Adult Acquired Flatfoot (AAFF) due to Stage 2 Posterior Tibial Tendon Dysfunction (S2-PTTD) can be a debilitating condition that affects quality of life.

Females between 50-65 years with pre-existing flat feet, hypertension, diabetes and obesity will be at risk.

The number of females between 50-65 years being treated for AAFF will reach its peak over the next 10-15 years.
The debilitating effects of AAFF/S2-PTTD can be minimized by understanding the pathomechanic pathway.

The lack of movement coupling between the foot and leg is the pathomechanic endpoint.

Part 1
Topics for Discussion
- Definition
- Significant Anatomy
- Prevalence
- Patient profile
- Pathomechanic pathway.

AAFF S2-PTTD is a symptomatic, progressive flatfoot deformity resulting from loss of function of the tibialis posterior muscle/tendon and/or the loss of integrity of the ligamentous structures supporting the joints of the arch and hindfoot.
The tibialis posterior is a bi-pennate muscle with oblique fibers connecting the tibia and fibula that functions to restrain internal rotation of the tibia during contact and midstance via eccentric contraction.

The tibialis posterior tendon has a zone of hypovascularity that is 14 mm proximal and 10 mm distal from the tip of the medial malleolus.
The tibialis posterior tendon has two abrupt changes of direction at the malleolar groove and at the navicular.

Normal tendons are characteristically composed of more than 95% type I collagen, with relatively small amounts of types III, IV and V collagen.

The tibialis posterior tendon inserts into 3 areas of the foot.

- Anterior
  - Navicular tuberosity
  - N-C joint capsule
  - 1st cuneiform

- Middle
  - 2nd cuneiform
  - 3rd cuneiform
  - Cuboid
  - P.longus tendon
  - 2nd, 3rd, 4th, 5th mets

- Posterior
  - Sustentaculum Tali

The "interosseous" or posterior band of the interosseous talocalcaneal ligament (ITCL) acts as a pivot.

Just anterior to the posterior articular facet of calcaneus
The cervical or anterior band of the (ITCL) is the strongest STJ ligament.

Talus rotates medially on calcaneus until anterior ITCL tightens.

The deltoid ligament is composed of superficial and deep bands:
- Superficial tibionavicular
- Superficial tibiocalcaneal
- Superficial tibiotalar
- Deep anterior tibiotalar
- Deep posterior tibiotalar

Tibiocalcaneal lig. (TCL) inserts on sustentaculum tali and restricts calcaneal eversion.

The deep anterior tibiotalar and posterior tibiotalar ligaments resist external rotation of the talus.
The spring ligament complex consists of the superior and inferior calcaneo-navicular ligaments as well as support from the TP tendon and deltoid ligament via insertions to spring ligaments.

The spring ligament complex creates a talar acetabulum that restrains multi-planar talar motion by functioning as a articular sling.

The superficial deltoid ligaments form a concavity around the head of the talus and insert into the entire length of the spring ligament.

The posterior tibial tendon helps to prevent medial/plantar talar head migration by providing 2 insertions into the spring ligament.

3 Spring ligament
4 Ant. Tibiotalar
4’ Tibionavicular
4” Tibiocalcaneal
A survey of 582 ♀ respondents over the age of 40 yrs from a British GP group practice revealed a **3.3% prevalence** of Stage 1 & 2 PTTD in previously undiagnosed patients.

<table>
<thead>
<tr>
<th>Age group in years</th>
<th>Respondents without PTTD (% of age group)</th>
<th>Respondents with PTTD (% of age group)</th>
</tr>
</thead>
<tbody>
<tr>
<td>40-49</td>
<td>56 (97.6%)</td>
<td>2 (2.2%)</td>
</tr>
<tr>
<td>50-55</td>
<td>60 (96.8%)</td>
<td>2 (2.2%)</td>
</tr>
<tr>
<td>55-60</td>
<td>67 (91.7%)</td>
<td>3 (4.2%)</td>
</tr>
<tr>
<td>60-65</td>
<td>64 (97.1%)</td>
<td>2 (2.9%)</td>
</tr>
<tr>
<td>65-70</td>
<td>57 (94.5%)</td>
<td>1 (1.7%)</td>
</tr>
<tr>
<td>70-75</td>
<td>62 (93.5%)</td>
<td>3 (4.2%)</td>
</tr>
<tr>
<td>75-80</td>
<td>55 (92.9%)</td>
<td>3 (5.0%)</td>
</tr>
<tr>
<td>80-85</td>
<td>31 (62.2%)</td>
<td>1 (3.9%)</td>
</tr>
<tr>
<td>≥85</td>
<td>2 (10.0%)</td>
<td>9 (45%)</td>
</tr>
</tbody>
</table>

Table 2: Prevalence of posterior tibial tendon dysfunction by age of respondents.

Richie observed that the majority cases of AAFF were unilateral in presentation and mostly female.

<table>
<thead>
<tr>
<th>Total Pts</th>
<th>Female</th>
<th>Male</th>
<th>Mean Age</th>
<th>Unilateral</th>
<th>Bilateral</th>
</tr>
</thead>
<tbody>
<tr>
<td>682</td>
<td>494</td>
<td>188</td>
<td>62 yrs</td>
<td>631</td>
<td>51</td>
</tr>
</tbody>
</table>

Holmes and Mann found that obesity, hypertension and diabetes mellitus were recurring medical problems in the AAFF patient profile.

- **n=67**
- 76% women,
- Avg. 57 years
- Obesity in 33% (p= .005)
- Hypertension in 34% (p = .025)

**Table 2**: Prevalence of posterior tibial tendon dysfunction by age of respondents.
The baby boom of 1945 - 1965 has had huge influence on Canadian life.

“Two thirds of everything can be explained by demographics”.

Comparing the 2007 vs. 1981 (40 to 69 yr) age band, the % of males and females whose waist circumference placed them at a high risk for health problems more than doubled.

The typical 45 year old ♀ in 2007 vs. 1981 has gained 12 lbs, 1.7 BMI, 2.8” of waist and 1.6” of hip circumference.
What is the pathomechanic pathway to AAFF/S2-PTTD?

Signal Hill, NFLD

The findings associated with the patient profile of AAFF/ S2-PTTD have been implicated as possible etiological factors or predisposing factors, however, the actual etiology remains controversial.


Holmes and Mann found 52% of the patient profile had either diabetes, hypertension, or obesity.

Vascular compromise
Poor collagen repair.

PTTD has a higher proportion of types III and V collagen which are thinner and may not be able to withstand tissue stresses leading to attenuation.


Tryfonidis et al reported 9 cases of surgically confirmed spring ligament tears caused by AAFF without PTTD.

<table>
<thead>
<tr>
<th>Patient Profile</th>
</tr>
</thead>
<tbody>
<tr>
<td>AAFF Patient Profile</td>
</tr>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Diabetes</td>
</tr>
<tr>
<td>Over 50</td>
</tr>
<tr>
<td>Vascular compromise</td>
</tr>
<tr>
<td>Poor collagen repair</td>
</tr>
<tr>
<td>Change in ratio of collagen fibre types in ligaments and tendons</td>
</tr>
</tbody>
</table>

Deland et al examined the MRIs of 31 confirmed cases of PTTD and concluded the most to least damaged ligaments were:

Superomedial calcaneonavicular
inferomedial calcaneonavicular
talocalcaneal interosseous

Bloome et al found a variation in the PTTT insertion into the spring ligament. Is this a possible etiology?


Table 1: Comparison of structure involved and number of confirmed cases within each PTT group:

<table>
<thead>
<tr>
<th>Structure</th>
<th>Number of Confirmed Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superomedial calcaneonavicular</td>
<td>23</td>
</tr>
<tr>
<td>Inferomedial calcaneonavicular</td>
<td>8</td>
</tr>
<tr>
<td>Talocalcaneal interosseous</td>
<td>8</td>
</tr>
</tbody>
</table>

Table 2: Insertion sites of the posterior tibial tendon.

<table>
<thead>
<tr>
<th>Number</th>
<th>Side</th>
<th>Size</th>
<th>Location/Site</th>
<th>MT</th>
<th>FTC</th>
<th>PL</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>2</td>
<td>Y</td>
<td>Y</td>
<td>2,5,4,6</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>3</td>
<td>Y</td>
<td>Y</td>
<td>2,5,4,6</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>4</td>
<td>Y</td>
<td>Y</td>
<td>2,5,4,6</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>5</td>
<td>Y</td>
<td>Y</td>
<td>2,5,4,6</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
</tbody>
</table>

Table 3: Comparison of structure involved and number of confirmed cases within each PTT group.

<table>
<thead>
<tr>
<th>Structure</th>
<th>Number of Confirmed Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superomedial calcaneonavicular</td>
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</tr>
<tr>
<td>Talocalcaneal interosseous</td>
<td>8</td>
</tr>
</tbody>
</table>

Acrometric variability and measurements requires CC: calcaneal, Y: talus, L: calcaneus, R: medial tib, P: posterior tib. Table 4: Table of confirmed cases, number of confirmed cases: AAFF, Posterior Tibial Tendinopathy, AAFF Patient Profile. Obesity, Vascular compromise, Poor collagen repair, Change in ratio of collagen fibre types in ligaments and tendons.
There is a high incidence of a pre-existing pes planus in AAFF/PTTD.

Obesity + Pre-existing flatfoot = Increased eccentric load on TP muscle-tendon

AAFF Patient Profile
Hypertension
Diabetes
Over 50

Obesity
Vascular compromise
Poor collagen repair
Change in ratio of collagen fibre types in ligaments and tendons
Lack of insertion into spring lig. from PTT

Spring lig. tears

Congenital Pes Planus and TPD:
Mann and Thompson (1985) 30%
Jahss (1991) 100%
Dyal et al (1997) 98%
Deland et al. concluded that the AAFF deformity associated with chronic PTTD “cannot be reproduced experimentally by releasing the tibialis posterior tendon alone.”


Chu et al. stated that to create flattening of the plantar arch, there is a need to cut, the spring and plantar ligaments and possibly the plantar fascia.


Niki et al. concluded that intact osteoligamentous structures can maintain alignment after the initial loss of PTT.


The creation of a flatfoot by ligament resection and subsequent PTT restoration could not significantly improve alignment.

Jennings and Christensen demonstrated that the spring ligament complex is the major stabilizer of the arch during midstance and that the posterior tibial tendon is incapable of fully accommodating for its insufficiency.

Imhauser et al. using an in-vitro model concluded that PTTD causes posterior shift in the center of pressure and abnormal loading of the foot’s medial structures.

Niki et al demonstrated that PT muscle had the greatest influence on hindfoot kinematics during heel rise.
“There is significant evidence to suggest that ligaments are responsible for the movement transfer coupling mechanisms between the foot and the leg.”

Hintermann et al reported the foot becomes partially disconnected from the tibia by transection of the deltoid and further disconnected after transection of the STJ interosseous.

- 8 cadaver models
- 0N, 200N, 400N, & 600N loads
- Sequential ligament release:

Complete ligament transection of medial ankle ligaments, spring ligament and plantar fascia.

Video courtesy of D. Richie DPM
Several cadaver studies on tarsal joint fusions have demonstrated that the talo-navicular (TNJ) is the keystone to joint motion in the rearfoot.

Hintermann applied a 600N load and internal rotation to the ankle complex with and without joint fusions.

<table>
<thead>
<tr>
<th>Ankle Load (600 N)</th>
<th>Int. Tibial Rotation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>2.2°</td>
</tr>
<tr>
<td>Ankle fused</td>
<td>1.7°</td>
</tr>
<tr>
<td>STJ fused</td>
<td>1.4°</td>
</tr>
<tr>
<td>T-NJ fused</td>
<td>0.9°</td>
</tr>
<tr>
<td>STJ &amp; T-NJ fused</td>
<td>0°</td>
</tr>
</tbody>
</table>


Astion et al demonstrated a TNJ fusion significantly reduced motion in the hindfoot and midfoot and excursion of the TP.

<table>
<thead>
<tr>
<th>Joint Fused</th>
<th>Retained ROM</th>
<th>Excursion of TP</th>
</tr>
</thead>
<tbody>
<tr>
<td>T-NJ</td>
<td>10% STJ, 10% CC 25% retained</td>
<td></td>
</tr>
<tr>
<td>STJ</td>
<td>26% TN 56% CC 46%</td>
<td></td>
</tr>
<tr>
<td>C-CJ</td>
<td>100% STJ, 67% TN 73%</td>
<td></td>
</tr>
</tbody>
</table>


O’Malley et al produced a flatfoot via sequential ligamentous release followed by joint fusions. The TNJ fusion most affected the hindfoot radiographic angles.

<table>
<thead>
<tr>
<th>Fusion of:</th>
<th>Radiograph Pre Release</th>
<th>C-C T-N</th>
<th>STJ T-N</th>
<th>TRIPLE T-N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lat T-N angle</td>
<td>0</td>
<td>23°</td>
<td>37°</td>
<td>3°</td>
</tr>
<tr>
<td>A-P T-N angle</td>
<td>0</td>
<td>6°</td>
<td>10°</td>
<td>0°</td>
</tr>
<tr>
<td>Hindfoot valgus angle</td>
<td>0</td>
<td>13°</td>
<td>19°</td>
<td>2°</td>
</tr>
</tbody>
</table>

Wulker found a TNJ fusion affected residual hindfoot motion the most as compared to a STJ or CCJ fusions.

<table>
<thead>
<tr>
<th>Fused Joint</th>
<th>Reduction of Motion at C - C STJ T - N</th>
</tr>
</thead>
<tbody>
<tr>
<td>C - C</td>
<td>98% N.Sig. N.Sig.</td>
</tr>
<tr>
<td>STJ</td>
<td>N.Sig. 98% 60%</td>
</tr>
<tr>
<td>T - N</td>
<td>98% 98% 98%</td>
</tr>
</tbody>
</table>

The talo-navicular joint is the pivotal joint in the adult acquired flatfoot deformity.

The TNJ has:
- strong reciprocal coupling to subtalar and tibial rotation
- dynamic support from the TP
- passive support spring lig. complex

Video courtesy of D. Richie DPM

Subtalar Arthrodesis

Talonavicular Arthrodesis

AAFF Patient Profile
- Hypertension
- Diabetes
- Over 50

Obesity
- Pre existing flat foot

Vascular compromise
- Poor collagen repair

Increase load on PT muscle and tendon
- Spring lig. tears
- Deltoid and STJ Lig. Damage

Movement coupling between foot and leg disrupted
- TNJ dysfunctional
The more massive tibia conveys most of the body weight directly on the talus and acts as a solid lever in ankle injuries.

The foot, on the other hand, is composed of numerous small bones which weaken it as a lever.

“When the foot is dorsiflexed at the ankle, the talus becomes firmly lodged in the tibiofibular socket and serves as part of the proximal lever of the leg.”

In the AAFF, the tibia becomes the dominant lever.

Unrestrained by a weakened TP, the internally rotating tibia drives the talus into adduction.

Internal rotation of tibia = Internal rotation of talus
Flemister et al using fresh frozen cadaver specimens demonstrated that the forefoot abduction deformity contributes significantly to PT muscle excursion length.

<table>
<thead>
<tr>
<th>Description</th>
<th>FF EV</th>
<th>FF EV</th>
<th>FT ADD</th>
<th>HT ADD</th>
<th>HT EV</th>
<th>HT EV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adduction</td>
<td>3.2 ± 0.7</td>
<td>3.1 ± 0.6</td>
<td>3.2 ± 0.5</td>
<td>3.2 ± 0.5</td>
<td>3.2 ± 0.5</td>
<td>3.2 ± 0.5</td>
</tr>
<tr>
<td>Neutral</td>
<td>3.2 ± 0.7</td>
<td>3.1 ± 0.6</td>
<td>3.2 ± 0.5</td>
<td>3.2 ± 0.5</td>
<td>3.2 ± 0.5</td>
<td>3.2 ± 0.5</td>
</tr>
<tr>
<td>Plantarflex</td>
<td>2.5 ± 0.7</td>
<td>2.0 ± 0.6</td>
<td>2.2 ± 0.6</td>
<td>2.2 ± 0.6</td>
<td>2.1 ± 0.6</td>
<td>2.1 ± 0.6</td>
</tr>
</tbody>
</table>

Table 3. Mean (± SE) of posterior tibial muscle excursion (cm) in each position for each foot position.

Arai et al demonstrated the motions in the frontal and transverse planes in the flatfoot condition increased the work of friction of the posterior tibial tendon in the retromalleolar region.

Table 4. AAFF patient profile

<table>
<thead>
<tr>
<th>Hypertension</th>
<th>Diabetes</th>
<th>Over 50</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre existing flat foot</td>
<td>Collagen fibre types in ligaments and tendons</td>
<td>Spring lig. tears</td>
</tr>
<tr>
<td>Vascular compromise</td>
<td>Poor collagen repair</td>
<td>Deltoid and STJ Lig. Damage</td>
</tr>
<tr>
<td>Change in ratio of collagen fibre types</td>
<td>Movement coupling between foot and leg disrupted</td>
<td>Tibia progressively allowed to internally rotate</td>
</tr>
<tr>
<td>Lack of insertion into spring lig. from PTT</td>
<td>Increase load on PT muscle and tendon</td>
<td>Tibia progressively allowed to internally rotate</td>
</tr>
</tbody>
</table>

Crim classified 4 pathological stages of PTTD using MRI

- **Tenosynovitis** – thickness and fluid within the sheath.
- **Grade I rupture** – thickness and foci of high signal within the tendon on T2.
- **Grade II rupture** – attenuation of the tendon, thinning.
- **Grade III rupture** – gap within the tendon, absent signal.

Wacker et al. observed atrophy of the PT muscle along with fatty infiltration in the cases of PTTD with tendon rupture when compared to the normal leg in pre surgical MRIs.

(mean 10.7%, p = 0.008).

Ringleb et al. demonstrated increased electromyographic activity and magnitude of the posterior tibialis during gait in 5 patients with S2-PTTD when compared to healthy individuals.
AAFF results from body weight, forward momentum, tibial rotation and dynamic contraction of all extrinsic muscles leading to:

- excessive load on the PT tendon
- followed by structural failure
- ending with loss of restraint of pronation moment in transverse and frontal planes
- resulting in the loss of movement coupling.

The number of females between 50-65 years being treated for AAFF will reach its peak over the next 10-15 years.

The lack of movement coupling between the foot and leg is the pathomechanic endpoint for AAFF.

Evaluating for the presence of movement coupling between the leg and foot is vital to the success of non operative treatment for AAFF.
Thanks for listening.

Acknowledgements
Paris Orthotics Ltd
Douglas Richie DPM