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NERVES INJURIES AND THEIR REPAIR

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Abstract: **Patients with nerve injury particularly extreme damage, regularly confront poor nerve recovery and not entirely utilitarian recuperation, even after surgical nerve repair. Recent studies about the new methodologies for the treatment of nerve injuries. Different than other tissues in the body, nerve recovery nerve regeneration is slow and usually inadequate. Not as much as half of patients who experience nerve repair after damage recapture great to amazing engine or tactile capacity. Our expanding information about nerve physiology and recovery far exceeds our surgical capacities to repair injured nerves and effectively recover engine and tangible capacity. Thus this review aims to summarizes treatments of nerve injures, and also describe why surgical management is still inadequate in using the new biological research that has documented the changes that occur after the nerve injury, which could cause suboptimal clinical outcomes.**

Keywords: **their repair, methodologies, engine and tangible capacity.**

1. INTRODUCTION

nerve injuries in the upper extremity are extremely common. The typical patient is usually young, sustaining a laceration from metal sharp objects or machinery [(1)]. To make the diagnosis of a nerve injury it is important to take a detailed history especially with regards to the timing of the event as this will help with guiding treatment. Sensation can be tested using several methods. Vibration is tested using a turning fork, which can be useful to test, as usually deficits occur before subjective complaints. Two-point discrimination is tested by using a paper clip and good for testing group of axons slow fibers. Pick-up tests have shown to be useful to test sensibility and tactile gnosis where the patient picks up the instructed item from a table full of multiple objects [(2,3)]. Sudomotor activity can be interestingly be assessed in children by the absence of wrinkling after water immersion or in adults using the sweat test [(3)]. Motor function should be tested as like the upper and lower limbs in neurological examination and graded from 0-5. Special tests can be used to support or confirm a nerve injury including electromyography (EMG), nerve conduction studies or electrical muscle stimulation.

Injuries to peripheral nerves are extremely common in many types of upper limb trauma. Injury to peripheral nerves can cause extreme dysfunction in the hand for the patient disrupting their professional and leisure activities. It is therefore vital that adequate treatment is available to repair peripheral nerves to prevent permanent financial loss for the patient as well as the healthcare economy [(4)].

Objectives:

Numerous studies indicate that surgical rapier of nerves after being injured and defects in humans that can be successfully treated by implantation of artificial nerve guides. This review provides a brief overview of various preclinical and clinical trials conducted to evaluate the utility of artificial nerve injuries repair for the regeneration of peripheral nerves. This review is also intended to help update surgeons on the rapid advances in surgical techniques that could save the function of nerve after been injured.

2. METHODOLOGY

We systematically reviewed the literature published from January 1980 to February 2016 using multiple databases to search the following: nerve injuries and graft, repair, reconstruction, nerve transfer, neurotization. Out of 934 studies

initially identified, 30 were included in analysis. We searched MEDLINE, the Cochrane Library, and CINAHL for English-language and human-only studies published before June 2016, We manually searched reference lists of pertinent reviews, included trials, and background articles on this topic to look for relevant citations our searches might have missed.

3. RESULTS

The goal of surgical repair is to repair the nerves so that function is restored to the area. Depending on the type and severity of the injury, but first we must discuss important aspects concerning the physiology and degeneration of nerve cells.

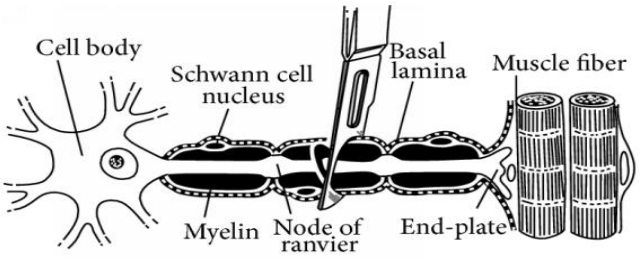
Key roles are played by cells other than neurons in the maintenance and function of the peripheral nerves. Schwann cells ensheath nerves in a layer of myelin and provide trophic support through the release of important neurotrophs such as Nerve Growth Factor (NGF). Myelin improves conduction velocity by limiting the sites of ionic transfer along the axon to the nodes of Ranvier, resulting in a faster, "jumping" action potential propagation that is termed saltatory conduction. The most heavily myelinated fibers are the large motor neurons (Type Aα), followed by afferent muscle spindles (Type Aβ). Nerve conduction velocities in theses neurons are approximately 30-120m/s. Unmyelinated neurons (Type C), such as the sensory neurons involved in transmitting pain and temperature and postganglionic sympathetics are the slowest, conducting at approximately 1-2 m/s (Table 1) (5,6,7).

Table.1: Nerve Fiber Types and Properties

Fiber Class	Myelin	Diameter (Mm)	Conduction Velocity (m/s)	Spinal Cord Tract	Location	Function
Aα	+	6-22	30-120	Ipsilateral dorsal column	Efferent to muscles	Motor
Aβ	+	6-22	30-120	Contralateral spinothalamic tract	Afferent from skin and joints	Tactile, proprioception
Aγ	+	3-8	15-35	Ipsilateral dorsal column	Efferent to muscle spindles	Muscle tone
Aδ	+	1-4	5-30	Contralateral spinothalamic tract	Afferent sensory nerves	Pain, cold, temperature, touch
B	+	1-3	3-15	Preganglionic	Preganglionic sympathetic	Various autonomic functions
sC	-	0.3-1.3	0.7-1.3	-	Postganglionic sympathetic	Various autonomic functions
dC	-	0.4-1.2	0.1-2.0	Contralateral spinothalamic tract	Afferent sensory nerves	Various autonomic functions Pain, warm, temperature, touch

The internal neuronal environment like all cells is in carefully controlled electrolyte homeostasis. Antegrade and retrograde axoplasmic transport cycles neurotransmitters and structural cell elements back and forth between the cell body and axonal tip. Any break or defect in the axonal or neuronal bilayer lipid membrane unless rapidly repaired results in an irreversible cascade of programmed cell death [(8)].

Axonal degeneration follows a sequence of events within the zone of trauma extending both proximally and distally (Figure 1). Disconnected axons and cell bodies (in proximal axon injuries) degenerate via a programmed cell death pathway called chromatolysis [(9,10)]. This focal degeneration is similar to what occurs in other traumatized tissues including skin and muscle [(11)]. However, the major difference compared to other tissues is that Wallerian degeneration of the distal axonal segment then occurs from the zone of trauma to the motor or sensory receptor some distance away. Wallerian degeneration ensues 24–48 hours after peripheral nerve injury and both the distal axons and surrounding myelin degenerate [(12)]. The proximal axonal segment also degenerates back to the adjacent node of Ranvier, the site of subsequent axonal regrowth.



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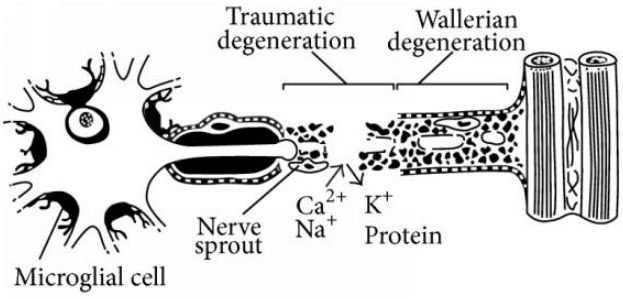

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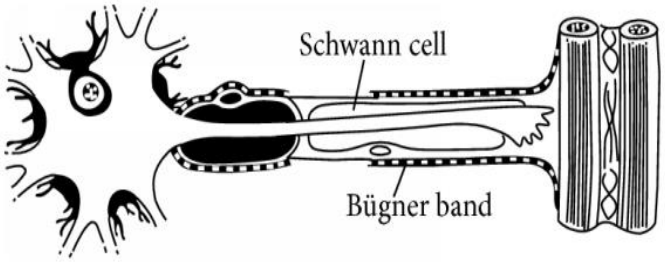

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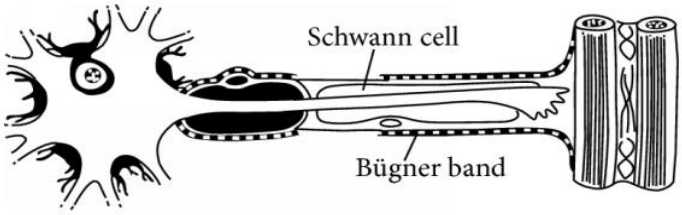

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Figure.1: Degeneration and regeneration after peripheral nerve injury [15]

classification of nerve injuries:

The earliest classification of nerve injury was made by Seddon in 1947 who described three injury grades (Table2) [14]. Neurapraxia is segmental myelin damage with an intact axon, usually caused by compression. There is a temporary focal conduction block that resolves completely within 12 weeks once myelination is restored. Axonotmesis from a crush mechanism is axonal injury where the connective tissue and nerve continuity remain intact. Wallerian degeneration ensues and slow axonal regeneration follows at a rate of 1 mm/day. Incomplete recovery is common, depending on the distance for regeneration between the injury and target tissue. Neurotmesis is complete physiological and anatomical transection of both axons and connective tissue. A neuroma may form but no spontaneous regeneration occurs without surgical intervention.

Table 2: Nerve injury classification in increasing severity.

Sunderland [13]	Seddon [14]	Features
Type 1	Neuropraxia	Damage to local myelin only
Type 2	Axonotmesis	Division of intraneural axons only
Type 3	Axonotmesis	Division of axons and endoneurium
Type 4	Axonotmesis	Division of axons, endo- and perineurium
Type 5	Neurotmesis	Complete division of all elements including epineurium
Type 6*	Mixed	Combination of types 2–4

Aspects of Nerve Repair:

Functional nerve recovery relies on motor axons correctly matched to motor endplates and sensory axons reaching their sensory receptors. Most studies have graded the success of nerve repair using the British Medical Research Council's system or its modified versions for the evaluation of motor and sensory return. Physical examination allows grading of sensory recovery from S0 to S5 and motor from M0 to M5 [16]. Mackinnon and Dellon reported in a 40-year compilation of data that after direct nerve coaptation 20–40% achieved very good (M4S3+) recovery after nerve repair but that few injuries recovered fully [17].

The results of nerve grafts (and allografts) are worse than for nerve coaptation. Grafts proximal to the elbow, more than 7 cm in length, older patients, and greater delay to nerve reconstruction are adverse prognostic features [18].

No alternatives to autologous nerve grafts have demonstrated equivalent outcomes in gaps >3 cm. For small gaps, the application of artificial resorbable nerve guides to bridge nerve defects up to 3 cm has the same success rate as nerve autograft repair, which results in recovery in up to 69% of cases [19].

In 1990 Sunderland summarized 40 years of clinical experience in nerve repair: early repairs are better than late; nerve coaptation is better than nerve grafts; young do better than old; distal repair is better than proximal repair; short grafts do better than long [20]. These principles remain equally as relevant today.

Glue Repair:

Advantages of an adhesive for nerve repair include ease of use, less tissue trauma, maintenance of nerve architecture, better fascicular alignment, and less scarring compared to microsutures [21].

The ideal glue should not induce fibrosis that can lead to nerve compression and in the case of substance interposition between nerves, it should not act as a barrier to nerve regeneration. The glue should provide adequate mechanical strength to prevent gapping or rupture at the initial repair and during the postoperative period.

Fibrin sealants have a proven track record as a safe and effective nerve glue [21]. The longest and greatest experience with nerve glue is in brachial plexus reconstruction. In this setting, fibrin glue has been indispensable. Narakas reported significantly reduced operative times and the ability to perform nerve repairs in areas where it was previously not possible [22,23]. Nerve glue allows repairs to be performed at or immediately within the bony foramen of a proximal nerve root where quality suture repair is not possible [21].

A systematic review of fibrin glue for peripheral nerve repair revealed 14 animal studies, 1 cadaver study, and 1 human study that fit the study criteria [24]. Most found fibrin glue repair to be equal or superior to suture repair.

Technique of Nerve Repair:

There are four main steps to a primary end-to-end repair – the most commonly used nerve repair technique.

1. Preparation – The nerve ends are prepared to get visible ends with necrotic tissue being removed with blades leaving two normal looking ends. Flexing the joint above the nerve injury and bone shortening can be given more length if this is required [25].

2. Approximation – The nerve ends are mobilized and brought together leaving a minimal gap by applying appropriate tension. Tensionless repairs have shown to have better outcomes. During the approximation the nerve ends can be mobilised but extensive intrafasicular dissection should be avoided [25].

3. Alignment – Bloods vessels must be aligned and proper rotational alignment undertaken [25].

4. Maintenance – The nerve repair is maintained by stitches into the epineurium, commonly 9-0 or 10-0 non-absorbable sutures. Hence, it is the epineural repair that keeps the repair together. The sutures need to be placed to avoid malrotation of the nerve ends. Sometimes individual fasiscular groups are identified for attachment (group fascicular nerve repair). These types of repair are usually preferred for larger nerves where sensory and motor fibers can be repaired separately [26].

Postoperatively nerve repairs should be protected by immobilization for 10-14 days and sometimes surgeons advocate up to six-weeks depending on the nerve injury severity and cause [25]. After this period full passive and active range of motion is initiated for rehabilitation [25]. Postoperatively axons may take time to learn how to process new information especially following sensory nerves [27,28]. Age is the most vital factor to determine the outcome of nerve repair and can account for 50% of the variance in success[29].

Another technique that can be used to repair nerves is the end-to-side nerve repair, which involves the attachment of one or two distal injured nerve end to the side of the uninjured nerve ends. This is a useful technique when the ends are not available as sources of axons [30].

4. CONCLUSION

Functional recovery after nerve repair has slowly improved since the development of microsurgical repair techniques more than 50 years ago. Nevertheless, many patients particularly with proximal nerve injuries suffer incomplete recovery and lifelong disability. Direct nerve repair yields the best results and nerve autografts remain the gold standard treatment for nerve gaps. Conduits have a limited role in small gaps <3 cm for sensory nerves, but this may expand in the future with improvements in conduit design.

The biological roadblocks to early and complete recovery remain Wallerian degeneration, slow axonal regeneration, and the effects of chronic axotomy on denervated muscles. Translational research therapies address some of these barriers and future advances in surgical care may come from enhancing axonal regrowth, electrically stimulating the distal motor target after injury, and most powerfully delaying or avoiding Wallerian degeneration.

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