

Chapter 5: Basic Principles of Electricity and Electrical Stimulating Currents

Daniel N. Hooker; William E. Prentice

OBJECTIVES

Following completion of this chapter, the student will be able to:

- Define common terminology related to electricity.
- Differentiate between monophasic, biphasic, and pulsatile currents.
- Differentiate between direct (DC), alternating (AC), and pulsatile (PC) currents.
- Explain current flow through various types of biologic tissue.
- Discriminate between series and parallel circuit arrangements.
- Discuss the various treatment parameters including waveforms, current modulation frequency, intensity, duration, polarity, and electrode placement that must be considered with electrical stimulating currents.
- Explain nerve, muscle, and nonexcitatory cell physiologic responses to electrical stimulation.
- Discuss the clinical goals of using electrical stimulating currents to stimulate either motor nerves to induce muscle contraction, or sensory nerves for the purpose of modulating pain.
- Differentiate between the various currents that can be selected on many modern generators including high volt, TENS microcurrent, Russian, interferential, premodulated interferential, low volt, and H-wave.
- Discuss additional uses for electrical currents including bone growth stimulation, functional electrical stimulation, and transcranial electrical stimulation.
- Be able to create a safe environment when using electrical equipment.

Many of the modalities discussed in this book may be classified as electrical modalities. These pieces of equipment have the capabilities of taking the electrical current flowing from a wall outlet and modifying that current to produce a specific, desired physiologic effect in human biologic tissue.

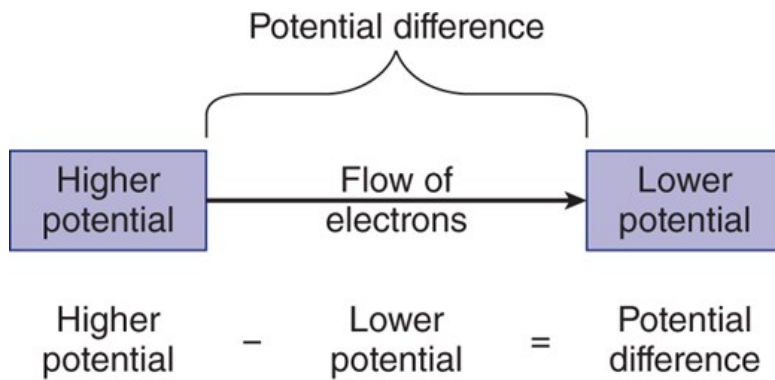
Understanding the basic principles of electricity usually is difficult even for the clinician who is accustomed to using electrical modalities on a daily basis. To understand how current flow affects biologic tissue, it is first necessary to become familiar with some of the principles and terminology that describe how electricity is produced and how it behaves in an electrical circuit.^{172,180,186}

COMPONENTS OF ELECTRICAL CURRENTS

All matter is composed of atoms that contain positively and negatively charged particles called **ions**. These charged particles possess electrical energy and thus have the ability to move about. They tend to move from an area of higher concentration toward an area of lower concentration. An electrical force is capable of propelling these particles from higher to lower energy levels, thus establishing **electrical potentials**. The more ions an object has, the higher its potential electrical energy is. Particles with a positive charge tend to move toward negatively charged particles, and those that are negatively charged tend to move toward positively charged particles ([Figure 5-1](#)).¹

Figure 5-1.

The difference between high potential and low potential is potential difference. Electrons tend to flow from areas of higher concentration to areas of lower concentration. A potential difference must exist if there is to be any movement of electrons.



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Electrons are particles of matter possessing a negative charge and very small mass. The net movement of electrons is referred to as an **electrical current**. The movement or flow of these electrons will always go from a higher potential to a lower potential.² An electrical force is oriented only in the direction of the applied force. This flow of electrons may be likened to a domino reaction.

The unit of measurement that indicates the rate at which electrical current flows is the **ampere**; 1 A is defined as the movement of 1 C or 6.25×10^{15} electrons/s. Amperes indicate the rate of electron flow, whereas coulombs indicate the number of electrons. In the case of therapeutic modalities, **current** flow is generally described in milliamperes (1/1000 of an ampere, denoted as mA) or in microamperes (1/1,000,000 of an ampere, denoted as μ A).³

The electrons will not move unless an electrical potential difference in the concentration of these charged particles exists between two points. The electromotive force, which must be applied to produce a flow of electrons, is called a **volt** and is defined as the difference in electron population (potential difference) between two points.⁴

Voltage is the force resulting from an accumulation of electrons at one point in an electrical circuit, usually corresponding to a deficit of electrons at another point in the circuit. If the two points are connected by a suitable conductor, the potential difference (in electron population) will cause electrons to move from the area of higher population to the area of lower population.

Commercial current flowing from wall outlets produces an electromotive force of either 115 or 220 V. The electrotherapeutic devices used in injury rehabilitation modify voltages. Electrical generators are sometimes referred to as being either low or high volt. These terms are not very useful, although some older texts have referred to generators that produce less than 150 V as *low volt* and those that produce several hundred volts as *high volt*.⁴

Electrons can move in a current only if there is a relatively easy pathway to move along. Materials that permit this free movement of electrons are referred to as **conductors**. **Conductance** is a term that defines the ease with which current flows along a conducting medium and is measured in units called siemens. Metals (**copper**, gold, **silver**, aluminum) are good conductors of electricity, as are electrolyte solutions, because both are composed of large numbers of free electrons that are given up readily. Thus, materials that offer little opposition to current flow are good conductors. Materials that resist current flow are called **insulators**. Insulators contain relatively fewer free electrons and thus offer greater resistance to electron flow. Air, wood, and glass are all considered insulators. The number of amperes flowing in a given conductor is dependent both on the voltage applied and on the conduction characteristics of the material.⁵

The opposition to electron flow in a conducting material is referred to as **resistance** or **electrical impedance** and is measured in a unit known as an **ohm**. Thus, an electrical circuit that has high resistance (ohms) will have less flow (amperes) than a circuit with less resistance and the same voltage.⁶

The mathematical relationship between current flow, voltage, and resistance is demonstrated in the following formula:

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$$\text{Current flow} = \frac{\text{Voltage}}{\text{Resistance}} \quad \text{Current flow} = \text{Voltage} \div \text{Resistance}$$

The above formula is the mathematical expression of **Ohm's law**, which states that the current in an electrical circuit is directly proportional to the voltage and inversely proportional to the resistance.⁷

An analogy comparing the movement of water with the movement of electricity may help to clarify this relationship between current flow, voltage, and resistance (Table 5-1). For water to flow, some type of pump must create a force to produce movement. Likewise, the volt is the pump that produces the electron flow. The resistance to water flow is dependent on the length, diameter, and smoothness of the water pipe. The resistance to electrical flow depends on the characteristics of the conductor. The amount of water flowing is measured in gallons, whereas the amount of electricity flowing is measured in amperes.

Table 5-1

Electron Flow as Analogous to Water Flow

ELECTRON FLOW	WATER FLOW
Volt	=Pump
Ampere	=Gallon
Ohm (property of conductor)	=Resistance (length and distance of pipe)

The amount of energy produced by flowing water is determined by two factors: (1) the number of gallons flowing per unit of time and (2) the pressure created in the pipe. Electrical energy or power is a product of the voltage or electromotive force and the amount of current flowing. Electrical power is measured in a unit called a **watt**:

$$\text{Watt} = \text{volts} \times \text{amperes} \quad \text{Watt} = \text{volts} \times \text{amperes}$$

Simply, the watt indicates the rate at which electrical power is being used. A watt is defined as the electrical power needed to produce a current flow of 1 A at a pressure of 1 V.

ELECTROTHERAPEUTIC CURRENTS

Electrotherapeutic devices generate three different types of current that, when introduced into biologic tissue, are capable of producing specific physiologic changes. These three types of current are referred to as direct (DC), alternating (AC), or pulsatile (PC).

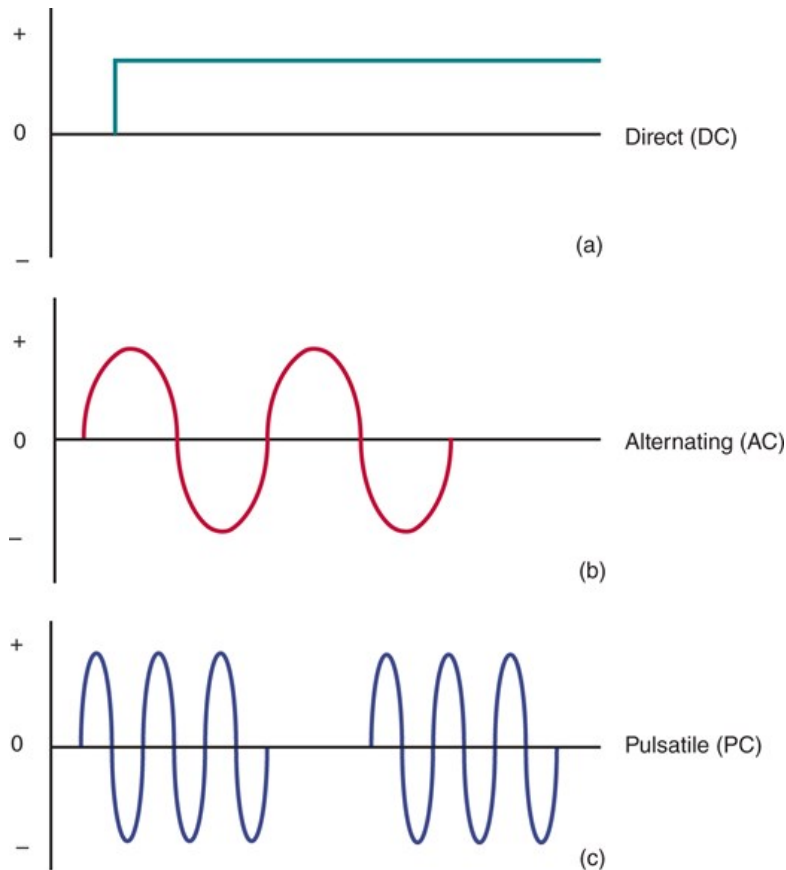
Types of electrical current are as follows:

- direct (DC);
- alternating (AC);
- pulsatile or PC.

Direct current or DC, also referred to in some texts as galvanic current, has an uninterrupted unidirectional flow of electrons toward the positive pole⁹ (Figure 5-2a). On most modern DC devices, the polarity and thus the direction of current flow can be reversed.⁸

Figure 5-2.

(a) Direct current (DC). (b) alternating current (AC). (c) Pulsatile current (PC).



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In **alternating current** or **AC**, the continuous flow of electrons is bidirectional, constantly changing direction or, stated differently, reversing its polarity. Electrons flowing in an **AC** always move from the negative to positive pole, reversing direction when polarity is reversed (Figure 5-2b).

Pulsatile current or **PC** usually contain three or more pulses grouped together and may be unidirectional or bidirectional (Figure 5-2c). These groups of pulses are interrupted for short periods of time and repeat themselves at regular intervals. PC are used in interferential and so-called Russian currents.^{10,11}

GENERATORS OF ELECTROTHERAPEUTIC CURRENTS

A great deal of confusion exists relative to the terminology used to describe electrotherapeutic currents.^{12,175} Basically, all therapeutic electrical generators, regardless of whether they deliver DC, **AC**, or **PC** through electrodes attached to the skin, are **transcutaneous electrical stimulators**. The majority of these are used to stimulate peripheral nerves and are correctly called **transcutaneous electrical nerve stimulators (TENS)**. Occasionally, the terms **neuromuscular electrical stimulator (NMES)** or **electrical muscle stimulator (EMS)** are used; however, these terms are only appropriate when the electrical current is being used to stimulate muscle directly, as would be the case with denervated muscle where peripheral nerves are not functioning. A **microcurrent electrical nerve stimulator (MENS)** uses current intensities too small to excite peripheral nerves. **Low-intensity stimulator (LIS)** is a term that has also been used to refer to **MENS**.^{10,13,14} Currently MENS and LIS are most often referred to simply as **microcurrent**.

Clinical Decision-Making Exercise 5-1

A student asks the clinical instructor the difference between a TENS unit and an NMES unit. How should the clinical instructor respond?

There is no relationship between the type of current the generator delivers to the patient and the type of current the generator uses as a power source (i.e., a wall outlet or battery). Generators that produce electrotherapeutic currents may be driven by either AC or DC. Devices that plug into the standard electrical wall outlet use AC. The commercially produced AC changes its direction of flow 120 times/s. In other words, there are 60 complete cycles/s. The number of cycles occurring in 1 second is called **frequency** and is indicated in hertz, pulses per second (pps), or cycles per second (cps). The voltage of electromotive force producing this alternating directional flow of electrons is set at a standard 115 or 220 V. Thus, commercial AC is produced at 60 Hz with a corresponding voltage of either 115 or 220 V.

Other electrotherapeutic devices are driven by batteries that always produce DC, ranging between 1.5 and 9 V, although the devices driven by batteries may, in turn, produce modified types of current.

ELECTRICAL CIRCUITS

The path of current from a generating power source through various components back to the generating source is called an electrical **circuit**.¹⁵ In a closed circuit, electrons are flowing, and in an open circuit, the current flow ceases. Electronic circuits are not ordinarily composed of single elements; they often encompass several branches or components with different resistances. The current in each branch may be easily calculated if the individual resistances are known and if the amount of voltage applied to the circuit is also known.¹⁶

With the development of the microelectronics industry, electrical circuits can be extremely complex. However, all electrical circuits have several basic components; a power source that produces voltage, and conducting medium that carries the flowing electrons. Finally, there is some component or group of components that is driven by this flowing current. These driven elements provide resistance to electrical flow.¹⁶

Series and Parallel Circuits

The components that provide resistance to current flow may be connected to one another in one of the two different patterns, a **series circuit** or a **parallel circuit**. The main difference between these two is that in a series circuit there is only one path for current to get from one terminal to another. In a parallel circuit, two or more routes exist for current to pass between the two terminals.

In a series circuit the components are placed end to end (Figure 5–3). The number of amperes of an electrical current flowing through a series circuit is exactly the same at any point in that circuit. The resistance to current flow in this total circuit is equal to the resistance of all the components in the circuit added together:

$$R_T = R_1 + R_2 + R_3 \quad R_T = R_1 + R_2 + R_3$$

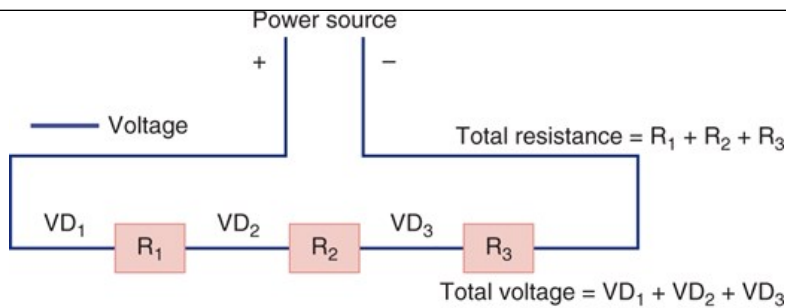
Electrical energy is required to force the current through the resistor, and this energy is dissipated in the form of heat. Consequently, there is a decrease in voltage at each component such that the total voltage at the beginning of the circuit is equal to the sum of the voltage decreases at each component:

$$V_T = V_{D1} + V_{D2} + V_{D3} \quad V_T = V_{D1} + V_{D2} + V_{D3}$$

In a parallel circuit, the component resistors are placed side by side and the ends are connected (Figure 5–4). Each of the resistors in a parallel circuit receives the same voltage.

Figure 5–3.

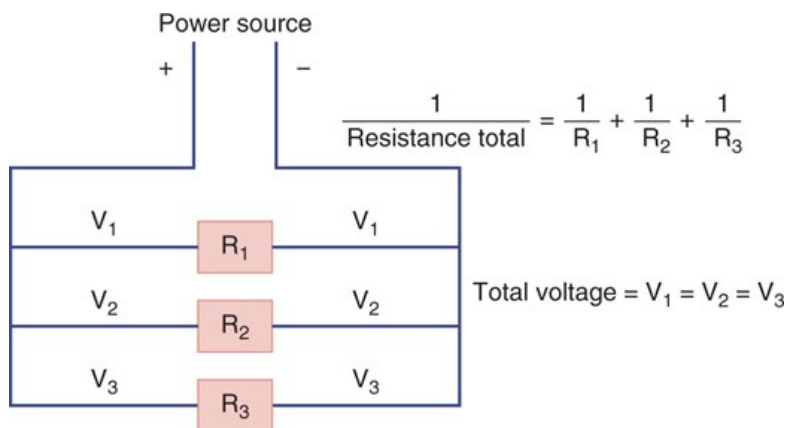
In a series circuit, the component resistors are placed end to end. The total resistance to current flow is equal to the resistance of all the components added together. There is a voltage decrease at each component such that the sum of the voltage decreases is equal to the total voltage.



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Figure 5-4.

In a parallel circuit, the component resistors are placed side by side and the ends are connected. The current flow in each of the pathways is inversely proportional to the resistance of the pathway. The total voltage is the sum of the voltages at each component.



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The current passing through each component depends on its resistance. Therefore, the total voltage will be exactly the same as the voltage at each component:

$$V_T = V_1 + V_2 + V_3 \quad V_T = V_1 + V_2 + V_3$$

Each additional resistance added to a parallel circuit in effect decreases the total resistance. Adding an alternative pathway, regardless of its resistance to current flow, improves the ability of the current to get from one point to another. The current will, in general, choose the pathway that offers the least resistance. The formula for determining total resistance in a parallel circuit according to Ohm's law is:

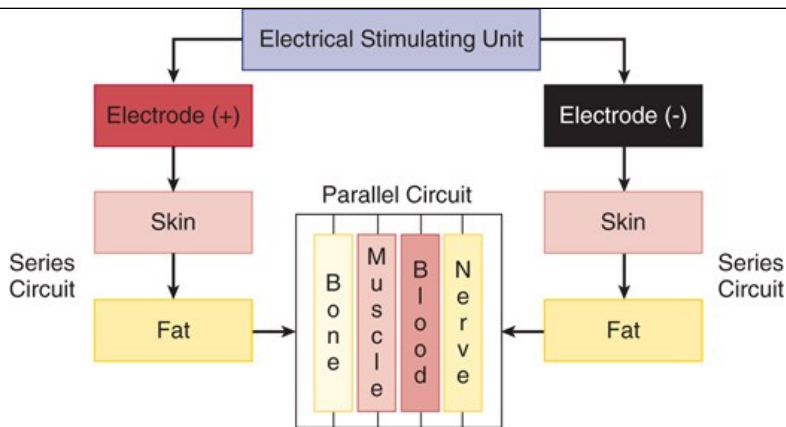
$$\frac{1}{R_T} = \frac{1}{R_1} + \frac{1}{R_2} + \frac{1}{R_3} \quad 1/R_T = 1/R_1 + 1/R_2 + 1/R_3$$

Thus, component resistors connected in a series circuit have a higher resistance and lower current flow, and resistors in a parallel circuit have a lower resistance and a higher current flow.

The electrical stimulating units, in general, make use of some combination of both series and parallel circuits.¹⁷ For example, to elicit a muscle contraction, the electrodes from an electrical stimulating unit are placed on the skin (Figure 5-5). The current from those electrodes must pass directly through the skin and fat. The total resistance to current flow seen by the electrical stimulating unit is equal to the combined resistances at each electrode. This passage of current through the skin is basically a series circuit.

Figure 5-5.

The electrical circuit that exists when electrons flow through human tissue is in reality a combination of a series and a parallel circuit.



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After the current passes through the skin and fat, it comes in contact with a number of different types of biologic tissues (bone, connective tissue, blood, muscle). The current has several different pathways through which it may reach the muscle to be stimulated. The total current traveling through these tissues is the sum of the currents in each different type of tissue, and because there are additional tissues through which current may travel, the total resistance is effectively reduced. Thus, in this typical application of a therapeutic modality, both parallel and series circuits are used to produce the desired physiologic effect.

Current Flow through Biologic Tissues

As stated previously, electrical current tends to choose the path that offers the least resistance to flow or, stated differently, the material that is the best conductor.¹⁸ The conductivity of the different types of tissue in the body is variable. Typically, tissue that is highest in water content and consequently highest in ion content is the best conductor of electricity.

The skin has different layers that vary in water content, but generally the skin offers the primary resistance to current flow and is considered an insulator. Skin preparation for the purpose of reducing electrical impedance is of primary concern with electrodiagnostic apparatus, but it is also important with electrotherapeutic devices. The greater the impedance of the skin, the higher the voltage of the electrical current must be to stimulate underlying nerve and muscle. Chemical changes in the skin can make it more resistant to certain types of current. Thus, skin impedance is generally higher with DC than with biphasic current.¹⁹

Blood is a biologic tissue that is composed largely of water and ions and is consequently the best electrical conductor of all tissues. Muscle is composed of about 75% water and depends on the movement of ions for contraction. It tends to propagate an electrical impulse much more effectively in a longitudinal direction than transversely. Muscle tendons are considerably more dense than muscle, contain relatively little water, and are considered poor conductors. Fat contains only about 14% water and is thought to be a poor conductor. Peripheral nerve conductivity is approximately six times that of muscle. However, the nerve generally is surrounded by fat and a fibrous sheath, both of which are considered to be poor conductors. Bone is extremely dense, contains only about 5% water, and is considered to be the poorest biologic conductor of electrical current. It is essential for the clinician to understand that many biologic tissues will be stimulated by an electrical current. Selecting the appropriate treatment parameters is critical if the desired tissue response is to be attained.²⁰

CHOOSING APPROPRIATE TREATMENT PARAMETERS

To make the treatment options very simple for the clinician, the equipment manufacturers have created preset treatment protocols for each type of current. A clinician may choose the preset protocols or can choose to manually alter a number of treatment parameters including waveforms, current modulation, frequency, intensity, duration, and polarity. He or she must also choose the size and placement location of the electrodes.

WAVEFORMS

The term **waveform** indicates a graphic representation of the shape, direction, **amplitude**, **duration**, and pulse frequency of the electrical current the electrotherapeutic device produces, as displayed by an instrument called an oscilloscope.

Waveform shapes are as follows:

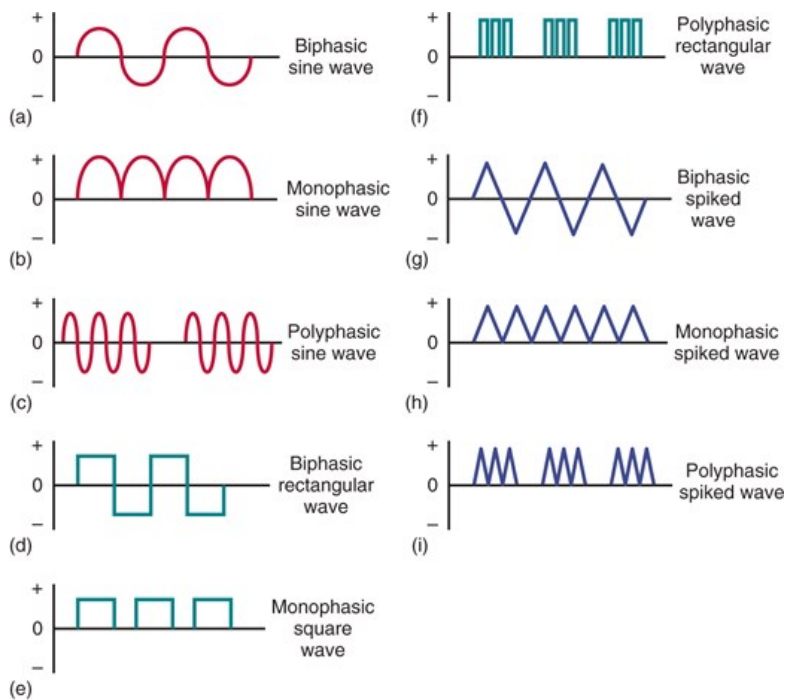
- sinusoidal;
- rectangular;
- square;
- spiked.

Waveform Shape

Electrical currents may display a *sinusoidal*, *rectangular*, *square*, or *spiked* waveform shape, depending on the capabilities of the generator producing the current (Figure 5–6). The waveform shapes may be *monophasic*, *biphasic*, or *polyphasic*.

Figure 5–6.

Waveforms of AC, DC, or PC current may be either sine, rectangular, square, or spiked in shape.



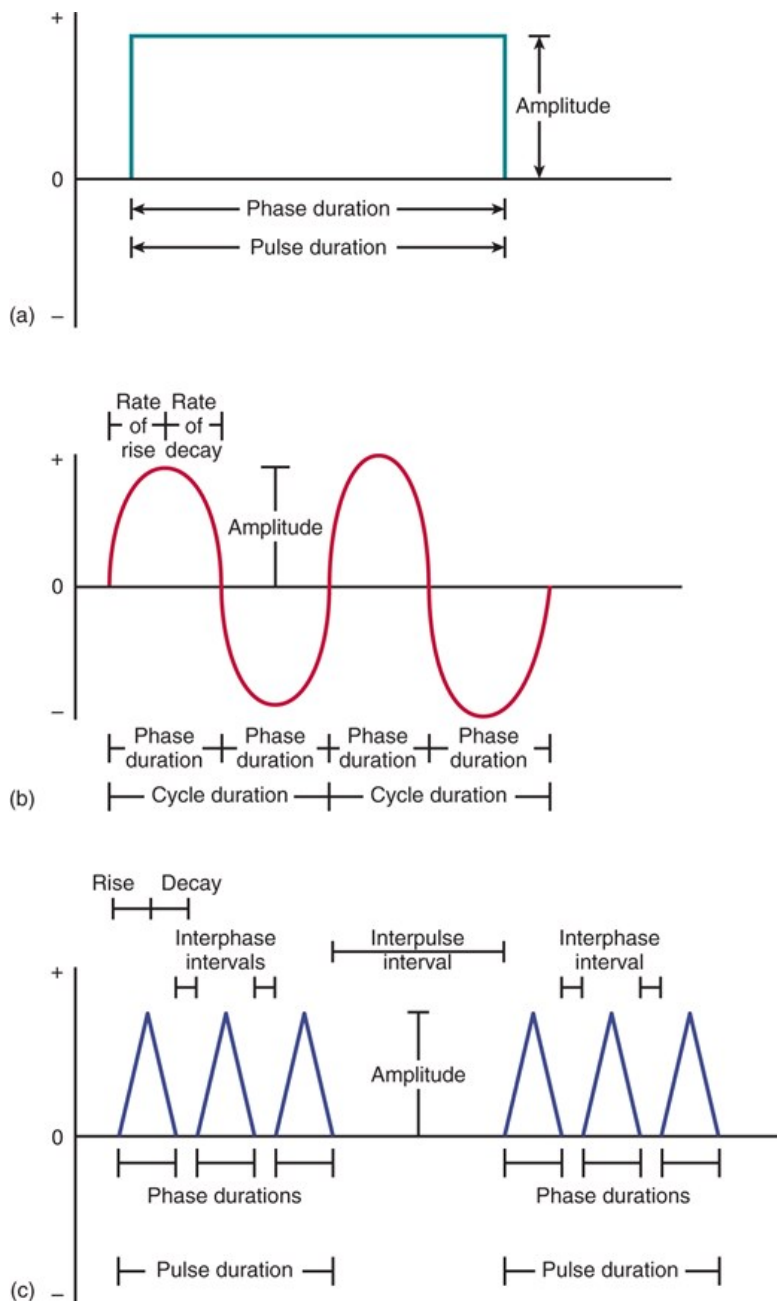
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Pulses versus Phases and Direction of Current Flow

On an oscilloscope, an individual waveform is referred to as a **pulse**. A pulse may contain one or more **phases**, which is that portion of the pulse that rises in one direction either above or below the baseline for some period of time. Direct current is unidirectional and has only one phase (Figure 5–7a). Thus the waveform for DC current is said to be *monophasic*. It produces waveforms that have only a single pulse and phase, which are the same (Figure 5–7a). Because current flow is unidirectional, it always flows in the same direction toward either the positive or negative pole. With DC the terms pulse duration and phase duration only indicate the length of time that current is flowing.

Figure 5–7.

Characteristics of (a) monophasic current, (b) biphasic current, and (c) polyphasic current.

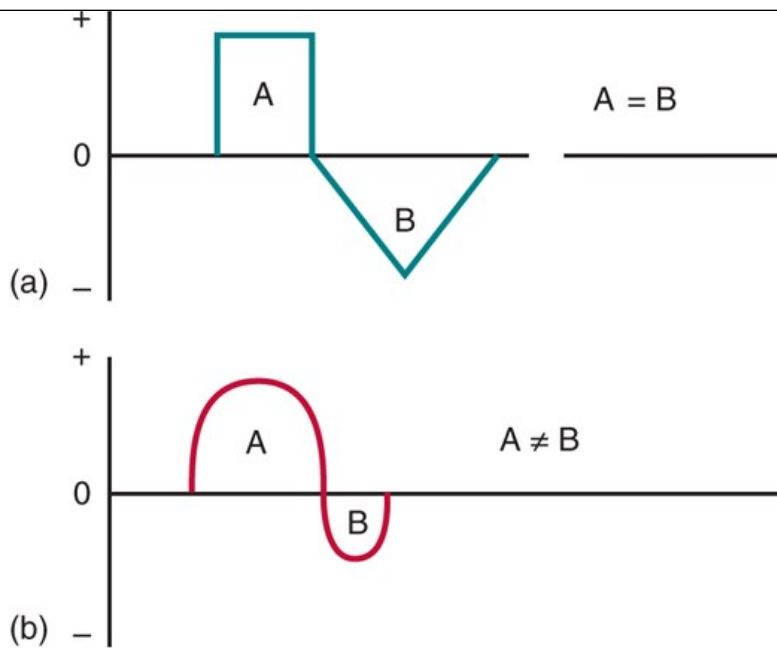


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Conversely, alternating current produces waveforms that are *biphasic*, meaning there are two separate phases during each individual **cycle**. (Cycle applies to biphasic current, whereas *pulse* applies to monophasic current.) Current flow is bidirectional, reversing direction or polarity once during each cycle. Biphasic waveforms may be symmetric or asymmetric.¹¹ A biphasic symmetric waveform has the same shape and size for each phase in both directions (Figure 5-7b). In contrast, a biphasic asymmetric waveform has different shapes for each phase (Figure 5-8a). Asymmetric waveforms can be either balanced or unbalanced. If the phases are balanced, the net charge in each direction is equal. If the phases are unbalanced, one phase has a greater net charge than the other and some movement of ions will occur (Figure 5-8b).

Figure 5-8.

Asymmetric waveforms. (a) Balanced asymmetric current. (b) Unbalanced asymmetric current.



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Polyphasic waveforms are representative of electrical current that is conducted as a series of pulses of short duration (milliseconds) and may be either monophasic or biphasic. The time that each pulse lasts is called the **phase duration**. Sometimes single pulses may be interrupted by an **interphase interval**. Pulse duration is the sum of all phases plus the interphase interval. With PC there is always a short period of time when current is not flowing between the two phases called the **interpulse interval** (Figure 5-7c).

Pulse Amplitude

The amplitude of each pulse reflects the intensity of the current, the maximum amplitude being the tip or highest point of each phase (see Figure 5-7). Amplitude is measured in amperes, microamperes, or milliamperes. The term *amplitude* is synonymous with the terms *voltage* and *current intensity*. Voltage is measured in volts, microvolts, or millivolts. The higher the amplitude, the greater the peak voltage or intensity. However, the peak amplitude should not be confused with the total amount of current being delivered to the tissues.

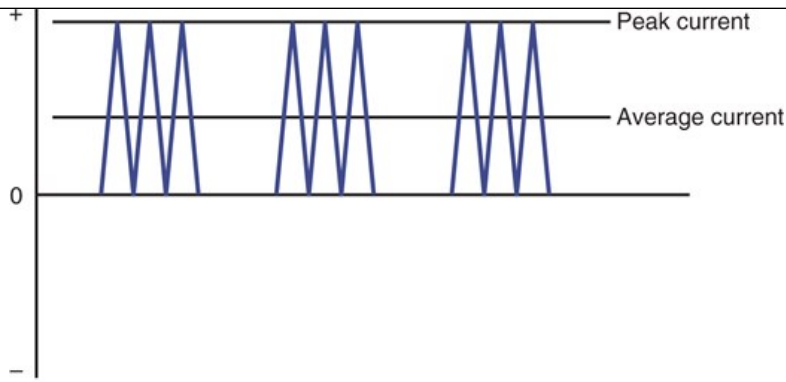
On electrical generators that produce short-duration pulses, the total current produced is low compared with peak current amplitudes owing to long interpulse intervals that have current amplitudes of zero. Thus, the **total current** (average), or the amount of current flowing per unit of time, is relatively low, ranging from as low as 2 mA to as high as 100 mA in some interferential currents (IFC). Total current can be increased by either increasing pulse duration or increasing pulse frequency or by some combination of the two (Figure 5-9).

Clinical Decision-Making Exercise 5-2

An injured lacrosse player has a strain of the right quadriceps muscle group. The clinician has decided to use an electrical stimulator to induce a muscle contraction and is explaining how the electricity will do this when the athlete becomes fearful that there will be an electrical shock. What should the clinician explain about using electrical current to reassure the patient?

Figure 5-9.

Average current is low compared with peak current amplitudes due to long interpulse intervals.



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Pulse Charge

The term **pulse charge** refers to the total amount of electricity being delivered to the patient during each pulse (measured in coulombs or microcoulombs). With monophasic waveforms, the phase charge and the pulse charge are the same and always greater than zero. With biphasic waveform the pulse charge is equal to the sum of the phase charges. If the pulse is symmetric, the net pulse charge is zero. In asymmetric pulses the net pulse charge is greater than zero, which is a monophasic waveform by definition.¹⁰

Pulse Rate of Rise and Decay Times

The **rate of rise** in amplitude, or the rise time, refers to how quickly the pulse reaches its maximum amplitude in each phase. Conversely, **decay time** refers to the time in which a pulse goes from peak amplitude to 0 V. The rate of rise is important physiologically because of the **accommodation** phenomenon, in which a fiber that has been subjected to a constant level of depolarization will become unexcitable at that same intensity or amplitude. Rate of rise and decay times are generally short, ranging from nanoseconds (billionths of a second) to milliseconds (thousandths of a second) (see Figure 5-6):

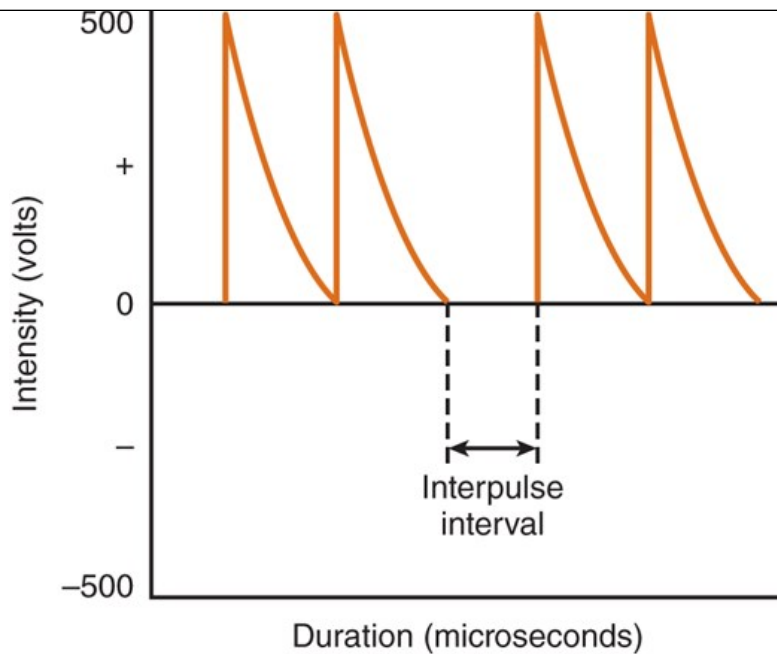
Amplitude = Voltage = Current intensity Amplitude=Voltage=Current intensity

By observing the different waveforms, it is apparent that the sine wave has a gradual increase and decrease in amplitude for biphasic, monophasic, and polyphasic waveforms (see Figure 5-6a-c). The rectangular wave has an almost instantaneous increase in amplitude, which plateaus for a period of time and then abruptly falls off (see Figure 5-6d-f). The spiked wave has a rapid increase and decrease in amplitude (see Figure 5-6g-i). The shape of these waveforms as they reach their maximum amplitude or intensity is directly related to the excitability of nervous tissue. The more rapid the increase in amplitude or the rate of rise, the greater the current's ability to excite nervous tissue is.

Many high-volt direct currents make use of a twin peak spiked pulse of very short duration (170 microseconds) and peak amplitudes as high as 500 V (Figure 5-10). Combining a high peak intensity with a short phase duration produces a very comfortable type of current as well as an effective means of stimulating sensory, motor, and pain fibers.²¹

Figure 5-10.

Most DC generators produce a twin peak spiked pulse of short duration and high amplitude.



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Pulse Duration

The **duration** of each pulse indicates the length of time current is flowing in one cycle. With monophasic waveforms, the phase duration is the same as the pulse duration and is the time from initiation of the phase to its end. With biphasic waveforms, the pulse duration is determined by the combined phase durations. In some electrotherapeutic devices, the duration is preset by the manufacturer. Other devices have the capability of changing duration. The phase duration may be as short as a few microseconds or may be a long-duration DC that flows for several minutes.

With PC, and in some instances with DC and AC currents, the current flow is off for a period of time. The combined time of the pulse duration and the interpulse interval is referred to as the **pulse period** (see Figure 5-7).

Pulse Frequency

Pulse frequency indicates the number of pulses or cycles per second. Each individual pulse represents a rise and fall in amplitude. As the frequency of any waveform is increased, the amplitude tends to increase and decrease more rapidly. The muscular and nervous system responses depend on the length of time between pulses and on how the pulses or waveforms are modulated.²² Muscle responds with individual twitch contractions to pulse rates of less than 50 pps. At 50 pps or greater, a tetanic contraction will result, regardless of whether the current is alternating, direct, or pulsatile.

Currents have been clinically labeled as low, medium, or high frequency, and a great deal of misunderstanding exists over how these frequency ranges are classified.¹⁰ Generally, all stimulating currents are low frequency and deliver between one and several thousand pulses per second. A number of so-called medium-frequency currents have been developed that have frequencies of 1000 pps to as high as 10,000 pps. However, these so-called medium-frequency pulses are in reality groups of pulses combined as bursts that range in frequency from 1 to 200 pps. These modulated bursts are capable of producing a physiologically effective frequency of stimulation only in this 1–200 pps range owing to the limitations of the absolute refractory period of nerve cell membranes. High frequency currents would be greater than 10,000 pps. Therefore, many of the claims of equipment manufacturers relative to medium-frequency currents are inaccurate.¹⁰

Current Modulation

The physiologic responses to the various waveforms depend to a large extent on current modulation. **Modulation** refers to any alteration in the amplitude, duration, or frequency of the current during a series of pulses or cycles.

The types of current modulation are as follows:

- continuous
- burst
- beat
- ramping

Continuous Modulation

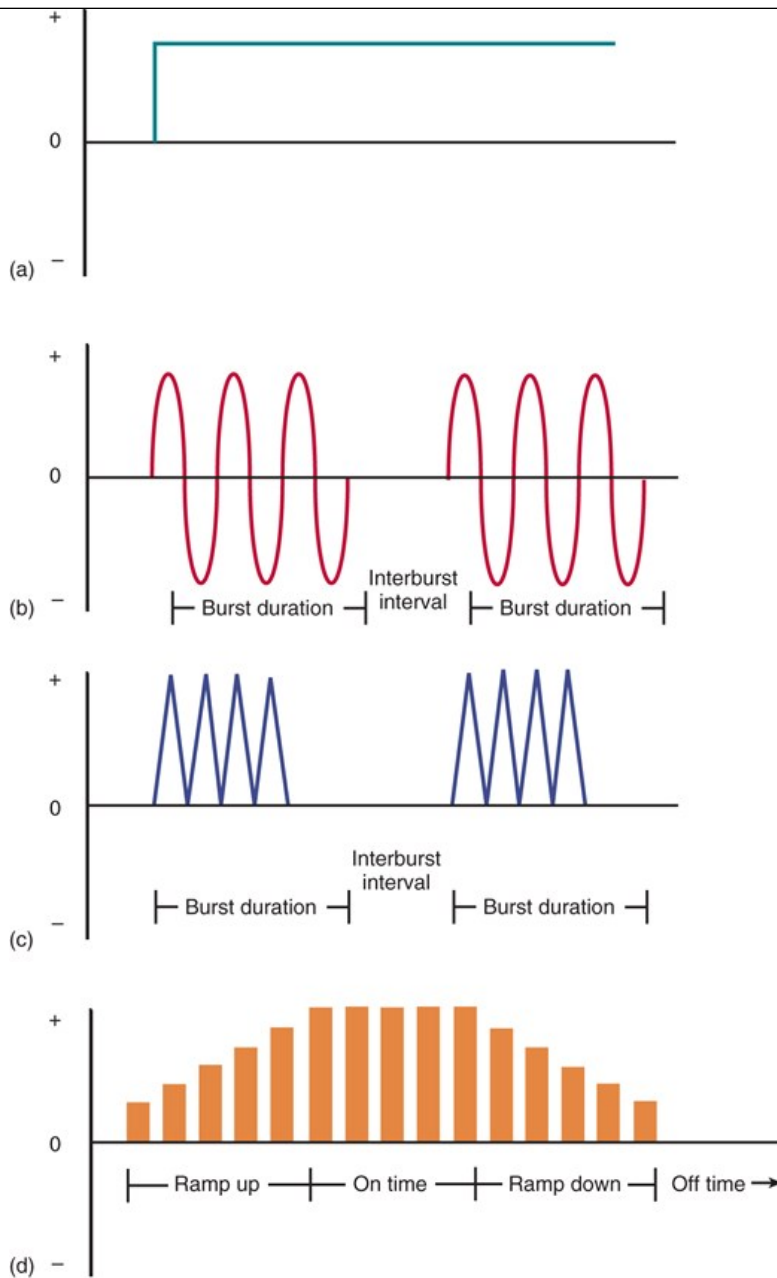
With continuous modulation the amplitude of current flow remains the same for several seconds or perhaps minutes as determined by the on/off time. Continuous modulation is usually associated with long-pulse-duration direct current (Figure 5–11a). With direct current, flow is monophasic and always in a uniform direction. In the discussion of physiologic responses to electrical currents, it was indicated that positive and negative ions are attracted toward poles or, in this case, electrodes of opposite polarity. This accumulation of charged ions over a period of time creates either an acidic or alkaline environment that may be of therapeutic value. This therapeutic technique has been referred to as **medical galvanism**. The technique of **iontophoresis** also uses continuous monophasic current to transport ions into the tissues (see Chapter 6). If the amplitude is great enough to produce a muscle contraction, the contraction will occur only when the current flow is turned on or off. Thus, with direct continuous current, a muscle contraction will occur both when the current is turned on and when it is turned off.

Clinical Decision-Making Exercise 5–3

The clinician is interested in producing a tetanic muscle contraction. What treatment parameter can be adjusted to produce this type of contraction?

Figure 5–11.

Current modulation may be (a) continuous current, (b) burst-modulated alternating current, (c) burst-modulated pulsatile current, and (d) ramp-up and/or ramp-down modulation.



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Burst Modulation

Burst modulation occurs when pulsatile or alternating current flows for a short duration (milliseconds) and then is turned off for a short time (milliseconds) in a repetitive cycle (Figure 5-11b and c). With pulsatile current, sets of pulses are combined. These combined pulses are most commonly referred to in the literature as **bursts**, but they have also been called *packets*, *envelopes*, or *pulse trains*.²³ The interruptions between individual bursts are called **interburst intervals**. The interburst interval is much too short to have any effect on a muscle contraction. Thus, the physiologic effects of a burst of pulses will be the same as with a single pulse.¹⁰ Some machines allow the clinician to change the burst duration and/or the interburst interval.

Beat Modulation

A beat modulation will be produced when two interfering alternating currents with differing frequencies are delivered to two separate pairs of

electrodes through separate channels within the same generator (see Figure 5–33). The two pairs of electrodes are set up in a crisscrossed or cloverleaf-like pattern so that the circuits interfere with one another. This interference pattern produces a beat frequency equal to the difference in frequency between the two alternating current frequencies. As an example, one circuit may have a fixed frequency of 4000 Hz, while the other is set at a frequency of 4100 Hz, thus creating a beat frequency of 100 beats/s. This type of beat-modulated AC is referred to as *IFC* and/or *premodulated interferential* and will be discussed later in this chapter.

Ramping Modulation

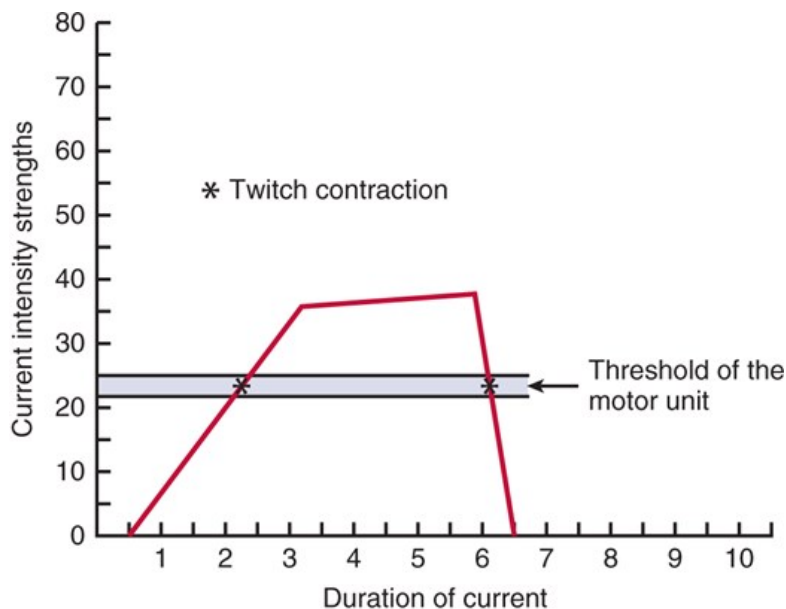
In **ramping** modulation, also called surging modulation, current amplitude will increase or ramp up gradually to some preset maximum and may also decrease or ramp down in intensity (Figure 5–11d). Ramp-up time is usually preset at about one third of the on time. The ramp-down option is not available on all machines. Most modern stimulators allow the clinician to set the on and off times between 1 and 10 seconds. Ramping modulation is used clinically to elicit muscle contraction and is generally considered to be a very comfortable type of current since it allows for a gradual increase in the intensity of a muscle contraction.

Frequency

To understand electrically stimulated muscle contractions, we must think in terms of multiple stimuli rather than a simple DC response. The motor nerves are not stimulated by a steady flow of DC. The nerve repolarizes under the influence of the current and will not depolarize again until a sudden change in current intensity occurs. If continuous direct current were the only current mode available, we would get a muscle contraction only when the current intensity rose to a stimulus threshold. Once the membrane is repolarized, another change in the current intensity would be needed to force another depolarization and contraction (Figure 5–12).

Figure 5–12.

Direct current influence on a motor unit.



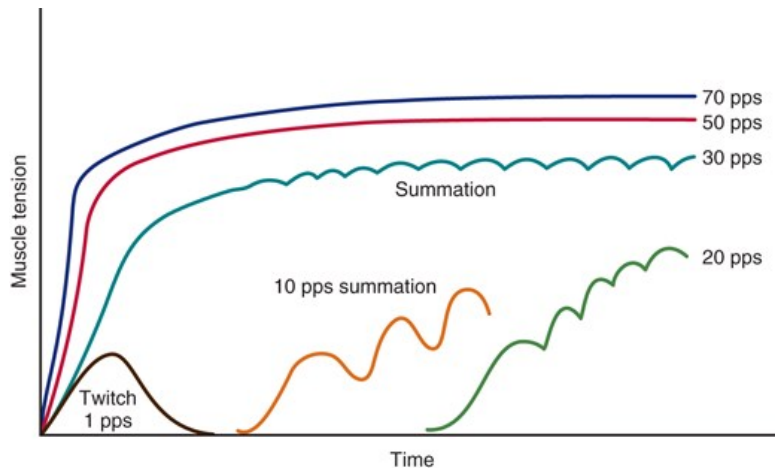
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Frequency indicates the numbers of impulses or cycles produced by an electrical stimulating device in 1 second and is referred to as cycles per second (cps), pulses per second (pps), or hertz (Hz). It can determine the type of muscle contraction elicited. The amount of shortening of the muscle fiber and the amount of recovery allowed to the muscle fiber are a function of the frequency. The mechanical shortening of the single muscle fiber response can be influenced by stimulating again as soon as the tissue membrane repolarizes. Only the membrane has the absolute refractory period; the contractile mechanism operates on a different timing sequence and is just beginning to contract. When the muscle membrane receives a second stimulus, the myofilaments are already overlapping, and the second stimulus causes an increased mechanical shortening of the muscle fiber. This process of superimposing one twitch contraction on another is called *summation of contractions*. As the number of twitch contractions per second increases,

single twitch responses cannot be distinguished, and **tetanization** of the muscle fiber is reached (Figure 5-13). The tension developed by a muscle fiber in tetany is much greater than the tension from a twitch contraction.¹⁸⁵ This muscle fiber tetany is strictly a function of the frequency of the stimulating current; it is not dependent on the intensity of the current.^{24,25} In general, a higher frequency can be used to produce an increase in muscle tension due to the summative effects, while a lower frequency is more often used for muscle pumping and edema reduction.

Figure 5-13.

Summation of contractions and tetanization.



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Intensity

Increasing the intensity of the electrical stimulus causes the current to reach deeper into the tissue. Depolarization of additional nerve fibers is accomplished by two methods: higher threshold fibers within the range of the stimulus are depolarized by the higher-intensity stimulus and fibers with the same threshold but deeper in the structure are depolarized by the deeper spread of the current. High-volt currents are capable of deeper penetration into the tissue than low-volt currents and may be desirable when stimulating deep muscle tissue. This is one of the most significant differences between high- and low-volt currents.^{8,25}

Duration

We also can stimulate more nerve fibers with the same intensity current by increasing the length of time (duration) that an adequate stimulus is available to depolarize the membranes. Greater numbers of nerve fibers then would react to the same intensity stimulus, because the current would be available for a longer period of time.^{2,24,26} This method requires the use of a stimulator with an adjustable duration.

Polarity

With any electrical current, the electrode that has a greater number of electrons is called the *negative electrode* or the **cathode**. The other electrode has a relatively lower number of electrons and is called the *positive electrode* or the **anode**. The negative electrode attracts positive ions, and the positive electrode attracts negative ions and electrons. With biphasic waves, these electrodes change polarity with each current cycle.

- negative electrode: cathode;
- positive electrode: anode;
- muscle contraction: negative active electrode;
- cathode: distal;

- anode: proximal.

With a direct current, the clinician can designate one electrode as the negative and one as the positive, and for the duration of the treatment the electrodes will provide that polar effect. The polar effect can be thought of in terms of three characteristics: (1) chemical effects, (2) ease of excitation, and (3) direction of current flow.^{2,24,25,27–29}

Chemical changes occur only with long-duration continuous current.

Clinical Decision-Making Exercise 5–4

A clinician is using an electrical stimulator to induce a muscle contraction of the rectus femoris. The active electrode is placed over the motor point of the muscle and the dispersive electrode is placed under the leg. What changes in the setup of the electrodes and/or changes in current parameters can be made to reach the threshold of depolarization for this muscle?

Chemical Effects

Changes in pH under each electrode, a reflex vasodilation, and the ability to facilitate movement of oppositely charged ions through the skin into the tissue (iontophoresis) are all thought of as chemical effects. A tissue-stimulating effect is ascribed to the negative electrode. To create these effects, longer pulse durations (>1 minute) are required.^{27,29–31} The bacteriostatic effect is achieved at either the anode or cathode with intensities in the 5–10 mA range, although at 1 mA or below the greatest bacteriostatic effect was found at the cathode.³² Another study using treatment times exceeding 30 minutes found some bacteriostatic effect of high-voltage pulsed currents.³³

Ease of Excitation of Excitable Tissue

The polarity of the active electrode usually should be negative when the desired result is a muscle contraction because of the greater facility for membrane depolarization at the negative pole. However, current density under the positive pole can be increased rapidly enough to create a depolarizing effect. Using the positive electrode as the active electrode is not as efficient, because it will require more current intensity to create an action potential. This may cause the patient to be less comfortable with the treatment. In treatment programs requiring muscle contraction or sensory nerve stimulation, patient comfort should dictate the choice of positive or negative polarity. Negative polarity usually is the most comfortable in this instance.^{2,25,34}

Direction of Current Flow

In some treatment schemes, the direction of current flow also is considered important. Generally speaking, the negative electrode is positioned distally and the positive electrode proximally. This arrangement tries to replicate the naturally occurring pattern of electrical flow in the body.^{27,35}

The direction of current flow could also influence shifting of the water content of the tissues and movement of colloids (fluid suspension of the intracellular fluid). Neither of these phenomena is well documented or understood, and further study is needed before clinical treatments are designed around these concepts.^{2,36,37}

True polar effects can be substantiated when they occur close to the electrodes through which the current is entering the tissue. In laboratory situations in physics, polar effects occur in very close proximity to the electrode. To cause these effects, the current must flow through a medium. If the tissue to be treated is centrally located between the two electrodes, results cannot be assigned to polar effects.²⁷ Clinically, polar effects are an important consideration in iontophoresis, stimulating motor points or peripheral nerves, and the biostimulative effects on nonexcitatory cells.

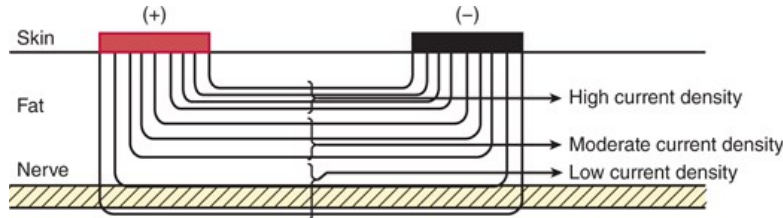
Current Density

The **current density** (amount of current flow per cubic volume) at the nerve or muscle must be high enough to cause depolarization. The current density is highest where the electrodes meet the skin and diminishes as the electricity penetrates into the deeper tissues (Figure 5–14).^{2,24} If there is a

large fat layer between the electrodes and the nerve, the electrical energy may not have a high enough density to cause depolarization (Figure 5–15).

Figure 5–14.

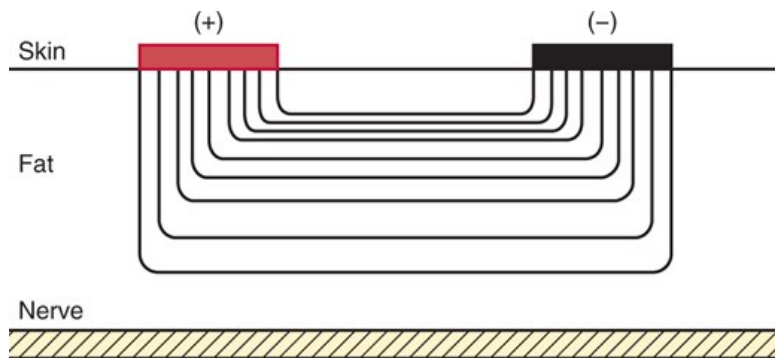
Current density using equal size electrodes spaced close together.



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Figure 5–15.

Equal size electrodes spaced close together on body part with thick fat layers. Thus, the electrical current does not reach the nerve.

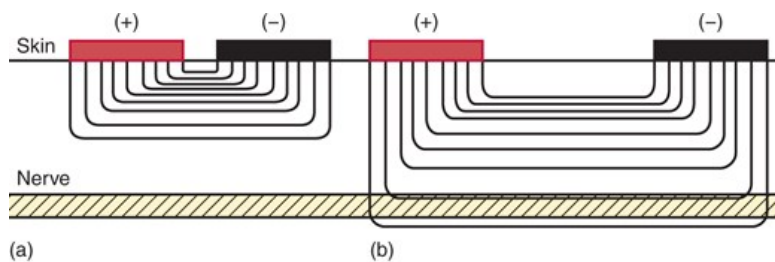


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If the electrodes are spaced closely together, the area of highest current density is relatively superficial (Figure 5–16a). If the electrodes are spaced farther apart, the current density will be higher in the deeper tissues, including nerve and muscle (Figure 5–16b).

Figure 5–16.

(a) Electrodes are very close together, producing a high-density current in the superficial tissues. (b) Increasing the distance between the electrodes increases the current density in deeper tissues.

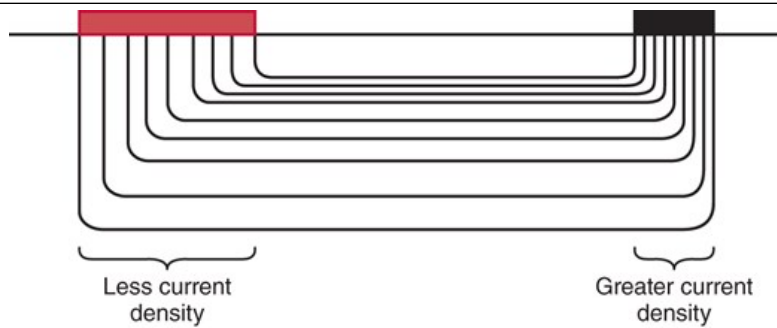


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Electrode size will also change current density. As the size of one electrode relative to another is decreased, the current density beneath the smaller electrode is increased. The larger the electrode, the larger the area over which the current is spread, decreasing the current density (Figure 5–17).^{2,8,24,25,38}

Figure 5–17.

The greatest current density is under the small or active electrode.



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Using a large (dispersive) electrode remote from the treatment area while placing a smaller (active) electrode as close as possible to the nerve or muscle motor point will give the greatest effect at the small electrode. The large electrode disperses the current over a large area; the small electrode concentrates the current in the area of the motor point (Figure 5-17).

Electrode size and placement are key elements the clinician controls that will have great influence on results. High current density close to the neural structure to be stimulated makes success more certain with the least amount of current. Electrode placement is likely one of the biggest causes of poor results from electrical therapy.

Electrode Placement

Several guidelines will help the clinician select the appropriate sites for electrode placement when using any of the treatment protocols aimed at the electrical stimulation of sensory or motor nerves. Electrodes should be placed where the clinician feels will be the most effective location and then moved in a trial-and-error pattern until a specific treatment goal is achieved. The following patterns may be used:

1. Electrodes may be placed on or around a painful area.
2. Electrodes may be placed over specific dermatomes, myotomes, or sclerotomes that correspond to the painful area.
3. Electrodes may be placed close to the spinal cord segment that innervates a painful area.
4. Peripheral nerves that innervate the painful area may be stimulated by placing electrodes over sites where the nerve becomes superficial and can be stimulated easily.
5. Vascular structures contain neural tissue as well as ionic fluids that would transmit electrical stimulating currents and may be most easily stimulated by electrode placement over superficial vascular structures.
6. Electrodes may be placed over trigger point or acupuncture point locations.³⁹
7. Electrodes should be placed over motor points of the muscle or at least over the muscle belly of the muscle in which you are trying to elicit a contraction.
8. Both acupuncture and trigger points have been conveniently mapped out and illustrated. A reference on acupuncture and trigger areas is included in Appendix A. The clinician should systematically attempt to stimulate the points listed as successful for certain areas and types of pain. If they are effective, the patient will have decreased pain. These points also can be identified using an ohm meter point locator to determine areas of decreased skin resistance.
9. Combinations of any of the preceding systems and bilateral electrode placement also can be successful.^{28,40,41}
10. A **bipolar** application of electrodes uses electrodes of the same size in the same general treatment area (Figure 5-18a). Since the size of the electrodes is the same, the current density under each electrode is essentially the same. Thus, the physiologic effects under each electrode should be the same. However, if one electrode is located over a motor point and the other is not, a muscle contraction may occur at lower current amplitude over the motor point.

11. A **monopolar** application of electrodes uses one or more small active electrodes over a treatment area and a large dispersive electrode placed somewhere else on the body (Figure 5–18b). The higher current density is under the smaller or active electrode, and thus a desired physiologic response will likely occur at the active electrode.
12. A **quadripolar** technique uses two sets of bipolar electrodes, each of which comes from a completely separate channel on the electrical stimulator (Figure 5–18c).
13. Crossing patterns are used with interferential and premodulated IFC. They involve electrode application such that the electrical signals from each set of electrodes add together at some point in the body and the intensity accumulates. The electrodes are usually arranged in a crisscross pattern around the point to be stimulated (Figure 5–19). If you wish to stimulate a specific superficial area, the electrodes should be relatively close together. They should be located so that the area to be treated is central to the location of the electrodes. If pain is poorly localized pain (e.g., general shoulder pain) and seems to be deeper in the joint or muscle area, spread the electrodes farther apart to give more penetration to the current.

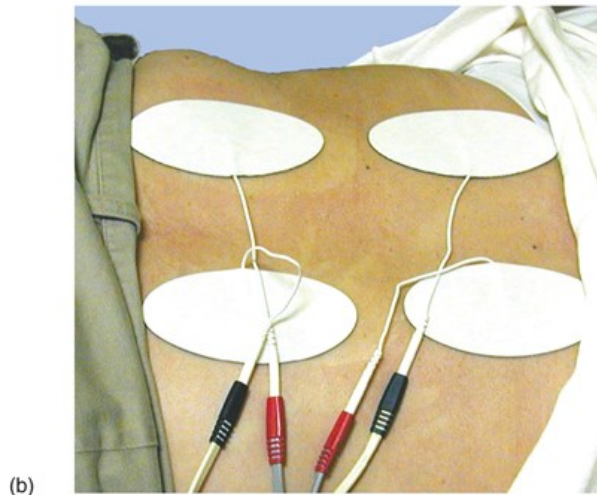
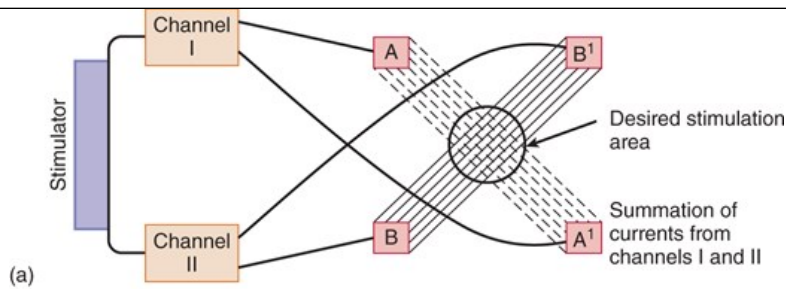
Figure 5–18.

Electrode setup: (a) bipolar, (b) monopolar, and (c) quadripolar.



Figure 5–19.

(a) Current flow is from A to A¹, and B to B¹. As the currents cross the area of stimulation, they summate in intensity. (b) Typical crossing pattern for electrodes.



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The clinician should not be limited to any one system but should evaluate electrode placement for each patient. The effectiveness of sensory or motor stimulation is closely tied in with proper electrode placement. As in all trial-and-error treatment approaches, a systematic, organized search is always better than a “shotgun,” hit-or-miss approach. Numerous articles have identified some of the best locations for common clinical problems, and these may be used as a starting point for the first approach.⁴² If the treatment is not achieving the desired results, the electrode placement should be reconsidered.

Clinical Decision-Making Exercise 5–5

How can the clinician make adjustments in the electrode placement to increase the current density in the deeper tissues?

On/Off Time

Most electrical generators allow the clinician the capability of setting the ratio of time the electrical current will be on and the time it will be off. The lower the ratio of on time to off time, the less total current the patient will receive. On some generators this on/off time is referred to as the *duty cycle*.

PHYSIOLOGIC RESPONSES TO ELECTRICAL CURRENT

Electricity has an effect on each cell and tissue that it passes through.^{43,44} The type and extent of the response are dependent on the type of tissue and its response characteristics (e.g., how it normally functions or changes under normal stress) and the nature of the current applied (current type, intensity, duration, voltage, and density). The tissue should respond to electrical energy in a manner similar to that in which it normally functions.³⁸

The effects of electrical current passing through the various tissues of the body may be thermal, chemical, or physiologic.⁴⁵ All electrical currents cause a rise in temperature in a conducting tissue.⁴⁶ The tissues of the body possess varying degrees of resistance, and those of higher resistance should heat up more when electrical current passes through. As indicated previously, the electrical currents used for stimulation of nerve and muscle have a

relatively low average current flow that produces minimal thermal effects.

Clinically, clinicians use electrical currents to produce either muscle contractions or modification of pain impulses through effects on the motor and sensory nerves. This function is dependent to a great extent on selecting the appropriate treatment parameters based on the principles identified in this chapter.⁴⁶

Electrical currents are also used to produce chemical effects. Most biologic tissue contains negatively and positively charged ions. A continuous direct current will cause migration of these charged particles toward the pole of opposite polarity, producing specific physiologic changes.

Direct and Indirect Physiologic Effects

These physiologic responses to electrical stimulating currents can be broken into direct and indirect effects. There is always a direct effect along the lines of current flow and under the electrodes. Indirect effects occur remote to the area of current flow and are usually the result of stimulating a natural physiologic event to occur.^{8,47}

If a certain effect is desired from stimulation, goals must be established to achieve the specific physiologic response as a goal of treatment. These responses can be grouped into two basic physiologic responses: excitatory and nonexcitatory.

The excitatory is the most obvious and the one that has been used the most often in the past in treating patients. In the clinical setting, we spend most of our time trying to get the excitatory response from the nerve cells. Patients perceive excitatory responses as electrical sensation, muscle contraction, and electrical pain. Physiologically, the nerves that affect these perceptions fire in that order as the stimulus intensity is increased gradually. Nerves have very little discriminatory ability. They can tell only if there is electricity in sufficient magnitude to cause a depolarization of the nerve membrane. They have very little regard for the different shapes and polarities of waveforms. To the nerve cell, electricity is electricity. As in all things dealing with higher-level organisms, the range of responses to the same stimulus is wide, depending on the environmental and systemic factors.

All perception is a product of the brain's activity of receiving the signal that a nerve has been stimulated electrically. This further enlarges the broad range of systemic effects that occur in response to the electrical stimulation.

Stimulation events will change the body's perception. As the strength of the current increases and/or the duration of the current increases, more nerve cells will fire. As the strength of the stimulus increases and these events occur, certain quality judgments about the electrical stimuli are made. Is the current pleasant or unpleasant? Is the intensity of the stimulus weak or strong? The broad range of individual responses to these quality judgments has a significant impact on the beneficial effects of this therapy.

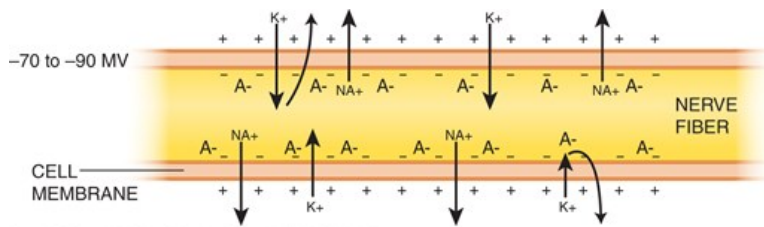
Nerve Responses to Electrical Currents

Nerves and muscles are both excitable tissues. This excitability is dependent on the cell membrane's **voltage-sensitive permeability**. The nerve or muscle cell membrane regulates the exchange of electrically charged ions between the inside of the cell and the environment outside the cell. This voltage-sensitive permeability produces an unequal distribution of charged ions on each side of the membrane, which in turn creates a potential difference between the charge of the interior of the cell and that of the exterior of the cell. The membrane then is considered to be polarized. The potential difference between the inside and outside is known as the **resting potential**, because the cell tries to maintain this electrochemical gradient as its normal homeostatic environment.⁴³

Both electrical and chemical gradients are established along the cell membrane, with a greater concentration of diffusible positive ions on the outside of the membrane than on the inside. Using the continuous activity of the sodium pumps in the nerve cell membrane, the nerve cell continually moves Na^+ from inside the cell to outside the cell membrane while voltage-activated potassium channels allow K^+ to move into the cell. This maintains the larger concentration of K^+ on the inside of the cell membrane. The overall charge difference between the inside and the outside of the membrane creates an electrical gradient at its resting level of -70 to -90 mV (Figure 5–20). As Guyton explains, “The potential is proportional to the difference in tendency of the ions to diffuse in one direction versus the other direction.”³² Two conditions are necessary for the membrane potential to develop: (1) the membrane must be semipermeable, allowing ions of one charge to diffuse through the pores more readily than ions of the opposite charge; and (2) the concentration of the diffusible ions must be greater on one side of the membrane than on the other side.^{32,43}

Figure 5–20.

Nerve cell membrane with active transport mechanisms maintaining the resting membrane potential.



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The resting membrane potential is generated because the cell is an ionic battery whose concentration of ions inside and outside the cell is maintained by regulatory Na^+K^+ pumps within the cell wall. In addition to the ability of the nerve and muscle cell membranes to develop and maintain the resting potential, the membranes are excitable.^{32,48}

To create transmission of an impulse in the nerve tissue, resting membrane potential must be reduced below a threshold level. Changes in the membrane's permeability then may occur. These changes create an **action potential** that will propagate the impulse along the nerve in both directions from the location of the stimulus. An action potential created by a stimulus from chemical, electrical, thermal, or mechanical means always creates the same result, membrane **depolarization**.

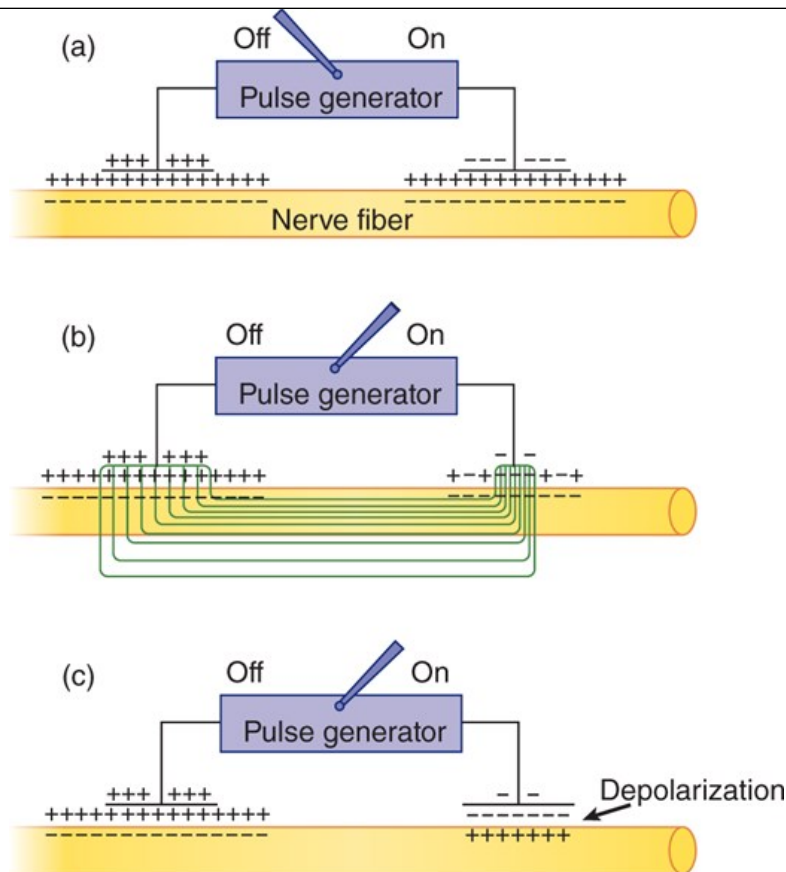
Not all stimuli are effective in causing an action potential and depolarization. To be an effective agent, the stimulus must have an adequate intensity and last long enough to equal or exceed the membrane's basic threshold for excitation. The stimulus must alter the membrane so that a number of ions are pushed across the membrane, exceeding the ability of the active transport pumps to maintain the resting potentials. A stimulus of this magnitude forces the membrane to depolarize and results in an action potential.^{2,32}

Depolarization

As the charged ions move across the nerve fiber membranes beneath the anode and cathode, membrane depolarization occurs. The cathode usually is the site of depolarization (Figure 5–21a). As the concentration of negatively charged ions increases, the membrane's voltage potential becomes low and is brought toward its threshold for depolarization (Figure 5–21b). The anode makes the nerve cell membrane potential more positive, increasing the threshold necessary for depolarization (Figure 5–21c). The cathode in this example becomes the active electrode; the anode becomes the indifferent electrode (dispersive). The anode and cathode may switch active and indifferent roles under other circumstances.^{2,8,24} The number of ions needed to exceed the membrane pump's ability to maintain the normal membrane resting potential is tissue dependent.

Figure 5–21.

(a–c) Depolarization of nerve cell membrane.



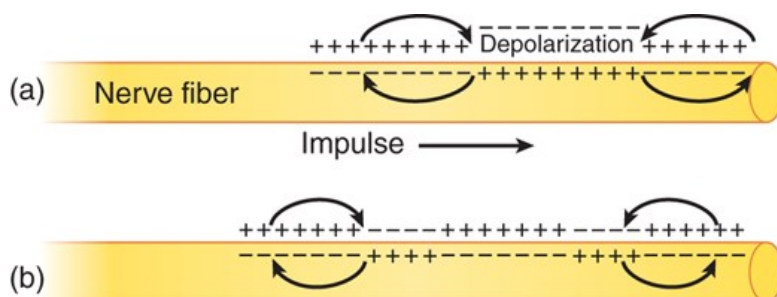
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Depolarization propagation. Following excitement and propagation of the impulse along the nerve fiber, there is a brief period during which the nerve fiber is incapable of reacting to a second stimulus. This is the **absolute refractory period**, which lasts about 0.5 microsecond. Excitability is restored gradually as the nerve cell membrane repolarizes itself. The nerve then is capable of being stimulated again. The maximum number of possible discharges of a nerve may reach 1000/s, depending on fiber type.^{2,24,32,49}

The difference in electrical potential between the depolarized region and the neighboring inactive region causes a small electrical current to flow between the two regions. This forms a complete local circuit and makes the depolarization self-propagating as the process is repeated all along the fiber in each direction from the depolarization site. Energy released by the cell keeps the intensity of the impulse uniform as it travels down the cell.^{2,24,32,49} This process is illustrated in Figure 5-22.

Figure 5-22.

(a and b) Propagation of a nerve impulse.



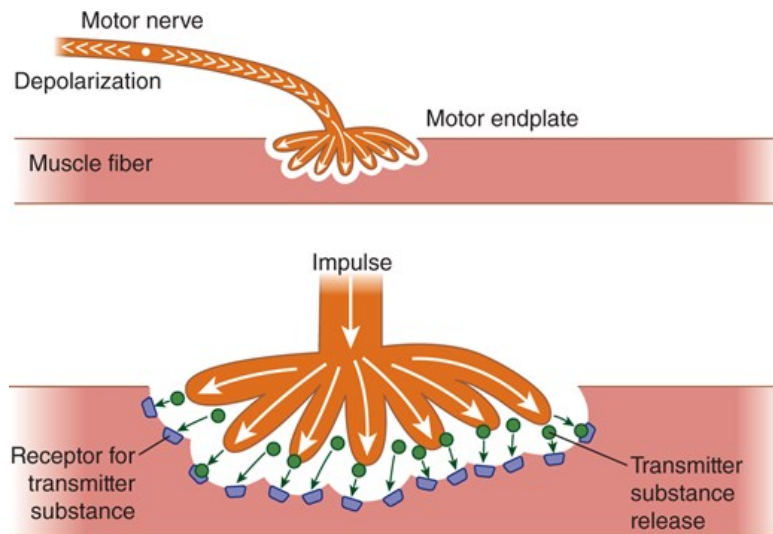
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Depolarization effects. As the nerve impulse reaches its effector organ, either another nerve cell or a muscle, the impulse is transferred between the

two at a motor endplate or synapse. At this junction, a neurotransmitter substance is released from the nerve. If the effector organ is a muscle, this neurotransmitter substance causes the adjacent excitable muscle to contract, resulting in a single twitch muscle contraction (Figure 5-23).^{2,24} This contraction, initiated by an electrical stimulus, is the same as a twitch contraction coming from voluntary activity.

Figure 5-23.

Change of electrical impulse to transmitter substance at the motor endplate. When activated, the muscle cell membrane will depolarize and contraction will occur.



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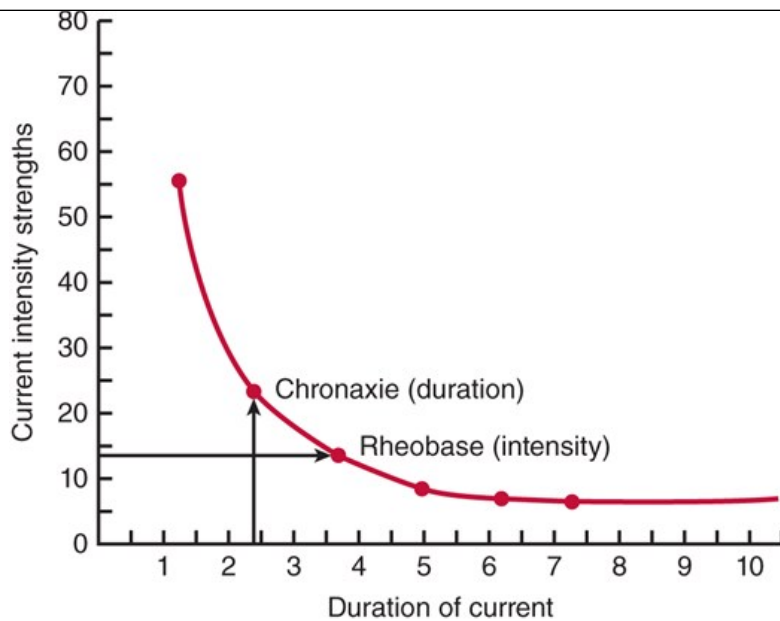
Strength–Duration Curve

The *strength–duration (SD) curve* is a graphic representation of the threshold for depolarization of a particular nerve fiber (Figure 5-24). A sufficient amount of electrical current must be delivered to make a nerve depolarize. As illustrated, there is a nonlinear relationship between current duration and current intensity, in which shorter-duration stimuli require increasing intensities to reach the threshold for depolarization of the nerve.

Rheobase is a term that identifies the specific *intensity* of current necessary to cause an observable tissue response (i.e., a muscle contraction) given a long current duration. **Chronaxie** identifies the specific length of time or *duration* required for a current of twice the intensity of the rheobase to produce tissue excitation.

Figure 5-24.

Strength–duration curve.

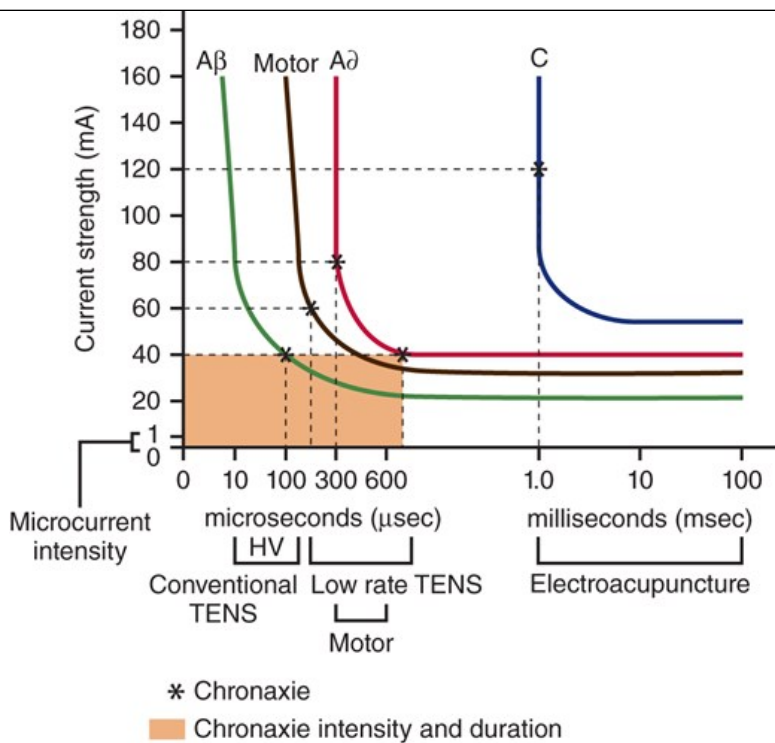


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Different sizes and types of nerve fibers have different thresholds for depolarization and thus different SD curves (Figure 5-25). A β fibers require the least amount of electrical current to reach their threshold for depolarization followed by motor nerve fibers, A δ fibers, and finally C fibers. The curves are basically symmetric, but the intensity of current necessary to reach the membrane's threshold for excitation differs for each type of nerve fiber.^{2,32,41,50} By gradually increasing the current intensity and/or current duration, the first physical response would be a tingling sensation caused by depolarization of A β fibers, followed by a muscle contraction when motor nerve fibers depolarize, and finally a feeling of pain from depolarization of A δ fibers and then C fibers.

Figure 5-25.

Strength-duration curves for A β sensory, motor, A δ sensory, and C (pain) nerve fibers. Durations of several electrical stimulators are indicated along the lower axis. Corresponding intensities would be necessary to create a depolarizing stimulus for any of the nerve fibers. Microcurrent intensity is so low that the nerve fibers will not depolarize. This current travels through other body tissues to create effects.



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Equipment manufacturers use the SD curves in choosing their preset pulse durations to be effective in depolarizing nerve fibers.

Muscular Responses to Electrical Current

To reemphasize, normally a muscle contracts in response to depolarization of its motor nerve. Stimulation of the motor nerve is the method used in most clinical applications of electrically stimulated muscle contractions. However, in the absence of muscle innervation, it is possible for a muscle to contract by using an electrical current that causes the muscle membrane, rather than the motor nerve, to depolarize. This will create the same muscle contraction as a natural stimulus.

The **all-or-none response** is another important concept that is relevant when applying electrical current to nerve or muscle tissue. Once a stimulus reaches a depolarizing threshold, the nerve or muscle membrane depolarizes, and propagation of the impulse or muscle contraction occurs. This reaction remains the same regardless of increases in the strength of the stimulus used. Either the stimulus causes depolarization (the all) or it does not cause depolarization (the none). There is no gradation of response; the response of the single nerve or muscle fiber is maximal or nonexistent.^{2,24,25} This all-or-none phenomenon does not mean that muscle fiber shortening and overall muscle activity cannot be influenced by changing the intensity, pulses per second, or duration of the stimulating current. Adjustments in current parameters can cause changes in the shortening of the muscle fiber and the overall muscle activity.

Stimulation of Denervated Muscle

Electrical currents may be used to produce a muscle contraction in **denervated muscle**. A muscle that is denervated is one that has lost its peripheral nerve supply. The primary purpose for electrically stimulating denervated muscle is to help minimize the extent of atrophy during the period while the nerve is regenerating. Following denervation, the muscle fibers experience a number of progressive anatomic, biochemical, and physiologic changes that lead to a decrease in the size of the individual muscle fibers and in the diameter and weight of the muscle. Consequently, the amount of tension that muscle can generate will decrease and the time required for the muscle to contract will increase.^{51,52} These degenerative changes progress until the muscle is reinnervated by axons regenerating across the site of the lesion. If reinnervation does not occur within 2 years, it is generally accepted that fibrous connective tissue will have replaced the contractile elements of the muscle and recovery of muscle function is not possible.^{52,174,177}

A review of the literature indicates that the majority of studies support the use of electrical stimulation of denervated muscle. These studies generally indicate that muscle atrophy can be retarded, loss of both muscle mass and contractile strength can be minimized, and muscle fiber size can be

maintained by the appropriate use of electrical stimulation.^{53–55} Electrically stimulated contractions of denervated muscle may limit edema and venous stasis, thus delaying muscle fiber fibrosis and degeneration.⁵² However, there also seems to be general agreement that electrical stimulation has little or no effect on the rate of nerve regeneration or muscle reinnervation.

A few studies have suggested that electrical stimulation of denervated muscle actually may interfere with reinnervation, thus delaying functional return.^{56,57} These studies propose that the muscle contraction disrupts the regenerating neuromuscular junction retarding reinnervation, and that electrical stimulation may traumatize denervated muscle since it is more sensitive to trauma than normal muscle.^{52,56,58,178}

Treatment Parameters for Denervated Muscle are as Follows:

1. A current with an asymmetric, biphasic waveform with a pulse duration less than 1 millisecond may be used during the first 2 weeks.⁵⁹
2. After 2 weeks, an interrupted square wave DC and a progressive exponential wave DC, each with a long pulse duration of greater than 10 milliseconds, or a sine wave AC with a frequency lower than 10 Hz will produce a twitch contraction.⁵² The length of the pulse should be as short as possible but long enough to elicit a contraction.⁶⁰
3. The current waveform should have a pulse duration equal to or greater than the chronaxie of the denervated muscle.
4. The amplitude of the current along with the pulse duration must be sufficient to stimulate a denervated muscle with a prolonged chronaxie while producing a moderately strong contraction of the muscle fibers.
5. The pause between stimuli should be 1:4 or 5 (15–40 mA) longer (about 3–6 seconds) than the stimulus duration to minimize fatigue.⁶⁰
6. Either a monopolar or bipolar electrode setup can be used with the small-diameter active electrode placed over the most electrically active point in the muscle. This may not be the motor point since the muscle is not normally innervated.
7. Stimulation should begin immediately following denervation using three stimulation treatments per day involving three sets of between 5 and 20 repetitions that can be varied according to fatigability of the muscle.⁵²
8. The contraction needs to create muscle tension, so joints may need to be fixed or isotonic contraction for end-range positions may be needed.

Biostimulative Effects of Electrical Current on Nonexcitatory Cells

Electrical stimulating currents can have an effect on the function of nonexcitatory cells, which will respond to electrical current in ways consistent with their cell type and tissue function. We have discussed how electrical currents cause depolarization of excitable cells that compose nerve tissue and muscle tissue. Electrical stimulation of the appropriate frequency and amplitude may be able to activate the receptor site on nonexcitable cells and stimulate the same cellular changes as the naturally occurring chemical molecular stimulation. The cell functions by incorporating a multitude of chemical reactions into a living process. It is conceivable that the appropriate electrical signal could create more specific sites for enzymatic activity, thereby changing or stimulating cell function.¹⁵

Cells seem responsive to steady DC gradients. The cells move or grow toward one pole and away from the other. The electric field created by the direct current may help guide the healing process and the regenerative capabilities of injured or developing tissues.^{15,59}

Cells also may respond to a particular frequency of current. The cell may be selectively responsive to certain frequencies and unresponsive to other frequencies. Some researchers claim that specific genes for protein manufacture can be activated by a certain shaped electrical impulse. This frequency could change in certain ways according to the cellular state. This phenomenon has been termed the **“frequency window” selectivity** of the cell.⁴³

Overall we see that small-amplitude direct currents are intrinsic to the ways the body works to grow and repair. Clinically if we can duplicate some of these same signals, we may be successful in using electrotherapy in the most efficient manner.

CLINICAL USES OF ELECTRICAL STIMULATING CURRENTS

Essentially, electrical stimulating currents may be used to stimulate either motor nerves to induce muscle contraction, or sensory nerves for the purpose of modulating pain.

Therapeutic Uses for Electrical Stimulation of Motor Nerves

A variety of therapeutic gains can be made by electrically stimulating a motor nerve to produce a muscle contraction:

1. muscle reeducation;
2. muscle pump contractions;
3. retardation of atrophy;
4. muscle strengthening.

Muscle fatigue should be considered when deciding on treatment parameters. The variables that have an influence on muscle fatigue are the following:

1. intensity: combination of the pulse stimulus's amplitude intensity and the pulse duration;
 2. the number of pulses or bursts per second;
 3. on time;
 4. off time.
-

CASE STUDY 5-1**ELECTRICAL STIMULATING CURRENTS: STRENGTHENING OF INNERVATED MUSCLE**

Background: A 22-year-old woman sustained a grade II MCL sprain of the left knee 3 days ago in an auto accident, and is being treated with plaster immobilization for 3 weeks. She is not able to generate a maximal isometric quadriceps contraction voluntarily. The cast has been modified to accommodate electrodes over the femoral nerve and the motor point of the vastus medialis muscle. There are no restrictions on the amount of force she is allowed to produce during a knee extension effort.

Impression: Grade II MCL sprain of the left knee, with inability to generate maximal isometric force of the knee extensors.

Treatment Plan: A 5-day per week schedule of electrical stimulation was initiated. A polyphasic waveform was selected, with a 2500-Hz carrier wave, with an effective frequency of 50 Hz (10 milliseconds on, 10 milliseconds off). The stimulator was set to ramp the current up for 6 seconds, maintain the current at a specific amplitude for 10 seconds, and then drop to zero with no ramp; rest time was 50 seconds, giving an effective duty cycle of 1:5 (10 seconds on, 50 seconds off). Each treatment session began with 10 repetitions at a comfortable stimulus amplitude, followed by three sets of 10 repetitions each with the maximal amount of current tolerable. A 2-minute rest separated the sets. During the 10 seconds on time, the current amplitude was adjusted to the maximal amount the patient was able to tolerate. The patient was encouraged to contract the quadriceps femoris muscle group as the current was delivered.

Response: The patient's tolerance for the electrical stimulation gradually increased during the first week, and then reached a plateau; this plateau was maintained for the next 2 weeks. On removal of the cast, there was no measurable or visible atrophy of the left thigh. A rehabilitation program of active range of motion, strengthening exercise, and functional activities was initiated, and the patient returned to full, pain-free activity 3 weeks following cast removal.

Discussion Questions

- What tissues were injured or affected?
- What symptoms were present?
- In what phase of the injury-healing continuum did the patient present for care?
- What are the physical agent modality's biophysical effects (direct, indirect, depth, and tissue affinity)?
- What are the physical agent modality's indications and contraindications?
- What are the parameters of the physical agent modality's application, dosage, duration, and frequency in this case study?
- What other physical agent modalities could be used to treat this injury or condition? Why? How?

The rehabilitation professional employs physical agent modalities to create an optimum environment for tissue healing while minimizing the symptoms associated with the trauma or condition.

Muscle force is varied by changing the intensity to recruit more or less motor units. It can also be varied to a certain degree by increasing the summing quality of the contraction with high burst or pulse rates. The greater the force, the greater the demands on the muscle, the greater the occlusion of muscle blood flow, and the greater the fatigue. If high muscle forces are not required, the intensity and frequency can be adjusted to desired levels but fatigue can still be a factor. To minimize fatigue associated with forceful contractions, a combination of the lowest frequency and the higher intensity will keep the force constant.⁶¹

If high force levels are desired, then higher frequencies and intensities can be used. To keep the muscle fatigue as low as possible, the rest time between contractions should be at least 60 seconds for each 10 seconds of contraction time. A variable frequency train, in which a high-frequency stimulus and then a low-frequency stimulus is used, will also help minimize fatigue in repetitive functional electrical stimulation (FES).⁶¹

Neuromuscular-induced contraction at the higher torques is associated with patient perceptions of pain, from either the current used or the intensity

of the contraction. This is often a limiting factor in the success of any of the following protocols. Each patient needs supervision and satisfactory clinician confidence for the most effective compliance with the treatment goals.^{61,62}

When using electrical stimulation for muscle contraction, motor point stimulation can give the best individual muscle contraction. To find the motor point of a muscle, a probe electrode should be used to stimulate the muscle. Stimulation should be started in the approximate location of the desired motor point. (See Appendix A for motor point chart.) The intensity should be increased until contraction is visible, and the current intensity should be maintained at that level. The probe should be moved around until the best visible contraction for that current intensity is found; this is the motor point.^{24,63} By choosing this location for stimulation, the current density can be increased in an area where numerous motor nerve fibers can be affected, maximizing the muscular response from the stimulation.

Muscle reeducation. Muscular inhibition after surgery or injury is the primary indication for muscle reeducation.¹⁸¹ If the neuromuscular mechanisms of a muscle have not been damaged, then central nervous system inhibition of this muscle usually is a factor in loss of control. The atrophy of synaptic contacts that remain unused for long periods is theorized as a source of this sensorimotor alienation. The addition of electrical stimulation of the motor nerve provides an artificial use of the inactive synapses and helps restore a more normal balance to the system as the ascending sensory information will be reintegrated into the patient's movement control patterns. A muscle contraction usually can be forced by electrically stimulating the muscle. Forcing the muscle to contract causes an increase in the sensory input from that muscle. The patient feels the muscle contract, sees the muscle contract, and can attempt to duplicate this muscular response.^{24,50,64,65,182} The object here is to reestablish control and not to create a strengthening contraction.

Protocols for muscle reeducation do not list specific parameters to make this treatment more efficient, but the criteria listed in the treatment protocol for muscle reeducation are essential.

Treatment Parameters for Muscle Reeducation are as Follows:

1. Current intensity must be adequate for muscle contraction but comfortable for the patient.
2. Pulse per duration should be set as close as possible to chronaxie for motor neurons (300–600 microseconds).
3. Pulses per second should be high enough to produce a tetanic contraction (35–55 pps) but adjusted so that muscle fatigue is minimized. Higher rates may be more fatigue producing than rates in the midrange of tetanic contraction.
4. On/off cycles should be based on the equipment parameters available and the clinician's preference in teaching the patient to regain control of the muscle. Currents that ramp up or down will require longer on times, so the effective current is on for 2–3 seconds. Off times can either be a 1:1 contraction to recovery ratio or 1:4 or 5, depending on the clinician's preference or the patient's attention span and/or level of fatigue.
5. Interrupted or surged current must be used.
6. The patient should be instructed to allow just the electricity to make the muscle contract, allowing the patient to feel and see the response desired. Next, the patient should alternate voluntary muscle contractions with current-induced contractions.
7. Total treatment time should be about 15 minutes, but this can be repeated several times daily.
8. High-voltage pulsed or medium-frequency alternating current may be most effective.^{24,64,66}

CASE STUDY 5-2**ELECTRICAL STIMULATING CURRENTS: REEDUCATION OF INNERVATED MUSCLE (2)**

Background: A 16-year-old male underwent arthroscopic partial medial meniscectomy on the right knee yesterday. He is to begin ambulation with crutches, weight bearing as tolerated, today. Clinic policy states that patients must be able to produce an active quadriceps femoris contraction prior to crutch-walking instruction. However, the patient is unable to produce an active contraction of the quadriceps femoris muscle. There is minimal pain and swelling, but after working with the patient for 15 minutes, he remains unable to contract the quadriceps femoris.

Impression: Status postarthroscopic surgery on the right knee with inhibition of quadriceps femoris control.

Treatment Plan: Using a pulsatile monophasic waveform generator, a course of electrical stimulation was initiated. The cathode (active, negative polarity) was placed over the motor point of the vastus medialis, and the anode (inactive, positive polarity) was placed on the posterior thigh. The frequency was set at 40 pps. Using an uninterrupted (1:0) duty cycle, the amplitude was set to a level that produced a visible contraction, but was below the pain threshold. After establishing the stimulus amplitude, the duty cycle was then adjusted to deliver 15 seconds of stimulus followed by 15 seconds of rest; the current was not ramped, so the effective duty cycle was 1:1. The patient was encouraged to contract the quadriceps femoris during the stimulation for the first five stimulations, and then was asked to contract the quadriceps femoris before the stimulus was delivered.

Response: After 20 repetitions of the stimulus, the patient was able to initiate a contraction of the quadriceps femoris before the current was delivered. The electrical stimulation was discontinued, and the patient was able to continue to contract the quadriceps femoris voluntarily. He was then instructed in crutch walking, and routine postoperative rehabilitation was initiated.

Discussion Questions

- What tissues were injured/affected?
- What symptoms were present?
- What phase of the injury-healing continuum did the patient present for care in?
- What are the physical agent modality's biophysical effects (direct/indirect/depth/tissue affinity)?
- What are the physical agent modality's indications/contraindications?
- What are the parameters of the physical agent modality's application/dosage/duration/frequency in this case study?
- What other physical agent modalities could be utilized to treat this injury or condition? Why? How?
- Why was the patient unable to contract the quadriceps femoris following surgery?
- Why was the ability to contract the quadriceps femoris a prerequisite to crutch ambulation?
- What is the difference (pathway and physiology) between the voluntary muscle contraction and the induced (stimulated) contraction?
- How did the electrical stimulation assist the patient in regaining the ability to voluntarily contract the muscle?
- What is a viable alternative approach to assisting this patient?
- What would you suspect if there were no responses to the electrical stimulation?
- Why was the amplitude of stimulus set below the pain threshold?

The rehabilitation professional employs physical agent modalities to create an optimum environment for tissue healing while minimizing the symptoms associated with the trauma or condition.

CASE STUDY 5-3**ELECTRICAL STIMULATING CURRENTS: REEDUCATION OF INNERVATED MUSCLE**

Background: A 23-year-old man experienced a Sunderland grade V lesion of the left radial nerve as a result of an open fracture of the humerus sustained in a motorcycle accident. The injury occurred 2 years ago. There was an unsuccessful primary repair of the nerve injury; because there was no evidence of reinnervation, a sural nerve graft was completed 1 year ago. Again, there was no evidence of reinnervation, so the distal attachment of the flexor carpi radialis (FCR) was transferred to the posterior aspect of the base of the third metacarpal to provide wrist extension. The tendon transfer was completed 3 weeks ago. The wrist and forearm have been immobilized until yesterday, and the patient has been referred for rehabilitation. The surgeon has cleared the patient for gentle FCR contraction.

Impression: Posttendon transfer with lack of voluntary control.

Treatment Plan: Using a pulsatile biphasic waveform generator, a course of therapeutic electrical stimulation was initiated. A bipolar electrode arrangement was used, with one electrode over the motor point of the FCR and the other electrode approximately 4 cm distal, over the FCR. The pulse rate was set at 40 pps, and the effective duty cycle was set at 5:5 (5 seconds on, 5 seconds off), with a 2-second ramp up and a 2-second ramp down (so the total time the current was delivered was 7 seconds, with 7 seconds between stimulations). The current amplitude was adjusted to achieve a palpable contraction of the FCR, but no wrist motion, and the treatment time was set to 12 minutes, so as to achieve approximately 50 contractions.

Response: Treatment was conducted daily for 3 weeks, with gradual increases in the current amplitude and number of repetitions. At this time, the patient was able to initiate wrist extension independent of the electrical stimulation, and was discharged to a home program.

Discussion Questions

- What tissues were injured or affected?
- What symptoms were present?
- What phase of the injury-healing continuum did the patient present for care in?
- What are the physical agent modality's biophysical effects (direct, indirect, depth, and tissue affinity)?
- What are the physical agent modality's indications and contraindications?
- What are the parameters of the physical agent modality's application, dosage, duration, and frequency in this case study?
- What other physical agent modalities could be used to treat this injury or condition? Why? How?
- What structures are involved with a Sunderland grade V peripheral nerve injury?
- What is involved in a sural nerve graft? What was the surgeon trying to achieve?
- What factors led to the failure of the primary radial nerve repair and the sural graft?
- Why did the surgeon wait nearly a year after the primary repair to do the sural graft and nearly a year after the sural graft to perform the tendon transfer?
- Will wrist extension in the absence of extensor digitorum communis function really increase the patient's function? Why or why not?

The rehabilitation professional employs physical agent modalities to create an optimum environment for tissue healing while minimizing the symptoms associated with the trauma or condition.

Muscle pump contractions. Electrically induced muscle contraction can be used to duplicate the regular muscle contractions that help stimulate circulation by pumping fluid and blood through venous and lymphatic channels back into the heart.^{67,183} A discussion of edema formation is included

in Chapter 15. Using sensory-level stimulation has also been found to control and minimize edema formation in sprain and contusion injuries in animals.

Electrical stimulation of muscle contractions in the affected extremity can help in reestablishing the proper circulatory pattern while keeping the injured part protected.⁶⁸⁻⁷¹

Treatment Parameters for Muscle Pumping Contraction to Reduce Edema are as Follows:

1. Current intensity must be high enough to provide a strong, comfortable muscle contraction.
2. Pulse duration is preset on most of the therapeutic generators. If adjustable, it should be set as close as possible to the duration needed for chronaxie (300–600 microseconds) of the motor nerve to be stimulated.
3. Pulses per second should be in the beginnings of tetany range (35–50 pps).
4. Interrupted or surged current must be used.
5. On time should be 5–10 seconds.
6. Off time should be 5–10 seconds.
7. The part to be treated should be elevated.
8. The patient should be instructed to allow the electricity to make the muscles contract. Active range of motion may be encouraged at the same time if it is not contraindicated.
9. Total treatment time should be between 20 and 30 minutes; treatment should be repeated two to five times daily.
10. High-voltage or medium-frequency alternating current may be most effective.^{13,36,72-74}
11. Use this protocol in addition to normal ice for best effect.^{36,75}

Retardation of atrophy. Prevention or retardation of atrophy has traditionally been a reason for treating patients with electrically stimulated muscle contraction. The maintenance of muscle tissue, after an injury that prevents normal muscular exercise, can be accomplished by substituting an electrically stimulated muscle contraction. Injury to a muscle often results in a decrease in voluntary activation of a muscle resulting in muscle weakness. This decreased voluntary activation can be associated with a clinical impairment characterized by a reflex inhibition of the motor neuron pool in uninjured surrounding muscles that is referred to as *arthrogenic muscle inhibition (AMI)*.¹⁸⁷ AMI decreases the ability for the muscle to recruit motor neurons during a contraction, thus limiting its potential to generate force. It has been suggested that reflex inhibition be addressed before engaging in rehabilitative exercise by focusing on disinhibiting the affected muscle prior to strengthening thereby creating a more optimal neural environment for normal functional patterns.¹⁸⁷ Transcutaneous electrical nerve stimulation (TENS) has been shown to disinhibit motor neuron pool excitability and to increase volitional activation. The electrical stimulation reproduces the physical and chemical events associated with normal voluntary muscle contraction and helps to maintain normal muscle function. In designing a program, the clinician should try to duplicate muscle contractions associated with normal exercise routines.¹⁸⁸

Treatment Parameters for Retardation of Atrophy are as Follows:

1. Current intensity should be as high as can be tolerated by the patient. This can be increased during the treatment as some sensory accommodation takes place. The contraction should be capable of moving the limb through the antigravity range or of achieving 25% or more of the normal **maximum voluntary isometric contraction (MVIC)** torque for the muscle. The higher torque readings seem to have the best results.
2. Pulse duration is preset on most of the therapeutic generators. If it is adjustable, it should be set as close as possible to the duration needed for chronaxie (300–600 microseconds) of the motor nerve to be stimulated.
3. Pulses per second should be in the tetany range (50–85 pps).
4. Interrupted or surge-type current should be used.
5. On time should be between 6 and 15 seconds.
6. Off time should be at least 1 minute.
7. The muscle should be given some resistance, either gravity or external resistance provided by the addition of weights or by fixing the joint, so that the contraction becomes isometric.
8. The patient can be instructed to work with the electrically induced contraction, but voluntary effort is not necessary for the success of this treatment.
9. Total treatment time should be 15–20 minutes, or enough time to allow a minimum of 10 contractions; some protocols have been successful with three sets of 10 contractions. The treatment can be repeated two times daily. Some protocols using battery-powered rather than line-powered units have advocated longer bouts with more repetitions, probably because of low contraction force.
10. High-volt or medium-frequency alternating current should be used.^{50,65,76–78}

Muscle strengthening. Neuromuscular electrical stimulation combined with voluntary exercise to increase muscle strength and functional performance has been used during rehabilitation of various musculoskeletal injuries.^{79–85} Electrical stimulation not only has the potential to disinhibit a muscle as discussed previously, but also to activate a greater proportion of type II (fast-twitch) muscle fibers that are essential for higher levels of force production which can result in improved functional performance.^{79–85}

Treatment Parameters for Muscle Strengthening are as Follows:

1. Current intensity should be high enough to make the muscle develop 60% of the torque developed in an MVIC.
2. Pulse duration is preset on most therapeutic generators. If adjustable, it should be set as close as possible to the duration needed for chronaxie (300–600 microseconds) of the motor nerve to be stimulated. In general, longer pulse durations should include more nerves in response.
3. Pulses per second should be in the tetany range (70–85 pps).
4. Surged or interrupted current with a gradual ramp to peak intensity is most effective.
5. On time should be in the 10- to 15-second range.
6. Off time should be in the 50-second to 2-minute range.
7. Resistance usually is applied by immobilizing the limb. The muscle is then given an isometric contraction torque equal to or greater than 25% of the MVIC torque. The greater the percentage of torque produced, the better the results are.
8. The patient can be instructed to work with the electrically induced contraction, but voluntary effort is not necessary for the success of the treatment.
9. Total treatment time should include a minimum of 10 contractions, but mimicking normal active resistive training protocols of three sets of 10 contractions can also be productive. Fatigue is a major factor in this setup. Electrical stimulation bouts should be scheduled at least three times weekly. Generally, strength gains will continue over the treatment course, but intensities may need to increase to keep pace with the most current maximum voluntary contraction torques.
10. High-volt or a medium-frequency Russian current is the current of choice.^{50,61,62,64,65,76–78,86}

Clinical Decision-Making *Exercise 5–6*

A clinician is using electrical stimulation for muscle strengthening following a hamstring muscle strain. What treatment parameters will likely be most effective in improving strength?

CASE STUDY 5-4**ELECTRICAL STIMULATING CURRENTS: STRENGTHENING OF INNERVATED MUSCLE (2)**

Background: A 33-year-old woman sustained an isolated rupture of the left anterior cruciate ligament (ACL) 2 weeks ago while skiing. Three days ago, she underwent an arthroscopically assisted intra-articular reconstruction of the ACL using an autologous patellar ligament graft. She is now weight bearing as tolerated with axillary crutches, is using a removable splint, and has been cleared for accelerated rehabilitation.

Impression: Postoperative ACL reconstruction.

Treatment Plan: In addition to the standard active strengthening and range of motion exercise and physical agent modalities to control postoperative pain and swelling, a course of electrical stimulation for strengthening was initiated. The splint was removed, and the patient was seated on an isokinetic testing and training device, with the left knee in 65 degrees of flexion and the device set at a speed of 0°/s (isometric). A pulsatile polyphasic electrical stimulator was used, with electrodes placed over the motor points of the vastus medialis and vastus lateralis muscles. The stimulator produced a 2500-Hz carrier wave, with an effective frequency of 50 Hz (10 milliseconds on, 10 milliseconds off). A 2-second ramp-up and then a 2-second ramp-down setting was selected, with a total duty cycle of 10:50 (14 seconds on, 50 seconds off), and the current amplitude was adjusted to maximal tolerance during every third stimulation. Fifteen cycles were administered, and then the patient rested for 5 minutes; this was repeated twice, for a total of 45 contractions per treatment session. The patient was treated three times per week for a total of 5 weeks.

Response: A linear increase in force produced during electrical stimulation, as well as maximal isometric force production, was recorded over the 5 weeks of treatment. The patient's gait and range of motion improved, and she was discharged to a home program at the end of treatment.

Discussion Questions

- What tissues were injured or affected?
- What symptoms were present?
- What phase of the injury-healing continuum did the patient present for care in?
- What are the physical agent modality's biophysical effects (direct, indirect, depth, and tissue affinity)?
- What are the physical agent modality's indications and contraindications?
- What are the parameters of the physical agent modality's application, dosage, duration, and frequency in this case study?
- What other physical agent modalities could be used in the rehabilitation of this injury? Why? How?
- Why was the training of the quadriceps femoris conducted at 65 degrees of flexion? What biomechanical factors favor training at this joint angle as opposed to full extension of the knee?
- What effect did the electrical stimulation have on the healing rate of the reconstruction? On the patient's return to function?

The rehabilitation professional employs physical agent modalities to create an optimum environment for tissue healing while minimizing the symptoms associated with the trauma or condition.

CASE STUDY 5-5**ELECTRICAL STIMULATING CURRENTS: PAIN MODULATION**

Background: A 47-year-old man sustained a closed crush injury of the right foot in a construction accident 12 weeks ago. Radiographs revealed no bone injury, and the physical examination indicated that the neurovascular structures were intact. A pneumatic immobilization device was applied to the right leg in the emergency department, the patient was supplied with axillary crutches, and he was instructed to avoid weight bearing on the right foot until he was cleared by his family physician. The immobilization device was removed 6 weeks ago, and the patient was instructed to begin progressive weight bearing and to exercise the foot on his own. He has now been referred to you because of a progressive increase in burning pain in the foot and leg, with swelling and extreme sensitivity to touch. The patient refuses to bear weight on the foot and is not wearing a sock or shoe on the right foot.

Impression: Complex regional pain syndrome (CRPS) type I (aka reflex sympathetic dystrophy).

Treatment Plan: A pulsatile biphasic current was delivered to the right leg, with electrodes over the anterior and posterior compartments. The frequency was 2 pps, and the amplitude was above the patient's pain threshold but below pain tolerance; a strong muscular twitch response was elicited. The current was delivered without interruption (duty cycle of 1:0) for 60 seconds. When the current was turned off, the patient's foot was brushed lightly with the therapist's hands. The process was repeated a total of 10 times in the initial treatment session, and the patient was instructed to attempt the brushing process at home.

Response: After the initial 60 seconds of current at the first treatment session, the patient was able to tolerate 5 seconds of light touch. After the 10th period of stimulation, the patient was able to tolerate 45 seconds of moderate touch. Treatment was repeated 3 days per week for 2 weeks, at which time the patient was able to tolerate a sock and shoe, was partial weight bearing, and continued the desensitization process on a home program.

Discussion Questions

- What tissues were injured or affected?
- What symptoms were present?
- What phase of the injury-healing continuum did the patient present for care in?
- What are the physical agent modality's biophysical effects (direct, indirect, depth, and tissue affinity)?
- What are the physical agent modality's indications and contraindications?
- What are the parameters of the physical agent modality's application, dosage, duration, and frequency in this case study?
- What other physical agent modalities could be used to treat this injury or condition? Why? How?
- What is CRPS type I?
- What is the difference between CRPS type I and CRPS type II?
- Why was low-frequency TENS selected for this patient? Would other forms of TENS (e.g., conventional, hyperstimulation) have been effective? Why or why not?
- Is it likely that CRPS could have been prevented in this patient? How?

The rehabilitation professional employs physical agent modalities to create an optimum environment for tissue healing while minimizing the symptoms associated with the trauma or condition.

The Effect of Noncontractile Stimulation on Edema

Ion movement within biologic tissues is a basic theory in the electrotherapy literature.

Since 1987, numerous studies using rat and frog models have helped to more clearly define the effects of electrical stimulation on edema formation and reduction.^{7,21,68–70,87} The muscle pumping theory discussed previously has seemed to be the most viable way to affect this problem.²⁸ Most of the recent studies have focused on a sensory-level stimulation. Early theory supported the use of sensory-level DC as a driving force to make the charged plasma protein ions in the interstitial spaces move in the direction of the oppositely charged electrode. In addition, some of the early basic science indicated that electrical stimulation decreases capillary permeability which is likely the primary cause of edema formation. Taylor demonstrated an increased lymphatic uptake of labeled **albumin** within rats treated with sensory-level high-voltage stimulation.⁶⁷ However, there was no significant reduction in the limb volume. They hypothesized that the electric field introduced into the area of edema facilitated the movement of the charged proteins into the lymphatic channels. When the lymphatic channel volume increased, so too did the contraction rate of the smooth muscle in the lymphatics. They also hypothesized that stimulation of sensory neurons may cause an indirect activation of the autonomic nervous system. This might cause release of adrenergic substances that would also increase the rate smooth muscle contraction in the lymphatic tissue.

Another proposed mechanism is that a microamp stimulation of the local neurovascular components in an injured area may cause a vasoconstriction and reduce the permeability of the capillary walls to limit the migration of plasma proteins into the interstitial spaces. This would retard the accumulation of plasma proteins and the associated fluid dynamics of the edema exudate. In a study on the histamine-stimulated leakage of plasma proteins, animals treated with small doses of electrical current produced less leakage.⁶⁷ The underlying mechanisms were a reduced pore size in the capillary walls and reduced pooling of blood in the capillaries, which could have been initiated by hormonal, neural, mechanical, or electrochemical factors.

It has been demonstrated on laboratory animals that high-voltage pulsed current effectively curbs edema formation if applied immediately after acute injury. The effects were most significant when the treatments were continuous throughout the period when edema was actively forming.⁶⁸

Treatment Parameters for Edema Control are as Follows:

1. Current intensity of 30–50 V or 10% less than that needed to produce a visible muscle contraction is most effective.
2. Preset short-duration currents on the high-voltage equipment are effective.
3. High pulse frequencies (120 pps) are most effective.
4. Interrupted monophasic currents are most effective. Biphasic currents showed increases in volume.
5. The animals treated with a negative distal electrode had a significant treatment effect. The animals with a positive distal electrode showed no change.
6. Time of treatment after injury: the best results were reported when treatment began immediately after injury. Treatment started after 24 hours showed an effect on the accumulation of new edema volume but showed no effect on the existing edema volume.
7. A 30-minute treatment showed good control of volume for 4–5 hours.
8. The water immersion electrode technique was effective, but using surface electrodes was not effective.
9. High-volt pulsed generators were effective, and low-volt generators were not effective.^{8,18,33,75,88–98}

THERAPEUTIC USES OF ELECTRICAL STIMULATION OF SENSORY NERVES

Gate control theory. Providing maximum sensory cutaneous stimulation to peripheral sensory A β fibers when there is pain in a certain area will generally “close the gate” to painful afferent impulses being transmitted to the spinal cord on A δ and C fibers at the spinal cord level. As long as the stimuli are applied, the perception of pain is diminished. Electrical stimulation of sensory nerves will evoke the gate control mechanism and diminish awareness of painful stimuli.^{2,35,40,42,46,76,78,99–104} This type of treatment is referred to as a *conventional, high-frequency, or sensory-level TENS*

treatment and is the most commonly used TENS protocol. The intensity is set only high enough to elicit a tingling sensation but not high enough to cause a muscle contraction. Pain relief lasts while the stimulus is turned on, but it usually abates when the stimulation stops. Normally patients apply the electrodes and leave them in place all day, turning the stimulus on for approximately 30-minute intervals.

Treatment Parameters for Conventional TENS Treatment (Gate Control) are as Follows:

1. Current intensity should be adjusted to tolerance but should not cause a muscular contraction—the higher the better.
2. Pulse duration (pulse width) should be 75–150 microseconds or maximum possible on the machine.
3. Pulses per second should be 80–125, or as high as possible on the machine.
4. A transcutaneous electrical stimulator waveform should be used (most commonly asymmetric biphasic, but it can be symmetric biphasic and less commonly monophasic).
5. On time should be continuous mode.
6. Total treatment time should correspond to fluctuations in pain; the unit should be left on until pain is no longer perceived, turned off, and then restarted when pain begins again.
7. If this treatment is successful, you will have some pain relief within the first 30 minutes of treatment.
8. If it is not successful, but you feel this is the best theoretical or most clinically applicable approach, change the electrode placements and try again. If this is not successful, then using a different theoretical approach may offer more help.
9. Any stimulator that can deliver this current is acceptable. Portable units are better for 24-hour pain control (see [Figure 5–21](#)).^{40,42,105}

Descending pain control theory. Intense electrical stimulation of the smaller peripheral A δ and C fibers that transmit pain causes stimulation of the midbrain, pons, and medulla. In turn, this causes the release of enkephalin through descending neurons, which blocks the pain impulses at the spinal cord level (see [Figure 3–9](#)).⁴⁸ Cognitive input from the cortex relative to past pain perception and experiences also contributes to this descending mechanism control. This type of treatment is referred to as a **low-frequency** or **motor-level** TENS treatment. The intensity is set high enough to elicit both a tingling sensation and a muscle contraction. Pain relief with motor-level TENS should be expected to take longer than with conventional TENS (15–60 minutes), but the relief likely will last longer (>1 hour).

Treatment Parameters for Low-frequency or Motor-level TENS are as Follows:

1. Current intensity should be high enough to elicit a muscle contraction.
2. Pulse duration should be 100–600 microseconds.
3. Pulses per second should be <20 pps.
4. On time should be 30 seconds to 1 minute.
5. Stimulation should be applied over points where it is not difficult to elicit a motor response such as a motor point or even over acupuncture and trigger points.
6. Selection and number of points used vary according to the part treated but they do not necessarily have to be over the area of pain.
7. If this treatment is successful, pain will be relieved in 15–60 minutes but relief may last longer than 1 hour.
8. If this treatment is not successful, try different electrode setups by expanding the treatment points used.

Endogenous opiate pain control theory. Electrical stimulation of sensory nerves may stimulate the release of β -endorphin and dynorphin from the pituitary gland and the hypothalamus into the cerebral spinal fluid. The mechanism that causes the release and then the binding of β -endorphin, dynorphin, and ultimately enkephalin to some nerve cells is still unclear. It is certain that a diminution or elimination of pain perception is caused by applying a noxious electrical current to areas close to the site of pain or to acupuncture or trigger points, both local and distant to the pain area.^{48,76,94,106–112}

To use the influence of hyperstimulation analgesia and β -endorphin release, a point stimulation setup must be used.¹⁰⁹ This approach utilizes a large dispersive pad and a small pad or handheld probe point electrode. The point electrode is applied to the chosen site, and the intensity is increased until the patient perceives it. The probe is then moved around the area, and the patient is asked to report relative changes in perception of intensity. When a location of maximum-intensity perception is found, the current intensity is increased to noxious but tolerable levels.¹¹³ This is much the same as finding a motor point, as described earlier.^{48,114}

β -Endorphin stimulation may offer better relief for the deep aching or chronic pain similar to the pain of overuse injury. The intensity of the impulse is a function of both pulse duration and amplitude. Comfort is a very important determinant of patient compliance and, thus, the overall success of treatment. Greater pulse widths tend to be more painful. The method of delivering TENS is less tolerable because the impulse intensity is higher.

A combination of noxious point stimulation and transcutaneous electrical nerve stimulation may be used. The transcutaneous electrical nerve stimulation applications should be used as much as needed to make the patient comfortable, and the intense point stimulation should be used on a periodic basis. Periodic use of intense point stimulation gives maximal pain relief for a period of time and allows some gains in overall pain suppression. Daily intense point stimulation may eventually bias the central nervous system and decrease the effectiveness of this type of stimulation.²⁶

Treatment Protocols for Noxious-level TENS are as Follows:

1. Current intensity should be high, at a noxious level: muscular contraction is acceptable.
2. Pulse duration should be 100–1000 microseconds.
3. Pulses per second should be between 1 and 5.
4. High-volt pulsed current should be used.
5. On time should be 30–45 seconds.
6. Stimulation should be applied over trigger or acupuncture points.
7. Selection and number of points used vary according to the part and condition being treated.
8. A high-volt PC or a low-frequency, high-intensity machine is best for this effect.^{48,102,103}
9. If stimulation is successful, you should know at the completion of the treatment. The analgesic effect should last for several (6–7) hours.
10. If not successful, try expanding the number of stimulation sites. Add the same stimulation points on the opposite side of the body, add auricular (ear) acupuncture points, and add more points on the same limb.

Clinical Decision-Making Exercise 5–7

The clinician is treating a myofascial trigger point in the upper trapezius. He decides to use a point stimulator for the purpose of pain modulation. What treatment technique will likely be most effective?

CLINICALLY USED ELECTRICAL STIMULATING CURRENTS

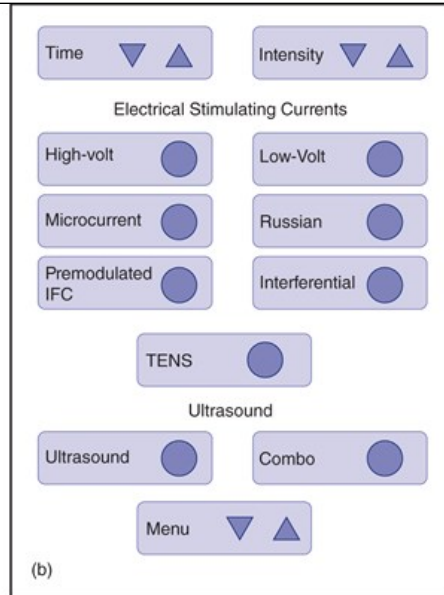
Over the years, advances in technology have enabled manufacturers of electrical stimulators to offer sophisticated pieces of equipment that allow the clinician the flexibility of making choices when it comes to selecting the most appropriate type of currents and treatment parameters to accomplish a specific treatment goal.¹⁷⁹ The newest electrical stimulating units are capable of outputting multiple types of current including high volt, TENS, microcurrent, Russian, interferential, premodulated interferential, and low volt (Figure 5–26). Table 5–2 provides a list of indications and contraindications for using the various types of electrical currents. A detailed discussion of these various types of current follows.

Figure 5–26.

Most electrical stimulating units allow the clinician to choose from a variety of current choices. Some units offer multiple modality options. (a) A combination of electrical stimulating unit and ultrasound. (Courtesy DonJoy Global) (b) Control panel for selecting current options.



(a)



(b)

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Table 5-2

Summary of Indications and Contraindications for Electrical Stimulating Currents

INDICATIONS
Modulating acute, postacute, and chronic pain
Stimulating contraction of denervated muscle
Retarding atrophy
Muscle strengthening
Muscle reeducation
Muscle pumping contraction
Curbing edema formation
Decreasing muscle spasm
Decreasing muscle guarding
Stimulating the healing process
Wound healing
Fracture healing
Tendon healing
Ligament healing

Stimulating nerve regeneration
Stimulating peripheral nervous system function
Changing membrane permeability
Synthesizing protein
Stimulating fibroblasts and osteoblasts
Regenerating tissue
Increasing circulation through muscle pumping contractions

CONTRAINDICATIONS

Pacemakers
Infection
Malignancies
Pregnancy
Musculoskeletal problems where muscle contraction would exacerbate the condition

High-Volt Currents

High-volt currents are widely used for a variety of clinical purposes: eliciting muscle contractions, pain control, and curbing edema. Although high-volt current is most commonly used to cause muscle contraction, it should be made clear that other types of electrical currents—Russian, interferential, premodulated interferential, or biphasic—may also be used. While high-volt current can also be used to control pain, it is not the current of choice. Many of the devices that generate high-volt current are not portable. Thus, TENS would be a better treatment modality choice for long-term pain relief. The efficiency and effectiveness of treatment can be increased by following the protocols as closely as possible with the available equipment. A high-volt current is a twin-peaked pulsed waveform that has a long interpulse interval (see [Figure 5-7](#)).

Transcutaneous Electrical Nerve Stimulation (TENS)

Asymmetric biphasic currents are found on the majority of portable TENS units ([Figure 5-27](#)). The term *transcutaneous electrical nerve stimulation* has become closely associated with pain control. Clinically, efforts are made to stimulate the sensory nerves to change the patient's perception of a painful stimulus coming from an injured area. A TENS unit consists of an electrical signal generator, a battery, and a set of electrodes. The units are small and programmable, and the generators can deliver trains of stimuli with variable current strengths, pulse rates, and pulse widths. To understand how to maximally affect the perception of pain through electrical stimulation, it is necessary to understand pain perception. The gate control theory, the descending control theory, and the endogenous opiate pain control theory are the theoretical basis for pain reduction phenomena. These theories were covered in depth in [Chapter 4](#).

Figure 5-27.

Portable TENS unit. (Courtesy DonJoy Global)



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Clinical Decision-Making Exercise 5–8

How should a clinician go about setting up a conventional TENS treatment for a sore biceps muscle?

Microcurrent

Generators that produce subsensory-level stimulation were originally called microcurrent electrical neuromuscular stimulators (MENS). However, the stimulation pathway is not the usual neural pathway, and these machines are not designed to stimulate a muscle contraction.

Consequently, this type of generator was subsequently referred to as a microcurrent electrical stimulator (MES). *Low-intensity stimulation (LIS)* is another currently used term in an ongoing evolution of terminology relative to this type of stimulation. A review of the current existing literature shows the term *microcurrent* to be the most widely used term to refer to this type of current.

Microcurrent < 1 mA

Perhaps the most important point to emphasize is that microcurrents are not substantially different from the currents discussed previously. These currents still have a direction, and both biphasic and monophasic waveforms are available. The currents also have amplitude (intensity), pulse duration, and frequency. The characteristic that distinguishes this type of current is that the intensity of the stimulus is limited to 1000 μA (1 mA) or less in microcurrent, whereas the intensity of the standard low-voltage equipment can be increased into the milliamp range.¹¹⁵

The generators can generate a variety of waveforms from modified monophasic to biphasic square waves with frequencies from 0.3 to 50 Hz. The pulse durations are also variable and may be prolonged at the lower frequencies from 1 to 500 milliseconds. This varies as the frequency changes or is preset when PC are used. Many of these devices are made with an impedance-sensitive voltage that adapts the current to the impedance to keep the current constant as selected.¹¹⁶

If the current generator can be adjusted to allow increases of intensity above 1000 μA , the current becomes like those previously described in this text. If the current provokes an action potential in a sensory or motor nerve, the results on that tissue will be the same as previously described for the sensations or muscle contractions caused by other currents.

Most of the literature on microcurrents and subsequently on subsensory stimulators has been generated by researchers interested in stimulating the healing process in fractures and skin wounds. Subsequent research aimed at identifying why and how microcurrents work. The best researched areas of application of microcurrents are in the stimulation of bone formation in delayed union or nonunion of fractures of the long bones. Most of this research was done using implanted rather than surface electrodes, and most has used low-intensity direct current (LIDC) with the negative pole placed at the fracture site.^{8,86,117} We are in danger of generalizing treatments for all problems based on success in this one area. These applications were intended to mimic the normal electric field created during the injury and healing process.^{38,91} At present, these electrical changes are poorly understood, and the effects of adding additional electrical current to the normal electrical activity created by the injury and healing process are still being investigated.

Microcurrent effects are as follows:

- analgesia;
- fracture healing;
- wound healing;
- ligament and tendon healing.

Microcurrent stimulation has been used for two major effects:

1. analgesia of the painful area¹⁸⁴
2. biostimulation of the healing process, either for enhancing the process or for acceleration of its stages.^{73,173}

CASE STUDY 5-6**ELECTRICAL STIMULATING: ANALGESIA**

Background: A 52-year-old woman is 9 months post-hemilaminectomy and discectomy without fusion at L5-S1 due to a herniated disc with compromise of the S1 nerve root. The surgery resulted in relief of the peripheral pain, weakness, and sensory loss, but persistent pain in the lumbosacral spine and buttock prevents the patient from engaging in rehabilitation exercises effectively.

Impression: Status postspinal surgery with persistent postoperative pain; no neural deficit.

Treatment Plan: The patient was already being treated with a hot pack prior to exercise; conventional TENS was added to the treatment regimen. Electrodes were placed at the L3-4 interspace and over the greater trochanter. A pulsatile biphasic waveform was selected, with a rate of 60 pps, an amplitude between the sensory and motor thresholds, and a duty cycle of 1:0 (uninterrupted). The stimulation was delivered for the 10-minute heat application and remained in place during the therapeutic exercise, as well as for 30 minutes following the exercise.

Response: The patient experienced a 60% reduction in the symptoms during the exercise; this enabled the patient to perform the exercise through a greater range and with a greater effect. The effect of the TENS began to diminish after 8 weeks, but the pain had diminished to manageable levels such that the patient was able to continue the rehabilitation program without the TENS.

Discussion Questions

- What tissues were injured/affected?
- What symptoms were present?
- What phase of the injury-healing continuum did the patient present for care in?
- What are the physical agent modality's biophysical effects (direct/indirect/depth/tissue affinity)?
- What are the physical agent modality's indications/contraindications?
- What are the parameters of the physical agent modality's application/dosage/duration/frequency in this case study?
- What other physical agent modalities could be utilized to treat this injury or condition? Why? How?
- What factors led to the selection of conventional TENS?
- What would be the advantages and disadvantages of low TENS for this patient?
- What is the theoretical mechanism of action of conventional TENS?
- Why did the effect of the TENS diminish over time?
- Would you characterize the patient's pain as chronic or acute? Why? Are there different optimum forms of electrical stimulation for pain relief dependent on the nature of the pain?

The rehabilitation professional employs physical agent modalities to create an optimum environment for tissue healing while minimizing the symptoms associated with the trauma or condition.

Analgesic Effects of Microcurrent

The mechanism of analgesia created by microcurrent does not fit into our present theoretical framework, as sensory nerve excitation is a necessary component of all three models of electroanalgesia stimulation. At best, microcurrent can create or change the constant DC flow of the neural tissues, which may have some way of biasing the transmission of the painful stimulus. LIS may also make the nerve cell membrane more receptive to neurotransmitters that will block transmission. The exact mechanism has not yet been established. The research is not supportive of the effectiveness

of microcurrent for pain reduction.^{118,119} This lack of consensus and disagreement in the research give the clinician limited security in devising an effective protocol. Most of the research uses delayed onset muscle soreness (DOMS) or cold-induced pain models, and results show no difference between microcurrent and placebo treatments.^{54,73,89,103,104,120–130}

Biostimulative Effects on the Healing Process

Promotion of wound healing. Low-intensity direct current has been used to treat skin ulcers that have poor blood flow. The treated ulcers show accelerated healing rates when compared with untreated skin ulcers. Other protocols have been successful using the anode in the wound area for the entire time. High-voltage stimulation also has been used in a manner similar to the negative–positive model presented. The intensity was adjusted to give a microamp current.

The mechanism by which microcurrent stimulates healing is elusive, but cells are stimulated to increase their normal proliferation, migration, motility, DNA synthesis, and collagen synthesis. Receptor levels for growth factor have also shown a significant increase when wound areas are stimulated.^{30,50,100,131–138} The naturally occurring electrical potential gradients are enhanced following electrical stimulation.⁵⁴ Details of how electrical stimulating currents are used in wound healing were presented in [Chapter 3](#).

Treatment Parameters for Wound Healing are as Follows:

1. Current intensity is 200–400 μA for normal skin and 400–800 μA for denervated skin.
2. Long pulse durations or continuous uninterrupted currents can be used.
3. Maximum pulse frequency.
4. Monophasic DC is best. Microcurrent stimulators can be used but other generators with intensities adjusted to subsensory levels also can be effective. A battery-powered portable unit is most convenient.
5. Treatment time is 2 hours followed by a 4-hour rest time.
6. Utilize two to three treatment bouts per day.
7. The negative electrode is positioned in the wound area for the first 3 days. The positive electrode should be positioned 25 cm proximal to the wound.
8. After 3 days the polarity is reversed and the positive electrode is positioned in the wound area.
9. If infection is present, the negative electrode should be left in the wound area until the signs of infection are not evident. The negative electrode remains in the wound for 3 days after the infection clears.
10. If the wound size decreases to a plateau, then return the negative electrode to the wound area for 3 days.

Promotion of fracture healing. The use of subsensory DC may be an adjunctive modality in the treatment of fractures, especially fractures prone to nonunion. Fracture healing may be accelerated by passing a monophasic current through the fracture site. Getting the current into the bony area without an invasive technique is difficult.^{43,46,48,58,86,121,139–141}

Using a standard transcutaneous electrical nerve stimulation unit, Kahn reported favorable results in the electrical stimulation of callus formation in fractures that had nonunions after 6 months.¹⁴² This information is based on a case study. Results of a more extensive population of nonunions have not been documented.

Treatment Protocols for Fracture Healing are as Follows:

1. Current intensity was just perceptible to the patient.
2. Pulse duration was the longest duration allowed on the unit (100–200 milliseconds).
3. Pulses per second were set at the lowest frequency allowed on the unit (5–10 pps).
4. Standard monophasic or biphasic current in the transcutaneous electrical stimulating units was used.
5. Treatment time was from 30 minutes to 1 hour, three to four times daily.
6. A negative electrode was placed close to but distal to the fracture site. A positive electrode was placed proximal to the immobilizing device.
7. If four pads were used, the interferential placement described earlier was used.
8. Results were reassessed at monthly intervals.¹⁴²

Promotion of healing in tendon and ligament. There are only a few research studies on the biostimulative effect of electrical stimulation on tendon or ligament healing. Both tissues have been found to generate strain-generated electrical potentials naturally in response to stress. These potentials help signal the tissue to grow in response to the stress according to Wolff's law.

In an experimental study on partial division of dog patellar tendons treated with 20- μ A cathodal stimulation, the stimulated tendons showed 92% recovery of normal breaking strength at 8 weeks.¹⁴³

Tendon stimulated in vitro in a culture medium showed increased fibroblastic cellular activity, tendon cellular proliferation, and collagen synthesis. The rate at which stimulated tendons demonstrated histologic repair at the injury site was also significantly accelerated over the control group.¹⁴⁴ Litke and Dahners studied rat medial collateral ligament (MCL) injuries treated with electrical stimulation. The treated group showed statistical significance in the rupture force, stiffness, energy absorbed, and laxity.⁵⁶

As can be seen by the previous sections, microcurrent can be a valuable addition to the clinical armamentarium of the clinician, but it is untested clinically.

Microcurrent is a case where more may not be better. For electricity to produce these effects: (1) cells must be current sensitive; (2) correct polarity orientation may be necessary; and (3) correct amounts of current will cause the cells to be more active in the healing process.

If results are not positive, then reduce the current and/or change polarity. Weak stimuli may increase physiologic activity, whereas very much stronger stimuli abolish or inhibit activity.

Most generators in use today are capable of delivering microcurrent. Simply turn the machine on but do not increase the intensity to threshold levels. This can also be a function of current density using electrode size and placement as well as intensity to keep current in the microampere range. The clinician is certainly entitled to be very skeptical of the manufacturers' claims until more research is reported. Existing protocols for use are not well established, which leaves the clinician with an insecure feeling about this modality.

Russian Currents (Medium-Frequency Current Generators)

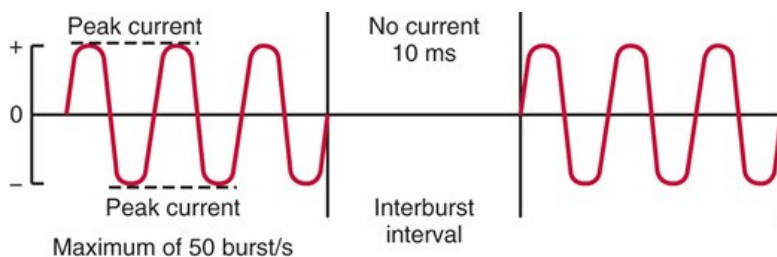
This class of current generators was developed in Canada and the United States after the Russian scientist Yadou M. Kots presented a seminar on the use of electrical muscular stimulators to augment strength gain.¹⁴⁵ The stimulators developed after this presentation were termed **Russian current** generators. These stimulators have evolved and presently deliver a medium-frequency (2000–10,000 Hz) pulsatile biphasic waveform. The pulse can be varied from 50 to 250 microseconds; the phase duration will be one half of the pulse duration, or 25–125 microseconds.¹⁴⁶ As the pulse frequency increases, the pulse duration decreases.^{45,91,147}

Russian current produces two basic waveforms: a sine wave and a square wave cycle with a fixed intrapulse interval. The sine wave is produced in a burst mode that has a 50% on/off time. According to SD curve data, to obtain the same stimulation effect as the duration of the stimulus decreases, the intensity must be increased. The intensity associated with this duration of current could be considered painful.

To make this intensity of current tolerable, it is generated in 50-bursts-per-second envelopes with an interburst interval of 10 milliseconds. This slightly reduces the total current but allows enough of a peak current intensity to stimulate muscle very well (Figure 5-28). If the current continued without the burst effect, the total current delivered would equal the lightly shaded area in Figure 5-29. When generated with the burst effect, the total current is decreased. In this case, the total current would equal the darkly shaded area in Figure 5-30. This allows the patient to tolerate greater current intensity. The other factor affecting patient comfort is the effect that frequency will have on the impedance of the tissue. Higher-frequency currents reduce the resistance to the current flow, again making this type of waveform comfortable enough that the patient may tolerate higher intensities. As the intensity increases, more motor nerves are stimulated, increasing the magnitude of the contraction.⁸⁰ Because it is a fast-oscillating biphasic current, as soon as the nerve repolarizes it is stimulated again, producing a current that will maximally summate muscle contraction.^{23,148} The primary clinical use of Russian current is for muscle strengthening.

Figure 5-28.

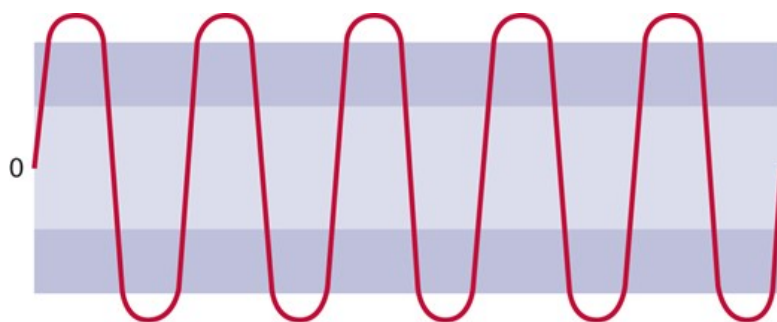
Russian current with polyphasic AC waveform and 10-millisecond interburst interval.



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Figure 5-29.

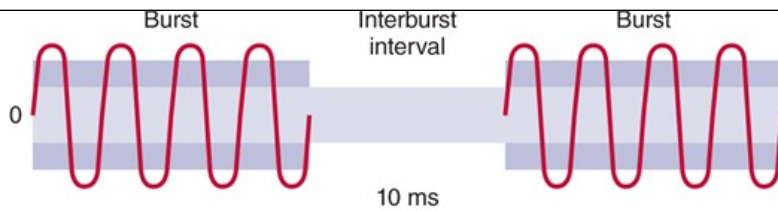
Russian current without an interburst interval. The lightly shaded area is equal to the total current.



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Figure 5-30.

Russian current with an interburst interval. Darkly shaded area represents total current, and light shading indicates total current without the interburst interval.



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The frequency (pulses per second or, in this case, bursts per second) is a variable that can be controlled to make the muscle respond with a twitch rather than a gradually increasing mechanical contraction. Gradually increasing the numbers of bursts interrupts the mechanical relaxation cycle of the muscle and causes more shortening to take place (see [Figure 5-13](#)).²⁵

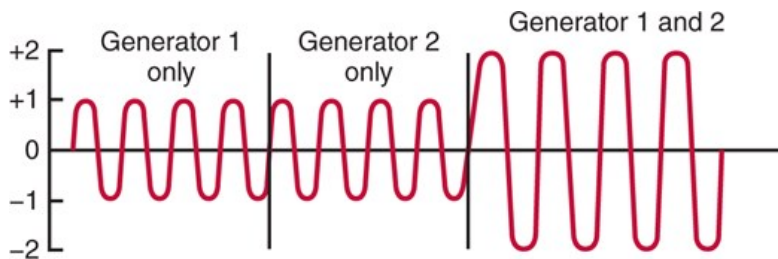
Interferential Currents

The research on and use of IFC has taken place primarily in Europe. An Austrian scientist, Ho Nemec, introduced the concept and suggested its therapeutic use. The theories and behavior of electrical waves are part of basic physics. This behavior is easiest to understand when continuous sine waves are used as an example.

With only one circuit, the current behaves as described earlier; if put on an oscilloscope, it looks like generator 1 in [Figure 5-31](#). If a second generator is brought into the same location, the currents may interfere with each other. This interference can be summative—that is, the amplitudes of the electrical wave are combined and increase ([Figure 5-31](#)). Both waves are exactly the same; if they are produced in phase or originate at the same time, they combine. This is called **constructive interference**.

Figure 5-31.

Sine wave from generator 1 and sine wave from generator 2 showing a constructive interference pattern.

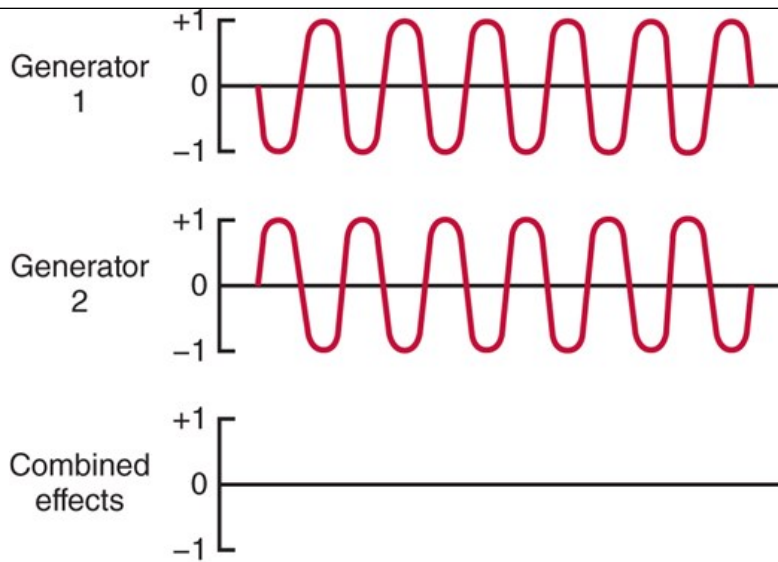


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If these waves are generated out of sync, generator 1 starts in a positive direction at the same time that generator 2 starts in a negative direction; the waves then will cancel each other out. This is called **destructive interference**; in the summation the waves end up with an amplitude of 0 ([Figure 5-32](#)).

Figure 5-32.

Sine wave from generator 1 and sine wave from generator 2 showing a destructive interference pattern.

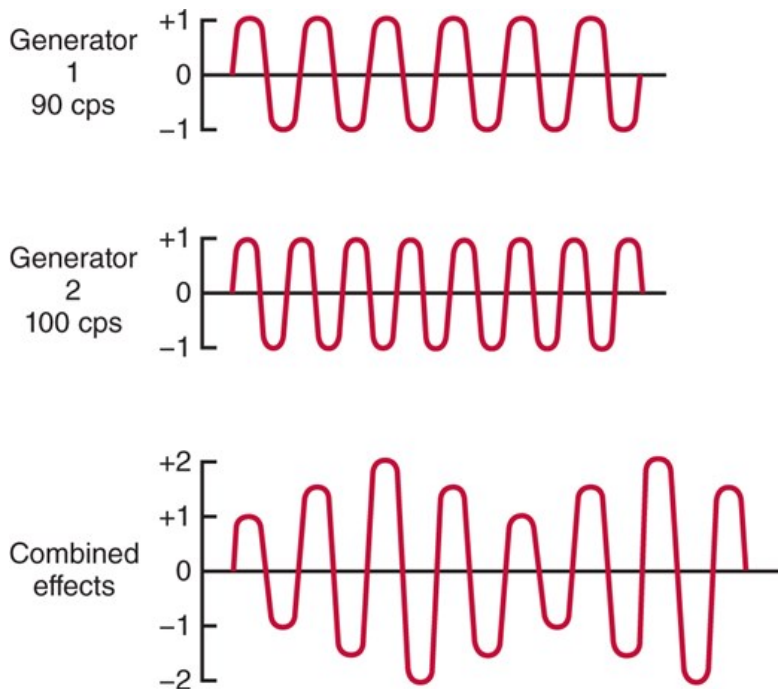


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To make this more complex, assume that one generator has a slightly slower or faster frequency and that the generators begin producing current simultaneously. Initially, the electrical waves will be constructively summated; however, because the frequencies of the two waves differ, they gradually will get out of phase and become destructively summated. When dealing with sound waves, we hear distinct beats as this phenomenon occurs. We borrow the term *beat* when describing this behavior. When any waveforms are out of phase but are combined in the same location, the waves will cause a beat effect. The blending of the waves is caused by the constructive and destructive interference patterns of the waves and is called *heterodyne* (Figure 5-33).^{91,149}

Figure 5-33.

Sine wave from generator 1 at 90 cps and sine wave from generator 2 at 100 cps showing the heterodyne, or beating pattern, of interference.



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The heterodyne effect is seen on an oscilloscope as a cyclic, rising and falling waveform.¹⁵⁰ The peaks or beat frequency in this heterodyne wave

behavior occur regularly, according to the difference of each current. With IFC, one generator produces current at a frequency of 4080 pps. The second generator outputs current at a frequency of 4080 pps. Thus, the beat frequency would be 80 pps:

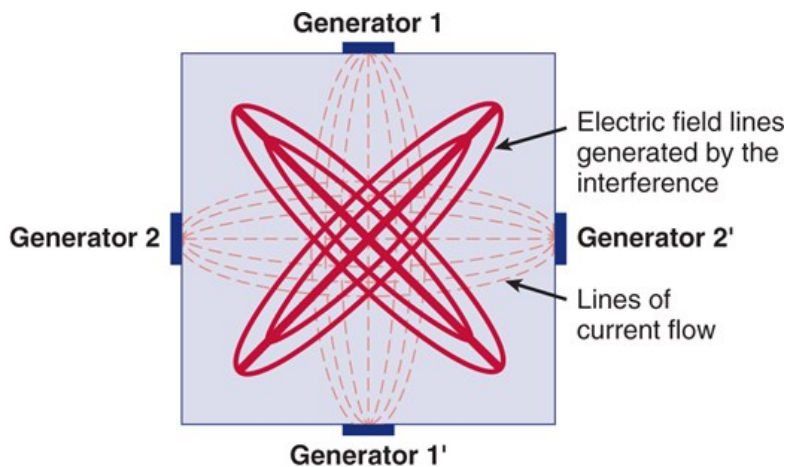
$$4080 \text{ pps} \sum 4000 \text{ pps} = 80 \text{ pps beat frequency} \quad 4080 \text{ pps} \sum 4000 \text{ pps} = 80 \text{ pps beat frequency}$$

In electrical currents, this beat frequency is, in effect, the stimulation frequency of the waveform because the destructive interference negates the effects of the other part of the wave. The intensity (amplitude) will be set according to sensations created by this peak.⁹¹ When using an interference current for therapy, the clinician should select the frequencies to create a beat frequency corresponding to his or her choices of frequency when using other stimulators: 20–50 pps for muscle contraction, 50–120 pps for a conventional TENS treatment, and 1 pps for endogenous opiate pain modulation.

When the electrodes are arranged in a square alignment and IFC are passed through a homogeneous medium, a predictable pattern of interference will occur. In this pattern, an electric field is created that resembles a four-petaled flower, with the center of the flower located where the two currents cross and the petals falling between the electrical current force lines. The maximum interference effect takes place near the center, with the field gradually decreasing in strength as it moves toward the points of the petal (Figure 5–34).⁹¹ Because the body is not a homogeneous medium, we cannot predict the exact location of this interference pattern; we must rely on the patient's perception. If the patient has a localized structure that is painful, locating the stimulation in the correct location is relatively easy. The clinician moves the electrode placement until the patient centers the feeling of the stimulus in the problem area.^{91,149} When a patient has poorly localized pain, the task becomes more difficult. See the discussion in the section “Electrode Placement” for a general discussion on the effect of electrode movement. The engineers added features to the generators and created a scanning IFC that moves the flower petals of force around while the treatment is taking place. This enlarges the effective treatment area. Additional technology and another set of electrodes create a three-dimensional flower effect when one looks at the electric field. This is called a **stereodynamic interference current**.^{91,149}

Figure 5–34.

Square electrode alignment and interference pattern of current in a homogeneous medium.



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All these alterations and modifications are designed to spread the heterodyne effect throughout the tissue. Because it is controlled by a cyclic electrical pattern, however, we actually may be decreasing the current passed through the structures we are trying to treat. The machines seem complex but lack the versatility to do much more than the conventional TENS treatment.^{25,151}

CASE STUDY 5-7**ELECTRICAL STIMULATION: CONTROL OF SWELLING**

Background: A 43-year-old woman, recreational runner, sustained a grade II ankle sprain (inversion stress) approximately 4 hours prior to presentation for treatment. She is ambulatory in a touch weight-bearing mode with crutches and is not in acute distress. Positive signs are limited to the ankle, which demonstrates 3+/4 swelling, marked restriction in range of motion, and point tenderness over the anterior talofibular and calcaneofibular ligaments. There is no loss of ligamentous stability.

Impression: Grade II ankle sprain, with significant swelling.

Treatment Plan: In addition to therapeutic exercise, electrical stimulation was selected to assist in the reduction of the swelling. A monophasic pulsatile waveform generator was selected, and the cathode (negative polarity) was placed over the anteriolateral aspect of the ankle, with the anode over the posterior leg. The pulse rate was set at 120 pps, with the amplitude between the sensory and motor thresholds. Stimulation was applied for 30 minutes daily.

Response: The swelling was reduced by approximately 30% after the initial treatment, but had returned the next day. Over the next 5 days, the swelling was markedly reduced following treatment, but regressed by about 50% by the next day. Electrical stimulation was discontinued after 7 days. A progressive rehabilitation program was initiated the first day, and the patient returned to full activity after 3 weeks.

Discussion Questions

- What tissues were injured/affected?
- What symptoms were present?
- What phase of the injury-healing continuum did the patient present for care in?
- What are the physical agent modality's biophysical effects (direct/indirect/depth/tissue affinity)?
- What are the physical agent modality's indications/contraindications?
- What are the parameters of the physical agent modality's application/dosage/duration/frequency in this case study?
- What other physical agent modalities could be utilized to treat this injury or condition? Why? How?
- Is electrical stimulation the most effective means to control the swelling in this patient's ankle? What approach might be more or equally effective?
- How will the swelling affect the healing of the injured tissues?
- How will the swelling affect the ability of the patient to perform therapeutic exercise?
- What are the physiologic mechanisms for the swelling? For the resolution of the swelling?
- Why is the term "swelling" used in lieu of "edema" or "effusion"?

The rehabilitation professional employs physical agent modalities to create an optimum environment for tissue healing while minimizing the symptoms associated with the trauma or condition.

Nikolova¹⁵² has used IFC for a variety of clinical problems and found it effective in dealing with pain problems (e.g., joint sprains with swelling, restricted mobility and pain, neuritis, retarded callus formation following fractures, pseudarthrosis).¹⁰³ These claims are supported by other researchers. Each of these researchers used slightly different protocols in treating the different clinical problems. To be successful in achieving the desired results with IFC, the clinician must thoroughly review existing protocols and acquire a good working knowledge of the application techniques.

Clinical Decision-Making Exercise 5–9

When using IFC to treat muscle guarding in the low back, how should the electrodes be placed?

Premodulated Interferential Current

In recent years, a second method of creating the interference effect has been developed, which is referred to as premodulated interferential. Premodulated IFC is available on most of the newer electrical stimulating units. In the premodulated setting, both generators of the unit output a frequency of 4000 Hz. However, each generator has the ability to premodulate or burst the frequency within the unit.¹⁵³ The unit has the capability of perfectly synchronizing these bursts in the same polarity, at the same time to create premodulated interferential.¹⁵⁵

Units that are capable of premodulation are not necessarily premodulated interferential. They may only provide premodulation for the purpose of bipolar (two electrodes) stimulation. While both create the interferential effect, there may be some advantages to the premodulated technique.¹⁵⁵

The true interferential provides an uninterrupted, constant 4000-Hz frequency to the tissue. This will create a numbness beneath the electrodes that the patient will perceive as a reduction in the intensity of the current. With premodulated interferential, however, since the current is being burst inside the unit itself, numbness does not occur and a larger treatment area is established with the actual therapeutic frequency.¹⁵⁵

Low-Volt Currents**Medical Galvanism**

The application of continuous low-voltage monophasic current causes several physiologic changes that can be used therapeutically. The therapeutic benefits are related to the polar and vasomotor effects and to the acid reaction around the positive pole and the alkaline reaction at the negative pole. The clinician must be concerned with the damaging effects of this variety of current. Acidic or alkaline changes can cause severe skin reactions.² These reactions occur only with low-voltage continuous DC and are not likely with the high-voltage pulsed generators. The pulse duration of the high-volt pulsatile generators is too short to cause these chemical changes.³¹

Low-volt currents also have a vasomotor effect on the skin, increasing blood flow between the electrodes. The benefits from this type of DC are usually attributed to the increased blood flow through the treatment area.²

Iontophoresis

DC has been used for many years to transport ions from the heavy metals into and through the skin for treatment of skin infections or for a counterirritating effect. Iontophoresis is discussed in detail in [Chapter 6](#).

Treatment Precautions with Continuous Monophasic Currents

Skin burns are the greatest hazard of any continuous monophasic current technique. These burns result from excessive electrical density in any area, usually from direct metal contact with skin or from setting the intensity too high for the size of the active electrode. Both these problems cause a very high density of current in the area of contact.^{25,114}

Treatment Protocols for Low-volt Current are as Follows:

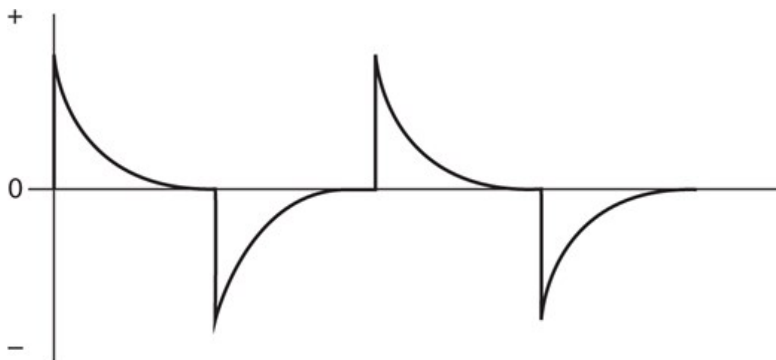
1. Current intensity should be to the patient's tolerance; it should be increased as accommodation takes place. These intensities are in the milliamperage range.
2. Continuous monophasic current should be used.
3. Pulses per second should be 0.
4. A low-voltage monophasic current stimulator is the machine of choice.
5. Treatment time should be between a 15-minute minimum and a 50-minute maximum.
6. Equal-sized electrodes are used over gauze that has been soaked in saline solution and lightly squeezed.
7. Skin should be unbroken.^{25,114,142}

H-Wave Stimulation

The H-Wave® is an electrical stimulating device which generates current that has been used for treating lymphedema, swelling, and pain. It is designed to specifically and directly stimulate the small smooth-muscle fibers within lymphatic vessels to enhance lymphatic flow and allow for improved metabolic waste removal that leads to fluid shifts, edema reduction, and thus improved tissue oxygenation.²⁰⁰ The H-wave is a bipolar exponentially decaying waveform with low pulse amplitude (<10mA) long-duration (fixed at 16 ms), and low-frequency (2–60 Hz) designed to stimulate a muscle contraction and modulate pain (Figure 5–35). The findings from a meta-analysis indicate a moderate to strong effect of the H-Wave® device in providing pain relief.²⁰⁰

Figure 5–35.

H-WAVE bipolar exponentially decaying waveform.



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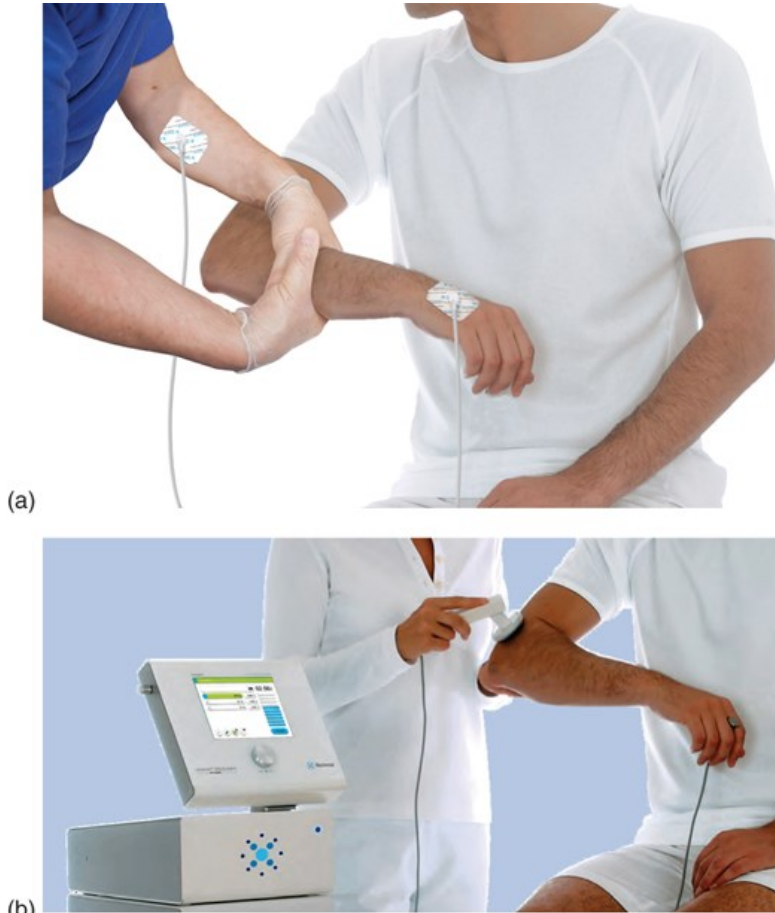
DEEP OSCILLATION THERAPY (HIVAMAT)

The technique of deep oscillation® therapy originated in Germany in 2007. A Hivamat®200 [Histological (HI) Variable (VA) Manual (MA) Technique(T)] is a device that produces an electrical alternating (AC) current at a frequency that ranges from 5 to 200 Hz. This biphasic current creates an electrostatic field in the tissues. With the original Hivamat® 200 unit, both the clinician and patient are connected to the unit; the clinician by an electrode attached to their forearm, and the patient by a hand-held metal conducting electrode. The clinician wears vinyl, non-conductive gloves which serve as an insulating medium to the hands that become one of the electrodes. The optimum therapeutic effect is on dry skin only and the application of powder to the treatment area facilitates this. The hands move over the skin to providing a deep tissue massage to the underlying tissues (Figure 5–36a). The

therapeutic effect is based on the electrostatic field set up between the clinician's hands and the patient's skin. Since the current is alternating, the polarity changes rapidly between the two electrodes. When the clinician's electrode (the hands) is negative, positive ions are attracted to that electrode. When the electrode becomes positive, the positive ions are repelled thus creating an oscillation of charged ions in the tissue. As the clinician's hands move over the skin, this oscillation causes a rhythmical deformation or vibration of the tissues being treated. The newer Hivamat® 200 Evident still has a hand-held titanium patient electrode, but an applicator (similar to an ultrasound applicator) has replaced the hands as the second electrode (Figure 5–36b).

Figure 5–36.

HIVAMAT. (a) Originally, deep oscillation therapy used the hands as electrodes. (b) The Hivamat® 200 Evident uses an applicator electrode to replace the hands. (Courtesy Richmar)



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Deep oscillation therapy has been shown to improve wound healing, inflammation, pain, and to facilitate resorption of lymphedema.²⁰¹ It has been indicated for use in musculoskeletal injuries but few studies on its effectiveness exist.²⁰¹ The mechanism by which deep oscillation therapy achieves results has been attributed to mobilization of interstitial fluids that enhances lymphatic drainage thus reducing inflammation.²⁰² Additionally, stimulation of mechanoreceptor cells has been attributed to a reduction of pain.²⁰²

For acute injuries, initial treatment should use high frequencies (80–200 Hz) for a surface effect and gentle vibration, medium frequencies (25–80 Hz) for more intense vibration and treating pain, and low frequencies (2–25 Hz) to provide a deep vibration for treating lymphedema.

BONE GROWTH STIMULATORS

Generally, bone fractures heal normally with standard care. Occasionally, the healing process stops due to added risks or complications. *Delayed*

union refers to a decelerating bone healing process. *Nonunion* is considered to be established when the fracture site shows no visibly progressive signs of healing, without giving any guidance regarding the time frame. A reasonable time period for lack of visible signs of healing is 3 months. It has been shown that electrical current can stimulate bone growth and enhance the healing process.¹⁵⁴

Several electrical bone growth stimulators are available. These stimulators attempt to produce electromagnetic fields similar to those that normally exist in bone. The *noninvasive type* of stimulator is comprised of coils or electrodes, placed on the skin near the fracture site. Noninvasive bone growth stimulators generate a weak biphasic electrical current within the target site using small electrodes placed on either side of the fracture.¹⁵⁵ These are worn for 24 hours per day until healing occurs or up to 9 months. A second type of noninvasive bone growth stimulator uses pulsed electromagnetic fields delivered via treatment coils placed directly onto the skin and are worn for 6–8 hours per day for 3–6 months. There is also a noninvasive stimulator that uses ultrasound.

The *invasive type* of stimulator includes percutaneous and implanted devices. The percutaneous type involves electrode wires inserted through the skin into the bone while implanted devices include a generator placed under the skin or in the muscles near the fracture site. The implanted devices are surgically placed and later surgically removed. Invasive devices use DC.¹⁵⁵ The implantable device typically remains functional for 6–9 months after implantation. Although the current generator is removed in a second surgical procedure when stimulation is completed, the electrode may or may not be removed. Invasive bone growth stimulation is used only in spinal fusion surgery, and is not used in the appendicular skeleton.

FUNCTIONAL ELECTRICAL STIMULATION

Since the mid-1980s researchers have experimented with using computer-controlled electrical currents that stimulate the peripheral nervous system for the purpose of providing dynamic assistance in functional activities, such as walking or upper extremity function.¹⁵⁶ Used primarily in patients who have sustained spinal cord injury or suffered a stroke, **FES** utilizes multiple-channel electrical stimulators controlled by a microprocessor to recruit muscles in a programmed synergistic sequence that will allow the patient to accomplish a specific functional movement pattern.^{113,156} Even though this technique has been used effectively in short-term management of a variety of dysfunctions, there are many practical considerations for use that might impede or limit the long-term independent usefulness of FES by a patient.¹⁵⁷

Currently, the majority of FES programs are limited to the use of surface electrodes that are difficult to adhere to the skin and to maintain positioning at the appropriate stimulation point.¹⁵⁸ For FES to be useful to the patient on a daily basis, the electrodes, and possibly the stimulator itself, will need to be implanted directly into the muscle or on a nerve.¹⁵⁹ Research is ongoing toward this end, but to date no acceptable system has been developed.

The existing computer control systems for FES also need to be refined if they are to be both useful and safe for the patient. Either the control systems must use a preset activation sequence that will allow the patient to execute a specific task or there must be some type of feedback from the stimulated neuromuscular systems so that the computer can make the appropriate movement corrections to ensure the safety of the patient. The development of a “closed-loop” feedback control system that would allow the computer to compensate for uneven terrain or to adjust the speed and frequency of movement presents a major challenge to researchers working in this area.¹⁶⁰

Although multichannel microprocessors may be preprogrammed to execute a variety of specific movement patterns, how those programs will be activated presents another obstacle for development of FES systems. Foot switches or crutch switches may potentially be used to trigger a desired response, although there are limitations to the number of switches that a spinal cord or stroke patient would actually be able to use.¹¹³ Some of the upper extremity control devices have used movements of the contralateral shoulder to trigger a response. Verbal commands recognized by the computer also have been used to control stimulation of muscle in various functional tasks.¹⁵⁷

Presently long-term independent use of FES is practical for only a few problems.^{157,176} Certainly as new technologies continue to become available, ongoing clinical research will make FES increasingly practical for various patient populations. The future of FES holds many exciting possibilities for patients and clinicians alike.

Clinical Uses of FES

FES has a number of clinical applications.¹⁶¹ Initially, FES was used for stroke patients with a foot drop to assist dorsiflexion. Subsequently, it was

found to be more useful in treating patients with incomplete spinal cord injury who have good stance stability but are unable to achieve adequate flexion during the swing phase of gait.¹⁵⁷

FES has been used with some success, enabling patients to stand, transfer, ambulate on level surfaces, and even ascend stairs on a limited basis using a walker or crutches in a closely supervised environment.^{107,162–166} Spinal cord patients have used computer-controlled FES to allow them to exercise on bicycle ergometers to improve cardiorespiratory endurance and fitness.^{167,168}

Control of muscles in the upper extremity using multiple-channel stimulation has allowed paraplegic patients to use the muscles of the hand and forearm of the paralyzed limb in functional grasp patterns. FES has also been used effectively in managing shoulder subluxation in the hemiplegic patient.¹⁵⁷

TRANSCRANIAL ELECTRICAL STIMULATION

Non-invasive stimulation of the brain using electrical currents is currently being used for the treatment of chronic pain. Currently, four different types of treatments have been used to stimulate the brain: transcranial direct current stimulation (tDCS), cranial electrotherapy stimulation (CES), repetitive transcranial magnetic stimulation (rTMS), and reduced impedance non-invasive cortical electrostimulation (RINCE). With each of these treatments, the intended outcome is to reduce pain by altering activity in the areas of the brain that are involved in pain processing.¹⁹⁶

Transcranial direct current stimulation (tDCS) uses large electrodes applied to the scalp directly over the targeted brain area to deliver a continuous direct current of <2mA to the cortex. CES is pulsed low intensity (<2mA) electrical current applied using electrodes clipped to the patient's earlobes. Like tDCS, RINCE uses scalp electrodes at specific stimulation frequencies that allow deeper cortical penetration and modulation of lower-frequency cortical activity. Repetitive transcranial magnetic stimulation (rTMS) stimulates the cerebral cortex by using a coil applied to the scalp that induces an electrical current using rapidly changing magnetic fields.¹⁹⁶

A recent meta-analysis has suggested there is low or very low-quality evidence that tDCS, rTMS, CES, and RINCE are not effective for treating chronic pain and thus cannot be recommended in routine clinical care.¹⁹⁶

PLACEBO EFFECT OF ELECTRICAL STIMULATION

There is a major placebo effect in all that we do in providing any therapy to our patients. This placebo effect is a basic and extremely important tool to help us achieve the best results. Our attitude toward the patients and our presentation of the therapy to them are crucial. When the clinician demonstrates a sincere interest in the patient's problems, the patient uses that interest to add to his or her own conviction and motivation to get well.

This perceptual change is influenced by many factors at the cognitive and affective levels. When these factors are active, real physiologic changes occur that assist in the healing process. The clinician should not intentionally deceive the patient with a sham treatment but should use the treatment to have the best impact on the patient's perception of the problem and the treatment's effectiveness.

The treatment will work better if the patient has a profound belief in its ability to alleviate the problem. To gain the most from this effect, the patient needs to be intimately involved with the treatment. We must educate, encourage, and empower the patient to get better. Giving the patient the knowledge and ability to feel some control and to be self-determined in healing reduces the stress of injury and enhances the patient's recovery powers. In stressful situations any measure of control lessens the extent of the stress and results in the improvement of disease resistance or injury recovery factors that will improve treatment outcomes.²⁶

WHAT DOES THE MOST RECENT BEST-AVAILABLE EVIDENCE SAY ABOUT THE EFFECTIVENESS OF ELECTRICAL STIMULATING CURRENTS AS A CLINICAL TREATMENT MODALITY?

The following statements are direct quotes from the most recent randomized controlled trials and systematic reviews found in *the Cochrane Database of Systematic Reviews* and in *PubMed* that focus on the efficacy of electrical stimulating currents as a therapeutic technique.

- “Treatment of chronic low back pain with TENS demonstrated significant pain reduction. The application of TENS may lead to less pain medication usage and should be incorporated into the treatment armamentarium for chronic low back pain.”¹⁸⁹
- “TENS reduces pain intensity over and above that seen with placebo (no current) TENS when administered as a stand-alone treatment for acute pain in adults.”¹⁹⁰
- “We could not confirm that transcutaneous electrostimulation is effective for pain relief. The current systematic review is inconclusive, hampered by the inclusion of only small trials of questionable quality.”¹⁹¹
- “TENS and acupuncture-like TENS are shown to be effective in pain control over placebo in this review.”¹⁹²
- “Patients treated with electrical stimulation as an adjunct for bone healing have significantly less pain and experience lower rates of radiographic nonunion or persistent nonunion. No difference was seen with regards to functional outcomes in a limited number of trials.”¹⁹³
- “This review of the literature suggests that regardless of the waveform or parameters used, current evidence does not support the use of electrical stimulation for reducing edema, decreasing pain, or improving function following acute lateral ankle sprain more effectively than no ES.”¹⁹⁴
- “Evidence from the meta-analysis suggests that functional electrical stimulation is beneficial in improving aspects of everyday activity performance after stroke.”¹⁹⁵
- “The available evidence suggests that low-frequency rTMS, rTMS applied to the pre-frontal cortex, CES and tDCS are not effective in the treatment of chronic pain.”¹⁹⁶
- “We cannot make any definite statements on the efficacy and clinical usefulness of electrotherapy modalities for neck pain. Since the evidence is of low or very low quality, we are uncertain about the estimate of the effect.”¹⁹⁷
- “Electroacupuncture treatment can relieve the pain of osteoarthritis of the knees and improve comprehensive aspects of knee osteoarthritis and the quality of life of patients with knee osteoarthritis.”¹⁹⁸
- “Interferential current as a supplement to another intervention seems to be more effective for reducing pain than a control treatment at discharge and more effective than a placebo treatment at the 3-month follow-up. However, it is unknown whether the analgesic effect of IFC is superior to that of the concomitant interventions.”¹⁹⁹

SAFETY IN THE USE OF ELECTRICAL EQUIPMENT

Electrical safety in the clinical setting should be of maximal concern to the professional clinician. Too often there are reports of patients being electrocuted as a result of faulty electrical circuits in whirlpools. This type of accident can be avoided by taking some basic precautions and acquiring an understanding of the power distribution system and electrical grounds.¹⁶⁹

The typical electrical circuit consists of a source producing electrical power, a conductor that carries the power to a resistor or series of driven elements, and a conductor that carries the power back to the power source.

Electrical power is carried from generating plants through high-tension power lines carrying 2200 V. The power is decreased by a transformer and is supplied in the wall outlet at 220 or 120 V with a frequency of 60 Hz. The voltage at the outlet is AC, which means that one of the poles, the “hot” or “live” wire, is either positive or negative with respect to other neutral lines. Theoretically, the voltage of the neutral pole should be zero. Actually, the voltage of the neutral line is about 10 V. Thus, both hot and neutral lines carry some voltage with respect to the earth, which has zero voltage. The voltage from either of these two leads may be sufficient to cause physiologic damage.

The two-pronged plug has only two leads, both of which carry some voltage. Consequently, the electrical device has no true **ground**. The term *true ground* literally means the electrical circuit is connected to the earth or the ground, which has the ability to accept large electrical charges without becoming charged itself. The ground will continually accept these charges until the electrical potential has been neutralized. Therefore, any electrical

charge that may be potentially hazardous (i.e., any electricity escaping from the circuit) is almost immediately neutralized by the ground. If an individual were to come in contact with a short-circuited instrument that was not grounded, the electrical current would flow through that individual to reach the ground.

Electrical devices that have two-pronged plugs generally rely on the chassis or casing of the power source to act as a ground, but this is not a true ground. Therefore, if an individual were to touch the casing of the instrument while in contact with some object or instrument that has a true ground, an electrical shock may result. With three-pronged plugs, the third prong is grounded directly to the earth and all excess electrical energy theoretically should therefore be neutralized.

By far the most common mechanism of injury from therapeutic devices results when there is some damage, breakdown, or short circuit to the power cord. When this happens, the casing of the machine becomes electrically charged. In other words, there is a voltage leak, and in a device that is not properly grounded electrical shock may occur (Figure 5-37).

Figure 5-37.

When a therapeutic device is not properly grounded, there is danger of electrical shock. This is a major problem in a whirlpool.



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The magnitude of the electrical shock is a critical factor in terms of potential health danger (Table 5-3). Shock from electrical currents flowing at ≤ 1 mA will not be felt and is referred to as **microshock**. Shock from a current flow greater than 1 mA is called **macroshock**. Currents that range between 1 and 15 mA produce a tingling sensation or perhaps some muscle contraction. Currents flowing at 15–100 mA cause a painful electrical shock. Currents between 100 and 200 mA may result in fibrillation of cardiac muscle or respiratory arrest. When current flow is above 200 mA, rapid burning and destruction of tissue occur.¹⁷⁰

Table 5–3

Physiologic Effects of Electrical Shock at Varying Magnitudes

INTENSITY (mA)	PHYSIOLOGIC EFFECTS
0–1	Imperceptible
2–15	Tingling sensation and muscle contraction
16–100	Painful electrical shock
101–200	Cardiac or respiratory arrest
>200	Instant tissue burning and destruction

Most electrotherapeutic devices (e.g., muscle stimulators, ultrasound, and the diathermies) are generally used in dry environments. All new electrotherapeutic equipment being produced has three-pronged plugs and is thus grounded to the earth. However, in a wet or damp area the three-pronged plug may not provide sufficient protection from electrical shock. We know that the body will readily conduct electricity because of its high water content. If the body is wet or if an individual is standing in water, the resistance to electrical flow is reduced even more. Thus, if a short should occur, the shock could be as much as five times greater in this damp or wet environment. The potential danger that exists with whirlpools or tubs is obvious. The ground on the whirlpool will supposedly conduct all current leakage from a faulty motor or power cord to the earth. However, an individual in a whirlpool is actually a part of that circuit and is subject to the same current levels as any other component of the circuit. Small amounts of current therefore can be potentially harmful, no matter how well the apparatus is grounded. For this reason in 1981 the National Electrical Code required that all health care facilities using whirlpools and tubs install **ground-fault interruptors (GFI)** (Figure 5–38). These devices constantly compare the amount of electricity flowing from the wall outlet with the whirlpool turbine with the amount returning to the outlet. If any leakage in current flow is detected, the ground-fault circuit breaker will automatically interrupt current flow in as little as one fortieth of a second, thus shutting off current flow and reducing the chances of electrical shock.¹⁷¹ These devices may be installed either in the electrical outlet or in the circuit breaker box.

Figure 5–38.

A typical ground-fault interrupter (GFI). Originally published in *The Family Handyman*. Copyright © 2007 by RDA Enthusiast Brands, a division of Trusted Media Brands, Inc. Used by permission. All rights reserved.



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Regardless of the type of electrotherapeutic device being used and the type of environment, the following safety practices should be considered.

1. The entire electrical system of the building or training room should be designed or evaluated by a qualified electrician. Problems with the electrical system may exist in older buildings or in situations where rooms have been modified to accommodate therapeutic devices (e.g., putting a whirlpool in a locker room where the concrete floor is always wet or damp).
2. It should not be assumed that all three-pronged wall outlets are automatically grounded to the earth. The ground must be checked.
3. The clinician should become very familiar with the equipment being used and any potential problems that may exist or develop. Any defective equipment should be removed from the clinic immediately.
4. The plug should not be jerked out of the wall by pulling on the cable.
5. Extension cords or multiple adaptors should never be used.
6. Equipment should be reevaluated on a yearly basis and should conform to National Electrical Code guidelines. If a clinic or athletic training room is not in compliance with this code, then there is no legal protection in a lawsuit.
7. Common sense should always be exercised when using electrotherapeutic devices. A situation that appears to be potentially dangerous may in fact result in injury or death.

Clinical Decision-Making Exercise 5–10

When installing a whirlpool in the hydrotherapy area, the clinician must always be concerned about the possibility of electrical shock. What measures can be taken to reduce the possibility of electrical shock?

SUMMARY

1. Electrons move along a conducting medium as an electrical current.
2. A volt is the electromotive force that produces a movement of electrons; an ampere is a unit of measurement that indicates the rate at which electrical current is flowing.
3. Ohm's law expresses the relationship between current flow voltage and resistance. The current flow is directly proportional to the voltage and inversely proportional to the resistance.
4. Electrotherapeutic devices generate three different types of current, direct (DC), alternating (AC), and pulsatile (PC), which are capable of producing specific physiologic changes when introduced into biologic tissue.
5. Confusion exists relative to the terminology used to describe electrotherapeutic currents, but all therapeutic electrical generators are transcutaneous electrical stimulators, regardless of whether they deliver AC, DC, or PC through electrodes attached to the skin.
6. The term *pulse* is synonymous with *waveform*, which indicates a graphic representation of the shape, direction, amplitude, duration, and pulse frequency of the electrical current the electrotherapeutic device produces, as displayed by an instrument called an oscilloscope.
7. Modulation refers to any alteration in the magnitude or any variation in duration of a pulse (or pulses) and may be continuous, interrupted, burst, or ramped.
8. The main difference between a series and a parallel circuit is that in a series circuit there is a single pathway for current to get from one terminal to another, and in a parallel circuit two or more routes exist for current to pass.
9. The electrical circuit that exists when electron flow is through human tissue is in reality a combination of both a series and a parallel circuit.
10. The effects of electrical current moving through biologic tissue may be chemical, thermal, or physiologic.
11. When an electrical system is applied to muscle or nerve tissue, the result will be tissue membrane depolarization, provided that the current has the appropriate intensity, duration, and waveform to reach the tissue's excitability threshold.
12. Muscle and nerve tissue respond in an all-or-none fashion; there is no gradation of response.
13. Muscle contraction will change according to changes in current. As the frequency of the electrical stimulus increases, the muscle will develop more tension as a result of the summation of the contraction of the muscle fiber through progressive mechanical shortening. Increases in intensity spread the current over a larger area and increase the number of motor units activated by the current. Increases in the duration of the current also will cause more motor units to be activated.
14. Nonexcitatory cells and tissues respond to subsensory electrical currents that can alter how the cell functions following injury.
15. The newest electrical stimulating units are capable of producing multiple types of current including high volt, TENS, microcurrent, Russian, interferential, premodulated interferential, low volt, and H-wave.
16. Electrically stimulating muscle contractions using primarily high-volt current are used clinically to help with muscle reeducation, muscle contraction for muscle pumping action, reduction of swelling, prevention or retardation of atrophy, and muscle strengthening.

17. TENS applications are generally used for stimulating sensory nerve fibers and modulating pain. TENS' current parameters can be modified to modulate pain through gate control, descending mechanisms, and endogenous opiate mechanisms of pain control.
18. Microcurrent uses subsensory electrical currents primarily to achieve biostimulative effects in healing of bone and soft tissues.
19. Russian current delivers a medium-frequency biphasic waveform and is used primarily for muscle strengthening.
20. Interferential and premodulated IFC rely on the combined effects of currents produced from two separate generators and are used primarily for pain management.
21. Low-volt currents are continuous monophasic current. Their primary use involves polar effects (acid or alkaline), increased blood flow, bacteriostatic effects (through the negative electrode), and migration and alignment of cellular building blocks in the healing processes.
22. Bone growth stimulation, functional electrical stimulation, and transcranial electrical stimulation are more recent uses for electrical stimulating currents.
23. Electrical safety is critical when using electrotherapeutic devices. It is the responsibility of the clinician to make sure that all electrical modalities conform to the National Electrical Code.

REVIEW QUESTIONS

1. How are the following electrical terms defined: *potential difference*, *ampere*, *volt*, *ohm*, and *watt*?
2. What is the mathematical expression of Ohm's law and what does it represent?
3. What are the three different types of electrical current?
4. What is a transcutaneous electrical stimulator and how is it related to a TENS unit?
5. What are the different types of waveforms that electrical stimulating generators may produce?
6. What are the various pulse characteristics of the different waveforms?
7. How can electrical currents be modulated?
8. What are the differences between series and parallel circuits?
9. How does electrical current travel through various types of biologic tissue?
10. What physiologic responses can be elicited by using electrical stimulating currents?
11. Explain the concept of depolarization of muscle and nerve in response to electrical stimulation.
12. What do the strength-duration curves represent?
13. How should electrical stimulating currents be used with denervated muscle?
14. What are the effects of electrically stimulating nonexcitatory cells and tissues?
15. What treatment parameters must be considered when setting up a treatment using electrical stimulating currents?
16. What are the various therapeutic uses of electrically stimulated muscle contractions?
17. How can electrical stimulating currents be used to modulate pain?
18. What are the clinical applications for using low-voltage DC?

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19. What are the various physiologic effects of using microcurrent?
20. Are there advantages to using IFC as opposed to premodulated IFC or other types of electrical stimulating currents?
21. What steps can the clinician take to ensure safety of the patient when using electrical modalities?

SELF-TEST QUESTIONS

True or False

1. Electrons tend to flow from areas of low concentration to areas of high concentration.
- a. True
- b. False
2. Insulators resist current flow.
- a. True
- b. False
3. The greater the voltage, the greater the amplitude is.
- a. True
- b. False
4. The cathode is the negatively charged electrode in a DC system.
- a. True
- b. False
5. *Chronaxie* refers to the minimum current intensity needed for tissue excitation if applied for a maximum time.
- a. True
- b. False
6. The electrode with the greatest current density is the active electrode.
- a. True
- b. False

Multiple Choice

7. A particle of matter with very little mass and a negative charge is a(n)
- a. ion
- b. electron
- c. neutron
- d. proton

8. What is the name of the unit measuring the force necessary to produce electron movement?

- a. ampere
- b. coulomb
- c. volt
- d. watt

9. In _____ current, electron flow constantly changes direction.

- a. alternating
- b. direct
- c. pulsatile
- d. galvanic

10. When the current increases gradually to a maximal amplitude, it is known as

- a. burst
- b. ramping
- c. modulation
- d. galvanic

11. In _____ circuits, electrons have only one path to follow.

- a. galvanic
- b. parallel
- c. resistor
- d. series

12. Physiologic response(s) to electrical current include

- a. thermal
- b. chemical
- c. physiologic
- d. all of the above

13. All whirlpools and tubs in a health care setting must have

- a. GFI
- b. a three-pronged outlet
- c. an insulated cord
- d. a waterproof motor

- 14.** During the absolute refractory period the cell is not capable of
 - a.** depolarization
 - b.** an action potential
 - c.** twitch muscle contraction
 - d.** all of the above
- 15.** The part of the cell responsible for transmitting messages to other cells via ionic, electrical, or small molecule signals is the
 - a.** electret
 - b.** gap junction
 - c.** dipole
 - d.** cell membrane pump
- 16.** To _____ current density in deeper tissue, the electrodes must be placed _____.
 - a.** increase, closer
 - b.** increase, further apart
 - c.** decrease, closer
 - d.** decrease, further apart
- 17.** Electrical stimulation may release enkephalin and endorphin to cause pain relief. What is the name of this pain control method?
 - a.** gate control theory
 - b.** central biasing theory
 - c.** opiate pain control theory
 - d.** placebo effects
- 18.** Two currents combine and the amplitude decreases. This is called
 - a.** destructive interference
 - b.** constructive interference
 - c.** heterodyne current
 - d.** beat current
- 19.** Which of the following currents is a pulsatile biphasic wave, generated in bursts, designed to create muscle contraction?
 - a.** LIS
 - b.** iontophoresis
 - c.** IFC

d. Russian

20. Increased blood flow between electrodes is an effect of which of the following?

a. IFC

b. function electrical stimulation

c. LIS

d. medical galvanism

SOLUTIONS TO CLINICAL DECISION-MAKING EXERCISES

5-1

The terms *TENS* and *NMES* are for all intents and purposes interchangeable in their physiologic effects. Both units can be used to stimulate peripheral motor or sensory nerves.

5-2

The clinician should make it perfectly clear that even though the generator is producing a high-voltage current, the amperage is very small in the milliamp range and thus the total amount of electrical energy being output to the patient is very small. It is important to explain exactly what the patient will feel, especially if this is the first time that he or she has experienced electrical stimulation.

5-3

The clinician can simply increase current intensity sufficiently to produce a muscle contraction and then adjust the frequency to approximately 50 pulses/s. This will produce a tetanic contraction regardless of whether biphasic, monophasic, or PC is being used.

5-4

The current density under the active electrode could be increased by using a smaller electrode. The current intensity, the current duration, or a combination of the two may be increased to cause a depolarization.

5-5

The size of the active electrode can be decreased, which will increase current density under that electrode. The active electrodes can be moved further apart. The current intensity can be increased, and the current duration may also be increased.

5-6

A medium-frequency AC stimulator should be used. Frequency should be set at 20–30 Hz using an interrupted or surge modulation. On time should be set at about 20 seconds with off time also set at 20 seconds. On most generators of this type, pulse duration is preset. Intensity should be increased to elicit a strong muscle contraction that moves the lower leg through its antigravity range. The patient should be instructed to simultaneously produce a voluntary muscle contraction.

5-7

In treating both trigger points and acupuncture points, the clinician should use a direct current with the frequency set between 1 and 5 Hz, and pulse duration between 100 and 1000 microseconds. Intensity should be increased to the point where there is a muscle contraction, and then increased further until it is somewhat painful. The point should be stimulated for 45 seconds.

5-8

In a conventional TENS treatment, the goal is to provide as much sensory cutaneous input as possible. Thus, both the frequency and the pulse

duration should be set as high as the unit will allow. The intensity should be increased until a muscle contraction is elicited, and then decreased slightly until the patient feels only a tingling sensation. If using a portable unit, the treatment may continue for several hours if necessary or until the pain subsides.

5-9

The four electrodes should be set up in a square pattern with the target muscle in the center of the square so that the maximum interference will take place where the electric field lines cross at the center of the pattern.

5-10

The four electrodes should be set up in a square pattern with the target muscle in the center of the square so that the maximum interference will take place where the electric field lines cross at the center of the pattern.

5-11

The National Electrical Code requires that all whirlpools have GFI installed to automatically shut off current flow. In addition, the clinician should not allow the patient to turn the whirlpool on and off. This is especially important when the patient is already in contact with the water. Extension cords or multiple adaptors should never be used in the hydrotherapy area.

REFERENCES

1. Licht S. *Therapeutic Electricity and Ultraviolet Radiation*. Vol IV. 2nd ed. Baltimore, MD: Waverly; 1969.
2. Watkins A. *A Manual of Electrotherapy*. 3rd ed. Philadelphia, PA: Lea & Febiger; 1968.
3. Valkenberg V. *Basic Electricity*. Clifton Park, NY: Delmar Learning; 1995.
4. Chamishion R. *Basic Medical Electronics*. Boston, MA: Little, Brown and Company; 1964.
5. Stillwell G. *Therapeutic Electricity and Ultraviolet Radiation*. 3rd ed. Baltimore, MD: Williams & Wilkins; 1983.
6. Bergueld P. *Electromedical Instrumentation: A Guide for Medical Personnel*. Cambridge: Cambridge University Press; 1980.
7. Thornton RM, Mendel FC, Fish DR. Effects of electrical stimulation on edema formation in different strains of rats. *Phys Ther*. 1998;78(4):386-394. [PubMed: 9555921]
8. Alon G, DeDomeico G. *High Voltage Stimulation: An Integrated Approach to Clinical Electrotherapy*. Chattanooga, TN: Chattanooga Corp; 1987.
9. Shriber W. *A Manual of Electrotherapy*. 4th ed. Philadelphia, PA: Lea & Febiger; 1975.
10. Alon G. Principles of electrical stimulation. In: Nelson R, Currier D, eds. *Clinical Electrotherapy*. Norwalk, CT: Appleton & Lange; 1999.
11. DeDomenico G. *Basic Guidelines for Interferential Therapy*. Sydney, Australia: Theramed; 1981.
12. Holcomb WR. A practical guide to electrical therapy. *J Sport Rehabil*. 1997;6(3):272-282.
13. Myklebust B, Robinson A. Instrumentation. In: Snyder-Mackler L, Robinson A, eds. *Clinical Electrophysiology, Electrotherapy and Electrotherapy and Electrophysiologic Testing*. Baltimore, MD: Lippincott Williams & Wilkins; 2007.
14. Robinson A. Basic concepts and terminology in electricity. In: Snyder-Mackler L, Robinson A, eds. *Clinical Electro-physiology, Electrotherapy and Electro-physiologic Testing*. Baltimore, MD: Lippincott Williams & Wilkins; 2007.

15. Carlos J. Clinical electrotherapy part I: physiology and basic concepts. *Phys Ther.* 1998;6(4):44.
16. Cohen H, Brunilik J. *Manual of Electroneuromyography*. 2nd ed. New York: Harper & Row; 1976.
17. Griffin J, Karselis T. *Physical Agents for Physical Therapists*. Springfield, IL: Charles C Thomas; 1988.
18. Taylor K, Mendel FC, Fish DR. Effect of high-voltage pulsed current and alternating current on macromolecular leakage in cheek pouch microcirculation. *Phys Ther.* 1997;77(12):1729–1740. [[PubMed: 9413451](#)]
19. Kitchen S, Bazin S. *Electrotherapy: Evidence-based Practice*. Wernersville, PA: Harcourt Health Sciences; 2001.
20. Kahn I. *Principles and Practice of Electrotherapy*. Philadelphia: Elsevier Health Sciences; 2001.
21. Wolf S. *Electrotherapy: Clinics in Physical Therapy*. Vol 2. New York: Churchill Livingstone; 1981.
22. Nalty T, Sabbahi M. *Electrotherapy Clinical Procedures Manual*. New York: McGraw-Hill; 2001.
23. McLoda TA, Carmack JA. Optimal burst duration during a facilitated quadriceps femoris contraction. *J Athletic Train.* 2000;35(2):145–150.
24. Benton L, Baker L, Bowman B. *Functional Electrical Stimulation: A Practical Clinical Guide*. Downey, CA: Rancho Los Amigos Hospital; 1981.
25. Nelson R, Currier D. *Clinical Electrotherapy*. Norwalk, CT: Appleton & Lange; 1999.
26. Howson D. *Report on Neuromuscular Reeducation*. Minneapolis, MN: Medical General; 1978.
27. Becker R, Selden G. *The Body Electric*. New York: Harper Collins; 1998.
28. Maurer C. The effectiveness of microelectrical neural stimulation on exercise-induced muscle trauma [abstract R200]. *Phys Ther.* 1992;725:574.
29. Randall B, Imig C, Hines HM. Effect of electrical stimulation upon blood flow and temperature of skeletal muscles. *Arch Phys Med.* 1952;33:73–78.
30. Gault W, Gatens P. Use of low-intensity direct current in management of ischemic skin ulcers. *Phys Ther.* 1976;56:265–269. [[PubMed: 1083031](#)]
31. Newton R, Karselis T. Skin pH following high voltage pulsed galvanic stimulation. *Phys Ther.* 1983;63:1593–1596. [[PubMed: 6604927](#)]
32. Guyton A. *Textbook of Medical Physiology*. Philadelphia, PA, PA: WB Saunders; 2005.
33. Kincaid C, Lavoie K. Inhibition of bacterial growth in vitro following stimulation with high voltage monophasic pulsed current. *Phys Ther.* 1989;69:651–655. [[PubMed: 2501804](#)]
34. Delitto A. A study of discomfort with electrical stimulation. *Phys Ther.* 1992;72:410–424. [[PubMed: 1589461](#)]
35. Melzack R. Prolonged relief of pain by brief, intense transcutaneous electrical stimulation. *Pain.* 1975;1(4):357–373. [[PubMed: 141644](#)]
36. Mohr T, Akers T, Landry R. Effect of high voltage stimulation on edema reduction in the rat hind limb. *Phys Ther.* 1987;67:1703–1707. [[PubMed: 3499622](#)]
37. Reed B. Effect of high voltage pulsed electrical stimulation on microvascular permeability to plasma proteins: a possible mechanism in minimizing edema. *Phys Ther.* 1988;68:491–495. [[PubMed: 2451258](#)]
38. Alon G. High voltage stimulation: effects of electrode size on basic excitatory responses. *Phys Ther.* 1985;65:890. [[PubMed: 3873661](#)]

39. Travell J, Simon D. *Myofascial Pain and Dysfunction: The Trigger Point Manual*. Baltimore, MD: Williams & Wilkins; 1998.
40. Lampe G. A clinical approach to transcutaneous electrical nerve stimulation in the treatment of chronic and acute pain, Minneapolis, July 1978. *Conference presentation*.
41. Wolf S. *Electrotherapy*. New York: Churchill Livingstone; 1981.
42. Lampe G. Introduction to the use of transcutaneous electrical nerve stimulation devices. *Phys Ther*. 1978;58:1450–1454. [[PubMed: 217028](#)]
43. Charman R. Bioelectricity and electrotherapy—towards a new paradigm? Part 1, the cell. *Physiotherapy*. 1990;76:452–491; Charman R. Part 2, cellular reception and emission of electromagnetic signals. *Physiotherapy*. 1990;76:502–518; Charman R. Part 3, bioelectric potentials and tissue currents. *Physiotherapy*. 1990;76:643–654; Charman R. Part 4, strain generated potentials in bone and connective tissue. *Physiotherapy*. 1990;76:725–730; Charman R. Part 5, exogenous currents and fields—experimental and clinical applications. *Physiotherapy*. 1990;76:743–750.
44. Selkowitz D. High frequency electrical stimulation in muscle strengthening. *Am J Sport Med*. 198;17:103–111.
45. Charman R. Bioelectricity and electrotherapy—towards a new paradigm. Part 6, environmental current and fields—the natural background. *Physiotherapy*. 1991;77:8–13; Charman R. Part 7, environmental currents and fields—man made. *Physiotherapy*. 1991;77:129–140; Charman R. Part 8, grounds for a new paradigm? *Physiotherapy*. 1991;77:211–221.
46. Brighton C. Bioelectric effects on bone and cartilage. *Clin Orthop*. 1977;124:2–4.
47. Clements F. Effect of motor neuromuscular electrical stimulation on microvascular perfusion of stimulated rat skeletal muscle. *Phys Ther*. 1991;71:397–406. [[PubMed: 2027896](#)]
48. Castel J. *Pain Management with Acupuncture and Transcutaneous Electrical Nerve Stimulation Techniques and Photo Simulation (Laser)*. *Symposium on Pain Management, Walter Reed Army Medical Center*; November 13, 1982.
49. Becker R. The bioelectric factors in amphibian-limb regeneration. *J Bone Joint Surg (Am)*. 1961;43-A:643–656. [[PubMed: 14448529](#)]
50. Lomo T, Slater C. Control of [acetylcholine](#) sensitivity and synapse formation by muscle activity. *J Physiol*. 1978;275:391. [[PubMed: 204772](#)]
51. Clemente F, Barron K. Transcutaneous neuromuscular electrical stimulation effect on the degree of microvascular perfusion in autonomically denervated rat skeletal muscle, *Arch Phys Med Rehabil*. 1996;77(2):155–160. [[PubMed: 8607739](#)]
52. Cummings J. Electrical stimulation of denervated muscle. In: Gersch M, ed. *Electrotherapy in Rehabilitation*. Philadelphia, PA: FA Davis; 1992.
53. Chu C. Weak direct current accelerates split-thickness graft healing on tangentially excised second-degree burns. *J Burn Care Rehabil*. 1991;12:285–1293. [[PubMed: 1939298](#)]
54. Gersch MR. *Electrotherapy in Rehabilitation*. Philadelphia, PA: FA Davis; 2000.
55. Gorgey A, Dudley G. The role of pulse duration and stimulation duration in maximizing the normalized torque during neuromuscular electrical stimulation. *J Orthop Sports Phys Ther*. 2008;38(8):508.
56. Litke D, Dahners L. Effect of different levels of direct current on early ligament healing in a rat model. *J Orthop Relat Res*. 1994;12:683–688.
57. Schimrigk K, McLaughlen J, Gruniger W. The effect of electrical stimulation on the experimentally denervated rat muscle. *Scand J Rehabil Med*. 1977;9:55. [[PubMed: 302482](#)]
58. *Instruction Manual for Electrostim*. Promatek: Canada; 1989:180–182.

59. Kosman A, Osborne S, Ivey A. Comparative effectiveness of various electrical currents in preventing muscle atrophy in rat. *Arch Phys Med Rehabil.* 1947;28:7. [PubMed: 20281416]
60. Thom H. Treatment of paralysis with exponentially progressive current. *Br J Phys Med.* 1957;20:49. [PubMed: 13413165]
61. Binder-MacLeod S, Snyder-Mackler L. Muscle fatigue: clinical implications for fatigue assessment and neuromuscular electrical stimulation. *Phys Ther.* 1993;73:902–910. [PubMed: 8248298]
62. Dallmann S. Preference for low versus medium frequency electrical stimulation at constant induced muscle forces [abstract R345]. *Phys Ther.* 1992;725:5107.
63. Unger P. A randomized clinical trial of the effects of HVPC on wound healing [abstract R294]. *Phys Ther.* 1991;715:5118.
64. Currier D, Lehman J, Lightfoot P. Electrical stimulation in exercise of the quadriceps femoris muscle. *Phys Ther.* 1979;59:1508–1512. [PubMed: 515187]
65. Eriksson E, Haggmark T. Comparison of isometric muscle training and electrical stimulation supplement, isometric muscle training in the recovery after major knee ligament surgery. *Am J Sports Med.* 1979;7:169–171. [PubMed: 313717]
66. DeVahl J. Neuromuscular electrical stimulation (NMES) in rehabilitation. In: Gersh M, ed. *Electrotherapy in Rehabilitation*. Philadelphia, PA: FA Davis; 1992.
67. Taylor K, Mendel F. Effect of high-voltage pulsed current and alternating current on macromolecular leakage in hamster cheek pouch microcirculation. *Phys Ther.* 1997;77(12):1729–1740. [PubMed: 9413451]
68. Mendel F, Dolan M. Effect of high-voltage pulsed current on recovery after grades I and II lateral ankle sprains. *Journal of Sport Rehabilitation.* 2010;19:399–410. [PubMed: 21116009]
69. Dolan M, Mychaskiw A, Mendel F. Cool-water immersion and high-voltage electric stimulation curb edema formation in rats. *J Athletic Train.* 2003;38(4):225–230.
70. Dolan M, Mychaskiw A, Mattacola C, Mendel F. Effects of cool-water immersion and high-voltage electric stimulation for 3 continuous hours on acute edema in rats. *J Athletic Train.* 2003;38(3):325–329.
71. Hopkins J, Ingersoll C, Edwards J. Cryotherapy and transcutaneous electric neuromuscular stimulation decrease arthrogenic muscle inhibition of the vastus medialis after knee joint effusion. *J Athletic Train.* 2002;37(1):25–31.
72. Cook T, Barr J. Instrumentation. In: Nelson R, Currier D, eds. *Clinical Electrotherapy*. Norwalk, CT: Appleton & Lange; 1999.
73. Denegar C. The effects of low-volt microamperage stimulation on delayed onset muscle soreness. *J Sport Rehabil.* 1993;1:95–102.
74. Reed A, Robertson V, Low J. *Electrotherapy Explained: Principles and Practices*. Burlington, MA: Elsevier Science and Technology; 2006.
75. Flicker MT. *An Analysis of Cold Intermittent Compression with Simultaneous Treatment of Electrical Stimulation in the Reduction of Postacute Ankle Lymphadema* [unpublished master's thesis]. Chapel Hill, NC: University of North Carolina; May 1993.
76. Salar G. Effect of transcutaneous electrotherapy on CSF B-endorphin content in patients without pain problems. *Pain.* 1981;10:169–172. [PubMed: 6267542]
77. Selkowitz D. Improvement in isometric strength of the quadriceps femoris muscle after training with electrical stimulation. *Phys Ther.* 1985;65:186–196. [PubMed: 3871529]

78. Siff M. Applications of electrostimulation in physical conditioning: a review. *J Appl Sport Sci Res.* 1990;4:20–26.
79. Holcomb W, Rubley M, Girouard T. Effect of the simultaneous application of NMES and HVPC on knee extension torque [abstract]. *J Athletic Train.* 2004;39(suppl 2):S–47.
80. Holcomb W, Rubley M, Miller M. The effect of rest intervals on knee-extension torque production with neuromuscular electrical stimulation. *J Sport Rehabil.* 2006;15(2):116.
81. Laufer Y, Ries JD, Leininger PM, Alon G. Quadriceps femoris muscle torques and fatigue generated by neuromuscular electrical stimulation with three different waveforms. *Phys Ther.* 2001;81(7):1307–1316. [[PubMed: 11444994](#)]
82. Lewek M, Stevens J, Snyder-Mackler L. The use of electrical stimulation to increase quadriceps femoris muscle force in an elderly patient following a total knee arthroplasty. *Phys Ther.* 2001;81(8):1565–1571. [[PubMed: 11688592](#)]
83. Valma J, Robertson A, Ward R. Vastus medialis electrical stimulation to improve lower extremity function following a lateral patellar retinacular release. *J Orthop Sports Phys Ther.* 2002;32(9):437–446. [[PubMed: 12322810](#)]
84. Van Lunen B, Carroll C, Gratias K. The clinical effects of cold application on the production of electrically induced involuntary muscle contractions. *J Sport Rehabil.* 2003;12(3):240–248.
85. Windley T. The efficacy of neuromuscular electrical stimulation for muscle-strength augmentation. *Athletic Ther Today.* 2007;12(1):9.
86. Currier D, Mann R. Muscular strength development by electrical stimulation in healthy individuals. *Phys Ther.* 1983;63:915–921. [[PubMed: 6856678](#)]
87. Karnes JL, Mendel FC, Fish DR. High-voltage pulsed current: its influence on diameters of histamine-dilated arterioles in hamster cheek pouches. *Arch Phys Med Rehabil.* 1995;76(4):381–386. [[PubMed: 7717840](#)]
88. Bettany J. Influence of high voltage pulsed current on edema formation following impact injury. *Phys Ther.* 1990;70:219–224. [[PubMed: 2315384](#)]
89. Brown S. The effect of microcurrent on edema, range of motion, and pain in treatment of lateral ankle sprains [abstract]. *J Orthop Sports Phys Ther.* 1994;19:55.
90. Cosgrove K, Alon G. The electrical effect of two commonly used clinical stimulators on traumatic edema in rats. *Phys Ther.* 1992;72:227–233. [[PubMed: 1584856](#)]
91. Fish D. Effect of anodal high voltage pulsed current on edema formation in frog hind limbs. *Phys Ther.* 1991;71:724–733. [[PubMed: 1946611](#)]
92. Griffin J. Reduction of chronic posttraumatic hand edema: a comparison of high voltage pulsed current, intermittent pneumatic compression, and placebo treatments. *Phys Ther.* 1990;70:279–286. [[PubMed: 2185495](#)]
93. Lea J. The effect of electrical stimulation on edematous rat hind paws [abstract R379]. *Phys Ther.* 1992;72:5116.
94. Mendel F. Influence of high voltage pulsed current on edema formation following impact injury in rats. *Phys Ther.* 1992;72:668–673. [[PubMed: 1508974](#)]
95. Miller BF, Gruben KG, Morgan BJ. Circulatory responses to voluntary and electrically induced muscle contractions in humans. *Phys Ther.* 2000;80(1):53–60. [[PubMed: 10623959](#)]
96. Miller M, Cheatham C, Holcomb W. Subcutaneous tissue thickness alters the effect of NMES. *J Sport Rehabil.* 2008;17(1):68. [[PubMed: 18270388](#)]
97. Mulder G. Treatment of open-skin wounds with electric stimulation. *Arch Phys Med Rehabil.* 1991;72:375–377. [[PubMed: 2059103](#)]

98. Taylor K. Effect of a single 30-minute treatment of high voltage pulsed current on edema formation in frog hind limbs. *Phys Ther.* 1992;72:63–68. [PubMed: 1728050]
99. Bishop B. Pain: its physiology and rationale for management. *Phys Ther.* 1980;60:13–37. [PubMed: 6243183]
100. Cheing G, Hui-Chan C. Analgesic effects of transcutaneous electrical nerve stimulation and interferential currents on heat pain in healthy subjects. *J Rehabil Med.* 2003;35(1):15. [PubMed: 12610843]
101. Laughman R, Youdes J, Garrett T. Strength changes in the normal quadriceps femoris muscle as a result of electrical stimulation. *Phys Ther.* 1983;63:494–499. [PubMed: 6601279]
102. Melzack R, Stillwell D, Fox E. Trigger points and acupuncture points for pain: correlations and implications. *Pain.* 1977;3(1):3–23. [PubMed: 69288]
103. Melzack R. *The Puzzle of Pain*. New York: Basic Books; 1973.
104. Rolle W, Alon G, Nirschl R. Comparison of subliminal and placebo stimulation in the management of elbow epicondylitis [abstract R280]. *Phys Ther.* 1991;715:5114.
105. Marino A, Becker R. Biologic effects of extremely low-frequency electric and magnetic fields: a review. *Phys Chem.* 1977;9:131–143.
106. Clement-Jones V. Increased β -endorphin but not met-enkephalin levels in human cerebrospinal fluid after acupuncture for recurrent pain. *Lancet.* 1980;8:946–948.
107. Evans T, Denegar C. Is transcutaneous electrical nerve stimulation (TENS) effective in relieving trigger point pain? *J Athletic Train.* 2002;37(suppl 2S):S–103.
108. Malezic M, Hesse S. Restoration of gait by functional electrical stimulation in paraplegic patients: a modified programme of treatment. *Paraplegia.* 1995;33(3):126–131. [PubMed: 7784113]
109. Malizia E. Electroacupuncture and peripheral β -endorphin and ACTH levels. *Lancet.* 1979;8:535–536.
110. Norcross M, Guskiewicz K, Prentice W. The effects of electrical stimulating currents on pain perception, plasma cortisol, and plasma b-endorphin for DOMS [abstract]. *J Athletic Train (Suppl).* 2004;39(2):S–48.
111. Snyder-Mackler L, Garrett M, Roberts M. A comparison of torque generating capabilities of three different electrical stimulating currents. *J Orthop Sports Phys Ther.* 1989;10:297–301. [PubMed: 18796954]
112. Wolf S. Perspectives on central nervous system responsiveness to transcutaneous electrical nerve stimulation. *Phys Ther.* 1978;58:1443–1449. [PubMed: 217027]
113. Denegar C. Influence of transcutaneous electrical nerve stimulation on pain, range of motion, and serum cortisol concentration in females experiencing delayed onset muscle soreness. *J Orthop Sports Phys Ther.* 1989;11:100–103. [PubMed: 18796921]
114. *Notes on Low Volt Therapy*. White Plains, NY: TECA Corp; 1966.
115. Alon G. “Microcurrent” stimulation: a progress report 1998. *Athletic Ther Today.* 1998;3(6):15.
116. Picker R. Current trends: low volt pulsed microamp stimulation. Parts 1 and 2. *Clin Manage.* 1989;9:11–14, 28–33.
117. Becker R, Bachman C, Friedman H. The direct current control system. *N Y J Med.* 1962;62:1169–1176.

118. Bonacci JA, Higbie EJ. Effects of microcurrent treatment on perceived pain and muscle strength following eccentric exercise. *J Athletic Train*. 1997;32(2):119–123.
119. Tan G, Monga T, Thornby J. Electromedicine: efficacy of microcurrent electrical stimulation on pain severity, psychological distress, and disability. *Am J Pain Manage*. 2000;10(1):35–44.
120. Allen JD, Mattacola CG, Perrin DH. Effect of microcurrent stimulation on delayed-onset muscle soreness: a double-blind comparison. *J Athletic Train*. 1999;34(4):334–337.
121. Ansoleaga E, Wirth V. Microcurrent electrical stimulation may reduce clinically induced DOMS. *J Athletic Train*. 1999;34(2):S–67.
122. Haynie L, Henry L, VanLunen B. Investigation of microcurrent electrical neuromuscular stimulation and high-voltage electrical muscle stimulation on DOMS. *J Athletic Train (Suppl)*. 2002;37 (2S):S–102.
123. Hewlett K, Kimura I, Hetzler R. Microcurrent treatment on pain, edema, and decreased muscle force associated with delayed-onset muscle soreness: a double-blind, placebo study [abstract]. *J Athletic Train*. 2004;39(suppl 2):S–48.
124. Jeter J, Valcenta D. The effects of microcurrent electrical nerve stimulation on delayed onset muscle soreness and peak torque deficits in trained weight lifters [abstract PO-R065-M]. *Phys Ther*. 1993;735:5–24.
125. Johnson MI, Penny P, Sajawal MA. Clinical technical note: an examination of the analgesic effects of microcurrent electrical stimulation (MES) on cold-induced pain in healthy subjects. *Physiother Theory Pract*. 1997;13(4):293–301.
126. Kulig K. Comparison of the effects of high velocity exercise and microcurrent neuromuscular stimulation on delayed onset muscle soreness [abstract R284]. *Phys Ther*. 1991;715:5115.
127. Rapaski D. Microcurrent electrical stimulation: comparison of two protocols in reducing delayed onset muscle soreness [abstract R286]. *Phys Ther*. 1991;715:5116.
128. Ross S, Guskiewicz K. Effect of balance training with and without subsensory electrical stimulation on postural stability of subjects with stable ankles and subjects with functional ankle instability [abstract]. *J Athletic Train*. 2005;40(suppl 2):S–70.
129. Wolcot C. A comparison of the effects of high voltage and microcurrent stimulation on delayed onset muscle soreness [abstract R287]. *Phys Ther*. 1991;715:5116.
130. Young S. Efficacy of interferential current stimulation alone for pain reduction in patients with osteoarthritis of the knee: a randomized placebo control clinical trial [abstract R088]. *Phys Ther*. 1991;715:552.
131. Carley P, Wainapel S. Electrotherapy for the acceleration of wound healing: low-intensity direct current. *Arch Phys Med Rehabil*. 1985;66:443–446. [PubMed: 3893385]
132. Chreng N, Van Houf H, Bockx E. The effects of electric current on ATP generation, protein synthesis, and membrane transport in rat skin. *Clin Orthop Relat Res*. 1982;171:264–272.
133. Gentzkow G. Electrical stimulation to heal dermal wounds. *J Dermatol Surg Oncol*. 1993;19:753–758. [PubMed: 8349916]
134. Griffin J. Efficacy of high voltage pulsed current for healing of pressure ulcers in patients with spinal cord injury. *Phys Ther*. 1991;71:433–444. [PubMed: 2034707]
135. Howson DC. Peripheral neural excitability. *Phys Ther*. 1978;58:1467–1473. [PubMed: 217029]

136. Leffmann D. The effect of subliminal transcutaneous electrical stimulation on the rate of wound healing in rats [abstract R166]. *Phys Ther*. 1992;725:567.
137. Weiss D, Kirsner R, Eaglstein W. Electrical stimulation and wound healing. *Arch Dermatol*. 1990;126:222–225. [PubMed: 2405781]
138. Wood J. A multicenter study on the use of pulsed low-intensity direct current for healing chronic stage II and stage III decubitus ulcers. *Arch Dermatol*. 1993;129:999–1009. [PubMed: 8352625]
139. Connolly J, Hahn H, Jardon O. The electrical enhancement of periosteal proliferation in normal and delayed fracture healing. *Clin Orthop*. 1977;124:97–105.
140. Pettine K. External electrical stimulation and bracing for treatment of spondylolysis—a case report. *Spine*. 1993;188:436–439.
141. Szabo G, Illes T. Experimental stimulation of osteogenesis induced by bone matrix. *Orthopaedics*. 1991;14:63–67.
142. Kahn J. *Low-voltage Technique*. 4th ed. Syosset, NY: Joseph Kahn; 1983.
143. Stanish W, Gunnlaugson B. Electrical energy and soft tissue injury healing. *Sport Care Fitness*. 1988;8(5):12–14.
144. Nessler J, Mass P. Direct current electrical stimulation of tendon healing in vitro. *Clin Orthop Relat Res*. 1987;217(3):303–312.
145. Ward A, Shkuratova N. Russian electrical stimulation: the early experiments. *Phys Ther*. 2002;82(10):1019–1030. [PubMed: 12350217]
146. Delitto A. Introduction to “Russian electrical stimulation”: putting this into perspective. *Phys Ther*. 2002;82(10):1017–1018. [PubMed: 12350216]
147. Goodgold J, Eberstein A. *Electrodiagnosis of Neuromuscular Diseases*. Baltimore, MD: Williams & Wilkins; 1980.
148. Comeau M, Brown L, Landrum J. The effects of high volt pulsed current vs. Russian current on the achievable percentage of MVIC [abstract]. *J Athletic Train*. 2004;39(suppl 2):S–47–S–48.
149. Franklin ME. Effect of varying the ratio of electrically induced muscle contraction time to rest time on serum creatine kinase and perceived soreness. *J Orthop Sports Phys Ther*. 1991;13:310–315. [PubMed: 18784397]
150. Svacina L. Modified interferential technique. *Pain Control*. 1978;4(1):1–2.
151. Snyder S. Opiate receptors and internal opiates. *Sci Am*. 1977;236:44–56. [PubMed: 190676]
152. Nikolova L. *Treatment with Interferential Current*. New York: Churchill Livingstone; 1987.
153. Draper D, Knight K. Interferential current therapy: often used but misunderstood. *Athletic Ther Today*. 2006;11(4):29.
154. Driban J. Bone stimulators and microcurrent: clinical bioelectrics. *Athletic Ther Today*. 2004;9(5):22.
155. Reiff, M Interferential Therapy: Tips for effective treatment, <http://www.medicalproductsonline.org/inth.html>.
156. Larsson L. Functional electrical stimulation. *Scand J Rehabil Ed Suppl*. 1994;30:63–72.
157. Baker L, McNeal D, Benton L. *Neuromuscular Electrical Stimulation*. Downey, CA: Rancho Los Amigos Medical Center; 1993.
158. Heller B, Granat M, Andrews B. Swing-through gait with free-knees produced by surface functional electrical stimulation. *Paraplegia*. 1996;34(1):8–15. [PubMed: 8848326]

159. Agnew W, McCreery D, Bullara L. Effects of prolonged electrical stimulation of peripheral nerve. In: Agnew W, McCreery D, eds. *Neural Prosthesis: Fundamental Studies*. Englewood Cliffs, NJ: Prentice-Hall; 1990.
160. Yamamoto T, Seireg A. Closing the loop: electrical muscle stimulation and feedback control for smooth limb motion. *Soma*. 1986;4:38.
161. Kumar V, Lau H, Liu J. Clinical applications of functional electrical stimulation. *Ann Acad Med*. 1995;24(3):428–435.
162. Bogataj U, Gros N, Kljajic M. The rehabilitation of gait in patients with hemiplegia: a comparison between conventional therapy and multichannel functional electrical stimulation therapy. *Phys Ther*. 1995;75(6):490–502. [PubMed: 7770495]
163. Kagaya H, Shimada Y. Restoration and analysis of standing-up in complete paraplegia utilizing functional electrical stimulation. *Arch Phys Med Rehabil*. 1995;76(9):876–881. [PubMed: 7668962]
164. Kralj A, Badj T, Turk R. Enhancement of gait restoration in spinal cord injured patients by functional electrical stimulation. *Clin Orthop*. 1988;233:34.
165. Mannheimer J, Lampe G. *Clinical Transcutaneous Electrical Nerve Stimulation*. Philadelphia, PA: FA Davis; 1984.
166. Stallard J, Major R. The influence of orthosis stiffness on paraplegic ambulation and its implications for functional electrical stimulation (FES) walking. *Prosthet Orthot Int*. 1995;19(2):108–114. [PubMed: 8570380]
167. Bradley M. The effect of participating in a functional electrical stimulation exercise program on affect in people with spinal cord injuries. *Arch Phys Med Rehabil*. 1994;75(6):676–679. [PubMed: 8002768]
168. Triolo RJ, Bogie K. Lower extremity applications of functional neuromuscular stimulation after spinal cord injury. *Top Spinal Cord Inj Rehabil*. 1999;5(1):44–65.
169. Gersh MR. Microcurrent electrical stimulation: putting it in perspective. *Clin Manage*. 1990;9(4):51–54.
170. Myklebust B, Kloth L. Electrodiagnostic and electrotherapeutic instrumentation: characteristics of recording and stimulation systems and principles of safety. In: Gersh MR, ed. *Electrotherapy in Rehabilitation*. Philadelphia, PA: FA Davis; 2001. [PubMed: NBK68483] [PubMed: 2708166]
171. Porter M, Porter J. Electrical safety in the training room. *Journal of Athletic Training*. 1981;16(4):263–264.
172. American Physical Therapy Association. *Electrotherapeutic Terminology in Physical Therapy: APTA Section on Clinical Electrophysiology*. Alexandria, VA: American Physical Therapy Association; 2000.
173. Chan H, Fung DT. Effects of low-voltage microamperage stimulation on tendon healing in rats. *J Orthop Sports Phys Ther*. 2007;37(7):399. [PubMed: 17710909]
174. Cole B, Gardiner P. Does electrical stimulation of denervated muscle continued after reinnervation, influence recovery of contractile function. *Exp Neurol*. 1984;85:52. [PubMed: 6734785]
175. Cromwell L, Arditti M, Weibell F. *Medical Instrumentation for Health Care*. Englewood Cliffs, NJ: Prentice-Hall; 1991. [PubMed: NBK234400] [PubMed: 3271666]
176. FDA clears restorative therapies functional electrical stimulation. *J Orthop Sports Phys Ther*. 2008;38(3):163.
177. Gutman E, Guttman L. Effect of electrotherapy on denervated and reinnervated muscles in rabbit. *Lancet*. 1942;1:169.
178. Herbison G, Jaweed M, Ditunno J. [Acetylcholine](#) sensitivity and fibrillation potentials in electrically stimulated crush-denervated rat skeletal

muscle. *Arch Phys Med Rehabil.* 1983;64:217. [PubMed: 6847358]

179. Holcomb W, Rubley M. Effect of the simultaneous application of NMES and HVPC on knee extension torque. *J Sport Rehabil.* 2007;16(4):307. [PubMed: 18246897]

180. Kloth L, Cummings J. *Electrotherapeutic Terminology in Physical Therapy.* Alexandria, VA: Section on Clinical Electrophysiology and the American Physical Therapy Association; 1990.

181. Mintken P, Carpenter K. Early neuromuscular electrical stimulation to optimize quadriceps muscle function following total knee arthroplasty: a case report. *J Orthop Sports Phys Ther.* 2007;37(7):364. [PubMed: 17710905]

182. Petterson S, Snyder-Mackler L. The use of neuromuscular electrical stimulation to improve activation deficits in a patient with chronic quadriceps strength impairments following total knee arthroplasty. *J Orthop Sports Phys Ther.* 2006;36(9):678–685. [PubMed: 17017273]

183. Taylor K. Effect of electrically induced muscle contraction on post traumatic edema formation in frog hind limbs. *Phys Ther.* 1992;72:127–132. [PubMed: 1549633]

184. Weber W. The effect of MENS on pain and torque deficits associated with delayed onset muscle soreness [abstract R034]. *Phys Ther.* 1991;715:535.

185. Wilding S, Miller K, Stone M. Increasing electrical stimulation frequency above cramp threshold frequency increases the strength and duration of electrically induced muscle cramps. *J Athletic Train.* 2009;44(suppl):S89.

186. Zbar P, Rockmaker G, Bates D. *Basic Electricity: a Text-Lab Manual.* New York: McGraw-Hill; 2000.

187. Pietrosimone B, Hopkins J, Ingersoll C. Therapeutic modalities: the role of disinhibitory modalities in joint injury rehabilitation. *Athl Ther Today.* 2008;13:2–5.

188. Son SJ, H Kim H. Effects of transcutaneous electrical nerve stimulation on quadriceps function in individuals with experimental knee pain. *Scand J Med Sci Sports.* 2016;26(9):1080–1090. [PubMed: 26346597]

189. Jauregui J, Cherian J. A meta-analysis of transcutaneous electrical nerve stimulation for low back pain. *Surg Technol Int.* 2016;28:296–302. [PubMed: 27042787]

190. Johnson M, Paley C. Transcutaneous electrical nerve stimulation for acute pain. *Cochrane Database Syst Rev.* 2015;6:CD006142.

191. Rutjes A, Nuesch E. Transcutaneous electrostimulation for osteoarthritis of the knee. *Cochrane Database Syst Rev.* 2009;(4):CD002823.

192. M. Osiri, V. Welch, L. Transcutaneous electrical nerve stimulation for knee osteoarthritis. *Cochrane Database Syst Rev.* 2000;(4):CD002823.

193. Aleem I. Efficacy of Electrical Stimulators for Bone Healing: A Meta-Analysis of Randomized Sham-Controlled Trials. *Sci Rep.* 2016;6:31724.

194. Feger M, Goetschius J. Electrical stimulation as a treatment intervention to improve function, edema or pain following acute lateral ankle sprains: a systematic review. *Phys Ther Sport.* 2015;16(4):361–369. [PubMed: 25791198]

195. Howlett O, Lannin N. Functional electrical stimulation improves activity after stroke: a systematic review with meta-analysis. *Arch Phys Med Rehabil.* 2015;96(5):934–943. [PubMed: 25634620]

196. O'Connell N, Wand B. Non-invasive brain stimulation techniques for chronic pain. *Cochrane Database of Syst Rev.* 2014;4CD008208.

197. Kroeling P, Gross A. Electrotherapy for neck pain. *Cochrane Database of Syst Rev.* 2013;8:CD004251.

198. Shim J, Jung J. Effects of electroacupuncture for knee osteoarthritis: a systematic review and meta-analysis. *Evid Based Complement Alternat*

Med. 2016;2016:3485875.

199. Fuentes J, Armijo O. Effectiveness of interferential current therapy in the management of musculoskeletal pain: a systematic review and meta-analysis. *Phys Ther.* 2010;90(9):1219–1238. [PubMed: 20651012]

200. Blum K, Chen A. The H-Wave device is an effective and safe non-pharmacological analgesic for chronic pain: a meta-analysis. *Advanced Therapy.* 2008;25(7):644–657.

201. O'Brien C, Watson A. Deep Oscillation® Therapy in the treatment of lateral epicondylalgia: a pilot randomized control trial. *J Sports Med Dopng Stud.* 2016;6:3

202. Gasbarro V, Bartoletti R, Tsolaki E, Sileno S, Agnati M, et al. Role of Hivamat (Deep Oscillation®) in the treatment for the lymphedema of the limbs. *Eur J Lymphol.* 2006;16:13–15.

SUGGESTED READINGS

Abdel-Moty E, Fishbain D, Goldberg M. Functional electrical stimulation treatment of postradiculopathy associated muscle weakness. *Arch Phys Med Rehabil.* 1994;75(6):680–686. [PubMed: 8002769]

Akyuz G. Transcutaneous electrical nerve stimulation (TENS) in the treatment of postoperative pain and prevention of paralytic ileus. *Clin Rehabil.* 1993;7(3):218–221.

Allen J, Mattacola C, Perrin D. Microcurrent stimulation effect on delayed onset muscle soreness. *J Athletic Train.* 1996;31:S–47.

Alon G. *High Voltage Stimulation: A Monograph.* Chattanooga, TN: Chattanooga Corporation; 1984.

Alon G. *Electrical Stimulators.* Chattanooga, TN: Chattanooga Corporation; 1985 [video presentation].

Alon G, Allin J, Inbar G. Optimization of pulse duration and pulse charge during TENS. *Aust J Physiother.* 1983;29:195. [PubMed: 25025827]

Alon G, Bainbridge J, Croson G. High-voltage pulsed direct current effects on peripheral blood flow. *Phys Ther.* 1981;61:678.

Alon G, Kantor G, Ho H. Effects of electrode size on basic excitatory responses and on selected stimulus parameters. *J Orthop Sports Phys Ther.* 1994;20(1):29–35. [PubMed: 8081407]

Alon G, Kantor G, Smith GV. Peripheral nerve excitation and plantar flexion force elicited by electrical stimulation in males and females. *J Orthop Sports Phys Ther.* 1999;29(4):208–217. [PubMed: 10322593]

American Physical Therapy Association. *Electrotherapeutic Terminology in Physical Therapy.* Alexandria, VA: APTA Publications; 2000.

Andersson S. Pain control by sensory stimulation. In: Bonica JJ, Liebeskind JC, Albe-Fessard DG, eds. *Advances in Pain Research and Therapy.* Vol 3. New York: Raven; 1979:569–584.

Andersson S, Hansson G, Holmgren E. Evaluation of the pain suppression effect of different frequencies of peripheral electrical stimulation in chronic pain conditions. *Acta Orthop Scand.* 1979;47:149.

Arnold P, McVey S. Functional electric stimulation: its efficacy and safety in improving pulmonary function and musculoskeletal fitness. *Arch Phys Med Rehabil.* 1992;73(7):665–668. [PubMed: 1622323]

Aubin M, Marks R. The efficacy of short-term treatment with transcutaneous electrical nerve stimulation for osteo-arthritic knee pain. *Physiotherapy*. 1995;81(11):669–675.

Baker L. Neuromuscular electrical stimulation in the restoration of purposeful limb movements. In: Wolf SL, ed. *Electrotherapy—Clinics in Physical Therapy*. New York: Churchill Livingstone; 1981.

Baker L, McNeal D, Benton L. *Neuromuscular Electrical Stimulation: A Practical Guide*. Downey, CA: Rancho Los Amigos Medical Center; 2000.

Balogun J, Onilari O. High voltage electrical stimulation in the augmentation of muscle strength: effects of pulse frequency. *Arch Phys Med Rehabil*. 1993;74(9):910–916. [PubMed: 8379835]

Bending J. TENS relief of discomfort. *Physiotherapy*. 1993;79(11):773–774.

Benton L, Baker L, Bowman B. *Functional Electrical Stimulation: A Practical Clinical Guide*. 2nd ed. Downey, CA: Professional Staff Association of Rancho Los Amigos Medical Center; 1981.

Berlandt S. Method of determining optimal stimulation sites for transcutaneous nerve stimulation. *Phys Ther*. 1984;64:924. [PubMed: 6610182]

Binder S. Electrical currents. In: Wolf S, ed. *Electrotherapy*. New York: Churchill Livingstone; 1981.

Binder-Macleod S, McDermond L. Changes in the force–frequency relationship of the human quadriceps femoris muscle following electrically and voluntarily induced fatigue. *Phys Ther*. 1992;72(2):95–104. [PubMed: 1549641]

Bowman B, Baker L. Effects of waveform parameters on comfort during transcutaneous neuromuscular electrical stimulation. *Ann Biomed Eng*. 1985;13:59–74. [PubMed: 3873884]

Brown I. *Fundamentals of Electrotherapy, Course Guide*. Madison, WI: University of Wisconsin Press; 1971.

Brown M, Cotter M, Hudlicka O. The effects of long-term stimulation of fast muscles on their ability to withstand fatigue. *J Physiol (Lond)*. 1974;238:47.

Brown M, Cotter M, Hudlicka O. Metabolic changes in long-term stimulated fast muscles. In: Howland H, Poortmans JR, eds. *Metabolic Adaptation to Prolonged Physical Exercise*. Basel: Birkhauser; 1975.

Burr H, Harvey S. Bio-electric correlates of wound healing. *Yale J Biol Med*. 1938;11(2):103–107. [PubMed: 21433802]

Burr H, Taffel M, Harvey S. An electrometric study of the healing wound in man. *Yale J Biol Med*. 1940;12:483. [PubMed: 21433903]

Butterfield DL, Draper DO, Ricard M. The effect of high-volt pulsed current electrical stimulation on delayed-onset muscle soreness. *J Athletic Train*. 1997;32(1):15–20.

Buxton B, Okasaki E, Hetzler R. Self selection of transcutaneous electrical nerve stimulation parameters for pain relief in injured athletes. *J Athletic Train*. 1994;29(2):178.

Byl N, McKenzie A, West J. Pulsed microamperage stimulation: a controlled study of healing of surgically induced wounds in Yucatan pigs. *Phys Ther*. 1994;74(3):201–211. [PubMed: 8115454]

Caggiano E, Emrey T, Shirley S. Effects of electrical stimulation or voluntary contraction for strengthening the quadriceps femoris muscles in an aged male population. *J Orthop Sports Phys Ther*. 1994;20(1):22–28. [PubMed: 8081406]

Campbell J. A critical appraisal of the electrical output characteristics of ten transcutaneous nerve stimulators. *Clin Phys Physiol Meas*. 1982;3:141. [PubMed: 6981485]

Carmick J. Clinical use of neuromuscular electrical stimulation for children with cerebral palsy, part 1, lower extremity. *Phys Ther.* 1993a;73(8):505–513. [PubMed: 8337238]

Carmick J. Clinical use of neuromuscular electrical stimulation for children with cerebral palsy, part 2, upper extremity. *Phys Ther.* 1993b;73(8):514–522. [PubMed: 8337239]

Chan C, Chow S. Electroacupuncture in the treatment of post-traumatic sympathetic dystrophy (Sudek's atrophy). *Br J Anesth.* 1981;53:899.

Chase J. Elicitation of periods of inhibition in human muscle by stimulation of cutaneous nerves. *J Bone Joint Surg.* 1972;54:173–177.

Cook H, Morales M, La Rosa E. Effects of electrical stimulation on lymphatic flow and limb volume in the rat. *Phys Ther.* 1994;74(11):1040–1046. [PubMed: 7972365]

Cooperman A. Use of transcutaneous electrical stimulation in the control of post operative pain. Results of a prospective, randomized, controlled study. *Am J Surg.* 1977;133:185.

Curico F, Berweger R. A clinical evaluation of the pain suppressor TENS, Fairleigh Dickinson University School of Dentistry, 1983. *Curr Opin Orthop.* 1993;4(6):105–109.

Currier D, Mann R. Pain complaint: comparison of electrical stimulation with conventional isometric exercise. *J Orthop Sports Phys Ther.* 1984;5:318. [PubMed: 18806388]

Currier D, Petrilli C, Threlkeld A. Effect of medium frequency electrical stimulation on local blood circulation to healthy muscle. *Phys Ther.* 1986;66:937. [PubMed: 2940607]

Currier D, Ray J, Nyland J. Effects of electrical and electromagnetic stimulation after anterior cruciate ligament reconstruction. *J Orthop Sports Phys Ther.* 1993;17(4):177–184. [PubMed: 8467342]

DeGirardi C, Seaborne D, Goulet F. The analgesic effect of high voltage galvanic stimulation combined with ultrasound in the treatment of low back pain: a one-group pretest/post-test study. *Physiother Can.* 1984;36:327.

Dimitrijevic M. Mesh-glove. 1. A method for whole-hand electrical stimulation in upper motor neuron dysfunction. *Scand J Rehabil Med.* 1994a;26(4):183–186. [PubMed: 7878391]

Dimitrijevic M. Mesh-glove. 2. Modulation of residual upper limb motor control after stroke with whole-hand electric stimulation. *Scand J Rehabil Med.* 1994b;26(4):187–190. [PubMed: 7878392]

Dolan M, Mendel F, Fish D. Effects of high voltage pulsed current on recovery following grade I and II lateral ankle sprains. *J Athletic Train.* 2009;44(suppl):S57.

Draper V, Lyle L, Seymour T. EMG biofeedback versus electrical stimulation in the recovery of quadriceps surface EMG. *Clin Kinesiol.* 1997;51(2):28–32.

Eisenberg B, Gilal A. Structural changes in single muscle fibers after stimulation at a low-frequency. *J Gen Physiol.* 1979;74:1.

Eriksson E, Haggmark T, Kiessling KH. Effect of electrical stimulation on human skeletal muscle. *Int J Sports Med.* 1981;2:18. [PubMed: 7333731]

Ersek R. Transcutaneous electrical neurostimulation—a new modality for controlling pain. *Clin Orthop Relat Res.* 197;128:314.

Faghri P, Glaser R, Figoni S. Functional electrical stimulation leg cycle ergometer exercise: training effects on cardiorespiratory responses of spinal cord injured. *Arch Phys Med Rehabil.* 1992;73(11):1085–1093. [PubMed: 1444777]

Faghri P, Rodger M, Glaser R. The effects of functional electrical stimulation on shoulder subluxation, arm function recovery, and shoulder pain in hemiplegic stroke patients. *Arch Phys Med Rehabil*. 1994;75(1):73–79. [PubMed: 8291967]

Ferguson A, Granat M. Evaluation of functional electrical stimulation for an incomplete spinal cord injured patient. *Physiotherapy*. 1992;78(4):253–256.

Finlay C. TENS: an adjunct to analgesia. *Can Nurse*. 1992;88(8):24–26.

Fleischli JG, Laughlin TJ. Electrical stimulation in wound healing. *J Foot Ankle Surg*. 1997;36(6):457. [PubMed: 9430002]

Fourie JA, Bowerbank P. Stimulation of bone healing in new fractures of the tibial shaft using interferential currents. *Physiother Res Int*. 1997;2(4):255–268. [PubMed: 9408935]

Fox F, Melzack R. Transcutaneous electrical stimulation and acupuncture: comparison of treatment for low back pain. *Pain*. 1976;2:141. [PubMed: 141018]

Frank C, Schachar N, Dittrich D. Electromagnetic stimulation of ligament healing in rabbits. *Clin Orthop Relat Res*. 1983;175:263.

Gallien P, Brisso R, Eyssette M. Restoration of gait by functional electrical stimulation for spinal cord injured patients. *Paraplegia*. 1995;33(11):660–664. [PubMed: 8584301]

Garrison S. *Handbook of Physical Medicine and Rehabilitation*. Philadelphia, PA: Lippincott Williams and Wilkins; 2003. [PubMed: NBK222017]
[PubMed: 3186378]

Geddes L. A short history of the electrical stimulation of excitable tissue. *Physiologist*. 1984;27(suppl):1. [PubMed: 6371852]

Geddes L, Baler L. *Applied Biomedical Instrumentation*. New York: Wiley; 1975.

Gellman H, Waters R, Lewonski K. Histologic comparison of chronic implantation of nerve cuff and epineural electrodes. *Adv Ext Control Hum Extrem*. 1990;12:160–183.

Godfrey C, Jayawardena H, Quance T. Comparison of electro-stimulation and isometric exercise in strengthening the quadriceps muscle. *Physiother Can*. 1979;31:265.

Gotlin R, Hershkowitz S. Electrical stimulation effect on extensor lag and length of hospital stay after total knee arthroplasty. *Arch Phys Med Rehabil*. 1994;75(9):957–959. [PubMed: 8085929]

Gould M, Donnermeyer D, Gammon GG. Transcutaneous muscle stimulation to retard disuse atrophy after open meniscectomy. *Clin Orthop Relat Res*. 1983;178:190.

Granat M. Functional electrical stimulation and hybrid orthosis systems. *Paraplegia*. 1996;34(1):24–29. [PubMed: 8848319]

Greathouse D, Nitz A, Matullonis D. Effects of electrical stimulation on ultrastructure of rat skeletal muscles. *Phys Ther*. 1984;64:755.

Guffey J, Asmussen M. In vitro bactericidal effects of high voltage pulsed current versus direct current against *Staphylococcus aureus*. *J Clin Electrophysiol*. 1989;1:5–9.

Gum SL, Reddy GK, Stehno-Bittel L, Enwemeka CS. Combined ultrasound, electrical stimulation, and laser promote collagen synthesis with moderate changes in tendon bio-mechanics. *Am J Phys Med Rehabil*. 1997;76(4):288–296. [PubMed: 9267188]

Halback J, Straus D. Comparison of electromyostimulation to isokinetic training in increasing power of the knee extensor mechanism. *J Orthop Sports Phys Ther*. 1980;2:20. [PubMed: 18810165]

Hamilton M, Anguish B, Koch D. Effects of high-voltage pulsed electrical current on pain, swelling and function following delayed onset muscle soreness. *J Athletic Train*. 2008;43(suppl):S86.

Higgins M, Eaton C. Nontraditional applications of neuromuscular electrical stimulation. *Athletic Ther Today*. 2005;9(5):6.

Holcomb W, Golestani S, Hill S. AQ comparison of knee extension force production with biphasic versus Russian current. *J Athletic Train*. 1999;34(2):S-17.

Holcomb W, Mangus B, Tandy R. The effect of icing with the Pro-Stim Edema Management System on cutaneous cooling. *J Athletic Train*. 1996;31(2):126-129.

Holcomb W, Rubley M. Periodic increases in neuromuscular electrical stimulation intensity eliminated significant knee extension torque decline. *J Athletic Train*. 2007;42(suppl):S134.

Houghton PE, Kincaid CB, Lovell M, et al. Effect of electrical stimulation on chronic leg ulcer size and appearance. *Phys Ther*. 2003;83(1):17-28. [PubMed: 12495409]

Ignelzi R, Nyquist J. Excitability changes in peripheral nerve fibers after repetitive electrical stimulation: implications in pain modulation. *J Neurosurg*. 1979;61:824.

Indergand H, Morgan B. Effects of high frequency transcutaneous electrical stimulation on limb blood flow in healthy humans. *Phys Ther*. 1994;74(4):361-367. [PubMed: 8140149]

Indergand H, Morgan B. Effect of interference current on forearm vascular resistance in asymptomatic humans. *Phys Ther*. 1995;75(5):306-312. [PubMed: 7899488]

Johnson MI. The mystique of interferential currents when used to manage pain. *Physiotherapy*. 1999;85(6):294-297.

Johnson MI, Tabasam G. A double-blind placebo controlled investigation into the analgesic effects of inferential currents (IFC) and transcutaneous electrical nerve stimulation (TENS) on cold-induced pain in healthy subjects. *Physiother Theory Pract*. 1999;15(4):217-233.

Jones D, Bigland-Ritchie B, Edwards R. Excitation and frequency and muscle fatigue: mechanical responses during voluntary and stimulated contractions. *Exp Neurol*. 1979;64:401. [PubMed: 428515]

Kahn J. *Low-volt Technique*. Syosset, NY: Joseph Kahn; 1983.

Karmel-Ross K, Cooperman D. The effect of electrical stimulation on quadriceps femoris muscle torque in children with spina bifida. *Phys Ther*. 1992;72(10):723-730. [PubMed: 1528965]

Karnes J. Effects of low-voltage pulsed current on edema formation in frog hind limbs following impact injury. *Phys Ther*. 1992a;72:273-278. [PubMed: 1584859]

Karnes J. Influence of high voltage pulsed current on diameters of arterioles during histamine-induced vasodilation [abstract R341]. *Phys Ther*. 1992b;725:5105.

Kim K, Saliba S. Effects of neuromuscular electrical stimulation after anterior cruciate ligament reconstruction on quadriceps strength, function, and patient oriented outcomes: a systematic review. *J Athletic Train*. 2009;44(suppl):S87.

Kono T, Ingersoll CD, Edwards JE. A comparison of acupuncture, TENS, and acupuncture with TENS for pain relief during DOMS. *J Athletic Train*. 1999;34(2):S-67.

Kostov A, Andrews B, Popovic D. Machine learning in control of functional electrical stimulation systems for locomotion. *IEEE Trans Biomed Eng*. 1995;42(6):541–551. [PubMed: 7790010]

Kramer J, Mendryk S. Electrical stimulation as a strength improvement technique: a review. *J Orthop Sports Phys Ther*. 1982;4:91. [PubMed: 18810104]

Kues J, Mayhew T. Concentric and eccentric force–velocity relationships during electrically induced submaximal contractions. *Phys Ther*. 1996;76(5):S17.

Lainey C, Walmsley R, Andrew G. Effectiveness of exercise alone versus exercise plus electrical stimulation in strengthening the quadriceps muscle. *Physiother Can*. 1983;35:5.

Lane J. Electrical impedances of superficial limb tissue, epidermis, dermis and muscle sheath. *Ann N Y Acad Sci*. 1974;238:812.

Latash M, Yee M, Orpett C. Combining electrical muscle stimulation with voluntary contraction for studying muscle fatigue. *Arch Phys Med Rehabil*. 1994;75(1):29–35. [PubMed: 8291958]

LeDoux J, Quinones M. An investigation of the use of percutaneous electrical stimulation in muscle reeducation. *Phys Ther*. 1981;61:678.

Leffman D, Arnall D, Holmgren P. Effect of microamperage stimulation on the rate of wound healing in rats: a histological study. *Phys Ther*. 1994;74(3):195–200. [PubMed: 8115453]

Levin M, Hui-Chan C. Conventional and acupuncture-like transcutaneous electrical nerve stimulation excite similar afferent fibers. *Arch Phys Med Rehabil*. 1993;74(1):54–60. [PubMed: 8420521]

Licht S. *Electrodiagnosis and Electromyography*. Vol 1. 3rd ed. Baltimore, MD: Waverly; 1971.

Licht S. History of electrotherapy. In: Stillwell GK, ed. *Therapeutic Electricity and Ultraviolet Radiation*. 3rd ed. Baltimore, MD: Williams & Wilkins; 1983.

Litke D, Dahners L. Effects of different levels of direct current on early ligament healing in a rat model. *J Orthop Res*. 1992;12:683–688.

Livesley E. Effects of electrical neuromuscular stimulation on functional performance in patients with multiple sclerosis. *Physiotherapy*. 1992;78(12):914–917.

Loeser J. Nonpharmacologic approaches to pain relief. In: Ng L, Bonica J, eds. *Pain, Discomfort and Humanitarian Care*. New York: Elsevier; 1980.

Loesor J, Black R, Christman A. A relief of pain by transcutaneous stimulation. *J Neurosurg*. 1975;42:308. [PubMed: 1117329]

Long D. Cutaneous afferent stimulation for relief of chronic pain. *Clin Neurosurg*. 1974;21:257. [PubMed: 4371289]

Macdonald A, Coates T. The discovery of transcutaneous spinal electroanalgesia and its relief of chronic pain. *Physiotherapy*. 1995;81(11):653–661.

Mannheimer C, Carlsson C. The analgesic effect of transcutaneous electrical nerve stimulation (TENS) in patients with rheumatoid arthritis. A comparative study of different pulse patterns. *Pain*. 1979;6:329. [PubMed: 313550]

Mannheimer C, Lund S, Carlsson C. The effect of transcutaneous electrical nerve stimulation (TENS) on joint pain in patients with rheumatoid arthritis. *Scand J Rheumatol*. 1978;7:13. [PubMed: 307810]

Mannheimer J. Electrode placements for transcutaneous electrical nerve stimulation. *Phys Ther*. 1978;58:1455. [PubMed: 311010]

Mao W, Ghia J, Scott D. High versus low-intensity acupuncture analgesic for treatment of chronic pain: effects on platelet serotonin. *Pain*. 1980;8:331. [PubMed: 7402692]

- Markov M. Electric current and electromagnetic field effects on soft tissue: implications for wound healing. *Wounds Comp Clin Res Pract*. 1995;7(3):94–110.
- Marvie K. A major advance in the control of post-operative knee pain. *Orthopedics*. 1979;2:129.
- Massey B, Nelson R, Sharkey B. Effects of high frequency electrical stimulation on the size and strength of skeletal muscle. *J Sports Med Phys Fitness*. 1965;5:136. [PubMed: 5850780]
- Mastbergen P, Lawson N, Meyer R. TENS application does not alter vibratory sensory threshold. *J Athletic Train*. 2009;44(suppl):S90.
- Matsunaga T, Shimada Y, Sato K. Muscle fatigue from intermittent stimulation with low and high frequency electrical pulses. *Arch Phys Med Rehabil*. 1999;80(1):48–53. [PubMed: 9915371]
- Mattison J. Transcutaneous electrical nerve stimulation in the management of painful muscle spasm in patients with multiple sclerosis. *Clin Rehabil*. 1993;7(1):45–48.
- McMiken D, Todd-Smith M, Thompson C. Strengthening of human quadriceps muscles by cutaneous electrical stimulation. *Scand J Rehabil Med*. 1983;15:25. [PubMed: 6828830]
- McQuain M, Sinaki M, Shibley L. Effect of electrical stimulation on lumbar paraspinal muscles. *Spine*. 1993;18(13):1787–1792. [PubMed: 8235862]
- Merrick MA. Research digest. Unconventional modalities: microcurrent. *Athletic Ther Today*. 1999;4(5):53–54.
- Meyer G, Fields H. Causalgia treated by selective large fibre stimulation of peripheral nerve. *Brain*. 1972;95:163. [PubMed: 5023085]
- Meyer R, Lawson N, Niemann A. TENS application alters constant pressure sensory threshold. *J Athletic Train*. 2009;44(suppl):S88.
- Michlovitz S. Ice and high voltage pulsed stimulation in treatment of acute lateral ankle sprains. *J Orthop Sports Phys Ther*. 1988;9:301–304. [PubMed: 18796993]
- Miller K, Knight K. The relationship between the beginning electrical stimulation frequency and a person's true cramp threshold frequency. *J Athletic Train*. 2009;44(suppl):S89.
- Milner-Brown H, Stein R. The relation between the surface electromyogram and muscular force. *J Physiol*. 1975;246:549. [PubMed: 1133787]
- Mohr T, Carlson B, Sulentic C. Comparison of isometric exercise and high volt galvanic stimulation on quadriceps, femoris muscle strength. *Phys Ther*. 1985;65:606. [PubMed: 3991805]
- Mostowy D. An application of transcutaneous electrical nerve stimulation to control pain in the elderly. *J Gerontol Nurs*. 1996;22(2):36–38.
- Munsat T, McNeal D, Waters R. Preliminary observations on prolonged stimulation of peripheral nerve in man. *Arch Neurol*. 1976;33:608. [PubMed: 962643]
- Myklebust J, ed. *Neural Stimulation*. Boca Raton, FL: CRC Press; 1985.
- Naess K, Storm-Mathison A. Fatigue of sustained tetanic contractions. *Acta Physiol Scand*. 1955;34:351. [PubMed: 13282743]
- Newing A, Tsang K, Thomas K. Concomitant application of ice and electrical stimulation does not improve pain threshold. *J Athletic Train*. 2008;43(suppl):S85.
- Newton R. Electrotherapy: selecting wave form parameters. Paper presented at the American Physical Therapy Association Conference; 1981;

Washington, DC.

Newton R. *Electrotherapeutic Treatment: Selecting Appropriate Wave Form Characteristics*. Clinton, NJ: Preston; 1984.

Owens J, Malone T. Treatment parameters of high frequency electrical stimulation as established on the Electrostim 180. *J Orthop Sports Phys Ther*. 1983;4:162. [PubMed: 18806445]

Packman-Braun R. Misconceptions regarding functional electrical stimulation. *Neurol Rep*. 1995;19(3):17–21.

Perroti A, Bay R, Snyder A. The influence of high volt electrical stimulation on edema formation following acute injury: a systematic review of the literature. *J Athletic Train*. 2008;43(suppl):S87.

Pert V. TENS for pain in multiple sclerosis. *Physiotherapy*. 1991;77(3):227–228.

Petrofsky J. Functional electrical stimulation, a two-year study. *J Rehabil*. 1992;58(3):29–34.

Picaza J, Cannon B, Hunter S. Pain suppression by peripheral stimulation, part I. Observations with transcutaneous stimuli. *Surg Neurol*. 1975;4:105. [PubMed: 1080893]

Pouran D, Faghri M, Rodgers M. The effects of functional electrical stimulation on shoulder subluxation, arm function recovery, and shoulder pain in hemiplegic stroke patients. *Arch Phys Med Rehabil*. 1994;75(1):73–79. [PubMed: 8291967]

Procacci P, Zoppi M, Maresca M. Transcutaneous electrical stimulation in low back pain: a critical evaluation. *Acupunct Electrother Res*. 1982;7:1.

Rabischong E, Doutrelot P, Ohanna F. Compound motor action potentials and mechanical failure during sustained contractions by electrical stimulation in paraplegic. *Paraplegia*. 1995;33(12):707–714. [PubMed: 8927410]

Rack P, Westbury D. The effects of length and stimulus rate on tension in the isometric cat soleus muscle. *J Physiol*. 1969;204:443. [PubMed: 5824646]

Ray R, Samuelson A. Microcurrent versus a placebo for the control of pain and edema. *J Athletic Train*. 1996;31:S–48.

Reddanna P, Moortly C, Govidappa S. Pattern of skeletal muscle chemical composition during in vivo electrical stimulations. *Ind J Physiol Pharmacol*. 1981;25:33.

Reismann M. A comparison of electrical stimulators eliciting muscle contraction. *Phys Ther*. 1984;64:751.

Requena B, Erelina J, Gapeyeva H. Posttetanic potentiation in knee extensors after high-frequency submaximal percutaneous electrical stimulation. *J Sport Rehabil*. 2005;14(3):248–257.

Rieb L, Pomeranz B. Alterations in electrical pain thresholds by use of acupuncture-like transcutaneous electrical nerve stimulation in pain-free subjects. *Phys Ther*. 1992;72(9):658–667. [PubMed: 1508973]

Rochester L. Influence of electrical stimulation of the tibialis anterior muscle in paraplegic subjects: 1. Contractile properties. *Paraplegia*. 1995;33(8):437–449. [PubMed: 7478737]

Roeser W, Meeks LW, Venis R, et al. The use of transcutaneous nerve stimulation for pain control in athletic medicine: a preliminary report. *Am J Sports Med*. 1976;4(5):210. [PubMed: 63248]

Romero J, Sanford T, Schroeder R. The effects of electrical stimulation of normal quadriceps on strength and girth. *Med Sci Sports Exerc*. 1982;14:194. [PubMed: 7109885]

Rosch P, Markov M. *Bioelectromagnetic Medicine*. New York: Informa Healthcare; 2004.

Rosenberg M, Vutyid L, Bourbe D. Transcutaneous electrical nerve stimulation for the relief of post-operative pain. *Pain*. 1978;5:129. [PubMed: 358067]

Rowley B, McKenna J, Chase G. The influence of electrical current on an infecting microorganism in wounds. *Ann N Y Acad Sci*. 1974;238:543. [PubMed: 4216282]

Schmitz R, Martin D, Perrin D. The effects of interferential current of perceived pain and serum cortisol in a delayed onset muscle soreness model. *J Athletic Train*. 1994;29(2):171.

Scott P. *Clayton's Electrotherapy and Actinotherapy*. 5th and 7th ed. Baltimore: Williams & Wilkins; 1965 and 1975.

Seib T, Price R, Reyes M. The quantitative measurement of spasticity: effect of cutaneous electrical stimulation. *Arch Phys Med Rehabil*. 1994;75(7):746–750. [PubMed: 8024418]

Selkowitz D. Improvement in isometric strength of the quadricep femoris muscle after training with electrical stimulation. *Phys Ther*. 1985;65:186. [PubMed: 3871529]

Shealey C, Maurer D. Transcutaneous nerve stimulation for control of pain. *Surg Neurol*. 1974;2:45. [PubMed: 4272574]

Simmonds M, Wessel J, Scudds R. The effect of pain quality on the efficacy of conventional TENS. *Physiotherapy (Can)*. 1992;44(3):35–40.

Sjolund B, Eriksson M. The influence of [naloxone](#) on analgesia produced by peripheral conditioning stimulation. *Brain Res*. 1979;173:295. [PubMed: 487091]

Sjolund B, Terenius L, Eriksson M. Increased cerebrospinal fluid levels of endorphin after electroacupuncture. *Acta Physiol Scand*. 1977;100:382. [PubMed: 920207]

Smith B, Betz R, Mulcahey M. Reliability of percutaneous intramuscular electrodes for upper extremity functional neuro-muscular stimulation in adolescents with C5 injury. *Arch Phys Med Rehabil*. 1994;75(9):939–945. [PubMed: 8085926]

Smith B, Mulcahey M, Betz R. Quantitative comparison of grasp and release abilities with and without functional neuro-muscular stimulation in adolescents with tetraplegia. *Paraplegia*. 1996;34(1):16–23. [PubMed: 8848318]

Snyder K, Meyer R, Neimann A. Transcutaneous electrical nerve stimulation (TENS) does not alter cold sensory detection threshold. *J Athletic Train*. 2009;44(suppl):S89.

Snyder-Mackler L, Delitto A, Stralka S. Use of electrical stimulation to enhance recovery of quadriceps femoris muscle force production in patients following anterior cruciate ligament reconstruction. *Phys Ther*. 1994;74(10):901–907. [PubMed: 8090841]

Standish W, Valiant G, Bonen A. The effects of immobilization and of electrical stimulation on muscle glycogen and myofibrillar ATPase. *Can J Appl Sports Sci*. 1982;7:267.

Stone JA. Prevention and rehabilitation. “Russian” electrical stimulation. *Athletic Ther Today*. 1997;2(3):27.

Stone JA. Prevention and rehabilitation. Interferential electrical stimulation. *Athletic Ther Today*. 1997a;2(2):27.

Stone JA. Prevention and rehabilitation. Microcurrent electrical stimulation. *Athletic Ther Today*. 1997b;2(6):15.

Sunderland S. *Nerves and Nerve Injuries*. Baltimore, MD: Williams & Wilkins; 1968.

- Szehi E, David E. The stereodynamic interferential current—a new electrotherapeutic technique. *Electromedica*. 1980;48:13.
- Szuminsky N, Albers A, Unger P. Effect of narrow pulsed high voltages on bacterial viability. *Phys Ther*. 1994;74(7):660–667. [PubMed: 8016198]
- Taylor M, Newton R, Personius W. The effects of interferential current stimulation for the treatment of subjects with recurrent jaw pain [abstract]. *Phys Ther*. 1986;66:774.
- Taylor P, Hallet M, Flaherty L. Treatment of osteoarthritis of the knee with transcutaneous electrical nerve stimulation. *Pain*. 1981;11:233. [PubMed: 7033891]
- Terezhalmay G, Ross G, Holmes-Johnson E. Transcutaneous electrical nerve stimulation treatment of TMJPD patients. *Ear Nose Throat J*. 1982;61:664.
- Thorsteinsson G, Stonnington H. The placebo effect of transcutaneous electrical stimulation. *Pain*. 1978;5:31. [PubMed: 353652]
- Tourville T, Connolly D, Reed B. Effects of sensory level high-volt pulsed electrical current on delayed onset muscle soreness. *J Athletic Train*. 2003;38(suppl 2S):S–33.
- Vrbov G, Hudlicka O. *Application of Muscle/Nerve Stimulation in Health and Disease*. New York: Springer; 2008.
- Wadsworth H, Chanmugan A. *Electrophysical Agents in Physical Therapy*. Marrickville, Australia: Science Press; 1983.
- Walsh D, Foster N, Baxter G. Transcutaneous electrical nerve stimulation parameters to neurophysiological and hypoalgesic effects. *Phys Ther*. 1996;76(5):552.
- Walsh D, McAdams E. *TENS: Clinical Applications and Related Theory*. Philadelphia, PA: WB Saunders; 1997.
- Ward A. *Electricity Waves and Fields in Therapy*. Marrickville, Australia: Science Press; 1980.
- Watson T. *Electrotherapy: Evidence Based Practice*. Philadelphia, PA: Churchill Livingstone; 2008.
- Weber M, Servedio F, Woddall W. The effects of three modalities on delayed onset muscle soreness. *J Orthop Sports Phys Ther*. 1994;20(5):236–242. [PubMed: 7827630]
- Wheeler P, Wolcott L, Morris J. Neural considerations in the healing of ulcerated tissue by clinical electrotherapeutic application of weak direct current: findings and theory. In: Reynolds D, Sjöberg A, eds. *Neuroelectric Research*. Springfield, IL: Charles C Thomas; 1971:83–96.
- Williams G, Krishnan C, Allen E. Torque-based triggering improves stimulus timing precision in activation tests. *J Athletic Train*. 2009;44(suppl):S88.
- Windsor R, Lester J. Electrical stimulation in clinical practice. *Phys Sports Med*. 1993;21(2):85–86, 89–92.
- Wolf S, Gersh M, Kutner M. Relationship of selected clinical variables to current delivered during transcutaneous electrical nerve stimulation. *Phys Ther*. 1978;58:1478–1483. [PubMed: 368821]
- Wolf S, Gersh M, Rao V. Examination of electrode placements and stimulating parameters in treating chronic pain with conventional transcutaneous nerve stimulation (TENS). *Pain*. 1981;11:37. [PubMed: 6975458]
- Wong R, Jette D. Changes in sympathetic tone associated with different forms of transcutaneous electrical nerve stimulation in healthy subjects. *Phys Ther*. 1984;64:478. [PubMed: 6709712]
- Yarkony G, Roth E. Neuromuscular stimulation in spinal cord injury: restoration of functional movement of the extremities, part 1. *Arch Phys Med*

Rehabil. 1992;73(1):78–86. [PubMed: 1729980]

Yarkony G, Roth E, Cybulski J. Neuromuscular stimulation in spinal cord injury II: prevention of secondary complications, part 2. *Arch Phys Med Rehabil.* 1992;73(2):195–200. [PubMed: 1543418]

Zecca L, Ferrario P, Furia G. Effects of pulsed electromagnetic field on acute and chronic inflammation. *Trans Biol Repair Growth Soc.* 1983;3:72.

GLOSSARY

absolute refractory period Brief time period (0.5 microsecond) following membrane depolarization during which the membrane is incapable of depolarizing again.

accommodation Adaptation by the sensory receptors to various stimuli over an extended period of time.

action potential A recorded change in electrical potential between the inside and outside of a nerve cell, resulting in muscular contraction.

all-or-none response The depolarization of nerve or muscle membrane is the same once a depolarizing intensity threshold is reached; further increases in intensity do not increase the response.

alternating current Current that periodically changes its polarity or direction of flow.

ampere Unit of measure that indicates the rate at which electrical current is flowing.

amplitude The intensity of current flow as indicated by the height of the waveform from baseline.

anode The positively charged electrode.

biphasic current Another name for alternating current, in which the direction of current flow reverses direction.

bursts A combined set of three or more pulses; also referred to as packets or envelopes.

cathode The negatively charged electrode.

chronaxie The duration of time necessary to cause observable tissue excitation, given a current intensity of two times rheobasic current.

circuit The path of current from a generating source through the various components back to the generating source.

conductance The ease with which a current flows along a conducting medium.

conductors Materials that permit the free movement of electrons.

constructive interference The combined amplitude of two distinct circuits increases the amplitude.

coulomb Indicates the number of electrons flowing in a current.

current density Amount of current flow per cubic area.

current The flow of electrons.

cycle Applies to biphasic current.

decay time The time required for a waveform to go from peak amplitude to 0 V.

denervated muscle A muscle that does not have nerve innervation.

depolarization Process or act of neutralizing the cell membrane's resting potential.

Destructive interference Combined amplitude of two distinct circuits decreases the amplitude.

direct current Galvanic current that always flows in the same direction and may flow in either a positive or a negative direction.

duration Sometimes also referred to as *pulse width*. Indicates the length of time the current is flowing.

electrical current The net movement of electrons along a conducting medium.

electrical impedance The opposition to electron flow in a conducting material.

electrical potential The difference between charged particles at a higher and a lower potential.

electron Fundamental particles of matter possessing a negative electrical charge and very small mass.

frequency window selectivity Cellular responses may be triggered by a certain electrical frequency range.

frequency The number of cycles or pulses per second.

functional electrical stimulation Utilizes multiple-channel electrical stimulators to recruit muscles in a programmed sequence that produces a functional movement pattern.

ground A wire that makes an electrical connection with the earth.

ground-fault interruptors (GFI) A safety device that automatically shuts off current flow and reduces the chances of electrical shock.

insulators Materials that resist current flow.

interburst intervals Interruptions between individual bursts.

interphase interval The interruptions between individual pulses or groups of pulses.

ion A positively or negatively charged particle.

iontophoresis Uses continuous direct current to drive ions into the tissues.

low-intensity stimulator (LIS) Another more current term for MENS.

macroshock An electrical shock that can be felt and has a leakage of electrical current of greater than 1 mA.

medical galvanism Creates either an acidic or an alkaline environment that may be of therapeutic value.

microcurrent electrical nerve stimulator (MENS) Used primarily in tissue healing, the current intensities too small to excite peripheral nerves.

microcurrent The term most commonly used to refer to MENS or LIS.

microshock An electrical shock that is imperceptible because of a leakage of current of less than 1 mA.

modulation Refers to any alteration in the magnitude or any variation in the duration of an electrical current.

monophasic current Another name for direct current, in which the direction of current flow remains the same.

neuromuscular electrical stimulator (NMES) Also called an electrical muscle stimulator (EMS), it is used to stimulate muscle directly, as would be the case with denervated muscle where peripheral nerves are not functioning.

ohm A unit of measure that indicates resistance to current flow.

ohm's law The current in an electrical circuit is directly proportional to the voltage and inversely proportional to the resistance.

parallel circuit A circuit in which two or more routes exist for current to pass between the two terminals.

phases That portion of the pulse that rises above or below the baseline for some period of time.

pulsatile currents Contain three or more pulses grouped together and can be unidirectional or bidirectional.

pulse charge The total amount of electricity being delivered to the patient during each pulse.

pulse period The combined time of the pulse duration and the interpulse interval.

pulse An individual waveform.

ramping Another name for surging modulation, in which the current builds gradually to some maximum amplitude.

ramping Another name for surging modulation, in which the current builds gradually to some maximum amplitude.

rate of rise How quickly a waveform reaches its maximum amplitude.

resistance The opposition to electron flow in a conducting material.

resting potential The potential difference between the inside and outside of a membrane.

rheobase The specific intensity of current necessary to cause an observable tissue response given a long current duration.

russian current A medium-frequency (2000–10,000 Hz) pulsatile biphasic wave generated in 50-bursts-per-second envelopes.

series circuit A circuit in which there is only one path for current to get from one terminal to another.

stereodynamic interference current Three distinct circuits blending and creating a distinct electrical wave pattern.

tetanzation When individual muscle twitch responses can no longer be distinguished and the responses force maximum shortening of the stimulated muscle fiber.

tetany Muscle condition that is caused by hyperexcitation and results in cramps and spasms.

transcutaneous electrical stimulator All therapeutic electrical generators regardless of whether they deliver biphasic, monophasic, or pulsatile currents through electrodes

transcutaneous electrical nerve stimulator (TENS) A transcutaneous electrical stimulator used to stimulate peripheral nerves.

volt The electromotive force that must be applied to produce a movement of electrons. A measure of electrical power.

voltage-sensitive permeability The quality of some cell membranes that makes them permeable to different ions based on the electrical charge of the ions. Nerve and muscle cell membranes allow negatively charged ions into the cell while actively transporting some positively charged ions outside the cell membrane.

voltage The force resulting from an accumulation of electrons at one point in an electrical circuit, usually corresponding to a deficit of electrons at another point in the circuit.

watt A measure of electrical power ($watt = volt \times ampere$).

waveform The shape of an electrical current as displayed on an oscilloscope.

LAB ACTIVITY: ELECTRICAL STIMULATION: ANALGESIA

DESCRIPTION

Electroanalgesia is arguably the most common use of therapeutic electricity. The use of therapeutic electricity for analgesia is often referred to as transcutaneous electrical nerve stimulation or TENS; however, all forms of therapeutic electricity that do not use implanted or needle electrodes are “transcutaneous,” and many forms stimulate nerves. Therefore, the term TENS should be discouraged. Although there are hundreds of different types of electrical stimulators available for use, there are essentially three levels in the body that may be affected.

The first level is the spinal gate. This level is activated by increasing the input to the spinal cord from large-diameter afferent neurons. The second level is referred to as the central bias mechanism, where intense small fiber afferent input activates a negative feedback loop through connections in the midbrain. Finally, some forms of electrical stimulation appear to stimulate the production of endogenous opiates, the endorphins.

Although stimulators have many different waveforms and modulations, there is no evidence that an “optimal” waveform exists. It is impossible to predict for an individual patient what type of current, electrode configuration, amplitude of stimulation, and so on will provide relief of pain. Therefore, electroanalgesia is somewhat of a trial-and-error phenomenon. This does not mean the approach should be haphazard; a systematic approach, based on clinical experience, is best.

Generally, there are three types of stimulation for electroanalgesia: conventional, low frequency, and hyperstimulation. Conventional generally has a pulse rate of 10–100 pps and is applied at an amplitude between sensory and motor thresholds. Low-frequency stimulation has a pulse rate of 1–5 pps, and an amplitude between motor and pain thresholds. Hyperstimulation generally uses a monophasic PC at a frequency of 1–128 pps and an amplitude to pain tolerance. It is often referred to as point stimulation.

PHYSIOLOGIC EFFECTS

Depolarization of peripheral nerves

THERAPEUTIC EFFECTS

Inhibition of pain perception

INDICATIONS

The obvious indication for electroanalgesia is pain. However, the cause of the pain should be identified prior to the use of electrical stimulation, and it must be remembered that the modulation of pain is not treating the cause of the pain.

CONTRAINDICATIONS

- Pregnancy
- Implanted electrical pacing devices (e.g., cardiac pacemaker, bladder stimulator)
- Cardiac arrhythmia
- Over the carotid sinus area
- Hypersensitivity (i.e., the patient who has a strong aversion to electricity, or the patient with certain types of catheters or shunts)

ELECTRICAL STIMULATION: ANALGESIA			
PROCEDURE	EVALUATION		
	1	2	3
1. Check supplies.			

a. Obtain towels or sheets for draping, and conductant.			
b. Check stimulator, electrodes, and cables for charged battery, broken or frayed insulation, and so on.			
c. Ensure the amplitude controls are at zero.			
2. Question patient.			
a. Verify identity of patient (if not already verified).			
b. Verify the absence of contraindications.			
c. Ask about previous treatments for current condition, and check treatment notes.			
3. Position patient.			
a. Place patient in a well-supported, comfortable position.			
b. Expose body part to be treated.			
c. Drape patient to preserve patient's modesty, and protect clothing, but allow access to body part.			
4. Inspect body part to be treated.			
a. Check light touch perception.			
b. Assess function of body part (e.g., ROM, irritability).			
5a. Apply conventional electrical stimulation.			
a. Place conductant on electrodes as indicated, secure electrodes to patient.			
b. Remind the patient to inform you when he or she feels something. Do not tell the patient what he or she will feel; for example, do not say, "Tell me when you feel a buzz or tingle."			
c. Adjust the pulse rate, pulse width, and mode of stimulation to desired settings if possible.			
d. Turn on the stimulator, and increase the amplitude slowly. Monitor the patient's response, not the stimulator.			
e. After the patient reports the onset of the stimulus, adjust the amplitude to a comfortable level, but make sure it is below motor threshold. If it is impossible to achieve suprasensory threshold stimulation without a motor response, turn the stimulator off and move the electrodes to another location.			
f. Set a timer for the appropriate treatment time and give the patient a signaling device. Make sure the patient understands how to use the signaling device.			
g. Recheck the patient after about 5 minutes. If the sensation has diminished, adjust the amplitude appropriately.			
5b. Apply low-frequency electrical stimulation.			
a. Place conductant on electrodes as indicated, secure electrodes to patient.			

b. Remind the patient to inform you when he or she feels something. Do not tell the patient what he or she will feel; for example, do not say, "Tell me when you feel a buzz or tingle."			
c. Adjust the pulse rate, pulse width, and mode of stimulation to desired settings if possible			
d. Turn on the stimulator, and increase the amplitude slowly. Monitor the patient's response, not the stimulator.			
e. After the patient reports the onset of the stimulus, adjust the amplitude to a comfortable level above motor threshold. The contraction should be just a twitch, not a strong contraction.			
f. Set a timer for the appropriate treatment time and give the patient a signaling device. Make sure the patient understands how to use the signaling device.			
g. Recheck the patient after about 5 minutes. If the sensation has diminished, adjust the amplitude appropriately.			
5c. Apply hyperstimulation electrical stimulation.			
a. Place conductant on "inactive" electrode; have the patient hold the electrode in his or her palm. Apply the conductant to points to be stimulated.			
b. If using electrical resistance to locate stimulation points, set sensitivity of ohm meter; set pulse rate, polarity, and length of stimulation to desired settings.			
c. Move the "active" electrode slowly in the area of the point to be stimulated until the area of minimal resistance is found; the pressure applied to the electrode must be constant.			
d. Tell the patient to report when the amplitude of stimulation is as high as he or she can tolerate. Activate the stimulation current, and increase the amplitude slowly. Monitor the patient's response, not the stimulator.			
e. After the patient reports that the stimulus is as much as he or she can tolerate, maintain constant pressure on the electrode. Stimulate the point two or three times, for 15–30 seconds each time.			
f. Repeat the process for each point to be stimulated.			
6. Complete treatment.			
a. When the treatment time is over, turn the intensity control to zero, and move the generator away from the patient; remove conductant with a towel.			
b. Remove material used for draping; assist the patient in dressing as needed.			
c. Have the patient perform appropriate therapeutic exercise as indicated.			
d. Clean the treatment area and equipment according to normal protocol.			
7. Assess treatment efficacy.			
a. Ask the patient how the treated area feels.			
b. Visually inspect the treated area for any adverse reactions.			

c. Perform functional tests as indicated.

LAB ACTIVITY: ELECTRICAL STIMULATION: REEDUCATION

DESCRIPTION

Electrical stimulation may be used to assist a patient in regaining the ability to voluntarily control a normally innervated muscle. Sometimes following surgery, a patient temporarily loses the ability to produce a muscle contraction. Probably the most common loss is of the quadriceps femoris following knee surgery. In addition, if a patient has undergone a tendon transfer, he or she may have difficulty recruiting the muscle to perform the new joint action.

The mechanism by which electrical stimulation aids in the recovery of volitional control of skeletal muscle is not clear, nor is the reason why volitional control is lost following surgery. The probable method of action is via stimulation of joint, muscle, and skin proprioceptors when the muscle produces joint motion.

PHYSIOLOGIC EFFECTS

Depolarization of peripheral nerves

THERAPEUTIC EFFECTS

Recovery of volitional control of skeletal muscle

INDICATIONS

The primary indication is loss of volitional control of a skeletal muscle following surgery or a tendon transfer.

CONTRAINDICATIONS

- Pregnancy
- Implanted electrical pacing devices (e.g., cardiac pacemaker, bladder stimulator)
- Cardiac arrhythmia
- Over the carotid sinus area
- Hypersensitivity (i.e., the patient who has a strong aversion to electricity, or the patient with certain types of catheters or shunts)

ELECTRICAL STIMULATION: REEDUCATION

PROCEDURE	EVALUATION		
	1	2	3
1. Check supplies.			
a. Obtain towels or sheets for draping, and conductant.			
b. Check stimulator, electrodes, and cables for charged battery, broken or frayed insulation, etc.			

c. Verify that the intensity control is at zero.			
2. Question patient.			
a. Verify identity of patient (if not already verified).			
b. Verify the absence of contraindications.			
c. Ask about previous exposure to electrotherapy; check treatment notes.			
3. Position patient.			
a. Place patient in a well-supported, comfortable position.			
b. Expose body part to be treated.			
c. Drape patient to preserve patient's modesty, and protect clothing, but allow access to body part.			
4. Inspect body part to be treated.			
a. Check light touch perception.			
b. Assess function of body part (e.g., ROM, irritability).			
5. Apply electrical stimulation for reeducation.			
a. Place conductant on electrodes as needed; secure electrodes to patient. Electrode location will vary depending on desired effect. Usually, the ideal location for the active electrode is over the motor point of the target muscle or the peripheral nerve trunk that supplies the target muscle.			
b. Remind the patient to inform you when he or she feels something. Do not tell the patient what he or she will feel; for example, do not say, "Tell me when you feel a buzz or tingle."			
c. Adjust the pulse rate, pulse width, and mode of stimulation to desired settings if possible.			
d. Turn on the stimulator, and increase the amplitude slowly. Monitor the patient's response, not the stimulator.			
e. After the patient reports the onset of the stimulus, adjust the amplitude to a comfortable level above motor threshold. Encourage the patient to try to volitionally contract the muscle before the stimulator does, and increase the force during the stimulation.			
f. Continue to monitor the patient during the duration of the treatment.			
6. Complete treatment.			
a. When the treatment time is over or the patient is able to control the muscle contraction, turn the intensity to zero, and move the generator away from the patient; remove conductant with a towel.			
b. Remove material used for draping; assist the patient in dressing as needed.			
c. Have the patient perform appropriate therapeutic exercise as indicated.			

d. Clean the treatment area and equipment according to normal protocol.			
7. Assess treatment efficacy.			
a. Ask the patient how the treated area feels.			
b. Visually inspect the treated area for any adverse reactions.			
c. Perform functional tests as indicated.			

LAB ACTIVITY: ELECTRICAL STIMULATION: STRENGTHENING

DESCRIPTION

Electrical stimulation is often used for increasing skeletal muscle strength by itself or in conjunction with active exercise. However, there is no evidence that the electrical stimulation by itself or in conjunction with active exercise is better than active exercise alone for muscle strengthening. Also, the increase in tension-developing capacity does not transfer to functional activities. Because of this, it is sometimes referred to as “electrical stimulation to increase isometric force development capacity.”

PHYSIOLOGIC EFFECTS

Depolarization of peripheral nerves

THERAPEUTIC EFFECTS

Increase in isometric force development capacity

INDICATIONS

The primary indication is muscle weakness. However, electrical stimulation is sometimes used in an attempt to prevent disuse atrophy during immobilization of a limb.

CONTRAINDICATIONS

- Pregnancy
- Implanted electrical pacing devices (e.g., cardiac pacemaker, bladder stimulator)
- Cardiac arrhythmia
- Over the carotid sinus area
- Hypersensitivity (i.e., the patient who has a strong aversion to electricity, or the patient with certain types of catheters or shunts)

ELECTRICAL STIMULATION: STRENGTHENING			
PROCEDURE	EVALUATION		
	1	2	3
1. Check supplies.			

a. Obtain towels or sheets for draping, and conductant.			
b. Check stimulator, electrodes, and cables for charged battery, broken or frayed insulation, etc.			
c. Verify that the intensity control is at zero.			
2. Question patient.			
a. Verify identity of patient (if not already verified).			
b. Verify the absence of contraindications.			
c. Ask about previous exposure to electrotherapy; check treatment notes.			
3. Position patient.			
a. Place patient in a well-supported, comfortable position.			
b. Expose body part to be treated.			
c. Drape patient to preserve patient's modesty, and protect clothing, but allow access to body part.			
4. Inspect body part to be treated.			
a. Check light touch perception.			
b. Assess function of body part (e.g., ROM, irritability).			
5. Apply electrical stimulation for muscle strengthening.			
a. Place conductant on electrodes as indicated; secure electrodes to patient. Electrode location will vary depending on desired effect. Usually, the ideal location for the active electrode is over the motor point of the target muscle, or the peripheral nerve trunk that supplies the target muscle.			
b. Remind the patient to inform you when he or she feels something. Do not tell the patient what he or she will feel; for example, do not say, "Tell me when you feel a buzz or tingle."			
c. Adjust the pulse rate, pulse width, and mode of stimulation to desired settings if possible.			
d. Turn on the stimulator, and increase the amplitude slowly. Monitor the patient's response, not the stimulator.			
e. After the patient reports the onset of the stimulus, adjust the amplitude to as high a level as the patient can tolerate.			
f. Set a timer for the appropriate treatment time and give the patient a signaling device. Make sure the patient understands how to use the signaling device.			
g. Recheck the patient after about 5 minutes. If the sensation has diminished, adjust the amplitude appropriately.			
6. Complete treatment.			
a. When the treatment time is over, turn the intensity control to zero, and move the generator away from the patient;			

remove conductant with a towel.			
b. Remove material used for draping; assist the patient in dressing as needed.			
c. Have the patient perform appropriate therapeutic exercise as indicated.			
d. Clean the treatment area and equipment according to normal protocol.			
7. Assess treatment efficacy.			
a. Ask the patient how the treated area feels.			
b. Visually inspect the treated area for any adverse reactions.			
c. Perform functional tests as indicated in Figure 5–30 . Russian current with an interburst interval. Darkly shaded area represents total current, and light shading indicates total current without the interburst interval.			