OBJECTIVES:

After listening to this presentation and studying the accompanying syllabus material, attendees will be able to:

1. Describe the breakdown of the gait cycle into swing vs. stance, and know the periods of single and double support.

2. Name the RLA (Rancho Los Amigos) phases of gait and their proportions of the cycle.

3. Define stride duration, swing duration, stance duration, stride length, step length, base of support width, cadence, and velocity, including the approximate normative values of each.

4. Describe the gross kinematics at the hip, knee, and ankle during normal gait.

5. Describe the major muscle activity during each RLA phase of gait.

6. Describe common orthopaedic gait deviations, including typical compensation that result.
Overview of Biomechanics

Of all human movements, walking has by far received the most study. What we learn from biomechanical analyses of walking provides a framework for studying all kinds of movements, such as reaching and grasping, sucking, mastication and swallowing, and movements of the eyes. Even for those clinicians who will not directly treat gait deviations, an understanding of gait biomechanics and a familiarity with normal gait will provide a quick window into the patient’s level of function because gait is such a common and readily observable activity that involves so much of the body.

Temporal and Spatial Measures

Temporal and spatial measures examine global aspects of gait. Because gait is a cyclical activity, the basic assumption is that one step is essentially the same as the next. Thus, a parameter such as stride length is expected to be characteristic of the person’s overall walking performance, not just the step(s) measured. The following list includes typical values for the common measures:

Stride: The period from contact on one extremity until the next contact on that extremity. One stride is one “cycle” of gait. A step is the period from contact on one extremity until contact on the opposite extremity

Stride Duration (cycle duration, cycle period): Males 1.1 sec/stride; Females 1.03 sec/stride

Stance Time (stance duration): 60% of one cycle of gait

Swing Time (swing duration): 40% of one cycle of gait

Single Support Time: 80% of one cycle

Double Support Time: 20% of one cycle

Stride Length: Male 1.5 meter; Female 1.3 meter

Step Length: half of stride

Base of Support Width: 2-12 cm between heels

Degree of Toe Out: ~ 7 degrees

Cadence (step rate): Male 110 step/min; Female 116 step/min

Velocity: Typical = 80 m/min; Male 80-91; Female 73-81
Phases of Gait

Gait, particularly walking, is a cyclic phenomenon that can be divided into segments, or phases. Two sets of terminology are currently in use: the traditional terminology and the Rancho Los Amigos (RLA) system. The traditional terminology developed as interest in gait rehabilitation mounted after WWII in the effort to improve lower extremity prosthetics. It describes gait in terms of discreet, momentary events, such as heelstrike, heel rise, and toe-off. The RLA terminology became increasingly popular in the late 1980’s and early 1990’s and is currently assuming a position as the preferred standard among clinicians. It describes gait more in terms of processes or segments of time, such as loading response, terminal stance, and pre-swing, and because it is semantically more generic and better encompasses the common features of normal and pathological gait. The traditional terminology uses the term “heel strike” where the RLA system uses the term “initial contact” to refer to the instant when the limb contacts the ground. “Initial contact” applies equally well to the gait of a child with cerebral palsy who actually makes contact with the toes as it does to the gait of a person with an amputated lower extremity or a person without disability who makes contact with the “heel”. In communicating with your colleagues and to understand the published literature, however, you will need to be fluent in both nomenclatures.

While the phases of gait defined in the RLA terminology are fine for walking, which is usually the focus of medical rehabilitation, additional nomenclature applies when studying running, which may be important in a sports-related, orthopedic practice. For both walking and running, stance gets much shorter and swing gets slightly shorter as speed increases. Thus, as speed increases, the proportion of the cycle devoted to stance decreases and the proportion of the cycle devoted to swing increases. For very fast sprinting, the absolute duration of swing may actually begin to increase as extremely high speeds are reached.

The following several pages provide graphic and written descriptions of the most important events and phenomena for each RLA phase of normal gait.
Initial Contact

You will want to know the material on this and the following pages so well that you can recite it in your sleep, backwards.

Initial Contact = Not really a phase of gait, but rather the instant when the foot hits the ground. Ideally, first contact is with the heel so that the limb is positioned to begin stance with a heel rocker. This is the beginning of DOUBLE LIMB SUPPORT. The position of the GRF vector at Initial Contact is sometimes misleading, so I have not displayed it above.

Muscle activity is in transition from Terminal Stance to Loading Response = preparation for weight acceptance.

Hip at ~30° flexion. Hip extensor activity.

Knee: ~5° flexion. Quad and hamstring activity, for stability.

Ankle: neutral. Dorsiflexor activity has been keeping the foot from plantarflexing and is now going to resist the plantarflexion moment of loading response (when initial contact is at the heel).
Loading Response - Beginning  Loading Response - End

HIP MOTION  KNEE MOTION  ANKLE MOTION

GROSS MUSCLE ACTIVITY

Loading Response begins w/ IC and continues until the other foot is lifted for swing. Shock is absorbed as weight is transferred to the outstretched limb.

- 0-10% of gait cycle
- Basically the opposite of Pre Swing
- The initial double stance period
- A critical phase (weight acceptance, lots of torque, potential for instability)
- Heel Rocker action
- External PF torque initially
- Critical events: Hip stability, controlled knee flexion and PF, pronation

Hip: From ~30° flexion may extend slightly. GRFV in front of hip = flexion torque, thus extensor activity

Knee: Begins at ~5° flexion (ideal for absorbing "shock"); ends ~20 flex (nearly its peak flexion). Motion: ~15° flex
GRFV moves posterior to knee (=external flexion torque, thus the internal extension torque)

Ankle: Begins at neutral with heel contact; this initiates a heel rocker and creates PF moment which is resisted by DF mms; once “footflat” is achieved PF ceases and DF begins, causing the plantarflexors to begin firing.
Motion: 8° PF until “footflat,” then 8° DF as tibia rocks forward.
Ankle is pronating* (eversion torque; TA and TP active)

* IR of tibia induces pronation
**MIDSTANCE**

- Interval = 10-30% of gait cycle

- It’s the beginning of single support.
- Starts as the other foot is lifted from the ground and continues until the body weight is aligned over the forefoot (and tibia of swing leg approaches vertical).
- Ends with heelrise
- A period of relative control, when your momentum moves your mass forward and slightly up (to the top of the pendulum) and great muscle activity is not usually required.

**Hip**: steadily extends toward neutral, achieving ~5° flexion
Motion= ~25° of extension. Abductor (frontal plane) mm activity! Otherwise minimal mm activity. GRFV near hip.

**Knee**: Flexion ceases early at ~20°, thereafter knee extends to ~8°. Thus motion= ~12° extension.
May be some hamstring activity early, but mostly extensor (quad) activity – this diminishes as GRFV passes thru, then anterior to the joint.

**Ankle**: Steadily dorsiflexes to ~10°. This is the ankle rocker which allows progression over the weightbearing limb.
-Motion: ~10° DF. Eccentric gastroc and soleus activity, acting to resist (slow down) DF.
Ideally, pronation slows & ceases early, followed by re-supination (and ER of the leg).
Terminal Stance completes the period of single support. Begins with heelrise and ends when the opposite foot strikes the ground. In this phase the body moves ahead of the forefoot.

30-50% of the gait cycle

High vertical forces with the initiation of "push-off" result in greater torque demands.

-Critical events: Heelrise: In spite of this the ankle is initially still dorsiflexing; DF slows then stops (due to gastroc/soleus activity), followed by PF; ↑gastroc/soleus activity; foot should be re-supinated and stable; Toe-rocker

Hip: Continued extension thru neutral to ~10° extension, for a total arc of ~15°. GRFV moves posterior to the joint, creating an extension moment. This is going to stimulate flexor activity eventually.

Knee: Continues extending to ~5° flexion, then reverses and flexes to ~12°; Motion during TSt: ~3° ext, ~7° flex. Little mm activity (possibly flexor). GRFV moves close to knee

Ankle: DF to a peak of ~12° DF, reversing near the end of the phase to reach ~10° DF. Motion: 2° DF, 2° PF

GRFV moves anterior to metatarsal heads. Eccentric, then isometric, then concentric PF activity: gastroc, soleus, TP, FDL, FHL – the whole enchilada!
**Pre-Swing** is the final phase of stance, and the 2nd period of double support. Begins with initial contact on the opposite side and ends with ipsilateral toe-off. Rapid unloading of the limb occurs as weight is transferred to the opposite side. Sometimes called “weight release” or “weight transfer” phase (I like Unloading Response).

Interval = 50-60% of gait cycle.

A major objective of Pre-Swing is to position the limb for swing.

- **Passive knee flexion to ~40°** and lots of PF

**Hip**: Reversal of direction, so that flexion to nearly neutral (0°) occurs. Motion = 10° flexion. Concentric hip flexor activity, a very important part of propulsion.

**Knee**: Rapid flexion to ~40° for an arc of 28°. Some rectus femoris & sartorius activity.

**Ankle**: Rapid PF from 10° DF to ~20° PF. Continued gastroc/soleus activity initially, but this drops off as weight is released and shifted onto the other limb.

Motion: 30° PF
Initial Swing is the first third of swing period. Begins with lift of the foot from the floor and ends when the swinging foot is opposite the stance foot.

Interval: 60-73% of gait cycle

Two important objectives are advancement of the limb from its trailing position and clearance of the swinging foot.

Hip: Rapid flexion from neutral to ~25° flexion, thus an arc of 25°. Hip flexor activity (this is Swing Phase folks!).

Knee: Continues flexing during most of this phase, reaching a peak of ~60°. Then motion reverses so that at the end of the phase a position of ~55 of flexion is achieved. Motion: ~20° flex, then 5° extension. Little mm activity (possibly some biceps femoris).

Ankle: A tiny bit of plantarflexion initially, but then dorsiflexion to about 10° in order to clear the toes. Motion: 3° PF, 10-12° DF Dorsiflexors are active.
**Mid Swing** begins when the swinging foot is opposite the stance foot and ends when the swinging limb is forward and tibia vertical (hip and knee flexion postures are approximately equal).

Interval=73-87% of gait cycle

Advancement of the swinging limb and clearance of the foot continue to be important objectives. Like Midstance, this is a period of relative control (you're sort of letting things happen).

- **Hip**: Flexion slows, then stops at the end of the phase, at a position of ~35° (of flexion). Motion = 10° of flexion. Flexor activity initially, then extensors kick in (hamstrings mostly) to slow down the swinging limb.

- **Knee**: Rapid extension to a position of ~20° flexion. Motion: ~35° of extension. A little hamstring activity toward the end of the phase, to keep the knee extension under control.

- **Ankle**: Dorsiflexion to neutral early on. Motion ~10° DF. Dorsiflexors continue to be mildly active to maintain the position of the ankle.
Terminal Swing: the final third of the swing period. Begins with a vertical tibia and ends when the foot strikes the floor/ground. Limb advancement is completed as the leg moves ahead of the thigh. The knee maximally extends.

Interval: 87-100% of gait cycle

Two important objectives are completion of limb advancement (deceleration of the swing limb) and preparation for stance.

Hip: Mostly holds steady, but may extend slightly to a position of ~30° (of flexion). Motion= 0-5° extension. Increasing activity in extensor muscles (decelerating the swinging limb).

Knee: Continues extending, nearly reaching neutral (0°), then near the end of the phase the knee flexes slightly to ~5° flexion (better for shock absorption). Motion: ~20° extension, 5° flexion. Initially mostly flexor activity, then extensors (quads) as well, for stability & in preparation for loading.

Ankle: remains in neutral. Increasing dorsiflexor activity at the end of the phase, which is helpful in preparation for the big plantarflexion moment that is about to occur.
Lower Extremity Kinematics

Sagittal plane motions

The following plates are graphic representations of the overall sagittal plane motions at the hip, knee, and ankle. It is recommended that you be able to describe such things as the position of each joint at the beginning and end of each phase, motions that are occurring during each phase, and total excursions at each joint during each phase. Hip, knee, and ankle sagittal plane motions and explanations during each phase of gait are shown in the previous section.
Motions in other planes

Pelvic Rotation
The pattern of pelvic rotation is fairly symmetrical. The pelvis rotates externally from initial contact until the onset of preswing, the first 50% of the cycle, and internally during preswing and swing, the second 50% (Note: External pelvic rotation means that if the stance limb were the right, the pelvis would be rotating to the right as seen from above).

Hip Internal and External Rotation
The hip rotates ± 8° from neutral in a monotonic fashion (i.e., like a sine-wave, with one maximum and one minimum). Peak internal rotation occurs during Pre-Swing, and peak external rotation occurs toward the end of Loading Response. That is, rotation at the hip trails pelvic rotation slightly -- so at the end of Loading Response the hip begins internally rotating and continues to do so until well into Pre-Swing when it begins externally rotating (and does so until well into Loading Response).

Hip Ab / Adduction
The hip ab- and adducts ± 7° from neutral. During the loading response, the pelvis undergoes a controlled drop on the contralateral side, thus the ipsilateral hip adducts under the eccentric control of gluteus medius and minimus. During midstance, the hip moves in the abductor direction, returning to neutral (level pelvis) by the onset of terminal stance and more or less remaining so until the onset of preswing. Just the opposite occurs thereafter, with rapid abduction as the limb is unloaded during preswing, etc. The “bump, flat, dip, flat” pattern characteristic of self-paced walking changes to a more sinusoidal pattern during rapid walking and running.
**Subtalar Joint**
The subtalar joints vary ± 5° from neutral in a roughly monotonic fashion. There is rapid eversion during the loading response, which slows greatly but continues into midstance. The subtalar joints begin re-supinating during midstance, ideally returning to neutral during terminal stance. Peak inversion occurs during preswing and the subtalar joints actually begin to return towards neutral before toe off. The return to neutral is completed during initial swing, and the subtalar joints hover near neutral for the remainder of swing, often entering initial contact in slight inversion (thus contact on postero-lateral heel).

**Lower Extremity Kinetics**
Below is a gross, graphic representation of muscular activity related to the hip, knee, and ankle during gait. Your goal is to understand and be able to describe the material in the graph from as many perspectives as possible = exactly what muscles are active and when, why they are active, what they are accomplishing, the position of the ground reaction force vector at any given time and its affect on torques at each joint, etc. Descriptions of muscular activity during each phase of gait are found in the first section.
COMMON PATHOLOGIC GAIT ABNORMALITIES

Normal walking is the standard against which pathology is measured. Efficiency is often reduced in pathology

**Focal Weakness**

Focal weakness of one or more LE muscle groups can be seen in a wide variety of disorders. The rehabilitation of the gait abnormalities due to focal weakness is relatively straightforward and relies primarily on substituting for the biomechanical deficits using bracing and assistive devices (plus strengthening if possible, of course). The more commonly encountered gait abnormalities are described below.

### Ankle Dorsiflexor Weakness

**COMMON ETIOLOGIES:**
Peroneal nerve injury at the fibular head due to trauma or compressive injuries; anterior horn cell disorders; peripheral neuropathy; severe L4 or L5 radiculopathies; myelodysplasia.

"NORMALLY": At foot contact, the dorsiflexors eccentrically contract to assist in limb loading and shock absorption as the foot plantarflexes from heel strike to a foot-flat position.

**PATHOLOGICAL PRESENTATION:**
With mild to moderate weakness, this motion is poorly controlled (restrained) leading to "foot slap", which is best observed as walking speed increases. Lateral ankle stability may be reduced (remember, the dorsiflexors also evert or invert), increasing the risk of sprains and injuries. When weakness is severe, heelstrike may be absent entirely because of an inability to dorsiflex the foot during swing.

During swing, toe clearance is reduced. This functionally lengthens the swing phase limb. A "steppage gait" (increased hip and knee flexion) is typically adopted to supply the necessary clearance.

Rx: AFO.

### Plantar flexor Weakness

**COMMON ETIOLOGIES:**
Tibial neuropathy from trauma; peripheral neuropathies (in combination with peroneal weakness); AHC disorders; plexopathies; S1 radiculopathies; myelodysplasia.

"NORMALLY": During stance, the plantarflexors normally undergo an initial eccentric contraction which controls forward tibial rotation. This is followed by a concentric contraction during pushoff which assists in moving the limb forward into swing.

**PATHOLOGICAL PRESENTATION:**
When weakness is present, excess anterior sagittal plane tibial rotation (ie, dorsiflexion) is present in mid and late stance (i.e. the foot remains dorsiflexed and heel rise is lost or attenuated). The rapid forward rotation of the tibia in stance moves the knee forward, prolonging the time during which the GRF line passes behind the knee. This increases stance phase knee flexion and the muscular demands on the quadriceps. The beneficial feature of the increase in knee flexion is to slow (but not prevent) trunk advancement over the stance phase leg. As the trunk continues its forward progression over the stance leg, the COM move further forward of the ankle joint, increasing the moment (torque) that is normally countered by the plantarflexors. This leads to a potentially unstable situation requiring than the stance limb be quickly unloaded to prevent dorsiflexion collapse. The contralateral leg step length (swing duration) is reduced so that double support is achieved early.

Rx = AFO
**Quadriceps Weakness**

**COMMON ETIOLOGIES**
- 2° femoral neuropathy from trauma; diabetic amyotrophic / mononeuropathy; AHC disorders;
- lumbar plexopathies; L3/4 radiculopathies.

“NORMALLY”:
At heelstrike, the quads normally eccentrically contract to control limb loading and prevent excessive knee flexion.

**PATHOLOGICAL PRESENTATION:**
With mild to moderate weakness, the knee is extended at or prior to heelstrike and knee flexion is eliminated or reduced. At times, this movement into full extension can be quite forceful, snapping the knee back. When normal plantarflexor and hip extensor strength is present, knee extension can be maintained by use of the hip extensors acting in a closed kinetic chain and/or by increased plantarflexor activity, which shifts the COP forward on the foot, in turn moving the GRF line in front of the knee.

With more severe weakness, the likelihood of knee instability and collapse increases. Additional strategies may be adopted to assist in knee control and to ensure that the GRF line always passes in front of the knee. These include forward trunk leaning, development of recurvatum, and use of upper extremities to assist in knee extension.

**Rx:**
With mild to moderate isolated weakness, use of a cane or other UE aid that allows for the shifting of the COM anterior to the knee for increased stability is usually adequate.

When paralysis is complete or when recurvatum develops, bracing may be needed, often an AFO with some PF built in.

**Hip Abductor Weakness**

**COMMON ETIOLOGIES:**
- Usually seen in combination with other proximal weakness from plexopathies, myopathies, AHC disease, myelodysplasia; may result from severe disuse/bed rest; often is a component of UMN gait; hip joint pathology.

“NORMALLY”:
During stance, the hip abductors stabilize the pelvis, limiting downward rotation in the frontal plane.

**PATHOLOGICAL PRESENTATION:**
During midstance, the pelvis drops toward the swing leg and there is visible lateral movement of the hip toward the stance leg. This gait pattern is known as the “uncompensated gluteus medius” or “Trendelenburg” gait and is most common when mild or moderate isolated hip abductor weakness is present. When weakness is more severe or hip pain is present, a common biomechanical compensation is to shift the COM toward the stance leg in order to decrease the stabilizing force required by the hip abductor. This compensated gait appears as a lateral shift and bending of the trunk over the stance–phase leg. Faster walking may mask the problem by reducing the time during which gravity can operate.

**Rx:**
Assistive devices used in contralateral hand allow development of a torque opposing the pelvic drop.
**Hip Extensor Weakness**

**COMMON ETIOLOGIES:**
With trauma to gluteal nerves, may see in isolation; more often seen in combination with other proximal weakness from plexopathies, myopathies, AHC disease, myelodysplasia.

“NORMALLY”: At heelstrike and in stance, the forward motion of the leg is slowed. Because of inertia, the trunk will tend to continue forward. The hip and back extensor muscles contract to control forward rotation of the trunk about the hip (pitch). In addition, hip extensors appear to also function in limb loading to assist in control of early knee flexion acting via the closed kinetic chain.

**PATHOLOGICAL PRESENTATION:**
With weakness, several compensatory patterns are observed. Walking speed is slowed to reduce forward momentum (often early strategy when weakness is bilateral and affects both hip and back extensors). The trunk COM is moved relatively posterior by increasing lumbar extension or posterior trunk lean. This allows the GRF line to pass close to or posterior to the hip, allowing gravity to assist in maintaining joint stability.

When weakness is bilateral or associated with limited hip extension, as is common in generalized myopathies, the increase in lordosis is often typically present. When weakness is isolated to gluteus maximus, there is a backward thrust or throwing of the trunk at heelstrike, which moves the trunk posteriorly. To reduce any tendency for the hip to move into flexion, there is a reduction in knee flexion and the limb is maintained in a more extended position.

Rx: 1° intervention with proximal weakness is strengthening when appropriate and the use of UE assistive devices to ensure trunk stability.

**Hip Flexion Contracture**

**COMMON ETIOLOGIES:**
Bed rest; joint disease; CDH; prolonged sitting posture (W/C).

“NORMALLY”: During stance, the hip joint normally moves from a position of about 20–30 degrees of flexion at initial contact to 10 degrees of extension in terminal stance as the trunk moves smoothly over the stance limb. Normally, the trunk remains vertically oriented over the stance limb as hip, knee and ankle motion are coordinated to keep the pelvis relatively level.

**PATHOLOGICAL PRESENTATION:** Hip joint pathology causes limitations primarily of internal rotation and extension through a combination of bony restrictions and soft tissue (anterior joint capsule) contracture.

When a hip flexion contracture is present, abnormalities will initially be seen during the latter half of stance, when maximal extension range is needed. When extension range is lacking, the pelvis must flex forward. Without any compensatory motion, this would force the trunk into a forward leaning position, moving the GRF anterior to the hip and increasing the hip extensor muscle torque required to stabilize the trunk. The most common compensatory strategy used by patients is to increase lumbar spine extension (i.e., lordosis) to allow the trunk to remain vertically oriented. Lumbar spine extension can effectively compensate for hip flexion contractures up to about 15 degrees. When hip flexion contractures exceed 15 degrees (a common occurrence) or there is limited lumbar spine extension range available (also common) the patient is forced to adopt a forward trunk tilt in terminal stance in order to complete the step.

An alternative strategy used by some patients to compensate for limited hip extension is increased knee flexion and ankle dorsiflexion (a “crouch” position). This strategy is uncommon because it ends to be very fatiguing and may increase pain in patients with hip joint arthritis.

With either gait pattern, there remains difficulty in advancing the trunk forward in terminal stance, which results in a shortened step length on the opposite leg.

Rx: Heat and stretch of anterior hip capsule & musculature to increase ROM; strengthen hip extensors in shortened range; use of UE aids to allow decreased muscular demands on hip and spine musculature.
Knee Flexion Contracture

COMMON ETIOLOGIES:
Patients with knee joint disease typically assume a resting posture of about 30 degrees of knee flexion as this decreases lateral forces and intra-articular pressure. The end result may be a knee flexion contracture.

“NORMALY”:
During stance, the knee joint moves from a fully extended position to about 10–15 degrees of flexion as the limb is loaded in early stance. This is followed by extension back to an almost straight knee in midstance, and finally rapid knee flexion in preswing.

PATHOLOGICAL PRESENTATION:
Mild degrees of knee flexion contracture (i.e. less than 15–20 degrees) are often difficult to detect with visual observation. The primary abnormality is a lack of full knee extension in stance, making the leg functionally short. This abnormality is more pronounced with rapid walking as the absence of full knee extension functionally shortens the leg, giving rise to a "short leg limp" and a mild reduction in contralateral step length. As the flexion contracture increases to more than 20–30 degrees, the lack of midstance knee extension becomes increasingly obvious. With increasing severity of the contracture, it is more difficult to advance the GRF vector anterior to the knee, its normal midstance position. This will force an increase in the muscular demands placed on the quadriceps muscle to maintain weight bearing through a flexed knee. Forward trunk leaning is used by some patients to lessen the quad demand, but this has the effect of causing compensatory hip flexion and ankle dorsiflexion, which also increases muscle demands.

Rx:
Heat and stretch of post-articular knee soft tissue to increase ROM (more difficult to achieve than increase in hip ROM); strengthen knee extensors in terminal extension; UE aids to decrease muscular demands.
Ankle Plantarflexion Contracture

COMMON ETIOLOGIES:
Most fixed ankle plantarflexion contractures are the result of prolonged passive positioning of the foot in plantarflexion (prolonged bed rest) or a result of prolonged positioning due to tone abnormalities. Dynamic loss of ankle range from plantarflexion muscle hyperactivity in UMN disorders results in similar biomechanical gait abnormalities.

"NORMALLY":
During stance, the ankle joint moves from a neutral position (90 degrees) at heel strike to 10 degrees of plantarflexion as the limb is loaded. This is followed by rapid movement into dorsiflexion (need about 10–15 degrees of dorsiflexion range of motion) which continues through midstance and early terminal stance. The ability to dorsiflex the foot in midstance is essential in allowing the smoothly controlled forward rotation of the tibia which in turn allows for a normal, smooth forward progression of the trunk over the stance limb. During swing, plantarflexion to a neutral position is needed to allow foot clearance to occur.

PATHOLOGICAL PRESENTATION:
In stance, an ankle plantarflexion contracture prevents smooth forward movement of the trunk, making it difficult to "step" through and complete a normal gait cycle. At heelstrike, a plantarflexion contracture will result in an absent heelstrike and floor contact either flatfoot or with the forefoot depending on the severity of the contracture. Floor contact with the foot plantarflexed moves the center of pressure well anterior to its usual location in early stance. This moves the GRF vector anterior to the knee, resulting in inappropriate knee extension or hyperextension during the loading response.

Several patterns of gait abnormalities and compensatory strategies can be seen with plantarflexor contractures. When no other problems are present, healthy individuals will often simply walk on the forefoot (“toe walking”). This requires good strength and the ability to walk at reasonable speeds since inertia is used to facilitate the progression of the trunk up and over the stance limb. More typically, plantarflexion contracture occurs in combination with changes in muscle tone, strength, voluntary control or with other joint abnormalities. In this context, the ability to compensate is more limited. In these patients, the plantarflexed foot moves the GRF vector anteriorly far earlier in the gait cycle than normal. This results in early and prolonged knee extension (or hyperextension), often persisting though stance. At times, this can result in a rather forceful and rapid snapping of the knee into extension, the so called “extensor thrust”.

When the plantarflexed foot prevents or severely limits forward rotation of the tibia, it becomes difficult for the trunk to progress forward over the stance limb. Increasing forward trunk lean moves the COM over the stance phase limb that remains extended at the knee and plantarflexed at the ankle. As long as the COM does not move beyond the base of support and there is adequate proximal muscle strength o control the trunk motion, this strategy allows upright posture to be maintained. It is associated, however, with very short step lengths (“step to” gait), slow walking speeds, and is usually seen in moderate to severely disabled patients.

Rx:
Prolonged stretch; serial casting to restore ROM; Achilles tendon lengthening; AFO if spasticity and dynamic posturing is primary problem; Adapt to deformity with use of heel wedge and shoe lift.
**Antalgic Gait**

Antalgic gait means that the pattern observed is a result of pain. Pain can cause a variety of responses, ranging from a lack of forceful activation up to a full blown flexor withdrawal reaction. In antalgic gait, the problem is chronic to one degree or another and the patient is attempting to compensate.

**COMMON ETIOLOGIES:**
Degenerative Joint Disease(DJD)/Osteoarthritis(OA), bony or soft tissue trauma, heelspur, etc.

**PATHOLOGICAL PRESENTATIONS:**
Most variants of antalgic gait tend to demonstrate generic features common to pain arising in many structures along with additional joint-specific abnormalities. The compensatory maneuvers used by patients are an attempt to achieve reduced weight bearing time on the painful limb, avoidance of impact loads, reduced joint excursion, and minimization of activity in muscles that cross the joint (decreases joint compressive forces). As a result, antalgic gaits from unilateral disease are characterized by slowed walking speed, asymmetry with a shortened stance phase on the painful limb, a tendency to stiffen the limb to avoid joint excursion, and an absence of forceful foot contact or pushoff.

### Hip Pain

When pain is due to hip joint pathology, a common adaptation used during stance phase is to bend the trunk over the painful stance phase limb. This brings the COM closer to the joint’s center of rotation (in the frontal plane), decreasing the joint compressive forces resulting from normal hip abductor activity. This gait pattern is essentially identical to the compensated gluteus medius gait described earlier. During swing phase, the hip is often carried in a mildly externally-rotated position, because this decreases tension on joint capsule. Patients with knee joint pain often keep the knee extended or in a slightly flexed position (especially if joint effusion is present) and the normal stance phase flexion-extension-flexion cycle of knee motion is absent or attenuated. There is a tendency to minimize knee extensor muscle activity in order to avoid joint compressive forces. Toe walking may be used to shift the GRF line anterior to the knee allowing passive stabilization without the need for knee extensor muscle activity. Some patients may carry the leg externally rotated during stance. This may be an alternate mechanism to passively stabilize the knee by using the collateral ligaments instead of knee extensor muscle activity.

### Knee Pain

When motion of the knee is painful even without weight bearing, swing phase is characterized by a stiff knee that requires circumduction or vaulting for clearance, single leg stance is shortened (to compensate, the step length may be lengthened on the affected side).

Rx: Pressure relief = unweight the knee as much as possible. Assistive device

### Arch Pain/Overpronation

The source of the pain is most commonly the attachment of the plantar fascia, the spring ligament, or the long plantar ligament.

**COMMON SOURCES OF THE PROBLEM (ETIOLOGY):** Overpronation, extended pronation, tight heelcord.

**PATHOLOGICAL PRESENTATION:**
May be very subtle, with no discernable gait deviation or perhaps a slightly decreased stance phase (or lengthened step) on affected side; most likely observation will be excessive rearfoot eversion during loading response and midstance, with pronounced medial protrusion of the talar head and collapse of the medial longitudinal arch; sometimes will manifest with weightbearing on the lateral surface of the foot (in an attempt to unweight the medial side).