
This article is posted on this site with the permission of the American Veterinary Medical Association, which holds the copyright for the article. For permission to distribute the article, in print or electronically, please contact the AVMA (dfagen@avma.org).

**University of Bristol - Explore Bristol Research**

**General rights**

This document is made available in accordance with publisher policies. Please cite only the published version using the reference above. Full terms of use are available: http://www.bristol.ac.uk/pure/about/ebr-terms
Cutaneous saphenous nerve graft for the treatment of sciatic neurotmesis in a dog

Nicolas Granger, DVM; Pierre Moissonnier, DVM, PhD; Laurent Fanchon, DVM; Antoine Hidalgo, DVM; Kirsten Gnirs, DVM; Stephane Blot, DVM, PhD

Case Description—A 2-year-old Griffon Vendéen was examined because of a 1-month history of right hind limb lameness after a traumatic injury.

Clinical Findings—Neurologic examination revealed monoparesis and anesthesia of the right hind limb distal to the stifle (femorotibial) joint except for the area supplied by the cutaneous saphenous nerve. Results of electromyographic testing were consistent with a severe lesion of the tibial and peroneal nerves at the level of the stifle joint.

Treatment and Outcome—Exploratory surgery revealed an 80-mm-long gap in both the peroneal and tibial branches of the right sciatic nerve. A section of the left cutaneous saphenous nerve was interposed to graft the nerve defects. The dog received joint mechanotherapy and electrophysiologic therapy during the reinnervation process. Ten months after surgery, the dog had recovered almost completely. Neurologic examination revealed diminished flexion of the tarsal and digital joints. Repeat electromyographic testing revealed no abnormal spontaneous electrical activity in the right hind limb musculature, and small compound muscle action potentials were recorded in the right interosseous and cranial tibial muscles.

Clinical Relevance—Without surgical treatment, neurotmesis injury results in poor recovery of motor and sensory functions and may result in amputation. Without surgical treatment, neurotmesis injury results in poor recovery of motor and sensory functions and may result in amputation. Even if the procedure is delayed until well after the injury, the sensory portion of the cutaneous saphenous nerve is a potential source of peripheral nerve for grafting in dogs. Reinnervation is a long-term process and physiologic support and owner involvement are necessary, but nearly complete functional recovery is possible. (J Am Vet Med Assoc 2006;229:82–86)

A 2-year-old sexually intact female Griffon Vendéen was referred to the National Veterinary School of Alfort with a 1-month history of lameness in the right hind limb after being gored by a wild boar. Prior to referral, a wound lateral to the stifle (femorotibial) joint had been treated and was healed. At the time of initial examination at the veterinary school, the dog had monoparesis of the right hind limb with plantigrade stance on the tarsal (hock) joint and knuckling of the dorsum of the paw. Severe muscle atrophy was evident distal to the stifle joint, and the skin was ulcerated on the dorsum of the paw. Neurologic examina-

From the Unité de Neurologie (Granger, Gnirs, Blot), Unité de Chirurgie (Moissonnier, Hidalgo), and the Unité de médecine de l’Elevage et du Sport (Fanchon), Ecole Nationale Vétérinaire d’Alfort, 7 avenue de General de Gaulle, 94704 Maison-Alfort Cedex, France. Address correspondence to Dr. Granger.
medial aspect of the left femoral region was prepared to expose the CSN for harvest as a source of nerve graft. Dense fibrous tissue caudal to the right stifle joint was removed. Neuromas were observed on the proximal end of the injured nerves, and degeneration of the distal axon segments was detected in both the tibial and peroneal nerves. The neuromas and degenerated axonal segments were transected back to expose viable nerve, leaving an 80-mm gap between the nerve ends. The CSN was isolated via soft-tissue dissection on the medial aspect of the femoral region on the contralateral limb and was transected just distal to the site where the muscular branch of the saphenous nerve and the femoral nerve exit (Figure 1). Sectioning of the CSN at 4 levels yielded 3 90-mm-long peripheral nerve grafts with diameters of 2 to 3 mm. Two grafts were interposed between the fascicular bundles of the tibial nerve endings, and 1 graft was interposed between the fascicular bundles of the peroneal nerve endings (Figure 2). Nerve grafts were sutured with a 7-0 non-absorbable monofilament material in an epineural-perineural technique under an operating microscope. The site was closed routinely. The total nerve regeneration distance was approximately 240 mm, corresponding to the 90 mm of peripheral nerve graft and the 150 mm between the end of the graft and the interosseous muscles. The dog was discharged with a protective boot for the right hind foot. A rehabilitation protocol, including passive range-of-motion exercises.
and twice-daily neuromuscular electrical stimulation of the quadriceps and posterior muscles of the right hind limb, was performed during the reinnervation process and convalescent period.

Three months after the surgery, the dog still had paresis in the right hind limb, but evidence of paresthesia was observed in the foot. Electromyographic evaluation was repeated 10 months after the surgery. At that time, the dog's neurologic status was dramatically improved. Paresthesia in the foot was no longer evident, but mild, intermittent lameness was observed when the dog moved at a trot. Amyotrophy of the cranial tibial and interosseous muscles was still palpable. Neurologic examination revealed normal postural reactions except for mild hypermetria during elicitation of the hopping reaction in the right hind limb. The cranial tibial reflex was intact, although diminished. The withdrawal reflex was complete in the stifle joint and was diminished in the hock and the digits. Electromyography revealed no abnormal electrical activity in the muscles of the affected limb. The M wave was elicited upon electrical stimulation of the tibial and peroneal nerves and was also recorded in the interosseous and cranial tibial muscles. Wave amplitudes were low for the tibial nerve (1.3 and 1.9 mV, respectively, for the distal and proximal stimulation sites at a stimulation voltage of 48 V; results for the left tibial nerve evaluated as a control were 44.9 mV amplitude. Nerve conduction velocity was 46 m/s.

Figure 3—Motor nerve conduction studies of the right peroneal nerve recorded during the first electromyographic evaluation (A) and 10 months after nerve graft surgery (B) in the same dog as in Figure 2. Upper traces were in response to stimulation of the distal segment of the nerve, and the lower traces were in response to stimulation of the proximal portion of the nerve. In panel A, notice the absence of recordable compound muscle action potentials (M waves). In panel B, notice that M waves were recorded upon nerve stimulation at both the proximal and distal sites. Also notice the polyphasic morphology of the M waves, including prolonged duration and reduced amplitude. Nerve conduction velocity was 46 m/s.

Discussion

Sciatic nerve injury in veterinary patients usually occurs secondary to pelvic or femoral fractures. Traumatic injury from bite wounds is less frequently reported. In France, hunting dogs are occasionally injured by wild boars. The sciatic nerve, the largest of the peripheral nerves, is relatively well protected from injury in its proximal extent (deep to the bone and heavy muscle masses), but is more vulnerable in the distal third of the femoral shaft, where it separates into the tibial and peroneal nerves.

Nerve damage should be evaluated by means of complete neurologic examination and electrophysiologic testing. Electromyographic and nerve conduction studies allow further characterization of structural nerve damage and diagnosis (eg, neuropraxia, axonotmesis, or neurotmesis). In the present study, spontaneous electromyographic activity was detected in the musculature of the distal portion of the right hind limb (eg, cranial tibial, gastrocnemius, and interosseous muscles), but not in the more proximally located muscles (eg, glutaeal, biceps femoris, semitendinosus, and semimembranosus muscles), indicative of a lesion distal to the sciatic nerve collaterals that innervate the proximal muscles (eg, those on the distal third of the femoral shaft). The fact that tibial and peroneal nerve M waves were absent, but that the semitendinosus and semimembranosus muscles contracted upon stimulation of the proximal segment of the sciatic nerve, suggested that there was a neurotmesis lesion in the same region. Such lesions are associated with a poor prognosis.

Alternative treatments to amputation in companion animals with sciatic neuropathy include hock-foot orthosis, in combination with or apart from peripheral nervous system surgery. Hock-foot orthosis has been described as improving patients’ willingness to ambulate, but complications associated with chronic tibio-tarsal hyperflexion can develop. When possible, surgical repair of sciatic neuropathy should be considered as a therapeutic option, regardless of how much time has elapsed since the original injury. Surgical exploration consists of detecting and exposing the proximal (neuroma) and distal (glioma) injured nerve segments and exposure of viable nerve endings.
In the authors' experience, if the length of the neuronal defect is more than 5 times the nerve diameter, nerve grafts should be used to fuse the proximal and distal stumps and allow functional recovery. In the dog of this report, the gap between the proximal and distal stumps of the sciatic nerve branches after dissection was 80 mm in length.

The contralateral CSN was chosen for grafting because it has been used experimentally as a source of autologous nonvascularized peripheral nerve for grafting procedures in dogs. The length and diameter of the CSN are appropriate for use in grafting procedures involving nerve gaps. In 1 study involving 67 dissected CSNs, the length of peripheral nerve graft that could be harvested ranged from 80 to 460 mm, and the mean nerve cross-section ranged from 1.8 to 3.0 mm. The CSN meets 2 criteria that are important in peripheral nerve grafting. The nerve's position beneath the subcutaneous tissues of the medial femoral region makes it easily accessible. Further, it is a sensory branch of the femoral nerve and supplies the skin on the medial aspect of the femoral region and on the dorsomedial aspect of the tarsus and metatarsus; harvest of the CSN, therefore, should lead to minimal postoperative discomfort and neurologic deficits. Adverse effects associated with removal of the CSN in the dog of this report were limited to definitive loss of cutaneous sensation in the medial femoral region.

Nerve grafting is similar in technique to perineurial fascicular repair. Fascicles in the proximal nerve segment are connected to corresponding fascicles in the distal segment by 1 or more grafts. We used 2 fascicles to graft the tibial nerve and 1 fascicle to graft the peroneal nerve. The number of graft segments needed can vary, but the cross-sectional area of all grafts should approximate that of the nerve to be grafted. After surgery, the limb should be protected for 7 to 10 days with semirigid external fixation before full movement of the limb is permitted. In the present report, the nerve graft length was of sufficient length that tension on the nerve was minimal irrespective of the limb's position. The dog's activity was limited during the 10 days after surgery, but normal activity was permitted after that period.

Accurate prognostication of the degree of functional recovery that may be expected after repair of an injured nerve is difficult. Many factors influence the quality of recovery after nerve repair, including patient age, timing of repair, type of injury, surgical technique, suture material used, length of the nerve defect, and nature of the soft tissue bed surrounding the repaired nerve. Healing, via axonal regeneration of nerve fibers from the proximal stump, occurs at a rate of 1 to 4 mm/d. In older animals, the rate of axonal regeneration is slower and the density of regenerating axons is lower. In free nerve grafts placed in a healthy soft tissue bed, revascularization begins on day 3 after implantation and progresses rapidly thereafter, resulting in adequate blood flow to support nerve growth by 1 week after reimplantation. In the dog of this report, the wound was aseptic and scar tissue was removed during the surgery, leaving a healthy bed of soft tissue around the graft. In humans, nerve grafts longer than 60 mm are correlated with poorer outcomes. In veterinary medicine, the prognosis for return to function is optimal if the length of nerve to be regenerated is less than 100 to 150 mm and minimal if the regeneration distance is longer than 250 to 300 mm. With regard to the 80-mm nerve defect in the dog reported here (length of regeneration, 240 mm; nerve grafts' length, 90 mm), the prognosis was guarded. The young age of the dog may have contributed to the favorable outcome. During the long regrowth period of the nerve over the graft to its end targets, irreversible muscle atrophy and fibrosis may develop in addition to joint ankylosis and stiffness. Functional recovery is directly related to the degree of muscle atrophy. Physical therapy, including neuromuscular electrical stimulation and active and passive range-of-motion exercises, should be initiated as early as possible.

Few investigations dealing with the neurophysiologic recovery of peripheral nerves after repair with an autogenous nerve graft have been reported in humans or dogs. Electromyography may provide neurophysiologic evidence of reinnervation, but the disappearance of spontaneous activity can also be observed in association with end-stage fibrosis. Nerve conduction studies yield definitive evidence of the muscle reinnervation. In the dog of this report, clinical improvement was obvious given the dog's ability to voluntarily flex the stifle joint 10 months after surgery, although motion in the hock was uncertain. At that time, disappearance of spontaneous muscle activity and recordable small M waves in the right interosseous and tibial cranial muscles were evidence of reinnervation and functional recovery of the distal limb muscles. In 1 study of the reinervation process in rats, spontaneous activity in muscles disappeared 10 weeks after regeneration began.

A compound muscle action potential is typically composed of a diphasic or triphasic waveform of short duration, resulting from simultaneous recruitment of all the muscle fibers innervated by the stimulated axons. In the dog of this report, nerve conduction studies and analysis of M-wave morphology yielded clues for assessing reinnervation. The polyphasic morphology of peroneal M waves is a result of desynchronized electrical activation of muscle fibers and primarily reflects differences in the conduction velocity and terminal length in newly formed individual axons. Low amplitudes of tibial and peroneal nerve M waves were a result of partial restoration of the number and size of nerve fibers. After nerve anastomosis, regenerating nerve fibers gradually increase in number over a period of years, although neither the original number nor diameter of fibers is restored. In humans, conduction velocity also increases slowly after anastomosis, reaching 60% of normal velocity within 4 years and a mean velocity of 85% of normal values after 16 years. In cats, 2-cm-long grafts are functional and retain their integrity and individual identity 6 to 12 months after surgery.

Regeneration of nerve fibers is frequently associated with other effects such as paresthesia. As a result of misdirected regrowth of axons, neuroma formation and spontaneous discharges of nerve impulses may occur. Spontaneous discharges may result from accumulation of sodium channels in the cell membranes of injured axons, resulting in axonal hyperexcitability.
Without treatment, neurotmesis of the proximal segment of the sciatic nerve results in poor return to motor and sensory functions of the affected limb and often leads to amputation. Nerve grafting appears to offer a viable treatment option. Early repair is recommended, but should not be undertaken until wound sepsis has resolved. The sensory branch of the CSN is a potential source of autologous nonvascularized peripheral nerve graft in dogs. Despite improvements in microsurgical techniques, the prognosis for restoration of function remains guarded in dogs with traumatic nerve injury, although electrophysiologic testing can aid in assessing initial injury and reinnervation. Physical therapy should be provided, and the owner must be committed to the postoperative recovery process over a long period of time.

References