Patient outcome after common peroneal nerve decompression

DOUGLAS B. HUMPHREYS, M.D., F.R.C.S.C., CHRISTINE B. NOVAK, M.S.,
AND SUSAN E. MACKINNON, M.D., F.R.C.S.C.

Division of Plastic and Reconstructive Surgery, Washington University School of Medicine,
St. Louis, Missouri

Object. This study examines common peroneal nerve decompression and its effect on nerve function.

Methods. Fifty-one peroneal nerve decompressions were retrospectively reviewed. All patients were evaluated preoperatively and postoperatively for motor and sensory function of the peroneal nerve as well as for pain.

Results. Postoperatively, 40 (83%) of 48 patients who had preoperative motor weakness had improvement in motor function. Likewise, 23 (49%) of 47 patients who had sensory disturbances and 26 (84%) of 31 patients who had preoperative pain improved after surgical decompression of the peroneal nerve.

Conclusions. Common peroneal nerve decompression is a useful procedure to improve sensation and strength as well as to decrease pain. (DOI: 10.3171/JNS-07/08/0314)

KEY WORDS • common peroneal nerve • compression neuropathy • decompression • outcome

Compression neuropathy is well documented in the literature. The results of chronic nerve compression have been studied and the pathophysiology of nerve compression is well understood. The changes seen in the nerves are dependent on the amount and duration of compression imposed on the nerve. These changes range from breakdown of the blood–nerve barrier, to subperineurial edema, to fibrosis and demyelination, and eventually Wallerian degeneration.

Common peroneal nerve neuropathy is not unlike carpal tunnel syndrome and can be thought of in a similar fashion. The end result of the neuropathy in both cases includes pain, numbness, and motor weakness. Peripheral nerves are particularly vulnerable to compression at specific anatomical areas. It is well established that the median nerve can become compressed at various points along its course; the most frequent site of compression occurs at the transverse retinacular ligament, causing carpal tunnel syndrome. Similarly, the common peroneal nerve can become entrapped at the neck of the fibula. Surgical exploration of the common peroneal nerve at the fibular head reveals distinct anatomical areas where the nerve can be compressed. The hypothesis of this study is that operative intervention in common peroneal nerve neuropathy favorably affects motor function, sensory function, and pain relief.

Clinical Material and Methods

Following institutional Human Studies Committee approval for this study, data were obtained through a retrospective review of all charts of patients who underwent

Abbreviations used in this paper: BMI = body mass index; MRC = Medical Research Council.

ERONEAL nerve compression neuropathy is a well-recognized entity that results in a variety of symptoms, including foot drop due to paralysis of the affected musculature as well as sensory disturbances over the lateral side of the lower extremity extending onto the dorsum of the foot. The diagnosis of compression neuropathy is made based on an understanding of the anatomy of the peroneal nerve. Surgical decompression of the common peroneal nerve at the fibular head has been described but results of that decompression procedure are rarely found in the literature.

The peroneal nerve arises from the L-4 to S-2 nerve roots. It is the lateral and smaller terminal division of the sciatic nerve. The sciatic nerve separates into the tibial and common peroneal nerves in the distal, posterior thigh. The common peroneal nerve subsequently travels around the fibular neck and then passes between the two heads of the peroneus longus muscle. The common peroneal nerve then divides into both the superficial and deep branches. The superficial branch supplies motor function to the peroneus longus and brevis muscles and then terminates as the sensory branch supplying sensation to the anterolateral skin of the leg and the dorsum of the foot. The deep branch supplies motor innervation to the tibialis anterior, extensor digitorum longus, extensor digitorum brevis and extensor hallucis longus muscles. The deep branch terminates as the sensory branch, which innervates the dorsal skin between the first and second toes.
common peroneal decompression by one surgeon between June 1, 1995, and July 31, 2001. A total of 48 patients underwent 51 peroneal nerve decompressions. Excluded from this review were three patients; two were lost to follow-up after their initial postoperative visit, and one was diagnosed with amyotrophic lateral sclerosis within weeks of surgery. All patients underwent common peroneal nerve decompression at the fibular head performed by the same surgeon (S.E.M.). No other ancillary procedures were performed at the same time (tarsal tunnel release or decompression of the deep peroneal nerve at the ankle). All patients were assessed preoperatively and postoperatively for motor function (strength), sensation, and pain.

Clinical Assessment

Basic health information was collected for each person. Each patient completed a standardized pain questionnaire, which included a self-assessment ranking of pain on a 10-cm visual analog scale as well as indicating pain distribution on a body diagram. All patients had motor and sensory function of the peroneal nerve clinically evaluated at their initial office visit. Motor function to the ankle dorsiflexors and evertors was assessed using the MRC scale. If motor function varied between the muscle groups supplied by the peroneal nerve, the lower value was assigned. Sensation was evaluated using the ten test. For this test, the patient ranks sensation on a scale of one to 10, with 10 equivalent to normal sensation. The ten test has been shown to have interobserver reliability for sensory testing with a correlation coefficient of 0.9148, with a 95% confidence interval of 0.8731 to 0.9432. The patients included in this series were evaluated for the possibility of peroneal nerve compression. Tinel signs were present at the fibular head in the vast majority of patients. This patient population was predominantly composed of three groups: those who had no identifiable cause of their peroneal nerve palsy (12 cases), patients who had previous orthopedic procedures (nine cases), and patients who had some trauma to the leg (27 cases). The patients who had had previous orthopedic surgeries were thought to have direct compression over the peroneal nerve at the fibular head from positioning or a stretch injury from retraction. Those who had had trauma to the lower extremity were believed to have a recoverable injury (Sunderland first-, second-, or third-degree), either by electrodiagnostic findings that demonstrated the presence of motor unit potentials suggesting continuity of the injury, or by the presence of a Tinel sign at the fibular head. Patients who on clinical examination had a very proximal Tinel sign not near the fibular head or who had electrodiagnostic studies showing no evidence of reinnervation in association with a history of severe trauma to the knee, such as that associated with severe ligamentous knee injury, were treated with nerve grafts or tendon transfers and were not included in this study.

Surgical Technique

All surgical procedures were performed after induction of general anesthesia with tourniquet control and 4.3 loupe magnification. The patients were positioned supine on the operating table with their affected leg flexed at the knee to approximately 60°. The incision was centered obliquely just below the fibular neck (Fig. 1a). Placement of the incision was critical to the procedure as it allowed easy access to the peroneal nerve both proximally and distally (Fig. 1b). The incision was carried down through the subcutaneous tissues while protecting the lateral sural cutaneous nerve and posterior femoral cutaneous nerve branches. The fascia was carefully opened to expose the common peroneal nerve (Fig. 1c) that is located proximally, posterior to the biceps femoris tendon.

The common peroneal nerve was followed around the fibular neck as it passed distally between the two heads of the peroneus longus muscle. The fascia overlying the peroneus longus muscle was divided (Fig. 1d) and the muscle was retracted distally (Fig. 1e). The muscle itself did not need to be divided, because once the fascia was opened it was easily retracted distally to provide exposure to the peroneal nerve. At this point the nerve typically made a sharp directional change. There was usually a fascial band directly over and under the nerve in this area that was considered the major site of compression. This fascia was excised (Fig. 1f) to allow the nerve to lie flat and without tension. The path of the nerve was traced distally into the substance of the lateral and anterior compartment muscles to ensure that there were no fascial elements, which would cause compression on the nerve. Frequently, there were separate fibrous bands within the muscle that compressed the nerve. If the nerve was elevated by the gastrocnemius muscle as it curved around the fibular neck, then a bed was created within the muscle to allow the nerve to run in a smoother, straighter course. As long as bands of Fontana were visible along the course of the nerve, a neurolysis was not performed; otherwise, a neurolysis was performed using micro-instrumentation.

Once all possible points of compression were released, the tourniquet was deflated and hemostasis was ensured. At this point, the nerve was stimulated for 10 to 15 minutes using a portable nerve stimulator (Vari-Stim III; Medtronic) at maximum intensity if patients had a motor deficit. The wound edges were infiltrated with long-lasting local anesthetic (bupivacaine) and then closed in a layered fashion. The lower extremity was dressed in a large bulky dressing for comfort. The patient’s leg was not splinted and ambulation with crutches was allowed immediately after surgery. Patients were examined within 48 hours of surgery at which time the original dressing was removed. Instructions were given about range of motion exercises for nerve gliding, a technique used to prevent scarring of the nerve to the surrounding tissues, and patient therapy was advanced, as tolerated, to full weight-bearing ambulation.

Data Analysis

Data were entered into a spreadsheet and analyzed using Statistica version 6.1 (StatSoft, Inc.). Means and standard deviations were calculated for demographic data. The relationship between preoperative and postoperative findings was evaluated using the sign test. The Fisher exact test was used to evaluate BMI and postoperative outcomes.

Results

A total of 51 common peroneal nerve decompressions were performed in 48 patients. The average patient age at the time of surgery was 41 ± 19 years. There were 24
right peroneal nerve decompressions and 27 left peroneal decompressions performed. Twenty of the patients were female and 28 were male; two females and one male had bilateral peroneal nerve decompressions performed at separate surgeries. The cause of the peroneal nerve compression was attributed to leg trauma in 27 patients, a surgical procedure in nine cases and no identifiable cause in 12 cases. The patients who had no identifiable cause for their peroneal nerve palsy reported onset of motor and/or sensory dysfunction with no obvious inciting event. Orthopedic procedures were associated with the majority of patients who developed a peroneal nerve palsy (total hip replacements, total knee replacements, and back surgery). No patients had open injuries that resulted in a neurotmetic injury, such that a nerve graft would be necessary. The patients in this series had compression or stretch from positioning or intraoperative traction. The average length of time from the onset of symptoms to surgery was 16 months (range 1–98 months).

Preoperatively, 47 of 51 lower extremities had weakness in the peroneal nerve muscles. Twenty-one of these patients had no motor function in the peroneal nerve innervated musculature (MRC Grade 0/5). Postoperatively, 40 (85%) of the 47 patients who had motor dysfunction preoperatively were noted to have improvement in their motor function (p < 0.01). Twenty-four (60%) of the 40 people who had improved motor function had MRC Grade 4/5 or 5/5 muscle strength. No patient had his or her motor function downgraded because of the surgery. Several patients recovered motor function within hours to days following surgery, which would suggest a neurapraxic conduction block-type injury. The majority of patients recovered over several months suggesting a Sunderland II or III degree injury that recovered with nerve regeneration at the typical rate of 1 inch per month.

Sensory disturbances were identified in 47 (92%) of the 51 peroneal nerve decompressions performed. Eleven of these patients reported no sensation in the peroneal nerve distribution whereas three reported hypersensitivity. Postoperatively, 23 of 47 patients reported improved sensation (p < 0.01). Of these 23 patients, 18 reported normal sensation in the peroneal nerve distribution. No patients had their sensation downgraded because of surgery.

When the relationship between postoperative recovery and BMI was evaluated it was found that patients who were overweight (BMI 25–29.9 kg/m²) and obese (BMI > 30 kg/m²) had more sensory postoperative disturbances than those patients who had an ideal body weight (BMI 20–24.9 kg/m²) or were thin (BMI < 20 kg/m²) (p < 0.01). No similar relationship was found for postoperative motor function.

Postoperatively, 31 (61%) of the 51 peroneal nerve decompressions were performed for pain relief. After surgery 26 (84%) of these 31 had improvement in their pain (p ≤ 0.01). Nineteen of these 26 patients reported complete resolution of their pain postoperatively. No patient’s pain increased as a result of surgery.
Patient outcome after common peroneal nerve decompression

Discussion

The peroneal nerve is the most common peripheral neuropathy of the lower extremity. The causes of peroneal palsy are multiple. Oppenheimer first reported peroneal nerve injury due to inversion ankle sprains, which tense the peroneus longus at the fibular neck producing increased traction on the peroneal nerve across the knee. There are many other causes of peroneal nerve palsy that have been reported including surgery (total knee arthroplasty, total hip arthroplasty, and high tibial osteotomies), habitual leg crossing, soft tissue tumors, ganglion cysts, Baker cysts, recent weight loss, squatting, and intermittent sequential pneumatic compression. This report describes a group of patients with compression neuropathy and not patients with direct neurotraumatic injuries to the peroneal nerve, who would require nerve repair, grafting, or nerve transplantation for reconstruction.

Compression neuropathies typically develop at the point where nerves cross joints. The peroneal nerve is no exception. The superficial position of the nerve as it passes around the fibular neck makes it vulnerable to trauma, crushing, and compression injuries. The peroneal nerve has larger fascicles with limited connective tissues compared with the tibial nerve, the other terminal branch of the sciatic nerve. In addition, the peroneal nerve is fixed both proximally and distally at the sciatic notch and the fibular neck. Where it crosses the joint, the peroneal nerve must make several directional changes to move from the back of the joint to the anterior and lateral compartments of the leg. Two relatively right-angle turns in proximity are likely to add to susceptibility of compression neuropathy. A cutaneous incision the surgical procedure is the precise preoperative marking of the incision, and typically the surgical incision is marked too far proximally. The surgeon needs to identify accurately the fibular head and mark the incision below the fibular head. The compression point is where the nerve passes around the fibular head into the muscle belly. There is a tendinous band directly above and below that needs to be excised and frequently there are separate tendinous bands within the muscle that need to be excised. In the patient sample that complained predominantly of pain, a small number may have had a period of significant pain relief followed by recurrence of pain. This occurs as the nerve regenerates distally in the leg and becomes entrapped along the course of the superficial peroneal nerve in the distal leg, where it can have its own separate tunnel, or more distally over the dorsum of the ankle where the deep peroneal nerve courses below the short extensor to the great toe. Clinical evaluation of these patients will show the presence of a Tinel sign at these entrapment points, and surgical release at these points will predictably relieve pain.

The senior author (S.E.M.) has had the opportunity to reexplore several peroneal nerves that have undergone previous decompressions. In these situations the release has typically not been carried out far enough distally. The nerve has been released at the noncompression point just proximal to the fibular head, and the release has not been carried distally enough into the peroneus musculature to achieve an adequate release. Finally, in patients with peroneal nerve dysfunction and a Tinel sign at the fibular head, consideration of a peroneal nerve decompression is appropriate. The presence of the Tinel sign at the fibular head suggests neural compromise at this location, just as a Tinel sign over the carpal tunnel would be an indication for carpal tunnel release in a patient complaining of median nerve dysfunction in the hand.

Peripheral neuropathies can be the result of chronic nerve compression, or due to an acute or chronic injury superimposed on subclinical nerve compression. Upton and McComas first introduced the idea of a “double crush” hypothesis in 1973. Their hypothesis is that an area of proximal nerve compression can cause distal sites to be more susceptible to nerve compression. The summation of compression along the entire nerve causes changes in the axoplasmic transport within the nerve, thus ultimately affecting nerve function. Each of these areas of constriction does not need to be sufficient enough to cause compression on its own but the additive effects of all the areas can be sufficient enough to result in clinical nerve dysfunction. This concept has been expanded to include the idea that a proximal nerve injury (excessive stretching) can render the distal nerve more susceptible to nerve compression. Also, systemic diseases such as diabetes can result in the nerve becoming more susceptible to compression.

Closed peroneal nerve injuries are typically treated nonoperatively. Many of these injuries do recover spontaneously over a period of weeks to months. Unfortunately, there remain a number of patients who do not recover and are left with persistent motor dysfunction, sensory disturbances, and pain. Historically, motor dysfunction was treated with splints and tendon transfers to help alleviate the foot drop associated with this injury and allow easier ambulation. Sensory and pain issues are treated medically or frequently go untreated as well. When nerves regenerate there is a tendency for any potential distal site of entrapment to cause a blockage in regeneration. Just as the carpal tunnel can impede median nerve regeneration in the upper extremity, superimposed compression of the peroneal nerve at the fibular head can limit recovery of a more proximal injury.

Conclusions

Our retrospective review suggests peroneal nerve decompression at the fibular head is a valid and effective procedure. Our study confirms the remarkable motor recovery documented by Thoma and colleagues; both studies describe a patient population in which there was a long delay between the onset of the common peroneal nerve palsy and operative decompression. Despite this delay, both patient populations showed significant recovery in motor function. Some of the most debilitating consequences of a nerve injury are the pain and sensory disturbances associated with these injuries. In this study, significant improvement in sensation and pain after peroneal nerve decompression was also reported.

Peroneal nerve decompression is a procedure that has been described for many years and first scientifically documented in 1925. Despite this long history, the procedure is infrequently performed. In this study, surgical decompression of the peroneal nerve resulted in significant improvement in motor and sensory function and in pain.

References


Accepted November 6, 2006.
Address reprint requests to: Susan E. Mackinnon, M.D., Division of Plastic and Reconstructive Surgery, Washington University in St. Louis, 660 South Euclid, Suite 5401, St. Louis, Missouri 63110. email: mackinnons@wustl.edu.