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REVIEWS

Integrated Therapeutic Strategies in the Rehabilitation of Upper-Limb Traumatic Peripheral Nerve Injuries

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Abstract

Traumatic peripheral nerve injuries of the upper limb remain a significant cause of long-term disability, chronic pain, and impaired quality of life, particularly among young and active individuals. While timely surgical repair restores structural continuity, functional recovery ultimately depends on early, sustained and neurophysiology-driven rehabilitation. Rehabilitation not only prevents secondary complications but also shapes the central and peripheral microenvironment required for reinnervation. Early postoperative care focuses on edema control, pain modulation, protective splinting and maintenance of passive mobility, establishing the biomechanical and neurobiological conditions for axonal regeneration. As reinnervation emerges, therapy transitions to progressive sensorimotor re-education, including graded sensory retraining, mirror-based visual feedback, bilateral tasks and neuromuscular facilitation to reverse cortical disorganization and reactivate functional motor units. Advanced rehabilitation incorporates task-specific practice, proprioceptive enrichment, neurocognitive methods and technology-assisted interventions such as neuromuscular electrical stimulation, virtual reality and biofeedback, all of which exploit activity-dependent plasticity to refine fine motor control. Effective management requires close collaboration among surgeons, physiotherapists and occupational therapists to align surgical strategies with biological regeneration timelines and individualized therapy goals. This review synthesizes current evidence from neurobiology, surgical reconstruction and rehabilitation science, emphasizing the integration of sensory re-education and neuroplasticity-based techniques to enhance long-term hand function following upper-limb peripheral nerve injuries.

Keywords: peripheral nerve injuries, trauma, upper limb, microsurgical repair, rehabilitation, neurocognitive methods.

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INTRODUCTION

Traumatic peripheral nerve injuries represent a major cause of chronic pain, functional disability and persistent decline in quality of life. They frequently affect young, active individuals and determine a substantial burden on healthcare systems[1,2].

Although clinically important, their true frequency after extremity trauma is still not well established. These lesions often produce severe and sometimes irreversible impairments in both motor and sensory function. In large trauma cohorts, peripheral nerve injuries (PNIs) are identified in roughly 2–3% of extremity trauma cases. They are typically associated with greater overall injury severity and poorer functional recovery, highlighting their substantial clinical and socioeconomic burden[3].

Within the upper limb, over half of nerve injuries involve the fingers or hand, with digital nerves being the most frequently affected (≈63%), followed by wrist or forearm lesions[4]. Sharp lacerations caused by knives or glass are common in distal injuries, whereas traction or avulsion mechanisms predominate in proximal patterns, particularly in brachial plexus injuries. Ballistic and crush mechanisms also contribute significantly to the peripheral nerve trauma[5,6]. Iatrogenic mechanism occurs for roughly one in six surgically treated nerve lesions and may arise across different surgical specialties, often leading to poorer recovery trajectories and carrying medicolegal implications[7].

Peripheral nerve injuries of the upper limb produce complex and interconnected motor, sensory and autonomic deficits that significantly impair daily function and quality of life. Motor alterations include weakness or paralysis of target muscles, resulting in loss of dexterity, grip strength and coordinated movement patterns, often accompanied by progressive atrophy and joint stiffness[5,8]. Sensory impairment manifests as hypoesthesia, paresthesia or anesthesia in the affected dermatomes, which leads to reduced tactile discrimination and difficulty in performing fine motor tasks[3]. Autonomic dysfunction may involve vasomotor and sudomotor disturbances, trophic skin changes and impaired thermoregulation, reflecting broader sympathetic involvement[4].

Regeneration in the peripheral nervous system occurs slowly, at a rate of approximately 1–3 mm per day. Consequently, long regeneration distances, such as those between proximal upper-limb lesions and distal target muscles, often lead to delayed reinnervation and poorer outcomes[9-11]. With prolonged denervation, the distal nerve stump, including Schwann cells, sensory receptors and muscle fibers, undergoes degenerative changes, reducing its capacity to support functional recovery. After peripheral nerve injury, cortical maps and sensorimotor representations reorganize rapidly. Without prompt and structured rehabilitation, the brain–nerve–muscle system may develop maladaptive patterns that preclude optimal recovery[12].

Early microsurgical repair and structured rehabilitation are therefore essential. Delayed interventions greatly decrease the likelihood of adequate recovery due to muscle fibrosis, Schwann cell degeneration, and cortical miswiring. Even when axons successfully reach their target organs, reinnervation may remain incomplete or misdirected, limiting the restoration of fine motor control and tactile discrimination.[13-15] Rehabilitation serves as a vital bridge between biological reinnervation and functional reintegration, ensuring that the potential for nerve recovery translates into good hand function[9,13].

During the early postoperative phase, rehabilitation focuses on edema control, pain modulation, wound protection and the maintenance of joint mobility to create optimal biomechanical conditions for nerve regeneration. As regeneration progresses, therapy shifts its focus to motor retraining and sensory re-education, both essential for re-establishing effective cortical representations and coordinated movement patterns[12]. Techniques such as graded sensory re-education, mirror therapy and task-oriented training have demonstrated potential in promoting cortical plasticity and improving functional outcomes[16].

The success of the recovery depends on a multidisciplinary approach integrating precise microsurgical intervention with individualized rehabilitation strategies. Close collaboration between surgeons, physiotherapists and occupational therapists ensures alignment between protective protocols and active functional goals. Rehabilitation transforms the biological potential for regeneration into meaningful, task-specific hand function through sustained, adaptive neuroplastic mechanisms[5].

This paper aims to provide an evidence-based overview of surgery and rehabilitation strategies following peripheral nerve injuries of the upper limb, emphasizing the continuum from early postoperative management to functional reintegration. Particular attention is given to sensory re-education methods that support adaptive neuroplasticity and optimize functional outcomes. Furthermore, the review highlights the importance of interdisciplinary collaboration among surgeons and physiotherapists in designing individualized rehabilitation protocols aligned with surgical repair and biological regeneration timelines. By integrating current evidence from neurobiological, surgical and therapeutic perspectives, this paper aims to provide practical and theoretical principles for improving long-term functional recovery after upper-limb peripheral nerve injuries.

PATHOPHYSIOLOGY AND MECHANISMS OF NERVE HEALING

Following peripheral nerve injury, the distal segment of the axon undergoes Wallerian degeneration, a highly regulated neurobiological process that enables subsequent axonal regeneration. This process involves the disintegration and phagocytic clearance of the distal axon and its associated myelin sheath, while the proximal axonal segment retains its regenerative capacity. Initially described by Augustus Waller in the 19th century, this degeneration is crucial for enabling nerve repair and regeneration. It begins with axonal fragmentation and myelin disintegration, followed by the activation of Schwann cells and infiltration of macrophages to clear debris. Schwann cells play a dual role by secreting neurotrophic factors and forming bands of Büngner, which guide regenerating axons toward their targets. An inefficient or delayed Wallerian degeneration can impair regeneration and functional recovery[9,11,17-20].

Axonal sprouting originates from the proximal stump, with new growth cones extending at an average rate of approximately 1 mm per day, depending on the injury level and patient factors[13,21]. Successful reinnervation depends on the maintenance of Schwann cell phenotype and the viability of the distal target. Prolonged denervation leads to reduced trophic support and impaired functional recovery[9,11]. Molecular mediators such as neurotrophic factors (NGF, BDNF, GDNF) and signaling pathways involving c-Jun, ERK and PI3K/Akt play pivotal roles in regulating axonal regeneration and remyelination[11].

In addition, the degree of functional restoration depends on the precision of axonal reinnervation and the central nervous system's ability to reorganize cortical representations in response to new peripheral input[12].

CLASSIFICATION OF PERIPHERAL NERVE INJURIES

Peripheral nerve injuries are conventionally described using two complementary classification systems: the Seddon classification and the more detailed Sunderland grading scheme. These classification systems provide a structured framework for understanding the extent of nerve damage, predicting recovery potential and guiding the selection of adequate therapeutic strategies[22-25].

Seddon's system divides nerve injuries into three major categories based on the degree of structural disruption[23-26]:

- Neurapraxia represents the mildest form, characterized by a transient conduction block resulting from focal myelin injury without axonal disruption. Because the underlying axons and connective tissue structures are preserved, recovery is typically complete once remyelination occurs.
- Axonotmesis reflects a more significant injury in which the axon is disrupted and Wallerian degeneration occurs distal to the lesion, but the endoneurial and perineurial connective tissues remain intact. This preserved architecture allows regenerating axons to follow their initial pathways, making functional recovery possible if the traumatic mechanism is removed.
- Neurotmesis represents the most severe lesion, involving complete disruption of the axon and all supporting connective tissue layers; in these cases, spontaneous recovery does not occur and surgical repair is required to restore continuity.

Sunderland expanded Seddon's classification model into a more detailed system to provide more anatomical specificity[23-28]:

- Grade I corresponds to neurapraxia and involves a purely functional conduction block without structural damage;
- Grade II injuries include axonal continuity loss with intact endoneurium, allowing recovery guided by the intact endoneurium;
- Grade III injuries disrupt both the axon and endoneurium, while the perineurium remains intact; this loss of internal organization poses the risk of axonal misdirection and incomplete functional recovery;
- Grade IV lesions extend the damage to the

perineurium, leaving only the epineurium intact and typically resulting in intraneural scarring that precludes axonal regeneration. Such injuries are unlikely to recover without surgical intervention.
- Grade V represents complete transection of the nerve, comparable to neurotmesis. All the nerve components are lost and microsurgical repair is required;
- A later addition by Mackinnon & Dellon, Grade VI, describes mixed injuries (spectrum of severities within the same nerve injury) in which different fascicles within the same nerve exhibit varying degrees of severity, resulting in highly variable clinical outcomes.

Together, the Seddon and Sunderland classifications form the basis of contemporary clinical decision-making in peripheral nerve trauma. By correlating structural damage with regenerative potential, these models assist clinicians in the assessment of prognosis, operative planning and informed patient counseling.

SURGICAL TREATMENT OF PERIPHERAL NERVE INJURIES

When the components of a peripheral nerve are completely disrupted or the nerve is transected, the only therapeutic option is surgical treatment. The choice of repair strategy depends on the severity of the injury, the size of the nerve gap and the available resources[25,28].

For clean transections with minimal nerve gaps, direct neurorrhaphy is the preferred approach. This involves tension-free microsurgical suturing of the nerve ends using epineural or perineural sutures under magnification, preferably using the operative microscope. When performed promptly after the injury, direct repair offers the highest potential for recovery by preserving the anatomical continuity of the nerve[29-31].

When a nerve cannot be repaired by direct neurorrhaphy, there are some options that a surgeon must consider, each with its indications and advantages: autologous nerve grafting, vascularized nerve grafting, nerve conduits, allografts and nerve transfers[22,29,32-35].

In cases where a nerve gap prevents direct repair, nerve grafting is the standard solution. Autografts, harvested from the patient's own body (like the sural nerve), are considered the gold standard due to their excellent biocompatibility and structural support for axonal regeneration. However, autografts have limitations, including donor site morbidity, limited availability, and the potential for mismatched nerve diameters. Allografts, derived from deceased donors, provide an alternative that eliminates donor site morbidity and offers better size matching. However, they require immunosuppressive therapy to prevent rejection, which can lead to significant side effects. Both approaches are effective for bridging gaps, with autografts being the preferred option when feasible[29,31,36-38].

Nerve conduits have emerged as a promising alternative to nerve grafts. These are used for nerve gaps usually smaller than 3 cm, so they can provide a scaffold through which the axons can regenerate while preventing protrusion of endoneural contents during healing. These tubular structures can be made from synthetic or biological materials. The ideal nerve conduit is biocompatible, biodegradable, and capable of creating a microenvironment conducive to nerve regrowth[29, 31,39-41].

For large nerve gaps or in cases where the vascular supply is compromised, vascularized nerve grafts are employed. These grafts, which include their own blood supply, enhance the survival and regenerative potential of the transplanted nerve tissue. However, this technique is more technically demanding[29,42]. In situations where neither direct repair nor grafting is feasible, nerve transfers are considered. This approach involves re-routing intact regional nerves to restore function to denervated areas. While effective, nerve transfers often require sacrificing the donor nerve's original function[29,31,43].

Adjunctive therapies, including neurotrophic factors, pharmacological agents, electrical stimulation, and targeted rehabilitation, play a vital role in promoting axonal regeneration and functional recovery. Experimental strategies such as gene therapy, stem cell applications, and tissue engineering are also gaining traction in the field of nerve repair. Emerging technologies like 3D printing and biodegradable scaffolds are being explored for their potential to create personalized solutions for complex nerve injuries[29,31,43-45].

REHABILITATION AFTER PERIPHERAL NERVE INJURY

Core goals and therapeutic aims of rehabilitation

Rehabilitation also serves as a first-line intervention, working in conjunction with surgical repair and guiding neural recovery through staged and task-specific interventions[25,46]. Early initiation of therapy prevents disuse complications, reduces maladaptive cortical reorganization and establishes a favorable biological and behavioral environment for regeneration. These rehabilitation principles are of utmost importance when referring to peripheral nerve injuries affecting the upper limb, a situation in which the functional implications are highly significant[16,47].

Objectives of rehabilitation include[16,46,48-51]:

- Protection of the repaired or grafted nerve and prevention of secondary complications.
- Preservation of joint and soft-tissue mobility.
- Progressive reactivation of sensory and motor pathways.
- Restoration of coordinated purposeful movement.
- Long-term reintegration into functional, vocational and social roles.

Basic principles of the rehabilitation process

According to Robinson and Shannon, rehabilitation after PNI should be *multidisciplinary, early and goal-oriented,* integrating pain management, protective positioning and progressive activation. The key components include range of motion and strengthening, sensory re-education, splinting and positioning and functional and behavioral training[46].

Maintaining the mobility of joints, tendons and soft tissues prevents adhesions and contractures. Early passive range-of-motion (ROM) exercises (Fig.1) progress to active and resisted movements as reinnervation occurs. Strengthening must be cautious, emphasizing low load and high control to avoid traction on healing nerves[52,53].

Fig 1. Continuous passive mobilization

Sensory recovery often lags behind motor regeneration, making early desensitization followed by progressively graded sensory discrimination training essential[16,54]. Patients are gradually exposed to varying textures, shapes and temperatures to retrain cortical recognition and reduce dysesthesia. Mirror therapy and bilateral integration tasks activate the mirror neuron system, promoting cortical remapping and sensorimotor congruence[12,55]. *Custom splints* are employed to protect nerve repairs, maintain functional alignment and prevent overstretching. Later, *functional splints* (Fig.2) support specific grasp and prehension patterns during daily activities[52].

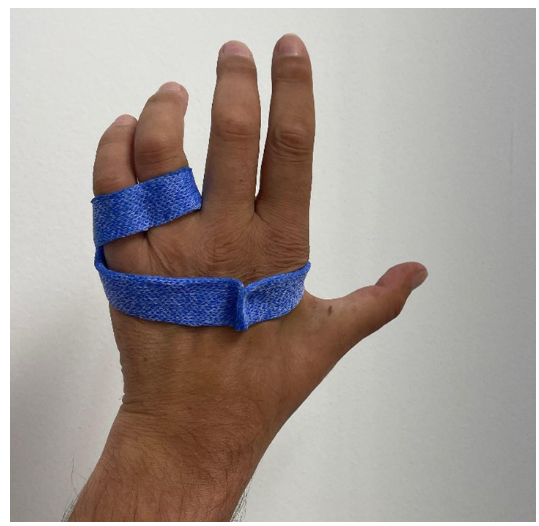

Fig 2. Anti-clow splint

Task-specific activities, such as grasp and release, grip modulation and bimanual coordination, are introduced early to reinforce cortical motor learning. Psychological and motivational aspects are emphasized equally, given their role in adherence and neuroplasticity[9,46].

Phases of rehabilitation after peripheral nerve injury

Rehabilitation after peripheral nerve injury is organized as a dynamic, overlapping continuum aligned with the biological timeline of axonal regeneration and soft tissue healing. Rather than rigid, isolated stages, these phases guide clinical decision-making and individualize progression based on surgical procedure, reinnervation status, pain, edema and functional demands.

5.3.1 Early Protective Phase (0–3 weeks)

The early postoperative phase begins immediately after surgery and extends through the first few postoperative weeks, which is a critical interval for ensuring the success of nerve repair and establishing the foundation for later functional recovery. The primary objectives during this period are protection of the surgical site, control of pain and edema, maintenance of passive joint mobility and prevention of muscle atrophy.

Immobilization is achieved using protective orthoses or splints, typically maintaining the wrist in slight extension and the metacarpophalangeal (MCP) joints in flexion, while allowing the interphalangeal (IP) joints to remain extended to minimize tension on repaired structures[16].

Edema control is addressed through limb elevation, gentle compressive dressings and manual lymphatic drainage techniques, which reduce interstitial pressure and promote venous return[5].

Pain management involves multimodal strategies such as pharmacologic therapy, cryotherapy and sensory desensitization, aiming to minimize nociceptive input that could otherwise reinforce cortical maladaptation[12]. Simultaneously, early passive mobilization (Fig. 3) of uninvolved joints and adjacent segments is essential to prevent stiffness, adhesion formation and disuse atrophy[13].

Fig 3. Manual passive mobilization

Comprehensive patient education regarding limb positioning, orthosis use and safe self-care practice ensures adherence to postoperative protocols and reduces the risk of compromising the repair. Psychological support and therapist-guided feedback are equally important for maintaining motivation, preventing learned non-use and encouraging early cortical engagement through mental imagery and observation-based activation of motor circuits[9,11].

Evidence suggests that early-initiated rehabilitation, started within the first days to weeks after hand trauma, can safely enhance functional recovery without compromising tissue integrity, provided that protocols are carefully adapted to the type and stability of the repair[46,48,56].

Controlled mobilization under protective protocols improves pain, edema and range of motion, supporting the concept that carefully timed intervention aligns with the biological pace of regeneration.

Together, these measures create an optimal biological and behavioral environment required for advancing into the next phases of sensorimotor re-education and functional reintegration.

5.3.2 Intermediate phase - Early active sensorimotor reeducation phase (3–8 weeks)

As axonal regeneration progresses and the first signs of reinnervation emerge, the intermediate phase (approximately 3–8 weeks postoperatively) marks the transition from protective immobilization to controlled activation of sensorimotor pathways. The therapeutic focus during this period shifts toward progressive mobilization, sensory re-education and early facilitation of neuromuscular activity, while maintaining protection of the healing nerve and surrounding soft tissues.

Gentle passive and active-assisted mobilization exercises are initiated to restore joint range of motion, prevent periarticular stiffness and stimulate proprioceptive feedback within safe mechanical limits[16]. Controlled movement not only preserves tendon gliding and soft tissue elasticity but also enhances intraneural blood flow and axonal nutrition, supporting the biological environment for regeneration[9].

Progressive sensory re-education becomes a key component of therapy once early reinnervation is expected. Programs follow a graded progression, beginning with texture discrimination and localization training and advancing toward object recognition and complex tactile tasks to restore cortical mapping and tactile awareness[12]. Early sensory re-education frequently incorporates visual-tactile integration, mirror therapy (Fig. 4) and bilateral tasks that engage both hemispheres and reinforce sensory–motor congruence through activation of mirror neuron networks[11]. These strategies help reduce hypersensitivity, enhance perceptual accuracy and promote adaptive cortical plasticity, all of which are important for functional recovery.

Fig 4. Mirror therapy exercises

Research on nerve transfer rehabilitation shows superior outcomes when early, repetitive and meaningful motor tasks are used to activate cortical networks. Such task-driven practice promotes adaptive neuroplasticity and consolidates newly reinnervated motor patterns into functional skills.[57]

In parallel, neuromuscular electrical stimulation (NMES) may be applied to maintain muscle trophicity, prevent denervation atrophy and facilitate voluntary contraction once motor unit reinnervation begins[13]. NMES (Fig. 5) provides valuable proprioceptive feedback to higher motor centers, reinforcing cortical reorganization and improving coordination between visual, sensory and motor systems.

Throughout this phase, patient tolerance, incision healing and nerve regeneration status should be closely monitored in order to ensure safety and optimize therapeutic progression. The therapist adapts exercise intensity and sensory input based on clinical feedback to prevent traction on the repair site while preparing the patient for the advanced phase of active strengthening and functional reintegration.

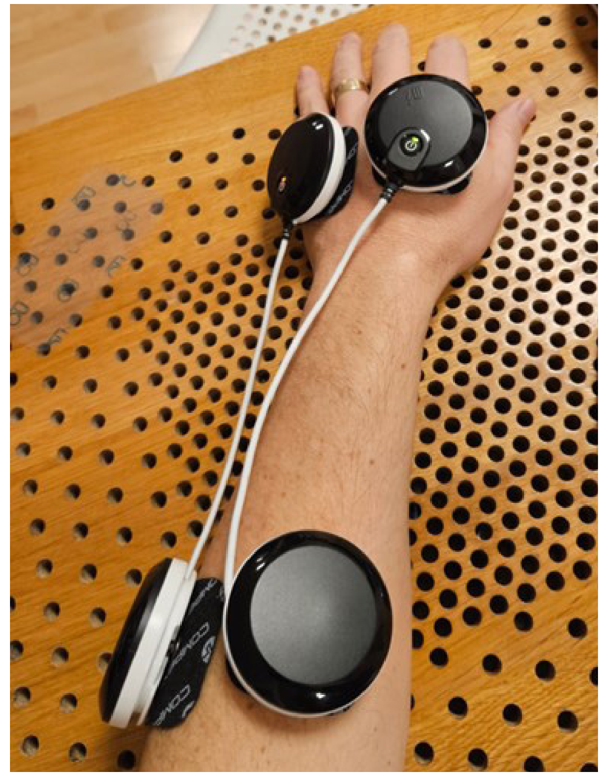

Fig 5. Neuromuscular electrostimulation

5.3.3 Advanced phase – Functional reintegration phase (approximately 2–6 months)

The advanced phase, beginning approximately eight weeks after surgery, represents the culmination of the rehabilitation process, during which the focus shifts from tissue protection and reinnervation to strength restoration, fine motor coordination and functional reintegration. At this stage, sufficient axonal regeneration and muscle reinnervation typically permit progressive loading and the reintroduction of complex, task-oriented activities.

Therapeutic goals emphasize graded strengthening and coordination exercises designed to enhance endurance, dexterity and control of reinnervated muscles[16]. Resistance training using putty, elastic bands or functional tools gradually increases the demand on precision grip, finger independence and coordinated wrist–hand movements. These are essential prerequisites for both occupational and daily living tasks[9].

As muscle strength and coordination return, functional re-education becomes the core of intervention. The focus is on task-oriented training that replicates real-life manual and professional activities such as grasping, pinching, manipulating small objects or handling work-specific instruments[13]. These repetitive, meaningful tasks reinforce the sensorimotor link between intention and execution, fostering cortical reorganization and enhancing the efficiency of motor control.

In parallel, neurocognitive and technology-assisted methods play a crucial role in optimizing central adaptation. Mirror therapy, constraint-induced movement therapy (CIMT) and virtual reality-based exercises activate mirror neuron networks and visual feedback mechanisms, promoting sensorimotor congruence and improving bilateral coordination[11,12].

All these interventions encourage active use of the affected limb, reduce compensatory non-use and strengthen cortical pathways associated with voluntary movement.

For patients with incomplete or delayed recovery, the introduction of adaptive strategies and assistive technologies, including orthoses, functional electrical stimulation or sensory substitution techniques, can significantly improve independence and quality of life[16]. Ultimately, this phase aims to achieve functional independence and skill refinement, integrating the rehabilitated limb into complex bimanual, vocational and leisure activities.

A multidisciplinary approach, uniting the expertise of surgeons, physiotherapists, occupational therapists and psychologists, remains essential to ensure that biological healing translates into meaningful, patient-centered function and long-term participation[13].

5.3.4 Chronic phase - Functional reconstruction and reintegration (>6 month)

The chronic phase of rehabilitation follows the period of biological nerve regeneration and functional reeducation, typically extending beyond 6–12 months post-injury. This phase focuses on optimizing residual function, compensating for irreversible deficits and supporting long-term adaptation and participation in personal and professional life. Despite significant advances in microsurgical techniques, some patients experience incomplete motor or sensory recovery, necessitating secondary reconstructive or compensatory interventions[13].

When reinnervation potential is limited, *tendon transfers* may be indicated to restore lost motor functions by rerouting active muscles to replace paralyzed ones, thereby improving grasp, pinch or wrist extension. The success of these procedures depends on careful donor selection, joint balance and subsequent intensive postoperative retraining to integrate new movement patterns into functional activities[5]. In parallel, *compensatory strategies* such as adaptive splinting, orthotic devices and assistive technology can further enhance independence and task performance, particularly for complex hand functions requiring precision and stability[16].

When complete functional restoration is not achievable, long-term management emphasizes compensation and participation.

O'Brien et al. demonstrated that structured hand therapy and workplace adaptation significantly improve return-to-work rates and psychosocial well-being, underscoring rehabilitation's societal impact.[58] Beyond physical recovery, the chronic phase also addresses *psychosocial and occupational reintegration*. Persistent sensory deficits, chronic pain or aesthetic concerns may affect self-image, mental health and social participation[12]. Psychological counseling, vocational retraining and workplace adaptation are therefore crucial components of long-term rehabilitation, aiming to restore confidence, autonomy and social engagement. A *multidisciplinary approach*, integrating surgical expertise with physiotherapy, occupational therapy, psychology and social support, ensures comprehensive recovery and sustained quality of life[9,11].

Therapeutic modalities and adjunctive interventions

Physical modalities serve as adjuncts to relieve pain, reduce inflammation and prepare tissues for active therapy.

Thermotherapy increases local blood flow, reduces muscle spasm and improves collagen elasticity. An optimal therapeutic heating range of 41–45 °C has long been recognized as effective for increasing soft-tissue extensibility and facilitating viscoelastic deformation, a concept grounded in classic experimental work by Lehmann et al. and consistently supported by contemporary thermotherapy research. Within this physiological window, tissue heating enhances collagen extensibility, improves perfusion and reduces muscle spindle sensitivity, thereby creating more favorable conditions for therapeutic exercise. This aspect was emphasized in neuromuscular rehabilitation frameworks such as those outlined by Krivickas. Methods include conductive (hot packs, paraffin baths), convective (whirlpool, fluidotherapy) and conversion modalities (ultrasound)[59-61].

Cryotherapy promotes vasoconstriction and analgesia, reducing edema and inflammatory activity. Effective skin cooling to 10–15 °C can relieve pain and improve mobility[62-64].

Therapeutic ultrasound remains a commonly used modality in peripheral nerve and soft-tissue rehabilitation due to its capacity to influence cellular activity and local physiological responses. Michlovitz highlights that ultrasound can enhance tissue healing by increasing cell membrane permeability, promoting ion exchange and stimulating local circulation, thereby creating a more favorable environment for nerve recovery[65]. Similarly, Scott et al. emphasize that ultrasound, used as part of a structured rehabilitation program for peripheral nerve injuries, may support symptom modulation, soft-tissue mobility and functional improvement when integrated appropriately within multimodal therapeutic strategies[66]. Together, these sources underline the rationale for incorporating ultrasound as an adjunct modality in evidence-based neuro-musculoskeletal rehabilitation.

Iontophoresis, using low-voltage direct current to facilitate the transdermal delivery of medications such as corticosteroids or lidocaine, represents a valuable modality in the management of inflammation and scar adhesions[52]. Recent literature highlights important technological and mechanistic advancements. Liatsopoulou et al. describe how iontophoresis enhances skin permeability and optimizes the transdermal transport of ionized agents, thereby strengthening its clinical applicability in rehabilitation settings[67]. These findings suggest that integrating iontophoresis within a structured physical therapy program may support the reduction of local inflammation, improve scar pliability and facilitate more efficient therapeutic mobility.

Transcutaneous electrical nerve stimulation (TENS) and neuromuscular electrical stimulation continue to be explored as adjuncts for pain modulation and muscle maintenance in partially denervated tissues[68]. Schimrigk et al. demonstrated early experimental evidence that electrical stimulation can attenuate muscle atrophy in denervated muscle, although without confirming a consistent influence on the reinnervation process[69]. More recent literature provides a refined understanding of these effects. Bersch et al. showed that electrical stimulation can induce favorable morphological and molecular adaptations in denervated intrinsic and extrinsic hand muscles, supporting the preservation of contractile properties during prolonged denervation[70]. Similarly, Ni et al. emphasize that while electrical stimulation can enhance local trophic support, modulate inflammatory responses and maintain muscle viability, evidence regarding its capacity to accelerate or improve true axonal reinnervation remains mixed[71]. Together, these reports prove that electrical stimulation may be valuable for symptom control and muscle preservation in the early and intermediate phases of recovery but should be applied within a comprehensive rehabilitation strategy rather than as a stand-alone intervention for promoting nerve regeneration.

Recent reviews emphasize that integrated, multimodal rehabilitation strategies that combine traditional therapeutic techniques with biologically active physical modalities offer the greatest potential for enhancing peripheral nerve regeneration and functional recovery. Chu et al. presented one of the most comprehensive syntheses to date, outlining how modern modalities such as low-intensity pulsed ultrasound (LIPUS), electrical stimulation (ES) and photobiomodulation (PBM) can be used synergistically alongside conventional exercise therapy. Experimental and preclinical evidence indicates that these modalities collectively promote accelerated axonal regeneration, improved microvascular perfusion and heightened cortical responsiveness, supporting more efficient sensorimotor reintegration. This recent literature marks a shift toward a multimodal rehabilitation paradigm, where physical therapy is not limited to restoring movement but actively modulates the cellular and neurophysiological processes underlying nerve repair[72].

Low-Intensity Pulsed Ultrasound (LIPUS) has received particular attention for its dual mechanical and thermal effects on nerve tissue. Preclinical and early clinical studies demonstrate that LIPUS can modulate inflammatory responses, promote Schwann cell proliferation and upregulate neurotrophic factors such as NGF through the ERK1/2-CREB-Trx-1 signaling pathway. Optimal parameters identified in experimental models include frequencies of approximately 800–1,000 kHz and intensities near 140 mW/cm² applied for about 10 minutes daily. *Electrical stimulation (ES)* represents another widely investigated technique. Both *postoperative electrical stimulation (PES)* and *conditioned electrical stimulation (CES)* have been shown to promote axonal outgrowth, enhance myelination and mitigate muscle atrophy after nerve repair. Frequencies around 20 Hz are most frequently reported, though lower frequencies (1–2 Hz) may induce more compact axonal organization with thicker myelin sheaths. ES appears to enhance axoplasmic flow, facilitate neurotrophin migration toward the cathode and improve synaptic reconnection. *Photobiomodulation (PBM),* using wavelengths within the 650–905 nm range, exerts photochemical effects on mitochondria, stimulating ATP production and neuronal metabolism. In contrast, shorter wavelengths (e.g., 473 nm blue light) may inhibit axonal regeneration. PBM is increasingly recognized for its potential to modulate oxidative stress and promote functional reinnervation[72-77].

Aerobic exercise has also emerged as a supportive intervention, improving peripheral circulation, oxygen delivery and neuronal metabolism. Although protocols remain heterogeneous, regular moderate aerobic activity appears to contribute to a more favorable neurotrophic environment and enhanced sensorimotor recovery. *Complementary modalities* such as hyperbaric oxygen therapy (HBOT), magnetotherapy and biofeedback have also been explored, though standardized parameters and high-quality evidence are still lacking[72].

Chu and colleagues emphasize the persistent challenges in this field, including the absence of standardized treatment protocols, variability in individual responses and limited long-term follow-up data. Consequently, they propose an integrated, multimodal rehabilitation approach, combining several physical therapy modalities to maximize the regenerative potential and functional outcome after peripheral nerve injury[72].

Range of motion and strengthening

Range-of-motion (ROM) exercises remain a cornerstone of upper-extremity rehabilitation, essential for preventing joint contractures, preserving soft-tissue mobility and minimizing fibrotic adhesions. Contemporary literature emphasizes that both passive techniques, whether therapist-applied or device-assisted and active, patient-initiated movements contribute to maintaining tendon gliding and optimizing collagen alignment during healing. Recent reviews highlight that early, controlled ROM promotes favorable biomechanical conditions for tendon excursion while reducing stiffness and improving functional recovery[78]. Similarly, clinical analyses of modern post-repair tendon protocols demonstrate that structured ROM interventions, including both active and passive components, are critical for reducing adhesion formation and enhancing digital mobility[79,80]. This recent knowledge reinforces the role of ROM as a fundamental therapeutic element in preventing secondary complications and supporting optimal outcomes in hand rehabilitation.

Strengthening exercises following peripheral nerve injury must be introduced cautiously, as reinnervating muscles are highly susceptible to fatigue and traction stress. Initial efforts should focus on low-load, high-control movements that promote motor unit recruitment and coordination without overloading vulnerable neuromuscular structures. As regeneration progresses and muscle endurance improves, resistance can be increased gradually to support hypertrophy, restore functional strength and reintegrate the affected limb into complex daily and occupational tasks[81].

Neurorehabilitation techniques

Modern rehabilitation following peripheral nerve injury increasingly integrates neurophysiological principles and technology-assisted methods to enhance cortical reorganization, sensory discrimination and functional recovery. The overarching goal of these interventions is to bridge the gap between peripheral regeneration and central adaptation, promoting restoration of the sensorimotor network and meaningful functional reintegration.

Sensory re-education is a central component of rehabilitation after peripheral nerve injury, aiming to reduce dysesthesia, normalize sensory processing and restore discriminatory touch as regenerating afferents reach their cutaneous targets. Early-phase interventions, such as desensitization, graded texture exposure and localization tasks, help modulate hypersensitivity and recalibrate cortical sensory responses[82-84].

As sensory recovery advances, structured discrimination activities, including shape, texture and two-point discrimination tasks, facilitate refinement of tactile gnosis and support functional hand use[52]. Cortically-based strategies, particularly mirror-induced visual–tactile feedback, have demonstrated the capacity to enhance cortical remapping and improve perceptual accuracy in patients with sensory deficits following nerve injury[55]. Together, classical frameworks and contemporary neurorehabilitation evidence support a progressive, multisensory program designed to exploit activity-dependent plasticity and strengthen sensorimotor representations during reinnervation.

Motor retraining focuses on precise, task-oriented repetition to promote adaptive cortical reorganization[9]. *EMG biofeedback* enhances voluntary muscle activation and prevents compensatory patterns[16]. *Robotic-assisted training* offers controlled, repeatable movements and early mobilization, facilitating coordinated strength recovery. *Neuromodulation techniques* such as transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) modulate cortical excitability and enhance synaptic plasticity when paired with peripheral stimulation. Preliminary studies suggest improved dexterity and sensory recovery when central and peripheral activations are synchronized. When combined with peripheral electrical stimulation (PES), these methods create synergistic effects by linking central and peripheral activation, thereby optimizing the reinnervation process and promoting synchronized recruitment of motor units. Although clinical evidence is still evolving, early trials suggest that neuromodulatory interventions can accelerate recovery of dexterity, sensation and cortical responsiveness in upper-limb nerve injuries[11,13,85,86].

Recent advances in *virtual and augmented reality* (VR/AR) have introduced immersive environments that stimulate both visual and proprioceptive feedback, enhancing engagement and promoting motor learning through action observation and embodiment[12]. *Haptic feedback systems* provide controlled tactile input and kinesthetic resistance, improving fine motor control and spatial perception. Additionally, serious gaming platforms are increasingly used to gamify repetitive rehabilitation tasks, increasing motivation and adherence while maintaining therapeutic precision[16]. Collectively, these technologies represent a new frontier in cortically driven, feedback-based rehabilitation, integrating neuroscience, engineering and behavioral science to maximize recovery potential after peripheral nerve injury.

Splinting and orthotic management

Custom-fitted splints play a crucial role in maintaining joint alignment, protecting soft-tissue repairs and preventing deformities. They should allow partial movement, preserve tactile feedback and avoid excessive pressure on delicate structures, with regular adjustments made as recovery progresses and functional demands evolve. Orthotic management is particularly essential following peripheral nerve repair, where protection of the coapted nerve, prevention of joint contractures and maintenance of optimal tendon–nerve relationships are critical for functional restoration. After nerve repair, splints are commonly used to position the extremity in a protective posture that reduces tension across the neurorrhaphy and minimizes traction during early mobilization phases[54]. Chan emphasizes that orthoses for peripheral nerve injuries must balance protection with the need to prevent secondary deformities such as clawing, MCP hyperextension, PIP flexion contractures or intrinsic imbalance, depending on the nerve involved. Devices may include resting hand splints, anti-claw orthoses, dorsal blocking splints or functional positioning orthoses tailored to the specific pattern of paralysis and muscle imbalance. As reinnervation advances, orthoses should be progressively modified to increase mobility while maintaining joint stability and safeguarding weakened musculature[87]. Collectively, contemporary evidence and foundational guidelines support a dynamic, phase-specific orthotic strategy that evolves in parallel with neural regeneration and the patient's functional progression.

Patient education and multidisciplinary collaboration

Education is a key determinant of long-term success because it empowers patients to understand their condition, adhere to therapy and adopt protective behaviors. Continuous feedback improves self-awareness, motivation and autonomy. Patients should understand the purpose of each therapeutic intervention and be encouraged to actively participate in their recovery. Empowering patients to integrate therapeutic principles into daily activities fosters adherence and accelerates reintegration into functional and occupational roles.

Effective rehabilitation demands *collaboration among surgeons, physiotherapy team and psychologists*, ensuring biological repair translates into meaningful function. *Multidisciplinary collaboration* represents a cornerstone of successful functional recovery after complex hand trauma, particularly during the chronic or reconstructive phase. Each specialist contributes a complementary perspective, ensuring an integrated approach that addresses the surgical, functional, psychological and occupational dimensions of rehabilitation. The hand surgeon focuses on restoring anatomical continuity and structural stability, while physiotherapists aim to optimize motion, enhance strength and facilitate the reintegration of fine motor skills required for daily and professional activities. The psychologist plays a key role in managing anxiety, depression and altered body image, which are common after severe injuries, thereby improving motivation and adherence to therapy[8,16,48,54,58,88-90]. Constant communication and reciprocal feedback among team members are essential for monitoring progress, adjusting therapeutic goals and preventing secondary complications. Through this ongoing cooperation, rehabilitation extends beyond physical recovery, evolving into a comprehensive process of restoring autonomy, identity and overall quality of life.

DISCUSSION

Peripheral nerve injuries represent a complex medical challenge, leading to sensory and motor deficits with significant psychosocial impairment that severely impact patients' quality of life, especially when the upper limb is involved.[6,25]

Peripheral nerve injuries need specific therapeutic strategies with tailored approaches to restore functional outcomes. The choice of repair method depends on the severity of the injury, the size of the nerve gap and the available resources.[28,31]

Nerve repair require precise microsurgical techniques, ranging from direct coaptation, applicable when no continuity defect is present (the ideal scenario) to situations in which a nerve gap necessitates reconstruction.[22,91] Optimal nerve coaptation consists of a tension-free approximation of healthy proximal and distal fascicles, supported by well-vascularized surrounding soft tissues, thereby providing an adequate environment to effective healing.[33,92,93]

For direct coaptation, several technical approaches have been described. Epineural repair represents the classic option, used in cases where global alignment of the nerve trunk is required. Epi-perineural repair, currently regarded as the optimal technique in most situations, enables more precise fascicular alignment by incorporating both the epineurium and perineurium into the suture, providing accuracy and safe tissue handling. Group fascicular repair is applied in selected cases, particularly when the internal nerve architecture is clearly identifiable and fine fascicular-level reconstruction is required. The choice of technique is determined by the clinical characteristics of the injury and by the degree of dissection that is permitted without compromising the nerve's vascularity.[22,94-96]

In the presence of a nerve gap, reconstructive options include autografts, still considered the gold standard in nerve repair, as well as allografts, nerve transfers and conduit-based techniques. Each of these methods has specific indications, dictated by the complexity of the trauma, the patient's condition, the available reconstructive resources and the surgeon's level of experience.[22,29, 32]

Multiple adjunctive techniques have been proposed to promote peripheral nerve regeneration, observing that even when microsurgical repair is executed at an optimal technical standard, the extent of functional recovery remains unpredictable and often limited. Figure 6 illustrates these principles of peripheral nerve repair.

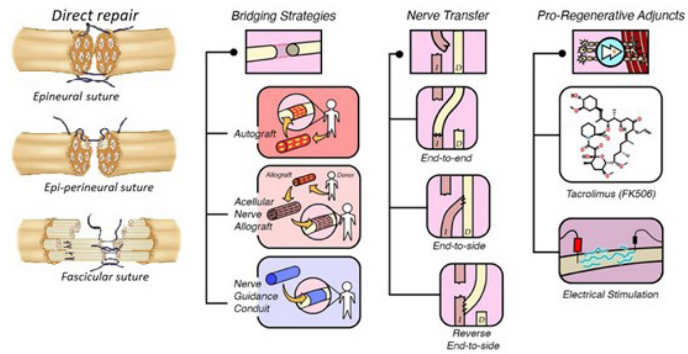

Fig 6. Peripheral nerve repair strategies[43,22,95,96]

The success of peripheral nerve regeneration depends on a combination of biological, mechanical and microenvironmental factors that modulate axonal growth and target reinnervation. Age plays a decisive role, as regenerative capacity declines with advancing age due to reduced Schwann cell responsiveness, diminished neurotrophic factor expression and slower axonal elongation[11]. The length of the nerve gap critically affects outcomes: longer defects require grafting or conduit repair, which increases the risk of axonal misdirection and incomplete reinnervation[13]. The type and timing of repair, whether direct end-to-end neurorrhaphy, nerve grafting or nerve transfer, also determine the alignment precision between fascicles and the time available for target reinnervation before irreversible muscle atrophy occurs[9]. Ischemia and vascular compromise at the repair site reduce oxygen and nutrient supply, impeding Schwann cell metabolism and axonal regeneration, while excessive fibrosis or scarring creates physical barriers and aberrant collagen deposition that obstruct regenerating axons[5].

Optimal outcomes depend on minimizing delays to repair, ensuring adequate vascular supply and implementing early rehabilitation strategies that prevent secondary tissue changes and support neuroplastic adaptation[16].

Peripheral nerve injury triggers a cascade of neuroplastic responses extending beyond the lesion site, profoundly affecting central sensorimotor organization. Functional and structural reorganization occurs within the primary somatosensory and motor cortices, where deprived representations of the affected limb undergo rapid remapping[12].

While early cortical plasticity may facilitate compensatory mechanisms, persistent sensory deprivation or maladaptive reorganization can lead to distorted body schema and impaired motor control[13].

Rehabilitation strategies harnessing mirror neuron activation, such as mirror therapy and active observation, promote sensorimotor congruence by re-engaging cortical networks responsible for both the execution and observation of movement[16]. The mirror neuron system, primarily located in the inferior parietal and premotor cortex, facilitates the restoration of coordinated motor output by linking visual feedback with motor intention and proprioceptive experience[12]. Through targeted interventions that combine graded sensory re-education, bilateral training and visual–motor coupling, therapists can guide adaptive neuroplasticity, supporting the reintegration of cortical representations with regenerating peripheral pathways[9,11]. This bidirectional interaction between peripheral regeneration and cortical reorganization underscores the need for early, multisensory rehabilitation that simultaneously addresses biological repair and central adaptation.

Rehabilitation after peripheral nerve injury is organized as a continuous, interdependent sequence of steps that parallels the biological processes of axonal regrowth and soft-tissue repair. Instead of being divided into strict stages, this framework supports clinical management by allowing adjustments based on the type of surgery performed, the extent of reinnervation and the patient's levels of pain, edema and functional activity. The figure 7 summarizes the essential phases of rehabilitation after peripheral nerve injury and the fundamental therapeutic principles, previously presented in extenso.

Ultimately, successful outcomes rely on a multidisciplinary model in which surgeons, physiotherapists and psychologists collaborate to align biological repair with functional reintegration. Future advances, driven by AI-based personalization, neuromodulation and predictive modeling, hold promise for transforming rehabilitation into a dynamic, evidence-guided process that adapts to the patient's evolving neural and functional state.[97-99]

By merging surgical precision with neuro-rehabilitative innovation, clinicians can more effectively restore autonomy and quality of life for individuals recovering from upper-limb peripheral nerve injuries.

CONCLUSION

Peripheral nerve injuries present a complex therapeutic challenge, requiring tailored approaches to restore functional outcomes. The choice of repair method depends on the severity of the injury, the size of the nerve gap and the available resources. While autografts remain the gold standard for nerve repair, advancements in nerve conduits and regenerative medicine offer promising avenues for improving outcomes.

Functional recovery after peripheral nerve injury depends not only on the quality of surgical repair but also on timely, individualized and sensorimotor-oriented rehabilitation that promotes cortical remapping and functional reintegration. The restoration of meaningful hand and upper-limb function requires

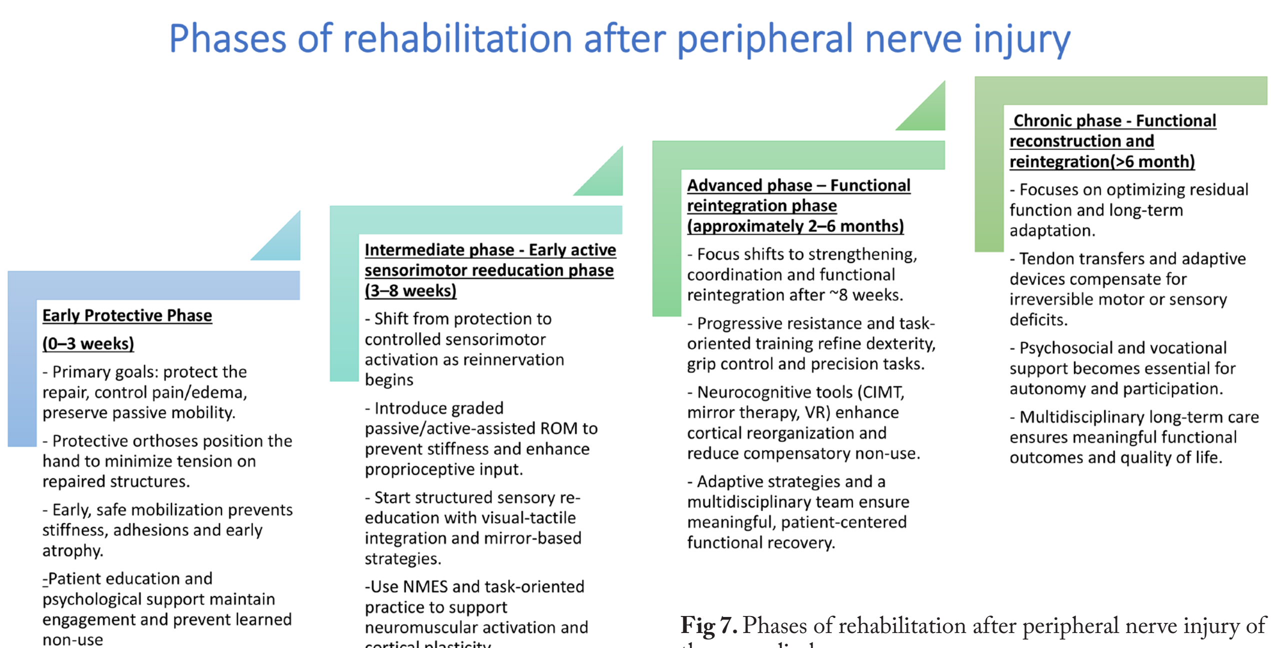

Fig 7. Phases of rehabilitation after peripheral nerve injury of the upper limb

a continuous interplay between peripheral biological regeneration, central nervous system plasticity and targeted therapeutic intervention. Early protection of the repair, progressive sensorimotor re-education and advanced neurorehabilitation strategies form the foundation of optimal recovery. A structured, phase-based rehabilitation program, spanning from early immobilization and edema control to late functional retraining, ensures safe tissue healing while stimulating adaptive reorganization of cortical and subcortical pathways. Evidence supports the integration of mirror therapy, task-specific practice, neuromuscular stimulation and immersive technologies such as virtual reality to reinforce sensorimotor congruence and enhance patient engagement. The inclusion of psychological assessment and patient-reported outcomes further ensures that rehabilitation addresses both the physiological and psychosocial dimensions of recovery.

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The authors did not use in this article generative AI and AI-assisted technologies.

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