The symptomatic adult-acquired flatfoot, also known as posterior tibial tendon dysfunction, continues to be an intriguing subject that has received considerable attention from researchers and clinicians over the past decade. Recent publications from multiple disciplines have enabled podiatric physicians to modify or even change their approach to evaluation and treatment of this common disorder.

Adult-acquired flatfoot is defined as a symptomatic, progressive deformity of the foot caused by a loss of dynamic and static supportive structures of the medial longitudinal arch. Although the condition begins with a loss of dynamic support from the posterior tibial tendon, the sequential ruptures of key ligaments in the ankle and hindfoot are the more important events leading to collapse of the arch and progressive disability of the patient.

The adult-acquired flatfoot almost always begins with a preexisting flatfoot and has a predilection to affect females over the age of 40. When the foot functions in a valgus position in the hindfoot and carries excessive body mass, the posterior tibial tendon has increased friction and gliding resistance along the medial malleolar gliding pulley. The combination of this mechanical strain with other metabolic factors will lead to a progressive attenuation and rupture of the posterior tibial tendon. However, visible change or collapse of the foot will not occur with simple rupture of the posterior tibial tendon. Instead, the loss of the posterior tibial tendon will cause a dysfunction of the foot during gait, which will then place progressive strain on key ligamentous structures in the hindfoot.

Increased load and strain will lead to rupture of the spring ligament, the interosseous talocalcaneal ligament and the long and short plantar ligaments. This will lead to a subluxation and triplane rotation of various joints that characterize the adult-acquired flatfoot: valgus alignment of the hindfoot, collapse of the medial longitudinal arch and abduction of the forefoot.

Emerging Concepts In The Staging Of Adult-Acquired Flatfoot

The challenge facing the clinician in the
initial evaluation of the adult-acquired flatfoot is determining the stage or severity of deformity in order to make appropriate treatment decisions. Currently, the accepted staging system for adult-acquired flatfoot relies on some variation of the original Johnson and Strom classification, which was originally published in 1989.6

There are numerous pitfalls with this classification primarily due to the reliance on subjective evaluation rather than objective findings. This classification defines Stage I adult-acquired flatfoot as tenosynovitis of the posterior tibial tendon with no evidence of collapse of the foot in comparison to the contralateral side. In Stage II, the posterior tibial tendon is attenuated or fully ruptured. Visible change in alignment of the foot has occurred but the deformity is still flexible. Stage III deformity is a rigid, non-reducible flatfoot deformity with complete rupture of the posterior tibial tendon. Myerson later added a Stage IV deformity, which is a valgus malalignment of the talocrural joint due to rupture of the deep deltoid ligament.7 Most updated variations focus on subdivisions of Stage II deformity.8-10 The biggest challenge for clinical decision making is with Stage II deformity. Stage II can have various levels of deformity, depending on the extent of ligament attenuation and stability of the medial column. Although initial treatment is almost always non-operative, the choice of modalities varies significantly depending on the level of deformity. The table “A Guide To The Updated Staging System For Adult-Acquired Flatfoot” on page 58 outlines the newer classification system along with key clinical exam features and also proposes certain treatment options that are relevant to each stage.9,10

In Stage I deformity, treatment focuses on treating the tenosynovitis. The fact that the posterior tibial tendon is still functioning in Stage I and there is no evidence of ligament attenuation means most of these patients can get successful treatment with short-term immobilization followed by long-term stabilization with custom functional foot orthotic therapy.11

In Stage II, the loss of ligament stabilization of the hindfoot causes a disruption of coupling or a lack of movement transfer between the foot and the leg. Foot orthoses are usually ineffective in this situation. Functional bracing with ankle foot orthoses has shown impressive results in resolving the symptoms of Stage II flatfoot and avoiding the need for surgery.12-16

In Stage III and IV flatfoot, functional bracing with more rigid devices is recommended but the results are not as positive as those for Stage II deformity.17 Surgery may be a better option for patients with

Here one can see the single foot heel rise test. Loss of stability of the midtarsal joint due to ligament disruption will cause plantarfexion of the rearfoot on the forefoot during contraction of the triceps surae.

Here one can see manual muscle testing for strength of the tibialis posterior. The examiner pushes against the plantar aspect of the first metatarsal and moves the foot into an everted and plantarflexed position. The examiner would ask the patient to push against his or her thumb.
Stage III or Stage IV flatfoot, but often other medical issues facing this patient population can increase complications and delay healing. In any case, the clinician must be able to evaluate the specific biomechanical findings carefully in each patient to choose the appropriate combination of surgical procedures to correct the deformity and restore mobility.

The following steps are recommended in the biomechanical exam of the patient presenting with a symptomatic flatfoot deformity. Since most cases of symptomatic adult-acquired flatfoot begin in one foot only, comparison to the asymptomatic side can be very helpful in measuring the progression of the deformity.

**What A Comprehensive Gait Analysis Can Reveal**

I suggest that the examination of the patient with symptoms of adult-acquired flatfoot begin with an evaluation of barefoot gait. Very quickly, the practitioner will learn the severity of the deformity and its symptoms. If the patient walks with a noticeable limp from significant pain, one may need to postpone the remainder of the biomechanical exam. Many of the clinical tests I describe here are invalid if pain causes splinting and reduced range of motion. In this case, I recommend immobilization with a walking boot for 14 days. After this period, the clinical examination may be much more accurate.

As with many of the clinical tests for adult-acquired flatfoot, gait analysis will allow a side-to-side comparison of deformity. The key differentiating feature comparing Stage I and II adult-acquired flatfoot is the fact that Stage I is characterized by tenosynovitis, but there is no visible collapse of the symptomatic foot in comparison to the contralateral side. While both feet look “flat and pronated,” one should evaluate the symptomatic side in Stage II for severity of rotation in all three body planes. As the flatfoot deformity progresses, three distinct changes occur. These changes are hindfoot eversion, forefoot abduction and lowering of the medial longitudinal arch, which will become accentuated during the late stance phase of gait.

Look for the timing of heel rise and evidence of inversion of the hindfoot during heel rise, which can indicate function of the posterior tibial tendon. Peak ankle joint dorsiflexion normally occurs at about 75 percent of the stance phase of gait. This is followed by rapid ankle joint plantarflexion, which will lead to visible heel rise. Heel rise will be delayed in Stage II flatfoot because the triceps has reduced leverage to plantarflex the entire foot as one stable unit.

Houck has shown that plantarflexion of the hindfoot will lower the medial longitudinal arch and dorsiflex the first metatarsal. Plantarflexion of the ankle occurs in the absence of heel rise as the rearfoot will plantarflex on the forefoot.
across an unstable midtarsal joint. The key features of Stage II adult-acquired flatfoot during gait are: increased ankle plantarflexion (without resulting heel rise); increased hindfoot eversion; increased first metatarsal dorsiflexion (lowering of medial longitudinal arch); and increased forefoot abduction.

After comparing foot alignment and function, evaluate the entire lower extremity as well as the hips, arms and shoulders for other postural alignment abnormalities. A common secondary deformity in Stage II and III adult-acquired flatfoot is the development of valgus alignment of the knee. This can cause symptoms and disability that will be as severe or worse than those in the foot itself.

**Essential Insights On The Static And Dynamic Stance Evaluations**

After watching the patient walk, ask him or her to stand next to a wall or countertop, and perform the single and double leg heel rise test. This is the single most important clinical exam to determine the stage of deformity in the adult-acquired flatfoot. Ask patients to touch the wall or countertop for balance only without pushing or pulling themselves upward.

The first test is the double leg heel rise test in which the patient does five consecutive heel rise maneuvers raising as high off the floor as possible. It is best to demonstrate these tests yourself to the patient in addition to providing verbal instruction. Look for the height that the patient can elevate the heels and also look for any evidence of inversion of the hindfoot as the heel rises off the ground. Look for asymmetry in both measurements.

Next, ask the patient to perform the single leg heel rise starting with the asymptomatic foot. Ask him or her to do five heel rises per leg and compare for height of heel rise and evidence of hindfoot inversion. Healthy patients should be able to lift their heel as high during single leg heel rise as they can during the double leg heel rise.

Lack of heel rise and inversion of the hindfoot is a reliable indicator of function of the tibialis posterior muscle as well as ligamentous stability of the midfoot. With loss of stability of the midfoot, the contraction of the triceps surae during heel rise will cause a plantarflexion of the rearfoot on the forefoot rather than a plantarflexion of the entire foot, which functions as one rigid lever. This loss of stability is a result of more than just an “unlocked” midtarsal joint. With a lack of ligament stabilization provided by the spring ligament and plantar ligaments, the forefoot becomes disconnected from the hindfoot. Inability to perform a single leg heel rise is the hallmark of Stage II adult-acquired flatfoot deformity. Impaired heel rise is present in early Stage II and diminishes to become completely absent in late Stage II.

While the patient is still standing, assess the “flexible versus rigid” deformity to dif-
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As part of the Silfverskiold test, compare passive range of dorsiflexion of the ankle with the knee flexed and extended as one can see above.

Diferentiate Stage II from Stage III flatfoot. The practitioner will attempt to move the patient’s foot into a rectus position at the hindfoot by manually inverting the heel or asking the patient to externally rotate the lower leg to supinate the hindfoot. If the hindfoot moves into a more vertical position from resting stance, the deformity is “reducible” and would still be “flexible” rather than rigid. However, the accurate evaluation of this test is quite subjective and evidence of flexibility versus rigidity can be difficult to determine, particularly with obese patients. One must correlate this evaluation with the off-weightbearing exam as well to stage the deformity.

The Hubscher maneuver, also known as the “Jack test,” can determine function of the windlass mechanism, which will be severely compromised by ligament disruption in the adult-acquired flatfoot. The examiner passively dorsiflexes the hallux and looks for movement transfer of supination of the hindfoot as well as external rotation of the tibia. In comparison to Stage I flatfoot, a Stage II deformity will lack tension in the plantar aponeurosis and connecting ligaments of the first ray, and no movement transfer will occur.

One should perform a modified Romberg test for balance with the patient in an upright standing position. It is often a revealing experience for both the clinician and the patient to learn how balance is severely compromised with the symptomatic foot. One must address this issue when prescribing a functional rehabilitation program for the patient with symptomatic adult-acquired flatfoot.

Pertinent Pearls On Conducting The Off-Weightbearing Exam

The off-weightbearing or open kinetic chain exam can reveal additional critical features of the patient’s own deformity to further aid in the staging and treatment recommendations. In addition to the general aspects of a biomechanical exam, I recommend the following additional tests.

Manual muscle testing for strength of the tibialis posterior. With the patient in a supine position, press your thumb against the plantar-medial aspect of the first metatarsophalangeal joint and move the foot into a plantarflexed and everted position at the ankle and subtalar joint. Ask patients to push their foot against your thumb. This will activate inversion of the foot using the tibialis posterior muscles. Assess the “push” or resistance produced by the patient’s symptomatic and asymptomatic foot. With this test, it is easy to detect a single grade of strength loss, which occurs with attenuation and eventual rupture of the posterior tibial tendon. Magnetic resonance imaging is rarely needed to document rupture because this test is very reliable when one combines it with other clinical findings.

Supination lag test. With the patient in a supine position and feet hanging in space off the end of the exam table, ask the patient to actively invert or supinate his or her feet. This is an open chain, active inversion movement of the ankle and hindfoot. Measure the ability of the feet to cross the midline of the body. With rupture of the posterior tibial tendon, the foot will not cross the midline. As deformity increases and rigidity of the hindfoot oc-
Staging of forefoot supinatus. As Stage II deformity increases, significant change will occur with frontal plane inversion of the forefoot and deformity in the medial column, which can provide a new sub-classification of Stage II as proposed by Myerson and cited by Haddad and colleagues. As the hindfoot moves into a valgus alignment during gait, the forefoot will invert in reciprocal fashion to keep the metatarsals flat on the ground. Furthermore, as ligaments progressively rupture in the flatfoot, deformity will occur in the medial column, causing a dorsiflexion alignment. As the forefoot inverts on the rearfoot and as the medial column dorsiflexes, an acquired forefoot varus deformity will occur. This is also known as forefoot supinatus. The severity of this deformity and ease of reduction will allow a sub-classification of Stage II.

To make an assessment of forefoot supinatus, evaluate the patient with him or her in a supine position, which is much easier for the patient than lying prone. In some cases, it may be more accurate to evaluate forefoot to rearfoot alignment with the patient in a prone position. The examination begins with the examiner holding the foot and grasping the heel, and moving the hindfoot to neutral or vertical. Then evaluate the frontal plane alignment of the forefoot to the rearfoot with the ankle dorsiflexed and then plantarflexed.

Myerson describes five classifications of Stage II AAF based upon this exam. In every case, one can correct the hindfoot to a vertical or neutral position.

A. Hindfoot valgus without residual forefoot supinatus.

B. Flexible forefoot supinatus. Inversion alignment of the forefoot becomes apparent when one corrects the hindfoot to neutral position, but the deformity reduces when the ankle plantarflexes.

C. Fixed forefoot supinatus. Inversion alignment of the forefoot does not reduce with ankle plantarflexion. The deformity is a frontal plane inversion across the midtarsal joint.

D. Forefoot abduction. This occurs at the tarsometatarsal joints, primarily at the first tarsometatarsal joint. One must determine this deformity by radiographic examination.

E. Medial ray instability. Ligament rupture leads to dorsiflexion instability of the first ray and can occur at the talonavicular joint, the naviculocuneiform joint or the medial cuneiform-first metatarsal joint, or any combination thereof. As with type C, this deformity does not reduce with plantarflexion of the ankle but the varus alignment of the forefoot is the result of dorsiflexion of the first ray rather than inversion of the entire forefoot.

It is helpful to assess true first ray instability versus forefoot inversion by holding the foot in the classic neutral suspension cast position with the forefoot...
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A Guide To The Updated Staging System For Adult-Acquired Flatfoot

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foot loaded and pronated at the midtarsal joint. The examiner then presses the first metatarsal downward in a plantarflexed direction. One can measure the relative flexibility or reducibility of the supinatus and make a visual assessment comparing first ray motion versus eversion of the entire forefoot. Using this assessment as well as weightbearing radiographs, one can make critical decisions regarding arthrodesis versus soft tissue corrective procedures on the medial column for stage II flatfoot.

Evaluation of the triceps and heel cord. Consider the powerful influence of the triceps in both the pathomechanics and the treatment of adult-acquired flatfoot. It is well recognized that the triceps through the tendo-Achilles is a significant deforming force in the progression of the adult-acquired flatfoot. When the tibialis posterior tendon fails to stabilize the hindfoot and midfoot, the triceps provides a plantarflexion force across the midfoot, which eventually leads to subluxation.

The valgus alignment of the hindfoot changes the direction and moment arm of the tendo-Achilles in the adult-acquired flatfoot. The rationale for the medializing calcaneal osteotomy is to realign the direction of force of the Achilles to provide an inversion moment on the rearfoot.

One can evaluate the deforming influence of the tendo-Achilles in the patient with any stage of flatfoot by performing a simple clinical test with the patient in a supine position. Starting with the hindfoot in neutral position, passively dorsiflex the entire foot with equal pressure against the plantar aspect of both the first and fifth metatarsal heads. With a valgus deforming tendo-Achilles, the hindfoot will noticeably move into a valgus position as the tendo-Achilles bears tension with passive ankle joint dorsiflexion. This rotation of the hindfoot into valgus with tensioning of the Achilles does not occur in healthy feet.

With the patient in a supine position, perform the Silfverskiold test to detect a contracture of the gastrocnemius versus a global contracture of the triceps or heel cord. Measure passive range of dorsiflexion of the ankle with the knee extended and then with the knee flexed. Significant reduction of dorsiflexion with the knee extended may be an indication for gastrocnemius recession.

In Summary

The adult-acquired flatfoot is a potentially disabling condition that results in significant biomechanical dysfunction of the human foot. One can evaluate and stage the progression of the disorder with numerous clinical tests. After assessing the stage or severity of the condition, the physician can select the appropriate conservative and surgical intervention.

Multiple studies have reported the success of non-surgical interventions, particularly for Stage II deformity, and one should consider conservative solutions before contemplating more disabling surgical procedures.

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References


Dr. Richie also writes a monthly DPM Blog for Podiatry Today. For recent blogs, visit http://bit.ly/xB1K0y.