Nerve Transfers in Patients with Brown-Séquard Pattern of Spinal Cord Injury: Report of 2 Cases

Thorbjorn Loch-Wilkinson[1,3], Stephen McNeil[1,3], Chris White[1,3], Christiaan Schrag[2], Rajiv Midha[1,3]

Key words
- Brown-Séquard injury
- Nerve transfer
- Surgical outcomes

Abbreviations and Acronyms
EMG: Electromyography
MRC: Medical Research Council

From the Departments of [1]Clinical Neurosciences and [2]Surgery and [3]Hotchkiss Brain Institute, University of Calgary, Calgary, Alberta, Canada

To whom correspondence should be addressed: Rajiv Midha, M.D., M.Sc. [E-mail: rajiv.midha@ahs.ca]

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BACKGROUND: **Use of distal nerve transfer for improving upper limb function has been well described for patients with tetraplegic spinal cord injury and brachial plexus injuries but has not previously been described for Brown-Séquard type spinal cord injury. We describe our experience with 2 cases of combined Brown-Séquard injury and unilateral brachial amyotrophy.**

CASE DESCRIPTION: **Patient 1, a 43-year-old woman, was involved in a motor vehicle accident and sustained left-side C5-7 level hemicord injury causing ipsilateral proximal arm weakness and sensory loss with contralateral hemisensory changes, neuropathic pain, and spasms. At 6 months after injury, she underwent a spinal accessory to suprascapular nerve, radial nerve triceps branch to axillary nerve, and ulnar fascicle to biceps transfer. At 2-year follow-up, she had improved function with Medical Research Council grade 4 power of shoulder abduction, elbow flexion, and internal and external rotation. Patient 2, a 38-year-old man, sustained a C4-5 fracture-dislocation in a motor vehicle accident and associated right-side hemicord injury involving the C5 and C6 myotomes with relatively preserved distal function. At 9 months after injury, he underwent radial nerve triceps branch to axillary nerve division and ulnar nerve fascicle to musculocutaneous nerve brachialis branch transfer. At 8 months after surgery, electromyography demonstrated evidence of further reinnervation of the deltoid muscle.**

CONCLUSIONS: **Our early experience of nerve transfer with 2 patients with combined Brown-Séquard cord injury and brachial amyotrophy indicated acceptable surgical safety and demonstrated encouraging results.**

INTRODUCTION

Upper extremity nerve transfer surgery in patients with spinal cord injuries, in particular, in tetraplegic patients, has been previously described.[1-6] Nerve transfers in tetraplegic patients involve transferring a donor nerve innervated from a supralesional site in the cord to nonfunctioning nerves innervated by the infralesional cord.[1] However, the use of nerve transfers from infralesional innervated sites is less established, and the utility of nerve transfer surgery has not been specifically described in patients with Brown-Séquard–type spinal cord injury. We describe our experience with 2 patients with Brown-Séquard spinal cord injury combined with brachial amyotrophy. Both patients demonstrated good results with surgery with minimal morbidity. Our experience with these 2 cases suggests that nerve transfer surgery using infralesional donor nerves may be indicated in select patients with Brown-Séquard spinal cord injury.

CASE DESCRIPTION

Patient 1

Patient 1 was a 43-year-old, right-handed, previously well woman and nonsmoker. She was involved in a motor vehicle accident and sustained a dens fracture and C5-6 Brown-Séquard–type left-side spinal cord injury in addition to limb, thoracic, and abdominal trauma, including rib fractures, pelvic and sternal fractures, and pulmonary contusions. She required emergency surgery for left nephrectomy, partial splenectomy, and repair of a left fifth metacarpal fracture. Her dens fracture was managed nonoperatively in a halo orthosis. She was initially managed at a regional hospital with complete left-side loss of motor function in the C5 and C6 myotomes and absent biceps and brachioradialis reflexes in addition to mild, British Medical Research Council (MRC) scale grade 4 triceps weakness. Sagittal and axial T2-weighted magnetic resonance imaging scans performed at her initial presentation demonstrated the odontoid fracture and a small dorsolateral epidural hematoma and subtle left-side cord signal change at the C4-5 level (Figure 1A and B).

On examination at 3 months after injury (her first visit to the tertiary peripheral nerve center), she had normal tone in both upper limbs, slightly increased left leg tone, and a slightly more brisk left ankle reflex. Knee reflexes were symmetric. She demonstrated profound wasting of the left deltoid, supraspinatus, infraspinatus, and rhomboid muscles. Supraspinatus and deltoid were isolated by stabilizing the

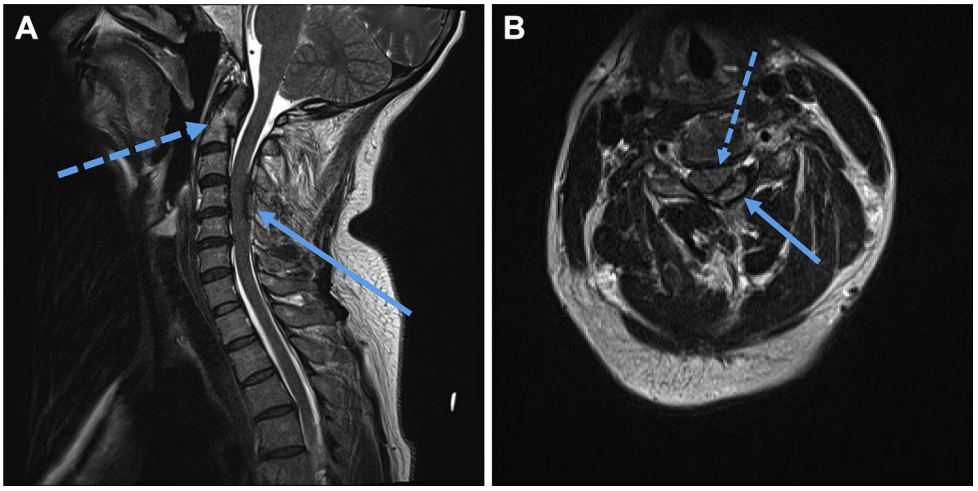

Figure 1. (**A**) Sagittal T2-weighted magnetic resonance imaging of patient 1 taken the day of presentation demonstrating a C2 odontoid fracture (*dashed arrow*) without cord compression and a C4-5 level injury with a small extradural hematoma causing mild cord compression and subtle cord signal change (*solid arrow*). (**B**) Axial T2-weighted magnetic resonance imaging of patient 1 demonstrating the extradural hematoma causing mild left cervical cord compression (*solid arrow*) and subtle T2 signal hyperintensity (*dashed arrow*).

scapula. She had 0/5 power of shoulder abduction, although she could achieve approximately 20° with scapular rotation from trapezius substitution, 0/5 external rotation, 4/5 internal rotation, 2/5 elbow flexion, and 4/5 wrist extension and pronation. Her hand function was relatively intact with at least grade 4/5 finger flexors and hand intrinsic muscle function. She had reduced left-side sensation to all modalities in the C6 dermatome with some hyperesthesia in the C5 and C7 dermatomes. She had right-side (contralateral) mild hemisensory changes, neuropathic pain in the buttock and sole of her foot, and lower limb spasms without motor weakness. Her lower limb motor, tone, and reflex examination was otherwise almost normal other than very mildly increased reflexes on the left (ipsilateral) side.

Electrodiagnostic studies demonstrated intact sensory nerve action potentials from the lateral antebrachial cutaneous nerve consistent with a preganglionic lesion. The electrodiagnostic studies indicated diffuse positive sharp waves and fibrillation potentials in all C5-6 innervated muscles with no motor units able to be elicited in the C5 and C6 innervated muscles except a single unit in biceps, which was very wide and polyphasic with low amplitude. The combined findings of preserved sensory nerve action potentials with no motor units elicited indicated a lower motor neuron type of injury consistent with injury to the anterior horn cells of the cord or a proximal nerve root rather than the brachial plexus. We excluded an upper motor neuron deficit in the C5-6 myotomes, where paralysis of muscles would be seen clinically, yet electrodiagnostic studies would show completely normal action potentials and no evidence of denervation changes on electromyography (EMG).

The patient gave her consent to undergo nerve transfer surgery, and a spinal accessory to suprascapular nerve, radial nerve long head of triceps branch to axillary nerve, and ulnar nerve fascicle to musculocutaneous nerve biceps branch transfer was performed at 6 months after initial injury. At surgery, there was no stimulation response of the suprascapular nerve, axillary nerve, or musculocutaneous nerve as expected. There were no complications from surgery, and the patient tolerated the procedure well.

At 5 months after surgery (11 months after injury), the patient had improved biceps function with 3/5 elbow flexion power and full range of motion. Some synkinetic motion of wrist flexion and elbow flexion was noted as expected. At this stage, she had not recovered any function of shoulder abduction (0/5) or shoulder external rotation (0/5), with no palpable contraction in the deltoid muscle and supraspinatus and infraspinatus muscles, all of which remained severely wasted.

At 1 year after surgery, she had improved biceps function to achieve grade 4/5 power of elbow flexion and had recovered shoulder abduction to 70° with 3/5 power. She had recovered 3/5 power in shoulder external rotation but without full range of motion. She had no significant downgrading of trapezius, triceps, or hand function. EMG demonstrated reinnervation of deltoid and infraspinatus muscles.

At 18 months after surgery (2 years after injury), she had recovered further with 80° of shoulder abduction with MRC grade 4−/5 power, grade 4/5 elbow flexion, grade 4−/5 shoulder external rotation, and grade 4/5 internal rotation. Forearm supination power was grade 4−/5, and pronation was grade 4/5. Trapezius, triceps, and hand function remained stable, and there was no evidence of downgrading from use of donor nerves. Rhomboid, supraspinatus, infraspinatus, and deltoid atrophy was still visible but improved, with greatest recovery of bulk in the middle deltoid fibers. At this stage, biceps and brachialis muscle bulk assessment was confounded owing to an interval

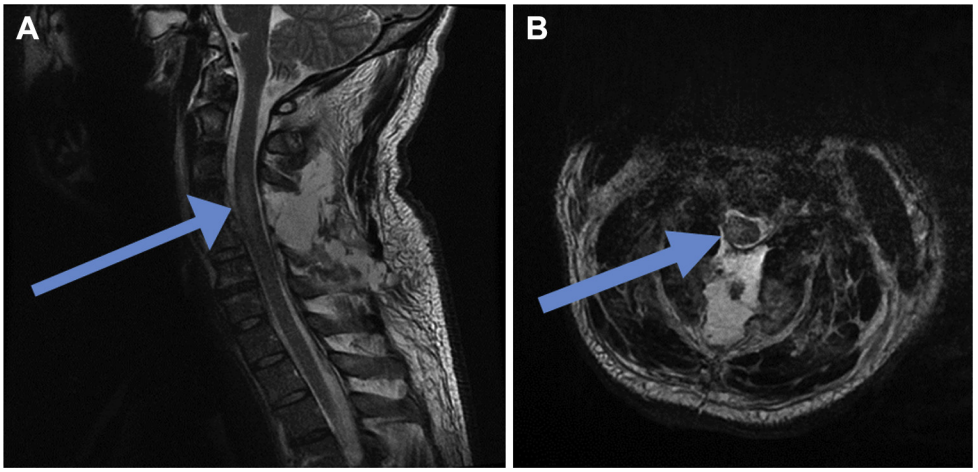

Figure 2. (**A**) Sagittal T2-weighted magnetic resonance imaging of patient 2 performed 1 day after spinal stabilization surgery for C4-5 fracture-dislocation demonstrating cord signal change from C4-6 (*solid blue arrow*). (**B**) Axial T2-weighted magnetic resonance imaging of patient 2 at C4-5 level demonstrating sharply demarcated right-side hemicord injury (*solid blue arrow*).

biceps muscle tear from a fall causing "balling up" of the muscle; however, there was no obvious decrease in elbow flexion power.

Patient 2

Patient 2 was a 38-year-old man who was involved in a motor vehicle collision and sustained a C5 lateral mass fracture-dislocation and an American Spinal Injury Association grade D spinal cord injury to the right hemicord causing a Brown-Séquard syndrome. He was treated with open posterior decompression and spinal stabilization of C4-6. T2-weighted axial and sagittal magnetic resonance imaging performed following spinal stabilization surgery demonstrated a sharply demarcated right hemicord injury extending from C4 to C6 (Figure 2A and B). There were no imaging findings to suggest root avulsion injury.

The patient was initially hemiplegic on the right but demonstrated some neurologic improvement after rehabilitation. At 3 months after injury, he had recovered ambulation with a walking stick but had right-side proximal arm weakness with MRC grade 0/5 shoulder abduction and 0/5 elbow flexion. He had better preservation of distal right upper limb function of forearm supination and pronation, elbow extension, and wrist extension and flexion and hand grip between 2/5 and 4/5 power. He had absent right proximal limb sensation over the C5 and C6 dermatomes with near-normal distal sensation. He had generally mild right lower limb weakness (4/5 power).

The patient underwent initial examination at our tertiary hospital peripheral nerve clinic at 8 months after injury where electrodiagnostic studies were performed. Radial, median, and ulnar sensory nerve action potentials were preserved. At this stage, he had established atrophy of deltoid, supraspinatus and infraspinatus muscles and absent biceps contraction with persisting 0/5 power of shoulder abduction and elbow flexion and decreased tone in C5 and C6 myotomes. More distally, he had some spasticity and difficulty activating muscle groups but good strength between grade 4 and 5 power. Electrodiagnostic studies of biceps, deltoid, and brachialis showed 3+ denervation changes and showed only a few motor units in the deltoid, biceps, and brachialis with decreased recruitment but good activation, suggesting weakness was due to a peripheral rather than a central nervous system problem. Triceps was normal on electrodiagnostic studies and had grade 4+/5 power. He had no denervation of flexor carpi ulnaris on needle EMG studies.

The patient was considered to have a combination of Brown-Séquard spinal cord injury with associated lower motor neuron—type injury likely involving the lower motor neurons of the metameric segment of the cord involving the C5 and C6 anterior horn cells and motor neurons or a combined cord injury and C5 and C6 root injury. Given the lower motor neuron injury, reconstructive procedures were considered to be time dependent. He was waitlisted for surgery and had a further evaluation 2 months later to ensure no further recovery had occurred that would mitigate the need to proceed to nerve transfer surgery.

By this stage at 9 months after injury, the patient had minimal biceps recovery with a flicker of contraction felt (MRC grade 1/5) but no functional elbow flexion. The deltoid remained atrophied; however, electrodiagnostic studies showed some partial reinnervation of the deltoid with a decreased number of units firing rapidly indicative of good central drive.[7] There was slight improvement in atrophy of the supraspinatus and infraspinatus. Shoulder abduction had improved slightly to achieve abduction of 25° in line with the body. With muscle substitution from the pectoralis, he could elevate his arm in an anterior direction to approximately 80°, and using gravity to passively flex his elbow, he could translate his forearm and hand to just reach his mouth. He had grade 4/5

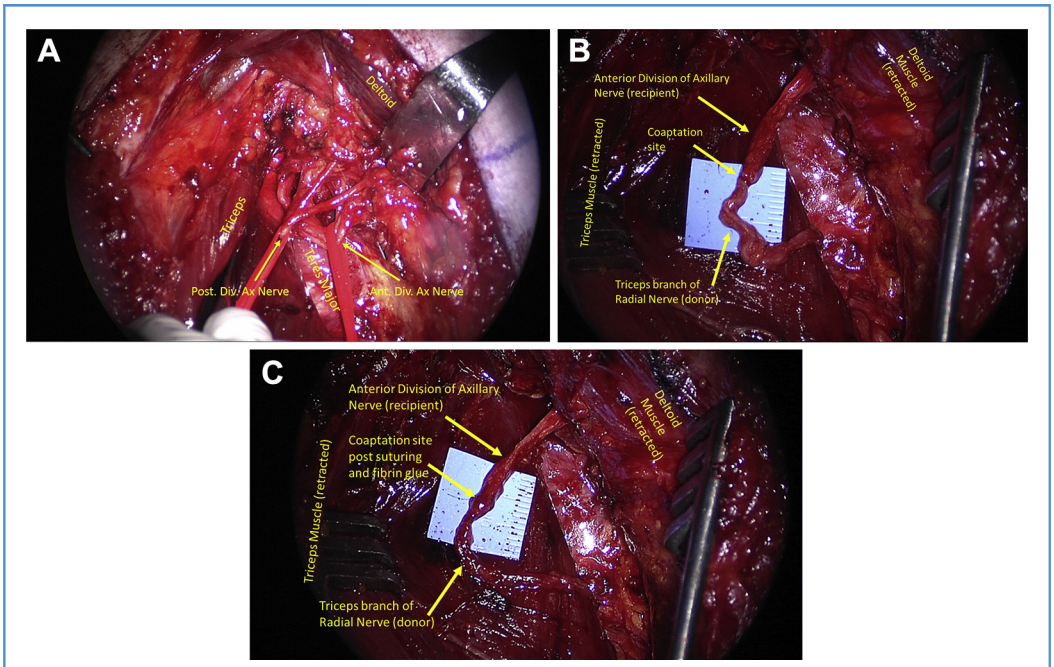

Figure 3. (**A**) Intraoperative image of patient 2 demonstrating exposure of the quadrangular space via a posterior approach. The upper loop is retracting the posterior division of the axillary nerve. The lower vessel loop is retracting the anterior division of the axillary nerve. (**B** and **C**) Intraoperative images of patient 2 showing coaptation of a long head of triceps branch of the radial nerve to anterior division of the axillary nerve before (**B**) and after (**C**) microsuture and fibrin glue application.

shoulder internal and external rotation. He had full range of motion with elbow extension and 4+/5 triceps power; however, he had some increased tone. Pronation power was slightly stronger than supination at 4−/5, and he had grade 5/5 wrist flexion and grade 4/5 in median and ulnar nerve innervated external flexors. However, he had persisting slight weakness of finger extensors and spasticity of the volar aspect of his forearm and fingers.

After detailed discussion with the patient, he underwent a radial nerve long head of triceps branch to axillary nerve anterior division transfer (Figure 3) and an ulnar fascicle (which stimulated to contract the flexor carpi ulnaris and flexor digitorum profundus) to a brachialis branch of the musculocutaneous nerve (Figure 4). During surgery, the musculocutaneous nerve branches to biceps and brachialis showed no contraction even to maximal electrical stimulation, but given the impression of clinical function in biceps before surgery, we elected not to direct the transfer to this muscle but rather to brachialis. Only the anterior division of the axillary nerve demonstrated muscle contraction with stimulus of anterior and middle deltoid fibers. One of 2 branches from the anterior division was selected as the recipient nerve from a long head of triceps branch of the radial nerve donor. The nerve coaptations were completed with 9-0 nylon suture and fibrin glue in a tension-free manner (Figure 3B and C and Figure 4B). The surgery was uncomplicated, and postoperatively there was no discernible change in baseline triceps strength or strength of flexor carpi ulnaris, triceps, or hand function. At 3 months after surgery, no discernible functional recovery had yet occurred, and his motor and sensory examination was unchanged. EMG of brachialis demonstrated no evidence of reinnervation at this stage.

At 8 months after surgery, the patient's motor examination was improved in both shoulder and elbow function. He demonstrated 30° of lateral and 110° of anterior shoulder abduction and grade 2 MRC strength in the deltoid, and EMG of the deltoid indicated further evidence of reinnervation in both anterior and middle deltoid. Elbow flexion was MRC grade 2, and he could bring his hand to mouth with gravity eliminated. Most elbow flexion was via biceps with no evidence of brachialis reinnervation apparent on EMG testing.

DISCUSSION

Patients with incomplete (American Spinal Injury Association grade C or D) spinal cord injuries are a heterogeneous group, and the injuries are complex from the point of view of upper limb reconstruction. Therefore, consideration for surgery requires an individualized approach, as many patients recover spontaneously.[8] Motor recovery typically occurs within 6 months in cases of Brown-Séquard injury, although it may be incomplete on the ipsilateral side.[9,10] In both cases described in this article, severe focal motor deficits were still present at 6 and 8 months after injury at the metameric level prompting consideration of reconstructive surgery.

Spinal cord function after cord injury requires an assessment to establish the extent of intact supralesional function, extent of the injured metamere, and the infralesional level of the cord. Injury of the

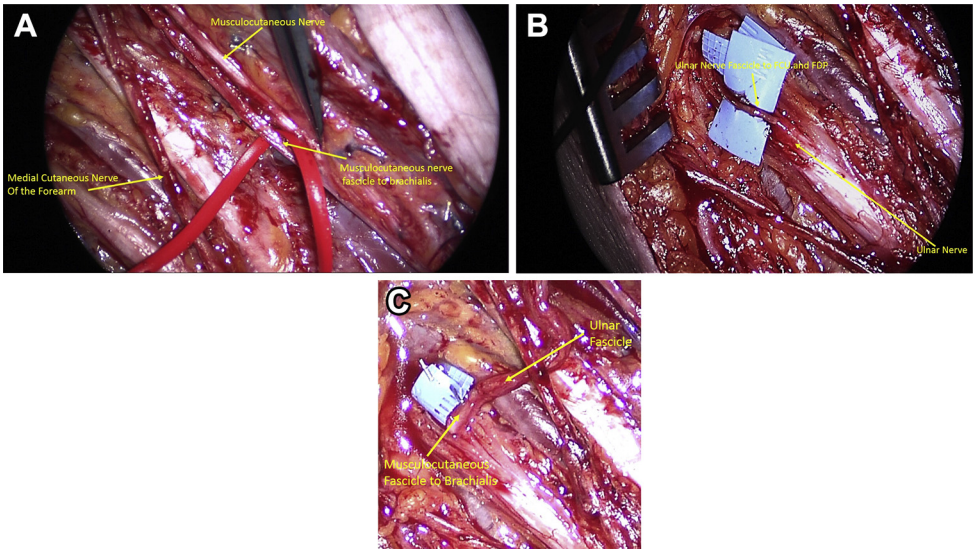

Figure 4. (**A**) Intraoperative image of patient 2 demonstrating exposure of the brachialis branch of the musculocutaneous nerve (with vessel loop). The medial cutaneous nerve of the forearm is also visible in the image. (**B**) Intraoperative image of patient 2 showing dissection of a donor ulnar fascicle (innervating flexor carpi ulnaris and flexor digitorum profundus) before coaptation. (**C**) Intraoperative image of patient 2 showing coaptation of an ulnar fascicle to the brachialis branch of musculocutaneous nerve.

spinal metamere entails segmental cord damage that includes injury to the lower motor neurons. The resulting denervation of the corresponding muscles is similar to a peripheral nerve palsy rather than an upper motor neuron type of injury from loss of cortical inhibition. Loss of the motor neuron pool from the injured metamere will eventually lead to degeneration of the motor endplate in muscle; therefore, reconstructive surgery to reinnervate the muscle is time dependent. Delay in diagnosis may lead to missed opportunity to reinnervate muscle with nerve transfer, leaving diminished options for reconstruction and functional improvement. Establishing the extent of an injured metamere can be challenging and relies on clinical assessment aided by spinal imaging and electrodiagnostic studies.[11] Clinical examination may demonstrate spasticity or spontaneous contraction of muscle groups innervated by spinal levels below (infralesional) the injured metamere, but given preservation of the motor neuron pool, muscles will not undergo atrophy or endplate degeneration. The extent of the injured metamere cannot be established in the setting of acute injury and is more accurately established by 4 months after injury.[11] Clinical examination demonstrates persisting palsy of the myotomal level of injury. EMG of metamere innervated muscle groups may demonstrate ongoing denervation changes (fibrillations and positive sharp waves) from motor neuron loss rather than evidence of reinnervation. Reduced amplitude of compound motor action potentials also suggests motor neuron or axonal loss. Surface electrical stimulation studies are of value in establishing lower motor neuron integrity and mapping muscle groups for partial or complete denervation.

Reconstructive procedures to improve upper limb function require a multidisciplinary approach that may require more than nerve surgery alone. Multiple surgical options can improve upper limb function, including nerve transfer, tendon transfers, tenotomy, tenodesis, arthrodesis, capsulotomies, and muscle lengthening.[8] Nerve transfers for upper limb reanimation have become increasingly popular but are best described for isolated brachial plexus or nerve root injuries. In spinal cord injury, use of nerve transfer reconstructive procedures is best described in tetraplegic patients.[1,3,6,11] Nerve transfers in this setting ordinarily use a donor nerve or fascicle with innervation from a supralesional location.[1,3,4] Use of infralesional innervated donor nerves in patients with spinal cord injury is performed only in very selected circumstances. The 2 patients described in this article had an unusual type of injury pattern involving a hemicord injury but with additional lower motor neuron involvement of either the anterior horn cells of the involved cord or the preganglionic nerve roots and had not recovered significantly at 6 and 8 months after injury. Neither patient had evidence of root avulsion, but evidence of spinal cord parenchymal injury was consistent with injury of a spinal metamere. Both patients had relatively preserved or had recovered motor function distal to the cord lesion, although with some upper motor neuron injury findings on examination (mild hyperreflexia or increased tone in infralesional innervated muscles). The concern in both patients was that a time-dependent risk of permanent muscle endplate degeneration existed owing to injury of the spinal metamere. Given there was good function infralesional to the level of injury, the risk of significantly downgrading function from use of an infralesional donor nerve

appeared acceptable in both patients. Both patients demonstrated encouraging functional or early electrophysiologic improvement with surgery; however, both patients will continue to be followed to assess the long-term outcome. Specific surgical selection criteria or contraindications may become clearer over time. In this small case series, good functional outcomes were achieved in patient 1, who had a less severe cord injury than patient 2. In patient 2, encouraging evidence of increased innervation of the deltoid was seen at 8 months, although we interpret this fact with caution given there were some preoperative indicators of spontaneous recovery in this muscle. As such, we do not ascribe with certainty the reinnervation of the deltoid to his nerve transfer surgery. We thought it appropriate to include both patients in the study to indicate the real-world experience of patients with this uncommon injury pattern.

CONCLUSIONS

In patients with a spinal cord injury involving damage to a spinal metamere causing proximal loss of upper limb function, acceptable results may be obtained from nerve transfers using a functional infralesional donor nerve if distal function remains relatively intact. The 2 patients in this series with Brown-Séquard–type incomplete cord injury and brachial amyotrophy had evidence of spinal metameric injury and therefore required reconstructive surgery in a similar manner to patients with a peripheral nerve injury. Although the Brown-Séquard spinal cord injury pattern described in the 2 patients in this case series is relatively uncommon, use of nerve transfer techniques could be considered a treatment option in select similar patients.

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