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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 monoplegia 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. If a nerve defect exists, nerve grafting should be considered, 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)

tion confirmed abnormal postural reactions in the limb in addition to a diminished withdrawal reflex in the stifle joint and absent flexion of the hock joint. The cranial tibial reflex was also absent. The character of the patellar reflex was consistent with pseudohyperreflexia. Cutaneous anesthesia was detected on the dorsal and plantar surfaces of the paw. These findings prompted a tentative diagnosis of a sciatic nerve lesion involving the tibial and peroneal nerve branches.

The radiographic appearance of the femur and tibia was considered normal. Electromyography[a] to characterize the sciatic nerve damage was performed with the dog anesthetized. Anesthesia was induced with IV administration of propofol[b] (6.5 mg/kg [3.0 mg/lb]) and maintained with inhaled isoflurane[c] and oxygen. Body temperature was maintained above 38.0°C (100.5°F) during electromyographic testing with a covered warming pad placed under the dog. During electromyography, spontaneous electrical activity (fibrillation potentials and positive sharp waves) was recorded, regardless of the position of the needle electrode, in the interosseous, gastrocnemius, and cranial tibial muscles of the affected limb. Motor nerve conduction studies consisted of distal and proximal stimulation of the tibial nerves, at locations proximal to the tarsus and caudomedial to the greater trochanter of the femur, respectively. Compound muscle action potentials (M waves) were recorded in the interosseous muscles. Distal and proximal stimulation of the peroneal nerves was conducted at the caudal aspect of the lateral head of the fibula and caudomedial to the greater trochanter, respectively. Compound muscle action potentials were recorded in the cranial tibial muscles. Motor nerve conduction velocities were calculated by dividing the distance between the proximal and distal points of stimulation by the proximal and distal M waves' latency difference. Motor nerve conduction studies revealed an absence of recordable tibial or peroneal M waves in the right cranial tibial and interosseous muscles after stimulation at proximal and distal sites on each nerve. Contraction of the semitendinosus and semimembranosus muscles was observed in response to stimulation of a proximal segment of the sciatic nerve. Neurotmesis of the tibial and peroneal nerves in the distal third of the femoral shaft was suspected.

Exploratory surgery was performed so that the extent of damage to the nerves could be determined. The skin on the lateral aspect of the right stifle region was aseptically prepared for surgery, and the skin of the

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 nonabsorbable monofilament[d] material in an epineural-perineural technique under an operating microscope. The site was closed routinely. The total nerve regener-

ation 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
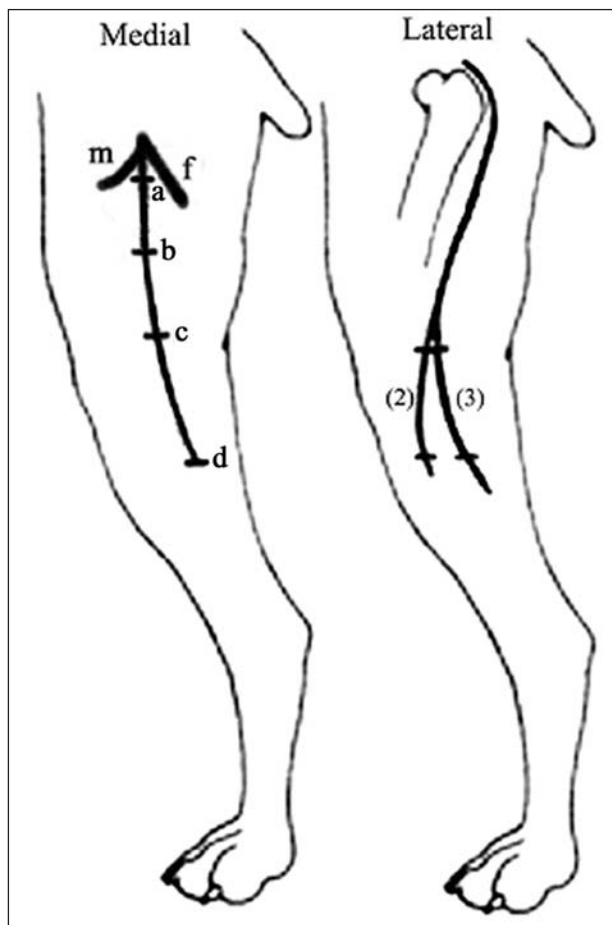

Figure 1—Schematic illustration of the medial and lateral aspects of the hind limb of a dog in the area of dissection of the CSN for use as a nerve graft. Possible sites (a, b, c, d) of nerve sectioning are indicated. The portion of the nerve distal to point d is too small in diameter for use as a graft. On the lateral aspect of the limb, the sites where the peroneal (2) and tibial (3) nerve defects were grafted in the dog of this report are indicated. m = Muscular branch of the saphenous nerve. f = Femoral nerve.

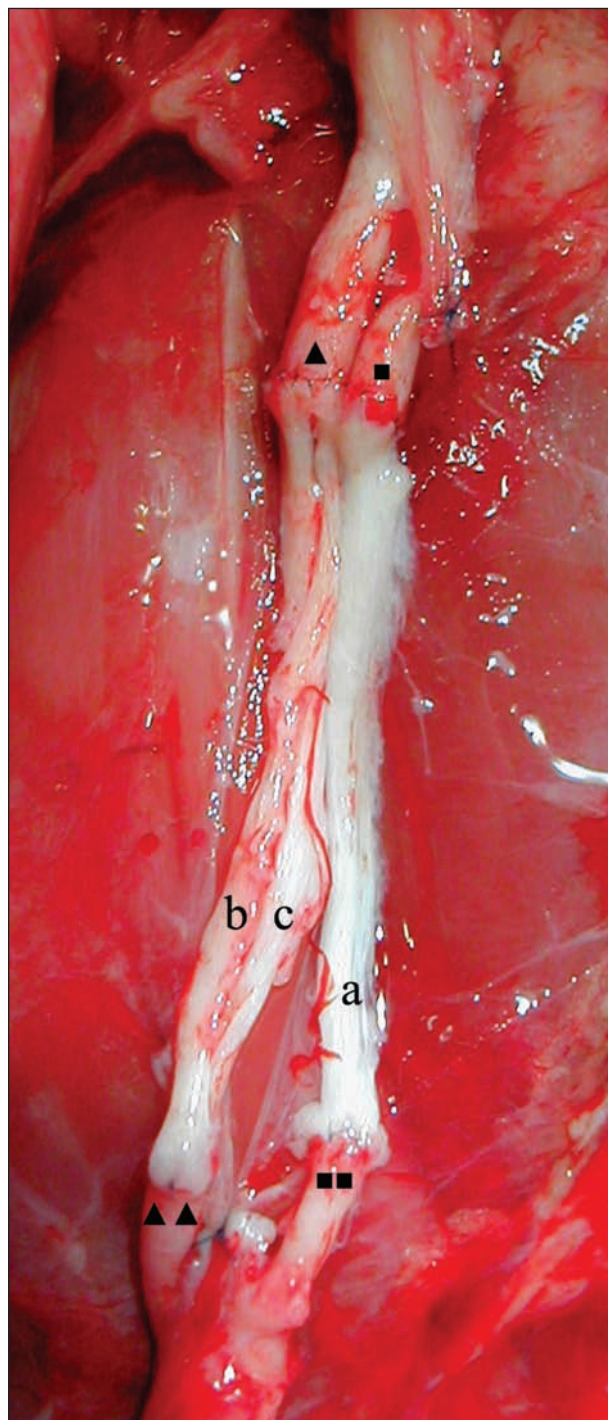Figure 2—Photograph of the site of nerve grafting during surgery in a dog with traumatic injury of the tibial and peroneal branches of the sciatic nerve in the right hind limb. The first nerve graft (a) was interposed between the proximal (black square) and distal (twin squares) peroneal nerve stumps, and the 2 other grafts (b,c) were interposed between the proximal (black triangle) and distal (twin triangles) tibial nerve stumps.
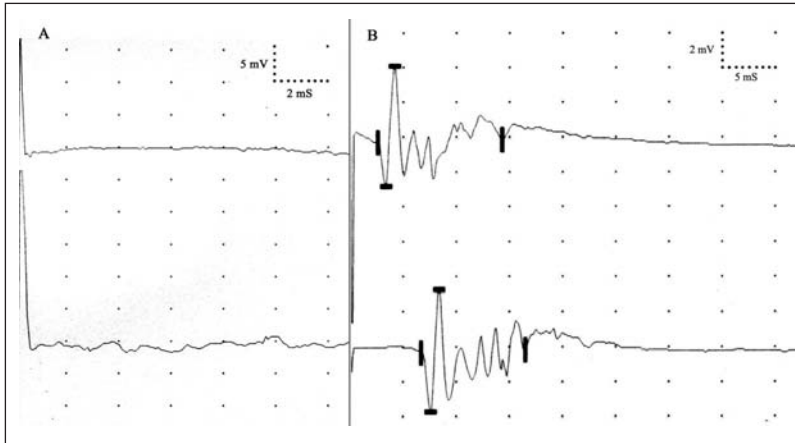
Figure 3—Motor nerve conduction studies of the right peroneal nerve recorded during the first electromyographic evaluation (A) and 10 months after nerve graft surgery (B) in the same dog as in Figure 2. Upper traces were in response to stimulation of the distal segment of the nerve, and the lower traces 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.

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 for both the distal and proximal nerve sites at a stimulation voltage of 32 V) and the peroneal nerve (wave amplitude of 7.0 mV for both the distal and proximal sites at a stimulation voltage of 128 V; results for stimulation of the left [unaffected] peroneal nerve were amplitudes of 30.4 and 29.1 mV, respectively, for the distal and proximal sites at a stimulation voltage of 42 V). The M waves of the peroneal nerve were polyphasic and of prolonged duration (duration between the first and last phase, 11.7 and 9.8 ms, respectively, for the distal and proximal sites; duration for the left peroneal

nerve, evaluated as a control, 7.1 and 7.3 ms, respectively, for stimulations at the distal and proximal sites; **Figure 3**). Duration of the right tibial nerve M wave was also increased (4.2 and 9.6 ms, respectively, for the distal and proximal stimulations; results for the left tibial nerve, evaluated as a control, 2.4 and 3.3 ms, respectively, for the distal and proximal stimulations). Nerve conduction velocities were low for the right tibial and peroneal nerves (35 and 46 m/s, respectively; velocities for the contralateral tibial and peroneal nerves, 63 and 84 m/s, respectively).

Discussion

Sciatic nerve injury in veterinary patients usually occurs secondary to pelvic or femoral fractures.[1,2] Traumatic injury from bite wounds is less frequently reported.[1] 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.[3]

Nerve damage should be evaluated by means of complete neurologic examination and electrophysiologic testing.[4-6] Electromyographic and nerve conduction studies allow further characterization of structural nerve damage and diagnosis (eg, neuropraxia, axonotmesis, or neurotmesis).[4-6] 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, gluteal, 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.[4,5,7]

Alternative treatments to amputation in companion animals with sciatic neuropathy include hock-foot orthosis, in combination with or apart from peripheral nervous system surgery.[8,9] Hock-foot orthosis has been described as improving patients' willingness to ambulate, but complications associated with chronic tibiotarsal hyperflexion can develop. When possible, secondary surgical repair of sciatic neuropathy should be considered as a therapeutic option, regardless of how much time has elapsed since the original injury.[10] 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.[11] The length and diameter of the CSN are appropriate for use in grafting procedures involving nerve gaps. In 1 study[11] 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.[12] 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.[13] 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 perineural fascicular repair.[7,10] 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.[10] After surgery, the limb should be protected for 7 to 10 days with semirigid external fixation before full movement of the limb is permitted.[10] In the dog of 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.[7,10] Healing, via axonal regeneration of nerve fibers from the proximal stump, occurs at a rate of 1 to 4 mm/d.[4,14] In older animals, the rate of axonal regeneration is slower and the density of regenerating axons is lower.[15] 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.[16] 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.[17] 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.[18] 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.[19,20] Physical therapy, including neuromuscular electrical stimulation and active and passive range-of-motion exercises, should be initiated as early as possible.[21]

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[22] of the reinnervation 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.[23] 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.[4] 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.[24,25] In cats, 2-cm-long grafts are functional and retain their integrity and individual identity 6 to 12 months after surgery.[26]

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.[4,7] Spontaneous discharges may result from accumulation of sodium channels in the cell membranes of injured axons, resulting in axonal hyperexcitability.[27]

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.

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a.   Viking IV, D Nicolet Biomedical Inc, Madison, Wis.
b.   Rapinovet, Schering-Plough Vétérinaire, Levallois-Perret, France.
c.   Isoflurane Belamont, Mundipharma SAS, Boulogne Billancourt, France.
d.   Prolene polypropylene monofilament, Ethicon SAS, Issy les Moulineaux, France.

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