Flexible Vertical Talus Syndrome: Its Relationship to Talipes Equinus

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The vertical talus syndrome is generally considered to be congenital or idiopathic; however, the author believes that the condition is common to more than one type of foot disease.

The syndrome is divided into two broad categories-flexible and rigid. In this paper, Dr. Rothbart discusses the flexible type and its relation to talipes equinus.

The etiology of a vertical talus is a controversial issue in the medical literature. Classically, this foot structure is considered congenital or idiopathic (1-6). However, everything from interuterine trauma to hormonal imbalances are incriminated (7). It is this author's opinion that the vertical talus is a radiologic finding common to more than one malady. For example, it is seen in a foot whose subtalar joint is pronated beyond the normal range of motion. Closed kinetic chain pronation is defined as eversion of the calcaneus and adduction, plantar-flexion of the talus. It is the excessive plantar-flexion of the astragalus that produces the flexible vertical talus. However, this pathomechanical entity must be carefully differentiated from the rather rare teratologic dislocation of the talonavicular joint which is present at birth, refractory to passive manipulation and unrelated to weight-bearing.

A flexible vertical talus commonly occurs in the presence of talipes equinus (8). An equinus deformity is defined as a limitation of ankle dorsiflexion. Specifically, at 50% stance phase (where the knee is fully extended and the subtalar joint is in its neutral position), the ankle joint is unable to dorsiflex at least 10 degrees (9-12). The importance of this range of motion becomes evident when one examines the kinesiology of a normal foot.

Normal Function (13). At heel contact, the vertical kinetic forces, which are at their maximum value, pass through the subtalar joint (14). The rearfoot prepares itself for this force by laying the entire inferior surface of the os calcis flush on the transverse plane. This wide base of support stabilizes the heel against the ground (15). Through 65-70% of stance phase the tibia is arcing anteriorly over the implanted foot. At midstance an angle of 80 degrees is formed between the longitudinal axis of the tibia and the plantar surface of the foot. That is, the foot is dorsiflexed 10 degrees to the leg. Concurrently, the tibia is fully extended at the knee joint. The propulsive phase of gait is initiated by heel lift, which occurs at between 65 and 70`7. of stance phase.

Abnormal Function. Premature heel lift suggests a limitation in the range of ankle joint dorsiflexion. When the talocrural joint locks, the forward momentum of the tibia forces the calcaneus off the ground. This shifts the

body's weight anteriorly through the midtarsal joint. However, in early stance phase the midtarsal joint is not prepared to receive this load. Instead, the active force of the body's mass acts as a strong pronator on the foot. A chain effect is set in motion which allows the heel to remain on the ground, but concomitantly produces: a) excessive pronation and partial subluxation within the midtarsal and subtalar joint (i.e., rocker-bottom foot), b) complete prolapsation of the foot's structural integrity, and c) a vertical talus (Fig. 1). It should be noted that the above set of pathodynamic events can be prevented by bending the knees or increasing the vertical oscillations within the lower extremity, clinically manifested as a bouncy gait.

Equinus Deformity (16). A talipes equinus deformity has several possible causes: a) It can occur from a shortage in the triceps surae. The muscle is physiologically normal, e.g., it is not contractured. However, it is simply too short for the bone and is referred to as a congenital equinus deformity. b) A tonic or clonic spasm of the gastrocnemius or soleus can limit the range of dorsiflexion at the ankle joint. Here the muscle belly is contracted or contractured and is referred to as an acquired equinus deformity. c) An ankle joint whose intrinsic bony structure prevents adequate dorsiflexion can produce an equinus deformity. This state is designated as an ankle block equinus deformity. d) A severe forefoot varum deformity.

A diagnosis of congenital talipes equinus is easily defended when examining the newborn; however, that same diagnosis becomes more formidable when evaluating adolescents with questionable past histories. In such cases a diagnosis of congenital talipes equinus is based upon: a) family history, b)

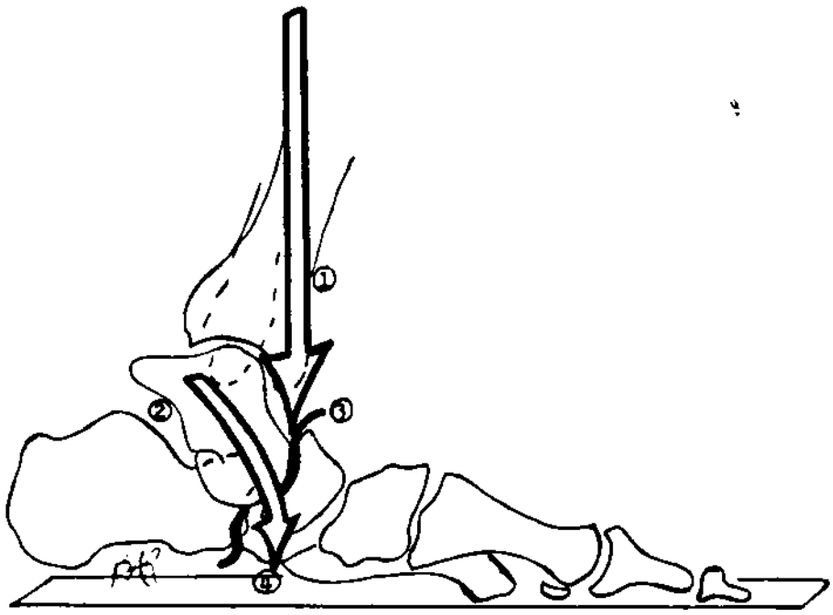

Figure 1. 1 , Forward displacement of body's vertical kinetic force; 2 , excessively pronated and subluxed subtalar joint; 3 , anteriorly broken syma-line; 4 , vertical talus.

x-rays, and c) the patient's response to treatment. The presence of an equinus condition in other members of the family suggests a hereditary factor. Radiographically, dorsoplantar and lateral views reveal a) flattening of the head of the talus, b) wedging of the navicular, c) decrease in the angle of calcaneal inclination, and d) a tendency toward a flexible vertical talus. If we do not see the above radiographic changes in adults or older children then an acquired condition of shorter duration is suspected. It must be kept in mind that a *chronic* acquired equinus deformity may present the same roentgenographic profile as the congenital form. In these instances the definitive diagnosis hinges on the patient's response to therapy. Utilizing an orthotic control that prevents excessive pronation, a patient with a congenital deformity either will break down the device or will not tolerate it unless the heel of the shoe is raised to compensate for the equinus. However, in many acquired equinus states, the orthotic will control pronation and result in stretching the muscle belly back to its normal physiological length if the cause of the spasm is resolved. Congentical equinus deformities are lengthened only by tendo achillis lengthenings, or tendon recessions.

Identification of the involved muscle group(s) is clinically obtained by noting the range of ankle dorsiflexion with the knee joint flexed and extended. Throughout the examination the subtalar joint must be held in its neutral position. This prevents the passive reconstruction of a rocker-bottom foot which can be mistaken for sagittal plane motion at the ankle joint.

A gastrocnemius type talipes equinus deformity is manifested *only* when the leg is extended on the thigh. Conversely, a short soleus exerts a limitation on ankle joint dorsiflexion regardless of the knee joint's position. Hamstring shortage, although not an equinus condition in the true sense of the term, creates an identical set of pathomechanical events by not allowing the knee joint to fully extend at midstance. The calcaneus is raised off the ground, limb instability is created, and the vertical talus syndrome ensues.

A chronic hamstring shortage, left untreated, can produce a dynamic contraction of the gastrocnemius: The knee joint is flexed, placing the belly of the gastrocnemius on a slack. The gastrocnemius compensates by contracture. Physiologically this reestablishes its muscular tonicity. Similarly, a shortage of the gastrocnemius can produce a dynamic contraction of the hamstrings.

An ankle block clinically mimics a short soleus in that a limitation of ankle dorsiflexion is seen with both the knee joint flexed and extended. A differential diagnosis is obtained with loaded lateral views of the dorsiflexed ankle joint and close inspection of the Achilles tendon with the knee flexed. A short soleus displays a chronically tight tendon, whereas x-rays delineate any bony involvement of the talocrural articulation.

Summary

The vertical talus syndrome is divided into two broad categories: flexible and rigid. Included in the former is the equinus deformity, and in the latter, the classical but rather rare congenital rigid convex pes planus.

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