Clinical application of sensory protection of denervated muscle

James R. Bain, M.D., M.Sc., F.R.C.S.C.¹, Yaniv Hason, M.D.⁴, Karen Veltri, M.Sc., Ph.D.¹, Margaret Fahnstock, Ph.D.², and Caroline Quartly, M.D., F.R.C.P.C.³

¹Department of Surgery, Division of Plastic Surgery, McMaster University, Hamilton, Ontario, Canada

²Department of Psychiatry and Behavioural Neurosciences, McMaster University, Hamilton, Ontario, Canada

³Department of Medicine, Division of Physical Medicine and Rehabilitation, McMaster University, Hamilton, Ontario, Canada

⁴McMaster University, Hamilton, Ontario, Canada

Abstract

Following proximal peripheral nerve injury, motor recovery is often poor due to prolonged muscle denervation and loss of regenerative potential. The transfer of a sensory nerve to denervated muscle results in improved functional recovery in experimental models. The authors here report the first clinical case of sensory protection. Following a total hip arthroplasty, this patient experienced a complete sciatic nerve palsy with no recovery at 3 months postsurgery and profound denervation confirmed electrodiagnostically. He underwent simultaneous neurolysis of the sciatic nerve and saphenous nerve transfers to the tibialis anterior branch of the peroneal nerve and gastrocnemius branch from the tibial nerve. He noted an early proprioceptive response. Electromyography demonstrated initially selective amelioration of denervation potentials followed by improved motor recovery in sensory protected muscles only. The patient reported clinically significant functional improvements in activities of daily living. The authors hypothesize that the presence of a sensory nerve during muscle denervation can improve functional motor recovery.

Keywords

muscle denervation; sciatic nerve palsy; sensory nerve

Poor muscle and nerve functional recovery following proximal peripheral nerve injury can result in a serious clinical problem. Incomplete or inappropriate muscle reinnervation,

Address correspondence to: James R. Bain, M.D., M.Sc., F.R.C.S.C., Department of Surgery, Division of Plastic Surgery, McMaster University, Hamilton, Ontario L8N 3Z5, Canada. bainj@hhsc.ca.
Current Address for Dr. Hason: 200 Danforth Avenue, 2nd Floor, Toronto, Ontario, Canada.
Current Address for Dr. Quartly: Department of Physical Medicine and Rehabilitation, Vancouver Island Health Authority, Foot & Leg Ulcer Clinic, Victoria, British Columbia.

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decreased receptivity of muscle to reinnervation, and muscle fiber apoptosis, necrosis, and fibrosis contribute to this functional deficit.^{4,5,13} Many strategies have been used to improve muscle recovery following these injuries, including distal motor neurotization,^{14,24} electrical stimulation,^{11,16} and pharmacological treatment^{6,19} to increase the rate of reinnervation.

There is conflicting evidence regarding the efficacy of these interventions. Distal motor nerve transfers have shown significant success as they provide healthy exogenous distal motor axons for timely reinnervation of the target musculature;²³ however, a suitable motor nerve do nor must be available for transfer, and this supply is limited.

Sensory protection is a method of improving muscle recovery after denervation by providing sensory nerve input into the denervated muscle while it is awaiting rein-nervation from its motor nerve. The procedure is based on observations of experimental data from the 1940s and 1950s.^{7,8,22} These researchers evaluated the physiological changes associated with sensory-to-motor nerve crossovers. No action potentials could be elicited by stimulating the transferred sensory nerve; however, other significant changes were observed in the muscle. We and others have reinvestigated experimental sensory nerve-to-motor transfer and demonstrated improved muscle weight and architecture.^{1,9,21} We have defined this effect as sensory protection of denervated muscle.⁹ Interestingly, if muscle treated with sensory protection is subsequently reinnervated by a motor nerve, even after prolonged periods of denervation, significantly improved muscle force can be measured in these muscles compared with those in denervated controls.¹

Complete sciatic nerve palsies rarely show good recovery below the knee given the prolonged reinnervation time to target musculature. Unfortunately, total hip arthroplasty is the most commonly reported cause of sciatic neuropathy (incidence of 0.5–2.0% among total hip arthroplasties).^{3,15,25} The peroneal division of the sciatic nerve is most commonly affected,¹⁵ followed by complete sciatic involvement. Most patients have a neurapraxia or axonotmesis and recover quite well, if not fully, whereas some suffer permanent disability.²⁵

Here we present a case of clinically applied sensory protection in a patient with a sciatic nerve palsy following total hip arthroplasty. This case is the first thoroughly reported instance of sensory protection with a major proximal nerve injury. Our end points were improved activities of daily living and thorough electrophysiological measurements of recovery. The limitations of a single case report apply.

Case Report

Approval for our study was obtained from the local institutional review board. The patient gave informed consent and was told that data concerning his case would be submitted for publication.

History and Examination

This previously healthy 62-year-old man with a prolonged history of osteoarthritis of the hip underwent total hip arthroplasty in April 1999. Immediately following surgery, he had paresthesia in his leg and foot and complete numbness and paralysis below the knee. He had

full knee extension but weak flexion. He had normal sensation in the perineum, hip, and buttock. Bladder and bowel function were normal. Initial electrodiagnostic studies showed profound loss in both the peroneal and tibial nerve distributions of the sciatic nerve.

There was no clinical improvement during the initial 3 months postsurgery, and electrodiagnostic studies confirmed the static nerve injury at the level of the notch. Magnetic resonance imaging confirmed a normal lumbosacral spine but did not show an obvious sciatic nerve lesion to account for the patient's deficit.

Operation

Three months after the arthroplasty, a combined procedure was undertaken, firstly, to deal with the proximal sciatic nerve neuroma and, secondly, to protect targeted muscles below the knee from atrophy until proximal nerve regeneration could occur. The right sciatic nerve was explored, and a neuroma-in-continuity was identified extending ~ 5 cm from the sciatic notch beneath the piriformis muscle. The piriformis muscle was released, the nerve was decompressed externally, and thorough internal neurolysis was performed with the aid of an operating microscope (Fig. 1). It was decided that the intraneural architecture was satisfactory postneurolysis and that resection and grafting were not required. Triamcinolone (40 mg) was topically applied to the nerve following decompression. Intraoperative direct sciatic nerve stimulation was performed and mirrored the preoperative findings of no distal motor response, even in the biceps femoris muscle. An anterior and posterior surgical exposure for nerve transfers was designed (Figs. 2 *left* and 3 *left*). The targets selected for sensory protection were the gastrocnemius (medial and lateral heads) and tibialis anterior muscles (Figs. 2 *right* and 3 *right*). The saphenous nerve was explored and divided distally to allow tension-free transfer into the popliteal fossa and across to the deep branches of the peroneal and tibial nerves (Fig. 4). The motor branches from the tibial nerve to the medial and lateral gastrocnemius nerve were identified where they become confluent exiting from the posterior tibial nerve, as was the branch from the deep branch of the peroneal nerve to the tibialis anterior muscle. The peroneal nerve was decompressed over the fibular head. A perineurial window was made in each recipient nerve, and an individual nerve fascicle from the saphenous nerve was repaired end-to-side to the distal nerves (Fig. 5).

Postoperative Course

The patient had no intraoperative or postoperative complications. He was monitored serially with both clinical and electrodiagnostic studies by an independent physiatrist. Threshold electrical stimulation was used for all distal muscles (not only sensory protected muscles).

Clinical and electrodiagnostic results are shown in Tables 1 and 2. There were no significant clinical changes for 5–6 months following surgery. As would be expected, the first muscles to show signs of recovery after the proximal nerve injury and sciatic nerve neurolysis were the lateral hamstring muscles. Both clinically and electrodiagnostically, reinnervation was observed in the biceps femoris and semimembranosus muscles 6 months postoperatively; however, no recovery was seen in the muscles below the knee other than in sensory protected muscles. These protected muscles were the only targets below the knee with electrophysiological responses. A distant attenuated voluntary motor unit was consistently

identified, and attempts at voluntary activity were associated with a reduction in the spontaneous discharge from the tibialis anterior muscle. This result was observed long before reinnervation from the sciatic nerve at the level of the piriformis muscle was possible but was temporally consistent with sensory protected fibers from the saphenous nerve that had regenerated into the muscle.

Compound motor action potential recordings in the left leg (control) showed normal electrical activity throughout. Clinical findings of power testing in the right foot inversion and eversion and hallux flexion and extension were completely absent at all test dates (data not shown). Knee extension was tested and was normal, as expected (not shown).

Sensory nerve action potential recording from the left and right sural nerve were performed as well. The left leg had full activity, whereas the right leg did not show any nerve response at any time. Sensory examination revealed normal sensation in the regions supplied by branches of the femoral nerve, as expected. Areas supplied by branches of the tibial and peroneal nerves showed markedly decreased or absent sensation.

By 1 year after surgery (16 months following denervation), reinnervation was robust in the lateral hamstring muscles and ankle dorsiflexion through the functioning tibialis anterior muscle was returning. Fibrillation was decreasing in the tibialis anterior muscle (Table 2), and compound motor action potential amplitude was increasing (Table 1). The patient also reported recovering the position sense of his ankle. A history of physical activity showed that he was unable to perform activities such as playing golf or swimming in the latter part of 1999 and early on in the year 2000. He later resumed swimming, cycling, and golf. He did not require an ankle-foot orthotic at all times. After 2 years, improvements continued both clinically and electrodiagnostically but were limited to the proximal musculature and the sensory protected muscles. No recovery occurred in the foot intrinsics or toe flexor or extensor muscles.

Discussion

Total hip arthroplasty is the most common cause of sciatic nerve palsy. Intraoperatively or postoperatively, nerve damage can occur due to acute or chronic compression, stretching, ischemia, or laceration.^{3,15} Some authors have argued that traction on the sciatic nerve due to intraoperative limb lengthening is the primary mechanism of injury, especially when the peroneal division is involved.^{3,25} Others have suggested that a direct causal relationship between limb lengthening and nerve injury is not so clear and that the true mechanism of injury is nerve compression.¹⁸ The architecture of the nerve can withstand modest stretch forces without sustaining any damage.²⁰ The intrinsic and extrinsic vasculature that run within the epineurium, perineurium, and endoneurium have a tortuous, coiled appearance with great reserve in length, permitting considerable change in position or length before blood flow is impaired.¹² In addition, the nerve's fascicular connective tissue elements accommodate moderate stretch forces, preventing force transmission to the axons.²⁰ Compression of the nerve can result not only in direct trauma, but also in ischemia, which has been associated with neuropathy.¹⁸ After 2–6 hours of ischemia, epineurial edema is observed but does not cause irreversible damage to the nerve. In contrast, 8 hours of

ischemia leads to permanent nerve injury.¹⁸ Thus, numerous mechanical forces can lead to nerve injury following arthroplasty.^{2,12}

The potential and time course for recovery depends on the extent of the nerve pathology: neurotmesis, axonotmesis, or neurapraxia.¹³ The complete sciatic nerve recovery rate following total hip arthroplasty is ~ 40%. Incomplete recovery with persistent mild deficits occurs in 44% of patients, and 15% of patients have a poor outcome.¹⁸ Furthermore, those who achieve complete recovery of function following sciatic nerve palsy from total hip arthroplasty usually do so by 21 months posttreatment.¹⁰

A poor clinical outcome also correlates with the distribution of nerve deficits. Lesions affecting the entire sciatic nerve have a poorer prognosis than those involving only the peroneal or tibial division. The peroneal nerve is more frequently affected, probably due to its superficial position in the sciatic trunk or its fixation at both the greater sciatic foramen and the neck of the fibula. Another possible reason for its vulnerability is its fascicular architecture.²⁰

The patient in the current case presented with a complete sciatic nerve palsy together with paresthesias in his leg and foot, complete numbness below the knee, and an inability to dorsiflex, plantar flex, and invert or evert the foot.^{3,18} He had full power extending the knee, and flexion was slightly decreased. He falls into Group 5 (out of 5) of Schmalzried et al.'s¹⁷ classification of sciatic nerve palsy. Patients in this group suffer from a neurological deficit that limits their ability to walk and forces them to change to a sedentary occupation. They exhibit gross impairment of motor function, suffer moderate to severe dysesthesia, or have a combination of these symptoms that impair their activities of daily living.

Edwards and associates³ have found that patients with this degree of severe nerve palsy demonstrate significant functional disability after an average of 6.9 years, with symptoms such as a flail ankle in association with paresis below the knee, painful dysesthesias that persist over the dorsum of the foot, foot drop, and weak recovery of plantar flexion. Interestingly, only knee flexion recovered function, whereas motor function below the knee, such as foot dorsiflexion and plantar flexion, did not recover. In contrast, the patient in the present case showed functional recovery of the tibialis anterior and gastrocnemius muscles within a period of only 1 year after the sensory protection operation (16 months after denervation). Furthermore, muscles that did not undergo sensory protection failed to show any clinical or electrophysiological recovery and clinically resembled the picture described by Edwards et al. Although we cannot attribute all of our patient's improvement to the sensory protection procedure (proximal muscle targets such as hamstrings normally recover some function), the time to reinnervation is one of the most significant factors affecting functional recovery of distal muscles following nerve injury. Sensory protection reduces or delays muscle fibrous and fat replacement and deterioration of the distal nerve stump,²¹ which impair receptivity to reinnervation. This case is the first clinical instance, paralleling findings in our experimental studies,^{1,9,21} of sensory reinnervation of muscle that can diminish denervation atrophy. Electrophysiological measures and activities of daily living were improved in this patient.

Conclusions

We presented a case of the clinical application of sensory protection of distal musculature in a patient who had suffered severe sciatic nerve palsy following total hip arthroplasty. Although these injuries have variable outcomes, based on the severity of our patient's injury we would not have expected him to recover distal muscle function without our treatment. We propose that the degree of recovery in electrophysiological measures and activities of daily living was a consequence of the muscles maintaining receptiveness to reinnervation because of the sensory protection nerve transfer.

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Fig. 1.

Drawing showing intraoperative exploration of the injured sciatic nerve. Once the piriformis muscle is reflected and the sciatic is identified, neurolysis is performed with the aid of an operating microscope. The procedure is continued until bands of Fontana are observed. Following neurolysis, deposteroid is injected locally.

Fig. 2. Schematic illustrating the step-by-step sensory protection procedure. *Left:* Anterior surgical exposure. *Right:* Anterior exposure to tibialis anterior muscle and deep branches of the peroneal nerve.

Fig. 3. Diagram demonstrating the step-by-step sensory protection procedure. *Left:* Posterior surgical exposure. *Right:* Posterior exposure to gastrocnemius muscles and their innervation.

Fig. 4.
Drawing showing exposure and distal division of the saphenous nerve in preparation for transfer.

Fig. 5. Schematic revealing sensory protection of the right tibialis anterior (*anterior view*) and gastrocnemius (*posterior view*) muscles by end-to-side connection of fascicles of the right saphenous nerve.

TABLE 1

Clinical and electrophysiological recovery data *

Parameter	Preop			Postop				
	04/15/99	05/06/99	07/12/99	10/14/99	01/13/00	05/11/00	08/17/00	04/23/01
CMAP studies								
peroneal nerve								
lt leg (control)	BK/EDB: L 12 msec, Amp 5.7 mV, CV 42 m/sec	NT	FH/EDB: L 11.5 msec, Amp 7.3 mV, CV 47 m/sec	K/SOL: L 34.4 msec, Amp 6.6 mV	NT	NT	NT	NT
rt leg	PF/TA: L 0, Amp 0, CV 0	A/EDB & K/TA: L 0, Amp 0, CV 0	PF/TA: L 12 msec, Amp 0.020 mV	A/EDB, K/TA, AK/TA, PF/Per Long, & K/SOL: L 0, Amp 0, CV 0	A/EDB, K/TA & AK/TA: L 0, Amp 0, CV 0	PF/TA: L 9.3 msec, Amp 0.070 mV, CV 19 m/sec; FH/TA: L 6.7 msec, Amp 0.060 mV	K/TA: L 8.9 msec, Amp 0.105 mV; AK/TA: L 11.5 msec, Amp 0.133 mV, CV 35 m/sec	AK/TA: L 2.0 msec, Amp 0.031 mV, CV 45 m/sec; BK/TA: L 5.6 msec, Amp 0.032 mV
tibial nerve								
lt leg (control)	A/AH: L 4.5 msec, Amp 14.1 mV; K/AH: L 13.4 msec, Amp 11.2 mV, CV 45 m/sec	K/SOL: L 33.6 msec, Amp 7.6 mV						
rt leg			A/AH: L 0, Amp 0, CV 0		A/AH: L 0, Amp 0, CV 0			A/AH: L 0, Amp 0, CV 0
clinical findings: rt leg [†]								
foot dorsiflexion		absent	absent	absent	absent	absent	2+	3+
foot plantar flexion		absent	absent	absent	absent	absent	absent	2+
knee flexion		absent	absent	1-2	3-4	4+	4+	4+
activity						weaning off splint, swimming daily, starting to play golf	resuming golf, biking	

*The construction x/y refers to stimulation site/recording site.

Abbreviations: A = ankle; AH = adductor hallucis muscle; AK = above knee; Amp = amplitude; BK = below knee; CMAP = compound motor action potential; CV = conduction velocity; EDB = extensor digitorum brevis muscle; FH = fibular head; K = knee; L = latency; NT = not tested; Per Long = peroneus longus muscle; PF = proximal fibula; SOL = soleus muscle; TA = tibialis anterior muscle.

[†]Muscle power was graded based on the Medical Research Council Scale.

TABLE 2
Electromyography recordings of sensory-protected muscles following denervation*

Muscle	Preop		Postop			
	07/12/99	01/13/00	05/11/00	08/17/00	04/21/01	
tibialis ant	2+ fibs, PSW's	3+ fibs, 1 distant MU recruitment	2+ fibs, 1-2 MU's	2+ fibs, 3-4 MU's	2+ fibs, large 6 MU's	
lat gastrocnemius	4+ fibs	4+ fibs	3-4+ fibs	3+	4+ fibs, 3-4 MU's	
medial gastrocnemius	4+ fibs	2+ fibs	3-4+ fibs		1+ fibs, small polyphasic MU	

* fib = fibrillation; MU = motor unit; PSW = positive sharp wave.