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Subtalar Joint Biomechanics

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SUBTALAR JOINT BIOMECHANICS

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Tom L. Baumgartner
Bachelor of Science in Physical Therapy
University of North Dakota, 1995

An Independent Study

Submitted to the Graduate Faculty of the

Department of Physical Therapy

School of Medicine

University of North Dakota

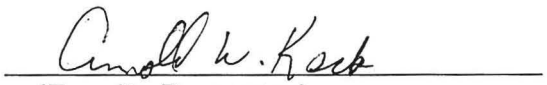
in partial fulfillment of the requirements

for the degree of

Master of Physical Therapy

Grand Forks, North Dakota
May
1996

This Independent Study, submitted by Tom L. Baumgartner 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)

(Graduate School Advisor)

(Chairperson, Physical Therapy)

PERMISSION

Title Subtalar Joint Biomechanics

Department Physical Therapy

Degree Master of Physical Therapy

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Date 12/07/95

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

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ABSTRACT

Subtalar joint movement is transmitted proximally to the lower extremity and distally to the forefoot during gait. Thus, the entire lower kinetic chain is influenced by abnormal subtalar joint biomechanics. If the subtalar joint is forced to compensate for structural deformities of the foot and leg, various lower extremity injuries are likely to develop. The purpose of this study is to examine the normal and abnormal biomechanics of the subtalar joint. In the process, the clinician will be able to identify various subtalar joint abnormalities and relate these to their respective lower kinetic chain pathologies. By correctly assessing the cause of the lower extremity injury, the examiner will be able to initiate the appropriate form of treatment.

CHAPTER I

INTRODUCTION

The foot is an important and often overlooked link in the lower kinetic chain, absorbing the equivalent of 639 metric tons of pressure each day.[1] It connects the body to the ground thereby facilitating movement. It must sustain weight bearing forces and provide stability without compromising mobility. During the normal walking cycle, the subtalar joint (STJ) must play two important roles. First, it must function as a shock absorber and mobile adaptor to accept uneven terrain. Second, it must provide a stable, rigid lever for push off in order to propel the body forward during gait. These are duties that each foot must sufficiently perform 10,000 to 15,000 times per day.[1] The importance of proper mechanics and functioning of the foot is apparent when an individual experiences pain and discomfort in the lower extremity. Faulty mechanical relationships between the rearfoot (STJ) and the forefoot can disrupt the whole lower kinetic chain, producing symptoms in the knees, hips, and low back.[2]

The normal foot functions as a mobile adaptor at heel strike, when the STJ begin to pronate from a slightly supinated position accompanied with internal rotation of the tibia. This dampens compressive forces and allows the lower extremity to accommodate to terrain.[1-3] The normal foot functions as a

rigid lever during the toe-off portion of gait. The STJ resupinates to accommodate the external rotation of the lower extremity at late stance phase. This supination of the STJ causes the joints of the foot to become rigid, thereby providing a firm base for propulsion at toe-off.[1,3]

Abnormal STJ pronation or supination due to congenital, acquired, or neuromuscular pathologies can disrupt the normal foot mechanics. This creates stresses on tissues which may contribute to injury throughout the lower kinetic chain.[1,3,4]

Problems can arise with excess pronation. For example, if the calcaneus or the forefoot are inverted excessively at heel strike, the STJ will be forced to hyper pronate in order for the foot to remain flat on the ground. Likewise, pathology may occur in a foot which pronates too long during stance phase of gait. This prolonged pronation causes inadequate supination when stability is needed for gait propulsion. These types of compensations can eventually strain the soft tissues responsible for controlling the amount of STJ pronation and internal rotation at the leg.[5]

When the STJ remains supinated throughout the gait cycle, lower extremity pathology is also likely to arise. If the STJ remains in a supinated position during the early part of stance phase, the foot will lack the ability to adequately perform the important functions of shock absorption and terrain accommodation.[6] Once again, this may ultimately lead to soft tissue strain as well as osseous disruption of the lower extremity. Furthermore, the foot often

exhibits lateral instability because of the lack of terrain adjustment due to insufficient STJ pronation.[3]

As a clinician, it becomes imperative to understand the intricate and complex functions of the STJ when dealing with lower kinetic chain pathologies. It would be naive to underestimate the role of the STJ when considering the important functions it plays as a mobile adaptor and rigid lever. Various anatomical structures in the lower extremity are excessively stressed when the STJ fails to exhibit proper mechanical alignment. This can contribute to a multitude of injuries, such as plantar fasciitis, tibialis posterior tendinitis, IT band syndrome, patellofemoral pain, and sacroiliac dysfunction to name a few.[2,3,7]

The purpose of this study is to examine the normal and abnormal biomechanics of the STJ in an attempt to describe how lower kinetic chain pathologies can be directly related to faulty STJ mechanics. This will, in the author's opinion, provide the clinician with the tools to successfully understand and identify the problematic link in lower extremity functioning, thus allowing for efficient and appropriate care.

CHAPTER II

STRUCTURAL ANATOMY

Osseous Anatomy

The foot and ankle are the focal points to which body weight is transmitted during standing, walking, and running. The foot is composed of 26 bones: 14 phalanges, 5 metatarsals, and 7 tarsals. Functionally, the foot can be divided into the anterior, middle, and posterior segments.[8,9] All three segments work together to provide the osseous support for the foot and ankle. The posterior segment, often referred to as the hindfoot or rearfoot, is made up of the calcaneus and the talus and is responsible for supporting the tibia.[10,11] This articulation between the talus and the calcaneus is called the STJ. It is this joint that will now be examined.

The talus forms the superior portion of the STJ and is often considered the mechanical keystone of the foot.[8] It is composed of a body, neck, and head. Superiorly, the talus articulates with the distal ends of the tibia and fibula forming the talocrural joint. The convex, saddle-shaped superior surface glides under the tibia during ankle motions.

The calcaneus is the largest and strongest bone in the foot, projecting posteriorly forming the prominence of the heel.[12] It articulates with the talus

superiorly and cuboid anteriorly. Laterally, the calcaneus has an oblique ridge called the peroneal trochlea in which the tendon of the peroneus longus muscle passes inferiorly.[8,12]

A shelf projects from the superior border of the medial surface of the calcaneus called the sustentaculum tali. This structure is important in supporting the talus on the calcaneus. Beath and Harris[4] have identified variations in the sustentaculum tali classifying the support as weak, moderate, or strong. The degree of support depends on how distally the anteromedial sustentaculum tali projects. With weak support, excessive talar plantarflexion and adduction (components of pronation) exist due to the lack of osseous support. As a result of weight bearing forces, the talus "falls" off the calcaneus producing a supple foot with decreased ability to lock at toe-off for propulsion. The sustentaculum tali also forms a fulcrum around which the tibialis posterior, flexor hallicus longus, and flexor digitorum longus tendons pass, allowing mechanical leverage for these muscles.[3] With the decreased area for a muscular fulcrum, efficiency of these muscles declines.

The STJ is a composite joint formed by three separate plane articulations between the talus and calcaneus allowing a combination of rolling and gliding motions.[13,14] The concave superoanterior portion of the calcaneus articulates with the convex inferior portion of the talus.[3,8,12,13] The articulation between the talus and the calcaneus is via three facets.[10,15,16]

The posterior facet joint, which is the largest of the three, is convex on the superior surface of the calcaneus and concave on the inferior surface of the talus. A separate joint capsule encases the posterior facet joint.[8,14,16,17] The anterior and middle facets are concave at the calcaneus and convex at the talus. These joints, along with the talonavicular joint, share a common joint capsule.[8,14,16,17] Situated between the anterior and middle facet joints is the sinus tarsi, a canal formed by a groove in the talus and calcaneus. Figure 1-1 illustrates the configuration of the STJ facets.

Hunt[17] classifies the facet articulations into three types. Type A, the most common joint type, is present when the STJ has three facet joints: anterior, middle, and posterior. However, on occasion, there are individual anatomical variations. When the anterior and middle facets are combined, forming one large joint with a separate posterior joint, the individual presents with a Type B STJ. The Type C joint exists when all three facets are continuous with only one large articulation.

Ligamentous Anatomy

The STJ is very stable, rarely dislocating because of the great tensile strength and vast number of ligaments. In addition to stabilizing the STJ itself, the ligaments also support the capsule surrounding the joint.[8,12] Due to weight bearing and ground reaction forces, these ligaments are put under sizable stress while attempting to maintain proper mechanical functioning of the STJ. Individuals with weak ligamentous support are extremely susceptible to overuse

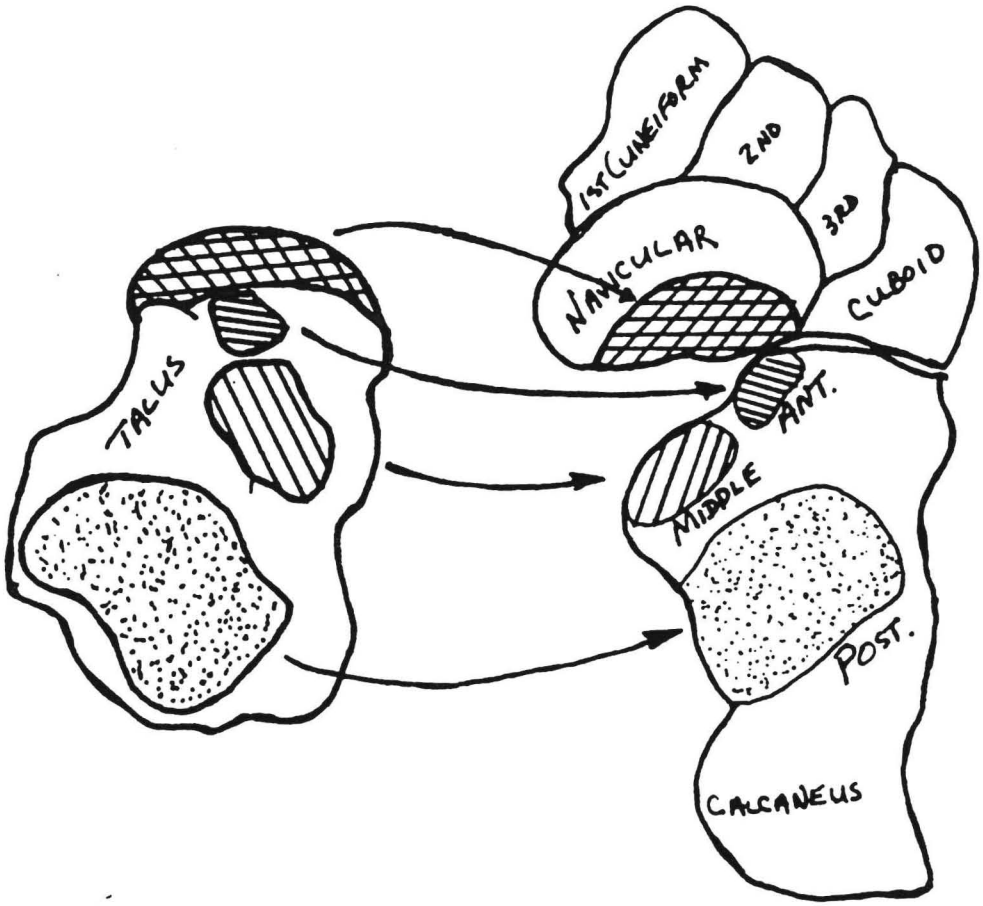

Figure 1-1 Subtalar Joint Facets

injuries in the lower extremity due to decreased stability. It is important to keep in mind that the ligaments are the most important stabilizers of the STJ.[3] The major static supporting structures of the STJ are the interosseous, axial, ligamentum cervicis, and the anterior, middle, lateral, and posterior talocalcaneal ligaments[17,18] (see Table 1-1).

The interosseous talocalcaneal ligament is found in the sinus tarsi (tarsal canal) dividing the STJ into anterior and posterior halves. The sinus tarsi forms a tunnel protecting the ligament from weight bearing forces.[13,14,17,18] The interosseous ligament is made up mostly of collagen fibers with little elastin, making it extremely strong.[13] Its function is to hold the talus and calcaneus together. Anatomically, the interosseous talocalcaneal ligament is derived of an anterior and posterior band. The anterior band runs superiorly, anteriorly, and laterally from the calcaneus to the talus while the posterior band runs superiorly, posteriorly, and laterally. In addition to preventing joint separation, both bands work together to limit inversion, therefore stabilizing the supinated foot. The ligament can be palpated at the large opening of the sinus tarsi located anterior to the fibular malleolus when the foot is in a supinated position.[10,13,14,17]

There are four talocalcaneal ligaments: the lateral, the posterior, the medial, and the anterior. These ligaments attach the talus to the calcaneus and the location of each on the STJ corresponds to its name. The lateral talocalcaneal ligament functions to limit inversion and dorsiflexion of the talus because its fibers run in a posterior and medial direction. The posterior

Table 1-1—Subtalar Joint Ligaments

LIGAMENTS	MOTIONS LIMITED
Interosseous	Inversion, Joint Separation
Anterior Talocalcaneal	Inversion
Medial Talocalcaneal	Eversion
Lateral Talocalcaneal	Inversion, Dorsiflexion
Posterior Talocalcaneal	Dorsiflexion
Ligamentum Cervicis	Inversion, Eversion, Joint Separation
Axial	Eversion

talocalcaneal ligament has fibers running in a vertical orientation, therefore limiting talar dorsiflexion. Inversion is held in check by the anterior talocalcaneal ligament because its fibers are aligned in a plantar, posterior, and lateral direction. The motion of eversion is limited by the plantar and posterior running fibers of the medial talocalcaneal ligament.

The prevention of talar and calcaneal separation is assisted by the ligamentum cervicis. This discrete fibrous band is located lateral to the interosseous talocalcaneal ligament inside the sinus tarsi, connecting the talus and calcaneus via two small tubercles. Several authors also conclude that the ligamentum cervicis aids in limiting inversion and eversion.[8,13,19]

Another important ligament supporting the STJ is the axial (cruciate) ligament. This ligament runs from the superior surface of the calcaneus to the inferior surface of the talus dividing the STJ into almost equal anterior and posterior portions. This ligament contains thick and strong collagen fibers which make it a strong antagonist to eversion movements. Clinically, overstretching of this ligament can result in valgus heel deformity.[19]

Other ligaments that help stabilize the STJ are the deltoid and lateral collateral ligaments of the ankle. The deltoid ligament is located medially and attaches the tibial malleolus to the talus, navicular, and calcaneus. The deltoid ligament becomes taut in eversion and also provides support to the medial longitudinal arch. The lateral collateral ligament of the ankle, which limits inversion motion, is made up of three small ligaments which attach the fibula to

the talus and calcaneus.[12,20] The extent to which the lateral collateral and deltoid ligaments provide STJ stability is presently controversial. Sectioning of these ligaments in cadavers does not appear to result in a substantial decrease in STJ stability.[13,21]

Muscular Anatomy

The muscles acting at the STJ have three primary functions: they stabilize, accelerate, and decelerate the foot.[2,22] It is important to remember that muscles are not the prime stabilizers of the STJ. Unlike the static stabilizers, the muscles will fatigue if relied heavily upon to provide support. When a muscle of the foot is described during locomotion, its function will be quite different than when the muscle contracts without resistance. This occurs because a muscle acting during movement (in the closed chain position) is contracting against the forces of gravity, kinetic forces, and the forces generated by contraction of other muscles.[2,3,13] In this section, the function of each muscle discussed will be in terms of its action during normal locomotion (closed chain). Only the muscles primarily acting at the STJ will be reviewed here.

The anterior tibialis is a thick, long muscle originating on the lateral condyle and superior half of the lateral surface of the tibia. It inserts on the medial and inferior surface of the medial cuneiform bone and on the base of the first metatarsal.[12] The primary function of this muscle during gait is to slightly supinate the STJ prior to heel strike. When the heel is in a supinated position at heel strike, it allows the STJ full range of motion for pronation throughout the

stance phase of gait to absorb ground reaction forces and accommodate to terrain.[23] With a weak anterior tibialis muscle, the heel strikes the ground with excessive pronation. This decreases the effectiveness of the STJ to efficiently absorb shock and accept terrain. It should be noted that this muscle cannot supinate the STJ after heel strike because of its short lever arm in the closed chain position.[1,2,24] In summary, the anterior tibialis provides its greatest effect at the STJ during the open chain activities of swing phase.

The extensor digitorum longus muscle functions as a stabilizer and accelerator of the foot during gait. It originates from the lateral condyle of the tibia, superior three-fourths of the anterior surface of the fibula and interosseous membrane. The middle and distal phalanges of the lateral four digits are the insertions.[12,25] This muscle becomes active at the STJ during the later portions of swing phase, providing a pronation force antagonistic to the supinating force of the anterior tibialis. If the extensor digitorum longus muscle functions inadequately, the STJ will supinate throughout swing phase of gait causing the foot to be excessively supinated at heel strike.[1,2] The anterior tibialis and extensor digitorum longus, therefore, function together to provide the essential amount of supination at heel strike for optimal foot functioning.

The peroneus tertius is a small muscle originating on the lower anterior surface of the fibula and inserting on the dorsal surface of the base of the fifth metatarsal bone. Some authors refer to this muscle's tendon as the fifth tendon of the extensor digitorum longus.[25] The functional actions of this muscle are not

at all dissimilar to that of the extensor digitorum longus. This muscle acts to prevent excessive supination of the STJ during late swing phase by providing an antagonistic pronation force to the anterior tibialis. Various authors[2,26] state that when the peroneus tertius muscle is inactive or absent, the extensor digitorum longus muscle can still satisfactorily limit exaggerated STJ supination at heel strike. Their observations indicate that the peroneus tertius muscle primarily functions synergistically with the extensor digitorum longus muscle.

The gastrocnemius and soleus muscles also demonstrate activity at the STJ. The gastrocnemius originates on the lateral and medial condyles of the femur, while the soleus originates on the posterior aspect of the head and upper neck of the fibula and soleal line of the tibia. These two muscles share a common tendon, the achilles, which inserts on the posterior surface of the calcaneus.[12,25] Together, the gastroc-soleus complex act to supinate the STJ and externally rotate the tibia during propulsive period of gait (toe-off), thus providing a stable lever for push-off.[1,2] In addition, these muscles forcibly plantarflex at toe-off initiating locomotion.

Located in the posterior compartment dep to the gastroc-soleus muscles is the posterior tibialis muscle. It originates on the posterior two-thirds surface of the tibia, fibula, and interosseous membrane. The distal attachments are the navicular tuberosity, lateral cuneiform, cuboid, and bases of second, third, and fourth metatarsal bones.[12,25] The posterior tibialis' tendon passes beneath the medial malleolus to its insertion. The medial malleolus serves as a pulley,

thereby increasing the efficiency of the posterior tibialis at the STJ. This muscle's most clinically significant function is to eccentrically decelerate the pronation occurring at the STJ and the internal rotation occurring at the leg during the contact phase (heel strike and foot flat) of gait.[27,28] Another major action is to supinate the STJ and externally rotate the tibia during the midstance period of gait. Unlike the anterior tibialis, the posterior tibialis is a strong supinator of the STJ during closed chain activities.

The flexor hallicus longus and flexor digitorum longus muscles share common functions at the STJ during locomotion. By assisting the posterior tibialis and gastroc-soleus muscles, the flexor hallicus longus and flexor digitorum longus help concentrically accelerate the STJ into a supinated position. They also externally rotate the leg during midstance in preparation for toe-off. Furthermore, they assist in deceleration of STJ pronation and tibial internal rotation at initial contact.[1,2,28] The flexor hallicus longus originates at the inferior two-thirds of the posterior surface of the fibula and interosseus membrane and inserts at the base of the distal phalanx of the great toe. The flexor digitorum longus originates on the medial part of the posterior surface of the tibia and fibula inferior to the soleal line and inserts on the base of the distal phalanx of the lateral four toes.[12,25]

The peroneus longus and brevis muscles exert an antagonistic action at the STJ to the supination forces produced by the posterior tibialis, gastroc-soleus, flexor hallicus longus, and flexor digitorum longus.[1] The peroneus

longus and brevis function together to eccentrically decelerate the speed of STJ supination by providing a pronation force. This action allows a smooth and controlled return of the STJ to its neutral position by late midstance.[1,2,28] These lateral compartment muscles originate on the lateral surface of the fibula (the longus at the head and upper two-thirds and the brevis at the inferior one-third).[12,25] The peroneus longus insets at the base of the first metatarsal and medial cuneiform bones while the peroneus brevis inserts on the lateral tuberosity of the fifth metatarsal.

It can be readily seen that the muscles discussed work in harmony with one another. Each muscle has an important action at the STJ which must be efficiently undertaken for optimal foot functioning. If a particular function fails to be adequately performed by the muscle in question, foot and lower extremity pathology are likely to follow. Table 1-2 lists the muscles and their respective STJ actions during locomotion.

Table 1-2—Actions of Muscles at STJ During Gait

MUSCLES	ACTIONS
Anterior Tibialis	Slightly supinate STJ prior to heel strike and control excessive pronation during swing phase.
Extensor Digitorum Longus	Prevents excessive supination of STJ prior to heel strike.
Peroneus Tertius	Prevents excessive supination of STJ prior to heel strike.
Gastroc-Soleus	Supinates STJ and provides propulsion forces at toe-off.
Posterior Tibialis	Decelerates pronation at STJ during contact phase and supinates STJ during midstance.
Flexor Digitorum Longus and Flexor Hallicus Longus	Decelerates pronation at STJ during contact phase and supinates STJ during midstance.
Peroneus Long and and Peroneus Brevis	Provide antagonistic pronation force at the STJ during midstance

CHAPTER III

NORMAL STJ BIOMECHANICS

It is important for the clinician to have a structural and functional understanding of the STJ in order to distinguish the normal biomechanics from the abnormal biomechanics. Cailliet[8] describes a normal functioning foot as one in which there is no pain, normal muscle balance, and a central heel. When the muscular and mechanical forces acting on the STJ are in sync, the foot acts as an efficient mobile adaptor and a powerful lever during toe-off.

Principles of Motion

The STJ provides movement in all three cardinal body planes: the sagittal, frontal, and transverse. Plantarflexion and dorsiflexion occur in the sagittal plane, adduction and abduction in the transverse plane, and inversion and eversion in the frontal plane.[3,10,13,20] The movement at the STJ is termed triplanar motion because the axis of motion is at approximately a 45-degree angle from all three body planes.

The STJ axis has an average angle of 42 degrees superior to the horizontal in the sagittal plane, and 23 degrees medial to the midline in the transverse plane[1,3,10,13,14] (see Figure 2-1). The axis travels from the posterolateral portion of the calcaneus to the anteromedial neck of the talus,

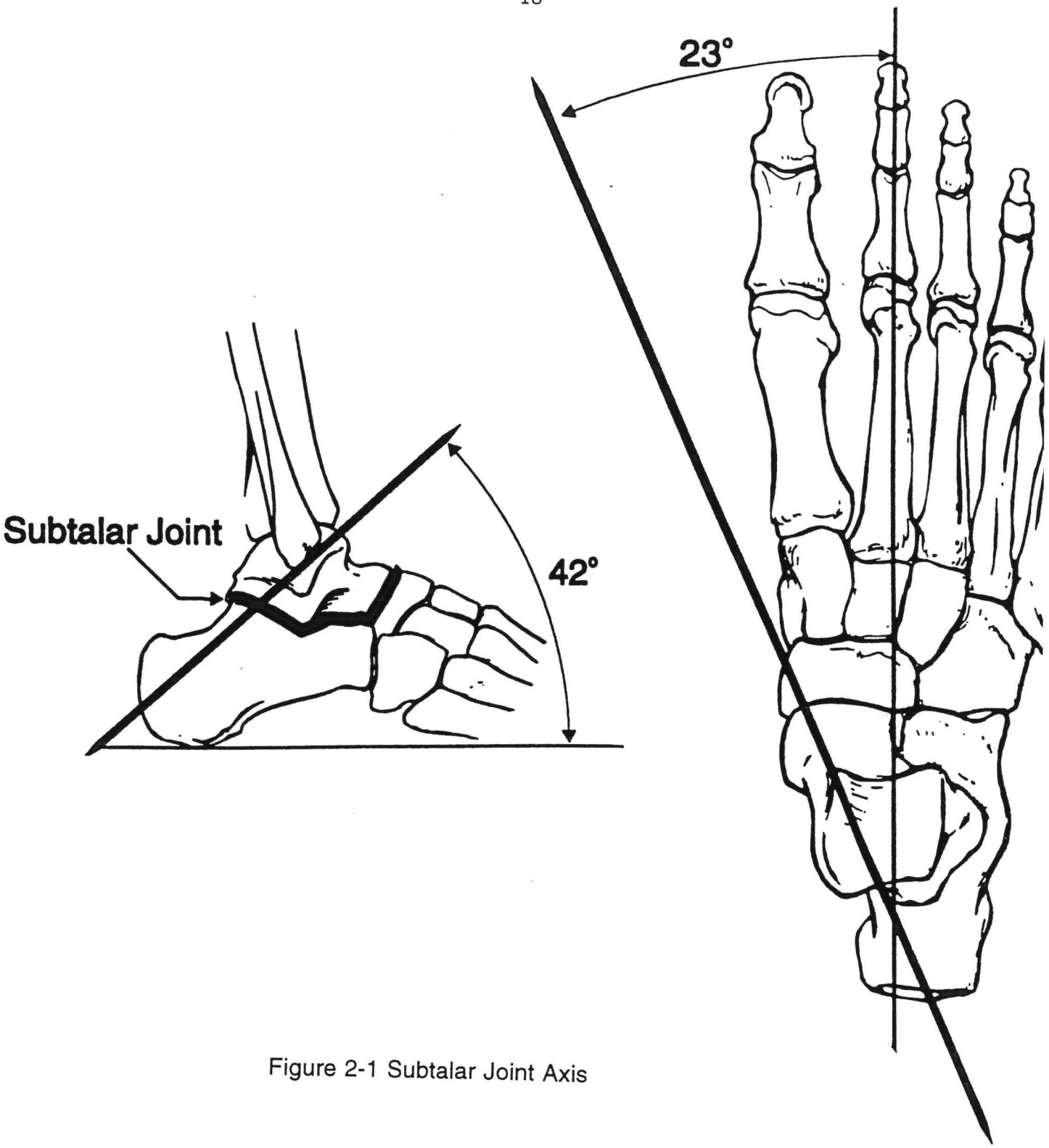

Figure 2-1 Subtalar Joint Axis

passing between the second and third toes.[29] There is much individual variation in the location of the STJ axis of motion. Numerous investigators report variations in the STJ axis position ranging from 20 to 68.5 degrees from the transverse plane and 4 to 47 degrees from the sagittal plane.[1,3,20,30] Ideally, when the orientation of the oblique axis of the STJ is equidistant from each plane of movement, the amount of sagittal, frontal, and transverse plane motions are equal.[1-3] If the axis runs more parallel to a specific plane, less motion is possible in that plane. For example, if the axis of motion lies closer to the frontal plane, there is less inversion/eversion and subsequently more dorsiflexion/plantarflexion movement available.[1-3] It is these instances in which the STJ inadequately functions due to the loss of true triplanar movement. This limits the STJ's ability to effectively compensate for various lower extremity motions during weight bearing activities.[1,2]

Clinically, the STJ axis is determined by comparing the range of rearfoot inversion/eversion with the range of tibial rotation during weight bearing supination and pronation. If the axis lies 45 degrees to the transverse plane, every one degree of rearfoot motion will produce one degree of tibial rotation. Therefore, if the STJ pronates five degrees, the tibia should be expected to internally rotate five degrees.[1,2] However, an axis positioned 70 degrees to the transverse plane will produce tibial rotation in amounts greatly exceeding STJ motion. This is significant because a high STJ axis (> 45°) is directly

responsible for chronic injuries to structures proximal to the STJ; while a low axis (< 45°) contributes to chronic injuries of structures distal to the STJ.[1]

The movement around the single oblique axis of the STJ consists of the triplanar motions of pronation and supination.[10,13,20] It should be kept in mind that supination and pronation are composite motions which can be broken down into movements occurring in each plane. However, these components do not and cannot occur independently.[13] Supination of the non-weight bearing STJ contains the component motions of inversion, adduction, and plantarflexion. Non-weight bearing pronation consists of the components of eversion, abduction, and dorsiflexion.[14,20,22] In the open kinetic chain, the calcaneus moves on a stable talus.

The functional biomechanics of the STJ occur during closed kinetic chain activities when the foot bears the weight of the body. The STJ is the first joint of the foot subject to the ground reaction forces of gait. When the calcaneus bears weight, it is unable to accomplish all of the components of triplanar motion.[13] It is still able to move in the frontal plane, providing inversion and eversion, but unable to dorsiflex/plantarflex or abduct/adduct. As previously mentioned, STJ motion cannot consist of one component independently from the other; therefore, the transverse and sagittal plane movements are performed by the talus.[13,20] In closed kinetic chain supination, the calcaneus inverts while the talus dorsiflexes and abducts. The opposite occurs during closed chain activities when the calcaneus everts and the talus plantarflexes and adducts.[20,28]

The Relationship Between the STJ and Lower Extremity Biomechanics

One of the primary relationships in the lower extremity is the interdependence between the STJ and the tibia.[13] In weight bearing, STJ motion absorbs the imposed lower extremity rotational stresses that would otherwise rotate the foot on the ground or disrupt the talocrural joint by rotating the mortise around a fixed talus.[13] It is important to keep in mind that open kinetic chain motions of the STJ and leg are independent of one another. In the closed kinetic chain, however, internal rotation of the leg occurs during STJ pronation because the talus is allowed to adduct and plantarflex while the calcaneus everts. Likewise, tibial external rotation in weight bearing produces STJ supination (talus dorsiflexes and abducts while the calcaneus inverts).[2,13,29]

It becomes apparent that the STJ functions as a torque converter for the lower extremity during closed chain situations. Transverse plane movements of the lower kinetic chain are attenuated at the STJ by pronation and supination. Likewise, STJ supination and pronation manifests as tibial external and internal rotation respectively.[29,30,31] During contact phase of gait, the tibia rotates an average of 10 to 19 degrees in the transverse plane.[32,33] Since the STJ axis is inclined at 42 degrees from the transverse plane, it is able to absorb these tibial rotations.[29,32-34] Instead of the foot rotating on the ground, the calcaneus inverts or everts depending on the movement which the tibia transmits to the talus. This is all possible because the STJ is able to convert transverse plane rotations of

the tibia to pronation/supination forces, thus completing the torque conversion.[3,20,32,33] Table 2-1 lists the component motions of the STJ in open and closed chain positions.

Range of Motion

Supination and pronation range of motion are difficult to assess because of the triplanar nature of the STJ and the varied inclination angle. However, the calcaneus component can be easily measured in both weight bearing and non-weight bearing. This is accomplished by measuring the angle of calcaneal inversion or eversion with respect to the tibia.

Normal range of motion of calcaneal inversion is 20 degrees, and calcaneal eversion is 10 degrees.[2,35,36] As discussed previously, the tibial rotation during weight bearing is 10 to 19 degrees.[32,33] This amount of tibial rotation is equivalent to the amount of abduction/adduction of the talus. Furthermore, this amount of abduction/adduction equals the range of calcaneal inversion/eversion measured with weight bearing.[2] Sgarlato[37] determined that the STJ ranges for inversion/eversion, abduction/adduction, and dorsiflexion/plantarflexion follow the ratio 4:4:1 respectively. That is, for every four degrees of calcaneal eversion, there should be four degrees of talar adduction (therefore, four degrees of tibial internal rotation), and one degree of talar plantarflexion. The above information illustrates the total interdependence of the STJ's component motions by virtue of their precise rhythmic relationships during closed chain functions.

Table 2-1—Subtalar Joint Component Motions

OPEN CHAIN	CLOSED CHAIN
Supination	Supination
Calcaneal inversion	Calcaneal inversion
Calcaneal adduction	Talar abduction
Calcaneal plantarflexion	Talar dorsiflexion
Pronation	Pronation
Calcaneal eversion	Calcaneal eversion
Calcaneal abduction	Talar adduction
Calcaneal dorsiflexion	Talar plantarflexion

Gait

During locomotion, the joints of the lower extremity exhibit a biomechanical complexity and symmetry as they move through the sagittal, frontal, and transverse planes. The STJ is an important link in the lower kinetic chain because it is able to change functions very rapidly, moving from a mobile adaptor to a rigid lever. By examining the functions of the STJ during normal gait activities, a better understanding of how faulty STJ mechanics influence lower extremity injuries becomes apparent. At this time, an in-depth review of the role of the STJ during the different phases of gait shall be explored.

The gait cycle is the basic reference used when describing locomotion. This refers to the period of time between successive ipsilateral heel strikes beginning when the heel makes ground contact and terminating when the same heel strikes the ground with the next step.[34] Stance phase, which occupies 60 percent of the gait cycle, is the portion of the cycle when the distal kinetic chain is fixed to the surface by ground reaction forces (closed chain).[1-3,8] This phase can be broken down into contact, midstance, and propulsion. Swing phase, by contrast, is the portion of the gait cycle in which the distal kinetic chain is freely mobile in space (open chain).[1,20]

Contact Phase

Contact phase can be broken down into heel strike and footflat. Just prior to heel strike, the STJ is in a position of supination with tibial external rotation. At heel strike, the lateral aspect of the posterior calcaneus meets the ground.

The calcaneus will evenly rest on the ground due to STJ pronation caused by the ground reaction forces and because the body weight is directed medially to the STJ. The STJ should be in a pronated position throughout heel strike and footflat phases, therefore providing shock absorption for the repetitiveness of the gait cycle while allowing the foot to be able to accommodate to terrain.[1-3,20,22,38,39] Normally, the STJ pronates only during heelstrike and footflat periods with various authors describing normal pronation range between 4 to 12 degrees from neutral (the discrepancy between these figures is related to the variable STJ axis amongst individuals).[2,34,38,40]

The combined talar movements of adduction and plantarflexion during pronation are the mechanism by which the STJ functions as a shock absorber.[1,9] Talar plantarflexion directly allows for shock absorption because it lowers the ankle mortise enabling the supporting musculature more time to dampen the body's momentum. Less force is absorbed by the soft tissues when more time is allowed to absorb these forces.[1,2,33] This reduces the magnitude of impact forces allowing for increased shock absorption. Furthermore, the STJ indirectly provides shock absorption as the adducting talus internally rotates the tibia, which subsequently flexes the knee. Flexion of the knee lessens potential injury by decreasing impact forces because the quadriceps musculature has more time to dampen ground reaction forces.[1,2,33] Also, STJ pronation at contact phase of gait shortens the lower extremity as much as 1 cm, therefore providing a dampening effect to ground reaction forces.[41]

STJ pronation during heel strike and footflat periods allows for surface adaptation by placing the two midtarsal joint axis in a parallel configuration thereby unlocking the midtarsal joint. This unlocking provides added range at the midtarsal joint for improved ability to accommodate terrain.[2,3,34]

Pronation of the STJ during heel strike and footflat phases is an entirely passive action. Mann et al[42] claim that this pronation is limited only by the placement of the axes of the STJ and midtarsal joint, the capsular attachments, and the extra-articular ligaments. However, after performing EMG studies, other authors[1,3] conclude that the posterior tibialis, flexor hallicus longus, flexor digitorum longus, and gastroc-soleus muscles play a role in controlling STJ pronation during contact phase by decelerating pronation. Apparently the constraints of STJ pronation are primarily osseous configuration and ligamentous support with muscular support contributing to a lesser degree.[1,3,29,32,39]

Midstance

Midstance phase of gait begins when the body is directed vertically over the tibia and terminates at heel strike. It is during this period of gait that the body begins single limb support. Throughout midstance period, the foot changes from a mobile adaptor to a rigid lever needed for propulsion. At this time, there is definite EMG activity of the STJ supinators, particularly the posterior tibialis, as these muscles move the STJ toward a supinated position.[2] The body weight tends to force pronation at the STJ during midstance. This

would tend to fatigue the muscles working to supinate the STJ against the body's weight. However, during midstance, the body laterally displaces over the stance leg which shortens the body weight's effective lever arm. This shortened lever arm decreases the body's STJ pronation force.[34]

In addition to the muscular action during midstance period, the STJ begins supinating due to the forward momentum provided by the contralateral extremity. The forward momentum of the swing phase leg externally rotates the pelvis which in turn externally rotates the stance leg. This causes the talus to abduct and dorsiflex and the calcaneus to invert; thus, the STJ supinates.[6,42,43] Furthermore, the forefoot becomes stable because STJ supination locks the midtarsal joint by decreasing the parallelism of the joint axes.[2,29,39] This action creates an osseous locking of the forefoot against the rearfoot which is a necessary prerequisite for normal gait because it minimizes muscular strain.[5,23] By the completion of midstance, the STJ supinates from a pronated position to its neutral position prior to heel lift. Likewise, the leg moves from an internally rotated position to its neutral alignment.

Propulsion

Heel lift period marks the beginning of the propulsive portion of the gait cycle. The STJ continues to supinate from its neutral position via the actions of the posterior leg muscles and the external rotation forces of the leg. These actions work to increase the efficiency of the propulsive lever arm required for effective toe off. STJ supination and further midtarsal joint locking is aided by

the contraction of the intrinsic muscles of the foot, especially the abductor hallicus.[1,2] Gray and Basmajian[44] conclude that the intrinsic muscles firing during late stance phase are more important in individuals with flat feet deformity in order to stabilize the forefoot due to an inadequately locked midtarsal joint.

The supinated position of the STJ at toe-off also functions to improve the efficiency of the peroneus longus by stabilizing the cuboid bone. This allows the muscle to function as prime propulsive agent because it can effectively stabilize and plantarflex the first ray, an extremely critical function during toe-off. The cuboid does not allow for an adequate angle of insertion for the peroneus longus muscle when the STJ is in a pronated position, thus decreasing the effectiveness of push off.[1-3,34] As can be seen, it is vital that the STJ is in a supinated position at toe-off in order for efficient propulsion to occur.

Swing Phase

The swing phase of gait begins with toe-off and ends with heel strike. The leg externally rotates briefly after toe-off and then internally rotates for the duration of swing phase.[33] The STJ moves into a pronated position shortly after toe-off and continues to pronate until midswing phase. The pronated alignment of the STJ during swing assists with toe clearance. Shortly after midswing, the STJ moves from a pronated to neutral position. By the end of the swing phase, the STJ starts to supinate in preparation for heel strike.[33,43]

Summary

At heel strike, the STJ begins in a supinated position followed by immediate pronation to absorb shock and to adjust to terrain. By late midstance, the STJ has supinated to is neutral configuration. Toe-off is accompanied by a supinated STJ to allow for a stable propulsive lever. The leg is in internal rotation during pronation and in external rotation during supination. It is important to keep in mind that the STJ is influenced by the proximal kinetic chain and vice versa.

CHAPTER IV

ABNORMAL STJ BIOMECHANICS

It is now time to examine the foot when the STJ demonstrates abnormal motion. Faults in the lower extremity can lead to changes at the STJ and faults at the STJ can cause deviation throughout the lower kinetic chain. Because the STJ exhibits triplanar motion, it has the ability to compensate for lower extremity deviations in all three planes.[2] STJ compensation is a normal function of the foot, allowing for both mobility and stability. As mentioned previously, the STJ compensates for the internal and external rotations of the leg by pronating and supinating respectively.

Normal STJ compensation is a temporary or intermittent condition required for effective functioning while abnormal compensation is a persistent condition which can lead to pathological functioning.[2] When the STJ is required to compensate for abnormal lower extremity mechanics, injury can occur due to repetitive trauma. The abnormal mechanics, which is usually a single plane deviation, are compensated in all three planes at the STJ, thus leading to abnormal function and pathology.[1-3] This compensation by the STJ in all three planes, instead of the plane which houses the deformity, is what produces the

destructive functioning.[2] These compensations are manifested by increased STJ pronation and supination.

Abnormal Pronation

Pronation at the STJ is considered abnormal if, during any period of gait, the amount of pronation is excessive or if pronation is occurring when the foot should be supinating. Abnormal pronation exists most often to compensate for osseous, functional abnormalities, or muscular imbalances of the lower extremity.[2] Excessive pronation is, essentially, a hypermobile deformity of the foot which subsequently leads to an inefficient lever arm for propulsion at toe-off. Also, excessive pronation directly leads to soft tissue breakdown due to the increased stress placed on the muscles because of ineffective action of ligaments and/or poor osseous stability. The soft tissue breakdown may result in the foot being unable to attenuate the forces of weight bearing. Eventually, this causes changes in muscle function as well as osteologic remodeling. These changes can produce a rigid hyperpronated foot (flat foot deformity) now unable to adequately act as a shock absorber or mobile adaptor.[3] It now becomes apparent how a hypermobile foot, due to excessive compensatory STJ pronation, can lead to a hypomobile foot unable to carry out its important objectives at heel strike.[2,3]

Classification

Abnormal pronation can be classified into three categories: congenital, acquired, and abnormal pronation secondary to neuromuscular diseases. It is

beyond the scope of this chapter to discuss STJ pathologies related to neuromuscular diseases.

Congenital Causes of Abnormal STJ Pronation

Congenital excessive STJ pronation can lead to flat foot deformities and abnormal STJ compensations in the adult.[44] The etiology is that of genetic influence or from malpositioning of the fetus in the uterus.[8] These congenital deformities can be classified as rigid or flexible.

Convex Pes Valgus.—Convex pes valgus (congenital vertical talus) is a congenital rigid disorder which the fetus acquires sometime within the first trimester of pregnancy.[8,45,46] Although it may occur in isolation, it commonly accompanies central nervous system pathologies, such as spina bifida and arthrogryposis.[46] However, the exact etiology of this deformity is presently unknown.

The clinical distinguishing characteristic of convex pes valgus is a dislocated navicular articulating with the dorsal neck of the talus. The STJ may also be dislocated in conjunction with the dislocated navicular. Furthermore, the calcaneus is in an everted and plantarflexed position in non-weight bearing. When the examiner dorsiflexes the infant's foot, the head of the talus forms a prominence at the medial longitudinal arch creating a "rocker bottom" appearance.[8,45,46] X-rays show the talus to be rotated in a plantar direction giving the appearance of a vertical valgus. This deformity, which very often requires surgery, places the STJ

n a hyperpronated configuration which predisposes the child to develop lower extremity pathologies later in life.[8]

Tarsal Coalition.—Another rigid congenital deformity which yields excessive STJ pronation is tarsal coalition. With this condition, there may be complete or incomplete fusion of the STJ along with the navicular-cuneiform articulation. Fusion of the talus and calcaneus is the most common manifestation of tarsal coalition. The fusion can occur at the anterior, middle, and posterior facet joints, thus severely limiting STJ motion.[8,45]

The joint coalition may be fibrous, cartilaginous, or osseous resulting in a rigid flatfoot deformity. This condition is often asymptomatic until the child reaches adolescence or early adulthood.[8] Clinically, the calcaneus demonstrates a valgus (everted) position when the calcaneal and talar coalition occurs, which creates a flattened medial longitudinal arch. This places the STJ in a constant position of pronation with little ability to adequately supinate for normal gait activities.[3,8,45] Eventually, this can lead to soft tissue trauma throughout the lower extremities, such as low back dysfunction, hip strain, medial knee pain, and "shin splints."[2,20]

Treatment is usually symptomatic using modalities such as ice, heat, and ultrasound. The individual may find relief by cutting back on excessive overuse activities like prolonged walking, standing, or intense exercise. Orthotic devices may prove helpful in controlling the excessive STJ pronation. In unresolving

ases, surgery may be required to resect the bar or fuse the STJ to increase foot stability.[8]

Talipes Calcaneovalgus.—The most common congenital flexible deformity which produces increased STJ pronation belongs to talipes calcaneovalgus. There is a high correlation between the presence of this deformity in an infant and subsequent flatfoot deformity in the older child.[45] Tachdjian[45] reports that a malpositioned fetus in utero is the cause of this deformity (the feet are held in an everted and dorsiflexed position beyond the second trimester). The foot is dorsiflexed and the calcaneus demonstrates an everted appearance. Also, upon X-rays, the plantarflexed talus can be observed.[3,8] Important clinical observations in talipes calcaneovalgus are the decreased range of plantarflexion (usually less than 90 degrees) at the ankle joint and a calcaneus positioned in eversion. These findings are the result of a tight achilles tendon.

Acquired Deformities

The acquired flatfoot deformity, due to excessive STJ pronation, develops after birth as a result of trauma, ligamentous laxity, or osseous abnormality of the STJ.[3] The STJ assumes the excessively pronated position as a result of compensations for intrinsic or extrinsic abnormalities of the lower extremity. It is important to keep in mind that when the available STJ pronation is used to compensate for a deformity, little will be available to fulfill the requirements of normal locomotion.[2]

Most often, the acquired deformity can be adequately treated with corrective orthotics. These devices, which are placed in the individual's shoes, are designed to place the STJ and forefoot in (or close to) the anatomical neutral position. This allows the foot to fulfill the required obligations of normal gait without detrimental compensations.[1,47] Fabrication of corrective orthotics is a precise science and a topic worthy of its own merit; therefore, it will not be discussed in detail here. The study of acquired deformities begins with those that are intrinsic to the foot.

Traumatic Flatfoot.—A ruptured posterior tibialis tendon is often a cause of acquired adult flatfoot deformity. As mentioned previously, the posterior tibialis muscle is a strong supinator of the STJ during midstance as well as a decelerator of STJ pronation during heel strike and foot flat. Basmajian[23] reports that this muscle is a crucial stabilizer of the rearfoot by preventing STJ valgus (eversion) deformities. Hence, when the posterior tibialis' tendon is ruptured or the muscle is weak, the static stabilizers of the foot (ligaments and bone) are excessively stressed trying to maintain proper mechanical foot alignment. The increase forced placed upon the STJ culminates in lax ligaments unable to properly stabilize the joint and eventually the destruction of the joint surfaces.[20,23] Because of the decreased support, the talus assumes a position of adduction and plantarflexion as the calcaneus everts. This, in essence, causes the calcaneus to sublux under the talus.

Clinically, the "too many toes sign" is a valuable indicator of a posterior tibialis rupture or muscle weakness. This is when the examiner can see four or five toes while viewing the posterior aspect of the patient's foot in the standing position. Normally, with a properly functioning posterior tibialis, only the first or second toes should be seen.[3]

Ligament Laxity.—When the individual presents with ligamentous laxity of the STJ ligaments or the medial arch supports, excessive STJ pronation resulting in an acquired flatfoot deformity often is manifest.[3,23] A child's foot has a certain amount of ligamentous laxity that is considered normal. As the child matures and gains body weight, the ligaments of the foot should increase in tensile strength. If, for some reason, these ligaments fail to gain sufficient tensile strength, the support of the STJ and foot will be greatly compromised. The weak ligaments are inadequately adjusted to handle the ground reaction forces during gait; therefore, they cannot properly stabilize the STJ. This results in a deficiency in the ability to supinate the foot during toe-off phase of gait.[8,23] This maintained pronation can lead to, once again, damage to the joint articular surfaces particularly in the STJ.

Bony Abnormalities of the STJ.—The importance of the anteromedial projection of the calcaneus (sustentaculum tali) was mentioned in a previous chapter. It is this structure which supports the talus (during heel strike and footflat) when the STJ is passively pronating.[2,11] If the osseous support of the sustentaculum tali cannot adequately hold the talus during weigh bearing, the

talus will excessively plantarflex and adduct (components of abnormal pronation). With weak bony support, the head and neck of the talus project over the anteromedial aspect of the calcaneus.[11] Likewise, the calcaneus moves into eversion. These excessive movements of the talus and calcaneus, due to osseous insufficiency, can lead to acquired abnormal pronation.

Compensatory Pronation Due to Intrinsic Deformity

The remainder of the intrinsic deformities discussed are those in which compensatory pronation occurs in order to achieve the basic biomechanical objective of the foot, which is to achieve a foot flat position during weight bearing. These deviant STJ compensations are discussed in terms of the relative positions of the rearfoot and forefoot when the STJ is in neutral. The STJ neutral position is that in which the least amount of stress to the joints and soft tissues of the foot occur. It is from the neutral position that the STJ has the most efficiency.[47]

When the STJ is in its neutral configuration, the vertical bisection of the posterior calcaneus should be parallel to the vertical bisection of the lower one-third of the tibia in both open and closed chain positions. The plane of the metatarsal heads should be perpendicular to the vertical bisection of the posterior calcaneus.[47-49] In other words, the bisection of the posterior calcaneus should be perpendicular to the ground while the plane of the metatarsals should be parallel to the ground.[48,49] The most common method of determining STJ neutral is to palpate the congruency of the talar head with the talonavicular joint

line while a dorsiflexion force is applied to the fourth and fifty rays to simulate weight bearing. This is most commonly performed in the open chain alignment. If the calcaneal bisection and lower leg bisection do not line up or the metatarsal plane is not perpendicular to the calcaneal bisection, STJ compensation will occur during gait (the extent depending on the amount of deviation). This compensation allows the foot to rest flat on the ground during stance phase.

Rearfoot Varus.—Rearfoot varus is an intrinsic frontal plane deformity resulting in compensatory pronation.[3] This deformity represents an osseous malformation in which the calcaneus fails to derotate from its infantile position.[47] In STJ neutral, the calcaneus is inverted (varus) relative to the vertical bisection of the posterior one-third of the tibia (figure 3-1). Because of the excessively inverted position of the rearfoot, initial ground contact occurs along the posterior lateral aspect of the calcaneus. To compensate for this deformity, the STJ must pronate excessively in order to place the medial part of the calcaneus on the ground.[1,3,49] Pronation may also occur at the midtarsal joint if the STJ lacks adequate range of motion to bring the forefoot to the ground. This abnormality is detrimental to the foot because, along with the normal pronation, compensatory pronation must also occur.[2]

In rearfoot varus deformity, the STJ is forced to pronate rapidly with extreme range of motion to compensate for calcaneal varus. The increase brought upon the STJ during contact phase can cause the soft tissues responsible for limiting STJ pronation to undergo microtrauma. This is due in

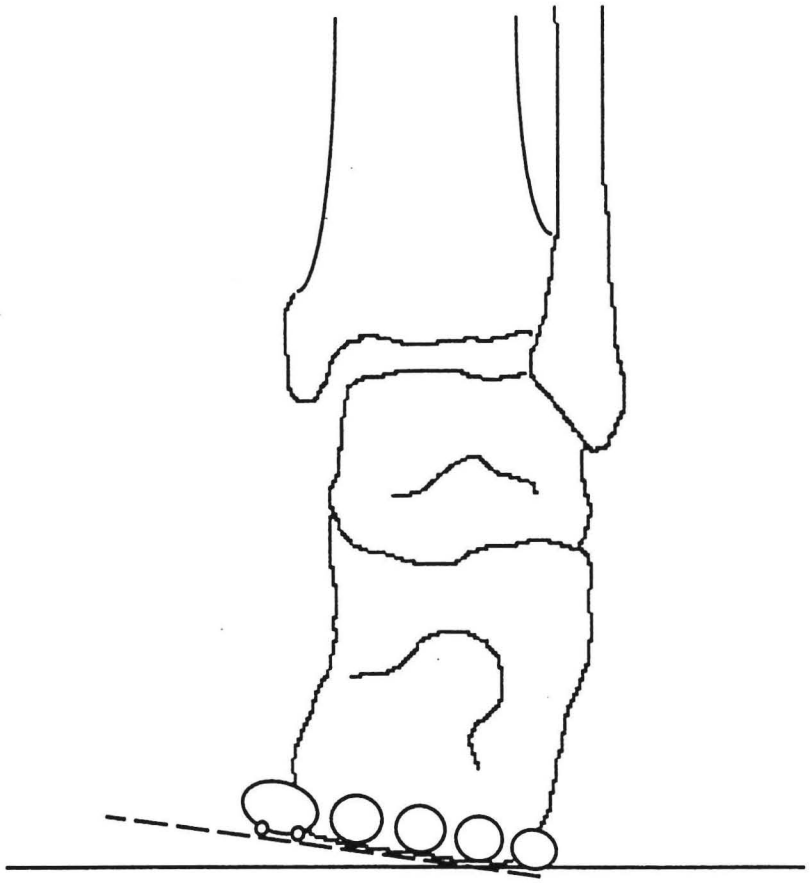

Figure 3-1 Rearfoot Varus

part to the greater repetitive forces these muscles encounter during eccentric loading.[2,3]

There are many clinical signs and manifestations of rearfoot varus which present the individual with lower extremity pain. Many of these clinical signs, however, are common not only in rearfoot varus but in any STJ deviation resulting in excessive pronation. Some of the more common symptoms are briefly discussed here; however, this by no means is a comprehensive list.

Plantar fasciitis is associated with a strain of the plantar fascia and medial longitudinal arch with symptoms usually over the medial calcaneal tubercle. This condition, due to constant repetitive trauma, may result in heel spurs over the calcaneal tubercle. Very often, the abductor hallicus muscle may be implicated due to its increased efforts to stabilize the forefoot.[2] The patient often complains of intense morning heel pain.

Hallux valgus is commonly seen in STJ pronation compensation deformities. This condition is described by the medial deviation of the first metatarsal and the subsequent lateral deviation of the distal phalanges. General ligamentous hypermobility in the foot can be implicated in this condition.[2,47]

Recurrent lateral ankle sprains may be common by virtue of the excessive teral heel strike of the calcaneus in the rearfoot varus deformity.[3] Posterior tibialis tendinitis is frequent due to the increase demand placed on the muscle while attempting to control large amounts of pronation.[7,20] This constant attempt

to control large amounts of pronation eccentrically eventually leads to microtrauma and subsequent irritation.

Medial knee pain (patellofemoral pain) can occur by way of the increased internal rotation occurring at the leg with excessive STJ pronation. This causes the ground reaction forces to be directed medially to the knee. Hence, pain is brought about by the stretching of the soft tissues at the medial aspect of the knee due to the increased valgus moment created at the knee.[1,2,20,47] In addition to soft tissue irritation, the patellofemoral joint is victimized because the valgus moment at the knee tends to cause the patella to rub against the edge of the patellofemoral groove.[7]

IT band syndrome is characterized by pain and tenderness at its insertion. This is caused by the tibia remaining in internal rotation during late stance phase because of excessive STJ pronation. The result is an increased amount of stress at the IT band insertion as well as constant irritation as the IT band constantly rubs across the femoral epicondyle.[22]

Hip and low back pain can also result from abnormal pronation. When the leg is held in internal rotation during late stance phase, the external rotators of the hip must work vigorously to try to control the internal rotation of the femur caused by these tibial forces. This action often produces a traction strain to the piriformis muscle manifesting in hip or low back pain. On occasion, the strained piriformis will irritate the sciatic nerve resulting in the typical pain and paresthesia of sciatica.[47]

The clinical examination is not complete unless the observer evaluates the patient's skin and shoe. Plantar callouses, usually between second and third metatarsal heads, are a common sign associated with rearfoot varus as is exaggerated shoe wear along the lateral border of the heel.[47]

Forefoot Varus.—Forefoot varus is the most common intrinsic deformity resulting in abnormal compensatory pronation.[2] McPoil[50] reports that this condition frequently results in many knee, hip, and pelvic disorders seen in the clinic. Forefoot varus is defined as an inversion of the forefoot on the rearfoot when the STJ is in neutral.[2,47,50] This deformity is caused by insufficient developmental rotation of the head of the talus,[47] although McPoil[50] claims that the etiology can be attributed to abnormality in the talonavicular and/or calcaneocuboid joints. To compensate for the inversion of the forefoot, the STJ must pronate through extreme ranges of motion in order to bring the medial portion of the forefoot to the ground (figure 3-2). This is achieved through talar adduction and plantarflexion and calcaneal eversion.[47,50]

The amount of STJ compensation for a forefoot varus deformity can be measured by the amount of calcaneal eversion in weight bearing.[2,3] For example, if there is six degrees of forefoot varus present, the calcaneus can be expected to evert six degrees. If enough STJ range of motion is not available to allow the forefoot to reach the ground, pronation at the midtarsal joint also occurs. It is important to be aware that the first ray may be plantarflexed when examining the foot in neutral. This is an attempt to assist the STJ in

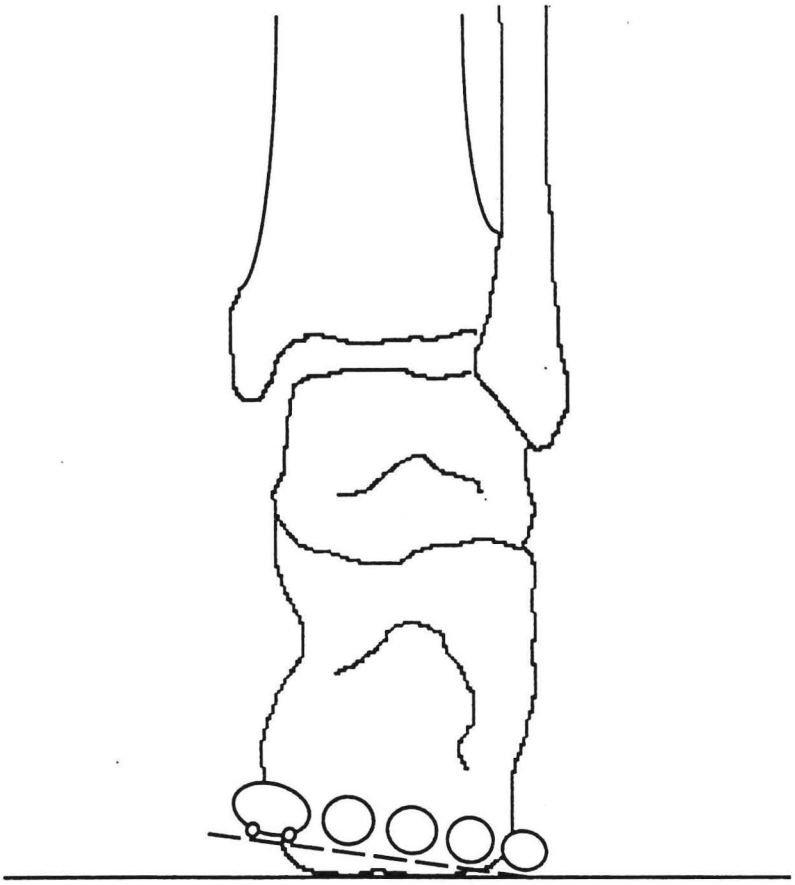

Figure 3-2 Forefoot Varus

compensating for a forefoot varus.[2,3] This may, to the novice clinician, disguise the presence of a forefoot varus because the plane of the first ray may not appear to be inverted. By carefully examining the position of rays two through five (they will still be inverted in the presence of a plantarflexed first ray) and by checking the amount of mobility in the first ray (the first ray will be flexible), an accurate diagnosis will be forthright.

It is important to remember that rearfoot varus and forefoot varus may be present in the same foot, thus forcing a greater degree of compensation. The forefoot varus deformity can be detrimental to the lower extremity because the STJ is maintained in end ranges of pronation and the leg is held in greater amounts of internal rotation. Once again, this intrinsic compensatory mechanism can lead to dysfunction throughout the lower kinetic chain. The lower extremity pathologies discussed under rearfoot varus can also be seen in the forefoot varus deformity by virtue of the same mechanisms.

Ankle Joint Equinus.—Ankle joint equinus can be defined as the lack of ankle dorsiflexion with the STJ in neutral (there needs to be at least ten degrees of dorsiflexion at the ankle joint during gait). The most common cause of this condition can be attributed to a short achilles tendon.[11,28,47] The increase pull from a tight achilles can force the calcaneus to compensate, thereby moving into eversion such that the lateral aspect of the tight tendon can be put on slack. Furthermore, if there is not sufficient dorsiflexion motion for gait activities, the tibia cannot move anterior to the talus during early midstance. This limitation

forces the STJ to pronate severely. The anterior movement of the talus occurring with STJ pronation compensates for the lack of anterior tibial movement.[3,47] In addition, the compensation for ankle joint equinus takes place prior to heel lift reducing the foot's ability to adequately supinate (figure 3-3).[47]

The clinical observations of this intrinsic sagittal plane deformity includes patellofemoral pain, plantar fasciitis, posterior tibialis tendinitis, low back and hip pain, and hallux valgus to name a few.[1,11,47]

Rotational Deformities.—Rotational deformities of the lower extremity are common extrinsic abnormalities resulting in excessive compensatory pronation. The etiology of rotational deformities of the femur and tibia are attributed to soft tissue abnormalities, malposition of the fetus in the uterus, genetic factors, and to certain pathologic sitting and sleeping postures.[51,52]

Excessive femoral anteversion (the relationship of the anterior rotation of proximal head and neck of the femur to the condyles of the distal femur) or medial femoral torsion (a medial directed rotation in the femoral shaft) are seen clinically in individuals demonstrating a toe-in gait pattern.[51,52] These patients often have excessive hip internal rotation (60-90 degrees) and decreased hip external rotation (less than 25 degrees).

Compensation throughout the lower extremity can occur if femoral anteversion or medial femoral torsion exist after the age of four or five years.[52] The child may develop excessive compensatory external tibial torsion (a transverse plane deformity due to rotation of tibia about its long axis) to reduce

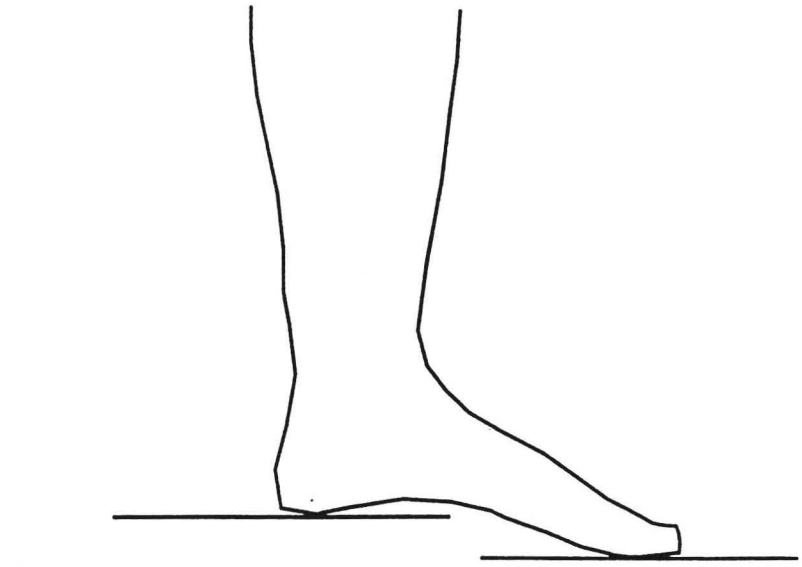

Figure 3-3 Ankle Joint Equinius

the clumsiness of gait created by the toe-in pattern.[52] This external tibial torsion, when greater than 20 degrees (normal is 20 degrees), increases the weight bearing force to the medial side of the foot. Therefore, the STJ undergoes abnormal compensatory pronation.

The abnormal STJ pronation can lead to medial knee pain and patellar malalignment due to the torsional stresses at the knee joint.[2,28] Furthermore, hip pain may result from strain to the external rotator musculature which must work hard to prevent toe-in.[53]

Leg Length Discrepancies.—Leg length discrepancies (LLDs) are another extrinsic deformity which can lead to increased STJ pronation. The LLDs can be divided into structural and functional leg length differences. Structural LLDs are true anatomic differences in the bone length of the femur, tibia, or both. A functional LLD is the shortening or lengthening of the limb due to joint contracture or muscle imbalance.[54]

Structural LLDs can be attributed to uneven lower extremity development in utero, fractures, sacral deformity, or unilateral coxa vara.[54] A functional LLD could result from ipsilateral hip flexor tightness which pulls the femur proximally, thus producing a short limb. Whatever the cause of the LLD, the most important biomechanical compensations occur at the STJ.[53] The STJ pronates on the long limb side in order to decrease the vertical height of the leg. In contrast, the STJ supinates on the short limb side in order to increase the length of the leg. The plantarflexion and adduction of the talus along with calcaneal eversion will

shorten the long leg by decreasing the height of the subtalar and talocrural areas. The opposite holds true for STJ supination.[3]

Once again, the associated signs and symptoms can exist when the STJ pronates to compensate for structural or functional LLDs. Table 3-1 illustrates the different forms of abnormal pronation previously discussed.

Abnormal Supination

Supination at the STJ is considered abnormal if the amount is excessive during any portion of gait or if supination occurs when pronation should.[2] Excessive supination is, essentially, a hypomobile deformity of the foot resulting from muscle imbalances, soft tissue contractures, and nerve disorders.[20] An individual presenting with excessive STJ supination has a decreased ability to function as a shock absorber or to adapt to terrain.[2,32,34] Unlike excessive pronation, an excessively supinated foot usually does not demonstrate a progressive breakdown in soft tissues. However, it is the inflexible supinated foot which leads to tissue inflammation and possible STJ destruction.[3]

The hypersupinated STJ, termed pes cavus foot, presents with a high medial longitudinal arch.[45,51] The etiology of a pes cavus foot is usually that of a neurologic disorder, contracture of soft tissues, or idiopathic.[3] Muscle group weakness of the extrinsic and/or intrinsic muscles can create a pes cavus foot. Weak dorsiflexors of the foot can cause the forefoot to drop, which can lead to a contracture of the plantar fascia and shortening of the gastroc-soleus. Also, the

Table 3-1—Abnormal STJ Pronation

I. Congenital Abnormal Pronation

A. Rigid deformities

1. Convex pas valgus (vertical talus)

2. Tarsal coalitions

B. Flexible deformities

1. Talipes Calcaneovalgus

II. Acquired Abnormal Pronation

A. Intrinsic deformities

1. Traumatic

2. Ligamentous Laxity

3. Bony Abnormalities

4. Compensatory pronation for intrinsic deformities

a. rearfoot varus

b. forefoot varus

c. ankle joint equinus

B. Extrinsic deformities

1. Rotational deformities of lower extremity

2. Leg length discrepancy

unopposed activity of the foot supinators, due to weak peroneals, produces a plantarflexed, adducted, and inverted foot position allowing for contracture of the plantar fascia.[2] Lastly, increased activity of the peroneus longus will plantarflex the first ray resulting in compensated STJ supination.[2,51]

Classification

This abnormal foot can be classified into three categories. These categories include congenital, intrinsic, and neurological mechanisms.

Congenital Talipes Equinovarus (Club Foot)

This congenital disorder, considered the most significant rigid disorder of the foot, is more prevalent in boys.[8,45] The exact etiology of clubfoot is unknown; however, genetic factors, malpositioned fetus in utero, and neuromuscular dysfunctions can be indicated.[8,45] The main clinical features in this deformity are inversion of the calcaneus, inversion and adduction of the forefoot, and equinus of the ankle and STJ.[51] There is also a contracture of the soft tissues (capsules, muscles, tendons, vessels, and skin) on the medial side of the foot. The underdevelopment of the peroneals as well as an underdeveloped and contracted gastroc-soleus is often evident.[8] The talus is the principal bone affected, with the anterior portion projecting medial and plantarly. The navicular is also displaced medially.[45] Clinically, the child bears weight on the lateral side of the foot (base of fifth metatarsal).

Treatment is started immediately after birth by the use of corrective casting. The goal of treatment is aimed at preventing bony and articular

damage. There is often irreversible muscle imbalance with recurrence possible later in life even after adequate correction early on.

Forefoot Valgus/Rigid Plantarflexed First Ray

Forefoot valgus deformity can be described as a forefoot which is everted relative to the bisection of the posterior aspect of the calcaneus in STJ neutral (figure 3-4).[5,20,47] The rigid plantarflexed first ray is described by ten neutral position of the first metatarsal head remaining below the level of the second through fifth despite pressure from an outside force. This configuration is similar to a plantarflexed first ray in the forefoot varus deformity except that in the latter the plantarflexed first ray is flexible. The etiology behind this condition includes congenital torsion of the head of the talus (resulting in eversion of the forefoot), post cerebral vascular accident, congenital first ray plantarflexion, or trauma.[1,5,35]

The clinical presentation of forefoot valgus with a plantarflexed first ray is that the medial portion of the forefoot contacts the ground before the lateral side. This causes the STJ to supinate in order to place the forefoot evenly on the ground.[1,5] The STJ compensates in the early stance phase (foot flat) of gait. Normal pronation, in this type of foot, does not occur. Therefore, the foot cannot effectively complete its role as a mobile adaptor. Because of this, the foot may develop postural instability to account for the decreased ability to adjust to uneven terrain.[5,20,47]

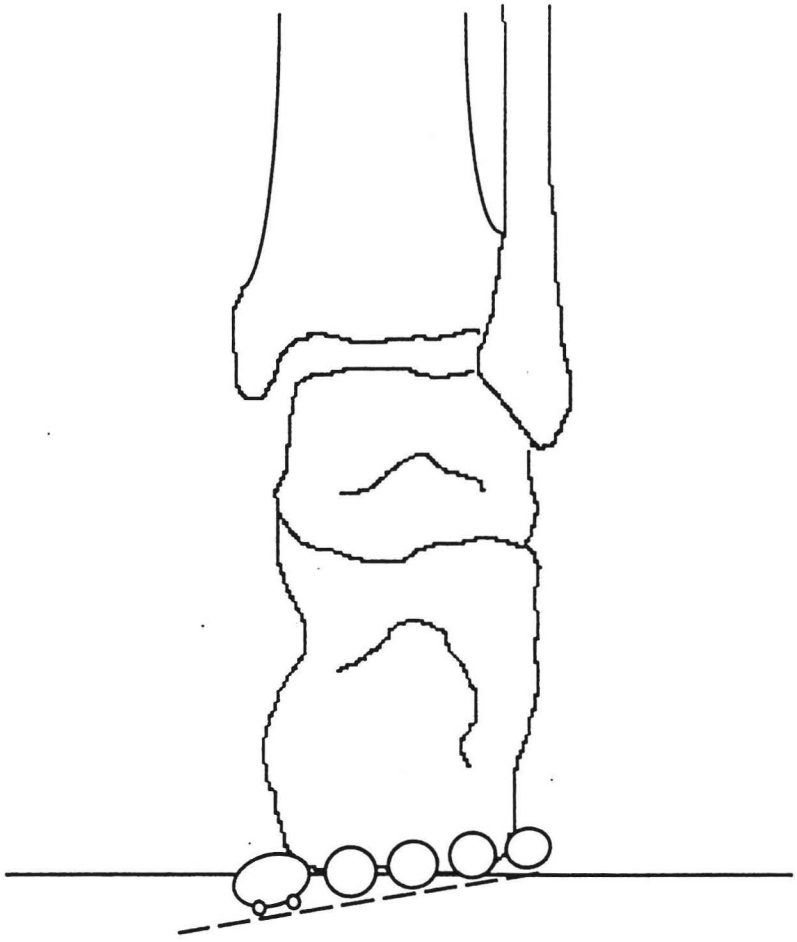

Figure 3-4 Forefoot Valgus

The ineffective pronation associated with forefoot valgus also compromises the ability of the lower extremity to absorb the ground reaction forces of gait. The leg is held in external rotation while the supination of the STJ is transmitted to a varus moment at the knee. The combined inefficiency of the foot to accommodate to terrain and to absorb shock will lead to lower kinetic chain pathologies. A few of these shall be discussed.

Stress fractures of the tibia are commonly seen in persons with the forefoot valgus syndrome because of inadequate shock absorption. This condition frequently occurs in runners because of the high impact forces present. Like in the rearfoot and forefoot varus deformities, IT band syndrome can be present in the pes cavus foot.[22] The excessively supinated STJ forces the tibia to externally rotate, thus producing a varus moment at the knee joint. This varus position of the knee pulls the IT band tightly against the lateral femoral condyle predisposing the insertion to frictional irritation. This varus at the knee may compromise patellar alignment thereby creating patellofemoral pain symptoms. Decreased shock absorption in the foot can lead to plantar fasciitis as this tissue must accommodate more of the ground reaction stresses.[47] Peroneal tendinitis is a symptom present in the forefoot valgus configuration. These muscles must contract vigorously to counteract the inverted position of the forefoot which can lead to microtrauma and inflammation. The inability to adjust to terrain through STJ pronation places an individual at risk for lateral ankle sprains. Lastly, the forefoot valgus presentation can lead to

painful and debilitating conditions of the foot, such as sesamoiditis and intermetatarsal neuralgia.

CHAPTER V

CONCLUSION

It was the author's objective to provide the clinician with the tools to understand the complexity of the STJ insofar that this joint will be included more frequently in the evaluation of lower extremity injuries. The STJ's many important functions in normal ambulation were demonstrated. It must act as a shock absorber by accommodating to terrain during heel strike and early midstance phases of gait. This is accomplished by STJ pronation. During toe-off portion of gait, the STJ supinates, thereby providing a stable base for propulsion.

Under normal circumstances, the biomechanics of the STJ allow the foot to accomplish its important functions. However, when structural deformities of the foot exist, the STJ must compensate to allow fluid functioning of the foot. These STJ compensations can lead to lower extremity pathologies which can be quite difficult to treat due to the repetitive mechanism of injury. Hence, it is important for the examiner to recognize the pathology and trace the symptoms to the source. Although treatment considerations were not integrated in this study, it is imperative that abnormal STJ mechanics be considered when treating lower extremity disorders.

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