

Muscle Weakness Or Paralysis With Compromise Of Peripheral Nerve

Muscle weakness or complete paralysis may be secondary to an interruption in the communication between the brain and nerve cells in the spinal cord that govern muscle activation, or to an interruption in the lower motor neuron or peripheral nerve [including the motor nerve cells in the spinal cord and their processes which connect with the muscle fibers], or both. Examples of a peripheral nerve involvement or injury include pain and weakness in the wrist and hand because of injury to the median nerve at the wrist, known as carpal tunnel syndrome, and weakness or paralysis of the muscles of facial expression because of injury to the facial nerve [known as Bell's Palsy]. There are a number of acquired or inherited neurological conditions in which the lower motor neuron is lost, such as poliomyelitis, the axonal form of Guillain-Barre syndrome, or amyotrophic lateral sclerosis [also known as Lou Gehrig's disease].

If the motor axon, or nerve fiber, is inactivated by injection of a toxin that prevents transmission at the neuromuscular junction, the muscle will respond in the same fashion as if the motor nerve has degenerated. This same phenomenon may be observed when there is severe pathology of myelin, the insulating layer found around large sensory and motor nerve fibers. So, it is possible that muscle will behave as though it has lost its nerve supply in severe demyelinating disease, including the severe demyelinating form of Guillain-Barre Syndrome.

In some conditions, it is possible to have involvement of the nerve pathways in the spinal cord and loss of the lower motor neuron. In spinal cord injury, for example, there is an injury to the spinal cord with a zone of injury in which the motor nerve cells in the cord may be destroyed. In addition, motor nerve cells in the cord below the zone of injury may be lost because of an alteration in circulation or a secondary site of injury. Spinal stenosis is another example with compromise of the spinal cord as well as involvement of the nerves as they exit from the vertebral canal. In this orthopaedic disorder, the bone changes that occur can constrict the spinal cord in the spinal canal and cause narrowing of the tunnels through which the spinal nerves exit resulting in compromise of the spinal cord and the spinal nerve[s].

Peripheral Nerve Dysfunction

Weakness or paralysis associated with peripheral nerve injury may be caused by a temporary block in nerve conduction [neuropraxia or nerve block], a partial loss of the motor fibers in the nerve [partial denervation of muscle], complete loss of nerve supply to the muscle [complete denervation] or loss of the myelin or insulation around the peripheral nerve [known as demyelination]. It is not uncommon to have more than one of these problems contributing to the muscle dysfunction. Rehabilitation efforts, including electrical stimulation, designed to strengthen muscle or improve muscle performance are guided by the specific nature of the reasons for muscle weakness.

For example, if the facial muscles are paralyzed and the facial nerve is only blocked [ie neuropraxia], muscle activity can be expected to return when the block spontaneously resolves or medical measures are taken to relieve the problem. Although most neuropraxias clear within a few weeks, some nerve blocks remain for several months. Muscle strengthening during the period of nerve block would be unsuccessful because the brain cannot communicate with the paralyzed muscles. If the nerve block

persists and there is a problem with disuse of the muscles, especially in the face where it is important to prevent the muscles on the normal side of the face from pulling the facial structures toward the stronger side, electrical stimulation of the muscles may be employed to help maintain the muscles and minimize loss of facial symmetry. Because the motor nerve is blocked, but remains anatomically intact, stimulation can be accomplished easily and comfortably.

If there is partial or complete loss of myelin, efforts to strengthen muscle or improve muscle performance for function will not be successful, or will be limited, until remyelination has finished.

If there is partial loss of motor nerve supply to the muscle, or partial denervation, it is necessary to wait for reinnervation of the muscle before setting goals for muscle improvement. Depending upon the location and severity of nerve injury, reinnervation may take approximately 3 months to 2 years. The injured motor fibers may regrow at a rate of approximately 1 mm/day. In addition, the healthy motor nerve fibers may sprout within the muscle and adopt the orphaned muscle fibers. This process requires approximately 6 months to complete. Each motor nerve fiber can adopt 5-6 times the number of muscle fibers that it originally served and so there is a tremendous potential for reinnervation of muscle by this mechanism.

There is controversy about the use of electrical stimulation in partially denervated muscle. It has been shown that regular ES of the denervated muscle fibers will suppress the chemical mediators required for the reconnection of the axon twig with the motor endplate on the muscle as well as reduce the random electrical activity of the orphaned muscle fibers [fibrillation] which is thought to be a signal for sprouting of the remaining healthy motor nerve. Electrical stimulation of the muscle fibers that retain a nerve supply [in the partially denervated muscle] may simulate voluntary muscle overuse and contribute to the suppression of the chemical mediators required for the reinnervation of the denervated fibers.

If there is a complete loss of nerve supply to the muscle, the only mechanism for primary muscle reinnervation is regrowth of the nerve fibers at approximately 1 mm/day. If the nerve injury is proximal in the neck or back region, for example, it will take approximately 2 years for reinnervation to occur in the hand and foot. During this time there will be severe changes within the muscle including loss of muscle bulk and circulation. The connective tissue, or supportive lattice structure of the muscle shrinks and becomes adherent. This process has been called fibrosis. After several months of complete denervation, the muscle membrane properties change, the chemical bonds become permanent in the fibrotic muscle and the muscle is relatively non-responsive to electrical stimulation.

For the patient who is expected to have nerve regrowth after complete denervation, it is important to minimize the fibrosis within the muscle connective tissue so that there will be movable muscle structure after the nerve reinnervates the muscle. This potential movement within the muscle is necessary for the reacquisition of the contractile elements or contractile proteins that make muscles work.

There is controversy in the literature about the necessity for electrical stimulation of completely denervated muscle that is expected to reinnervate. There is evidence that electrical stimulation of the denervated muscle will augment regrowth of the nerve.

Previous investigators who expected to prevent the loss of muscle mass, or prevent atrophy of muscle, were not successful. It is possible to retard the loss of muscle bulk, but atrophy is a natural response to complete loss of nerve supply to the muscle. Investigators who were interested in minimizing the connective tissue contracture, or fibrosis, have demonstrated the efficacy of electrical stimulation during the denervation period. The studies date back to the 1950's and the number of animal or human subjects, or sample sizes were small, but the results were applicable to clinical practice. Electrical stimulation is the only intervention that will maintain the connective tissue mobility within the denervated muscle, with the goal of having a mobile end-organ when reinnervation takes place.

Comfort is a critical issue in ES of completely denervated muscle. A relatively longer pulse duration is required to cause contraction of the denervated muscle than for the muscle that has a nerve supply. Although textbooks may state that Galvanic current [300+ mS of current flow] will activate denervated muscle, Galvanic is painful when it is turned up to elicit a muscle contraction. A simple electrical stimulation test with a skin electrode [the strength-duration curve] can identify the shortest possible pulse duration required to contract the denervated fibers. The importance of using the shortest possible pulse is illustrated in the case of complete loss of the facial nerve. This nerve carries motor fibers to the muscles of facial expression. The skin on the face is normal because another nerve, the trigeminal nerve, allows us to "feel" our face. The Bell's palsy patient with muscles that can respond to 5 mS pulses could comfortably use this current and would be compliant with a home protocol. This patient could not tolerate the pain of excessive stimulus duration with Galvanic current [295 mS of unnecessary current flow in every pulse].

There is evidence to support the discontinuation of electrical stimulation [for completely denervated muscle] at the first sign of muscle reinnervation. Repeated electromyographic assessment at the estimated time for reinnervation [ie 1 mm/day times the distance for nerve regrowth] is required so that stimulation may be continued at an appropriate time for each of the muscles in the proximal and distal limb. This clinical approach is based upon work demonstrating that electrical stimulation of denervated muscle will prevent the acceptance of a nerve graft during the period of stimulation. This is thought to be due to the suppression of chemical mediators and spontaneous electrical activity in the denervated muscle.

Once the critical reinnervation period has elapsed and there is evidence of reinnervation, electrical stimulation may be useful to augment exercise protocols or provide sensory input for regaining functional capabilities.

Electrical Stimulation Of Permanently Denervated Muscle For Function

Individuals with completely denervated muscles and no anticipated nerve regrowth have previously not been considered to be candidates for electrical stimulation devices for functional control of their paralyzed muscle. As indicated above, the stimulus duration must be increased to activate denervated muscle. In addition, the quality of ES contraction of denervated muscle is slower and sluggish when compared to the brisk response obtained in normal muscle.

There is a research interest in functional electrical stimulation of chronically denervated muscle. A few patients who have volunteered to participate in long-term research protocols have benefited from improved muscle excitability and enough improvement in electrically stimulated muscle response in the knee muscles that ES could be used to assist in standing from sitting without the use of bracing at the knee. Scientists from several countries in the European community will be investigating the changes in denervated muscle with electrical stimulation and the clinical efficacy of electrical stimulation systems for activation of long-term denervated muscle over the next 5-10 years.

Peripheral Nerve Involvement In Spinal Cord Injury

Although it is possible to have injury along the pathway of a peripheral nerve in addition to spinal cord injury, the loss of motor nerve cells in the spinal cord is more common. Because most muscles receive nerve supply from more than one level of the spinal cord, there may be partial denervation. For example, the muscles that straighten the knee [the quadriceps] receive motor nerve supply from the second, third and fourth lumbar segments of the spinal cord. If the motor cells at L4 are lost, the muscles will retain L2 and L3. The remaining motor nerve may sprout within the muscles and adopt or reinnervate the orphaned muscle fibers in the ensuing 6 months.

In the case of partial denervation of muscle, efforts to strengthen the quadriceps volitionally or with ES in the first 6 months after injury cannot be expected to be effective. Rehabilitation dollars reasonably would be invested in muscle strengthening after 6 months when the muscle fibers are supplied by a nerve.

If all of the motor nerve cells responsible for innervating a muscle are lost, there is no natural potential for reinnervation. If the cells at L2,3 and 4 are lost, for example, the quadriceps will be completely denervated. As discussed above, ES has not been clinically employed to provide functional use of completely denervated muscle. There is, however, a research interest in this area.

Peripheral Nerve Involvement In Acquired And Inherited Disorders

Because of the variation in the type and severity of peripheral nerve involvement in acquired and inherited disorders, each individual patient requires thorough electrodiagnostic assessment prior to determining the most appropriate treatment. The principles discussed above do apply to candidacy for muscle strengthening or for controlling muscle with electrical stimulation.

Contributor:

J.M. Campbell, Ph.D., P.T.

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P.M. Meadows, M.S., B.M.E., Secretary IFESS
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