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Adult Acquired Flatfoot Deformity

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ABSTRACT

Tibialis posterior tendon insufficiency can be the cause or the result of Adult Acquired Flatfoot Deformity. Other causes include midfoot arthritis, spring ligament rupture, accessory navicular detachment, acute rupture of tibialis posterior tendon or deep deltoid ligament, hallux valgus. In the early stages the hindfoot remains flexible, whilst later it can become rigid. Ankle valgus alignment and degeneration can also be present in some patients. Orthotics and physiotherapy constitute the first line treatment. Open or endoscopic tibialis posterior tendon debridement is indicated in the earlier stage, when no deformity is present. Calcaneal medialisation osteotomy and flexor digitorum tendon transfer are usually performed in flexible flatfeet, whilst additional medial (Cotton medial cuneiform osteotomy or midfoot arthrodesis) and/or lateral column (calcaneal lengthening osteotomy or calcaneocuboid arthrodesis) procedures, can be considered if the forefoot remains supinated after hindfoot neutralisation. Rigid feet can require corrective arthrodesis of the subtalar, talonavicular +/- calcaneocuboid joints. Gastrocnemius or Achilles tendon lengthening are often required to allow deformity correction. Ankle arthritis requiring usually tibiotalocalcaneal arthrodesis.

KEY WORDS: Flatfoot; planovalgus; pes planus; tibialis posterior

1. Introduction

The term "Adult Acquired Flatfoot Deformity" (AAFD) includes a variety of disorders that result in foot hyperpronation, valgus heel alignment and dropped medial arch of the foot. In the past the term "Tibialis Posterior Tendon Dysfunction" has been used, instead, to describe the same foot deformity [1-7]. However it has been realised that primary causes (**Table 1**), other than loss of strength and function of tibialis posterior (TP), may also be responsible for the development of pes planus deformity. Thus, TP tendinopathy and/or degeneration may be secondary to the development of planovalgus foot deformity, due to the increased stresses on the medial side of the foot during stance and gait. One has to also distinguish between deformities that first occur in adult life and those in childhood and adolescence, as the latter have different causes and a different management approach may be needed. Of course, children's flatfoot deformities will gradually result in worsening deformities in adults.

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TABLE 1. *Causes of pes planus*

Tibialis posterior tendinopathy or rupture (80%)
Spring ligament degeneration or acute rupture
Midfoot joints arthritis
Tarsal coalition
Hallux Valgus
Rheumatoid arthritis
Neurological disorders

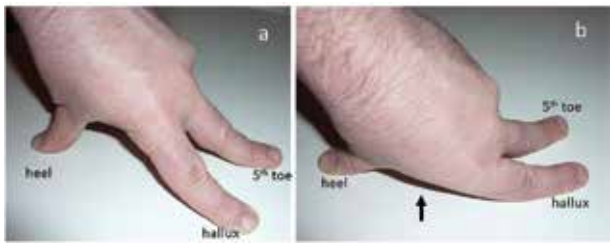

Fig. 1: *In this model a human hand stimulates the standing foot. When the arch is normal (a), the forces are distributed in a balanced fashion between heel, hallux and 5th toe. In the presence of a dropped medial arch (b), the medial aspect is overloaded (arrow) during stance.*

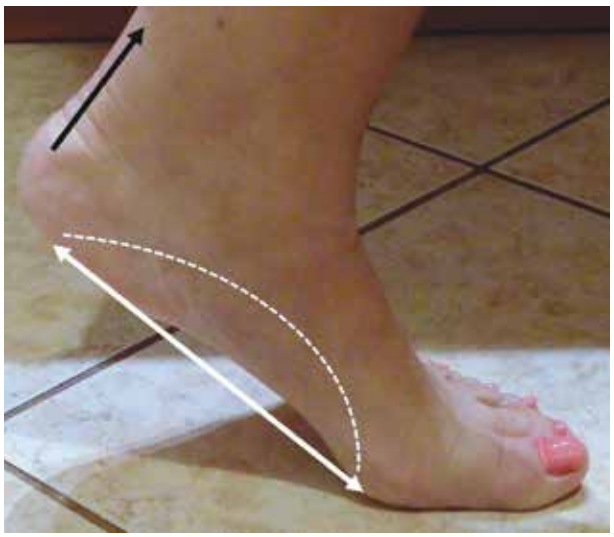

Fig. 2: *The action of the gastroc-soleus complex, through the Achilles tendon (black arrow), results in tightening of the plantar fascia (double ended white arrow), stabilisation of the midfoot and the medial arch, to allow the push-off phase of gait.*

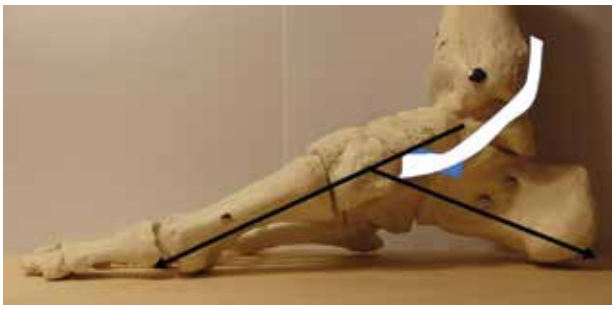

Fig. 3: *The foot medial arch is a result of the anatomic relation between the medial column and the calcaneus (black arrows), connected by the talus. The calcaneonavicular ("spring") ligament (blue), and tibialis posterior tendon (white) support the medial aspect of the talus.*

2. Clinical Anatomy, Pathomechanics and Aetiology

Some basic anatomic and biomechanical principles have to be taken into consideration, in order to understand the development of foot deformities. The foot functions as a tripod (**Fig. 1**), with the pressures being distributed between the heel, the first ray (1st metatarsal and hallux) and the 5th ray (5th metatarsal and 5th toe), during stance. The "windlass mechanism" is essential, to allow lifting the heel off the ground [1,2] (**Fig. 2**) .

The lateral foot column consisting of the calcaneus, cuboid and 4th, 5th metatarsals is relatively "flat", almost parallel to the ground, whilst the medial column (talus, naviculum, medial cuneiform, 1st metatarsal, hallucal phalanges), "descends" at an angle to the ground, in the standing foot. The middle rays (intermediate and lateral cuneiform bones and 2nd, 3rd metatarsals), are connected with strong ligaments, have little flexibility and connect the two columns distally. The normal medial arch of the foot is thus formed because of the inclination of the medial column, in relation to the calcaneum. The talus "sits" on the calcaneum (posterior and middle facets), whilst the talar head is not supported by osseous structures but is suspended on the so called "spring" (calcaneonavicular) ligament and is dynamically stabilised by the tibialis posterior tendon (**Fig. 3**). Thus, the medial foot arch consists of the 1st metatarsal, medial cuneiform, naviculum, talus and calcaneus. The foot arch resembles the "Roman arch" in terms of engineering

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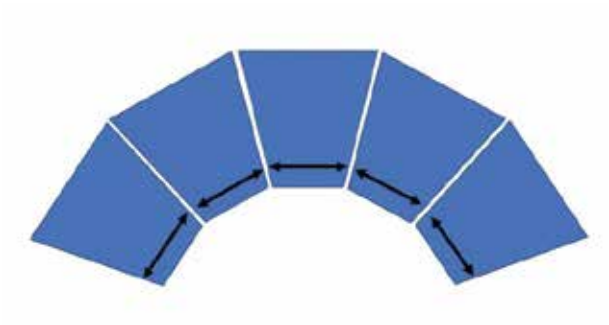

Fig. 4: *Roman arch: Its structural integrity and stability are based on the principle of balancing opposing forces.*

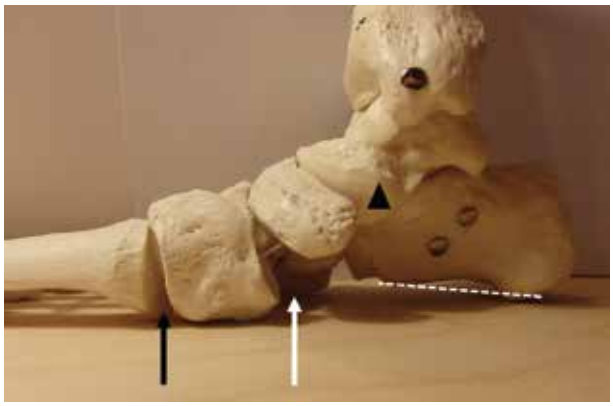

Fig. 5: *Arthritis at the first tarsometatarsal (black arrow), or medial naviculocuneiform (white arrow) joint, results in loss of integrity of the medial arch. The medial soft tissue structures (spring ligament, tibialis posterior tendon) cannot provide enough stability, the talar head shifts medially, and the talonavicular joint becomes uncovered (black triangle). The subtalar joint is also destabilised and calcaneal pitch angle drops (white dotted line). The foot appears flat.*

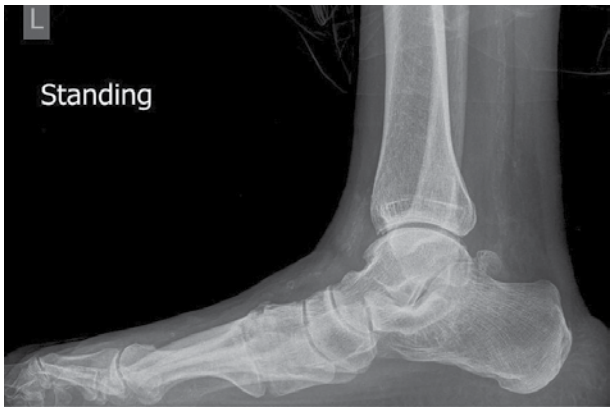

Fig. 6: *Degenerative changes at the first tarsometatarsal joint resulted in midfoot collapse and planovalgus deformity.*

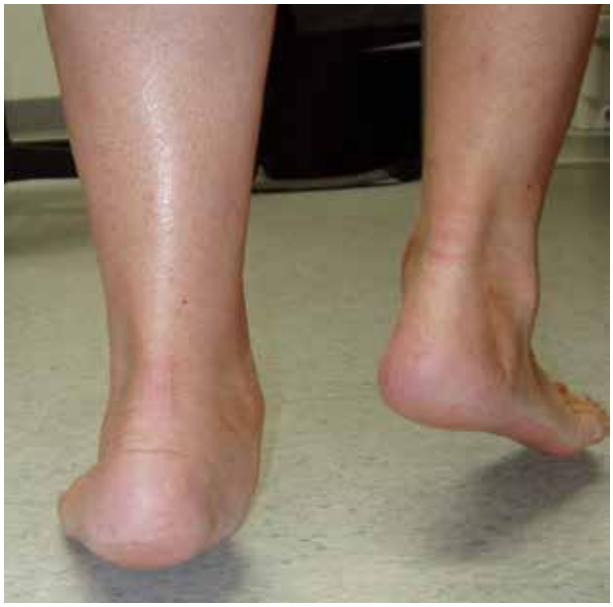

Fig. 7: *The left heel (pes planus deformity) does not invert (remains valgus), during heel rise.*

(**Fig. 4**). In the foot, bones are connected with strong ligaments, and as mentioned earlier, the action of muscles, tendons and the tightening effect of the plantar fascia (windlass mechanism) [1,2].

Any reason that causes soft tissue laxity in the medial column and dysfunction of the windlass mechanism, can result in drop of the medial arch. Midfoot joints (tarsometatarsal, naviculocuneiform) arthritis results in loss of cartilage, and thus the ligamentous structures will loosen, as the reactive forces between the bones forming the medial column, cannot maintain stability (**Fig. 5**). This is seen on lateral foot weight bearing radiographs as "midfoot sag" (**Fig. 6**). Secondarily, due to biomechanical changes, the foot hyperpronates, cannot supinate, medial structures become overloaded during gait, TP tendon is "overused" and can become tendinopathic. In such case, midfoot arthritis (post-traumatic or idiopathic) is the primary condition and TP dysfunction the result [3-7].

The talus has no tendon attachments, thus the dynamic stabilisers of the hindfoot act beyond the Chopart's joint. This makes the talus prone to dis-

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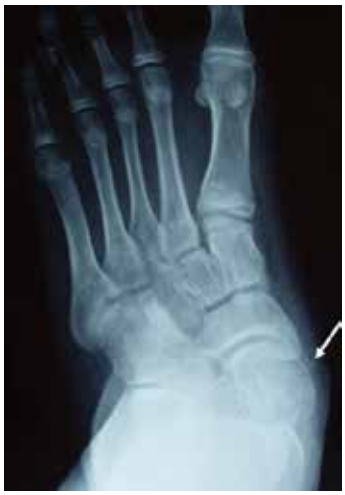

Fig. 8: *Talonavicular joint becomes incongruent and the talar head uncovered medially (arrow), in advanced pes planus.*

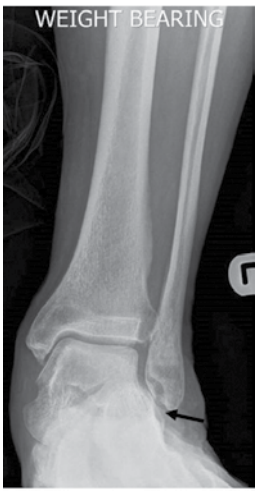

Fig. 9: *Lateral pain can occur as a result of impingement between fibula and calcaneus in severe heel valgus alignment.*

placement and loss of normal anatomic relationship, in cases of imbalance of the acting forces around the talus. This, for example, could be a result of TP weakness, because of acute rupture (rare), or tendinopathy (common). TP has a wide attachment of the naviculum and some fibers onto the medial cuneiform, and its action "locks" the talus and the talonavicular joint during the midstance phase of gait, produces initial plantar flexion and supination of the foot, allowing tightening of the plantar fascia (windlass mechanism), and the transverse tarsal joints, increases the strength of push-off, and finally the heel inverts at the subtalar joint and raise from the ground. Thus, TP dysfunction does not allow normal foot supination and heel inversion (**Fig. 7**). The heel remains in valgus, and peroneal tendons' action remains unopposed, thus resulting in increasing pronation, and a vicious cycle starts. After some time, the valgus heel alignment is worse, so the Achilles tendon attachment is transferred laterally. This results in abnormal mechanics of the gastroc-soleus complex, thus affecting the windlass mechanism [1,2].

Another reason for the foot to become flat is injury to the calcaneonavicular (spring) ligament. Loss of support of the talar head can result in overload of TP (dynamic stabiliser), and can subsequently lead to TP tendinopathy, pain, progressive planovalgus deformity, as mentioned earlier. Often, in long-standing AAFD, these two conditions (TP tendinopathy and spring ligament degeneration or rupture) co-exist, and it is difficult to say which is the primary initiator of the biomechanical events causing deformity [1-7].

A proportion of adult patients will present with worsening pes planus deformity that started in adolescent life, as a result of tarsal coalition. Some movement at the subtalar joint may be allowed, in fibrous coalitions, but generally the foot is chronically stiff. At some stage, due to the abnormal mechanics, the subtalar joint becomes arthritic and painful. TP tendinopathy may result as a secondary effect of the medial foot overload and the progressive deformity, that can be made worse by peroneal tendons overuse and secondary spasticity, that drives the foot into hyperpronation [8].

Hallux valgus (as the primary problem) may also result in progressive hindfoot valgus deformity as the function of the foot as a "tripod" is affected (**Fig. 1**). Medial shift of load during gait, will put TP and Achilles tendons in suboptimal position of action, and will initiate the sequence of pathomechanical events mentioned above.

In progressive planovalgus foot deformities, the heel remains in valgus, and a side effect is the functional shortening of the gastroc-soleus complex and the Achilles tendon.

Gradually, foot hyperpronation causes permanent changes in the alignment of bones (stiff deformity). The talus remains adducted and the talonavicular

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TABLE 2. *Johnson & Strom classification of Tibialis posterior tendon dysfunction*

	Stage I	Stage II	Stage III
Pain	Mild, medial	Moderate, medial	Severe, medial and lateral
Examination			
Swelling, tenderness	Mild, tenderness along TP	Moderate, tenderness along TP	Significant, tenderness along TP
Heel-rise test	Normal	Weakness	Weakness
"Too many toes" sign	Absent	Present	Present
Deformity	No	Yes, flexible	Yes, fixed
Pathologies	Normal TP, paratendinitis	Longitudinal tears of TP (tendinopathy)	Disrupted TP (severe tendinopathy)
Images	Normal	Deformity	Arthritis
Treatment	Conservative, tenosynovectomy	Flexor digitorum longus transfer	Triple arthrodesis

(TP: Tibialis posterior tendon)

joint becomes unstable and the talar head appears "uncovered" in the dorsoplantar weight bearing radiographic view (**Fig. 8**). The calcaneus cannot invert and remains in excessive valgus, and this can cause lateral ("subfibular") impingement and pain (**Fig. 9**). Thus, talonavicular and subtalar joints become eccentrically loaded and degenerative changes can occur over the years. Lateral loading at the ankle joint may cause ankle arthritis, as well, in some patients. The forefoot is affected as well, and often hallux metatarsophalangeal joint arthritis and hallux rigidus may accompany pes planus deformities.

3. Clinical presentation and diagnosis

Patients usually present with pes planus deformity and medial +/- lateral hindfoot pain. Sometimes they complain of easy fatigue when walking. Often, they have symptoms associated with the forefoot (big toe pain at the hallux MTP joint, or metatarsalgia, related to forefoot overload). Others may also have heel pain at the plantar (plantar fascia origin) or posterior (Achilles tendon) aspect. Thorough clinical examination is essential. The patient should be examined standing, gait should be observed and also non-weight bearing to assess joints active and passive range of motion, and the tender spots or areas in the patient's foot. Silfverskioldt test [9] is essential to assess calf muscle tightness (**Fig. 10**). The patient is asked to perform the "single heel rise" test (**Fig.** 7) to evaluate function of TP and flexibility of the subtalar joint.

Plain radiographs (ankle and foot weight bearing views) are needed (**Fig. 6, 8, 9**). More advanced imaging can be required. Ultrasound scans are helpful in examining TP tendon, whilst MR imaging to assess the condition of tendons, potential degeneration of joints, and to reveal/exclude the possibility of tarsal coalition in stiff subtalar joints. CT co-registered bone scan (SPECT-CT) can also be helpful in identifying the painful arthritic joints [10].

4. Classifications

Over the years several classification systems have been proposed. Johnson and Strom's classification (1989), distinguished between three stages of TP tendon dysfunction, based on the location and intensity of pain, presence of deformity, and hindfoot flexibility [11,12], (**Table 2**).

Myerson was later credited with the modification of Johnson and Storm's classification, describing a fourth stage, characterised by valgus alignment and lateral arthritis at the ankle joint [13]. Myerson proposed in 2007 [14] a comprehensive, detailed, but also quite

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TABLE 3. *Myerson's AAFD classification*

Stage	Clinical findings	Imaging	Treatment
I			
A	TP tenderness, normal anatomy	Normal	Immobilisation, orthotics, NSAID's, tenosynovectomy
B	TP tenderness, normal anatomy	Normal	
C	Slight hindfoot valgus, normal anatomy	Slight hindfoot valgus	
II			
A1	Supple hindfoot valgus, flexible forefoot varus	Hindfoot valgus, Meary's line disrupted, Loss of calcaneal pitch	Orthosis, medial displacement calcaneal osteotomy, Achilles tendon or gastrocnemius lengthening and flexor digitorum longus transfer if deformity corrects only with ankle plantar flexion
A2	Supple hindfoot valgus, fixed forefoot varus		Orthosis, medial displacement calcaneal osteotomy, flexor digitorum longus transfer, Cotton osteotomy
B	A2 + forefoot abduction	Talonavicular joint uncovered, forefoot abduction	Orthosis, medial displacement calcaneal osteotomy, flexor digitorum longus transfer, lateral column lengthening
C	B + medial column instability, first ray dorsiflexion with hindfoot correction, sinus tarsi pain	First tarsometatarsal plantar gapping	Medial displacement calcaneal osteotomy, flexor digitorum longus transfer, Cotton osteotomy or medial column fusion
III			
A	Rigid hindfoot valgus, lateral hindfoot pain (sinus tarsi)	Subtalar joint space loss, angle of Gissane sclerosis, hindfoot valgus	Triple arthrodesis or custom bracing if not surgical candidate
B	A + forefoot abduction	A+ forefoot abduction	A+ lateral column lengthening
IV			
A	Supple ankle valgus	Ankle valgus	Surgery aiming at plantigrade foot+ deltoid reconstruction
B	Rigid ankle valgus		Tibiotalocalcaneal arthrodesis

complicated, classification system, that takes into consideration clinical appearance, radiographic findings, joints flexibility or rigidity and proposes treatment. Myerson's classification describes four stages and their subdivisions (**Table 3**).

Although this is a difficult classification to remember, studying the classification, one can become familiar with the different pathologies and deformities that are evident in AAFD. Furthermore, it takes into consideration the position of the forefoot and midfoot (tarsometatarsal joint 1) flexibility or rigidity.

Parsons et al. [15] suggested that stage II should be subdivided based on the severity and flexibility of forefoot supination when the hindfoot is brought

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TABLE 4. *Raikin's classification of AAFD*

Stage	Hindfoot	Midfoot	Ankle
Ia	Posterior tibial tendon tenosynovitis		
		Neutral	Neutral
Ib	Posterior tibial tendinitis without deformity	Mild flexible mid-foot supination	Mild valgus (<5°)
IIa	Flexible planovalgus (<40% talar uncoverage,<30° Meary's angle, incongruency angle 20°–45°)	Mid-foot supination without radiographic instability	Valgus with deltoid insufficiency
IIb	Flexible planovalgus (>40% talar uncoverage, >30° Meary angle, incongruency angle >45°)	Mid-foot supination with instability	Valgus with deltoid insufficiency and tibiotalar arthritis
IIIa	Flexible planovalgus (<40% talar uncoverage,<30° Meary's angle, incongruency angle 20°–45°)	Arthritic changes isolated to medial column	Valgus secondary to bone loss in lateral ankle compartment (deltoid normal)
IIIb	Flexible planovalgus (>40% talar uncoverage, >30° Meary angle, incongruency angle >45°)	Medial and middle-column mid-foot arthritic changes	Valgus secondary to bone loss in lateral ankle compartment and deltoid insufficiency

into neutral position. This is a clinically significant observation, as it can affect the choice of surgical procedures that are needed, to allow the foot to be plantigrade after hindfoot correction.

Raikin et al. [16] proposed another classification in 2012, taking into account the condition of the hindfoot, midfoot and ankle (**Table 4**).

Raikin's classification system is quite complex and maybe not so "user-friendly". One can note that stage I is characterised by neutral hindfoot, stages IIa and IIIa by moderate hindfoot deformity, whilst stages IIb and IIIb by severe hindfoot deformity. Arthritic changes in the midfoot are seen in stage III, whilst different degrees of ankle deformity and/or instability are seen in stages II and III.

Interestingly, none of the above classifications are validated. It seems that Johnson and Storm's classification system (from 1989) is the simplest and most widely used. Stage I is characterised by medial pain, TP tenosynovitis and no deformity. Nonoperative management is recommended. In stage II disease, we are dealing with flexible planovalgus deformities and significant weakness and degeneration/tears of TP. Calcaneal osteotomy for deformity correction and tendon transfer is indicated. Stage III includes stiff deformities with arthritic changes. One has to take into consideration stage IV (credited to Myerson) that describes ankle valgus (flexible or rigid), that usually required tibiotalocalcaneal arthrodesis. It is important to remember midfoot flexibility versus rigidity and Parson's remark about and forefoot alignment after hindfoot correction, as this may have implications on surgical treatment options [15].

5. Treatment

Irrespective of staging of AAFD, nonoperative management is indicated first for 6 months, before any decisions regarding corrective surgeries are met. An exclusion to this concept includes acute soft tissue injuries (e.g. tibialis posterior tendon or deep deltoid ligament ruptures) that result in rapid onset development of medial hindfoot instability and progressive deformity, especially in younger patients. In those patients early surgical management may be indicated.

5.1 Nonoperative treatment

Non-surgical management options include modifications of activities, weight loss, analgesics and anti-inflammatories, physiotherapy (to strengthen TP and functionally lengthen the calf muscles) and orthotics. Various types of orthotics have been described for AAFD management. These include insoles, ankle

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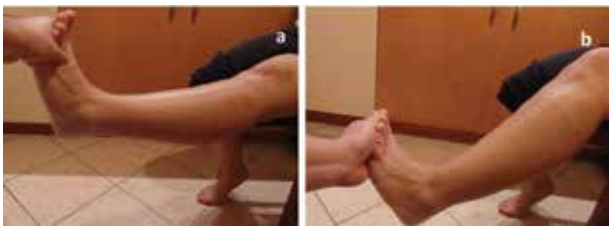

Fig. 10: *Gastrocnemius muscle origin is at the femoral condyles (proximal to the knee), so it bridges two joints (knee and ankle). In this case the ankle cannot be dorsiflexed beyond neutral* ***(a)****, thus calf muscles are tight. When the knee flexes* ***(b)****, the gastrocnemius muscle relaxes, and the ankle can be dorsiflexed more. This shows that gastrocnemius, only, is tight. If increased ankle dorsiflexion could not be achieved even with the knee flexed, then not only the gastrocnemius, but the gastroc-soleus / Achilles tendon, are tight. This has implications in management (which structure – gastrocnemius muscle, or Achilles tendon - to release).*

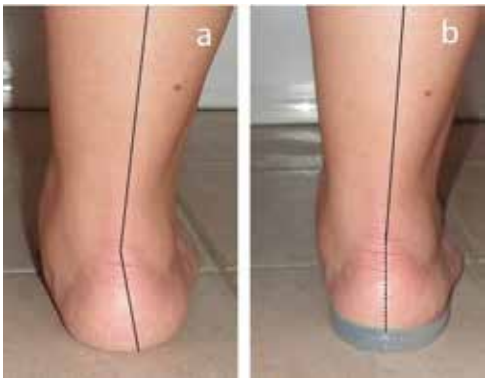

Fig. 11: *In flexible planovalgus foot deformity, the valgus heel* ***(a)*** *is neutralised in relation to the tibia (black lines), using a medial heel wedge insole* ***(b)****.*

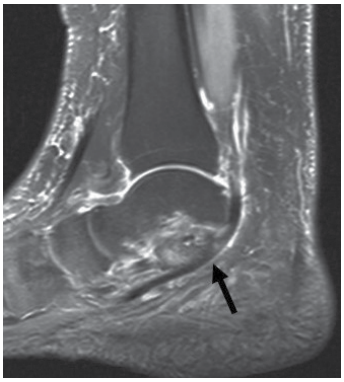

Fig. 12: *MRI showing significant tendinopathy of tibialis posterior tendon (arrow).*

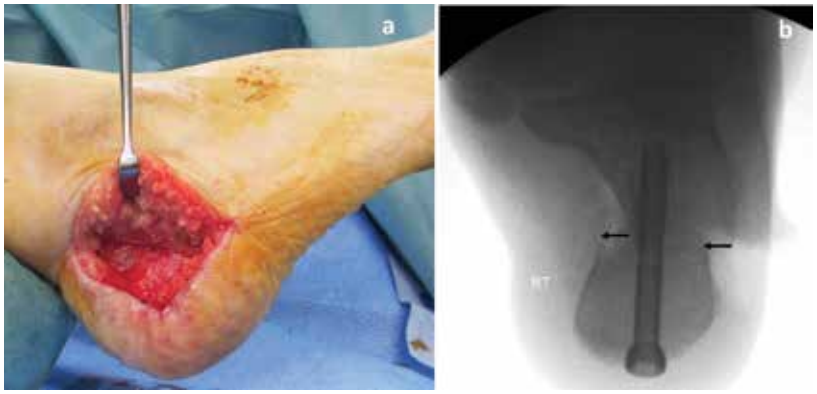

Fig. 13: *A lateral heel "L"-shaped incision* ***(a)*** *can be used to perform calcaneal osteotomy. The calcaneal tuberosity is shifted medially, as shown on intraoperative axial fluoroscopy view* ***(b)****.*

braces, and custom-made boots. In flexible deformities, corrective insoles (with medial heel corrective wedge and soft arch support) may be sufficient. As the foot is flexible, the corrective medial heel wedge may be able to restore neutral hindfoot alignment (**Fig. 11**). It is important that soft arch support is used, to avoid applying pressure to the painful medial arch of the foot if hard materials are used. It is important to emphasize that the heel wedge (and not the arch support), is the corrective part of the insole. In rigid deformities on the other hand, one should only use accommodative (not corrective) insoles, as the stiff joints will not allow neutralisation of hindfoot alignment. Ankle braces and rigid supportive shoes are more ap-

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Fig. 14: *Gastrocnemius muscle contracture often requires release, at proximal medial gastrocnemius approx. 2.5 cm below the knee crease (a), or at the musculotendinous junction (b). If the gastroc-soleus complex is tight, Achilles tendon lengthening through a percutaneous "triple-cut" can be performed (c).*

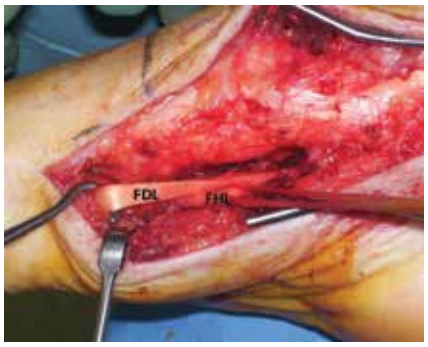

Fig. 15: *Flexor digitorum longus (FDL) tendon is harvested in the midfoot region, after it is dissected carefully from Flexor hallucis longus (FHL).*

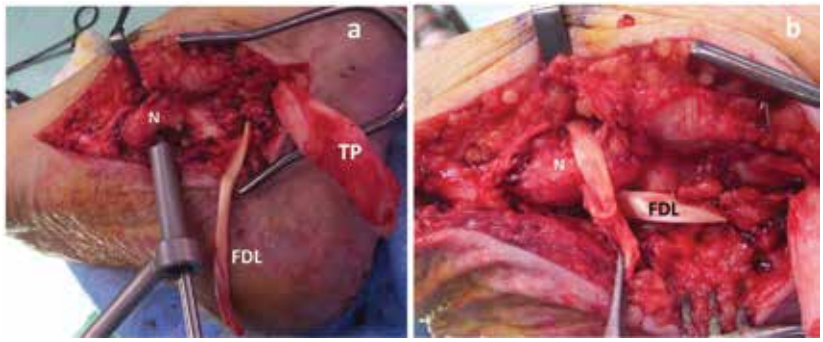

Fig. 16: (a)*: Tibialis posterior tendon (TP) is significantly thickened and tendinopathic. Flexor digitorum longus (FDL) has been retracted proximally, and a 5mm tunnel is drilled in the navicular bone (N).*
(b)*: FDL is passed through the osseous tunnel from plantar to dorsal, and once tightened with the foot in inversion, it is sutured to itself.*

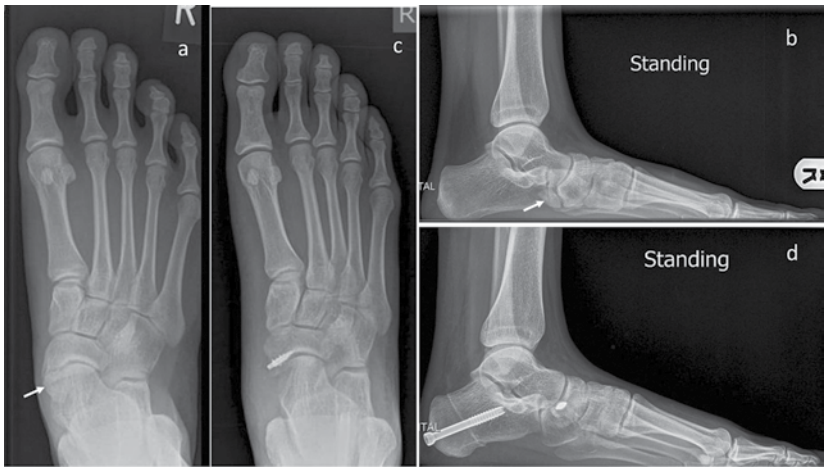

Fig. 17: *A 22- year old patient has significant medial pain and worsening planovalgus deformity, as a result of a "foot sprain" that caused detachment of the accessory navicular bone (arrow; a,b). Calcaneal medial shift osteotomy was performed to correct hindfoot valgus, whilst the accessory naviculum was excised and tibialis posterior was re-attached using an anchor. Very good deformity correction was achieved (c,d).*

propriate for rigid AAFDs (e.g. in stages III and IV, according to Johnson and Storm) [1,3-7].

5.2 Surgical treatment

Surgery is required when nonoperative management has failed and the patients accepts the risk and the recovery time associated with surgery.

Acute TP or deltoid ligament ruptures, may require early surgical reconstruction, to avoid progressive deformity. Often the ruptured tissues may be degenerate and augmentation using autologous tendon grafts, allografts, or orthobiologics. If the injury is chronic (beyond six weeks) and a degree of hindfoot valgus is already present, calcaneal medialisation osteotomy is also needed.

In feet without significant deformity and confirmed

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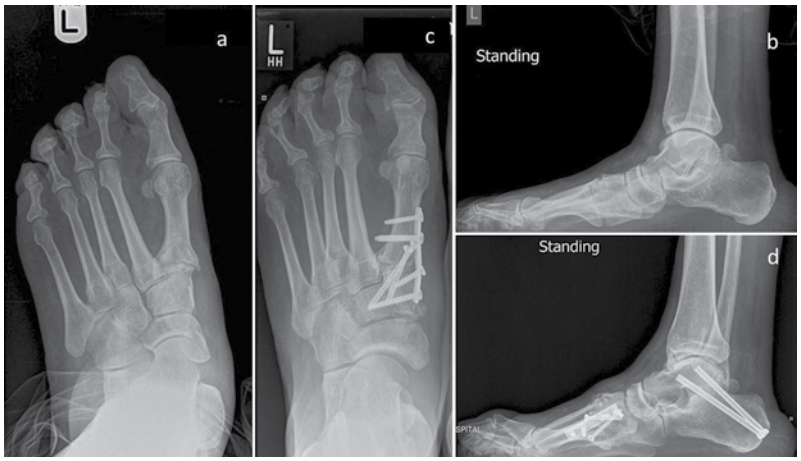

Fig. 18: *A complex deformity (a,b) required corrective fusions of the first tarsometatarsal and the subtalar joints (c,d).*

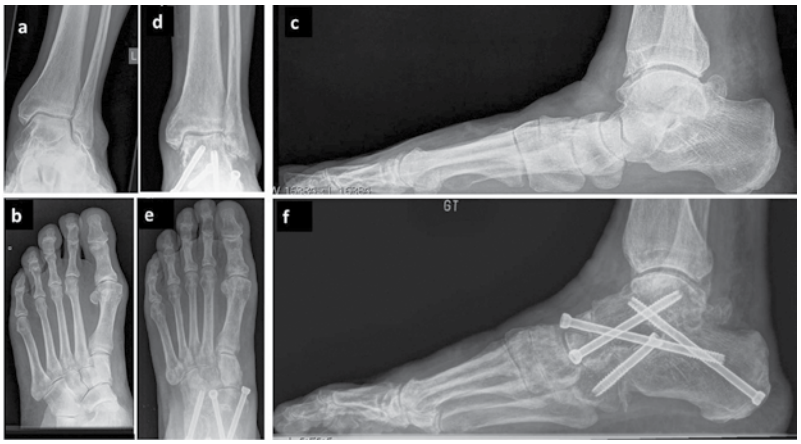

Fig. 19: *Severe planovalgus deformity with arthritic changes (a,b,c), was corrected with a triple hindfoot arthrodesis (d,e,f) in a 67-year old patient.*

(e.g. on ultrasound or MRI) TP tenosynovitis, open or endoscopic tendon debridement is an option. When longitudinal tears or significant tendinopathy (**Fig. 12**) is found, open tendon repair is needed [1-7].

In the presence of planovalgus deformity, with flexible subtalar joint, and no arthritic changes, joint preserving procedures are indicated [3-7,17]. Calcaneal (medial shift) osteotomy is mandatory (**Fig. 13**). Not only does it neutralise the weight bearing axis, and reduces the load to the medial side of the foot, but it is also considered a tendon transfer, as it places the insertion of Achilles tendon and the origin of plantar fascia to a more favourable biomechanical position (from excessive valgus to more neutral), in order for the windlass mechanism to be more effective during stance and gait [3-7,17]. It can be performed through an "L-shaped" lateral incision, or an oblique incision at the level of the osteotomy (taking care to avoid damage to the sural nerve), or through stab incisions using special instruments and fluoroscopy (minimally invasive technique) [18].

One has to emphasize the necessity for calf muscle lengthening, in the majority of patients requiring hindfoot valgus correction. Given that the Achilles tendon insertion has "moved" laterally in those patients, the gastroc-soleus muscle complex (triceps surae) becomes functionally tight. The Silfverskioldt test [9], that should have been performed preoperatively, will show whether the surgeon has to only lengthen the gastrocnemius [performing a proximal medial gastrocnemius release [19], or lengthening at the musculotendinous junction/ Strayer's procedure [20], or the Achilles tendon (performing formal Z-leghtnening, or a triple cut "Hoke" procedure) [3] (**Fig. 14**).

TP tendon exploration reveals the degree of degeneration. In mild tendinopathy one can consider debridement and repair (tubularisation using absorbable suture). In more severe tendinopathy, tendon transfer is indicated. Flexor digitorum longus (FDL) is usually

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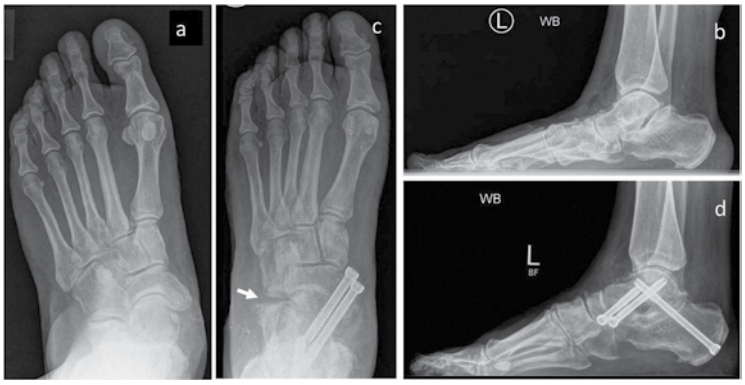

Fig. 20: *In a patient with fixed deformity and arthritis (a,b), "double" hindfoot corrective arthrodesis (of talonavicular and subtalar joints) was performed, preserving the calcaneocuboid joint, which is distracted (c, arrow) whilst deformity was corrected (c,d).*

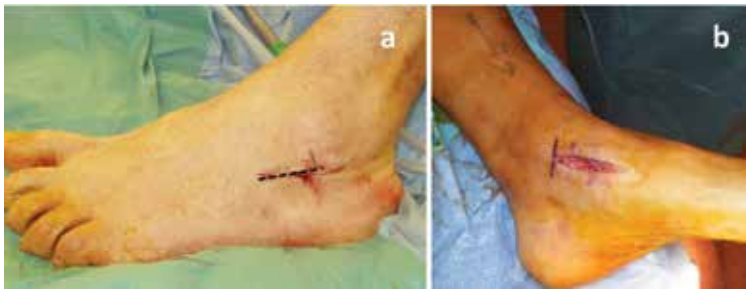

Fig. 21: *Lateral (sinus tarsi) (a) and medial (b) approaches for triple arthrodesis.*

used (**Fig. 15 and 16**). It lies next to TP, and functions along the same direction as TP. The surgeon can also assess tendon elasticity by pulling the tendon from a proximal to distal direction, in order to decide whether TP will be sacrificed (when the tendon appears stiff, without significant elasticity), or augmented by FDL (performing proximal tenodesis of TP to FDL, whilst FDL is attached to the foot navicular bone). The author recommends that, in cases of significant TP tendinopathy and degeneration and pain in the region of the medial malleolus or more proximally, TP should be sacrificed to avoid continuous postoperative symptoms.

Some patients can develop AAFD as a result of an accessory navicular foot bone. Those patients develop medial pain and progressive planovalgus deformity usually as a result of an avulsion injury that de-stabilises the synchondrosis between the accessory navicular and the "main" navicular bone. Thus, tibialis posterior becomes weak, without inherent tendinopathy or rupture. In those patients, calcaneal osteotomy restores hindfoot alignment, whilst excision of accessory navicular and TP re-attachment (Kidner procedure) [21] restores strength and function of TP (**Fig. 17**).

Recently another method of surgical treatment of flexible pes planus has been proposed, using a sinus tarsi implant, the so called "arthroereisis screw ", to stop pronation at the subtalar joint [22-24]. At the same time a medial soft tissue procedure can be performed. The users of the technique advocate removal of the implant 6-12 months later, as in the meantime the foot should have become dynamically stable. The theoretical advantage of this technique – that was initially introduced to treat paediatric flatfoot deformities - is that a calcaneal osteotomy is not performed. However, there are no comparative studies to support superiority of one technique over the other.

Once the hindfoot has been reduced to more neutral, relative hindfoot – forefoot alignment has to be assessed. In feet of residual forefoot supination, one can consider a plantarflexion (dorsal closing wedge) medial cuneiform (so called "Cotton") osteotomy. Alternatively, arthrodesis of first tarsometatarsal and/or naviculocuneiform joints can be performed [1,3-7] (**Fig.18 and 19**).

Midfoot arthrodesis [25] (involving tarsometatarsal and /or naviculocuneiform joints), is generally indicated in patients with "midfoot sag" on the lateral weight bearing foot radiograph.

Some authors advocate lateral column lengthening in feet with severe planovalgus (e.g. >40% uncovered

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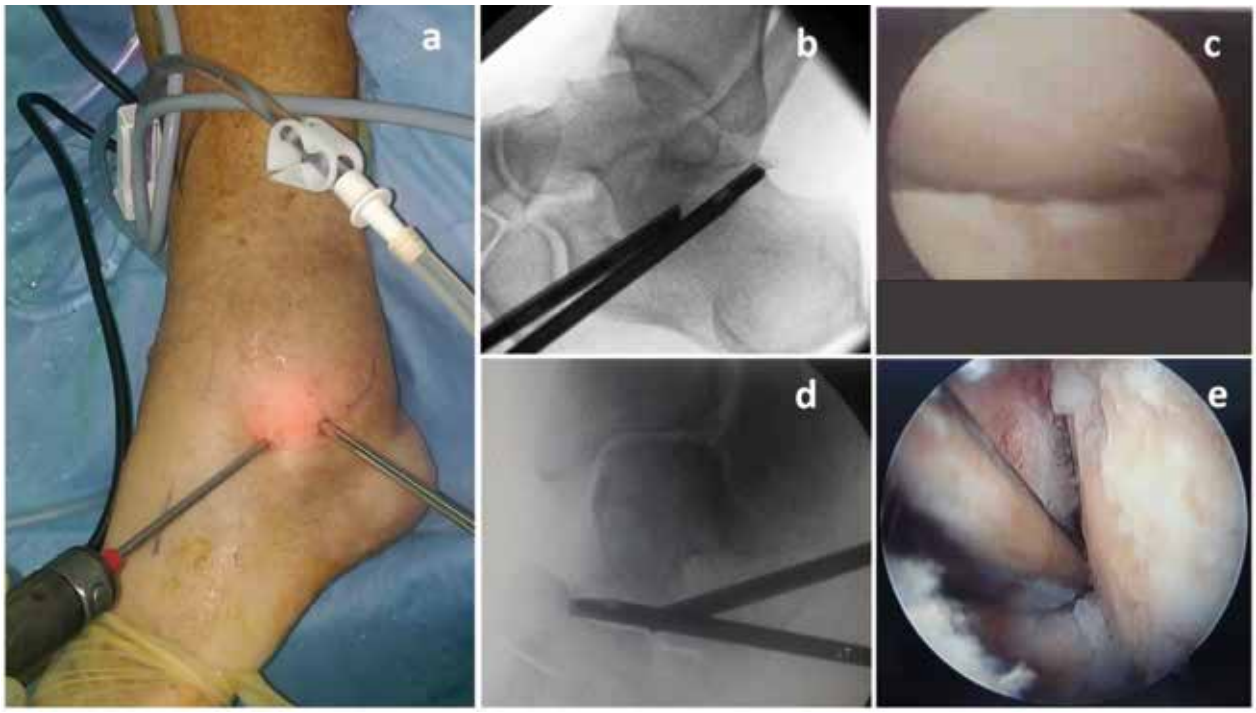

Fig. 22: *Two sinus tarsi endoscopic portals (a) allow preparation of subtalar (b,c), talonavicular (d,e), +/- calcaneocuboid arthrodesis.*

talar head at the talonavicular joint, on the weight-bearing view). This procedure requires a calcaneal or cuboid osteotomy, interposing a bone block taken from the medial cuneiform or allograft. Lateral column lengthening can be associated with stiffness and sometimes pain at the calcaneocuboid joint or the lateral midfoot joints (tarsometatarsal joints 4 and 5). Another option is to perform calcaneocuboid arthrodesis, interposing bone block. This carries a higher risk for non-union [1,3,4,7].

Rigid hindfoot valgus is usually accompanied by arthritic changes in some of the hindfoot joints (subtalar, talonavicular, calcaneocuboid) and then triple hindfoot arthrodesis is required (**Fig. 19**). It is debatable whether all three joints need to be fused, as studies have shown that "double arthrodesis" of subtalar and talonavicular joints, only, is sufficient [26] (**Fig. 20**). Given that corrective arthrodesis "opens" the lateral side (supinating the hindfoot), it could distract the calcaneocuboid joint, predisposing to non-union if arthrodesis is attempted. Triple arthrodesis requires usually two surgical approaches. A lateral "sinus tarsi" approach for subtalar and calcaneocuboid joints (along the line that connects the tip of the fibula and the 4th metatarsal base), and a medial approach along TP tendon (between the insertions of tibialis anterior and posterior tendons) (**Fig. 21**). The lateral approach is at risk of wound dehiscence (related to deformity correction that stretches the lateral approach skin edges), so many surgeons advocate one medial approach only to approach subtalar and talonavicular joints, avoiding fusion of the calcaneocuboid joint. If sufficiently trained, the surgeon can also prepare the joints arthroscopically using two sinus tarsi portals [27] (**Fig. 22**).

Ankle valgus alignment associated with AAFD, if flexible, may be treated effectively with deltoid ligament reconstruction and osteotomy (distal tibia/fibula or calcaneus). In the presence of significant degeneration and rigidity, tibiotalocalcaneal arthrodesis is the most reliable option. Depending on the patients' profile, and after discussion of the increased associated risk, one can also consider triple arthrodesis, soft tissue (deltoid ligament, TP) reconstruction and total ankle replacement (simultaneously or at a later stage) for some of these patients.

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6. Conclusion

It becomes apparent that preoperative planning is quite complicated. The aim of surgery is to produce a plantigrade foot, balancing forces between medial and lateral column, also taking into consideration and adjusting alignment between hindfoot and forefoot. The à la carte surgery is often required, depending on clinical and imaging findings. Thus, clinical assessment and preoperative evaluation and planning, as well as surgical management, requires the depth knowledge and experience in foot and pathology and surgery.

Conflict of interest:

The authors declared no conflicts of interest.

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Gougoulias N. Adult Acquired Flatfoot Deformity. *Acta Orthop Trauma Hell* 2018; 69(2): 85-98.

ΠΕΡΙΛΗΨΗ

Η ανεπάρκεια του οπισθίου κνημιάιου τένοντα μπορεί να είναι αιτία επίκτητης βλαισοπλατυποδίας των ενηλίκων. Άλλες αιτίες αποτελούν η αρθρίτιδα του μέσου ποδός, η ρήξη του πτερνοσκαφοειδούς συνδέσμου, η αποκόλληση του επικουρικού σκαφοειδούς οστού, η οξεία/τραυματική ρήξη του οπισθίου κνημιαίου τένοντα, ή του δελτοειδούς συνδέσμου, το βλαισό μεγάλο δάκτυλο. Στα αρχικά στάδια το πόδι παραμένει εύκαμπτο, ενώ αργότερα μπορεί να γίνει δύσκαμπτο. Κάποιοι ασθενείς μπορεί να παρουσιάσουν και βλαισότητα με εκφυσλιστικές αλλοιώσεις στην ποδοκνημική άρθρωση. Η αρχική αντιμετώπιση περιλαμβάνει την εφαρμογή ορθωτικών και φυσικοθεραπεία. Ανοικτός ή ενδοσκοπικός καθαρισμός του οπισθίου κνημιαίου έχει ένδειξη στο αρχικό στάδιο, αν δεν υπάρχει παραμόρφωση. Οστεοτομία ραιβοποίησης της πτέρνας και τενοντομεταφορά του καμπτήρα του μεγάλου δακτύλου ενδείκνυται σε εύκαμπτη βλαισοπλατυποδία. Αν το πρόσθιο πόδι παραμένει σε υπτιασμό μετά τη διόρθωση του οπισθίου ποδιού, μπορεί να απαιτηθεί συμπληρωματική επέμβαση, στην έσω κολώνα (οστεοτομία Cotton του έσω σφηνοειδούς ή αρθρόδεση μέσου ποδιού), ή την έξω κολώνα του ποδιού (οστεοτομία επιμήκυνσης πτέρνας ή αρθρόδεση πτερνοκυβοειδούς). Δύσκαμπτες παραμορφώσεις απαιτούν διορθωτικές αρθροδέσεις της υπαστραγαλικής, αστραγαλοσκαφοειδούς +/- πτερνοκυβοειδούς άρθρωσης. Επιμήκυνση Αχιλλείου τένοντα ή γαστροκνημίου μυός χρειάζεται συχνά συμπληρωματικά, ώστε να επιτραπεί η διόρθωση της παραμόρφωσης. Σε συνυπάρχουσα αρθρίτιδα της ποδοκνημικής, συνήθως απαιτείται κνημαστραγαλοπτερνική αρθρόδεση.

ΛΕΞΕΙΣ ΚΛΕΙΔΙΑ: πλατυποδία, βλαισοπλατυποδία, βλαισό πόδι, οπίσθιος κνημιαίος