# NeuroMuscular Electrical Stimulation

## - A Practical Guide -

3rd Edition

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**Rehabilitation Engineering Program**
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The clinical information in this text is based on programs developed at Rancho Los Amigos Medical Center, and through integration of current literature in the area of NMES. These programs do not represent the only ways electrical stimulation may be used. Improvements and modifications in treatment programs have been made from earlier editions of this text, and future research will require continued modifications.

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# HISTORY OF ELECTRICAL STIMULATION

Treating disease with electricity has intrigued man for centuries. Early civilizations prescribed shock-producing elements of the environment, such as electric fish and rubbed amber, to cure ills ranging from headache to hemorrhage. Nineteenth century investigators, including Faraday and Duchenne, designed mechanical current generators and established methods of 'localized electrization' over muscle motor points.[78] At the same time, imaginative entrepreneurs promoted an assortment of electric inventions claiming to do everything from extracting poison from the body to strengthening the generative organs and restoring manly vigor.[84] Although federal regulation has controlled more contemporary therapeutic applications of electrical current, technological advancement has expanded the possibilities of its use. The limits of electrical stimulation have not been reached, and even its current sophisticated uses soon will be as antiquated as the methods of the past.

## HIGHLIGHTS IN EARLY ELECTRICAL STIMULATION

Torpedo fish, capable of generating substantial electric shocks of 100-150 volts, were recommended as therapeutic agents as early as 400 B.C.[26,84,125] Topically applied to the head, these electric fish were reported to relieve headache;[125] placed under the feet, the torpedo cured arthritis.[78] A diet including boiled torpedo was prescribed for asthma.[84] Even hemorrhoids were effectively treated, although this application was not described!

Amber, a fossilized resin, was noted by the ancients to produce shocks after being rubbed. It was given as a pill to cure inflammation, hemorrhage and nausea.[26] A derivative of *electrica*, from the Latin word for amber, electricity was used in the 1600s to describe the force which tingled the senses and moved the limbs.[78] By 1744 static electricity was being used to treat paralysis. Kratzenstein, a German physician, described "a woman who lost the paralysis in her small finger within one quarter hour by electrification."[84]

In 1745 the invention of the Leyden jar, forerunner of the electrical capacitor, extended the applications of electricity by providing the ability to store quantities of charge for later use. Such disorders as kidney stones, sciatica, epilepsy and angina pectoris, as well as paralysis, were soon reported to be 'successfully' treated with electricity.[84] A representative cure is described in the following 1753 report by the practitioner Samuel Quelmalz:

> "A young man of 18 with hemiplegia of two years duration was unable to stand or walk and had lost his speech. His fingers were held in voluntary flexion so that he was unable to put on his shoes by himself. His arm was motionless and his hand cold...I applied some shocks to his hand in the morning and again in the afternoon. After a few days he returned and was able to move the arm more freely and also to speak with greater ease. Electric shocks were given once or twice a week. Soon he recovered so much function that he no longer complained of inability to finger the violin as he had previously." [84]

In spite of convincing testimonials of the therapeutic value of 'electric shocks,' it was not until 1791 that published reports linked muscle contraction to electrical stimulation of nerve. Luigi Galvani observed that application of dissimilar metals to the nerve of frog muscle induced muscular contraction.[84] Galvani assumed that 'animal electricity' was generated by the nervous tissue and stored in muscles, which he compared to Leyden jars, and that the metal only provided a path to discharge the inherent energy.[78,84] Alessandro Volta, however, contradicted this assumption and charged that muscular contractions were not autogenic but actually were due to quantities of electricity produced intrinsically at the junction of the different metals. The controversy was resolved several years later when Volta constructed a dependable source of continuous electric current in 1799.[78] The forerunner of the battery, this 'voltaic pile' consisted of alternating silver coins and zinc discs, each separated by a water-soaked card.[26] The steady output of the device was named 'galvanic current.' [84] When current was applied to muscle, Volta noted that "... a contraction takes place only at the first flow of electricity, and sometimes also at the breaking of the circuit." [78]

In 1801 the importance of the rate of amplitude rise (or increase) of the stimulating current was observed by Ritter. Using a battery of cells in series, Ritter gradually increased the current to the muscle by including cells in the circuit one at a time, and then slowly reduced it by removing them. He concluded that "if the exciting stimulus were not applied with briskness, the muscle would not contract." [78]

Although Galen had divided nerves into sensory and motor classifications in the second century, it was not until 1822 that Magendie made a formal distinction between them. This led to the first experimentation with 'electropuncture,' in which electric current was applied to needles inserted into muscles and nerves, a process similar to Japanese acupuncture. Muscle contraction was produced, but the associated pain and lack of therapeutic benefits led to the method's decline.[78]

Duchenne de Boulogne, was nevertheless intrigued by electropuncture and continued to explore its use during the 1830s. His design of cloth-covered, transcutaneous electrodes and method of 'localized electrization' over specific areas of muscles (later determined by Remak to be points of entry of the muscular nerves) contributed to labeling Duchenne the 'father of electrotherapy.' [26,78,84]

In 1831 Michael Faraday advanced electrical hardware with his electromagnetic machine, the forerunner of the electric generator and many devices used in electrotherapeutics today. The alternating current, induced in a metal wire rotated in a magnetic field, was termed 'faradic' by medical practitioners.[84] Until Duchenne introduced surface electrodes, the method for general faradization consisted of seating the patient, deprived of most of his clothing, with bare feet upon a sheet of copper. The copper was connected to one pole of the generator, while the other electrode consisted of a wet sponge or the operator's hand applied to the appropriate area of the patient's body.[26]

In 1840 the introduction of electricity into muscles was recommended as a diagnostic tool. Several investigators noted that paralyzed muscle responded to galvanic but not to faradic current. It also was noted that if a continuous current was rapidly interrupted, no contraction was produced by a stimulated muscle when the interruptions exceeded a certain rate. An important conclusion in electrodiagnosis was drawn -- that the duration of the current was a deciding factor in eliciting contraction. Other investigators confirmed the time factor of stimulation. In 1909 Madame and Louis Lapicque developed the first expression of adequate stimulus, including both time and intensity. Using a pulley circuit breaker, which afforded greater accuracy of measurement, Lapicque named the threshold of excitation 'rheobase.' He called the minimal duration of current needed to excite the tissue at double the rheobase intensity the 'chronaxie.'[78]

Curves of threshold stimuli at varying current intensities and durations were graphed for laboratory animals by Engelmann in 1870, but it was not until 1916 that Adrian mapped strength-duration curves for healthy and diseased human muscle.[78] In plotting these relationships, Adrian noted a significant shift in the curves for muscle with nerve degeneration and certain characteristic changes in muscle undergoing neural regeneration.[125]

In 1907 Bordet was the first to use accommodation measurements to identify denervation.[78,125] He observed that muscles with intact nerves adapted to stimuli which increased in intensity over a period of time, while muscles deprived of their nerve supply did not demonstrate that property. The difference in accommodation of innervated and denervated muscle became the basis of two electrodiagnostic tests, the galvanic-tetanus ratio and the progressive current ratio.[78]

With increased understanding of electrical stimulation, and such improved devices as the battery and the induction coil, enthusiasts of electrotherapeutics searched for diseases to cure. The latter half of the nineteenth century became the 'Golden Age of Medical Electricity,' and most physicians in America possessed at least one electrical stimulator.[84]

Devices providing high frequency currents were said to relieve the pain of rheumatism, neuralgia, gout, fractures, bruises and cuts. One such machine, called an 'electrostatic bath,' showered the entire body with charges. Presumably the device had beneficial effects, although it is unclear from the descriptive procedure:

> "Placed on the insulated stool...the patient is more or less highly charged with electricity, which is silently received without pain, as it does not pass by disruption. The hair is deflected from the scalp, the surface becomes warm, and cutaneous circulation is active, the face flushed, the action of the heart is quickened, and the pulse is more rapid. A general sense of tingling in the skin is experienced, and an abundant perspiration breaks out over the body. If now the knuckle of the operator, or a brass knob, is presented to any part of the body, a spark passes with a stinging sensation, and a weal is ultimately formed."[84]

Hydroelectric baths, resembling whirlpool tanks, were advertised to be "an auxiliary in the treatment of diseases of woman...a boon of greater value to her than has been discovered during the last 50 years."[84] Cardiocirculatory problems, on the other hand, were treated by immersing the extremities in the four-cell bath of Schnee.[84]

A patent was issued in 1898 for a device which claimed to be able to extract poison from the body. The inventor described his method:

> "For vegetable poisons I employ a vegetable receiver instead of a mineral or copper one and for animal poisons I use an animal receiver, such as raw meat, the device being capable of use with the...kind of receiver applicable to the kind of poison desired to be extracted..."[84]

The 'Pulvermacher Bi-Polar Electric Belt and Suspensory Appliance (Latest Improvement),' featured in the Pulvermacher Galvanic Company's 1889 catalogue, promised a "...cure of weak, nervous and debilitated conditions of the generative organs." Its description was detailed:

> "Continuous electric currents are constantly traveling through the generative organs, the small of the back and every adjacent part, at once healing, strengthening and invigorating the organs and speedily removing every symptom of waste and decay..."
>
> "Happily for the sufferer, these disorders, in all their various forms, yield readily to electricity... This treatment is now offered to every sufferer, and may be relied on for a complete cure, and restoration to health and manly vigor."[84]

## CONTEMPORARY DEVELOPMENTS IN ELECTRICAL STIMULATION

In addition to -- or in spite of -- these novel uses of electrical stimulation, progress continued to be made in understanding basic relationships between electricity and physiologic processes. In 1887 the electromotive changes accompanying the human heart beat were demonstrated by Augustus Waller. Using the capillary electrometer, which allowed heart currents to be recorded in a manner that could be photographed, Waller called this representation of heart

electrical activity an 'electrogram' or 'cardiogram.' Einthoven, who invented the string galvanometer and used it for electrical recording in 1903, later introduced the word 'electrocardiogram.'[26]

The door to cardiac pacemaking was opened in 1931 by Albert Hyman, who demonstrated that animals with induced cardiac arrest could be successfully resuscitated with electric currents. In 1952 Paul Zoll showed that an artificial pacemaker could maintain a human heart beat for at least a limited period. Longer term cardiac maintenance was accomplished by Furman and Schwedel in 1958, in a patient who was paced without complications for 96 days. Although these early pacemakers were awkward, nonportable units, technological improvements during the following few years resulted in reliable, implanted pacemakers which required no outside charging. The fact that millions of patients have been implanted with cardiac pacemakers is a tribute to success in at least one area of electrical stimulation.

Notable improvement in electrodiagnostic techniques occurred after Piper recorded voluntary contractions in human muscles with the string galvanometer in 1907, and subsequently pioneered the development of electromyography. In 1929 Adrian designed the coaxial needle electrode, which made possible the detection of the potential developed by a single muscle fiber. Using a loudspeaker, Adrian also introduced the electromyographic sound record. In 1935 Lindsley made the first recordings from a patient with myasthenia gravis and demonstrated marked fluctuations in the amplitude of motor unit responses during contraction. Denny-Brown and Pennybacker differentiated between fasciculation and fibrillation in 1938. In 1941 the characteristic potentials of myotonia were recorded, as well as the rhythmic potentials in the rigid muscles of patients with Parkinsonism. In 1950 a relationship between muscle electrical potential and force of contraction was recognized.[78]

An increase in the number of peripheral nerve injuries during World War II also increased the interest in electrotherapy. Improved electronics, such as Bauwen's constant current impulse generator, made determination of chronaxie values and strength-duration curves less tedious and less time consuming.[78] The use of electric current for stimulation, as well as for diagnosis of peripherally denervated muscle, became common.

Electrodiagnostic procedures moved beyond the muscle with measurement of nerve conduction velocities. In 1948 Hodes, Larrabee and German stimulated nerves at different levels and noted temporal differences in muscle response. They determined neural conduction time by correlating these differences in response time with the length of the neural pathway.[78]

In addition to cardiac pacemakers and more refined electrodiagnostic procedures, contemporary uses of electrical current have ranged from treatment for chronic pain to overcoming paralysis. The term *NeuroMuscular Electrical Stimulation* (NMES) has been officially identified with the external control of

innervated, but paretic or paralytic muscles by electrical stimulation of the corresponding intact peripheral nerves.[43] The purpose of such external activation is to achieve therapeutic goals. When the specific goals entail functional and purposeful movements, and when the NMES intervention is planned for long term management of these movements, the specialized term *Functional Electrical Stimulation* (FES) is applied. Thus, FES is a subset of treatment programs of the more broadly defined term NMES.[43]

One of the first clinical applications of this concept of FES/NMES was a single-channel electrical stimulation system, designed to dorsiflex the ankle of a hemiparetic patient during the swing phase of gait.[77] Since 1960 many modifications of this device have been developed and evaluated. It was estimated in 1976 that various systems had been made available to approximately 2,000 patients to correct 'foot drop.'[100] Correction of more complex anomalies of hemiparetic gait has been attempted by patterned excitation of up to 16 different muscles during the gait cycle.[138,140] Both implanted[151] and surface[6,7,71,140] electrodes have been used in these various applications of lower extremity FES.

Electrical stimulation has also been used to evoke and control upper extremity muscle activity in patients with central nervous system disorders. Applications range from cyclical stimulation for contracture correction[14] to programs incorporating various audio and visual feedback of activity with NMES,[20] to more sophisticated implanted systems for enhanced hand function in the quadriparetic patient.[65]

Improved electronic technology and hardware have made other contemporary applications of electrical stimulation almost too numerous to describe. Several of these technologies include: 1) electrophrenic respirators which artificially ventilate high level spinal cord injured patients;[84] 2) both implanted[1,84] and transcutaneous devices[84] which are used successfully to treat chronic pain; 3) stimulators which void the bladder for paralyzed patients[84,104] and treat urinary and anal incontinence;[84] 4) stimulators implanted in the cerebrum and/or cerebellum to reduce spasticity and intractable epileptic seizures;[29,33] 5) implanted,[18] as well as surface[4,22] stimulators to treat and correct scoliosis; and 6) implanted cochlear systems for individuals with hearing impairments.[74]

It is difficult to estimate future applications of electrical stimulation within the medical and therapeutic domain. Current studies are focusing on designing visual prostheses, while cortical stimulators, which alter behavior, already have been developed.[84] Only the imagination limits the ways that the motor, sensory and emotional centers of man might be augmented or governed by artificial stimulation. It is likely that ethical questions, rather than technological answers, will determine the boundaries.

# PHYSIOLOGIC BASIS OF EXCITABLE TISSUE

The excitability of nerve and muscle provides the basis for the therapeutic use of electrical stimulation. This discussion is intended to be a general review of the fundamental neurophysiology of nerve and muscle excitation, so that the principles of electrically stimulated responses can be clearly understood. This is not intended to be an extensive review of electrophysiology, and it is assumed that the reader is familiar with the material included. Many textbooks, monographs and articles address nerve and muscle excitation in a detailed manner and should be consulted for further study.[32,56,59,64,67,152]

## NERVOUS TISSUE AND THE NERVE IMPULSE

The basic unit of nervous tissue which supports the communication network of the body is the nerve cell, consisting of a cell body and its fiber processes of axon and dendrites. This discussion will emphasize the peripheral fibers, particularly excitation of those concerned with motor and sensory functions. Three main types of nerve fiber have been identified -- groups A, B and C. Various characteristics of these fiber types are listed in Table 1.

Generally, the greater the fiber diameter the more rapid the conduction velocity, the lower the threshold of excitability to a peripherally applied electrical stimulus, the larger the recorded electrical response (action potential), the shorter the duration of the excitatory response, and the shorter the refractory period.[64] The A and B fibers are myelinated, which acts to increase conduction velocity, while the C fibers are almost devoid of myelin. The A fibers are largest (2-20μ in diameter) and thus conduct the most rapidly (12-120 m/sec). They are both motor and sensory in function and may be subdivided on the basis of conduction velocity into alpha, beta, gamma and delta fibers.[64] The B fibers are smaller (1-3μ) and slower (3-15 m/sec) than A fibers, and are autonomic in function.[64] The C fibers, the smallest (less than 1μ diameter) and slowest (2 m/sec or less) of the principal fiber types, are found in cutaneous and visceral nerves, and are associated with pain information, reflex responses and autonomic functions.[64] The A, B and C nerves travel together between the spinal cord and periphery, forming bundles of fibers of different sizes and functions. When an electrical stimulus is applied to a peripheral nerve, not only will motor fibers be excited, but also sensory fibers, if the parameters of the stimulus meet or exceed the thresholds of excitation.[67] It is unlikely, however, that direct activation of B and C fibers would occur under

**TABLE 1.** Mammalian Nerve Fiber Types and Characteristics

| Fiber Type | Fiber Diameter (μ) | Conduction Velocity (m/sec) | Spike Duration (ms) | Absolute Refractory Period (ms) | Peripheral Organ | Receptor Organ | Function |
|---|---|---|---|---|---|---|---|
| A fibers (motor) | | | | | | | |
| alpha | 12-20 | 70-120 | 0.4-0.5 | 0.4-1 | Muscle | | Somatic motor |
| gamma | 3-6 | 15-30 | | | | | Motor to muscle spindle |
| A fibers (sensory) | | | | | | | |
| Group Ia | 12-20 | 70-120 | 0.4-0.5 | 0.4-1 | Muscle | Annulospiral spindle endings | Proprioception |
| Group Ib | | | | | | Tendon organs of Golgi | |
| Group II | | | | | 1. Extensor muscles | Flower spray of spindle | Touch, pressure, vibratory receptors |
| beta | 5-12 | 30-70 | 0.4-0.5 | 0.4-1 | 2. Flexor muscles | Flower spray of spindle | |
| | | | | | 3. Skin | Touch-pressure receptors | |
| Group III | 2-5 | 12-30 | 0.4-0.5 | 0.4-1 | 1. Muscle | Unknown pain receptors(?) | Pain - (fast?), temperature (cold, heat) |
| delta | | | | | 2. Skin | Pain | |
| B fibers | 1-3 | 3-15 | 1.2 | 1.2 | | | Preganglionic sympathetics |
| C fibers Group IV | 0.5-1 | 0.5-2 | 2 | 2 | Muscle and skin | Pain | Pain - (slow), temperature, mechanoreceptors |

normal levels of therapeutic electrical stimulation, and very unlikely when the short stimulus pulses of NMES are used.

The nerve action potential (AP) is the message unit used by the nervous system to transmit information over a distance. Informational content of a stimulus depends primarily on the frequency of impulses transmitted by a nerve fiber, the number of fibers activated, and the synaptic connections that a nerve makes. Each AP is fundamentally identical within the same nerve, although slight variations occur between different nerves. The AP may be characterized as a brief electrical event of approximately one millisecond (ms) duration, involving up to a 120 millivolt potential change across the nerve membrane. The AP is propagated between the periphery and the central nervous system (CNS), and within the CNS by afferent and efferent axons. Although all of the underlying membrane events are not fully understood, an action potential, once generated, is normally self-sustaining throughout its travel along the nerve process.

The continuity of the myelin encircling the larger peripheral nerve fibers is interrupted regularly for short distances along the fiber, forming areas of exposed nerve called 'nodes of Ranvier.'[64] Although the lipid based myelin insulation is merely a structural modification around the nerve fiber and does not alter the mechanism of membrane excitation, it allows the nerve impulse to 'jump' between nodes, a phenomenon referred to as saltatory conduction. The effect of saltatory conduction can be to increase conduction velocity for a given fiber diameter more than 25-fold.[67,142]

As has been generally known for a quarter of a century, the electrical excitability of the nerve membrane is dependent on its voltage-sensitive permeability.[32,59] Due largely to structural characteristics of the membrane, and maintained over long periods of time by active transport through metabolic membrane pumps, the nerve cell interior is rich in potassium ions and poor in sodium ions (Fig. 1). Both ions tend to flow passively through the cell membrane down their concentration and electrotonic gradients. However, in its resting state the membrane is much more permeable to potassium than to sodium or other cellular cations. The resulting unequal ionic distribution maintains a resting membrane potential of 70-90 millivolts, with the inside electrically negative with respect to the outside. A similar situation is found in muscle fibers.

The general development and transmission of the AP in nerve and muscle is well understood, although details are complex and subject to debate. In the most simple terms, the AP is triggered by a stimulus which reduces the membrane potential (i.e., the difference between inside and outside voltage). The triggering stimulus may be natural, such as a touch activation of a sensory receptor, or may be induced by an externally applied current source, as in the case of NMES. The trigger stimulus, regardless of its source, results in an explosive opening of sodium

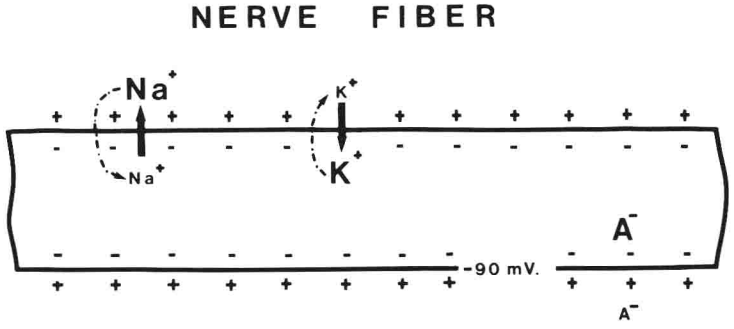


**FIGURE 1. Nerve Fiber.** A membrane potential of approximately -90 millivolts is established in the normal resting nerve fiber. Concentration differences of sodium ($Na^+$) and potassium ($K^+$) ions, as well as of cellular anions ($A^-$) exist due to the structural characteristics of the membrane. The passive diffusion (broken arrows) is countered by the active transport system (solid arrows), allowing a steady state condition to prevail.

selective membrane channels, and the beginning of an inward flow of sodium ions driven by the large concentration and electrotonic gradients. The membrane potential is briefly reversed to a positive potential, the inside assuming an electrically positive relationship with respect to the extracellular fluids. During the action potential, potassium ions also cross the membrane, exiting from the nerve fiber interior. As the equilibrium potential for sodium is approached, the driving force of sodium decreases and finally stops as the membrane channels for sodium close. This closure of the sodium gates appears to be timed, related to the initial triggering stimulus. The return to a selective potassium permeability restores the original diffusional and electrotonic gradients and results in a reestablishment of the potassium dominated resting potential.[64,67] This transient process is completed in about a millisecond for the nerve and within a few milliseconds for the muscle.

The event that triggers the generation of the AP is called an adequate stimulus. Many different kinds of stimuli, including chemical, electrical, thermal or mechanical, may act to excite the nerve, but the mechanism of excitation is always membrane depolarization. To be effective in causing depolarization, a stimulus must satisfy certain criteria. An adequate stimulus must be of sufficient magnitude (intensity) and must be applied for a long enough time to equal or exceed the threshold of excitation for the tissue. The threshold for nerve and muscle tissue differs; thus, the adequate stimulus for both tissue types will vary.

Nerve differs in threshold from muscle tissue and variations exist even among nerve fibers themselves. The basic relationship between a particular tissue and the intensity and duration of an adequate *electrical* stimulus traditionally is represented as a 'strength-duration' or intensity-duration curve. Such a curve compares the length of time the stimulus current of a particular intensity must be applied to a tissue to excite it. Figure 2 shows a schematic representation of the variation in

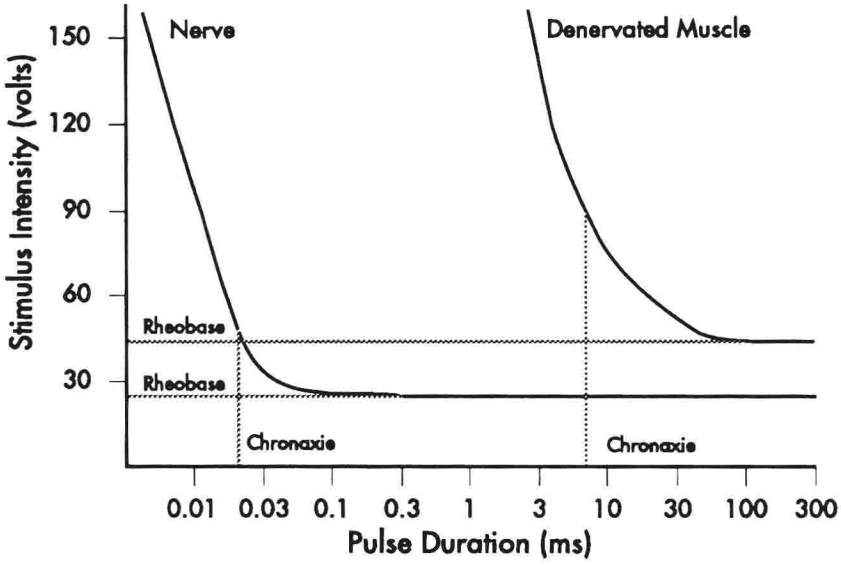


**FIGURE 2. Strength-Duration Curves.** Each curve relates the intensity (strength) of an electrical stimulus to the length of time it must be applied to an excitable tissue to produce a response. The left curve represents the excitability of a nerve, the right curve characterizes the excitability of denervated muscle. Rheobase, the intensity of current just sufficient to excite the tissue with an infinitely long pulse, and chronaxie, the length of time a current at twice rheobase intensity must be applied to produce an identical response, are shown.

excitability between nerve and muscle. Important concepts to note are: 1) stimuli of shorter duration require rapidly increasing intensities in order to excite the tissue; 2) stimuli of extremely short duration will not cause depolarization except with intensities impractically high for most clinical purposes; and 3) conversely, a point is reached with low intensity stimuli when no AP is generated regardless of the duration the current is applied.[45] The magnitude of current just sufficient to cause excitation of a particular type of tissue with a very long pulse duration (at least 300-1,000ms) is called *rheobase.* Rheobase is measured as an intensity, preferably as current. Often, in the past, rheobase was reported as the voltage output of the *stimulator,* without regard for the amount of current passing into the tissues. However, it is the actual *current* which determines excitation. Specifying

voltage output does not yield current unless the impedance of the tissue/electrode interface is also known. The use of voltages has contributed greatly to inaccuracies and inconsistencies associated with the clinical measurement of rheobase.

Because the rheobase of both nerve and muscle is relatively low and fairly similar, it has become customary to measure the length of time (pulse duration) required for threshold activation at a current intensity which is twice the rheobase. This value is reported as *chronaxie*. As seen in Figure 2, chronaxies for nerve and muscle are significantly different. Although the use of rheobase and chronaxie measures have been largely superseded by the electromyograph (EMG), there are some pathologic conditions where the surface assessment of excitable tissue is as accurate, or potentially more accurate, than the EMG.[47]

The familiar expression 'all-or-none' refers to the fact that an action potential elicited by a 'just threshold' stimulus is the same as an AP activated by a much stronger stimulus. At a given moment a nerve fiber fires maximally, if at all, and the propagated AP in a single fiber cannot be graded by altering the intensity or the duration of the triggering stimulus. During the AP and the propagation of the impulse, there exists a brief period in which the nerve is completely unexcitable to a second stimulus, regardless of intensity. This interval, called the *absolute refractory period*, is approximately the same duration as the action potential.[64] It occurs due to the changes in potassium and sodium ion conductances and the membrane state evoked by the first AP. Excitability is restored as the nerve's threshold to a second excitation returns to normal, and resting conditions reoccur (Fig. 3). Because of the absolute refractory period, a nerve cannot achieve a state of continuous excitation or a condition analogous to tetanization of muscle contraction. Fusion and summation of impulses do not occur in nerves; rather, within the limits of its excitability under normal conditions, the nerve fiber responds to stimulation with discrete, equal amplitude action potentials, and may be graded in its response only in terms of the *rate* of firing.

In mammalian nerves, the maximum AP frequency is approximately 1,000 per second.[64] Relatively high frequencies are routinely used physiologically within the sensory nervous system to allow gradation of the intensity of a sensation. Motor nerves, on the other hand, are generally not driven to high frequencies during normal function. In the practical motor function of man, most voluntary activity of large trunk and limb muscles is achieved with rates on the order of 20-25 action potentials per second or less.[56,64] Possibly related to this limited physiologic need, motor nerves demonstrate an extended *relative* refractory period which follows the absolute refractory time. Different motor nerves have longer or shorter relative refractory periods, ranging from 5 to over 100 milliseconds. During the relative refractory period, the motor neuron is less excitable than it is at rest, and requires a greater amount of current, either synaptic or electrical, to cause it to fire.

The information coded as individual action potentials must be transferred between nerves and from nerve to muscle or other effector organ if a behavioral