

# Lower Extremity Nerve Trauma

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## Abstract

*Peripheral nerve injuries of the lower extremity (LE) are frequently encountered in orthopaedic practice. They can be traumatic or iatrogenic. Proper and timely diagnosis and treatment are the keys to optimizing outcomes. This paper reviews and discusses the basic anatomy and physiology of nerve injury and the current literature on the incidence, pathogenesis, diagnosis, management and outcomes of sciatic, femoral, peroneal, and tibial nerve injuries. The purpose of this review is to suggest a protocol for evaluation and management of LE nerve injuries.*

Peripheral nerve injuries of the lower extremity are frequently encountered in orthopaedic practice.<sup>1</sup> Traumatic or iatrogenic nerve injuries increase morbidity and affect functional outcomes. The rate of spontaneous recovery reported in the literature is highly variable, but a prevalence of unsatisfactory recovery is frequently described. A number of factors influence outcome. They include the nerve involved and the location of the lesion, the extent of the injury, and the timing of treatment. Iatrogenic

injuries may be preventable, and the surgeon should be aware of the risks of nerve injury during lower extremity orthopaedic procedures. Once an injury occurs, the only factors under the physician's influence are the timing and manner of intervention. This review provides orthopaedic surgeons a working knowledge of the diagnosis and management of lower extremity nerve injury so that optimal outcomes can be achieved.

## Gross Anatomy

The femoral nerve originates from the lumbar plexus, specifically from the posterior divisions of the ventral rami of the L2-L4 lumbar roots, and runs under the lateral margin of the psoas muscle. Approximately 4 cm proximal to the ilioinguinal ligament, the nerve runs on top of the iliacus but still under its fascia. It then passes deep to the inguinal ligament, lateral to the femoral artery, and after a few centimeters splits into multiple branches. It innervates the sartorius, pectineus, and quadriceps. Its sensory branches become the saphenous, the intermediate femoral cutaneous, and the medial femoral cutaneous nerves.

The sciatic nerve originates from the sacral plexus, specifically from the anterior division of the plexus (roots L4-S3) contributing to the tibial division, and from the posterior division of the plexus (roots L4-S2) contributing to the peroneal division. An important aspect of the sciatic nerve anatomy is that the peroneal division is more superficial and smaller than the tibial division, playing a role in its higher susceptibility to injury. The sciatic nerve exits the pelvis in the greater sciatic notch, inferior (and anterior) to the piriformis muscle. At this level, the sciatic nerve appears as one structure (peroneal and tibial enveloped in a common capsule). Anatomic variations are found in up to 48% of specimens, with the nerve passing above or through the piriformis muscle or splitting into its divisions in the pelvis and passing through or around the piriformis as two separate

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divisions.<sup>2</sup> The nerve then lies posterior to the short external rotators, posterior to the adductor magnus but anterior to the hamstring muscles, and passes deep to the long head of biceps femoris. In the thigh, the tibial division innervates the long head of the biceps femoris, the semitendinosus, and the semimembranosus. The peroneal division innervates only the short head of the biceps femoris. Just above the popliteal fossa, the common capsule is lost, and the sciatic nerve splits into the common peroneal (fibular) nerve and the tibial nerve. Branches of the common peroneal nerve and of the tibial nerve form the sural nerve, which provides sensory innervation of the dorsolateral foot.

The common peroneal nerve (CPN) continues along the medial margin of the biceps femoris, towards the posterior fibular head. It passes around the lateral aspect of the proximal fibula, through a fibrous tunnel composed of the peroneus longus aponeurosis and a superficial aponeurosis between soleus and peroneal fascia. This tethering is yet another factor that contributes to its higher injury rate. Under the peroneus longus, the nerve then divides into the superficial and deep components. The superficial peroneal nerve runs under the peroneus longus and innervates it and the peroneus brevis. The superficial peroneal nerve provides sensory innervation to the dorsum of the foot. The deep peroneal nerve runs in the anterior compartment and innervates the tibialis anterior, extensor hallucis longus, extensor digitorum longus, and peroneus tertius. In the foot, it also innervates the extensor hallucis brevis, the extensor digitorum brevis, and the first web space.

The tibial nerve lies in the popliteal fossa, lateral to the artery and vein. The tibial nerve then runs deep to the gastrocnemius and soleus but superficial to the deep posterior compartment. A sensory branch, the medial sural cutaneous nerve, contributes to the sural nerve and innervates the dorsolateral skin of the foot. The tibial nerve then continues to innervate the muscles of the posterior compartments of the leg. It also innervates the majority of the intrinsic muscles of the foot via the medial and lateral plantar nerves and is responsible for protective sensation of the sole of the foot.

### Microscopic Nerve Anatomy

On a microscopic level, nerves consist of several anatomic layers.<sup>3</sup> The individual axons of the nerve are surrounded by the endoneurium and subsequently bound together in fascicles by the perineurium. The individual fascicles are the smallest units of the nerve that can be manipulated with current surgical techniques. While considerable branching and fusion of these fascicles occur, there is also functional segregation, which allows for identification and surgical manipulation of fascicles specific to a certain muscle group.<sup>4</sup> The outer layer is termed the epineurium and has the most abundant connective tissue elements of the nerve. The epineurium is most easily manipulated surgically and allows for nerve repair.

In addition, some investigators identify the loose areolar tissue surrounding the nerve, termed “paraneurium” or

“mesoneurium.” This tissue permits gliding of the nerve and is the location of blood vessels that then pass through the epineurium to supply the nerve trunk. The presence of the mesoneurium and its characteristic blood supply assures that the nerve can generally be safely mobilized over a long course, thus permitting surgical dissection.<sup>4</sup>

### Pathology of Nerve Injury—Macroscopic

The mechanisms of traumatic nerve injury are varied. Simple lacerations are perhaps the easiest to manage because the nerve ends can often be primarily repaired. This is in contrast to segmental loss, crush, compression, and stretch events, which frequently result in a large zone of injury and are difficult to manage. Traction is one of the most common mechanisms associated with traumatic and iatrogenic nerve injuries. It has been shown that stretch of as little as 6% of nerve length can disrupt the blood supply and lead to injury.<sup>5,6</sup>

Iatrogenic nerve injury is of particular interest to the orthopaedic surgeon. Traction is likely the most common mechanism, but nerves also can be injured by sutures, injections, and orthopaedic hardware.<sup>7-9</sup> Limb lengthening can be a factor; for example, 2.7 cm to 4.4 cm is thought to be a threshold for causing sciatic nerve injury.<sup>10</sup> Tourniquet application has been implicated; animal studies demonstrate perineurial edema after 2 to 4 hours of compression and irreversible loss of nerve function after 6 to 8 hours. Two hours appears to be a safe time frame, as microcirculation is rapidly restored.<sup>11</sup>

### Pathology of Nerve Injury and Regeneration—Microscopic

Two major classifications of nerve injury exist.<sup>12,13</sup> Seddon classified injuries into three grades: neurapraxia, axonotmesis, and neurotmesis.<sup>12</sup> Neurapraxia is essentially a conduction block due to demyelination. It is the least severe with preserved axonal and connective tissue and a good prognosis. Axonotmesis is defined by the severing of axons but variable preservation of connective tissues. It has a variable prognosis. Neurotmesis is a complete severing of axon and connective tissue; recovery is not expected without surgical repair. Sunderland proposed a more precise classification in which grades 1 to 5 are used to describe the nerve injury.<sup>13</sup> Grade 1 corresponds to neurapraxia and Grade 5 to neurotmesis. Grades 2 to 4 represent axonal degeneration with varying degrees of connective tissue injury. Sunderland Grade 2 (axonal degeneration with connective tissue, nerve fiber, and nerve trunk continuity) has a good prognosis. In Grade 3, axonal degeneration and loss of endoneurium may occur, and nerve fiber continuity recovery may be incomplete. In Grade 4, axonal degeneration is coupled with loss of endoneurium, perineurium, and nerve fiber continuity; spontaneous recovery becomes less likely due to scarring (Table 1).

Wallerian degeneration is the process of degeneration that involves the axon and the myelin distal to the site of

**Table 1** Nerve Injury Classifications

Seddon Grade	Sunderland Grade	Structures Injured	Prognosis for Spontaneous Recovery
Neuropraxia	1	Myelin	Full
Axonotmesis	2	Myelin, Axons	Functional
	3	Myelin, Axons, Endoneurium	Incomplete
	4	Myelin, Axons, Endoneurium, Perineurium	None
Neurotmesis	5	Myelin, Axons, Endoneurium, Perineurium, Epineurium	None

injury. It begins immediately after the injury but is not completed until several weeks. It is important to understand the timeframe for this process, because it will affect results of electrodiagnostic testing as discussed later in this review. Once Wallerian degeneration is complete, recovery can occur provided the connective tissue architecture is preserved. After the myelin debris is cleared, the regenerating growth cone of the axon follows along a hollow tube of connective tissue and Schwann cells, known as bands of Bungner. A specialized chemical milieu is required for this process, with molecules, such as nerve growth factor and glial cell line-derived neurotrophic factor, implicated in the process.<sup>14,15</sup>

One of the major factors in the success of nerve recovery is time. The speed of nerve regeneration has been determined to be 1 to 3 mm per day.<sup>4,14</sup> As a result, distal nerve injuries recover rapidly, while proximal injuries have poor recovery potential. As much as 1% of ultimate function may be lost for every 6 days the nerve repair is delayed.<sup>5</sup> Further delay results in increased neuronal cell death and decreased neurotrophic ability of Schwann cells.<sup>4,14</sup> In addition, the target organs are prone to degeneration and atrophy. The neuromuscular junction requires trophic support by the nerves and through mechanisms that are not fully understood degenerates when denervated. Degeneration starts to occur at 6 to 18 months, although some useful functional recovery may still occur. Furthermore, muscle atrophy and fibrosis progress and become complete by 3 years.<sup>4</sup>

### Injury Assessment

Timely and accurate diagnosis is crucial to achieving good outcomes. The mechanism of injury must be noted, as it will affect clinical decision-making. A sharp, penetrating injury implies an acute laceration amenable to immediate repair. Conversely, a gunshot wound or a massive crush injury creates a large zone of injury for which delayed exploration may be warranted. Similarly important is the timing of the neurologic deficit. A delayed presentation implies an evolving lesion, such as a hematoma, and in these cases, an urgent nerve exploration has been advocated.<sup>16,17</sup> The presence of severe or unremitting neurogenic pain may be a sign of ongoing injury and an indication for surgical exploration.<sup>8,18,19</sup> A complete history will also include the time since injury, as this will impact the efficacy of repair and may lead the physician to suggest other treatments, such as

tendon transfers. Older age is associated with poorer prognosis.<sup>20</sup> Any comorbidities that may affect outcome, such as a scarred or dysvascular tissue bed, diabetes, or smoking, should be noted.<sup>4,20-24</sup>

A detailed motor and sensory exam should be performed. While light-touch sensation is the easiest to test, a complete examination includes assessment of two-point discrimination, sharp-dull sensation, and Semmes-Weinstein monofilament testing. For evaluation of muscle strength, the Medical Research Council (MRC) grading system should be used (Table 2). In the lower extremity, typically muscle strength of grade 3 to 4 is needed to achieve a satisfactory clinical outcome. More specific grading systems have been devised for individual nerves in the lower extremity and may be useful for assessment of outcomes.<sup>9,18,25-27</sup>

Imaging is an important modality in evaluation of nerve injury. Magnetic resonance imaging (MRI) is commonly available and is often performed as part of the general traumatic workup. MRI may be indicated acutely to help localize or diagnose a sharp laceration. When performed as part of follow-up, newer MRI techniques may allow for differentiation between neuropraxia, axonotmesis, and neurotmesis.<sup>28</sup> Ultrasound can also be a useful diagnostic modality. In CPN injuries related to knee dislocations, ultrasound was used to correctly diagnose neurotmesis injuries and define the extent of intraneural disruption, helping to guide treatment.<sup>29</sup>

Nerve conduction studies are the mainstay of the diagnostic techniques. Electrodiagnostics can be used to localize the lesion, to determine the extent of the injury, and to follow recovery. Timing of nerve conduction studies is important. Immediately after the injury, prior to completion of Wallerian degeneration, a nerve conduction study will demonstrate a conduction block at the site of the injury but continued

**Table 2** MRC Grades of Muscle Strength

Grade	Motor Function
0	No movement or contraction
1	Trace movement or fasciculations
2	Active motion with gravity eliminated
3	Active motion against gravity only
4	Active motion against some resistance
5	Full strength

conduction distally, due to the still functioning remaining axon. However, once Wallerian degeneration is complete after several weeks, there will be a loss of the distal conduction in a complete axonal injury. Persistent conduction at that time suggests preservation of axons (neuropraxia). On electromyography (EMG), findings such as fibrillations and positive sharp waves suggest muscle denervation due to an axonotmetic injury. Intraoperatively, a nerve stimulator may be used to help localize the injury, as well as assess recovery potential and guide surgical treatment. It has been suggested that at 3 months, spontaneous recovery potential is as high as 90% in the presence of action potential transmission across a neuroma; therefore, only neurolysis is indicated in those cases.<sup>5</sup> Conversely, lack of signal transmission indicates a need for neuroma excision and repair or nerve grafting.

### Overview of Treatment Options

Complete and technical descriptions of surgical treatment options for nerve injury are outside the scope of this review; however, a brief overview of treatment options is provided with detailed descriptions of etiology and outcomes for specific nerve lesions.

Observation as the primary treatment modality may be appropriate for injuries that have a high likelihood of being neurapraxic or if the deficit involves only pain or sensory loss. During observation, bracing and stretching may be needed to prevent contractures and permit ambulation. Serial clinical examinations should be combined with nerve conduction studies to demonstrate recovery. The indication for surgical treatment is lack of recovery after 2 to 5 months of close observation.

Surgical treatment ranges from simple to complex. Neurolysis involves freeing the nerve from scar tissue. It can be external or internal, during which individual nerve fascicles are dissected. Direct repair can be either fascicular or epineural. Epineural repair involves placing sutures in the epineurium, whereas fascicular repair attempts to align individual groups of fascicles together. Nerve grafting is employed if the gap in the nerve is so large that a direct tension-free repair is not feasible. Sural nerve autograft is commonly employed in the lower extremity, as it is easily harvested and has low donor site morbidity. In addition, allograft products are available. Multiple studies have shown that 6 cm is the upper length limit for successful nerve grafting in the lower extremity, although satisfactory results have been reported with gaps as long as 20 cm.<sup>30-33</sup> Nerve transfer utilizes a portion of an intact nerve to reinnervate a key muscle or muscle group. Because the nerve transfer is typically performed distally, it tends to shorten recovery time and minimize the problem of neuromuscular junction and muscle degeneration. It does, however, require a functioning donor nerve, and may result in weakening of the donor muscle group.<sup>34</sup> Free-functioning muscle transfer has been described for the lower extremity, and may provide motor power in the case of a chronic injury well past the time limit for nerve

regeneration.<sup>35</sup> Finally, tendon transfers are utilized to restore mobility and function. They can be used in situations in which functioning donor muscles are available (such as in isolated CPN injury) and can lead to good functional results with minimal donor site morbidity.<sup>36</sup>

### Femoral Nerve

Traumatic injuries to the femoral nerve are relatively rare, mostly due to the relatively protected anatomic position of the nerve.<sup>25,37</sup> In the setting of penetrating groin trauma, lacerations to the nerve can be associated with vascular injury. Fracture-related injuries are rare; Gruson and Moed<sup>35</sup> reported four cases of femoral nerve injuries out of 726 operatively treated acetabulum fractures; two of those cases were believed to be iatrogenic. Similarly, the overall rate of femoral nerve injuries in total hip arthroplasty (THA) is low: 0.13% in a large study encompassing 24,469 cases.<sup>38</sup> A higher rate (2.3%) may be associated with anterolateral approaches as reported by Simmons and coworkers.<sup>39</sup>

Injury to the femoral nerve results in the denervation of hip flexors and knee extensors. This results in loss of terminal knee extension and knee and patellar instability. Patients may have difficulty negotiating inclines and stairs and rising from a chair; however, ambulation ability is good due to the stabilizing effects of other muscle groups. Overall, the natural history of femoral nerve injuries, based on the literature, is favorable. In one series, all 10 patients spontaneously recovered good function by the end of follow-up.<sup>39</sup>

Despite the majority of iatrogenic femoral nerve lesions being “in-continuity,” recovery is not universal. Kim and colleagues<sup>23</sup> reviewed 10 patients with iatrogenic femoral nerve lesions who demonstrated no recovery after 6 to 12 months. Mechanisms of injury were postulated to be post-operative hematoma, cement extrusion, trauma from retractors, and bone or prosthesis malpositioning. Three patients were managed with neurolysis and one with a simple repair; these patients had good functional recovery. The remaining six patients had a large zone of no conduction and required nerve grafting; only two achieved a functional recovery.<sup>25</sup> The same investigators also described 19 patients with persistent femoral nerve lesions associated with hip or pelvic fractures. Nine were treated with neurolysis, and all nine improved. Two required repair, and eight required nerve grafting; only half had a good result. Overall, the investigators recommend exploration of the nerve if no recovery is noted after 3 to 4 months, although useful recovery could still be obtained at up to 14 months post-injury due to the relatively short distance between the lesion and innervated muscles.<sup>25</sup>

If nerve repair fails, a transfer of the biceps femoris and semitendinosus to the patella can be performed. The benefits of this transfer are to prevent lateral subluxation of the patella and to minimize knee buckling. In a series of seven patients reported by Fansa and Meric,<sup>40</sup> all regained strength against gravity, and all were able to independently climb stairs. Four patients had no extension lag, and the remaining three had

20° to 30° extension deficit.

### Sciatic Nerve

The sciatic nerve has two distinct divisions: tibial and peroneal. The peroneal division is significantly more susceptible to injury than the tibial division for several reasons. The peroneal nerve is tethered proximally in the greater sciatic notch and distally where it passes through fibrous tunnel around the knee; therefore, it has limited mobility of only 0.5 cm. It also has less connective tissue and fewer and larger fascicles than the tibial division. The distance to reinnervation of key muscle groups is longer than that for the tibial nerve, and as a result, the peroneal division is injured more frequently and recovery is often worse than for the tibial division.<sup>6,34</sup>

The incidence of sciatic nerve injury associated with fractures, particularly of the acetabulum, ranges from 7.9% to 75%, depending on the type of fracture and the extent of the neurologic injury (partial versus complete).<sup>5,41-45</sup> Risk factors for nerve injury associated with acetabulum fractures include concomitant hip dislocation or involvement of the posterior column. While the majority of these lesions may be “in-continuity” on gross examination, nerve conduction testing has demonstrated axonotmesis in at least one study, suggesting a lower potential for spontaneous recovery.<sup>41</sup> Acetabular fracture surgery has also been associated with sciatic nerve injuries. Early series reported rates as high as 18% with the Kocher-Langenbeck approach, but Letournel and Judet felt that this was caused by traction on the nerve during the surgery.<sup>41,44,46</sup> Once this was recognized and the technique changed to the use of transcondylar traction, hip extension, and knee flexion, the rate of nerve injury decreased dramatically to 3%.<sup>44,46</sup> More recent series report a lower rate of 1%.<sup>43</sup> The extended iliofemoral approach is at highest risk, followed by combined, ilioinguinal, and Kocher-Langenbeck approaches.<sup>42</sup> The use of nerve monitoring, particularly somatosensory evoked potentials (SSEPs), to prevent iatrogenic nerve injury is controversial but not currently supported in the literature. Baumgartner and associates reported a decrease from 24% to 5% rate of sciatic nerve palsy with monitoring, but their sample was small and statistical significance was not reached.<sup>47</sup> In a larger series of 140 unmonitored and 112 monitored cases, Haidukewych and coworkers had an overall 5.6% rate of sciatic nerve injury but with a higher rate in the monitored group, attributed to selection bias and learning curve. Crucially, when the investigators considered the 10 patients that were monitored and still sustained an injury, 7 had no detected abnormalities intraoperatively, implying only a 30% sensitivity of this technique.<sup>42</sup>

In THA, the incidence of sciatic nerve injury varies between 0.16% and 8% depending on the series.<sup>38,48-50</sup> Known risk factors include developmental hip dysplasia, posterior approach, revision surgery (3.2%), limb lengthening, female gender, and younger age. A large review of the literature identified a 0.79% incidence of sciatic nerve injuries out of

a total of 24,469 patients.<sup>38</sup> The most common mechanisms were tension, direct trauma from retractors, and postoperative hematoma, but direct laceration and sutures have also been implicated.<sup>8</sup> Farrell and colleagues reviewed 27,004 primary THAs, and reported 44 patients (0.16%) with sciatic nerve injury, 61% of whom presented with a complete motor deficit.<sup>48</sup> In their series, 16 patients had an isolated complete peroneal lesion, 11 had complete involvement of both tibial and peroneal divisions, but no patients had an isolated complete tibial lesion.

Hip arthroscopy also carries an approximately 1.7% risk of sciatic nerve injury. A recent study demonstrated that the amount of traction weight, not time in traction, is the most important risk factor in these cases.<sup>51</sup>

The natural history of sciatic nerve injury is variable.<sup>16,38,41,48,52</sup> A complete lesion without recovery leads to a 100% limb disability.<sup>5</sup> Letournel and Judet noted a 57% rate of complete or good recovery in their series of fracture-associated sciatic nerve palsies.<sup>44</sup> While only 6% required bracing and two-thirds of patients were able to perform their normal activities, a substantial number of patients were considered slightly or substantially disabled.<sup>44</sup> Severe peroneal division injuries have been shown to have a worse prognosis than tibial division injuries, and failure to recover nerve function has been associated with significantly worsened short musculoskeletal function assessment (SMFA) scores.<sup>41,52</sup> A large literature review of injuries associated with THA found 44% fair and 15% poor outcomes.<sup>38</sup> Incomplete sciatic nerve lesions have better prognosis than complete lesions, and in cases of postoperative hematoma causing nerve compression, urgent evacuation results in better outcomes than expectant management.<sup>16,48</sup>

Results of operative treatment are mixed. Yeremeyeva and colleagues explored 15 iatrogenic injuries after a period of observation with no recovery and found that despite the nerve appearing grossly intact, half of the lesions had no NAPs across the lesion requiring neuroma resection. The rates of good recovery for the peroneal component were only 25% for neurolysis and 14% for grafting. The tibial division recovered well, with 57% to 75% good results.<sup>8</sup> Kim and Murovic reported on a large series of sciatic nerve repairs, noting that primary repair had better results than grafting, and neuroma-in-continuity or compression injuries had good results as long as there was NAP conduction across the neuroma. Results of primary repair of the tibial division were superior to peroneal, and results at the thigh were better than at the buttock. At the buttock, primary and graft repairs of the tibial division resulted in 62% to 73% good outcomes, versus 24% to 30% for the peroneal division. At the thigh level, good results were seen in 80% to 93% and 45% to 69% for the tibial and peroneal divisions, respectively.<sup>18,31</sup>

### The Common Peroneal Nerve

The incidence of traumatic injury to the common peroneal nerve at or below the knee is from 1.2% to 3% in tibial plateau fractures but can be as high as 75% in multiligament

mentous knee injury, particularly with postero-lateral knee dislocations.<sup>6,53-55</sup> Risk factors include obesity, male gender, and fibular head fracture.<sup>6</sup> There is a statistically significant association between the peroneal nerve injury and vascular injury in knee dislocations, necessitating a high index of suspicion in these cases.<sup>56</sup> Nerve rupture rate can be as high as 40%.<sup>56</sup>

Iatrogenic injuries to the CPN have been described with high tibial osteotomies (HTO) and total knee arthroplasties (TKA). HTO is associated with a 4.9% rate of CPN injury, with a high rate of complete recovery; there is no significant difference in the rate of injury between medial opening and lateral closing-wedge osteotomies.<sup>57,58</sup> The reported rate of CPN injury in TKA varies from 0.3% to 9.5% depending on the series; the largest series of primary TKA report a low rate of 0.3%.<sup>59-61</sup> A number of risk factors have been associated with an increased risk for CPN palsy and include revision surgery, preoperative valgus deformity, rheumatoid arthritis, history of previous HTO, prolonged tourniquet time, and history of laminectomy.<sup>60-62</sup>

Untreated CPN injury results in foot drop, a supination (equinovarus) deformity, a steppage gait, and 30% to 35% limb disability.<sup>5</sup> Bracing with an ankle-foot orthosis is effective and commonly used and should be employed after the injury to facilitate ambulation and prevent joint contracture. As a permanent solution, bracing suffers from poor acceptance, particularly in younger patients.<sup>63</sup> If a recovery occurs, the superficial peroneal nerve is expected to recover first, with peroneal muscle contractures evident at about 5 months, followed by the deep peroneal nerve with tibialis anterior contractures evident at 12 months. It may take as long as 30 months for the recovery to be complete.<sup>26</sup> The literature on the natural course of non-operatively treated peroneal palsies associated with knee dislocations or TKA demonstrates a wide range of functional recovery, with lower functional scores in patients without recovery.<sup>56,59-61,64,65</sup> The rate of full recovery of partial peroneal palsies ranges from 76% to 87%, and the rate for spontaneous recovery of complete lesions ranges from 20% to 35%. Caution should be used when interpreting these outcome studies, as none of these series specified the extent of "partial" recoveries or specifically defined what is meant by "incomplete" injuries, which makes it difficult to compare results.

Surgical treatment is indicated if there is no clinical or electrodiagnostic evidence of recovery or if the nerve can be explored during reconstructive surgery after a knee injury. Results of operative treatment depend on a number of factors, such as the type of injury, the type of repair, the elapsed time, and others, making interpretation of literature challenging.<sup>32,64,66-68</sup> Neurolysis has resulted in good recovery in 71% to 97% of patients, likely due to lesser degrees of neurologic injury.<sup>26,32,64</sup> A recent large series by Kim and coworkers reported excellent results for neurolysis alone; there was an 88% rate of functional return in all neurolysis cases and 93% (65/70) in cases of nerve stretch or contu-

sion.<sup>26</sup> Direct repair had an 84% rate of useful functional recovery, and grafting resulted in a 75% success rate for graft lengths less than 6 cm dropping sharply to 16% to 38% with increasing graft lengths.<sup>26</sup>

Tendon transfers can be used to restore dorsiflexion after a failed nerve reconstruction, using the tibialis posterior and flexor digitorum longus tendons to restore ankle and toe dorsiflexion.<sup>6,63</sup> Good results are reported, with 81% of patients regaining active dorsiflexion past neutral, and 50% gaining grade 4 strength or higher.<sup>63</sup> Flatfoot is a possible complication but is rare in this setting due to peroneal musculature paralysis, which therefore cannot contribute to the flatfoot deformity.<sup>63</sup>

## Tibial Nerve

Unlike the CPN, the tibial nerve is not as vulnerable to injury due its better blood supply, larger number of fascicles and connective tissue, and lack of tethering. In addition, the innervation necessary for gastrocnemius muscle to achieve function is more proximal and relatively small, permitting better functional recovery.<sup>31</sup>

Isolated tibial nerve injuries have been described with distal femur, tibia, and calcaneal fractures, ankle or knee arthroscopic surgery, TKA, or direct penetrating injury.<sup>27,69-72</sup> Overall these are rare, comprising only 4% of major peripheral nerve injuries associated with trauma, and even more rare in knee arthroscopy (Table 3).<sup>71,73</sup>

With a complete tibial nerve injury, the amount of limb disability ranges from 35% to 70%, dependent on the location of the injury. The major deformity is calcaneus, and loss of plantar sensation can be problematic and is poorly tolerated. Up to 71% of patients may have trophic and vasomotor changes, anhidrosis, cracked skin, and ulcers associated with repeated foot trauma. Patients have difficulty with ambulation, and may lack push-off power. Bracing is challenging, and a rigid ankle brace is needed.<sup>5,33,36</sup>

Non-operative treatment of isolated tibial nerve injury at or below the knee is generally felt to have a higher success rate than other nerves in the LE, but good data is lacking. It is reasonable to assume that if a complete nerve lesion is suspected, it will not recover without repair, and surgical management is indicated. Overall, the results of repair are reassuring, and expected time for recovery is 4 to 8 months.<sup>5</sup> Good recovery is expected in the majority of cases both with direct repairs or grafting. Older series quoted 39% to 62% sensory and 27% to 79% motor recovery.<sup>5</sup> Kim and colleagues reported on surgical treatment of 38 tibial nerve injuries at the level of the knee and leg and 33 tibial nerve injuries at the level of the foot and ankle. Neurolysis resulted in good recovery in 95% and 74% in higher- and lower-level injuries, respectively. Only two primary repairs were reported, and both patients did well. Grafting (5 cm to 11 cm in length) was performed in 16 knee and leg-level injuries, with a 94% rate of grade 3 or better motor recovery. In ankle-level injuries, grafting resulted in 64% restoration

**Table 3** Summary of Incidences of Lower Extremity Nerve Injuries

	Traumatic	Iatrogenic
Femoral Nerve	Acetabulum fractures: 0.55%	THA overall: 0.13% THA - Anterolateral approach: 2.3%
Sciatic Nerve	Acetabulum fractures and hip dislocations: 7.9% to 75%*	Acetabulum fracture surgery: 1% to 3% Primary THA: 0.16% THA for DDH: 5.2% Revision THA: 3.2% Hip arthroscopy: 1.7%
Common Peroneal Nerve	Tibial plateau fractures: 1% to 3% Knee dislocations: 10% to 75%	HTO: 4.9% Primary TKA: 0.3% to 1.3%
Tibial Nerve	Tibial fractures: 0.5%	Knee arthroscopy: < 0.004%

of adequate sensation. Interestingly, the results of grafting at the knee level were nearly identical to results after primary repair or external neurolysis, confirming the high potential for recovery.<sup>27</sup> Others have reported good results with grafting at the ankle level, with grafts as long as 18 cm.<sup>74</sup>

Tendon transfers have been described to restore plantar flexion.<sup>36</sup> The tibialis anterior tendon is detached from its insertion and passed through the interosseous membrane and is attached to the calcaneus. The peroneus longus tendon is detached as well and is added to the transfer to balance it and prevent valgus deformity, relegating ankle dorsiflexion to the remaining long toe extensors. Triple arthrodesis with shortening of the talar neck has been suggested to complement the transfer, to allow posterior displacement of the calcaneus and increase the lever arm of the transfer.

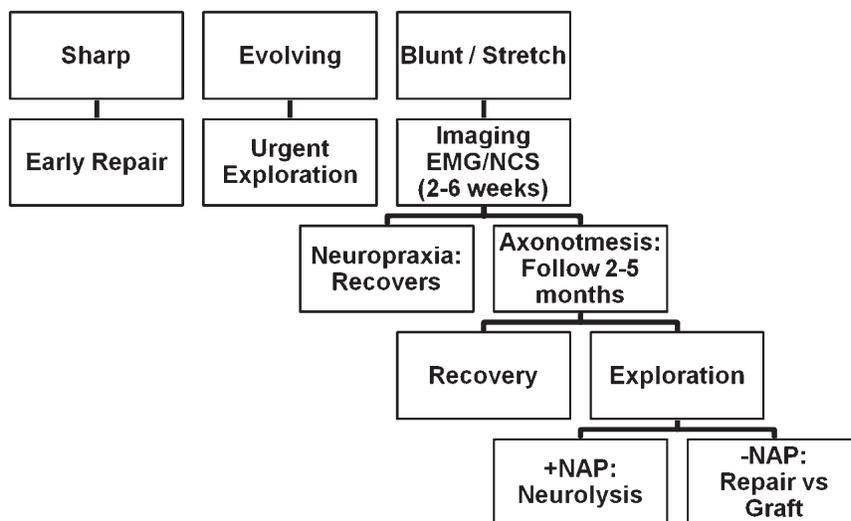
**Summary and Conclusions**

The incidence of LE nerve injury ranges from less than 1% in hip and knee arthroplasty to as high as 75% in knee dislocations (Table 3). Most traumatic and iatrogenic lower-extremity nerve injuries likely to be encountered by orthopaedic surgeons are grossly discontinuity, yet frequently

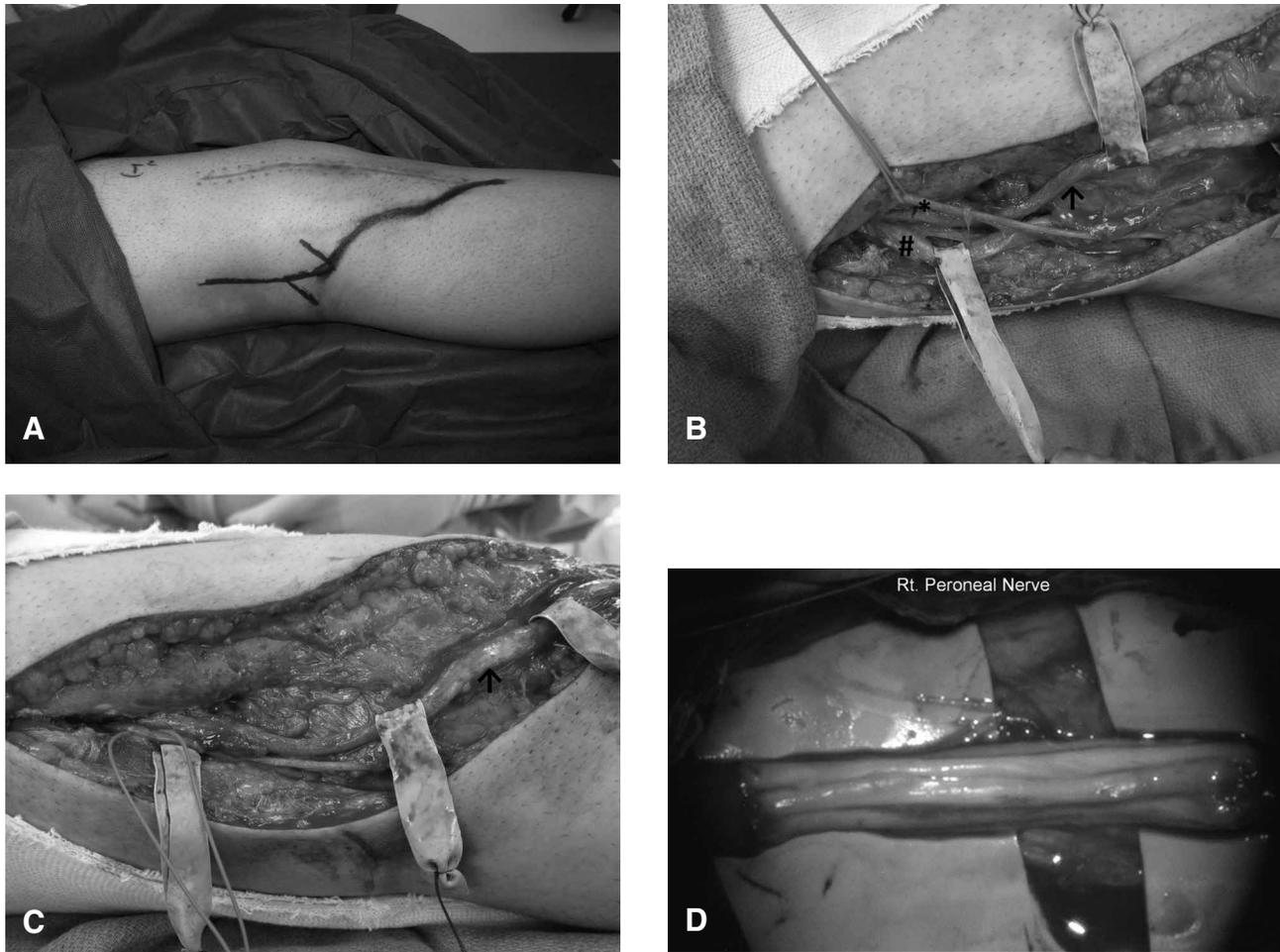
involve axonotmesis and should not be assumed to be simple neuropraxias. This distinction can be made with a thorough history and physical examination, serial electrodiagnostic studies, or advanced imaging.

The natural course is favorable for the femoral and tibial nerves but disappointing for both sciatic and CPN around the knee. Outcomes of repair vary widely, and no randomized studies exist, making comparisons between operative and non-operative treatments difficult. The results of femoral and tibial operative repair are superior to sciatic and CPN, and lesions that can be managed by neurolysis have a better prognosis than repair or grafting, particularly for sciatic and CPN.

It is essential that orthopaedic surgeons be aware of the general principles of management of lower extremity nerve injury. With this in mind, we propose an overall protocol for diagnosis and treatment of lower extremity traumatic and iatrogenic nerve injuries (Fig. 1). For sharp lacerations, early surgical exploration is warranted, but in gunshot wounds or severe nerve contusion, a delay of several weeks is recommended to allow for full delineation of zone of injury. An evolving injury, such as a postoperative sciatic or CPN palsy,



**Figure 1** Suggested protocol for management of traumatic and iatrogenic LE nerve injuries.



**Figure 2** A 19-year-old male sustained a right knee dislocation, with a complete common peroneal nerve palsy confirmed with electrodiagnostic studies. He failed to show clinical improvement for 3 months and was indicated for open exploration. **A**, A curvilinear incision is designed with a z-plasty at the popliteal crease to prevent scar contracture. **B**, A wide dissection is performed identifying the common peroneal (black arrow), tibial (#), and sural (\*) nerves. **C**, A neuroma-in-continuity (black arrow) is identified in the common peroneal nerve. **D**, The neuroma is resected, and cable grafting with sural nerve autograft is performed. (Images courtesy of Robert M. Szabo, M.D.)

developing on the first or second postoperative day may be reversible and should be addressed rapidly. The dressings should be loosened; the hip and the knee should be flexed to decrease tension on the sciatic and peroneal nerves, and if a hematoma is suspected, it should be evacuated. For blunt trauma and stretch injuries, an initial period of observation is warranted. Electrodiagnostics should be performed after 2 to 6 weeks, and imaging can be considered. Recovery should be followed with serial examinations, and findings of axonotmesis should lead to a consideration of operative management. If no recovery is observed after 2 to 5 months, the nerve should be explored and managed appropriately based on intraoperative findings, with neurolysis, repair, or grafting if the defect is large (Fig 2).

Future research may shed light on mechanisms of nerve regeneration and lead to advances that improve the outcomes of nerve injury. Regardless, orthopaedic surgeons will likely remain the first and sometimes only physicians

that manage patients with lower extremity nerve injuries. Thus, orthopaedic surgeons must be aware of the prognosis, proper diagnostic methods, and management options so that patients can be counseled appropriately, proper treatment can be instituted, and necessary referrals are not delayed.

#### Disclosure Statement

None of the authors have a financial or proprietary interest in the subject matter or materials discussed, including, but not limited to, employment, consultancies, stock ownership, honoraria, and paid expert testimony.

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