CONTINUING EDUCATION
for Physical Therapists

EVIDENCE-BASED ELECTRICAL STIMULATION

PDH Academy Course #PT-1801 | 3 CE HOURS

Course Abstract

This course examines the evidence behind the use of electrical stimulation in therapeutic settings. It covers muscle re-education, spasticity management, edema management, wound healing, pain control, and iontophoresis.

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Approvals

To view the states that approve and accept our courses, visit www.pdhtherapy.com/physical-therapy/.

Target Audience & Prerequisites

PT, PTA – no prerequisites

Learning Objectives

By the end of this course, learners will:

- ❏ Recognize at least 2 specific principles of electricity
- ❏ Identify at least 2 physiological effects of electrical stimulation
- ❏ Recall at least 4 contraindications associated with electrical stimulation
- ❏ Recognize at least 2 evidence-based principles pertaining to each of the 6 clinical applications of electrical stimulation

Timed Topic Outline

I. History of Electrical Stimulation (5 minutes)

II. Principles of Electricity (5 minutes)

III. Physiology of Electrical Stimulation (5 minutes)

IV. Contraindications and Applications (150 minutes)
Muscle Re-education; Spasticity Management; Edema Management; Wound Healing; Pain Management; Iontophoresis

V. Conclusion, References, and Exam (15 minutes)

Delivery Method

Correspondence/internet self-study with interactivity, including a provider-graded multiple choice final exam. *To earn continuing education credit for this course, you must achieve a passing score of 80% on the final exam.*

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Course Author Bio and Disclosure

Dawn T. Gulick, PT, PhD, ATC, CSCS, is a Professor of Physical Therapy at Widener University in Chester Pennsylvania. She has been teaching for over 20 years. Her areas of expertise are orthopedics, sports medicine, modalities, and medical screening. As a clinician, she has owned a private orthopedic/sports medicine practice. She also provides athletic training services from the middle school to elite Olympic/Paralympic level. As a member of the Olympic Sports Medicine Society, Dr. Gulick has provided medical coverage at numerous national and international events. As a scholar, Dr. Gulick is the author of 4 books (*Ortho Notes, Screening Notes, Sport Notes, Mobilization Notes*), 4 book chapters, and over 50 peer-reviewed publications, and has made over 100 professional and civic presentations. She is the developer of a mobile app called iOrtho+ (Apple, Android, & PC versions), and the owner of a provisional patent.

Dr. Gulick earned a Bachelor of Science in Athletic Training from Lock Haven University (Lock Haven, PA), a Master of Physical Therapy from Emory University (Atlanta, GA), and a Doctorate of Philosophy in Exercise Physiology from Temple University (Philadelphia, PA). She is an AMBUCS scholar and a member of Phi Kappa Phi Honor Society. As a licensed physical therapist, she has direct access authorization. She also is a certified strength and conditioning specialist.

Dawn T. Gulick sells iOrtho+, a mobile app, through Therapeutic Articulations LLC; receives royalties as an author with F. A. Davis Publishing; and received a stipend as the author of this course. She has no other relevant financial or nonfinancial relationships to disclose.

History of Electrical Stimulation

Electrical stimulation has a very long history, dating back many centuries. The earliest applications by Egyptians, Romans, and Greeks involved using fish to generate electrical discharges. Electric eels, cat fish, and torpedo fish were used to treat a variety of pathologies. In the 18th century, Luigi Galvani discovered that passing a current through the spinal cord of a frog resulted in muscle contractions.

Michael Faraday dedicated his life to the study of electromagnetism. Through his work, by the end of the 19th century, electro-stimulation was being used in a therapeutic manner. In the mid-20th century, electrical stimulation was popularized through use in sport. Advances in neurology and muscle physiology helped to identify the importance of stimulation of fast and slow muscle fibers. Concepts such as iontophoresis were used and stimulators became small enough to be portable. Today, electrical stimulation is used in a plethora of diagnostic and therapeutic methods. Muscle re-education, edema management, wound healing, pain control, and iontophoresis are a few of the techniques discussed in this course.

Principles of Electricity

Prior to addressing the physiologic influence of electrical stimulation, it is important to define the terminology associated with electrophysiology. Below are a few basic terms needed to effectively apply electrical principles to therapeutic interventions:

- **Cation = (+) ion**
- **Anion = (-) ion**
- **Cathode = (-) electrode**
- **Anode = (+) electrode**

Furthermore, current is defined as direct (DC) or alternating (AC). Direct current vs alternating current wave forms are displayed in figure 1.

DC is a continuous, uninterrupted, unidirectional flow of charged particles. It is also known as "Galvanic" current (HVGS = high volt galvanic stimulation) and results in a polar effect under each electrode.

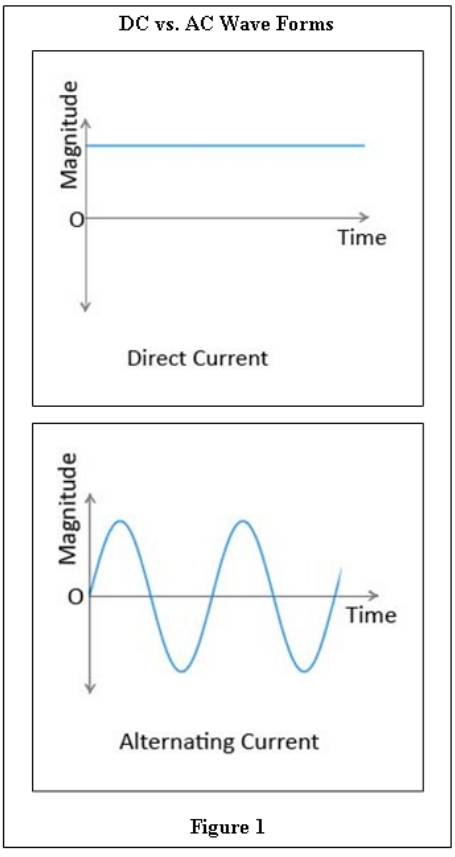


Figure 1

AC is a continuous, uninterrupted, bidirectional flow of charged particles. This is analogous to fluid in a closed system moving back and forth and results in no net accumulation of ions, i.e. no polar effect under either electrode. Alternating current is also known as "Faradic" and is a biphasic wave. Examples of AC, symmetrical, biphasic waves are shown in figure 2. However, AC waves can also be asymmetrical as shown in figure 3. When a wave form is asymmetrical it simply means the shapes are different. In spite of the different shapes, the area within the shape may be equal (balanced) or unequal (unbalanced), as also shown in figure 3. The unbalanced state will result is a slight polarity of the alternating current but it is rarely equivalent to that of a direct current.

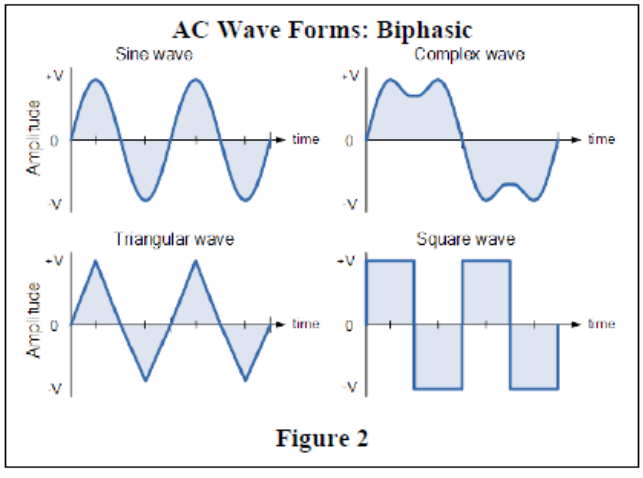


Figure 2

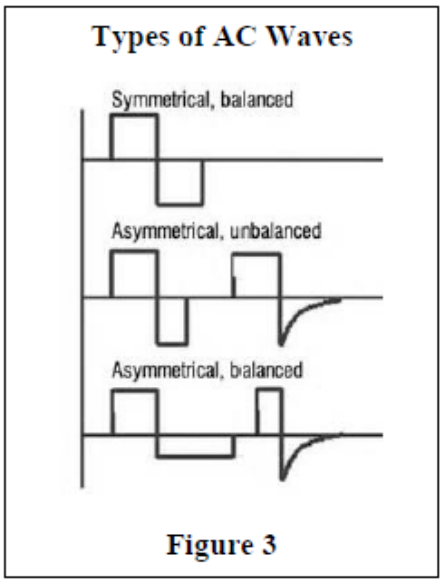


Figure 3

Other terms used to describe wave characteristics include *phase duration, pulse duration, peak amplitude, rise time,* and *duty cycle.*

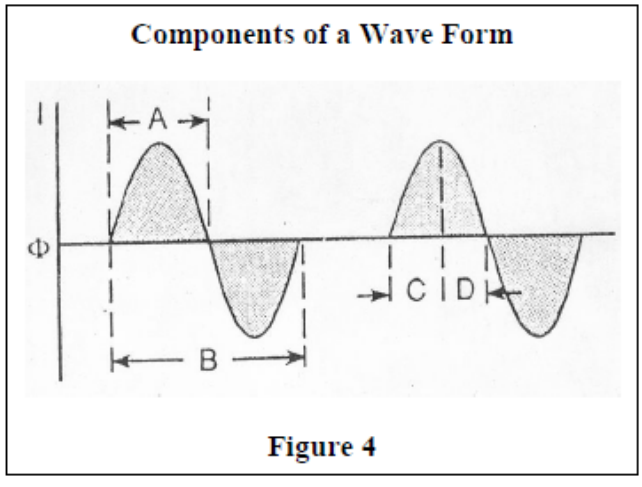


Figure 4

Phase duration is the time elapsed between the beginning and the end of one pulse of a wave (figure 4A).

Pulse duration is the time elapsed between the beginning and end of all phases of a single pulse (figure 4B). Sometimes pulse duration is referred to as pulse width.

Peak amplitude is the maximal current reached in any given phase. It can be the maximal current in either a positive or negative deflection. The area under the curve of a wave form is the product of the duration on the x-axis and the amplitude on the y-axis. Kramer et al (1984), Walmsley et al (1984), Snyder-Mackler et al 1989) have all published works supporting the asymmetric over the symmetric waveform for maximal quadriceps force production. There has been

a near-linear relationship identified between "current intensity" and "force of contraction" (Ferguson et al 1989, Underwood et al 1990). However, by increasing the duration of the stimulus (figure 5, 2nd wave form), the intensity of the stimulus can be reduced to achieve the same stimulus output. Since high intensities can result in increased heat production, the advantage of using a greater stimulus duration is to allow the intensity to be lower for a given output (figure 5).

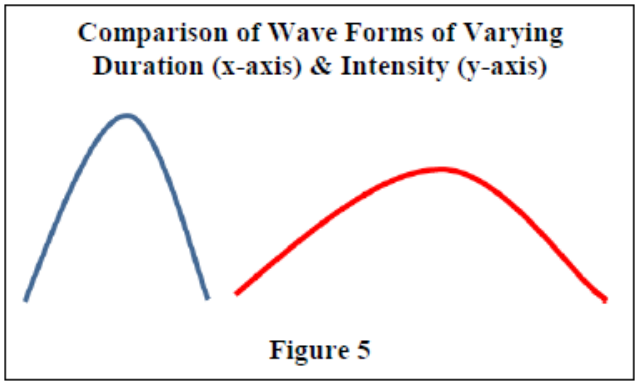


Figure 5

Rise time is also known as ramp time (figure 4C). It is the time from the onset of the current to the peak amplitude in a phase. Rise time is part of the "on time" of the stimulus. A rapid rise time can be used when increased power is the desired goal. By definition, power is force divided by time. Thus, the faster the onset of the stimulus, the more powerful the contraction. However, a slow rise time of a stimulus allows a patient to work with the stimulus to achieve a strong contraction. It is also helpful when patients are a bit frightened by the use of electrical current. A 1-2 second rise time gives them a chance to build up to the maximal intensity. As a clinician, it is important to recognize that rise time is part of the total "on time." For example, if the rise time is 3 seconds and the total time is 10 seconds, the patient is only a maximal intensity for 7 seconds. Fall time (figure 4D) is also known a decay time, the time from peak current back to baseline.

Duty cycle is defined as the ratio of "on time" to "total time" of the stimulus. Duty cycle is an important component of muscle re-education. The ratio of the duty cycle will be discussed in greater detail in the muscle re-education section.

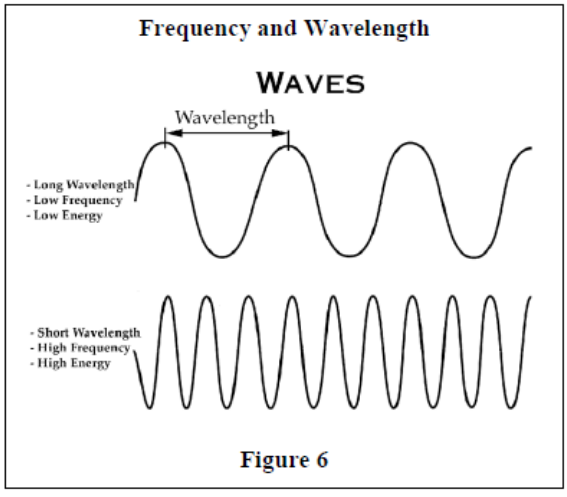


Figure 6

Frequency of electrical stimulation is defined as the number of pulses (complete oscillations) per second (pps). The unit of measure for frequency (pulse rate) is hertz (Hz). The association between frequency and the wavelength is inversely related. As the frequency increases, the wavelength becomes shorter (figure 6).

Multiple parameters combine to influence the response to electrical stimulation. The next three tables look at three parameters in isolation, to differentiate between them.

When frequency changes, so does the perception of the stimulus. Table 1 of pulse rate and frequency perception provides insight into what the patient feels as the frequency of stimulation changes from the ability to identify individual pulses to tingling, a visible contraction to tetany and paresthesia.

Table 1: Pulse Rate / Frequency Perception
1-5 pps = individual pulses
5-10 pps = increased pulsing
10-20 pps = mild tingling
20-50 pps = significant tingling/contraction
50-100 pps = tetany
>100 pps = paresthesia

Intensity of stimulation can also be described in a similar way (table 2). This is important because on many electrical stimulation devices, the intensity dial is arbitrary, i.e. numbered 0-10 with no indication of the actual output in amps. For example, an intensity of 7 on one device will not equate to a 7 on another device. As the battery charge fades, the electrode placement changes, or the electrode begins to break down, the intensity of the stimulation may be altered. Although it may appear strange to encourage the qualitative description of the stimulus and not the quantitative value, it is important for the clinician to describe intensity by the desired outcome.

Table 2: Intensity Perception
Subsensory = barely perceptible tingling (5-10 mA)
Sensory = perceptible tingling (10-20 mA)
Motor = muscle contraction (20-60 mA)
Noxious = uncomfortable & intolerable (>80 mA)

Table 3: Duration Perception
Barely perceptible tingling (40 µsec)
Deeper tingling (80 µsec)
Motor response (120 µsec)
Paresthesia & tetany (> 160 µsec)

Finally, stimulus duration is a measure of time (table 3). It is sometimes erroneously referred to a pulse width because it is the time from the beginning to the end of a pulse. As previously stated, the longer the duration of the stimulus for a given intensity, the greater the charge of the stimulus, i.e. phase charge is the area under the curve.

Physiology of Electrical Stimulation

Action potential propagation depends upon fiber diameter and myelination (table 4).

Table 4: Fiber Types, Description, & Conduction Velocity		
Type	**Description**	**Conduction Velocity**
Type A fibers	Large & myelinated (sensory & motor)	4 – 120 m/sec
Type B fibers	Small & myelinated (sensory & motor)	3 – 15 m/sec
Type C fibers	Small & unmyelinated (sensory)	0.5 – 4 m/sec

Likewise, tissue temperature is directly related to conduction velocity. Thus, when temperature goes up, so does conduction velocity.

Muscle fibers respond differently to voluntary stimulus than that of an electrical stimulation unit. Fast twitch fibers are innervated by alpha fast twitch neurons and are responsible for producing force needed for power and strength activities. Slow twitch fibers are innervated by alpha slow twitch neurons and are responsible for sustained contractions. Normal voluntary contractions recruit slow twitch fibers first and then sequentially add fast twitch fibers until sufficient force can be generated to perform the task desired. However, electrical stimulation recruits fast twitch fibers first. This is actually a convenient process since when an injury/pathology occurs, the fast twitch fibers are the first to atrophy. Thus, using electrical stimulation for muscle re-education challenges the fibers which are in the greatest need of strengthening.

Electrical stimulation is capable of producing changes at the cellular, tissue, segmental, and systemic levels. Table 5 below summarizes the changes that occur at each of these levels.

Table 5: Physiologic Changes with Electrical Stimulation			
Cellular	**Tissue**	**Segmental**	**Systemic**
• Excitation of peripheral nerves • Changes of membrane permeability • Modification of fibroblast/clast formation • Modification of osteoblast/clast formation • Modification of microcirculation • Alteration of protein & blood-cell concentration • Alteration of enzymatic activity • Alteration in protein synthesis • Modification of mitochondrial size & concentration	• Skeletal muscle contraction; effects muscle strength, contraction speed, fatigability • Smooth muscle contraction or relaxation & effect on blood flow • Tissue regeneration (bone, ligaments, connective & dermal) • Tissue remodeling (softening, stretching, decreasing viscosity & fluid absorption) • Changes in tissue thermal & chemical balance	• Muscle group contractions that effect joint mobility • Muscle pumping action that effects lymphatic drainage, venous & arterial blood flow	• Analgesic effects via beta-endorphins, enkephalins, dopamines • Analgesic effects via neurotransmitters; serotonin, substance P • Circulatory effects via polypeptides; VIP vasoactive intestinal polypeptides)

Contraindications and Applications

Prior to discussing the indications for electrical stimulation, it is important to cover the contraindications and precautions (table 6).

Table 6: Electrical Stimulation Contraindications & Precautions	
Contraindications	**Precautions**
Not in the thoracic region of an individual with an arrhythmia, congestive heart failure, or recent myocardial infarction	In the area of the uterus during pregnancy (effects are unknown)
Not with a cardiac pacemaker, defibrillator, or deep brain stimulator	In conjunction with diathermy
Not over the carotid sinus or transcerebral or individuals with epilepsy	In the area of the bladder (may interfere with normal function)
Not over cancerous (malignant) regions	Over scar tissue
Not over staples or external pins	In patient with history of metastatic disease
Not on a patient with TB	In patient who cannot provide feedback
Not in the area of a thrombi	In a patient with poor sensation
	

As we embark on the discussion of the various types of electrical stimulation, there are a few points worthy of noting. Under the umbrella of electrical stimulation we have AC muscle re-education, DC wound care, TENS (transcutaneous electrical nerve stimulation) pain management, and iontophoresis. However the nomenclature can be confusing. For example, all TENS units are electrical stimulation units but not all electrical stimulation units are TENS. Likewise, electrical stimulation units can be powered by batteries or an electrical outlet. Yet the source of the power for the unit is **not** necessarily the output of the unit. For instance, an electrical stimulation unit plugged into the wall is receiving its power via an AC, but the unit can be set up to distribute a DC. A power inverter in the "guts" of the unit makes that conversion.

That being said, let's get to the heart of the course… clinical application.

Muscle Re-Education

The application of electrical stimulation to augment voluntary muscle contraction has been used for many decades. There are a few pearls that are important to appreciate before discussing the most appropriate treatment parameters.

First, the order of recruitment is different for voluntary versus ES-induced contractions. Electrical stimulation (ES) reverses the order of recruitment of motor units observed with voluntary muscular contraction (Paillard, Noe, Passelergue, & Dupui; 2005). With ES, large motor units are recruited before small motor units, whereas voluntary muscle contractions recruit small motor units before large. This keeps us from slamming our coffee cup into our teeth each morning: sequentially adding more and more small motor units to a task allows us to modulate our force production to meet the needs without going overboard. But since fast twitch fibers atrophy quicker than slow twitch fibers, it is actually advantageous to have ES target the fast twitch fibers. These fibers are the ones in greatest need of stimulation.

Second, ES can only elicit a contraction up to 80% to 90% of the maximal voluntary contraction. Thus, ES cannot produce supra-normal contractions and there is no added benefit of ES when weakness is not present (Paillard, Noe, Passelergue, & Dupui; 2005).

This concept is supported by the work of Holcomb, Rubley, and Girouard (2007). They studied 14 healthy individuals over three testing conditions for 10 seconds each. Condition 1 was maximal voluntary knee extension force. Condition 2 was maximal knee extension with ES to maximal comfort (33 pps, 2 second ramp, 10 seconds on, 30 seconds off). Condition 3 was passive knee extension with ES only. Mean torque values are displayed in table 7.

Table 7: Outcome Data of Holcomb, Rubley, & Girouard (2007)			
Condition	**Treatment**	**Mean Torque**	**% of Maximal Contraction**
1	Maximal voluntary contraction	1000.1 ± 167.4	
2	Maximal voluntary contraction with ES	1015.0 ± 191.1	101.5%
3	ES to maximal comfort only	430.4 ± 121.2	43%

When comparing the outcomes, it is important to recognize when ES was imposed on voluntary muscle contraction, 101.5% of the maximal voluntary isometric contraction (MVIC) was achieved. But when ES was applied without voluntary effort, only 43% of MVIC was achieved.

Another study by Palmieri-Smith, Thomas, Karvonen-Gutierrez, and Sowers (2010) assessed the influence of ES on quadriceps strength/activation in women with osteoarthritis. They rendered treatment three times per week of four weeks. The treatment parameters were similar to Piva et al (2007) with a slightly lower frequency of 50 pulses per second. However, no improvement in quad strength, WOMAC score, or 40 foot walk test occurred.

So what was different? In Palmieri-Smith et al there was no voluntary contractions with the ES. This may be a critical component to achieving strength gains. The take-home message is one must always remember to instruct the patient to "work with" the ES to achieve maximal strength benefits. As evidenced by the percentages above, the benefits are enormous.

Third, ES strength gains are greater when the current

is strong enough to produce a maximal contraction. Paillard, Noe, Passelergue, and Dupui (2005) demonstrated the combination of ES and voluntary contraction can theoretically activate more motor units than voluntary contraction alone, i.e. an increase of the contraction force. In a therapeutic context, concentric training programs using ES superimposition compensate for volume and muscle strength deficit with more efficiency than programs using either separately. With eccentric activity, ES increased torque over voluntary conditions with velocities from (-) 30°/sec to (-) 120°/sec. Voluntary eccentric actions are limited by neural inhibition mechanism protecting muscle from extreme tension (simulate GTO and inhibit 1a afferents). ES stimulates cutaneous receptors to decrease neural inhibition & increase motor unit recruitment to increase force production.

The indications for muscle ES include the enhancement of a muscle contraction, strengthening a muscle for correct use, re-educating a muscle after surgical transfer, increasing blood flow, facilitating range of motion (ROM), and the performance of functional activities. The parameters selected for optimal outcomes are as follows:

- **Current** = pulsed biphasic (AC)
- **Amplitude** = maximum tolerable
- **Rate of stimulation** = 30-50 pps
- **Duty cycle** = 1:3-6
- **Ramp** = 2 sec for strength; none for power
- **# of contractions** = 10-20 maximal contractions

Any biphasic wave form appears to be effective as long as the phase charge (amplitude x duration) is sufficient to command a maximal contraction.

The rate of stimulation is dictated by that which is needed to achieve tetany. In other words, we want a strong by smooth contraction not individual muscle twitches. With an increasing frequency, the summation of the twitches results in a smooth tetanizing contraction (figure 7). The upper extremities tend to need a lower frequency than the lower extremities, hence the range of 30 to 50 pulses per second.

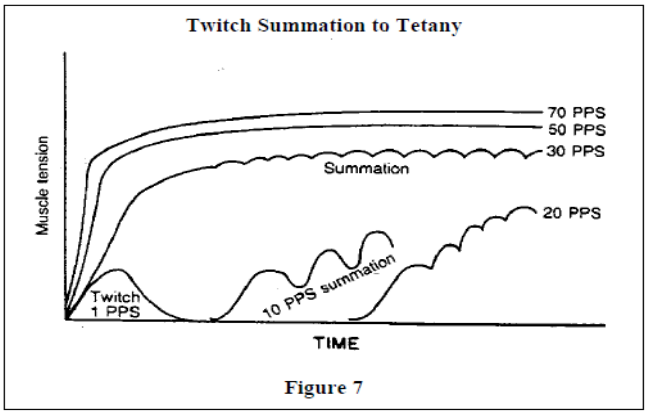


Figure 7

As for duty cycle, the ratio depends on the goal. If increased muscle endurance is the desired outcome, a low duty cycle would be desirable, i.e. 1:2 or 1:3 to minimize the time for recovery. If increased strength is desired, a higher ratio of 1:5 or 1:6 would be recommended. The body of literature by Kotz has recommended these parameters to allow for sufficient fuel recovery for the next contraction to be maximal. Contractions in less time will result in sub-maximal contractions and less than optimal strength gains. Figure 8 displays the relationship of recovery time to the strength of subsequent contractions. Thus, prolonged, low force level contractions can increase endurance via increased muscle oxidative enzymes, increased myoglobin (O2 transport protein), increased number of capillaries, and increased oxidation of fats. When using ES for intermittent, high force level contractions to increase strength, there is an increased content of muscle contractile proteins and an increased number of cross-bridges formed to enhance force production.

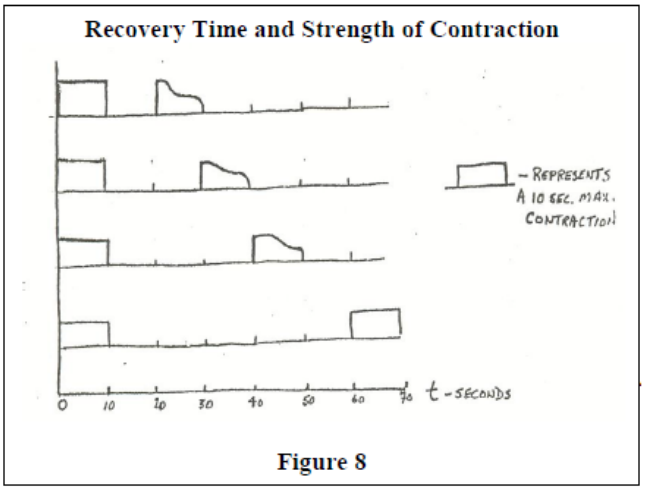


Figure 8

There is ample evidence to support the physiologic changes that occur with ES. Parker, Bennett, Hieb, Hollar, and Roe (2003) studied the strength response in human quadriceps femoris muscle via two ES programs. Twenty-seven subjects were randomly assigned to one of three groups: control, ES twice per week, ES three times per week. The ES groups received 10 minute treatments at a frequency of 50 pps at a 1:6 duty cycle (10 sec on: 50 sec off). Treatments were rendered over four weeks. The maximal voluntary isometric contraction (MVIC) was assessed pre-test and after each week of treatment. The results demonstrated significant increases (P = .021) in the quadriceps femoris muscle strength when ES was rendered three times per week.

Obajuluwa (1991) studied the effect of 10 weeks of ES on quadriceps muscle strength and thigh circumference. Twenty-four men had their right quadriceps treated three times per week and the left quadriceps was the control (no treatment).

Both strength and thigh circumference significantly increased with ES. The strength changes were apparent at five weeks and the increase in thigh circumference at 10 weeks. Likewise, Piva, Goodnite, Azuma, Woollard, Goodpaster, Wasko, and Fitgerald (2007) examined the influence of ES and volitional exercise on individuals with rheumatoid arthritis. They conducted 60 sessions over 16 weeks using ES and exercise. ES parameters were 75 pulses per second (4 second ramp, 6 second max contraction) with a 50 second rest (1:6 ratio). Repetitions were progressed from 10 contractions per leg to 30 repetitions over a two week period. The outcomes were an 18.5% increase in quadriceps strength and a 24% improvement in the sit-to-stand test. Furthermore, as per CT imaging, those patients who completed the greatest number of ES and exercise sessions had the greatest gains in muscle cross sectional area. This is not new physiologic data. We know this to be true for active strengthening but this research simply confirms that what we know about active strengthening also applies to ES.

Avramidis, Karachalios, Popotonasios, Sacorafas, Papathanasiades, and Malizos (2011) sought to answer the question "Does electric stimulation of the vastus medialis influence rehab after total knee replacement?" Patients were randomly divided into two groups (n=70). Group A received ES and physiotherapy while group B received physiotherapy only for six weeks. Group A had a statistically significant improvement in walking speed, Oxford Knee Score, and American Knee Society function score at 6 weeks (p=0.003, 0.001, and 0.001, respectively) and at 12 weeks (all p=0.001). Group A also had a statistically significant increase in the SF-36 physical component summary score at 6, 12, and 52 weeks (all p=0.001). Thus, the results support the efficacy of ES of the vastus medialis muscle in addition to conventional physiotherapy improves functional recovery and early rehabilitation after TKR. Steven-Lapsley et al (2011) compared the application of ES within 48 hours of a total knee arthroplasty (TKA) to standard rehabilitation (n=66). ES was delivered via a symmetrical, biphasic current at 50 pps for 15 seconds with a 3 second ramp and 45 seconds off time. This represents a 1:4 duty cycle. Treatment was applied twice daily at the maximum tolerable intensity for 15 contractions each over six weeks. Both groups completed a standard rehab protocol. Outcome data for muscle strength, functional performance, and self-report measures were obtained before surgery and 3.5, 6.5, 13, 26, and 52 weeks after TKA. Interestingly, at 3.5 weeks after surgery, the TKA group with ES was significantly better for all outcome measures than the standard rehab group. At 52 weeks, the differences between groups were attenuated, but improvements with ES were still significant. These studies substantiate the value of ES in the recovery of joint arthroplasty. The improvement of functional activities in both the short and long-term can have a substantial influence on quality of life.

The effect of ES on vertical jump was examined by Paillard, Noe, Bernard, Dupui, and Hazard (2008). Twenty-seven healthy, trained males were randomly assigned to three groups: control, strength, and endurance. The control group received no treatment. The strength group was treated three times per week (TIW) over five weeks at 80 pulses per second (pps) for 15 minutes. The endurance group were also treated TIW for five weeks but the stimulus was 25 pps for 60 minutes. Both treatments were to the quadriceps muscle. The strength and endurance groups demonstrated increases in vertical jump of 5 and 3 cm, respectively. The increases occurred after one week of ES and remained at that level after five weeks. The authors concluded ES training for endurance did not interfere with vertical jump performance. However, endurance was not tested.

Balogun, Onilari, Akeju, and Marzouk (1993) utilized high volt (HV) ES for muscle strengthening. The study examined three different frequencies: 20, 45, and 80 pps. The right quadriceps was treated and the left was the control. The duty cycle was 1:6 (10 sec on, 50 sec off) for 10 maximal contractions. Treatment was rendered three times per week for 6 weeks. After six weeks, the results for the three groups were not significantly different with a 24% and 10% increase in isometric knee extension force in the right and left leg, respectively. The strength gains were maintained for at least three weeks. The conclusions were that HV improved strength but none of the pulse frequencies selected offered a significant clinical advantage. This is not a commonly used technique since HV is a direct current. Not only does HV tend to be uncomfortable with a polar effect under the electrode, it is more likely to produce an adverse skin reaction. In addition, with the short pulse duration of HV, a markedly greater intensity is needed to obtain a tetanizing contraction. With the increased intensity comes the byproduct of heat and potential adverse skin reactions.

When administering ES for muscle re-education, the parameters discussed are important, but so is electrode placement. To achieve a maximal contraction for a given phase charge, the impedance of the stimulus should be minimized. To do this, good skin preparation and appropriate electrode placement is imperative. Utilizing motor points, the location where the motor nerve enters the muscle, is optimal. A motor point is the point of least resistance to achieve a muscle contraction, i.e. it is the most electrically excitable location. There are two ways to identify a motor point: use a motor point reference chart, or manual motor point mapping. Motor point charts are valuable and can serve as a valuable reference but every person is unique. As such, there are subtle differences that can exist. The technique for manual motor point mapping is a simple process to ensure optimal electrode placement. For example, if one wishes to facilitate wrist extension, the sequence is outlined in table 8:

Table 8: Steps of Manual Motor Point Mapping
• Position the patient with the upper extremity supported and the forearm in pronation • Place one electrode on the upper arm of the patient • Place one electrode in the clinician's hand • Set the parameters to the desired frequency with the stimulus on continuous, i.e. do not set the duty cycle at this time (you do not want the current to cycle on and off while identifying motor points) • Clinician places one finger on the patient's forearm – this completes the circuit • Turn the intensity up until the clinician feels a notably "buzz" – it is not necessary for the patient to feel the current • While maintaining constant pressure, slowly move across the skin of the forearm – do **not** lose contact with the patient's skin • The area at which the current is greatest represents the point of minimal impedance – this is the motor point • Wipe this area with an alcohol wipe and move the electrode from the patient's upper arm to the motor point • Repeat this process to identify a second motor point • Wipe the second area with an alcohol wipe and move the electrode from the clinician to the second motor point • At this time, the clinician should set the duty cycle and increase the intensity to the achieve a motor response

This motor point mapping technique is particularly helpful if you are dealing with a denervated muscle. When a muscle is denervated, Wallerian degeneration occurs. Regeneration and collateral sprouting during the recovery process may result in the motor point moving distally. Thus, motor point charts may not be appropriate for this population. In addition, due to the physiologic changes, a long pulse duration (500-1000 µsec) is needed for denervated muscle (strength-duration curve: figure 9). AC pulse duration is typically too short to achieve a contraction. As such, a direct current is recommended.

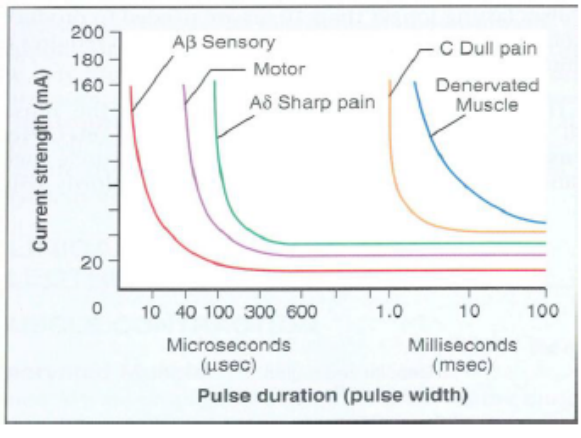


Figure 9

Attention should be given to one additional feature. When electrodes are placed on a muscle with the goal of achieving an effective contraction, the distance between the electrodes is important. The further apart the electrodes are placed, the deeper the depth of penetration. When one is working with relatively small or thin muscles, the stimulus may actually penetrate through the target muscle to the antagonist. This could result in a co-contraction instead of isolated muscle contraction of the agonist. That is not to say a co-contraction is a bad thing. In fact, co-contractions are important for joint stabilization. However, this goal can be better achieved by using two channels of ES: one channel on the agonist and one on channel on the agonist and then setting the parameters for a synchronized contraction.

Spasticity Management

Simply put, spasticity is related to central nervous system damage and is a state of increased muscle tone and an increased stretch reflex. The use of ES is directed at managing spasticity not "curing" it. The techniques put forth have been used to temporarily abate the magnitude of the tone, at best.

There are four theories in the literature upon which spasticity management is based. They are:

- Stimulation of the antagonist
- Tetanic activation of agonist to fatigue
- Alternate activation of spastic and antagonist muscle
- Transcutaneous neuromuscular stimulation

Stimulation of the antagonist. The stimulation of the peripheral nerve to antagonist involves the excitation of the Ia muscle spindle afferent fibers, action potentials travelling to the spinal cord, and excitation of interneurons which in turn inhibit the spastic muscles.

For the stimulation of the antagonist, the electrodes are placed on the antagonist to the spastic muscle, i.e. electrodes on the triceps if the biceps tone is increased. The technique has been reported to provide a 90% decrease in spasticity for 10 minutes to two hours. The rationale for the reduction in tone has been proposed to be related to post-tetanic potentiation and reciprocal inhibition. This theory was supported by a case report published by Pease (1998). A 26 year old man with spastic paraparesis, gait dysfunction, and bilateral lower extremity spastic muscle tone was the subject for the study. Before treatment, gait analysis revealed 63% of normal velocity and a crouched gait

pattern with excessive EMG activity in the hamstrings and gastrocnemius muscles. Prior physical therapy and baclofen was not effective in managing his tone. Neuromuscular Electrical Stimulation (NMES) was applied to the quadriceps and dorsiflexors two to three times per week. After three months, there was a 27% increase in gait velocity with an increase in both cadence and right step length. Right hip and bilateral knee stance position returned to normal (rather than "crouched"). The conclusion was ES to the antagonist was valuable for managing muscle spasticity.

ES was also used in conjunction with Bobath techniques for patient post cerebral vascular accident (CVA). Two groups of post-CVA patients were treated with Bobath techniques or Bobath techniques with ES (9-minutes daily for 20 sessions). Bakhtiary and Fatemy (2008) reported the combined treatment resulted in increased dorsiflexion motion, decreased plantarflexion tone using the Ashford Scale, and increased dorsiflexion strength, but there was no change in the H-reflex. The conclusion was the combination of Bobath inhibitory techniques and ES may help to reduce spasticity effectively in stroke patients.

Tetanic activation of agonist to fatigue. Tetanic activation of agonist is based on the concept of fatiguing out the spastic muscle. Thus, electrodes are placed directly on the motor points of the spastic muscle. The fatigue may be a result of neuromuscular junction fatigue or the depletion of calcium released at the post-synaptic binding sites. In either case, a 20-30% reduction in spasticity has been reported within two to three minutes that lasted for several hours.

In either of the above applications (table 9), there is one important concept to consider. The rise time of the amplitude of treatment must be long to minimize the abrupt onset of the intensity to avoid a withdrawal reflex or startle response by the patient which may increase tone. Although there is limited evidence on the management of spasticity, it is believed to be safe to render treatment daily.

Table 9	Stimulation of the Antagonist	Tetanic Activation of Agonist to Fatigue
Current	Intermittent AC	Continuous AC
Amplitude	Maximal	Maximal
Pulse Duration	As long as possible	As long as possible
Rate of Stimulation	50 pps	100-120 pps
Duty Cycle	1:2	None
Treatment Duration	10 minutes	15 minutes

Alternate activation of spastic and antagonist muscle. The third approach was simply a combination of the other two theories. The thought being, if both stimulation of the antagonist and stimulation of the agonist worked to a limited degree, perhaps the combination of the two would be better than the sum of their parts. It is an interesting concept but it has not been demonstrated to be effective.

Transcutaneous neuromuscular stimulation. Another approach to the management of spasticity is to use transcutaneous neuromuscular stimulation (TENS). Armutlu, Meric, Kirdi, Yakut, and Karabudak (2003) found high frequency TENS (100 Hz) to be successful in managing spasticity. They rendered a 20 minute treatment daily to 10 individuals with spasticity secondary to multiple sclerosis. After four weeks of treatment, there were statistically significant reductions in spasticity of both extremities as assessed by myoelectric activity and the Modified Ashworth Scale ($P < 0.05$). However, the Ambulation Index level was not improved.

Likewise, the effect of TENS to spastic plantar flexors of people who experienced a stroke was found to be successful (Laddha, Ganesh, Pattnaik, Mohanty, Mishra, 2016). It is unlikely that a single session of TENS would have any significant impact on spasticity. Thus, the researchers looked to examine the effects of five sessions per week over six weeks. The post stroke participants were randomly assigned to one of three groups: 1) task oriented exercises, 2) TENS for 30 minutes and task oriented exercises, and 3) TENS for 60 minutes and task oriented exercises. TENS treatments were performed with the electrodes placed over the common peroneal nerve. Both TENS groups resulted in reduced spasticity of the ankle plantar flexors. There was also improved walking ability but no difference in the timed up and go test (TUG) scores between groups. The authors found success but suggested an increase in the treatment duration to enhance results.

Finally, a systematic review by Mills and Dossa (2016) reviewed 14 randomized controlled trials. They reported the use of TENS in combination with active therapy (i.e. exercise and task-related training) produced better outcomes than a single therapeutic modality.

Edema Management

Edema occurs when small blood vessels become "leaky" and fluid is released into nearby tissues. As the extra fluid builds up, swelling of the tissue occurs. Trauma is one of several reasons for swelling/edema to transpire. Blood vessels are interwoven with skeletal muscle tissues. The close relationship reduces the diffusion distance for oxygen, nutrients, waste products, as well as fluid. Blood flow within muscles is influenced by contraction and relaxation. During contraction, the vessels within the muscles are compressed. Contraction results in reduced arterial inflow and increased venous outflow. Relaxation increases arterial inflow and reduces venous outflow. Muscular activity along with the presence of one-way valves in the veins helps to drive blood back to the heart. However, when trauma occurs and pain results, immobility and/or limb dependence can magnify the quantity of the edema.

ES can be used to palliate edema but the technique

applied is based on the stage of the injury and the ability to generate a voluntary contraction. There are two theories behind the management of edema: muscle pumping or a polarity.

Muscle pumping. As stated above, muscle pumping influences the movement of fluid. With slight modifications to the parameters discussed for muscle re-education, ES can be used to mitigate edema. Let's look at an example to best describe the process. Imagine an individual status-post Colles fracture. Edema is present in the wrist and hand. By placing electrodes of channel #1 on the forearm extensors and the electrodes of channel #2 on the forearm flexors, a pattern of reciprocal contractions can be achieved. The ES is used to supplement the voluntary muscle contraction to pump the fluid out of the extremity. The parameters include the use of an AC unit at a pulse rate similar to that of muscle re-education to achieve a tetanic contraction. This is usually 30-50 pps (upper extremity < lower extremity). However, the duty cycle tends to be shorter for edema management because muscle contractions are not maximal. Thus, recovery time and replacement of adenosine triphosphate (ATP) is not critical. A slow and rhythmic reciprocal contraction can be achieved by setting the device to stimulate channel #1 for 2-5 seconds followed by channel #2 for 2-5 seconds. The use of a ramp time is optional. The intensity of the stimulus should be sufficient to achieve a "comfortable" muscle contraction, it does not need to be maximal or noxious. The treatment time may range from 20-30 minutes and may be performed multiple times per day. This technique has the dual benefit of reducing edema while helping to stimulate the surrounding muscles to take the limb through the available range of motion.

Polarity. An alternative method for managing edema is to use polarity, i.e. DC. The polar reactions can have a significant impact on various tissues. The reactions at each pole are:

Anode:

- acidic reaction
- hardening effect
- vasoconstriction

Cathode:

- alkaline reaction
- softening effect
- vasodilation

Under the positive electrode, blood cells tend to clump with vasoconstriction. In contrast, the negative current results in vasodilation and repels the negatively charged blood cells and proteins. This can cause a "fluid shift." Given the diverse influence of the two polarities, two examples can help one see how the applications are very different. If one sustains a significant trauma and edema is anticipated, the immediate application of a positive current could facilitate vasoconstriction and minimize the "leaking" of vessels to prevent the edema from occurring. The prevention is typically accompanied by ice, compression, and elevation (I.C.E) to achieve a maximal effect. Whereas, if the edema is already present, the negative charge could be used to facilitate a "fluid shift." So let's look at the clinical application of the latter.

Let's imagine a second degree lateral ankle sprain that is in the subacute stage with significant edema and ecchymosis. Given the presence of the edema, there are two options for treatment: 1) reciprocal muscle pumping or 2) negative-polarity fluid shifting.

Let's explore the set-up for each in table 10:

Table 10: Edema Management Options

Reciprocal muscle pumping using AC	Negative-polarity fluid shifting using DC

• Quantify edema via volumetry, girth or figure-8 measures
• Position the patient with the ankle elevated
• Electrode placement:
  ○ Option #1
  ▪ Place channel #1 electrodes on the motor points of the ankle plantarflexors (gastroc/soleus muscles)
  ▪ Place channel #2 electrodes on the motor points of the ankle dorsiflexors (anterior tibialis muscle)
  ○ Option #2
  ▪ Place channel #1 electrodes on the motor points of the ankle invertors (anterior & posterior tibialis muscles)
  ▪ Place channel #2 electrodes on the motor points of the ankle evertors (peroneals/fibularis muscles)
• Set the pulse rate at 40-50 pps
• Set the pulse duration as wide/high as possible
• Set the "on" time at 4-5 seconds and "off" time at 4-5 seconds, i.e. duty cycle of 1:2 for each channel
• Set the intensity to achieve a strong but comfortable muscle contraction
• Set the treatment time to 20-30 minutes
 	
Note: In addition to the application of the ES with elevation, the application of an ice pack could also be considered.
	• Quantify edema via volumetry girth or figure-8 measures
	• Position the patient
	  ○ Option A = elevate ankle
	  ○ Option B = place the ankle in a plastic container of cold water
	• Place the positive electrode (anode) on the thigh – note the anode electrode should be significantly larger than the "active" negative elective. This electrode is known as the inactive or dispersive pad. By being larger, the current density is lower and the stimulus perception is much lower than that of the active electrode.
	• Negative electrodes (cathode)
	  ○ Option A = place 1-2 electrodes over the edematous region
	  ○ Option B = place the negative lead in the container of water and tape the lead to the side of the container to keep it from coming out (it is not necessary to place an electrode on the wire to complete the circuit and render a safe treatment)
	• Pulse rate at approximately 120 pps (rate has not been specifically defined in the literature)
	• Set the pulse duration as wide/high as possible
	• Set the current to continuous
	• Set the intensity to a strong but comfortable stimulus
	• Set the treatment time to approximately 30 minutes

Several studies have been published demonstrating the effective use of ES on edema management.

Mendel et al (1992) studied the use of four 30-minute sessions of high-voltage pulsed current (HVPC) administered at 120 pps on edema. The study was performed on rats with one trauma-induced extremity treated and the other trauma-induced extremity serving as the control. After two treatments, there was a significant reduction in trauma compared to the control.

Sandoval, Ramirez, Camargo, Russo, and Salvini (2015) assessed the effect of high voltage pulsed current (HVPC) via a negative polarity on acute ankle inflammation. They divided 64 rats into four groups:

- Inflamed and treated with (-) HVPC
- Inflamed and placebo HVPC
- Normal and treated with (-) HVPC
- Control

HVPC was rendered at 100 Hz with a sub-motor intensity for 45 minutes over three consecutive days. Outcome measures were pain, hind-foot volume, serum histamine and albumin levels. Although there was no different in pain or edema levels between the inflamed groups, albumin levels were reduced but were not different. Histamine level only briefly increased (24 hours) in the treatment groups. Therefore, the results did not support the use of these parameters for the treatment of acute joint inflammation. However, it does make sense that if the negatively charged albumin can be dispersed from the injured area, the accumulated fluid will move with it. The decision to use a negative polarity which facilitates vasodilation in this acute situation may have had an impact.

Dolan, Mychaskiw, and Mendel (2003) compared cool-water immersion and high voltage (HV) stimulation on edema formation in rats. Thus treatment was rendered prior to, in anticipation of, edema formation. They examined three treatment groups:

1. Cool water immersion (55° F)
2. Cathodal HVPC, 120 pps, 90% of visible contraction, 30 min on:30 min off over 4 hours
3. Combination of cool water immersion and HVPC

They reported volumes of all treated limbs were smaller than control. Thus they concluded all treatments were effective in curbing edema but the combination treatment was not more effective than either treatment alone.

Dolan, Mychaskiw, Mattacola, and Mendel (2003) also studied treatment on acute edema.

The effects of cool water immersion (CWI) and HV stimulation were delivered as follows:

1. CWI at 55° F x 3 hours
2. HV stim x 3 hours
3. CWI x 1 hour followed by HV stimulation x 2 hours

Again, the results were all treated limb volumes were smaller than control. However, since the effects were only during the application, it was suggested repeated application may be appropriate.

Dolan, Graves, Nakazawa, Delano, Hutson, and Mendel (2005) assessed the effects of ibuprofen and HV stimulation on edema formation after blunt trauma to limbs of rats. Three treatment groups were as follows:

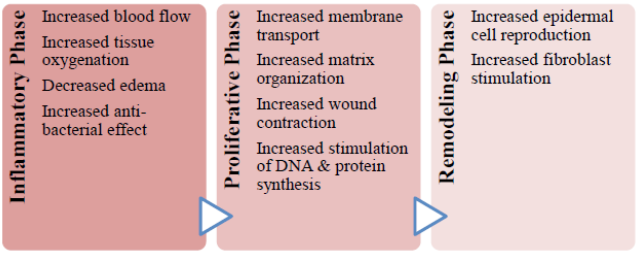
1. Ibuprofen (15mg/kg is equivalent to an 800 mg dose in an adult)
2. Cathode HVPC
3. Combination ibuprofen and cathode HVPC

All treatments effectively curbed edema after injury by roughly 50% relative to untreated injured control.

In a systematic review by Snyder, Perotti, Lam, and Bay (2010), eleven studies meet the inclusion criteria. The available evidence supports using a negative polarity at 120 pps, and an intensity of 90% of a visual motor contraction. To curb edema formation four 30-minute treatment sessions (30-minute rest in between) were recommended. To suppress the present of existing edema, a single continuous 180-minute treatment should be administered.

Wound Healing

Wound healing is a dynamic and complex process involving three overlapping phases (Reid & Zhao, 2014; Ud-Din & Bayat, 2014):



Healing begins with the development of a blood clot, i.e. platelet growth factor activates macrophages and fibroblasts. Neutrophils clean up the cell debris. Re-epithelialization begins within hours. Epidermal cells at the wound edges begin to proliferate. Fibroblasts continue to deposit and remodel the extracellular matrix as new blood vessels form. The wound closes in and the connective tissue increases in strength (Reid & Zhao, 2014).

ES has an extensive history dating back to the 17th century. It was not until the 1830's that the detection of "skin wound electric potentials" led to theories on the use of ES for wound healing. Undamaged skin has an endogenous electrical potential and a

transcutaneous current potential of 20-50 mV (Ud-Din & Bayat, 2014). When a defect in the skin occurs, a "current of injury" or "skin battery" is created. In-vitro studies have supported the theory that membrane depolarization and ion shifts are involved in the changes noted with ES. Interestingly, the use of sodium and calcium channel blockers has been shown to mitigate the effects of ES (Ennis, Lee, Gellada, Corbiere, & Koh, 2016).

When wounds fail to close, Kloth (2014) suggests it is likely the endogenous electric field is askew or absent. Many other factors can delay wound closure. Diabetes, vascular insufficiency, age, nutritional deficiencies, and smoking are just a few of the factors resulting in the formation of chronic wounds. ES can be combined with standard wound care to facilitate wound healing.

There is currently a substantial body of work to verify that ES enhances blood flow, tissue oxygenation, antibacterial effects, and migration of lymphocytes, fibroblasts, macrophages, and keratinocyctes. More specifically, the cathode enhances mobility of epithelial, fibroblast, and keratinocyte cells, while the anode enhances mobility of macrophages and neutrophils. Since sinusoidal AC has no polarity (it is a balanced charge), it cannot mimic the physiological properties induced by a DC signal (Kloth, 2014; Ud-Din & Bayat, 2014). Microcurrent, low voltage pulsed current, and high voltage galvanic stimulation (HVGS) all have a monophasic waveform. Microcurrent is known as an "electrical whisper." Low voltage (20-35V) pulsed current delivers a continuous DC with a long duration waveform. HVGS, also known as twin-peaked ES, delivers a double pulse with a high peak (150-500V) and short duration (<200 usec).

Microcurrent & Low Voltage Pulsed Current. Wolcott, Wheeler, Hardwicke, and Rowley (1969) published one of the earliest studies on the effects of microcurrent stimulation on wound healing. These researchers applied stimulation in the range of 200 - 800 microamps to a wide variety of wounds. ES demonstrated 200 - 350% faster healing rates than controls. In addition, the tensile strength of the tissue was stronger and they also noted antibacterial effects.

Cheng et al (1982) studied the effects of various intensities of electric currents on three variables associated with the healing process. They found at 500 microamps, ATP generation increased about 500% and amino acid transport was increased by 30% to 40% above currents levels of 100 to 500 microamps. Yet, when currents were increased to the milliampere range (103 increase), ATP generation was depleted, amino acid uptake was reduced, and protein synthesis was inhibited. These outcomes imply the higher milliamp currents inhibit healing whereas the lower microampere currents promote healing. To that end, the work of Stanish (1984) found implanted electrodes delivering 10-20 microamps of electrical current hastened recovery from ruptured ligaments and tendons. In fact, Stanish was able to reduce the normal 18-month recovery period to only six months.

Dr. Reinhold Voll and his colleagues were able to chart the effects of specific frequencies in the range of 0.5 - 1.0 Hz on different tissues. They reported this very low frequency range is resonant with the normal electrical activity of the human body. Voll et al documented the following effects:

- Spasmolysis of smooth muscles
- Tonification of elastic fibers, i.e. increased lung capacity in emphysema patients
- Reduction of inflammatory processes
- Reduction of degenerative process by restoring diffusion-osmotic equilibrium
- Restoration of polarization to the nerves
- Stimulus of ATP function

Talebi, Torkman, Firouzabadi, Mofid, Shariat, and Kahrizi (2007) examined 39 male guinea pigs with full thickness skin incisions. They were divided into three groups: control, DC anodal, DC cathodal. Surface skin potential was measured before injury, immediately after injury, and over the first 21 days of healing. The DC intensity delivered was that of a micro-amperage level. The anodal current was shown to accelerate the bioelectric events of wound healing to more rapidly return the wound potential to its pre-injury status.

Chronic dermal ulcers were successfully treated by a combination of cathodal and anodal pulsed ES (Feedar, Kloth, Gentzkow, 1991). They randomly assigned 47 patients into a treatment or control group. The treatments were progressed as the condition of the wound progressed. All treatments were 30-minutes and began with cathodal stimulation at 128 pps and a peak amplitude of 29.2mA twice daily. The polarity was changed every three days until the wound reached a stage II classification. Then the frequency was reduced to 64 pps and the polarity changed daily until the wound healed. After four weeks of treatment, there was significant improvement in the treatment group. Wounds were 44% and 67% of their original size in the treatment and control groups, respectively. The healing rates were calculated to by 14% and 8.25%, respectively. Thus, this unique ES protocol was deemed effective for wound healing. Perhaps there is value in capitalizing of the benefits of both the cathode and anode characteristics to retard microorganism growth and facilitate cellular migration, respectively.

Litke and Dahners (1994) studied the effect of low level direct current on early healing of medical collateral ligaments (MCL) in rats (n=87). All rats underwent MCL transection bilaterally. One MCL was the control and the other was treated with ES. The cathode was positioned distal to the injury site and the anode proximal. Current was delivered for 12 continuous

days. The three treatment groups were based on the intensity of the stimulation. Group 1 received 10-100 nanoAmps (nA), group 3 received 1-20 microAmps (uA), and group 3 was stated to be in between groups 1 and 3. After 12 days, group 1 showed no positive effects, group 2 showed slight improvement but not statistically significant, and group 3 had the greatest effect. Histological analysis revealed increased maximal rupture force, energy absorbed, and stiffness with decreased laxity at 8.6 ± 5.9 uA. Likewise, despite limited information on the parameters implemented, several other studies all demonstrated significantly accelerated healing (Gault & Gatens, 1976; Carley & Wainapel, 1985; Wirsing, Habrom, Zehnder, Friedli, & Blatti, 2013; Wood, Evans, Schallreuter, et al, 1993).

High Voltage Galvanic Stimulation (HVGS). As stated, HVGS is a mono-phasic, pulsed, twin-peaked waveform (figure 10) which involves the use of significantly high voltage. Selection of polarity is an important parameter. If the wound is infected, the polarity should be (-) to create an alkaline reaction to inhibit bacterial growth and increase blood flow. If the wound is "clean," i.e. not infected, the polarity of choice is (+) for cell proliferation and to promote the migration of cells toward the center of the wound. As will be discussed, some studies advocate the switching back and forth between polarities to increase blood flow and stimulate cell proliferation.

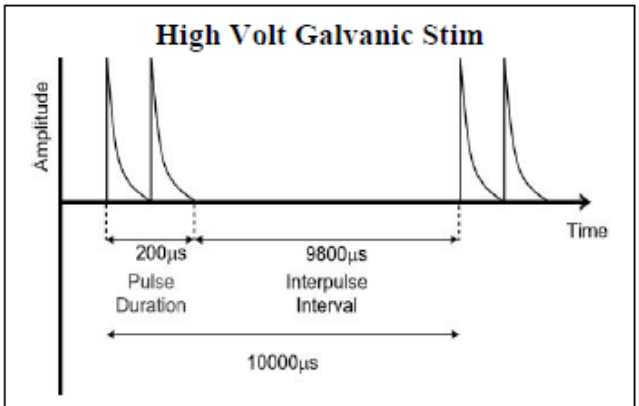


Figure 10

HVGS treatment procedure is identified in table 11.

Table 11: HVGS Treatment Procedure

- Remove dressing if present and measure the wound
- Electrode placement:
  - Prepare the electrodes to match the size of the wound – the electrode for the wound is typically made out of aluminum foil and wrapped in a saline-soaked, sterile gauze pad. An alligator clip is used to attach the lead wire to the gauze pad.
  - The inactive/dispersive pad must always be at least twice the size of the active electrode and secured on a non-treatment surface
  - Care should be taken to secure the electrodes with Velcro straps or ace wraps to minimize the use of adhesive substances on the skin. After all, if the patient is having challenges with wound healing, one would not want to create any further skin damage.
- Set rate at ~50 pps when using (-) polarity and ~80 pps with (+) polarity; frequency parameters are not well supported in the literature
- Current intensity should begin between 100V - 150V (subthreshold muscle contraction) – the first treatment is a trial for future intensity settings
- Treatment time is either twice daily for 30 minutes or once daily for 60 minutes

At the conclusion of the treatment, the gauze pad of the active electrode should be viewed. The desired outcome is a serosanguinous discharge, i.e. consisting of both bloody and serous fluid. If the gauze has just serous drainage, the intensity was under-dosed and should be increased for the next treatment. If the gauze is very bloody, the intensity was over-dosed and should be decreased for the next treatment. The voltages of endogenous human skin wound electric fields usually range from 100-300mV/mm. The voltages used for HVGS wound care treatment is notably higher because there is a significant drop in voltage between the electrodes and the cells in the wound (Reid & Zhao, 2014).

Evidence supporting these parameters has been described by several researchers.

Asadi, Torkaman, and Hedayati collaborated to publish three studies (2011, 2014, 2017).

The first study (2011) examined the effect of sensory and motor electrical stimulation in vascular endothelial growth factor (VEGF) of muscle and skin in full-thickness wound (n=48 rats). Three treatment groups were: 1) Sensory cathode DC at 600uA, 2) Motor cathode DC at 100 Hz, 2.5-3.0 mA, 3) Control. All treatments were for one hour every other day. The results revealed no difference in VEGF on Day 3 but VEGF level was significantly higher in the sensory DC stimulation on Day 7. Sensory ES parameters may be more effective in promoting wound healing.

The second study (2014) compared sensory and

motor ES on fibroblastic growth factor (FGF), inflammation, vascularization, and mechanical strength of wounds (n=96). The treatment groups received 10 one-hour sessions every other day. On the third day, the results showed that not only was the FGF greater for the sensory group, but the FGF of the motor group was less than control. There was no significant difference in strength or stiffness. They concluded the sensory ES may have a beneficial effect in the early stages of wound healing by inducing the release of angiogenic factors.

The third study (2017) examined the angiogenic effects of low-intensity cathodal DC on ischemic diabetic foot ulcers. Thirty type 2 diabetes patients with ischemic foot ulcerations were randomly assigned to ES or control. The ES group was treated one hour per day, every other day for four weeks (12 sessions). Outcome measures were hypoxic inducible factor-1a (HIF-1a), nitric oxide (NO), vascular endothelial growth factor (VEGF), and soluble VEGF receptor-2 (sVEGFR-2). HIF-1a and VEGF levels were significantly increased with ES but there was no change in NO or sVEGFR-2 levels. The researchers concluded low-intensity cathodal DC stimulation can be a promising way of promote angiogenesis to successfully treat diabetic wounds. Diabetes is a common complication in wound healing and is associated with abnormalities of the extracellular matrix. The effect of HVPC on type 1 collagen, a-smooth muscle actin, and transforming growth factor-b1 (TGF-b1) was investigated by Kim, Cho, and Lee (2014). Thirty rats were divided into three groups: non-diabetic control, diabetic control, and diabetic treatment. The diabetic treatment group received 40-minutes of HVPC daily for one week. The control groups received sham interventions. As expected, the wound closure was delayed in the diabetic rats as compared to non-diabetic control but those treated with HVPC showed accelerated wound closure and healing ($p < 0.01$), as well as restored expression levels of collagen-I ($p = 0.02$), a-SMA ($p = 0.04$), and TGF-b1 ($p = 0.01$) mRNAs. The researchers concluded HVPC may be beneficial for enhancing the healing of diabetic wounds.

Griffin, Tooms, Mendius, Clifft, Vander Zwaag, and El-Zeky (1991) studied the efficacy of high voltage pulsed current for healing of pressure ulcers in patients with spinal cord injury. Patients received high-voltage pulsed current (HVPC) or placebo for one hour a day over 20 consecutive days. ES parameters were negative polarity, 100 pps, and an intensity of 200 V. The surface area of the ulcers was measured before treatment and after treatment days 5, 10, 15, and 20. The ulcers of the HVPC group demonstrated significantly greater percentage-of-change when compared to the placebo group.

Houghton et al (2003) studied the influence of HVGS on chronic leg ulcers. The 45-minute treatment was rendered three times per week at a frequency of 100 Hz and an intensity of 150 V. At the end of four weeks of treatment, the wound size of the treatment group was 43% smaller and that represented an improvement of over twice that of the control group. Another study by Houghton et al (2010) also used HVGS for pressure ulcers in spinal cord injured individuals (n=34) but the parameters were very different. The two groups were HVGS with standard wound care compared to standard wound care alone. The treatment utilized multiple parameters:

- 20 minutes of stimulation at 100 Hz followed by
- 20 minutes of stimulation at 20 Hz followed by
- 20 minutes of no stimulation
- This sequence was repeated for 8 hours per day

The active electrode was initially the cathode but it was alternated every week. The intensity of treatment was 50-150V. After three months, the HVGS group had 70% ± 25% of wound closure verses the control was 36% ± 61% closure ($p=0.048$).

Merriman, Hegyi, Albright-Overton, Carlos, Putnam, and Mulcare (2004) compared four electrical stimulation parameters on staphylococcus aureus growth in vitro. The one hour treatments over three consecutive days were as follows:

1. Continuous direct micro-current at 500 uA
2. High voltage pulsed direct current at 100 pps, 250 V
3. Low voltage pulsed direct current at 128 pps, 30 mA
4. Low voltage pulsed alternating current at 128 pps, 30 mA

After the ES treatment, the zone of inhibition surrounding each electrode was measured. This zone revealed a significant inhibitory effect for continuous direct micro-current and high voltage pulsed DC, but not for either low voltage technique. These data suggest that for infected wounds, high voltage DC and continuous direct micro-current treatments may have an initial bacterial inhibitory effect.

Finally, Pellett (2000) conducted a review of the literature on wound healing. The review concluded there were numerous studies which demonstrated physiological effects of electrical stimulation on cells and tissues. Increased blood flow, stimulation of cellular responses and inhibition of bacterial growth using direct current can enhance healing. A summary of the generally acceptable parameters in this review were DC polarity at 50 to 128 pps and below a palpable muscle contraction. Treatment should be at least 40 minutes and rendered five days per week.

In addition to the wound healing influence of an electrical current, the polarity also appears to have an inhibitory effect on bacterial growth. As previously

stated, both DC and HVPC are more effective at inhibiting bacterial growth than are other types of ES. More specifically, the results of most studies support the application of cathodal microcurrent stimulation for bacterial growth inhibition (Asadi & Torkaman, 2014).

In 2015, Wirsing, Habrom, Zehnder, Friedli, and Blatti reported the use of a new ES technique in which wireless micro current stimulation (WMCS) was used to treat chronic wounds. WMCS was used to transfer current to the surface of a wound in 47 patients with wounds. The stimulus was administered two to three times per week for 45-60 minutes per session. The intensity was 1-5 A. After two weeks, visual progress was observed in all patients. After eight weeks, the mean reduction in wound size was 95%. There were no reported clinical side effects and the authors stated there are advantages compared with the previous methods of ES, as it is contactless, free of pain and very easy to use. Perhaps there will be more research on this method in the future.

Despite a plethora of excellent wound care studies demonstrating efficacy, there is not a "best" set of parameters we should all be using. This is similar to the ongoing quest for the "best" exercise protocol. We have an overabundance of suggested exercise regimes for strengthening but no "one size fits all" option. Thus, we need to consider the evidence provided and apply the techniques to the specific population being treated.

Pain Management

Transcutaneous electrical nerve stimulation (TENS) has been used since the late 1960's for pain management. *TENS pain management is transient and is not meant to cure a pathology, but rather to mitigate the pain level while healing is occurring*. Some of the conditions for which TENS may be used to address pain include arthritis, tendonitis, bursitis, headaches, myofascial trigger points, post-surgical, and other chronic conditions.

It has been suggested that TENS reduces sensitization that occurs with chronic pain. Sensitization has been associated with enhanced excitability of neurons in a given pathway. Neurons fire spontaneously and have an increased response to noxious stimuli as well as innocuous (not harmful or offensive) stimuli. Chronic pain conditions have also been associated with a loss of descending pain inhibition (Sluka, Bjordal, Marchand, & Rakel, 2013). This combination contributes to the increase in pain. However, there is evidence that strong intensity TENS reduces central excitability and restores inhibition in animals (DeSantana, da Silva, DeResende, & Sluka, 2009). Sluka et al (2013) hypothesized that repeated TENS may cause "rebooting" of the sensitization process(es).

In addition, as you review the literature in this course, remember TENS is not a "stand-alone" modality. In some studies, TENS has been shown to have an effect on resting pain but evidence will be presented that TENS appears to be more effective for reducing pain associated with movement. Rakel and Frantz (2003) demonstrated a limited effect of TENS on resting pain but a significant influence on walking and deep breathing. In long term use (>6 months), studies have shown significant reductions in pain with activities and the use of medication and health services (Chabal, Fishbain, Weaver, & Heine, 1998; Fishbain, Chabal, Abbott, et al, 1996).

Negative effects of TENS use have been minimal but one must consider the potential interactions with opioid use. Low frequency TENS (1-10 Hz) activates the mu-opioid receptors and high frequency TENS (50-150 Hz) activates the delta-opioid receptors (Kalra, Urban, & Sluka, 2001; Sluka, Deacon, Stibal et al, 1999; Sluka & Walsh, 2003). Thus, one should take care in administering TENS to individuals using opioids. Low frequency TENS will be ineffective with opioids but high frequency TENS was still effective.

We will explore a variety of theories associated with the wave form, frequency and intensity used to manage pain. These mechanisms include:

- Frequency Specific Microcurrent – DC
- Conventional TENS – AC (Gate Control Theory)
- Brief Intense TENS – AC (Central Biasing Theory)
- Low Rate TENS – AC (Opiate Pain Control Theory)
- Analgesic Nerve Block – AC (Wedesky Inhibition)

Frequency Specific Microcurrent (FSM) has been traced back to the early 1900's when Dr. Albert Abrams reported all matter radiates electromagnetic energy. Furthermore, it is theorized each tissue in the body has individualized frequencies, and varying conditions influence the frequencies. Injury may result in the electrons in the affected tissue taking on a different vibrational characteristic. Electrons may interact/resonate based on the thermodynamic and electromagnetic conditions created by the molecules configuration. Electrons are stable in an "energy well" and may require an energy "boost" to move from one energy well/orbit to another (Young, 2017). FSM is "frequency specific" in that it claims to match the frequencies of the affected tissues to counter/neutralize them. The frequency thought to address or neutralize the condition is applied via channel A while the frequency thought to address the tissue is applied via channel B. McMakin (2017) reports the combination of the two frequencies appear to have a therapeutic effect.

All frequencies used in FSM are less than 1000 Hz. Specific frequency claims are as follows:

- 0.3 Hz = healing
- 3 Hz = stimulating acupuncture points
- 30 Hz = controlling pain

- 300 Hz = reducing edema and stimulating lymphatic flow

Furthermore, the intensity of the current has been reported to influence physiologic functions. Cheng (1982) showed that microcurrent increased ATP production in rat skin by 500% as long as the current was below 500 µA. When the current was increased to 1000 µA (1 mA), the ATP production was reduced. The current also increased amino acid transport into the cell by 70% and waste product removal. When the current was increased to 5000 µA (5mA), protein synthesis was suppressed. The implications for human healing and repair are interesting.

Fujiya et al (2015) examined the influence of microcurrent on the regenerative process of injured skeletal muscle. Mice (n=30) were randomly assigned to two groups: cardiotoxin-injected or cardiotoxin-injected with microcurrent ES. Cardiotoxin was injected into the tibialis anterior (TA) to initiate necrosis. Forty-eight hours after the injection, one limb was treated with 10uA current at 0.3 Hz and 250 mseconds for 60-minutes. The treatment was three days per week for three weeks. At weeks 1, 2, and 3 post-injection, the TA was dissected out. Based on muscle weight, muscle protein content, the mean cross-sectional areas of muscle fibers, the relative percentage of fibers having central nuclei, and the number of muscle satellite cells, microcurrent was deemed successful in facilitating the regeneration process.

Curtis, Fallow, Morris, and McMakin (2010) examined the efficacy of FSM on delayed onset muscle soreness (DOMS). Healthy participants (n=35) underwent an eccentric DOMS producing exercise program for the hamstrings. One leg served as the control and the other leg was treated with microcurrent ES for 20 minutes. Graphite gloves were used to deliver a positive current proximally and a negative current to the distal hamstrings. The 200 mA treatment was administered in the following sequence:

- 18 Hz via channel A (pathology specific) and 62 Hz via channel B (tissue specific) for four minutes
- 124 Hz via channel A and 62 Hz via channel B for one minute
- 142 Hz via channel A and 191 Hz via channel B for one minute
- 40 Hz via channel A and 116 Hz via channel B for four minutes
- 40 Hz via channel A and 62 Hz via channel B for two minutes
- 142 Hz via channel A and 191 Hz via channel B for two minutes
- 49 Hz via channel A and 62 Hz via channel B for one minute
- 142 Hz via channel A and 191 Hz via channel B for one minute

Perceived soreness measured by a visual analogue Scale (VAS) was reported to be significantly reduced in the treatment leg at 24, 48, and 72 hours. This is interesting since 48-72 hours after the DOMS producing exercise program is when one would expect the VAS to be highest.

Conventional TENS, also known as the Gate Control Theory of pain management, was first reported by Melzack and Wall in 1965 (figure 11). The theory asserts that when an electrical current is applied to a painful area, the transmission of the pain to the brain via the small diameter fibers is inhibited by the large diameter, highly myelinated, proprioceptive sensory nerves, thus "closing the gate" to the perception of pain (Kerai et al, 2014). Furthermore, it has been proposed that release of endogenous opioids activates descending inhibitory pathways. The structures involved in this process include the periaqueductal gray (PAG) sending projections to the nucleus raphe magnus in the rostral ventral medulla (RVM) which in turn send projections to the spinal dorsal horn. Stimulation of either the PAG or RVM results in inhibition of the dorsal horn nucleus and spinothalamic tract cells.

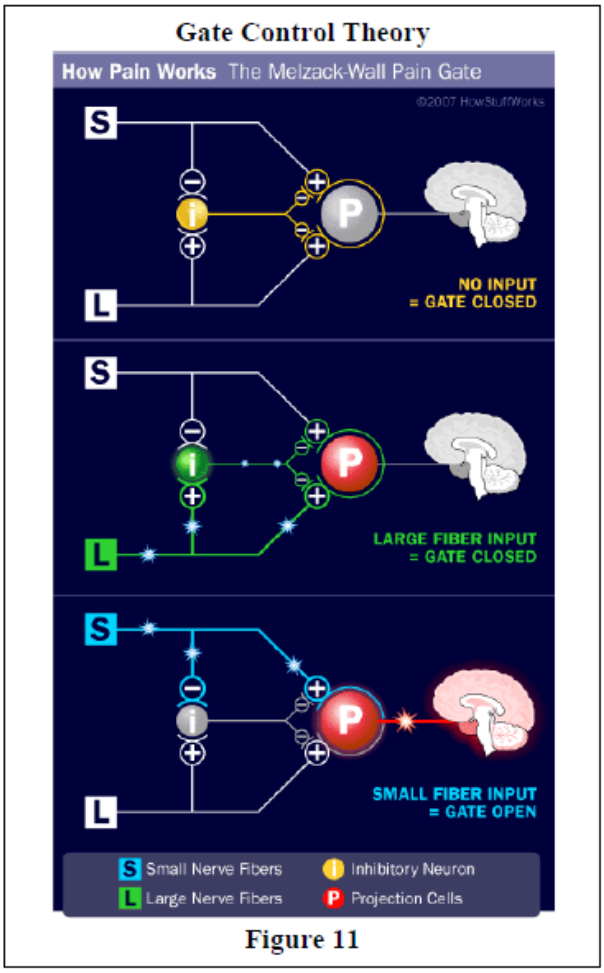


Figure 11

Different frequencies of TENS have been shown to activate specific opioid receptors. For example, low frequency TENS causes activation of the δ-opioid receptors and high frequency TENS activates the μ-opioid receptors. These receptors activate the PAG-RVM pathway (Kerai et al, 2014).

The use of ES to stimulate the large diameter, fast-conducting, highly myelinated A-beta fibers has been demonstrated to reduce pain. The parameters for ES have included the use of a continuous alternating current delivered at 100-120 pps to a tolerable sensory level (no muscle contraction). High frequency TENS is typically more comfortable. The electrodes should be placed in the direct vicinity of the pain for a 15-20 minute treatment duration. The release of enkaphalins takes about 10-20 minutes and pain relief is sustained for about 30 minutes after the cessation of treatment.

Conventional TENS can be used effectively in many ways. One clinical example is a patient with osteoarthritis of the knee. The patient may begin therapeutic intervention with a moist heating pad and ES to the knee or a warm-up on a stationary bike with ES to the knee. After "warm-up," the patient moves on to a therapeutic exercise program of joint mobilization, strengthening, and/or stretching. Then treatment is concluded with ice application. Throughout the entire treatment, the ES unit will remain in place, i.e. electrodes around the knee. This entire treatment duration of the ES would be approximately 40-50 minutes. This example is consistent with the following literature.

Since 1965, numerous studies have investigated the effect of the gate control theory.

Atamaz, Durmaz, Baydar, Demircioglu, Iyiyapici, Kuran, Oncel, and Sendur (2012) compared the influence of various modalities on pain associated with knee osteoarthritis. This was a multi-center study in which patients were randomized into six groups:

- TENS sham
- TENS – AC at 80 Hz, 10-30mA intensity x 20 minutes
- Interferential current (IFC) sham
- IFC – AC at 100 Hz, sensory threshold intensity x 20 minutes
- Shortwave diathermy (SW) sham
- SW – 27.12 MHz at 3.2 Watts

Interventions were administered five times per week for three weeks and exercises were given. Pain was assessed via a visual analogue scale (VAS), time to walk 15 meters, range of motion, WOMAC, and medication intake were all measured. Although all groups had a significant improvement in all parameters, only the treatment groups (not sham), had a significant reduction in pain medication.

Cheing, Tsui, Lo, and Hui-Chan (2003) sought to identify the optimal treatment duration for the administration of conventional TENS. They examined the influence of 100 Hz TENS on pain associated with knee osteoarthritis. Treatment durations were 20, 40, 60 minutes and a 60 minute placebo. Treatment was provided five days per week for two weeks. The outcome measure was VAS. The results can be best summarized in table 12.

Table 12: Magnitude & Duration of Pain Relief

Treatment	Pain Reduction	Post-stimulation Analgesia
TENS 20	54.59%	168 minutes
TENS 40	83.40%	256 minutes
TENS 60	68.37%	258 minutes
Placebo	6.14%	35 minutes

The conclusion was that 40 minutes of TENS at 100 Hz provided optimal pain reduction for the longest period of time. This type of information is very helpful in determining the appropriate evidence-based treatment parameters. This is analogous to another decision-making process: soft tissue stretching. Evidence has demonstrated 15-seconds of static stretching increases tissue elongation but 30-seconds were statistically better. Although 60-seconds of stretching also increased tissue elongation, it was not significantly better than 30-seconds. Research such as this contributes to the implementation of best practice.

Moore and Shurman (1997) combined neuromuscular electrical stimulation (NMES) with TENS to manage chronic back pain. Four treatment protocols were rendered in random order for five consecutive hours per day for two days. There was a two day hiatus between treatment conditions. The protocols were very unusual, consisting of the treatment times mapped out in table 13:

- NMES = AC stimulation at 5 second on time, 15 second off time at 70 Hz and an intensity of 1-100mA.
- TENS = continuous AC stimulation at 100 Hz and an intensity of 0-60 mA
- NMES/TENS = alternating NMES and TENS
- Placebo = no stimulation administered

Table 13. Treatment Time for Each Method

NMES	10 min NMES	130 min no treatment	10 min NMES	130 min no treatment	10 min NMES
TENS	5 hours TENS				
Combo	90 min TENS	10 min NMES	90 min TENS	10 min NMES	90 min TENS
Placebo	5 hours no treatment				

The conclusion was that the combined NMES/TENS treatment consistently produced greater pain reduction and pain relief than placebo, TENS, or NMES. NMES alone was less effective but did produce as much pain relief as TENS. There were a number of concerns about this study. NMES is not typically used for pain relief and the intensities identified were quantified but not functionally described. Given that the stimulus tolerated by any individual is predicated on the location and size of the electrodes, this may be a confounding variable. The broad range of the NMES intensity may have produced a motor response, i.e. muscle contraction in some people and not in others. Likewise, the TENS intensity may have been maximal or noxious in some people and sub-motor/sensory in others. Thus, the lack of standardized response makes generalized conclusions challenging.

A pair of noteworthy studies conducted in central Europe explored the impact of TENS on acute trauma to the low back and hip pain.

Bertalanffy, Kober, Bertalanffy, Gustorff, Gore, Adel, and Hoerauf (2005) administered a single TENS treatment to individuals suffering from low back pain. These individuals were transported by ambulance to a hospital. While in transit, they were randomly assigned to a treatment or sham group. The TENS treatment group received a 30-minute treatment at a 100 Hz frequency 200 usec pulse duration and 2 mA intensity. They collected data on pre- and post-transports pain and anxiety levels using a visual analog scale (VAS).

Pain levels were:

	Beginning of Transport	End of Transport
TENS VAS	79.2 +/- 6.5 mm	48.9 +/- 8.2 mm
Sham VAS	75.9 +/- 16.4 mm	77.1 +/- 11.2 mm

Anxiety levels were:

	Beginning of Transport	End of Transport
TENS VAS	81.7 +/- 7.9 mm	69.2 +/- 12.1 mm
Sham VAS	84.5 +/- 5.8 mm	83.5 +/- 8.9 mm

Likewise, Lang, Barker, Steinlechner, Gustorff, Puskas, Gore, and Kober (2007) administered a single TENS treatment to individuals suffering from nonlife-threatening hip trauma. These individuals were transported by ambulance to a hospital. The exact same treatment protocol (30-minute treatment at a 100 Hz frequency 200 usec pulse duration and 2 mA intensity) or a sham treatment was randomly assigned to this population.

VAS pain assessment was:

	Beginning of Transport	End of Transport
TENS VAS	89 +/- 9 mm	59 +/- 6 mm
Sham VAS	86 +/- 12 mm	79 +/- 11 mm

Thus, TENS was found to be a valuable and fast-acting pain relieving treatment under difficult circumstances of "out-of-hospital rescue." The technique was easy to use and there were no side effects noted. A systematic review by Simpson, Fouche, Thomas, and Bendall (2014) supported the results of these two studies.

Another study examined the influence of TENS on phantom limb pain. Kawamura et al (1997) applied TENS to the contralateral limb corresponding to the portion of the amputated limb where the patient felt phantom limb pain. The treatment involved 30-minutes of ES three times per day over a 9 week period. Pain level via a VAS was the outcome measure. ES was found to significantly relieve degree, duration, and area of pain. Final VAS of 1.2 +/-1.9 compared with the initial VAS of 5.5 +/- 1.7 ($p<0.001$) resulted in better appetite, sleep, and prosthetic use. This method of pain management has been shown to be efficacious.

Ardic, Sarhus, and Topuz (2002) compared two modes of ES to treat myofascial neck pain. They administered 20 minutes of TENS to a myofascial trigger point (MTrP) in the upper trapezius. The frequency was 60 Hz, pulse duration 100 usec, and intensity to a level a sensory paresthesia. The second group received muscle ES at 25 Hz, 250 usec, a duty cycle of 1:3 (3 sec on: 6 sec off), and an intensity to achieve a strong muscle contraction. The third group was a control of no ES. All groups completed a self-stretching program three times a day. Outcome measures of VAS, cervical ROM, and pain pressure threshold (PT) were assessed before treatment, after two weeks of treatment, and after three months of treatment. The researchers found such a strong statistically significant improvements in the treatment groups over the control at the two-week assessment, they made the ethical decision to treat the control group. Although thought provoking, there are concerns about the parameters of this study. First, the manuscript did not reveal how often the participants were treated over this three month time frame. Second, there was no information about the stretching program implemented. Third, they spoke about the physiology behind the treatment parameters for group #1 as complying with the "gate control theory" of pain management. However, 60 Hz is not consistent with the frequency for "conventional" TENS. The frequencies used in this study really don't fit into any of the ranges suggested for pain management via conventional, brief-intense, or low rate TENS.

Azatcam et al (2017) also examined the effectiveness of TENS on myofascial pain (n=75). However, they also included the use of Kinesio Taping. So the three

randomly assigned groups were: TENS (60 Hz, 100 msec, paresthesia intensity), Kinesio Taping, and control. The TENS groups was treated for 20 minutes, five times per week for two weeks. The Kinesio Taping group was taped two times per week for two weeks. Stretching was to be performed three times per day at 10 repetitions each. However, the duration of the stretching, i.e. hold time was not revealed. Nor were the type or number of exercises. Nonetheless, VAS, pain pressure threshold (PPT), cervical ROM and Neck Disability Index measures were evaluated pre-treatment, post-treatment, and three months after treatment. All groups improved for all outcome measures with only the VAS achieving significance. Again, as previously discussed, the TENS frequency of 60 Hz is not consistent with any TENS theory (Opiate Theory =2-10 Hz, Gate Control = 100-150 Hz). Yet, several studies have been found to report this frequency as being successful.

In a completely different venue of pain management, Dowswell, Bedwell, Lavender, and Neilson (2009) published a systematic review of TENS for pain relief in labour. In summary, 19 studies of 1671 women concluded although women receiving TENS were less likely to report severe pain, there was little difference in pain ratings between TENS and control groups. Nonetheless, the majority of women using TENS said they would be willing to use it again in a future labour. No adverse events were reported for the mother or baby. Several years later, Santana, Gallo, Ferreira, Duarte, Quintana, and Marcolin (2016) examined the influence of TENS on the various stages of labor. They randomized patients (n=46) in labor into two groups: treatment and control. Four electrodes were placed on the T10 to L1 and S2 to S4 paravertebral regions. A stimulus to tolerance at 100 Hz and 100 usec was administered for 30 minutes. The results were statistically significant for pain reduction using a VAS. The treatment group demonstrated a 11 mm reduction in pain while the control group had a 4 mm increase in pain. Thus, TENS may be a viable intervention to reduce pain and/or postpone the need for pharmacological pain relief.

Noehren, Dailey, Rakel, Vance, Zimmerman, Crofford, and Sluka (2015) reported preliminary results in a non-pharmacological pain management option for individuals with fibromyalgia. An AC waveform at a frequency modulated between 10 and 100 Hz and the intensity as high as tolerable suggested changes in pain with movement, function, and pain physiology. Prior works have placed emphasis on the fact that pain is not the only indicator of effectiveness. Function is frequently not addressed in studies yet it is more likely to be reduced than pain at rest.

Facci, Nowotny, Tormem, and Trevisani (2011) compared TENS with interferential current (IFC) and control for individuals with chronic low back pain. The TENS parameters were 20 Hz at a strong but comfortable intensity for 30-minutes. The IFC was administered at a medium frequency of 4000 Hz with a modulation frequency of 20 Hz. This means the AC waves intersect to result in a different of 20 Hz. In other words, channel #1 delivers 4000 Hz and channel #2 delivers 4020 Hz with a delta of 20 Hz. Ten treatments were rendered over two weeks. There was no difference between the groups in duration of analgesia or the Roland-Morris Disability Questionnaire. However, the TENS, IFC, and control groups had an 84%, 75%, and 34% reduction in the use of medication, respectively. The reduced need for "rescue" medication can be extremely valuable. The reduced risk of addiction to narcotics cannot be underestimated.

Brief Intense TENS is based on the Central Biasing Theory of pain relief. Very simply, the intense stimulation of C-fibers for brief periods of time can be used to produce hyperstimulation analgesia. The parameters recommended to achieve this effect are the use of a continuous alternating current at a rate of 100-120 pps. The maximal stimulus is delivered for 30-60 second bouts. Relief has been suggested to last about five to ten times the duration of the stimulus. Thus, relief is for minutes not long-term, i.e. 30 second stimulus may mollify pain for 2.5 - 5 minutes. Although this technique is not frequently used in clinical practice, this type of pain management lends itself to mitigating pain to perform another technique, i.e. brief intense TENS prior to performing transverse friction massage, scar mobilization, or breaking of soft tissue adhesions.

Effects of brief high intensity electrical stimulation were examined on patients with chronic pain (Jeans, 1979). Participants reported pain for a mean duration of 5.7 years. The pathologies included low back pain, general musculoskeletal pain, neuralgia, causalgia, and phantom limb pain. Participants were divided into treatment and sham groups. Electrode placement was over the painful area and a distal trigger point for both groups. Participants received two treatments per day over four consecutive days. The outcome measure was the McGill Pain Questionnaire before and after treatment and a demonstrated significant reduction in pain intensity and analgesic at two weeks following treatment. Jeans concluded the noxious stimulation at the site of the pain is significantly more effective than placebo at relieving chronic pain.

Koke et al (2004) explored pain management with three types of TENS: high frequency, low intensity and high frequency, high intensity was compared to a control. Their cross-over design (n=180) incorporated two weeks of one treatment followed by a 2 week wash-out period prior to the other treatment being applied. The order of treatments was randomized. The parameters are recapped in table 14:

Table 14: Koke et al (2004) Parameters			
	High frequency-low intensity	High frequency-high intensity	Control
Frequency	80 Hz	80 Hz	30 Hz
Pulse duration	90 msec	250 msec	250 msec
Intensity	Sensory	Maximal tolerable	Self-selected
Treatment time	1 hour	30 minutes	Self-selected
Treatment frequency	4-6x/day	4-6x/day	Self-selected

The study reported no difference in the pain perception of the three groups. Yet, 56% continued the TENS use after the study with 42% still used the TENS six-months later. Unfortunately, this study has flaws. Allowing a "control" group to self-select the parameters of a treatment fails to have any standardization to the group. Since no "true" control group was used, one cannot draw conclusions on the effectiveness. In addition, when TENS is used for long periods of time, it may result in a "cross tolerance." In other words, treatment can backfire after prolonged use because the system becomes depleted of endorphins and serotonin.

Opiate Pain Control Theory utilizes low rate TENS to stimulate A-delta and C-fibers to release enkephalins and beta-endorphins to mitigate pain. The parameters include AC delivery at a rate of 1-5 Hz at a long pulse duration with a noxious level of stimulation. A treatment time of 40-60 minutes is recommended since the onset of relief can take 15-30 minutes. The duration of the relief can be 2-6 hours. This can lend itself to a very functional application of pain management. For example, imagine this scenario: A person is experiencing pain on a relatively constant basis. S/He could apply a TENS unit in the morning upon waking for 40-60 minutes, turn the unit off but leave all the electrodes in place. The unit could be placed in a pocket or clipped on a belt to drive to work. The individual would then turn the unit back on during the lunch break for 40-60 minutes. After work the stimulus could be re-applied over the dinner hour and even an hour before bedtime if desired. This pattern of application would give the individual pain relief for 4-6 hour blocks of time. This is just one example of the practical application of low rate TENS use. Now let's look at the literature to reveal the evidence.

Law and Chein (2004) sought to identify the optimal stimulation frequency of transcutaneous electrical nerve stimulation on people with knee osteoarthritis. A double-blind study of 34 subjects was randomly allocated into four groups: 1) TENS at 2 Hz, 2) TENS at 100 Hz, 3) TENS alternating 2 and 100 Hz (3 seconds at 2 Hz and 2.5 sec at 100 Hz), and 4) placebo. Treatments were administered five times per week over two weeks. The intensity was administered to "comfort" and was reported to produce paresthesia and mild twitches. The researchers also stated the intensity was increased after five minutes if the subject "accommodated." The outcome measures were VAS, timed up-and-go test, and range of motion. All three treatment protocols were found to be superior to placebo at reducing knee pain but there was no difference between the groups. The parameters of this study address two different theories of pain management. Group #1 frequency is consistent with the Opiate Pain Control Theory (low rate TENS) but the intensity is notably lower than recommended. Whereas, the group #2 frequency and intensity are consistent with the Gate Control Theory (conventional TENS). Group #3 is a hybrid of the two theories. Table 15 is a summary of the VAS values for each group:

Table 15: Law & Chen (2004) Results				
	Baseline	After Session #1	After Session #10	2 Week Follow-up
Low Frequency	6.6	4.6	1.4	1.6
High Frequency	5.2	2.6	0.7	0.9
Alternating Low & High Frequency	5.4	2.3	1.1	1.6
Control	5.8	4.6	4.1	4.4

Chesterton, Foster, Wright, Baxter, and Barlas (2003) studied a variety of TENS frequencies and intensities on pressure pain threshold of 240 volunteers. The treatment groups were:

- Sham
- Control
- Frequency of 110 Hz, intensity strong but comfortable
- Frequency of 110 Hz, intensity highest tolerable
- Frequency of 4 Hz, intensity strong but comfortable
- Frequency of 4 Hz, intensity highest tolerable

Treatments were 30 minutes in duration. The high frequency, high intensity stimulation groups, showed rapid onset and significant hypoalgesic effects. This effect was sustained for 20 minutes after stimulation ceased. All other TENS groups showed hypoalgesic responses similar to the sham group.

Noaham and Kumbang (2008) reviewed the influence of TENS on chronic pain. Only 25 randomized controlled trials (RTCs) were included. They found TENS to provide an analgesic outcome compared to control in 13 of 22 studies. For studies involving multiple TENS doses, eight of 15 were favorable to TENS treatments. When comparing high frequency to low frequency TENS, seven out of nine found no difference. Overall, the authors stated "published literature on the subject lacks the methodological rigor or robust reporting needed to make confident assessments of the role of TENS in chronic pain management."

Maayah and Al-Jarrah (2010) examined the effect of a single 60-minute TENS treatment at 2-8 Hz on subjects with neck pain. The authors deemed the treatment effective despite a variety of intensities and the failure to control analgesic intake.

Zeng, Li, Yang, Deng, Yang, Zhang, and Lei (2015) conducted a systematic review that explored six forms of ES:

- High frequency TENS
- Low-frequency TENS
- Neuromuscular ES
- Interferential current (IFC)
- Pulsed ES
- Noninvasive interactive neurostimulation.

They identified 20 studies addressing knee osteoarthritis that meet their criteria. IFC was deemed the only significantly effective treatment for pain intensity. As a medium-frequency current, IFC is known to have lower skin impedance which may enhance depth of penetration. This systematic review was consistent with a Cochrane review in 2009 but in conflict with the review by Davis and Mackay in 2013.

The intensity of ES has been deemed important for maximal current charge for muscle reeducation and has been correlated with the success of wound care but the need to adjust the intensity throughout treatment has been controversial. Defrin, Ariel, and Peretz (2005) examined 62 people with chronic knee pain due to osteoarthritis. The participants were divided into six groups:

- Noxious stimulation that was not adjusted during treatment
- Noxious stimulation that was increased (adjusted) during treatment
- Innocuous unadjusted
- Innocuous adjusted
- Placebo
- Control

Twelve treatments of interferential current (IFC) were delivered for 20-minutes sessions every other day. The results were all treatment groups had a reduction in VAS. There was a statistically significant reduction in noxious stimulation groups over that of the innocuous stimulation groups. However, there was no significant difference in pre-to-post VAS between adjusted and unadjusted current groups. Thus, three conclusions can be drawn: 1) IFC is effective for chronic osteoarthritic knee pain, 2) noxious stimulation is more effective than innocuous stim, and 3) fading current sensation during treatment does not impede the analgesic effect.

Several years later, Pantaleao, Laurino, Gallego, et al (2011) sought to address the same question. They divided 56 healthy individuals into four groups:

- Control
- Placebo
- Fixed TENS amplitude
- Adjusted TENS amplitude

The outcome measure was pressure pain threshold (PPT) assessed before, during, and after 40-minutes of ES. The adjusted amplitude results in a greater PPT than all other groups. This suggests it is important to increase the TENS amplitude to get the maximal analgesic effect. This is contrary to the results of Defrin, Ariel, and Peretz (2005). So the question remains, do you increase the current when a patient reports the intensity feels less than that at the beginning of treatment? Perhaps the answer may be frequency dependent. Defrin, Ariel, and Peretz (2005) used medium frequency AC and did not find it beneficial to increase the current but Paleao et al (2011) used low frequency current and found it advantageous to increase the current. This amplifies the underlying issues of ES research. It is challenging to control all of the parameters involved to identify best-practice.

Typically pain management has utilized AC devices. Yet, a study on the treatment of pain associated with lateral epicondylitis used a DC mode (Nourbakhsh & Fearony, 2008). A stimulus to tolerance was applied to each point tender area of the elbow at 4 Hz for 30 seconds. All points were stimulated three times and subjects were treated for six sessions over a 2-3 week period. The treatment group receive DC stimulation has an increased grip strength ($p < 0.04$), decreased pain intensity ($p < 0.01$), decrease in limited activity due to pain ($p < 0.003$), and an increased functional level ($p < 0.01$). Thus, treating active points of tenderness was effective in managing the symptoms of chronic lateral epicondylitis.

Although the majority of studies support the use of TENS for pain management, a number of studies refute its effectiveness.

A systematic review by Kerai, Saxena, Taneja, and Sehrawat (2014) from July 2012 to January 2014 reported most studies demonstrated a reduction of pain and supplemental analgesics. However, when studies do not provide the parameters, i.e. frequency, intensity, and pulse duration, or placement of the electrodes, it is not possible to compare the data. If a variety of outcome measures are used or patient populations vary, generalizations are limited.

Sawant, Dadurka, Overend, and Kremenchutzky (2015) conducted a systematic review of the efficacy of TENS for pain relief in people with multiple sclerosis. Four studies meet their criteria. Two studies reported no difference between high and low frequency TENS and placebo. Another study found TENS to be equivalent to medication. Only one study found both high and

low frequency TENS to be better than placebo. One significant issue with this review is that none of the studies utilized quality of life measures following pain relief. As identified by other studies, pain relief at rest is not nearly as valuable as pain relief with activity.

Table 16 summarizes the parameters discussed for the three theories of pain management to simplify the process and provide a quick-glance reference:

Table 16: Comparison of AC Pain Management Theories

Theory	Gate Control Theory (Conventional TENS)	Central Biasing Theory (Brief Intense TENS)	Opiate Control Theory (Low Rate TENS)
Physiology	Stimulation of the A-beta sensory fibers to block the A-delta & C-fibers; as long as the sensory fibers are firing, the gate to the pain is "closed"	Intense stimulation of the C-fibers to produce hyperstimulation analgesia	Stimulation of the A-delta & C-fibers lead to the release of enkephalins & beta-endorphins to decrease pain
Electrode Placement	Directly around the painful region	Directly over the painful site	Non-specific because of the systemic response
Rate	100-120 pps	100-120 pps	1-3 pps
Intensity	To sensory tolerance but no muscle contraction	Maximal tolerated; Noxious	Maximal tolerated; Noxious
Treatment Duration	~ 40 minutes	30-60 seconds	60 minutes
Time of Onset of Relief	10-20 minutes	Within a couple of minutes	15-30 minutes for the release of enkephalins
Duration of Relief	~ 30 minutes after the removal of the stimulation	5-10x the duration of treatment, i.e. minutes	2-6 hours (B-endorphins half-life is 4 hours)

Thus the take-home message for TENS can be summarized into a few important points:

- The level of hypoalgesic efficacy of TENS is clearly dependent on the combination of parameters selected (defined in terms of intensity, frequency, and stimulation site) and the experimental pain model
- Stimulation amplitude has been shown to be critically important to obtain a positive effect. TENS delivered at a strong but comfortable intensity was more effective at providing an analgesic effect than that delivered at or below sensory level (Sluka, Bjordal, Marchand, & Rakel, 2013).
- Lee et al (1985) demonstrated that low frequency TENS (<10 Hz) produced greater inhibition than high frequency TENS (>50 Hz). Furthermore, increasing the activation of the Aδ nociceptors produced a greater inhibition than increased intensity to the A-beta fibers.
- Although TENS has not been shown to be effective under all circumstances, it may be equally as effective as many modalities. Many studies do not provide details of the stimulation parameters. Many studies use a variety of outcome measures, some active and some passive. Moreover, electrode placement and duration of relief are variable. Animal models have been helpful in advancing the understanding of the influence of TENS but there is still more to learn. Given the numerous side-effects of pharmaceutical pain management and the risk of addiction, TENS can be a very efficacious alternative.

Analgesic Nerve Block. There is one other technique that does not fall into the main stream theories of pain management but is worthy of discussion: Analgesic Nerve Block. This technique uses a medium frequency AC mode to keep a nerve in the refractory phase of an action potential. The mechanism for this "blocking" is through the use of a medium frequency current (5000 Hz) which causes depolarization of the nerve membrane and inhibition of nerve conduction. With an increased refractory period, high frequency discharges are blocked and low frequencies are transmitted. This effect is known as "Wedensky Inhibition." When "Wedensky Inhibition" is induced, the involved muscles go through a series of contractions with each contraction being weaker than the previous one until the muscle is no longer able to contract. This inhibitory technique may last from 12-15 minutes.

This technique was tested by Gulick, Borger, and McNamee (2007) on an individual with adhesive capsulitis. As one may know, when the inferior capsule of the shoulder becomes adhered, elevation and ER are notably limited. The subscapularis is often very tender to palpation, as is the coracohumeral ligament. This case report indicated the 64 year old female was treated three times per week with moist heat to the inferior capsule and ES to the subscapularis (2 electrodes) and the C5-T1 segmental innervation (2 electrodes). Treatment began with moist heat engulfing the involved shoulder in the supine position. After 10 minutes of heat, ES was added for the next 10 minutes (heat remained on the patient for a total of 20 minutes). The ES was rendered with an OmniStim 500® using the pre-programmed analgesic nerve block parameter (continuous, non-modulated, medium frequency current of 5000 Hz). The intensity of the stimulus was increased to the maximal level of comfortable tolerance. At the conclusion of the ES, the therapist assisted the patient in performing 10 contract/relax activities, alternating between shoulder IR and ER (5 second hold, 30 second stretch). The patient then performed 10-minutes of Pendulum (Codman's) exercises with a 2-pound cuff weight

on the wrist of the involved upper extremity. At the end of four weeks, the patient demonstrated an 83% improvement in the Shoulder Pain and Disability Index (SPADI) score, a 33-35% increase in shoulder internal/external rotation ROM, and a 18 cm improvement in the rotational lack measurement. Thus, by blocking the subscapular muscle, the clinician can utilize techniques to stretch the capsule to improve motion & function.

To date, there does not appear to be any other publications using this methodology.

In short, there are numerous combinations of five diverse theories of pain management. They all have different treatment durations with different mechanisms and time of pain relief. With so many options, it is challenging to determine the best choice. Fortunately if a patient does not get relief with one technique, there are other options. Remember, ES for pain management is an excellent alternative to pharmacological intervention but it is not meant to be used independently or for extensive periods of time.

Iontophoresis

Iontophoresis is a method of local transfer of ionized medication and non-medicated substances through the skin and into the target tissues using polarized electrical stimulation (Brown, Lauber, & Cappaert, 2015). Put another way, it is the use of a DC device to facilitate topically applied active ions from heavy metals into and through the skin (figure 12). Medications used must be both water and lipid soluble. DC units are used to insure unidirectional ion flow. The theory is simple physics: the electrical principle of opposite charges attracting and like charges repel on another. Thus, when one places a negatively charged ion in an electrode, places the electrode on the skin, and the negative wire delivering the current on the electrode, the two charges will repel one another and the only direction the ion can go is into the skin.

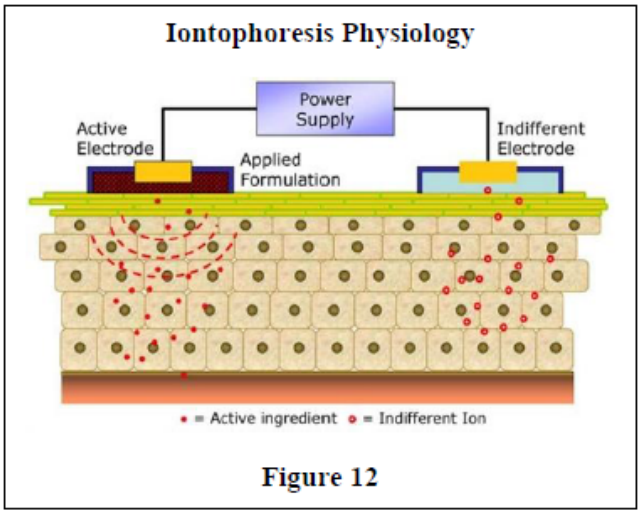


Figure 12

Iontophoresis dates back to 1908 but the development of a crude medical device for the use on scar tissue on burn victims was not available until 1965 (University of Texas). In 1977, the Phoresor, by Motion Control, was developed to provide anesthesia prior to the use of large-bore needles for dialysis. It evolved into being used for many other substances.

The chart below is a list of the numerous ions and the polarity for which they are theorized to be indicated. Many of these ions have very little evidence to support their efficiency and as such are used very infrequently in the clinic. The preponderance of iontophoresis treatments uses acetate, ketaprofen, dexamethasone, or lidocaine. The indications for iontophoresis include inflammation, analgesia, scar modification, wound healing, and calcium deposits, to name a few (table 17).

Table 17: Iontophoresis Ions, Polarity, & Indications

IONS	POLARITY	INDICATIONS
Acetate	Negative	calcium deposits
Atropine sulfate	Positive	hyperhidrosis
Calcium	Positive	spasmodic conditions, tics
Chlorine	Negative	soften scars & adhesions, i.e., sclerolytic agent
Citrate	Negative	rheumatoid arthritis
Copper	Positive	fungus infections i.e., athlete's foot
Dexamethasone	Negative	Inflammation, i.e. tendonitis, bursitis
Glycopyronium bromide	Positive	hyperhidrosis
Hyaluronidase	Positive	edema reduction
Hydrocortisone	Positive	rheumatoid arthritis, myositis, bursitis
Iodine	Negative	Adhesive capsulitis, sclerolytic
Ketaprofen	Negative	Inflammation, i.e. tendonitis, bursitis
Lidocaine	Positive	Pain for acute conditions
Lithium	Positive	gouty tophi & hyperuricemia
Magnesium	Positive	muscle relaxant, vasodilator, mild analgesic
Potassium iodide	Negative	scar tissue
Salicylate	Negative	myalgias, arthritis (analgesic)
Xylocaine	Positive	bursitis, neuritis (analgesic)
Zinc	Positive	Slow-healing dermal ulcers

First, why should iontophoresis be elected over other clinical options such as injections or oral medication? Perhaps a chart of advantages and disadvantages (table 18) of each of these modalities would be the most straightforward approach (Belanger, 2015):

Table 18: Modality Comparisons

	Advantages	Disadvantages
Injection	• Medication can be placed at a precise location • The depth of delivery can be precise • The process bypasses the need for the gastrointestinal system, liver, and kidney to metabolize large quantities of medication • There is a known amount of drug delivered to the tissue	• Invasive • Painful (or in some cases fearful) • Potential risk for infection • A professional is required for delivery • Costly
Oral Medication	• Quick and easy to administer • Systemic delivery = can address a large variety of tissues • Non-invasive • Can be administered independently	• Must pass through gastrointestinal system, liver, and kidney to metabolize the drug • Due to variability in metabolism, the actual amount of drug delivered to the tissue is unknown
Iontophoresis	• Local delivery to a specific tissue • Non-invasive so no risk of infection • The process bypasses the need for the gastrointestinal system, liver, and kidney to metabolize large quantities of medication	• Superficial delivery so target tissue must be appropriate • A professional is required for delivery • Mild skin irritation

A plethora of studies will be summarized to provide clinical justification for the use of iontophoresis. Prior to that, though, let's consider the early years of this technique, when there was uncertainty about some of the methods of delivery. For example, could more than one ion be delivered at one time? Did both ions need to be of the same polarity or could the polarity be switched mid-treatment? Electrodes were not as sophisticated as the current buffered electrodes. Studies like that of Pellecchia, Hamel, and Behnke (1994) examined the combination of modalities and transverse friction massage to that of iontophoresis for infrapatellar tendinitis. The iontophoresis treatment consisted of six sessions in which dexamethasone and lidocaine were both delivered. All four measures were used to assess patient status: a functional index questionnaire, a visual analog pain scale, a rating of tenderness with palpation of the involved tendon, and the number of step-ups needed to elicit pain, improved in the iontophoresis group. Whereas, only the step-ups showed significant improvement in the modality and transverse friction group. The researchers suggested that iontophoresis may be more effective and efficient in decreasing pain, reducing inflammation, and promoting healing in patients with infrapatellar tendinitis. Yet the use of two different ions needing two different polarities (Dex = negative; Lidocaine = positive) for delivery poses more questions about the process. Current buffered electrodes would no longer make the phoresing of two opposing ions possible.

A study by Gokoglu et al (2005) compared iontophoresis with dexamethasone to a local corticosteroid injection for carpal tunnel syndrome (CTS). Patients with clinical and electrophysiologic evidence of CTS (n=48) were divided into one of two groups: 40 mg of methylprednisolone acetate injected locally in the carpal tunnel or iontophoresis with of dexamethasone sodium phosphate. Outcome measures were a functional status scale, a symptom severity scale, and visual analog scale. There was a significant difference in all outcomes scales in both treatment groups at week 2 and week 8 compared to baseline. However, symptom relief was greater with the injection of corticosteroids.

Likewise, Stefanou, Marshall, Holdan, and Siddiqui (2012) compared treatments for lateral epicondylitis. They randomized 82 people into one of three groups: 10 mg dexamethasone iontophoresis delivered over 24 hours, 10 mg dexamethasone injection, and 10 mg triamcinolone injection. They all underwent the same therapeutic exercise program. Grip strength, pain, and functional scores were assessed. The grip strength was reported to be better with the iontophoresis group in the short term but by the 6-month follow-up, all groups had equivalent results.

In a recent study by Brown, Lauber, and Cappaert (2015), they explored the effect of dexamethasone iontophoresis on musculoskeletal conditions. They reported on three studies using dexamethasone with one deemed effective and two not effective. When exploring the parameters of each study, the conditions being treated can play a very significant role in determining the medication employed. Dexamethasone is an anti-inflammatory medication, yet two of the conditions, carpal tunnel syndrome and epicondylalgia, do not necessarily involve inflammation. Thus, it is not surprising that these conditions would not respond to iontophoresis. Whereas, epicondylitis is an inflammatory condition and did demonstrate improvement with six treatments at a 40 mA-min dosage.

Ketoprofen is another non-steroidal anti-inflammation (NSAID) medication used in iontophoresis. Panus, Ferslew, Tober-Meyer, and Kao (1999) compared iontophoresis with passive drug permeation. Their methodology utilized the medial thigh of swine to iontophorese 750 mg of ketoprofen via the cathode at 4 mA for 40 minutes. The passive drug

comparison was applied to the opposite thigh. The two conditions yielded equivalent results for the concentration of ketoprofen in the skin and fascia. However, the concentration in the first centimeter of muscle was significantly greater in the iontophoresis group. Of course the study was performed on swine but the skin composition is very similar to humans. The dosage of 160 mA-min is double that which is usually recommended but it is still valuable clinical information.

The use of dexamethasone, acetic acid, and placebo were assessed for the treatment of plantar fasciitis. Osborne and Allison (2006) administered six treatments to the plantar surface of the foot over a two week period to individuals in one of three groups:

- iontophoresis with 0.4% dexamethasone
- iontophoresis with 5% acetic acid
- placebo

All groups also received continuous LowDye taping and stretching instructions for the gastrocnemius/soleus muscles. At the end of the two weeks, all groups improved in pain and stiffness but the acetic acid group showed a greater improvement both after treatment and at a four week follow-up.

These results lend themselves to an interesting discussion about the physiologic effect of acetic acid. Although the dexamethasone group improved, it was not significantly better than the acetic acid response. Perhaps this may be related to the ability of the acetic acid to facilitate the absorption of plantarfascial spurring. The formation of a horizontal plantar spur is the desperate attempt of the calcaneus to hold onto the plantarfascia (figure 13). As one knows, bones are made of calcium carbonate and not fluid soluble. However, when treated with acetic acid, the calcium carbonate is transformed into calcium acetate which IS fluid soluble. Hence, acetic acid contributes to the reabsorption of the spur. This is consistent with the work of Gulick, Bouton, Detering, Racioppi, and Shafferman (2000). They performed pre-treatment radiographs on 10 individuals diagnosed with plantar fasciitis. They treated these individuals with acetic acid iontophoresis three times per week for four weeks. The iontophoresis dosage was 40 mA-min. At the end of the treatments, radiographs were retaken. The pre- and post-radiographs were interpreted by three different podiatrists who were blinded to the treatment and sequence of the imaging. Eight of the 10 participants demonstrated a reduction in the size and/or density of the calcaneal spurring.

Plantarfascia Heel Spur

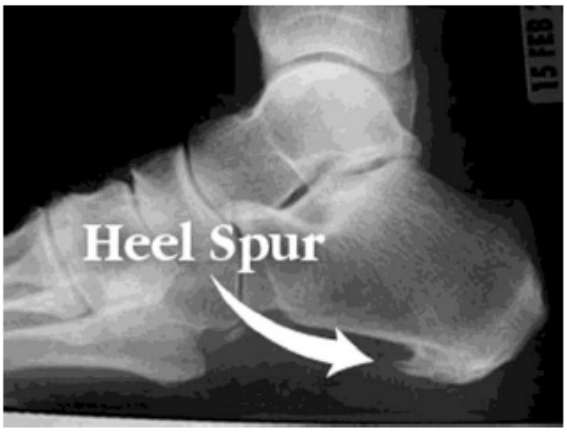


Figure 13

Despite the long term use of dexamethasone sodium phosphate (DSP) with iontophoresis, there is controversy over if, and how much, medication reaches the tissue. Joshi, Stagni, Cleary, Patel, Weiss, and Hagins (2014) compared the DSP concentrations with and without iontophoresis. The researchers delivered the active and passive iontophoresis to the forearm of eight healthy adults. Two mL of 4.4 mg/mL DSP was delivered at 4 mA for 20 minutes (80mA-min dosage). Interstitial fluid concentrations were measured by cutaneous microdialysis for two hours. Seven of the eight iontophoresis sites were reported to contain quantifiable levels of DSP but none of the passive samples contained the drug. This study demonstrates that iontophoresis significantly ($p < 0.0001$) increases delivery of DSP to the dermis when compared with passive delivery, and that microdialysis is an appropriate method to monitor DSP delivery.

Furthermore, to explore the depth of penetration, Gurney, Wascher, Eaton, Benesh, and Lucak (2008) performed a very unique experiment. They capitalized on the intention to extract the semitendinosis tendon from a patient for an anterior cruciate ligament reconstruction and performed an iontophoresis treatment with dexamethasone prior to extraction. A single 40 mA-minute dose was administered to 16 adults. The tendon slip was extracted within four hours. High-performance liquid chromatography mass spectrometry was used to analyze the concentration of dexamethasone in the tendon. The average concentration of dexamethasone was 2.9 ng/g of tendon tissue. Hence, iontophoresis appears to facilitate transmission of dexamethasone into connective tissue with skinfold thickness up to at least 30 mm.

So what are the important elements related to the depth of penetration? The primary resistance to ion transfer is the skin. It is the hair follicles and sweat glands that are areas of decreased resistance. Therefore,

tissue hydration is very important to enhance the permeability of the hair follicles and sweat glands. Skin that is dry or callused will often fail to conduct a current as well as transmit a medication. With aging, the stratum corneum decreases its permeability. Drying and a decrease in the microvasculature blood supply to the dermis can make the skin more resistant to the passage of hydrophilic substances through the skin. This could decrease the overall effectiveness of the therapeutic intervention (Brown, Lauber, & Cappaert, 2015).

In addition, one other topic should be addressed regarding the transmission of a medication. The use of other modalities before or after iontophoresis may influence the medication transmission. For example, if a moist heating pack were applied prior to or after an iontophoresis treatment, the vasodilation resulting from the heating has the potential to carry the medication away from the target tissue. On the contrary, the application of ice or a cold pack can cause vasoconstriction which at first glance may appear to be favorable to keep the medication local. However, analgesia is also a result of cold application and this could be problematic before iontophoresis in providing feedback on the treatment intensity. Yet, ice application after iontophoresis may facilitate vasoconstriction and keep the medication in the local tissue.

That being said, the next topic is the form of iontophoresis delivery. The standard method uses a clinical phoresor (figure 14) but alternative methods are disposable electrodes with low voltage batteries built into the patch (figure 15).

Standard Iontophoresor Set-up

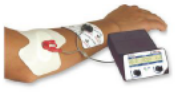


Figure 14

Sample Low Voltage Patches with Batteries Built-in

Figure 15

Regardless of the delivery method, there are a few important items that should be addressed. Table 19 provides the sequence of tasks in the rendering of an iontophoresis treatment.

Table 19: Sequence of Tasks of an Iontophoresis Treatment

- Determine the pathology present
- Identify the appropriate ion for the given treatment
- Identify the appropriate polarity for the selected ion
- Rule out the possibility of a drug allergy to the selected ion by placing a drop on the skin to determine if there is a reaction
- Inspect the body part for rashes, lacerations, or infections
- **NEVER** shave the area immediately before treatment
- Evaluate the patient's sensation of the treatment area
- Explain the procedure to the patient, i.e. there will be a tingling sensation that is dependent on the method of delivery. A standard phoresor will be a strong tingling sensation but a disposable low-voltage electrode will provide a very slight tingling sensation. The stimulus should **not** burn
- Prepare the electrodes
  - Standard phoresor = the appropriate quantity of the medication is injected into the active electrode and the inactive electrode is usually self-adhering with no additional preparation needed.
    - Electrode sizes include 1.5, 2.0, 2.5, & 4.0 cc
  - Low-voltage built-in battery = place the appropriate quantity of medication in the active well and saline in the inactive well.
- Cleanse the skin thoroughly with soap and water prior to the application of the electrode
- Secure the electrode(s) to the desired location. Care should be taken to **never** press on the center of the electrode for fear of pushing the fluid outward and interfering with the adhesive. Apply even pressure all around the perimeter (on the adhesive portion).
- Treatment time is dependent on the intensity of the stimulation.
  - Standard electrodes are usually applied for 15-40 minutes
  - Low-voltage electrodes can remain in place for 24 hours
- Skin should be monitored throughout treatment

As indicated earlier, dosage is a product of intensity and time. Intensity ranges from 0.1 to 4.0 mA depending on the device. Dosage is recommended to be in the range of 40 to 80 mA-minutes (Rothstein et al, 1998). One should treat on the lower end of the range if the individual is fair skinned, red-haired, and freckled, i.e. the individual who tends to burn easily at the beach. Individuals with darker skin tones can often be dosed at the upper end of the range. Thus, lower voltage devices require longer treatment times but they are designed to be worn throughout the day when on some surfaces (shoulder, elbow, knee) and over-night for others (ankle, foot, hand). The standard electrodes are designed to render the medication relatively quickly (15-40 minutes) but there may be a benefit to leaving the electrode on the patient after the removal of the phoresor. Once the phoresing process has begun, some have questioned the potential for passive diffusion continuing to the transmit medication. However, if the electrode is going to be left on, one should always be briefly peel it back to check the skin at the conclusion of the treatment. This is done because adverse reactions may happen. These include allergic responses, redness and even burns, chemical reactions due to the acidic or alkaline reaction of the direct current. Good skin and electrode preparation can help abate the adverse reactions. Increased skin resistance can produce heat as a byproduct and produce a skin burn. In addition, one should **never** re-use an iontophoresis electrode. Although very similar

to all other electrical stimulation interventions, contra-indications for iontophoresis are listed in table 20.

Table 20: Iontophoresis Contraindications
• any conditions that are contraindicated for electrical stimulation • never use an ion to which a patient is allergic • never on a desensitized area • never over denuded areas or new scar tissue • never allow the metal electrode to touch the skin • never apply to patients that do not understand the sensation of burning or cannot provide feedback! • never move or remove the electrodes while the unit is active • never use an electrode again

Beyond the literature to substantiate the use of iontophoresis, there are some frequently asked questions which are clinically important. These include:

What factors are important about electrode filling?	•Always fill the electrode with exactly the volume stated on the packaging •Always fill the electrode immediately before use to avoid inactivating the buffering & denaturing the medication
How many treatments of iontophoresis can be administered?	•All modalities should be constantly re-evaluated •If after 2-3 treatments, there is not a change in patient's status, a change of treatment should be considered •There is no maximum number of treatments but progress needs to be occurring to continue
How frequently can iontophoresis be rendered?	•Recommendations are TIW but never over an area on which the skin of a previous treatment has not returned to normal
What are the adverse effects of iontophoresis?	•Erythema, blistering •Dryness, flaking, itching

As for the handling of a medication, each professional must have a firm understanding of the practice act under which s/he is governed. The issues include is a prescription needed for the medication, who should obtain the medication, how is the medication cost/payment managed, and how is the medication stored? One should be sure to know the answers to these issues before embarking on the administration of an iontophoresis treatment.

Summary

In conclusion, clinicians have a wide variety of tools in the toolbox of therapeutic interventions. The choice of tools to use should be founded on evidence-based clinical decisions. This is not always as easy as it may sound since some of the literature is controversial. But choices should be based on physiologic principles and one should not be afraid to take a step back, re-evaluate, and change the intervention if it is not working. Detailed documentation of the intervention is critical for consistency. All parameters should be identified as well as the patient response.

Images

Figure 1

http://blog.electricalcommunity.com/difference-between-ac-and-dc/

Figure 2

http://www.electronics-tutorials.ws/accircuits/acp2.gif

Figure 11

http://s.hswstatic.com/gif/pain-2.gif

Figure 12

http://www.electrotherapy.org/modality/iontophoresis

References

1. Ardic F, Sarhus M, Topuz O. Comparison of two different techniques of electrotherapy on myofascial pain. Journal of Back & Musculoskeletal Rehabilitation. 2002;16(1):11-16
2. Armutlu K, Meric A, Kirdi N, Yakut E, Karabudak R. The effect of TENS on spasticity in multiple sclerosis patients: a pilot study. Neurorehabilitation and Neural Repair. 2003;17(2):79-82
3. Asadi MR, Torkaman G, Hedayati M, et al. Angiogenic effects of low-intensity cathodal direct current on ischemic diabetic foot ulcers: A randomized controlled trial. Diabetes research and clinical practice. 2017;21(127):147-155
4. Asadi MR, Torkaman G, Hedayati M. Effect of sensory & motor electrical stim in vascular endothelial growth factor expression of muscle & skin in full-thickness wound. Journal of Rehabilitation Research & Development. 2011, 48(3), 195-202.
5. Asadi MR, Torkaman G. Bacterial inhibition by electrical stimulation. Advances in wound care. 2014;3(2):91-97
6. Avramidis K, Karachalios T, Popotonasios K, Sacorafas D, Papathanasiades AA, Malizos KN. Orthopedics. 2011 Mar; 34(3): 175
7. Azatcam G, Atalay NS, Akkaya N, Sahin F, Alsoy S, Zincir O, Topuz O. Comparison of effectiveness of TENS and Kinesio Taping added to exercises in patients with myofascial pain syndrome. Journal of Back and Musculoskeletal Rehabilitation. 2017;30(2):291-298.
8. Bakhtiary AH, Fatemy E. Does electrical stimulation reduce spasticity after stroke? Clinical Rehabilitation 2008; 22: 418–425
9. Balogun JA, Onilari OO, Akeju OA, Marzouk DK. Archives of Physical Medicine & Rehabilitation. 1993 Sep; 74(9): 910-916
10. Becker RO, Spadaro JA, Marino AA. Clinical experiences with low density current stimulation of bone growth. Clinical Orthopedics. 1977;124:75-83
11. Belanger A-Y. Therapeutic Electrophysical Agents. 3rd ed. Wolters Kluwer Health, Phila, 2015
12. Brighton CT. The semi-invasive method of treating non-union with direct current. Orthopedic Clinics of North America. 1984;15:33-45
13. Brown CD, Lauber CA, Cappaert T. The effect of dexamethasone iontophoresis on decreasing pain and improving function in patients with musculoskeletal conditions. Journal of Sport Rehabilitation. 2015;24:327-331
14. Carley PJ, Wainapel SF. Electrotherapy for acceleration of wound healing: Low intensity direct current. Archive of Physical Medicine & Rehabilitation. 1985;66:443-446.
15. Chabal C, Fishbain DA, Weaver M, Heine LW. Long-term transcutaneous electrical nerve stimulation use: impact on medication utilization and physical therapy costs. Clinical Journal of Pain. 1998;14:66-73
16. Cheng N, Van Hoof H, Bockx E, Hoogmartens MJ, Mulier JC, DeCijcker FJ, Sansen WM, De Loecker W. The effects of electric currents on ATP generation, protein synthesis, and membrane transport of rat skin. Clinical orthopaedics and related research. 1982;171:264-272
17. Cheng N. The effect of electric currents on ATP generation, protein synthesis and membrane transport in rat skin in clinical orthopedics. 1982;171:264-272.
18. Chesterton LS, Foster NE, Wright CC, Baxter GD, Barlas P. Effects of TENS frequency, intensity and stimulation site parameter manipulation on pressure pain threshold in healthy human subjects. Pain. 2003;106(1-2):73-80.
19. Curtis D, Fallow S, Morris M, McMakin C. The efficacy of frequency specific microcurrent therapy on delayed onset muscle soreness. Bodyworks & Movement Therapy. 2010;14:272-279
20. Defrin R, Ariel E, Peretz C. Segmental noxious versus innocuous electrical stimulation for chronic pain relief and the effect of fading sensation during treatment. Pain. 2005;115(1-2):152-160.
21. DeSantana JM, da Silva LF, DeResende MA, Sluka KA. Transcutaneous electrical nerve stimulation at both high and low frequencies activates ventrolateral peraqueductal grey to decrease mechanical hyperalgesia in arthritic rats. Neuroscience. 2009;163:1233-1241.
22. Dolan MG, Graves P, Nakazawa C, Delano T, Hutson A, Mendel FC. Effects of Ibuprofen & HV Stim on Acute Edema Formation After Blunt Trauma to Limbs of Rats. JAT. 2005;40(2):111–115
23. Dolan MG, Mychaskiw AM, Mendel FC. Cool-water immersion & HV stimulation curb edema formation in rats JAT. 2003; 38(3): 225-30
24. Dolan MG, Mychaskiw AM, Mattacola CG, Mendel FC. Effects of CWI & HV stim for 3 continuous hrs on acute edema in rats JAT. 2003; 38(4): 325-9
25. Dowswell T, Bedwell C, Lavender T, Neilson JP. TENS for pain relief in labour. The Cockrane database of systematic reviews. 2009:15(2): CD007214

26. Ennis WJ, Lee C, Gellada K, Corbiere TF, Koh TJ. Advanced technologies to improve wound healing: Electrical stimulation, vibration therapy, and ultrasound – What is the evidence? Plastic Reconstruction Surgery. 2016;138:94S-104S

27. Facci LM, Nowotny JP, Tomem F, Trevisani VF. Effects of TENS and IFC in patients with nonspecific chronic low back pain: randomized clinical trial. Sao Paulo Medical Journal. 2011;129(4):206-216.

28. Feedar JA, Kloth LC, Gentzkow GD. Chronic dermal ulcer healing enhanced with monophasic pulsed electrical stimulation. Physical Therapy. 1991;71(9):639-649.

29. Ferguson JP, Blackley MW, Knight RD, Sutlive TG, Underwood FB, Greathouse DG. Effects of varying electrode site placements on the torque output of an electrically stimulated involuntary quadriceps femoris muscle contraction. Journal of Orthopedic & Sports Physical Therapy. 1989;11(1):24-29.

30. Fishbain DA, Chabal C, Abbott A, et al. TENS treatment outcome in long-term users. Clinical Journal of Pain. 1996;12:201-214.

31. Fujiya H, Ogura Y, Ohno Y, Goto A, Nakamura A, Ohashi K, Uematsu D, Aoki H, Musha H, Goto K. Microcurrent electrical neuromuscular stimulation facilitates regeneral of injured skeletal muscle in mice. Journal of Sports Science & Medicine. 2015;14(2):297-303

32. Gault WR, Gatens PF. Use of low intensity direct current in management of ischemic skin ulcers. Physical Therapy. 1976;56:265-269

33. Gokoglu F, Findikoglu G, Yorgancioglu ZR, Okumus M, Ceceli E, Kocaoglu S. Evaluation of iontophoresis & local corticosteroid injection in the treatment of CTS. American Journal of Physical Medicine & Rehab. 2005 Feb; 84(2): 92-96

34. Griffin JW, Tooms RE, Mendius RA, Clifft JK, Vander Zwaag RV, El-Zeky F. Efficacy of high voltage pulsed current for healing of pressure ulcers in patients with spinal cord injury. Physical Therapy. 1991 Jun; 71(6): 433-444

35. Gulick DT, Borger A, McNamee L. Effects of Analgesic Nerve Block Electrical Stimulation on Shoulder Pain, Range of Motion, & Function in a Patient with Adhesive Capsulitis. Physiotherapy Theory & Practice, 2007

36. Gulick DT, Bouton K, Detering K, Racioppi E, Shafferman R, Effects of Acetic Acid Iontophoresis on Heel Spur Reabsorption. Case Reports in Physical Therapy. 2000; 3(2):1-7

37. Gurney B, Wascher D, Eaton L, Benesh E, Lucak J. The effect of skin thickness and time in the absorption of dexamethasone in human tendons using iontophoresis. Journal of Orthopedic and Sports Physical Therapy. 2008;38(5):238-245

38. Holcomb, Rubley, & Girouard. Journal of Sport Rehabilitation, 2007, 16, 307-318

39. Houghton PE, Campbell KE, Fraser CH, Harris C, Keast DH, Potter PJ, Hayes KC, Woodbury MG. Electrical stimulation therapy increases rate of healing of pressure ulcers in community-dwelling people with spinal cord injury. Archives of Physical Medicine & Rehabilitation. 2010;91:669-678

40. Houghton PE, Kincaid CB, Lovell M, Campbell KE, Keast DH, Woodbury MG, Harris KA. Effect of electrical stimulation on chronic leg ulcer size and appearance. Physical Therapy. 2003;83(1):17-28.

41. http://www.itechmedicaldivision.com/en/brief_history_of_electro_stimulation-t-2.html

42. Joshi A, Stagni G, Cleary A, Patel K, Weiss DS, Hagins M. Iontophoresis successfully delivers dexamethasone sodium phosphate to dermis as measured by microdialysis. Journal of Pharmaceutical Sciences. 2014;103(1):191-196

43. Kalra A, Urban MO, Sluka KA. Blockade of opioid receptors in rostal ventral medulla prevents antihyperalgesia produced by TENS. Journal of Pharmacological Exp Ther. 2001;298:257-263

44. Kerai S, Saxena KN, Taneja B, Sehrawat L. Role of TENS in post-operative analgesia. Indian Journal of Anaesthesia. 2014;58(4):388-383.

45. Kim TH, Cho HY, Lee SM. High-voltage pulsed current stimulation enhances wound healing in diabetic rats by restoring the expression of collagen, a-smooth muscle actin, and TGF-b1. Tohoku Journal of Experimental Medicine. 2014;234(1):1-6

46. Koke A, Schouten JSAG, Lamerich-Geelen MJH, Lipsch JSM, Waltje EMH, van Kleef M, Patijn J. Pain reducing effect of three types of TENS in patients with chronic pain: A randomized crossover trial. Pain. 2004;108:36-42

47. Kramer J, Lindsay D, Magee D, Mendryk S, Wal T. Comparison of voluntary and electrical stimulation contraction torques. Journal of Orthopaedic & Sports Physical Therapy. 1984;324–331

48. Laddha D, Ganesh GS, Pattnaik M, Mohanty P, Mishra C. Effect of TENS on plantar flexors muscle spasticity and walking speed in stroke patients. Physiotherapy Research International. 2016;21(4):247-256

49. Law PP, Chein GL. Optimal stimulation frequency of transcutaneous electrical nerve stimulation on people with knee osteoarthritis. Journal of Rehabilitation Medicine. 2004;36(5):220-225

50. Lee KH, Chung JM, Willis WD. Inhibition of primate spinothalamic tract cells by TENS. Journal of Neurosurgery. 1985;62:276-287

51. Litke DS, Dahners LE. Effects of different levels of direct current on early ligament healing in a rat model. Journal of orthopaedic research. 1994;12(5):683-688

52. Maayah M, Al-Jarrah M. Evaluation of TENS as a treatment of neck pain due to musculoskeletal disorders. Journal of Clinical Medicine Research. 2010;2(3):127-136

53. Marco, FB. Brief History of Electro-Stimulation

54. McMakin C. Frequency specific microcurrent. 2017. https://frequencyspecific.com/faq/

55. Mendel FC, Dolan MG, Fish DR, Marzo J, Wilding GE. Effect of High-Voltage Pulsed Current on Recovery After Grades I & II Lateral Ankle Sprains. Journal of Sport Rehab. 2010;19:399-410

56. Mendel FC, Wylegala JA, Fish DR. "Influence of High Voltage Pulsed Current on Edema Formation following Impact Injury in Rats." Physical Therapy 72 (1992): 668-73.

57. Merriman HL, Hegyi CA, Albright-Overton CR, Carlos J Jr., Putnam RW, Mulcare JA. A comparison of 4 electrical stim types on staphylococcus aureus growth in vitro. Journal of Rehabilitation Research & Development. 2004 Mar-Apr; 41(2): 139-146

58. Mills PB, Dossa F. TENS for management of limb spasticity: A systematic review. American Journal of Physical Medicine & Rehabilitation. 2016;95(4):309-318

59. Nnoaham KE, Kumbang J. TENS for chronic pain. The Cochrane database of systematic reviews. 2008;16(3):CD003222

60. Noehren B, Dailey DL, Rakel BA, Vance CG, Zimmerman MB, Crofford LJ, Sluka KA. Effect of transcutaneous electrical nerve stimulation on pain, function, and quality of life in fibromyalgia: a double-blind randomized clinical trial. Physical Therapy. 2015;95(1):129-140.

61. Nourbakhsh MR, Fearony FJ. An alternative approach to treating lateral epicondylitis. Clinical Rehab 2008;22:601–609

62. Obajuluwa VA. Physiotherapy Theory & Practice. 1991 Sep; 7(3): 191-197

63. Osborne HR, Allison GT. British Journal of Sports Medicine. Treatment of plantar fasciitis by LowDye taping and iontophoresis 2006 Jun; 40(6): 545-549

64. Paillard T, Noe F, Bernard N, Dupui P, Hazard C. Effects of two types of neuromuscular electrical stimulation training on vertical jump performance. Journal of Strength and Conditioning Research. 2008;22(4):1271-1278

65. Paillard T, Noe F, Passelergue P, & Dupui P. Sports Med 2005; 35 (11): 951-966

66. Palmieri-Smith RM, Thomas AC, Karvonen-Gutierrez C, Sowers MF. Physical Therapy. 2010,90:1441-1452

67. Pantaleao MA, Laurino MF, Gallego NL, Cabral CM, Rakel B, Vance C, Sluka KA, Walsh DM, Liebano RE. Adjusting pulse amplitude during TENS application produces greater hypoalgesia. The Journal of Pain. 2011;12(5):581-590.

68. Parker MG ; Bennett MJ ; Hieb MA ; Hollar AC ; Roe AA. JOSPT. 2003 Dec; 33(12): 719-726

69. Pease WS. Therapeutic electrical stimulation for spasticity: quantitative gait analysis. American Journal of Physical Medicine & Rehabilitation. 1998 Jul-Aug;77(4): 351-355

70. Pellecchia GL, Hamel H, Behnke P. Treatment of infrapatellar tendinitis: a combo of modalities & transverse friction massage vs. iontophoresis. Journal of Sport Rehabilitation. 1994 May; 3(2): 135-145

71. Pellett R. Use of electrical stimulation in wound healing: a review of the literature. Acute Care Perspectives. 2000 Spring; 9(1): 9-15

72. Piva SR, Goodnite EA, Azuma K, Woollard JD, Goodpaster BH, Wasko MC, Fitgerald GK. PT. 2007, 87:1064-1077

73. Rakel B, Frantz R. Effectiveness of transcutaneous electrical nerve stimulation on post-operative pain with movement. Journal of Pain. 2003;4:455-464.

74. Reid B, Zhao M. The electrical response to injury: Molecular mechanisms and wound healing. Advances in Wound Care. 2014;3(2):184-201

75. Rothstein JM, et al. The Rehabilitation Specialist's Handbook. 1998. Philadelphia, F A Davis

76. Sandoval MC, Ramirez CR, Camargo DM, Russo TL, Salvini TF. Effect of high-voltage electrical stimulation on the albumin and histamine serum concentrations, edema, and pain in acute joint inflammation of rats. Brazilian Journal of Physical Therapy. 2015;19(2):89-96

-77. Santana LS, Gallo RB, Ferreira CH, Duarte G, Quintana SM, Marcolin AC. Transcutaneous electrical nerve stimulation reduces pain and postpones the need for pharmacological analgesia during labour. Journal of Physiotherapy. 2016;62(1):29-34

78. Sawant A, Dadurka K, Overend T, Kremenchutzky M. Systematic review of efficacy of TENS for management of central pain in people with multiple sclerosis. Multiple Sclerosis and Related Disorders. 2015;4(3):219-227

79. Simpson PM, Fouche PF, Thomas RE, Bendall JC. Transcutaneous electrical nerve stimulation for relieving acute pain in the prehospital setting: a systemic review and meta-analysis of randomized controlled trials. European Journal of Emergency Medicine. 2014;21(1):10-17.

80. Sluka KA, Bjordal JM, Marchand S, Rakel BA. What makes transcutaneous electrical nerve stimulation work? Making sense of the mixed results in the clinical literature. Physical Therapy. 2013;93(10):1397-1402

81. Sluka KA, Deacon M, Stibal A, et al. Spinal blockade of opioid receptors prevents the analgesia produced by TENS in arthritic rats. Journal of Pharmacological Exp Ther. 1999;289:840-846.

82. Sluka KA, Walsh D. Transcutaneous electrical nerve stimulation: basic science mechanisms and clinical effectiveness. The journal of Pain. 2003;4(3):109-121

83. Snyder AR, Perotti AL, Lam KC, Bay RC. Influence of High-Voltage Electrical Stimulation on Edema Formation After Acute Injury: A Systemic Review. Journal of Sport Rehab. 2010, 19, 436-451

84. Snyder-Mackler, L, Garrett M, Roberts M. A comparison of torque generating capabilities of three different electrical stimulating currents. Journal of Orthopedic & Sports Physical Therapy. 1989;10(8): 297-301.

85. Stefanou A, Marshall N, Holdan W, Siddiqui A. A randomized study comparing corticosteroid iontophoresis for lateral epicondylitis. Journal of Hand Surgery. 2012;37(1):104-109

86. Stevens-Lapsley JE, Balter JE, Wolfe P, Kohrt WM. Early Neuromuscular Electrical Stimulation to Improve Quadriceps Muscle Strength After Total Knee Arthroplasty: A Randomized Controlled Trial. Physical Therapy. 2011;92(2):210-226.

87. Talebi G, Torkaman G, Firouzabadi M, Mofid M, Shariat S, Kahrizi S. Effects of micro-amperage direct current stimulation on injury potential and its relation to wound surface area in guinea pig. Annual International Conference of the IEEE Engineering in Medicine & Biology Society. 2007;3516-3519

88. Ud-Din S, Bayat A. Electrical stimulation and cutaneous wound healing: A review of clinical evidence. Healthcare. 2014;2:445-467

89. Underwood FB, Kremser GL, Finstuen K, Greathouse DG. Increasing involuntary torque production by using TENS. Journal of Orthopedic & Sports Physical Therapy. 1990;12(3):101-104

90. Walmsley RP, Letts G, Vooys J. A comparison of torque generated by knee extension with a maximal voluntary muscle contraction vis-a-vis electrical stimulation. Journal of Orthopaedic & Sports Physical Therapy. 1984;6:10–17.

91. Wirsing PG, Habrom AD, Zehnder TM, Friedli S, Blatti M. Wireless micro current stimulation – an innovative electrical stimulation method for the treatment of patients with leg and diabetic foot ulcers. International Wound Journal. 2015;12(6):693-698

92. Wolcottt LE, Wheeler PC, Hardwicke HM, Rowley BA. Accelerated healing of skin ulcer by electrotherapy: preliminary clinical results. Southern Medical Journal . 1969;62(7):795-801

93. Wood JM, Evans PE, Schallreuter KU, et al. A multicenter study on the use of pulsed low-intensity direct current for healing chronic stage II & II decubitis ulcers. Archives of Dermatology. 1993;129:999-1009.

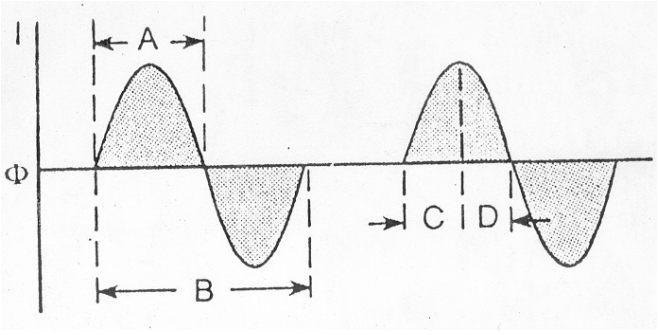
94. Young DG. Frequency specific microcurrent. 2017. http://www.angelfire.com/nd/drdavid/microcurrent.html

95. Zeng C, Li H, Yang T, Deng ZH, Yang Y, Zhang Y, Lei GH. Electrical stimulation for pain relief in knee osteoarthritis: systematic review and network meta-analysis. Osteoarthritis and Cartilage. 2015;23(2):189-202.

EVIDENCE-BASED ELECTRICAL STIMULATION

(3 CE Hours)

FINAL EXAM



1. **"C" on the image above is the _____.**
   a. Fall time
   b. Phase duration
   c. Pulse duration
   d. Rise time

2. **"Phase charge" is the area under the curve and is the product of _____.**
   a. Frequency & intensity
   b. Intensity & polarity
   c. Phase duration & intensity
   d. Polarity & phase duration

3. **Normal voluntary contraction recruits _____ twitch fibers first, and electrical stimulation recruits _____ twitch fibers first.**
   a. Fast; fast
   b. Fast; slow
   c. Slow; fast
   d. Slow; slow

4. **Which of the following is a "systemic" physiologic change from electrical stimulation?**
   a. Analgesic effects via beta-endorphins
   b. Modification of fibroblast/clast formation
   c. Skeletal muscle contraction
   d. Tissue remodeling

5. **Which of the following is NOT a contra-indication for electrical stimulation?**
   a. Cardiac pacemaker
   b. Individuals with epilepsy
   c. Individuals with a joint replacement
   d. In the area of a thrombi

6. **Based on the work of Holcomb, Rubley, and Girouard (2007), the "take home" message of the chart below is _____.**

Condition	Treatment	Mean Torque	% of Maximal Contraction
1	Maximal voluntary contraction	1000.1 ± 167.4	
2	Maximal voluntary contraction with ES	1015.0 ± 191.1	101.5%
3	ES to maximal comfort only	430.4 ± 121.2	43%

   a. Electrical stimulation should always be accompanied by a voluntary contraction
   b. Electrical stimulation should never be accompanied by a voluntary contraction
   c. There is no difference in the force production when electrical stimulation is added
   d. Voluntary force production is less than that of ES alone

7. **For strength-related muscle re-education, the parameter responsible for assuring fuel recovery for maximal subsequent contractions is _____.**
   a. A duty cycle of 1:2
   b. A duty cycle of 1:5
   c. A frequency of 20-30 pps
   d. A frequency >80 pps

8. **The objective of the manual motor point mapping technique is to find the site of _____.**
   a. Greatest intensity
   b. Highest impedance
   c. Largest duty cycle
   d. Lowest impedance

9. **All of the following theories have been suggested as viable methods to manage spasticity EXCEPT _____.**
   a. High volt galvanic current at 50 pps
   b. Stimulation of the antagonist at 50 pps & a duty cycle of 1:2
   c. TENS application at 100 pps
   d. Tetanic activation of the agonist at 100 pps in a continuous mode

10. Which of the following pulse rates (pps) are associated with a tetanic contraction?
a. 1 – 5 pps
b. 5 – 10 pps
c. 10 – 20 pps
d. 30 – 50 pps

11. Once edema has formed in an area of trauma, the parameter for the fluid shift approach are _____.
a. (-) current results in vasoconstriction & repels the (+) charged blood cells & proteins
b. (-) current results in vasodilation & repels the (-) charged blood cells & proteins
c. (+) current results in vasoconstriction & repels the (+) charged blood cells & proteins
d. (+) current results in vasodilation & repels the (-) charged blood cells & proteins

12. The evidence regarding wound healing supports the use of _____.
a. Alternating current & high-volt galvanic current
b. High-volt galvanic current & micro-current
c. Iontophoresis & alternating current
d. Micro-current & iontophoresis

13. Which of the following statements is correct about electrical stimulation for wound care?
a. (-) charge inhibits bacterial growth & decreases blood blow
b. (-) charge promotes cell migration
c. (+) charge creates an alkaline reaction
d. (+) charge promotes cell proliferation

14. At the conclusion of a wound care treatment, _____ drainage on the gauze pad indicates the patient was properly dosed.
a. Bloody
b. Cloudy
c. Serosanguineous
d. Serous

15. Based on the literature of Cheing, Tsui, Lo, and Hui-Chan (2003), the recommended treatment time for pain reduction and post-stimulation analgesia is _____.
a. 20 minutes
b. 40 minutes
c. 60 minutes
d. 120 minutes

16. The opiate pain control theory utilizes low rate TENS to stimulate _____.
a. A-delta & C-fibers via 1-5 Hz at a noxious intensity
b. A-beta via 1-5 Hz at a sensory intensity
c. A-delta via 100 Hz at a motor intensity
d. A-beta & C-fibers via 100 Hz at a sub-sensory intensity

17. Analgesic nerve block is theorized to utilize _____ to manage pain.
a. GTO inhibition
b. Muscle spindle facilitation
c. Raimiste facilitation
d. Wedensky inhibition

18. For iontophoresis, the _____ polarity is used for the delivery of _____.
a. Negative; lidocaine
b. Negative; ketoprofen
c. Positive; dexamethasone
d. Positive; acetic acid

19. An appropriate dosage for iontophoresis is _____.
a. 20 - 40 volts
b. 30 – 50 Hz
c. 40 – 80 mA-min
d. 100 – 120 amps

20. Which of the following statements is CORRECT?
a. All stim units are TENS units
b. If a unit is powered by a battery, it must be a DC unit
c. If a unit is powered via an AC plug, it can only be used for muscle re-education
d. Iontophoresis must be delivered via a DC unit

ANSWER SHEET

First Name: _____ Last Name: _____ Date: _____

Address: _____ City: _____

State: _____ ZIP: _____ Country _____

Phone: _____ Email: _____

License/certification # and issuing state/organization _____

Clinical Fellow: Supervisor name and license/certification # _____

Graduate Student: University name and expected graduation date _____

** See instructions on the cover page to submit your exams and pay for your course.

By submitting this final exam for grading, I hereby certify that I have spent the required time to study this course material and that I have personally completed each module/session of instruction.

Evidence-Based Electrical Stimulation Final Exam

1. Ⓐ Ⓑ Ⓒ Ⓓ	5. Ⓐ Ⓑ Ⓒ Ⓓ	9. Ⓐ Ⓑ Ⓒ Ⓓ	13. Ⓐ Ⓑ Ⓒ Ⓓ	17. Ⓐ Ⓑ Ⓒ Ⓓ
2. Ⓐ Ⓑ Ⓒ Ⓓ	6. Ⓐ Ⓑ Ⓒ Ⓓ	10. Ⓐ Ⓑ Ⓒ Ⓓ	14. Ⓐ Ⓑ Ⓒ Ⓓ	18. Ⓐ Ⓑ Ⓒ Ⓓ
3. Ⓐ Ⓑ Ⓒ Ⓓ	7. Ⓐ Ⓑ Ⓒ Ⓓ	11. Ⓐ Ⓑ Ⓒ Ⓓ	15. Ⓐ Ⓑ Ⓒ Ⓓ	19. Ⓐ Ⓑ Ⓒ Ⓓ
4. Ⓐ Ⓑ Ⓒ Ⓓ	8. Ⓐ Ⓑ Ⓒ Ⓓ	12. Ⓐ Ⓑ Ⓒ Ⓓ	16. Ⓐ Ⓑ Ⓒ Ⓓ	20. Ⓐ Ⓑ Ⓒ Ⓓ

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Evidence-Based Electrical Stimulation

(3 CE HOURS)

COURSE EVALUATION

Learner Name: _____

	Disagree				Agree	
Orientation was thorough and clear	1	2	3	4	5	
Instructional personnel disclosures were readily available and clearly stated	1	2	3	4	5	
Learning objectives were clearly stated	1	2	3	4	5	
Completion requirements were clearly stated	1	2	3	4	5	
Content was well-organized	1	2	3	4	5	
Content was at or above entry-level knowledge	1	2	3	4	5	
Content was substantiated through use of references, footnotes, etc.	1	2	3	4	5	
Content reflected stated learning objectives	1	2	3	4	5	
Exam assessed stated learning objectives	1	2	3	4	5	
Exam was graded promptly	1	2	3	4	5	
Satisfied with learning experience	1	2	3	4	5	
Satisfied with customer service (if applicable)	1	2	3	4	5	n/a

What suggestions do you have to improve this program, if any?

_____

_____

What educational needs do you currently have?

_____

_____

What other courses or topics are of interest to you?

_____

_____