Review Article

Nerve Injury Complicating Multiligament Knee Injury: Current Concepts and Treatment Algorithm

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Abstract

Multiligament knee injuries account for <0.02% of all orthopaedic injuries, and 16% to 40% of these patients suffer associated injury to the common peroneal nerve (CPN). The proximity of the CPN to the proximal fibula predisposes the nerve to injury during local trauma and dislocation; the nerve is highly vulnerable to stretch injury during varus stress, particularly in posterolateral corner injuries. CPN injuries have a poor prognosis compared with that of other peripheral nerve injuries. Management is determined based on the severity and location of nerve injury, timing of presentation, associated injuries requiring surgical management, and the results of serial clinical evaluations and electrodiagnostic studies. Nonsurgical treatment options include orthosis wear and physical therapy. Surgical management includes one or more of the following: neurolysis, primary nerve repair, intercalary nerve grafting, tendon transfer, and nerve transfer. Limited evidence supports the use of early one-stage nerve reconstruction combined with tendon transfer; however, optimal management of these rare injuries continues to change, and treatment should be individualized.

***JAAOS* Plus Webinar**

Join Dr. Moorman, Dr. Mook, and Dr. Leversedge for the *JAAOS* interactive webinar discussing "Nerve Injury Complicating Multiligament Knee Injury: Current Concepts and Treatment Algorithm," on Tuesday, June 18, 2013, at 9 PM Eastern. The moderator will be Peter Jokl, MD, the *Journal's* Deputy Editor for Sports Medicine topics.

To join and to submit questions in advance, please visit the OrthoPortal website: http://orthoportal.aaos.org/jaaos/

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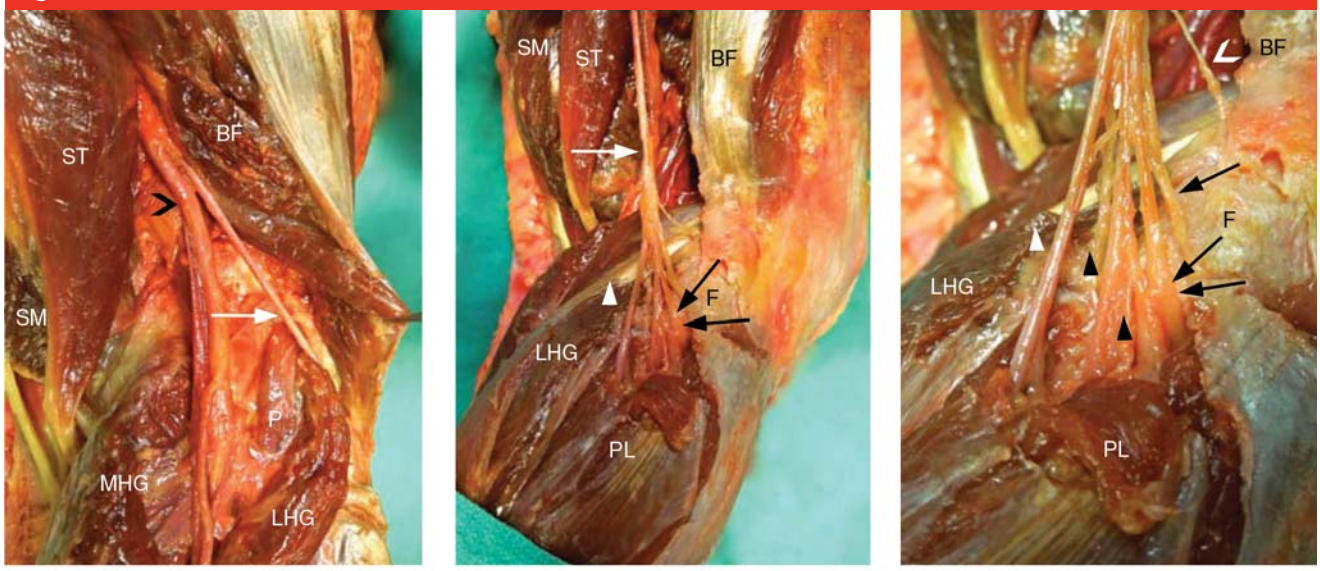
Multiligament knee injuries (MLKIs) associated with knee dislocations account for <0.02% of all orthopaedic injuries; however, this may be an underestimation because some knee dislocations may spontaneously reduce at the time of injury and go unrecognized.[1] Common peroneal nerve (CPN) injury occurs in 16% to 40% of patients with knee dislocation.[2] The prognosis for nerve recovery associated with MLKIs is generally poor and depends on the extent of disruption of the normal neural anatomy.[2]

Acute knee dislocations are caused by high-energy trauma such as motor vehicle collisions and industrial injuries, as well as lower-energy trauma such as that sustained during sports participation and falls.[3]

The four primary ligamentous stabilizers of the knee are the anterior cruciate ligament, posterior cruciate ligament, medial cruciate ligament, and lateral collateral ligament. Disruption of two or more of these ligaments may occur with knee dislocation, resulting in an MLKI. MLKIs may be associated with vascular injury or neurovascular injury or both, and the clinician should have a high index of suspicion for neurovascular deficits during the global assessment.[1,2]

Figure 1



Photographs of cadaver dissection of the popliteal fossa and posterolateral corner of a right knee. **A,** Bifurcation of the tibial nerve (black chevron) and the common peroneal nerve (CPN [white arrow]) in the proximal popliteal fossa. **B,** Branching of the CPN proximal to the fibula (F). **C,** Close-up view of branching of the peroneal nerve into superficial and deep divisions, the motor branches to the short head of the biceps femoris (BF) and peroneus longus (PL) muscles, and an articular branch with the PL muscle reflected from its origin. LHG = lateral head of the gastrocnemius muscle, MHG = medial head of the gastrocnemius muscle, P = popliteal muscle, SM = semimembranosus muscle, ST = semitendinosus muscle, white triangle = superficial peroneal nerve, black triangle = motor nerve branches to the PL, black arrow = articular branch of the common peroneal nerve, double arrow = deep peroneal nerve, white chevron = nerve to the short head of the BF

Injury to the CPN may be associated with sensory and/or motor deficits. The severity of the neurologic impairment can range from a mild stretching injury (ie, neurapraxia), to nerve rupture or laceration with an open injury, to neurotmesis.[4] Few evidence-based guidelines exist to guide the management of these complex injuries, particularly in light of the limited ability to determine the extent of nerve injury and establish a prognosis for nerve recovery. Careful patient evaluation and individualized treatment are paramount.

Anatomy

The CPN lies close to the posterolateral corner (PLC) of the knee joint and the proximal fibula, which places the nerve at risk of injury during varus stress, local trauma, and knee dislocation[5,6] (Figure 1). In the distal one third of the thigh, the sciatic nerve bifurcates into the CPN and the tibial nerve. Prior to exiting the popliteal fossa, the CPN, situated anterior to the conjoined biceps femoris tendon and posterior to the lateral head of the gastrocnemius muscle, innervates the short head of the biceps femoris muscle. The CPN courses distally and superficially, covered by only subcutaneous tissue

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and skin, traveling lateral to the proximal fibula.

A consistent vascular supply to the CPN arises from an unnamed branch of the popliteal artery within the proximal popliteal fossa. However, more distally, at the level of the knee joint, the vascular supply becomes more tenuous, relying on small vasa nervorum derived from the anterior recurrent tibial artery.[7] In contrast, the tibial nerve remains protected within the popliteal fossa as it courses between the popliteus muscle and the popliteal fascia before entering the deep posterior compartment of the lower leg, and it receives predictable vascular contributions from the popliteal and the posterior tibial arteries.[7] The tibial nerve is less likely to be injured during knee dislocation,[1,2] which may be due in part to its protected location and more consistent blood supply.

In approximately 80% of patients, the CPN splits into the superficial and deep divisions at or distal to the fibular neck; however, in up to 10% of persons, bifurcation of the CPN occurs proximal to the lateral joint line.[8] The superficial peroneal nerve innervates the peroneus longus and brevis muscles, which function primarily to plantarflex the first ray and evert the ankle, respectively. The deep peroneal nerve innervates four extrinsic muscles (ie, peroneus tertius, tibialis anterior, extensor hallucis longus, extensor digitorum longus) to dorsiflex the foot and extend the toes. Injury to the CPN may cause a motor palsy involving these muscle groups; often, the clinical presentation involves foot drop or loss of ankle dorsiflexion, as well as a relatively unsteady gait. The terminal branches of the peroneal nerve provide sensory innervation for the dorsal foot and the first web space. Patients with knee injury may experience sensory disturbances in these distributions.

Mechanism of Injury

At the time of knee dislocation, disruption of the PLC is associated with an increased incidence of CPN injury.[1,2] Isolated PLC injuries are rare but may result from a posterolateral force applied to the tibial plateau with the knee near full extension.[9] Other mechanisms of PLC injury include isolated severe varus bending moments, external rotatory torque on the tibia, combined hyperextension and external rotation forces, and both contact and noncontact hyperextension moments.[10]

Nerve Injury Classification

Two nerve injury classification systems are applicable to the diagnosis and management of CPN injuries (Table 1). Seddon[12] stratified peripheral nerve injuries into three classes: neurapraxia (mild), axonotmesis (moderate), and neurotmesis (severe). Sunderland[13] modified that system to account for the variable outcomes of axonotmetic injuries. The histology of nerve injury and the regenerative response follow a predictable sequence of pathophysiologic events; the complexity of this biologic process highlights the guarded prognosis for CPN recovery following injury (Table 2).

Clinical Examination

Serial comprehensive examinations should be carefully documented and a thorough history obtained that includes the mechanism of injury, postinjury interventions, and previous and current symptoms. Most knee dislocations are caused by high-energy trauma; thus, concomitant injuries should be noted. Resuscitation and a global clinical assessment at the time of initial presentation are prioritized. Lower limb evaluation in the setting of knee trauma should consider the integrity of the ligaments of the knee joint, and detailed evaluation of the sensory and motor functions of the CPN is essential. Injury to the CPN is suggested by inability to dorsiflex the foot or extend the toes, ankle eversion paralysis or weakness, and/or altered sensibility in the cutaneous nerve distributions; however, a more proximal nerve injury, such as from the lumbar spine, should be considered as well.

Muscle strength is graded from 0 to 5 using the British Medical Research Council (MRC) scale[15] (Table 3). Sensibility is evaluated subjectively by assessing the patient's ability to appreciate deep and superficial pain, light touch, and two-point discrimination. The presence of a Tinel sign, or percussion of the injured nerve that causes paresthesia in its sensory distribution, can be used to trace the progress of a regenerative CPN over time. In the absence of indications for emergent or urgent surgical intervention, serial examinations are conducted to monitor the neurovascular status of the affected limb.

Adjuvant Studies

Imaging

Standard radiographs are routinely obtained in the evaluation of acute knee injuries. However, the use of MRI and ultrasonography continues to evolve for evaluating the macroscopic anatomy of neurologic injury and the relative zone of injury that may influence decisions regarding management. Enhanced resolution may improve the ability to correlate images with nerve function and, ultimately, with prognosis for neurologic recovery.

Radiography

Standard radiographs of the knee are indicated in the initial evaluation of

Table 1

Nerve Injury Classifications and Electrodiagnostic Findings[11]

Seddon	Sunderland	Histopathologic Features	Expected Recovery Response and Timeline	NCV	EMG
Neurapraxia (mild)	1	Normal nerve architecture: local loss of conduction	No degradation of axons. Full recovery in hours to weeks probable.	Usually preserved. Reduced SAP amplitude proximal to the injury and normal distal to the injury. Normal MUP.	No or few fibrillations
Axonotmesis (moderate)	2	Disrupted: axons Intact: endoneurium, perineurium, epineurium	Wallerian degeneration, proximal axon degeneration within the zone of injury, variable maintenance of supporting structures depending on the mechanism of injury. Full recovery in weeks to months possible. (Regeneration 1 mm/d.)	Normal or reduced to a degree dependent on the size of the zone of injury, injury type, amount of axonal degeneration, and the nerve types involved. Decreased SAP and firing rate of MUP.	Fibrillations
	3	Disrupted: axons, endoneurium Intact: perineurium, epineurium	Same as Sunderland grade 2	Same as Sunderland grade 2	Same as Sunderland grade 2
	4	Disrupted: axons, endoneurium, perineurium Intact: epineurium	Same as Sunderland grade 2	Same as Sunderland grade 2	Same as Sunderland grade 2
Neurotmesis (severe)	5	Complete transection of the nerve	Spontaneous recovery unlikely	Not measurable. SAP and MUP absent.	Innumerable fibrillations

EMG = electromyography, MUP = motor unit potentials, NCV = nerve conduction velocity, SAP = sensory action potentials

Table 2

Pathophysiology of Peripheral Nerve Injury

Time	Stage	Characteristics
At injury	Mechanical nerve injury	Axonal disruption (axonotmesis/neurotmesis) and proximal degeneration within the zone of injury. Some axonotmized neurons die without target support.
	Neuron cell body chromatolysis	Peripheral migration of surviving neuron nuclei. Production of reparative structural molecules.
48–160 h[11]	Wallerian degeneration	Degeneration of distal axon–Schwann cell activation and macrophage recruitment. The nerve-blood barrier is disrupted, which allows for clearance of inhibitory nerve outgrowth factors. Distal Schwann cells may become less able to support regenerating axons with time.[14]
Weeks to months	Growth cone formation/ elongation	Motile tip of the regenerative axon that responds to neurotrophic/neurotropic stimulation provided by end-organs and Schwann cells that have organized along the path of the distal axon
	Reinnervation	If the growth cone fails to reach its target organ, motor end plates are lost, muscles atrophy, and muscles eventually fibrose along the path of the distal axon. Nerve growth occurs at a rate of approximately 1 mm/d.

a patient with known or suspected MLKI. These are used to evaluate for associated bony injuries (Table 4) and to confirm concentric joint reduction. PLC injuries should be considered in the presence of local bony avulsion injuries (Figure 2), and the CPN should be carefully evaluated. During preoperative planning, dy-

namic varus stress radiographs may be useful to supplement MRI in determining the degree of PLC laxity.[19]

MRI

MRI is useful in evaluating potential ligamentous injury, detecting neurologic injury, and determining the soft-tissue response to injury (Figure 3). In conjunction with other studies and physical examination, MRI helps to confirm the presence and location of CPN injury.[4,20] MRI may be useful in determining surrounding fat planes, localized edema, presence of contusion, nerve fiber disruption, and encasing hematoma.[4,20] Increased signal intensity within the nerve following injury is seen on T2-weighted images at and distal to the site of nerve injury. In subacute images, chronic neuropathic changes may manifest as muscle edema or fatty infiltration within the anterior and lateral compartment musculature.[21]

More recently, magnetic resonance neurography has been used in both animal models and retrospective case series of peripheral nerve injuries.[22] This modality involves the use of short tau inversion recovery sequences to image peripheral nerves directly. Its precise role in the evaluation of peroneal nerve injury has yet to be defined; however, it seems to have the potential to accurately detect the early extent of nerve lesions and monitor their regeneration.

Ultrasonography

Ultrasonography is a dynamic imaging modality, and knee orientation can be manipulated to evaluate the continuity of the CPN (Figure 4). High-resolution ultrasonography can be used rather than MRI to detect the location and determine the severity of nerve injury.[4,23]

Ultrasonography has been used to accurately discern the specific location and length of CPN injury, the diameter of an injured but continuous CPN, and the presence of an obstructing hematoma or scar.[5] It is an efficient diagnostic tool for differentiating incomplete injury (ie, neurapraxia, axonotmesis) from complete injury (ie, neurotmesis).[4,23] This imaging tool is highly user dependent,[5,23] but it is promising when used by experienced clinicians.

Electrophysiologic Testing

Electromyography (EMG) and nerve conduction velocity (NCV) studies can be used in the assessment of the severity, location, and prognosis of nerve injury.[4,5] Baseline NCV and EMG studies are obtained approximately 6 weeks following injury if a functional neurologic deficit remains. These studies may be used for subsequent comparison at 3 and 6 months if neurologic recovery is incomplete and surgical reconstruction is being considered. Pertinent EMG findings corresponding to acute nerve injury include positive sharp waves and fibrillation potentials.[4] Chronic denervation is marked by fasciculations and complex repetitive discharges.[11]

Severity of nerve injury may correlate with NCV findings[4,8] (Table 1). In an incompletely damaged nerve, conduction velocity is slowed, whereas a completely severed nerve may lack motor control (measured in

Table 3

British Medical Research Council Scale for Evaluating Muscle Function[15]

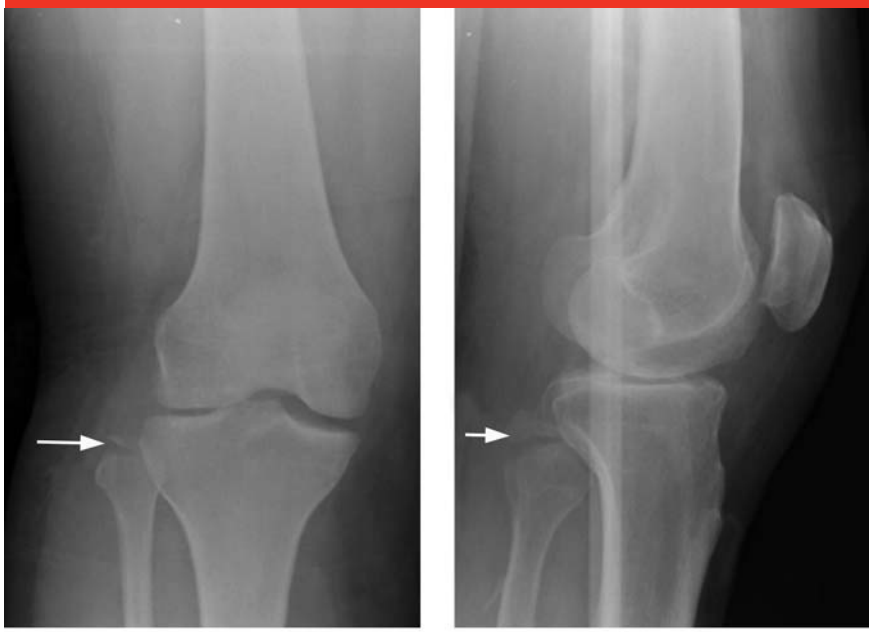
Grade	Description
0	No muscle contraction
1	Trace contraction
2	Contraction with resistance of gravity removed
3	Muscle contraction against gravity resistance only
4	Muscle contraction against some resistance
5	Normal muscle contraction against full resistance

Table 4

Avulsion Fractures That May Indicate Underlying Injury to the Lateral Knee Soft Tissues

Lesion	Injured Structure	Radiographic Appearance
Segond fracture[16]	Tibial insertion of the middle third of the lateral capsular ligament. Suspect for anterior cruciate ligament and lateral meniscal injuries.	AP knee: Elliptical osseous fragment parallel to the tibia, just distal to the lateral tibial plateau.
Arcuate complex avulsion (ie, arcuate sign)[17]	Fibular collateral, fabellofibular, popliteofibular, and arcuate ligaments	AP knee: Elliptical osseous fragment oriented orthogonally to the long axis of the tibia. Donor site originating from the fibular styloid process.
Biceps femoris avulsion[18]	Conjoined tendon of the biceps femoris	AP and lateral knee: Difficult to differentiate from the arcuate sign. More irregular osseous fragment found proximal and posterolateral to the fibula. Donor site is the fibular head. On the lateral view, found more posterior than the arcuate complex avulsion.

Figure 2



A B

AP (**A**) and lateral (**B**) non–weight-bearing radiographs of the knee demonstrating arcuate complex avulsion fracture (ie, arcuate sign). The arrows indicate a proximally displaced elliptical fracture fragment of the fibular styloid process.

motor unit potentials), sensation (measured in sensory action potentials), or both.[11]

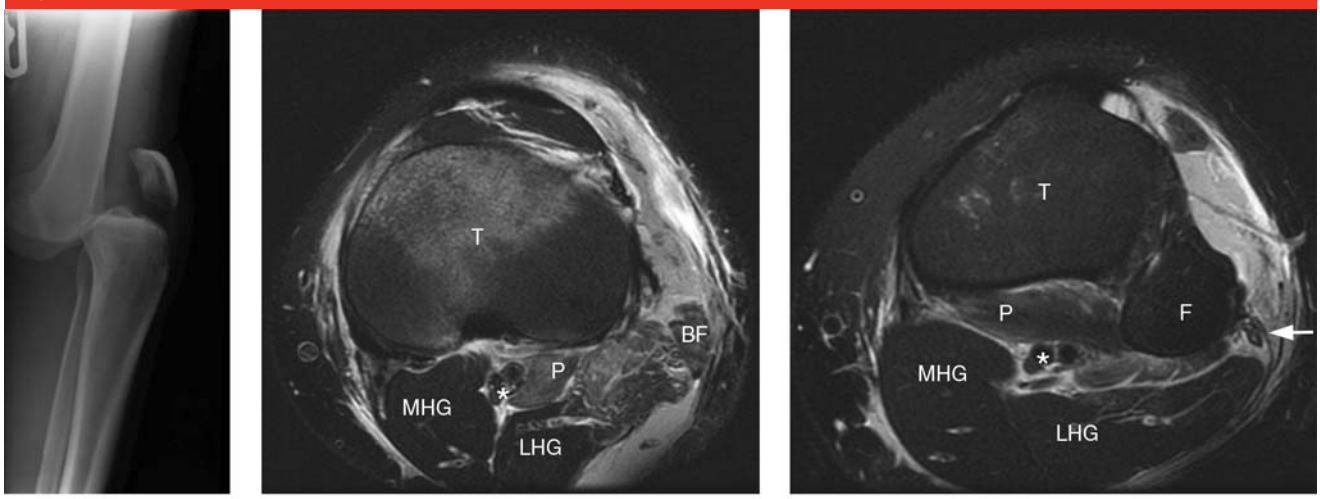
Management of CPN Injuries

Management goals for CPN injuries include promoting neurologic recovery, maximizing functional recovery, and minimizing risk and functional loss with nerve or musculotendinous reconstructive procedures. Prognosis and management are influenced by numerous factors, including patient age; timing of injury; mechanism of injury; longitudinal extent of CPN damage; distance of injury location from the nerve's distal targets; and associated soft-tissue, vascular, and bony injuries.

Nonsurgical

Nonsurgical management is prescribed in the presence of compelling

Figure 3



A B C

A, Lateral radiograph of a left knee demonstrating anteromedial knee dislocation before reduction. **B,** Axial T2-weighted fat-suppressed magnetic resonance image just distal to the level of the knee joint demonstrating increased signal intensity within the lateral gastrocnemius and popliteus muscle bellies, posterolateral corner, and lateral subcutaneous tissue. Normally, the common peroneal nerve (CPN) would be visualized deep to the biceps femoris muscle (BF); however, normal tissue planes are obscured by edema and hemorrhage. **C,** Axial T2-weighted fat-suppressed magnetic resonance image at the level of the proximal fibula, distal to the image in panel B, demonstrating increased signal intensity within the CPN (arrow), which is characteristic of injury. F = fibula, LHG = lateral head of the gastrocnemius, MHG = medial head of the gastrocnemius, P = popliteus, T = tibia, * = popliteal neurovascular bundle

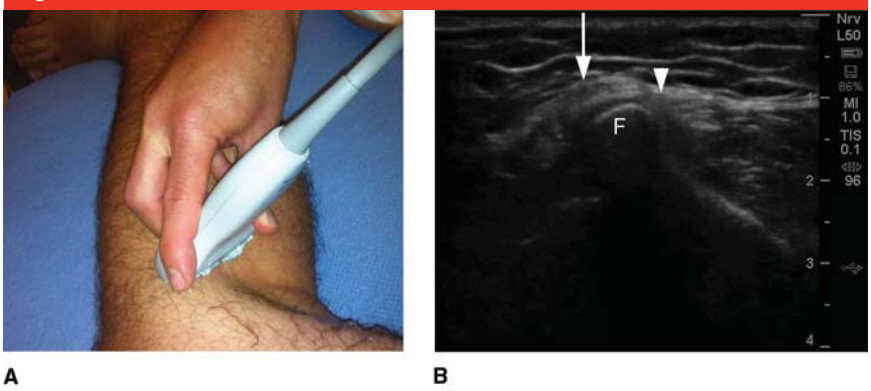
evidence supporting the spontaneous regeneration of the CPN (eg, serial clinical examination findings confirming improvements in sensory and motor function, EMG recordings depicting normal insertion activity postinjury). Every patient with CPN motor palsy should be fitted with an ankle-foot orthosis (AFO) and should undergo physical therapy to prevent equinovarus deformity by maintaining range of motion of the posterior ankle capsule, preventing heel cord contracture, and strengthening the remaining functional muscles.[1,4] Spontaneous CPN recovery after injury associated with MLKI occurs in 14% to 56% of cases.[24] Younger age (ie, <30 years) at the time of injury is the only variable that has been shown to be predictive of a higher likelihood of spontaneous CPN recovery.[25]

Surgical

Indications for Nerve Exploration/Neurolysis

The timing of repair or reconstruction of the ligamentous structures injured in an MLKI is controversial[26] and is dependent on numerous factors, including vascular status of the injured limb, joint stability, skin condition, and the status of other injuries. Early repair or reconstruction of the PLC is technically less challenging, and acute intervention may yield improved clinical outcomes[27] and permit direct visualization of the CPN. It is the authors' preference to reconstruct the PLC within 3 weeks of injury. Persistent CPN motor impairment at the time of acute reconstruction warrants exploration of the CPN and external neurolysis if it is incarcerated by hematoma, scar, or fracture. Information on the location of the nerve injury gained from imaging studies such as MRI and ultrasonography is helpful in surgical planning. Some authors have found the intraoperative use of ultrasonography to be invaluable in localizing nerve lesions.[28]

Figure 4



A B

A, Photograph demonstrating the orientation of the right leg and ultrasonography transducer that corresponds to the sonographic image in panel B. The proximal thigh is in the foreground, the popliteal fossa is to the right, and the foot can be seen in the upper left corner. **B,** Axial high-resolution sonogram (15.6 MHz transducer) of the split deep (arrow) and superficial (arrowhead) branches of the peroneal nerve, respectively, as they course around the proximal fibula (F). Normal atraumatic peripheral nerve echotecture is shown.

Neurolysis of the CPN at the time of acute or subacute PLC repair may improve functional outcomes. Thoma et al[29] retrospectively reviewed 20 patients with CPN injury, of whom 19 patients (95%) demonstrated improvement of at least one MRC grade for ankle dorsiflexion. Ten of these 19 patients regained motor function of grade 3 or better. Neurolysis was delayed >7 months in half of the patients, which indicates that even delayed management of an incomplete injury may result in improved ankle dorsiflexion strength. Nevertheless, these authors reported excellent results with early intervention. All three patients who underwent neurolysis within 4 months of injury improved from MRC grade 0 to at least grade 4. The natural history of these specific injuries is not known. Seidel et al[30] reported that 8 of 11 patients who underwent neurolysis for traumatic CPN injuries achieved a good functional outcome (MRC grade ≥4). Average surgical delay was 5 months. Although both studies are limited by lack of control groups and small sample size, the findings indicate that surgical neurolysis is indicated after 3 months if no electrical or clinical improvement is observed and the CPN is morphologically intact.

If acute intervention on the PLC is either not indicated or not possible, the patient is followed clinically, with electrodiagnostic testing at 6 weeks following injury and again at 3 months. Serial clinical examination findings and EMG and NCV results at 3 months assist in determining whether neurophysiologic testing is repeated at 6 months or if earlier surgical intervention is warranted. Prior to proceeding with surgery, repeat MRI and ultrasonography are recommended to help define the nature and extent of the zone of injury.

Indications for Direct Nerve Repair

Direct epineurial repair is the procedure of choice when the CPN is not in continuity and the zone of injury is small, thereby enabling end-to-end

repair without undue tension at the repair site. Because of the mechanism of injury associated with acute knee dislocations, the CPN fibers are often severely stretched, making end-to-end repair impossible without excessive tension.[1,4,31] Kim et al[32] reported that 16 of 19 patients who underwent end-to-end suture repair recovered motor function of MRC grade 3 or higher, which obviated the need for the use of AFOs to achieve functional gait mechanics.

Repair can be performed up to 12 months following the initial injury. However, delay of this length is controversial because the longer the interval between injury and repair, the greater the likelihood of adverse outcomes. Reinnervation of motor end plates is more time sensitive than that of sensory end-organs.[33] We agree with many of the authors who suggest that outcomes are best when repair is performed within 3 to 6 months of injury.[33,34]

Indications for Nerve Excision and Intercalary Nerve Grafting

If after at least 3 months of expectant management there is no clinical or electrical evidence of CPN reinnervation, regardless whether acute external neurolysis or direct repair were attempted acutely, the prognosis for a reasonable functional recovery is poor and surgical intervention is indicated.[1,34]

If tension-free repair has failed and/or is not feasible, additional options to promote reinnervation are considered. Tomaino et al[35] recommended cable grafting in the absence of positive EMG potentials even after neurolysis. Determining the length or zone of nerve injury is critical, and advanced imaging such as ultrasonography may assist in surgical planning.[5] If repair or intercalary nerve grafting is performed within the zone of injury, reinnervation may be limited by neuroma formation or fibrosis. Intraneural exposure with serial sectioning (ie, bread-loafing) and examination of the nerve can help to delineate normal fascicular anatomy. In general, an intercalary nerve graft measuring ≥4 cm is needed to span the entire zone of injury in cases of failed primary repair.[33]

The influence of length of the zone of injury has been demonstrated in several large series in which poorer prognoses were observed with the use of grafts measuring ≥6 cm.[30,32,34] In the largest study to date, Kim et al[32] reported functional outcomes following CPN injury for external neurolysis (121 patients), direct end-to-end repair (19 patients), and graft repair (138 patients). In the graft reconstruction group, 27 of 36 patients had a postoperative MRC grade 3 or above when a graft of <6 cm was used (75%). When the graft length was 6 to 12 cm, only 24 of 64 patients achieved an MRC of grade 3 or higher (38%). In persons with graft lengths of 13 to 24 cm, only 16% had a good outcome (6 of 38 patients). Recently, Cho et al[36] reported on outcomes of sports-related peroneal nerve injuries. Nerve gaps of <6 cm had a favorable functional outcome (MRC grade 3 or above) in 70% of patients, whereas gaps of 6 to 12 cm had only a 43% success rate and gaps of 13 to 24 cm had only a 25% functional success rate. Similar findings have been reported in other studies in which nerve grafting was the preferred management of CPN injury.[6,34]

Autogenous nerve graft remains the standard for the management of large peripheral nerve defects that require reconstruction because this graft type provides a nonimmunogenic and structurally inert scaffold for axonal regeneration.[37] Autogenous nerve grafts provide neurotrophic factors, extracellular matrix molecules, and viable Schwann cells not found in allografts or synthetic alternatives.

The sural nerve is the most commonly used autograft because of its potential length, diameter, proximity to the surgical field, and relatively low donor site morbidity.[38] However, the patient should be cautioned of the potential graft harvest morbidity, which includes leg pain resulting from neuroma formation, distal sensory changes, hematoma, and wound healing problems.[39] Preoperative EMG and NCV studies should include an assessment of the viability of the ipsilateral sural nerve; this nerve may have been injured at the time of injury. In the case of sural nerve injury, the surgeon may need to consider using contralateral sural graft harvest or an alternative nerve graft source instead.

Sedel and Nizard[40] caution against nerve grafting in the setting of concomitant vascular injury about the knee, even when adequately repaired or bypassed, due to disappointing outcomes. The use of a vascularized sural nerve graft (VSNG) has been described when vascular insult is suspected. Terzis and Kostopoulos[41] presented their long-term results of a series of 12 patients treated between 3 and 48 months from injury with VSNG using grafts between 6 and 35 cm in length. All patients treated within 6 months of injury, regardless of graft length (≤20 cm), achieved an MRC grade 4 of ankle dorsiflexion and/or eversion. Outcomes were substantially better if denervation time was ≤6 months at the time of surgery. For this reason, the authors concluded that VSNG should be considered when attempting to bridge nerve defects ≥13 cm in length, especially within 6 months of injury.

Nerve allografts offer an additional source for nerve reconstruction. Allografts have the advantages of unlimited supply, ready availability in most cases, and lack of harvest site

morbidity. However, they have a direct risk of infection, and they require the use of temporary systemic immunosuppression, which may preclude their use in patients with multisystem trauma. Giusti et al[42] recently showed autogenous nerve graft reconstructions to be superior to allograft reconstructions with regard to motor recovery in a rodent model. Based on the current evidence, we prefer the use of autogenous nerve grafts when possible.

Indications for Tendon Transfer, With or Without Nerve Reconstruction

Historically, tendon transfer was indicated only when time-dependent myoneural degeneration had occurred and/or previous attempts at reinnervation had failed. The posterior tibialis is the most commonly used tendon. It is transferred from the posterior compartment of the leg to the dorsal second or third cuneiform. The procedure enables the tibialis posterior to function as a dorsiflexor, resulting in improved gait mechanics and decreased reliance on assistive devices such as AFOs.

Equinovarus deformity is believed to result from an imbalance between the flexor and extensor muscles of the foot, and it is a complication of CPN injury following acute dislocation.[31,43] Recent studies have shown that early surgical intervention to correct imbalances with concomitant nerve reconstruction or repair can positively influence prognosis.[31,43]

Garozzo et al[43] and Ferraresi et al[44] compared combined CPN repair/reconstruction and posterior tibial tendon transfer (PTTT) with CPN repair/reconstruction alone in patient groups that were matched with regard to demographics, mechanism of injury, and surgical timing. Eighty-five percent of patients had improved motor function of the tibialis anterior muscle, peroneal muscles, and common toe extensors to MRC grade 3 or higher. Although the conclusions are limited by the small size of the control group, CPN repair/reconstruction combined with PTTT resulted in improved objective recovery of the CPN as measured clinically and on EMG. No patient who failed initial nerve repair or reconstruction and then underwent a delayed PTTT objectively recovered CPN reinnervation. The authors proposed that the early improvement in dorsiflexion provided by the PTTT allows for "internal rehabilitation" to maintain the flexibility of the ankle joint and surrounding musculature and fosters passive stimulation of the denervated muscles. They felt that this may be more effective than the combination of traditional physical rehabilitation and use of an AFO.

Our experience with delayed management of extensive CPN injury (>6 cm) with nerve reconstruction alone has been similarly disappointing, with outcomes comparable to those reported by Ferraresi and colleagues.[43,44] We have modified our treatment algorithm based on these findings and other available literature (Figure 5).

Previous studies indicate that patients who undergo PTTT—regardless of timing—can return to ambulation without an AFO.[4,31] Nonetheless, there are no reports describing return to competitive sports or participation in activity more strenuous than walking.[34] This is likely due in part to the inability to completely restore dorsiflexion and eversion strength, which affect gait mechanics.[45] It also may be complicated by the long-term increase in the risk of developing a pes planovalgus deformity and/or hindfoot arthrosis.[46] Although early PTTT may prove to be more advantageous for nerve recovery when combined with early nerve repair, it remains the standard of care for the management of equinovarus deformity in the setting of chronic CPN palsy.

Indications for Nerve Transfer

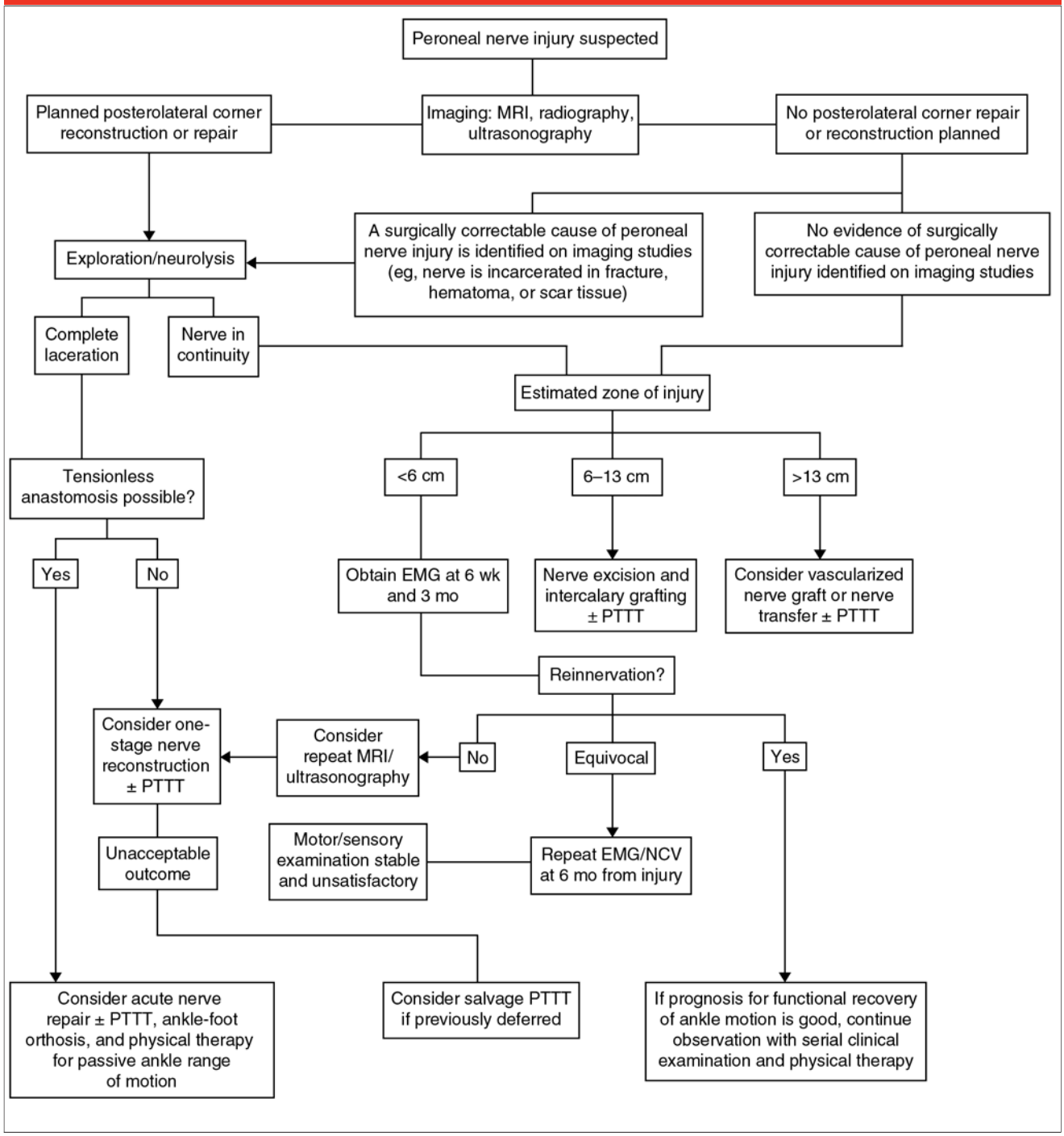
Nerve transfer involves the coaption of a functional but potentially expendable nerve with an injured nerve. In the case of an injured CPN, a tibial nerve branch or fascicle can be used to reanimate the tibialis anterior muscle.[47] With nerve transfer, one of these functional donor nerve fascicles can be placed closer to the motor end plate than to the site of original injury. This reduces the time and distance required for the regenerative nerve to migrate to the motor end plates.[48] For this reason, the advantage of nerve transfer over nerve repair or grafting may be fully realized in the setting of delayed surgical intervention; however, despite encouraging anatomic and limited outcomes studies, no evidence exists to support the benefits of nerve transfer over traditional methods of managing CPN injury.

Summary

CPN injuries associated with MLKI have a poor prognosis for recovery and are challenging to manage,[4,6,34] although emerging evidence and outcomes studies have improved our understanding of the natural history of these complex injuries. In the acute and subacute settings, the inability to quantify the pathophysiology of nerve injury despite advances in nerve imaging precludes the accurate determination of the prognosis for recovery and, therefore, adversely influences clinical decision-making.

Important factors that influence ultimate outcomes include the zone of injury, patient age, graft length, interval from injury to surgical repair, and severity of injury.[32] Nonsurgical management options include physical therapy in combination with or-

Figure 5



Evidenced-based treatment algorithm to help guide decision making when managing peroneal nerve injuries following multiligament knee injuries. EMG = electromyography, NCV = nerve conduction velocity, PTTT = posterior tibial tendon transfer

thoses. Surgical options include one or a combination of the following: neurolysis, primary nerve repair, intercalary nerve grafting, tendon transfer, and nerve transfer. Limited evidence exists in support of early one-stage nerve reconstruction combined with tendon transfers; however, optimal management of these rare injuries continues to change and should be individualized to each patient.[44]

References

Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, references 27 and 28 are level II studies. References 6, 19, 20, 22, 32, and 43 are level III studies. References 5, 9, 24, 29-31, 36, 39-41, 44, 45, and 47 are level IV studies. References 12-15, 17, 18, 35, and 46 are level V expert opinion. The remaining references are cadaver or animal studies, book chapters, or traditional clinical reviews.

References printed in **bold type** are those published within the past 5 years.

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