

## Review Article

# The Interdisciplinary Management of Foot Drop

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## Summary

**Background:** Foot drop can be caused by a variety of diseases and injuries. Although it is a common condition, its overall incidence has not been reported to date. Foot drop markedly restricts the everyday activities of persons suffering from it. There is, therefore, a need for an optimized strategy for its diagnosis and treatment that would be standardized across the medical specialties encountering patients with this problem.

**Methods:** This article consists of a review on the basis of pertinent publications retrieved by a search in the Pubmed/MEDLINE and Cochrane databases, as well as a description of the authors' proposed strategy for the diagnosis and treatment of foot drop.

**Results:** Foot drop can be due to a disturbance at any central or peripheral location along the motor neural pathway that terminates in the dorsiflexor muscles of the foot, or at multiple locations in series. Optimal localization of the lesion(s) is a prerequisite for appropriate treatment and a successful outcome. The most common causes are L5 radiculopathy and peroneal nerve injury. An operation by a neurosurgeon or spinal surgeon is a reasonable option whenever there is a realistic chance that the nerve will recover. In our opinion, any patient with a subjectively disturbing foot drop and a clinically suspected compressive neuropathy of the peroneal nerve should be informed about the option of surgical decompression of the nerve at the fibular head, which can be performed with little risk. In case of a permanent foot drop, some patients can benefit from muscle-transfer surgery. For spastic foot drop, the option of botulinum toxin injections should be evaluated.

**Conclusion:** The care of patients with foot drop could be optimized by interdisciplinary foot-drop clinics involving all of the relevant specialists. The goals of treatment should always be improved mobility in everyday life and the prevention of falls, pain, and abnormal postures.

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Foot drop can be unilateral or bilateral. Starting from a muscle strength test result of less than 3/5, i.e. when the foot can no longer be actively lifted against gravity, an abnormal gait pattern arises which affects both the stance phase and swing phase (1). In order to prevent the forefoot from catching on the ground, patients with foot drop perform compensatory hyperflexion in the hip and knee joints and in addition internal rotation of the foot in the transverse plane. In the long term, this gait pattern results in improper loading of the entire skeletal axis (2). As the result of the muscular imbalance, which causes permanent strain of the plantar flexors and shortening of the Achilles tendon, equinus position of the foot may develop. The only available prospective multicenter study published by Aprile et al. in 2005 found that patients with foot drop had a reduced quality of life with significantly poorer scores in the physical and psychosocial domains. Of the assessed patients, 69% required mobility aids (3).

Until now, there is no specific code of the International Classification of Disease (ICD) for foot drop as a sign. For example, ICD G57.3 (Lesion of lateral popliteal nerve) only refers to peripheral lesions and excludes all more proximally located lesions. Other codes potentially cover foot drop, but only as an addition to other signs and symptoms (*eTable 1*). Solely based on experiences in everyday clinical practice, it can be assumed that foot drop is quite common. An overall incidence of foot drop has not been reported in the literature. Prospective randomized trial are scarce. Retrospective data only refer to a part of the possible conditions or treatment modalities.

Depending on the initial situation, primary and secondary diagnoses, patients with foot drop encounter various specialties in the hospital or community-based care setting. In our experience, patients are not treated based on common standards. Existing treatment options are not consistently offered. It is necessary to integrate patients with foot drop in a clearly defined diagnosis and treatment algorithm.

## Methods

This review informs about various causes of foot drop and presents an algorithm for the diagnosis and treatment of the condition. The review is based on a selective search in the PubMed/MEDLINE and Cochrane databases, using the search terms “foot drop”,

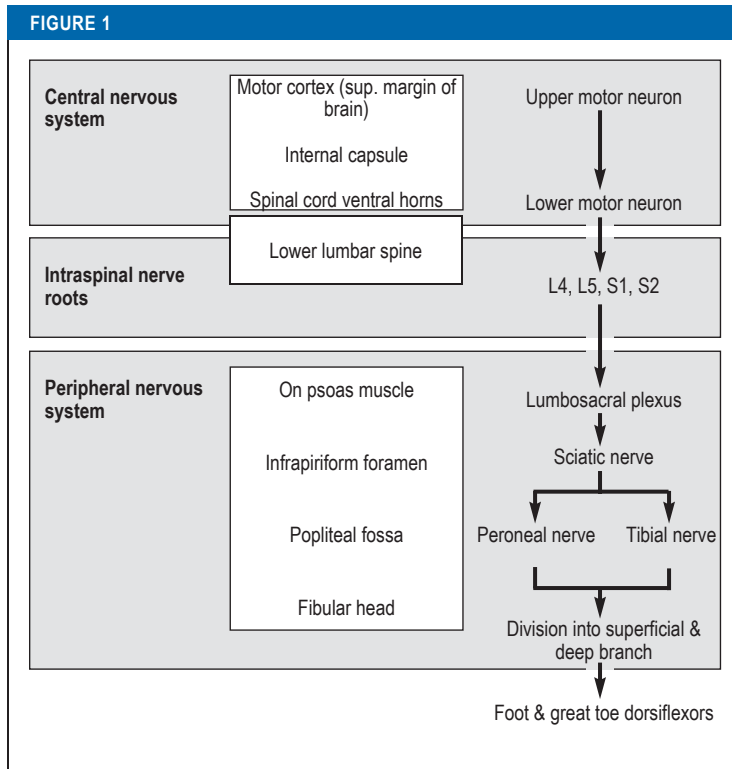
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Innervation pathway of the foot dorsiflexors

“dropfoot“, “gait disturbance“, “quality of life“, “peroneal nerve lesion“, “L5 nerve root“, “central causes of foot drop“, “tendon transfer“, and on the current guidelines on nerve injuries and lumbar disc herniation.

**Etiology**

In principle, injury to the motor pathways of the muscles involved in dorsiflexion of the foot can occur on any level, from central to peripheral, or simultaneously at multiple levels (*Figure 1, Table 1*). It is important for the treatment and prognostic assessment of foot drop to narrow down the possible location of the damage as precisely as possible. In 1973, Upton and McComas postulated that a nerve with a proximal injury is more prone to develop a lesion along its further distal course (4). Until today, this double-crush theory is still favored by several authors (5). However, more important appears to be the experience from clinical practice that many patients have multiple conditions which may be competing or additive causes of foot drop.

**Central causes**

The definition of “central” comprises the upper and lower motor neurons of the pyramidal tract in the brain and the spinal cord. Here, foot drop is frequently not an isolated finding, but part of a more extensive paresis pattern. The underlying pathologies can be characterized by displacement (for example, extra-axial tumors

[6] or tumor edema) or a destructive nature (ischemia, hemorrhage, inflammations, invasive tumors).

Central causes of foot drop were mainly described in case reports or case series some of which are already older (6–8). Johnson et al. estimated in a preliminary study that 20% of patients with anterior cerebral artery infarct (superior margin of the brain) or lacunar infarcts in the area of the internal capsule or the cerebral peduncles develop foot drops (9). Jakubowitz et al. report in a systematic review that 14% of all stroke patients have a residual foot drop as a permanent complication (10). Spinal cord lesions from the cervical spine to the conus medullaris are frequently associated with foot drop, besides other pareses. Van der Salm et al. found in a retrospective study on patients with incomplete spinal cord injury foot drop (plantar flexion included) in 76% of cases (11). Jellema et al. reported in a review that 78 to 100% of patients with spinal dural arteriovenous fistulas had a paresis of the lower limbs with gait abnormality at the time of diagnosis (12). Tokuhashi et al. report in a retrospective study that the majority of patients with disc herniation at the thoracolumbar junction had a paresis, especially the group with disc herniation at the T12–L1 level and bilateral or unilateral foot drop (13).

**Cause at the spinal nerves**

In the literature, the reported incidence rates for discogenic foot drop vary (14, 15). In a recent retrospective study from 2018, Ma et al. found that 23% of patients with disc herniation affecting the L5 nerve root had foot drop; here, the term “foot drop” was only used for muscle strength results below 4/5 (movement possible against some resistance, but not full strength). Risk factors for the development of paresis included concomitant polyneuropathy, extensive recessal or foraminal disc herniation, calcified disc herniation, and canal occupancy rates greater than 50% (15). Suzuki et al. conclude that noncontained sequestered disc herniations significant more frequently result in foot drop compared to subligamentous sequestered discs (16).

**Peripheral causes—lumbosacral plexus, sciatic nerve, peroneal nerves**

Injury to peripheral nerves may occur at any point in their course (*Figure 1*). Damage to the peroneal nerve is the most common cause of foot drop (3, 17). Additionally, peroneal nerve injury accounts for 15% of all mononeuropathies in adults (18, 19).

External and internal factors can cause peroneal nerve dysfunction. Mild pressure on the region of the fibular head, for example by placing the lower leg on it, is sufficient to induce a conduction block of the nerve. Space occupying lesions within or adjacent to the peroneal nerve are usually ganglion cysts, originating from the knee joint or the tibiofibular joint (20). An analysis of the trauma registry data of the German Society of Trauma Surgery (DGU, *Deutsche Gesellschaft für Unfallchirurgie*) performed in 2018

TABLE 1

**Causes of foot drop\***

	Mechanism	Causes
Central nervous system	Displacement/compression	<ul style="list-style-type: none"> <li>● Extra-axial brain tumors (e.g. meningioma, metastases)</li> <li>● Tumor edema</li> </ul>
	Destruction	<ul style="list-style-type: none"> <li>● Intra-axial tumors (e.g. glioma)</li> <li>● Hemorrhage</li> <li>● Ischemia</li> <li>→ Prerequisite: involving parts of the central region/pyramidal tract</li> </ul>
	Developmental disorder	<ul style="list-style-type: none"> <li>● Infantile cerebral palsy</li> </ul>
Intraspinal nerve roots L4/L5	Compression	<ul style="list-style-type: none"> <li>● Disc herniation</li> <li>● Stenosis</li> <li>● Neurinoma</li> <li>● Other tumors</li> </ul>
	Direct trauma	<ul style="list-style-type: none"> <li>● Nerve root avulsion (extremely rare)</li> <li>● Iatrogenic injury</li> </ul>
Peripheral nervous system	External compression	<ul style="list-style-type: none"> <li>● Positioning during surgery or prolonged bed rest</li> <li>● Plaster splint</li> <li>● Compression stockings</li> <li>● Pressure on lower leg (e.g. sitting with crossed legs)</li> </ul>
	Internal compression	<ul style="list-style-type: none"> <li>● Narrowness along fibular tunnel, myositis ossificans</li> <li>● Anterior compartment syndrome</li> </ul>
	Direct trauma	<ul style="list-style-type: none"> <li>● Fibula fractures</li> <li>● Adduction trauma with knee dislocation</li> <li>● Gunshot wound</li> <li>● Iatrogenic</li> </ul>
	Space-occupying lesions	<ul style="list-style-type: none"> <li>● Intra-/extra-neural ganglion cysts</li> <li>● Baker's cysts</li> <li>● Fibular bone tumors</li> <li>● Nerve tumors (neurofibroma, schwannoma, nerve sheath tumors)</li> <li>● Lipoma and others</li> </ul>
Other	Neuromuscular disease	<ul style="list-style-type: none"> <li>● Focal myopathies</li> <li>● Multifocal motor neuropathy</li> </ul>

\* adapted from Stewart 2008 (17)

showed that 1.8% of patients with lower extremity injury suffer concomitant nerve damage, and the peroneal nerve is with 56% the most commonly damaged nerve (21).

Another cause is iatrogenic injury, typically sustained during osteosynthetic or arthroscopic surgery. Kretschmer et al. showed in a large, retrospectively analyzed sample of trauma surgery patients that iatrogenic nerve injuries accounted for 17.4% of cases (22). Besides the accessory nerve, the peroneal nerve was the most commonly injured nerve.

**Other causes**

Metabolic disease as well as neurodegenerative, neuromuscular and inflammatory processes can also cause foot drop (Table 1).

**Diagnosis**

It is not always easy to clearly determine the cause of a sudden-onset foot drop. The main challenge is to differentiate between radiculopathy and peripheral nerve injury or a neurological systemic disease.

**Clinical examination**

Table 2 shows the association of individual muscles and their innervation. Knowledge of the sensory loss area as well as clinical signs and provocation tests can aid the diagnosis (eBox 1).

**Electrophysiological measurement**

Electrophysiological measurement is an established and sensitive method (23, 24) which can differentiate between a lesion of the long tracts, a radiculopathy and a peripheral nerve injury. Information about the location and severity/type of nerve lesion can be obtained (eBox 2).

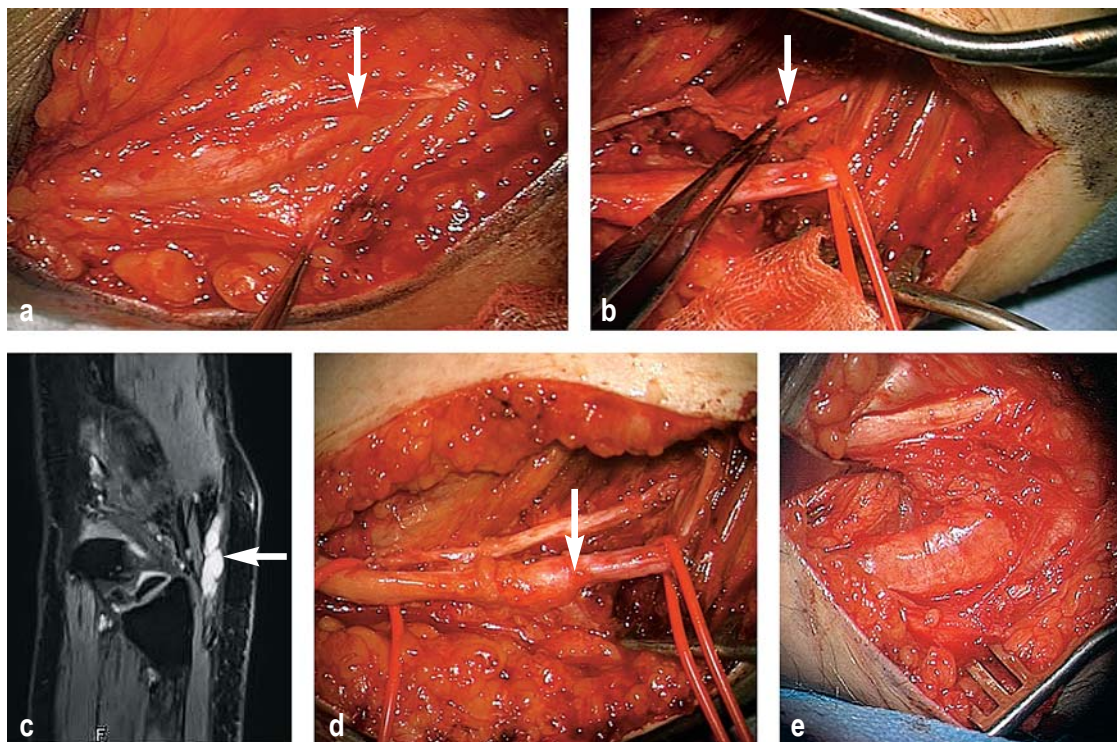
**Imaging**

Obtaining imaging studies is a standard diagnostic procedure, especially for the diagnosis of cerebral and spinal causes of foot drop (Figure 3). In the diagnosis of peripheral nerve abnormalities, they provide information about the exact location and morphology of a lesion. Besides magnetic resonance imaging (MRI) and MR neurography, nerve ultrasound is available as a

**Figure 2:**

**Peroneal nerve lesions**

- a) Compression of the common peroneal nerve on entry into the muscle compartment – split fascia (arrow)
- b) Injury to the superficial branch (arrow)
- c) Intraneural ganglion cyst – magnetic resonance imaging of the left upper/lower leg (arrow)
- d) and e) intraneural ganglion cyst – intraoperative finding



non-invasive and cost-efficient method. A retrospective study found a significant correlation between electrophysiologically detectable axonal damage and nerve changes visualized using ultrasound. However, no such correlation was found for the electrophysiological nerve conduction block (25).

**Management**

The available evidence with regard to treatment options is weak, in particular because foot drop as an autonomous condition independent of other diagnoses has so far received little attention in studies. One level IV study on the tendon replacement technique discussed below is available. By contrast, a 1b level of evidence is reached for the treatment with botulinum toxin due to the availability of prospective, randomized controlled trials on this subject (*eTable 2*).

In the treatment concept for the patient, the first step is to determine whether a surgical intervention offers a realistic chance of restoring nerve function. However, this requires that the etiology of the foot drop is completely understood. The problem is that restoration of the nerve structures is often no longer possible at the point in time when the etiology is known. Still, it is just as important to not deny patients with persistent foot drop access to supportive measures. When selecting these measures, differences in the original level of functioning, which do not necessarily correlate with the strength level of the paresis, and the patients' expectations with regard to their everyday mobility should be taken into account.

**Surgery for preservation and regeneration of nerve function**

As a general rule, causal treatment should initially be attempted in every patient with foot drop. Here, the range of treatment options extends from cerebral or spinal tumor resection to sequestrectomy and neurolysis for disc herniation to the complete spectrum of peripheral nerve surgery. To what extent one of these options is practical and useful in an individual patient depends on the patient-perceived limitations, the location of the lesion and the duration and severity of the paresis.

Currently, clinical data defining a precise time frame for surgery are scarce. Studies investigating the timing of surgery are largely experimental and focus on molecular factors of nerve degeneration and nerve regeneration (*eBox 3*). Fundamentally, repair processes are possible in the peripheral nervous system. As long as the nerve-cell nucleus is intact, axonal sprouting occurs for up to six months after the injury. However, the capacity for regeneration already starts to decline after three months because of a variety of changes in the distal stump (26). In the central nervous system, neural regeneration is inhibited by the extracellular environment. Consequently, the damage is usually irreversible. Here, surgery can improve signs and symptoms if, for example, a space-occupying lesion acts on a neural structure by displacing or compressing it, instead of destroying it by infiltrative growth.

In the current DGN guideline on lumbar radiculopathy, a relative and absolute indication for surgery is

TABLE 2

**Innervation of foot muscles\***

Muscle	Function	L5	S1	Deep peroneal nerve	Superficial peroneal nerve	Tibial nerve
Posterior upper leg muscles	Hip internal rotation Hip abduction Hip extension	x	x			
Tibialis anterior muscle	Foot dorsiflexion (inversion)	x		x		
Tibialis posterior muscle	Plantar flexion Inversion	x	x			x
Extensor hallucis longus/brevis muscles	Extension of great toe	x		x		
Extensor digitorum longus/brevis muscles	Toe extension 2–5 Eversion	x	x	x		
Peroneus muscles	Eversion	x	x		x	

\* adapted from Stewart 2008 (17)

described for muscle strengths of >3/5 and ≤ 3/5, respectively (27). Recovery is correlated with the severity of the paresis. There is no clear consensus to what extent the preoperative duration of the paresis correlates with recovery (28, 29). However, there is a trend to better recovery if the patient undergoes surgery within 48 hours of the onset of the paresis.

It has been observed that the peripheral peroneal nerve shows poorer regeneration compared to other nerves, both spontaneous and after surgery (30–32). If a common compression injury is suspected, a watch-and-wait strategy is justified; however, over the course of a week clear functional gains should be noted. Decompression of the peroneal nerve at the fibular head at least ensures that intrinsic compression is remedied. For the repair of the peroneal nerve, the principles of peripheral nerve surgery apply. Finally, it should be noted that the graft length, especially with this nerve, should not exceed 6 cm, because otherwise it will lead to significantly poorer outcomes (30, 31).

**Tendon transfers**

Tendon transfers to restore specific movements have been described since the end of the 19<sup>th</sup> century (33). In patients with irreversible loss of peroneal nerve function, the posterior tibial tendon is pulled through the interosseous membrane to the instep where it is anchored (Figure 4). In addition to the passive lifting achieved with this redirection, this procedure enables a certain degree of active control by the patients. One variation of this procedure is to attach the tibialis posterior tendon, which has been redirected to the front, with the tibialis anterior and the peroneal tendons to create a so-called stirrup. Even though the measured increase in foot lifting strength resulting from a tendon transfer is only about 30% of the full strength, a significant functional increase is achieved and the quality of life is satisfactorily improved (34, 35).

**Functional electrical stimulation**

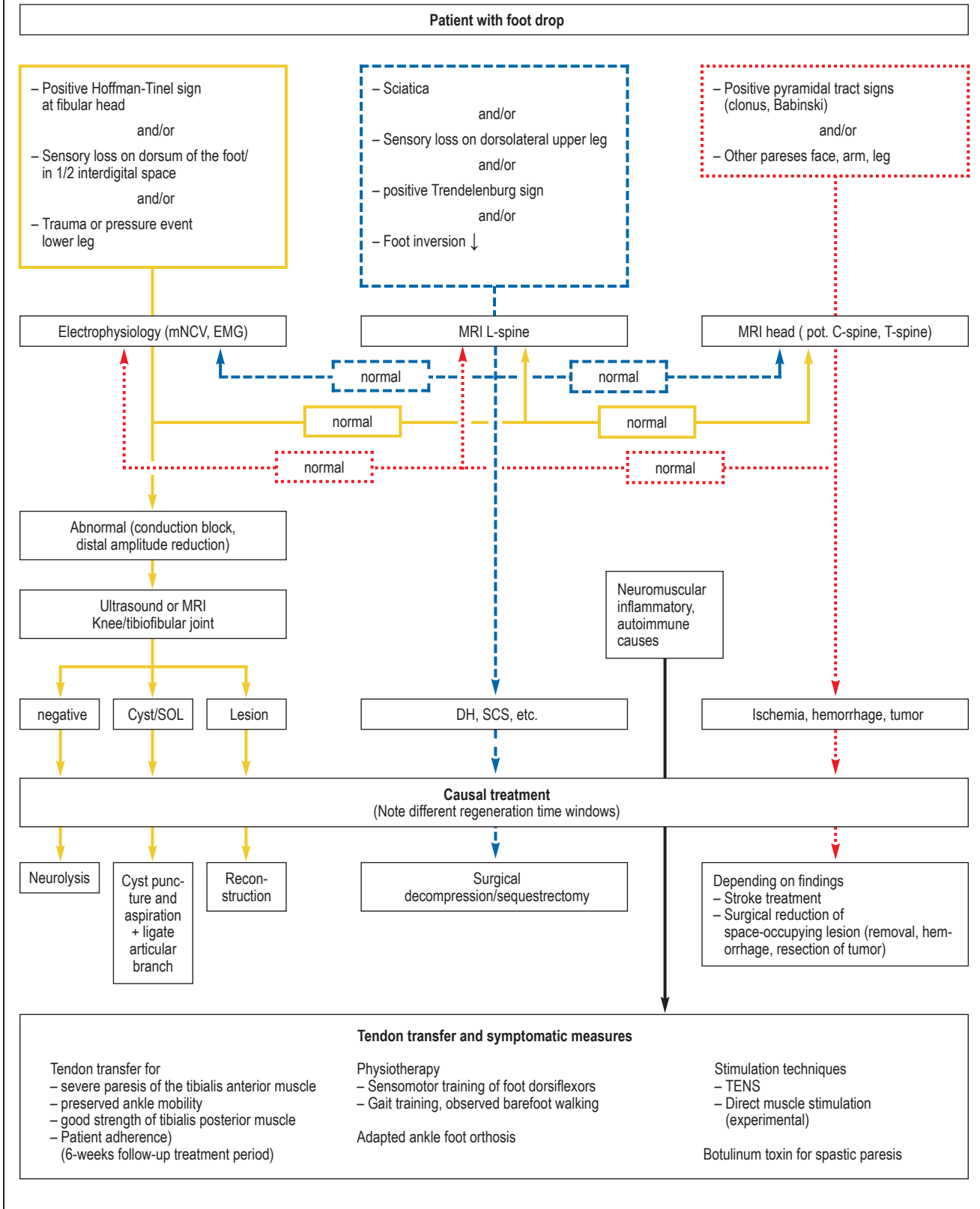
Direct muscular stimulation appears to help prevent muscle atrophy in the denervated area and therefore is generally useful in patients with foot drop as long as reinnervation is still pursued. Su et al. have shown in a recent animal experimental study that the interval between the start of stimulation and the time of denervation plays a role and best outcomes are achieved with a delayed initiation of electrical stimulation in relation to the time the lesion occurred (36). Transcutaneous electrical nerve stimulation (TENS), however, targets sensory skin nerves and thus rather helps to alleviate pain than to promote muscle fiber growth. Implantable neurostimulation devices were only temporarily available on the market and have so far failed to gain general acceptance.

**Botulinum toxin**

In patients with spastic foot drop, the option of treatment with botulinum toxin should be evaluated. Randomized, double-blind, placebo-controlled studies showed that injections of type A botulinum toxin into the calf muscles resulted in an effective, dose-dependent reduction in ankle spasticity (37, 38). These studies also found a reduced use of aids with botulinum toxin treatment. The time of onset of spasticity was not recorded. Another limitation of the two studies is the one-time injection of botulinum toxin with a follow-up period of only the expected duration of action. Both studies did not record the time of onset of spasticity.

There is conflicting evidence from studies on the effect on gait parameters. In patients with severe spasticity, treatment with botulinum toxin can be combined with physiotherapeutic redressement procedures. This approach involves the application of plaster casts with weekly increased dorsal extension to overcome concomitant shortening of the Achilles tendon by continuous stretching. In addition, there is evidence indicating that the pain associated with

FIGURE 3



**Diagnosis and treatment algorithm for foot drop**

DH, disc herniation; T-spine, thoracic spine; EMG, electromyography; C-spine, cervical spine; L-spine, lumbar spine; mNCV, motor nerve conduction velocity; MRI, magnetic resonance imaging; SOL, space-occupying lesion; SCS, spinal canal stenosis; TENS, transcutaneous electrical nerve stimulation



**Figure 4:**  
**Tibialis-posterior transfer**  
 a) Transferred tendon on target location on cuneiform bone  
 b) MRI cross-section with anchor (arrow)

spastic foot drop can be improved by botulinum toxin treatment. In 2014, botulinum toxin was approved for the treatment of the distal lower extremity in patients with stroke-related spasticity.

### Orthoses

Ankle foot orthoses, so-called peroneus splints, are designed to improve everyday mobility in patients with irreversible foot drop. There are dynamic and stiff versions of these orthoses available (39). Disadvantages include weight and appearance of the orthoses as well as pressure points due to insufficient adaption to the foot and shoe shape. In addition, the use of orthoses does not lead to a normal gait pattern. Patients should be encouraged to at times abstain from the use of the orthosis in a barrier-free environment.

### Training therapy

Independent of the surgical or conservative approach, every patient with foot drop should undergo targeted training. The therapeutic goals include (40):

- to reduce muscle fiber atrophy
- to preserve ankle mobility
- to promote reinnervation after nerve surgery
- to acquire central control after tendon transfer
- to improve gait steadiness in general.

### Conclusion and outlook

Foot drop is an underestimated sign. The available evidence is weak and data are mainly obtained from retrospective studies. This is in stark contrast to the fact that foot drop is at least by some of the affected patients viewed as an impairment with relevant impact on everyday activities. A comprehensive search for the cause of the patient's foot drop, including all possibilities of injury to the neuromuscular pathway, should be undertaken. There is a range of treatment options, some are causal and others symptomatic in nature. Typically, these treatments cannot be provided by one specialty alone.

In order to close information gaps and to simplify the logistic aspect of treatment for the patient, inter-

disciplinary outpatient clinics focusing on foot drop appear to be a practical strategy. Thus, close and regular cooperation between neurosurgeons, neurologists, specialist plastic and reconstructive surgeons or orthopedic and trauma surgeons with focus on foot surgery, as well as physiotherapists and orthopedic technicians is desirable.

The creation of a guideline of the Association of the Scientific Medical Societies in Germany (AWMF) on the management of foot drop should be discussed. Likewise, it should be considered to create a registry supported by several centers to obtain data of good quality with regard to foot drop etiology, symptom severity and successful management.

### Conflict of interest statement

The authors declare no conflict of interest.

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## Key messages

- Foot drop is not uncommon in the hospital and community-based care setting and should be regarded as an impairment with a relevant impact on the activities of daily living.
- The most common causes of foot drop are L5 radiculopathy and peripheral damage of the peroneal nerve.
- There is a lack of robust studies on the various treatment options.
- A systematic diagnostic work-up is required and should be initiated as early as possible so that any potentially treatable cause can be addressed within the nerve regeneration time window.
- Patients with severe paresis or outright paralysis of the foot dorsiflexor muscles should be informed about the possibility of tendon transfer, if no causal treatment option is available. In addition, supportive measures should be part of the care for these patients.

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### ► Supplementary material

For eReferences please refer to:  
[www.aerzteblatt-international.de/ref2019](http://www.aerzteblatt-international.de/ref2019)

eBoxes, eTables:  
[www.aerzteblatt-international.de/19m0347](http://www.aerzteblatt-international.de/19m0347)



Supplementary material to:

# The Interdisciplinary Management of Foot Drop

by Anne Elisabeth Carolus, Michael Becker, Jeanne Cuny, Rüdiger Smektala, Kirsten Schmieder, and Christopher Brenke

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**eReferences**

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**eTABLE 1**

**ICD-10 codes (potentially) including foot drop**

G57.0	Lesion of sciatic nerve
G57.3	Lesion of lateral popliteal nerve
G57.8	Other mononeuropathies of lower limb
G57.9	Mononeuropathy of lower limb, unspecified
M54.1	Radiculopathy
M51.0	Lumbar and other intervertebral disc disorders with myelopathy
M51.1	Lumbar and other intervertebral disc disorders with radiculopathy
M51.2	Other specified intervertebral disc degeneration
M51.3	Other specified intervertebral disc displacement
M51.8	Other specified intervertebral disc disorders
M51.9	Intervertebral disc disorder, unspecified
M48.0–M48.09	Spinal stenosis
G80.0–G80.9	Infantile cerebral palsy
G81.0–G81.9	Hemiparesis and hemiplegia
G82.0–G82.6	Paraparesis and paraplegia, tetraparesis and tetraplegia
G83.1	Monoparesis and monoplegia of lower limb
G83.9	Paralytic syndrome, unspecified
G99.2	Myelopathy in diseases classified elsewhere
G99.8	Other specified disorders of nervous system in diseases classified elsewhere

eTABLE 2

**Overview of foot drop treatments**

Type of treatment	Prerequisite	Evidence level	Key studies	Study design	Result
Early spinal nerve decompression (Sequestrectomy or bony decompression)	Corresponding finding in spinal MRI/CT		<ul style="list-style-type: none"> <li>● Szik guideline on lumbar radiculopathy, including:                             <ul style="list-style-type: none"> <li>– Petr. et al. 2017 (e1)</li> <li>– Postacchini et al. 2002 (28)</li> </ul> </li> </ul>	Retrospective cohort study, n = 330  Prospective cohort study, n = 116	If paresis <3/5 SG, surgery within 48 hours advantageous  Negative correlation of delayed surgery and more severe paresis with recovery
Depending on injury type:  Decompression of peroneal nerve at the fibular head or repair of injured peroneal nerve		IV	<ul style="list-style-type: none"> <li>– Kim et al. 2004 (31)</li> <li>– Maalla et al. 2013 (e2)</li> </ul>	Retrospective cohort study, n = 318  Retrospective cohort study, n = 15	If surgery <3 months and graft <6 cm recovery between 70–80%  Nerve decompression within the first 3 months after injury results in 80% in recovery → Note: 1. No RCTs or case-control studies, directly comparing these results with spontaneous recovery, are available. 2. Recovery of the peroneal nerve is typically poorer compared to other nerves.
Direct nerve stimulation			Taken off the market		
Tendon transfers	Good strength in tibialis posterior muscle	IV	<ul style="list-style-type: none"> <li>– Cho et al. 2017 (34)</li> <li>– Steinau et al. 2011 (35)</li> </ul>	Retrospective case-control study, n = 17  Prospective cohort study, n = 53	Only 33% improvement of total strength after tendon transfer, by foot function and gait significantly improved  1/3 of the original strength recovered after tendon transfer, but significant improvement in quality of life
Orthoses			– Van der Wilk et al. 2015 (39)	Literature review incl. 24 studies, altogether n = 394 patients	Comparison of different types of orthosis
Botulinum toxin	Spastic component of foot drop	Ib	<ul style="list-style-type: none"> <li>– Pitcock et al. 2003 (37)</li> <li>– Keji et al. 2010 (38)</li> </ul>	RCT, n = 234 RCT, n = 120	Dose-dependent effect on ankle spasticity
Targeted physiotherapy/sports therapy			– Willerslev-Olsen et al. 2015 (40)	Prospective cohort study, n = 60	In children with cerebral palsy, foot and great toe dorsiflexion were found significantly improved after 1 month with gait training → Note: Lack of studies on training to improve spinal/peripheral FD, but authors observed marked gait improvement

CT, computed tomography; FD, foot drop; SG, strength grade; MRI, magnetic resonance imaging; RCT, randomized controlled trial

## eBOX 1

**Work-up to narrow down potential etiologies of foot drop**

Central foot drop with cerebral lesion can occur without sensory deficits if it is caused by an anterior cerebral artery infarction, while this is rarely the case in patients with middle cerebral artery infarction. Furthermore, the latter is rather associated with a sensorimotor hemiparesis predominantly affecting the arm than with an isolated foot drop.

Because of the limited anatomical space, a spinal cord lesion can be associated with a broad spectrum of combinations of motor, sensory and autonomous deficits. It is rare that an injury to the spinal cord causes only motor deficits, but this may be the case e.g. in patients with poliomyelitis, spinal muscle weakness, amyotrophic lateral sclerosis (ALS) and hereditary spastic spinal paralysis. The level of a focal lesion can be concluded from the affected key muscles and dermatomes.

A radicular lesion of the L5 root is not only associated with foot drop, but also with paresis of the gluteus medius muscle and thus can cause a positive Trendelenburg's sign with typical waddling. The key muscle of the L5 root is the tibialis posterior muscle. Therefore, the unilateral absence of the tibialis posterior reflex can also be indicative of a radicular lesion. Here, it is essential to compare the affected side with the healthy side, because this reflex may be absent in healthy individuals too. According to the textbooks, sensory examination in patients with L5 syndrome reveals hypoesthesia of the lateral thigh, the ventral lower leg down to the instep. However, frequently examination findings are not conclusive because of overlapping dermatomes. Since the muscles involved in foot dorsiflexion have multi-segmental nerve supply, a complete foot drop rather indicates injury to the peroneal nerve and makes an isolated L5 radiculopathy unlikely.

Furthermore, the clinical examination can help to differentiate between a lesion of the common peroneal nerve and a lesion of one of its branches: Injury to the common peroneal nerve (or to the peroneal fibers of the sciatic nerve) results in a combined impairment of pronation in the ankle and dorsiflexion of the foot. The sensory deficit comprises the ventral lower leg down to the instep, including the area between the great toe and the second toe.

An isolated injury to the deep branch of the peroneal nerve results in foot drop without impairment of pronation. The sensory loss is limited to the area between the great toe and the second toe.

## eBOX 2

**Electrophysiological narrowing down of the etiologies of foot drop**

In nerve conduction studies (neurography), a circumscribed myelin lesion, e.g. as the result of chronic pressure, causes a conduction block. The response to stimulation proximal of the lesion is less intense compared to the response distal of the lesion.

In the presence of axonal damage, compound muscle action potentials (CMAP) have a low amplitude, regardless of the stimulation site. Ten to 14 days after a nerve injury, electromyography (EMG) shows abnormal spontaneous activity. If the damage is limited to myelin, no abnormal spontaneous activity is observed. In addition, the EMG detects reinnervation potentials which are of positive prognostic value even before clinical improvements are seen.

In addition, sensory neurography can differentiate between preganglionic and postganglionic nerve damage over time: In contrast to postganglionic lesions, preganglionic lesions do not result in the development of Wallerian degeneration in a proximal-to-distal manner. Thus, in patients with a postganglionic lesion, sensory neurography will show an abnormal finding in the clinical course which can be used to locate the lesion and to distinguish, for example, between a radicular lesion and a plexus lesion or peripheral nerve damage. By contrast, in acute cases sensory neurography will be unremarkable even if the patient reports sensory deficits.

Furthermore, neurography can detect any additional, potentially asymptomatic nerve lesions, for example as part of a polyneuropathy. In case of normal neurography findings, it can be useful to obtain sensory and motor evoked potentials to the peroneal nerve and the L5 dermatome.

## eBOX 3

**Repair processes in the peripheral and central nervous system**

In the peripheral nervous system, repair processes are fundamentally possible. As long as the nerve-cell nucleus is intact, axonal sprouting occurs for up to six months after the injury. However, due to the various changes in the distal stump, regeneration markedly declines already after 3 months (26) and consequently the outcomes of surgery are less favorable.

In the central nervous system, neural regeneration is inhibited by the extra-cellular environment to prevent uncontrolled growth processes. Thus, any damage is generally definite. Damaged tissue is quickly replaced by a glial scar.