
5. Anomalous innervation patterns

Upper-extremity nerve conduction velocity is, on average, 5 to 10 meters per second faster than the lower-extremity nerves. This may be because the lower extremity is longer than the upper extremity, so the cell bodies for lower-extremity nerves are farther away—which may make the lower-extremity axons less nutritionally competent. Presumably this has the effect of slightly slowing down conduction velocity.

Age has an influence on latency, amplitude, and conduction velocity values. Nerve conduction velocity does not achieve the normal adult values until age 7, although some elements of the PNS may not fully mature until age 14 to 18. Prior to age 7 (when myelination is not yet complete), NCV values are roughly half that of adult velocities. This chapter will not provide special information on conducting NCS with pediatric clients.

The relationship of aging on conduction velocity has been extensively studied, but there remains some debate on the precise effect of age on nerve values. It is generally accepted that beyond the age of 60, values decline 1 to 2 meters per second for each decade (over 60). Some authors contend that this decline actually begins after age 40, but that has been refuted by other investigators. In any event, even with this slight slowing that may occur with age, the conduction velocity should not drop below the lower limit of normal, even for those in their 80s.

Height, and consequently limb length, influences nerve conduction velocities. In general, the longer the limb, the slower the conduction velocity. This fact reinforces the importance of comparing conduction velocity values for both extremities to be certain that slowed conduction represents a disease state rather than the normal variation associated with limb length.

Limb temperature has a decided effect on distal latency and nerve conduction values. Because a cool limb conducts electrical signals more slowly than one of normal temperature, skin temperature is measured, recorded, and monitored during the ENMG examination. Upper or lower extremity limbs whose temperatures fall below 86°F (30°C) must be warmed with hot packs or warm towels until this threshold is achieved. Interestingly, sensory action potential amplitudes actually increase with a cool limb and might be a clue to warm the limb. If the latency of a sensory nerve action potential is slowed but the amplitude is higher than would be predicted, the examiner should suspect a cool limb. Efforts should be made to warm the limb to see if both values will return to the normal range.

Anomalous innervation patterns can present a confusing picture to the ENMG examiner, but teasing this possibility out is important to provide a clear picture to the referring individual. Approximately 20% of individuals have an anastomosis (nerve connection) between the median and ulnar nerve in the forearm; this is called a Martin-Gruber anastomosis (Fig. 17-9). The condition is characterized by median nerve fibers that cross over to join the ulnar nerve in the forearm proximal to the stimulation site at the wrist. When stimulation is carried out over the normal median nerve sites at the wrist and elbow, this pattern of nerve connection will usually result in abnormally high conduction velocity calculations (e.g., often as high as 100 meters per second). In addition to this abnormally high conduction velocity, there is usually a change in the waveform configuration between the stimulation sites at the wrist and the elbow—another clue that some unusual pattern is present. Being alert to the possibility of this nerve

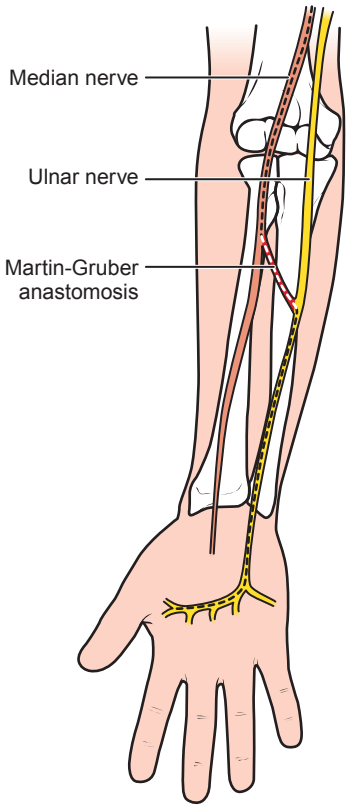


Fig 17 ■ 9 The classic Martin-Gruber anastomosis between the median and ulnar nerve is one example of anomalous innervations. Anatomical variances such as this will dramatically affect conduction velocity values.

arrangement will limit the likelihood of erroneous interpretation of nerve responses to stimulation.

Motor Nerve Conduction Study

Following the patient history and clinical examination, motor nerve conduction studies are often the first portion of the ENMG test and provide information about the function of the axons, myelin, and neuromuscular junction. The physiological response evoked by stimulating the nerve during this portion of the examination is called the *compound muscle action potential* (CMAP) and represents the simultaneous depolarization of all the individual motor units under the recording electrode (Fig. 17-10). The general procedure for obtaining motor nerve conduction and relevant portions of the waveform is described next.

Procedure

Motor conduction studies are generally accomplished by placing a pair of surface electrodes on a distal muscle supplied by the nerve being tested and then stimulating

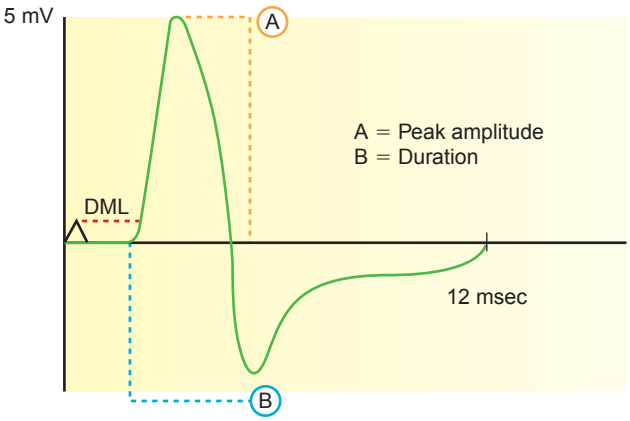


Fig 17 ■ 10 Standard compound muscle action potential (CMAP) for a typical motor nerve study following stimulation. The time from stimulus artifact to initial departure from the baseline is the distal motor latency (DML). Other important features of this response include the latency, duration of the waveform, its shape, and the area under the curve.

that nerve at various sites along its anatomical course. More specifically, the "active" electrode (of the pair) is placed as close to the anatomical motor point as possible; the "reference" electrode is placed over the nonexcitable tendon of that muscle. These two electrodes together are referred to as the *recording electrodes*. A ground electrode is also placed on nonexcitable tissue, often over a bony prominence, ideally between the stimulating and recording electrodes (Fig. 17-11).

The mixed peripheral nerve is electrically stimulated at some set distance from the active electrode in an effort to obtain a CMAP response. For upper-extremity nerves, this distance is usually 8 centimeters from the cathode of the stimulating device. For lower-extremity nerves, it

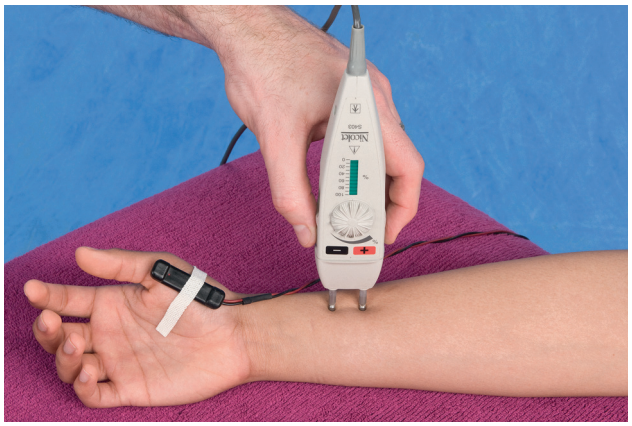


Fig 17 ■ 11 Standard setup for median nerve motor conduction study: A ground electrode is placed over the bony aspect of the dorsum of the hand, the black recording electrode is placed on the middle of the abductor pollicis brevis muscle, and the red reference electrode is placed on the volar surface of the thumb metacarpal phalangeal joint.

may vary between 8 and 12 centimeters. The stimulator's cathode is oriented so that it is closest to the muscle to be depolarized.

Key Point! A "mixed" peripheral nerve contains motor, sensory, and autonomic nerve fibers. Motor NCV testing examines conduction on the motor nerve portion of the mixed nerve.

The key here is to be consistent in electrode placement and distal stimulation distance. The intensity of stimulation should be advanced incrementally until a maximal CMAP is obtained; this response represents the distal motor latency (DML, also termed the *M-wave*) and is a reflection of the ability of the fastest conducting axons to conduct the electrical stimulation to the neuromuscular junction (NMJ), bridge the junction, depolarize the muscle fibers that they innervate, and contract the muscle. Because there is no consistent way for clinical ENMG testing to accurately calculate the rate of conduction across and distal to the NMJ, motor conduction velocity measurement requires at least two stimulation sites; the second site is referred to as the *proximal latency* and is generally obtained at some common anatomical site to ensure consistency within the test and between individuals tested.

The examiner measures the linear distance between the two stimulation sites, then subtracts the difference between the two latencies and simply divides the latency difference into the distance between the two stimulation sites to obtain the NCV between the two sites (see Fig. 17-8):

$$\text{Nerve Conduction Velocity (m/sec)} = \frac{\text{Distance Between Proximal and Distal Stimulation Sites (mm)}}{\text{Proximal Latency} - \text{Distal Latency (msec)}}$$

Note that the millimeters and the milliseconds cancel each other out in this calculation, which leaves the unit of conduction velocity as meters per second. Often a third (or even a fourth) stimulation site is warranted to more thoroughly evaluate the motor nerve conduction status; for example, above and below the elbow for the ulnar nerve to identify cubital tunnel syndrome or above and below the fibular head to document fibular nerve compression at this site. Typical values for distal motor latency, motor conduction velocity, and amplitude of the CMAP for commonly studied upper-and lower-extremity nerves are noted in Table 17-1.

TABLE 17–1. Normal Motor-Conduction Values of Commonly Tested Nerves in Adults

Nerve	Distal Latency (msec)	Conduction Velocity (m/sec)	CMAP Amplitude (mV)
Median			
Wrist–APB muscle (8 cm) Elbow–wrist	< 4.2	> 45	> 4
Ulnar			
Wrist–ADM muscle (8 cm) Elbow–wrist	< 4	> 45	> 3.5
Radial			
Forearm–EIP muscle (8 cm) Mid-humerus–forearm	< 3.5	> 45	> 2.5
Fibular (Deep Peroneal) (7 cm)			
Ankle–EDB muscle Above fibular head–ankle	< 6	> 40	> 3.5
Tibial (10 cm)			
Ankle–AH muscle Knee–ankle	< 6	> 40	> 3

ADM = abductor digiti minimi; AH = abductor halluces; APB = abductor pollicis brevis; CMAP = compound motor action potential; EDB = extensor digitorum brevis; EIP = extensor indicis proprius

What the Findings Mean

Taking a closer look at the distal motor latency CMAP, the ENMG examiner is interested in the time from the stimulus artifact (the instant the signal is delivered to the nerve) to the initial deflection of the waveform from the baseline. As noted previously, this value in milliseconds represents the conduction along the nerve, across the NMJ, and to all the muscle fibers innervated by the axons stimulated. If sufficient demyelination has occurred, this latency value will fall above the upper limit of normal and will be considered slowed or delayed. As such, the latency values reflect the state of the myelin surrounding the largest and fastest conducting axons of the nerve under investigation. Typical DML values for commonly studied upper and lower extremity nerves are noted in Table 17-1.

Key Point! Note that an increase in latency reflects a decrease in conduction velocity.

The examiner is also very interested in the amplitude of the evoked response, which is measured in millivolts (mV) and is described as representing the number of functioning motor units innervated by the nerve being studied. Localized nerve compression (e.g., carpal tunnel syndrome) or diffuse disease processes, such as advanced diabetes, adversely affect the number of functioning motor units in a muscle, thereby leading to a decreased CMAP amplitude. Some computer-based ENMG testing units have software programs that can provide "area under the curve," which is actually a more accurate measure of functioning motor units for any given muscle. However, amplitude is a well-accepted representation of this nerve feature and is normally measured from peak to baseline. The lower limit of amplitude values are also shown for upper- and lower-extremity nerves in Table 17-1.

Duration is the length of time from the initial deflection of the signal from the baseline until it crosses the baseline again. This feature of the nerve response indicates the state of myelination and is excessively prolonged in certain demyelinating disorders. Related to duration is the process called *temporal dispersion,* which represents the electrical activity of individual nerve fibers that may be slower in conducting velocity. Temporal dispersion spreads the waveform out (prolonging the duration) and makes it less uniform in shape (Fig. 17-12).

Nerve conduction velocity values are usually established for each laboratory, although many facilities rely on nationally accepted norms to guide practice and decisions. The examiner generally compares the obtained NCV values from both limbs for internal consistency and against standard laboratory ranges to identify abnormalities. Nerves that have been injured by trauma or disease will not conduct signals at normal speeds. Often the injured segment is identified by carefully comparing sequential segments of each nerve studied.

Key Point! A reduction in NCV of more than 10 meters per second between consecutive segments of the same nerve would indicate a possible nerve injury.

It should be noted that conduction velocities in the upper extremities are roughly 10 meters per second faster than expected in the lower extremity. Velocities in the order of 45 to 50 meters per second are considered the lower limit of normal for upper-extremity nerves; a value of 40 meters per second is considered the lower limit for lower-extremity nerves. One reason for the lowered conduction velocity for lower-extremity nerves is perhaps related to the sheer length difference between upper and lower extremities, which results in a reduced limb temperature that has a known slowing effect on conduction velocity.

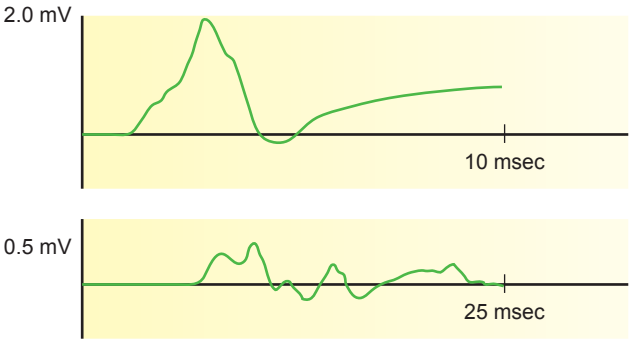


Fig 17 ■ 12 Temporal dispersion of the CMAP is caused by the relative difference in velocity of the fastest and slowest conducting fibers. Disproportionate slowing of some fibers occurs when nerves are compressed. The result is a CMAP that is increased in duration, reduced in amplitude, and less smooth in shape.

Which Nerves to Study?

Practitioners should be familiar with the routine motor nerve conduction techniques as they are most commonly performed and reported with normal values. The most commonly examined nerves and corresponding muscles are the following:

- Median nerve—electrodes on the abductor pollicis brevis
- Ulnar nerve—electrodes on the abductor digiti minimi
- Fibular nerve—electrodes on the extensor digitorum brevis
- Tibial nerve—electrodes on the abductor hallucis

Familiarization with the standard normal values for these four nerves will greatly enhance understanding in reading an ENMG report. Other nerves are less commonly studied but might include the following:

- Radial nerve—electrodes on the extensor indicis proprius
- Axillary nerve—electrodes on the middle deltoid
- Spinal accessory nerve (CN XI)—electrodes on the upper trapezius
- Femoral nerve—electrodes on the vastus medialis
- Fibular nerve—electrodes on the tibialis anterior

Sensory Nerve Conduction Study

Sensory nerve conduction studies are generally performed by placing a surface- or ring-recording electrode on the skin directly over a nerve trunk and stimulating the nerve to produce a sensory nerve action potential (SNAP) (Fig. 17-13). The SNAP is the summated response of all the depolarized sensory fibers in the mixed peripheral nerve in much the same way that the CMAP is the summated response of all the motor units responding to maximal electrical stimulation. However, there are some important differences that should be noted. The SNAP is a direct response having no intervening synapse like the NMJ for motor nerve conduction. Thus, a single point of stimulation provides sufficient information to measure a distal latency, obtain an amplitude, and (unlike motor conduction) attain a sensory conduction velocity.

Secondly, SNAPs (most often measured from peak to peak) are significantly smaller than CMAPs (Table 17-2).

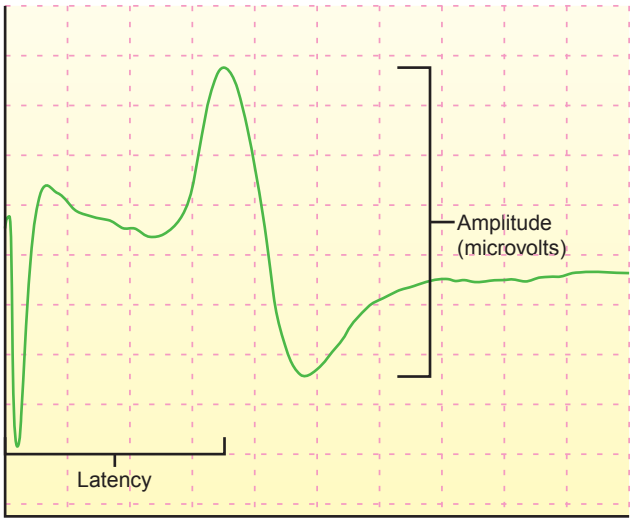


Fig 17 ■ 13 Typical sensory nerve action potential (SNAP) showing latency to the "takeoff," latency to the peak, and "peak-to-peak" amplitude. The amplitude of the sensory response (measured in microvolts) is significantly smaller than the CMAP amplitudes (measured in millivolts).

A typical CMAP may be on the order of 5 to 10 mV; a normal SNAP response may be as small as 5 to 10 microvolts (μV; remember that 1 mV = 1,000 μV). This represents a *1,000-fold difference,* so different sensitivity settings are required to obtain the much smaller SNAP response. The profound difference is because the CMAP is the summation of numerous muscle fiber action potentials that comprise several motor units. There are occasions when the sensory response is so diminutive that a special procedure called *signal averaging* is used to summate several responses and depict the summated waveform as a single response. This kind of additional signal processing is rarely needed for motor stimulation because the responses are usually large enough that it is unnecessary.

The depolarization produced by sensory nerve stimulation is propagated in both directions. As a result, the sensory nerve can be stimulated distally while recording proximally on the nerve trunk (the normal direction of afferent fiber depolarization). This is referred to as *orthodromic stimulation*. Conversely, the nerve can be stimulated at some proximal site, and the recording can be accomplished distally (the reverse of the normal direction of afferent fiber depolarization). This is referred to as *antidromic stimulation*. Both procedures are used by most examiners because the results are nearly identical for the two methods with no distinct advantage for either.

TABLE 17–2. Normal Values of Commonly Tested Sensory Nerves in Adults

Nerve	Distal Latency to Peak (msec)	Velocity (m/sec)	SNAP Amplitude (μV)
Median			
Wrist–digit (14 cm)	3.5 ± 0.5	50 ± 10	15–60
Palmar (7–9 mm)	< 2.5	—	> 10
Ulnar			
Wrist–digit (14 mm)	3 ± 0.5	50 ± 10	15–50
Palmar (7–9 mm)	< 2.5	—	> 10
Superficial Radial			
Forearm–wrist recording (12 cm)	< 3	50 ± 10	10–50
Superficial Fibular (Peroneal; 10–14 cm)	2.5 ± 0.5	45 ± 5	> 10
Sural (14 cm)	3.5 ± 0.5	45 ± 5	> 5

Key Point! *Orthodromic stimulation* refers to propagation of the electrical stimulus in the normal or customary direction. For motor testing, this is proximal to distal, while distal to proximal for sensory testing.

Procedure

The basic premise and setup for performing a sensory nerve conduction study is much like that of the previously described motor stimulation. Stimulation occurs by means of the adjustable stimulating probe placed over the course of the nerve, with the cathode (negative pole) positioned closest to the recording electrode. Of course, the examiner needs to determine whether to conduct the signal orthodromically or antidromically—unlike the motor study, which is always conducted orthodromically. The recording electrode pair can come in the form of a ring, often used for upper extremity nerve studies, or typical surface electrodes, used for both upper- and lower-extremity studies (see Fig. 17-7). The intensity of stimulation necessary to obtain a maximal response is usually less than that necessary in a motor study. For this reason, some examiners prefer to begin with the sensory portion of the NCS because it introduces the patient to the experience of electrical stimulation a bit more gently than the more intense stimulation necessary for most motor studies.

The parameters of interest with the sensory NCS are, in general, the same as those examined during the motor study and include the following:

1. Distal sensory latency (DSL)—the time from the stimulus artifact to either the onset or peak of the SNAP. As recording equipment has become more sophisticated and sensitive over time, the onset has become a more reliable parameter to measure. This was not true with most of the first- or even second-generation devices. Therefore, many examiners prefer to report both onset and peak latency values for SNAPs in their analysis. The accepted values for these various nerve responses have been established over predetermined distances between stimulation site and recording electrode.
2. Amplitude—a reflection of all the individual SNAPs summated into a compound SNAP. This parameter informs the examiner of the general function of the nerve axons in the segment studied. It is also reflective (by inference) of some important conclusions. Sensory nerve cell bodies are located in the dorsal root ganglion within the intervertebral foramen. Lesions located proximal to the ganglion (principally, radiculopathies) do not usually affect the cell body; consequently, the SNAPs are ordinarily normal. By contrast, brachial or lumbar plexopathies and other neuropathic conditions often result in reduced amplitude or absent SNAPs. As such, sensory nerve studies can be useful for delineating proximal or distal causes for sensory complaints. For instance, a lumbar radiculopathy can cause numbness, but the sural nerve SNAPs are invariably normal because the lesion (usually a disk herniation) is proximal to the dorsal root ganglion; therefore, the intact nerve cell body keeps the nerve and the distal responses normal.
3. Waveform, duration, and nerve conduction velocity are additional parameters that might be evaluated for

a sensory nerve study, but practically speaking, the DSL and amplitude are the most commonly reported values for most sensory nerves.

Which Nerves to Study?

Much like the motor conduction studies, there are basic nerves that tend to be studied routinely along with a host of less commonly studied sensory nerves. The more commonly examined sensory nerves include median, ulnar, radial, medial antebrachial cutaneous, sural, superficial peroneal, and saphenous nerves. However, there are times when specific additional nerves may need to be studied to answer a clinical question. For instance, if a referring physician was concerned that a patient was experiencing dysesthesia on the lateral side of the proximal thigh, symptoms consistent with a condition called *meralgia paresthetica,* failure to study the lateral femoral cutaneous nerve would prevent the electroneuromyographer from adequately establishing the potential source of the symptoms.

Central Conduction and Long-Loop Responses: F-Wave and H-Reflex

Although motor and sensory nerve stimulation studies as described thus far are the most common procedures undertaken during nerve conduction testing, other techniques have become somewhat routine because they are relatively easy to accomplish and provide additional information about the status of the nerve complex.

F-Wave

With motor conduction studies, we described a procedure whereby the motor nerve is stimulated proximally to obtain an orthodromically derived distal motor latency, also referred to as the *M-wave*. Simultaneously, the nerve conducts an action potential antidromically to the anterior horn cells in the spinal cord that, in turn, send back a small action potential that contracts some of the muscle fibers under the recording electrode on the distal muscle. This delayed, small action potential is referred to as the *F-wave,* so named because it was originally recorded from the foot muscles (Fig. 17-14). F-wave latencies are usually smaller than 500 μV. Because they must traverse the distance of the entire limb length twice, they are much longer than the M-wave latencies (from 20 to 32 msec for upper extremities and 42 to 58 msec for lower extremities).

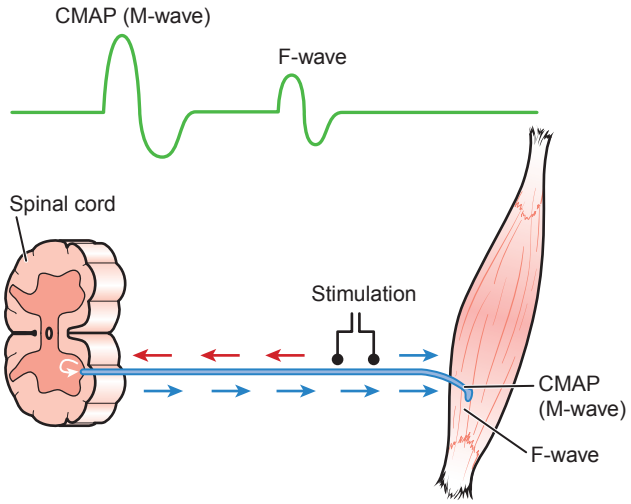


Fig 17 ■ 14 The F-wave is a long-loop response produced by stimulating the nerve at a distal site. The stimulus proceeds bidirectionally and produces the M response (DML) and also sends a volley proximally to the spinal cord. There, a number of anterior horn cells are activated, which then send a small response back to the distal muscle to be recorded as the F-wave. This response is slowed in conditions that affect the overall conductivity of the nerve (various neuropathies) and may also be slowed with focal demyelination or axonopathy in conditions affecting the nerve roots or the plexus.

The limb's length clearly will significantly impact this delayed response, so the patient's height (and limb length itself in some laboratories) is recorded for reference to normal values for these latencies.

The usual technique for this procedure is virtually identical to that for obtaining the DML, except that the cathode and the anode of the handheld stimulator are reversed so the primary action potential proceeds toward the spinal cord rather than toward the distal muscle. Besides having a much smaller response than the M-wave, the presence and shape of the F-wave is variable. Consequently, it is routine to obtain a number of F-wave responses (5 to 10, typically) and report the shortest latency as the accepted value. Because the series of 5 to 10 consecutive F-waves are obtained in response to maximal stimulation, this is one procedure that patients often find uncomfortable during the NCS.

The principal purpose for obtaining an F-wave response is to examine nerve conduction in proximal nerve segments for conditions like thoracic outlet syndrome, some radiculopathies, or diffuse demyelinating diseases. For patients who have significant distal neural compression (e.g., carpal tunnel syndrome), a prolonged F-wave does not contribute any specific information regarding the location of compression that is not already demonstrated with the normal DML and NCV testing. However, if a

patient demonstrates normal distal latencies and nerve conduction and yet exhibits prolonged F-waves, this strongly suggests that compromise of the neural system is located at a more proximal site such as the plexus or nerve root.

H-Reflex

Sometimes the standard nerve conduction and EMG examination does not identify a clear electrophysiological explanation for a patient's pain or numbness and tingling in a radicular pattern. More subtle tests of neural compromise may be useful at such a time. One such example is the *H-reflex*. This is an action potential response first described by Hoffman in 1922 and later named the H-reflex by Magladery and McDougal in 1950. It is most often undertaken for the tibial nerve when an S1 radiculopathy is suspected, although it is gaining some acceptance for the median nerve when a C7 nerve compression is suspected. A few investigators have described a femoral nerve H-reflex, but it has not become a routine test for ENMG examination.[16]

As noted previously, when an electrical stimulus is applied to a mixed motor and sensory nerve, action potentials are propagated bidirectionally. In the case of the tibial nerve H-reflex, an action potential will travel from the point of stimulation (popliteal space) to the spinal cord, where it synapses with an alpha-motor neuron in the anterior horn. The activated motoneuron results in an action potential propagated back to the peripheral muscle, in this case the medial head of the gastrocnemius, an S1 innervated muscle (Fig. 17-15). The time from the stimulus artifact to the muscle action potential is the H-reflex and ranges from 26 to 33 msec in most individuals. Like the F-wave, the H-reflex is also a limb length–dependent response. Nomograms are available that factor in age and height (limb length) for this response to see if the values obtained for the patient are falling within a predicted range.

The H-reflex is a very consistent reflex and represents the same pathway as the monosynaptic deep tendon reflex for the ankle that is elicited clinically. Although consistent, it is somewhat sensitive to voltage variation and is usually elicited in response to a submaximal, rather than a maximal, stimulation delivery. In fact, in many cases, a delivery of maximal levels of stimulation will cause the H-reflex (elicited by submaximal stimulation) to disappear while the M-wave dominates the screen. This

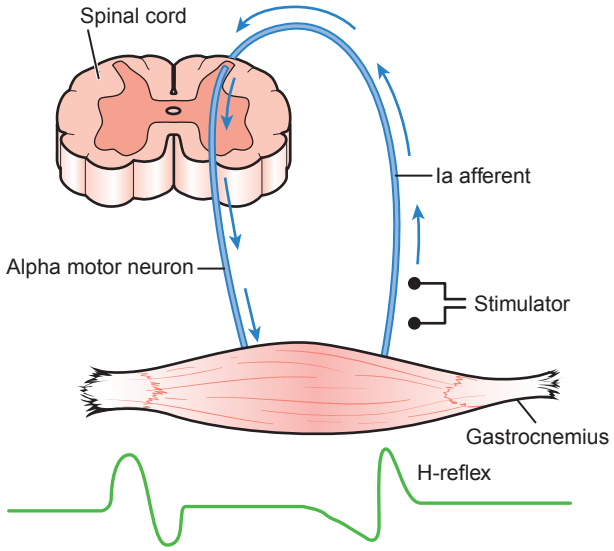


Fig 17 ■ 15 The H-reflex is another long-loop response evoked by stimulating the tibial nerve at the popliteal fossa and directing the signal toward the spinal cord. From there, a monosynaptic response sends a signal back to the recording site on the medial gastrocnemius muscle and is recorded as the H-reflex. This response is slowed in S1 radiculopathies, often when no other abnormalities are noted on the nerve conduction study or the EMG.

narrow window between achieving threshold for eliciting the H-reflex and obscuring the response by applying stimulation levels that are too high can make obtaining this response a challenge for the examiner. However, the information obtained with this procedure is important for corroborating the presence of conditions such as S1 and C7 radiculopathies and is worth the effort in most cases. To reiterate, the procedure for obtaining a tibial nerve (S1) H-reflex is as follows:

1. Place the active surface electrode on the medial head of the gastrocnemius muscle belly and place the reference electrode on the tendon of the muscle.
2. Stimulate the tibial nerve at the popliteal space with the cathode pointed toward the spinal cord.
3. Slowly increase the stimulus amplitude until a depolarization response is first noted. Continue increasing the amplitude until a maximal H-reflex is obtained.
4. Compare the obtained H-reflex value to:
   a. The contralateral H-reflex
   b. A predicted H-reflex normalized for age and limb length
5. Determine whether the difference between obtained and predicted H-reflex values exceeds acceptable variation (usually a difference of 1 msec is considered the acceptable variation).

Height and limb length are important factors when conducting long-loop latency responses (i.e., H-reflex, F-wave). The length of time for an electrical signal to depolarize the nerve in the direction of the spinal cord, synapse with an anterior horn cell that sends a signal back to the peripheral muscle (H-reflex), or activate a group of anterior horn cells to depolarize and send a signal to the peripheral muscle (F-wave) is dependent on the distance the signal must travel to accomplish such electrical events. In fact, nomograms have been devised in which an individual's height or limb length and age can be factored to arrive at a predicted latency value.

Coming to Some Conclusions: What Do We Know So Far?

At this point in the examination process, the motor and sensory conduction studies already described have been completed and the special responses of F-wave and H-reflex have been obtained. It is possible that the findings up till now require other nerves to be examined or even additional limbs (upper or lower, in the case of possible polyneuropathy) to be analyzed. Furthermore, we have not described the needle EMG portion of the examination or the findings from such a study.

In any event, this is a good time to evaluate what we know so far. First of all, beginning the process of analysis and determining an ENMG diagnosis is based on accurate awareness of normal ranges and values for the responses obtained during the NCS. An abridged list of typical latency, nerve conduction velocity, and amplitude values are noted in Table 17-1. The data collected in the study needs to be compared to these normal values to determine the status of the nerve.

When nerves are injured by disease or compression, their ability to conduct electrical signals is diminished or abolished. This abnormality, referred to as a *conduction block,* is what nerve conduction studies measure. A conduction block is present when the CMAP elicited from a more proximal site has less amplitude than the CMAP elicited at a distal site. This indicates that some of the fibers along the course of the nerve failed to conduct a signal but that the axons and myelin are normal distally. NCS is used to help determine if a patient's neural complaints are caused primarily from demyelination (e.g., nerve compression or diffuse disease), from axonal degeneration (e.g., dying back neuropathy, severe axon injury from compression), or from some combination of these two processes.

Key Point! The primary indicator of demyelination is prolonged distal latencies and slowing of nerve conduction velocity. The principal feature of axonal degeneration is reduced amplitude and area under the curve of the CMAP or SNAP.

Clinically, the most common categorization scheme for neural injuries was first described by Seddon in 1943 and includes neurapraxia, axonotmesis, and neurotmesis[17] (Fig. 17-16). *Neurapraxia* is the mildest form of peripheral nerve disorder and is characterized by a local conduction block without any axonal injury. We have all experienced very transient versions of this category when we have slept on our arm for a short period of time and then were unable to feel or move the limb for several seconds to a couple of minutes while blood flow was reestablished to the nerves and muscles. More serious neurapraxias are

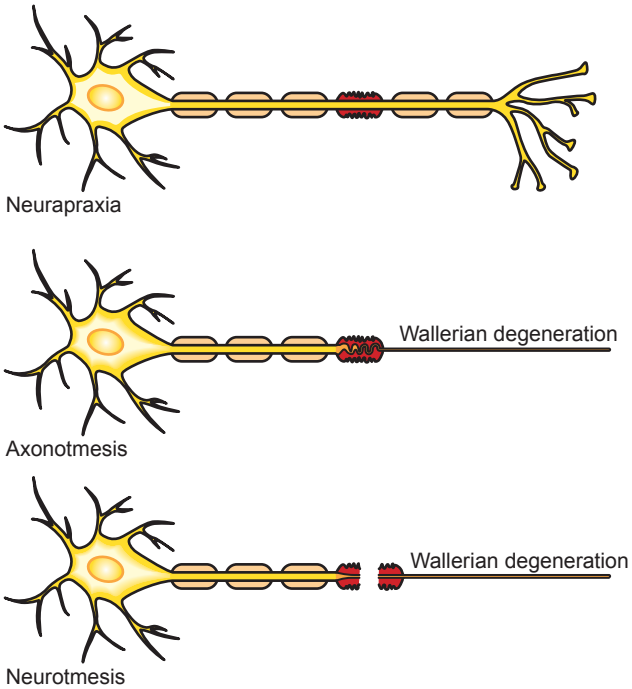


Fig 17 ■ 16 Standard description of nerve injury categories include neurapraxia (a myelin injury or condition), axonotmesis (axonal degeneration and myelin injury in most cases), and neurotmesis (myelin, axon, and connective tissue injury). Both axonotmesis and neurotmesis result in Wallerian degeneration, but neurotmesis is a far worse injury because the connective tissue sheath injury makes nerve regeneration less likely.

caused by sustained or intense nerve pressure, stretching, or focal inflammation that are believed to cause localized demyelination. Strictly speaking, the axons and surrounding connective tissue sheath of the nerve are intact with a neurapraxia.

Classic examples of this category of nerve injury are the occasional tourniquet palsy associated with compression during surgery and the ever popular "Saturday night palsy," named for the compression neuropathy of the radial nerve at the spiral groove of the humerus (usually caused by sustained focal nerve pressure from a long period of sleep secondary to inebriation or drug abuse). The effect on the electrophysiological examination is reduced nerve conduction velocities and proximal CMAPs (those obtained by stimulation above the focal demyelination). In some severe cases, conduction is blocked entirely, resulting in no response to stimulation across the injury site. If a pure neurapraxia has been sustained, the recovery usually takes weeks and occurs when remyelination of the large diameter axons is complete, or nearly so. If recovery takes several months, then it is possible that a second level of nerve injury, axonotmesis, has occurred.

Axonotmesis lesions are caused by more severe nerve pressure, stretching, or inflammation and are the result of a disruption of axonal continuity leading to some level of Wallerian degeneration (axonopathy). Interestingly, research has shown that this axonal degeneration occurs distal to the lesion and one node of Ranvier proximal to the lesion. However, the connective tissue layers (epineurium, perineurium, endoneurium) remain intact with an axonotmesis, so the opportunity for recovery is possible. The clinical effect of this injury is diminished, or there is loss of sensation if sensory nerves are involved and muscle weakness if motor axons are injured.

Electrophysiologically, the effect of axonotmesis is virtually identical to that of neurapraxia for the first 2 to 5 days following the lesion. Specifically, the NCS is normal below the level of the injury, but there is no response across the axons that have experienced conduction block. After this 2- to 5-day period, axonal degeneration occurs, and the involved axons do not respond to stimulation above or below the lesion. If a sufficient number of axons are involved, the amplitude of motor- and sensory-evoked potentials (CMAP, SNAP) will be reduced, although the conduction velocity will approximate the normal rate because the myelin (especially of the large fibers) is still intact.

Key Point! Provided that the connective tissue sheaths are intact, the prognosis for recovery from an axonotmesis is reasonably good, although not nearly as hopeful as that for neurapraxia. Recovery usually takes between 3 and 12 months for an axonotmesis and, generally, is an incremental process, proceeding at about 1 mm per day in most cases.

The most severe category of nerve injury is referred to as a *neurotmesis* and involves not only axonal degeneration, but also injury to some aspect of the connective tissue sheath around the nerve. The electrophysiological effects of a neurotmesis are indistinguishable from an axonotmesis with immediate loss of conduction ability across the lesion and loss of distal excitability over a 2- to 5-day period following the injury. What distinguishes neurotmesis from axonotmesis is the fact that the former shows no improvement with follow-up (serial) testing over time, indicating the absence of reinnervation of the injured neural structure. Because reinnervation by normal nerve regeneration processes is so tenuous for neurotmesis lesions, surgical exploration and repair may be necessary to extend any hope of recovery. Fortunately, most neural injuries do not fall into this category, so the prognosis for recovery is fair or good for most patients.

Additional techniques can identify within 1 cm the precise location of the conduction block, which is valuable information for the surgeon if surgical exploration and compression relief is contemplated. One such procedure is called the *inching technique* and involves the same setup as described for the motor conduction study but with additional stimulation sites at every centimeter from primary sites below and above the elbow. For a 12-cm segment, as noted in this brief case, the examiner would have 12 separate responses to stimulation to evaluate.

This case also underscores the limitation of the nerve conduction study because the presence of axonal degeneration is not clearly determined unless an EMG is also conducted to identify signs of abnormality associated with axonotmesis.

CASE STUDY 17-1 Nerve Conduction Velocity

JD is a 48-year-old male training for his first triathlon. For 6 weeks, he has had increasing right dominant elbow pain and numbness in the ulnar nerve distribution. He especially notices these symptoms when he is training for long periods of time on his bike. In addition, JD mentions that fastening the buttons on his shirt has become difficult because of numbness and clumsiness with his hands. He has no family history of nerve disorders, is not diabetic, and is 6 feet tall.

Clinical exam shows decreased two-point discrimination in the ulnar nerve distribution, a positive Tinel sign over the ulnar nerve in the cubital tunnel, and a mildly positive Wartenberg's sign (indicative of weakness of ulnar-innervated hand intrinsics). Further, he demonstrates tenderness over Guyon's canal in the hand, which is the area that he rests his weight upon during long training bike rides. Cervical spine screening exam is unremarkable. Thoracic outlet tests do not provoke any diminution in radial pulse.

RATIONALE FOR NERVE CONDUCTION STUDY

This patient has signs and symptoms of ulnar nerve compression at the elbow (i.e., cubital tunnel syndrome), although compression of the ulnar nerve at Guyon's canal, fairly common in competitive cyclists, may also explain the hand symptoms and intrinsic muscle weakness. Obtaining information from a nerve conduction of the upper extremities will help to localize the source of the problem more definitively, rule out the possibility of comorbidities (e.g., simultaneous ulnar nerve compression at the elbow and the wrist), and provide a more sound basis for therapeutic intervention and advising the patient about future effects of training.

NCS PLAN

Conduct bilateral upper-extremity nerve conduction studies; this includes motor, sensory, and long-loop responses (F-waves, H-reflexes) for the median and ulnar nerves.

FINDINGS

1. Median motor and sensory latencies and conduction values, F-wave, and H-reflexes are normal bilaterally.
2. Left ulnar nerve motor and sensory conduction values are normal.
3. Right ulnar nerve distal motor and sensory latencies are normal; the conduction velocity from below the cubital tunnel to the wrist is also normal.
4. Right ulnar nerve conduction velocity across the elbow drops to 35 meters per second, which is below the lower limit of normal for conduction (45 meters per second); amplitude of the CMAP drops from 8 milliamps (mA) in the wrist and forearm response to 3 mA for the across-the-elbow segment; right ulnar nerve F-wave is mildly prolonged.
5. Right ulnar nerve sensory conduction across the elbow is 27 meters per second, well below the lower limit of normal.

INTERPRETATION

1. Findings are consistent with ulnar nerve compression at the elbow; moderate severity.
2. The nerve conduction slowing and reduced amplitude of the CMAP across the elbow indicates involvement of myelin (neurapraxia), but definitive evidence of coincidental axonotmesis (axonal degeneration) is not established on the basis of nerve conduction findings alone. Proximal neural compression (nerve root compression, brachial plexopathy) does not seem likely on the basis of these findings but cannot be strictly excluded without additional testing.

PROCEDURES

The portion of the NCS that provided the most useful diagnostic information was the conduction study of the right ulnar nerve. As noted in the illustration, the recording electrode is placed on the abductor digiti minimi muscle, and nerve stimulation is carried out at a minimum of four locations:

1. Wrist: proximal to Guyon's canal
2. Distal to the elbow (just below the cubital tunnel)
3. Proximal to the elbow (just proximal to the cubital tunnel)
4. Axilla

A latency is obtained at each stimulation site. The distance between the contiguous sites is measured with a tape measure. In addition, the amplitude and shape of the CMAP is noted and recorded at each stimulation site. For the sake of simple computation, the distal motor latency (the one obtained by stimulating the ulnar nerve at the wrist) is 3 msec, and the latency just distal to the elbow is recorded as 7 msec—a difference of 4 msec. The distance between these two sites is 24 cm (240 mm). So according to the formula, our conduction velocity is obtained by dividing the difference (4 msec) between the two latencies into the distance between the two sites (240 mm), which yields a value of 60 meters per second—well within the

CASE STUDY 17-1 Nerve Conduction Velocity—cont'd

normal range of 45 to 70 m/sec. The across-the-elbow segment is 12 cm in length (120 mm), and the latency difference between the stimulation sites above and below is 2.92 msec. Dividing 2.92 msec into 120 mm yields a conduction value of 35 m/sec—well below the lower limit of the acceptable range (45 m/sec). In addition, the amplitude dropped precipitously from 8 milliamps (mA) below the elbow to 3 mV above the elbow site—evidence of partial conduction block.

The final segment of the motor study involves stimulating the ulnar nerve in the axilla and recording the distance between this new site and the one just above the elbow. Here we obtain a latency difference of 2 msec, and the distance is 12 cm (120 mm) once again. This results in a return to normal conduction of 60 m/sec, but the amplitude remains at 3 mV because the signal must go past the area of compression to reach the target organ where the recording electrodes are located (abductor digiti minimi [ADM] muscle of the hypothenar eminence).

The F-wave for the right ulnar nerve is 38 msec—prolonged for a 6-foot individual and significantly longer than the ipsilateral median nerve F-wave value of 29.8 msec. The sensory conduction for ulnar nerve follows a similar course with normal distal values (below the level of the compression) and slowed and reduced amplitude responses across the elbow, lending further evidence to the conclusion that the ulnar nerve is compressed at the cubital tunnel.

CLINICAL ELECTROMYOGRAPHY

Certain features of the second part of the ENMG—the needle EMG—provide important complementary information for clinical decision-making. Up to this point, the nerve has been artificially stimulated in order to evoke certain motor and sensory potentials, but now the interest is to monitor and explore the muscle-nerve complex without this externally applied stimulus. Although it is possible to examine basic muscle contractility by means of surface EMG electrodes, this technique provides very little diagnostic information about the motor unit. By contrast, a pin electrode inserted directly into the muscle belly yields a wealth of information, especially about the status of the motor unit. The one big disadvantage of needle EMG, of course, is the typical discomfort associated with the examination. Patients are sometimes comforted by being informed that the electrode is very thin (28 gauge in the case of many monopolar needles) and is coated by Teflon so it moves as smoothly as possible through the muscle. Nevertheless, this portion of the exam is somewhat uncomfortable but is generally very tolerable when conducted well.

The basic equipment needed to conduct an EMG examination is the same as that used for the NCS, except that no externally applied electrical stimulation is involved. There are a variety of needle types that can be used, such as a concentric, bipolar, or single-fiber needle, but the Teflon-coated monopolar electrode is by far the most common (see Fig. 17-6). There are specific reasons to use the previously mentioned electrodes, but we will restrict our discussion to the typical findings using the Teflon electrode, because that is the choice of most practitioners conducting ENMG examinations.

What Can Be Learned by Needle EMG That Has Not Already Been Determined by the NCS?

Although the NCS can reveal a demyelinating lesion, such as a focal nerve compression of the fibular nerve at the fibular head, NCS cannot determine the particular status of the motor units. Specifically, the NCS cannot accurately tell whether some axons have been injured in addition to the focal demyelination. Clinical testing is often not sensitive enough to identify subtle neural injury that may be causing symptoms.

This presents a conundrum that EMG examination is able to elucidate because the intramuscular nature of the study allows it to depict the discrete characteristics of individual motor units. On rare occasion, a patient with various forms of denervation develops fasciculations that are visible to the naked eye.

Key Point! It has been estimated that a patient with 50% loss of motor units can still provide two or three contractions graded as "normal" by voluntary muscle testing, although the patient often complains of fatigue or weakness.

To assess the integrity of the motor unit, it is necessary to examine the muscle with an electrode in very close proximity to the injured motor units. This can only be done with a needle electrode. Furthermore, in many conditions affecting the neuromuscular system, the nerve conduction study does not identify a clear location of the problem, nor does it accurately delineate the cause of the complaints. For instance, a lumbar radiculopathy often results in complaints of pain, numbness, and tingling. The clinical exam demonstrates subtle motor weakness in the lower extremity. Unfortunately, the motor and sensory NCS is invariably normal and sheds no delineating light on the location of the problem. However, the needle EMG exam can often identify patterns of partial denervation that correspond to a nerve root. The pattern of EMG muscle involvement confirms the probable location of the problem, identifies the severity of individual muscle involvement, and provides a hint at the prognosis.

To summarize the kind of information the needle EMG examination provides, we will provide standard clinical questions answered by the procedure. Robinson and Kellogg[13] have identified several such questions:

1. Is the muscle normally innervated, partially innervated (which means, if true, it is also partially denervated), or completely denervated?
2. Is there evidence of motor unit recovery, which means reinnervation is taking place?
3. Do the specific findings tend to be more consistent with a neuropathic or myopathic disorder (often determined by the nature of the motor unit recruitment pattern)?
4. Is the pattern of EMG abnormality most consistent with involvement of:
   a. Anterior horn cells (polio, ALS)
   b. The nerve root (herniated nucleus pulposus, tumor)
   c. The plexus (stretch, compression, tumor)
   d. Mixed nerves (cubital tunnel syndrome)
   e. Neuromuscular junction disease (myasthenia gravis)
   f. A myopathic disorder (facioscapulohumeral muscular dystrophy)
5. If a neuropathic condition is suspected, what is the location of the lesion?
6. Finally, does the pattern of muscle involvement correspond to a posterior primary rami distribution (paraspinal muscles), an anterior primary rami (extremity muscles), a combination of both, or is there cranial nerve involvement (such as seen with anterior horn cell disease like ALS)?

Just as certain principles guided the nerve conduction study, similar principles guide the EMG examination. These principles are predicated on the examiner having committed to memory which nerve root levels and specific nerves innervate individual muscles in the limb. The specific principles include:

1. Examining several muscles above and below the suspected site of the pathology. The muscles studied during an EMG examination are chosen for their likelihood of contributing information to the eventual or suspected diagnosis. If a nerve root injury is suspected, then the practitioner is required to choose muscles that would confirm and disprove this possibility. Alternatively, if a peripheral nerve lesion is anticipated, muscles supplied by other peripheral nerves than the one suspected must be examined along with muscles supplied by the nerve root levels that contribute to the peripheral nerve in question. This ensures that the examiner's conclusion from the EMG study is correct. In other words, the planning of the EMG exam (just as the planning of the NCS) must be done to avoid errors of clinical reasoning.
2. Examining muscles innervated by other nerves in the same extremity (as explained in No. 1 above).
3. Sampling several sites in each muscle tested (to obtain accurate representation). This sounds more uncomfortable than it actually is. The sampling should be done so that several motor unit regions can be accessed by means of redirecting the tip of the needle electrode to various depths in the muscle (Fig. 17-17).
4. Preparing to examine muscles in the contralateral extremity or upper/lower extremity. If the findings during the EMG examination indicate a more pervasive explanation for the patient's symptoms than was first suspected (e.g., a polyneuropathic process rather than a mononeuropathic lesion), then the examiner should extend the testing to include other limbs in the study to confirm or rule out this possibility.
5. Recalling proper time frame to conduct study to maximize information provided by the exam.

This last point needs some elaboration because it is often not well understood by referring sources for patients with apparent neuromuscular involvement. If a patient has a sudden onset of hand numbness and is referred for "EMG/NCS" just days after the first presenting symptoms occur, it is possible that the NCS may show some

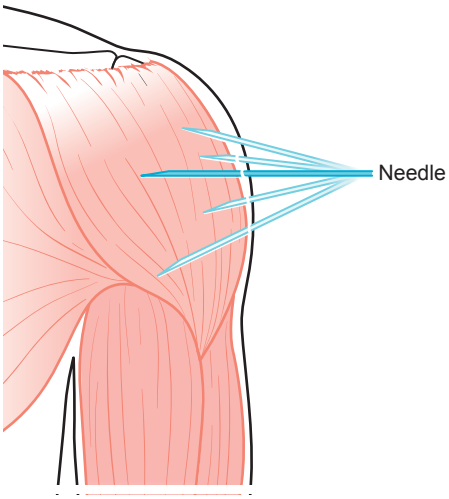


Fig 17 ■ 17 An example of examining various "quadrants" of the same muscle belly region through one insertion site. This limits the number of times the highly innervated investing deep fascia must be pierced to conduct a thorough EMG examination.

early signs of neural injury, but the EMG will not show abnormality until approximately 3 weeks. The time-honored standard time frame for signs of denervation to develop is 21 days following onset of symptoms. This is an average value because it is entirely possible for muscles in very close proximity to the site of a nerve injury to demonstrate EMG abnormalities as early as 10 to 14 days following onset of symptoms (e.g., paraspinal muscles with a nerve root compression). It is usually best to delay either the entire test or at least the EMG portion of the study until more accurate results are likely to be obtained.

Because the needle EMG study is not entirely pleasant, it is preferable not to conduct this portion of the exam when little or no diagnostic information is likely to be gained. By the same token, ENMG studies conducted many months after the onset of symptoms and signs of neural involvement may yield little helpful information to guide the practitioner's clinical decision-making. The reason for this is that a neural injury is generally followed by attempts at neural repair, which eventually cloud the electrophysiological picture. For example, 4 months after a disc herniation in the lumbar spine, the electrical evidence of denervation (spontaneous potential) has largely dissipated and been replaced by more subtle changes in motor recruitment characteristics. These alterations in recruitment profile are much harder to quantify and often do not proceed at the same rate in the various muscles that were formerly denervated. Meanwhile, the patient continues to have complaints of numbness, tingling, and pain in a radicular pattern. Although there are good clinical reasons to conduct ENMG examinations even at a late date, the clearest information provided by this test is obtained when the examination is performed in the optimal time frame.

A peculiar feature of EMG testing is not considered during the NCS portion of the examination—the *sound* of the responses. There is a very distinct sound of the responses to needle provocation and voluntary activation. The characteristic sounds are associated with each of the four segments of the EMG examination described here, listed in the order in which they are ordinarily conducted in a typical patient testing session:

1. Insertion
2. Rest
3. Minimal activation
4. Maximal activation (recruitment)

Insertion

When a needle electrode is first inserted into the muscle, there is a brief burst of electrical activity that corresponds to the electrode's movement; this is a normal response. Ironically, this activity has historically been referred to as *injury potentials,* although now the most common designation is *insertional activity*. It should last between 50 and 300 msec, although some practitioners doing this test include as much as 500 msec in the normal range. In any event, normal insertional activity ends abruptly when the electrode movement ceases. If the muscle membrane is more irritable than it should be, there will be prolonged depolarization that can continue far beyond this upper limit of 500 msec.

In cases of severe denervation in which the muscle membrane is extremely unstable, the insertional activity (membrane depolarization) will continue unabated. By contrast, sometimes the insertional activity is diminished or nearly absent. In most cases, this is the result of long-standing denervation and represents the effect on electrical activity of connective tissue or fatty infiltration into the muscle. Terms used to delineate the characteristics of the insertional activity include *normal, increased, sustained, decreased,* or *absent.* Abnormalities in insertional activity are not pathognomonic of any particular neural condition; they simply indicate an abnormality of the muscle membrane stability.

Most patients find the EMG examination uncomfortable because a needle electrode is used to obtain the signals. Nevertheless, to obtain a representative sample of the muscle's state, several sections of each muscle included in the study should be examined. In addition, three to four levels of depth should be explored, with each section of the muscle to be studied. The discomfort associated with the sheer number of needlesticks required to obtain reliable and representative information using these sampling criteria would be overwhelming for a majority of patients. Fortunately, multiple sections (quadrants) of the muscle can be accessed through a single needle insertion by carefully directing and redirecting the tip of the electrode to various regions to obtain a valid sample (see Fig. 17-17).

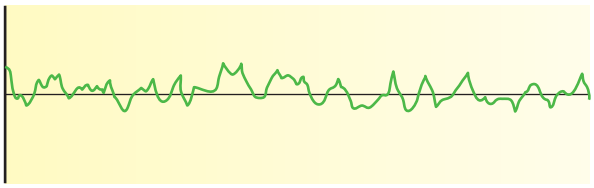
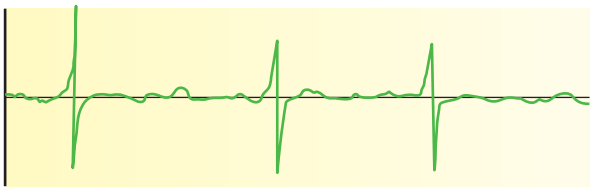
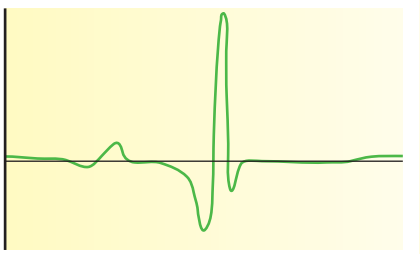
Rest

After assessing the insertional activity of a muscle quadrant, the examiner will "rest" (cease needle movement) to see if the muscle will return to electrical silence or whether spontaneous potentials will appear. At rest, the normal state of the motor unit and muscle fibers is complete electrical silence. One form of spontaneous electrical activity is entirely normal and is thought to occur when the tip of the electrode is near the neuromuscular junction and is producing a localized, transient depolarization. These rapidly firing potentials (2,000 to 3,000 Hz) are of very low amplitude (10 to 50 μV) and are characterized by an initial upward directed spike (negative); they are referred to as *miniature end-plate potentials* (MEPPs; Table 17-3).

The sound produced by this spontaneous activity is much like that heard when holding a large seashell to the ear. These potentials occur only in the end-plate zone, which is usually found in the middle of the muscle belly, the prime target area for needle examination.

Key Point! Note this oddity of EMG convention: For ENMG signals, upward deflections are designated as negative and those that are directed downward are considered positive. Although this is not in keeping with the usual designation in other areas of scientific exploration (usually we think of up as positive and down as negative), it has been the practice for more than 60 years of ENMG history and is not likely to change in the near future.

TABLE 17–3. Characteristics of Normal and Abnormal Potentials During Electromyography

	Wave	Image
Normal Potentials	Miniature end-plate potentials	
	End-plate spikes	
	Motor unit potential	

Another form of normal spontaneous potential is referred to as *end-plate spikes,* or *end-plate noise* (see Table 17-3). Like the MEPPs, the initial deflection for these potentials is generally upward (negative), although they are much larger—ranging from 100 to 200 μV. They are very short duration (1 to 4 msec) and usually fire at a slightly higher rate (100 to 300 Hz) than the abnormal wave with which they are most often confused: fibrillation potentials. The distinguishing difference is the initial upward deflection for end-plate spikes in contrast to the downward deflection for fibrillation potential. A peculiar feature of end-plate spikes is that they are invariably painful; fortunately, a very slight adjustment of the needle electrode tip will relieve the symptoms and usually oblate the end-plate signal.

Common forms of abnormal spontaneous potentials include:

- Positive sharp waves
- Fibrillations
- Fasciculations
- Complex repetitive discharges
- Myotonic discharges
- Myokymic discharges

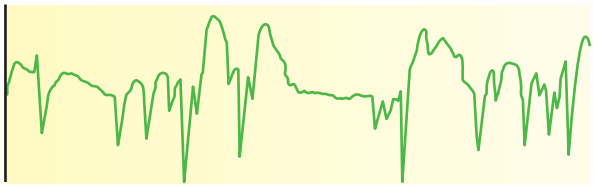
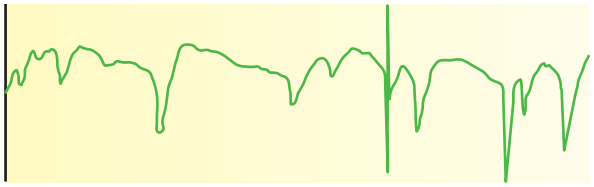
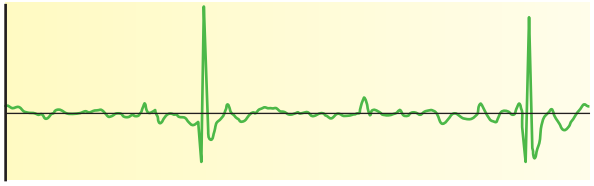
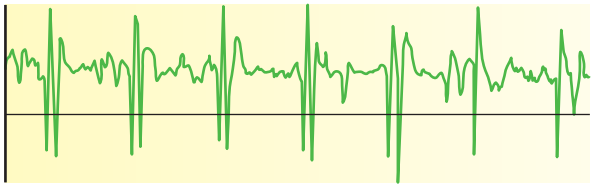
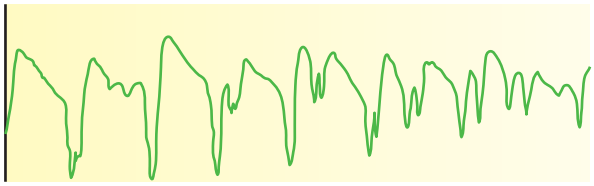
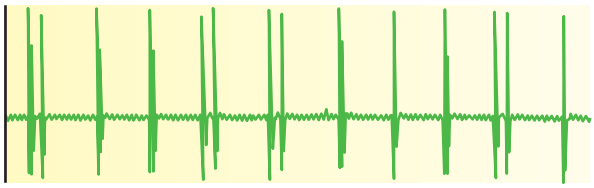
As their name suggests, *positive sharp waves* (PSWs) are positive-directed potentials, although this initial phase is followed by a low-amplitude, comparatively long-duration negative phase (see Table 17-3). The amplitude range noted in the literature is from as little as 10 μV to as high as 1,000 μV (1,000 μV = 1 mV). The rate of discharge is regular and ranges from 1 to 200 Hz. PSWs have a characteristic sound similar to a dull thud, much like that of a motor boat engine at low idle. Positive sharp waves result from an abnormally sensitive muscle membrane and probably represent the depolarization of a single muscle fiber, although their exact etiology has not been completely delineated. A motor unit firing from some distance may initially appear as a positive sharp wave, but three clues should alert the examiner to avoid this error in interpretation:

1. There is no negative initial deflection with a PSW, although this is often the case with a distant firing motor unit.
2. A motor unit's rate of firing is usually quite a bit lower in frequency compared to a PSW and is fairly rhythmic.

Amplitude	Rate	Duration	Sound	Characteristics
10–50 μV	2,000–3,000 Hz	0.5–1 msec	Large seashell held to the ear	Normally occurring insertional activity. Found only in the end-plate zone. Initial upward directed spike (negative).
100–200 μV	100–300 Hz	1–4 msec	Low-level murmur	Normally occurring insertional activity. Initial deflection upward (negative).
250 μV to 5 mV	Onset of 5–15 Hz up to 60 Hz	5–15 msec	Sharp, crisp discharge	Bi- or triphasic waves

Continued

TABLE 17–3. Characteristics of Normal and Abnormal Potentials During Electromyography—cont'd

	Wave	Image
Abnormal Spontaneous Potentials	Positive sharp waves	
	Fibrillations	
	Fasciculations	
	Complex repetitive discharges	
	Myotonic discharges	
	Myokymic discharges	

Amplitude	Rate	Duration	Sound	Characteristics
10 μV to as high as 1,000 μV	1–200 Hz	< 10 Hz up to 100 msec	Dull thud, like motorboat engine	Biphasic initially positive with long, low amplitude negative phase
20 μV to > 1 mV	1–30 Hz	< 5 msec	Rain on a tin roof	Initial positive deflection
Similar to normal motor unit potentials	1 Hz to 1 every few sec	Similar to normal motor unit potentials	Popping sound	Considered to be an involuntary motor unit firing. May be normal but considered abnormal if attended by other forms of spontaneous potentials.
Few to several hundred μV	20–100 Hz	10–50 msec	Machine gun firing	High-frequency polyphasic potentials initiated by needle movement or tapping, waning to low frequency. Not specific to particular condition but usually associated with chronic neuropathic processes.
10 μV to 1 mV (1,000 μV)	20–150 Hz	1–5 msec	Dive-bomber or chainsaw	Rhythmically waxing and waning potentials initiated by needle movement or tapping. Biphasic initially downward.
250 μV to 2 mV	2–60 Hz	Rapid, staccato discharge	Marching sound	Consecutively firing potentials that do not wax and wane; not specific to particular condition but clearly are associated with an unstable muscle membrane.

3. With effort, a volume-conducted motor unit (coming from a distance away) can be eliminated with sufficient patient relaxation, which is not the case for true spontaneous potentials. Often PSWs appear with other forms of spontaneous potentials, such as fibrillations and complex repetitive discharges and are seen with both neuropathic and myopathic conditions and with anterior horn cell disease.

Fibrillations are common spontaneous potentials with an initial positive deflection and are of very short duration (usually less than 5 msec). Reported amplitudes for fibrillations range from 20 μV to more than 1 mV, and they generally discharge somewhat irregularly at rates between 1 and 30 Hz (see Table 17-3). As with PSWs, fibrillations are the firing of individual muscle fibers because of membrane instability and hypersensitivity to acetylcholine. The typical sound of fibrillation potentials has been described as "rain on a tin roof" when projected through the EMG speaker. Together, PSWs and fibrillations have been called *denervation potentials* because the most common cause of these spontaneously firing potentials is muscle denervation. Strictly speaking, however, this designation is not correct because myopathic processes and anterior horn cell disease can generate these abnormal potentials. The presence of positive sharp waves and fibrillations are usually graded from 0 to 4. Findings of 3 to 4+ PSWs and fibrillations indicate more severe muscle membrane instability than a 1 to 2+ designation. Consequently, their prognosis for recovery is less. This method of quantification of spontaneous potentials is useful when serial or repeat studies are undertaken. Progression from one grade to another over time is a means to gauge improvement (or lack of it) and provides objective documentation for making predictions regarding recovery from neuromuscular injury or disease.

Another spontaneous potential that may occur during the "rest" segment of the study is a *fasciculation,* considered to be a nonvoluntary motor unit firing. Most people have experienced an involuntary twitch of the eyelid muscle. This is a benign, albeit annoying, form of fasciculation. Fasciculations have the appearance of normal motor units, but their sound is unique, probably because they are activated in relative isolation. They are characterized by a popping sound. If there is another motor unit fasciculating at some distance from the tip of the needle electrode, it will produce a dull thud sound. Together, the sound is typified by a random sequence of *pop, pop, thud, pop, pop, thud* sounds. Fasciculations may occur in otherwise normal individuals, but they are often observed in patients with anterior horn cell disease. If attended by other forms of spontaneous potentials, they are considered an abnormal waveform. They may also be present in other disorders, such as entrapment neuropathies and radiculopathies. Unlike PSWs and fibrillations, there is no attempt to grade fasciculations; simply noting their presence is the accepted standard.

Complex repetitive discharges (CRDs) are spontaneous potentials that are not specific to any particular condition but are usually associated with chronic neuropathic processes (see Table 17-3). These were formerly called *bizarre high-frequency discharges.* These waves may vary in shape from linked PSWs or fibrillations to multiple polyphasic-like waveforms (a phase is measured as each time the motor unit signal crosses the baseline). They usually fire initially at a high frequency (up to 100 Hz) and are evoked by movement of the electrode's tip and then wane down to a lower frequency of 20 to 30 Hz. CRDs tend to start and stop abruptly and sound like a machine gun firing. Although CRDs are not specific to any particular condition, they are usually associated with chronic neuropathic processes.

Myotonic discharges are rhythmic spontaneous potentials often initiated by needle movement or tapping (see Table 17-3). The initial frequency of firing for these potentials is very high—as much as 150 Hz—and then fade to a rate of 20 to 30 Hz over a couple of seconds. Myotonic discharges differ from CRDs in that, in addition to waning in frequency, they will also resume a higher frequency—waxing. This characteristic waxing and waning sound reminded early ENMG examiners of WWII dive-bomber sounds; at one time, these potentials were referred to as *dive-bomber potentials.* Usually the waveform for myotonic discharges corresponds to that of sharp waves or fibrillations. These spontaneous potentials are noted in patients with myotonic dystrophy and myotonia congenita but have also been observed in patients with chronic radiculopathies.

A final spontaneous potential is the *myokymic discharge.* Myokymia is a muscle disorder that produces wormlike contractions of long sections of muscle. The electrical representation of this disorder appears like consecutively firing fasciculation potentials and produces an unmistakable marching sound. Unlike myotonic

discharges, myokymia does not wax and wane. In fact, volitional activity does not seem to alter these abnormal potentials, and they have even been observed during sleep. Myokymic discharges appear most often in chronic conditions, radiation plexopathies, Bell's palsy, and the facial muscles of patients with multiple sclerosis. Like positive sharp waves and fibrillations, they are not specific to any particular neuromuscular disease but clearly are associated with an unstable muscle membrane.

Minimal Activation

Up to this point in the EMG examination, the patient has been asked only to tolerate the procedure and remain as relaxed as possible because insertional activity and spontaneous activity at rest are entirely passive electrical phenomena. However, during the third segment of the EMG, the patient's full cooperation is needed to accomplish the goal. Segment three is referred to as *minimal activation*. The primary goal is to assess the motor units volitionally recruited by the patient. A normal motor unit potential (MUP) has a duration of 5 to 15 msec, an amplitude of 250 μV to 5 mV, and an onset firing frequency of 5 to 15 per second (upper range less than 60 Hz) (Fig. 17-18). The typical configuration of an MUP is biphasic or triphasic, although up to four phases is still considered normal. More than four phases constitutes a "polyphasic" motor unit. Although generally considered an abnormal MUP, normal young to middle-aged individuals are "allowed" to have up to 15% of their volitionally recruited motor units appear as polyphasics. The proportion of polyphasic motor units increases with age and is considered a normal variation when up to 30% of the recruited motor units are polyphasic in those 60 years or older.

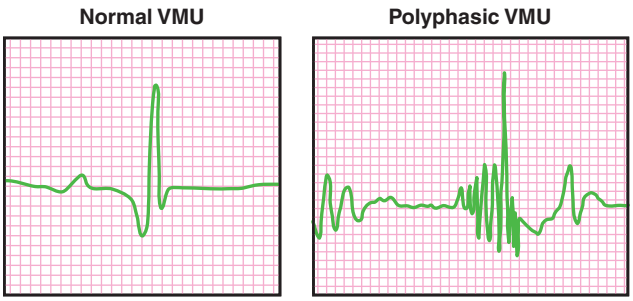


Fig 17 ■ 18 A normal motor unit is usually biphasic or triphasic, has a duration of 5 to 15 msec, an amplitude of 250 μV to 5 mV, and an onset frequency of 5 to 15 per second. A polyphasic motor unit is characterized by five or more phases and may be small during initial attempts at reinnervation or large in patients with chronic neuropathies.

A polyphasic motor unit is one that has five or more phases (see Fig. 17-18). Although these abnormal motor units can occur during the process of denervation, small low-amplitude, long-duration polyphasics usually indicate a recent attempt to reinnervate an injured or diseased motor unit. They have been given a special name—*nascent (polyphasic) potentials*—and are seen only during the early stages of reinnervation. When collateral or terminal sprouting by an injured or diseased axon occurs, the configuration of the motor unit changes because of the poorly synchronized conduction of its distal branches. As a result, the normal biphasic or triphasic configuration gives way to the polyphasic form. By contrast to the nascent polyphasic motor units, large-amplitude polyphasics are often observed in patients who have chronic neuropathies. Small-amplitude, short-duration polyphasics are believed to be a hallmark sign of myopathic disease.

One principle of volitional motor unit activation is that the first MUPs recruited are those with the lowest threshold. These tend to be the smallest motor units that are innervated by small-diameter alpha (α') motoneurons and are comprised primarily of slow-oxidative, fatigue-resistant muscle fibers. When greater levels of motor contraction are needed, these motor units increase their firing frequency and more motor units are recruited to meet this demand. Then, as still greater contraction strength is needed, larger motor units comprised of fast-twitch, fatigue-resistant muscle fibers are activated. Finally, when very strong muscle contractions are called for, the slow (red muscle fiber) motor units and the fastest-twitch, readily fatigable motor units are recruited at even faster frequencies. This process of recruiting motor units according to size and frequency in response to imposed demand is referred to as *rate coding;* it can be identified to a certain extent by electrophysiological assessment during the minimal activation portion of the examination. The size of the motor units (amplitude) and their frequency will characterize the recruited potentials as large or small, although this is often an academic point without a great deal of clinical utility.

Maximal Activation (Recruitment)

The final segment of the EMG examination is to evaluate the recruitment or interference pattern of each muscle. We have mentioned the process of motor unit recruitment, but here we are not interested in the characteristics

of the discrete units. Rather, the focus is to observe the orderly recruitment of MUPs and the ability to "fill the screen" with electrical activity while the patient begins with a minimal contraction and builds to a maximal contraction (Fig. 17-19). With full muscle contraction, the examiner should no longer be able to identify the baseline because the entire screen will be filled with summated motor units with amplitudes of roughly 4 mV from peak to peak (top of the positive phase to the bottom of the negative phase). The extent of electrical activity from maximal activity should "interfere" with the baseline. Many examiners refer to this pattern of activity as the *interference pattern.* Abnormalities noted during this segment of the study include the following:

- Neuropathic recruitment pattern
- Myopathic recruitment pattern
- Decreased activation

A neuropathic recruitment pattern is characterized by decreased recruitment for the entire muscle because a significant number of motor units have been lost through denervation (Fig. 17-20). Somewhat counterintuitively, any individual motor unit (whether healthy or diseased) in a muscle that has an overall decreased recruitment pattern will actually fire at a faster than normal frequency; this is usually referred to as *rapid firing rate* and is a classic sign of lower motor neuron injury or disease. The sound made by motor units exhibiting decreased recruitment and increased firing frequency is like that made by a playing card against moving bicycle spokes or rapidly running a stick along a picket fence. The rapid firing rate of the still-viable motor units in a muscle that is partially denervated is one good indication that a patient is fully cooperating during the examination. Failure to see an increased firing frequency in a muscle exhibiting weakness may suggest less-than-complete patient effort from pain, volition, or CNS involvement.

A myopathic recruitment pattern (Fig. 17-21) is characterized by small-amplitude, short-duration polyphasic motor units that appear almost immediately with little effort. In fact, it is almost impossible to isolate individual motor units in patients who have myopathic processes because they recruit their existing motor units so readily. Although this may initially appear as an actual increase in number of motor units recruited, the fact that it appears with almost no effort and that it is often attended by clinical weakness suggests that the muscle itself is diseased.

Decreased activation of motor units is an abnormality noted during recruitment, although it is often the examiner's subjective sense that leads to this designation. In the presence of organic lower motor neuron disease or injury, motor units may be decreased in number but will fire more rapidly to accomplish a task such as maximal muscle contraction. If the practitioner

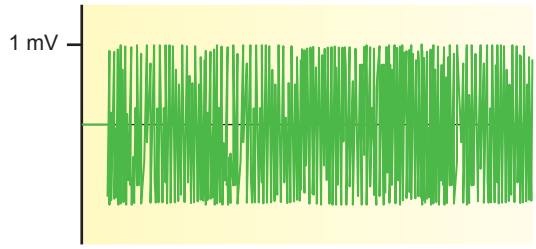


Fig 17 ■ 19 Full-screen interference indicates normal recruitment of motor units. The baseline is no longer visible because the electrical activity associated with depolarization of the large number of normal units has obscured it.

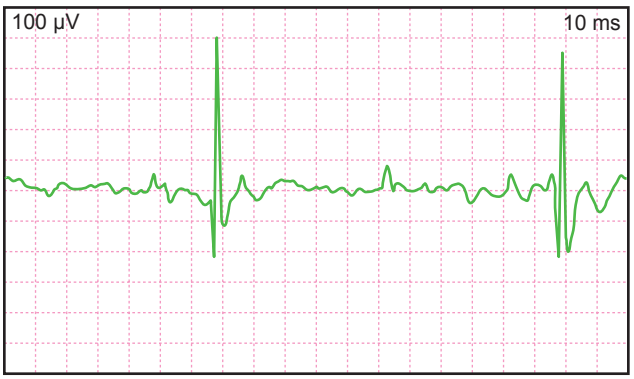


Fig 17 ■ 20 Neuropathic recruitment is characterized by a decreased number of motor units firing; those that are activated usually fire at a faster-than-normal rate—called *rapid firing rate.*

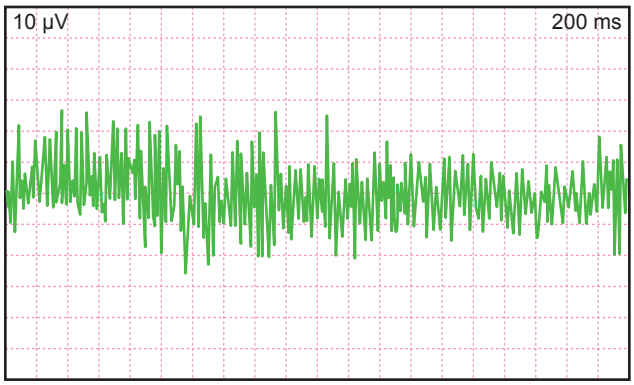


Fig 17 ■ 21 A myopathic recruitment pattern is noted for rapidly filling the screen with small-amplitude, short-duration polyphasic motor units accomplished with little effort.

asks the patient to give greater effort during a muscle contraction and the firing frequency remains unchanged, several explanations may apply. First, the patient may be experiencing pain that is prohibiting full cooperation, resulting in the expected increase in activation frequency. If this is the case, slightly adjusting the needle's tip may circumvent the problem, reduce the patient's discomfort, and allow a more robust contraction (characterized by the expected frequency of motor unit firing). This is a fairly common occurrence during a typical EMG examination.

Alternatively, a patient may not be giving full effort for reasons other than pain, such as secondary gain or symptom magnification. In this case, the decreased activation and failure to increase the motor unit firing frequency is entirely voluntary. It should be noted in the interpretation report that the motor unit firing is less than expected based on other findings. Results inconsistent with a failure to increase the frequency of motor unit firing and achieve full recruitment include the absence of any muscle atrophy, normal motor and sensory nerve conduction values, and no evidence of other electrophysiological abnormalities generally associated with denervation such as positive sharp waves or fibrillation potentials (Fig. 17-22).

Finally, when a central nervous system injury has resulted in motor weakness, a diminished number of motor units firing at a set frequency is the result of impaired signal to the anterior horn cells from higher centers. The patient may very well give the best effort, but the CNS lesion will prevent the lower motor neuron (anterior horn cells) from being activated, resulting in an increased firing rate. EMG of patients with CNS lesions is generally not recommended because it is not good at identifying the location and extent of such an injury. A summary of general EMG findings is noted in Table 17-4.

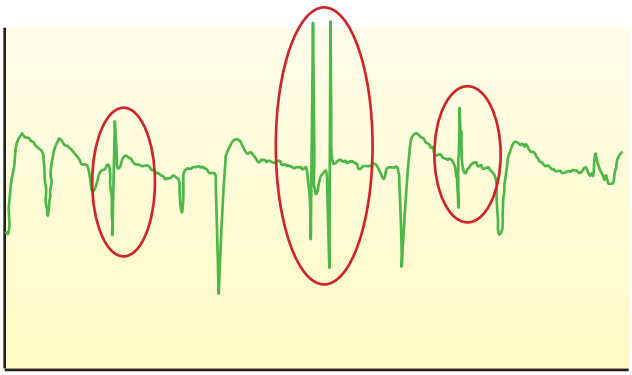


Fig 17 ■22 Positive sharp waves and fibrillation potentials (circled) are seen during EMG examination when axonal degeneration (denervation) has occurred. The extent of abnormal waves of this nature is usually graded from 1 to 4, depending on severity of involvement.

INTERPRETATION OF ELECTROPHYSIOLOGICAL EVALUATION FINDINGS

When the information from the NCS and the EMG examination has been obtained, the examiner must provide the referring practitioner with a summary of the findings and an interpretation that can answer the following primary questions:

1. Is this a normal or abnormal study?
2. If abnormal, are the findings more consistent with a neuropathic or a myopathic disorder?
3. If the examiner determines that the findings are most consistent with a neuropathic condition, does the lesion seem to affect the myelin or the axon preferentially?
4. If the lesion is neuropathic in its primary effect, does it appear to be focal (discrete site) or systemic?
5. Does the condition appear to be mild, moderate, or severe? Often the terminology used is *partial* or *complete,* which may refer to the extent of conduction block on NCS or spontaneous potentials (denervation-like potentials) on EMG. There is no certain way to distinguish between the denervation associated with an axonotmesis compared to a neurotmesis, outside of serial studies conducted over several months after finding a lesion.
6. Is there any hint that recovery might be occurring (e.g., presence of small-amplitude, polyphasic motor unit potentials on EMG)?

If ENMG examination provides answers to these questions, clinically useful information can be ascertained to establish the presence and severity of disease, chart a patient's course of care, and serve as an important indicator for prognosis.

Again, the questions used to construct and qualify the examination findings are time-dependent—that is, in the first few weeks that a disorder affecting the muscle and nerve is present, the abnormal findings (if there are any) would be primarily noted during the NCS. At the 3- to 4-week mark, abnormalities consistent with neurapraxia and axonotmesis would be present during

TABLE 17–4. Summary of EMG Findings

Testing Category	Normal Muscle	Peripheral Nerve Disorder	Myopathic Process
Insertional activity	Brief	Increased or prolonged	Brief or increased
Spontaneous activity	None	Present	Usually none
Minimal motor unit activation	Normal	Polyphasics, increased duration, large or small amplitude	Polyphasics, decreased duration, small amplitude
Maximal motor unit activation	Full interference	Reduced	Full with nominal activity

the NCS *and* the EMG. The EMG changes, in particular, would be consistent with a recent-onset condition, because the Wallerian degeneration and consequent muscle membrane instability associated with a neural injury are most prevalent in the 3- to 6-week mark. As time progresses, the NCS begins to return to some semblance of normalcy, and the acute changes associated with denervation (e.g., spontaneous potentials on the EMG) begin to give way to chronic motor unit recruitment findings. The full process of reinnervation may take several months to complete and often results in large-amplitude polyphasic motor units during recruitment, but little else will indicate presence of a neural insult. This is why an ENMG is especially useful for identifying abnormalities during the early stages of a neuromuscular disorder.

CASE STUDY 17-2 EMG Study

TM is a 59-year-old female who works as a forklift operator for a local bottling company. She has had a 2-month history of low back pain, left lower extremity pain, and paresthesias to the lateral side of the foot. She especially notices the discomfort and numbness in the foot after sitting for 30 minutes or longer at work. TM denies any history of recent trauma, but her physician has told her that she is "borderline diabetic," and she has been advised to control this condition with diet and exercise. Most troubling for TM is that she recently began a walking program to lose weight and is noticing a bandlike feeling around her ankle and a sense of weakness after walking for 10 minutes or more.

Clinical examination shows signs of neural tension in the left lower extremity with straight leg raise test and slump testing. Both of these tests refer pain from the lumbar spine into the lateral aspect of the gastrocnemius muscle. Sensory testing reveals diminished sensory acuity to crude and light touch in the S1 distribution and muscle weakness at 3+/5 in the gastroc-soleus complex, the tibialis posterior, and the fibularis longus. Great toe extensors are graded at 4/5, as are the tibialis anterior and the tensor fascia lata muscle. The deep tendon reflexes are normal at the knee; the right ankle jerk is graded as 2+, but the left ankle DTR is absent. Exquisite palpation tenderness is noted in the midline at L5–S1. The pain provocation tests for the sacroiliac joint are normal. The hip exam does not reveal any joint restriction.

SUMMARY OF NCS FINDINGS

Bilateral nerve conduction studies included the fibular and tibial motor nerves and F-waves, sural nerve sensory latencies, and the H-reflexes. The motor and sensory nerve conduction studies were normal, but the tibial nerve F-wave on the left side was mildly prolonged. In addition, the left H-reflex was significantly prolonged when compared to her uninvolved right side H-reflex.

RATIONALE FOR EMG

The NCS failed to identify a localized conduction block or area of segmental demyelination to explain TM's symptoms. The mildly prolonged F-wave and markedly prolonged H-reflex on the right side are certainly suggestive of a neural problem, but there is no definitive indication as to the severity or location of the problem. An EMG of the left lower extremity and paraspinal muscles should provide evidence of ongoing axonal degeneration if it is present. Recruitment characteristics of the muscles examined will also provide some information for clinical decisions regarding intervention.

EMG PLAN

Monopolar needle EMG examination of the left and right T12–S1 paraspinals and the following lower-extremity muscles will be conducted: tensor fascia lata, medial and lateral

CASE STUDY 17-2 EMG Study—cont'd

hamstrings, quadriceps femoris, tibialis anterior, tibialis posterior, fibularis longus, medial head of the gastrocnemius, and the soleus. These muscles will help to show whether the patient's complaints are arising from a radiculopathic process, a lumbosacral plexopathy, or some other cause.

FINDINGS

Monopolar needle EMG study of the left L5–S1 paraspinals and the lateral hamstrings, tensor fascia lata, medial gastrocnemius, tibialis posterior, and fibularis longus revealed sharp waves and fibrillation potentials, indicating ongoing axonal injury. Motor recruitment in these muscles was impaired, but early signs of recovery potentials (small amplitude polyphasic motor units) were noted. Muscles examined without any electrical evidence of abnormality included the T12–L4 paraspinals, quadriceps, tibialis anterior, and soleus muscles. The muscles examined revealed no evidence of myopathic motor units. No fasciculation potentials were noted at rest. No abnormality was identified in any of the similar muscles examined on the right side.

INTERPRETATION

1. Findings are consistent with a moderately severe radiculopathic process primarily affecting the (L5) S1 innervated muscles of the left lower extremity. Findings of similar abnormality in the left lumbar paraspinals clearly implicate the location of the neural injury to the level of the nerve root.
2. Early signs of reinnervation (small amplitude polyphasic motor units) indicate some attempt at neural recovery is ongoing and is likely to proceed over time.
3. No indication of abnormality is noted in the right paraspinals or lower extremity.

SUMMARY

EMG findings are best explained by a nerve root compression. Correlation with clinical exam findings and any imaging studies is recommended to identify the cause of the compression (e.g., herniated nucleus pulposus, tumor, etc.). The indication of ongoing reinnervation suggests that conservative management is appropriate for this patient, but vigilance should be maintained to detect any signs of progressive neurological deficit.

REPORTING RESULTS

After the examiner completes the technical aspect of data collection for the ENMG and has come to a conclusion after interpreting the information, the next task is to communicate the results to the referring physician in a manner that is readily understandable and usable.

Key Point! To arrive at the diagnosis, the information from the ENMG testing must be used in light of the clinical and historical information obtained from the patient. Imaging studies or other specialist test procedures (blood analysis) are also complementary in this process.

Summarizing the test results is the final step in the evaluation process. A sufficient history and brief details of a clinical examination should be given to justify the need for the ENMG testing. Then the data from the NCS are usually depicted in tabular form so the nerves studied and the numerical results are clearly identified (Table 17-5).

Many current EMG machines can generate reports that include the values obtained during the study and a comparison list of normal values. The normal values usually are obtained from a national database and the current literature and are programmed into the computer. Examiners should verify with their own laboratory that these values are appropriate for their particular setting; if not, more clinically applicable values should be used for comparison.

Key Point! A categorical statement should not be made about the cause of the findings because the ENMG cannot actually determine this.

Information from the needle EMG portion of the exam should follow and is usually presented in tabular format to include muscles studied and the response of each muscle to insertion, rest, minimal activation, and maximal activation (interference pattern with recruitment). The interpretation often presents, in list form, the abnormal findings from the study, an impression

TABLE 17–5. Sample Report Following ENMG Study: How ENMG Findings Are Reported

Brief History: Relevant history of patient described here

Brief Clinical Exam: Details of examination described here

Nerve Conduction Study

	(L) Median Nerve	(R) Median Nerve
DML	3.1 msec (8 mV)	5.8 msec (3.8 mV)
DSL (palmar-onset)	1.6 msec (42 μV)	3.2 msec (12 μV)
NCV (above elbow–wrist)	62 m/sec (8 mV)	54 m/sec (3.8 mV)
NCV (axilla–elbow)	63 m/sec (8 mV)	58 m/sec (3.8 mV)
F-wave	27.4 msec	32.1 msec
H-reflex	16.0 msec	16.2 msec
	(L) Ulnar Nerve	**(R) Ulnar Nerve**
DML	2.8 msec (8.4 mV)	3.2 msec (4.8 mV)
DSL (palmar-onset)	1.4 msec (40 μV)	2.4 (12 μV)
NCV (below elbow–wrist	58 m/sec (8.4 mV)	57 m/sec (4.2 mV)
NCV (across elbow)	62 m/sec (8.4 mV)	38 m/sec (1.8 mV)
NCV (axilla–elbow)	63 m/sec (8.4 mV)	58 m/sec (1.8 mV)
F-wave	27.6 msec	33.1 msec

EMG Findings

Muscle	Insertional Activity	Spontaneous Activity	Motor Units	Interference Pattern
Cervical paraspinals (C3–T1) R and L	Brief	None	Full	Not tested
Deltoid: R and L	Brief	None	Full	Full
Biceps: R and L	Brief	None	Full	Full
Triceps: R and L	Brief	None	Full	Full
Flexor carpi radialis: R and L	Brief	None	Full	Full
Flexor digitorum sublimis: R and L	Brief	None	Full	Full
Extensor digitorum communis: R and L	Brief	None	Full	Full
(L) Abductor pollicis brevis and opponens pollicis	Brief	None	Full	Full
(L) First dorsal interosseous	Brief	None	Full	Full
(R) Abductor pollicis brevis and opponens pollicis	Prolonged	1+ sharp waves and numerous fibrillations	Increased number of small amplitude polyphasic motor units	Partial interference pattern with motor unit dropout and rapid firing rate
(R) First dorsal interosseous	Prolonged	1+ sharp waves and numerous fibrillations	Increased number of small amplitude polyphasic motor units	Partial interference pattern with motor unit dropout and rapid firing rate
(R) Extensor pollicis longus	Brief	None	Full	Full
(R) Abductor digiti minimi	Prolonged	Occasional to 1+ sharp wave and fibrillations	Mixed polyphasic and normal motor units	Slight motor unit dropout and slight rapid firing rate

TABLE 17–5. Sample Report Following ENMG Study: How ENMG Findings Are Reported—cont'd

List of Abnormal Findings
Nerve Conduction Study
Using surface electrodes, bilateral median and ulnar motor and sensory nerves were tested. Testing also included bilateral F-waves and median nerve H-reflexes. F-waves are long-loop latencies that reflect the health of the entire length of the nerve. Median nerve H-reflexes are long-loop latencies specific to the C6–7 nerve root. All nerves tested had normal amplitudes, latencies, and nerve conduction velocities except: • Prolonged right median nerve motor and sensory latencies • Reduced right median nerve motor and sensory amplitudes • Prolonged right median nerve F-wave • Slowed right ulnar nerve motor conduction across the elbow • Reduced right ulnar nerve CMAP amplitude Additional right ulnar nerve abnormal findings include: • Prolonged sensory latency • Reduced sensory amplitude • Borderline reduced CMAP amplitude at the wrist and below elbow stimulation sites (compared to contralateral ulnar nerve values)
Needle EMG Study
Using a sterile monopolar needle electrode, bilateral upper extremity muscles were tested across C5–T1 myotomes and across peripheral nerves. All muscles tested exhibited normal insertional activity, absence of spontaneous activity at rest, and normal motor unit number, configuration, and recruitment except: • Right abductor pollicis and opponens pollicis demonstrated prolonged insertional activity, 1+ sharp waves and fibrillations at rest, increased number of small amplitude polyphasic motor units, and partial interference with motor unit dropout and rapid firing rate. • Right first dorsal interosseous and abductor digiti minimi exhibited prolonged insertional activity, occasional to 1+ sharp waves and fibrillations at rest, increased number of small amplitude polyphasic motor units, and partial interference with motor unit dropout and rapid firing rate.
Impression
This is an abnormal study showing electrophysiological evidence of (R) median nerve compression at or near the wrist (carpal tunnel region) characterized by segmental demyelination (nerve conduction findings) and axonal degeneration (needle EMG findings of denervation). Further, there is electrophysiological evidence of (R) ulnar nerve compression at or near the cubital tunnel of the elbow characterized by segmental demyelination (nerve conduction slowing) and axonal degeneration (needle EMG findings of denervation). There was no evidence of a diffuse peripheral polyneuropathic process, radiculopathy, or brachial plexopathy. No myopathic motor units were recruited and no fasciculations were noted at rest to suggest anterior horn cell involvement.

DML = distal motor latency; DSL = distal sensory latency; NCV = nerve conduction velocity; R & L = right and left.

as to whether the findings are most consistent with segmental demyelination or axonal degeneration, and a concluding remark about the likely location of the lesion should the ENMG identify one. It is common for a referring clinician to call after receiving the report to seek clarification regarding prognosis based on the findings and to obtain counsel about the potential benefit of conservative management of a patient's neuromuscular disorder. Clinical experience with such cases gained over the years may allow the ENMG examiner to provide additional useful information and recommendations based on the results of the testing and brief clinical exam.

DOES ENMG BEAR ANY RELATIONSHIP TO EMG BIOFEEDBACK?

EMG biofeedback (i.e., surface EMG), discussed in Chapter 13, uses electrical signals of muscle depolarization in much the same way that diagnostic EMG does but with two important distinctions. First, EMG biofeedback is an entirely therapeutic procedure for the purpose of helping a patient increase or decrease skeletal muscle activity. There is little diagnostic element to the biofeedback procedure. Second, EMG biofeedback almost always uses surface EMG electrodes (not needles),

CASE STUDY 17-3 Postpartum Femoral Nerve Palsy

BL is a 23-year-old woman with right lower-extremity motor and sensory complaints immediately following delivery of her first child, which was accomplished with epidural anesthesia. Weakness was particularly noted in the quadriceps muscle and numbness in the saphenous nerve distribution. These complaints appeared to peak several days after parturition and then improved slightly for a couple of weeks. However, 2 to 3 weeks after onset of symptoms, the patient remained unable to completely extend her right knee, and her right knee DTR was graded at 1+/4. Persistent weakness and numbness in the lower extremity suggested a neural injury, but it was unknown whether this represented a neurapraxia or axonotmesis. Further, normal iliopsoas muscle testing in the presence of 3/5 grade for quadriceps manual muscle testing indicated a discrete muscular branch of the femoral nerve as a possible location of this injury.

RATIONALE FOR EMG/NCS

Persistent weakness and complaint of numbness in the saphenous nerve distribution suggested injury to the femoral nerve. However, the history of this beginning immediately following administration of epidural anesthesia at the T12–L1 level also raised the possibility of a more proximal injury location (spinal nerve root level). The somewhat rapid partial resolution of symptoms and clinical findings indicated the likelihood of a neurapraxia rather than an axonotmesis. Prognosis for the former would be significantly better (especially the time frame for recovery) than the latter, which was of concern for this student hoping to return to full-time graduate studies. Consequently, it was determined to obtain additional clinical and electrophysiological information to more accurately identify the features of this apparent neural injury.

SUMMARY OF INITIAL EMG/NCS AND CLINICAL TESTING

At 12 days postdelivery, maximal voluntary isometric force (MVIF) of the right knee extensors was 13.9 lb on the right and 42.2 lb on the left. Maximal electrically elicited isometric force (MEIF) on the right side was 24.3 lb. Nerve conduction study of the femoral motor nerve and saphenous sensory nerve revealed the following abnormalities:

1. The noninvolved left femoral nerve CMAP was 12 mV (normal value) while the right involved CMAP was 2 mV, a six-fold decrement.
2. Similarly, the right saphenous SNAP was normal at 12 μV, but the left, involved side demonstrated a SNAP of 5 μV. The conduction velocities for the left and right femoral nerve were comparable and well within the normal range.
3. Monopolar needle EMG examination of femoral innervated right thigh musculature failed to reveal any evidence of muscle membrane instability (positive sharp waves or fibrillations) to suggest the presence of Wallerian degeneration, but all four portions of the quadriceps muscle showed motor unit recruitment impairment consistent with partial conduction block. There was no involvement of the abductor magnus (innervated by the obturator nerve) or the iliopsoas muscles.

INTERPRETATION OF INITIAL EMG/NCS AND CLINICAL FINDINGS

It was concluded that this was a positive nerve conduction study and EMG for probable early indication of moderate axonal injury to the right-sided L3–4 nerve roots and/or femoral nerve. Clinical findings of MVIF and MEIF reduction were consistent with femoral nerve impairment. Because EMG evidence of axonal injury takes an average of 21 days to develop following onset of injury, it was recommended that follow-up clinical and/or EMG and NCS be repeated within a 2- to 4-week time frame.

SUMMARY OF REPEAT EMG/NCS AND CLINICAL TESTING

Six weeks after onset of symptoms, repeat MVIF value was 73.8 lb, a greater than four-fold improvement from initial value. Similarly, the right femoral nerve CMAP had improved to 8.5 mV, slightly greater than a four-fold improvement. Needle EMG of the right lower extremity did not reveal any signs of ongoing denervation to suggest axonal injury. In addition, the motor recruitment abnormalities identified at the initial evaluation had virtually resolved.

SUMMARY

Nulliparity and prolonged second-stage labor are known risks for postpartum femoral nerve entrapment, and the potential for nerve root injury associated with epidural injection is present in this patient's history as well. Quadriceps, but not the iliopsoas, muscle involvement suggests entrapment distal to the inguinal ligament. Greater than 50% loss of peak CMAP initially, but less than 30% loss at 6 weeks strongly suggests a neurapraxic injury. Further, though there were motor unit recruitment impairments

CASE STUDY 17-3 Postpartum Femoral Nerve Palsy—cont'd

by needle EMG at the 2-week mark, these had virtually resolved by 6 weeks. These electrophysiological findings coincided with similar impairments and resolution noted with clinical testing for MVIF and MEIF. The value of obtaining EMG/NCS data on this patient was to confirm that this injury represented a neurapraxia rather than an axonotmesis. The student was able to resume all physical and academic requirements for her graduate study program within 4 weeks postpartum with no long-term sequelae.

and the equipment is usually smaller and more portable than diagnostic EMG units. Nevertheless, there is a connection between the two procedures because, although ENMG is a diagnostic test, its purpose is to answer a clinical question that leads to a more informed therapeutic intervention.

Consider this example: A patient who has had total knee arthroplasty (TKA) 3 weeks previous has been sent to the clinic to begin outpatient physical therapy. On clinical examination, you note that the patient has a 3/5 muscle contraction of the quadriceps. This finding represents a barrier to the normal course of exercise intervention. Although you are aware that pain, inhibition, and preoperative disuse atrophy can contribute to such a finding, you are also familiar with the literature that indicates that up to 70% of TKA patients have a femoral nerve injury produced by the compression of the pneumatic tourniquet used during the surgery to control excessive bleeding in the surgical field. Because you are not certain of the exact cause of this muscle impairment, you proceed with brief needle EMG of the quadriceps and other thigh muscles. You find that the patient has no increase in insertional activity and no spontaneous potentials at rest (i.e., no positive sharp waves or fibrillations). The motor units appear to be primarily biphasic and triphasic, although they are decreased in number. Efforts to recruit a full interference pattern are compromised initially by pain, but with continued effort, a full pattern is achieved. From this information, you conclude that the patient has *not* sustained a neural injury but is, indeed, suffering from postoperative pain, inhibition, and typical muscle atrophy.

This patient has plenty of viable motor units to recruit and thus is an ideal candidate for both EMG biofeedback as a primary adjunct to therapeutic exercise and high-amplitude neuromuscular electrical stimulation. Often, after three to five sessions of EMG biofeedback, the patient has mastered the ability to recruit available motor units without causing significant pain or experiencing inhibition. At that point, the biofeedback procedures can be discontinued and the patient can concentrate on therapeutic exercise activities. This is an example of how ENMG testing leads to a therapeutic intervention that makes primary use of EMG biofeedback to optimize outcome without tissue irritation or injury.

Documentation Tips

When documenting the procedures and results of an ENMG, the following should be presented:

- Medical screening/history findings
- Clinical exam findings
  - Neuro exam
  - Musculoskeletal screening exam
  - Height
  - Age
  - Surface temperature of the extremity
- Nerve conduction study
  - Nerve segment distance
  - Latency (motor, sensory)
  - Amplitude (motor, sensory)
  - Duration of the action potential
  - Shape (configuration) of the waveform
  - Nerve conduction velocity (motor, sensory)
- EMG
  - Needle type (e.g., monopolar Teflon, concentric, bipolar, etc.)
  - Muscles examined
    - Specific nerve supply
    - Nerve root derivation
    - Key findings
    - Insertion

  - Rest
  - Minimal activation
  - Maximal activation
- List of abnormal findings
- Interpretation/impression

Clinical Controversy

- Some controversy exists regarding the use of antidromic versus orthodromic sensory latency and conduction values. The orthodromic technique is certainly more consistent with the anatomical and physiological direction of impulse propagation (afferent: distal to proximal); however, antidromic technique is easier to use in many cases, and the amplitude of response is often larger than orthodromically obtained responses and is therefore easier to see and record.
- Many practitioners often identify a neural lesion as a "neurapraxia" or an "axonotmesis," but caution should be used here. Most neural lesions are a combination of myelin and axon involvement. Although it is true that a neural injury may be primarily myelin or axonal, they are rarely one or the other. Caution should be exercised in describing them as such.

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Glossary

Absolute atmosphere (ATA) The atmospheric pressure at sea level, equal to 1 ATA (760 mm Hg).

Acoustic impedance A material's ability to transmit sound, related to the molecular density and structure of the material. Impedance is inversely related to transmission.

Acoustic streaming The forward movement of fluid created by acoustic oscillations of ultrasound energy.

Allodynia Pain produced by an otherwise non-noxious stimulus, such as light touch of the skin following sunburn; a form of hyperalgesia.

Alpha motoneuron The large, lower motor neurons of the brain and spinal cord that innervate skeletal muscle.

Alternating current (AC) The uninterrupted bidirectional flow of ions or electrons that must change direction at least one time per second.

Ampere (A; amp) The unit of electrical current reflecting the volume of current (electrons) passing a given point in a given time; 1 amp = 1 coulomb/sec.

Amplification The act of repeatedly bouncing light between two parallel reflectors arranged at opposite ends of a lasing chamber, causing the excitation of more photons.

Analgesia The decrease or absence of pain.

Analgesic tolerance A decline or loss in the analgesic effectiveness resulting from repeated use of a given therapeutic intervention.

Ankle-Brachial Index (ABI) A method used to determine the presence and severity of peripheral arterial disease, calculated as the ratio of ankle systolic blood pressure to the arm systolic blood pressure.

Anode A point or region of a circuit that has a deficiency of electrons; also referred to as the *positive pole.*

Antidromic Conduction along a nerve in the direction opposite of normal; for example, proximal to distal for sensory and distal to proximal for motor axons.

Aquatic therapy A therapeutic intervention, usually performed in a pool that uses water to facilitate physiological effects or exercise programs.

Asymmetrical Condition when the amplitude and duration characteristics between the two phases of the biphasic waveform differ in any manner.

Attenuation A measure of the decrease in sound energy either by absorption, reflection, or refraction.

Axonotmesis Axonal degeneration and myelin sheath disruption that occurs distal to and one node of Ranvier proximal to the nerve lesion.

Balanced When the area under the curve of the first phase (i.e., phase charge) of a biphasic pulse is equal to that of the second phase.

Beam nonuniformity ratio (BNR) The ratio between spatial peak intensity and spatial average intensity of an ultrasound beam.

Beat frequency The frequency at which peak constructive or destructive interference occurs for interferential current; calculated as the difference in frequency between the currents interfered.

Bipolar An electrode configuration in which all the electrodes of a single electrical circuit are placed over the treatment area.

Body mass index (BMI) A value that represents a measure of mass, calculated as body mass (in kg) divided by height (in meters) squared (kg/m^2).

Buoyancy A force on a body immersed in a fluid that is equal to the weight of the fluid displaced by that object.

Burst A series of pulses or brief periods of alternating current delivered consecutively and separated from the next series or period.

Burst frequency The frequency at which bursts are generated.

Capacitance The amount of charge that a material can hold for a given voltage imposed upon it.

Capacitive or electric field method (diathermy) An applicator system that requires making the patient's tissues part of the dielectric of a capacitor.

Carrier frequency The frequency of the alternating current or pulse train that is interrupted into bursts.

Cathode A point or region of a circuit that has an excess of electrons; also referred to as the *negative pole*.

Causalgia Type 2 complex regional pain syndrome identified as painful burning sensations in an extremity that usually occurs along the distribution of a nerve.

Cavitation Pulsation of gas bubbles in biological tissues in response to the passage of ultrasound.

Central sensitization The amplification of neural signaling within the central nervous system that underlies the development of pain hypersensitivity.

Charge (*Q*) Electrical state obtained by the addition or removal of electrons; charge is measured in coulombs (C) or microcoulombs (μC).

Chemical mediators Substances located in inflammatory cells that attract and activate fibroblasts to the site of an injury; for example, histamines, cytokines, and leukotrienes.

Chronaxie The duration of a stimulus that is two times the rheobase amplitude and capable of eliciting a minimally detectable motor response. Chronaxie is used to assess the integrity of tissue, as healthy innervated tissue should have a chronaxie less than 1 msec.

Chronic venous insufficiency (CVI) Vascular disease that begins at the junction of superficial and deep vein systems and creates valvular incompetence.

Chronic wound A wound that deviates from the expected sequence of repair in terms of time, appearance, and response to aggressive and appropriate treatment.

Classification system for pain Developed by the International Association for the Study of Pain; it includes definitions for pain terms and descriptions of pain syndromes.

Claudication pain Pain in the calves when walking brought on by muscle ischemia; the cardinal symptom of peripheral arterial disease.

Cold bath Immersion of distal extremities in a tub or basin with circumferential contact of the cooling agent.

Cold packs Flexible frozen ice, gel, or liquid packs used for cryotherapy.

Cold urticaria Hypersensitivity to cold that results in a vascular skin reaction in response to cold exposure; typically characterized by smooth, itchy, elevated red patches.

Comorbidity Any existing medical illnesses concurrent with a primary disorder.

Complex regional pain syndrome (CRPS) Categorized as either type 1 (RSD) or type 2 (causalgia); both types are forms of pain in the sympathetic nervous system, either with or without known involvement of the peripheral nerves.

Complex repetitive discharges Abnormal spontaneous electrical potentials not specific to any particular condition but usually associated with chronic myopathic or neuropathic processes.

Compound motor action potential The summated response of all the depolarized motor units in a nerve.

Compression sleeve Sleeve donned on a limb with inflatable chambers to provide a compression force to the limb.

Condensations Areas of compression or increased molecular density in biological tissues in response to the passage of ultrasound.

Conduction A method of heat transfer between objects in direct contact with each other, where the kinetic motion of atoms and molecules of one object of higher energy (i.e., temperature) is passed to the other object of lesser energy.

Conductor A material that permits the flow of electrical current.

Constant current (CC) Related to Ohm's law ($I = V/R$) where flow of current is directly related to voltage; constant current devices maintain a constant current (I) by continually adjusting voltage (V).

Constant voltage (CV) Related to Ohm's law ($V = IR$); CV devices maintain a constant voltage (V) by continually changing current (I).

Continuous mode (1) Therapeutic ultrasound delivered without interruption at 100% duty cycle for the entire treatment period; (2) the uninterrupted delivery of pulsed electrical current.

Controlled–cold compression unit A device that alternately pumps cold water and air into a sleeve

that is wrapped around a patient's limb to provide cold therapy and compression.

Convection A method of heat transfer in which the heated molecules move or circulate from one place to another, such as the movement of water in a whirlpool.

Cosine law Maximum absorption of radiant energy occurs when the source is at a right angle to the absorbing surface; when the source is not at a right angle to the absorbing surface, the angle formed by the source and perpendicular to the absorbing surface determines the effect of the energy.

Coulomb The unit of measure of electrical charge; equal to 6.24151×10^{18} electrons.

Cryoglobulinemia A disorder characterized by the presence of an abnormal blood protein that, when exposed to cold temperatures, results in agglutination of serum proteins that can impair circulation, resulting in ischemia or gangrene.

Cryotherapy The use of cold modalities (e.g., ice, cold packs, cold compression devices, vapocoolant sprays) for therapeutic purposes.

Current The movement of ions or electrons in a conductor in response to a voltage force; represented by I (in amperes).

Current density A measure of the electrical charge per unit area of an electrode's cross-sectional area (mA/cm^2 or mm^2). Current density is inversely proportional to electrode size. Also referred to as *charge density*.

Cytokines Chemical mediator protein molecules that allow for receptor-mediated communication between cells to trigger cell transformation, secretion, migration, proliferation, and death.

Deep vein thrombosis (DVT) The presence of a thrombosis within the venous system.

Denervation Loss of nerve supply to muscles or other tissues.

Diabetic polyneuropathy The consequence of a diabetic vasculopathy, presented as multiple areas of nerve damage.

Diathermy A therapeutic modality device that produces radiofrequency radiation, usually used to heat through biological tissues.

Dielectric constant The ratio of the capacity of a material (tissue) to that of free space.

Diode A two-terminal electronic component that conducts electric current in only one direction.

Diplode A hinged drum connected to a diathermy device that enables one or more body-part surfaces to be treated simultaneously.

Direct current The continuous unidirectional flow of charged particles (electrons or ions) for at least 1 second.

Dosage (1) The total amount of energy or force delivered by a therapeutic modality to a patient during a treatment session; (2) the intensity or amplitude of electrical stimulation required to generate a specific muscular force or torque.

Duty cycle The percentage of the on-time to the total time (on-time plus off-time) of electrical current, multiplied by 100%.

Edema Presence of excess fluid in the interstitial space.

Effective radiating area (ERA) A measure of the actual cross-sectional area of the ultrasound beam as it exits the metal end plate of the transducer, expressed in square centimeters (cm^2).

Electroanalgesia The modulation of pain through the use of electrical stimulation.

Electrode The interface relaying current between an electrical stimulation device and the patient.

Electromigration Movement of charged particles in response to an applied voltage.

Electroneuromyography The recording of nerve and muscle activity in response to an electrical stimulus.

Electroosmosis Bulk or volume fluid flow in response to a voltage difference imposed across a charged membrane, such as the skin.

Electroporation The increase in the porosity of the superficial skin in response to electrical stimulation.

Endogenous opiates Hormones released into the blood as a part of the response to stressful stimuli that inhibit the perception and experience of pain.

End plate spikes Normal nonpropagated spontaneous electrical potentials observed when the tip of the EMG needle electrode is near a motor end plate. End plate spikes are induced by the release of acetylcholine from the presynaptic axon terminal in response to an action potential.

Enkephalin Inhibitory neuropeptide that is widely distributed in the central and peripheral nervous systems.

Evidence-based practice The clinical practice of basing patient-management decisions and interventions on evidence of effectiveness found in the scientific literature, patient values, and expert opinion.

Examination A sequential, iterative process that consists of three parts: history, systems review, and specific tests and measures.

Extracorporeal shock wave therapy (ESWT) Application of single-impulse, focused acoustical sound waves with a rapid rise in pressure to biological tissues.

Fall time The time required for the trailing edge of a single phase to return to the isoelectric line.

Fasciculations Repetitive twitchlike spontaneous electrical potentials that reflect discharge of single or multiple motor units.

FDA U.S. Food and Drug Administration.

FES Functional electric stimulation, usually indicating the use of neuromuscular electrical stimulation in substitution for an orthotic device.

Fibrillations Abnormal spontaneous electrical potentials indicative of unstable muscle membrane; thought to represent depolarization of a single muscle fiber.

Fluidotherapy A dry-heat modality that transfers heat energy by forced convection.

Frequency In electrotherapy, the number of cycles per second (cps, or hertz [Hz]) for an alternating current or the number of pulses per second (pps) for a pulsed current.

Functional limitations The inability to carry out specific functional activities.

F-wave An action potential produced by an antidromically directed motor stimulus that is part of neurodiagnostic testing used to examine conduction problems in the proximal and distal region of peripheral nerves.

Gauss (G) Unit used to express strength of a magnetic field; the relationship of the gauss and the tesla is 1 G = 10^{-4} T.

Granulation tissue New tissue that grows inward from surrounding healthy connective tissue; it is filled with new capillaries and is surrounded by fibroblasts and macrophages.

Gridding Treatment performed by a series of vertical and horizontal strokes with a laser applicator over the length and width of an area of skin.

Ground substance Amorphous gel that forms the extracellular matrix within a wound.

Growth factors Factors that stimulate cellular proliferation and tissue growth and repair.

High-voltage pulsed current (HVPC) A twin-peaked monophasic pulsed current with voltage up to 500 volts and very short pulse duration resulting in a current with a relatively low average current yet high peak voltage.

H-reflex An action potential produced by an orthodromically directed sensory stimulus that is part of neurodiagnostic testing that assesses the monosynaptic stretch reflex.

Hunting response Cold-induced vasodilation following the initial period of vasoconstriction, resulting in cyclic periods of vasodilation and vasoconstriction and in cyclic warming and cooling of the skin of the face, hands, fingers, feet, and toes.

Hydrostatic pressure Force exerted by water on a body or body part immersed in water.

Hydrotherapy Therapeutic use of water and water-based modalities.

Hyperalgesia Increased sensitivity to pain.

Hyperbaric oxygen therapy (HBOT) Inhalation of 100% oxygen in a pressurized hyperbaric chamber at a pressure greater than 1 absolute atmosphere (ATA).

Hyperemia Increased blood flow caused by a tissue temperature elevation.

Hyperstimulation A form of noxious-level stimulation often using monophasic currents of long pulse durations or direct current.

Hypertrophic scar Excessive collagen synthesis resulting in a raised scar that remains within the original boundaries of the wound.

Ice massage A cryotherapy technique in which ice is rubbed over a small area of the skin to produce rapid analgesia.

Impedance Frequency-dependent resistance to the flow of alternating current.

Inductive or magnetic field method (diathermy) Use of an inductive applicator in which an oscillating magnetic field produces "eddy" currents in the treated tissues, usually resulting in a tissue temperature rise.

Insertional activity Normal brief electrical activity noted upon insertion of EMG needle electrode within a muscle.

Insulator A material in which the movement of current (i.e., electrons or ions) is opposed or not free to move.

Interference pattern Visual disruption of the isoelectric line during electromyographic examination reflecting electrical potentials of multiple motor units during maximal voluntary muscle contraction. Individual motor units cannot be identified when a patient demonstrates a full interference pattern.

Interferential current An amplitude modulated form of alternating current commonly used for pain modulation (i.e., electroanalgesia).

Intermittent pneumatic compression (IPC) A modality that applies compressive force to a limb through a sleeve garment that alternately fills and empties with air pumped into the garment by an air compressor.

Intermittent traction A mechanical device that alternately applies and releases traction to the neck or back at preset intervals.

Interphase interval The time between successive phases of a single pulse.

Interpulse interval The time between successive pulses.

Intrapulse interval See *Interphase interval.*

In vitro studies Studies performed outside of a living organism.

In vivo studies Studies performed within a living organism.

Ionohydrokinesis See *Electroosmosis.*

Iontophoresis Use of direct current to induce the transcutaneous movement of ions across the skin into target tissues.

Keloid Raised scar that extends beyond the original boundaries of the wound and can invade surrounding tissue.

Laser Light amplification by stimulated emission of radiation.

Latency Time between an electrical stimulus and the initial deflection of the compound sensory or motor action potential.

Light Electromagnetic energy that is transmitted through space either as a propagated wave or as small parcels of energy called *photons*.

Longitudinal waves Ultrasound waves that are parallel to the direction of the sound beam.

Low-intensity direct current (LIDC) See *Microcurrent*.

Lymphedema Swelling of an extremity brought on by decreased ability of the lymph system to transport fluid, resulting in an increase in protein-rich fluid that damages artery and venous systems.

Magnetic field The attractive or repulsive force represented by field lines drawn around a magnet.

Magnet therapy The use of magnets for therapeutic purposes.

Manual traction Traction applied to a body part by the hands of a therapist or other health-care provider.

Mechanical traction Traction applied to the neck or back for a period of time via the use of an electrical or mechanical motor unit.

Microcurrent Current with a peak amplitude less than 1 mA.

Microstreaming Flux of ions present within and around cells; intracellular and extracellular fluids in tissue exposed to ultrasound energy.

Miniature end-plate potentials Normal spontaneous electrical potentials observed when the tip of the EMG needle electrode is near a neuromuscular junction, producing a localized, transient depolarization initiated by the spontaneous release of individual acetylcholine quanta from the presynaptic axon terminal.

Minimal erythema dose (MED) The smallest dose of ultraviolet light that produces an erythema that appears within 1 to 6 hours and fades without a trace within 24 hours.

Modulation The changing or alteration of specific parameters of an electrical stimulus, such as the frequency and amplitude.

Monochromatic infrared energy (MIRE) Refers to devices used to produce energy from the near-infrared portion of the magnetic spectrum for therapeutic purposes.

Monochromatic light Light that has a singular wavelength and therefore is one color.

Monode A drum attached to a diathermy device that is used to treat a single body surface.

Monophasic pulsed current The repeated delivery of monophasic pulses produced by intermittently interrupting a DC current source.

Monopolar An electrode configuration in which at least one electrode of a single circuit is placed over the intended treatment area with another electrode placed in a nontreatment area. Monopolar electrode placement is most commonly used with DC and monophasic pulsed currents and maintains a constant anode and cathode.

Motor unit An alpha motoneuron and all muscle fibers it innervates.

Myofascial pain syndrome Pain or autonomic phenomena referred from active myofascial trigger points with associated dysfunction.

Myokymic discharges Abnormal consecutively firing spontaneous electrical potentials that do not wax and wane and that are followed by a short period of electrical silence.

Myotonic discharges Abnormal rhythmic spontaneous electrical potentials initiated by movement or by tapping the inserted EMG needle electrode due to independent, repetitive discharges of single muscle fibers.

Neurapraxia The mildest form of peripheral nerve disorder characterized by local conduction failure or block across the affected segment without any axonal injury.

Neurolemma The outermost layer of the nerve membrane.

Neuropeptide Substance secreted at perivascular terminals of noradrenergic and cholinergic fibers; it has been shown to affect cellular events in all three phases of healing.

Neurotmesis Nerve injury characterized by disruption of the axon with damage to the associated connective tissue layers of the nerve.

NMES Neuromuscular electrical stimulation.

Nociception Response to noxious stimuli that results in perception of pain; the neural mechanism involved in detecting tissue damage.

Nociceptor Receptors located in tissues that are stimulated by noxious chemical, mechanical, or thermal stimuli associated with inflammation and tissue healing.

Nonionizing radiation Radiation with insufficient energy concentration to dislodge orbiting electrons from atoms.

Numerical pain rating scale A scale used by an individual to rate pain. It is most often marked 0 to 10 from one end to the other.

Ohm's law The relationship between resistance (R) and the flow of current (I), where current is directly proportional to the voltage force (V) and inversely proportional to resistance ($I = V/R$).

Orthodromic Conduction along a nerve in the normal direction of propagation; proximal to distal for motor and distal to proximal for sensory axons.

Paraffin wax A conductive thermal modality consisting of a mixture of seven parts wax to one part mineral oil.

Paresthesia An abnormal sensation often associated with pain localized to the nerve root or peripheral nerve sensory distribution.

Paroxysmal cold hemoglobinuria Release of hemoglobin into the urine from lysed red blood cells in response to local or general exposure to cold.

Pathology A wide variety of diseases that arise from different etiologies, including infection, trauma, and degenerative processes.

Peak area of maximal beam nonuniformity ratio Total area of peak intensity delivered from an ultrasound head.

Peak pulse power The power (in watts) delivered during a pulse of ultrasound.

Percutaneous electrodes Thin wires implanted near the motor point of a muscle.

Period The inverse of frequency (*f*) calculated as $1/f$. *Period* is the duration of one cycle in a repeated event such as alternating current.

Peripheral arterial disease (PAD) Obstructive atherosclerosis or arteriosclerosis obliterans.

Phase Flow of current in one direction from the isoelectric line; commonly expressed in microseconds.

Phase duration The time from the beginning of one phase to the end of that phase.

Phonophoresis The use of ultrasound to enhance the delivery of topically applied medications through the skin.

Photobiostimulation Application of laser light on biological tissues, causing photochemical interactions between photons and cells that result in increased cellular activity.

Piezoelectricity The phenomenon in which a crystal generates an electric voltage when mechanically compressed.

Polarity The property of having charge—either positive or negative.

Polymodal fibers Nerve fibers that respond to a variety of painful stimuli.

Positive sharp wave Abnormal spontaneous electrical potentials usually initiated by needle movement within the muscle thought to represent an unstable muscle membrane.

Power The rate at which energy is being produced; measured in watts.

Pressure sores Tissue necrosis that occurs when external forces are applied for a prolonged period of time. Compression of the integumentary tissue between the external force and a bony prominence, which results in a skin wound.

Prognosis A prediction of the probable or likely outcome or course of a disease, pathology, or impairment.

Proinflammatory Effects of ultrasound that augment both the quantity and quality of the healing process.

Proteinases Cellular enzymes necessary for pathogen control, cellular migration, and tissue remodeling.

Pulse An isolated electrical event separated from the next by a finite period of time.

Pulsed current The uni- or bidirectional flow of ions or electrons, which periodically ceases for a short period of time before the next electrical event.

Pulsed electromagnetic field (PEMF) A field that produces an electric current by inducing the movement of ions in body fluids.

Pulsed lavage with concurrent suction (PLWCS) Form of mechanical debridement that applies a stream or spray under controlled pressure to remove loosely adherent necrotic tissue or foreign material.

Pulsed radio frequency radiation (PRFR) Pulses of electromagnetic energy from the radio frequency part of the spectrum.

Pulsed ultrasound Noncontiguous or interrupted ultrasound.

Pulse duration Time from the beginning to the end of all phases of a single pulse, including any interphase interval.

Pulse period The pulse on-time plus the pulse off-time.

Pulse train See *Train*.

Quadrant testing Examination of motor units in multiple regions via single needle insertion completed by redirecting the needle tip without completely removing it from the muscle.

Quadripolar An electrode configuration in which the electrodes of two separate circuits are placed on the treatment area with intention for the currents to intersect; used with quadripolar interferential current.

Radiation The process by which energy is propagated through space.

Radio frequency (RF) radiation Propagating waves between the frequencies of 10 kilohertz (kHz) and 300 gigahertz (GHz) on the electromagnetic spectrum.

Ramp-down The time it takes for the current to decrease from peak amplitude to zero amplitude during any one on-time period.

Ramp-up The time it takes for the current to increase from zero amplitude to peak amplitude for any one on-time period.

Rarefactions Areas of decreased molecular density in tissues in response to the passage of ultrasound.

Raynaud's phenomenon A vasospastic disorder resulting in paroxysmal digital cyanosis with cold exposure.

Reflection The change in the path of propagation of a beam of energy occurring at a discontinuity in the acoustical impedance.

Reflex sympathetic dystrophy (RSD) Type 1 complex regional pain syndrome that is identified when it is unknown whether there is any peripheral nerve involvement.

Refraction The bending of waves as they pass from one medium to another, proportional to the difference in acoustic impedance.

Resistance Opposition to current flow.

Rheobase The minimum strength of an electrical stimulus of infinite duration that is capable of eliciting a minimally detectable motor response.

Rise time The time required for the leading edge of a single phase to reach peak amplitude.

Russian current A burst-modulated form of alternating current that is commonly used for activation of skeletal muscle.

Saltatory conduction The propagation of action potentials along myelinated axons between nodes of Ranvier.

Sarcolemma The cell membrane of a muscle cell.

Scan See *Vector scan.*

Segmental demyelination A focal conduction abnormality characterized by the intermittent absence of myelin along normally myelinated axons.

Sensory-level stimulation The threshold at which an electrical stimulus elicits a sensory response.

Sensory nerve action potential (SNAP) The summated response of all the depolarized sensory fibers in a nerve.

Sonic Accelerated Fracture Healing System (SAFHS) Specific ultrasound units designed to deliver fixed parameters—20% duty cycle, 1.5 MHz, I_{SATA} = 30 mW/cm^2 for 20 minutes—for the purpose of promoting fracture healing.

Spasticity Velocity-dependent increase in resistance to passive stretch associated with exaggerated deep tendon reflexes.

Spatial average intensity (I_{SA}) A measure of the average acoustic power of ultrasound across the ERA of the transducer; expressed in watts per square centimeter (W/cm^2).

Spatial average temporal average intensity (I_{SATA}) An intensity value calculated by multiplying I_{SATP} by the duty cycle of the applied ultrasound.

Spatial average temporal peak intensity (I_{SATP}) The spatial average intensity of an ultrasound beam between interruptions during pulsed ultrasound.

Spatial peak intensity (I_{SP}) The acoustic power of an ultrasound beam at its highest point.

Specific gravity The ratio of the density of a substance to the density of water at 39.2°F (4°C).

Specific heat A measure of the amount of energy (heat) required to heat a material and thus the amount of energy (heat) stored in a material.

Standing wave When waves are in phase with each other, their energies are added together, creating an area of more intense energy in the tissue.

Static magnets Magnets used with constant poles for therapeutic purposes.

Streamline or laminar flow Occurs when each particle of the fluid follows a smooth path without crossover of paths.

Strength-duration curve A plot of the range of combinations of current amplitude and pulse duration that result in depolarization of sensory or motor nerves.

Suberythemal dose (SED) The situation in which none of the ultraviolet light exposure spots on the skin produce a minimal erythemal dose.

Suprathreshold stimulus An electrical stimulus exceeding the minimal threshold stimulus required to elicit depolarization.

Sustained (static) traction Form of traction applied continuously to a body part by an electrical or mechanical device.

Sweep Modulation of the beat frequency of interferential current.

Swing Denotes the temporal characteristics of the sweep of interferential current.

Symmetrical When the amplitude and duration characteristics of the two phases of the biphasic waveform are identical.

Temporal peak intensity The maximal intensity of the pulses in pulsed ultrasound.

Temporal (time) average intensity The average intensity of pulsed ultrasound, taking into account peak intensity and duty cycle.

TENS Transcutaneous electrical nerve stimulation.

Tesla (T) Unit used to measure the strength of a magnetic field. The relationship of tesla to gauss is 1 G = 10^{-4} T.

Therapeutic modalities Devices or techniques applied to a patient as part of a plan of care for a therapeutic purpose.

Thermal conductivity A measure of the efficiency of a material or tissue in conducting heat.

Thermal medium The product of thermal conductivity, density, and specific heat.

Thermoreceptors Receptors in the skin that respond to changes in temperature.

Thermotherapy Modalities primarily used to cause an increase in tissue temperature.

Traction The process of drawing or pulling apart a body part, usually a joint or joints.

Train The uninterrupted generation of pulses at a fixed frequency or a brief period of alternating current.

Transducer The part of an ultrasound device that houses a piezoelectric crystal where high-frequency electrical energy is converted to ultrasound energy.

Transverse waves Ultrasound waves perpendicular to the direction of the sound beam.

Trigger point A palpable band or nodule within the muscle or connective tissue that refers pain, elicits a local twitch response when pressed, and is generally hypersensitive.

Turbine A motor that agitates water in a whirlpool tank to create a "whirlpool" effect.

Turbulent flow The flow of fluids in erratic, small, whirlpool-like circles called *eddy currents* or *eddies*.

Ultraviolet An electromagnetic energy, invisible to the human eye, that lies between visible light and x-ray on the electromagnetic spectrum.

Ultraviolet light A (UVA) Also known as *long-wave UV,* it is nonionizing and produces fluorescence in many substances.

Ultraviolet light B (UVB) Also known as *middle-wave UV,* it is nonionizing and produces most skin erythema.

Ultraviolet light C (UVC) Ultraviolet light defined as the wavelength 180–250 nm.

Unbalanced When the area under the curve of the first phase (i.e., phase charge) of a biphasic pulse is unequal to that of the second phase; the result is a net charge, either positive or negative.

Urine incontinence Inability to control bladder function.

Vapocoolant spray A cold therapy delivery method in which one sprays rapidly evaporating chemicals on the skin; used for temporary pain relief and for preparation prior to stretching muscles with active trigger points.

Vasoconstriction Contraction of vascular smooth muscle, which results in decreased diameter of blood vessels and decreased blood flow.

Vasodilation Relaxation of vascular smooth muscles, which results in increased diameter of blood vessels and increased blood flow.

Vector scan The modulation of the amplitude of one or both currents of interferential current, which results in a rhythmic change in position of the interference pattern or vector.

Venous ulcers Chronic venous insufficiency that leads to the formation of a chronic wound.

Viscosity The internal friction present in liquids secondary to the cohesive forces between the molecules.

Visual analog scale (VAS) A scale using visual input for an individual to rate pain, most often a scale 10 cm in length.

Voltage An electrical force capable of moving charged particles through a conductor between two regions or points secondary to a potential difference between the points.

Wallerian degeneration Axon degeneration distal to the site of nerve transaction.

Waveform A description or visual representation of the characteristics of an electrical current, including shape, magnitude, and duration, depicted on an amplitude–time plot.

Zero net DC A state of no net charge between phases of a bi- or polyphasic waveform.

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