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Clinical Applications of Electrical Stimulation in Peripheral Nerve Injuries and Entrapment Neuropathies: A Narrative Review

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Abstract

Introduction and purpose: Peripheral nerve injuries and entrapment neuropathies are associated with chronic pain, sensory and motor dysfunction. Although the nervous system has some regenerative capacity, recovery is often incomplete. Electrical stimulation (ES) may enhance axonal regeneration, reinnervation and functional recovery. This study aimed to summarize current evidence on the clinical use of ES in these conditions and to assess its role in everyday practice.

Material and methods. A narrative review was performed using PubMed and Google Scholar databases. The search included 22 studies published between 2009 and 2026. Eligible sources included books, original and retrospective studies, reviews, systematic reviews and randomized controlled trials. Case reports, conference abstracts without full text, non-English publications, duplicates, and studies not directly related to the topic were excluded.

Results. ES was associated with improved outcomes in several clinical settings. Postoperative ES after carpal tunnel and cubital tunnel decompression improved electrophysiological and

functional outcomes. In carpal tunnel syndrome, interferential current therapy provided greater pain relief and better nerve function recovery than splinting or TENS. ES after digital nerve repair improved sensory recovery, while intraoperative ES after neck dissection was associated with better shoulder function and electrophysiological results. However, the evidence is limited by small sample sizes and restricted applicability of some outcome measures in everyday practice.

Conclusions. ES appears to be a promising adjunctive treatment in peripheral nerve injuries and entrapment neuropathies, but further studies are needed to standardize protocols and confirm its effectiveness.

Key words: “peripheral nerve injury”; “entrapment neuropathies”; “electrical stimulation”; “neuromuscular electrical stimulation”; “functional recovery”; “nerve regeneration”

1. Introduction

The peripheral nervous system (PNS) is a complex network of nerves that functionally connects various parts of the body to the central nervous system (CNS). (1)

Unlike the CNS, the PNS has a greater capacity for regeneration. (1) Nevertheless, peripheral nerve injuries (PNIs) often lead to long-term impairment in quality of life. Their clinical presentation varies widely, and delayed or unsuccessful treatment may result in chronic pain, sensory disturbance, and motor deficits. (2)

PNIs are a relatively common consequence of trauma. According to Torge Huckhagel et al., additional nerve injuries occur in approximately 1.8% of patients with lower limb trauma and 3.3% of those with upper limb trauma. (3) (4)

Following traumatic nerve transection, Wallerian degeneration develops in the distal nerve stump. In the absence of neuronal contact, supportive Schwann cells gradually lose their ability to promote axonal regeneration. As a result, although peripheral nerves have some regenerative potential, spontaneous recovery is often incomplete and treatment is required in most cases. (5)

Entrapment neuropathies are another important category of peripheral nerve disorders and occur as a result of chronic nerve compression or mechanical injury. The most common examples include carpal tunnel syndrome and ulnar neuropathies at the cubital tunnel. Although their pathophysiology differs from that of acute traumatic nerve injuries, they may also lead to chronic pain, sensory dysfunction and motor deficits. Early recognition and appropriate treatment are essential to prevent progression and minimize the risk of permanent nerve damage. Management may involve avoiding repetitive movements, splinting, injection therapy or surgical decompression. (6)

Functional recovery is particularly challenging when long nerve segments are damaged and reinnervation must occur over greater distances. Therefore, optimizing clinical management is essential to improve outcomes and prevent insufficient or failed regeneration, which may result in disability and neuropathic pain. In this context, electrical stimulation (ES) appears to be a promising therapeutic approach, as it may enhance reinnervation and improve functional recovery. (7) (5)

The aim of this narrative review is to evaluate the current literature on the clinical application of electrical stimulation in peripheral nerve injuries and entrapment neuropathies, summarize the available evidence, and discuss its potential role in everyday clinical practice.

2. Materials and methods of research

A narrative review was performed using the PubMed and Google Scholar databases. The search covered studies published between 2009 and 2026. The following keywords were used: “*traumatic peripheral nerve injuries*”, “*peripheral nerve injury*”, “*peripheral nerve trauma*”, “*entrapment neuropathies*”, “*electrical stimulation*”, “*neuromuscular electrical stimulation*”, “*brief electrical stimulation*”, “*rehabilitation*”, “*functional recovery*” and “*nerve regeneration*”. Inclusion criteria comprised books, retrospective studies, reviews, systematic reviews, original studies, and randomized controlled trials. Exclusion criteria included case reports, conference abstracts without full text, non-English publications, duplicate records, and studies not directly related to peripheral nerve regeneration or electrical stimulation. The final selection was based on relevance to the aim of the review. A total of 114 records were identified, of which 22 were included in the final analysis.

3. Research results

3.1. Clinical background of traumatic peripheral nerve injuries

The nervous system is classically divided into the central nervous system (CNS), which comprises the brain and spinal cord, and the peripheral nervous system (PNS), which connects the CNS with peripheral tissues and organs. The peripheral nervous system contains different types of nerve fibers with distinct functions: sensory fibers transmit information from peripheral tissues to the CNS, whereas motor fibers convey signals from the CNS to target muscles and other effector organs. (8) Traumatic peripheral nerve injuries (PNIs) most frequently occur with motor vehicle accidents, penetrating trauma following stab wounds, gunshot wounds, and stretch or crush injuries associated with falls or fractures. Depending on the mechanism, PNIs may be sharp or blunt, and may involve nerve transection, laceration, contusion, stretching or partial disruption. Such injuries may result in neuroma formation or lesions in continuity, and restrict functional recovery. (9)

The level and severity of nerve injury are major determinants of prognosis and treatment strategy. In 1943, Seddon classified nerve injuries into three categories: neurapraxia, axonotmesis, and neurotmesis. This system was expanded by Sunderland in 1951 and further developed into a five-grade classification. (10) In the context of mononeuropathies, including entrapment neuropathies, the simplified Seddon classification remains particularly useful. (6) (Table 1).

Table 1. Seddon and Sunderland classifications of peripheral nerve injuries, including typical mechanisms and characteristics of injury.

| Seddon | Sunderland | Typical mechanism | Characteristics of injury |
|-------------|------------|-------------------------------------|---|
| Neuropraxia | Grade I | Focal demyelination and/or ischemia | Conduction of nerve impulses is blocked in the injured area, resulting in motor and sensory deficits. |

| | | | |
|-------------|-----------|-------------------------|--|
| | | | The axon and all connective tissue layers, including the endoneurium, perineurium, and epineurium, remain intact. |
| Axonotmesis | Grade II | Crush or stretch injury | The epineurium is intact, while the perineurium and endoneurium may be disrupted. Wallerian degeneration occurs in the axon. |
| | Grade III | | |
| | Grade IV | | |
| Neurotmesis | Grade V | Complete transection | The axon, myelin sheath, all connective tissue layers (endoneurium, perineurium and epineurium) are completely severed. |

Source: Adapted from the literature on peripheral nerve injury classification. (11)

Notes: Seddon classification distinguishes three major types of peripheral nerve injury: neuropraxia, axonotmesis, and neurotmesis. In the more detailed Sunderland classification, these correspond to Grade I, Grades II–IV, and Grade V, respectively.

Peripheral nerve injury initiates a cascade of physiological and cellular changes. Following injury, the proximal axonal segment retracts to the region of the nearest Ranvier's node, while the distal segment undergoes axonal degeneration and Wallerian degeneration. Within 24–48 hours, the axonal cytoskeleton and cell membrane begin to disintegrate into small debris, while Schwann cells undergo demyelination, dedifferentiation and proliferation. Afterward Schwann cells upregulate regeneration-associated genes and secrete extracellular matrix molecules, trophic factors, cytokines, and chemokines that promote axonal regrowth and recruit immune

cells. Macrophages, neutrophils, and other leukocytes clear myelin debris by phagocytosis and further contribute to Wallerian degeneration. (12)

Despite the regenerative capacity of the PNS, recovery is often incomplete, particularly in severe injuries. For this reason, various therapeutic strategies have been developed to enhance peripheral nerve regeneration, including surgical interventions, physical rehabilitation, pharmacological therapies, nanotechnology-based approaches, neurotrophic factors, stem cell therapies, and extracellular vesicles. (1)

3.2. Mechanisms of action of electrical stimulation

The first studies investigating electrical stimulation (ES) as a potential treatment for peripheral nerve injury (PNI) began in the mid-twentieth century. In 1952, Hoffman demonstrated that stimulation at different frequencies could accelerate axon sprouting, laying the foundation for later studies on the regenerative effects of ES. Several mechanisms have been proposed to explain the beneficial effects of ES on peripheral nerve regeneration. Following peripheral nerve injury, the production of nerve growth factor (NGF) by Schwann cells (SCs) is reduced, which limits neural repair. ES appears to counteract this effect by activating molecular pathways involved in axonal regeneration. (7)

One proposed mechanism involves ES-induced glutamate secretion by SCs, which may increase the release of Schwann cell-derived exosomes and elevate intracellular calcium (Ca^{2+}) levels. (13) Intracellular Ca^{2+} waves go from the injured axon to the neuronal cell body, where they promote upregulation of brain-derived neurotrophic factor (BDNF) and its receptor tropomyosin receptor kinase B (TrkB). This leads to elevation of cyclic adenosine monophosphate (cAMP) levels and increased expression of regeneration-associated genes, including $\text{T}\alpha 1$ tubulin and growth-associated protein-43 (GAP-43). ES may also increase polysialic acid concentration, which could improve the precision of axon regrowth by enhancing collateral sprouting and influencing arborization fields. (14)

ES has also been shown to affect intracellular signaling pathways associated with neurite outgrowth. In particular, activation of p38 mitogen-activated protein kinase (MAPK) may promote the activation of cAMP response element-binding protein (CREB), thereby supporting axonal growth and regeneration. (15)

Another important mechanism may involve the interaction between regenerating axons and Schwann cells through exosome-mediated signaling. ES may enhance this process by promoting the transport of Schwann cell-derived exosomes containing mRNA, miRNA, and proteins to injured axons, thereby providing transcripts for local translation. In addition, ES may reduce the activity of the RhoA pathway, which normally inhibits axonal extension, thus promoting neurite outgrowth. (14)

Finally, ES may also modulate the post-injury immune response. ES can induce a shift in macrophage phenotype toward a predominantly pro-repair state, which may accelerate myelin debris clearance and promote remyelination. (16)

Through these combined neurotrophic, intracellular, axon–Schwann cell interactions, and immunomodulatory effects, ES may create a more favorable condition for nerve regeneration.

4. Clinical applications of electrical stimulation

4.1. Carpal tunnel syndrome - Postoperative electrical stimulation after decompression

The electrical stimulation (ES) following carpal tunnel decompression was first evaluated in a pilot study published in 2010. All included patients presented with thenar muscle atrophy and motor unit number estimation (MUNE) findings indicating at least 50% axonal loss. The control group included 10 patients, whereas the stimulation group consisted of 11 patients who received one hour of postoperative ES (20 Hz, 4–6 V, 0.1–0.8 ms).

The outcomes were more favorable in the ES group, with significant electrophysiological improvement observed at 6–8 weeks after surgery, compared with 12 months in the control group. Moreover, by 12 months, all motoneurons in the ES group had established functional connections with denervated muscle fibers. In contrast, surgery alone was insufficient to

produce significant improvement in muscle reinnervation, even one year after the procedure. (17) (18)

4.2. Carpal tunnel syndrome - Non-invasive electrotherapeutic approaches in entrapment neuropathies

In a 2013 prospective, single-blind, single-center, randomized, three-arm parallel-group study, patients with idiopathic carpal tunnel syndrome were assigned to one of three treatment groups: splint therapy, transcutaneous electrical nerve stimulation (TENS), or interferential current (IFC) therapy. The splint group included 22 patients who wore a resting wrist-hand splint at night for 3 weeks. The TENS group included 20 patients who received 15 sessions of TENS (100 Hz, 80 ms; 20 min, 5 sessions/week). The IFC group included 21 patients who underwent 15 sessions of interferential current therapy (base frequency 4,000 Hz, modulation frequency 20 Hz, ΔF 10 Hz, slope 1/1, quadripolar mode; 20 min, 5 sessions/week).

After 3 weeks, no significant difference in clinical improvement was observed between the TENS and splint groups. In contrast, IFC therapy resulted in significantly greater pain relief and better recovery of nerve function than splint therapy. Moreover, IFC therapy produced significantly greater improvements in pain relief, symptom severity, functional capacity, and electrophysiological recovery than TENS therapy. (19)

4.3. Digital nerve lacerations - Electrical stimulation as an adjunct to surgical treatment

A double-blind randomized clinical trial published in 2015 investigated the effects of postoperative ES in patients with complete digital nerve lacerations. Following surgical repair, 18 patients received one hour of continuous ES (20 Hz, <30 V, 0.1–0.4 ms), while 18 patients received sham stimulation.

Patients in the ES group recovered near-normal sensation within 5 to 6 months, whereas most patients in the control group did not achieve comparable recovery. Sensory outcomes were significantly better in the stimulated group. Although a trend toward improved functional

recovery was also observed, the difference did not reach statistical significance. This finding should be interpreted in the context of the sensory nature of the digital nerve, which makes objective assessment of functional outcomes more difficult. (20)

4.4. Spinal accessory nerve after neck dissection - Electrical stimulation as an adjunct to surgical treatment

A double-blinded randomized clinical trial published in 2018 evaluated the effect of ES on the spinal accessory nerve (SAN) in 54 patients undergoing neck dissection for head and neck cancer. In the treatment group, one hour of intraoperative ES (20 Hz, 3–5 V, 100 μ s) was applied to the SAN after completion of the procedure, whereas patients in the control group received no stimulation.

At 12 months, the ES group showed significantly better preservation of shoulder function and greater improvement in electrophysiological outcomes than the control group. These findings suggest that intraoperative ES may help reduce postoperative functional deficits associated with SAN injury during neck dissection. (21)

4.5. Cubital tunnel syndrome - Electrical stimulation as an adjunct to surgical treatment

Postoperative ES has also been investigated as an adjunct to surgery in compressive neuropathies. In a randomized, double-blind, placebo-controlled trial published in 2020, 31 patients with severe cubital tunnel syndrome underwent surgical decompression. Of these, 11 received surgery alone, whereas 20 received surgery followed by one hour of postoperative ES (20 Hz, <30 V, 0.1 ms). Patients in the control group received sham stimulation.

Three years after surgery, motor unit number estimation (MUNE) was significantly higher in the ES group than in the control group. The mean improvement in key pinch strength was nearly three times greater in stimulated patients than in controls. Other functional outcomes, such as grip strength, as well as physiological measures, including compound muscle action potential (CMAP), showed significantly greater improvement in the ES group. These findings indicate

that postoperative ES may enhance muscle reinnervation and functional recovery after surgery for severe cubital tunnel syndrome. It may serve as a clinically useful adjunct in patients for whom recovery after conventional treatment is often suboptimal. (22)

5. Discussion

The findings of this review suggest that electrical stimulation (ES) is a promising adjunctive strategy in the management of peripheral nerve injuries and entrapment neuropathies. Its main advantages include low invasiveness and the possibility of integration with both surgical treatment and rehabilitation programs.

The available studies indicate that ES has already been applied in different clinical settings. In entrapment neuropathies, it has been used as a postoperative intervention, particularly after carpal tunnel and cubital tunnel decompression. (17) (22)

In carpal tunnel syndrome, ES has also been investigated as a non-invasive rehabilitation approach. (19)

In oncological surgery, intraoperative ES has been evaluated following neck dissection involving the spinal accessory nerve. (21)

In traumatic nerve injuries, ES has been studied after surgical repair of digital nerve lacerations. (20)

More favorable outcomes were observed in patients treated with ES than in control groups without stimulation. The stimulated groups showed significant improvements in electrophysiological parameters, including motor unit number estimation (MUNE) and compound muscle action potential (CMAP), as well as better preservation of muscle function and strength. ES was also associated with improved functional reinnervation, including the establishment of functional connections with denervated muscle fibers. In addition, patients receiving ES demonstrated better sensory recovery, pain relief, reduced symptom severity, and improved functional capacity.

An important limitation of the available research is the small sample size in most studies, which reduces the strength of the evidence and limits the generalizability of the findings. Larger studies would allow for a more reliable assessment of treatment effects and improve the ability to draw broader clinical conclusions. In addition, many of the outcome measures used in research, such as motor unit number estimation (MUNE), are currently available mainly in laboratory settings, which limits their applicability in routine clinical practice. Cost-effectiveness concerns and technical limitations further hinder the broader adoption of ES.

Therefore, although ES appears to be a promising therapeutic adjunct, further high-quality research is needed before it can be routinely recommended in standardized clinical practice.

Future research should focus on standardizing ES protocols and determining in which clinical settings this therapy is most effective. It would also be valuable to evaluate ES in combination with surgery, rehabilitation, pharmacological treatment, or regenerative therapies. Particular attention should be given to long-term clinical outcomes, such as motor and sensory recovery, pain reduction, quality of life, and functional improvement.

6. Conclusions

Electrical stimulation (ES) is used across several clinical settings involving peripheral nerve injuries and entrapment neuropathies. In carpal tunnel syndrome, postoperative ES after decompression was associated with significant electrophysiological improvement, and after 12 months all motoneurons in the stimulated group had established functional connections with denervated muscle fibers. (17) ES has also been evaluated as a non-invasive therapeutic approach in carpal tunnel syndrome, where interferential current therapy was associated with greater pain relief, better recovery of nerve function, and superior improvements in symptom severity, functional capacity, and electrophysiological outcomes. (19) In cubital tunnel syndrome, postoperative ES resulted in significantly higher motor unit number estimation (MUNE), greater improvement in key pinch strength, and better functional and physiological outcomes, including grip strength and compound muscle action potential (CMAP). (22) In oncological surgery, intraoperative ES after neck dissection involving the spinal accessory

nerve was associated with better preservation of shoulder function and improved electrophysiological outcomes. (21) In traumatic nerve injuries, ES applied after surgical repair of digital nerve lacerations was associated with recovery of near-normal sensation within 5 to 6 months. (20) Taken together, these findings suggest that ES may be a valuable adjunct to surgical and rehabilitative treatment.

Disclosure

The authors declare no relevant disclosures.

Supplementary Materials

Not applicable.

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Conflicts of Interest

The authors declare no conflict of interest.

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