Second Edition

Clinical Electrotherapy

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Notice: Our knowledge in clinical sciences is constantly changing. As new information becomes available, changes in treatment and in the use of drugs become necessary. The authors and the publisher of this volume have taken care to make certain that the doses of drugs and schedules of treatment are correct and compatible with the standards generally accepted at the time of publication. The reader is advised to consult carefully the instruction and information material included in the package insert of each drug or therapeutic agent before administration. This advice is especially important when using new or infrequently used drugs.

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Preface

The purpose of this second edition of *Clinical Electrotherapy* is to update the electrotherapy nomenclature into the standardized format developed by the Section on Clinical Electrotherapy, American Physical Therapy Association. The standardized electrotherapy nomenclature was developed to improve communication among all professional groups who use and manufacture electrotherapy equipment.

The chapters have been revised to reflect the standard terminology used in electrotherapy. Each chapter contributor has been diligent in updating the scientific information that has been published in the past several years. Great care was taken by the chapter contributors to improve the content of each area. The student and clinician will find this edition both informative and current.

Research related to microcurrent electrical neuromuscular stimulation (MENS) is slow in developing despite increasing clinical interest. Chapter 3 offers an introduction to MENS as a therapeutic modality. The reader should be aware that MENS electrical characteristics do not significantly differ from those of existing electrostimulators. The singular difference between MENS and existing equipment is the pulse durations. Existing stimulators are capable of producing subthreshold stimuli as are the MENS-specific equipment. Randomized, controlled clinical studies are desperately needed to critically examine the stated therapeutic claims of MENS.

The reader may experience some overlap of electrophysics throughout the text. The editors have made a conscious effort to leave some of the overlap in the second edition. Repetition in some content is not necessarily a bad idea. One author may present the information in a slightly different manner, which may make all the difference for a student or clinician. The reader is offered

different approaches and sometimes different opinions for neuromuscular electrical stimulation (NMES). The different opinions result because electrotherapy remains, to some extent, a science based on the clinical model. The chapter authors are all authorities in their area and as such provide a valuable useful introduction for the variety of uses for NMES.

We have added a new chapter to the second edition. Magnetic stimulation of skeletal muscle may be a breakthrough in electrotherapy. Magnetic stimulation for chronic inflammation is also presented. These magnetic stimulation devices are currently available. For the results of ongoing clinical investigations, please review current peer-reviewed journals. This edition will serve to introduce the emerging technology of magnetically induced neuromuscular stimulation for the clinician.

The chapter on NMES for pain suppression is completely new, while chapters on biofeedback and electrophysiologic evaluation have had major revisions. We have also added wound healing by NMES to this edition. The remaining chapters have been revised to reflect recent scientific information, but with little structural change from the first edition.

This book is more than just a guide for students and clinicians. We have made every attempt to provide the scientific basis for electrotherapy. This edition offers theory, detailed review of state-of-the-art subject matter, and an immediate reference for the practitioner who seeks an answer to his or her problems on patient management techniques in clinical electrotherapy. This book is inclusive of electrotherapy as presently understood, although much research is needed to support its efficacy. Clinical electrotherapy also offers considerable potential in the successful management of patients and healthy subjects in athletic training.

Roger M. Nelson, PhD, PT
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Contents

CHAPTER 1

Review of Physiology

Nancy L. Urbscheit

The use of electrical stimulation by a physical therapist is effective in treating various disorders. It is essential, however, that the therapist possess intimate knowledge, not only of the disorder to be treated, but also of the mechanism by which electrical stimulation affects tissues in the path of the current. If such knowledge is either absent or ignored, the physical therapist will be unable to select effective, safe, and comfortable procedures of electrical stimulation.

This chapter discusses the properties of muscle and nerve cell membranes and their response to electrical stimulation. The purpose of the chapter is to provide the therapist with the knowledge necessary to understand the phenomena of both natural and invoked discharge of excitable membranes.

PROPERTIES OF EXCITABLE CELL MEMBRANES

Resting Membrane Potentials

Before the manner in which nerve and muscle cells respond to electrical stimulation can be considered, it is necessary to understand the normal properties of the cells. Both nerve and muscle cells are enveloped by a membrane that separates a charge across the membrane. This charge is maintained as long as the cell is healthy and undisturbed. This resting charge can be measured experimentally and is typically between 60 and 90 millivolts (mV), the inside of the cell being negative with respect to the outside. The charge on the membrane is a result of an unequal concentration of ions on either side of the membrane. In a normal muscle or nerve cell, the sodium (Na^+) concentration

is higher outside the cell whereas the potassium (K^+) concentration is higher inside the cell. These concentration differences are maintained by an active pump that expels Na^+ and takes in K^+ on a one-to-one exchange (Fig. 1-1A). Each ion, however, will attempt to diffuse across the membrane passively in an attempt to equalize its concentration (Fig. 1–1B). This diffusion process is much more successful for K^+ than for Na^+, because the membrane has a greater permeability to K^+ than to Na^+. As the passive diffusion of the ions continues, a charge develops on the membrane. As positively charged K^+ ions leave the cell, a net negativity develops inside the cell. The diffusion of K^+ ions out of the cell is eventually retarded as the negative charge inside the cell increases. The force acting on K^+ ions to move out of the cell to equalize the concentration is eventually matched by an opposing force to return the ions to the cell, due to the negative charge. When these forces are equal, K^+ ions will be in equilibrium, with one K^+ ion leaving the cell for every one that enters. When K^+ is at equilibrium in its passive movement in and out of the cell, the cell membrane will be charged at -100 mV.

As was mentioned earlier, however, the resting membrane potential is -60

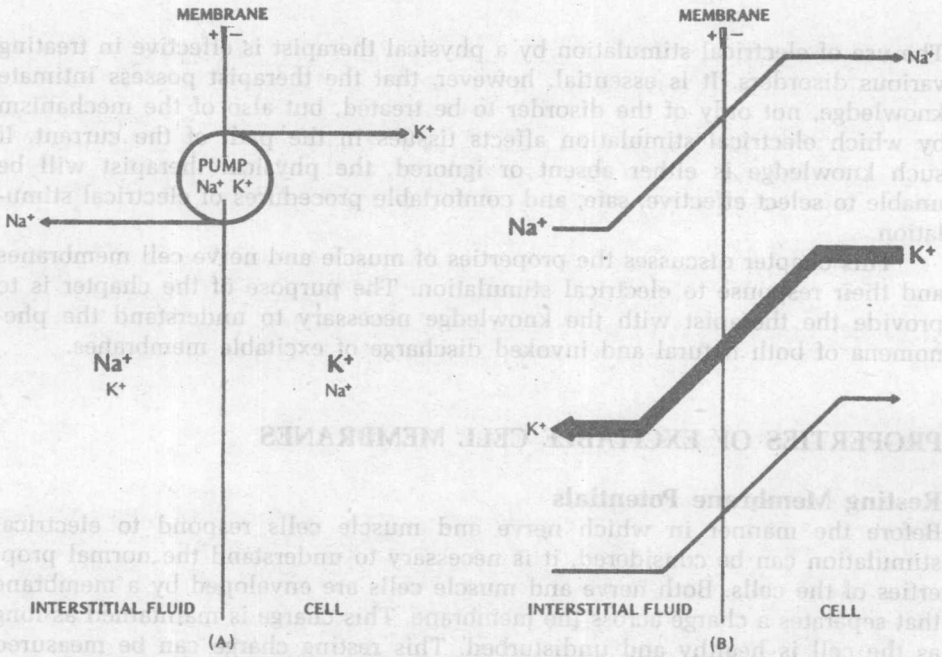

Figure 1–1. A. Active movement of Na^+–K^+ ions across an excitable membrane via an active Na^+–K^+ pump. **B.** Passive movement of Na^+ and K^+ ions. Each arrow represents the direction of movement of an ion. The width of an arrow is proportional to the number of ions moving and the slope of the arrow is proportional to the driving force.

mV. Therefore, in the typical resting excitable cell, K^+ ions are not at equilibrium, and more potassium will be passively leaving the cell than entering it.

Why does the resting membrane fail to remain at −90 mV, the equilibrium potential for potassium? Sodium ions will also be passively moving across the membrane in an attempt to equalize the concentration difference (Fig. 1–2). In the case of the Na^+ ions, the passive diffusion will be into the cell. The passive movement of Na^+ into the cell will not be as large as the passive movement of K^+ out of the cell because of the lower membrane permeability to Na^+. The inward movement of the Na^+ ions will reduce the negative charge developed by the outward leak of the K^+ ions. The movement of the Na^+ and K^+ ions reaches a steady state at approximately the resting membrane potential of −60 mV. At this potential, however, Na^+ has not equalized its concentration, and forces still exist to passively move it into the cell. In order for Na^+ to be at equilibrium (one Na^+ ion passively entering per every one that leaves), the membrane would have to be charged at +50 mV inside with respect to the outside. The reason the resting membrane potential normally lies much closer to the equilibrium potential of K^+ is that the excitable membrane is much more permeable to K^+.

In summary, the resting membrane potential of an excitable membrane is the consequence of both the concentration differences across the membrane and the different permeabilities of the resting excitable membrane to Na^+ and K^+.

Discharge of an Action Potential

Most excitable cells in the body spend little time at their resting membrane potential because excitable cells are continuously subjected to events that change the membrane's permeability to Na^+ and K^+ ions. If a cell membrane is exposed to a neural transmitter or a sensory stimulus, the cell may undergo a slight increase in its permeability to Na^+. As the number of Na^+ ions moving into the cell increases, the cell membrane will undergo a reduction of its negative charge (depolarization). If this depolarization reaches a certain critical level (threshold), Na^+ permeability will increase explosively, and Na^+ ions will rush into the cell (Fig. 1–2). The membrane potential will rapidly change to +25 to +35 mV because of the influx of these positive ions. Yet, this increase in permeability to Na^+ ions lasts only very briefly.

The initial depolarization of the membrane also increases the membrane's permeability to K^+. This increase in permeability to K^+ occurs a little more slowly than the increase to Na^+ and will reach a peak soon after the membrane has closed down again to Na^+ ions (Fig. 1–2). This latter change in the membrane's permeability to K^+ causes the membrane potential to become negative, reaching the equilibrium potential of −100 mV for potassium (hyperpolarization). The increase in permeability to K^+ ions is also very brief. The active Na^+–K^+ pump will return the ions to their original concentrations, and the resting membrane potential will be restored quickly. This sudden, rapid alteration in the membrane's potential is known as an *action potential*. The

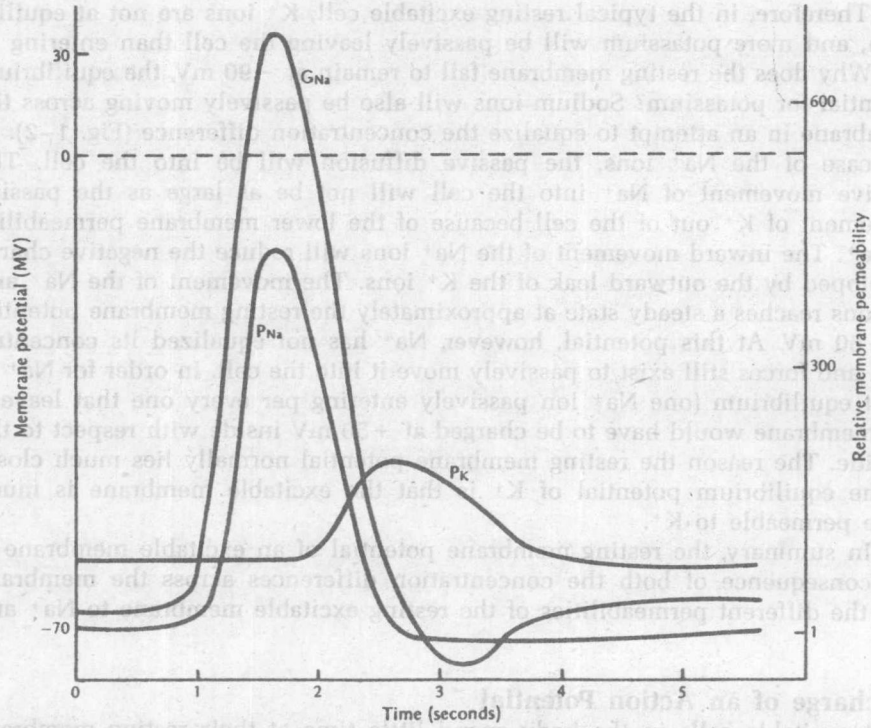

Figure 1–2. Changes in the permeability of K^+ (P_K) and Na^+ (P_{Na}) during an action potential.

ability of a membrane to generate an action potential is the property that defines an excitable membrane.

Although normal excitable cells undergo action potentials very frequently, there do exist conditions that will cause an excitable membrane to fail. If a membrane has been depolarized at a subthreshold level for a period of time, an action potential will not be evoked, even if the normal threshold depolarization occurs. The membrane can discharge an action potential, however, if depolarization reaches a level that is much higher than the normal firing threshold. The excitable membrane in these circumstances is said to have *accommodated.* The subthreshold, prolonged depolarization apparently raises the threshold level of depolarization at which the sudden explosive increase in permeability to Na^+ occurs. A strong, slowly rising depolarization may also prevent the initiation of an action potential by blocking the increase in permeability to Na^+.

If a membrane becomes sufficiently hyperpolarized, it may also be unable to discharge an action potential, because the firing threshold cannot be easily reached.

Propagation of an Action Potential

An action potential occurring in one region of an excitable membrane can trigger an action potential in a neighboring region of the membrane. The depolarization occurring in the beginning of an action potential causes a localized flow of current around the site of the action potential. This current may cause a threshold depolarization of the neighboring membrane, which may evoke an action potential. If the excitable membrane is very large, such as in a nerve axon or a muscle fiber, the action potential can be propagated over the entire membrane. If propagation is occurring in the normal direction (for example, progressing in a proximal direction for a sensory fiber), it is called *orthodromic conduction*. *Antidromic conduction* occurs in the direction opposite to normal and can be evoked by electrical stimulation.

The speed at which an action potential is propagated varies from one excitable membrane to another. In nerve cell fibers that are unmyelinated, action potentials create localized eddy currents that cross the membrane (Fig. 1–3A). The span of membrane encompassed by the eddy currents is small because of the high resistance of the membrane. In nerve membranes that are myelinated, the local current generated by an action potential travels inside the fiber, because the myelin prevents the eddy currents from crossing the membrane. The next action potential will occur only where a break in the myelin exists. Conduction in a myelinated fiber is much faster than in an unmyelinated fiber. In the unmyelinated fibers, numerous small areas of the membrane must sequentially undergo an action potential to propagate the length of an excitable membrane. In myelinated fibers, the action potential skips from node to node; thus, fewer action potentials will be propagated along the length of the membrane. Each action potential lasts approximately 1.0 milliseconds (ms); thus, the fewer action potentials produced during propagation of activity along the length of a nerve fiber, the less time to conduct from one end to the other.

Conduction velocity within a group of either unmyelinated or myelinated

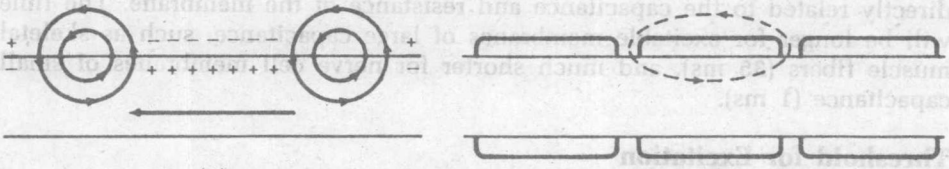

Figure 1–3. Local current flow generated by an action potential in nerve axons. **A.** Eddy currents produced by an action potential in an unmyelinated axon. The arrow indicates direction of impulse propagation. Propagated impulses would proceed only in the orthodromic direction, as the membrane would be refractory in the antidromic direction. **B.** Local current flow evoked by an action potential in a myelinated axon. The current crosses the membrane only at a node.

fibers will vary, as well. The larger the diameter of the fiber, the less resistance offered to conduction currents generated by action potentials, and thus, the faster the speed of conduction of an action potential. Nerve fibers also conduct faster if the temperature is raised.

Conduction of an action potential along a nerve fiber can be stopped in a number of ways. If a nerve fiber is exposed to local pressure or to anoxia, that region of the fiber will begin to depolarize slowly. If this occurs, the sudden, explosive increase in permeability to Na^+ ions will not occur; therefore, propagation of an action potential will cease in this area. An injection of a local anesthetic (such as novocaine) will block conduction of an action potential in the region by preventing the sudden increase in permeability to Na^+ ions. Extreme cooling can also stop conduction.

RESPONSE OF AN EXCITABLE MEMBRANE TO ELECTRICAL STIMULATION

The behavior of an excitable membrane can be modified by the application of electrical current through two external electrodes. The membrane will undergo depolarization at the cathode (negative electrode) and hyperpolarization at the anode (positive electrode).

Subthreshold Currents

If the amplitude of the electrical stimulus is too weak to produce a threshold depolarization at the cathode, an action potential will not take place. An understanding of subthreshold current flow is a prerequisite to a discussion of how an action potential is generated by electrical stimulation. A membrane exposed to a subthreshold electrical stimulus does not immediately change its charge. Upon exposure to a maintained electrical stimulus, the excitable membrane will eventually reach a steady state in which the charge on the membrane reaches its peak in response to the externally applied current. The amount of time that it takes an applied current to charge the cell membrane is directly related to the capacitance and resistance of the membrane. The time will be longer for excitable membranes of large capacitance, such as skeletal muscle fibers (35 ms), and much shorter for nerve cell membranes of small capacitance (1 ms).

Threshold for Excitation

If the applied current depolarizes the membrane to threshold, the membrane will fire an action potential. Whether the membrane reaches the threshold depends partially upon the stimulus amplitude. The stimulus amplitude required, however, varies from membrane to membrane, and even from minute to minute in the same membrane. For example, narrow-diameter fibers require a higher stimulus amplitude to reach threshold because of the high internal resistance of such fibers to current flow. In addition, a fiber that has been exposed to a continuous level of depolarization at the cathode may eventually

become inexcitable unless the stimulus amplitude is increased (known as *accommodation*, or *depolarization block*).

The duration of the stimulus also affects whether the membrane reaches firing threshold. If a 1.0-ms pulse is applied to a skeletal muscle cell membrane, the membrane will not fire regardless of the amplitude of the stimulus. The amount of time that a skeletal muscle cell needs to reach a maximum charge in response to an externally applied current is approximately 35 ms. To ensure that the membrane responds to the stimulus, a pulse duration closer to 35 ms will be needed. If a moderately shorter stimulus is used, a stronger stimulus amplitude may cause the membrane to fire. The relationship between the threshold stimulus amplitude and the stimulus duration needed to evoke a consistent response is plotted on a strength—duration curve.

Refractory Periods

When an electrical current is applied to a nerve cell membrane, the stimulation may be applied in pulses separated by time. Even if the pulses are of sufficient duration and amplitude, the membrane may not discharge to the second pulse if it occurs too close to the first because the membrane needs approximately 0.5 ms to recover its excitability after an action potential. This recovery time is called the *absolute refractory period*. A higher-amplitude stimulus may be needed before the membrane will again fire for a period between 0.5 and 1 ms after having discharged an action potential. This time of recovery is called the *relative refractory period*. The refractoriness of the membrane limits the maximum frequency of discharge that an excitable membrane can undergo.

Stimulating Nerve Trunks

When therapists apply electrodes for electrical stimulation, the path of current will most likely include nerve fibers of different diameters. Large fibers require the lowest stimulus amplitude and shortest pulse duration to reach threshold because of their lower axonal resistance. Thin fibers require a higher stimulus amplitude and longer duration to reach threshold. Fibers that provide the sensation of cutaneous pain are typically of small diameter. Thus, use of short pulse duration, lower stimulus amplitude, or both, may reduce the painful sensation accompanying electrical stimulation.

When stimulating a nerve to evoke a muscle contraction, not all of the alpha motor axons in the nerve will discharge at the same threshold or stimulus amplitude. Because of differences in both axonal diameter and anatomical orientation to the current, the stimulus amplitude may have to be quite high (sometimes beyond tolerance) to recruit all of the motor axons.

SPECIFIC RESPONSES OF MUSCLE TO ELECTRICAL STIMULATION

In a normal muscle, electrical stimulation evokes a contraction by excitation of the nerve rather than by excitation of the muscle directly. Nerve fibers need

only a short pulse duration to be able to discharge, whereas muscle requires a much longer pulse duration in order to respond. Few of the electrical stimulators in use today have pulse durations long enough to excite a muscle membrane directly.

Critical Fusion Frequency

If a muscle nerve is exposed to a single electrical pulse of adequate duration and amplitude, a single twitch contraction results. The evoked twitch is a synchronous contraction of all of the motor units whose alpha motor axons are excited by the pulse. The tension produced by a group of motor units may be increased if a volley of pulses, rather than a single pulse, is used. When muscle twitches are evoked in rapid succession, there may not be enough time for complete relaxation between twitches. If the tension developed during one twitch fuses with that of the successive twitch, the tension will be cumulative. When the twitches are completely fused (Fig. 1–4), the contraction produced is said to be *tetanic*. The frequency of pulses at which this occurs is called the *critical fusion frequency* (CFF).

The CFF of each muscle depends upon its individual twitch duration. Postural muscles have slow twitch times (approximately 75 ms) and, therefore, undergo a tetanic contraction at around 13 to 15 pulses per second (pps). By contrast, muscles in the hands have faster twitch times (approximately 25 ms), thereby undergoing a tetantic contraction around 40 pps.

Gradation of Tension

The contractions evoked by electrical stimulation are nonphysiologic, in that they do not duplicate the normal recruitment and firing patterns of motor units. During a normal contraction, motor units fire asynchronously. Therefore, a complete tetanic contraction is not possible. Normally, motor units can be recruited to produce very fine, smooth gradations of tensions. With electrical stimulation, however, the motor units participating fire synchronously and are recruited in larger groups; hence, it is difficult to produce small, smooth increments of tension. If a weak, but smooth, contraction of a muscle is desired during electrical stimulation, the CFF of a muscle should be used (to

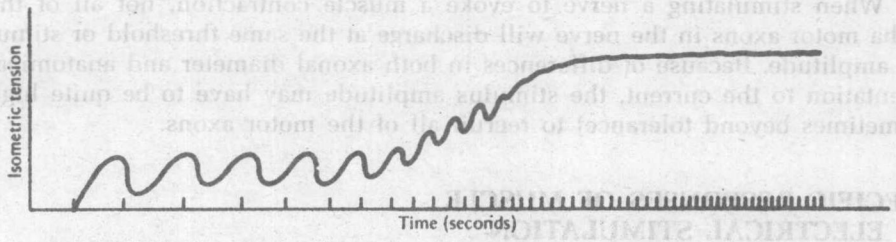

Figure 1–4. Accumulated tension of isometric twitches of skeletal muscle produced by gradually increasing the frequency of electrical stimulation.