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An Analysis of Pathological Gait

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AN ANALYSIS OF PATHOLOGICAL GAIT

by

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An Independent Study

Submitted to the Graduate Faculty of the

Department of Physical Therapy

School of Medicine

University of North Dakota

in partial fulfillment of requirements

for the degree of

Master of Physical Therapy

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1997

This Independent Study, submitted by Michael Norio Nakamoto in partial fulfillment of the requirements for the Degree of Master of Physical Therapy from the University of North Dakota, has been read by the Faculty Preceptor, Advisor, and Chairperson of Physical Therapy under whom the work has been done and is hereby approved.

(Faculty Preceptor)

Beverly Johnson

(Graduate School Advisor)

Thomas Mohr

(Chairperson, Physical Therapy)

PERMISSION

Title	An Analysis of Pathological Gait
Department	Physical Therapy
Degree	Master of Physical Therapy

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Signature Michael N. Nakamoto

Date December 20, 1996

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CHAPTER I

INTRODUCTION

The study of human motion has intrigued man since the time of Aristotle. Quantitative study of human gait in the modern era began in the early nineteenth century.[1] In recent years, the interest in the study of human locomotion and movement has grown. Computer technology is used to gather massive amounts of data from a variety of equipment such as electromyographic (EMG) amplifiers, force transducers, foot switches and electrogoniometers, to name a few. From this data base, averages of many studies and measurements have been calculated as a model for the "average" or "normal" gait.[1,2,3,4] Many years of research have provided for the present knowledge of gait and have laid the groundwork for detailed methods for analyzing gait.[3-25]

Walking is a learned activity that is unique to each individual. Yet, because everyone is designed with the same basic anatomic and physiological components, human walking is performed in a similar manner in all healthy adults. The purpose of walking is to transport the body from one place to another in a safe and energy efficient manner.[1,26] The structural design of the body permits the individual to traverse over various types of terrain, up and down inclines and stairs, and over obstacles at various speeds. To transport the body safely, the neuromuscular system must provide adequate shock absorption, maintain an upright posture of the upper body, prevent collapse of the lower limbs, and achieve safe trajectory of the foot.[8,22]

Thus, walking involves the control and integrated activity of muscles acting across many joints.

When an individual's ability to walk proficiently becomes impaired through disease, trauma, or the aging process, compensatory mechanisms and motions may develop to overcome the physical and/or neurological deficits. When these newly acquired, or abnormal walking mechanisms are substituted for a person's normal walking motion, pathological gait patterns develop.

Gait analysis involves recognizing any deviations in gait, determining what these deviations are, and using those determinations and findings to select the appropriate treatment.[1,2,25] Total evaluation of the gait pattern should include the analysis of the head, arms, and trunk (HAT), pelvis, hips, knees, ankles and feet.[3] Clinicians will frequently assess the affects the treatment protocols by observing the gait of their patients. The knowledge base required to determine a cause of any pathology in a patient's gait pattern include the integration of knowledge of normal gait, the anatomy involved, and moment arm lengths that act at each joint.[22,27]

The most common method used in clinics today is observational gait analysis. It is a convenient method that does not require expensive equipment. Observational gait analysis involves viewing and observing a patient's gait pattern and then comparing it to the parameters of "normal" gait. Joint motion is perhaps most frequently observed in this technique. The criteria for normal gait on clinical or visual examination is the absence of apparent abnormalities. It requires careful observation by a trained observer and is usually best done systematically, one joint at a time.[2,3,24,27,28]

Several observational gait analysis protocols have been developed. These techniques are used to direct the observer to a specific joint or body part during a given point in the patient's gait cycle.[28] One such protocol was developed by the Physical Therapy Department of the Rancho Los Amigos Medical Center.[5,6] This method has made gait analysis more objective than in the past.[20] The Rancho Los Amigos (RLA) technique provides a systematic assessment of the movement patterns of the trunk, pelvis, hip, knee, ankle, and foot during specific points in the gait cycle.[28,25] It also describes common gait deviations that can occur at these body segments. The observing therapist decides whether a deviation is present and records the timing and occurrence of the deviation.[28]

The benefits of gait analysis are many. They include:[2]

1. A more accurate diagnosis being made.
2. Assistance with treatment choice.
3. More accurate and objective assessment of the efficacy of the chosen treatment.
4. Comparisons of the relative merits of treatment techniques can be made.
5. Innovative methods of treatment may be suggested.
6. Recorded data to avoid mistakes of relying on memory from previous observations.
7. Readily available data may facilitate clinical discussions among disciplines.

Traditional terminology used to describe gait include heel strike, foot flat, midstance, heel off, toe off, acceleration, midswing, and deceleration.[24,26,28] However, these terms are not appropriate for some disabilities.[24] Therefore, to avoid inaccuracies and confusion RLA developed another system of describing gait using period of time. These are initial contact, loading response, midstance, terminal stance, preswing, initial swing, midswing, and terminal swing.[5,6]

The purpose of this literature review is twofold. First, to provide the reader with a basic understanding of the terminology and mechanisms of normal gait, and second, to provide a tool which can be used to identify some common pathological gait patterns and their possible causes. To accomplish this, this paper will present: 1) a description of the gait cycle as proposed by the Rancho Los Amigos Medical Center, 2) normal and pathological gait components of the foot, ankle, knee and hip, and 3) a case study and video recording of the gait pattern of a person with an incomplete spinal cord injury. It is hoped that the reader will gain a better understanding of human locomotion and use this knowledge in identifying and properly treating patients with pathological gait.

CHAPTER II

PHASES OF GAIT

Normal human gait is a sequence of reciprocal limb motions which progress the body along while maintaining balance, conserving energy, and absorbing the impact of the ground as contact is made.[5,26] It involves a series of orchestrated interactions between the total body mass and the two lower limbs. As the body moves forward, one lower extremity provides a source of support and propulsion, while the other is advanced to become the new mobile support.[5,26,29] Body weight is transferred from one limb to the other at the time when both feet are in contact with the ground. This sequence of events is continually repeated as the individual walks toward a desired destination.[5,26,30,31,40]

Gait is a very complex activity. Many years of research and improved technology have provided comprehensive methods to analyze gait. This chapter will begin to define the basic components and terminology that describes a gait cycle as derived by the Pathokinesthiology and Physical Therapy Departments of the Rancho Los Amegos Medical Center.[5,6] Since gait consists of a series of interactions that continuously flow from one event to the next, there is no specific event that marks the start or end of a cycle.[5,32] Generally, a gait cycle is described by a single sequence of events performed by one limb (referred to as the reference limb). The moment ground contact is made by the reference limb is commonly considered the start of a gait cycle. It ends with the same foot striking the ground again.[5,6,26,29,30,31,32,33,37,40]

Periods of Gait

Traditionally, the gait cycle is divided into two periods: stance and swing.[5,6,7,10,26,29,31] Stance is used to designate the entire period in which the reference foot is on the ground. It begins the moment one extremity contacts the ground and continues until the same extremity is lifted from the ground. Stance can also be divided into three intervals according to the pattern of floor contact: initial double stance, terminal double stance, and single limb support.[5] Initial and terminal double stance mark the beginning and the end of the stance period, during which both feet are in contact with the ground. Weight of the body mass is alternately transferred from one limb to the other.[1,30,33] During most of the double stance intervals, body weight is not evenly distributed over the two supporting limbs.[5,6,26,30] Single limb support begins when the opposite foot is lifted for the swing phase and the reference limb is left to support the entire weight of the body.

The swing period refers to the time the reference extremity is in the air during limb advancement. It begins as soon as the reference foot is lifted off the ground and ends just before the same foot makes contact with the ground again. Swing is comprised of three components: 1) an initial period in which the rate of swing can be altered, 2) a transition period, and 3) a final segment in which the speed of the limb reversed.[30] The swinging limb acts as a pendulum in which the inertia of its segments determine the duration of the swing. Variation in cadence can be achieved by accelerating or decelerating the pendulum limb during the initial segment of the swing period.[5,30]

The normal distribution of time between the two periods during the gait cycle is approximately 60% for stance and 40% for swing.[1,5,7,26,30,32] Percentages will vary according to the walking speed of each individual. Walking faster lengthens the single limb support time, while decreasing time in double stance.[1,21,30,33]

Functional Tasks

In order to walk, an individual must be able to accept and support the weight of the body with one lower extremity, and swing the other forward. Thus, gait can be defined in terms of three basic tasks that must be accomplished during the stance and swing periods for an individual to progress toward a desired destination. They are: 1) Weight Acceptance (WA), 2) Single Limb Support (SLS), and 3) Swing Limb Advancement (SLA).[5,6,26] Weight acceptance and SLS are accomplished during the stance period and SLA in the swing period.

Weight acceptance is the most demanding of the three tasks, and it begins the stance period of the gait cycle. Three functional patterns are needed to complete this task:[5] 1) a rapid transfer of weight onto an extended limb, 2) shock absorption as contact is made with the ground, and 3) maintenance of stability as the body moves in a forward path. During SLS, the body moves forward over a single, stable limb, starting when the opposite foot comes off the ground and continuing until the same foot contacts the ground again. Swing limb advancement is characterized by the unloading of the reference limb as the foot is lifted from the ground. The extremity is brought forward from behind the body and positioned out in front of the body as the knee extends to take the next step.[5,6]

Functional Phases of Gait

The sequence of events that constitute a gait cycle have been overviewed along with the basic divisions of gait and the elements that identify each period. Focus will now be directed on the functional phases of gait which are the basic components that allow the stance and swing periods to occur. The functional phases of gait provide a way to better understand the relationships between the interactions of the foot, ankle, knee, and hip as they progress through the gait cycle and will be central in describing normal and pathological gait patterns throughout this literary review. Understanding how each joint functions individually, as well as a whole unit, is important in assessing the effects of disability.[5]

The eight phases of gait as described by the Rancho Los Amigos Medical Center[5,6] are initial contact (IC), loading response (LR), midstance (MSt), terminal stance (TSt), preswing (PSw), initial swing (ISw), midswing (MSw), and terminal swing (TSw). Each of the eight phases represents a 'period of time' in the gait cycle and has a specific objective or goal that, when accomplished, allows for the progression of the events needed to complete a gait cycle. As these phases are sequentially performed, it allows the lower extremities to carry out the three functional tasks (WA, SLS, and SLA).[5]

Initial contact is considered the start of the gait cycle.[5] However, Devita[8] proposes toe off to be a better convention to use as the start of gait. Initial contact begins the moment the foot of the reference limb makes contact with the ground. This phase marks the start of initial double stance. In normal gait, the heel is usually the point of contact. In pathological gait, however, it is possible for either the entire foot or the toes to make initial contact, rather than the heel. The principle objective of this phase is to correctly position the foot as it comes into contact with the ground. The body begins to decelerate as it prepares to make contact and absorb the shock from the ground forces.[5,26,30,32]

Loading response occurs immediately after initial contact from 0% to 10% of the gait cycle and continues until the opposite limb lifts off the ground.[5,32] The body decelerates as the shock of impact is absorbed.[14] The objective is to maintain a smooth progression of forward motion as the body mass is slowed to prepare for its downward path from its peak in midstance. Force plate studies show deceleration amounts of 20% to 30% of body weight during normal gait.[30]

In **midstance**, the body is in SLS. The interval of this limb is from 10% to 30% of the gait cycle.[5,26,32] Primary goals of this phase are maintaining stability at the hip and knee by utilizing momentum, while advancing the body over a stationary limb.[5,6] This phase starts when the opposite limb is lifted from the ground and continues to a position where the body has progressed over and ahead of the supporting reference extremity. Here the body's center of gravity (COG) reaches its highest point as the body positions itself directly over the supporting limb.[5,7,26,32]

Terminal stance (30% - 50% of the gait cycle) continues on from the end of midstance to a point just before the opposite foot makes ground contact, or following the lifting of the heel (heel off) of the reference foot.[5,26,32] The main objective of terminal stance is to provide proper acceleration and to ensure that there is adequate step length.[6] The acceleration is fueled by the body's COG as it moves down and forward from its highest point reached during midstance. As the COG moves in front of the base of support, the body falls anteriorly and toward the unsupported side.[5,6,26]

Preswing is the final phase of the stance period and is the second of the double limb stances of the gait cycle. This sequence comes at the 50% - 60% interval of the gait cycle.[5,26] It is the period from heel off to toe off, as the contralateral limb begins a stance period with initial contact. The principle goal of preswing is to position the reference extremity so it can swing forward. This is accomplished by shifting the weight from the reference limb onto the opposite limb.[5,6]

Initial swing (60% - 73% of the gait cycle) comprises approximately one third of the swing period.[5] Onset occurs when the reference foot is lifted off the ground and ends when the swinging limb reaches maximum knee flexion and advances to a point opposite the stationary, non-reference stance limb. The critical goal of this phase is to allow the reference extremity to clear the ground to be able to advance.[6,26,32]

Midswing follows immediately after maximum knee flexion of the swing limb occurs and continues until the limb is forward of the body and the tibia is in a vertical position relative to the ground. Midswing encompasses the 73% - 87% interval of the gait cycle.[5] The objective of midswing is to maintain foot clearance while advancing the reference limb.[6] In other words, the foot is kept at a distance above the ground until the swing limb is ready to make contact with the ground. In normal gait, foot clearance is only 0.87 cm. during midswing.[5,26,30]

Terminal swing (87% - 100% of the gait cycle) is the final phase of the swing period.[5] It begins at the vertical tibia and ends just before the reference foot contacts the ground again. As the knee extends, deceleration of the reference limb and repositioning of the foot for contact with the ground are the primary goals. This phase is crucial as it sets up for initial contact marking the onset of the next gait cycle.[5,6,26,30]

CHAPTER III

BASIC FUNCTIONS

As a walking entity, the body can be divided into two functional units, the upper body and the lower extremities. The head, arm, neck, trunk comprise the upper body unit and is designated by the term HAT.[10] Maintaining neutral vertebral and postural alignment are the major requirements of the HAT, and its contribution to the act of walking is very minimal. Forces from the arm swing and hip movements also help to stabilize the HAT during normal gait.[5,14] The lower extremity unit includes the pelvis and the lower limbs. The pelvis is considered to be part of both the HAT and lower extremity units because it provides a base from which the HAT rides on the hip joints and it serves as a mobile link between the lower extremities.[5] Four basic functions must be performed by the lower extremities as it carries the HAT along its desired pathway:[5,8] 1) maintain upright stability despite an ever changing posture, 2) generate a propulsive force for progression, 3) absorb the impact of floor contact with each stride, and 4) minimize the required amount of muscular activity to conserve energy.

Upright Stability

Maintaining a stable upright position depends on the functional balance between the alignment of the body and muscle activity at each joint. Without proper restraints, each body segment will fall from the pull of gravity. There are three factors that make it difficult for the body to maintain a stable upright posture: 1) body weight alignment, 2) skeletal properties, and 3) multisegmented lower limbs.[5]

The most significant is the alignment of body weight. During gait, three external forces act on the body: inertia, gravity, and the ground reaction force vector (GRFV), or body vector. Inertial properties of the body segments create a force that is proportional to, but opposite in direction to the accelerative force. Gravity acts as a force that is directed downward through the center of mass of each segment. The GRFV represents the force of the ground on the foot, of equal magnitude, but opposite in direction to the force applied to the ground by the body, through the foot while it is in contact with the ground.[8] Internal forces created by muscles, tendons and ligaments are used to counter the external forces. The relationship between the body vector and the alignment of the various joints determine the magnitude and degree of instability, and consequently, the magnitude and intensity of the internal forces required to maintain stability.[5,26]

Progression

During walking, the lower extremities rely on various mechanisms and forces to assist in moving the body forward. The momentum created by the forward fall of the body is the primary force. The ankle and foot perform rocking actions at the heel, ankle and forefoot of the stance limb to maintain and control momentum. The hips generate a force to accelerate the swing limb to advance the HAT past the vertical position of the support limb. Both the swing leg and stance leg act simultaneously to accelerate the HAT forward.[5,14]

From quiet standing, the body performs three actions to begin walking. It starts with a slight shift of body weight toward the side of the swing leg. Next, all weight is shifted laterally onto the stance limb. Finally, the body weight is transferred forward on the stance limb as body weight falls forward and the swing leg is lifted.[5]

Once the swing (reference) limb has advanced and takes on the role of the stance limb, body weight is dropped onto that limb. The force generated by the forward fall is directed toward the ground. The first of three rocker mechanisms, the heel rocker, is used to redirect the force in a forward direction.[5] Initially, floor contact is made by the rounded surfaces of the calcaneus. As body weight moves forward over the limb it causes the foot to fall toward the ground. Activity by the pretibial muscles control the rate at which the foot drops. In this manner, the tibia is drawn forward. At the same time the quadricep muscles link the femur and tibia while restraining the rate of knee flexion; thus the entire leg is pulled forward.[5,14]

Progression is sustained next by the ankle rocker. As the forefoot strikes the floor, the tibia continues its advancement through passive dorsiflexion (DF) created by the momentum. Muscle action by the soleus plays a major role in the ankle rocker mechanism. It contracts to stabilize the tibia for knee extension. At the same time, with assistance from the gastrocnemius, it controls the progression of the tibia.[5]

As the weight of the HAT falls beyond the supporting foot progression accelerates. The GRFV moves to the metatarsal heads to create a forefoot rocker. As the body weight moves over the forefoot rocker, the heel is lifted, and preparations are made to accelerate the limb from preswing through its advancement in swing.[5]

A series of interactions between the ankle, knee and hip continues the progression of the reference limb through preswing. First, the ankle is dorsiflexed, which places the body vector over the metatarsal heads. Second, transfer of the body weight to the contralateral limb rapidly unloads the stance limb. Third, contraction of the gastrosoleus muscle plantar flexes the foot on the forefoot rocker. This advances the tibia and flexes the knee. Lastly, the adductor longus muscle acts to flex the hip and to keep the body from falling medially.[5,14]

Progressional force is maintained by the reference limb as the hip continues to flex and advance the limb. An additional force is needed to propel the body mass up and forward over the contralateral stance limb. This is supplied by a combination of knee extension and further advancement of the thigh of the reference limb.[5] Active knee extension places the weight of the tibia to the front of the swinging limb and provides enough pulling force to maintain momentum. At the end of terminal swing the limb is properly positioned to accept the falling body weight and begin the next cycle of progression.[5,14]

Shock Absorption

The immediate response to floor contact at heel strike is passive ankle plantar flexion (PF). Pretibial muscles act to slow the rate of PF, reducing the rate at which falling body weight is transferred onto the floor.[5,6,32] In working to control ankle PF during loading response, the pretibial muscles pull the tibia forward by means of its attachment to the foot and tibia. The forward motion of the tibia along with a flexion moment created by the GRFV, causes the knee to flex. Activation of the quadricep muscles restrains knee flexion and provides another means of shock absorption as some of the loading force is transferred to the quadriceps, thus reducing the impact of the loading forces on the joints. Another shock absorbing mechanism occurs at the pelvis. As weight acceptance of one limb unloads the weight from the other, the pelvis and HAT drop toward the non-weight bearing extremity. By controlling the rate of the pelvic drop, the abductor muscles of the support limb help to absorb the impact of limb loading.[5]

Energy Conservation

Moving the body through space with the least amount of energy expenditure is the primary objective during normal walking. The energy cost of locomotion is dependent on the muscular effort required to maintain stance stability and to advance the lower extremities along the desired path. A person's endurance will depend on the amount of energy expended during muscle activity.[5] Energy expenditure in human walking is frequently linked to the movement of the body's COG. If changes in the body's COG are abrupt and large, more energy is expended.[29] To maximize energy efficiency, two mechanisms are used during gait: 1) monitoring of the COG's alignment, and 2) selective muscular control. Both act to reduce the intensity and duration of muscular activity.[1,5,6]

The major mechanism in conserving energy is to minimize the displacement of the body's COG from its line of progression. In normal human walking, the COG follows a smooth sinusoidal curve in both sagittal and coronal planes.[14,19] Located anteriorly to the tenth thoracic vertebrae, the COG averages less than five cm of total displacement during normal gait.[1,5,26,29] To minimize the COG's displacement, the body uses a combination of six motion patterns known as the determinants of gait. They are: 1) pelvic rotation, 2) pelvic tilt, 3) early knee flexion, 4) foot and ankle motion, 5) late knee flexion, and 6) lateral pelvic rotation.[5,7,14,26,34,35,36] These determinants function as a result of proper posturing of the lower extremities and pelvis.

As the swing limb advances, the pelvis is rotated forward in the transverse plane. This rotation lengthens the stride length by increasing the distance between the points of floor contact. The COG descends to its lowest position during the periods of double stance as pelvic rotation functions to minimize the caudal displacement of the COG.[1,5,7,26,29,34,35,37]

During single limb support, the COG reaches its highest point in the gait cycle. A lateral tilt or drop of the pelvis on the side of the swing limb lowers the COG and decreases its potential vertical displacement. The combined weight of the HAT and the swinging limb is balanced and controlled by the hip abductors of the stance limb. For the pelvic tilt to be effective, full knee flexion is necessary.[1,5,7,26,29,34,35,36]

Knee flexion during stance occurs immediately after initial contact as the knee flexes approximately 15° during limb loading.[5,29] Flexion shortens the length of the supporting limb and lowers the COG to keep it from rising as high as it could if the knee were extended. Knee flexion occurs a second time in the stance period during preswing and serves the same purpose.[1,5,7,26,29,35,36]

Foot and ankle motion after heel strike and at toe off is PF. These gait components place the ankle at its lowest and highest position and gives length to the lead and trailing limb, respectively.[5,36] Ankle motion, combined with the movements of the knee and foot, prevent abrupt changes in the vertical displacement of the COG, thereby smoothing the pathway of the COG and reducing deceleration and acceleration.[1,5,7,26,29,35,36]

Lateral pelvic displacement occurs in the frontal plane as body weight is loaded and unloaded between the alternating support limbs. This motion is minimized by two factors.[1] First, the natural valgus at the knee reduces the width of the base of support and decreases the medial-lateral displacement of the COG. If the long bones of the lower extremity were formed in a straight line from hip to foot, the lateral shift of the COG would more than double. Second, as the stance limb is loaded, there is a slight increase in knee abduction which brings the COG closer to the supporting foot.[1,5,7,26,29,35,36]

The second mechanism used to minimize the amount of energy expended comes by the timing of serial muscle response and the use of passive posturing and momentum. Muscles are able to relax and minimize their activities when momentum and ligamentous or fascial tension stabilize joints to counter the moments created by the GRFVs.[5]

The ability to maintain an upright posture while generating forces needed for limb and body advancement, as well as dispersing the impact of limb loading and conserving energy require accurate and detailed coordination of all components involved in human walking. Any change in kinetics alters the way in which the body is able or unable to progress through the gait cycle.[22] The following chapter will discuss the elements involved in making these basic functions of gait possible.

CHAPTER IV

COMPONENTS OF NORMAL GAIT

Normal human gait is a series of repeated sequential events which interact with one another to propel the body along a desired path in the most efficient manner. The lower extremities along with the pelvis make up the primary unit that provides the necessary mechanisms involved in gait. Eleven articulations and forty-seven muscles work together on a timely basis to move this multisegmented unit forward.[5] This chapter will describe the forces acting at the foot, ankle, knee, and hip joints and the mechanisms which must occur at each joint to achieve a normal gait pattern.

The Foot

The foot consists of three articulations that have major functional significance during human normal gait walking. They are: 1) subtalar (ST), 2) midtarsal (MT), and 3) metatarsal phalangeal (MTP) joints.

The ST joint lies between the talus and the calcaneus. It functions to accommodate vertical weight bearing, it adds mobility to the coronal and transverse planes of the ankle, and it modifies the motions of the other joints of the foot. [5,35]

The MT joint links the midfoot to the forefoot. It is comprised of two articulations--the talonavicular and the calcaneocuboid. Motion here aids in absorbing the demands placed on the foot by the loading response phase.[5,26]

The MTP joints provide a wide base of support across the forefoot. The articulation between the metatarsal heads and the phalanges give the foot the ability to roll over the metatarsal heads instead of the tips of the toes. Adjustment by the proximal phalanges provide stability during forward progression of the body over the foot.[5,26,35]

Motions of the Foot

In the coronal plane the ST joint inverts and everts the foot around an oblique axis.[35] At heel strike the foot is inverted then quickly everts during loading response and returns to an inverted position throughout terminal stance. The foot is a rigid lever when inverted, and becomes a mobile adaptor in the everted position.[38]

Motions at the MT joint have to do with the flattening and recovery of the arch. During forefoot contact through early midstance, the arch is flattened and is restored at terminal stance.[5]

The MTP joints are in a dorsiflexed (i.e. extended) position at initial contact. At the end of loading response and through midstance they return to a neutral position. Heel rise at the onset of terminal stance places the MTP joints in an extended position again. This allows maximal floor contact in preswing. During initial swing MTP DF is decreased and maintained in a slight position through swing phase. At terminal swing MTP extension increases in preparation for initial contact.[5]

Muscle Activity at the Foot

Ten muscles are involved in controlling the foot during gait. Muscle control within the foot begins in the hindfoot, progresses to the forefoot, and finally to the toes.[5] Five muscles are invertors (supinators) of the foot, including the tibialis posterior, tibialis anterior, flexor digitorum longus (FDL), flexor hallicus longus (FHL) and soleus. They cross the ST joint medially.[11] On the lateral side of the ankle lay five evertor muscles. They are the extensor digitorum longus (EDL), peroneus tertius, gastrocnemius, peroneus longus and brevis.[5,26]

Functions of the Foot

Shock absorption, weight bearing stability, and progression are a product of muscular control and motion of the foot during gait.[5] These events occur sequentially from initial contact to preswing.

The mechanisms used for shock absorption are ST eversion and MT DF. Subtalar eversion occurs at heel strike because the calcaneus is lateral to the longitudinal axis of the tibia, causing the talus to evert under the load.[11] As the foot descends towards the floor, the foot moves laterally, decreasing calcaneal support of the talus. This brings the talus to an inverted position which causes the tibia to rotate medially. By decelerating ST eversion, the invertor muscles act to absorb some of the impact of floor contact.[11,35] Dorsiflexion of the MT joint at the onset of midstance also assists in shock absorption. It follows ST eversion occurring at loading response.[5]

The greatest demand for foot stability happens at heel rise when the forefoot briefly supports the body.[5] With the advancement of the body mass over the foot, a need for MT stability increases as load demands on the forefoot increases. By reversing its position from eversion to inversion, the talus locks the MT joints and changes the foot from a mobile adaptor to a rigid support. Inversion moves the calcaneus under the talus and externally rotates the tibia. Synergistic activity of the two peroneal muscles prevent excessive inversion during heel rise and preswing.[5,26,35]

To optimize the effects of the forefoot rocker, it is essential to control the motion of the MTP joints.[5] During midstance the MTP joints lie in a neutral position.

However, the MTP joints must extend as the body mass continues forward and the heel rises, placing the body weight entirely on the forefoot.[5,26]

Control by the toe flexors determine the shape and stability of the forefoot rocker. Floor contact by the distal phalanges help broaden and stabilize the base of support. Forefoot contact is further enhanced as the toe flexors bring the base of the proximal phalanges in line with the metatarsal heads. Consequently, weight bearing pressure is minimized.[5,26]

The insertion of the peroneus longus onto the first metatarsal also improves the support area of the forefoot. Plantar flexing the first ray strengthens the weight bearing capacity on the medial aspect of the forefoot. This becomes significant when the body weight is transferred toward the first ray in preparation for weight acceptance of the contralateral limb.[5]

Ankle

The ankle is unique in that it is able to take a vertical weight-bearing force, coming through the leg (tibia) and disperse that force to a horizontal support system[5] (foot). Within the ankle are two separate, functioning joints: the ankle (tibiotalar) joint is the junction between the tibia and the talus.[5,26,35] The ST joint links the talus to the calcaneus. The talus acts as a weight-bearing link between the tibia and calcaneus and it provides three dimensional mobility for the two single axis joints.[5,26,35]

Motion of the Ankle

The primary motions of the tibiotalar joint are PF and DF[5,26,35]. During each gait cycle the ankle will twice make a transition between PF and DF.[5] At initial contact the ankle is at or near neutral. The first PF motion immediately follows during loading response as the foot is projected towards the floor. When the forefoot makes contact with the ground, the foot becomes stationary. The tibia then becomes the moving segment as the body mass moves over the foot, causing the ankle to dorsiflex. Dorsiflexion continues through midstance and part of terminal stance. This is followed by a rapid ankle PF at the onset of preswing. The final motion, DF, occurs at toe off which restores the ankle to a neutral position and allows the foot to clear the ground as it swings forward to prepare for the next heel strike.[5,23,38]

Muscle Activity at the Ankle/Foot

The muscles controlling ankle motion in the sagittal plane are specific to the phases of gait. Plantar flexors contract throughout stance, while the dorsiflexors are active during swing. The exception occurs during the loading response phase where the dorsiflexors work to monitor the rate of ankle PF.[5]

Muscle activity in the ankle provides weight acceptance during stance and propulsion at toe off.[23] Perry,[5] however, reports muscle action of the triceps surae, before toe off, is sufficient only to eccentrically support heel rise. No force is added to thrust the body forward. The pre-tibial muscles, tibialis anterior (TA), extensor hallicus longus (EHL), extensor digitorum longus (EDL), and peroneus tertius, work to dorsiflex the ankle as well as eccentrically controlling PF. Muscle activity is greatest during preswing and early loading response.[23,35]

Preswing initiates pretibial muscle activity into DF. Tibialis anterior activity greatly increases throughout initial swing to control ground clearance of the foot as the transition from stance to swing is made.[6,23] Muscle activity in midswing is highly variable. Perry[5] describes it as minimal and Smidt[38] found it to be non-existent in some individuals. In terminal stance dorsiflexors renew their activity as the foot is positioned for stance. Muscle action is intensified during the transition from initial contact to loading response. By the end of loading response, all dorsiflexor activity is terminated. The tibialis anterior contributes to ankle stabilization at the later stage of loading response.[5,38]

Ankle plantar flexors are comprised of seven muscles. Two of which account for more than 90% of the functional torque produced by the group.[5] The triceps surae (soleus and gastrocnemius) have that capacity because of their size and lever arm advantage. The other five (posterior tibialis, flexor digitorum longus, flexor halliais longus, peroneus brevis, peroneus longus) are relatively small and contribute to PF motion to a much lesser degree. The triceps surae is activated towards the end of loading response. Both the soleus and gastrocnemius continue to work through midstance and peak torque is reached at the onset of terminal stance. Muscle activity of the triceps surae declines rapidly and ends shortly after preswing.[5,23] Alignment of the five smaller muscles result in low plantar flexor capabilities. Instead, they function more to control the ST joint and other articulations in the foot.[5] Function of the long toe flexors is to stabilize the toe during metatarsal contact of the foot. Onset of the muscular activities in the peroneus longus and peroneus brevis begin during midstance and ends in mid preswing. The peroneals and the tibialis anterior work as antagonists to control inversion/eversion of the foot. During walking, they control weight distribution on the lateral aspect of the foot in stance and orientation of the foot in swing.[5,35,38]

Functions of the Ankle

Initial Contact.--Ankle position is at neutral (0°) to initiate heel rocker.[5,6] The GRFV is posterior to the ankle joint and the stance limb is prepped to respond in preserving progression and provide shock absorption. The ankle, being in neutral, provides sufficient time for heel only support.[5] Dorsiflexion pull from the pretibial muscles give support to the foot. A lack of precision in controlling ankle position into PF will reduce heel rocker potential proportionately.[5,6,8,32]

Loading Response.--The first PF motion occurs during this phase. Heel rocker is used to preserve forward motion because the line of the body vector force is directed toward the ground.[5] Vector force provides limb stability but not progression. With GRFV positioned posterior to the ankle joint and the rapid limb loading a PF moment of the foot is produced. Pretibial muscles act aggressively to reduce the rate of PF.[8,32] By doing this, the heel support period is extended and the tibia is drawn forward. The body mass is also drawn forward on the heel by the tibial advancement and by the passive drop of the foot to the ground.[5,6,32,8]

Midstance.--The major components of this phase include the first arc of ankle DF and the ankle rocker for progression.[5,6] Stability of the stance limb is maintained by the soleus. Throughout midstance, GRFV advances along the foot in response to the momentum of the contralateral swing limb and the forward fall of the body weight. A DF torque is created to move the tibia forward over the foot from a PF position to DF. The heel and forefoot remain in contact with the ground. The soleus and gastrocnemius react to slow the initial rate of DF as the forefoot contacts the ground. This controls the rate of tibial advancement.[5,6] Soleus activity is the dominant decelerating force due to its direct tie to the tibia and calcaneus. The gastrocnemius has no direct tie to the tibia because of its attachment at the distal femur. Its posterior position to the knee joint also makes it a knee flexor. This increases the demand on the quadricep muscles, which can be a problem for those who lack normal strength and control.[5,6,8,30,32,] Terminal Stance.--Heel rise with continued ankle DF and the use of the forefoot rocker for progression are the major achievements of terminal stance.[5,6] The body vector is aligned on the forefoot. Triceps surae act to stabilize ankle position as the heel rises off the ground and the tibia continues to advance. The forefoot becomes the only supporting surface as the metatarsal heads provide a forefoot rocker to sustain progression. Maximal DF torque is generated by the position of the GRFV in creating a lever arm the length of the forefoot.[5] This, in combination with the falling body weight and heel rise, demands strong triceps surae activity to maintain ankle stability with minimal motion. The body's COG is positioned anterior to the base of support by the DF motion and the raising of the heel. By progressing past the metatarsal head axis, the COG causes the foot to roll on the forefoot which in turn increases the height of heel rise. Dorsiflexion torque is increased which allows a free forward fall that creates a force to advance the body forward. Terminal stance ends when the contralateral limb makes initial contact and double limb stance provides stability again.[5,6,8,26,32,39]

Preswing.--Primary function of preswing is to initiate knee flexion for swing. Intensity in activity of the plantar flexor muscles is greatly reduced following the start of double limb support. The need for strong eccentric stabilization of the ankle is diminished as the body weight is shifted onto the contralateral limb. Muscle activity of the soleus and gastrocnemius rapidly declines, but contracts just enough to provide a force that accelerates the advancement of the limb to head into the swing phase.[5] During most of the phase the foot does not bear significant weight which demonstrates double stance involves bilateral foot contact but not with equal weight distribution.[5,6,14,26,32,39]

Initial Swing.--Initial swing is highlighted by floor clearance of the foot for limb advancement. The onset of initial swing begins at the moment of toe off. With the foot and tibia trailing behind the body, the ankle is placed in a PF position. A rapid change to DF occurs to allow the foot to clear the ground. Pretibial muscles act to bring the ankle towards neutral as it approaches midswing. Toe extensor muscles place toes in an extended position to assist in foot clearance.[5,6,32]

Midswing.--Ankle DF and floor clearance are continued through mid swing. The tibialis anterior and EHL reach peak torque early during this phase as it must resist the downward torque created by the weight of the tibia. Ankle DF is maintained at neutral or slightly above. Muscle action declines toward the end of midswing as an isometric contraction is used to hold the ankle in neutral.[5,6,32]

Terminal Swing.--Motion here requires the support of the ankle at neutral as the limb prepares for initial contact. Pretibial muscle activity increases to maintain the ankle at neutral to ensure optimal heel contact with the floor. The increased activity is likely in response to the inertia of the foot as the tibia is advancing. Terminal swing prepares the pretibial muscles for the upcoming demands of initial contact.[5,6,32]

Knee

The knee joint is one of the largest joints in the body and is the most complex.[5,26,32] Functionally, it is responsible for supporting and transferring weight and providing mobility for limb advancement during locomotor activities. Motion in the knee joint occurs in the sagittal, coronal, and transverse planes during gait. The largest motion takes place in the sagittal plane with smaller arcs in the coronal and transverse. Functional responsibility of the knee is closely tied to the ankle and hip through the activity of the several two joint muscles that control the knee.

Motions of the Knee

Sagittal motion measured at the junction between the femur and tibia is called knee flexion and extension. The knee passes through four alternating motions of flexion and extension during each gait cycle.[5,7,15,38,40] The full range of motion needed at the knee in normal walking falls within 0° to 60° of flexion.[5,26,15,38]

At initial contact the knee is positioned at approximately 0°-5° of flexion. During loading response the knee rapidly flexes to 15°- 20°.[5,6] As the weight of the body is shifted over to the stance limb, the flexed knee is under maximum weight bearing load. The knee begins to extend as the limb continues through midstance and into terminal stance. Heel rise brings the knee from an extended position to slightly flexed (approximately 7°).[5] Onset of double limb stance brings rapid knee flexion. At the end of preswing, knee flexion is at 40°, and continues through toe off and reaches its peak of 60° at initial swing.[5,6] During midswing the knee changes direction towards extension. Knee extension persists through terminal swing and reaches maximum extension just before the end of the swing period. Posture of the knee at the end of terminal swing is one of slight flexion (average 5°) as the limb prepares for ground contact.[5,7,15,17,38,40]

Muscle Activity of the Knee

Muscle activity is responsible for providing limb mobility and stability for walking. Fourteen muscles act to control the knee during gait. The design of the lower limbs allow for efficient use of two joint muscles to fire with minimal change in length and allows for intervals of inactivity for energy conservation. Each muscle reacts to the demands of the individual phases of gait.[5]

Knee extension is primarily controlled by the quadriceps. Three vasti heads (vastus intermedius, vastus medialis, vastus lateralis) of the quadriceps cross only the knee joint. The rectus femoris crosses both the knee and hip joint. Activity of the three vasti heads begin in terminal swing, continues through the early stance period, and rapidly decreases at midstance until activity ceases early in midstance.[5] The rectus femoris, in contrast, has a relatively short period of activity which lies between late preswing and early initial swing. Other authors[14,38] show rectus femoris activity to parallel that of the vasti heads. The upper gluteus maximus also assists in knee extension by way of its attachment to the iliotibial band (ITB). Contribution by this muscle begins in late terminal swing and ends by the middle of midstance.[5,8,35]

The popliteus and short head of the biceps femoris (BFSH) are two muscles that act directly on the knee to produce flexion. Activity of the politeus muscle has been reported to occur during midstance while contributing to the deceleration of knee extension.[38] Perry[5] found no consistent pattern of popliteus activity, except that it was used during all gait phases except initial and midswing. The FSH works during initial and midswing, with possible activity during terminal stance. The remainder of the hamstring muscles, the biceps femoris long head (BFSL), semitendinosis, semimembranosis help to flex the knee during mid and terminal swing. Between late loading response and the onset of preswing, the gastrocnemius helps to control the rate of knee flexion.[5,38,40] From the time of its onset, intensity of the gastrocnemius' activity increases until it peaks in terminal stance, where it begins to decline and ends at the start of preswing.[5,38,40] In swing, knee flexion is produced through two hip flexor muscles--the sartorius and gracilis. Muscle activity for both occur in initial and early midswing.[5,38]

Functions of the Knee

Three functional requirements are placed on the knee during human walking. In stance, the knee absorbs shock as weight is loaded on the limb and provides weight bearing stability in extension. Progressional mobility is provided during the swing period as the knee must rapidly flex to allow foot clearance. In normal gait, appropriate timing and intensity of muscle contraction provide effective responses to meet the functional demands placed on the lower extremities.[5,8,35]

Initial Contact.--Position of the knee at initial contact is nearly at full extension (approximately 5° of flexion). Muscle activity during terminal swing of the three vasti heads and the upper gluteus maximum/ITB which continue into initial contact, along with the GRFV bring aligned anterior to the knee, act together to create the extended knee posture. The knee is in a fixed position to accept the weight bearing load. Hamstrings work to protect the knee from hyperextending.[5,6,40]

Loading Response.--As body weight continues to be loaded onto the stance limb, the initial stability of the knee is disrupted momentarily. The knee is placed in a flexed posture to absorb the shock at limb loading. This shock absorbing mechanism is initiated by the heel rocker as the tibia is rolled forward over the foot and the GRFV is shifted posterior to the knee to create a flexor moment at the knee.[25] Concentric contraction by the hamstrings contribute to flexion motion. The vasti heads of the quadricep muscle work eccentially to control speed and range of knee flexion to approximately 15°.[6] Also, the heel rocker redirects some of the initial loading force into the quadricep muscles. Strength of the quadricep muscles active during loading response is essential to limb stability as the shock absorbing mechanism creates an unstable weight bearing posture at the knee.[5,6,8,32,35]

Midstance.--As the total body weight is transferred onto the flexed knee just after loading response, the quadriceps (vasti group) react to inhibit further knee flexion. A transition towards extension begins as the knee moves from 15° of flexion at the end of loading response to 5° flexion at the end of midstance.[5,6] Motion is made possible by several factors. Although not completely extended, stance stability is optimum in this position. Restraint and stabilization of the tibia by the soleus muscle allows the femur to advance faster than the tibia and minimizes the activity of the quads. As the contralateral limb advances ahead of the stance limb and the body mass moves forwards, the ankle dorsiflexes and the knee extends, bringing the GRFV closer to the knee joint. The vector passes anterior to the knee joint axis and creates an extensor moment. The vasti group become inactive at this point and the knee is stabilized by the passive extensor force and by the tricep surae muscles which restrains the tibia.[5,6,8,31,32,35]

Terminal Stance.--Terminal stance earmarks the completion of knee extension in the stance period. No muscles are actively maintaining knee extension at this point. Stability during terminal stance is a result of a combination of factors. Plantar flexors stabilize the tibia as the femur continues to advance. Momentum of the contralateral swing limb is maintained as the extremity moves further past the body's COG.[5] The body's forward fall over the stance limb is facilitated by the forefoot rocker which keeps the GRFV anterior to the knee joint axis. Knee extension is also provided indirectly through the tensor fascia latae (TFL), which places tension on the ITB, while restraining hip hyperextension. A potential for hyperextension exists with all the mechanisms working at the knee joint. However, the popliteus and gastrocnemius provide a flexor force posteriorly to prevent any strain.[5,31,32]

Towards the end of terminal stance, the knee begins a motion in the opposite direction. This is caused by the loss of tibial stability when the GRFV moves further ahead on the MT joints. Restraints on the tibia are lost, leading to ankle DF, which causes the heel to lift off the ground and the knee is flexed to a position of about 5°.[5] This unlocks the knee to provide the flexion required at initial swing.[5,6,31,32]

Preswing.--To prepare the limb for swing, a set of mechanisms are needed to provide adequate knee flexion. The GRFV moves to the end of the MTP joints and posterior to the knee joint to create a flexion torque.[5,31,32] Unopposed, the flexion torque leads to rapid flexion of the knee. The heel continues to rise and the tibia rolls forward freely. Minimal plantar flexor activity help to accelerate heelrise and tibial motion.[35] Direct knee flexion force is provided by the gastrocnemius and popliteus muscles. As weight is rapidly shifted to the opposite limb, the reference extremity is free to passively flex to 40° which will allow for easy toe clearance. Any excessive passive flexion (knee flexion which proceeds faster than limb unloading) is restrained by the rectus femoris.[5,6,8,31,32,35]

Initial Swing.--During initial swing, the limb is trailing behind the body with the knee flexed, leaving the foot in a toe down position. This posture adds length to the reference limb from the hip to the toe, and makes it functionally larger than normal standing distance from hip to ground in this position.[5] Ankle DF alone is not sufficient to completely clear the toe from the floor for limb advancement. Consequently, knee flexion becomes the critical motion required for this phase to further progression of the swing limb. To provide sufficient foot clearance the knee must be flexed to 60° in order for the limb to advance from its trailing position.[5,7] Achieving 60° of knee flexion depends on the completion of several mechanisms. First, knee flexion of 40° needs to be accomplished at preswing. After toe off, acceleration by the hip flexors generate energy to accelerate swing leg.[14] The advancing femur along with the inertia of the tibia produce knee flexion that is mainly passive.[18,32] The BFSH contributes to the knee motion by actively contracting. Minimal activity from the sartorius and gracilis can also aid in the knee and hip flexion, simultaneously. To avoid excessive hip flexion, the rectus femoris will pull the tibia forward and increase hip flexion at the same time.[5,7,14,26,31]

Midswing.--At this phase the foot has moved ahead of the hip joint and no longer is dependent on the position of the knee for foot clearance. Thus, knee motion into extension begins in order to complete limb advancement and prepare for floor contact. Using gravity as the primary force, the knee flexors relax to allow the tibia to advance from a trailing position to a vertical one.[5] Momentum created by the continuing hip flexion aids the force of gravity on the tibia. The knee quickly extends to approximately 25°. Lamont[32] and Norkin[26] report knee extension up to 30°. The BFSH may be active in controlling the rate of extension.[5,31]

Terminal Swing.--Functions of terminal swing include the completion of the step length and preparation for stance. The knee continues to extend as the quadriceps (vasti heads) actively lift the weight of the tibia against gravity. Since hip flexion at this point is undesirable, the rectus femoris is not used to extend the knee.[5] The hamstrings contract eccentrically to prevent excessive knee extension as well as the rate of tibial motion, and decelerates the forward motion of the extremity. At the end of terminal swing, knee extensors stabilize the knee for initial contact.[5,26,31,32,35,38,41]

Hip

The hip joint is formed by the union of the acetabulum of the pelvis and the head of the femur. As it is the link between the upper body and the lower extremities, its primary function is to support the weight of the HAT during static and dynamic activities. The hip provides a pathway for transmission of force between the pelvis and the lower extremities.[5,26]

Motions of the Hip

Motion of the hip during human walking is normally measured as the displacement of the femur from a vertical position. In the sagittal plane, the hip moves between two arcs of motion during the gait cycle. During stance the hip is in extension and flexes during swing. Anterior pelvic tilt of 10° in normal walking will increase hip motion in flexion and decrease it in extension.[5] Perry[5] reports the average range in normal hip motion during gait is 40°. Peaks in hip motion are 10° in extension and 30° in flexion. Position of the thigh at initial contact is 30°of flexion, and is held approximately through loading response. At the onset of midstance the hip begins to extend and reaches a neutral position (0°) about the time of heel off. The hip extends to its peak position of 10° at 50% of the gait cycle, at the end of terminal stance. Going into preswing the hip reverses direction towards flexion. Neutral position is reached at the end of the stance period. During initial swing the thigh flexes to 15°. Thigh position increases to 25° of flexion in midswing and holds this approximate position within 5° through terminal swing.[5,7,26,38]

Motion of the pelvis is influenced by the swing limb. Consequently, the hip moves in a small range of adduction and abduction in the coronal plane. At initial contact the hip is adducted to approximately 10°, then to 5° of adduction in loading response.[5]

The hip moves into a neutral posture during mid and terminal stance and settles in a relative abducted position in initial swing.[7,38]

The hip is at or near neutral in the transverse plane at initial contact.[5,6] Internal rotation proceeds and peaks at the end of loading response, as the contralateral limb unweights itself. Throughout the rest of the stance period the thigh externally rotates until it peaks at the end of preswing onset of initial swing brings on a change in direction as the hip medially rotates during the swing period to prepare for floor contact.[5,7,35]

Muscle Activity at the Hip

Muscles around the hip function to stabilize the pelvis on a moving base of support as well as to mobilize the hip. The pelvis also provides a stable base from which the trunk muscles control the forward motion of the trunk.[7] As an individual walks, muscle groups perform their primary functions throughout the phase of gait.[5]

Muscles acting to extend the hip joint include the hamstrings, adductor magnus and gluteus maximus. Hip extensor activity begins from late midswing through to loading response.[5] Components of the hamstrings involved in extending the hip joint, (semitendinosis, semimembranosis and BFLH) begin activity in late midswing. Peak intensity is reached in the early part of terminal swing, declines toward the end of the swing period through initial contact, and ceases activity during early loading response. The hamstrings remain inactive during the remainder of the gait cycle.[5,6,42] Activity of the adductor magnus beings toward the end of terminal swing. Intensity progressively increases through initial contact, but begins to decline in early loading response. Muscle action ceases near the end of loading response and throughout the rest of the cycle.[5,6]

The gluteus maximus is the primary, single joint hip extensor.[5] It contributes initially to stabilize the hip, then to hip extension.[38] Functionally, the muscle can be divided into upper and lower parts. The upper portion works as a hip abductor, the lower half as a hip extensor. At the start of terminal swing, muscle activity of the lower gluteus maximus begins. Intensity increases at initial contact, peaks during the middle of loading response, and declines to a point of inactivity at the end of loading response.[5,6,7,35]

Phasic activity of the hip abductors occur during the stance period of walking and function to stabilize the pelvis. The TFL, gluteus medius, gluteus minimus, and the upper portion of the gluteus maximus make up the abductor group.[5,6,43] Activity pattern of the gluteus minimus closely follows that of the gluteus medius.[5,38] Description of the muscle activity of the medius/minimus, will focus on the gluteus medius. Activity of the gluteus medius begins at the end of terminal swing. Intensity peaks immediately after initial contact and diminishes through midstance until activity ceases prior to initial double limb stance. The upper gluteus maximus also begins activity during terminal stance, peaking at loading response and continuing through midstance.[5,6,7,26,35,43]

The TFL, because of its postural advantage, is the primary hip abductor.[42,43] Muscle activity of the TFL is separated into an anterior and posterior portion. The anterior fibers are active in terminal stance, firing at a relatively low level of intensity. Posterior fiber activity is at a moderate level and begins at the onset of loading response and continues through terminal stance.[5,6,38]

Hip flexor muscles essentially control hip extension during toe off and contract concentrically to initiate swing. Muscular activity begins late in terminal stance, continues through initial swing to early midswing.[5] The adductor longus is the first to be activated in late terminal stance and remains active through initial swing. Brief activity from the rectus femoris follows from preswing to early initial swing. Gracilis, sartorius, iliopsoas muscles show similar activity patterns during initial swing. Hip flexor activity during midswing is inconsistent and there is no activity in terminal swing.[5,6,7,26]

Behavior of the hip adductors vary greatly among individuals and between researchers.[5,7,26] The adductor longus, adductor magnus and gracilis are the major adductor muscles that can be identified by EMG studies.[5] In addition to the function in the sagittal plane, the adductors act to keep the extremities near midline.[7]

Function of the Hip

Demands made on the hip during normal walking must be accommodated by muscular control specific for each phase of gait. Muscle activity around the hip stabilize the HAT over the lower extremities and initiates limb movement. Forces generated by the hip musculature can vary from 1.5 to 5 times body weight during walking.[32,38] An understanding of the coordinated interplay of motion, muscle function and force vectors will make clear the complexity of hip function in human walking.

Initial Contact.--With the hip flexed to 30°, the limb is positioned at a diagonal to the floor, and the forward momentum of the trunk creates an unstable posture at floor contact.[5] A potential for the heel to slide forward is present. However, stability is maintained by floor friction and the vertical force vector created by the loading of the limb.[5] Hip extensor muscle (gluteus maximus, hamstrings) action restrain the flexor moment of the GFRV as it is positioned anterior to the hip.[6,31,32,42]

Loading Response.--The task, placed on loading response is to maintain weight bearing stability while contending with a hip flexion posture of 30°, a flexor torque created by the anterior location of the GRFV, and the forward momentum of the body mass, all of which create an unstable situation. Stability is maintained by the hip extensors. The lower gluteus maximus and the adductor magnus provide direct response due to their single joint alignment at the hip. Stabilization of the trunk by the hip extensors also assists in limb stability. It provides a link between the femur and the pelvis to use the forward momentum of the body mass to indirectly draw the thigh back and thus extend the knee.[5,6,8,31]

Activity of the hip extensor muscles become dormant by the end of loading response as the force vector moves closer to the hip joint.[32,38] Passive hip extension begins as the vasti group of the quadriceps act to restrain knee flexion. This is made possible by the heel rocker that links the tibia to the foot. The quadriceps link the femur to the tibia which pulls the femur forward along with the advancing tibia.[5,6,8]

A rapid transfer of body weight moves the HAT forward and lateral unto the loading extremity. This creates a demand for active lateral stabilization of the pelvis over the hip.[5] An adduction torque created at the hip causes the contralateral pelvis to drop. The abductor muscles (gluteus medius, upper gluteus maximus, post. TFL) react quickly and intensely to meet the rapid unloading and high mechanical demand in the frontal plane.[5,6,26,38]

Midstance.--During midstance the hip travels from its flexed position towards extension. As the body mass moves forward over the supporting limb, the GFRV becomes aligned with the hip joint center early in the phase. By the end of midstance the force vector is posterior to the hip joint axis, thus allowing the extensor muscles to relax.[5] The hip abductors realign the pelvis to a neutral position in the coronal plane. Medial and lateral stability demands are diminished. The gluteus maximus ceases activity and the TFL begins to work to prevent excessive pelvic tilt as the opposite limb is unweighted.[5,7]

Terminal Stance.--The forefoot rocker continues forward progression of the body mass. The force vector is positioned posterior to the hip joint creating a hip extensor torque to keep the hip joint stable. Trailing the body, the stance limb is pulled into extension and then hyperextension. Controlling the rate and degree of passive extension is left to the TFL.[43] It also supplies the low level abductor force needed at the hip.[5,6,31]

Preswing.--This phase is called the interval of limb acceleration because the hip is moved quickly from extension to flexion.[5] Thigh motion in the sagittal plane returns the hip to a neutral posture. The rectus femoris provides hip flexion force while restraining knee flexion. Passive abduction is controlled by the adductor longus as weight is transferred to the opposite extremity.[5,31,32]

Initial Swing.--Recovery of the trailing limb generated by the momentum initiated during preswing is the primary action here.[5] Momentum created by the swinging limb helps to carry the body past the contralateral limb. Control of the swinging limb is maintained through the combined efforts of the gracilis, sartorius and rectus femoris. The gracilis provides adduction, internal rotation and hip flexion, while the sartorius responds to the abductor and external rotation forces about the hip and assists in flexing the hip. The rectus femoris corrects any excessive knee flexion to preserve the motion of hip flexion.[5,6,31,32,42]

Midswing.--The hip continues to flex but in an almost completely passive manner. Momentum is the primary force to advance the swing limb as hip flexor activity ceases. Activity of the hamstrings begin in midswing.[5,6,26,31]

Terminal Swing.--This phase of gait is the transitional period between swing and stance.[5,44] Hip flexion stops in order to prepare the limb for stance. In order for the reference extremity to be positioned optimally for initial contact, several mechanisms must occur. First, hamstrings act to restrain further hip flexion. Second, motion of the femur and tibia must be coordinated to obtain proper limb position. This is done by the hamstrings which simultaneously restrain the thigh while decelerating knee motion by controlling the influences of momentum and the pull of the quadriceps on the tibia. At the end of terminal swing hamstring activity is reduced and the gluteus maximus and adductor magnus being low level activity in preparation for their role in weight acceptance. Lastly, the gluteus medius counteracts the previous adductor motion initiated by the hip flexors.[5,6,31,32,40]

It is important for the physical therapist to be familiar with the normal ranges, movement patterns, and muscle activity required at each joint during normal gait. Tables 1 and 2 present the components associated with the foot, ankle, knee, and hip during the stance and swing periods. By understanding the mechanisms that produce a normal gait pattern, the therapist will be better equipped to identify any deviations.

Table 1--Gait Analysis and Stance Phase[5,6,28]

Phase	Joint	Motion	Moment	Muscle Activity
Initial Contact	Ankle/Foot	0° Neutral	Plantar Flexion	Pretibial muscles act to give support to the foot.
	Knee	0° - 5° Flexion	Flexion	Quadriceps contact to hold knee in extensions and to prepare limb for weight acceptance. Hamstrings eccentrically keep knee from hyperextending.
	Hip	30° Flexion	Flexion	Hip extensors contract to restrain flexion movement.
Loading Response	Ankle/Foot	10° Plantar Flexion	Plantar Flexion	Pretibial muscles contract eccentrically to control plantar flexion movement.
	Knee	15° - 20° Flexion	Flexion	Quadriceps work eccentrically to appose flexion movement and to control amount of flexion. Hamstrings contract concentrically to contribute to flexion movement.
	Hip	30° Flexion	Flexion	Gluteus maximus and adductor magnus act to stabilize hip joint and to oppose flexion movement. Hip extensor activity decreases and ceases by the end of LR.
Midstance	Ankle/Foot	5° - 10° Dorsiflexion	Dorsiflexion	Gastrocnemius and soleus contract eccentrically to slow the initial rate of DF and control rate of tibial advancement.

Table 1 (cont.)

Phase	Joint	Motion	Moment	Muscle Activity
	Hip	0° - 5° Flexion (Neutral)	Extension	Hip extensors are inactive. TFL contracts to prevent excessive pelvic tilt during single limb support.
Terminal Stance	Ankle/Foot	10° Dorsiflexion	Dorsiflexion	Triceps surae act to control DF movement and to stabilize ankle position as the tibia advances and the heel rises off the ground.
	Knee	5° Flexion to 0° Neutral	Extension	Quadriceps remain inactive.
	Hip	10° Hyperextension	Extension	TFL contract to control rate and degree of passive extension
Preswing	Ankle/Foot	20° Plantar Flexion	Dorsiflexion	Muscle activity of the gastrocnemius and soleus decreases. Plantar flexors provide just enough force to accelerate limb into swing phase. Tibialis anterior and toe extensors eccentrically control plantar flexion.
	Knee	40° Flexion	Flexion	Rectus femoris may act to restrain rapid passive knee flexion.
	Hip	10° Hyperextension to 0° Neutral	Extension	Rectus femoris contracts to provide hip flexion force. Adductor longus controls passive abduction.

Table 2--Gait Analysis: Swing Phase[5,6,28]

Phase	Joint	Motion	Moment	Muscle Activity
Initial Swing	Ankle/Foot	10° Plantar Flexion	None	Pretibials work to initiate dorsiflexion. Toe extensor muscles extend toes to assist with foot clearance.
	Knee	60° Flexion	None	Biceps femoris (short head), gracilis and sartorius contract concentrically.
	Hip	15° Flexion	None	Gracilis, sartorius and rectus femoris act to control swinging limb.
Midswing	Ankle/Foot	Neutral	None	Pretibial muscles and toe extensors contract to bring ankle to neutral and prevent the toes from dragging on the ground.
	Knee	25° Flexion	None	Little or no activity by the quadriceps. Biceps femoris (short head) may be active to control rate of extension.
	Hip	30° Flexion	None	Hip flexors inactive as momentum acts as primary force to flex hip. Hamstrings begin activity.
Terminal Swing	Ankle/Foot	Neutral	None	Activity of pretibial muscles increase to maintain ankle position.
	Knee	0° - 5° Neutral	None	Quadriceps contract concentrically to carry the weight of the fibia against gravity and to stabilize knee in extension in preparation for initial contact. Hamstrings work eccentrically to control rate of extension and to decelerate the forward motion of the swing limb.

Table 2 (cont.)

Phase	Joint	Motion	Moment	Muscle Activity
	Hip	30° Flexion	None	Hamstrings contract eccentrically to limit flexion gluteus maximus and adductor magnus begin activity in late part of phase to prepare limb for weight acceptance.

CHAPTER V

PATHOLOGICAL GAIT

To identify and evaluate gait problems properly, the physical therapist must understand what the problem is, where and when it presents, and why it occurs. In other words, the clinician must be able to compare what the patient presents with to the normal patterns of gait. Pathological gait mechanisms develop as a result of the loss of normal functioning of muscles and/or their coordination. From a functional perspective, gait pathologies can be categorized based on their appearance during the gait, cycle, and the segment at which they occur.[1] This chapter will describe common functional gait deviations that may be seen throughout a variety of pathologies, rather than describing them by specific diseases, and the mechanisms that cause them.

Foot

Premature Heel-Off

The inability to keep the heel in contact with the ground in any part of initial contact, loading response and midstance is considered abnormal. Premature heel rise is a subtle deviation when it occurs during loading response and midstance and is often overlooked.[5] Premature heel-off during stance is attributable to either excessive PF or excessive knee flexion.[5,7,20,28] Early heel rise also may occur as a voluntary mechanism to gain more height (by excessive PF) for a short limb to provide floor clearance for the opposite swing limb.[5]

Delayed Heel-Off

Prolonged heel contact with the floor during terminal and preswing is an indication of either PF weakness or excessive PF.[5,6] Delay of heel rise is significant in terminal stance because it interferes with progression of the body over the forefoot. It also shortens the step length of the contralateral extremity.[5,6,7,28]

Foot Flat Contact

This gait deviation is identified by the simultaneous contact of the forefoot and heel with the ground.[5,6,20] It is caused by weak or flaccid ankle dorsiflexors which places the knee in a flexed posture. The foot flat contact provides an immediate base of support but adequate quadricep muscle strength is necessary to control the flexed knee.[5] No heel rocker is present which decreases the forward momentum of the tibia, as well as the limb's ability to absorb the shock of floor contact.[5,6,7,20,26]

Forefoot Contact

Forefoot contact results from a combination of excessive ankle PF and knee flexion which causes the toes to make initial contact instead of the heel.[5] It reduces the forward momentum of the tibia and decreases the shock absorption capacity of the extremity.[1,5,26,28]

Foot Slap

A week tibialis anterior allows the foot to fall to the ground in an uncontrolled manner immediately following initial contact. Shock absorption is reduced as knee flexion is limited and the advancement of the tibia is decreased.[5,6,28]

Ankle

Excessive Plantar Flexion

Excessive ankle PF results in a loss of forward progression as the tibia is restricted from advancing over the ankle and forefoot. Step length is reduced as well as gait velocity. PF limits the knee from flexing, thereby affecting the ability of the limb to absorb the impact of floor contact.[5] During swing, the plantar flexed ankle interferes with foot clearance and with the foot position for initial contact.[5,7,20,32]

Causes of excessive PF include pretibial muscle weakness, soleus and gastrocnemius spasticity, and voluntary excessive ankle PF.[5,7] Activity of the pretibial muscles during limb loading controls the PF moment at the ankle. The inability to produce a sufficient DF force allows the foot to drop uncontrolled. If only the tibialis anterior is unable to function, the foot will drop on the medial side.[5] Inadequate activity by the tibialis anterior results in the alteration of the heel rocker during initial contact and loading response, and impedes foot clearance in midswing (toe drag).[5,26,16]

An extensor synergy pattern can produce excessive activity of the triceps surae muscles which results in a plantar flexed ankle posture that affects the gait pattern from initial contact to preswing.[5] The extensor pattern begins during terminal swing when the quadricep muscles start to extend the knee to prepare the extremity for floor contact. Synergistic activation of the gastrocnemius and soleus cause the ankle to move from a dorsiflexed to a plantar flexed position.[26] However, during initial and midswing, activation of the dorsiflexors positions the ankle near neutral to aid in foot clearance. This reversal of ankle motion is what differentiates an extensor pattern from a plantar flexion contracture.[5]

Voluntary excessive ankle PF can be used by a patient as a means to reduce the impact of the loading response when weak quadricep muscles are unable to restrain the flexion movement at the knee. The ankle is placed in a plantar flexed posture of about 10° just before initial contact.[5] This causes a low heel strike (shortened period of isolated heel contact) that reduces the effect of the heel rocker. The soleus restrains the tibia from advancing during stance and keeps the knee in extension throughout preswing. Consequently, heel contact is maintained and peak DF occurs in late preswing instead of terminal swing.[5,7]

Excessive Dorsiflexion

A gait pattern characterized by ankle DF beyond neutral is considered as abnormal for all phases of gait except midstance and terminal stance.[5] Excessive DF also indicates conditions where normal plantar flexion is lacking and is functionally more significant in stance than swing.[5,20] Soleus weakness can cause excessive DF resulting in a loss of tibial stability and causes an abnormal flexed knee posture. This in turn places a greater demand on the quadriceps to stabilize the knee during the stance period.[5,28] In midstance, the tibia is allowed to rapidly advance over the foot creating an abnormal amount of ankle DF. The alignment of the tibia promotes knee flexion and the increased support needed by the quadriceps. Difficulty in stabilizing the ankle and knee results in a shortened period of single limb support. Step length becomes shorter than normal because the normal toe-off segment at the end of stance is lost. Soleus weakness also results in the loss of heel rise in terminal stance as knee extension is replaced by knee flexion.[5] If an individual has normal strength of the quadriceps muscles, the individual will not try to compensate for the weakness. Instead, the patient will walk with the knee in flexion.[5,6,7,26,28]

Knee

Limited Flexion and Excessive Extension at Knee

The interchanging pattern between knee flexion and extension provide for a smooth progression throughout the gait cycle. Because of this relationship, pathology can influence both motion, as limited knee flexion leads to excessive knee extension. Lack of knee flexion during loading response, preswing, initial swing, and midswing has significant impact in human walking. Differing pathologies are responsible for the lack of flexion in stance and in swing, and for the differences in substitution patterns.[5] In loading response, weak quadricep muscles are unable to restrain the flexing knee. An extended knee posture is substituted to compensate for the instability in weight bearing at the knee. As a result, shock absorption and forward momentum of the tibia are reduced.[5,6] Also, there is potential for trauma to occur to the posterior knee capsule with repeated hyperextension.

Two mechanisms can prevent knee flexion in loading response. One is hip extension caused by the gluteus maximus and adductor longus. The other is by the soleus muscle causing a premature ankle PF to block the advancement of the tibia and to limit rocker action. Inability to adequately flex the knee during preswing does not allow for proper transition from stance to swing to occur.[5] The ankle is excessively dorsiflexed and the heel maintains floor contact which makes toe-off difficult to achieve. The knee remains in an extended posture until initial swing begins as compensation for weakness of the quadriceps.[5,28] Inadequate knee flexion in initial swing causes the toe to drag on the ground and blocks limb advancement. Without sufficient knee flexion, the length of the swing limb becomes relatively longer than the contralateral stance extremity, thus causing the toe to drag along the floor. Limited knee flexion during midswing is perpetuated from the deviation in initial swing.[5,6,7,26,28]

Knee hyperextension during midstance and terminal stance provides stability to an otherwise unstable knee during weight bearing. Activity by the soleus and an extensor movement at the knee maintains the extended posture. Heel contact is prolonged through terminal stance for optimum foot support.[5]

Limited Extension and Excessive Flexion at the Knee

Inadequate knee extension is commonly seen in three phases of gait: midstance, terminal stance, and terminal swing.[5] In midstance, a reversal in direction from knee flexion to knee extension does not occur. In terminal stance knee extension to or near neutral, is not achieved. Both deviation normally stem from the increased flexion occurring in loading response, reduces single limb stability and increases the demand on the quadriceps.[5,6,32] Inability of the knee to achieve an extended posture during terminal stance leaves the limb unprepared for initial contact and decreases the step length of the reference limb.[5,6,26,28,32]

Excessive flexion can occur during the loading response and midswing phases of gait. During loading response, the knee flexes more than 25° and places an increased demand on the quadricep muscles to stabilize the knee.[5,20] An increase in hip flexion normally account for the excessive knee flexion during midswing.[5,6,20,28]

Hip

Limited Extension and Excessive Flexion of Hip

Limitations in hip extension obstructs or delays forward progression, decreases step length, and makes for an unstable posture during the weight bearing phases of gait such as midstance and terminal stance.[5] A lack of hip extension during midstance causes a greater anterior pelvic tilt and brings the trunk forward in a flexed position ahead of the hips.[20] This places the body vector anterior to the hip joint which increases the demand on the hip extensors because of the flexion movement. The trunk compensates to counteract the flexion movement by increasing its lumbar lordosis in order to bring the HAT back over the hips.[5] Another compensating posture commonly seen with limited hip extension is flexed knees. This allows the pelvis to keep its normal alignment. However, this crouched posture places greater demands on the quadricep muscles, and requires an increase in ankle DF or heel rise to phase into terminal stance. Anterior pelvic tilt along with the lumbar lordosis is perpetuated in terminal stance when inadequate hip extension is present. This reduces the normal position of the trailing limb, deters body advancement and shorts the step length.[5,6,20]

Excessive hip flexion is most significant in preswing, initial swing, and midswing.[5] Limited hip extension through stance fosters a flexed hip posture as transition into preswing is made and can continue into initial swing. In midswing, an increase in hip flexion is often used to intentionally clear the foot when knee flexion is inadequate, plantar flexion is excessive or the swing limb is longer than the stance limb.[5,6,28]

Common pathologies such as spastic hip flexors, flexion contractures, or pain can either restrict hip extension or promote hip flexion. Shortening of the flexor muscles, joint capsule, or ITB are common causes of limited extension at the hip. Hip flexor spasticity is the result of upper motor neuron lesions which can enhance the stretch reflex in these muscles. Pain can be induced through conditions like arthritis, which cause inflammation in the hip joint. Pain is greatest when tension is placed on the hip joint, especially during single limb support. Increased hip flexion is used to reduce the pressure within the joint. As a result, duration of single limb support is shortened as well as step length.[5]

Limited Hip Flexion

Limited hip flexion due to pathology can become evident during initial swing through initial contact. Inadequate hip flexion (less than 15°) during initial swing contributes toe drag and ankle PF, and consequently, hinders limb advancement.[5,6] The lack of hip flexion in initial swing will commonly remain throughout midswing and initial contact, resulting in a shorter step length. Normal loading response can be disrupted if knee flexion and ankle PF are limited.[5,6,28]

Muscle weakness is the primary reasons why adequate hip flexion is not achieved throughout the various phases of gait. Loss of speed and proper range is a product of weak or flaccid hip flexors. With upper motor neuron lesions, flexor patterns may assist the patient in gaining necessary hip flexion to advance the limb during initial and midswing.[5]

To accommodate for the lack of hip flexion, several substitution patterns are commonly used to clear the ground for limb advancement. The limb can be circumducted by hiking the ipsilateral pelvis up and rotating it forward, while the hip is in abduction. By bringing the knee to an excessively flexed position, the patient can indirectly flex the hip to advance the limb. Other substitution patterns include vaulting of the contralateral limb and lateral lean of the truck toward the opposite side.[5]

Excessive Hip Adduction

Increase in hip adduction during the stance period of gait can be a result of weak hip abductors and/or spasticity or contracture of the adductors.[5] All of which will produce a pelvic drop on the contralateral side. Excessive adduction can increase the relative length of the limb and obstruct foot clearance, it will decrease the base of support, and jeopardize limb stability.[5,6]

Abductor weakness is evident during single limb support. As the swing limb is lifted, activity of the hip abductors (gluteus medius) on the stance leg is unable to control the pelvic drop on the swing side.[5] Excessive fall of the pelvic and trunk continues throughout stance until the swing limb makes ground contact and the body weight can be transferred to the new stance limb. Deviations created by spastic or contractured adductors are present throughout the gait cycle.[5,6,26,28]

Excessive adduction in swing causes the swing limb to cross over medially as it is being advanced. This deviation is termed as a "scissor gait."[5] If severe enough, the swing limb may cross midline and make ground contact in a position that obstructs the forward swing of the other limb. Adductor muscles acting as primary hip flexors in the absence of adequate iliacus activity, produce the "scissor gait." Spastic adductors, influenced by the stretch reflex created by the weight of the swing limb, can also produce the same deviation.[5,6]

Excessive Hip Adduction

Excessive abduction of the hip during stance increases the base of support and reduces the relative length of the limb in swing.[5] Contracture of the hip abductors displaces the stance limb laterally and causes an ipsilateral drop of the pelvis. Adductor weakness or a tight ITB can also cause the excessive abduction posture. However, with ITB tightness, hip abduction accompanies hip extension and a neutral position may be achieved with the hip in flexion. During the swing period, circumduction is used as a means to advance the limb in the absence of adequate hip flexion.[5,28]

Tables 3 through 5 describe some common deviations found in pathological gait. Possible compensations and causes as well as an analysis are presented as a reference for the reader to use. The next chapter will present an opportunity to use the information from this literature review to perform an actual gait analysis.

Table 3--Common Deviations: Ankle and Foot[5,6,28]

Phase	Deviation	Description	Possible Compensations	Possible Causes	Analysis
Initial Contact	Foot Slap	At heel strike, the foot falls uncontrolled to the floor.	To avoid foot slap, the foot may be placed flat on the ground or the toes may be used for initial contact.	Flaccid or weak dorsiflexors.	Look for low muscle tone at ankle. Look for increased hip and knee flexion during swing.
	Toe First	Toes contact ground instead of heel. Plantar flexed posture may be maintained throughout stance, or heel may contact the floor.		Leg length discrepancy; contract heel cord; PF contraction; spasticity of plantar flexors; flaccid dorsiflexors; painful heel.	Compare leg lengths; check for hip and/or knee flexion contractures. Look at muscle tone and timing of activity of plantar flexors. Check for heel pain.
	Foot Flat	Entire foot contacts ground.		Excessive fixed dorsiflexion; flaccid or weak dorsiflexors.	Check ROM at ankle. Check for hyperextension at the knee.
Midstance	Excessive Plantar Flexion	Tibia does not advance to neutral from 10° plantar flexion in LR.		Flaccid or weak dorsiflexors; spastics soleus and gastrocnemius; weak quadriceps.	Check for weak quadriceps, hyperextension at the knee or hip; backward or forward leaning of the trunk. Check for dorsiflexor weakness.

Table 3--Common Deviations: Ankle and Foot[5,6,28] (Cont.)

Phase	Deviation	Description	Possible Compensations	Possible Causes	Analysis
	Excessive Dorsiflexion	Tibia advances too rapidly over the foot causing an abnormal flexed knee posture.	Ankle may be maintained in plantar flexion. If the foot is flat on the floor, the dorsiflexion movement is eliminated and a 'step to' gait is produced.	Inability of plantar flexors to control advancement of tibia. Knee or hip flexion contractures.	Check strength of ankle muscles, knee and hip flexors, ROM, and trunk position.
	Premature Heel-Off	Heel does not keep contact with ground.		Spasticity of plantar flexors; excessive hip flexion.	Check for spasticity of plantar flexors, quadriceps, hip flexors and adductors.
Terminal Swing to Preswing	Delayed Heel Rise	Insufficient transfer of body weight from hind foot to the fore foot.	Whole foot is lifted off the ground.	Flaccid or weak plantar flexors; excessive plantar flexion; pain in forefoot.	Check ROM at ankle and foot. Check muscle function and tone at muscles controlling the ankle.
Swing	Toe-Drag	Insufficient dorsiflexion and toe extension. Fore foot unable to clear ground for advancement.	Hip and knee flexion may be increased to prevent toe drag. The swing leg may be circumducted or hip hiked. Vaulting may occur with the contralateral limb.	Flaccid or weak dorsiflexors and toe extensors. Inadequate knee or hip flexion. Spasticity of plantar flexors.	Check ROM of ankle, knee, and hip. Check muscle tone at ankle, knee and hip.

Table 4--Common Deviations: Knee[5,6,28]

Phase	Deviation	Description	Possible Compensations	Possible Causes	Analysis
Initial Contact to Loading Response	Excessive Flexion	Knee flexes or "buckles" rather than extend as foot makes contact with ground.	Plantar flexion at ankle so that entire foot makes ground contact instead of the heel. Plantar flexion eliminates the flexion movement.	Spastic knee flexors or weak or flaccid quadriceps. Short length of contralateral limb.	Check for pain in knee; tone of knee flexors; strength of knee extensors; leg lengths; anterior pelvic tilt.
Midstance	Excessive Extension	Tibia remains behind ankle joint during single limb support, as the body weight moves over the foot.	Ankle plantar flexion or backward lean of trunk	Flaccid or weak quadriceps. Gluteus maximus extends hip or soleus muscle causes premature ankle plantar flexion.	Check for tone and strength of knee and ankle flexors, and ROM of ankle.
Terminal Stance to Preswing	Limited Knee Flexion	Less than normal amount of knee flexion (40°).		Spastic or overactive quadriceps and/or plantar flexors.	Check tone in hip, knee, and ankle muscles.
Initial Swing to Midswing	Excessive Flexion	Knee flexes more than 60°.		Decreased knee flexion in preswing.	Check tone in hip, knee, and ankle muscles.
	Limited Flexion	Knee does not flex to 60°.	Increased hip flexion, circumduction or hiking.	Pain in knee, decreased ROM of knee, extensor spasticity.	Assess for knee pain and knee ROM. Check muscle tone at knee and hip.

Table 5--Common Deviations: Hip[5,6,28]

Phase	Deviation	Description	Possible Compensations	Possible Causes	Analysis
Initial Contact	Excessive Flexion	Flexion exceeding 30°.	Increased lumbar lordosis to prevent excessive hip flexion and to eliminate hip flexor moment.	Hip and/or knee flexion contractures. Knee flexion caused by weak soleus and quadriceps.	Check hip and knee ROM and strength of soleus and quadriceps.
	Limited Flexion	Hip flexion less than 30°.	Increased knee flexion, circumduction, vaulting on the contralateral leg, and lateral lean of trunk toward opposite side.	Weakness of hip flexors or gluteus maximus. Limited ROM of hip.	Check strength of hip flexors and extensors, and hip ROM.
Midstance	Limited Extension	The hip does not attain neutral position. Trunk flexed forward ahead of hips.	Increased lumbar lordosis to lean trunk backwards. Knees placed in flexed posture.	Hip flexion contracture, spasticity in hip flexors. Painful hip.	Check ROM of hip and tone of hip muscles. Assess for hip pain.
	Excessive Adduction	Hip abductors or stance limb unable to control pelvic tilt on side of contralateral swing limb.		Weak hip abductors, primarily gluteus medius, on side of stance limb. Spasticity or contractures of adductors.	Check strength of hip abductors, tone of hip muscles, and hip ROM.

Table 5--Common Deviations: Hip[5,6,28] (Cont.)

Phase	Deviation	Description	Possible Compensations	Possible Causes	Analysis
	Excessive Abduction	Stance leg is postured in an abducted position.		Contracture of hip abductors; weak adductors; or ITB tightness.	Check for tone of hip abductors and ITB and strengthening of adductors.
Swing	Circumduction	A lateral circular motion of the entire limb to advance the swing leg.		Used to compensate for weak hip flexors or the inability to shorten the leg to clear the ground.	Check strength of hip and knee flexors and ankle dorsiflexors. Check for extensor pattern and ROM of hip, knee, and ankle.
	Hip Hiking	Shortening of the swing limb by contraction of the quadratus lumborum.		Used to compensate for inadequate knee flexion or ankle dorsiflexion. Also, to compensate for extensor pattern of swing leg.	Check strength and ROM of hip, knee, and ankle. Check muscle tone at knee and ankle.
	Excessive Flexion	Hip flexion greater than 30°.		Voluntary hip flexion to shorten limb due to a foot drop. Flexor pattern.	Check ROM and strength of ankle and foot. Check for flexor pattern.
	Limited Flexion	Hip flexion less than 30°.		Weak hip flexors; limited hip ROM; hip pain; hypertonic hip extensors.	Check strength and ROM of hip. Check tone of hip extensors and assess for hip pain.

Table 5--Common Deviations: Hip[5,6,28] (Cont.)

Phase	Deviation	Description	Possible Compensations	Possible Causes	Analysis
Phase Swing	Excessive Adduction	Swing limb crosses medially as it is being advances--scissor gait.		Adductor muscles acting in place of primary hip flexors spasticity of hip adductors.	Check strength of hip flexors and tone of adductors.

CHAPTER VI

CASE STUDY

Injury to the spinal cord presents profound ramifications for an individual. Tremendous challenges that affect every aspect of his or her life must be met and dealt with. The role of a physical therapist is to help facilitate the individual's return to as high of a functional, independent, and fulfilling lifestyle as possible. With the proper knowledge to train the person in the skills that will be needed, as well as an understanding of the issues related to the disability, a therapist will be more effective in assisting the patient through the rehabilitation process.

One of the issues that is of great importance to the spinal cord injured person is walking. The ability to walk gives a greater sense of one's independence and functional role in society.[45] A patients' prognosis for ambulation after spinal cord injury (SCI) is dependent on the level and completeness of the injury and the strength in the lower extremities.[46] Incomplete injuries to the spinal cord can spare motor pathways below the level of injury and provide enough control and strength for ambulation. However, the potential quality and degree to which the individual can walk can be affected by the patient's level of conditioning, conditioning potential, motivation, presence of spasticity, muscle weakness and coactivation.[46,47]

This case study is an example of an individual with an incomplete SCI who achieved wheelchair independence and was discharged functionally as a home ambulator. He received treatment as an outpatient at the Rehab Hospital in

Grand Forks, North Dakota, for fourteen weeks. A video recording of the patient's gait pattern was taped at the eleventh week, post operatively, and every two weeks thereafter, up until his discharge. The video tape will be used as a tool in conjunction with the case study and literature review to describe basic gait pathologies that can be found in persons with SCI.

For the purpose of this case study, five basic gait deviations will be used to observe, analyze, and monitor the progress of the patient in his ambulatory skills. These are: 1) foot contact during initial contact, 2) excessive knee extension in midstance, 3) delayed heel rise during terminal stance, 4) limited hip flexion and/or excessive knee flexion in swing, and 5) limited knee extension at terminal swing.

The patient was a 30 year old male who worked as a detention officer in Montana. On June 28, 1996, he suffered an acute flexion injury to his thoracic spine during a rodeo accident. He was pinned against a rail in a Brahma bull chute by his horse and immediately lost the feeling in his legs. He was taken to a hospital in Billings, Montana, where he was diagnosed with a T_{12}-L_1 fracture and incomplete paraplegia. The patient underwent Harrington Rod placement, fusion from T_{10}-L_2, and placed in a body jacket that same day. His post-operative course was unremarkable. Past medical history of the patient showed a L_5-S_1 fusion for spondylolisthesis in 1983. Otherwise, no significant past medical history.

A month later, the patient was referred to the Rehab Hospital where he was evaluated on July 29, 1996. The evaluation described the patient to be well muscled, but he had lost a considerable amount of weight since the accident. Neurological assessment showed diminished sensation to light touch at L_2-L_5 for both lower extremities and an absence of sensation at S_1, and below. Proprioception was absent, bilaterally, at the ankles, but present at the knee and hip joints. Presence of spasms in the bilateral triceps surae was reported by the patient. Passive range of motion was limited to 10° and 5° of DF in the right and left ankles, respectively. Straight leg raises (SLR) was limited to 45° on the right and 35° on the left because of hamstring tightness. Hip internal rotation was significantly limited bilaterally. Strength assessment of the lower extremities showed a severe decrease in strength, left greater than the right (Table 6). The patient was at a non-ambulatory status. Standing in the parallel bars required maximum assistance to keep both knees from buckling because of the weak muscles because lower extremities had very little weight bearing capacity, most of his support came through his arms.

Table 6.--Lower Extremity Strength Assessment

	Admission		Discharge	
	R	L	R	L
Hip Flexion	4	1	4	2-
abduction	1+	1	2	2
adduction	3+		4	2
IR	NT	NT	2-	4
ER	NT	NT	4	3
extension	NT	NT	1	2+
Knee Flexion	3+	1	3+	3-
extension	4	1	5	2
IR	NT	NT	4	3+/4
ER	NT	NT	4	3+/4
Ankle DF	0	2	1	2
PF	0	3	1	4
Long Toe Extension	1	3	0	3

0 = Total paralysis
1 = Palpable contraction
2 = Active movement with gravity eliminated
3 = Active movement against gravity
4 = Active movement against moderate resistance
5 = Active movement against full resistance
NT = Not tested

In terms of gait, the problems the patient encountered involved his weak ankle dorsiflexors which produced an excessive PF or toe drag during swing. Weak plantar flexors on the right gave no stability to the ankle, resulting in tibial instability during stance which caused the knee to flex more than normal and placed added demand on an already weakened quadricep muscle. On the left leg, the weak quadriceps required excessive knee extension during loading response and preswing to compensate for the instability at the knee. Limited left hip flexion due to the weak hip flexors contributed to toe drag and hindered limb advancement. Also, the body jacket which provides stability to the trunk and allows for early mobility, limited his motion in all planes of the thoracic and lumbar spine.

His treatment plan included gait training, lower and upper extremity strength training, and stretching, fitting for orthotics, and functional wheelchair training. The patient was reported to be cooperative and highly motivated to work with physical therapy. During the subsequent weeks of rehabilitation, the patient began pool therapy where he learned to advance his left leg while walking in the parallel bars. He progressed to gait training in the parallel bars in the PT gym and then to a wheeled platform walker with moderate assistance of two people. He continued to make functional gains in the strength of his lower extremities and the weight bearing tolerance in his left leg improved as well. As a result, he began to use a front wheeled walker (FWW) in gait training at minimal assistance of one person with ace wrap used to position his ankles into DF because of the weakness of his ankle dorsiflexors.

At the time of the first videotaping, the patient was ambulating for 250 feet with the FWW at standby assistance (SBA). However, he still required a significant amount of upper extremity support as his legs were not capable of supporting his body weight. He had been fitted for a rigid ankle foot orthosis (AFO) on the right leg and was using a toe-strap on the left. He had just begun to gait train with forearm crutches for 50 feet and required moderate assistance of two people. The following is a description of the patient's gait pattern as observed during the video recordings. At each session, the patient was asked to ambulate using the FWW, with and without his foot orthoses, and the forearm crutches, with the foot orthoses. The video demonstrates the gait deviations that arose as a result of his injury to his spinal cord. The gait pathologies posed a problem for the patient as he worked to gain greater independence and a more efficient gait.

Week Eleven Post Op

The patient ambulated with a FWW without the use of the AFO or toe strap. As a result he had difficulty controlling his ankles during swing and made foot flat contact at initial contact. Toe drag was present due to weak dorsiflexors (less than poor grade) which forced him to increase his knee and hip flexion to clear his foot during swing (right > left). In midstance, the patient used greater knee extension to stabilize the limb because of the weak quadriceps (less than poor grade). Weak ankle PF contributed to the delay heel rise during terminal stance and the patient had difficulty extending his knee and flexing his hip during terminal swing. All of which resulted in a shortened step length.

Next, the patient ambulated with the rigid AFO on the right leg and the toe strap on the left leg using the FWW and later, the forearm crutches. All gait deviations were present except the AFO and toe strap stabilized and positioned the ankles in DF to allow foot clearance during swing and allowed for heel strike at initial contact.

Balance and stability were an issue when he ambulated with the forearm crutches and he required moderate assistance of two people. Because of the high energy expenditure associated with the four point gait, the patient required a rest period after ambulating approximately fifty feet. Walking with the FWW provided more stability for the patient.

Week Thirteen Post Op

The patient was ambulating with the forearm crutches for 250 feet with moderate assistance of two people. He was fitted with a leaf spring AFO for his left leg. He continued to make functional strength gains, increased the ROM of his SLR to 78° and 67° for the left and right legs, respectively. Gait deviations were present while patient ambulated with the FWW and AFO's. The left AFO stabilized his ankle in DF, but it forced the knee into a flexed posture. His left quadricep muscle was not strong enough to control and stabilize the knee joint. Consequently, the left knee would keep buckling unless he consciously put the effort to extend it to keep himself upright during the stance period. His gait became more static because he had to concentrate on keeping his left knee extended during each gait cycle. Also, the patient appeared to be using pelvic rotation and momentum to advance his left leg from preswing to terminal swing, in order to compensate for weak hip flexors and knee extensors.

Week Fifteen Post Op

The patient was now ambulating with the forearm crutches and AFO's for 500 feet with supervision. His body jacket was discontinued and the left AFO was trimmed to give greater flexibility. He began gait training with a wide base quad cane (WBQC) and was able to ambulate 50 feet with moderate assistance of two people. The patient continued to make small gains in his lower extremity strength and ambulation skills. Gait was slow and unsteady and the gait deviations were more pronounced when he used the quad cane to walk. Using the forearm crutches, he exhibited improvement in his step length and the duration of heel contact increased. When he ambulated with the FWW, he demonstrated a smoother gait pattern and relied less on his upper extremities for support and balance. The pattern of "kicking out" his left leg to advance it along with knee extension in midstance, delayed heel rise, and limited knee extension at terminal swing were still present. The patient ambulated using the FWW, without the AFO's. Lack of ankle control, toe drag, foot flat contact, increased knee/hip flexion in swing were present.

Week Seventeen Post Op

This was the last video session before the patient's discharge. Gait with the bilateral AFO's and forearm crutches showed marked improvement from the first videotaping. The transition from one gait phase to the next was more fluid than in the past. Heel strike was observable and consistent. There was less dependence on stabilizing the knee in extension during midstance (right leg more so than the left), delayed heel rise in terminal stance was not as obvious, and hip flexion in swing and knee extension in terminal swing improved in both lower limbs.

At discharge, the patient was able to static stand in an unsupported position for two minutes. He was able to ascend and descend a flight of stairs, with the use of the bilateral railings, with close supervision. Strength in both lower extremities improved somewhat (Table 6) and there was no noticeable increase in the tone of his lower extremities.

Discussion

Knowledge of the level of the SCI and the degree of any existing spasticity will enable the clinician to anticipate gait abnormalities in patients with SCI's who have the ability to ambulate.[46] This knowledge, along with an accurate assessment and an understanding of the needs and goals of the patient will help the physical therapist prescribe the proper assistive devices that will enable the SCI person the best opportunity to ambulate in the most efficient manner possible. The use of an AFO is primarily to control ankle motion by assisting DF or PF. The posterior leaf spring AFO assist ankle DF. It provides more motion and assists to lift the foot during the swing period for ground clearance.[48] The leaf spring AFO was used on this patient's left ankle because he had fair grade in his plantar flexors, but a poor grade for his dorsiflexors. A more rigid AFO that restricts ankle motion in the transverse plane as well as in DF and PF was used on the patients right leg because of his flaccid or weak ankle muscles.[28,49]

Walking with crutches is a difficult task for paraplegics. The energy cost to ambulate for such individuals is considerable.[47,50,51,45] Alternate four point gait has a phasic pattern and allows the individual to feel he or she is walking "normally." It is a relatively slow gait and has been described as the most difficult of the paraplegic gaits.[45]

The patient's upper body strength, motivation and improvement in his leg strength gave him the ability to perform a reciprocal gait using the forearm crutches. However, without adequate strength in the left quadriceps muscles, the patient will continue to have problems with the stability of the knee during stance. Because the AFO is designed to stabilize the ankle and not the knee, he will need to compensate by actively keeping the knee in an extended posture. Without improvement in his quadriceps strength he will not achieve a complete status as a community ambulator, thus requiring dependency on the wheelchair for long distances. The patient was discharged on October 31, 1996, to his home in Montana. He will continue outpatient therapy there to work on strengthening and gait training.

Video recording the gait pattern of a SCI person can be a useful tool for the physical therapist to objectively monitor the progress, or lack of, in the individual's rehabilitation process. It can also be used as motivation for the patient as he or she observes the improvements. With proper guidance from the rehabilitation team an individual with a SCI, who has retained the ability to ambulate, will, in many cases, continue to gain strength, increase the capacity to exercise and improve gait performance.[50] This was evident in the case study presented here.

CHAPTER VII

CONCLUSION

Gait assessment has become an important and useful tool for patient evaluations in physical therapy. It is used to identify deviations in a patient's gait by comparing it to "normal" gait, to determine the degree of abnormality, to identify the causes of the abnormal gait pattern, and as a tool to reassess and evaluate the efficacy of treatment.[2,4,24,52] Because most physical therapy clinics do not have the resources or the personnel to maintain a full diagnostic lab, careful observation by trained observers is the most convenient way to analyze gait.[22,27]

Recently, video recordings have been used in addition to observational gait analysis. Video allows adequate analysis of joint motion through its play back and stop frame capabilities. Videotaping allows the therapist to view gait patterns repeatedly without fatiguing the patient.[27,52]

However, gait assessment like most clinical measurement is subjective and open to observer error. Measurement error can be caused by poor observation training and skills, personal biases, and a limited capacity of human perception. The difficulty in observing gait is the observer's task of objectively trying to differentiate deviation from the "normal" pattern through rapidly changing motions in a three dimensional object.[2,22,27] Even with the use of a video recording of patients with gait pathology, interrater reliability in observational gait analysis was shown to be low to moderate.[27,52] Despite these flaws of the system, there is great potential in observational gait analysis. Future research is needed to develop and improve techniques that will be convenient and more reliable in clinical settings.

This literature review of normal and pathological gait is intended to assist the reader in understanding the complex mechanisms involved in human walking. With the numerous methods and technologies used today, this review is not meant to be an extensive profile of gait analysis. Instead, the aim is to provide a basic foundation and understanding of the challenges involved in gait analysis. Considerable training and constant practice are necessary to develop observational skills required to perform any observational gait analysis.

Since a large percentage of patients seen for physical therapy have some sort of gait deviation,[2] perhaps a greater emphasis might be placed in academic course work in teaching about normal as well as pathological gait. Gait analysis, along with sound clinical judgment, are important in determining factors involved in pathological gait and the subsequent selection of appropriate treatment.

APPENDIX A

Observational Gait Analysis

Patient's Name________________________ Date________________________

Age________________ Sex__________ Therapist________________________

Diagnosis________________________ Assistive Devices________________________

		STANCE										SWING							
		IC		LR		MSt		TSt		PSw		ISw		MSw		TSw		POSSIBLE CAUSES	ANALYSIS
JOINT	DEVIATION	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R		
Ankle/ Foot	None																		
	Foot Flat																		
	Foot Slap																		
	Premature Heel Off																		
	Delayed Heel Off																		
	Excessive Plantar Flexion																		
	Excessive Dorsiflexion																		
	Toe Drag																		
Knee	None																		
	Excessive Flexion																		
	Limited Flexion																		
	Excessive Extension																		
	Limited Extension																		
Hip	None																		
	Excessive Flexion																		
	Limited Flexion																		
	Limited Extension																		
	Excessive Adduction																		
	Excessive Abduction																		

Comments:

REFERENCES

1. Delisa JA. Rehabilitation Medicine: Principles and Practice. 2nd ed. Philadelphia, Pa: Lippincott Co; 1993:122-130.

2. Little H. Gait Analysis for Physiotherapy Departments: A review of current methods. Physiotherapy; 67(11):334-337.

3. Leavitt LA, Zunig EN, Calvert JC, et al. Gait analysis of normal subjects. South Med J. 1971;64(9):1131-1138.

4. Gronley JK, Perry J. Gait analysis techniques: Rancho Los Amigos gait laboratory. Phys Ther. 1984;63:1831-1838.

5. Perry J. Gait Analysis: Normal and Pathological Function. Thorofare, NJ: Slack Inc.; 1992.

6. Rancho Los Amigos Medical Center. Observational Gait Analysis Handbook. Downey, Calif; Los Amigos Research and Education Institute, Inc.; 1993.

7. Inman VT, Ralston HJ, Todd F. Human Walking. Baltimore, Md; Williams & Williams Co; 1981.

8. Skinner SR, Antonelli D, Perry J. Functional demands on the stance limb in walking. Orthopedics. 1985;8(2):355-361.

9. Murray MP, Drought AB, Kory AC. Walking patterns of normal men. J Bone Joint Surg. 1963;46A(2):335-360.

10. Elftman H. The Functional Structure of the Lower Limb. New York, NY; McGraw-Hill Book Co; 1954:411-436.

11. Perry J. Anatomy and biomechanics of the hindfoot. Clin Orthop. 1983;177:9-16.

12. Kabada MP, Ramakaishan HK, Wooten ME. et al. Repeatability of kinematic, kinetic, and electromyographic data in normal adult gait. J Orthop Res. 1989;7:849-860.

13. Seichert N, Senn E. Clinical meaning of the torgue between stance leg and ground for the analysis of gait mechanism. Clin Investig. 1993;71:214-220.

14. Dillingham TR, Lehmann JF, Price R. Effect of lower limb on body propulsion. Arch Phys Med Rehabil. 1992;73:647-652.

15. Hang YS. Biomechanics of the knee joint in normal and pathological gait: an electrogoniometric study. J Formosan Med Assoc. 1983;82:169-189.

16. Kettlekamp DB, Johnson RJ, Smidt GL, et al. An electrogoniometric study of knee motion in normal gait. J Bone Joint Surg. 1970;52A(4):775-790.

17. Loslever P, Laassel EM, Angue JC. Combined statistical study of joint angles and ground reaction forces using component and multiple correspondence analysis. IEEE Trans Biomed Eng. 1994;51(12):1160-1167.

18. Mansour JM, Audri ML. The passive elastic moment at the knee and its influence on human gait. J Biomech. 1986;19(5):369-373.

19. Tesio L, Civaschi P, Tessari L. Motion of the center of gravity of the body in clinical evaluation of gait. Am J Phys Med. 1985;64(2):57-65.

20. Cerny A. Pathomechanics of stance: clinical concepts for analysis. Phys Ther. 1984;64(12):1851-1859.

21. Larsson LE, Odenrick P, Weitz P. et al. The phases of the stride and their interaction in human gait. Scand J Rehab Med. 1980;12:107-112.

22. Winter DA. Knowledge base for diagnostic gait assessment. Med Prog Technol. 1993;19:61-81.

23. Kameyama O, Agawa R, Okamoto T, Kumamoto M. Electric discharge patterns of ankle muscles during the normal gait cycle. Arch Phys Med Rehabil. 1990;71:969-974.

24. Harris GF, Wertsch JJ. Procedure for gait analysis. Arch Phys Med. Rehabil. 1994;75:216-223.

25. Stanic U, Bajd T, Valencic V, et al. Standardization of kinematic gait measurements and automatic pathological gait pattern diagnostics. Scand J Rehab Med. 1977;9:95-105.

26. Norkin C., Levangie P. Joint Structure & Function: A Comprehensive Analysis. Philadelphia, Pa; F.A. Davis Co. 1992.

27. Krebs DE, Edelstein JE, Fishman S. Reliability of observational kinematic gait analysis. Phys Ther. 1985;65(7):1027-1033.

28. O'Sullivan SB, Schmitz TJ. Physical Rehabilitation: Assessment and Treatment. 3rd ed. Philadelphia, Pa; F.A. Davis Co; 1994.

29. Goodgold J. Rehabilitation Medicine. St. Louis, Mo: The C.V. Mosby Co; 1988.

30. Gage JR. An overview of normal walking. Instr. Course Lect. 1990;39(37):291-303.

31. Hughes J, Jacobs N. Normal human locomotion. Prosthet Orthot Int. 1979; 3(1):4-12.

32. LaMont JG. Functional anatomy of the lower limb. Clin Plastic Surg. 1986;13(4):571-579.

33. Novecheck TF. Walking, running, and sprinting: A three dimensional analysis of kinematics and kinetics. Instr Course Lect. 1995;44:497-506.

34. Stokes VP, Anderson C, Frossberg H. Rotational and translational movement features of the pelvis and thorax during adult locomotion. J Biomech. 1989;22(1):43-50.

35. Donatelli, RA. Biomechanics of the Foot and Ankle. Philadelphia, Pa: F.A. Davis Co; 1996:90-111.

36. Kaplan PE, Cerullo LJ. Stroke Rehabilitation. Boston, Mass: Butterworth Publishers; 1986:128-133.

37. Thomas SS, Barnett S. Walking through gait analysis. Orthop Nurs. 1994;13(6):7-13.

38. Smidt GL. Gait in Rehabilitation. New York, NY: Churchill Livingston Inc. 1990.

39. Lehmann JF. Push off and propulsion of the body in normal and abnormal gait: Correction by ankle-foot orthoses. Clin Orth Rel Res. 1993;288:97-107.

40. Gilbert JA, Maxwell GM, McElhaney JH, et al. A system to measure forces and moments at the knee and hip during level walking. J Orth Res. 1984;2(3):281-288.

41. Winter DA. Foot trajectory in human gait: a precise and multifactorial motor control task. Phys Ther. 1992;72(1):45-54.

42. Galley PM, Forster AL. Human Movement. 2nd ed. New York, NY: Churchill Livingston; 1987:227-237.

43. Gottschalk F, Kourosh S, Leveau B. The functional anatomy of tensor fascia latae and gluteus medius and minimus. J Anat. 1989;166:179-189.

44. Devita P. The selection of a standard convention for analyzing gait data based on the analysis of relevant biomechanical factors. J Biomech. 1994;27(4):501-508.

45. Crosbie WJ, Nicol AC. Reciprocal gait in paraplegia. Paraplegia. 1990;28:353-363.

46. Krawetz P, Nance P. Gait analysis of spinal cord injured subjects: effects of injury level and spasticity. Arch Phys Med Rehabil. 1996;77:635-638.

47. Waters RL, Yakura JS, Adkins R. et al. Determinants of gait performance following spinal cord injury. Arch Phys Med Rehabil. 1989;70:811-818.

48. Lehmann JF, Esselman PC, Ko MJ, et al. Plastic ankle-foot orthoses: evaluation of function. Arch Phys Med Rehabil. 1983;64(9):402-407.

49. Somers MF. Spinal Cord Injury: Functional Rehabilitation. East Norwalk, Conn: Appleton & Lange; 1992.

50. Yakura JS, Waters RL, Adkins RH. Changes in ambulation parameters in spinal cord injury individuals following rehabilitation. Paraplegia. 1990;28:364-370.

51. Waters RL, Yakura JS, Adkins RH. Gait performance after spinal cord injury. Clin Orth Rel Res. 1993;288:87-96.

52. Eastlack ME, Arvidson J, Snyder-Mackler L, et al. Interrater reliability of videotaped observational gait analysis assessments. Phys Ther. 1991;71:465-472.

53. Heinamann AW, Magiera-Planey R, Schiro-Geist et al. Mobility for persons with spinal cord injury: An evaluation of two systems. Arch Phys Med Rehabil. 1987;68:90-93.

54. Bromley I. Tetraplegia and Paraplegia: A Guide for Physiotherapists. 4th ed. New York, NY: Churchill Livingstone; 1991:164.

55. Fletcher GF, Banja JD, Jann BB, et al. Rehabilitation Medicine: Contemporary Clinical Perspectives. Philadelphia, Pa: Lea & Febiger; 1992:395-397.

56. Shurr DG, Cook TM. Prosthetic & Orthotics. Norwalk, Conn; Appleton & Lange; 1990:123-127.