Primary nerve grafting in traumatic injuries is rarely performed because of the uncertainty of the extent of injury, the limited availability of nerve grafts, and the damage to adjacent soft tissue. In this report the authors present two cases of acute nerve grafting after trauma—the first of the common peroneal nerve and the second of the ulnar nerve above the elbow—with sensory and motor recovery. Although compelling general arguments against primary posttraumatic nerve grafting exist, these cases illustrate that, in certain favorable and critical clinical situations, acute nerve grafting may be successful.


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Based on the work of Millesi and colleagues, nerve repair with autografts has become a standard microsurgical procedure. Nerve grafting has been used to reconstruct nerve gaps electively in all body regions, including the lower extremities, with useful results.

Nerve graft reconstructions are rarely performed at the time of traumatic injury. Standard practice generally consists of secondary nerve grafting 3 to 8 weeks after injury. The rationale for this delay is both well established and based on a number of empirical considerations. The extent of nerve damage may be difficult to assess accurately on initial exploration, and errors of estimate could lead to unnecessarily extensive nerve resection or grafting between damaged nerve margins. Donor nerve available for use as graft is a finite resource and should not be used in less than optimal circumstances. Time lost observing an ultimately unsuccessful nerve repair delays definitive repair and rehabilitation. Complications of the traumatic wound, including soft-tissue loss and infection, could expose or otherwise compromise the nerve reconstruction.

The disadvantages of delayed grafting are also notable. They include the requirement of a second operation and the difficulties of reoperative exploration of the nerve injury site through established scar and soft-tissue reconstruction. The necessity of delayed nerve grafting has been questioned in cases in which the defect was created surgically. In selected cases of surgically defined nerve trauma secondary to tumor resection, acute nerve grafting has been performed successfully. The data from such cases supports the possibility that traumatic nerve injuries could be reconstructed acutely if the wound was explored, defined, and closed under circumstances similar to a surgical resection. We present 2 patients—the first underwent acute peroneal nerve grafting after a power saw injury, and the second underwent acute ulnar nerve grafting after gunshot injury—managed according to the strategy used in repair of surgically defined nerve defects.

Patient Reports

Patient 1
A 36-year-old man sustained a power saw injury to his left leg in September 1991. Examination shortly after injury revealed a 12-cm laceration across the posterolateral surface of the popliteal fossa. Operative exploration revealed that the lateral biceps femoris tendon had been divided completely and that the common peroneal nerve had been injured grossly (Fig 1). The peroneal nerve was explored with an operating microscope. All fascicular portions of the nerve were found to be divided, although a posterior segment of epineurium and underlying fascia remained in continuity.

Microscopic debridement of all damaged nerve left a 3-cm defect that could not be repaired.
primarily despite mobilization of both nerve segments. The margins of the nerve defect after debridement appeared completely uninjured, and resection of the traumatized skin appeared to allow closure with undamaged tissue. Acute nerve reconstruction was therefore elected. Two 3-cm nerve grafts were taken from the right sural nerve and were used to reconstitute the injured nerve. Tendon repair was performed, and a rotation flap from the popliteal fossa was used to close the defect. The wound healed without complication.

The patient’s knee was immobilized for 4 weeks and quickly regained full range of motion. At 6 months the patient had paraesthesias of the first web space and some dorsiflexion and eversion of the ankle. During the next 12 months he developed strong ankle dorsiflexion and eversion, as well as dorsiflexion of the small toes and weak dorsiflexion of the great toe. The patient reported sensation to light touches at the first web space. When last examined 4 years after the injury, the patient was fully active and working, with occasional lateral foot pain attributed to some residual peroneal muscle weakness and mild adduction of the forefoot. The patient’s symptoms were well controlled by customized work boots.

An initial electromyogram acquired 7 months after surgery showed reinnervation of the peroneus muscle and proximal reinnervation of the tibialis anterior. Two subsequent studies performed during the next 8 months showed progressive, abundant reinnervation in the territory of the left peroneal nerve, including the extensor hallucis longus. No clinical or electromyographic evidence of extensor digitorum brevis reinnervation has been noted.

**Patient 2**

In November 1999, a 36-year-old right-handed man presented to the emergency room after a gunshot injury to the left arm. Physical examination showed an entry wound just above the antecubital fossa and an exit wound on the medial side of the left arm 6 cm above the medial epicondyle. The patient was taken to the operating room shortly afterward and was found to have
a partially ruptured brachialis muscle. The brachial artery and the ulnar nerve were divided completely. The brachial artery was repaired with 6-0 Prolene sutures. The ulnar nerve was explored under the microscope and was then debrided to healthy-looking fascicles, resulting in a gap of 3 cm. The gap could not be approximated without tension. The left sural nerve was harvested and three cable grafts were used to reconstitute the ulnar nerve. The brachialis muscle was repaired with 2-0 Vicryl sutures and the wound was closed in layers, taking care to keep the nerve grafts on a vascular bed. The left elbow was splinted in flexion for 3 weeks.

Electromyographic studies performed 4 months after injury showed reinnervation of the flexor carpi ulnaris. Eight months after injury the patient had grade 3 power in the flexor digitorum profundus of his little finger. Tinel’s sign had progressed to the mid forearm level. Eleven months after injury the patient had grade 4 power in the flexor digitorum profundus of the little finger and flexor carpi ulnaris. He had grade 3 power in the interossei (Fig 2). Twelve months after sustaining the injury, Tinel’s sign had progressed to the palm level. A repeat electromyogram 18 months after surgery showed further progress in reinnervation of the flexor digitorum profundus, and the hypothenar muscles showed isolated reinnervation. The ulnar portion of the dorsum of the hand regained protective sensation, with a two-point discrimination of 14 mm. He also regained protective sensation up to the proximal interphalangeal joint of the ring and little fingers.

Discussion

The case reports of peroneal and ulnar nerve repair presented here suggest that, in specific cases, acute posttraumatic nerve grafting can be successful. The benefits to the patient are dramatic: definitive repair in a single operation with early recovery and rehabilitation. The general reservations about acute posttraumatic nerve grafting may certainly be respected in wounds with ragged, indefinable injuries. In wounds that can be explored and debrided to create an approximation of a surgical resection, however, our experience leads us to recommend consideration of acute grafting.

We propose the following criteria for serious consideration of acute posttraumatic nerve grafting: existence of a substantial functional deficit because of a nerve injury, clearly defined margins of the nerve defect, and secure soft-tissue coverage. Supporting conditions for considering acute posttraumatic nerve grafting include anticipated difficulty in reexposure of the injury site, and expendable nerve graft sources. These may consist of subtotal sural nerve harvests, “spare parts” nerves retrieved from amputated parts not undergoing replantation, and nerve harvested from flap donor sites.

The functional outcomes of our 2 patients appear very good when they are examined in the general context of peroneal and ulnar nerve injuries in general. Intraoperative microscopic dissection helped to identify normal, healthy fascicles at both ends. If substantial defects exist after debridement, and a tension-free primary repair cannot be performed, then immediate nerve grafting may be attempted to repair these nerve lacerations.

The common peroneal arises from the sacral plexus and is a terminal branch of the sciatic nerve. Complete transection of the common peroneal nerve results in loss of dorsiflexion and eversion of the foot, as well as minor sensory losses. Before application of microsurgical techniques to peroneal injuries, functional return of the motor deficit was reported in 36% or less of patients after surgical repair. Using microsurgical technique and grafting, Wood reported 6 functional results in 11 cases requiring nerve grafting. Demuynck and Zuker reported one functional result in two cases of peroneal nerve grafting in children. Stancic and associates achieved functional results with nerve grafts in four of seven peroneal injuries in wartime victims.

The ulnar nerve arises from the medial cord of the brachial plexus (C8, T1). It supplies the flexor carpi ulnaris, flexor digitorum profundus to the ring and little fingers, the ulnar lumbrical, the hypothenar muscles, the palmar and dorsal interosseous muscles, and the adductor pollicis. Ulnar nerve injury, therefore, creates severe losses of extrinsic and intrinsic muscle function
Fig 2. (A) Abduction of fingers after ulnar nerve graft above the elbow. (B) Adduction of fingers. (C) Complete fist formation showing functioning of profundis. (D) Abduction of fingers viewed dorsally 18 months after repair.
as well as a sensory loss of the ulnar border of the hand. Before use of microsurgical techniques in the repair of ulnar nerve lesions, good sensory and motor return were obtained in 22% and 44% of primary and secondary repairs respectively of ulnar nerve repairs in the arm.\textsuperscript{18,19} Millesi and colleagues\textsuperscript{1} introduced microsurgical nerve grafting into the treatment of these injuries and reported useful motor return in an initial series of 39 patients. Later reports, however, have described good functional results in 52% or less of cases.\textsuperscript{20,21}

From the perspective of the current expectations of outcomes following peroneal and ulnar nerve repair, our patients have experienced good results. We propose that more frequent consideration of acute posttraumatic nerve grafting may improve results in treatment of these difficult injuries.

References


Invited Discussion: Dellon: Acute Nerve Grafting

A. Lee Dellon, MD

Sud and colleagues have described clearly the arguments against primary autologous nerve grafting, with which I am in complete agreement: (1) difficulty in determining the proximal and distal extent of the zone of injury to the nerve, (2) risk of committing scarce nerve graft donor material to an acute wound, and (3) delay in doing a secondary reconstruction because of the length of time required to determine whether the primary graft has been successful. They go on to describe 2 patients in whom they have disregarded these arguments to perform primary autologous nerve grafts: a 36-year-old man with a common peroneal nerve division by a power saw and a 36-year-old man with an ulnar nerve division by a gunshot proximal to the elbow. The results they obtained in these patients are good, but not excellent, and these authors justify their results by comparison with the literature that, although sparse, demonstrates that it is unusual to get better than a good result in cases such as these. The patients were followed for just 15 months and 12 months respectively, and it may be assumed that each of them would achieve further improvement with time.
There are three specific points to be made with respect to the general concept presented here:

First, known sites of compression proximal or distal to the nerve reconstruction should be decompressed initially or subsequently once neural regeneration becomes impeded or stops. Second, use of the bioabsorbable, polyglycolic acid Neurorotube (Neuroreogen, LLC, Bel Air, MD) can avoid the need to use autologous nerve graft donor material for defects ≤ 3 cm (as in each of these 2 patients). Third, postoperative monitoring of peripheral nerve function and sensory reeducation should be included routinely after nerve repair or reconstruction.

It has been demonstrated that a repaired peripheral nerve will regenerate distally only to have its progress stopped at a known site of compression. The known site of common peroneal nerve compression, beneath the fascia of the peroneus longus at the fibular neck, was within the operative site of the first patient reported. The fascia of the peroneus longus should be divided transversely, proximally and distally, and the nerve released into the anterolateral compartment. Frequently, there will be a fibrous band deep to the peroneus longus that must also be divided. The known site of ulnar nerve compression, the cubital tunnel, was related immediately to the gunshot that divided the brachial artery at this level. The swelling from the trauma was highly likely to create a secondary ulnar nerve compression at this level even if the nerve had not been injured directly. An ulnar nerve transposition, preferably by a musculofascial lengthening technique, is indicated for ulnar nerve reconstruction proximal or distal to the elbow, not necessarily to gain length along the course of the ulnar nerve to minimize the need to perform a nerve graft, which is possible sometimes, but to prevent secondary chronic nerve compression from compromising the result of the nerve reconstruction, be it done primarily or secondarily. It is very likely that the final result in each of the 2 patients reported by Sud and colleagues would be improved by these distal nerve decompressions. As the peripheral nerve regenerates distally over time, it may even become necessary to decompress the known distal sites of compression for each of these nerves (i.e., the superficial peroneal nerve in the distal leg and the deep peroneal nerve beneath the extensor hallucis brevis, and the ulnar nerve in Guyon’s canal). The need to do this can be identified by the development of a positive Tinel’s sign at each of these sites.

The extent of the peripheral nerve that must be resected to avoid the zone of injury produced by trauma is related to the mechanism of injury and cannot be determined until 3 weeks after the injury. Therefore, if primary nerve reconstruction is to be performed, an estimate of the zone of injury must be made based on the mechanism of injury. A knife may damage just a few millimeters at each edge of the transection, whereas a vibrating quarter-inch saw blade may damage a centimeter at each edge of the transection. A nerve defect of 3 cm can be bridged by neural regeneration through a polyglycolic acid bioabsorbable Neurorotube without the need for nerve growth factors or basement membrane. This was demonstrated in monkeys and humans. The first randomized, prospective study of nerve reconstruction ever conducted was reported recently to give significantly better results with this technique than with either a primary nerve repair or a nerve graft for nerve defects ≤ 3 cm. It is reasonable, therefore, given the situation presented by the 2 patients reported by Sud and colleagues, that, in the future, a nerve defect of as long as 3 cm be created, and a Neurorotube can be placed into the defect instead of an autologous nerve graft. This saves the patient a nerve graft donor site scar and sensory loss, saves anesthesia time, and may well be all that is necessary. If, however, at 6 months, there is not sufficient evidence of appropriate neural regeneration, then a secondary, traditional, interposition interfascicular autologous nerve graft can be performed.

Monitoring peripheral nerve function is done most easily by evaluation of sensibility in the territory innervated by the sensory portion of the reconstructed nerve. Today this is best accomplished using the Pressure-Specified Sensory Device (Sensory Management Services, LLC, Baltimore, MD). Measurements with this noninvasive and nonpainful computer-assisted technique document neural regeneration, and because they are not painful this testing lends itself to sequential studies to document clinical results of nerve reconstruction. The sensory recovery in the little finger and dorsum of the hand, as well as the dorsum of the foot, would be improved with sensory rehabilitation techniques.
A criticism may be aimed at the photodocumentation used by Sud and colleagues to demonstrate motor reinnervation. For the patient with peroneal nerve reconstruction, the photo is taken in an oblique view and from above, not lateral, so that the true extent of dorsiflexion cannot be determined. Indeed, it appears as if the ankle, which is probably stiff anyway, does not hang, as a “drop foot,” but also does not extend as much as even 90 deg of extension. So motor reinnervation has not been clinically very successful. For the patient with ulnar nerve reconstruction, abduction of the little finger can be accomplished with the extrinsic muscle—the extensor digiti minimi—by its insertion into the proximal phalanx of the little finger, and therefore these photos do not demonstrate intrinsic muscle function. Furthermore, the view of the hand with a grip shows the thumb with a positive “Froment’s sign,” demonstrating failure to reinnervate the first dorsal interossei. The flexed little finger can be accomplished by “trapping” it with the ring finger. Proof for a photo would be with the proximal interphalangeal joint held in extension and the distal interphalangeal flexed.

I think that more acute nerve reconstructions will be performed in the future, now that there is the availability of bioabsorbable nerve conduits, thus obviating the primary objection to primary nerve grafting: potential loss of autologous nerve graft donor material. A primary Neurotube reconstruction may fail just as a primary autologous nerve graft may fail, but there is still always the alternative of the classic, secondary autologous nerve graft as a lifeboat. When Sud and colleagues performed their acute nerve grafting, the Neurotube had not yet been approved by the Food and Drug Administration (late in 1999), and so they are to be congratulated for bringing to our attention the concept of primary nerve reconstruction.

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