Surgical Treatment of Foot Drop: Patient Evaluation and Peripheral Nerve Treatment Options

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KEYWORDS

• Foot drop • Peroneal nerve • Direct repair • Neurolysis • Decompression • Nerve graft • Nerve transfer

KEY POINTS

- A detailed clinical history, physical examination, electrodiagnostic studies, and advanced imaging modalities are all helpful diagnostic tools in the evaluation of the patient presenting with foot drop.
- Acute surgical exploration and primary nerve repair is associated with the best postoperative functional outcomes, but is indicated in only select clinical scenarios such as following acute traumatic or iatrogenic injuries with a known or suspected sharp laceration to a peripheral nerve.
- In-situ neurolysis/decompression may be effective for the treatment of conducting neuromas in continuity, whereas autologous nerve grafting may be required for treatment of nonconducting neuromas or traumatic segmental neural injuries not amenable to tension-free primary repair.
- There is limited evidence supporting the use of nerve transfers for the management of foot-drop.

INTRODUCTION/BACKGROUND

Foot drop is a common clinical condition which presents with both sensory deficits and weakness or complete paralysis of ankle dorsiflexion. Patients with foot drop may also present with weakness of the lateral and/or posterior compartments of the leg depending on the location of the pathologic neural lesion. Foot drop is associated with substantial gait abnormalities and a significant increase in the risk of falls and injury.[1,2] The pathophysiology of foot drop is diverse and may be multifactorial in nature. The most common etiology of foot drop is peripheral compression of the common peroneal nerve (CPN), responsible for innervating the tibialis anterior—the primary dorsiflexor of the foot.[1,2] However, foot drop may also occur secondary to peripheral nerve lesions distal or proximal to the level of the CPN.

Effective surgical management of foot drop is highly dependent on the mechanism of initial injury, the duration of clinical symptoms, the severity of neural injury, and the capacity for spontaneous recovery. This review will provide an overview of the surgical nerve repair and reconstructive treatment options available for the management of foot drop as well as their associated clinical outcomes.

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ETIOLOGIES OF FOOT DROP

Traumatic peripheral nerve injuries are common causes of foot drop. Acute neural injuries may occur secondary to blunt contusions, stretch/traction injuries, crushing injuries, and sharp lacerations due to penetrating trauma. The mechanism of injury and associated zone of neural injury have significant implications for the capacity for spontaneous improvement of neural deficits in patients with foot drop following acute trauma. Foot drop may also occur due to peripheral nerve compression at any location along the path of the sciatic and peroneal nerves. The most common etiology of foot drop is compression of the CPN at the level of the fibular neck. Peripheral nerve compression may occur secondary to soft tissue and bony masses, external compression such as due to intraoperative positioning or plaster casting, or repetitive compression from functional activities such as habitual leg crossing or squatting. Iatrogenic injury is also an important cause of foot drop, and orthopedic surgical procedures are the most common cause of iatrogenic peripheral nerve injuries requiring intervention. Nerve palsies resulting in foot drop are well-reported albeit uncommon complications following total hip and total knee arthroplasty, arthroscopic surgical procedures of the knee, and lower extremity fracture fixation. Finally, peripheral nerve lesions secondary to disc herniation or spinal stenosis, external compression, or iatrogenic injury at the level of the lumbar spine and lumbosacral plexus may also result in clinical foot drop. For a detailed description of the diverse etiologies of foot drop, the authors direct readers to the Dwivedi and colleagues' article "Surgical Treatment of Foot Drop: Pathophysiology & Tendon Transfers for Restoration of Motor Function," in this issue.

PATIENT EVALUATION OVERVIEW

Clinical History

Evaluation of the patient presenting with foot drop should begin with a detailed clinical history. It is important to determine the quality and severity of the patient's symptoms, ranging from mild sensory deficits, and motor weakness to dense numbness and flaccid paralysis, as this may influence clinical decision-making. The temporal evolution of the patient's symptoms, including acuity of onset, duration of symptoms, and change in severity over time, should be assessed. Any history of acute injury or trauma before the onset of symptoms should be elicited. Perhaps most importantly, the functional impact of the patient's symptoms on their quality of life and ability to participate in their desired activities should be assessed. The clinician should have a transparent conversation with the patient regarding their most significant functional deficits and realistic goals for recovery. Is the patient a competitive athlete striving to return to high-level sports? Or would the patient be satisfied with the ability to ambulate comfortably in the community using a walker for assistance? Establishing a mutual understanding of patient expectations is critical to creating an effective, patient-centered treatment plan.

Physical Examination

A detailed physical examination of the patient with foot drop is incredibly useful in establishing the severity of the patient's neurologic deficits and localizing the pathologic lesion. A comprehensive motor and sensory examination of the bilateral lower extremities should be performed. The lower extremity should be closely inspected for signs of muscle atrophy, Muscle strength is graded from 0 to 5 using the British Medical Research Council (MRC) scale.[3] Patients with a lesion at the level of the CPN will present with the classic clinical picture of weakness of ankle dorsiflexion, great toe and lesser toe dorsiflexion, and foot eversion with sensory deficits in both the deep and superficial peroneal nerve dermatomes. Ankle inversion strength will be preserved due to the maintenance of tibialis posterior innervation through the tibial nerve. Patients presenting with an isolated lesion of the deep peroneal nerve, although uncommon, would present with preserved foot eversion strength and normal sensation in the superficial sensory nerve distribution. Patients presenting with foot drop secondary to a complete lesion of the sciatic nerve will present with the aforementioned deficits as well as motor and sensory deficits in the tibial nerve distribution. These patients will also have weakness with ankle plantarflexion, great toe and lesser toe plantarflexion, and foot inversion as well as sensory deficits in the tibial and sural nerve dermatomes. Patients with a partial lesion of the sciatic nerve may present with incomplete motor and sensory deficits of varying severity in both the common peroneal and tibial nerve distributions, depending on the fascicular involvement of the sciatic nerve.

Focal tenderness to palpation and a Tinel's sign with radiating pain or paresthesias along the course of a peripheral nerve are useful clinical examination signs which can be used to localize a pathologic lesion. These signs should be used to examine the course of the sciatic

nerve in the posterior thigh, the CPN as it winds around the fibular neck, the deep peroneal nerve along the anterolateral leg, and the superficial peroneal nerve over the lateral compartment as well as its medial and lateral branches at the level of the anterior ankle. The popliteal fossa and course of the CPN at the level of the fibular neck should also be carefully palpated to evaluate for any compressive masses. Lower extremity reflexes including patellar and Achilles reflexes should be evaluated. These reflexes will be normal in the setting of a lesion at, or distal, to the level of the CPN. Abnormally diminished patellar or Achilles reflexes are suggestive of a lower motor neuron lesion proximal to the CPN, such as at the level of the sciatic nerve or the lumbosacral plexus involving the L4 and/or S1 nerve roots. Lower extremity hyperreflexia or other long tract signs such as pathologic clonus or an abnormal Babinski should raise high suspicion for an upper motor neuron lesion affecting the central neural axis. Finally, the patient should be asked to ambulate for evaluation of gait. The ability to actively dorsiflex one's ankle to neutral is critical in achieving foot clearance during gait. Additionally, eccentric contraction of the ankle dorsiflexors following heel-strike allows for the controlled return of the forefoot to the ground during normal walking gait. Patients presenting with complete foot drop may demonstrate the characteristic slap gait during ambulation, with the forefoot uncontrollably striking the ground following heel-strike. These patients may also demonstrate a characteristic steppage gait with compensatory hyperflexion of the hip and knee to achieve foot clearance during swing-through phase of ambulation.

Patients with foot drop may pose a diagnostic challenge due to their wide spectrum of clinical presentation. Patients may present with highly variable lower extremity motor and sensory deficits dependent on both the location and severity of the pathologic lesion. All patients with foot drop should undergo a detailed clinical examination, and in the setting of uncertainty regarding the location of the lesion or disease severity, further diagnostic tests may be necessary.

Electromyography/Nerve Conduction Study

Following a detailed history and physical examination, electrodiagnostic studies by both needle electromyography (EMG) and nerve conduction study (NCS) are valuable tools that assist with localization of the neural lesion and zone of injury, evaluation of injury severity, and monitoring for nerve recovery. EMG assesses a nerve's resting membrane potential and electrical response to a stimulus, while NCS assesses the integrity and conductive ability of a nerve. These studies are particularly helpful when evaluating potential etiologies of foot drop apart from CPN injury at the level of the fibular neck. It is important to perform EMG and NCS studies within the distributions of the CPN, L4-S2 nerve roots, lumbosacral plexus, and sciatic nerve to accurately localize the site of the injury; thus, the dorsiflexors and evertors of the foot, extensors of the toes, gastrocnemius, tibialis posterior, hamstring, short head of the biceps femoris, and gluteal muscles should be tested.[1] Electrodiagnostic studies may also be performed every 6 to 12 weeks following initial diagnosis to monitor for axonal regeneration and nerve recovery.

Baseline electrodiagnostic testing should be performed in all patients presenting with new-onset foot drop. In cases of foot drop resulting from acute traumatic injury, delayed testing should occur 4 to 6 weeks following the traumatic event. EMG/NCS testing earlier than 4 weeks following initial injury may result in false negative study results as Wallerian degeneration is not complete until approximately 3 weeks postinjury.[4,5] Repeat testing is particularly helpful in cases of axonal loss whereby electrodiagnostic studies may reveal increased nerve activity and motor unit recruitment before clinical signs of recovery are evident. In this instance, repeat testing should be performed every 6 to 12 weeks to monitor for improvement.

Latency, nerve conduction velocity (NCV), and amplitude are the 3 primary measures of nerve function assessed by NCS. Latency and NCV provide insight into the integrity of the myelin sheath surrounding the axon, while amplitude reflects the number of viable, conducting axons within the nerve. These parameters are directly influenced by the nature and severity of the neural injury. In neurapraxic injuries, NCS will reveal delayed distal latencies and NCVs with amplitude unaffected. Conversely, axonotmetic injuries will demonstrate decreased amplitudes on NCS due to axonal loss. In addition to these three parameters, analysis of the resulting sensory nerve action potentials (SNAPs) and compound motor action potentials (CMAPs) include examination of both the duration and shape of these waveforms. SNAP duration reflects synchrony of conduction through individual nerve fibers, whereas CMAP also reflects conduction through contraction of the muscle fibers.[4,5]

Assessment of EMG involves examining activity at three phases: insertional activity, resting phase, and activation phase. In the first 3 weeks following nerve injury when Wallerian degeneration is occurring, EMG will show increased insertional activity secondary to a hyperexcitable membrane. The resting phase will also demonstrate features of hypersensitivity in the form of fibrillation potentials and positive sharp wave potentials due to spontaneous depolarization. Active denervation will reveal prominent fibrillation and positive sharp wave potentials. Features of hypersensitivity in the resting phase can be observed within days following the nerve injury and can last for months thereafter. Motor unit action potentials (MUAPs) are analyzed in the activation phase and provide significant insight into both the nature and severity of the neural injury. In neurotmetic injuries, MUAPs will be completely absent whereas in axonotmetic injuries MUPAs may be absent or decreased. The amplitude and duration of the waveform also provides insight into the timing of the injury. Subacute injuries are associated with increased MUAP duration, while chronic injuries are associated with increased MUAP amplitude. During the regeneration phase, MUAP duration may vary while MUAP amplitude will be decreased.[4,5]

Dy and colleagues[4] and Lee and colleagues[5] have both described the principles of EMG/NCS interpretation and the authors direct readers to their reviews for further details on the fundamentals of these electrodiagnostic studies.

Imaging

Diagnostic imaging studies may be helpful adjuncts to a detailed history and physical examination in certain clinical scenarios. Plain radiography should be considered as part of the initial workup for patients presenting for evaluation following an acute traumatic injury, as well as for evaluation of appropriate implant positioning in the setting of postoperative nerve palsy following total hip arthroplasty, total knee arthroplasty, and high tibial osteotomy. Ultrasound and MRI may be useful diagnostic tools for the assessment of perineural scarring, focal nerve enlargement, and nerve continuity in the setting of traumatic or iatrogenic injuries. MRI may be used for assessment of multiligamentous knee injuries as well as for detailed evaluation of intraneural and extraneural compressive masses. An image-guided needle biopsy may be necessary for definitive pathologic diagnosis for masses with concerning features. Referral to an orthopedic oncologist should be considered for these lesions. Surgical excision of an intraneural lesion should only be performed by an experienced peripheral nerve surgeon with a fundamental understanding of oncologic principles. Finally, radiographic evaluation of the lumbar spine including plain radiographs, CT, and MRI may be useful modalities for the evaluation of foot drop believed to be secondary to lumbosacral nerve root compression.

Classification of Nerve Injury

All of the clinical information gathered through a careful history, detailed physical examination, electrodiagnostic studies, and/or advanced imaging must be considered together to determine the location and severity of the neural lesion responsible for the patient's foot drop. In 1942, Seddon[6] described a classification system for the severity of peripheral nerve injuries based on the degree of structural disruption of the peripheral nerve architecture. In 1951, Sunderland[7] published a modification to this classification to better describe the severity and clinical consequences of intermediate-grade peripheral nerve injuries (Table 1). The Seddon and Sunderland classification systems provide useful frameworks through which to consider the prognosis of peripheral nerve injuries and ultimately guide management. Neurapraxic injuries, marked by a conduction block with possible segmental demyelination but without axonal loss, generally have a good prognosis with spontaneous recovery and should be managed conservatively. Neurotmetic injuries, marked by complete discontinuity of the peripheral nerve, carry no capacity for spontaneous recovery and require surgical intervention for return of motor and sensory function. Intermediate-grade axonotmetic injuries, marked by axonal loss resulting in Wallerian degeneration but without complete nerve transection, carry a more variable prognosis and should be monitored closely, with surgical intervention generally indicated in circumstances without spontaneous improvement in motor and sensory deficits.

CONSERVATIVE MANAGEMENT OPTIONS

Initial conservative management of foot drop is appropriate for most patients presenting with evidence of a neurapraxic or low-grade axonotmetic injury, as described by the Seddon and Sunderland classification systems.[6,7] Conservative management consists primarily of activity modification such as the cessation of habitual leg crossing or prolonged squatting/kneeling, provision of an ankle-foot orthosis (AFO) to

Table 1
Seddon and Sunderland classifications of peripheral nerve injuries

Seddon	Sunderland	Neural Injury	Potential for Recovery
Neurapraxia	I	Intrafasciular edema, conduction block with possible segmental demylenation; no axonal loss	Full/excellent; 1 wk – 3 mo
Axonotmesis	II	Axonal disruption with intact endoneural tube	Full/good; 1-6 mo
Axonotmesis	III	Axonal disruption with torn endoneurium	Incomplete/fair; slow recovery, may be >12 mo
Axonotmesis	IV	Axonal disruption with torn endoneurium & perineurium; intact epineurium	Incomplete/poor; neuroma in continuity
Neurotmesis	V	Complete nerve discontinuity	No recovery without surgical intervention

Data from Seddon HJ: A classification of nerve injuries. Br Med J 1942;2(4260):237-239 and Sunderland S: A classification of peripheral nerve injuries producing loss of function. Brain 1951;74(4):491-516.

assist with ambulation, and physical therapy for the initiation of active and passive range of motion exercises to maintain full passive joint range of motion. Patients should be examined every 4 to 6 weeks for repeat assessment of motor strength and sensory deficits. A Tinel's sign that advances distally along the course of a nerve on repeat clinical assessments is a useful sign of regenerating axonal injury. A strong Tinel's sign that remains stationary is concerning for a nonregenerating injury and neuroma formation. Repeat EMG/NCS studies may be performed at 6 to 12 weeks to assess for early recovery of a neurapraxic or axonotmetic injury. Patients who fail to demonstrate significant spontaneous recovery on repeat clinical assessments and/or electrodiagnostic studies may be candidates for surgical intervention to regain sensorimotor function.[1,8–11]

SURGICAL TREATMENT OPTIONS

Acute Surgical Exploration and Primary Nerve Repair

Timing of surgical intervention for the management of foot drop is controversial and is dependent on a variety of clinical factors. Acute surgical exploration and primary nerve repair are indicated in only select clinical scenarios, as in most circumstances, time must be allowed for the zone of peripheral nerve injury to declare itself or to monitor for spontaneous recovery of nerve function. In the setting of an acute traumatic or iatrogenic injury with a known sharp laceration of the nerve, acute surgical exploration and primary nerve repair are warranted. This may occur such as during stab injuries to the extremity or intraoperative injuries during open or arthroscopic procedures around the knee and posterior femur. Patients who are found to have a new postoperative foot drop after open reduction and internal fixation of periarticular fractures about the knee or percutaneous lower extremity procedures (ie, varicose vein procedures, less invasive stabilization system tibial plating) may also be candidates for acute intervention.[9,12,13] Patient who are found to have a discrete compressive mass causing their neurologic deficits may also benefit from early intervention for surgical excision following a thorough workup and evaluation of the mass. Finally, patients with CPN palsy following a multiligamentous knee injury or periarticular tibia fracture may benefit from a form of early intervention, although surgical timing and intra-operative management of neural injuries is controversial within this population. Communication with the surgeon performing the multiligamentous knee reconstruction or fracture fixation for these patients should begin early, before the patient's sentinel surgical procedure. In general, the authors provide technical assistance if requested for intraoperative assessment of neural injury and possible CPN decompression, but do not advocate for CPN reconstruction less than 3 weeks from injury as the zone of neural injury has yet to declare itself within this time period. Acute repair or nerve reconstruction is only performed within the first 3 weeks if the nerve is found to be in discontinuity, through either preoperative imaging or intraoperative assessment. A detailed description of the authors' approach to surgical timing within this population can be found in a recent review by Dy and colleagues.[11]

Early intervention should focus on a thorough exploration and evaluation of the peripheral nerve to identify the site of injury. In the setting of a partial or complete sharp transection of the nerve, the nerve must be sufficiently mobilized with the aim of performing a primary nerve repair with a tension-free coaptation. The nerve stumps are prepared with use of a number 15 or 11 scalpel blade against a wooden disposable tongue blade to identify the level of healthy-appearing nerve architecture with pooching individual fascicles. In the acute setting following a sharp laceration before neuroma formation has occurred, minimal nerve stump resection is typically necessary. There is no clear clinical consensus on the optimal surgical repair technique, although most peripheral nerve surgeons elect to perform an epineural repair using 8 to 0 or 9 to 0 monofilament nylon suture.[14–17] Giddins and colleagues[17] demonstrated that 8 to 0 nylon suture tended to pull out of repaired nerve endings and 10 to 0 nylon sutures typically failed under tension, whereas 9 to 0 nylon suture withstood the greatest distractive force. Prior studies have failed to demonstrate any single superior epineural or fasicular repair technique.[18] It is our preference to perform an epineural repair using several 9 to 0 monofilament nylon sutures to achieve a tension-free coaptation. If the nerve ends cannot be approximated without rupture of a 9 to 0 nylon suture or without gapping at the repair site, the nerve coaptation is under excessive tension and alternate techniques such as use of a small nerve graft must be considered. Following primary repair, in our practice the nerve coaptation site is then reinforced with fibrin glue,[19,20] although augmentation with a variety of bioabsorbable nerve wraps/conduits has also been described.[21]

Delayed Surgical Exploration and Neurolysis/Decompression

In the absence of a known sharp laceration or acute iatrogenic injury to a nerve, most patients presenting with foot drop will initially undergo a period of conservative management with serial examinations to assess for spontaneous recovery of nerve function. Delayed surgical intervention for exploration and neurolysis/decompression of the nerve may be warranted for patients who fail to show improvements in motor and sensory function within 3 to 6 months. This is most commonly required for foot drop secondary to common peroneal palsy, the most common compressive neuropathy of the lower extremity.[22,23] Delayed surgical exploration and neurolysis/decompression may also be indicated following blunt trauma, traction injuries, and crush injuries which fail to spontaneously improve by 3 to 6 months, as well as for persistent motor and sensory deficits following total hip arthroplasty and total knee arthroplasty despite conservative management. Decompression may be necessary at the level of the CPN at the fibular neck, the deep or superficial peroneal nerves in the distal leg, or the sciatic nerve in the posterior thigh depending on the location of neural injury. Patients presenting with foot drop secondary to ballistic injuries typically have an extensive zone of neural injury and are not good candidates for decompression alone. Patients with signs of active muscle denervation such as fibrillations and positive sharp waves on their initial EMG/NCS studies may benefit from early nerve exploration and decompression. Patients with motor and/or sensory abnormalities without signs of active muscle denervation on their initial EMG/NCS undergo initial conservative management. If a repeat EMG/NCS at 3 months suggests a neurapraxic or axonometic injury without any improvement in motor or sensory signals or new signs of active muscle denervation, the authors recommend proceeding with surgical exploration and decompression at that time. There is no clear consensus regarding optimal management if the 3-month EMG/NCS demonstrates signs of nerve recovery such as increased recruitment of motor unit potentials or voluntary motor units. Some peripheral nerve surgeons would advocate for decompression of the nerve at this time, while others would continue to monitor for an additional 3 to 6 months for continued spontaneous recovery. There is also controversy surrounding timing of intervention for foot drop secondary to multiligamentous knee injuries. In this scenario, the authors generally do not advocate for early exploration and reconstruction of the nerve less than 3 weeks from injury to allow time for the zone of injury to present itself. Nerve exploration and decompression with possible nerve grafting may be considered greater than 3 weeks from injury at the time of ligament reconstruction if preoperative imaging demonstrates nerve discontinuity, or at 3 months in the setting of neurapraxic or axonotmetic injuries without significant improvement on repeat EMG/NCS studies.

Author's Preferred Technique: Common Peroneal Nerve Decompression at the Fibular Neck

The most common site of peripheral nerve entrapment or injury resulting in foot drop is at

the level of the CPN at the fibular neck. Prior studies have demonstrated favorable results following surgical exploration and neurolysis of the CPN at this level with 88% of patients recovering useful function in one series.[24] Our preferred surgical technique for CPN decompression at the fibular neck is as follows:

The patient is positioned in the supine position with a bump under the ipsilateral hip and a padded foam leg ramp under the operative knee. The fibular head is palpated and marked. A 6–8 cm curvilinear incision is made 1–2 cm distal to and centered around the fibular head. The skin and subcutaneous tissue is sharply incised using a number 15 scalpel and the dissection is carried deeply to the level of the fascia overlying the lateral compartment, ensuring to raise full-thickness soft tissue flaps. Care is taken to avoid injury to the lateral sural cutaneous nerve in the proximal aspect of the incision. The CPN may be palpated along its course just distal to the fibular head in slim patients, although this may be difficult in patients with a higher body mass index. Rather, the CPN is identified just posterior to the biceps femoris tendon within the proximal portion of the surgical incision and is followed distally to the level of the fibular neck. The CPN is neurolysed carefully along its course using tenotomy scissors. The fascia overlying the lateral compartment and the lateral $^1/_2$ of the anterior compartment is sharply incised. The first primary site of nerve compression that is now encountered is the posterior crural intermuscular septum. Tenotomy scissors are used to carefully define the anterior and posterior surfaces of the septum and the septum is sharply divided in its entirety, ensuring to avoid injury to any small perforating vessels. A secondary site of nerve compression may be found posterior to the CPN at this level, at the leading edge of the anterior fascia overlying the soleus muscle. If this fascia is felt to be tight and a source of compression, it is carefully divided in its entirety. The CPN is then neurolysed medially until the second primary site of nerve compression is identified: the anterior crural intermuscular septum. The anterior and posterior surfaces of this septum are defined in a similar fashion using tenotomy scissors and the septum is carefully divided along its entire width. The third and final primary site of nerve compression which must be identified is the innominate septum between the extensor hallucis longus and tibialis anterior muscle bellies, and is encountered approximately 1 cm medial to the anterior intermuscular septum. The innominate septum is then carefully divided in its entirety. The CPN is then confirmed to be completely decompressed without any remaining sites of nerve compression along its entire length around the fibular neck to the tibial crest medially. The branching point of the CPN into the deep peroneal nerve, superficial peroneal nerve, and the anterior recurrent branch is visualized and confirmed to be decompressed. If there is any compression noted of the proximal superficial peroneal nerve, the lateral compartment fascia overlying the nerve is released for 1–2 cm distally along the course of the nerve. The wound is then irrigated thoroughly and a layered closure is performed using 3 to 0 Monocryl deep dermal sutures and a running 4 to 0 Monocryl subcuticular suture.

Nerve Grafting and Nerve Transfers

For patients in which a neuroma in continuity is identified during surgical exploration, the nerve should be stimulated intraoperatively with a nerve stimulator. If a nerve action potential (NAP) is unable to conduct across the neuroma, neurolysis alone will not yield satisfactory clinical recovery postoperatively and excision of the neuroma must be performed.[24–26] In this circumstance as well as in situations when there is traumatic segmental loss of the peripheral nerve or when there is a wide zone of injury (often seen following blunt, traction, and crush injuries), primary nerve repair with a tension-free coaptation may not be possible. In these patients, interposition cable nerve grafting may be performed. For reconstruction of a mixed motor and sensory peripheral nerve such as the sciatic or CPN with >1 cm gap, the authors recommend use of an autologous sural nerve graft harvested from the contralateral lower extremity. The ipsilateral sural nerve may also be used if preoperative EMG/NCS studies demonstrate a viable nerve without any evidence of injury. Autologous nerve graft provides a structurally inert and nonimmunogenic scaffold for axonal regeneration as well as neurotrophic factors and viable Schwann cells which are not present in nonautologous grafting alternatives.[8,27] It is critical to place the intercalary nerve graft outside of the zone of injury to facilitate axonal regeneration and avoid neuroma formation. Serial sectioning of the nerve must be performed intraoperatively until normal fascicular architecture is noted on both ends of the nerve stumps. Similar to primary nerve repair, the graft must be fixed to the proximal and distal nerve stumps through tension-free coaptations.

Additional sources of nerve graft for peripheral nerve reconstruction in the setting of foot

drop have been proposed and include nerve allograft as well as biologic and synthetic nerve conduits. Although limited series have demonstrated early favorable results with use of these alternatives in the setting of short-gap, small diameter nerve defects, the literature base supporting their use for larger diameter peripheral nerves is poor.[28,29] The authors do not recommend use of alternatives to nerve autograft for reconstruction of mixed motor and sensory peripheral nerve defects, such as in the surgical management of foot drop. It should be noted that there is no clear consensus among peripheral nerve surgeons regarding the optimal management of foot drop secondary to high-grade axonometic or neurotmetic injuries at the level of the CPN. As primary nerve repair is often impossible for these patients, some surgeons advocate for nerve grafting for shorter gaps <6 to 12 cm while others recommend early referral to a foot & ankle specialist for consideration of tendon transfers.[1,11]

Another potential treatment option for reconstruction of a peripheral nerve defect when primary repair is not possible is a nerve transfer. A nerve transfer entails the coaptation of a well-functioning and expendable whole donor nerve, or nerve fascicle, to a more important, injured recipient nerve to reinnervate the recipient nerve's downstream motor targets. A potential advantage of a nerve transfer over nerve grafting is the ability to coapt the donor nerve close to the target motor end plate, thereby decreasing the distance required for axonal regeneration and consequently the time until reinnervation.[30] A nerve transfer also requires creation of only one nerve coaptation rather than 2 required for a nerve graft (proximal and distal), thereby theoretically resulting in only one potential site of axonal loss as the nerve regenerates across the coaptation. Although a number of upper extremity nerve transfers have become well established in the management of upper extremity peripheral nerve lesions, clinical results following lower extremity nerve transfers have not been as reliable. A limited number of lower extremity nerve transfers for management of foot drop have been described, including transfer of a motor branch of the tibial nerve or soleus nerve to the deep peroneal nerve.[31–33] Although initial evaluation of these nerve transfers yielded promising clinical results,[33] further investigations have demonstrated poor reliability both between and within centers.[34,35] Another potential alternative involves performing a reverse end-to-side nerve transfer for foot drop in order to augment a regenerating peripheral nerve with additional donor axons to accelerate target muscle reinnervation.[36] Ultimately, the use of peripheral nerve transfers in the management of foot drop is controversial and is supported by a limited base of literature with mixed clinical results. The authors do not currently use nerve transfers in our practice for the management of foot drop, although this is an area of continued investigation.

Patients with refractory foot drop despite nerve reconstructive efforts may be candidates for dynamic tendon transfers to restore active ankle dorsiflexion. The authors direct readers to the associated chapter "Surgical Treatment of Foot Drop: Pathophysiology & Tendon Transfers for Restoration of Motor Function" for the authors' algorithm regarding the use of tendon transfers for the management of foot drop.

OUTCOMES

Foot drop secondary to CPN palsy following knee dislocation has been associated with poorer functional outcomes. Krych and colleagues[37] found that of 27 patients with peroneal nerve palsy who underwent multiligamentous knee reconstruction, 35% required use of an AFO at final follow-up. Only 83% of patients with a partial nerve palsy and 38% of patients with a complete nerve palsy recovered antigravity ankle dorsiflexion despite treatment, which consisted of a combination of conservative management, neurolysis, nerve transfers, and tendon transfers.[37] Further studies have demonstrated similar results, with spontaneous recovery of motor and sensory deficits following knee dislocation occurring in only 14% to 56% of patients.[38] Younger age (<30 years) has been demonstrated to be predictive of a higher likelihood of spontaneous nerve recovery following knee dislocation.[39]

Outcomes Following Nerve Neurolysis/ Decompression and Nerve Grafting

When properly indicated, the use of nerve decompression/neurolysis for treatment of foot drop has been shown to have favorable outcomes. In an analysis of 318 operatively treated CPN injuries, Kim and colleagues[24] demonstrated that 88% of patients with recordable intraoperative NAPs across the zone of neural injury recovered useful function following neurolysis. Seidel and colleagues[40] demonstrated that of 22 patients with a traumatic peroneal nerve injury who had recordable intraoperative NAPs, consistent with a neuroma in continuity,

73% had a good functional outcome following neurolysis with recovery of MRC 4 or 5 strength, obviating the need for an AFO. Thoma and colleagues examined 20 patients who underwent neurolysis for CPN palsy and found that 95% experienced improvement of at least one MRC grade in motor strength postoperatively, and all 3 patients who underwent neurolysis within 4 months of injury had improvement from MRC 0 to MRC 4 or 5 following intervention. Similar favorable results have been demonstrated following neurolysis for more proximal injuries, such as lesions in continuity of the sciatic nerve. Kim and colleagues[41] examined outcomes in 353 operatively treated sciatic nerve injuries and found that between 71% and 96% of patients with recordable intraoperative NAPs recovered at least MRC grade 3 motor strength postoperatively. Murovic[42] reported good functional recovery in 78% to 95% of patients who underwent neurolysis for thigh-level sciatic nerve injuries and in 69% to 86% of patients who underwent neurolysis for buttock-level sciatic nerve injuries, with better recovery in the tibial than the CPN division.

When a tension-free coaptation is technically possible, primary nerve repair for management of foot drop secondary to traumatic or iatrogenic injuries has demonstrated favorable outcomes. Kim and colleagues[24] reported that 16 of 19 patients (84%) who underwent end-to-end suture repair following CPN injury recovered at least MRC 3 strength by 24 months and did not require use of an AFO. Gürbüz and colleagues[43] reported that 4 of 7 patients who underwent end-to-end suture repair following peroneal nerve injury had recovered M4 or M5 function at a mean follow-up of 30 months. Similar outcomes have been reported following primary suture repair of sciatic nerve injuries resulting in foot drop, although clinical recovery seems more limited following injury to the peroneal division of the sciatic nerve. In the largest series to date, Kim and colleagues[41] and a subsequent review by Murovic[42] reported that following primary suture repair, 73% of buttock-level and 93% of thigh-level tibial division injuries recovered at least MRC 3 motor strength, compared with 30% of buttock-level and 69% of thigh-level peroneal division injuries.

If a tension-free nerve repair is not feasible or has previously failed, intercalary autologous nerve grafting may be performed to facilitate axonal regeneration. Several large series have demonstrated that outcomes following autologous nerve grafting for management of foot drop are most dependent on the length of the zone of neural injury and thus the graft length necessary for reconstruction. In the largest study to date, Kim and colleagues[24] reported outcomes of 138 patients who underwent graft reconstruction for management of CPN injury. Of 36 patients who required a graft <6 cm in length, 27 (75%) had a good functional outcome with recovery of at least MRC 3 strength postoperatively. When a longer graft length of between 6 and 12 cm was required, only 24 of 64 patients (38%) recovered at least MRC 3 strength postoperatively. In patients requiring a graft length of 13 to 24 cm, only 6 of 38 (16%) had a good outcome. Seidel and colleagues[40] reported that 4 of 9 patients (44%) who underwent autologous sural nerve grafting for traumatic peroneal nerve injuries with a graft length <6 cm recovered MRC 4 or 5 motor strength, compared with only 1 of 9 patients (11%) in which a graft length 6 cm was required. Cho and colleagues[44] recently reported outcomes following surgical management of 84 sports-related CPN injuries. In their series, 70% of patients who required an autologous nerve graft <6 cm in length had a good outcome with recovery of at least MRC 3 strength postoperatively, compared with 43% of patients who required a graft length between 6 and 12 cm and only 25% of patients who required a graft length between 13 and 24 cm. In the largest study of its kind to date, Kim and colleagues[41] reported outcomes following graft repair for proximal sciatic nerve injuries and demonstrated that functional recovery is particularly limited following peroneal division injury. In their series, a good functional outcome (recovery of MRC grade 3 strength) was achieved in 21 of 34 (62%) patients with buttock-level and 43 of 54 (80%) of patients with thigh-level tibial division injuries, compared with only 9 of 37 (24%) of patients with buttock-level and 22 of 49 (45%) of patients with thigh-level peroneal division injuries.

In a recent review of 28 studies evaluating outcomes following the surgical management of CPN injuries, George and Boyce[45] reported good functional recovery of MRC grade 4 or 5 motor strength in 80% of patients following neurolysis, 37% of patients following direct suture repair, and 36% of patients following nerve grafting. Good functional outcomes were achieved in 44% of patients who underwent nerve grafting within 6 months and 64% of patients requiring a graft length <6 cm, compared with only 12% of patients who underwent nerve grafting after 12 months and 11% of patients who required a graft length greater than

12 cm. In general, the authors agree with most peripheral nerve surgeons that autologous nerve grafting is a valuable reconstructive option for the management of foot drop in the setting of neural injuries with a gap <6 cm in length. A detailed, informed discussion regarding the prognosis and timeline of recovery following nerve grafting versus dynamic tendon transfers must be had with the patient preoperatively in the setting of anticipated nerve gaps of 6 to 12 cm. The authors currently do not perform nerve grafting for the management of foot drop in the setting of nerve gaps greater than 12 cm, and these patients are referred to a foot and ankle specialist for consideration of dynamic tendon transfers.

Outcomes Following Nerve Transfer

Multiple lower extremity nerve transfers for the management of foot drop have been described, although with mixed reported outcomes in the literature. An early series by Nath and colleagues[33] in 2008 reported favorable outcomes of 14 patients who underwent nerve transfer of either a motor fascicle of either the superficial peroneal or the tibial nerve to the deep peroneal nerve. In this series, 11 of 14 (79%) of patients recovered MRC grade 3+ to 4+/5 ankle dorsiflexion strength, 1 patient recovered MRC grade 3 strength, and 2 patients had no motor recovery postoperatively. However, later studies evaluating outcomes following nerve transfer for foot drop have demonstrated mixed results both within and between institutions. Flores and colleagues[31] reported that only 2 of 10 (20%) patients with follow-up who underwent a transfer of the soleus branch of the tibial nerve to the deep peroneal nerve achieved a good functional outcome of MRC 3 or 4 ankle dorsiflexion strength. Giuffre and colleagues[35] reported that only 4 of 11 (36%) of patients who underwent transfer of a fascicle of the tibial nerve to the motor branch of the tibialis anterior recovered MRC grade 3 or 4 ankle dorsiflexion strength postoperatively, and 4 of 11 (36%) of patients did not achieve any motor recovery. In a series of 6 patients who underwent transfer of the soleus branch of the tibial nerve to the deep peroneal nerve, Emamhadi and colleagues[32] reported that only 2 patients (33%) achieved recovery of at least MRC grade 3 ankle dorsiflexion strength postoperatively. In a recent meta-analysis of 14 studies with a total of 41 patients who underwent nerve transfer of tibial nerve (n = 36) or superficial peroneal nerve (n = 5) fascicles to the deep peroneal nerve (n = 24) or tibialis anterior branch (n = 17), Head and colleagues[34] demonstrated a bimodal distribution of motor recovery postoperatively with a mean MRC grade of only 2.1 for ankle dorsiflexion strength at final follow-up. Given the limited evidence of their efficacy in the literature, the authors do not currently use nerve transfers in our practice for the surgical management of foot drop.

SUMMARY

Foot drop, marked by partial or complete lower extremity sensorimotor palsy resulting in weakness with ankle dorsiflexion, is a common condition with a wide spectrum of clinical presentation. Underlying etiologies of foot drop are diverse in nature and a detailed clinical history and physical examination are critical in understanding the pathophysiology of foot drop and the capacity for spontaneous neural recovery. Electrodiagnostic studies are useful tools in determining injury severity, evaluating for signs of active muscle denervation, and actively monitoring for signs of spontaneous neural recovery. Initial treatment options for most cases of foot drop entail conservative measures including activity modification, functional bracing, and physical therapy to maintain passive joint range of motion. Surgical nerve repair and reconstructive options for foot drop are diverse and depend largely on the mechanism and zone of neural injury, the time elapsed since onset of symptoms, and the degree of spontaneous recovery of motor, and sensory deficits. Acute surgical exploration and primary nerve repair are indicated in the setting of acute penetrating trauma and sharp lacerations of a peripheral nerve. Functional outcomes are favorable following neurolysis alone or nerve reconstruction with an autologous nerve graft <6 to 12 cm in length, while outcomes following reconstruction with an autologous graft greater than 12 cm in length are comparatively poor. Although allogeneic, bioartificial, and synthetic nerve graft alternatives are available commercially, the authors do not recommend their use for the treatment of mixed sensorimotor peripheral nerve lesions resulting in foot drop. Nerve transfers are effective reconstructive options for upper extremity peripheral nerve lesions, but the evidence supporting their use in the lower extremity is limited, and the authors do not currently use nerve transfers for the management of foot drop. Dynamic tendon transfers are indicated for patients with refractory, severe foot drop with adequate tibial nerve motor function.

CLINICS CARE POINTS

- A detailed clinical history in patients presenting with foot drop is essential and should elicit the mechanism of injury, the severity of sensorimotor deficits, the duration of symptoms, and the degree of spontaneous recovery since symptom.
- A comprehensive motor and sensory physical examination is critical and special attention should be paid to muscle strength testing and detection of any tibial nerve deficits (most often indicating a level of injury proximal to the knee and potentially limiting options for dynamic tendon transfer) and an advancing Tinel's sign (suggestive of axonal regeneration).
- EMG/NCS studies are useful diagnostic tools and should be performed at least 4 to 6 weeks following an acute traumatic injury. Neurapraxic injuries demonstrate prolonged sensory/motor latencies and/or conduction velocities without decreased amplitudes, while axonotmetic and neurotmetic injuries demonstrate diminished amplitudes consistent with axonal loss. Fibrillations and positive sharp waves are indicators of active muscle denervation. Absent MUAPs are suggestive of neurotmetic or high-grade axonotmetic injuries.
- Indications for acute exploration and primary nerve repair for foot drop include known sharp peripheral nerve lacerations secondary to acute trauma or iatrogenic injury and new, severe postoperative deficits.
- Neurolysis alone is indicated for neuromas in continuity capable of conducting NAPs across the zone of injury, while autologous nerve grafting must be performed for nonconducting neuromas or segmental nerve loss.
- In the setting of CPN palsy following multiligamentous knee injury, early communication with the surgeon performing the knee reconstruction is essential. If requested, the authors provide technical assistance for intra-operative assessment of neural injury and possible CPN decompression, but do not advocate for CPN reconstruction less than 3 weeks from injury as the zone of neural injury has yet to declare itself within this time period.
- Patients with a zone of injury >6 to 12 cm or presenting greater than 12 mo from the onset of foot drop should be strongly considered for dynamic tendon transfers rather than nerve reconstruction.

DISCLOSURE

One author (J.E. Johnson) is a consultant for Arthrex and Stryker and has equity in CrossRoads Medical, but has no financial conflicts of interest with this topic. The other authors have nothing to disclose.

REFERENCES

1. Stewart JD. Foot drop: where, why and what to do? Pract Neurol 2008;8(3):158–69.
2. Poage C, Roth C, Scott B. Peroneal Nerve Palsy: Evaluation and Management. J Am Acad Orthop Surg 2016;24(1):1–10.
3. Medical Research Council; Nerve Injuries Research Committee. Aids to the investigation of the peripheral nervous system. London: His Majesty's Stationery Office; 1942.
4. Dy CJ, Colorado BS, Landau AJ, et al. Interpretation of Electrodiagnostic Studies: How to Apply It to the Practice of Orthopaedic Surgery. J Am Acad Orthop Surg 2021. https://doi.org/10.5435/JAAOS-D-20-00322.
5. Lee DH, Claussen GC, Oh S. Clinical nerve conduction and needle electromyography studies. J Am Acad Orthop Surg 2004;12(4):276–87.
6. Seddon HJ. A classification of nerve injuries. Br Med J 1942;2(4260):237–9.
7. Sunderland S. A classification of peripheral nerve injuries producing loss of function. Brain 1951;74(4):491–516.
8. Mook WR, Ligh CA, Moorman CT 3rd, et al. Nerve injury complicating multiligament knee injury: current concepts and treatment algorithm. J Am Acad Orthop Surg 2013;21(6):343–54.
9. Pulos N, Shin EH, Spinner RJ, et al. Management of iatrogenic nerve injuries. J Am Acad Orthop Surg 2019;27(18):e838–48.
10. Garg B, Poage C. Peroneal nerve palsy: evaluation and management. J Am Acad Orthop Surg 2016;24(5):e49.
11. Dy CJ, Inclan PM, Matava MJ, et al. Current concepts review: common peroneal nerve palsy after knee dislocations. Foot Ankle Int 2021;42(5):658–68.
12. Giannas J, Bayat A, Watson SJ. Common peroneal nerve injury during varicose vein operation. Eur J Vasc Endovasc Surg 2006;31:443–5.
13. Pichler W, Grechenig W, Tesch NP, et al. The risk of iatrogenic injury to the deep peroneal nerve in minimally invasive osteosynthesis of the tibia with the less invasive stabilisation system: A cadaver study. J Bone Joint Surg Br 2009;91:385–7.
14. Rowshan K, Jones NF, gupta R. Current Surgical Techniques of Peripheral Nerve Repair. Oper Tech Orthop 2004;14:163–70.
15. Dahlin LB. Techniques of peripheral nerve repair. Scand J Surg 2008;97(4):310–6.

16. Goldberg SH, Jobin CM, Hayes AG, et al. Biomechanics and histology of intact and repaired digital nerves: an in vitro study. J Hand Surg Am 2007;32(4):474–82.
17. Giddins GE, Wade PJ, Amis AA. Primary nerve repair: Strength of repair with different gauges of nylon suture material. J Hand Surg Br 1989;14:301–2.
18. Spinner RJ, Kline DG. Surgery for peripheral nerve and brachial plexus injuries or other nerve lesions. Muscle Nerve 2000;23(5):680–95.
19. Ornelas L, Padilla L, Di Silvio M, et al. Fibrin glue: an alternative technique for nerve coaptation–Part I. Wave amplitude, conduction velocity, and plantar-length factors. J Reconstr Microsurg 2006;22(2):119–22.
20. Ornelas L, Padilla L, Di Silvio M, et al. Fibrin glue: an alternative technique for nerve coaptation–Part II. Nerve regeneration and histomorphometric assessment. J Reconstr Microsurg 2006;22(2):123.
21. Aberg M, Ljungberg C, Edin E, et al. Clinical evaluation of a resorbable wrap around implant as an alternative to nerve repair: A prospective, assessor-blinded, randomised clinical study of sensory, motor and functional recovery after peripheral nerve repair. J Plast Reconstr Aesthet Surg 2009;62:1503–9.
22. Masakado Y, Kawakami M, Suzuki K, et al. Clinical neurophysiology in the diagnosis of peroneal nerve palsy. Keio J Med 2008;57(2):84–9.
23. King J. Peroneal neuropathy. In: Frontero J, Silver K, Rizzo T, editors. Essentials of physical medicine and rehabilitation. 2nd edition. Philadelphia: Saunders; 2008. p. 389–93.
24. Kim DH, Murovic JA, Tiel RL, et al. Management and outcomes in 318 operative common peroneal nerve lesions at the Louisiana State University Health Sciences Center. Neurosurgery 2004;54(6):1421–8 [discussion: 1428–9].
25. Oberle JW, Antoniadis G, Rath SA, et al. Value of nerve action potentials in the surgical management of traumatic nerve lesions. Neurosurgery 1997;41(6):1337–42 [discussion: 1342–4].
26. Nelson KR. Use of peripheral nerve action potentials for intraoperative monitoring. Neurol Clin 1988;6(4):917–33.
27. Ray WZ, Mackinnon SE. Nerve problems in the lower extremity. Foot Ankle Clin 2011;16(2):243–54.
28. Moore AM, Kasukurthi R, Magill CK, et al. Limitations of conduits in peripheral nerve repairs. Hand (N Y) 2009;4(2):180–6.
29. Ray WZ, Mackinnon SE. Management of nerve gaps: autografts, allografts, nerve transfers, and end-to-side neurorrhaphy. Exp Neurol 2010;223(1):77–85.
30. Brown MC, Holland RL, Hopkins WG. Motor nerve sprouting. Annu Rev Neurosci 1981;4:17–42.
31. Flores LP, Martins RS, Siqueira MG. Clinical results of transferring a motor branch of the tibial nerve to the deep peroneal nerve for treatment of foot drop. Neurosurgery 2013;73(4):609–15 [discussion: 615–6].
32. Emamhadi M, Naseri A, Aghaei I, et al. Soleus nerve transfer to deep peroneal nerve for treatment of foot drop. J Clin Neurosci 2020;78:159–63.
33. Nath RK, Lyons AB, Paizi M. Successful management of foot drop by nerve transfers to the deep peroneal nerve. J Reconstr Microsurg 2008;24(6):419–27.
34. Head LK, Hicks K, Wolff G, et al. Clinical outcomes of nerve transfers in peroneal nerve palsy: a systematic review and meta-analysis. J Reconstr Microsurg 2019;35(1):57–65.
35. Giuffre JL, Bishop AT, Spinner RJ, et al. Partial tibial nerve transfer to the tibialis anterior motor branch to treat peroneal nerve injury after knee trauma. Clin Orthop Relat Res 2012;470(3):779–90.
36. Kale SS, Glaus SW, Yee A, et al. Reverse end-to-side nerve transfer: from animal model to clinical use. J Hand Surg Am 2011;36(10):1631–9.e2.
37. Krych AJ, Giuseffi SA, Kuzma SA, et al. Is peroneal nerve injury associated with worse function after knee dislocation? Clin Orthop Relat Res 2014;472(9):2630–6.
38. Plancher KD, Siliski J. Long-term functional results and complications in patients with knee dislocations. J Knee Surg 2008;21(4):261–8.
39. Peskun CJ, Chahal J, Steinfeld ZY, et al. Risk factors for peroneal nerve injury and recovery in knee dislocation. Clin Orthop Relat Res 2012;470(3):774–8.
40. Seidel JA, Koenig R, Antoniadis G, et al. Surgical treatment of traumatic peroneal nerve lesions. Neurosurgery 2008;62(3):664–73 [discussion: 664–73].
41. Kim DH, Murovic JA, Tiel R, et al. Management and outcomes in 353 surgically treated sciatic nerve lesions. J Neurosurg 2004;101(1):8–17.
42. Murovic JA. Lower-extremity peripheral nerve injuries: a Louisiana State University Health Sciences Center literature review with comparison of the operative outcomes of 806 Louisiana State University Health Sciences Center sciatic, common peroneal, and tibial nerve lesions. Neurosurgery 2009;65(4 Suppl):A18–23.
43. Gürbüz Y, Sügün TS, Özaksar K, et al. Peroneal nerve injury surgical treatment results. Acta Orthop Traumatol Turc 2012;46(6):438–42.
44. Cho D, Saetia K, Lee S, et al. Peroneal nerve injury associated with sports-related knee injury. Neurosurg Focus 2011;31(5):E11.
45. George SC, Boyce DE. An evidence-based structured review to assess the results of common peroneal nerve repair. Plast Reconstr Surg 2014;134(2):302e–11e.