Management of acute nerve injuries of the hand

Feiran Wu
Chye Yew Ng

Abstract

Acute nerve injuries of the hand are common and demand a methodical approach to their assessment and management. The decision to explore a hand wound is generally made based on careful clinical assessment. Different repair and reconstructive techniques are employed depending on the mechanism of injury, the type of nerve injured, the length of any defect and the timing of surgery. This article aims to provide a current review of the principles of nerve repair and reconstruction in the hand.

Keywords nerve allograft; nerve conduit; nerve injury; nerve reconstruction; nerve repair; vein conduit

Introduction

Injuries of the hand are common and among the affected structures are peripheral nerves with an incidence of 0.14/1000 inhabitants/year.[1] Despite the advances in understanding the pathophysiology of peripheral nervous system injury and regeneration, as well as advancements in microsurgical techniques, peripheral nerve injuries remain a challenge for reconstructive surgeons. This article aims to provide a current review of the principles and indications of nerve repair and reconstruction in the hand.

Assessment

All patients with a potential nerve injury should be assessed on an individual basis. The typical patient who sustains an acute nerve injury to the hand is a young male through domestic or industrial accidents.[2] The injury can be a result of sharp or blunt trauma, with glass being the most common cause, either by falling through glass, onto glass or from hand-held glass objects. Occupational injuries involving machinery are likely to cause more extensive injuries or a mangled extremity.[2]

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 to guide treatment. Sensory and motor function should be carefully examined and documented. Light touch is easily examined in the acute setting by the Ten test.[3,4] In this test, a normal area of the body is initially stroked by the examiner and told that this is equivalent to a 10 on a 1-to-10 scale. The examiner subsequently strokes the normal or '10' area, while simultaneously and with equal pressure strokes the abnormal area and asks the patient how this compares with the normal area on a 1-to-10 scale, with 10 being the best sensibility that can be appreciated. The Ten test has been found to correlate well with other tests of hand sensibility such as with a Semmes–Weinstein monofilament test, has good inter- and intra-observer reliability, and is accurate in detecting very early loss of sensibility when moving and static two-point discrimination appear normal.[5] Moving and static two-point discrimination should be assessed by a two-point discriminator. The normal static value is less than 6 mm in adults at the fingertips.[6] Motor function should be graded according to the Medical Research Council (MRC) grades of 0–5.

Sympathetic unmyelinated fibres in a peripheral nerve are among the most resistant to mechanical trauma.[7] As such, damage to these fibres suggests a more severe injury. Disturbance of sympathetic function is therefore an important early feature of nerve damage. The skin wrinkle test is an effective and simple test for autonomic dysfunction that can be performed acutely. By simply wrapping the traumatized digit in a wet swab on admission to the accident department, the lack of skin wrinkling within a few minutes will indicate autonomic dysfunction and the presence of a more serious nerve injury.[7]

Classification

Sir Herbert Seddon classified peripheral nerve injuries into three types in 1942, based on the anatomic layers of the nerve that have been injured and the potential functional outcomes from these injuries.[8,9] In neurapraxia the axon remains in continuity and the epineurium, perineurium and endoneurium remain intact. There may be disruption of the myelin layer encasing the nerve fascicles. There is no distal Wallerian degeneration but there is a focal area of nerve conduction block. Recovery is usually complete and occurs in days to weeks. In axonotmesis the axon is disrupted, but the supporting structures of Schwann tubes, endoneurium and perineurium remain intact. Recovery could be complete because the endoneural conduits remain intact, and regenerating axons do not need to traverse a nerve coaptation site. Neurotmesis refers to division of a nerve where all essential parts are destroyed. However, this can also include a neuroma-in-continuity where interruption can occur without apparent loss of continuity. Complete recovery will not occur in neurotmesis without surgical repair.

In 1951, Sunderland felt there were limitations to this classification and proposed a new classification system based on the histological examination of injured nerves.[10] He proposed five degrees of nerve injury arranged in ascending order of severity and affect successively: (i) conduction in the axon, (ii) continuity of the axon, (iii) the endoneurial tube and its contents, (iv) the funiculus and its contents, and finally (v) the entire nerve trunk. Mackinnon and Dellon later introduced a type VI injury, to denote a mixed nerve injury with a combination of grades I to V lesions, which is more representative of the nerve injuries seen in a clinical setting.[11]

Lundborg, in 1988, proposed two mechanisms for grade I conduction block injuries.[12] He described type A injuries were due to intraneural circulatory arrest, causing a metabolic block

Feiran Wu *MA MB BChir (Cantab) FRCS (Tr & Orth) Training Interface Group Hand Fellow, Upper Limb Unit, Wrightington Hospital, Wigan, UK. Conflicts of interest: none declared.*

Chye Yew Ng *MBChB (Hons) FRCS (Tr & Orth) DipHandSurg (Br & Eur) Consultant Hand and Peripheral Nerve Surgeon, Upper Limb Unit, Wrightington Hospital, Wigan, UK. Conflicts of interest: none declared.*

with no nerve fibre pathology, leading to clinical changes that are immediately reversible when the insult is removed. Type B injuries are a result of intraneural oedema, leading to increased endoneurial fluid pressure. These injuries are reversible once the oedema resolves but this process can take days or weeks.

A simplified and more pragmatic classification was described by Birch and colleagues,[13] which divides nerve injuries into non-degenerative and degenerative lesions. These classifications are summarized in Figure 1.

Treatment

Timing of nerve repair

Timing of nerve repairs depends on the type of nerve and the mechanism of injury sustained, the condition of the wound, as well as the vascular supply of the nerve bed. In sharp nerve transections with a good blood supply, a clean wound bed and with no or minimal crushing element, primary nerve repair is the best option for restoring function.[14] Historically, nerve repairs were performed 3 weeks after initial injury, but Mackinnon et al. demonstrated better outcomes from immediate reconnection of the transected nerve.[15] Currently, primary surgery is performed within 72 hours when possible, but can be performed up to 7 days following nerve injury.[16]

In cases of blunt trauma, the degree of damage to the nerve stumps can be more precisely evaluated after several weeks. The time delay is limited, however, by the viability of surrounding Schwann cells and endoneural tubes. It is essential for regenerating axons to reach the endoneural tube within 12–18 months after injury, otherwise degeneration will occur and target muscle atrophy becomes irreversible.[17]

Direct repair

Primary immediate repair should be the method of choice for neurobiological reasons and is easier to perform than a delayed repair. The main principles of repair include careful mobilization and debridement of the severed nerve ends, followed by approximation in the correct orientation without undue tension.[16,18] The nerve should be dissected from a healthy area, safely down to the zone of injury. Skeletal stability is a prerequisite and associated soft tissue or bony injuries will also need to be considered. Micro-instrument use is essential, under magnification with loupes or microscope, to minimize further tissue trauma. Microscope use has been shown to be superior to loupes for digital nerve repair in a cadaveric model,[19] but this difference has not been replicated in the clinical setting.[20]

End-to-end repair techniques include epineural repair, group-fascicular repair, and fascicular repair. The fascicular pattern and epineural vessels can be used as a visual aid for the correct orientation of the nerve ends. Tension to a peripheral nerve can compromise blood supply to the nerve ends with subsequent effects on the viability of Schwann cells, which leads to impaired axonal outgrowth. Therefore, it is of the utmost importance to perform a tension free repair. The joints should be placed in a neutral position and the suture size of choice is used to perform an epineural stitch. A rule-of-thumb is that if the nerve ends cannot be coaptated without tearing, then there is too much tension and a conduit or graft is indicated.

In epineurial repair, our preference is to use 9-0 or 10-0 nylon sutures in digital nerve repairs and 8-0 nylon sutures in larger trunk repairs at the wrist. The number of sutures should be the minimum required to ensure coaptation of the nerve ends. Grouped fascicular repair can be applied to a crush nerve injury,

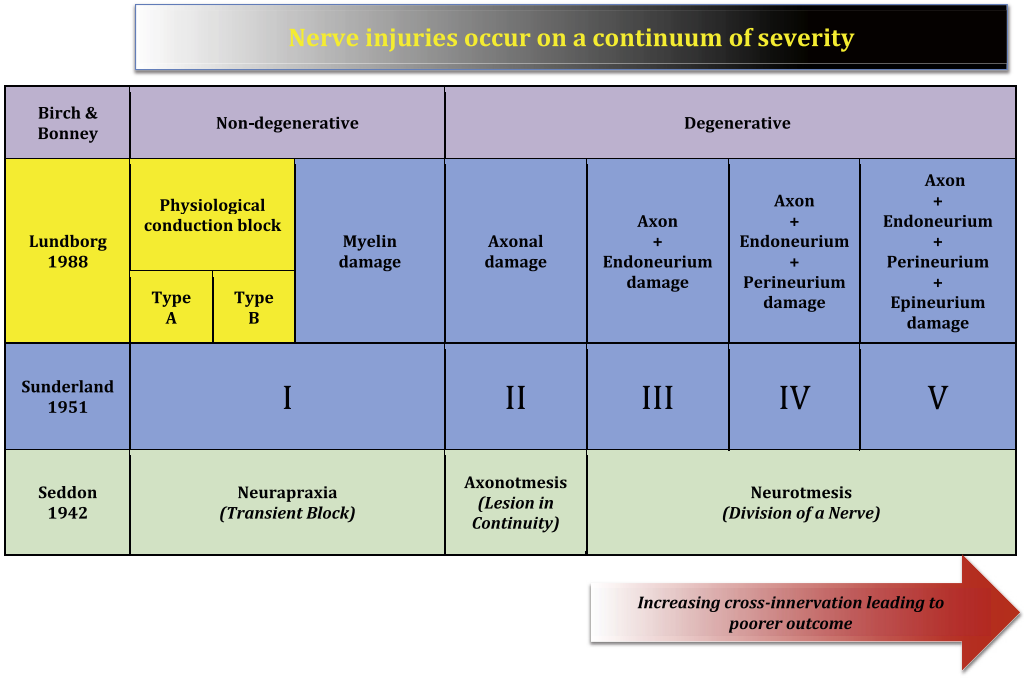

Figure 1 A summary of classifications of peripheral nerve injuries. Historical references are covered in the text.

or a delayed nerve repair that require trimming of the nerve ends, particularly in mixed nerves where it is easy to identify groups of matching fascicles. In this technique, the epineurium is retracted prior to debridement and corresponding fascicles are coaptated with two to three sutures passing through the interfascicular epineurium. The functional results of group fascicular repairs have not been found to be more superior than that of epineurial repair. Better fascicle alignment afforded by a fascicular repair is potentially offset by the increased scarring due to the increased dissection required, when compared to a simpler epineural repair.[21]

Commercial fibrin glue can be used as an alternative to sutures, or more typically as an adjunct to epineural sutures. There is a lack of literature comparing the efficacy of fibrin glue to suture repair in a well-designed clinical trial, but a systematic review involving animal studies has shown this to be a viable technique for peripheral nerve repair compared to microsuturing.[22]

Nerve grafts

In case of nerve gaps, which cannot be coaptated without undue tension, then a nerve reconstruction may be performed. The gold standard remains the use of autologous nerve grafts, although other strategies, such as nerve allografts and synthetic nerve conduits have been introduced.[23] Nerve grafts have shown superior results when compared with direct repairs performed under undue tension that produced nerve ischaemia.[24,25] Injured nerve endings can retract up to 28% in delayed nerve repairs compared to 4% in primary repairs, thereby increasing the need for the use of nerve grafts or conduits.[26]

Donor nerves can be harvested from different locations and should be selected on the basis of the size and length of the defect of the specific nerve to be reconstructed. Common sites include the sural nerve, lateral antebrachial cutaneous nerve and the terminal branch of the posterior interosseous nerve (PIN). For digital defects, we prefer the use of the terminal PIN, which lies in the dorsal fourth compartment, as it leaves no motor or sensory deficit. The distal end of the anterior interosseous nerve can be used as a donor for defects of the deep branch of the ulnar nerve or the recurrent motor branch of the median nerve.[27] For the bridging of larger nerve defects, such as in the radial, ulnar and median nerves, the sural nerve can be used with application of several cables. The superficial radial nerve and lateral antebrachial cutaneous nerve may be considered in cases of irreparable damage proximally, but these should be a last resort as they are more prone to the formation of painful neuromas.[28]

Alternative reconstruction techniques

Autologous nerve grafting has significant limitations: 1. Donor site morbidity caused by the need for an additional skin incision and dissection; 2. Limited availability of donor tissue; 3. The risk of neuroma formation at the donor site; 4. Less than expected recovery of motor and sensory function at the host site.[29]

Alternative techniques involve the use of processed nerve allografts (PNA) and nerve conduits, which are non-neural, hollow, tubular interposition substitutes to bridge a nerve gap. This aims to bridge the peripheral nerve discontinuity to allow axonal regeneration and growth through the allograft towards the distal nerve.[29] PNAs consist of human nerves that are prepared with enzymatic decellularization, but with preservation of the epineurium and internal fascicular architecture. They are stored frozen until implantation and are available in a range of sizes. Immunosuppressive treatment is not required, and the typical length of an allograft implant is 1–3 cm. A number of single-armed studies involving this graft have demonstrated favourable recovery following its use, with results comparable to those published on nerve autograft reconstruction and with no adverse events described.[30–32] For gaps larger than 25 mm, a report from the RANGER study registry (an active database that collects data on the use and outcomes of PNAs using standardized collection methods) showed recovery in 86% of cases in terms of static two-point discrimination and Semmes-Weinstein monofilament testing with no adverse events.[33] Recent guidance issued by the UK National Institute for Health and Care Excellence (NICE), supports the use of PNAs for the reconstruction of digital nerve discontinuities (Figure 2a and 2b).

An ideal nerve conduit should be biocompatible, biodegradable, flexible, highly porous, compliant, neuroinductive and neuroconductive.[34] Types of conduit materials include biological (vein or rarely artery) or synthetic that can be absorbable or non-absorbable. Autologous venous nerve conduits have been shown to be comparable to nerve autografts for digital gaps $\leq$3 cm.[35] The vein harvested should be reversed and needs to be longer than the size of the defect, as 2 mm of the nerve ends are inserted into the vein lumen and sutured in place. There are concerns, however, that conduits made of biological tissue can lead to fibrosis, cell infiltration and loss of mechanical precision.[36]

Non-absorbable synthetic conduits, such as silicone tubes, have been used successfully.[37] There are concerns, however, that these materials can cause chronic inflammation which can prevent the passage of nutrients and cause the tube to collapse, leading to compression of the nerve.[38,39] Braga Silva et al. evaluated 26 patients with median and/or ulnar nerve injuries who were treated with silicone tubes. Although the outcome was good or very good in 73% of patients, the conduit had to be removed in 7 patients (27%) due to discomfort.[40]

Absorbable synthetic materials are biocompatible and reduce the chance of an immune response. The degradation rate and mechanical properties can also be controlled, creating an opportunity to individualise patient treatment,[36] although the published clinical evidence has not shown improved clinical outcomes compared with conventional methods. Currently available bio-absorbable synthetic conduits include collagen conduits, polyglycolic acid tubes (PGA) and Neurolac (poly DL-lactide-e-caprolactone) nerve guides. For collagen conduits, single-armed studies have demonstrated favourable results, particularly for defects smaller than 20 mm.[41–43] However, a prospective randomized study of collagen conduits versus conventional repair in 43 patients did not demonstrate a significant difference in outcome.[44] PGA tube reconstructions were analysed by Mackinnon, who found 86% or 15 cases achieved 'excellent' or 'good' outcomes based on static and moving two-point discrimination, but 27% of patients complained of significant pain.[45] Rinker et al. demonstrated no difference in outcome between PGA conduits versus vein grafts in a prospective randomized study, but the PGA group had two cases of extrusion.[46] The Neurolac nerve guide was compared to conventional microsuture repair in a randomized study. There was no

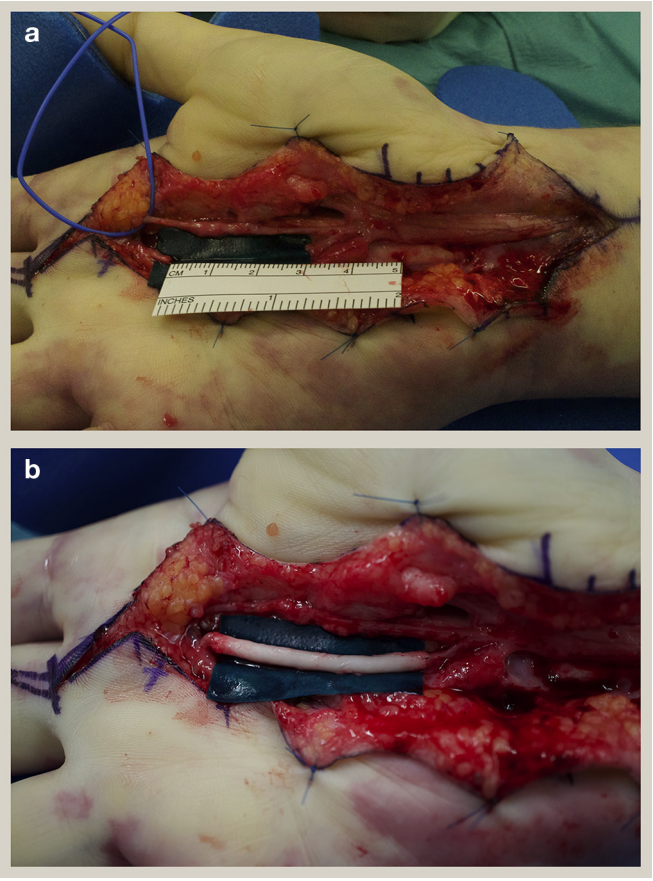

Figure 2 Clinical photographs of (a) a digital nerve defect of 3 cm and (b) its reconstruction with a processed nerve allograft.

difference in sensory recovery between the two groups and more complications were reported in the Neurolac nerve guide group.[47]

The repair of motor and mixed nerves presents additional challenges secondary to the need for precise identification of the fascicles. Motor pathways differ from sensory pathways and there is fundamental evidence that a pure sensory nerve graft is more effective in promoting sensory rather than motor axon regeneration.[48,49] Currently the best evidence for nerve conduits in mixed nerves comprise two prospective randomized controlled trials that evaluated nerve gaps less than 6 mm with diameters between 3.0 and 7.0 mm, both of which reported excellent results for median and ulnar nerves.[44,50] When evaluating PNAs for mixed and motor nerves, an industry-sponsored RANGER study showed meaningful recovery in 80% of such patients ($n = 16$),[51] although there are criticisms regarding the heterogeneity of patient demographics and outcome assessments.

Postoperative management

In our practice, following the repair of clean, uncomplicated digital nerve divisions, early active mobilization is encouraged. Prolonged immobilization may be detrimental and can lead to increased stiffness and cold intolerance.[52] Nerve repair at the wrist should be protected from excessive stretch with dorsal splinting for approximately 6 weeks, although the degree of immobilization is often dictated by associated flexor tendon injuries and their management. Gentle, active mobilization within the confines of the splint is commenced early to reduce the risk of adhesion formation.

Outcomes

There is no consensus on the standardized evaluation covering all types of nerve injuries, although in general a good to excellent result is indicated by the MRC grading of M3, S3 (muscle function against gravity, recovery of pain and touch sensibility with disappearance of over-response). A validated scoring system for median and ulnar nerve repairs at the wrist level does exist (i.e. the Rosen score) but is infrequently used.[53]

Post-traumatic nerve regeneration is a complex biological process where the outcome depends on multiple biological and environmental factors such as survival of nerve cells, axonal regeneration rate, extent of axonal misdirection, type of injury, type of nerve, level of the lesion, length of nerve defect (if present), age of the patient, timing of surgery, skill level of the surgeon and compliance to rehabilitation.[50] Better outcomes are associated with younger patients, a pure sensory nerve injury, and when the repair is performed early.

Summary

Management of nerve injuries of the hand and wrist demand a methodical approach. The decision to explore a hand wound is generally made based on clinical assessment. The gold standard to treat a divided nerve is a tension-free neurorrhaphy. Segmental nerve defects can be reconstructed with autologous nerve graft, conduits or PNAs, depending on the level and length of the defect. Rehabilitation with early mobilization and sensory re-education are vital to achieving an optimal outcome. ◆

REFERENCES

1 Asplund M, Nilsson M, Jacobsson A, Holst von H. Incidence of traumatic peripheral nerve injuries and amputations in Sweden between 1998 and 2006. *Neuroepidemiology* 2009; **32:** 217–28.
2 McAllister RM, Gilbert SE, Calder JS, Smith PJ. The epidemiology and management of upper limb peripheral nerve injuries in modern practice. *J Hand Surg Br* 1996; **21:** 4–13.
3 Strauch B, Lang A, Ferder M, Keyes-Ford M, Freeman K, Newstein D. The ten test. *Plast Reconstr Surg* 1997; **99:** 1074–8.
4 Strauch B, Lang A. The ten test revisited. *Plast Reconstr Surg* 2003; **112:** 593–4.
5 Patel MR, Bassini L. A comparison of five tests for determining hand sensibility. *J Reconstr Microsurg* 1999; **15:** 523–6.
6 Dellon AL, Mackinnon SE, Crosby PM. Reliability of two-point discrimination measurements. *J Hand Surg Am* 1987; **12:** 693–6.
7 Tindall A, Dawood R, Povlsen B. Case of the month: the skin wrinkle test: a simple nerve injury test for paediatric and uncooperative patients. *Emerg Med J* 2006; **23:** 883–6.
8 Seddon HJ. A classification of nerve injuries. *Br Med J* 1942; **2:** 237–9.
9 Seddon HJ. Three types of nerve injury. *Brain* 1943; **66:** 237–88.
10 Sunderland S. A classification of peripheral nerve injuries producing loss of function. *Brain* 1951; **74:** 491–516.
11 Mackinnon SE, Dellon AL. Surgery of the peripheral nerve. Thieme Medical Publishers, 1988.
12 Lundborg G. Nerve injury and repair. 2nd edn. Churchill Livingstone, 2005.

13 Birch R, Bonney G, Wynn Parry CB. Surgical disorders of the peripheral nerves. Edinburgh: Churchill-Livingstone, 1998.
14 Siemionow M, Brzezicki G. Chapter 8: Current techniques and concepts in peripheral nerve repair. *Int Rev Neurobiol* 2009; **87:** 141–72.
15 Mackinnon SE. New directions in peripheral nerve surgery. *Ann Plast Surg* 1989; **22:** 257–73.
16 Dvali L, Mackinnon S. Nerve repair, grafting, and nerve transfers. *Clin Plast Surg* 2003; **30:** 203–21.
17 Campbell WW. Evaluation and management of peripheral nerve injury. *Clin Neurophysiol* 2008; **119:** 1951–65.
18 Dahlin LB. Techniques of peripheral nerve repair. *Scand J Surg* 2008; **97:** 310–6.
19 Bernstein DT, Hamilton KL, Foy C, Petersen NJ, Netscher DT. Comparison of magnification in primary digital nerve repair: literature review, survey of practice trends, and assessment of 90 cadaveric repairs. *J Hand Surg Am* 2013; **38:** 2144–50.
20 Thomas PR, Saunders RJ, Means KR. Comparison of digital nerve sensory recovery after repair using loupe or operating microscope magnification. *J Hand Surg Br* 2015; **40:** 608–13.
21 Houschyar KS, Momeni A, Pyles MN, et al. The role of current techniques and concepts in peripheral nerve repair. *Plast Surg Int* 2016; 4175293.
22 Sameem M, Wood TJ, Bain JR. A systematic review on the use of fibrin glue for peripheral nerve repair. *Plast Reconstr Surg* 2011; **127:** 2381–90.
23 Dahlin LB, Wiberg M. Nerve injuries of the upper extremity and hand. *EFORT Open Rev* 2017; **2:** 158–70.
24 Birch R, Raji AR. Repair of median and ulnar nerves. Primary suture is best. *J Bone Joint Surg Br* 1991; **73:** 154–7.
25 Lundborg G, Rydevik B. Effects of stretching the tibial nerve of the rabbit. A preliminary study of the intraneural circulation and the barrier function of the perineurium. *J Bone Joint Surg Br* 1973; **55:** 390–401.
26 Trumble TE, Shon FG. The physiology of nerve transplantation. *Hand Clin* 2000; **16:** 105–22.
27 Slutsky D. A practical approach to nerve grafting in the upper extremity. *Atlas Hand Clin* 2005; **10:** 73–92.
28 Flannery OM, Roche SJ, Ng CY. (vi) Management of acute nerve injuries of the hand. *Orthop Traumatol* 2014; **28:** 230–5.
29 Braga Silva J, Marchese GM, Cauduro CG, Debiasi M. Nerve conduits for treating peripheral nerve injuries: a systematic literature review. *Hand Surg Rehabil* 2017; **36:** 71–85.
30 Taras JS, Amin N, Patel N, McCabe LA. Allograft reconstruction for digital nerve loss. *J Hand Surg Am* 2013; **38:** 1965–71.
31 Karabekmez FE, Duymaz A, Moran SL. Early clinical outcomes with the use of decellularized nerve allograft for repair of sensory defects within the hand. *Hand* 2009; **4:** 245–9.
32 Brooks DN, Weber RV, Chao JD, et al. Processed nerve allografts for peripheral nerve reconstruction: a multicenter study of utilization and outcomes in sensory, mixed, and motor nerve reconstructions. *Microsurgery* 2012; **32:** 1–14.
33 Rinker B, Zoldos J, Weber RV, et al. Use of processed nerve allografts to repair nerve injuries greater than 25 mm in the hand. *Ann Plast Surg* 2017; **78**(6S suppl 5)**:** S292–5.
34 Muheremu A, Ao Q. Past, present, and future of nerve conduits in the treatment of peripheral nerve injury. *Biomed Res Int* 2015; **2015:** 237507.
35 Chiu DT, Strauch B. A prospective clinical evaluation of autogenous vein grafts used as a nerve conduit for distal sensory nerve defects of 3 cm or less. *Plast Reconstr Surg* 1990; **86:** 928–34.
36 Biazar E, Khorasani MT, Montazeri N, et al. Types of neural guides and using nanotechnology for peripheral nerve reconstruction. *Int J Nanomed* 2010; **5:** 839–52.
37 Lundborg G, Rosén B, Dahlin L, Holmberg J, Rosén I. Tubular repair of the median or ulnar nerve in the human forearm: a 5-year follow-up. *J Hand Surg Br* 2004; **29:** 100–7.
38 Lohmeyer JA, Siemers F, Machens H-G, Mailänder P. The clinical use of artificial nerve conduits for digital nerve repair: a prospective cohort study and literature review. *J Reconstr Microsurg* 2009; **25:** 55–61.
39 Zhu Y, Wang A, Patel S, et al. Engineering bi-layer nanofibrous conduits for peripheral nerve regeneration. *Tissue Eng C Methods* 2011; **17:** 705–15.
40 Braga Silva J. The use of silicone tubing in the late repair of the median and ulnar nerves in the forearm. *J Hand Surg Br* 1999; **24:** 703–6.
41 Taras JS, Jacoby SM, Lincoski CJ. Reconstruction of digital nerves with collagen conduits. *J Hand Surg Am* 2011; **36:** 1441–6.
42 Bushnell BD, McWilliams AD, Whitener GB, Messer TM. Early clinical experience with collagen nerve tubes in digital nerve repair. *J Hand Surg Am* 2008; **33:** 1081–7.
43 Schmauss D, Finck T, Liodaki E, et al. Is nerve regeneration after reconstruction with collagen nerve conduits terminated after 12 months? the long-term follow-up of two prospective clinical studies. *J Reconstr Microsurg* 2014; **30:** 561–8.
44 Boeckstyns MEH, Sørensen AI, Viñeta JF, et al. Collagen conduit versus microsurgical neurorrhaphy: 2-year follow-up of a prospective, blinded clinical and electrophysiological multicenter randomized, controlled trial. *J Hand Surg Am* 2013; **38:** 2405–11.
45 Mackinnon SE, Dellon AL. Clinical nerve reconstruction with a bioabsorbable polyglycolic acid tube. *Plast Reconstr Surg* 1990; **85:** 419–24.
46 Rinker B, Liau JY. A prospective randomized study comparing woven polyglycolic acid and autogenous vein conduits for reconstruction of digital nerve gaps. *J Hand Surg Am* 2011; **36:** 775–81.
47 Bertleff MJOE, Meek MF, Nicolai J-PA. A prospective clinical evaluation of biodegradable neurolac nerve guides for sensory nerve repair in the hand. *J Hand Surg Am* 2005; **30:** 513–8.
48 Brushart TM. Preferential reinnervation of motor nerves by regenerating motor axons. *J Neurosci* 1988; **8:** 1026–31.
49 Brushart TME, Seiler WAIV. Selective reinnervation of distal motor stumps by peripheral motor axons. *Exp Neurol* 1987; **97:** 289–300.
50 Lundborg G, Rosén B. Hand function after nerve repair. *Acta Physiol (Oxf)* 2007; **189:** 207–17.
51 Cho MS, Rinker BD, Weber RV, et al. Functional outcome following nerve repair in the upper extremity using processed nerve allograft. *J Hand Surg Am* 2012; **37:** 2340–9.
52 Clare TD, de Haviland Mee S, Belcher HJCR. Rehabilitation of digital nerve repair: is splinting necessary? *J Hand Surg Br* 2004; **29:** 552–6.
53 Rosén B, Lundborg G. A model instrument for the documentation of outcome after nerve repair. *J Hand Surg Am* 2000; **25:** 535–43.