

Understanding Adult-Acquired Flatfoot

■ By Séamus Kennedy, BEng (Mech), CPed

Adult-acquired flatfoot (AAF) is the term used to describe the progressive deformity of the foot and ankle that, in its later stages, results in collapsed and badly deformed feet. Although the condition has been described and written about since the 1980s, AAF is not a widely used acronym within the O&P community—even though orthotists and pedorthists easily recognize the signs of the condition because they treat them on an almost daily basis. AAF is caused by a loss of the dynamic and static support structures of the medial longitudinal arch, resulting in an incrementally worsening planovalgus deformity associated with posterior tibial (PT) tendinitis.

Over the past 30 years, researchers have attempted to understand and explain the gradual yet significant deterioration that can occur in foot structure, which ultimately leads to painful and debilitating conditions—a progression that is currently classified into four stages. What begins as a predisposition to flatfoot can progress to a collapsed arch, and then to the more severe posterior tibial tendon dysfunction (PTTD). Left untreated, the PT tendon can rupture, and the patient may then require a rigid AFO or an arthrodesis fixation surgery to stabilize the foot in order to remain capable of walking pain free.

Foot Mechanics

The symptom most often associated with AAF is PTTD, but it is important to see this only as a single step along a broader continuum. The most important function of the PT tendon is to work in synergy with the peroneus longus to stabilize the mid-tarsal joint (MTJ). When the PT muscle contracts and acts concentrically, it inverts the foot, thereby raising the medial arch. When stretched under tension, acting eccentrically, its function can be seen as a pronation retarder. The integrity of the PT tendon and muscle is crucial to the proper function of the foot, but it is far from the lone actor in maintaining the arch. There is a vital codependence on a host of other muscles and ligaments that when disrupted leads to an almost predictable loss in foot architecture and subsequent pathology.

The proper alignment of the foot's bones forms a robust arch when the facets settle snugly in place. Structural soundness is maintained by compression, much the way stones in a Roman arch remain in place. In an abnormal foot, if the facets become misaligned, the structure becomes “unlocked,” stability is lost, and excessive forces are placed on the muscles and ligaments.

The bones of a normal foot become naturally close packed as



Unilateral calcaneal eversion and forefoot abduction are typical signs in Stage II.

heel lift begins. This occurs automatically as the foot resupinates and the operation of the windlass mechanism pulls the plantar fascia taut. However, if the foot remains in pronation and fails to resupinate at the critical point of heel lift, the facets do not marry and the body weight, now directly overhead, acts as a deforming force on the unlocked joints. One clinical indication of this is prolonged pronation of the subtalar joint (STJ), seen as extended calcaneal valgus, late in the gait cycle. In addition, any process that

restricts the smooth motion of the first metatarsophalangeal joint (MPJ) and inhibits the windlass mechanism can leave the foot unlocked.

One can also view the foot as being supported by a web of ligaments and tendons that crisscross underneath the midfoot. The PT tendon courses from medial to lateral in an anterior direction, and the peroneal tendons cross lateral to medial. The long and short plantar ligaments and the spring ligament connect the tarsus bones. If the bones have not aligned correctly to form a rigid support structure for propulsion, these ligaments can begin to attenuate, leading to their deterioration and a loss in foot shape. In one study, in order to accomplish the collapse of the hindfoot and midfoot seen in Stages II and III PTTD deformities, researchers had to sever the spring ligament, plantar aponeurosis, deltoid ligament, talocalcaneal ligament, long and short plantar ligaments, and the medial calcaneal-cuboid ligament.¹ This proves that the severity of AAF goes far beyond being a strain on the PT tendon. Predisposing factors for AAF include obesity, diabetes, hypertension, and rheumatoid arthritis. Another reported risk factor is being a sedentary, middle-aged to elderly woman. The progression of AAF was originally classified into distinct stages by Johnson and Strom in 1989.² The stages and associated treatment protocols have since been further refined, and Stage II has been subdivided into five categories, A through E.

Treatment

Although AAF is not reversible without surgery, appropriate treatment should address the patient's current symptoms, attempt to reduce pain, and allow continued ambulation. In the early stages, orthotic and pedorthic solutions can address the loss of integrity of the foot's support structures, potentially inhibiting further destruction.³⁻⁵ As a general principle, orthotic devices should only block or limit painful or

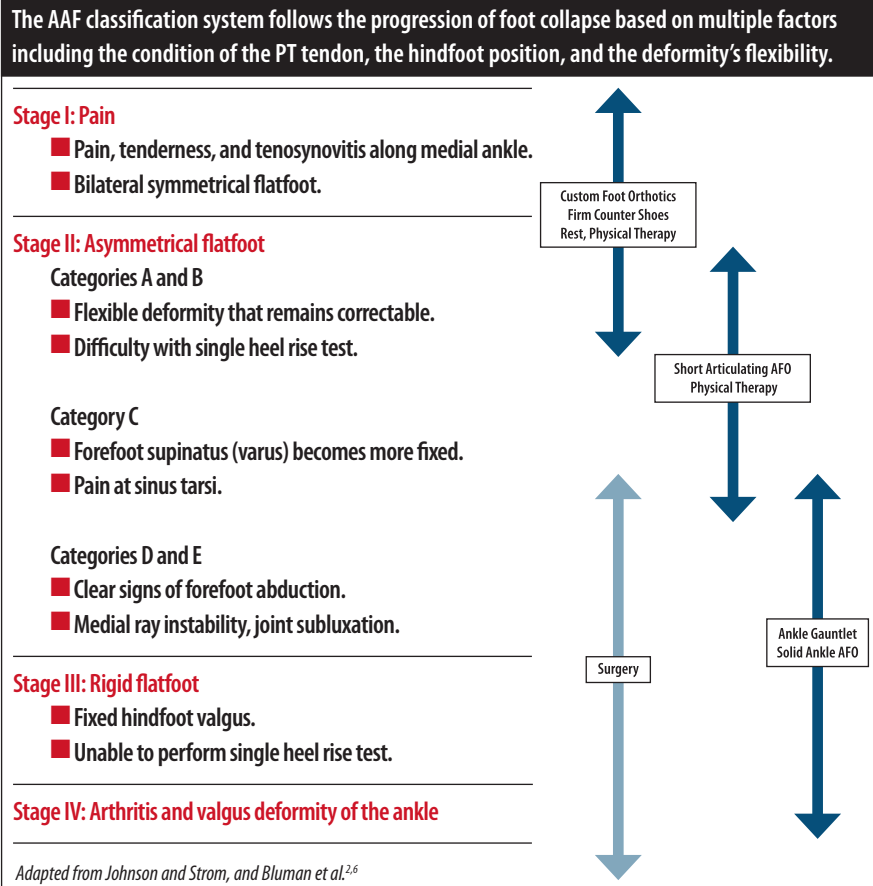
destructive motion without reducing or restricting normal motion or muscle function. Consequently, the treatment must match the stage of the deformity.

In Stage I, the patient can be prescribed custom foot orthotics and well-fitting, supportive shoes. The goal is to support the foot and, if possible, minimize the effects of late-stage pronation. Orthotic modifications, such as deep heel cups, medial heel skives, medial flanges, and rearfoot varus posts, can help redirect and alter ground reaction forces. In some cases, requesting a first ray cutout or kinetic wedge can restore lost motion at the first MPJ, which will enhance the stabilizing effect of the windlass mechanism. Some physicians recommend that patients with Stage I AAF wear hiking boots with high, stiff counters to reinforce the medial column and provide support above and below the ankle. Anti-inflammatory medications may also be prescribed.

As Stage II develops, foot orthotics may no longer offer sufficient support to control the foot and ankle complex. Early in Stage II, physical therapy to strengthen and rehabilitate the weakened PT muscle and tendon can be beneficial. In normal foot function there is an internal rotation of the tibia when the foot pronates, and an external rotation of the tibia during supination. As ligaments in the foot attenuate and eventually rupture, this important tibial coupling effect is lost; the foot becomes increasingly disconnected from tibial rotation. It becomes necessary to graduate the patient to an AFO that will control the foot and maintain the coupling of motion across the ankle joint.

Short, articulating AFOs, such as the Richie Brace®, are well suited to early Stage II treatment. The custom foot orthotic, deep heel cup, and high medial and lateral struts enhance the tibial coupling. Patients tend to accept these devices as they can often be worn in sneakers or extra-depth shoes. More importantly, using pivots at the ankle enables full range of motion in the sagittal plane and allows the patient to walk relatively normally. Hinged devices are advantageous as they still recruit regular muscle activity, which helps control forefoot abduction.

Later in Stage II and into Stage III it becomes necessary to provide more control. If the midfoot becomes destabilized and forefoot abduction continues, ankle gauntlets, such as Arizona-type AFOs, can be employed. Gauntlets made from rigid polypropylene are more restrictive and may require a change of footwear to a wider- lasted shoe with an open throat. Chukka-style or high-top shoes are recommended as they provide more support and help keep the AFO properly seated. A rocker sole can be added to the shoe to replace some of the lost ankle motion. There are also several surgeries that can be performed to selectively stabilize the major joints in the foot and improve



the biomechanics and foot function. In cases that are more advanced, such as if the patient is heavy or not a good surgical candidate, it may be necessary to consider a solid-ankle AFO or even a traditional double metal upright-style brace.

In its simplest form, AAF is recognized as the painful, incremental deterioration of the support structures of the normal foot. PTTD is just one step on a longer journey that typically begins with a preexisting flatfoot condition and late-stage pronation. When the bones of the foot remain unlocked late in midstance, the MTJ is compromised, undue strain is placed on critical ligaments, and proper structure in the foot is gradually lost. Appropriate orthotic treatment supports and retards the progress of AAF, especially in Stages I and II. In the worst cases, reconstructive surgeries or complete ankle immobilization may be necessary. For a more complete review of the etiology and staging of AAF, read “Biomechanics and Clinical Analysis of the Adult Acquired Flatfoot,” D. Richie, *Clinics in Podiatric Medicine & Surgery*, 2007 24 (4): 617–44. **O&P EDGE**

Séamus Kennedy, BEng (Mech), CPed, is president and co-owner of Hersco Ortho Labs, New York, New York. He can be contacted via e-mail at seamus@hersco.com or by visiting www.hersco.com

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