Treatment of Median Nerve Transection

18

Julie Balch Samora and Philip E. Blazar

Introduction

Upper limb peripheral nerve injury can have devastating effects on functional ability [1]. Etiologies of peripheral nerve injury can include penetrating laceration, crush, traction, ischemia, thermal necrosis, electric shock, radiation, and vibration [2, 3]. The first description of median nerve lesions was by Stopford in 1918 [4]. Upon evaluation of 1111 peripheral nerve injuries of the upper limb, there were 211 (19%) median nerve injuries [5]. Lacerations account for 30% of peripheral nerve injuries [2]. Nerve repair was reported as early as the seventh century, when Paul Aegina approximated cut nerve ends [6]. This chapter will discuss the neuropathology of median nerve injury, clinical examination, indications for surgery, surgical treatment options, rehabilitation, and outcomes after median nerve transection.

J.B. Samora
Nationwide Children's Hospital,
700 Children's Drive, Columbus, OH 43205, USA
e-mail: julie.samora@nationwidechildrens.org

P.E. Blazar (✉)
Brigham and Women's Hospital,
75 Francis Street, Boston, MA 02115, USA
e-mail: pblazar@partners.org

Neuropathology

In order to best treat peripheral nerve injuries, having an understanding of the basic anatomy is important. The individual myelinated axons and unmyelinated groups of axons are surrounded by endoneurium. Fascicles are collections of axons which are surrounded by perineurium. The internal epineurium lies between fascicles, and the external epineurium surrounds the nerve trunk. Whereas the endoneurium is longitudinally oriented, the epineurium and perineurium are circumferential [7] (see Fig. 18.1). The Seddon classification (1943) [8] includes neurapraxia, axonotmesis, and neurotmesis. This chapter will focus on the latter two. The axon is damaged or destroyed in axonotmesis, but the connective tissue is maintained. In neurotmesis, the nerve trunk is completely disrupted with no continuity and disrupted connective tissue.

Wallerian degeneration occurs with disruption of the axon [9], and repair and regeneration occur following nerve injury. With lesions involving fewer than 20–30% of the axons, recovery is mostly by collateral sprouting from surviving axons and occurs over 2–6 months [2]. When more than 90% of axons are injured, the primary mechanism of repair is regeneration from the injury site and depends largely on the age of the patient; distance from the injury site, but is also affected by the level of injury; and local biologic

S.F.M. Duncan, R. Kakinoki (eds.), *Carpal Tunnel Syndrome and Related Median Neuropathies*, DOI 10.1007/978-3-319-57010-5_18

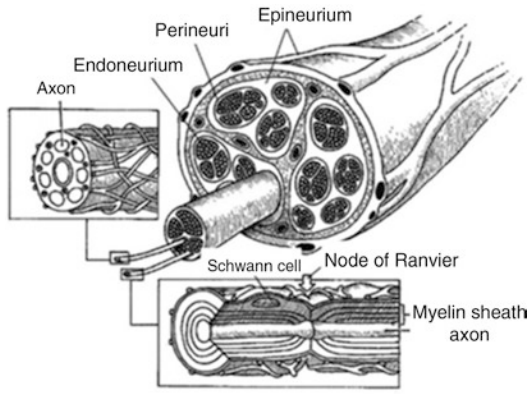

Fig. 18.1 Cross-sectional appearance of peripheral nerve. Obtained from: Biazar E, Khorasani MT, Montazeri N et al. Types of neural guides and using nanotechnology for peripheral nerve reconstruction. Int J Nanomedicine. 2010; 5: 839–852

factors. The neuron's capability to sustain regenerative attempts persists for at least 12 months after injury. Poor functional recovery occurs if the growth cone fails to reinnervate the motor end plate by 12 months due to secondary end-organ degeneration [10] leading to time dependence after injury for those injuries involving motor components.

Clinical Examination

Patients with median nerve injuries can present acutely or in delayed fashion. Generally, they are open injuries with sensory deficits involving the thumb, index, long, and radial half of the ring fingers. Partial injuries are more common than complete injuries, and lacerations at the wrist are more common than at the elbow. Depending on the level of injury, motor deficits can involve the pronator teres, flexor carpi radialis, palmaris longus, flexor pollicis longus, and flexor digitorum profundus muscles to the index and long fingers. If the entire nerve has been injured proximally, thenar muscle weakness will be observed [11]. A Tinel's sign at the site of nerve injury will develop, and distal propagation of the Tinel's is a good marker for axon regeneration after repair.

Indications for Surgery

When nerve continuity is uncertain, one can take an observatory approach to determine if there is either clinical or EMG evidence of reinnervation. The mechanism of injury (e.g., sharp vs. blunt) will help guide treatment. Axon regrowth from the proximal stump optimally occurs at 1 mm/day after about a 1-month delay [2]. Irreversible muscle atrophy occurs anywhere from 12 to 18 months. Schwann cells and endoneurial tubes remain viable for 18–24 months after injury. An advancing Tinel's sign can help clarify if reinnervation is occurring. If deficits persist past 3 months or if there is no evidence for reinnervation clinically, authors agree that there has typically been axonal damage [12]. Any patients without evidence of clinical recovery should undergo surgical exploration by 6 months, and some surgeons advocate for even earlier intervention for more proximal injuries [2].

Electrodiagnostic studies may provide information to help guide when to proceed with surgical exploration. The optimal timing is still debated. Although loss of amplitude of compound muscle action potential and nerve action potential is complete by 11 days after injury [2], electrical studies performed before 3 weeks after injury can be unreliable. Within the first week, electrodiagnostic tests can be useful for localization and determining complete from incomplete injuries. At 1–2 weeks, they can help distinguish axonotmesis or neurotmesis from neurapraxia. At 3–4 weeks, after fibrillation potentials have had a chance to develop, this provides the most information from a single study. At 3–4 months, they may provide information regarding reinnervation [2].

Partial Nerve Lacerations

There are times when the nerve is not entirely transected and there is no universal agreement on management of these lesions. Options include conservative management, nerve grafting, and repair of the lacerated fascicular groups only. Depending on the severity of the lesion, the

deficit, and the aforementioned factors affecting recovery, treatment is individualized. Early end-to-end microsurgical repair of lacerated fascicular groups has been shown to result in good motor and sensory outcomes (BMRC 4 and 3.81, respectively) [13].

Surgical Options

The principal surgical options for complete lacerations include neurolysis, primary end-to-end repair, nerve grafting, and nerve transfer [14]. Primary nerve repair involves direct end-to-end suture of separated nerve ends [15] and has the best prognosis [16] (see Fig. 18.2). The indications for primary repair include the ability to directly approximate nerve ends without undue tension in a nerve that has not sustained either a crush injury or mechanical disruption. If there is tension on the repair, ischemia will occur, leading to dysfunction. If nerves are stretched by 8–10%, blood flow is reduced by half [17]. A basic clinical tenet is that if one cannot coapt two nerve ends with a single 9-0 nylon suture, there is too much tension [18]. However, there is little clinical literature supporting this assertion.

Tissue approximation and alignment will be easier with earlier repair [19], with most surgeons preferring to operate before 2 weeks. Furthermore, with earlier repair, there is improved neuron survival [20] and decreased fibrosis of the distal stump [21] (see Fig. 18.3). The definition of secondary repair is end-to-end suture 2–3 weeks after injury. If a tension-free repair cannot be achieved, primary repair should be abandoned for another method.

Neurorrhaphy can be performed with sutures, fibrin glue, or nerve tubes. One must dissect to scar-free, healthy appearing tissue. The injured portion of the nerve must be removed to expose healthy nerve with a visible fascicular pattern. It is of paramount importance to align the proximal and distal stumps. One must utilize both surface landmarks and other indicators to properly align the nerve fascicles. Recommendations for alignment include visualizing fascicular patterns, using surface vessels as markers and any obliquity of the injury. Some authors have reported on using histologic acetylcholinesterase staining to identify motor axons and carbonic anhydrase staining to identify sensory fibers [22], but this adds a great deal of time to the operative procedure. Others have advocated "awake" electrical nerve stimulation to identify motor fibers [23]. Neither of these methods has resulted in significantly improved results.

Ends should be lined up such that the fascicular groups are gently touching. One may place sutures in the epineurium, which is less traumatic but may not adequately approximate the deep fascicles. A fascicular repair involves dissecting the epineurium and suturing the perineurium of each fascicle. Grouped fascicular repair minimizes nerve trauma and allows for improved alignment. There

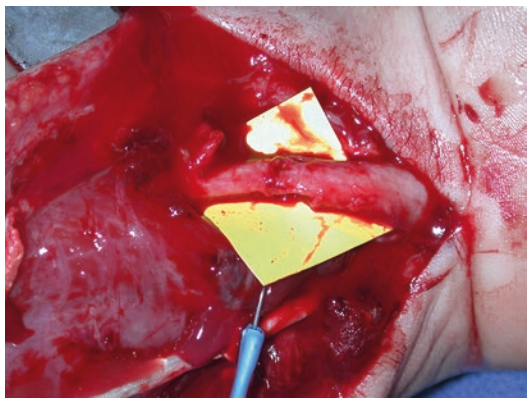

Fig. 18.2 Epineural repair of median nerve in the setting of a spaghetti wrist

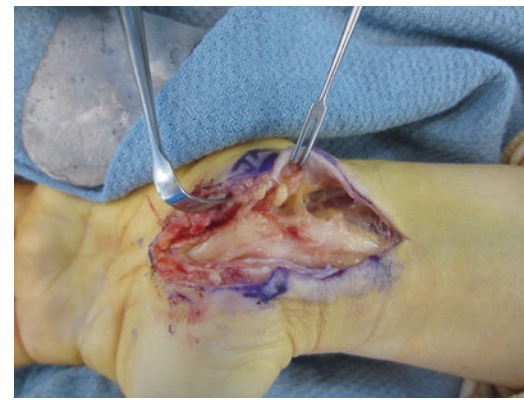

Fig. 18.3 This patient had a median nerve laceration and was taken to surgery at 2 weeks post-injury. Scar has already formed

have been no studies documenting superiority of one technique over another [24].

Fibrin-based tissue glue is becoming more popular for coaptation of the nerve ends. Its advantages include efficiency, simplicity, minimal trauma, and creation of a barrier to invading scar tissue [25]. Furthermore, there is animal data suggesting fibrin glue can promote angiogenesis, stimulate chemotaxis and leukocytosis, enhance macrophage proliferation, and provide hemostasis [6, 26, 27]. Fibrin glue does not appear to be a barrier to regeneration [28], but may have inferior holding strength [29, 30].

In secondary reconstruction with retraction of nerve endings and a large gap, end-to-end neurorrhaphy is no longer an option. Nerve diameter, gap size, and quality of the injured nerves influence the decision-making process. When a tension-free neurorrhaphy is not feasible, nerve grafting is typically the first option. Nerve graft can be autogenous (e.g., sural) or allograft (e.g., cadaver). The gold standard for a long gap is autograft. A variety of options exist, although the most commonly utilized donor is the sural nerve. Advantages of using sural nerve include a lack of motor deficit, a fairly superficial dissection to harvest, and a relatively lengthy course of the nerve with limited branches. Disadvantages of using autograft include limited availability, sensory donor nerves instead of mixed nerves as options, the obligate loss of nerve function, scarring, and painful neuroma formation [31].

Dissection, scar removal, neuroma resection, and management of median nerve injuries with a gap rest on the knowledge of interfascicular relationships and nerve architecture. It is important to align the median nerve and place the autograft accordingly in as near anatomic position as possible. Authors have previously identified the internal topography of the median nerve [32, 33]. In the upper two thirds of the forearm, the motor branches to the extrinsic muscles lie about the periphery, on the radial and ulnar aspect [32]. The sensory branches to the hand, the thenar motor branches, and the palmar cutaneous branch are in the central and dorsal quadrant. In the distal third, the thenar motor, lumbrical and sensory components are segregated and can be isolated.

Processed nerve allografts provide decellularized and predegenerated human nerve tissues which maintain the microarchitecture including the epineurium, fascicles, endoneurial tubes, and microvasculature [34, 35]. Processed nerve allograft may provide a viable option for mixed nerves and has the advantage of avoiding donor morbidity and decreasing surgical time [34]. The thickness and length of the processed autograft are also variable, and they are available in a range of sizes. Furthermore, they can be easily obtained. It is unclear, however, if there is a limit in the efficacy based on the length of the gap.

Nerve substitutes can include vein grafts, synthetic nerve conduits, and Schwann cell-lined nerve conduits [35, 36]. Nerve conduits are also an option for small gaps or partial lacerations. Lundborg and Hansson originally presented the concept of nerve entubulation in 1980 [37]. Conduits available include those made of collagen, polyglycolic acid (PGA), and polycaprolactone. In 1997, Lundborg et al. [38] performed a prospective, randomized clinical study comparing conventional microsurgical repair of median and ulnar nerves to using silicone tubes, with gaps measuring 3–4 mm between nerve ends. They found no difference in sensory or motor function between the two groups. Lundborg et al. [39] demonstrated that the median nerve can regenerate across 5 mm gaps equivalently to direct repair. However, a recent report was published describing four cases of failed conduit-based major nerve reconstructions [40].

A combination approach can also be made. For example, there is one case report describing reconstruction of a 4 cm median nerve graft with a piece of autogenous median nerve placed in a bioabsorbable conduit [41]. At 2 years after surgery, the patient had 7 mm moving and static two-point discrimination to the thumb and had recovered palmar abduction and EMG evidence of reinnervation of the abductor pollicis brevis.

It is beyond the scope of this chapter, but branches of the radial or ulnar nerve have been transferred to median nerve branches in the forearm, hand, or even digits [42–44]. Nerve transfers utilize intact motor nerves with a minor function to reinnervate critical muscles. One can

join the distal end of the cut normal nerve with the distal stump of the injured nerve. Another option is to perform neurorrhaphy of selected fascicles from a normal nerve to an injured nerve. End to side involves taking the end of a healthy donor nerve to the side of a target nerve distal to the site of injury [45].

Rehabilitation

There is a lack of consensus on postoperative immobilization and rehabilitation after median nerve repair or grafting. The rehabilitation decision is generally patient and surgeon dependent. Tactile gnosis is generally not regained in adult patients after injury to a major nerve trunk [24, 46]. Sensory reeducation has been proposed and combines techniques to help patients with sensory impairment to learn to interpret the altered neural impulses by attempting to reprogram the brain [46, 47]. In 40 patients with low median nerve complete transection and repair, 20 were rehabilitated with a sensory reeducation program, and 20 had no further treatment than the initial therapy. In the first group, locognosia (ability to localize touch) was significantly improved compared to group B, but static and moving two-point discrimination was not different [48]. In a systematic review evaluating the effects of sensory reeducation programs on functional hand sensibility after median and ulnar nerve repair, there was limited evidence to support the use of early or late sensory reeducation programs [49].

Outcomes

The best-known scale for sensibility and motor grading is the British Medical Research Council (MRC) scale, which is the most widely accepted classification system to score outcome of peripheral nerve injuries [50–52]. Functional outcomes have been assessed successfully using the DASH including the functional symptom score, with strong relation found with motor and sensory recovery [53]. DASH score, Rosen score, and Highet score were found to correlate significantly when evaluating outcomes of median and ulnar nerve injuries [54]. Studies have not universally utilized the same outcome assessments, making comparisons difficult.

There are multiple factors that influence recovery, such as cooperative and motivation of the patient, hand therapy, cognitive capacity, psychological stress due to the trauma, and comorbidities such as diabetes and alcoholism [55]. Age, gap length, and delay to surgery greatly influence outcome after repair of median and ulnar nerve transection injuries [55, 56]. Time of improvement can be variable as well, with one study indicating that grip and tip-pinch strength improve over a period of 3 years following median or ulnar nerve lesions [57]. Return to work after isolated median nerve injuries is influenced by level of education, type of job, and compliance with hand therapy, with 80% of workers returning to work within 1 year [58].

Primary repair produces superior results compared to those of delayed repair [59, 60]. In a series of 2181 acute nerve injuries, a primary repair was achieved in 87% of the cases with end-to-end approximation [18]. In median nerve injuries at the wrist, protective sensation in the fingertips can be reliably restored by direct suture or nerve grafting [55]. However, more proximal injuries have had less sensory recovery due to the long distance between the site of injury and the target cutaneous receptors in the fingertips.

In a long-term outcome study, 71 median and ulnar lesions were assessed 8 years after microsurgical repair and were classified according to the DASH, the Rosen's hand protocol, and the Highet scale. Patients regained approximately 70% of their original hand function [54]. Satisfactory motor (M4/M5) and sensory (S3+/4) recovery occurred in more than 50% of patients if the delay in repair was less than 3 months and the gap was less than 6 cm [56]. In 28 patients undergoing primary repair of a sharp transection of the median nerve at the wrist, S4 was elicited in 36%, S3+ in 29%, and S3 in 14%. This study demonstrated a significant correlation between age and functional sensibility [61].

Birch and Raji [59] reported on repair of 108 median and ulnar nerves after a clean laceration in the forearm with 48 undergoing primary repairs. None of these repairs failed, which they defined as motor grade of 3 or less, trophic changes, lack of sweating, no sensation or the presence of severe cold sensitivity, and general hypersensitivity. On the other hand, Mailander et al. [62] reported on ten median nerve repairs at the wrist with only 40% achieving S4, whereas 90% achieved at least an M4. Puckett and Meyer [63] reported on a series of 38 volar wrist lacerations involving either or both of the median and ulnar nerves (age range 1–61 years). Only 19 of 37 (51%) patients regained moving two-point discrimination better than 12 mm.

Hudson and de Jager [64] reported on 15 patients with spaghetti wrist who underwent primary repair of median and ulnar nerves at the wrist and found better functional results for median repairs compared to ulnar nerve repairs. Two children recovered 2PD of less than 10 mm with another two patients achieving 2PD between 10 and 15 mm. Hudson et al. [65] evaluated 18 children who underwent primary epineurial repair of median nerve lacerations and found mean static 2PD was 5 mm and motor strength of opponens pollicis was 4.5 on the MRC scale. Distal injuries fared better than proximal injuries.

In autograft repair of median nerve injuries, meaningful recovery was observed in 67% of patients [66]. In a functional outcome study following nerve repair using processed nerve allograft, there were no adverse events, and overall meaningful recovery was found in 75% of median nerve repairs [34].

In a study using tube conduits for mixed nerve injuries, functional recovery in gaps between 2 and 25 mm was only obtained in 1 of the 12 patients [67]. In contrast, Ruijs et al. [55] reported a 52% success rate in motor outcomes from mixed nerve repairs using conduits. Dienstknecht et al. [68] found purified type 1 bovine collagen conduits to be a good option for median nerve injury in the distal forearm in nerve gaps ranging from 1 to 2 cm. They found static two-point discrimination to be less than 6 mm in three patients, between 6 and 10 mm in four patients, and over 10 mm in two patients.

A prospective multicenter registry of peripheral nerve injuries associated with orthopedic trauma has been established and may lead to prospective studies to better evaluate outcomes following repair and reconstruction [69].

Conclusions

Median nerve injuries can lead to devastating consequences, and unfortunately the results frequently lead to some loss of function despite surgical repair. Early diagnosis and treatment is of paramount importance. Primary repair of median nerve injury consistently leads to best outcomes. Alternative treatments include autograft, allograft, and nerve conduits, particularly when a primary repair would require undue tension. In select injuries, nerve transfers are viable options.

References

1. Novak CB, Anastakis DJ, Beaton DE, et al. Patient-reported outcome after peripheral nerve injury. J Hand Surg [Am]. 2009;34:281–7.
2. Campbell WW. Evaluation and management of peripheral nerve injury. Clin Neurophysiol. 2008;119(9):1951–65.
3. Robinson LR. Traumatic injury to peripheral nerves. Suppl Clin Neurophysiol. 2004;57:173–86.
4. Stopford JS. The variation in distribution of the cutaneous nerves of the hand and digits. J Anat. 1918;53:14–25.
5. McAllister RM, Gilbert SE, Calder JS, et al. The epidemiology and management of upper limb peripheral nerve injuries in modern practice. J Hand Surg. 1996;21B:4–13.
6. Lin MY, Manzano G, Gupta R. Nerve allografts and conduits in peripheral nerve repair. Hand Clin. 2013;20(3):331–48.
7. Sunderland S. The anatomy and physiology of nerve injury. Muscle Nerve. 1990;13:771–84.
8. Seddon H. Three types of nerve injury. Brain. 1943;66:237–88.
9. Koeppen AH. Wallerian degeneration: history and clinical significance. J Neurol Sci. 2004;220:115–7.
10. Konofaos P, Ver Halen JP. Nerve repair by means of tubulization: past, present, future. J Reconstr Microsurg. 2013;29(3):149–64.

11. Pederson WC. Median nerve injury and repair. J Hand Surg Am. 2014;39(6):1216–22.
12. Mackinnon SE, Dellon AL. Surgery of the peripheral nerve. New York: Thieme; 1988.
13. Hurst LC, Dowd A, Sampson SP, et al. Partial lacerations of median and ulnar nerves. J Hand Surg. 1991;16(2):207–10.
14. Spinner RJ, Kline DG. Surgery for peripheral nerve and brachial plexus injuries or other nerve lesions. Muscle Nerve. 2000;23:680–95.
15. Allan CH. Primary nerve repair: indications and results. J Hand Surg. 2004;4(3):195–9.
16. Diao E, Vannuyen T. Techniques for primary nerve repair. Hand Clin. 2000;16:53–66. viii
17. Driscoll PJ, Glasby MA, Lawson GM. An in vivo study of peripheral nerves in continuity: biomechanical and physiological responses to elongation. J Orthop Res. 2002;20:370–5.
18. de Medinaceli L, Prayon M, Merle M. Percentage of nerve injuries in which primary repair can be achieved by end-to-end approximation: review of 2,181 nerve lesions. Microsurgery. 1993;14:244–6.
19. Dahlin LB. Techniques of peripheral nerve repair. Scand J Surg. 2008;97:310–6.
20. Ma J, Novikov LN, Kellerth JO, et al. Early nerve repair after injury to the postganglionic plexus: an experimental study of sensory and motor neuronal survival in adult rats. Scand J Plast Reconstr Surg Hand Surg. 2003;37:1–9.
21. Lundborg G, Rosén B. Hand function after nerve repair. Acta Physiol (Oxford). 2007;189:207–17.
22. He YS, Zhong SZ. Acetylcholinesterase: a histochemical identification of motor and sensory fascicles in human peripheral nerve and its use during operation. Plast Reconstr Surg. 1988;82:125–32.
23. Al-Qattan MM. Refinements in the technique of 'awake' electrical nerve stimulation in the management of chronic low ulnar nerve injuries. Injury. 2004; 35:1110–5.
24. Lundborg G. A 25-year perspective of peripheral nerve surgery: evolving neuroscientific concepts and clinical significance. J Hand Surg. 2000;25(3):391–414.
25. Isaacs J. Treatment of acute peripheral nerve injuries: current concepts. J Hand Surg. 2010;35(3):491–7.
26. Hall H. Modified fibrin hydrogel matrices: both 3D-scaffolds and local and controlled release systems to stimulate angiogenesis. Curr Pharm Des. 2007; 13(35):3597–607.
27. Sameem M, Wood TJ, Bain JR. A systematic review on the use of fibrin glue for peripheral nerve repair. Plast Reconstr Surg. 2011;127(6):2381–90.
28. Palazzi S, Vila-Torres J, Lorenzo JC. Fibrin glue is a sealant and not a nerve barrier. J Reconstr Microsurg. 1995;11:135–9.
29. Cruz NI, Debs N, Fiol RE. Evaluation of fibrin glue in rat sciatic nerve repairs. Plast Reconstr Surg. 1986;78: 369–73.
30. Nishimura MT, Mazzer N, Barbieri CH, et al. Mechanical resistance of peripheral nerve repair with biological glue and with conventional suture at different postoperative times. J Reconstr Microsurg. 2008;24(5):327–32.
31. Taras JS, Jacoby SM, Lincoski CJ. Reconstruction of digital nerves with collagen conduits. J Hand Surg [Am]. 2011;36(9):1441–6.
32. Jabaley ME, Wallace WH, Heckler FR. Internal topography of major nerves of the forearm and hand: a current view. J Hand Surg. 1980;5(1):1–18.
33. Williams HB, Jabaley ME. The importance of internal anatomy of the peripheral nerves to nerve repair in the forearm and hand. Hand Clin. 1986;2(4):689–707.
34. Cho MS, Rinker BD, Weber RV, et al. Functional outcome following nerve repair in the upper extremity using processed nerve allograft. Journal of Hand Surgery. 2012;37(11):2340–9.
35. Mackinnon SE, Doolabh VB, Novak CB, et al. Clinical outcome following nerve allograft transplantation. Plast Reconstr Surg. 2001;107:1419–29.
36. Weber RV, Mackinnon SE. Bridging the neural gap. Clin Plast Surg. 2005;32:605–16.
37. Lundborg G, Hansson HA. Nerve regeneration through preformed pseudosynovial tubes: a preliminary report of a new experimental model for studying the regeneration and reorganization capacity of peripheral nerve tissue. J Hand Surg [Am]. 1980;5(1): 35–8.
38. Lundborg G, Rosén B, Dahlin L, et al. Tubular versus conventional repair of median and ulnar nerves in the human forearm: early results from a prospective, randomized, clinical study. J Hand Surg. 1997;22(1): 99–106.
39. Lundborg G, Rosén B, Dahlin L, et al. Tubular repair of the median or ulnar nerve in the human forearm: a 5-year follow-up. J Hand Surg Br. 2004;29(2): 100–7.
40. Moore AM, Kasukurthi R, Magill CK, et al. Limitations of conduits in peripheral nerve repairs. Hand. 2009;4(2):180–6.
41. Hung V, Dellon AL. Reconstruction of a 4-cm human median nerve gap by including an autogenous nerve slice in a bioabsorbable nerve conduit: case report. J Hand Surg. 2008;33(3):313–5.
42. Brunelli GA. Sensory nerve transfers. J Hand Surg. 2004;29B:557–62.
43. Tung TH, Mackinnon SE. Nerve transfers: indications, techniques, and outcomes. J Hand Surg. 2010; 35A:332–41.
44. Bertelli JA, Ghizoni MF. Very distal sensory nerve transfers in high median nerve lesions. J Hand Surg Am. 2011 Mar;36(3):387–93.
45. Matsuyama T, Macky M, Midha R. Peripheral nerve repair and grafting techniques: a review. Neurol Med Chir (Tokyo). 2000;40:187–99.
46. Dellon AL, Jabaley ME. Reeducation of sensation in the hand following nerve suture. Clin Orthop. 1982;163:75–9.
47. Wynn Parry CB, Salter M. Sensory reeducation after median nerve lesion. Hand. 1976;8:50–257.

48. Mavrogenis AF, Spyridonos SG, Antonopoulos D, et al. Effect of sensory re-education after low median nerve complete transection and repair. J Hand Surg. 2009;34(7):1210–5.
49. Miller LK, Chester R, Jerosch-Herold C. Effects of sensory reeducation programs on functional hand sensibility after median and ulnar repair: a systematic review. J Hand Ther. 2012;25(3):297–306.
50. Allan HA. Functional results of primary nerve repair. Hand Clin. 2000;16:67.
51. Dellon AL, Curtis R, Edgerton M. Reeducation of sensation in the hand after nerve injury and repair. Plast Reconstr Surg. 1974;53:297–305.
52. Seddon HJ. Peripheral nerve injuries. Medical research council special report no. 282. London: Her Majesty's Stationary Office;1954
53. Jaquet JB, Kuypers P, Kalmijn S, et al. The DASH: a useful assessment method to evaluate functional recovery following median and ulnar nerve injuries. J Hand Surg. 2003;28:16.
54. Vordemvenne T, Langer M, Ochman S, et al. Long-term results after primary microsurgical repair of ulnar and median nerve injuries. A comparison of common score systems. Clin Neurol Neurosurg. 2007;109:263–71.
55. Ruijs A, Jaquet J, Kalmijn S, et al. Median and ulnar nerve injuries: a meta-analysis of predictors of motor and sensory recovery after modern microsurgical nerve repair. Plast Reconstr Surg. 2005;116(2):484–94.
56. Ruijs A, Jaquet J, Giele H, et al. Meta-analysis: repair of median and ulnar nerve transection injuries. J Hand Surg. 2003;28:16.
57. Ghandi A, Jaquet JB, Kalmijn S, et al. Motor recovery following median and ulnar nerve injuries. Journal of Hand Surgery. 2003;28:16.
58. Bruyns CNP, Jaquet JB, Schreuders TAR, et al. Predictors for return to work in patients with median and ulnar nerve injuries. J Hand Surg. 2003;28A:28–34.
59. Birch R, Raji AR. Repair of median and ulnar nerves. Primary suture is best. J Bone Joint Surg. 1991;73B:154–7.
60. Merle M, Amend P, Cour C, et al. Microsurgical repair of peripheral nerve lesions. Periph Nerve Repair Regen. 1986;2:17–26.
61. Selma P, Emre O, Oguz P, et al. Evaluation of the improvement of sensibility after primary median nerve repair at the wrist. Microsurgery. 1998;18:192–6.
62. Mailander P, Berger A, Schaller E, et al. Results of primary nerve repair in the upper extremity. Microsurgery. 1989;10:147–50.
63. Puckett CL, Meyer VH. Results of treatment of extensive volar wrist lacerations: the spaghetti wrist. Plast Reconstr Surg. 1985;75:714–21.
64. Hudson DA, de Jager LT. The spaghetti wrist. Simultaneous laceration of the median and ulnar nerves with flexor tendons at the wrist. J Hand Surg. 1993;18B:171–3.
65. Hudson DA, Bolitho DG, Hodgetts K. Primary epineural repair of the median nerve in children. J Hand Surg. 1997;22B:54–6.
66. Kim DH, Kam AC, Chandika P, et al. Surgical management and outcomes in patients with median nerve lesions. J Neurosurg. 2001;95(4):584–94.
67. Chiriac S, Facca S, Diaconu M, et al. Experience of using the bioresorbable copolyester poly(DL-lactide-E-caprolactone) nerve conduit guide Neurolac for nerve repair in peripheral nerve defects: report on a series of 28 lesions. J Hand Surg. 2012;37B:342–9.
68. Dienstknecht T, Klein S, Vykoukal J, et al. Type I collagen nerve conduits for median nerve repairs in the forearm. Journal of Hand Surgery. 2013;38(6):1119–24.
69. Shores JT, Gaston GR, Reider L, et al. A prospective multicenter registry of peripheral nerve injuries associated with upper and lower extremity orthopedic trauma. Clinical Paper 80, session 15, ASSH meeting, 2014