Dual reinnervation of biceps muscle after side-to-side anastomosis of an intact median nerve and a damaged musculocutaneous nerve

Case report

Tene A. Cage, M.D., Neil G. Simon, M.B.B.S., Suzanne Bourque, M.D., Roger Noss, Ph.D., D.ABNM, John W. Engstrom, M.D., Jeffrey W. Ralph, M.D., and Michel Kliot, M.D.

Departments of Neurological Surgery and Neurology, University of California, San Francisco, California

Traumatic peripheral nerve injury can lead to significant long-term disability for previously healthy persons. Damaged nerve trunks have been traditionally repaired using cable grafts, but nerve transfer or neurotization procedures have become increasingly popular because the axonal regrowth distances are much shorter. These techniques sacrifice the existing nerve pathway, so muscle reinnervation depends entirely on the success of the repair. Providing a supplemental source of axons from an adjacent intact nerve by using side-to-side anastomosis might reinnervate the target muscle without compromising the function of the donor nerve.

The authors report a case of biceps muscle reinnervation after side-to-side anastomosis of an intact median nerve to a damaged musculocutaneous nerve. The patient was a 34-year-old man who had sustained traumatic injury primarily to the right upper and middle trunks of the brachial plexus. At 9 months after the injury, because of persistent weakness, the severely damaged upper trunk of the brachial plexus was repaired with an end-to-end graft. When 8 months later biceps function had not recovered, the patient underwent side-to-side anastomosis of the intact median nerve to the adjacent distal musculocutaneous nerve via epineural windows. By 9 months after the second surgery, biceps muscle function had returned clinically and electrophysiologically. Postoperative electromyographic and nerve conduction studies confirmed that the biceps muscle was being reinnervated partly by donor axons from the healthy median nerve and partly by the recovering musculocutaneous nerve.

This case demonstrates that side-to-side anastomosis of an intact median to an injured musculocutaneous nerve can provide dual reinnervation of the biceps muscle while minimizing injury to both donor and recipient nerves.

Key Words • nerve injury • reinnervation • side-to-side anastomosis • electromyography • peripheral nerve

Traumatic injury involving peripheral nerves can lead to substantial impairment of one or more muscle groups, thereby leaving an otherwise healthy person with a significant disability. For these patients, preoperative studies, including electromyography, nerve conduction studies, and magnetic resonance neurography, can help to characterize the location and extent of nerve injury. For severe peripheral nerve injuries that directly damage axons, regeneration is required for the recovery of function. For axonotmetic injuries, no direct surgical repair of the nerve is required because the pathways allowing for nerve regeneration remain intact. However, for neurotmetic injuries in continuity, resection of intraneural scar tissue is required, followed by direct surgical repair of the 2 ends of the nerve, either with primary suture repair or with the aid of a nerve graft or synthetic tube graft. For some cases, it is difficult to distinguish axonotmetic and neurotmetic grades of nerve injury preoperatively or even intraoperatively. For such cases, it would be advantageous to construct a nerve repair that allows for possible axon regeneration along the original nerve pathway while also providing a supplemental source of axons from an adjacent intact nerve without compromising its function. We describe one such construct: a side-to-side anastomosis between an intact median nerve and the adjacent severely damaged musculocutaneous nerve via epineural windows and primary suture repair. Postoperative clinical and electrophysiological evidence showed successful dual reinnervation of the target biceps muscle by axons from both nerves.
Case Report

History and Examination. The patient was a 34-year-old man who had been involved in a motorcycle accident resulting in a right brachial plexus injury that most severely affected the upper and middle trunks. Immediately after the accident, the patient had no motor function of the right suprascapularis, infraspinatus, deltoid, biceps, triceps, finger extensor, and wrist extensor muscles. He had decreased strength in the finger flexor, abductor, and adductor muscles and decreased sensation to light touch over the right thumb and shoulder. Electromyography confirmed injury to the right upper and middle trunks and relative sparing of the lower trunk.

The patient subsequently underwent 2 surgical brachial plexus repairs: the first, 9 months after the initial injury; and the second, described here, 17 months after the accident. During the first repair, extensive scarring of a segment of the upper trunk was found. Intraoperative neurophysiological monitoring revealed no motor response to direct stimulation of the upper trunk. However, somatosensory evoked potential signals recorded from the scalp were present with upper trunk stimulation. Therefore, the scarred section of the upper trunk was excised and the ends were cut back to expose viable nerve fascicles on either side. The cut ends were then repaired using a 7-mm-diameter, 2-cm-long, synthetic NeuraGen (Integra LifeSciences Corp.) interposition tube graft in an end-to-end repair.

By 8 months postoperatively (17 months postinjury), strength had improved in lower trunk–supplied muscles in the right forearm and hand, and electromyography indicated reinnervation of the suprascapularis muscle on the right. However, clinical examination and electromyography showed no evidence of reinnervation of the biceps, triceps, or deltoid muscles. Because of the patient’s persistent disability, a second brachial plexus exploration was undertaken with the aim of returning function to the biceps muscle.

Operation. Preoperative baseline motor evoked potentials were not recordable from the right biceps, triceps, or deltoid muscles, and baseline somatosensory evoked potentials elicited positive responses from the ulnar and median nerves on the right. The brachial plexus, including the musculocutaneous nerve providing motor supply to the biceps muscle, was identified. Although the musculocutaneous nerve was found to be in continuity, it was encompassed by thick, scarred adhesions. We performed a circumferential neurolysis, which enabled us to electrically interrogate the nerve (Fig. 1B and C). Direct electrical stimulation of the nerve yielded no motor response in the biceps muscle. However, transcranial stimulation of the motor cortex yielded positive compound nerve action potentials along the proximal, but not the distal, musculocutaneous nerve, suggesting the presence of regenerating axons that had not yet traversed the full length of the nerve to reinnervate the biceps muscle. Electrical stimulation of the median nerve yielded normal motor responses in the appropriate muscles of the forearm and hand.

Given these intraoperative findings, we left the musculocutaneous nerve intact while providing it with a potential source of supplemental axons from the adjacent functioning median nerve. We approximated the musculocutaneous nerve and median nerves in parallel at a site where focal stimulation of the median nerve resulted in vigorous contraction of the flexor carpi radialis muscle. A small (1–2 mm) epineural window was cut along the medial aspect of the musculocutaneous nerve and the lateral aspect of the median nerve at the same level. The epineural sheaths were then approximated and sutured to each other to create a side-to-side anastomosis between the musculocutaneous and median nerves (Fig. 1D). The nerves were sutured together with two 7-0 monofil monocryst interrupt suture, wrapped in Surgicel (Ethicon Inc.), and then covered with Tisseel fibrin glue (Baxter BioSurgery; Fig. 1E and F). When the anastomosis was completed, signals through the median nerve were confirmed to be at baseline.

Postoperative Course. The patient returned for follow-up electromyography and nerve conduction testing at 4 and 9 months after the second surgery. At the 4-month visit, clinical testing revealed no voluntary contraction of the right biceps muscle but full strength for wrist flexion, finger flexors, thumb abduction, and the opponens pollicis muscle. Electromyography of the biceps muscle identified sparse motor units, which were maximally activated during flexion of the arm at the elbow combined with flexion of the wrist. No acute denervation was found in muscles supplied by the median nerve.

At the 9-month visit, clinical examination demonstrated Grade 1/5 power in the biceps muscle, which increased to 3/5 with concomitant pronation of the forearm (as rated on the Medical Research Council scale). Electrodiagnostic studies were performed, recording from a concentric needle electromyography electrode in the biceps muscle with stimulation of the median nerve at the elbow and wrist (Fig. 2A). Late responses to stimulation at both sites (Fig. 2B) suggested functional communication between the median and musculocutaneous nerves. Electromyography used 2-channel recording with needle electrodes placed in the biceps and flexor carpi radialis muscles. Sparse motor units in the biceps muscle were identified when the patient was asked to flex the elbow alone (Fig. 2C) or flex the wrist alone (Fig. 2D). When the patient was asked to flex the wrist and elbow together, the density of motor units in the biceps muscle increased significantly (Fig. 2E). These electrodiagnostic results demonstrated dual reinnervation of the biceps muscle through both the musculocutaneous nerve and the side-to-side anastomosis of the median and musculocutaneous nerves.

Discussion

Side-to-side anastomosis between a healthy median nerve and a severely injured musculocutaneous nerve demonstrated meaningful improvement of function. Neurophysiological studies identified a dual source of axons reinnervating the biceps muscle. Reinnervation was indicated by 1) regenerating axons traversing the original musculocutaneous nerve pathway that were activated when the patient voluntarily tried to flex the elbow, and 2) axons...
Muscle reinnervation after side-to-side nerve repair

that sprouted from the adjacent median nerve and regenerated across the side-to-side anastomotic site into the distal musculocutaneous nerve to innervate the biceps muscle and produce contraction when the patient voluntarily contracted muscles supplied by the median nerve. This observation is promising because side-to-side nerve anastomosis has not routinely been used as a surgical technique; the case reported here suggests that this procedure is useful as an adjunct to the surgical treatment of severe traumatic brachial plexus injuries.

Electromyography conducted before the second surgery confirmed an absence of motor unit action potentials in the right biceps, triceps, and deltoid muscles despite the use of an interposition graft 8 months earlier to repair the upper trunk. However, electromyography and nerve conduction velocity testing conducted after side-to-side anastomosis of the median and musculocutaneous nerves indicated dual reinnervation of the biceps muscle. Postoperative electromyography confirmed that elbow and wrist flexion produced motor unit activation in the right biceps brachii muscle. We propose that augmented biceps muscle contraction produced with cocontraction of muscles supplied by the median nerve resulted from reinnervation of the musculocutaneous nerve via donor axons from the median nerve via way of the anastomotic site. For this to have occurred, some axons in the median nerve would have to have sprouted an axonal branch, which entered the musculocutaneous nerve. This theory is supported by the presence of late responses when recording from the biceps muscle after electrical stimulation of the median nerve at the elbow and the wrist. Although a heteronomous biceps reflex can be produced by stimulation of the median nerve at the elbow, it is not produced by stimulation of the median nerve at the wrist. Hence, the late responses might be a consequence of the median-to-musculocutaneous nerve anastomosis and might represent axon reflexes (A wave) or F-wave responses relayed to the biceps via the donor median nerve axons, although a long loop (transcortical) reflex cannot be ruled out. Clinical examination, electromyography, and nerve conduction velocity studies indicated preserved function of the muscles supplied by the median nerve, including the flexor carpi radialis, pronator teres, adductor pollicis brevis, opponens, and finger flexor muscles, without postoperative compromise.

The standard surgical repair technique for cases of severe neurormetic traumatic postganglionic brachial plexus injury is end-to-end anastomotic repair with or without a graft and/or distal neurorization that uses an intact nerve in an end-to-end manner. End-to-side neurorization repairs have also been described. For the patient in the present case, an end-to-end partial median to musculocutaneous neurorization repair at the time of the second operation would have damaged some median nerve donor axons and could have compromised axons already regenerating along the musculocutaneous nerve. Instead, the side-to-side technique enabled continued primary regeneration through the injured musculocutaneous nerve and augmented its repair with axonal contributions from the neighboring median nerve without causing damage to this nerve.

Several other groups have described side-to-side nerve repair in animals and in humans with promising results of improved motor function in the recipient nerve. However, these groups detail a technique in which patients received a combination end-to-end anastomosis at the site of nerve injury as well as a side-to-side neurorraphy distal to the site of injury in the same operation. The technique that they describe for the side-to-side neurorraphy involves incisions in both the epineurium and perineurium of the donor and recipient nerves. We have shown that nerve regeneration is possible by using side-
to-side anastomosis with an epineural window that does not damage axons in the donor nerve, as evidenced by the preservation of normal median nerve function and the absence of electromyography muscle injury potentials during the side-to-side surgical anastomosis. Although these findings cannot definitively prove a lack of injury to the median nerve axons, they strongly suggest the presence of intact nerve fascicles at the conclusion of the anastomosis. This type of repair also avoids disrupting axonal pathways that may or may not contain regenerating axons in the damaged nerve.

Intraoperative electrophysiological monitoring and stimulation played a major role in the intraoperative decision-making process. Both musculocutaneous and median nerves were systematically tested for somatosensory evoked potentials, motor evoked potentials, and motor responses in muscles through direct electrical stimulation. After motor evoked potentials were detected in the proximal musculocutaneous nerve, preservation of the regenerating axons and their potential substrate pathways became an important goal, as did preservation of intact median nerve function.

Although these results in a single patient are encouraging, further clinical and animal studies are necessary to optimize this procedure and to determine its success rate for restoring useful function.

Conclusions

Side-to-side anastomosis was performed between a healthy median nerve and a severely injured musculocutaneous nerve 17 months after a severe brachial plexus in-
Muscle reinnervation after side-to-side nerve repair

jury involving the upper and middle trunks. Postoperative clinical and electrophysiological evaluations demonstrated dual reinnervation of the biceps muscle by axons regenerating along the original musculocutaneous pathway and by axons sprouting from the intact median nerve. In the setting of a severe nerve injury, side-to-side anastomosis through an epineural window may be a viable repair option that enables continued axon regeneration along the original injured nerve pathway while simultaneously augmenting the repair process with axons from an intact adjacent nerve without compromising its function.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Kliot. Acquisition of data: Kliot, Cage, Noss, Engstrom, Ralph. Analysis and interpretation of data: Kliot, Cage, Simon, Noss, Ralph. Drafting the article: Cage, Simon. Critically revising the article: all authors. Reviewed the final version of the manuscript on behalf of all authors: Kliot. Study supervision: Kliot.

Acknowledgment

The authors thank Dr. Michael T. Lawton (University of California, San Francisco), whose vascular neurosurgery lecture (in which he discussed and showed examples of side-to-side vascular bypass procedures to revascularize brain territories) inspired us to perform an analogous surgical repair on a damaged peripheral nerve.

References


Manuscript submitted December 9, 2012. Accepted May 13, 2013. Please include this information when citing this paper: published online June 14, 2013; DOI: 10.3171/2013.5.JNS122359.

Address correspondence to: Michel Kliot, M.D., Department of Neurological Surgery, University of California San Francisco, Room M780, Box 0112, 505 Parnassus Ave., San Francisco, CA 94143-0112. email: KliotM@neurosurg.ucsf.edu.