

SECTION ONE

Clinical Observation

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INTRODUCTION

The ability to walk upright is a defining characteristic of man. Gait is the manner in which walking is performed and can be normal, antalgic, or unsteady. Gait analysis can be assessed by various techniques but is most commonly performed by clinical evaluation incorporating the individual's history, physical examination, and functional assessment. Gait abnormalities can be more precisely examined through the use of gait laboratories. These laboratories utilize surface EMG activity of muscles, force plates, and kinematic evaluation of the lower limbs. They are highly specialized units that assess various gait abnormalities from individuals with neuromuscular disorders to high-level athletics. While some clinical impressions have been shown to be incorrect by the use of gait lab technology, the clinical evaluation still remains the essential component in determining the etiology and the treatment plan for gait problems. A proper clinical evaluation should always precede any gait lab assessment.

Normal Gait

The determination of abnormal gait requires one to first have an understanding of the basic physiology and biomechanics of normal gait (1,2,3). The gait cycle is a time interval or sequence of motion occurring from heelstrike to heelstrike of the same foot. The gait cycle has been broadly divided into two phases: stance phase and swing phase. These phases can then be further subdivided and discussed in terms of percentage of each

within the gait cycle. This is diagrammatically represented in **Figure 1**, by Verne T. Inman, MD, PhD.

The stance phase is 60 percent of the gait cycle and can be subdivided into double-leg and single-leg stance. In double-leg stance, both feet are in contact with the ground. At an average walking speed, it represents 10 percent of the entire gait cycle, but decreases with increased walking speed and ultimately disappears as one begins to run. At slower walking velocities the double-leg support times are greater. Single-leg stance comprises up to 40 percent of the normal gait cycle. The muscles that are active during the stance phase act to prevent buckling of the support limb. These include the tibialis anterior, the quadriceps, the hamstrings, the hip abductors, the gluteus maximus, and the erector spinae (1,4,5).

The swing phase is described when the limb is not weight bearing and represents 40 percent of a single gait cycle. It is subdivided into three phases: initial swing (acceleration), midswing, and terminal swing (deceleration). Acceleration occurs as the foot is lifted from the floor and, during this time, the swing leg is rapidly accelerated forward by hip and knee flexion along with ankle dorsiflexion. Midswing occurs when the accelerating limb is aligned with the stance limb. Terminal swing then occurs as the decelerating leg prepares for contact with the floor and is controlled by the hamstring muscles.

Determinants of Gait and Energy Conservation

During gait, three main events occur in which energy is consumed. This includes controlling forward

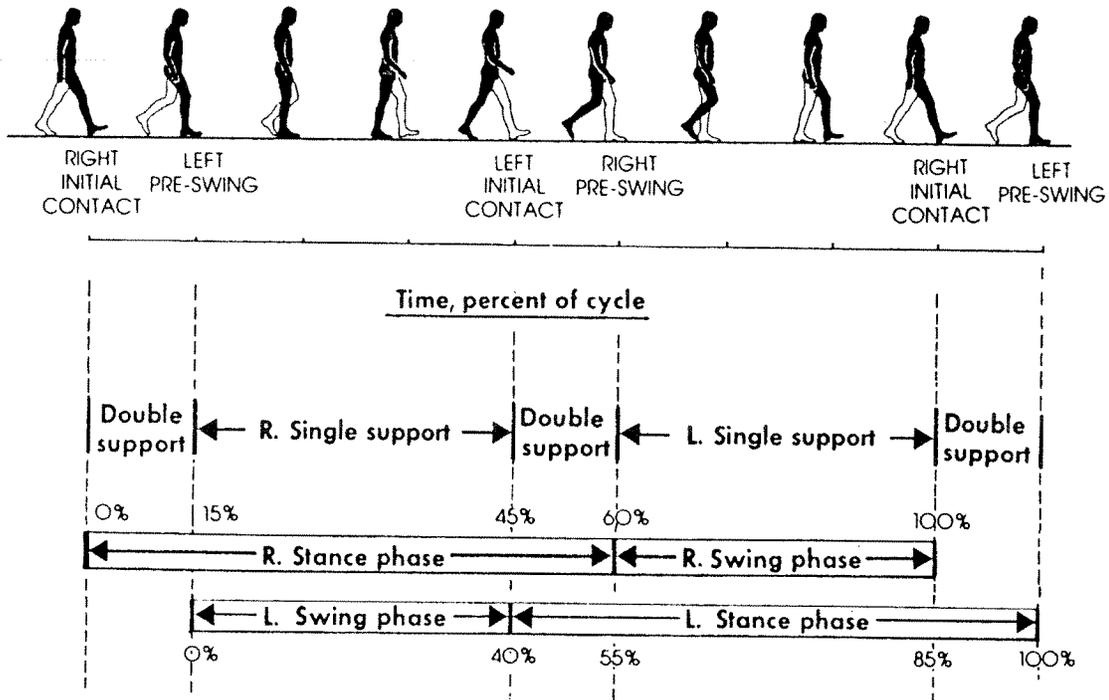


Figure 1.

Time dimensions of the gait cycle. (Reprinted, with permission, from a chapter by V.T. Inman et al., which appeared on page 26 of *Human Walking*, edited by Rose and Gamble and published by Williams & Wilkins, Baltimore, MD; 1981.)

movement during deceleration toward the end of swing phase, shock absorption at heelstrike, and propulsion during push off, when the center of gravity is propelled up and forward (6,7). Muscle activity used during the gait cycle is noted in **Table 1**.

A human's center of mass (COM) is located just anterior to the second sacral vertebra, midway between both hip joints. The least amount of energy is required when a body moves along a straight line, with the COM deviating neither up nor down, nor side to side. Such a straight line would be possible in normal gait if man's lower limbs terminated in wheels instead of feet. This obviously is not the case, thus, our COM deviates from the straight line in vertical and lateral sinusoidal displacements.

With respect to vertical displacement: the COM goes through rhythmic upward and downward motion as it moves forward. The highest point occurs at midstance, the lowest point occurs at time of double support. The average amount of vertical displacement in the adult male is approximately 5 cm.

With respect to lateral displacements: As weight is transferred from one leg to the other, there is shift of the pelvis to the weight-bearing side. The oscillation of the COM amounts to side-to-side displacement of approximately 5 cm. The lateral limits are reached at mid-stance.

In his classic article, Inman describes the components of gait (8). These are referred to as the six determinants of gait (**Table 2**). He describes several mechanical factors that help to flatten the arc in the vertical and horizontal (lateral) planes reducing displacement of the body's COM and thereby reducing the energy expenditure. The net effect is a smooth, sinusoidal translation of the COM through space along a path that requires the least amount of energy. Any pathology that increases the vertical distance between the high and low points, increases the energy cost of ambulation.

First determinant: pelvic rotation in the horizontal plane. This allows the swinging hip to move forward faster than the stance hip (1-3,8,9). Pelvic rotation

Table 1.
Primary muscular activity during the gait cycle.

Muscular Activity	Muscles	Period
Shock Absorbers	Quadriceps Dorsiflexors	Weight-Loading
Stabilizers	Gluteus Maximus, Medius, & Minimus Tensor Fascia Lata Erector Spinae	Stance-Phase
Foot Lift Off	Flexor Digitorum Longus Flexor Hallucis Longus Gastrocnemius Peroneus Longus and Brevis Soleus Tibialis Posterior	Weight-Unloading
Accelerators	Adductor Longus and Magnus Iliopsoas Sartorius	Weight-Unloading
Foot Controllers	Extensor Digitorum Longus Extensor Hallucis Longus Tibialis Anterior	Swing-Phase
Decelerators	Gracilis Semimembranosus Semitendinosus Biceps Femoris	Swing-Phase mid-swing to initial-contact

Table 2.
Determinants of Gait.

Determinant	COM Displacement	Effect
First Pelvic Rotation About the vertical axis, alternating to the right and to the left relative to line of progression	Decreased 4° of each side from a total of 8° Reduces the drop in COM during double limb support	Energy conservation saves the COM drop at its lowest point 6/16 inch (elevates end or arc)
Second Pelvic Tilt At horizontal axis at midstance	Reduces the peak of COM during single limb support	Energy conservation by shortening the pendulum of the leg (3/16 inch) at the high part of arc (depresses summit arc)
Third Knee flexion in stance	High point of COM further reduced by knee flexion in midstance	Energy conservation by decreasing rise of arc (7/16 inch) by walking over a bent knee (depresses summit arc)
Fourth & Fifth Foot and ankle mechanism	Combination of foot and ankle motion with knee motion smoothes the COM change in direction	Flattens and slightly reverses arc of translation (decreased 3/16 inch)
Sixth Lateral displacement of pelvis	Must bring COM above support point to balance on one leg	Lateral displacement of the pelvis is largely abolished by the presence of the tibial-femoral angle. There is a side-to-side sway of 1.7 inch radius

COM = Center of Mass

occurs anteriorly on the swinging limb and posteriorly during midstance. It is maximal just before heelstrike with a total motion of pelvic rotation of 3–5° to each side. Pelvic rotation also produces a longer stride length for the same amount of hip flexion of the advancing leg and hip extension of the retreating leg. Thus, it allows for longer steps without changing the COM displacement significantly.

Second determinant: pelvic tilt in the frontal plane. As the pelvis on the swing leg is lowered, the hip abductors of the stance hip control pelvic tilt. During normal gait, the pelvis drops 4–5° away from the stance leg and toward the swing leg. This pelvic dip decreases horizontal displacement of the COM during single limb support.

Third determinant: knee flexion, which acts to decrease vertical displacement of the COM. This occurs during midstance, as knee flexion to approximately 15° occurs under the control of eccentric quadriceps contraction and remains flexed until the foot is flat on the ground. These first three determinants save one inch of vertical displacement with each stride.

Fourth and Fifth determinants: involve control of the knee-ankle-foot motion. This synchronized movement results in eccentric control of plantar flexion of the ankle and knee flexion, which occurs during the first portion of the stance phase. These factors help to avoid abrupt changes of the lowest portion of COM arc, producing a smooth, sinusoidal curve instead of an arched pattern.

Sixth determinant: lateral pelvic movement. This is the lateral sway or side-to-side oscillation that occurs with each step. This defines the motion of the COM in the horizontal plane. The shifting of the pelvis occurs over the supporting foot to provide stability during the stance phase. The extent of sway is determined by the base of support. Normal knee valgus between the femur and tibia helps to reduce the amount of pelvic shifting required for stability and allows the feet to be closer together during forward progression.

Murray et al. (10) determined parameters of gait in nondisabled men. They found that the mean duration of the gait cycle was 1.03 seconds. The steps per minute were 117 (90–120 steps) and the average comfortable walking speed was 2.8 miles per hour. The average stride length was 70–82 cm and the average stride width was 8 cm, with a foot angle of 6.7°. Ostrosky et al. compared gait characteristics in young and old subjects and found that older people demonstrate less knee

extension and a shorter stride length compared with younger people (5).

The clinical evaluation of gait occurs within the context of a detailed history and physical examination. The history may reveal complaints of pain, weakness, or instability. In addition, it is important to know the individual's past medical history to be aware of underlying neurologic or musculoskeletal problems. The examination must include a detailed musculoskeletal and neurologic examination. It must address an evaluation of the person's muscle strength, joint range of motion, tone, and proprioception. The musculoskeletal examination should include, at a minimum, the joint above and below the area of complaint. The entire kinetic chain, which includes the spine and the upper limb, should be considered. The history and physical are helpful in focusing the differential diagnosis of the complaint. The observation and evaluation of gait can occur either before or after the physical examination and is included as a part of the physical examination. It is the authors' preference to evaluate the individual's gait after a detailed history and physical examination. **Figure 2** and **Table 3** summarize the main muscle actions and their timing during the gait cycle.

GAIT ANALYSIS

The observation of gait begins with a general assessment, noting symmetry and smoothness of movements of the various body parts (**Table 4**). The clinician should take note of the cadence (steps/minute), base width, stride length, arm swing, movement of the trunk, and rise of the body. The observer must then look at the individual segments of the kinetic chain as the subject ambulates, including the head, shoulders, arms, trunk, pelvis, hips, knees, ankles, and feet. Specifically, the clinician examines the head position, whether the shoulders are depressed, elevated, protracted, or retracted. The amount of arm swing can be categorized as normal, increased, or decreased. The trunk may have a forward or backward lurch or a list to the right or left. The pelvis may be hiked, level, dropped, or fixed. The hip may demonstrate increased extension, flexion, rotation, circumduction, or an adducted or abducted posture. The knee is observed for proper flexion, extension, and general stability in the various phases of the gait cycle. The ankle is examined for plantarflexion and dorsiflexion, as well as eversion and inversion.

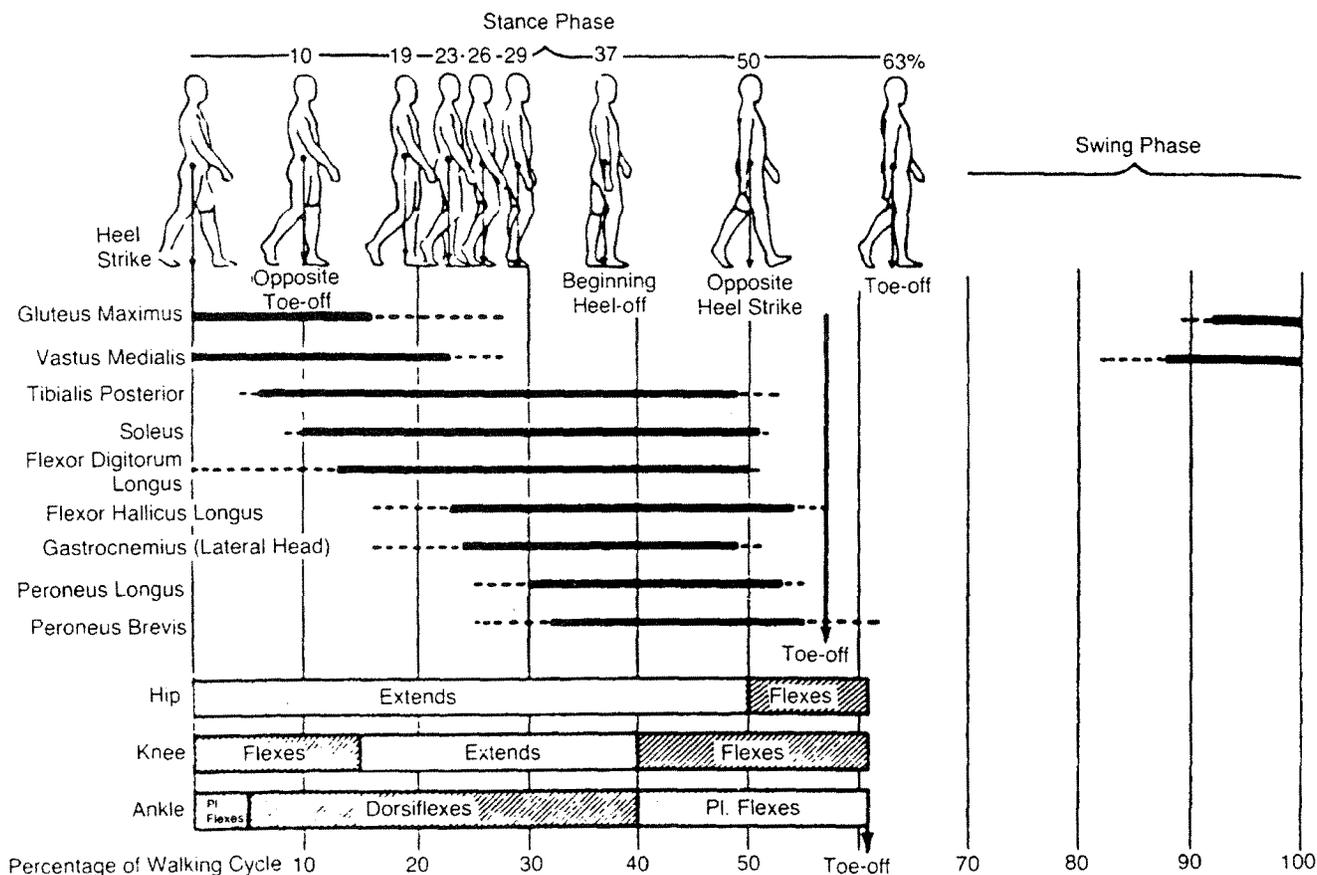


Figure 2.

On-off patterns of electromyographic activity of ankle plantar flexors. (Reprinted, with permission, from an article entitled "An electromyographic study of the plantar flexors of the ankle in normal walking on the level," by DH Sutherland, which appeared on page 66 of the *Journal of Bone and Joint Surgery* Vol. 48A:1966.)

Finally, the foot is observed for proper push off and excessive pronation and supination during weight bearing. If pain is experienced during walking, the subject should so indicate, so that its position in the gait cycle can be identified.

Because the entire gait cycle ends in a little over one second, a systematic and disciplined approach must be used to clinically evaluate a person's gait. Subjects should be viewed from the front, side, and behind, while they are wearing a minimal amount of clothing. The front view is helpful in viewing any deviations of the trunk or pelvis. One can also look for proper upper limb swing, which is usually opposite of the pelvis and lower limb. Upper limb swing helps to balance and smooth the forward progression of the body. The side view is helpful in examining exaggerations of spinal motions

(e.g., hyperlordosis and hip motion). It is also best for observing the load response of the stance leg. One can look for ankle plantarflexion, knee flexion followed by ankle dorsiflexion, and hip and knee extension, as the leg progresses to midswing. The ankle should demonstrate proper plantarflexion at initial contact and then dorsiflexion in midstance through just before heel off. The posterior view is probably best for observing pelvic abduction or adduction in determining whether there is a Trendelenberg gait.

PATHOLOGIC GAIT

Pathologic gait patterns can be broadly divided into either neuromuscular or musculoskeletal etiologies

Table 3.

Main muscle function for unimpaired ambulation.

Muscle	Gait Cycle Function
Gastrocnemius and soleus	Midstance to heelstrike
Gluteus maximus	Heelstrike to midstance
Gluteus medius and minimus	Heelstrike to toe off
Hamstrings	Midswing to heelstrike
Iliopsoas and adductors	Toe off to midswing
Quadriceps	Heelstrike to midstance Toe off to midswing
Tibialis anterior and peroneals	Heelstrike to foot flat Toe off to heelstrike

Table 4.

Gait: Major points of observation.

1. Cadence a. Symmetrical b. Rhythmic	6. Pelvic a. Anterior or posterior tilt b. Hike c. Level
2. Pain a. Where b. When	7. Knee a. Flexion, extension b. Stability
3. Stride a. Even/uneven	8. Ankle a. Dorsiflexion b. Everision, inversion
4. Shoulders a. Dipping b. Elevated, depressed, protracted, retracted	9. Foot a. Heelstrike b. Push off
5. Trunk a. Fixed deviation b. Lurch	10. Base a. Stable/variable b. Wide/narrow

(3,5,11,12). Gait deviations may be a result of structural abnormalities of the bone, joints, or soft tissue. Limitations of lower limb joint mechanisms will usually be compensated by increased motion at the joints above and below (12). Other general causes of pathologic gait include neuromuscular and myopathic conditions or painful segments of the lower limb kinetic chain. Generally, as the efficiency of the gait pattern is reduced, the energy expenditure is increased.

Common Musculoskeletal Causes of Pathologic Gait

Hip Pathology

Osteoarthritis is the most common abnormality of the hip resulting in gait abnormalities. The first changes

noted are diminished hip range of motion especially in internal rotation and flexion. This often results in exaggerated compensatory motion in the lumbar spine and the opposite unaffected hip. In severely restricted hip joints, there will be a reduction in hip flexion in the swing phase and in hip extension during the stance phase. These restrictions will be somewhat compensated by other joints (e.g., hip hiking on the unaffected side or “tiptoeing” on the affected side).

The antalgic gait is the most common pattern seen in individuals with a painful hip. This is characterized by avoidance of weight bearing on the affected side and a decrease in the stance phase on that limb in an attempt to unload the mechanical stresses on the painful hip joint. In addition, a trunk lurch toward the painful hip of the stance leg brings the COM over the joint and decreases the mechanical stress across the joint. This is done by dipping the shoulder on the affected side, elevating the opposite shoulder and shifting the pelvis over the stance leg during the stance phase of the gait cycle. During the swing phase, the hip is slightly flexed, externally rotated, and abducted in order to relax the joint capsule and ligaments to reduce joint tension. Heelstrike tends to be avoided in persons with a painful hip in order to prevent jarring and excess loading of the joint.

Knee Pathology

In general, a painful knee is maintained in slight flexion throughout the gait cycle. This is especially true if there is an intra-articular effusion, as slight flexion reduces the tension on the knee joint capsule (12). Compensation for knee flexion involves the avoidance of heelstrike and toe walking on the affected side. This type of antalgic gait may result from any painful condition of the knee joint including a meniscal tear, loose body, fracture, infection, or inflammatory synovitis.

Ligamentous instability of the knee can result in variable gait presentations depending on the ligament involved. The most common gait pattern seen in ligamentous laxity, hyperextension, or “recurvatum,” is a result of a loss of muscular control of the knee secondary to various neuromuscular problems. In these cases, the knee must rely on the static stabilizers (i.e., the ligaments and joint capsule), which become stretched and lax over time. During the stance phase of the gait cycle, the knee hyperextends, which, over time, leads to degenerative changes of the knee joint.

Another abnormal gait pattern associated with instability of the knee ligaments is the varus thrust gait pattern seen in persons with injuries of the posterior-lateral corner of the knee. These injuries usually involve a combined injury to the posterior cruciate ligament, lateral collateral ligament, posterior joint capsule, and the popliteus tendon. The combined injuries, can lead to significant functional impairment requiring reconstructive surgery. The gait pattern seen in these people is characterized by varus thrust, which occurs at the knee during the stance phase of gait. They should be differentiated from isolated injuries of the lateral collateral or posterior cruciate ligaments, which generally have a good prognosis with nonoperative treatment.

The quadriceps avoidance gait occurs in those who have suffered an injury to their anterior cruciate ligament (ACL). The quadriceps muscle provides an anterior force to the tibia, which becomes a problem in someone with an ACL deficiency, as the tibia is prone to anterior subluxation. The person will attempt to decrease the load response phase on the affected limb by decreasing the stride length and avoiding knee flexion during the mid-portion of stance (13,14).

Knee-joint contractures will also lead to abnormal gait patterns. A flexion contraction of the knee will cause signs of a short leg limp. A flexion contracture of less than 30° becomes more pronounced with faster walking speeds, while contractures of more than 30° are apparent with normal walking speeds (12). The gait is characterized by toe walking on the affected side and a steppage gait or hip hiking on the unaffected side.

Foot and Ankle Pathology

Painful conditions of the foot and ankle from trauma, inflammatory disorders, degenerative arthritis, and so forth, will result in an antalgic gait pattern. There will be an attempt to limit weight bearing through the affected area. The stride length will be greatly shortened and normal heel-to-toe motion will be lost. If the problem involves the forefoot, the person will tend to avoid plantarflexion and toe off. If the problem involves the ankle or hindfoot, then the person will avoid heelstrike at initial contact and will ambulate with a tiptoeing gait on the affected side with compensations on the unaffected side.

People with ankle instability will have great difficulty with supporting body weight during initial contact on the stance leg. At contact, the unstable ankle will often buckle with a resultant antalgic gait limiting the load response phase on the affected side.

Joint contractures of the ankle are often seen after trauma, immobilization, and neurologic problems affecting the muscles of the ankle and foot. The most common contracture seen in clinical practice is contracture of the gastrosoleus complex or “heel cord.” A tight or contracted heel cord will result in a steppage type gait pattern. There will be a loss of normal heel contact and heel-to-toe motion, along with exaggerated hip and knee flexion during the swing phase in order to clear the toe. In long-standing contracture, hyperextension of the ipsilateral knee may occur as plantarflexion at the ankle causes an extension moment at the knee.

Problems of the hindfoot, particularly of the calcaneus, will produce a similar gait pattern (e.g., elimination of heelstrike and a promotion of toe contact during stance). These problems include calcaneal fractures, plantar fasciitis, stress fractures of the ankle or calcaneus, and so forth. An antalgic or avoidance gait with a decrease in the loading of the heel is the typical pattern. In contrast, problems of the forefoot (sprain, fracture, arthritis, metatarsalgia, etc.) will result in an antalgic gait, which minimizes loading on the forefoot by decreasing plantarflexion during the stance phase and push off. People with these problems will tend to increase loading to the heel and hindfoot, and shorten the time of forefoot loading.

Leg Length Discrepancy

Leg length discrepancy can be the result of various factors affecting any segment of the kinetic chain including scoliosis and contracture of the hip, knee, and ankle, and is termed a “relative” leg length discrepancy. A true leg length discrepancy is the result of asymmetry in length of the pelvis, femur, or tibia. In either case, a leg length discrepancy can result in pelvic obliquity with a drop of the pelvis, decreased hip and knee flexion, ankle plantarflexion, and/or hyperpronation, which all occur ipsilateral to the shortened side. It is important to determine the etiology of the leg length discrepancy and to properly treat the underlying cause rather than treating all leg length discrepancy with a heel lift. In leg length discrepancy of less than 1.27 cm during the entire stance phase, one sees dipping of the shoulder on the affected side and a compensatory pelvic drop. There is an apparent elevation of the shoulder on the opposite (swing side) and an exaggerated flexion of the hip, knee and ankle on the ipsilateral side. For shortening more than 3.81 cm, he or she will walk on

tiptoes on the shortened limb during the stance phase with full knee extension.

Neurologic Causes of Abnormal Gait

Any dysfunction of the central nervous system, spinal cord, peripheral nerve(s), or muscle(s) can result in an abnormal gait (2,3,12). It is important to know the segmental innervation of the trunk and lower limbs to evaluate for abnormal gait patterns, particularly the peripheral nerve innervation of each muscle and region. In addition, neurologic injury may result in changes in motor tone and control. The more common disease problems leading to pathologic gait will be reviewed.

Hemiplegic Gait

Cerebrovascular injuries commonly result in various gait abnormalities, the most common of which is the hemiplegic gait. It is characterized by abnormal arm swing with the arm carried in adduction with flexion at the shoulder, elbow, wrist, and fingers. Also, in many people, there is an extensor synergy of the affected lower limb, consisting of extension, adduction, and internal rotation at the hip, extension at the knee, and plantarflexion and inversion of the ankle and foot. This synergy pattern is often initiated by weight bearing over the involved limb and can be useful in supporting the subject. The hemiplegic gait tends to be quite slow with a decrease in step length and an increase in the stance phase with circumduction to allow toe clearance. Compensatory changes include hip hiking from lack of knee flexion of the stance leg, a decreased lateral shift over the affected side, a lack of heelstrike secondary to the plantarflexion of the ankle, and recurvatum of the affected knee. The extension moment at the knee is created by the plantar flexion moment occurring at the ankle. Swing phase is characterized by an absent or markedly reduced knee flexion due to quadriceps spasticity. The flexor synergy gait occurs less commonly and consists of hip flexion, abduction and external rotation, knee flexion, and ankle dorsiflexion. This synergy pattern does not allow the person to stand, thereby eliminating ambulation potential.

Spastic Gait

A spastic gait can develop from an insult to the central nervous system that affects motor tone, particularly of the lower limbs. This can result in "scissoring" of the lower limbs from over-activity of the hip adductors and a narrow, crossing base. There is associated tiptoeing to maintain balance and great effort

is exerted to swing the legs forward, all of which create an unsteady fatiguing gait. In addition, isolated muscles or muscle groups may develop increased tone and spasticity. For example, spasticity of the tibialis posterior, a powerful plantarflexor and inverter of the foot, causes significant changes in gait during both the stance and swing phases. During stance phase, the initial contact will occur on the lateral aspect of the foot and plantar flexion at the ankle results in an extension moment at the knee. Plantarflexion will also result in a relative lengthening of the limb; often causing dragging of the toes and requiring increased hip and knee flexion.

Parkinsonian Gait

Parkinson's disease results from lesions of the basal ganglia affecting motor control and function bilaterally. It is characterized by a paucity of movement of the facial, trunk, and upper and lower limb muscles. This results in a gait that is slow and shuffling with short rapid steps described as being festinating. The trunk is flexed forward and the person may have difficulties with stops and turns, appearing to chase after his or her COM (12). Joint motion is reduced due to rigidity and there is usually little or no arm swing to help in balancing the individual, with falls being a common result.

Ataxic Gait

Injury to the cerebellum or its pathways may disrupt the normal coordination and precision of motor function. The gait of these individuals will be unsteady and associated with a broad standing base and a lurching or staggering of the trunk and lower limbs. Movements are uncoordinated and appear exaggerated (4). Leg placement will be variable and reproducibility is lost. An ataxic gait may also be seen in persons with sensory deficits of the lower limb. In these people, the base is wide, and there may be slapping of their feet as they hit the ground. In addition, these individuals will tend to look at their feet due to the lack of proprioceptive feedback and, therefore, have more problems at night or in the dark.

Isolated Motor Weakness Gait Problems

Gluteus Maximus (Lurch) Gait

The gluteus maximus, a major hip extensor and stabilizer of the trunk, prevents the trunk from falling forward as the COM moves forward at heelstrike. In weakness of the gluteus maximus, the hip is supported by the ligament of Bigelow, which becomes taut in

hyperextension. The individual will throw the hip backward with a "lurch" using abdominal and paraspinal muscle activation just after heelstrike on the affected side. The backward trunk lurch persists throughout stance to maintain the gravitational force line behind the hip axis locking the hip in extension. There is an apparent forward protrusion of the affected hip due to the exaggerated trunk motion and the person may also hold the shoulders backward to keep the center of gravity behind the hip joint. The hamstring muscles will often compensate for isolated gluteus maximus weakness resulting in a near normal gait pattern; however, these muscles are often affected together (e.g., in S-1 radiculopathy).

Gluteus Medius (Trendelenberg) Gait, Uncompensated or Compensated

In uncompensated gluteus medius weakness, there is a drop of the pelvis more than the usual 5° on the unaffected side beginning with heelstrike on the affected side and continuing until heelstrike on the unaffected side. There is also a lateral protrusion of the affected hip. In compensated gluteus medius gait due to severe or total paralysis of the hip abductors, the pelvic drop appears to be less as the subject laterally bends the trunk over the hip and drops the shoulder on the affected side. This serves to keep the center of gravity over the hip, which decreases the muscle force required to stabilize the pelvis. With both compensated and uncompensated gait, because the affected leg becomes functionally longer, there is an increase in hip and knee flexion and ankle dorsiflexion. This steppage gait allows for toe clearance.

Hip Flexor Weakness

Hip flexors are the major accelerators in the swing phase of gait. Weakness of the hip flexors results in a limp starting during the stance phase of gait at push off persisting throughout the swing phase of the affected side. The subject will demonstrate a trunk lurch backward and toward the unaffected side from push off to midswing. This results in locking of the hip joint on its ligaments, with further extension of the trunk as a unit from push off to midswing carrying the affected leg forward. The inertia generated from trunk and hip activity carries the limb into flexion. The stride thus becomes shortened on the affected side.

Quadriceps Weakness

Weakness of the quadriceps is most apparent during heelstrike through the stance phase of gait.

However, the limp affects all phases of the gait cycle. The affected knee must be locked in hyperextension at or preceding heelstrike by compensatory activity of the gluteus maximus extending the femur and the soleus, which extends the tibia. Extension at the femur results in flexion of the trunk and an extension moment at the knee. Some people place their hand on their thigh at heelstrike and stance to assist the knee into this extended position. With rapid walking, the affected leg lags during swing phase resulting in excessive heel rise. Repetitive hyperextension of the knee results in stretching of the ligaments and capsule of the knee and resultant recurvatum of the knee during the stance phase.

Ankle Dorsiflexor Weakness (Drop Foot, Slap Foot, or Steppage Gait)

With mild weakness, the gait abnormality will be noted at heelstrike and results in loss of plantarflexion control. Heelstrike to foot-flat phase occurs rapidly and the foot may slap at heelstrike, as eccentric control of the dorsiflexors is decreased. In severe weakness or paralysis, the foot will fall into plantarflexion during swing phase, presenting as a footdrop. Heelstrike is absent and the person comes down with the toes first or with the entire foot. This will cause a relative lengthening of the limb, compensated for by exaggerated hip and knee flexion to allow for toe clearance (steppage gait).

Gastrosoleus Weakness

Gastrosoleus weakness results in loss of ankle dorsiflexion control. Heel off will be delayed and the push off phase will be decreased. This results in a lag of forward movement of the pelvis on the unaffected side at the time of heelstrike and on the affected side during push off. There will be a shortening of the stride on the unaffected side due to the delay of forward movement of the ipsilateral hip. Altered ground reactive forces lead to a flexion moment behind the knee, which can result in knee buckling (10).

CONCLUSION

The ability to walk upright is a key functional activity which, when performed abnormally, impacts adversely on activities of daily living. The clinical evaluation of gait abnormalities, performed in conjunction with a thorough history and physical examination, is an important undertaking. These gait abnormalities

result from various neuromusculoskeletal disorders and can often be detected during the screening evaluation. Making the proper diagnosis is important in allowing for appropriate rehabilitation and/or orthotic strategies. Occasionally, for managing complicated spasticity or for determining surgical correction, a formal gait laboratory evaluation may be necessary.

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