



# FOOT DROP

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I HAVE NO DISCLOSURES

## WHAT IS FOOT DROP?

- Weakness or inability to dorsiflex the ankle
- Common condition with significant functional impact
- Affects mobility, fall risk, and quality of life
- Many potential causes

## MOTOR PATHWAY TO DORSIFLEXORS

Cerebral cortex → corticospinal tract → anterior horn cells

L4-L5 nerve roots → lumbosacral plexus

Sciatic nerve → common peroneal nerve

Deep peroneal nerve → tibialis anterior, extensor hallucis longus, extensor digitorum longus

## COMMON ETIOLOGIES

L5 radiculopathy

Common  
peroneal  
neuropathy

OTHER  
IMPORTANT  
ETIOLOGIES

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Central nervous system lesions (stroke, MS, spinal cord injury)

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Lumbosacral plexopathy

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Sciatic neuropathy

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Polyneuropathy (diabetes, toxic, hereditary)

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Anterior horn cell disease

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Myopathy

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## MECHANISMS OF PERONEAL NEUROPATHY

**Compression/Entrapment:** Prolonged positioning,  
habitual leg crossing, casts/braces

**Traction/Stretch:** Knee dislocation, ankle inversion injuries

**Transection/Laceration:** Trauma, surgical injury

**Intraneural ganglion cyst:** Rare but important to identify

## ANATOMICAL CONSIDERATIONS

Vulnerable at fibular  
head

Superficial location  
makes it susceptible to  
compression



# CLINICAL EVALUATION - HISTORY

**Onset:** Acute vs. gradual

**Mechanism:** Trauma, surgery, positional, spontaneous

**Associated symptoms:** Pain (radicular vs. local), sensory changes, weakness in other muscle groups

**Medical history:** Diabetes, prior back problems, recent procedures

**Functional impact:** Gait changes, falls, use of assistive devices

## RED FLAGS

Bilateral symptoms

Bowel/bladder  
dysfunction

Progressive weakness

Upper motor neuron  
signs



## PHYSICAL EXAMINATION

- Ankle dorsiflexion strength (tibialis anterior)
- Great toe extension (extensor hallucis longus)
- Ankle eversion (peroneus longus/brevis)
- **Hip abduction strength** - Critical differentiating test
  - Weak hip abduction → L5 radiculopathy
  - Normal hip abduction → Peroneal neuropathy

## PHYSICAL EXAM – ADDITIONAL FINDINGS

- **Sensory Examination:**
  - L5 dermatome (lateral leg, dorsum of foot)
  - Superficial peroneal distribution (dorsum of foot)
  - Deep peroneal distribution (first web space)
- **Reflexes:**
  - Ankle reflex (S1) - should be normal in isolated foot drop
  - Knee reflex (L4) - assess for multilevel involvement

## GAIT ASSESSMENT

- Steppage gait pattern
- Circumduction
- Foot slap during heel strike
- Trendelenburg (if hip abductor weakness)





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# ORTHOTIC INTERVENTION

## Ankle-Foot Orthoses (AFO):

First-line treatment for most patients

Prevents foot drop during swing phase

Reduces fall risk and improves gait efficiency



# PHYSICAL THERAPY

## **Therapeutic Exercise Program:**

Strengthening of dorsiflexors and compensatory muscles

Range of motion to prevent contractures

Gait training with assistive devices

Balance and proprioception training



## SURGICAL INTERVENTION

Peroneal nerve  
decompression at the  
fibular head



## SURGICAL INTERVENTION

### Indications for Tendon Transfer

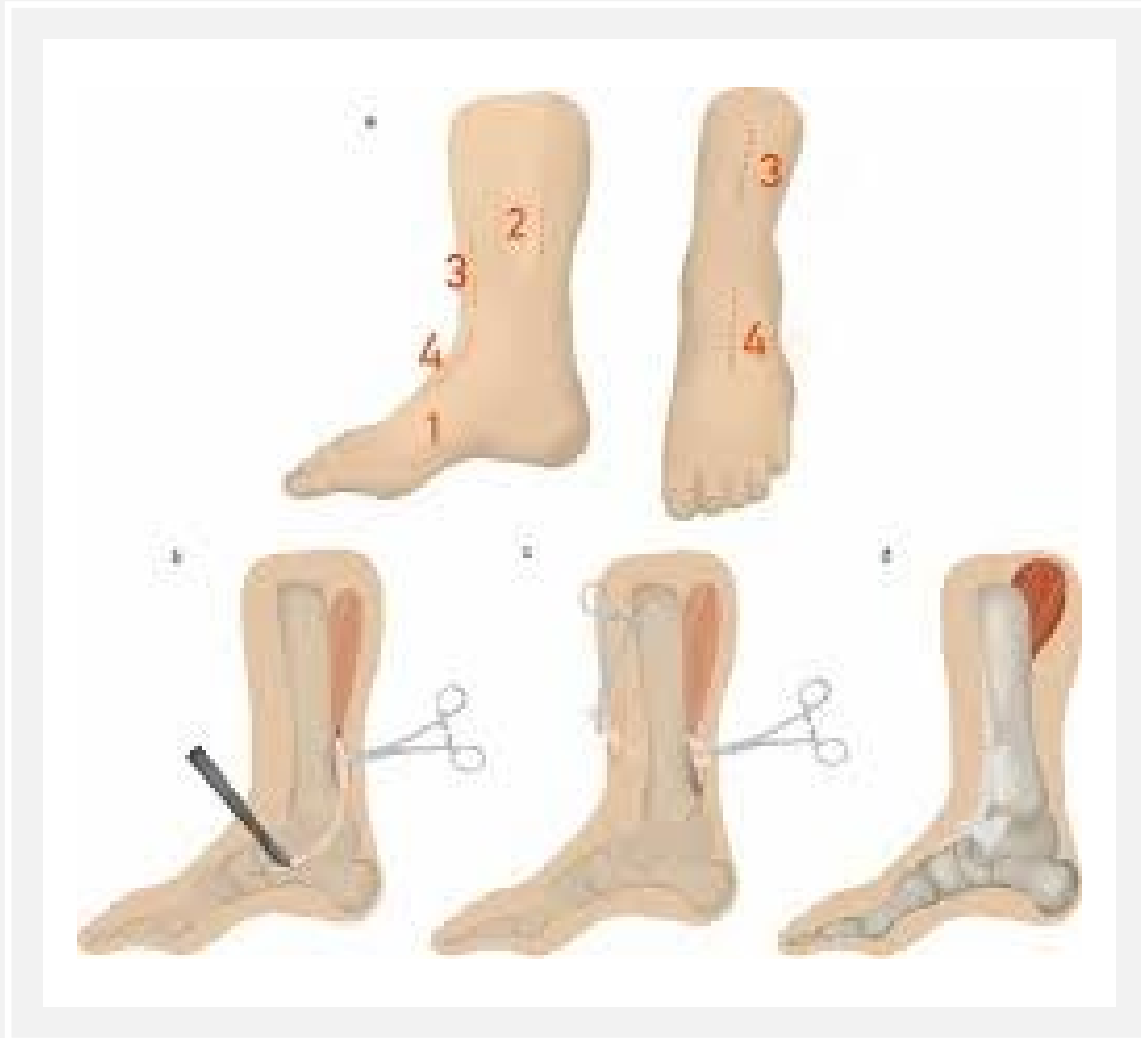
Chronic foot drop (>2 years without recovery)

Failed nerve decompression

Functional disability despite conservative management

### Common Procedures:

Posterior tibial tendon transfer (most common)



## PREREQUISITES



Adequate donor muscle strength



Correctible deformity



Adequate soft tissue coverage

# SPASTIC FOOT DROP

Address contractures,  
spasticity, then weakness



## TREATMENT OPTIONS

Botulinum toxin injections for spasticity

Serial casting for contractures

AFO for support

Tendon lengthening procedures



## DIABETIC NEUROPATHY

Length-dependent process

Often bilateral and symmetrical

Associated with sensory loss

## MANAGEMENT CONSIDERATIONS

Glycemic control optimization

Careful skin monitoring with AFO use

Fall prevention strategies

Multidisciplinary approach (endocrinology, podiatry)

CASES

## THE LEG CROSSER

38-year-old male software engineer, presents with 3-week history of progressive right foot slapping while walking

Works 10–12 hours/day at desk, habitually crosses right leg over left

Recently lost 15lbs on a new diet

Noticed tingling on top of right foot 4 weeks ago, then weakness

No back pain, no trauma, no prior surgeries

## EXAMINATION

Ankle dorsiflexion: 3/5

Great toe extension: 3/5

Ankle eversion: 4/5

Ankle plantarflexion: 5/5

Ankle inversion: 5/5

Hip abduction: 5/5

Tinel's sign positive at fibular head

Sensory loss over dorsum of foot

# ELECTRODIAGNOSTICS

Peroneal motor NCS: Conduction block across fibular head  
(50% amplitude drop)

Superficial peroneal SNAP: Reduced amplitude

Tibial motor NCS: Normal

EMG: Fibrillations in tibialis anterior; normal medial head of gastrocnemius, short head of biceps femoris and lumbar paraspinals

## DISCUSSION

Classic compressive peroneal neuropathy at fibular head

Weight loss is a well-known risk factor

Conduction block indicates neurapraxia — favorable prognosis

Management: Behavior modification (stop crossing legs), AFO, PT

Expected recovery: 3–6 months with conservative management

If no improvement by 3 months → consider MR neurography if available/MRI knee and surgical decompression

## THE DIAGNOSTIC DILEMMA

62-year-old male with diabetes presents with 6-week history of left foot drop and low back pain radiating to the lateral leg

Type 2 diabetes for 15 years, A1c 8.2%

Chronic low back pain, worsened 6 weeks ago after lifting heavy box

Left leg pain radiating from buttock to lateral calf

Numbness in left foot

No bowel/bladder dysfunction

# EXAMINATION

Ankle dorsiflexion: 2/5

Great toe extension: 1/5

Ankle eversion: 4/5

Ankle inversion: 4/5

**Hip abduction: 4/5**

Positive straight leg raise

Decreased sensation L5 dermatome AND dorsum of foot

Ankle reflex intact, knee reflex intact

Mild stocking-glove sensory loss bilaterally (baseline neuropathy)

# DIAGNOSTICS

MRI L spine: Large L4-L5 paracentral disc herniation with severe L5 nerve root compression

## **Electrodiagnostics:**

Peroneal motor NCS: Reduced amplitude bilaterally (worse on left), no conduction block at fibular head

Superficial peroneal SNAP: Absent bilaterally

Tibial motor NCS: Mildly reduced amplitude bilaterally

EMG: Fibrillations in left tibialis anterior, tibialis posterior, and gluteus medius; active denervation in left L5 paraspinals

Right side: Chronic neurogenic changes in distal muscles



## LEARNING POINTS

**Dual pathology:** L5 radiculopathy superimposed on diabetic polyneuropathy

Hip abductor weakness and paraspinal denervation confirm radicular component

Bilateral SNAP abnormalities suggest underlying polyneuropathy

Diabetes is an independent risk factor for foot drop with disc herniation

This patient has multiple poor prognostic factors: diabetes, severe weakness, and nerve compression on MRI

Surgical consultation warranted — early decompression associated with better motor recovery

## LEARNING POINTS

In diabetic patients, consider dual pathology

Double crush phenomenon — a proximal lesion (radiculopathy) makes the nerve more vulnerable to distal compression, and vice versa

Electrodiagnostics are essential to sort out the contributions of each lesion

## THE STROKE PATIENT

68-year-old male, 3 weeks post-right MCA stroke, referred to inpatient rehabilitation with left foot drop

Acute right MCA ischemic stroke with left hemiparesis

Received IV tPA within window

Left upper extremity: 2/5 throughout

Left lower extremity: Proximal 3/5, ankle dorsiflexion 1/5

Significant left ankle plantarflexor spasticity (Modified Ashworth 2)

Unable to ambulate independently

## EXAMINATION

Left ankle dorsiflexion: 1/5 with increased tone

Passive ankle dorsiflexion limited to neutral due to gastrocnemius/soleus tightness

Hyperreflexia left lower extremity

Left ankle clonus

## DISCUSSION

Upper motor neuron foot drop  
Spasticity is the primary barrier,  
not just weakness

Management hierarchy:

Address contracture →

Manage spasticity → Address  
weakness

## Treatment plan:

Rigid AFO initially for safe gait training

Aggressive stretching and serial casting if contracture develops

Botulinum toxin to gastrocnemius/soleus if spasticity limits function

Transition to articulated AFO as spasticity improves



TREATMENT  
PLAN

## ESSENTIAL POINTS

**Accurate localization is critical** - check hip abduction strength and electrodiagnostics

**Most common causes:** L5 radiculopathy and peroneal neuropathy

**AFO is first-line** for most patients, get patients into PT

**Tendon transfer for chronic cases** (>2 years)

**Interdisciplinary approach** optimizes patient care

**Goals:** Improved mobility, fall prevention, quality of life



THANK YOU