

Tibialis Posterior as A Cause of AAFD - 'Not Guilty Your Honour'

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ABSTRACT

Recent changes in the perception of the primary lesion responsible for the pathogenesis of adult acquired flatfoot deformity (AAFD) has emerged. Studies question the essential role of the tibialis posterior (TP) and demonstrate its absence does not necessarily lead to planovalgus. The significant role the static restraints play, specifically the spring ligament (SL), its contribution to the medial arch stability, and how its failure precipitates AAFD, are increasingly recognised. It is now addressed as part of the treatment planning^[1,2,3]. This article aims to evaluate new and existing theories that allow us to draw these conclusions.

Key words: Foot and ankle; Posterior tibial tendon dysfunction; spring ligament; Adult acquired flatfoot deformity; Plantar calcaneonavicular ligament; Medial longitudinal arch

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BACKGROUND AND OBJECTIVES

Adult acquired flatfoot deformity (AAFD) has become synonymous with posterior tibialis tendon dysfunction (PTTD) and the two terms are used interchangeably. Much of our understanding of AAFD had been entirely influenced by the work of Johnson and Strom in 1989^[4] and is thought to be secondary to the dysfunction of the posterior tibialis tendon (PTT) that arises from the tibialis posterior (TP) muscle. Whilst the PTT is the second strongest tendon after the Achilles tendon, its excursion is only 2 cm^[5]. Small increases in length due to synovitis, tears, elongation and rupture is assumed to lead to a significant breakdown in its function, resulting in medial longitudinal arch breakdown and AAFD. Johnson and Strom proposed a staged classification based on progressive failure of the PTT^[4] and correlating foot clinical findings with this. In stage 2, tendon elongation results in planus deformity, implying the PTT is the primary dynamic stabiliser of the medial longitudinal arch. The assumption has been that spontaneous primary synovitis and secondary stretching and tears of the tendon cause structural changes in the foot, fixed deformity and degeneration as the end point. This is a fundamentally flawed position. Whilst degeneration, attenuation, and eventual rupture of the PTT are important parts of the pathology of the AAFD, it is insufficient to explain its deformity. The failure of the SL complex is the key step in the AAFD and this is thought to occur early in stage 0^[6,7], when deformity of the affected foot is not clinically visible. We aim to look at the current evidence that renders TP the unlikely initiating factor of painful planovalgus in AAFD.

ANATOMICAL PREDISPOSITION TO BIOMECHANICAL OVERLOAD

The TP muscle forms from the fourth week of gestation. By 8 weeks, somite derived skeletal muscle is well-formed^[8]. The TP origin is the proximal tibia, fibula, and interosseous membrane. The fibular origin is the strongest with short excursion multipennate muscle

fibres that have powerful contraction. The soleus and gastrocnemius account for the majority of the strength in ankle plantar flexion with minimal contribution from TP^[9]. TP synovitis is unlikely with no underlying biomechanical cause. Its anatomy may help explain its increased susceptibility to biomechanical overload. Fibula origin fibres ‘sling’ around the medial malleolus with some fibres re-inserting as lateral as the base of the 4th and 5th metatarsals. This ‘sling effect’ is far greater than the flexor hallucis longus (FHL) and flexor digitorum longus (FDL) which have a more linear path around the medial malleolus making the TP more biomechanically susceptible to valgus. This increases gliding frictional resistance of the PTT in planovalgus by 30%. Arai *et al*^[10] describes increased friction of the PTT in AAFD in the transverse and frontal planes which increases upon foot loading^[11]. Increased resistance was not present in FDL and FHL in a simulated flatfoot condition. This may reflect their orientation at origin and insertion. The retromalleolar region has a section of fibrocartilage on the anterior surface of the PTT that assists gliding and protects against compression and shear stress. This fibrocartilage is associated with areas of lower blood flow to the PTT^[12] possibly predisposing it to tendinopathy. There is no evidence to substantiate this. Furthermore, components of the PTT insertion are differentially affected^[13]. The anterior component is the largest component and is often affected, inserting on the navicular tuberosity and the medial cuneiform. The middle component gives attachment to the medial limb origin of flexor hallucis brevis and has a wide insertion on second and third cuneiforms, cuboid and the base of second, third, fourth, and fifth metatarsals. A final recurrent slip inserts on the anterior sustentaculum tali^[13]. The middle and recurrent slip insertions seem to be less affected.

VASCULAR THEORY

This theory is based on the assumption that a relative hypoxic region in the retromalleolar region of the PTT is responsible for the onset of degradation. In experimental models, healthy tendons are resilient to trauma. McMaster’s rabbit Achilles tendon studies demonstrate tendons do not rupture when subjected to severe strain^[14]. Fractures, tendon avulsions and muscle belly ruptures occur prior to tendon failure. Vascular obstruction however, results in tendon ruptures, when subject to the same strain, hence hypoxia’s potential contribution to tendonopathy. A precarious TP blood supply has been speculated as a cause of tendinopathy. A study by Manske *et al*^[15] showed 90% (27/30) of specimens had a vessel entering the tendon at 4.8 ± 0.8 cm proximal to the medial malleolus, and 100% (30/30) of specimens had a vessel entering the tendon 1.9 ± 0.8 cm distal to the medial malleolus. A relative hypovascular region starting 2.2 ± 0.8 cm and ending 0.6 ± 0.6 cm proximal to the medial malleolus, having an average length of 1.5 ± 1.0 cm is therefore present. Furthermore, Petersen *et al.* in 2002^[12] showed no immunostaining of laminin in the anterior tendon portion that passes behind the medial malleolus suggesting an avascular region. Simultaneous injection of Technetium 99 solution, Indian ink and gelatin into the anterior and posterior tibial arteries of fresh frozen bodies demonstrated the posterior tibial artery as the dominant blood supply and the PTT portion that curves around the malleolus was not vascularized. The insertion however was well vascularized. However, Frey^[16] looked at tendon micro-vascularization using conventional injection methods (Spalteholtz’s technique). While suggesting a reduced blood supply in the retromalleolar region, there was no avascular zone. Prado *et al*^[17] evaluated the vascular density of the PT after direct observation under a light microscope of histologic cuts stained with Masson’s Trichrome. There was no difference in vascular density at different sites of the PTT.

Arguments against a vascular theory are many. Anatomical studies do not conclusively prove an avascular zone at microvascularization level. A relative hypovascular zone is present throughout an individual’s life yet symptom onset is predominantly around 40 years of age, and not an older age group where the circulation may further be impaired. Also, anatomically, tendinopathy occurs at rich blood supply areas in the tendon, such as the insertion. In diabetics where the blood supply of the tendon can be severely reduced due to proximal vasculopathy and critical ischemia, there is no demonstrated increased incidence of PTTD. This is despite non-enzymatic glycosylation of tissues that stiffen tendons and decrease their ability to tolerate strain^[18]. A deficient blood supply alone, fails to explain why tendons with similar relative decrease in blood supply such as FDL and FHL, fail to become tendinopathic, despite being subjected to similar tibial loading. The unilateral presentation of AAFD despite good blood supply in both limbs, questions if a vascular hypothesis contributes to precipitating spontaneous tendon inflammation^[19].

COMPUTATIONAL

Finite element modelling of the foot has been used to estimate forces generated in foot tendons and bones as a result of ligament deficiencies. Cifuentes *et al.* showed an intact SL decreases stresses in PTT. SL insufficiency however, significantly increased stresses in the PTT and muscles that act to stabilise the first ray. The lack of a TP does not increase stress in the SL. Their model found that the PTT has a secondary role to the plantar fascia (PF) and the SL in medial arch support^[20].

Xu *et al*^[21] used finite element analysis and were able to reconstitute the foot arch by reconstructing the spring ligament. Non-anatomic reconstruction of the spring ligament provided the greatest correction to arch stability. Spring ligament reconstruction may mitigate against the need for non-anatomic bony procedures and preserve the triple joint complex.

Wong *et al*^[22] using finite element analysis, demonstrated that the loss of PF integrity or the SL leads to the increased stresses through the lesser metatarsal ligaments in the foot and destabilisation of the foot ligaments. Hence the protective effects of the medial ligaments were critical to the function and protection of the lateral ligamentous complex.

Iaquinto *et al*^[23] showed a 300% increase in *strain* in the SL in their model after PF division compared to when the PF was intact. These models show that ligament integrity of different structures is interdependent. The SL, plantar ligaments and PF are 3D structures engaging different bones, at different foot arch depths, suggesting they have overlapping functions. Their model shows that as tissues fail, the remaining structures have a relatively higher stress, increasing their chance of failure as loads surpass the ultimate strength of tissues.

CONTRIBUTION OF AGING AND COLLAGEN DEGRADATION

Collagen is part of connective tissue and is found in cartilage, ligaments, tendons, bones and skin. Ligaments consist of collagen bands intermingled with elastic fibers capable of supporting hundreds of pounds of stress per square inch. Loss occurs at 1-2% per year declining after 25 years of age. Visible skin changes reflect ligament changes. Barros *et al*^[24] looked at aging’s effect on the elastic and collagen fibers in the human cervical ligament and showed in 17 patients (age 16 to 69 years) how the fraction of collagen and elastic fibers changes with age. Age progressively increases elastic fibers

responsible for elasticity. However, these fibres showed increased degenerative changes with age. With age there is a decrease in oxytalan elastic fibers responsible for resistance allowing increased stiffness. This decline caused ligaments, tendons, bones and cartilage to become less flexible and more brittle with time. Evidence shows tendons and ligaments change but do not degenerate with age^[25]. Proteoglycan reduction and increased cross linking makes them stiffer and less capable of tolerating load^[26]. Thus, older people exposed to moderate loads have no increased risk of tendinopathy. Age related stiffness associated with an increased BMI and activity biomechanically disadvantage some structures, leading to overload. Both a decline in tendons ability to load and an increase in BMI leads to repeated overload below the ultimate tensile stress but above their endurance limit leading to eventual strain failure.

BIOMECHANICAL

Planovalgus feet does not equate to the presence of AAFD. Prior to the onset of AAFD the planus foot starts off with a degree of intrinsic stability, enough to prevent foot tendons reacting. Dyal *et al*^[19] demonstrated in patients presenting with painful planus, 70% of contralateral feet had asymptomatic planus showing planus alone does not cause pain. Planovalgus feet destabilise easier, being biomechanically disadvantaged, thus developing pain from reactive tendons and impingement. Instability and valgus impingement pain from talonavicular instability may present in cavus, planus and neutral feet. Instability is key to determining if planus feet become painful. Cavus feet are less biomechanically predisposed to medial column instability and often have an overactive TP that is causal to its cavus. Despite overactivity, calcaneonavicular ligament instability predisposes the PTT overload. Pes cavus with planus reflects feet that are predominantly cavus but have overriding planus symptoms.

Immediate arch collapse with SL sectioning does not occur^[2]. On cyclical loading, ligament attenuation allows visible arch collapse as the talar head subluxes through a lax medial buttress allowing foot hyperpronation. Ground reaction forces through a stable first ray provides a supinating vector that counteracts subtalar pronation. Thus, first ray destabilisation in the presence of a failed SL progress into planovalgus. Evidence comes from cadaver studies^[27].

CADAVER STUDIES

Stage 1 TP synovitis reflects overload synovitis arising from failed ligaments such as the SL. This causes a lateral plane deformity, that in the initial stages cannot be perceived on weight bearing radiographs. Gravity stresses the SL in the wrong plane and its early strain should be assessed in the perpendicular plane.

Jennings and Christianson^[28] assessed 5 cadaver feet mounted in a 3-dimensional kinematic, custom-loaded frame, to quantify the rotation of the talus, navicular and calcaneus before and after sectioning the SL complex, whilst incrementally tensioning the PTT. They showed SL sectioning led to instability and changes in talar, navicular, and calcaneal rotations, which an intact tensioned PTT was unable to compensate for. They concluded that the SL complex was the major arch stabilizer during mid-stance and its evaluation and repair in flatfoot reconstruction was undertaken.

A cadaveric study by Deland *et al*^[29] showed that the planovalgus deformity was recreated by systematically cutting key ligamentous structures. The deformity was corrected by reconstructing the SL alone using a bone/tendinous graft.

A further^[30] cadaveric study sequentially sectioned TP/FDL and the SL in feet and crucially found that only after sectioning the SL that

lateral translation of the foot/valgus was induced that could not be compensated for by tensioning a loaded FDL.

Reeck *et al*^[31] looked at the determining the magnitude of force transmission to the talus by its inferior articulation. Cadaver feet were axially loaded through the tibia and fibula on a mounted frame with and without a tensile load through tendons. Eighteen specimens were tested in three positions of gait. A pressure-sensitive film was inserted into the subtalar posterior and anteromedial facets, talonavicular joint and between the talar head and superomedial part of the SL. Contact areas and force transmitted across the articulations were greatest at near toe-off position, in the subtalar posterior facet, anteromedial facet, talonavicular joint and SL articulation showed decreasing amounts of contact area and force transmission. Loss of PTT tension did not increase talar head contact forces. SL fibre orientation was thought to contribute to stabilization to talar head to medial displacement.

Niki *et al*^[32] demonstrated in eight cadaver leg and foot specimens when tensioning the TP when the ligaments are weakened, failed to restore normal joint kinematics. In the first part of their experiment, they loaded cadaver feet to simulate heel strike, midstance and heel rise both with and without TP function. Each specimen was sectioned to create a simulated flatfoot deformity. 3-D kinematic orientation of the hindfoot complex was recorded with simulated loading. Small but significant changes in the angular orientation of the hindfoot complex were observed. The greatest angular changes were recorded during heel rise. They concluded, in feet with intact ligamentous structures, after the release of the PTT, loading the foot maintained normal alignment. Subsequent restoration of TP function did not correct a flatfoot deformity in the absence of soft tissues. Because TP had little effect in correcting foot attitude they concluded AAFD pathogenesis was due to gradual attrition of ligamentous structures caused by ambulation on an unlocked valgus hindfoot.

These studies demonstrate the pathogenesis of AAFD is the failure of static restraints and most importantly the SL. Evidence of what happens to the foot after SL failure is alluded to in several studies.

AFTER SPRING LIGAMENT FAILURE

Chu in 2001^[33] examined 7 cadaver feet using clamps to grasp the Achilles, peroneus longus, peroneus brevis, FDL, and FHL tendons with cable wires to pneumatic cylinders. Cyclic loading (3000 cycles) at 735 newtons with radiographic evaluation, concluded no change in the lateral talocalcaneal angle. However, after SL division and dynamic cyclical loading they were able to create a flatfoot model with a decrease in talar-first metatarsal angle and loss of height of the medial cuneiform. Crucially, the experiment demonstrated proximal integrity of the SL protects more distal plantar ligaments. Failure of the SL results in the failure of the first plantar TMT ligaments.

This corroborates with clinical data demonstrating in ankle fractures with complete deltoid ruptures also disrupts SL integrity leading to first ray dorsal instability around 6 months of injury^[7]. The deltoid and the SL are anatomically linked and weakening one weakens the other. The exact time of the SL failure was known thus allowing us the establishment of a time frame to develop other ligament failure. Feet that presented 6 months or less after the injury did not develop first ray instability. This is consistent with Chu *et al*^[33] as in both scenarios, cyclical foot loading in the absence of a SL results in planovalgus and dorsiflexion failure of the first ray. An intact SL protects the stability of the first ray. These studies establish the essential lesion in AAFD is failure of the static restraints, principally the SL and the PF medially. Laxity of the interosseous, cervical, superficial deltoid and deltoid ligaments may subsequently occur. Integrity of deltoid and superficial deltoid may also protect the integrity of SL.

The PF's role in maintaining the medial longitudinal foot arch cannot be underestimated. Huang^[34] showed that arch strength was maintained principally by 4 ligaments, the PF, the SL, the long and short plantar ligaments. The loss of all the ligaments resulted in a loss of approximately 40% foot stability as measured by arch height and foot length. Amongst these four ligaments, the relative contribution of the PF to medial arch integrity was over 50%. The PF also has three fold strength in maintaining the medial arch compared to the PTT.

The SL and PF have a joint role in maintaining foot arch. The final common pathway to AAFD development is talonavicular instability from SL failure. Loss of one ligament overloads the other beyond its ultimate tensile stress. Crary *et al*^[35] showed that cyclical loading of the cadaveric foot after PF sectioning caused SL permanent strain/plastic deformation. All feet subsequently went into the planus. This correlates with clinical studies^[36] looking at feet with unilateral plantar fasciitis. It is assumed that pain arises from tensile overload/failure of the medial PF insertion. Affected feet demonstrate a higher contemporaneous incidence of talonavicular/SL strain and first ray instability regardless of foot shape. Graham *et al*^[37] showed 93.5% of patients with chronic recalcitrant plantar fasciitis demonstrated a change in talar declination angle or talar/2nd metatarsal angle. They concluded that the subtalar joint should be evaluated and options to realign the subtalar joint should be considered. These clinical studies reinforce the theory that SL and PF have a combined role in maintaining foot arch height and failure^[36,37]. Hinterman showed deltoid ligament insufficiency also has significant effects on the transfer of the couple to the hindfoot^[38].

GAIT ANALYSIS

The tibia has a total rotation of about 30 degrees from heel strike to toe off. Internal tibial rotation unlocks the midfoot and allows calcaneal eversion^[39]. The presence of an intact SL provides a medial static restraint to this motion. At terminal stance, in closed chain motion the tibia externally rotates^[39] and the TP concentrically contracts. The talar head externally rotates over the fixed calcaneus locking the midfoot and allowing midfoot stability at toe off. A talus that starts excessively in external rotation fails to lock the midfoot and allows it to come under strain.

Tome and colleagues^[40] used a multi segment foot model with a marker on the navicular to measure the medial longitudinal arch in 12 patients with AAFD compared with 10 healthy controls. In the patients with AAFD, greater forefoot abduction was present throughout stance compared with controls. The medial longitudinal arch angle was significantly lower through pre-swing in the patients with AAFD thus suggesting compromise of the arch integrity due to failed ligaments, specifically the plantar aponeurosis and the SL. Alignment loss at the arch suggests that an osseous locking mechanism did not exist in the foot. Wang^[41] *et al* from the meta-analysis showed more conclusive changes in the forefoot (increased dorsiflexion and abduction) and hindfoot (increased plantarflexion and eversion) kinematics in AAFD during stance. These changes arise due to loss of ligamentous integrity.

RADIOLOGICAL

Both ultrasound (USS) and MRI have been used to image the SL and establish normal and abnormal SL in AAFD^[42,44], however MRI reports often fail to comment on the SL.

Tanaka and Kudo^[43] looked at SL strain in healthy asymptomatic feet. Functional evaluation of the SL using USS compared thickness between 102 non weightbearing and 90% weight bearing feet. SL

thickness was not significantly different between weight bearing and non-weight bearing feet in males and females (Thickness was 2.42 ± 0.38 mm (NWB) vs 2.26 ± 0.31 mm (WB) in males, and 2.07 ± 0.28 mm (NWB) vs 1.93 ± 0.29 mm (WB) in females). SL thickness was 2.0-2.8 mm in males and 1.8-2.4 mm in females ($p < 0.01$). The integrity of the SL using axial thickness as a measure of strain, showed no significant strain develops in the weight bearing healthy foot. Weak correlation between arch height and SL thickness ($p < 0.05$) was present. The decreased thickness in women may contribute to an increased occurrence in women.

MRI studies^[44] show that the SL is abnormal in patients with AAFD, in the presence of acute or chronic deformity, in isolation or associated with talonavicular dislocation, deltoid ligament changes, and PTT abnormalities. Common reported signs are thickening/thinning of the SL, increased signal on proton density with fat suppression (PD FS) or T2-weighted sequences, thickening greater than 5 mm, thinning less than 2 mm, and partial/complete discontinuity and defects at the junction of tibio-spring ligament with the superior-medial portion of the SL^[44].

Deland^[29] *et al* evaluated 31 MRI scans with evidence of degeneration/ tear of the PTT. The superomedial portion of the SL was attenuated or torn in 87% of the patients and the talocalcaneal interosseous ligament was affected in 48% of the patients. Although signal change in the PF was present in 26% of the patients, no partial or complete tears were noted.

Balen and Helms^[45] looked at 25 patients with advanced PTT and retrospectively examined them for SL, sinus tarsi, and PF abnormalities and compared to 25 patients with normal tendons. 92% had abnormalities in the SL and 72% had changes in the sinus tarsi. They concluded advanced PTT injury has a high association with SL injuries.

William *et al*^[3] evaluated 161 patient MRI's and lateral weight bearing radiographs for at least one of the following being present: talo-first metatarsal angle $\geq 5^\circ$, calcaneal pitch $\leq 20^\circ$, and talocalcaneal angle $\geq 45^\circ$. A positive finding for ≥ 1 measurement identified a radiographic planovalgus position of the foot. The radiographic deformity was then analysed against MRI evidence of SL or PTT abnormalities. SL abnormalities were strongly associated with all three radiographic planovalgus foot parameters. $p < 0.0001$. PTT changes failed to demonstrate significance $p = 0.04$.

Ormsby *et al*^[46] were able to use MRI to evaluate the tibionavicular component of the superficial deltoid ligament in AAFD, which is not routinely imaged in MRI sequences the tibionavicular (TN) ligament plays an important role. However, this ligament is not imaged in routine magnetic resonance imaging (MRI) sequences.

The relationship between radiographic flatfoot and SL tears was further evaluated by Pate *et al*^[47]. They identified 58 patients of which 29 patients had confirmed SL tears and 29 patients had intact SLs intraoperatively. 5 radiographic parameters were measured. A lateral talar-first metatarsal angle >30 degrees was the most significant predictor ($p < 0.001$) of intraoperative SL attenuation and had a sensitivity and specificity of 65% and 100%. We believe this correlation arises from dorsiflexion failure of the first metatarsal secondary to SL failure and the talonavicular axis.

STUDIES WITH IATROGENIC LOSS OF TIBIALIS POSTERIOR

Ligamentous instability cannot solely occur due to failure of a single tendon in the presence of other inverters, flexors and an intact tendoachilles. Failure of TP is assumed to arise from synovitis,

adhesions, degeneration, thinning, elongation and rupture of the PTT. However, the PTT is used as a non-phasic transfer for foot drop and despite the presence of peroneus brevis, feet do not collapse into planus. Despite iatrogenic loss of TP, the tibialis anterior and long flexors keep the foot balanced and fail to induce planovalgus.

In 2001, Yeap *et al*^[48] published a key paper describing the results of PTT transfers. These procedures were performed as a treatment for drop foot and in a series of 12 patients. None developed planovalgus deformity that we might have expected. The mean follow up was 90 months (range 24 to 300).

Pecheva *et al*^[49] showed in an up to 8 year follow up of 10 patients who underwent both phasic and non-phasic transfers of TP, none developed AAFD. They used lateral translation scores as an indirect measure to assess SL strain. Only one patient developed strain, but no feet developed planus despite some feet undergoing a concomitant first ray dorsiflexion osteotomy to correct cavus.

A study by Mizel^[50] looked at ten patients with traumatic common peroneal nerve palsy who had undergone TP transfer to the anterior midfoot for foot drop. At 74.9 months follow-up (18-351 months) all patients' feet were assessed clinically for planovalgus, range of motion and strength. Weight bearing lateral radiographs were used to assess both feet. In no case was valgus hindfoot deformity associated with the loss of TP.

Whilst the scale of these studies is modest and the demographics vary from AAFD population, they question the traditional assumption that dysfunction/elongation and tears of the PTT causes the collapse of the medial longitudinal arch. Despite a functioning peroneus brevis and absent TP, feet do not develop planovalgus as suggested in stage 2 Johnson and Stroms classification^[4].

FLEXOR DIGITORUM LONGUS TENDON TRANSFERS

Iatrogenic subtraction of the PTT do not result in unstable planus. However, when excised and substituted, FDL is often used. FDL lies adjacent to PTT and relatively easy to substitute to provide an inversion moment/ supination force to the hindfoot. Studies have demonstrated that despite an 'unaffected tendon' being transferred restoration of foot alignment and instability does not occur.

Goldner^[51] and colleagues first proposed the concept of transferring the FDL tendon in the treatment of PTTD in 1974 and then Mann popularized it in 1983^[52]. FDL generates half the inversion torque of the PTT^[53]. It also has 2 times the excursion. Murray and coworkers showed that the FHL tendon has a twofold greater strength than the flexor digitorum longus^[53]. The futility of an isolated FDL transfer has been shown in the following studies:

A study at the University of Utah demonstrated that the FDL transfer to the navicular reduced the capacity of this tendon to invert the hindfoot by 36 percent in comparison to leaving the tendon in its native state^[54].

A study at Lehigh University used multi-segmented foot modeling to assess the effectiveness of the medial displacement calcaneal osteotomy to correct AAFD and showed no improvement in abnormal loading or deforming joint moments in the medial arch with FDL tendon transfer^[55].

The ability to resist lateral translation (used indirectly as a measure of talonavicular laxity) with an FDL transfer showed no improvement in lateral translation when loaded in a cadaver model^[30], thus showing its failure to compensate for a failed SL.

Vaudreuil *et al* used a cadaveric robotic gait simulator to study the effects of the FDL tendon transfer on a flatfoot model. No

improvement of foot alignment or function occurred with this procedure^[56]. Furthermore, Spratley *et al* in a cadaveric study determined the FDL tendon transfer actually increased loads on the medial arch and medial heel, exacerbating the deforming forces of the AAFD^[57].

The FDL is unable to compensate for the lack of TP power. Transferring a tendon decreases its power by changing its axis, phase or excursion subtly or due to secondary complications such as adhesions. Even if the FDL increases in axial size by 44%, thus increasing its amplitude, it is still less strong compared to the initial TP^[58]. These studies further demonstrate it fails to compensate for the PTT or the SL. We postulate its hypertrophy occurs secondary to the continuous overload of the FDL from a persistently failed SL and biomechanical instability. No documented evidence of hypertrophy has occurred in the non-transferred tendon.

Isolated FDL transfer fails due to the same reason TP fails. Tradition justifies the FDL tendon transfer, but some studies show it has little biomechanical effect on the flatfoot deformity^[59]. FDL transfers may weaken the inversion power of the flexor digitorum longus when compared to doing no transfer at all^[54].

Although Mann popularised this treatment the most likely benefit of the procedure is the calcaneal osteotomy and not the FDL. It is the osteotomy that offloads the talonavicular axis and not supinating effects of the FDL transfer. The calcaneal shift offloads the talonavicular axis^[20] and changes calcaneal inversion torque that is lost as the subtalar axis externally rotates as the SL becomes lax or the foot pronates^[60].

The latter use of an osteotomy improves the results of a transfer. Many authors believe that it is remarkable that a procedure that by itself showed no benefit in AAFD, has continued to be a standard of operative care for over 40 years, despite biomechanical studies demonstrating the futility of substituting the FDL tendon for the PTT^[59].

CLINICAL DATA

Orr *et al*^[61] described 6 female feet, presenting with isolated SL rupture and a normal PTT. All of the patients achieved normal foot positions following surgery to the SL itself and/or bony fusion.

Borton and Saxby demonstrated isolated SL failure without PTT synovitis leading to planovalgus foot^[62]. Planovalgus occurring without PTT synovitis has also been reported by Orr *et al*^[61].

These and other studies show the development of the planus foot with isolated SL failure in the absence of TP synovitis.

Alessio Bernasconi *et al*^[63] reviewed the literature on synovectomy procedures for the treatment of PTT synovitis. PTT tenosynovectomy and debridement was successful in reporting complete relief of pain in a mean 80.5% of cases. However, all these studies have investigated small samples (7 to 19 patients) without comparative design and critical analysis of complications. They concluded stage I had a superior success rate (91%) associated with a faster recovery time. All data were based on case series. Follow up was at best short to medium. Long term data with significant numbers on this procedure is not present. We believe that its benefit may be short-lived without correction of the biomechanics.

Future areas to further investigate currently being undertaken in centres to compare the effect of tenoscopy alone to offloading the talonavicular axis with a calcaneal osteotomy. The aim of this would be to see if offloading the talonavicular axis is superior to simple synovectomy. If PTT synovitis was a secondary reactive synovitis and not the primary problem, failure to address the biomechanics may have inferior results.

RHEUMATOLOGICAL

Information from rheumatology data does not mirror PTT dysfunction, as rheumatoid arthritis is a synovial inflammatory disorder that affects both joints and tendons, however valuable information may be ascertained from how its inflammation may be casual in the development of AAFD. Spontaneous local synovitis of PTT, described in stage 1 is unlikely and erroneous^[4]. Synovitis in tendons and joints arise primarily due to either a primary inflammatory arthropathy or secondarily due to overuse such as in peroneal overload in the cavus feet or 2nd toe transfer synovitis in hallux valgus. In inflammatory disorders such as rheumatoid arthritis, synovitis weakens structures that support the foot such as joint capsules, ligaments and tendons. Despite inflammatory arthropathies having predilection to certain joints, widespread chronic synovitis also affects PTT. However, the incidence of AAFD in this population is only reported at 11% using stringent criteria^[64].

Several studies predate the use of antimetabolites. These studies imply the prevalence of synovitis may be greater due to the lack of anti-TNF drugs that would help control inflammatory arthropathy better. Spiegel^[65] observed in patients who had rheumatoid disease less than 5 years, only 8% demonstrated hindfoot changes. Kirkham^[66] reported 50 patients with rheumatoid arthritis and concluded that there were no instances of PTT dysfunction. They believed that longitudinal arch collapse and hindfoot valgus deformity was a consequence of joint destruction of the hindfoot and not posterior tibial dysfunction.

Jahss^[67] has found that rheumatoid arthritis exists in 7% of cases presenting with hindfoot valgus and loss of medial longitudinal arch. On surgical exploration, the PTT was relatively unaffected despite a rheumatoid process, with hindfoot deformity occurring as a consequence of subtalar joint involvement. This suggests planovalgus was primarily a joint/ligament pathology and didn't occur due to the dysfunction of the PTT.

Michelson *et al*^[64] showed an 11% to 64% incidence of PTT dysfunction in rheumatoid patients, depending on the diagnostic criteria used. 99 patients (73 females) with an average patient age of 58.4 were evaluated. The average time since diagnosis of rheumatoid arthritis was 13.5 years. Using three criteria for diagnosis, 11% of patients were thought to have PPTD only. The results of this study suggest that valgus deformity of the hindfoot in rheumatoid patients results from exaggerated pronation forces on the weakened and inflamed subtalar joint. These forces are caused by alterations in gait secondary to symmetrical muscular weakness and the effort of the patient to minimize pain in the feet.

These studies demonstrate that the presence of primary synovitis alone, that may occur in inflammatory arthropathies does not necessarily cause planovalgus as suggested in stage 1 of the Johnson and Strom classification.

NEUROPHYSIOLOGICAL STUDIES/CHANGE IN ACTIVITY OF SEVERAL TENDONS

Keenan *et al*^[68] looked at the causes of hindfoot valgus deformity in patients with rheumatoid arthritis and characterized the effects of the deformity on gait. Two groups of patients were assessed clinically, radiographically, and with gait analysis. Group 1 consisted of seven patients with seropositive rheumatoid arthritis and normal foot alignment. Group 2 consisted of ten patients with rheumatoid arthritis and valgus hindfoot deformity. No muscular imbalance, equinus contracture, valgus deformity, or isolated deficiency of the TP/PTT

(weakness, synovitis, or tendon rupture) contributed to the valgus deformity. Keenan also found in valgus deformity, electromyography demonstrated increased intensity and duration of activity of the TP, in an effort to support the failing foot arch.

Ringleb *et al*^[69] found TP electromyographic activity was increased in the five patients with valgus hindfoot. If the AAFD is principally a disease of the PTT, then one questions why the whole tendon unit changes its pattern of behaviour. We assume that increased activity would generate a greater force thus dynamically assisting the foot greater and preventing arch failure. EMG studies have demonstrated the over activity of several tendons in stage 2 flat feet including that of the peroneus longus (PL).

Ringleb^[69] and colleagues studied the kinematics, plantar foot pressures, and electromyography activity in 5 patients with PTTD. As with previous studies, increased forefoot dorsiflexion across the midfoot joints was observed in the patients with PTTD. In addition, a varus motion of the forefoot during heel rise was measured in patients, whereas healthy controls showed forefoot pronation during this phase, indicating healthy function of the first ray and engagement of the windlass mechanism. In this study, patients with PTTD exhibited greater activity of the PL. We postulate overactivity of PL was to compensate for loss of lateral column height and help plantarflex the first ray which becomes dorsally unstable in the sagittal plane. Also, increased activity of the gastrocnemius was measured in the PTTD group, suggesting excessive work to plantarflex the ankle with an unstable midfoot.

The increased activity these studies demonstrate most likely represents its activity increasing to compensate for failed ligaments and this overloading the TP prior to the onset of tibialis synovitis. This is consistent with increased stresses in the tendons demonstrated by computational modelling.

CONCLUSIONS

Evidence that the tibialis posterior is not the primary lesion responsible for the pathogenesis of AAFD comes from several study disciplines. Recent attempts to reclassify AAFD are based upon SL integrity^[6]. Furthermore, recent attempts to rename the pathology as PCFD (progressive collapsing foot deformity) and remove the term tibialis posterior has been undertaken. Most significantly, they acknowledge that failure of static restraints is responsible for the pathology^[70]. Whilst they question the presence of stage 1, we believe that it represents an early clinical manifestation of a reactive tibialis posterior prior to dorsal first ray destabilisation. Stage 0 disease has been described as the failure of the SL prior to the failure of more distal ligaments leading to first ray instability or the onset of a clinically reactive PTT^[6]. We believe that this is the essential lesion that precipitates subsequent stages of AAFD. Assessment of SL integrity is largely a lateral plane deformity that can be identified and assessed clinically, prior to the onset of planus^[6].

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