


9:45 – 10:45 am

**Walking the Line:  
A Primer on Gait Analysis**

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**Presenter Disclosure Information**

The following relationships exist related to this presentation:

- ▶ Salvatore Mangione, MD: No financial relationships to disclose.

**Off-Label/Investigational Discussion**

- ▶ In accordance with pmICME policy, faculty have been asked to disclose discussion of unlabeled or unapproved use(s) of drugs or devices during the course of their presentations.

**GAIT – WHY BOTHER?**

- Gait disturbances are common.
- They are especially common in the *elderly*, affecting 15% of subjects above 60, 25% of those older than 80, and 50% of people above 85.
- In fact, half of all nursing home residents have problems walking.
- In Goteborg, Sweden, one in four 79-year olds had to use a mechanical aid, and none could comfortably walk at 1.4 m/sec, which is the norm for street crossing.
- Hence, gait disturbances are a common risk factor for *falls*, injuries & loss of independence.
- In a study of more than 1,000 community living residents older than 72 years of age, 50% had suffered at least one fall during a 2.5 year period.
- Of these, 25% experienced a serious injury, and 5% had a fracture.
- In fact, gait disturbances are an important contributor to *hip* fractures — the sixth leading cause of death in the elderly.
- There are good historical precedents for this...

**1. Flexed Posture**

- Head bent down
- Spine flexed
- Elbows, Hips and Knees are also flexed

In Ambulation:

- Trunk further bent forward
- Upper Extremities do not swing
- Lower Extremities remain flexed

**2. Shuffling Gait**

- Reduced Step *Height*
- Reduced Step *Length*
- Shortened *Stride*
- Feet barely clearing the ground

**GAIT NEUROPHYSIOLOGY – A PRIMER**

- *Stance* is the position assumed by a *standing person*, or *station* (from the French derivative of the Italian "stanza"). It is also one of the phases of ambulation.
- *Gait* is instead the individual's *ambulating style* (from the Old Norse "gata", path), which is often so unique to be recognizable from a distance.

- In fact, gaits can say a lot not only about neuromuscular (patho)physiology, but also about *mood* (like depression), occupation, and even *character*.

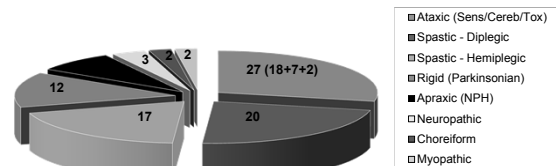
- *Stance* begins when one heel strikes the ground and it lasts for the entire period during which that foot stays grounded.
- Hence, it is a *weight-bearing* phase.

- *Stance* is mostly due to contraction of the extensors: the gluteus maximus early on, quadriceps in the middle, and plantar flexors (soleus and gastrocnemius) towards the end.
- *Swing* is instead mostly due to contraction of the flexors: iliopsoas (for hip), hamstrings (for knee), and tibialis anterior (for ankle).
- **Through it all the Long Extensors dorsiflex foot for toe clearance.**

- Stance and swing make up a stride, which corresponds to the interval between the time one heel hits the floor until it strikes it again.
- For 20–25% of the cycle the stance of the two legs overlaps, insofar as both feet are on the ground (double-limb support). This time increases with age.

### GAIT – IMPORTANT DIAGNOSTIC CLUES

- Symmetry versus asymmetry
- Height, length and cadence of steps (length of *stride*)
- Presence of muscle weakness (*hypotonia*)
- Presence of stiffness in the limbs (*hypertonia*)
- Presence of bladder or bowel dysfunction (*spastic paraplegia*)
- Difficulty in initiating or terminating walking (*Parkinson's*)
- Association with vertigo or light-headedness (*cerebellar, vestibular*)
- Association with pain, numbness, or tingling in the limbs (*neuropathy*)
- Worsening of disturbance at night (*posterior column disease*)
- *Acute onset* — suggesting *vascular disease* versus *drugs* (alcohol, benzodiazepines, neuroleptics, and orth. hypotension-inducing agents)



Neuro-muscular Causes of Gait Impairment

### ABNORMAL GAITS "THE MAGNIFICENT 7"

- Besides *antalgic* ambulation (limp), there are *seven gaits* that are useful because of their *localizing value*, insofar as they can pinpoint both the *level* and the *system* involved (sensory, cerebellar, basal ganglia, extra-pyramidal, pyramidal, peripheral neural, and muscular):
  1. **Ataxic** – Sensory/Cerebellar/Toxic
  2. **Dyskinetic/Choreiform** – Basal Ganglia
  3. **Hypokinetic/Rigid** (Parkinsonian/Apraxic – Extra-Pyramidal/NPH)
  4. **Spastic/Hemiplegic** – Pyramidal
  5. **Spastic/Diplegic** (Myelopathic) – Pyramidal
  6. **Neuropathic** – Motor (Weak gait)
  7. **Myopathic** – Muscular (Weak gait)

\* Remember the difference between the two forms of hypertonicity, "Rigid" And "Spastic".

### GAIT – AUGENBLICK ASSESMENT

From front, back and side, assess:

- Posture (normal, stooped, kyphotic /simian-like and swaying)
- How the patient *gets up* from a chair (Parkinson's or myodystrophy)
- How the patient *initiates walking* (also useful in Parkinson's)
- How the patient walks at a *slow pace*
- How the patient walks at a *fast pace*
- How the patient *turns*
- How the patient *walks on toes* (Parkinson's can't do it, but also sensory ataxia, spastic hemiplegia, or paresis of the soleus/gastrocnemius)
- How the patient *walks on heels* (diagnostic in foot drop, spastic paraplegia or motor ataxia)

## GAIT – AUGENBLICK ASSESSMENT

- How the patient stands (normal vs wide-based)
- How the patient stands with eyes first open and then closed (Romberg's)
- How the patient walks with eyes first open and then closed (sensory ataxia worsens with closed, whereas cerebellar ataxia does poorly either way)
- How the patient cope with sudden postural challenges, such as modest nudging or pull from behind after adequate warning; inadequate postural reflexes (as often seen in nursing home residents) will cause a few steps of retropulsion, and even a tendency to fall backward.
- How the patient walks a straight line in tandem (i.e., heel to toe) – this is useful in all gait disorders because it narrows the base.

- Always inspect the upper extremities and observe how they behave during walking!

## ANTALGIC GAIT

### Antalgic (Coxalgic) Gait

- The limp caused by a painful hip is characterized by shifting of the upper torso toward the painful side during the single-limb stance phase on the affected hip.
- This results from an unconscious adaptive response to decrease the joint reaction force on the painful hip.

- From the Greek "against the pain", this is a "limp" caused by discomfort on weight bearing.
- It is an antalgic strategy used by patients with either:
  - > *Hyperesthesia* of neurologic disease
  - > *Pain* in one of the weight-bearing joints (hips, knees, ankles, or just the bottom of the feet)
- The latter is very common, since by age 75, 85% of the population will have osteoarthritic changes of the large joints.
- These typically present in a unique (and thus diagnostic) fashion:

- 1) *Coxarthrosis* causes a *coxalgic gait*, characterized by limited range of hip extension and a "lateral (or adductor) lurch." This is an excessive lateral shift of the patient's upper body *toward the affected side* when standing on the painful limb, which effectively relocates the center of gravity, thus reducing the weight load.
- 2) *Gonarthrosis* is instead associated with an antalgic gait wherein the knee is stiff and there is inability to flex or extend the leg.
- 3) Finally, if the pain originates in the *foot*, there will be an incomplete (and very gentle) contact with the ground.
  - Gaits of knee or foot pain are mostly *antalgic*, insofar as they are characterized by a shortened stance on the affected limb, but not as much by the *lateral lurch* that characterizes *coxalgic* gaits.

## PATHOLOGICAL GAITS "THE MAGNIFICENT 7"

### Antalgic – Joint/Skeleton

## ATAXIC GAIT

- This is the unsteady and uncoordinated walk of *ataxia*, with a wide base and feet that are thrown outward.
- An ataxic gait can be *sensory* or *cerebellar*.
- In *sensory ataxia*, the cerebellum is intact while the afferent input is compromised.
- This is usually *sensory* (dorsal columns), but could also be visual, vestibular, and even auditory.

## ATAXIC GAIT – CEREBELLAR ATAXIA

- In contrast to sensory ataxia, opening (or closing) the eyes neither improves nor worsens stance (negative Romberg's)
- Since the cerebellum is responsible for proper balance and posture, a cerebellar gait results from either *primary cerebellar disease* or *alcohol intoxication*.
- Hence, it differs from sensory ataxia since it is associated with other signs of cerebellar deficit, such as *dysmetria*, *dysarthria*, *nystagmus*, *hypotonia*, and *intention tremor*.

## PATHOLOGICAL GAITS “THE MAGNIFICENT 7”

Antalgic – Joint/Skeleton

1. Ataxic – Sensory/Cerebellar/Toxic

## PATHOLOGICAL GAITS “THE MAGNIFICENT 7”

Antalgic – Joint/Skeleton

1. Ataxic – Sensory/Cerebellar/Toxic
2. Diskinetic/Choreiform – Basal Ganglia

## PARKINSONIAN GAIT

- Common (1.5% of the population older than 65.)
- Rigidity, bradykinesia and tremor are the hallmark.
- The *frozen posture* and the *shuffling gait* are its hallmarks.
- In fact, they are so typical that in the absence of tremor they provide the most reliable sign of the disease.
- The main features are *axial rigidity* and *shuffling*.

## PARKINSONIAN GAIT

- This results in a slow walk characterized by a series of small (reduced *stride*), and narrow-based steps that barely clear the ground (reduced *height*).
- As a result gait is *very slow*.
- Patients find it especially difficult to *initiate the gait*, not only when trying to rise from a chair, but also when starting to walk after long standing.
- Very characteristic are also the *freezing episodes*, which typically occur when crossing a threshold, facing a door, turning a corner, or simply transitioning from hardwood to carpet floor.
- Turns are also rather slow (*en bloc*) due to bradykinesia and postural instability.

## FESTINATION

- *Festination* is usually a late phenomenon, resulting from all the aspects of the disease:
  - > Flexion of hips and knees
  - > Forward stoop
  - > Shuffling steps
- Yet, it's primarily caused by the *forward leaning* (and advancing center of gravity), which force the patients to keep on moving in order to regain their center of gravity.
- Still, festination is often inadequate, thus resulting in frequent falls.

### PARKINSON'S "STANCE"

- This is also stooped, rigid, and primarily flexed at multiple levels:
  - *Head* (bent downward)
  - *Thoracic spine* (bent forward)
  - *Arms* (moderately flexed at the elbows)
  - *Legs* (mildly flexed at hips and knees)
- 75% of patients will stand by *leaning forward from the waist*, often resulting in instability.
- This bent and forward-leaning posture may resemble the "simian stance" of spinal stenosis, which is *antalgic* since it reduces the pull on the compressed lumbosacral nerves)
- Still, in contrast to spinal stenosis the stance of Parkinson's is *completely painless*.

### (1) NPH – MAGNETIC GAIT

- The first and cardinal sign of NPH.
- May be progressive, as hydrocephalus expands into the lateral ventricles.
- It's caused by traction on periventricular lumbosacral motor fibers.
- Often presenting as *unsteadiness* and *imbalance*, especially on stairs and curbs.
- Typically bradykinetic (*very* slow), wide-based, short-stepped, shuffling (little knee flexion), and "magnetic".
- Patients don't lean forward but backward, and they may swing arms.

### PATHOLOGICAL GAITS "THE MAGNIFICENT 7"

#### Antalgic – Joint/Skeleton

1. Ataxic – Sensory/Cerebellar/Toxic
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3. Hypokinetic/Rigid (Parkinsonian/Apraxic – Extra-Pyramidal/NPH)

### SPASTIC/HEMIPLEGIC GAIT

- *Upper extremity adduction and flexion* at all levels (elbow, wrist, and fingers).
- *Lower extremity extension* at all levels (hip, knee, and ankle.)
- The foot is *internally rotated*.
- There is no arm swing.

### SPASTIC/HEMIPLEGIC GAIT

- Spastic hemiplegic patients have great difficulty in flexing the involved hip and knee, and also in dorsiflexing the ankle (which thus remains flexed downward and inward — *equinovarus deformity*).
- As a result, they do not drag the foot limply behind them, but they *swing it onto the affected side in a half circle (circumduction)*
- Thus, the foot scrapes the ground on its lateral edge, in a typical equinovarus wear-and-tear of the shoes.
- Given the inability to flex the knee, the upper body lurches to the opposite side (compensating for circumduction), and the walk is overall difficult and slow.

### PATHOLOGICAL GAITS "THE MAGNIFICENT 7"

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4. Spastic/Hemiplegic – Pyramidal

### SPASTIC/DIPLEGIC GAIT

- This is the stiff-legged gait of patients with *myelopathy*
- It is characterized by reduced toe clearance in the swing phase, requiring *symmetric circumduction* of the lower extremities.
- Later it becomes wide-based, unsteady, shuffling, jerky, and spastic.

### STOOPED GAIT OF LUMBAR SPINAL STENOSIS

- Dull or aching back pain spreading to legs
- Numbness/paresthesias in legs, calves or buttocks
- Weakness or loss of balance
- ↓ Exercise tolerance (*neurogenic claudication*)
- Symptoms worsened by walking/standing
- Symptoms relieved by:
  - Bending over or leaning forward
  - Lying down
  - Sitting
  - Put feet on a raised rest

### SPASTIC DIPLEGIC GAIT

- In contrast to *spastic hemiplegia* the arms of spastic diplegia are essentially normal.
- And in contrast to *Parkinson's*, toes of spastic paraplegia always stay on the ground.
- Parkinson also lacks the back pain.

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### “FOOT DROP” (HIGH STEPPAGE GAIT)

- 1) High Steppage:
  - Knees are raised unusually high to allow the drooping foot to clear the ground.
  - Yet, since the toes of the lifted foot remain pointed *downward*, they may still scrape the floor, thus resulting in frequent stumbles and falls.
  - A foot drop can often be diagnosed by simply looking at the patient's shoes, since wear-and tears will be typically asymmetric, and affecting especially the toes.
- 2) “Foot slap”: After the heel touches the ground the forefoot is brought down suddenly and in a slapping manner. This “stamping gait” creates a typical double loud sound of contact (first heel, then forefoot)\*

\* Only two gaits “slap the ground”: sensory ataxia and foot drop.

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6. Neuropathic – Motor (Weak gait)

### ANSERINE (WADDLING) GAIT

- Bilateral pelvic girdle weakness, resulting in a *waddling* (or anserine) gait.
- Note the *lumbar hyperlordosis*, with shoulders thrust backwards and abdomen being protuberant.
- This places the center of gravity behind the hips, so that the patient does not fall forward as a result of weak back and hip extensors.

### ANSERINE (WADDLING) GAIT – ROLE OF HIP ABDUCTORS

- The gluteus medius originates on the *ilium* (between ant. and post. gluteal lines), eventually terminating on the lateral surface of the greater trochanter.
- Its contraction pulls the two insertion sites toward one another, thus elevating the opposite side of the pelvis.
- Its weakness causes contralateral sagging of the pelvis (Trendelenburg Sign).

### TRENDELENBURG SIGN

- It is a phenomenon that typically occurs when the subject stands on one leg.
- Contraction of the *gluteus medius* of the weight-bearing limb prevents the pelvis from tipping toward the opposite (and unsupported) side, thus keeping it level.
- When the gluteus medius is weak, the contralateral hemipelvis tips instead *down*, the buttock sags, and the unsupported leg hangs lower.
- That's a positive Trendelenburg sign.

### TRENDELENBURG - CAUSES

- The two most common causes are:
  - 1) *Weak hip abductors*:
    - Muscular atrophy / dystrophy
    - Spinal nerve root lesions (paralyzing the superior gluteal nerve — hence, L5)
    - Polyomyelitis
  - 2) *Hip disease* (especially congenital dislocation, but also fracture of the greater trochanter, which is the distal insertion site for the gluteus medius)

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