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The role of medial ligaments and tibialis posterior in stabilising the medial longitudinal foot arch: a cadaveric gait simulator study

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ABSTRACT

Background: Debate exists whether adult acquired flatfoot deformity develops secondary to tibialis posterior (TibPost) tendon insufficiency, failure of the ligamentous structures, or a combination of both.
Aim: The aim of this study is to determine the contribution of the different medial ligaments in the development of acquired flatfoot pathology. Also to standardise cadaveric flatfoot models for biomechanical research and orthopaedic training.
Methods: Five cadaveric feet were tested on a dynamic gait simulator. Following tests on the intact foot, the medial ligaments – fascia plantaris (FP), the spring ligament complex (SLC) and interosseous talocalcaneal ligament (ITCL) – were sectioned sequentially. Joint kinematics were analysed for each condition, with and without force applied to TibPost.
Results: Eliminating TibPost resulted in higher internal rotation of the calcaneus following the sectioning of FP and SLC ($d > 1.28$, $p = 0.08$), while sectioning ITCL resulted in higher external rotation without TibPost ($d = 1.24$, $p = 0.07$). Sequential ligament sectioning induced increased flattening of Meary's angle.
Conclusion: Function of TibPost and medial ligaments is not mutually distinctive. The role of ITCL should not be neglected in flatfoot pathology; it is vital to section this ligament to develop flatfoot in cadaveric models.

1. Introduction

Adult acquired flatfoot deformity is a frequent clinical condition. This deformity results in a variety of symptoms ranging from mild functional limitations to severe pain dependent on the affected structures. It can also lead to severe mobility issues, such as arthritis in foot joints. Although flatfoot deformity in an early stage can be corrected to some extent with the use of insoles, surgery is the only effective way to restore foot function and relieve pain in more advanced stages of the pathology [1]. The mechanism of acquired flatfoot development is not fully understood yet, primarily due to the complexity of the involved anatomical structures and their functional interdependence. This knowledge gap hinders surgeons in developing satisfactory surgical interventions to correct the deformity. Moreover, it also complicates the standardisation of cadaveric flatfoot models for biomechanical research and orthopaedic training.

Abbreviations: TibPost, tibialis posterior; FP, fascia plantaris; SLC, spring ligament complex; ITCL, interosseous talocalcaneal ligament; CT, computed tomography; Tib-Cal, tibiocalcaneal alignement; Tal-Cal, talocalcaneal joint; Tal-Nav, talonavicular joint; Tal-MT-1, talometatarsal alignement; AA, average angle; ROM, range of motion

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Debate exists whether adult acquired flatfoot deformity develops secondary to tibialis posterior (TibPost) tendon insufficiency or failure of the medial ligamentous structures. These medial ligaments consist of the fascia plantaris (FP), the interosseous talocalcaneal ligament (ITCL) and the spring ligament complex (SLC) which includes the tibionavicular, tibiospring and the calcaneonavicular ligaments [2,3]. Several investigators [4,5] suggest that failure of the SLC is crucial in flatfoot deformity. *In vitro* [6,7] and in vivo [8] studies suggest that the medial arch ligamentous structures - SLC and FP – are the major stabilizers, and that intact TibPost function is incapable of compensating for their dysfunction [9,10]. On the contrary, other investigators [11–13] report TibPost function is essential to maintain the medial arch in dynamic weight bearing conditions, and have therefore hypothesised that TibPost dysfunction is the leading cause for adult acquired flatfoot deformity. This is echoed in the debate on the treatment of the pathology, where the current focus is on restoring TibPost function.

In vitro biomechanical studies reported in the literature, typically assess the role of the SLC at the static midstance foot position [4,14], while also studying failure of FP and ITCL in the development of a flatfoot deformity [14]. Previous cadaveric studies have failed to induce a flatfoot deformity without sectioning all ligaments, or have stressed the foot specimen with cyclic loading thereby straining the remaining ligaments in a uncontrolled manner [5,10]. McCormack indicated that division of the SLC, followed by subsequent cyclic axial loading, represented the most physiological flatfoot surrogate for in vitro experiments [15]. Based on our clinical experience, we believe that acquired flatfoot deformity results from a combined injury to the medial ligaments, and that the role of ITCL cannot be overlooked in this. Moreover, we believe a functional TibPost is insufficient to prevent flat foot deformity in presence of injury to the medial ligaments. Enhanced insight in the contribution of the various ligaments and tendons towards the support of the medial foot arch will improve surgical management of this debilitating condition.

Therefore, the aim of this cadaveric study was to evaluate the impact of a sequential sectioning of the medial ligaments (FP, SLC and ITCL), and the stabilising role of TibPost on the foot-and-ankle kinematics, using a dynamic and actively-loaded gait simulator to replicate physiological loading conditions on the joint complex. We hypothesised that sectioning the ligaments would alter hindfoot kinematics, with each sequential ligament sectioning progressively increasing these alterations. We also hypothesised that the absence of TibPost function would result in the amplification of these alterations observed in each condition.

2. Methods

2.1. Specimen preparation

Six fresh-frozen cadaveric feet (4 females, 2 males; 4 right, 2 left) of median age 78 years (range: 64–91 years) were obtained for this study following ethical approval from the institutional ethical committee. Computed tomography (CT, slice thickness: 0.6 mm; Siemens Somatom Force, Siemens Healthcare, Erlangen, Germany) and manual examination was used to include specimens with no signs of ligament injury, previous foot surgery or significant osteoarthritis. One specimen was excluded following visual inspection for flatfoot deformity, which involved monitoring the medial longitudinal arch height while manually loading the feet in the midstance position.

All specimens were stored at −20 °C. After thawing at room temperature for 12 h, the fibular and tibial shafts were resected at 25 cm above the foot sole. All soft tissue was removed at 20 cm from the sole, except for the nine extrinsic muscle tendons – peroneus longus, peroneus brevis, tibialis anterior, extensor digitorum longus, extensor hallucis longus, TibPost, flexor digitorum longus, flexor hallucis longus and triceps surae – which were resected at the distal musculotendinous junctions. The fibula was fixed to the tibia with a cortical bone screw to stabilise the syndesmosis. The proximal end of the tibia was fixed in a cylindrical steel pot using polyester resin (Motip Dupli BV, Wolvega, The Netherlands) to allow fixation of the specimen to the gait simulator. The cutis and subcutis were resected at the medial side of the ankle. A triangular shaped area from the medial malleolus down to the medial calcaneal tuberosity and proximally to the base of the first metatarsal was exposed, thereby uncovering the following structures in the medial arch – TibPost tendon, deltoid ligament, medial malleolus, sustentaculum tali, the neurovascular bundle running beneath the sustentaculum tali, fascia plantaris, and the SLC.

2.2. Testing on gait simulator

Specimens were mounted on a previously validated physiological gait simulator (Fig. 1), which replicated the stance phase of a gait cycle in cadaveric specimens (duration: 1 s) [16–19]. Pneumatic actuators attached to the tendons of six muscle groups – Peronei (peroneus longus, peroneus brevis), Anterior Extensors (tibialis anterior + extensor hallucis longus, extensor digitorum longus), Achilles (triceps surae), tibialis posterior, flexor hallucis longus and flexor digitorum longus – applied tensile loads corresponding to muscle forces representative of the stance phase of gait. Electric servomotors were used to replicate flexion-extension at the knee and anterior displacement of the limb. A pneumatic actuator in-line with a force plate was operated on inertial control to mimic the vertical ground reaction force variations during stance [17].

Intracortical screws (dia: 4 mm, length: 50 mm; ICOS, New Deal, France) were inserted in the tibia, talus, calcaneus, navicular and first metatarsal of each specimen. A cluster of four LED markers was rigidly mounted on each screw and relative positions with respect to corresponding bones were registered on CT scans. An optical motion capture system (frequency: 100 Hz; Krypton, Metrix, Belgium) was used to track the bone trajectories during the gait cycle.

Each specimen was tested on the simulator for three cycles with force application on all muscle groups, and three cycles without force application on TibPost. (Fig. 1).

2.3. Surgical procedure

All surgeries were performed by one surgeon whilst the foot was mounted on the gait simulator. Care was taken to keep the foot hydrated during the experiment by regularly spraying it with water and a phosphate-buffered saline solution.

The medial ligaments were sectioned sequentially in the following order – first the FP [5,7], followed by the SLC, and finally the ITCL. The FP and SLC were sectioned under direct vision through the medial approach. The ITCL was sectioned by inserting a blade between talus and calcaneus just anterior of the posterior facet of the talocalcaneal joint and rotating the blade towards the middle facet of the talocalcaneal joint.

The specimens underwent the same aforementioned testing protocol in the gait simulator – three cycles each with and without force on TibPost – after each sequential state of ligament sectioning.

2.4. Data analysis

Marker trajectories were used to calculate 3D rotation angles – inversion-eversion about the anteroposterior axis, internal-external rotation about the proximal-distal axis, and plantar-dorsiflexion about the mediolateral axis – for the tibiocalcaneal alignement (Tib-Cal) representing the hindfoot, the talocalcaneal joint (Tal-Cal) representing the subtalar joint, the talonavicular joint (Tal-Nav) and the talometatarsal alignement(Tal-MT1). Kinematic data were normalised for 0–100% of the stance phase and post-processed using a low-pass

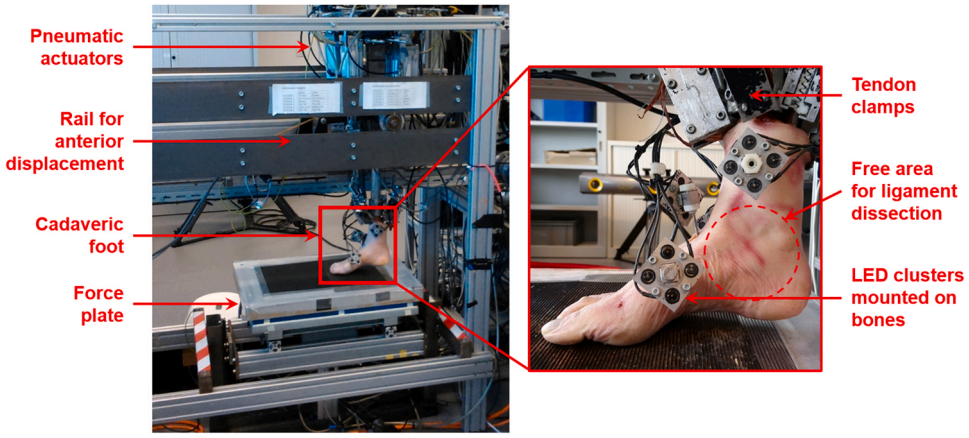

Fig. 1. Experimental setup showing the primary components of the gait simulator along with a zoomed-in view of the mounted cadaveric specimen with LED clusters attached for capturing the trajectories of bones using an optical motion capture system.

filter (cut-off frequency: 6 Hz; Matlab R2019b, Mathworks, Natick, USA). The mid-stance position during the gait simulation was used as a reference to offset kinematic data, thereby allowing inter-specimen comparisons. Mean trajectories across three trials were used to calculate the average angle (AA) and range of motion (ROM) over the gait cycle for the aforementioned articulations. AA indicated a global deviation from intact kinematics due to ligament rupture, while ROM suggested potential excessive or restricted mobility at a certain joint.

Since data were found to deviate from the normal distribution using the Shapiro-Wilk test, non-parametric tests were used to compare data. For each of the aforementioned articulations, a Wilcoxon signed-rank test (significance: $p < 0.1$; Matlab, MathWorks, Natick, USA) was used to compare AA and ROM following each sequential ligament sectioning (FP, SLC, ITCL) with respect to the native condition. The Wilcoxon signed-rank test (significance: $p < 0.1$) was also used to compare AA and ROM for each condition (Native, FP, SLC, ITCL) with and without the force application on the TibPost tendon. The Cohen's d test was used to calculate the effect size of the aforementioned comparisons (significant effect: $d > 0.5$) [20].

3. Results

3.1. Tib-Cal inversion-eversion

Sectioning FP resulted in an increase in mean varus (d=0.86) as compared to the native condition; however, the varus decreased after each subsequent sequential sectioning (Fig. 2). Switching off the TibPost reduced varus and increased ROM following sectioning FP and ITCL ($d > 0.66$), although results were not statistically significant (Table 1).

3.2. Tib-Cal internal-external rotation

A significant influence of the TibPost function was observed on mean internal-external rotation following ligament sectioning (Fig. 2). Switching off the TibPost resulted in higher internal rotation of the calcaneus following the sectioning of FP ($d = 1.54$, $p = 0.08$) and SLC ($d = 1.28$, $p = 0.08$), while sectioning ITCL resulted in higher external rotation without TibPost ($d = 1.24$, $p = 0.07$) (Fig. 2).

3.3. Tal-Cal inversion-eversion (Valgus heel)

An increase in mean varus was noticeable following sectioning SLC ($d > 0.78$), although this was not statistically significant (Fig. 3). No significant difference was seen in AA or ROM between conditions simulated with or without TibPost.

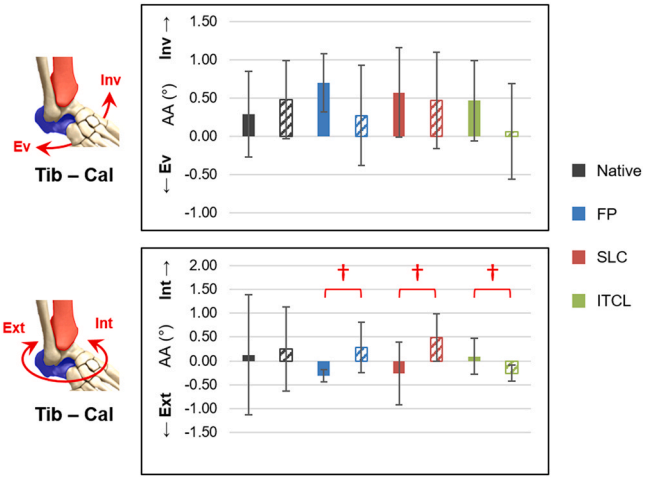

Fig. 2. Average angles (AA) for inversion (Inv) - eversion (Ev) and internal (Int) - external (Ext) rotation at the tibiocalcaneal joint (Tib-Cal) in the native condition (black) and following sectioning of fascia plantaris (FP; blue), spring ligament complex (SLC; red), and interosseous talocalcaneal ligament (ITCL; green) measured over the simulated stance phase of the gait cycle with (solid) and without (hatched) tibialis posterior. [Data represented as mean ± SD across five specimens. The dagger (†) represents statistically significant differences between the same condition simulated with and without tibialis posterior ($p < 0.1$). No statistically significant differences were observed for any condition with respect to the native condition simulated with tibialis posterior (solid black)].

3.4. Tal-Cal internal-external rotation

Switching off the TibPost resulted in a more externally rotated calcaneus following sectioning ITCL ($d = 0.61$), although this was not statistically significant (Fig. 3). A higher range of calcaneal rotation was observed following sectioning FP ($d = 0.82$, $p = 0.08$) and SLC ($d = 0.73$).

3.5. Tal-Nav internal-external rotation (Talonavicular coverage angle)

An increase in mean external rotation of the navicular was observed following simulations without TibPost, most noticeably following sectioning ITCL ($d = 0.96$), although this was not statistically significant (Fig. 4).

3.6. Tal-MT1 plantar-dorsiflexion (Meary's angle)

Sequential sectioning of the medial ligaments resulted in an increased flattening of the medial arch (Fig. 5). Mean metatarsal

Table 1
Average angles (AA) and ranges of motion (ROM) of various joints involving the tibia (Tib), talus (Tal), calcaneus (Cal), navicular (Nav) and first metatarsal (MT1) in the native condition and following sectioning fascia plantaris (FP), spring ligament complex (SLC), and interosseous talocalcaneal ligament (ITCL) measured over the simulated stance phase of the gait cycle with and without tibialis posterior (TP). [Data represented as mean ± SD across five specimens. Each condition has been compared to the native condition simulated with TP, with corresponding effect sizes (d_n). Each condition without TP has also been compared to the corresponding condition with TP, with corresponding effect sizes (d_{tp}). Shaded red cells represent statistically significant comparisons ($p < 0.1$), with bolded green values representing significant effect sizes ($d > 0.5$). InvEv: inversion(+) eversion(-), IErot: internal(+) external(-) rotation, PDF: plantar(+) dorsi(-) flexion].

JOINT (ANGLE)		Native		FP		SLC		ITCL	
		withTP	noTP	withTP	noTP	withTP	noTP	withTP	noTP
Tib – Cal (InvEv)	AA	0.3±0.6	0.5±0.5	0.7±0.4	0.3±0.7	0.6±0.6	0.5±0.6	0.5±0.5	0.1±0.6
			d_n=0.35	d_n=0.86	d_n=0.03	d_n=0.49	d_n=0.30	d_n=0.32	d_n=0.39
		d_{tp}=0.35		d_{tp}=0.80		d_{tp}=0.17		d_{tp}=0.70	
	ROM	6.2±3.2	6.5±4.1	5.5±2.1	7.6±4.0	5.5±2.6	5.8±2.3	4.9±2.1	6.9±3.7
			d_n=0.09	d_n=0.26	d_n=0.40	d_n=0.23	d_n=0.12	d_n=0.49	d_n=0.20
		d_{tp}=0.09		d_{tp}=0.67		d_{tp}=0.13		d_{tp}=0.66	
Tib – Cal (IErot)	AA	0.1±1.3	0.3±0.9	-0.3±0.1	0.3±0.5	-0.3±0.7	0.5±0.5	0.1±0.4	-0.3±0.2
			d_n=0.11	d_n=0.49	d_n=0.16	d_n=0.39	d_n=0.38	d_n=0.03	d_n=0.43
		d_{tp}=0.11		**d_{tp}=1.54**		**d_{tp}=1.28**		**d_{tp}=1.24**	
	ROM	6.9±5.9	6.2±3.2	4.1±1.6	4.9±2.3	5.6±4.3	4.2±1.3	4.1±2.7	4.2±1.7
			d_n=0.14	d_n=0.66	d_n=0.46	d_n=0.25	**d_n=0.63**	d_n=0.60	d_n=0.63
		d_{tp}=0.14		d_{tp}=0.40		d_{tp}=0.44		d_{tp}=0.02	
Tal – Cal (InvEv)	AA	-0.1±0.6	0.0±0.6	-0.1±0.5	0.1±0.2	-0.7±0.8	-0.9±0.4	-0.6±0.7	0.0±0.9
			d_n=0.18	d_n=0.08	d_n=0.55	d_n=0.78	d_n=1.52	d_n=0.66	d_n=0.20
		d_{tp}=0.18		d_{tp}=0.52		d_{tp}=0.41		d_{tp}=0.72	
	ROM	5.5±1.5	5.2±1.4	4.9±0.4	5.1±1.1	5.9±1.2	5.1±1.0	5.4±2.7	5.8±3.2
			d_n=0.17	d_n=0.52	d_n=0.28	d_n=0.32	d_n=0.27	d_n=0.02	d_n=0.13
		d_{tp}=0.17		d_{tp}=0.24		d_{tp}=0.70		d_{tp}=0.12	
Tal – Cal (IErot)	AA	0.0±0.4	-0.1±0.5	-0.1±0.7	-0.1±0.4	-0.1±0.8	-0.1±0.7	-0.2±0.3	-0.5±0.7
			d_n=0.24	d_n=0.28	d_n=0.30	d_n=0.17	d_n=0.15	d_n=0.47	d_n=0.88
		d_{tp}=0.24		d_{tp}=0.07		d_{tp}=0.03		d_{tp}=0.61	
	ROM	4.6±1.7	4.9±2.5	6.3±2.4	5.2±0.5	6.1±2.4	4.3±2.0	5.1±3.4	4.5±1.2
			d_n=0.13	**d_n=0.82**	d_n=0.47	d_n=0.73	d_n=0.17	d_n=0.16	d_n=0.12
		d_{tp}=0.13		d_{tp}=0.65		**d_{tp}=0.83**		d_{tp}=0.24	
Tal – Nav (IErot)	AA	-0.2±0.8	-0.6±1.1	-0.1±1.2	-0.7±0.6	-0.3±0.3	-0.4±1.1	-0.3±1.5	-1.2±1.3
			d_n=0.40	d_n=0.12	d_n=0.72	d_n=0.24	d_n=0.19	d_n=0.07	d_n=0.96
		d_{tp}=0.40		d_{tp}=0.65		d_{tp}=0.05		d_{tp}=0.66	
	ROM	7.0±2.4	10.1±5.9	7.8±2.4	6.6±1.7	10.1±2.7	4.9±1.3	7.3±2.8	8.6±4.7
			d_n=0.70	d_n=0.36	d_n=0.17	**d_n=1.24**	d_n=1.07	d_n=0.11	d_n=0.45
		d_{tp}=0.70		d_{tp}=0.59		d_{tp}=2.50		d_{tp}=0.36	
Tal – MT1 (PDF)	AA	-0.8±0.5	-0.9±0.9	-1.1±0.9	-1.2±0.7	-1.2±1.5	-1.7±0.9	-1.2±0.5	-1.2±0.8
			d_n=0.06	d_n=0.42	**d_n=0.62**	d_n=0.36	**d_n=1.21**	d_n=0.69	d_n=0.60
		d_{tp}=0.06		d_{tp}=0.09		d_{tp}=0.37		d_{tp}=0.10	
	ROM	10.3±0.3	9.8±1.3	12.5±3.2	12.3±2.0	14.8±3.9	13.3±2.8	13.5±3.4	13.8±3.4
			d_n=0.47	d_n=0.99	d_n=1.48	**d_n=1.66**	d_n=1.54	**d_n=1.38**	d_n=1.49
		d_{tp}=0.47		d_{tp}=0.07		d_{tp}=0.47		d_{tp}=0.08	

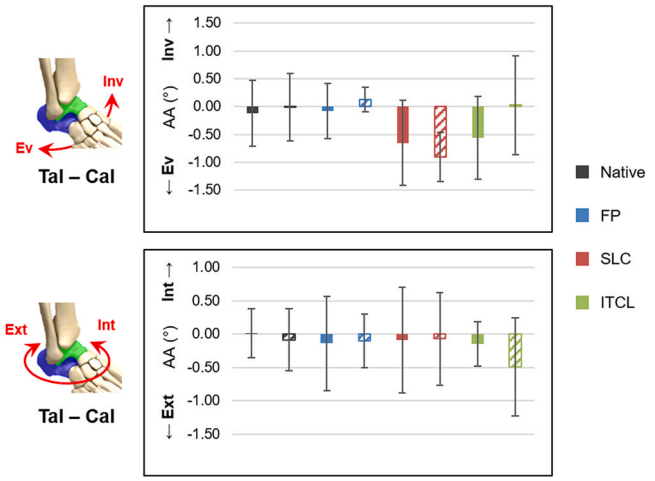

Fig. 3. Average angles (AA) for inversion (Inv) - eversion (Ev) and internal (Int) - external (Ext) rotation at the talocalcaneal joint (Tal-Cal) in the native condition (black) and following sectioning of tablefascia plantaris (FP; blue), spring ligament complex (SLC; red), and interosseous talocalcaneal ligament (ITCL; green) measured over the simulated stance phase of the gait cycle with (solid) and without (hatched) tibialis posterior. [Data represented as mean ± SD across five specimens. No statistically significant differences were observed for any condition with respect to the native condition simulated with tibialis posterior (solid black). No statistically significant differences were observed between the same condition simulated with and without tibialis posterior ($p > 0.1$)].

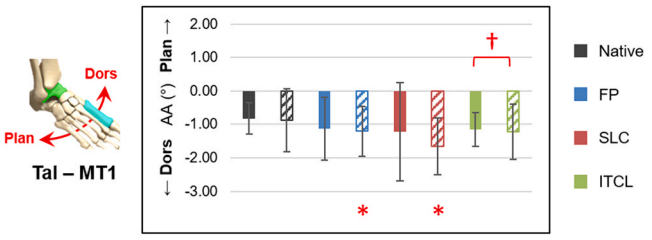

Fig. 5. Average angles (AA) for plantarflexion (Plan) – dorsiflexion (Dors) at the talometatarsal joint (Tal-MT1) in the native condition (black) and following sectioning fascia plantaris (FP; blue), spring ligament complex (SLC; red), and interosseous talocalcaneal ligament (ITCL; green) measured over the simulated stance phase of the gait cycle with (solid) and without (hatched) tibialis posterior. [Data represented as mean ± SD across four specimens. The asterisk (*) represents statistically significant differences with respect to the native condition simulated with tibialis posterior (solid black). The dagger (†) represents statistically significant differences between the same condition simulated with and without tibialis posterior ($p < 0.1$)].

dorsiflexion was greater for all sectioned conditions as compared to the native condition following TibPost elimination ($d > 0.6$). Metatarsal ROM was also higher for all sectioned conditions ($d > 0.99$), with statistically significant results following sectioning the SLC ($d = 1.66$, $p = 0.07$) and ITCL ($d = 1.38$, $p = 0.1$).

4. Discussion

Although much has been written on the anatomy of the medial ankle and hindfoot ligaments, literature on the behaviour of the individual tarsal bones after sequential sectioning of the ligaments during the load bearing condition of the foot is missing. Furthermore, the contribution of TibPost in assisting stabilisation of the medial arch has not been well documented. Our cadaveric gait simulator study assessed these conditions in vitro for various tarsal kinematics at physiological loading regimes. Sequential sectioning of the ligaments showed increased alterations towards a flatfoot deformity. The TibPost force was unable to fully compensate for the ligamentous loss of medial arch support. Only after sectioning the FP, SLC and also ITCL a foot mimicking the deformities of an acquired flat foot deformity was created.

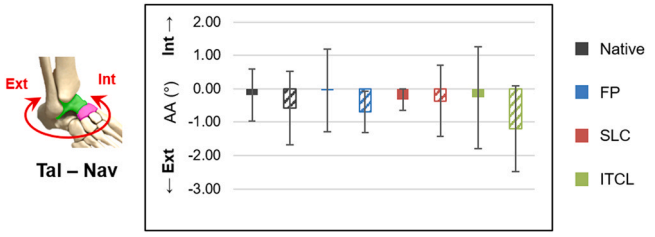

Fig. 4. Average angles (AA) for internal (Int) – external (Ext) rotation at the talonavicular joint (Tal-Nav) in the native condition (black) and following sectioning fascia plantaris (FP; blue), spring ligament complex (SLC; red), and interosseous talocalcaneal ligament (ITCL; green) measured over the simulated stance phase of the gait cycle with (solid) and without (hatched) tibialis posterior. [Data represented as mean ± SD across five specimens. No statistically significant differences were observed for any condition with respect to the native condition simulated with tibialis posterior (solid black). No statistically significant differences were observed between the same condition simulated with and without tibialis posterior ($p > 0.1$)].

Medial ligament sectioning, including the ITCL, caused tibiocalcaneal valgus and a calcaneal external rotation at subtalar level. Both deformities were larger when the TibPost force was eliminated. The decrease in tibiocalcaneal inversion in the absence of an active TibPost force was expected owing to its primary action as foot inverter. Also, sequential ligament sectioning tended to reduce talocalcaneal varus, and created an external rotation of the calcaneus, most noticeably after sectioning the ITCL. These findings were similar to the study by Watanabe et al. reporting the importance of TibPost in flatfoot deformity [21]. However, since sections in our study were performed sequentially, we found that significant calcaneal valgus and external rotation occurs only after ITCL sectioning. TibPost was, for a great part, able to compensate for calcaneal valgus, although this correction was only partial after ITCL sectioning. Interestingly, before sectioning the ITCL, the calcaneus internally rotated with sequential sectioning of the medial ligaments. A possible explanation for this could be the pivoting of the calcaneus around the strong ITCL. Only after sectioning the ITCL, the anterior part of the calcaneus was able to translate laterally and rotate externally. This led to an increase in the talocalcaneal angle, which is well known as a characteristic of an acquired flatfoot deformity [22].

Talonavicular coverage, indicated by the internal rotation of the navicular relative to the talus, noticeably decreased following TibPost elimination. The TibPost was able to largely compensate for the extensive ligament loss at this level and in this direction. Sectioning the SLC increased the ROM at the talonavicular joint with a functioning TibPost; the ROM reduced after eliminating the TibPost, but was accompanied by external rotation of the navicular. However, failure of both the TibPost and the medial ligaments, including the ITCL, caused a noticeable increase in navicular external rotation, although not statistically significant. Thus, an increased contribution of the TibPost was observed in talonavicular joint stabilisation following medial ligament failure.

Ligament sectioning increased flattening of the medial arch. TibPost elimination prior to ligament sectioning did not result in alterations in the medial arch. However, FP and SLC sectioning led to a significant increase of first metatarsal dorsiflexion, more prominently after TibPost elimination. This indicated the importance of TibPost on the alignment of the first metatarsal. ITCL sectioning did not influence this deformity further. This is in line with findings of Williams et al. who reported a decrease in Meary's angle as the strongest radiographic finding associated with SLC failure [8].

Our research highlights the importance of the ITCL in acquired flatfoot. A failure of the ITCL was vital to obtain a cadaveric flatfoot model that mimics the acquired flatfoot deformity observed clinically. The involvement of additional ligaments apart from the SLC in this deformity might explain why a simple SLC repair is potentially inferior to a SLC repair coupled with corrective osteotomies and/or augmentation [23,24]. We acknowledge that an anatomical repair of

both the SLC and the ITCL is probably unachievable since this would be too extensive; moreover, it would still not guarantee native physiological attachments, mechanical properties and proprioception in the reconstructed ligaments. Also ligament reconstructions using tendon grafts and especially internal braces risk to create acute stops in the physiologic movements, altering biomechanics of the foot and provoking disabilities [25]. Another proposition could be the lateral column lengthening, which has been shown to be superior to a medial slide calcaneal osteotomy in correcting the midfoot deformity [24]. This procedure rotates the cuboid and anterior talar facet inwards correcting the calcaneal external rotation provoked by ITCL failure. Thus, it unloads the medial ligaments, and limits the need for a strong anatomical ligamentous reconstruction.

Findings from our study also show that the function of the TibPost and the medial ligaments is not mutually distinctive, i.e. their stabilising effects are complementary and interdependent. The insertion of the TibPost tendon is very large and branches out of the medioplantar ligaments [26], thereby influencing the intrinsic tightness of these ligaments. This ought to be vital in transferring stresses in the complex bio-tensegrity system [27]. Failure of one element potentially increases the stress on the other, i.e. a loss of tension in the TibPost potentially results in the ligaments losing tension. Similarly, if the ligaments are damaged, the TibPost loses an indirect attachment point to the tarsals. Therefore, it is not uncommon to see associated failure of the TibPost and SLC [28]. This complex anatomy also explains how the TibPost is able to control deformities occurring proximally to its insertion. For instance, the underlying mechanism of how the TibPost blocks the external rotation of the calcaneus without having attachments on the calcaneus has already been described by Lapidus (1963). Owing to the TibPost tendon insertion on the cuboid and the calcaneocuboid ligaments, TibPost contraction internally rotates the calcaneus. This induces a medial shift in the middle and anterior calcaneal facets, which blocks external rotation of the talus [29].

One of the limitations of this cadaveric study was the use of five specimens. Although similar studies with comparable number of specimens have been reported in the literature [30], qualitative differences reported in this study which are not statistically significant will need to be strengthened in future work. Secondly, only one sequence of sectioning was studied due to the limited number of specimens. Although ITCL was sectioned last, because it is most approachable only after the SLC has been sectioned, an alternative could be to section it through the sinus tarsi. Lastly, further biomechanical tests, apart from replicating gait cycles on a physiological simulator, need to be performed to identify the role of these different ligaments in stabilising the subtalar and midfoot joints.

5. Conclusion

The function of the tibialis posterior tendon and the medial ligaments is not mutually distinctive, i.e. their stabilising effects are complementary and interdependent. Failure of the and interosseous talocalcaneal ligament is necessary to develop a cadaveric flatfoot model. This ligament should not be neglected in understanding flatfoot pathology.

Declarations of interest

None.

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