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Acquired flatfoot in adults

Master's diploma thesis

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Declaration of authorship

I declare that the work presented here is my own, to the best of my knowledge and belief, with use of information gathered from various online journals, articles and books, consultations and seminars. Under no circumstances, has any work been copied, forged or changed and where formulations and ideas are taken from sources, it has been, with every effort, cited as such.

Prague, August 2017 Mohamed Khadura

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Abstract

Title: Acquired flatfoot in adults

Background:

Flatfoot deformity is one of the common diagnoses in the orthopedic field and physiotherapy, its common in adults as acquired, there is still a large incidence of flatfoot in adolescence and many cases of adult flatfoot are frequently presented as residual pediatric flatfoot. The treatment also has wide field according to type of cause.

Aims:

The aim of this literature review was to enhance knowledge on the persistence of the acquired flatfoot condition in adult's population by determining the causes of flatfoot with contributing factors that affect the medial longitudinal arch and progression of the deformity, and review of the most common treatment.

Method:

An electronic database search was conducted to obtain articles from relevant journals (from early 1990 to end 2015). The information was collected also from textbooks.

Results:

Regarding the causes of acquired flatfoot in adult, a good base of evidence stems from a number of articles review, the posterior tibialis tendon dysfunction is the most cause of acquired flatfoot.

The sensory motor stimulation technique is not the most treatment used to treat acquired flatfoot in adults and the most used treatment is the surgical procedure.

Conclusion:

Regarding the causes of adults acquired flatfoot in our results and the evidence from a number of articles review, we found that the most common cause of the

acquired flatfoot in adults is the posterior tibialis tendon dysfunction.

The surgical is more using than conservative treatment, and sensory motor stimulation technique is most using to treat flatfoot associated with dysfunction of plantar intrinsic muscles.

Keywords:

Acquired, Flatfoot, Longitudinal arch, Posterior tibialis tendon dysfunction, Intrinsic foot muscles, Conservative treatment and Sensory motor stimulation technique.

Abstrakt

Název: Získané plochonoží u dospělých jedinců

Úvod: Plochá noha je jednou z častých diagnóz jak ve fyzioterapii, tak v ortopedii. V dospělosti je tato deformita velmi běžná jako vada získaná. V dospívání je v mnoha případech výskyt plochých nohou prezentován jako zbytková pediatrická plochá noha. Léčba může být různá podle typu příčiny.

Cíle: Cílem této práce bylo zvýšit povědomí a rozšířit znalosti o přetrvávání získané vady plochých nohou v populaci dospělých pomocí určení příčin plochých nohou s přispívajícími faktory, které ovlivňují mediální podélný oblouk nohy a průběh deformity, dále vytvořit přehled nejčastější léčby.

Metody: Bylo provedeno elektronické vyhledávání v databázích s cílem získat články z příslušných zdrojů - časopisů (od počátku roku 1990 do konce roku 2015), a mimo jiné byly informace shromážděny z učebnic.

Výsledky: Při hledání příčin získané deformity plochých nohou u dospělých je dobrým výchozím zdrojem množství recenzí a článků, ze kterých vyplývá, že nejčastější příčinou této vady je dysfunkce svalu Tibialis posterior.

Technika senzomotorické stimulace není nejčastěji používanou léčbou získaného plochonoží u dospělých, nejpoužívanější je však chirurgické řešení.

Závěr: Pokud jde o příčiny získané ploché nohy v dospělosti, zjistili jsme na základě našich výsledků a výsledků vyhledávání z mnoha zdrojů, že nejčastější příčinou je dysfunkce svalu tibialis posterior.

Nejčastěji používanou technikou pro ovlivnění léčby ploché nohy spojených s dysfunkcí krátkých svalů planty je senzomotorická stimulace.

Klíčová slova: Získaná plochá noha, podélný oblouk klenby nohy, dysfunkce svalu Tibialis posterior, krátké svaly nohou, konzervativní léčba a technika senzomotorické stimulace.

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List of abbreviations

AAFD- Adult Acquired Flatfoot Deformity

PTTD- Posterior Tibialis Tendon Dysfunction

ROM- Range of Motion

EMG- Electromyography

MRI- Magnetic Resonance Image

MTP- Metatarsophalangeal

TENS- Transcutaneous Electrical Nerve Stimulation

SMST- Sensory Motor Stimulation Technique

1. Introduction

Flatfeet deformity is characterized by loss of the medial longitudinal arch, forefoot abduction, hindfoot eversion, and often Achilles tendon contracture. Flatfeet are postural deformity in which the arches of the foot collapse, with the entire sole of the foot coming into complete or near-complete contact with the ground.

Flatfeet are wide term used to describe a decrease longitudinal foot arch with calcaneus valgus. We can divide them in two groups (Classification of flatfeet)

Congenital flatfeet (Flatfeet in children - pes planovalgus):

During foot developing until 6-7 years of age. The lower extremity is physiological positioned in calcaneal valgus, knee valgus and hip valgus and internal rotation. Around the age of 6 years, the knee joint axis straightens and the calcaneal valgus decreases. Calcaneal valgus greater than 20 degrees is defined as pathological. Next calcaneal valgus, this deformity also includes internal rotation of the ankle axis (Blackman et al., 2009).

Flexible – pes calcaneovalgus when child with flexible flatfoot stand, the arch of the foot disappears. Upon sitting or when the child is on tiptoes, the arch reappears. Although called "flexible flatfoot," this condition always affects both feet, but the rigid flatfoot – congenital steep of talus the foot arch disappears in all condition of the foot with loading or without loading.

Acquired flatfoot:

This is static foot deformity that develops due to long-term overuse. It can develop from childhood flatfeet or in a foot without any previous deformity. Next long-term exposure to static loading, poorly fitting footwear and hormonal imbalances (pregnancy, menopause) also contribute to the development of flatfoot (Kolář et al., 2013).

Flatfoot can also develop as an adult ("adult acquired flatfoot") due to injury, illness, unusual or prolonged stress to the foot, faulty biomechanics, or as part of the normal aging process, with increase connective tissue laxity this is most common in women over 40 years of age. Known risk factors include obesity, hypertension and diabetes. Flatfoot can also occur in pregnant women as a result of temporary changes, due to increased elastin (elasticity) during pregnancy. However, if developed by adulthood, flat feet generally remain flat permanently (Blackman et al., 2009).

Adult Acquired Flatfoot Deformity (AAFD) can be as resulting by degenerative disease in malalignment of the midfoot and hindfoot secondary to posterior tibialis tendon dysfunction and increasing implication of ligament pathologies or neuromuscular disease – paresis, myopathies (Spratley et al., 2013).

Adult acquired flatfoot deformity is a common disorder characterized by collapse of the medial longitudinal arch, forefoot abduction, and hindfoot eversion can be happened by Posterior Tibialis Tendon Dysfunction (PTTD) is associated with adult acquired flatfoot deformity in both a chronic and a traumatic fashion although the exact role of the failure of the posterior tibialis tendon is not completely known. The valgus deformity resulting from collapse of medial supporting ligaments leads to increased eversion of the calcaneus due to the position of the Achilles tendon lateral to the axis of rotation of the subtalar joint. Degradation of the medial supporting ligaments over time results in a painful and visibly deformed flatfoot (Vaudreuil et al., 2014).

Rehabilitation is indicated for patient with flatfoot, physical therapy includes sensorimotor exercises, foot sole facilitation, training the foot pressure distribution, the practice of three point support and “short foot” with lower extremity in neutral alignment. Treatment also incorporates soft tissue techniques; foot joint mobilization and stretching of the short muscles and relaxation witch have hyper tone (Kolář et al., 2013).

1. 1. Methodology

- **Research questions**

Q1- The main cause of acquired flatfeet is the dysfunction of the tibialis posterior muscle?

Q2- The sensory motor stimulation technique is the most effective therapeutic technique for the acquired flatfeet in the adults?

- **Aims**

The aim of this thesis is to search, analyze and present up to date evidence based information concerning practical and theoretical knowledge concerning patients who suffer from acquired flatfeet in adults by represent the rehabilitation programs.

- **Theoretical justification**

Although there have been published many studies about acquired flatfoot, still there are several etiologies. Due to the fact that these theories differ from each other in such a great manner, it makes it difficult to focus on specific similarities or even draw comparisonable lines between them.

- **Methodology**

The diploma thesis will be written in the form of a literature review.

Population: No strict criteria have been established for the populations investigated in the individual studies to be reviewed. For each article or/and study, the following criteria about the population studied will be noted:

- Male, Female
- Age: 18 to 88 years
- Activity level:
    - Professional athlete
    - Non-professional athlete
    - Non-sport

- Health state:
    - Injured and non- Injured
    - Non-operational and after operational

- Without neurological problems

Measurements:

- Static posture
- Dynamic movements
- Foot print
- X-ray
- Electromyography (EMG)
- Magnetic Resonance Image (MRI)
- Functional tests
- Cadaveric foot model
- Changing of plantar pressure

Method of data gathering: Articles for analysis will be gathered from available Internet resources. Attempt will be made to gather data for general discussion from so called "grey literature".

Inclusion criteria:

- Search in following databases / search engines: EMBASE, EBSCO, Ovid, ProQuest, Medline and Wiley Interscience.
  - Search with a combination of the following words: flatfeet, tibialis posterior and foot arch.
- Articles and case reports published in the year 1990 or later.

Exclusion criteria: □

- Written in language other than English, Arabic. □
- Subjects with diseases that can have impact on the cause of flatfeet as neurological diseases. □

1. 2. Anatomy of ankle joint and foot

The tarsus (ankle) is the proximal region of the foot and consists of seven tarsal bones (Figure 1). The tarsal bones are much greater in size than the small carpal bones, with the two most proximal bones (the talus and calcaneus) being significantly larger than their more distal counterparts. Joints between tarsal bones are called inter tarsal joints.

The tarsals include the talus (ankle bone) and calcaneus (heel), located in the posterior part of the foot. The calcaneus is the largest and strongest tarsal bone.

The anterior tarsal bones are the navicular and three cuneiform bones called the third (lateral), second (intermediate), and first (medial) cuneiforms, and the cuboid.

The talus is the only bone of the foot that articulates with the fibula and tibia. It articulates on one side with the medial malleolus of the tibia and on the other side with the lateral malleolus of the fibula. These articulations form the Talocrural (ankle) joint.

The metatarsus is the intermediate region of the foot and consists of five metatarsal bones (Figure 1). Each metatarsal consists of a proximal base, an intermediate shaft, and a distal head. The metatarsals articulate proximally with the first, second, and third cuneiform bones and with the cuboid to form the tarsometatarsal joints. Distally, they articulate with the proximal row of phalanges to form the metatarsophalangeal joints.

The phalanges comprise the distal component of the foot (toes). They are numbered (I to V) beginning with the great toe (Hallux), from medial to lateral (Figure 1).

Each phalanx (singular) consists of a proximal base, an intermediate shaft, and a distal head. Joints between phalanges of the foot, like those of the hand, are called Inter-phalangeal joints (Tortora et al., 2012).

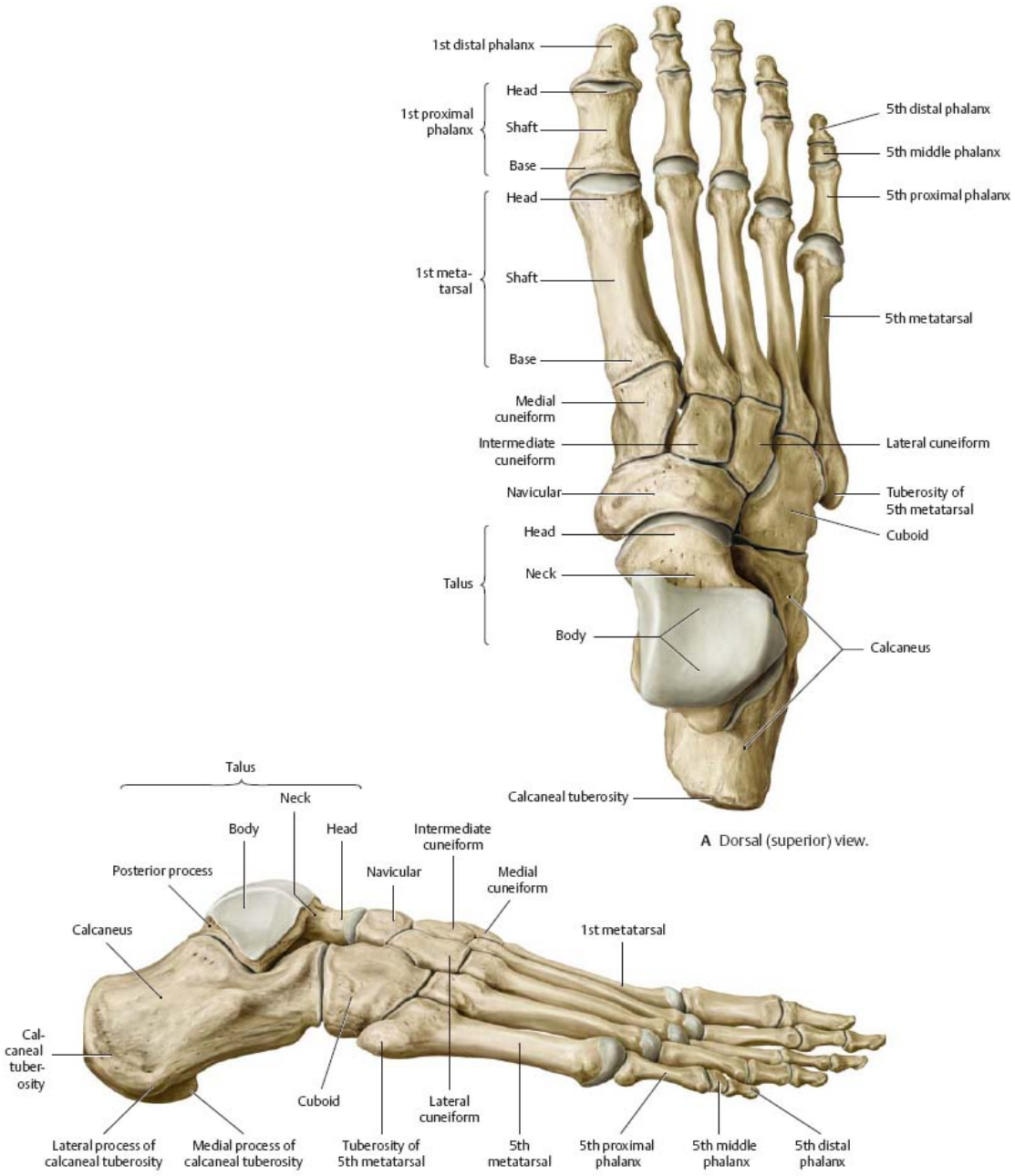


Fig. 1: Anatomy of the foot and ankle joint (Gilroy et al., 2008).

1. 3. Stability of ankle joint and foot

The ankle joint gains its stability from the joint capsule and ligamentous support. The joint capsule is anteriorly and posteriorly weak, but enforced by collateral ligaments on both sides (Kolář et al., 2013).

Supporting ligaments group which are consisted of three:

- Anterior inferior tibiofibular ligament, it is flat and strong ligament runs from the anterior edge of the lateral malleolus to the anterolateral tubercle of the tibia.
- Posterior inferior tibiofibular ligament, it is consist of superficial and deep portion. The deep portion runs from the posterior margin of tibia to the posteromedial aspect of the distal fibula. The superficial portion functions along with the anterior inferior tibiofibular ligament to ensure that the fibula remains held tightly with tibia.
- The interosseous ligament is a thickening of the interosseous membrane. This ligament is more flexible and allows a subtle diastasis of the tibia and fibula during ankle dorsiflexion (Norkus et al., 2001).

Lateral Ankle stability confirmed by lateral collateral ligaments, which consists of the anterior talofibular ligament, calcaneofibular ligament and the posterior talofibular ligament. The anterior talofibular ligament is a thickening of the anterior capsule and its primary function of it is to resist inversion when the ankle is in plantar flexion position, also resists external tibial rotation and anterior draw of the talus (Burks & Morgan, 1994). The calcaneofibular ligament lies in a horizontal position during ankle plantar flexion but comes to be vertically during dorsal flexion. It remains under tension throughout this arc of movement, although the tension is greatest during ankle dorsal flexion where the ligament most effectively resists inversion. During ankle plantar flexion the calcaneofibular ligament guides calcaneal inversion, which may be observed when stand on tiptoes with normal feet. The posterior talofibular ligament runs from the posterior lateral part of the talus to the lateral malleolus (Kjaersgaard et al., 1988). Lateral collateral ligament is the weaker ligament than medial collateral ligament, which clinically predisposes the lateral malleolus to injuries (subluxation, dislocation) during inversion (Kolář et al., 2013). The sequence is anterior talofibular ligament, calcaneofibular ligament and posterior talofibular ligament. Once the anterior talofibular ligament has ruptured there is a significant increase in internal hindfoot rotation, which predisposes to further ligament injury (Kjaersgaard et al., 1988).

The medial collateral ligament *"Deltoid ligament"* consists of three parts: (tibiocalcaneal, tibiotalar and tibionavicular). It is strongest ligament stabilizing the ankle joint (Attarian et al., 1985; Kolář et al., 2013). The ligament is formed from superficial part and deep part. The superficial deltoid is divided into three slips, which originate from the anterior inferior medial malleolus (tibia) and insert into the navicular (tibionavicular ligament) and the sustentaculum tali and tuberosity of the calcaneus (tbiocalcaneal ligament). This ligament acts mainly to prevent eversion of hindfoot. The deep deltoid ligament originates from the posterior border of the anterior colliculus, intercollicular groove and posterior colliculus before running transversely to insert into the non-articular surface of the medial talus (tibiotalar ligament). The posterior medial aspect of this ligament is covered by tibialis posterior tendon sheath (Golano et al., 2010).

1. 4. Development of ankle joint and foot

At the 4th week of gestation, the development of the foot begins (Bosch et al., 2007). Between the 16th week and the 28th week of gestation, the foot grows by means of torsion of the talus on the calcaneus. This growth enhances the feet to become everted and gradually makes the feet become a more neutral position (Bernhardt, 1988). The length of the foot at birth is an average of 7-10 cm. half of the adult foot length is reached by first year after birth (Bosch et al., 2007).

The average width of the foot is half of its length at birth and a third of its length in an adult (Bernhardt, 1988) it is common to have up to 5 degrees of forefoot varus up until the age of 2 years. Later this forefoot varus decreases by means of rotational changes in the talus, caused by rotational changes in the tibia. The Hindfoot (calcaneus) is in varus position of 22 degrees in relation to the tibia. This growth continues to rotate into valgus position until it reaches a more perpendicular position by the time of maturity (Dalley & Moore, 2004).

The foot of the newborn is quite elastic and flexible, there is high range of motion (ROM) of the dorsiflexion and forefoot adduction more than planter flexion and forefoot abduction, and the metatarsals are also in adduction, but this decreases with age (Bernhardt, 1988). A child younger than the age of 3, goes through rapid growth changes that result in many findings that may seem abnormal, especially when comparing them to adult-like patterns, therefore characteristics of the development are

more significant as the child grows (Sass & Hassan, 2003). When a child below the age of 3 presents with flat feet, it is almost meaningless, unless in case of a rigid-type, because of the presence of the fat pad, lack of ligament tightness, muscle strength and walking experience. However as the child grows, the presence of flatfeet can be a sign of a delay in development or a deformity and becomes more of a concern that may need further evaluation (Zoran et al., 2009).

The longitudinal bony structure of the foot arch is found at birth. However, in infants, it is filled with fatty tissue leading to the impression of flat foot. In infants, the heel is positioned in slight varus together with forefoot supination and accompanied by bilateral genu varum (Kolář et al., 2013). The correction of genu valgum and development of the medial longitudinal arch usually occur around the same period of time, from 5-6 years of age (Leung et al., 2005).

1. 5. Biomechanics of ankle joint and foot

The biomechanics of the foot and ankle are important to the normal function of the lower extremity. The foot is the terminal joint in the lower kinetic chain that opposes external resistance. The movement of the foot and ankle influences the ability of the lower limb to attenuate the forces of weight bearing. It is important for the lower extremity to distribute compressive, tensile, shearing, and rotatory forces during the stance phase of gait. Inadequate distribution of these forces could lead to abnormal stress and the eventual breakdown of connective tissue and muscle. The combined effect of muscle, bone, ligaments, and normal foot biomechanics will result in the most efficient force attenuation in the lower limb.

The foot must bear combined forces that reach more than 50% of the individual's body weight. In the normal weight distribution of the foot, the body's weight is in fact distributed over three points mainly. These points form a triangle and they are: the heel, under the heads of the first and fifth metatarsals (Dalley & Moore, 2004). The first metatarsal is thicker than the others because it bears more weight (Tortora et al., 2012)**,** The ankle joint is usually characterized as a uniaxial hinge joint with one degree of freedom (Kolář et al., 2013).

During walking, the talus transmits about half the weight of the body to the calcaneus. The remainder is transmitted to the other tarsal bones (Tortora et al., 2012).

Normal biomechanics of the foot and ankle can be divided into static and dynamic components.

1. 5. 1. Static biomechanics of ankle joint and foot

Static biomechanics when the foot is free to move and the leg is fixed (open kinetic chain), the subtalar joint is a gliding joint in supination. The calcaneus moves anteriorly, inferiorly, and medially under the talus. During pronation, it moves posteriorly, superiorly, and laterally (Otis, 1988). The talus can be palpated laterally during supination as the calcaneus moves medially, and palpated medially during pronation as the calcaneus moves laterally. The subtalar joint is a hinge or uniaxial joint with an axis running downward, posteriorly, and laterally. This joint orientation results in triplanar motion, providing pronation and supination and in the sagittal orientation there is wide variation of the subtalar joint with respect to the horizontal plane and its rotation from the long axis of the foot (Inman, 1976).

The subtalar joint contributes more to inversion/eversion the more closely its axis is aligned with the longitudinal axis of the foot, and contributes more to abduction/adduction the more closely its axis is aligned with the longitudinal axis of the leg. The subtalar joint orientation also affects motion in the transverse view, providing more eversion/inversion and less dorsiflexion/plantar flexion as the subtalar joint axis approaches the longitudinal axis of the foot.

The transverse tarsal joint (Chopart's joint) amplifies and is interdependent with motions of the ankle joint and subtalar joint. As the subtalar joint moves into pronation, the transverse tarsal joint is pulled toward pronation resulting in flattening of the medial longitudinal arch and increasing flexibility of the foot and than pulled toward supination which makes elevation of the arch and increasing rigidity of the foot.

Tarsometarsal joints (Lisfranc joint) are providing motion, which is translatory or planar. The first ray connects the first metatarsal and medial cuneiform bones. The axis of motion is uniaxial and triplanar, combining dorsiflexion and inversion or plantar flexion and eversion with little contribution to abduction or adduction.

Rays two through four provide flexion extension motion while the fifth ray allows pronation and supination to occur between the metatarsal and the cuboid.

The metatarsophalangeal joints are biaxial, providing pure dorsal flexion/plantar flexion and abduction and adduction. The interphalangeal joints are hinge joints, permitting pure flexion and extension (Garbalosa et al., 1994). The static tripod or

semi-rigid is supporting the body weight, which characteristics of each individual foot will influence its dynamic response to walking and running.

1. 5. 2. Dynamic biomechanics of ankle joint and foot

The dynamic function is required for foot during walking and the body requires a flexible foot to accommodate the variations in the environment and semi-rigid foot, which can act as a spring and lever arm for the push-off during gait and rigid foot to stabilize the body weight.

During normal walking, the pelvis goes to maximum rotation in each gait cycle and the tibia is rotating in the same period (Lnman et al., 1981). The lower limb generally rotates internally during swing phase and in early stance phase and then rotate externally until complete the stance phase and toe-off has occurred (Manley, 1980).

At heel-strike, the tibia is rotated internally and the ankle joint with slight plantar flexion or in its neutral position (Soderberg, 1986). The foot continues toward the floor immediately following heel-strike, with the dorsiflexor muscles controlling this plantar flexion movement to prevent the foot from slapping down to the foot flat position (foot drop), the rotation of the tibia is transmitted through the ankle mortise the talus (Mann & Inman, 1964) and this rotation combined with ankle plantar flexion and tends to shift the forefoot medially. When the heel contact with the floor the body weight transmitted to the talus, creating a pronation at the subtalar joint, which turn stresses the structures of the medial arch.

The talus rotates medially on the calcaneus about the subtalar axis, forcing the calcaneus into pronation position. The foot quickly pronates (Wright et al., 1964) and this gives free motion at the Chopart joint and that remains the foot flexible, distal to the navicular and cuboid, and can bend into close contact with the supporting surface. At the foot flat position and the forefoot is fixed on the ground, the lower limb begins rotating externally. The external rotation of the ankle mortise is transmitted to the talus as external rotation continues; the foot supinates, producing increased stability at the Chopart's joint and along the longitudinal arch of the foot and this stability improved by the increasing body load being carried (Mann & Inman, 1964).

When the leg has passed over the foot, ankle dorsiflexion is initiated. After heel rise, the ankle joint moves back into plantar flexion, forcing the metatarsophalangeal joints to dorsiflexion. Since the plantar aponeurosis wraps around the metatarsal heads, a "windlass effect" takes place, which increases tension

across the longitudinal arch, further elevating the arch and increasing foot stability. Before toe-off, the combination of weight bearing, windlass effect, and supination ensures that the foot is in a maximally stable position for push-off (Chan & Rudins, 1994) after that the foot rotates medially and again the pronating the foot and unlocking the Chopart's joint the foot returns to its flexible state for the swing phase of gait (Basmajian, 1985).

1. 5. 3. Foot muscle activity during gait cycle

The activity of lower extremity muscles during walking have shown many of the changes in levels of EMG activity occur at 15-20% of the gait cycle, when the foot adapts to the supporting surface (Basmajian, 1985).

The tibialis anterior: has major activity at the end of swing to keep the foot in a dorsal flexed position (Winter & Yack, 1987). Immediately after heel contact, the tibialis anterior generates forces to lower the foot to the ground in opposition to the plantar flexing ground reaction forces. In some individuals, the tibialis anterior plays a minor role in pulling the leg forward over the foot shortly after foot flat and the other activity commences the toe- off and results in dorsiflexion for foot clearance during mid-swing.

The extensor digitorum: activates with the tibialis anterior. It functions after heel contact and to dorsal flexion the foot and toes for clearance during swing. The minor activity during push-off and appears to be a cocontraction to stabilize the ankle joint.

The gastrocnemius and soleus: begins just prior to heel contact and rises during stance, reaching peak just before mid-push-off (50% stride). From foot flat to 40% stride, the muscles lengthen as the leg rotates forward for controlling the ankle. During push- off, the posterior calf muscles shorten to actively plantar flex the foot and to generate an explosive push-off. Activity rapidly drops until toe-off, where low level of gastrocnemius activity continues into swing, probably showing the gastrocnemius acting as a knee flexor.

The peroneus longus: activity during weight acceptance, which appears to stabilize the ankle and during push-off (50%) acting as a plantar flexor and during early swing is likely a co-contraction to the tibialis anterior to control the amount of foot dorsiflexion and supination (Basmajian & Stecko, 1963; Mann & Inman, 1964).

The group of intrinsic muscles: covered by the plantar fascia (flexor digitorum brevis, abductor hallucis, and abductor digiti minimi) active at 35% of the gait cycle during the heel rise the concentration of body weight on the forefoot, and the beginning of foot resupination and to assists the foot in becoming more rigid as it resupinates (Cavanagh et al., 1981).

1. 6. Kinesiology of ankle joint and foot

The foot includes the segments of lower extremity, located in the distal part to the ankle joint. The foot, divided into two corresponding lines at Chopart's joint (transvers tarsal joint) and Lisfranc's joint (tarsometatarsal joint) (Figure 2).

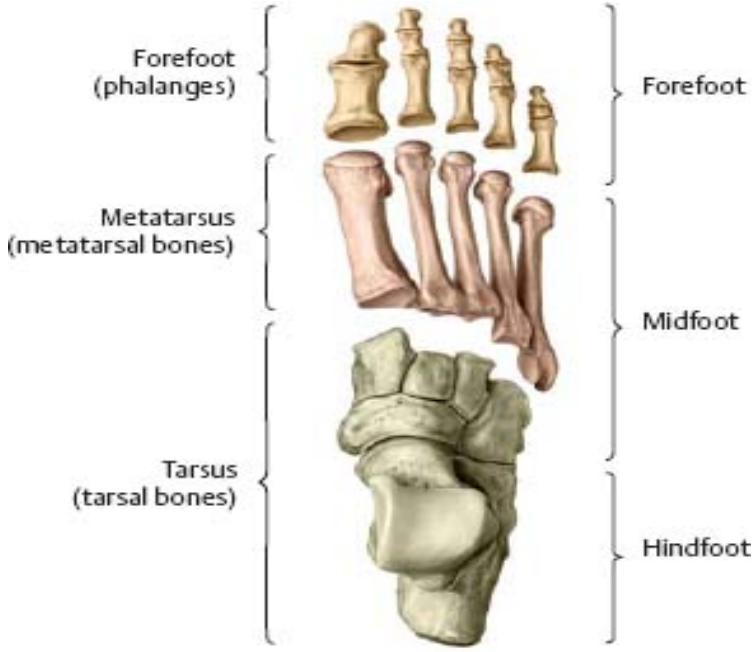


Fig. 2: The joints and foot segments (Gilroy et al., 2008).

The foot can be divided into 3 parts:

- Hindfoot (posterior tarsals): formed by two large tarsal bones (talus and calcaneus)
- Midfoot (front tarsals): formed by five small tarsal bones (cuboid, navicular and cuneiforms)
- Forefoot (metatarsal and toes): formed by the bones of the instep and phalanges of the toes.

Chopart's joint divides the Hindfoot from the midfoot and forefoot. The majority of the movement between the hindfoot and forefoot happen at Lisfranc's joints.

Talocrural joint (upper ankle joint):

The Talocrural joint is a combined joint; it is formed by distal end of the tibia and the fibula with talus. The articulation between the tibia and fibula forms a morphological fork sitting on the head of the talus, the trochlea of the talus is wider anteriorly. Consequently, malleoli are pushed away one another with foot dorsiflexion (Kolář et al., 2013). It functions almost as a uni-planar hinge joint. The talus is wider anteriorly than posteriorly and this shape provides bony stability during dorsiflexion. There is a small degree of coronal plane rotation. As a result dorsiflexion causes the forefoot to point laterally whilst plantar flexion causes the forefoot to point medially (Wright et al., 1964). The normal ROM is 10 – 20º of dorsal flexion and 25 – 30º plantar flexion. Full ankle dorsiflexion provides only 11º of internal tibial rotation and during toe-off requires 19º of internal tibia rotation to allow a propulsive gait (Ramachandran, 2011).

Lower ankle joint:

It is an articulation between the talus and the other bones that allows an oblique sloping of the foot structure in relation to the talus set on a bifurcate formation of the talocrural joint. It is an independent joint between the posterior surface of mutual articulation between the talus and calcaneus. The anterior section is further divided into a medial segment (talocalcaneonavicular articulation) connecting the two anterior joint surfaces under the head of the talus with the navicular bone. This composite is laterally attached to an articulation between the calcaneus and cuboid (articulation calcaneocubiod).

Subtalar joint (articulatio subtalaris):

This joint consists of the following two separate joints: the posterior articulation is formed between the inferior posterior talar facet (facies articularis talaris posterior) and the superior posterior facet of the calcaneus head (faciet articulatio calcanearis posterior) (Dawe & Davis, 2011; Kolář et al., 2013). The anterior articulation is formed between the anterior superior facets of the talar head and the concave surface of the navicular. This talocalcaneonavicular joint functions as a ball and socket. These two joints are separated by the sinus tarsi and have separate joint capsules although they share a similar axis of rotation (Hertel, 2002) (Figure 3).

The axis passes obliquely from the posteriolateral side to the anteriormedial, and at the same time from the lower posterior aspect anteriorly and superiorly. This

axis determines the motions of the entire lower ankle joint. The subtalar joint results primarily in foot rotation in the frontal plane, in inversion, in eversion (or supination and pronation), partially in adduction and abduction in the transverse plane due to the fact that the movement axis also forms a certain angle with it (Kolář et al., 2013).

The subtalar joint is able to move to inversion by 20º and eversion by 5º in the normal foot. This is reduced in patients with flat feet and may be reduced to around 12º (Nordin & Frankel, 2001). In normal feet, 1º of tibia rotation results in 1º of subtalar motion (Ramachandran, 2011) (Figure 4).

Transverse Tarsal joint (Chopart's joint):

The Chopart's joint (midtarsal joint and articulatio tarsi transversis) is the articulation between the talus, navicular and the cuboid with the calcaneus. This joint includes almost the entire anterior section of the lower hindfoot joint with the exception of the talocalcaneal portion at the medial aspect. However, the Chopart's joint anatomically formed by two joints (calcaneocuboid and talonavicular). From the kinesiological perspective, it is considered a functional unit that closely cooperates with other foot joints (Kolář et al., 2013) (Figure 3).

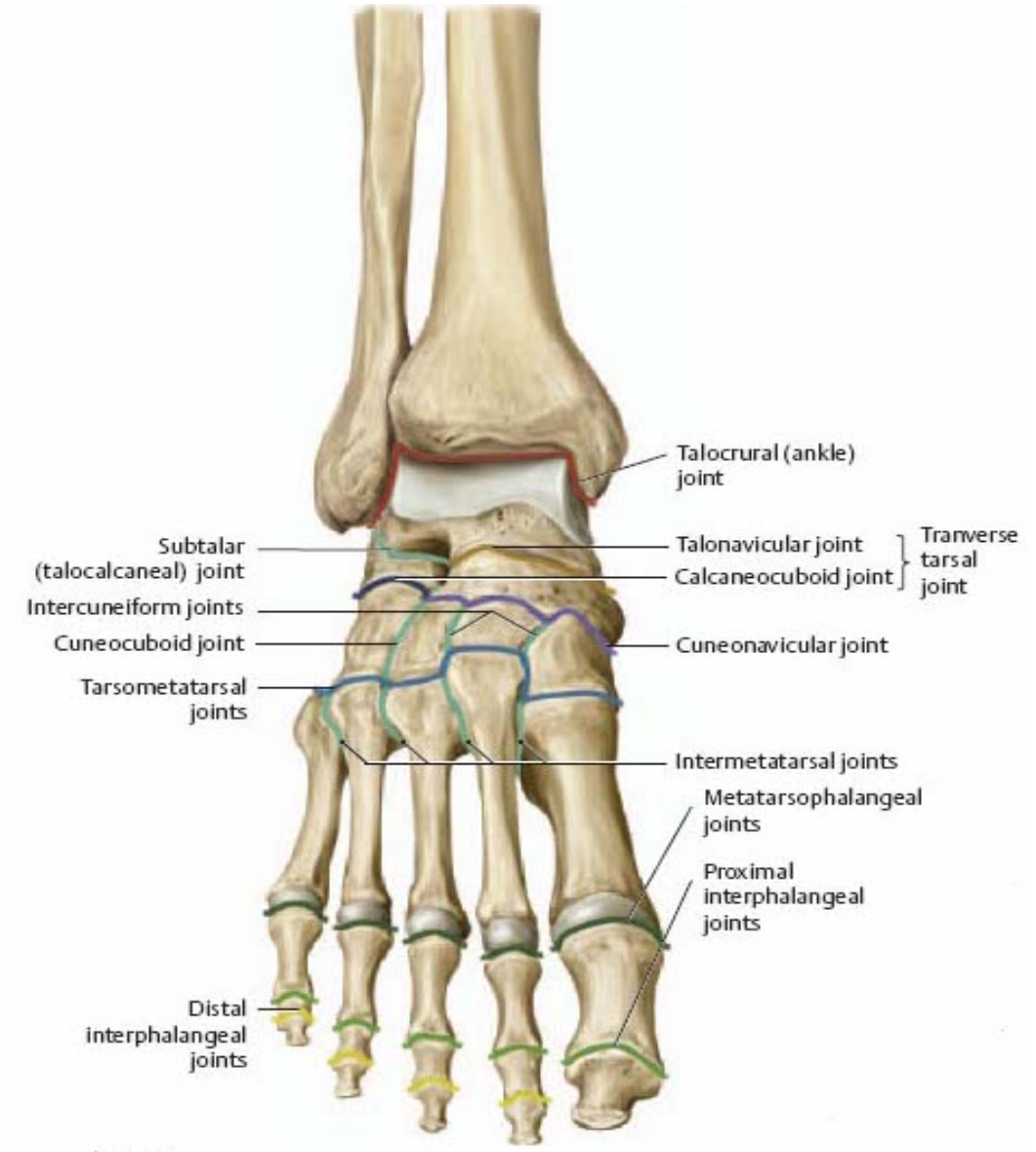


Fig. 3: The subtalar joint and transverse joint (Gilroy et al., 2008).

Transverse tarsal motion is key to movement between flexibility and rigidity of the midfoot during gait (Dawe & Davis, 2011). The movement of the joint is described as rotation around two axises: longitudinal axis and oblique axis. The course of the longitudinal axis allows movements primarily in the frontal plane supination and pronation or inversion and eversion. This allows the Forefoot and midfoot to keep contact with floor without taking in consideration the alignment of the hindfoot with subtalar joint movements. The oblique axis of the Chopart's joint, in comparison to the longitudinal axis, is oriented steeper and more obliquely.

Its course is similar to the talocrural joint axis and its larger deviation from the transverse and sagittal planes. In the sagittal plane approximately 52° from the

transverse plane, and in the transverse plane 57° from the sagittal plans, allows significant movement especially in such a plane. As a result, dorsiflexion with simultaneous abduction or planter flexion with simultaneous adduction. The range and the course of such movements is always accompanied and influenced by co-movements in the talocrural joint (Kolář et al., 2013).

During the first 15% of the stance phase the lower limb internally rotates and this movement has the effect of pronating the foot, which allows the midfoot to become flexible and than the foot is able to adapt to uneven ground. As the body weight passes over the planted foot in late stance the heel inverts, supinating the forefoot and locking the Chopart's joint. This makes the midfoot more rigid and allows effective transmission the force from the forefoot to the ground (Ramachandran, 2011).

Tarsometatarsal joint (Lisfranc's joint)

The Lisfranc's joint contribute little to midfoot flexibility. There little movement results from a gliding motion in this Joint and the stability results from a high degree of congruency and also from strong ligaments support. Lisfranc's ligament runs from the medial cuneiform to the second metatarsal. Movement at the first and second tarsometatarsal joint is considerably less than that at the fourth and fifth tarsometatarsal joint. The motion at first tarsometatarsal joint is 3.5º flexion, extension and 1.5º pronation, supination. This compared to 9º flexion, extension and 9º pronation, supination at the fourth and fifth tarsometatarsal joint (Ouzounian & Shereff, 1989).

Movement in the lower ankle joint:

These movements are combined movements based on mutual relationship between individual joint components. The fact that the talus and calcaneus are articulating in two areas, posteriorly at subtalar joint and anteriorly at an almost spherical talocalcaneonavicular joint, allows for a single oblique axis for the combined movements of these two bones and consequently all the tarsals and entire foot.

The axis of these movements begins on the external part of the posterior border of the calcaneus obliquely, moves forward and medially into the neck of the talus and overhead the navicular. It is angled from the inferior-posterior to superior-anterior.

The movement of the tarsals occurs in harmony and includes the following:

- Foot inversion, which is accompanied by plantar flexion with adduction and supination.
- Foot eversion, which is accompanied by dorsal flexion with abduction and pronation.
- Small movement at the Chopart joint line, which are significant for flexibility of the foot as a whole (Kolář et al., 2013) (Figure 4).

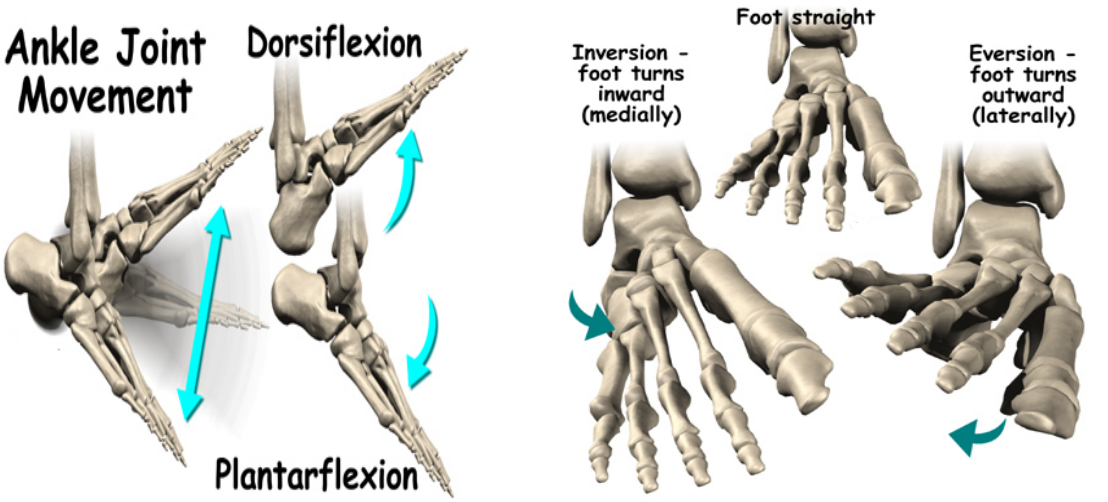


Fig. 4: The ankle joint movements (De Maeseneer et al., 2015).

Complex movements of the upper (Talocrural) and the lower ankle joint:

As a result of movement in the subtalar joint is primary rotation of the foot around the longitudinal axis in supination and pronation. Conversely, the talocrural joint permits for a full ROM in the sagittal plane (dorsal and planter flexion), which linked to abduction and adduction given the oblique course of the axis. In such manner, both joints complement one another in their functions, they from a complex posterior portion of the foot allowing movement in all three planes. Their function is closely connected to the Chopart's joint function.

The kinetic of common joints of the foot, which formed by the talocrural, subtalar and Chopart's joints. Movements in this joint occur along two parallel axises: the axis of the upper talocrural joint and the axis of lower subtalar joint (Figure 4).

With a ROM restriction in one joint, a compensatory increase in the ROM in the other joint occurs. With increased external rotation of the foot, during walking with the toes pointing outward, range of the motion in the subtalar joint is increased while

it becomes decreased in the talocrural joint. Walking with the toes pointed inward presents the opposite situation (Kolář et al., 2013).

1.7. Foot arches

The bones of the foot are arranged in two arches (Figure 5). The arches enable the foot to support the weight of the body, provide an ideal distribution of body weight over the soft and hard tissues of the foot, and provide leverage when walking. The arches are not rigid; they yield as weight is applied and spring back when the weight is lifted, thus helping to absorb shocks (Tortoraen, 2012).

The foot skeleton is arched longitudinally and transversely. The talus in the area of the navicular fibrocartilage is the highest point on the volar side of the foot skeleton. The architecture of the spongious bone reflects the course of girders in a dome and forms arches from the distal end of the tibia via the talus back into the calcaneus and forward all the way to the metatarsal heads.

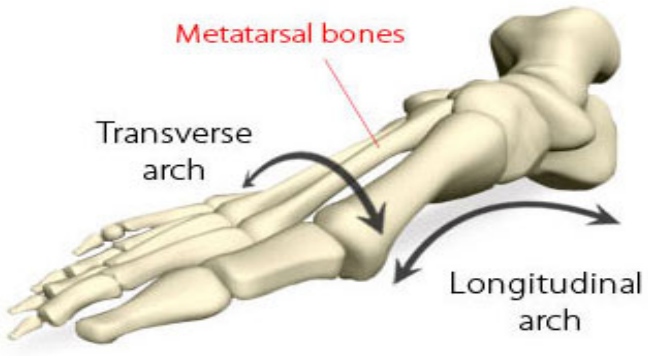


Fig. 5: Longitudinal arch and transverse arch (De Maeseneer et al., 2015).

The foot has been perceived as a tripod, with the heel and the heads of the 1st and 5th metatarsals as the points of support, connected by three arches: the medial longitudinal arch, the lateral longitudinal arch and the transverse arch of the forefoot. An arch shaped structure based on the anatomy of the bones in conjunction with intact ligaments and muscles has been perceived as crucial to the spring action and shock absorption by the forefoot and the longitudinal arches of the foot (Kanatli et al., 2003).

Longitudinal arch:

The foot has two longitudinal arches, medial and lateral longitudinal arches. The medial longitudinal arch is higher on the tibia side (Kolář et al., 2013) confirmed by the talus, calcaneus, navicular, medial, inter- mediate and lateral cuneiforms and

the first three metatarsals. The talus placed on the apex of the arch and confers stability by acting as a wedge between the calcaneus and navicular. There are a number of important static stabilizers of the arch. The primary and the most important stabilizer is the plantar fascia, followed by the short and long plantar ligaments and than spring ligaments (plantar calcaneonavicular ligament) (Kitaoka et al., 1997). The ligaments would not be able to support the foot arch without the other structures (Kolář et al., 2013), which called dynamic stabilization, releasing the dynamic structures without releasing any static stabilizers has only a modest effect on arch height (Kitaoka et al., 1997). The dynamic stabilization including the muscles that help support the foot longitudinal arch along the sole of the foot are (tibialis posterior, flexor digitorum longus, flexor hallucis longus and superficial short plantar muscles), superficial plantar aponeurosis and a tendinous band under the foot with the help of the tibialis anterior pulls up on the tibial side of the foot (Kolář et al., 2013), and releasing posterior tibial tension to be a reduction of 0.5 mm in arch height (Dawe & Davis, 2011).

There are two models for considering the stability of the medial longitudinal arch they are the beam model and the truss model.

In the beam model (Figure 5) is apply loading to the apex of the arch with generating compressive forces on the dorsal surface and tensile forces on the plantar surface. Stability in this model results from bony congruency and attachments of the ligaments (Figure 6).

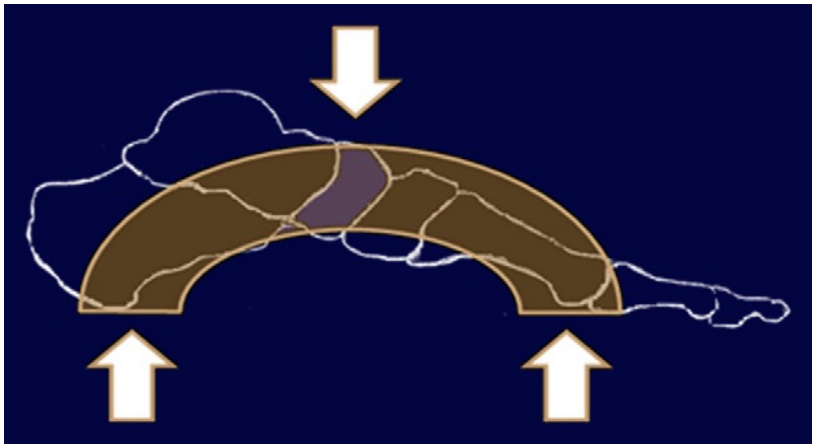


Fig. 6: The beam model for stability of the medial longitudinal arch. This model assumes arch stability from bony contact and ligamentous support (Dawe & Davis, 2011).

In the truss model (Figure 6) there is a triangular arrangement of foot structures. The bones of the arch are able to pivot about their apex whilst the tough

plantar fascia forms the third side. This is firmly attached to the medial and lateral calcaneal tuberosity proximally and its slips insert into the plantar plate and the fibrous flexor sheathes distally (Sarrafian, 1987) (Figure 7).

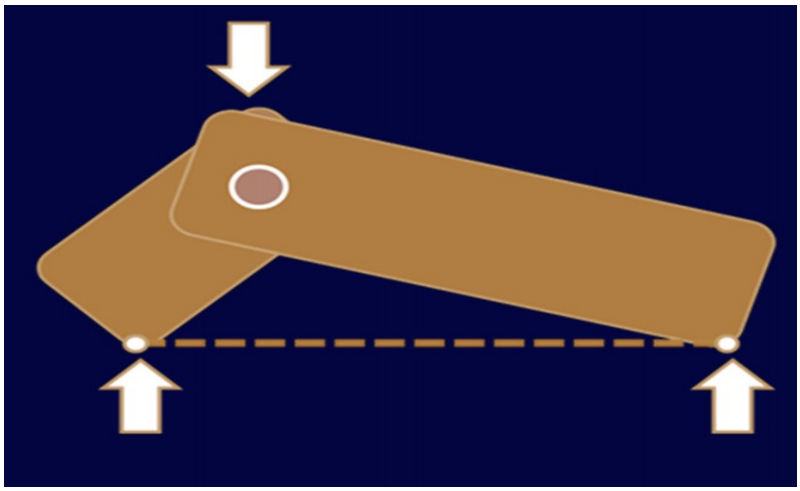


Fig. 7: The truss model for medial longitudinal arch support. Half the body-weight passes through the apex of the arch whilst standing. The ends of the ach are unable to move apart due to the tight plantar fascia, which connects them (dashed line). Hence arch height is maintained (Dawe & Davis, 2011).

The windlass effect of the medial longitudinal arch is raised on dorsiflexion of the first metatarsophalangeal joint. The plantar fascia spans this joint and has minimal elasticity. With considering the truss model of arch stability, dorsiflexion of the first metatarsophalangeal joint makes the plantar side of the triangle shorter, which has to result in drawing the calcaneus closer to the metatarsal head and lead to increase the arch height (Hicks, 1954).

Transverse Arch:

The transverse arch of the foot made up of series of arch between the posterior part of the metatarsus and the anterior part of the tarsus and have a shape of a half dome and is most prominent at the level of the cuneiforms and cuboid bones and The best advantage of the transverse arches can be seen in the region of the cuneiform bones which is strengthened by the interosseous, plantar and dorsal ligaments, intrinsic muscles that support the longitudinal arch also and the tendon of the peroneus longus and it adapts according to the position of the two main rays of the foot present in the tarsal segment at various heights from the floor (Kolář et al., 2013).

2. Special part

In this special part we will review the causes of adult acquired flatfoot deformity in different diagnostic methods perspective, based on the muscles and ligaments dysfunction, with the treatment and rehabilitation outcomes.

2. 1. Causes of acquired flatfeet in the adults and treatment procedures

Adult acquired flatfoot deformity (AAFD) is a degenerative disease characterized by chronic changes in the joints alignment of the foot leading to pain and dysfunction. While the etiology of the problem is unknown completely, the loss of posterior tibialis tendon strength and mechanical degradation of ligament support in the midfoot and hindfoot, namely the spring ligament, talocalcaneal interosseous ligaments, fibers of the anterior deltoid and the long and short plantar ligaments, they can be key factors of the acquired flatfoot (Blackman et al., 2009).

Flatfoot is a developmental or acquired deformity that is progressive and is characterized by plantar medial rotation of the talus, decrease in the medial arch height, supination and abduction of the forefoot. The Achilles tendon may shorten and exert a valgus moment on the calcaneus, producing heel valgus (Mann & Coughlin, 1993). The posterior tibialis tendon may weaken and tear the talonavicular capsule, the spring ligament, the tibionavicular ligament, the long and short plantar ligaments and the plantar aponeurosis may become stretched (Arangio et al., 1998; Kitaoka et al., 1998; Thordarson et al., 1998).

The muscles imbalance has ability to generate force can influence on the medial longitudinal arch (gastrocnemius, soleus, tibialis posterior, tibialis anterior, flexor hallucis longus, abductor halluces, flexor digitorum longus, short plantar muscles, peroneus longus and brevis) (Hunt & Smith, 2004; Kolář et al., 2013; Mann, 1992).

We can divide the causes of acquired flatfoot into, which influence on the medial longitudinal arch structural: PTTD, ligament insufficiency, (spring ligament, deltoid ligament and plantar fascia), intrinsic muscles dysfunction, Achilles tendon tightness, tibialis anterior injury, foot muscle muscles imbalance.

2.1. 1. Posterior tibialis muscle dysfunction

The posterior tibialis muscle originates from the posterior interosseous membrane and the proximal 2/3 of the adjacent posterior tibia and fibula, forming the deep posterior compartment with popliteus, flexor hallucis longus and digitorum longus, the muscle tendon structure starts at the distal third of the calf before passing behind the medial malleolus and runs along a groove deep to the deltoid ligament, changing direction in its sheath towards the navicular tuberosity. It then inserts into the navicular tuberosity and the plantar surface of the medial cuneiform by an anterior slip, the posterior slip inserts into the plantar surfaces of the cuneiforms and the base of the 2nd to 4th metatarsals (Figure 8) and its innervated by the tibial nerve (Frey et al., 1990).

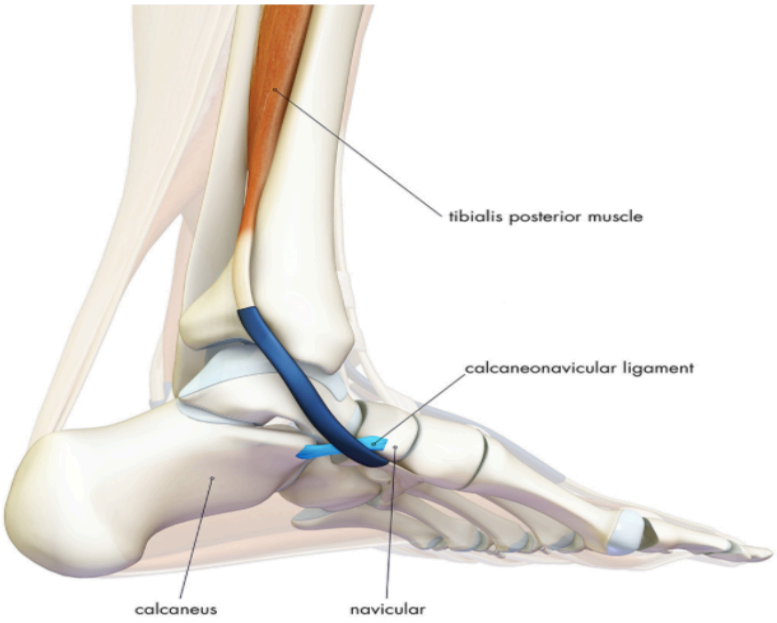


Fig. 8: Tibialis posterior muscle, medial view of left foot (Gilroy et al., 2008).

Tibialis posterior is the primary dynamic stabilizer of the medial longitudinal arch. It elevates the medial longitudinal arch by inverting and plantar flexing the foot, locking the Chopart's joint, inverting the subtalar joint and stabilizing the hindfoot and the midfoot and they are rigid enables a more effective action of gastrocnemius and soleus muscles during mid stance and propulsion in the gait cycle (Alvarez et al., 2006).

The tibialis posterior muscle acts during the stance phase of gait, particularly at heel-off, to invert the hindfoot, lock the calcaneo-cuboid, talo-navicular and transverse tarsal joints, thus creating a rigid lever to propel the foot over the metatarsal heads (Baumhauer, 1997).

The tibialis posterior tendon has a relatively large cross sectional area of the foot, twice that of peroneus brevis, which acts like primary agonist (Trnka, 2004). As it has only a short excursion, it will fail in its principle function as the primary stabilizer of the medial longitudinal arch. As a consequence, the midfoot does not lock during propulsion and the contraction of the gastrocnemius and soleus muscles causes excessive forces at the Chopart's joints leading to collapse of the medial longitudinal arch and eversion of the subtalar joint. To keep the foot abduction takes place at the talonavicular joint, giving rise to the radiographic appearance of an uncovered navicular (Ness, 2008).

Clinicians and biomechanists believe that the main function of the posterior tibialis tendon is to facilitate the smooth transition of the center of pressure from the heel to the forefoot through the stance phase of gait (Baumhauer, 1997). When the posterior tibialis tendon is damaged, it is believed that its ability to do so is compromised. Therefore, when evaluating PTTD it is critical to monitor the changes that occur in the center of pressure and the plantar pressure distribution (Hansen et al., 2001; Kitaoka et al., 1997; Niki et al., 2001).

Etiology of posterior tibialis tendon dysfunction

Posterior tibialis dysfunction is associated with adult acquired flatfoot deformity in both a chronic and a traumatic fashion or acute condition (Vaudreuil et al., 2014) although the exact role of the failure of the posterior tibialis tendon is not completely known (Myerson, 1996).

Women in middle age are commonly affected by PTTD and the incidence increases with age. Other risk factors include hypertension, obesity, diabetes mellitus, and seronegative arthropathies (Holmes & Mann, 1992). There are a number of reports in the literature associating both oral corticosteroid intake and local injection of steroids with rupture of the tendon (Holmes & Mann, 1992; Myerson, 1996). The presence of an accessory navicular may interfere with the function of tibialis posterior and be a risk factor (Myerson, 1996).

The pathological process in PTTD may be degenerative, inflammatory or repeated micro-traumatic in nature. Histological findings include mucinous degeneration, vascular hyperplasia and tendon sheath hyperplasia (Arangio et al., 2007), or it can be direct injury, pathologic rupture, idiopathic rupture and functional rupture may be the most appropriate proposal, other causes of PTTD, such as postoperative tibialis posterior tendon failure after tendon transfer, tibialis posterior tendon dislocation and others, may be added (Hintermann, 1995).

The stages of the PTTD according to Myerson are including four stages

- First stage: the tibialis posterior tendon is inflamed but the arch is maintained and this stages the patient will often describe medial ankle discomfort along the course of the tendon.
- Second stage: the muscle tendon is not longer functional and there is acquired flatfoot.
- Third stage: the flatfoot is appeared and the subtalar joint valgus cannot passively reduced.
- Fourth stage: there are arthritic changes with ankle joint (Myerson, 1996; Edwards et al., 2008).

If there is an element of tenosynovitis (tendon sheath inflammation) of tibialis posterior, there may be swelling around the medial malleolus and tenderness along the course of the tendon behind the medial malleolus. As the condition progresses, tenderness initially present along the tendon may disappear, and patients may complain of fatigue and aching in the leg and a reduction in walking distance. With further progression they may notice a change in the shape of the foot with a loss of the medial arch and the heel drifting into valgus (Niki et al., 2001).

Commonly patients complain of loss of function rather than a change in the shape of their foot. They may also have symptoms of instability, a limp and inability to walk on uneven surfaces (Alvarez et al., 2006). In the later stages the patient may complain of lateral ankle pain as the fibula impinges on the calcaneus (Mosier et al., 1999). Patients find standing on tiptoe difficult and notice asymmetrical wear along the medial sole of their shoes. One must always consider PTTD in middle aged or elderly patients with lateral hindfoot pain and not forget that other foot pathologies may initiate medical attendance and incorrect initial diagnosis of hallux valgus, hallux

rigidus and metatarsalgia may be the initial diagnosis in primary care (Pearlman, 1996).

Study of (Imhauser et al., 2004) with purpose to develop and validate the foot and ankle at the heel-off instant of gait by matching the location of the center of pressure on the plantar surface of the foot. The experiments were performed on five amputated lower limbs through the proximal third of the tibia. Each subject was verifying that no pre-existing foot pathologies were present.

Results that the posterior tibialis tendon plays a fundamental role in shifting the center of pressure anteriorly at heel-off during the gait and unloading the posterior tibialis tendon caused significant posterior movement in the center of pressure for the intact and flatfoot conditions. This also resulted in a medial shift in the force acting on the forefoot (see Figure 9).

The forefoot loads shifted medially after a flatfoot was created even when the posterior tibialis tendon remained loaded. The spatial relationships of the bones of the arch and the bones of the hindfoot also changed significantly for the intact specimen, and to a lesser extent after a flatfoot.

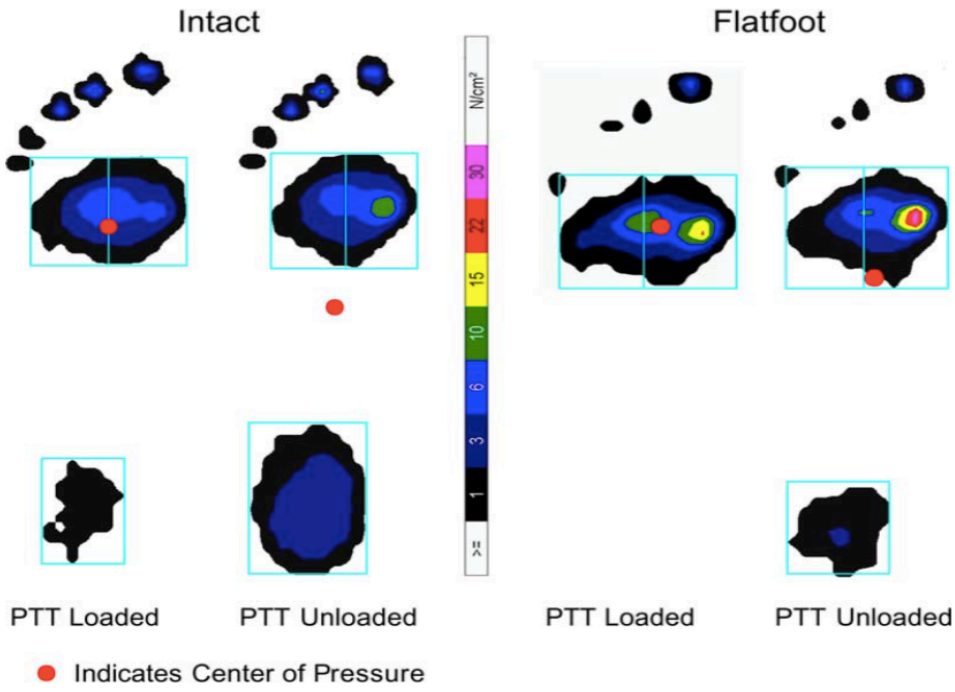


Fig. 9: The changes in the plantar pressure distribution and location of center of pressure are shown for one specimen in all of the experimental conditions (Imhauser, 2004).

The effect of PTTD on the plantar pressure characteristics (center of pressure location, medial to lateral forefoot force ratio and hindfoot to forefoot force ratio), and on the spatial relationship between the bones comprising the hindfoot and the arch (medial longitudinal arch and transverse arch) both in the intact foot and in the foot with a flatfoot deformity was determined.

The results of this study indicate two functions of the posterior tibialis muscle during the heel-off stage of gait: the first is shifting the center of pressure anteriorly and second is preventing the forces acting on the foot from shifting medially as illustrated by the sample pressure, this study indicate also that the posterior tibialis tendon cannot lock the hindfoot and the arch in the intact, stable configuration at the heel-off instant in the presence of a flatfoot deformity (Imhauser et al., 2004).

These results supported the conclusion of (Niki et al., 2001) who stated that reconstructing only the posterior tibialis tendon when a flatfoot deformity is present does not restore normal arch and hindfoot kinematics, he observed that the tibialis posterior muscle has the greatest influence hindfoot kinematics during the heel-rise phase of gait, which is documented the activity of tibialis posterior by using EMG and this study investigated the effects of tibialis posterior rupture on the kinematics of the hindfoot complex (talus, navicular, cuboid and calcaneus) and the effectiveness of surgical reconstruction of the tibialis posterior tendon in the presence of a flexible flatfoot deformity. They concluded that an acquired flatfoot deformity resulting from a compromised tibialis posterior could not be corrected by surgically repairing this tendon alone.

Study (Kiter et al., 1999) by using MRI of 27 patients with an accessory navicular and tibialis posterior dysfunction found that the insertion of tibialis posterior was solely into the accessory navicular without slips to the plantar surfaces of the metatarsals in the majority. This may be the reason that PTTD leads to an acquired flatfoot deformity. Conversely, the flatfoot deformity may be a predisposing factor in the onset of PTTD. This tendon also acts to lock the bones of the arch and the hindfoot in a stable configuration at heel-off, but a flatfoot deformity compromises this ability.

There are various stages in the development of PTTD, ranging from an asymptomatic predisposition to its development, to a complete rupture of the tibialis posterior tendon. A classification into 3-5 stages has been proposed in number of studies (Deeymaeker & Wouters, 1994; Klaue, 1994; Mueller, 1991). From a practical point of view, the classification into three stages is recommended (Hintermann, 1995; Hintermann, 1997).

- *The first stage* (symptomatic stage) is characterized by the symptoms of tibialis posterior tendinitis, and progressive flatfoot.

- *The second stage* (functional rupture) is characterized by complete or functional rupture of the tibialis posterior tendon associated with increasing severity of symptoms, but the flatfoot deformity is still supple. This situation is very disabling, and most patients will have presented for treatment at this stage.

- *The third stage* in the development of tibialis posterior dysfunction is the end stage, characterized by rapid progression of flatfoot and fixation of the foot deformity

Clinical signs and symptoms tibialis posterior rupture

Patient with the tibialis posterior complaints depend on the stage at which he present. In acute stage patients describe medial ankle pain with or without swelling which is worse with weight bearing activities. The onset of the problem may be temporarily associated with some minor trauma or it may not be preceded any history of trauma.

Patients who present a few months after the onset of symptoms often have decreased their activity level because of pain. They may have noticed gradual arch loss and its subsequent further decrease. But often the forefoot abduction not appears initially and increases in heel valgus that begins to develop. As the forefoot abduction getting worse, patients may describe decreasing in strength in the affected foot with a lack of ability to push-off. Some patients begin to develop sinus tarsi and lateral hindfoot pain.

In the chronic stage, the medial pain of hindfoot may diminish to the point that the lateral hindfoot and sinus tarsal pain become the predominant complaint of the patient, along with difficulty in fitting shoes. As they experience more subluxation of the subtalar and talonavicular joints, it may starts to complain of early morning stiffness and pain on both medial and lateral aspects of the foot in the region of the

subtalar and talonavicular joints. This may indicates the onset of secondary arthritis (Hintermann, 1995).

The other study (Hintermann, 1997) by treated operatively 38 patients with I-II-III stages PTTD showed that the prognosis is better when the treatment starts early, and the treatment was depend on the stage of the disease and the severity of the symptoms. It includes other non-operative options, such as rest, immobilization, anti-inflammatory medication and operative options, as tendon transfer, astenosynovectomy, calcaneal osteotomy and various arthrodesis techniques.

The prognosis becomes worse with increasing of the age of the patient. As the PTTD has passed the stage II, the prognosis seems to be better after bony correction (calcaneal osteotomy or arthrodesis) than after soft tissue procedures should be adapted in every single case to the special needs of the patient's foot.

The purpose of other study of (Hintermann, 1996) was to validate the clinical finding by surgical exploration and by compare the method (first metatarsal rise sign) sensitivity with other common clinical signs. While other clinical signs (too many toes, the double-heel rise and the single-heel rise) were noted to be negative in 20% to 35%.

This study included 21 consecutive feet with PTTD of 19 patients. The deformity was fixed in 9 feet and supple in 12 feet. They found the first metatarsal rise sign to be positive in all cases of PTTD. This simple clinical test enables us to recognize and treat a dysfunction of the tibialis posterior tendon at an early stage, when the foot is still supple. As the foot deformation progresses, early treatment may be the most effective measurement in preventing long-term functional impairment.

Patients tested while they were standing with fully weight bearing on both feet. When the leg of the affected foot is taken with one hand and externally rotated, or when the heel of the affected foot is taken with one hand and brought passively into a varus position, the head of the first metatarsal raised in the case of PTTD and remained on the ground in normal posterior tibialis function. They found in all cases of PTTD that first metatarsal rise sign is positive.

The abduction deformity of the forefoot and valgus deformity of the hindfoot that produced the typical flatfoot were described as the hallmark of this disorder.

More recently, the first metatarsal rise sign has been suggested to be the most reliable sign in detecting PTTD even in a very early stage (Hintermann, 1996).

In study of treatment effect for 64 patients with diagnosis tibialis posterior tendonitis, they treated with physical therapy modalities, medications, such as non-steroidal anti-inflammatory drugs, oral administration or local infiltration of corticosteroid; and orthotics or bracing such as a foot orthoses and arch and ankle brace, low-articulating ankle-foot orthosis, cast-boot or shoe modifications. The result of this study reported 87% success rate, which do not required further surgical treatment (Nielsen et al., 2011).

In other study involved 47 consecutive patients with I, II stage of PTTD (Alvarez et al., 2006). The patients treated non-operative management by foot orthosis, high repetition exercises, plantar flexion activities and high repetition home program exercise that included stretching exercises for gastrocnemius and soleus. The strengthening exercises for tibialis posterior, peroneus, tibialis anterior and gastrocsoleus muscles and included isokinetic exercises, exercise band, heel rises, toe walking (type balance exercise) and home exercises for each muscle group. The result showed success defined as 10% strength deficit (compared with the uninvolved side). With this treatment protocol 89% of patients were satisfied. The results reported by this study assessing non-operative management of PTTD with success rates ranging from 67-90%.

Dysfunction of the posterior tibialis tendon typically is unilateral condition caused by pathologic changes within the tendon. The arch deformity is usually progressive and results in a flexible to rigid flatfoot, depending on the stage of the condition (Lee et al., 2005) Initial treatment options for adult flexible flatfoot include the following: physical therapy, activity modifications, weight loss, anti-inflammatory medications, immobilization, footwear modifications and orthotic management encompasses a broad spectrum of devices that includes foot orthoses or ankle-foot orthoses, either prefabricated or custom molded (Figure 10. a, b).

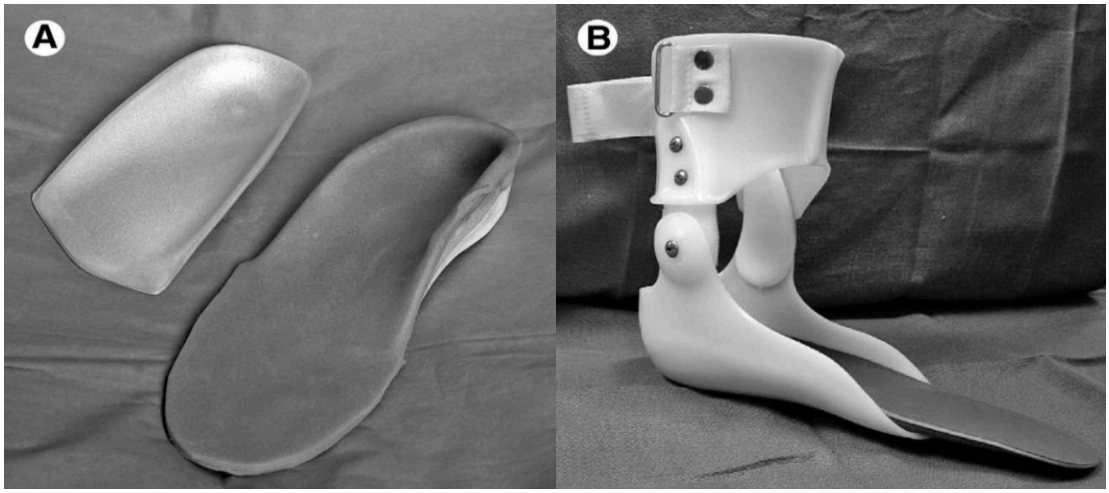


Fig. 10: Orthotic management of flatfoot is accomplished with foot orthoses (Lee et al., 2005)

Surgical treatment techniques of adult flexible flatfoot are including osteotomy, arthroereisis and arthrodesis. And the other soft tissue procedures are including tibialis posterior tendon advancement, flexor digitorum longus tendon transfer and reconstruction of the spring ligament.

The effect of the medial calcaneal osteotomy displacement and the medial plantar fasciotomy on arch configuration in a foot model showed that medial displacement calcaneal osteotomy corrected the foot deformity. In study (Arangio & Salathe, 2001) they compared between the reaction of a normal foot, a flatfoot and a flatfoot with a medial displacement calcaneal osteotomy by applied load of 683n are analyzed and the distribution of support among the heads of metatarsal and moment about various joints are computed.

They found from the result that the total force in the medial plantar fascia increased by 7% in the foot model relative to the normal foot and does not revert to normal after medial displacement calcaneal osteotomy, which can use it in a foot model to decrease the load on the medial arch.

Patients with tibialis posterior dysfunction have significantly reduced stride length, cadence and walking speed, which explains why patients may present with reduced function rather that altered foot shape as their primary complaint (Arangio & Salathe, 2001).

There are different types of the flexible acquired flatfoot deformity due to the multiple joints involved and according to (joint and plane) deformity that could guide optimal surgical correction. There is currently surgical treatment and the most effective for the flexible flatfoot deformity. The reconstructive procedures including a central calcaneal osteotomy, a medial column stabilization procedure, flexor digitorium transfer and a gastrocnemius muscle or Achilles tendon lengthening.

The method of study was 26 feet in 21 patients with age average of 35 years; Diagnoses included specific conditions such as posterior tibialis tendon pathology and accessory navicular bone. Degenerative joint pathologies and patients with a rigid deformity with pathological neuromuscular conditions were excluded.

The indication for surgery to restore normal foot alignment with good function from painful flexible flatfoot deformity than 1 year after initiation of conservative treatment like orthoses, insoles and physical therapy (Klaue et al., 2013).

Study of (Ettore, 2013) suggested conservative management recommended for all patients affected by any stage of PTTD should undergo non-surgical treatment before considering surgery. Physical therapy consisting of Achilles stretching, inversion and toe flexor strengthening along with proprioception exercises is recommended once the pain has subsided.

Surgery treatment is indicated in patients who didn't respond after 3 months or more of conservative management.

Stage I of PTTD procedures and medial calcaneal osteotomy require postoperative no weight bearing in splint for 2 weeks followed by a removable cast for at lest 4 weeks then progressed to full weight bearing after this period. ROM exercises can start at 2 weeks and strengthening exercises start at 12 weeks after surgery.

Stage II AAFD presents with a mild to moderate flexible deformity without abduction through the talonavicular joint. Different operative approaches are available to treat the deformity, including subtalar arthroereisis, medial calcaneal osteotomy and tendon transfers.

Stage II AAFD presents with abduction deformity through the talonavicular joint. The treatment for this stage is controversial in terms of adding lateral column lengthening to the media calcaneal osteotomy, flexor digitorum longus transfer and gastrocnemius recession. Spring ligament repair or reconstruction. In this stage of AAFD postoperative care consists of no weight bearing in first 2 weeks in splint, followed by a cast or removable boot for 6 weeks. ROM exercises can start at 2 weeks after surgery. And full weight bearing after 8 weeks depend on patient healing.

Stage III AAFD consists of fixed deformity involving the triple-joint complex (subtalar, calcaneocuboid and talonavicular joint). Surgical correction is achieved at talonavicular and subtalar joints, where the most deformity occurs. Triple arthrodesis results foot rigidity and difficulty on uneven ground, while increases the risk of develop ankle arthritis. Postoperative care in a cast for 10 to 12 weeks no weight bearing, followed by removable boot for 4 weeks with progressive weight bearing.

Stage IV deformity doesn't need to replace the ankle joint. And reconstruction can be performed for deltoid ligament with tendon transfers. They recommend that patient should be surgically treated before progression to stage III (Ettore, 2013).

2. 1. 2. Intrinsic foot muscles dysfunction

There are two groups of toe flexor muscles, namely extrinsic muscles and intrinsic muscles. The extrinsic muscles running across the ankle joint include the flexor digitorum longus and flexor hallucis longus. On the other hand, the intrinsic muscles include flexor digitorum brevis, abductor hallucis, flexor hallucis brevis, abductor digiti minimi, and dorsal interossei muscles, and they do not across the ankle joint and do not contribute to ankle joint movement. Both the intrinsic muscles and extrinsic muscles are stretched by dorsal flexion of the metatarsophalangeal (MTP) joints, and the extrinsic muscles are only relaxed by plantar flexion of the ankle joint (Refshauge et al., 1995).

At the neutral position of the MTP joints and ankle joint, both the intrinsic muscles and extrinsic muscles can produce similar force. However, the produced force ratio of the extrinsic muscles to the intrinsic muscles is relatively large at the neutral position of the ankle and MTP joints as compared to at 20° plantar flexion of the ankle joint with 45° dorsal flexion of the MTP joints, and this is likely the reason for the lack of a significant correlation between maximal voluntary contraction torque

and navicular drop in this condition. From these results suggest that the intrinsic foot muscles contribute greatly to the construction of the medial longitudinal arch and that navicular drop relates to maximal plantar flexion torque of MTP joints at 20° plantar flexion of the ankle joint with 45° dorsal flexion of the first MTP joint. These results support a previous finding that navicular drop is correlated with EMG activity of the abductor hallucis muscle (Nam et al., 2012).

The exhibited maximal torque of the MTP joints is affected by the ankle joint angle (Williams & Stutzman, 1959), and a relationship between joint angle and plantar flexion torque of the MTP joints has been reported previously (Goldmann & Brüggemann, 2012).

Plantar toe intrinsic muscles:

Plantar intrinsic foot muscles such as the flexor digitorum brevis, abductor hallucis, flexor hallucis brevis, abductor digiti minimi, and dorsal interossei muscles (Figure 11).

Dysfunction of any of these intrinsic muscles supporting the medial longitudinal arch may predispose individuals to hyper-pronation related overuse injuries (Headlee et al., 2008).

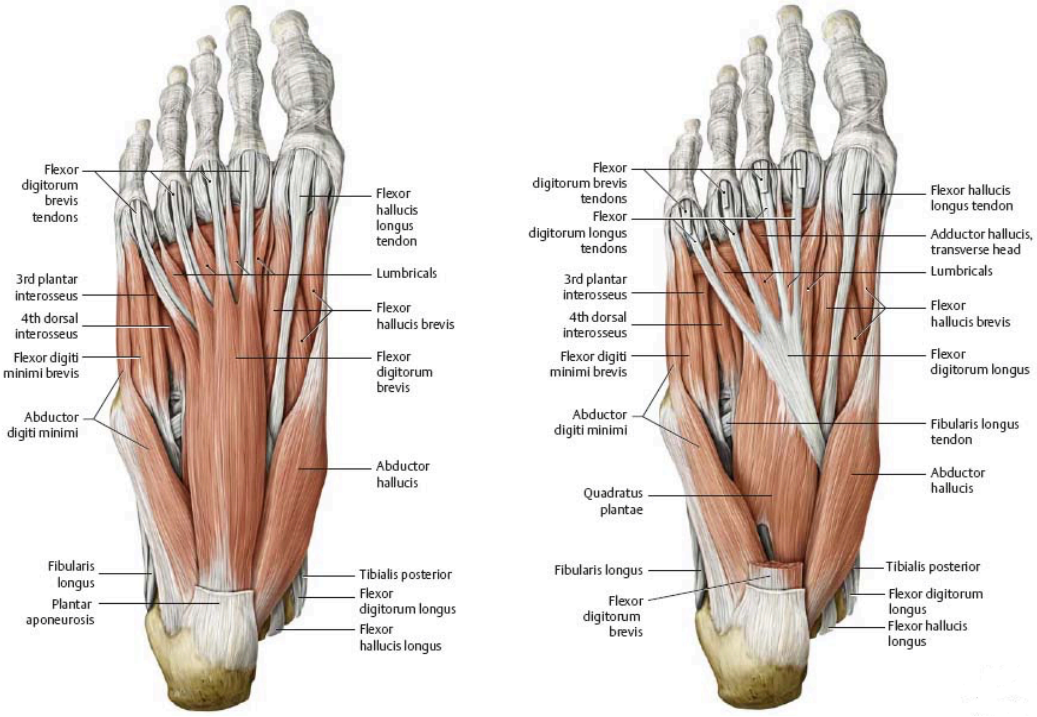


Fig. 11: Superficial and second layer of the intrinsic muscles, plantar view (Gilroy et al., 2008).

The abductor hallucis muscle is the most medial muscle in the first layer of the

intrinsic muscles of the sole of the foot innervated by posterior tibial nerve. The origin of the muscle is the posterior medial calcaneus tuberosity on the plantar medial aspect from deep fascia and an inserted distally into the tibial sesamoid and that lifting the muscle to give it an inverted 'V' shaped configuration (Kura et al., 1997). Abductor hallucis muscle has eight specimens, strong and thickened fibers of the deep fascia arising from the medial malleolus and deltoid ligament were pass over the abductor hallucis muscle, then turn back underneath to insert into the deep fascia overlying the flexor digitorum brevis muscle, holding up the abductor hallucis like a sling, other fibers continued to the medial tuberosity of the calcaneus, blending with the fibers of the abductor hallucis muscle arising from the calcaneus. The abductor hallucis itself had a 'V' shaped configuration, with one limb running from the sling plantarwards and towards the calcaneal tuberosity and a second limb running distally towards the hallux. It's working as an abductor and flexor of the first MTP joint of the hallux (Wong, 2007).

The abductor hallucis plays a significant role in gait; it has two kinematic activity: the first during the late stance and toe-off phases of gait, and the second moving the center of pressure forward and medially at toe-off (Whittle, 2002), on other hand based of the subtalar axis the abductor hallucis muscle acting between the medial malleolus and the calcaneal insertion of the muscle be well positioned to act as an inverter of the subtalar joint (Van den Bogert et al., 1994).

All specimens also demonstrated a fascial sling in the hindfoot, lifting the abductor hallucis muscle to give it an inverted 'V' shaped configuration. Simulated contraction of the abductor hallucis muscle caused flexion and supination of the first metatarsal, inversion of the calcaneus, and external rotation of the tibia, consistent with elevation of the arch. The abductor hallucis muscle acts as a dynamic elevator of the arch (Wong, 2007).

Navicular drop provides a composite measure of pronation with greater inferior displacement of the navicular during weight bearing representing more pronation (Menz, 1998; Picciano et al., 1993), there some studies used same method.

The aim of (Saeki et al., 2015) study was to determine the relationship between navicular drop and plantar flexion torque of the first and second to fifth MTP joints. The method of this study was ten healthy young men subjects participated.

The procedure by performed maximal voluntary isometric contraction of plantar flexion of the first MTP joint.

They found there through this study relation between the navicular drop and the strength of the intrinsic toe plantar flexion muscles, they suggested that exercises targeting the intrinsic muscles to improve excessive navicular drop and also can help prevent sport related disorders which caused by excessive navicular bone drop, on the other hand navicular drop relates to MTP joint muscles strength in plantar flexion where the intrinsic toe plantar flexion muscles are capable of exerting force (figure 12).

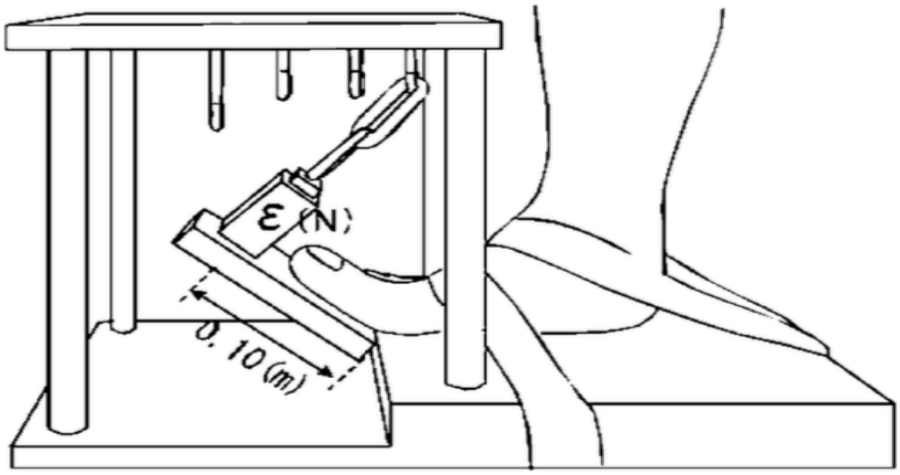


Fig. 12: Structure of the torque meter used to measure isometric plantar flexion torque at the MTP joints. MTP joint plantar flexion torque was calculated using the following formula: plantar flexion torque (Saeki et al., 2015).

The medial longitudinal arch is affected not only by the toe flexor muscles but also by the muscle that did not contribute to MTP joint plantar flexion (e.g., the peroneus longus and tibialis posterior muscles). This result suggests that exercises targeting the intrinsic muscles may help improve excessive navicular bone drop and improve the height of longitudinal arch (Saeki et al., 2015).

The functional way to assess the contribution of the intrinsic foot muscles in maintaining the medial longitudinal arch may be through fatiguing exercise of these muscles. The plantar intrinsic muscle fatigue causes a decrease in power output resulting in a reduced work capacity (Fitts, 1996).

In study to assess the effect of foot intrinsic muscle fatigue on pronation for (Headlee et al., 2008) was assessed with navicular drop, during static stance. For twenty-one healthy young adults, by measuring navicular drop before and after fatiguing exercise of the plantar foot intrinsic muscles (Figure 13). Muscle activity was measured with surface EMG. The EMG of the abductor hallucis muscle was recorded during maximum voluntary isometric contractions, the results of the study showed that intrinsic foot muscles play a role in support of the medial longitudinal arch.

The intrinsic muscle function in maintaining the integrity of the medial longitudinal arch and these muscles can be effectively treat injuries that are linked to hyper-pronation of foot (flatfoot). Disrupting the function of the intrinsic muscles lead to increase in pronation as assessed by navicular drop.

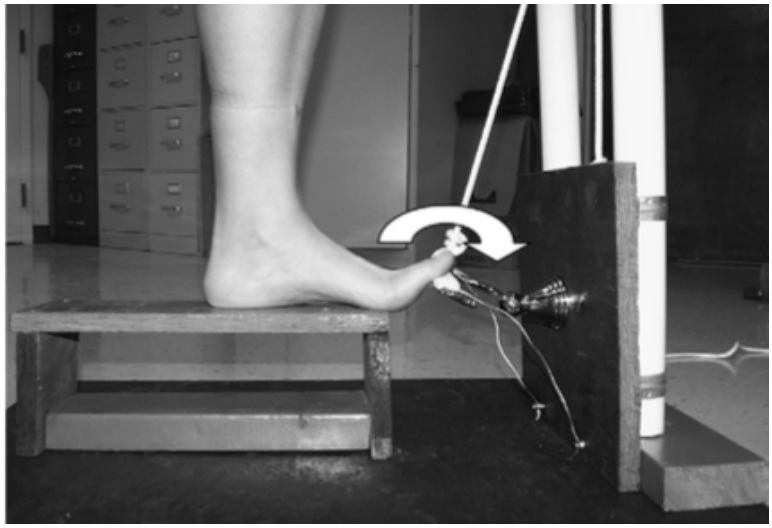


Fig. 13: Custom-fabricated pulley system positioned for fatiguing exercises (Headlee et al., 2008).

Increased pronation was seen in subjects after fatigue of the plantar intrinsic muscles of the foot regardless of baseline longitudinal arch height, as assessed by navicular drop, in static stance, which mean the function of the intrinsic foot muscles adversely affected the structural integrity of the medial longitudinal arch in static stance regardless of subjects base on the navicular drop measurement.

Treatment of flatfoot, which related injuries, has generally focused on limiting pronation through use of taping techniques and orthotic devices. The treatment procedure should also including the dynamic control of pronation during gait through neuromuscular exercises purely rather than the mechanical, which means of limiting pronation with orthotics to control the foot pronation (Headlee et al., 2008).

Study to compare the abductor hallucis muscle activity and the medial longitudinal arch angle during short foot exercise (Figure B14) and toe curl exercise (Figure C14) while sitting or in one leg standing position, for twenty subjects with neutral foot alignment participated in this study. The activity of the abductor hallucis measured by EMG.

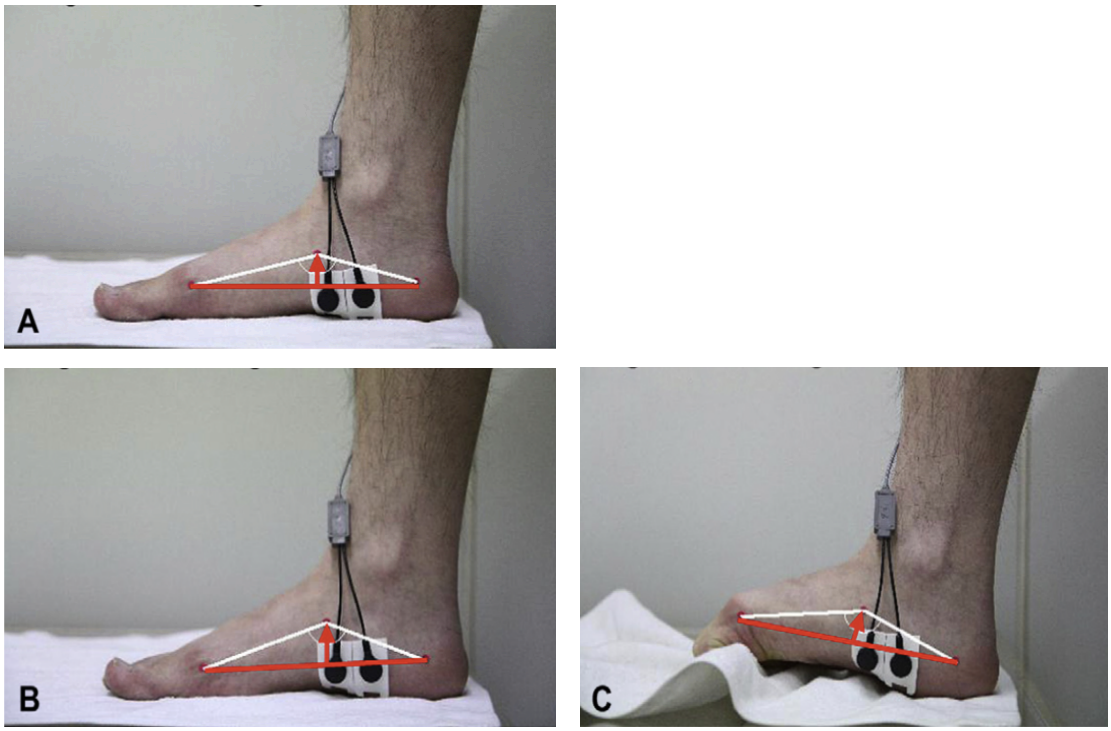


Fig. 14: Types of exercises (A: resting, B: short foot exercise, C: toe curl exercise) (Gazdag & Cracchiolo, 1997).

They found that the abductor hallucis muscle was significantly more activated in a short foot, without the interphalangeal joints flexed, than in toe curl exercise, with interphalangeal joints in flexion. The medial longitudinal arch angle in short foot exercise was significantly smaller angle than in toe curl exercise in both the sitting position (Figure 15). On other hand significant increaseing in the abductor hallucis activity from sitting to one leg standing position, there is no significant difference in medial longitudinal arch angle between the sitting and one leg standing positions.

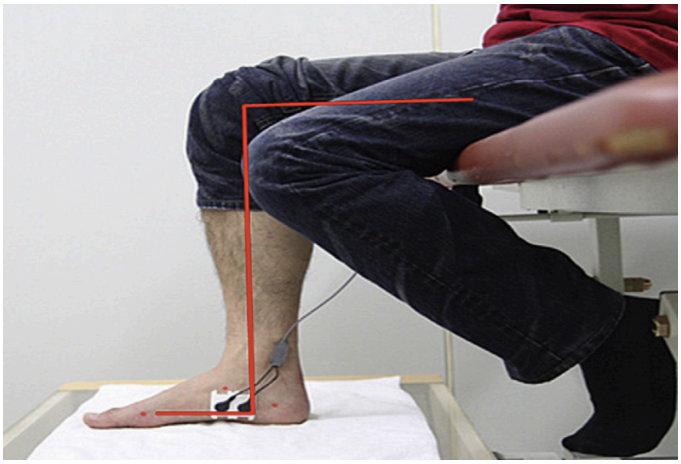


Fig. 15: Siting position for exercises (Gazdag & Cracchiolo, 1997).

The conclusion of the study, that short foot exercise (one of sensor motor stimulation exercises) is more beneficial than the toe curl exercise in comparing the activity of the abductor hallucis muscle and in preventing lowering of the medial longitudinal arch, both of them considered as strengthening exercise. In addition, based on the level of the foot intrinsic muscle performance, exercise position should be considered when performing "short foot exercise" (Gazdag & Cracchiolo, 1997).

The therapeutic approach of sensor motor stimulation basis on that movement cannot be accomplished without coordination of the afferent pathways and the efferent pathway. The therapy concept is basis of the proprioceptive Neuromuscular Facilitation (PNF). The sole receptors can be facilitated in different ways: by stimulation of skin receptors or more effectively, by active contraction of intrinsic muscles of the foot by forming "short foot exercise" without activation of the long toe flexors is more effective. And other various balance exercises by using wobble and rocker boards, rolls from plastic material, balance shoes help to configure the short foot.

Sensor motor stimulation uses for motor re-education to achieve a quality of movement patterns that is as close to normal as possible, helps to improve muscle coordination and motor regulation and increase the speed of the muscle contraction and improve muscle imbalance, which needed to protect the joint and to prevent injury and micro-injury in particular. Used originally to improve the unstable ankle after injury (Liebenson, 2007).

While the important contribution of the intrinsic foot muscles to controlling pronation during static stance, had a functional role in maintaining the medial longitudinal arch. The activity of foot intrinsic muscles showed influents on height of the foot arch, these muscles are important to proper foot function (Fiolkowski et al., 2003).

The studies (Fiolkowski et al., 2003; Gaillet et al., 2004) and other studies have suggested that the intrinsic muscles have an important role in foot function, measured the longitudinal arch height before and after Lidocaine injection into the posterior tibial nerve at the ankle joint, and they noted a drop in navicular height of 3.8mm performed transcutaneous electrical nerve stimulation (TENS) to the abductor halluces muscle. Pedobarographic data demonstrated increased plantar pressure on the anterior medial part of the sole, lateral displacements of the anterior maximal pressure point and the foot thrust center, and external rotation of the leg (Wong, 2007).

The dysfunction of the plantar intrinsic foot muscles can be by blocking tibial nerve transmission to the intrinsic foot muscles produces a significant increase in pronation in static stance as assessed by navicular drop in studies of (Fiolkowski et al., 2003) for 10 subjects (9 males and 1 females, the ages between 21 and 35 years) were tested bilaterally for myoelectric activity and navicular drop before and after the anesthetic injection. The electrodes of EMG ware located by palpation for the abductor hallucis.

Resulted that navicular drop measurement with pronation in static stance, increased significantly after fatiguing contractions of the plantar intrinsic foot muscles. These results reinforce recent findings that the intrinsic foot muscles provide substantial support to the medial longitudinal arch in static stance, the intrinsic foot muscles play an active role in dynamic longitudinal arch support, meaning they function during gait but not in static stance. And when there is complete loss of function of the plantar intrinsic musculature as seen when blockage of a tibial nerve, navicular drop significantly increases indicating a loss of the integrity of the medial longitudinal arch.

In the course of rehabilitation from foot injury, or as a procedure for preventing injuries, strengthening of the intrinsic muscles of the foot may be a factor to consider in people who present with problems stemming from excessive pronation. These finding have an implication for clinicians who are treating individuals whose

injuries directly involve the medial longitudinal arch or those injuries emanating from excessive pronation. Strengthening of intrinsic and extrinsic muscles may help to increase muscular support of the arch, and thus increase control of pronation.

Study to evaluate the effects of transcutaneous electrical stimulation of the abductor hallucis muscle in the loaded foot on the change of plantar pressures, as measured by digital baropodograms. After physical examination for 30 subjects (19 males, 11 females) they considered to have flatfoot on one side.

Post contraction by electric, detected on baropodograms increased plantar pressure on the anterior-medial part of the sole and displacements of the lateral part, which consistent with foot inversion, induce external rotation of the tibia on the stimulated side, with change of plantar pressure and thrust the pressure in the posterior part of the footprint. Conclusions of the abductor hallucis electrical stimulation in the loaded foot induces immediate specific changes in baropodogram indices, some of changes persist 2 months later (Gaillet et al., 2004).

Clinical research for (Jam, 2004) exists on the contribution, evaluation and retraining of the intrinsic foot muscles for pain syndromes related to abnormal control of pronation, the purpose of this clinical paper was to review the potential functions of the intrinsic foot muscles and their role in maintaining and dynamic controlling the medial longitudinal arch.

According to this research the evaluation of the plantar intrinsic foot muscles are following these steps:

- The examiner is standing front a wall with the feet apart and knees slightly flexed. The hands fingertips lightly placed on the wall.
- In order to achieve subtalar joint neutral, gently supinate the feet by lifting the toes off from the floor (Figure 16A), then slowly drop the toes down but maintain the arch, the rise is result of windlass effect via plantar fascia.

  If this procedure difficult, the other way by achieve approximation the head of first metatarsal actively toward the heel (short foot), without flexing the other toes (Figure 16B).
- With keeping the arch rise, stand on one leg for 30 seconds and observe the navicular height (Figure 16C), repeat the process on the other feet.

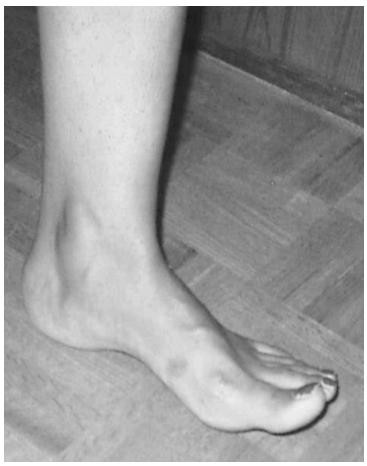


Fig. 16A: Active extension of the toes

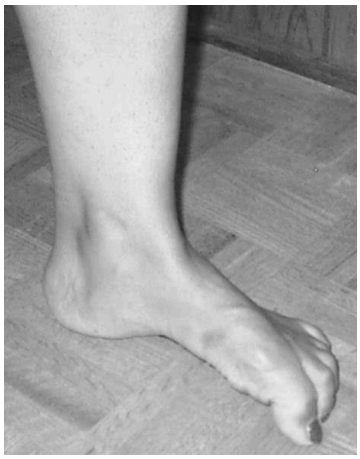


Fig. 16B: Intrinsic muscles activation with incorrect pattern

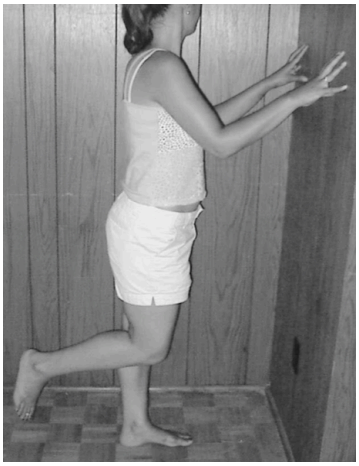


Fig. 16C: Evaluation position (Jam, 2004)

The therapist can evaluate over activity of the extrinsic foot muscles by occasionally tries to lift the toes passively from the floor, if observed during the test. The muscle control can classified as fair or poor. Evaluated as fair if unsteadiness of the neutral navicular height and/or over activity of the muscles, are evaluated as poor, if unable to maintain the neutral navicular height at all and/or over activity during most of the 30-second test.

A common exercise with compensatory patterns used by physiotherapists for retraining the plantar intrinsic muscles is the towel toe curls (Figure 17) this exercises primarily require the use of the flexor digitorum longus, flexor hallucis longus and tibialis anterior muscle for strengthen these extrinsic muscles may be benefit for patient with general foot and ankle weakness (Tab. 1).

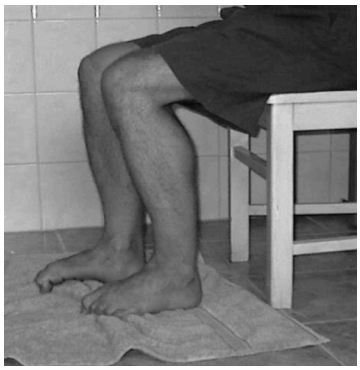


Fig. 17: Towel toe curl exercise (Jam, 2004)

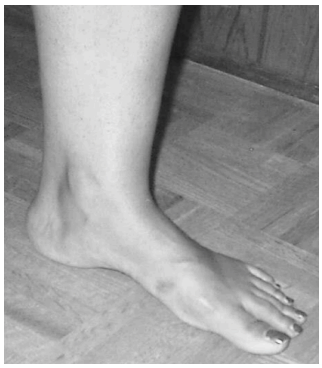


Fig. 18: Intrinsic foot muscle activation with correct patterning (Jam, 2004)

Therapeutic exercise program should be easy to understand and perform in most

setting. The following suggested exercise fits these criteria.

- Standing front of a wall with the fingertips may be lightly placed on the wall and the feet shoulder width apart and knees slightly flexed.
- In order to increase the longitudinal arch height, gently supinate and actively try to make short foot, without flexing the toes (Figure 18). Often the gluteal muscles need to be activated to facilitate femoral and tibial lateral rotation, which assist the supination.
- While actively maintaining the arch, stand on one leg if it's not difficult. The knee on the weight bearing side should be flexed 10-20 to activate the quadriceps muscle and the potential facilitation of the intrinsic muscles.
- Hold the position for 10 seconds with maintaining the arch height without any compensatory extrinsic muscles activity. Following slowly eccentric control the foot to pronate and to a relaxed state for 1-2 seconds, actively re-supinate and repeat steps 3-4.
- Perform up to 5 repetitions of this concentric, isometric and slow eccentric retraining exercise, several times per day.

The retraining exercise can progressed by gradually lifting the fingers off the wall and performing them in unsupported one leg standing with open eyes and eventually with eyes shut. Instructed to perform active supination during daily activity to restore motor control. Losing of foot intrinsic muscles function due to fatigue could result in a loss of structural support and motion control for medial longitudinal arch (Jam, 2004).

Traditional concept	**New proposed concept**
Excessive pronation is the primary biomechanical cause of many foot and ankle disorders	Lake of control into pronation is the primary biomechanical cause of many foot and ankle disorders
Rigid foot orthotics are clinically essential and should be the primary management option for many foot and ankle pain syndromes related to abnormal pronation	Prior to prescribing orthotics retraining of the intrinsic foot muscles may also be clinically valuable for the management for some foot and ankle pain syndromes related to abnormal pronation

Rigid orthotics are required to maintain the subtalar neutral position and to stop or limit pronation in many foot and ankle syndromes related to abnormal pronation	Semi-rigid orthotics may also be effective for maintaining the subtalar neutral position and controlling pronation in some foot and ankle pain syndromes related to abnormal pronation
The purpose of retraining the intrinsic foot muscles is to change the static foot posture and the height of medial longitudinal arch	The purpose of retraining the intrinsic foot muscles is neither to change the static foot posture and the height of medial longitudinal arch, but to help dynamically control the arch following the heel strike
Intrinsic foot muscles can be retrained using phasic towel toe curl exercises	Intrinsic foot muscles may be effectively retrained using specific tonic exercises in isolation of the superficial muscles

Table 1: Summary of the traditional versus a new proposed concept regarding the intrinsic foot muscles according to of this study (Jam, 2004)

2. 1. 3. Achilles tendon tightness

Triceps surae muscle (gastrocnemius and soleus) tightness can lead to laxity in the foot arch supporting structures of the midfoot, also that midfoot laxity can predispose patients to triceps surae muscle tightness (Van Boerum & Sangeorzan, 2003) and because the Achilles tendon insertion lies laterally to the axis of the subtalar joint the triceps surae muscle tightness can increases the amount of hindfoot eversion in flatfeet and initial soft tissue laxity has led to some degree of calcaneal eversion (Deland et al., 2005). The triceps surae muscle has the most significant forefoot abducting and arch flattening effect of the extrinsic muscles of the foot.

The overloading of the Achilles tendon in normal foot and in flatfoot resulted primarily changes in the sagittal plane. The most significant changes with Achilles over pull included talar plantar flexion, the navicular-to-talus dorsiflexion and 1st metatarsal-to-talus dorsiflexion, all consistent with flattening of the medial longitudinal arch. Forefoot abduction, another characteristic of flatfeet, was also increased. The 1st metatarsal abducted relative to the calcaneus, and the navicular

abducted relative to the talus, consistent with an increase in the talonavicular coverage angle (Thordarson et al., 1995).

In study of the ligament attenuation and Achilles tendon over-pull, it was including 10 fresh-frozen human cadaver feet (range of age 56 to 88 years, 8 females, 2 males). Due to large forces applied to the Achilles tendon, which simulate the mid-stance phase of gait (30% gait cycle).

The Achilles tendon over-pull resulted significant differences in foot bone positions primarily in the sagittal plane and analysis showed decrease of the plantar pressures force under the hindfoot and increase force under the 1st metatarsal head and generates significant calcaneal eversion, which prove that the Achilles tendon contribute in many flatfeet deformity, particularly in longitudinal arch depression, forefoot abduction and increased medial forefoot pressure. No significant force changes were seen under the medial or lateral midfoot, hallux or toes (Blackman et al., 2009).

The flatfoot due to the injury and surgery treatment may be caused by anatomical defects, such as varus of the forefoot, ligaments laxity, weakness or contracture of the gastrocnemius and soleus muscles. Hyperpronation of the foot can cause secondary effects in the lower extremity such as tibialis posterior tendinitis.

In one study involving 101 patients who were operated to treat ruptures of the Achilles tendon (86 males, 15 females. range of age 19 to 71years), the longitudinal arch height of the foot was measured clinically by footprint. Feet were classified using a three points scale: (a) high arch, (b) normal arch, and (c) flatfoot.

Combination height of longitudinal arch and under pronating alignment of the ankle was classified; 19 of patients have high arch, 67 normal arch and 3 flatfeet. Hyperpronation of the ankle not predispose factors for the Achilles tendon rupture and the high longitudinal arches were over represented, being associated with less pronation of the ankle and less varus in the forefoot and for flatfoot as well (Leppilahti et al., 1998).

The Triceps surae contractures can be associated with foot and ankle pathological and Achilles tendon contractures can shift plantar foot pressure from the heel to the forefoot.

In study to assess the effects of gastrocnemius and soleus contracture on midfoot plantar pressure by determine whether isolated gastrocnemius contractures and the method was using 10 fresh frozen cadavers.

In a static model (Figure 19), by increase the force through triceps surae the weight bearing shifted from plantar pressure from the hindfoot to the midfoot and forefoot.

The results of this study associated with clinical contracture with foot and ankle disorders including diabetic foot ulcers and Morton's metatarsalgia, which have similar effects with triceps surae. And they found a statistically significant increase in percent force at midfoot with increasing triceps surae force.

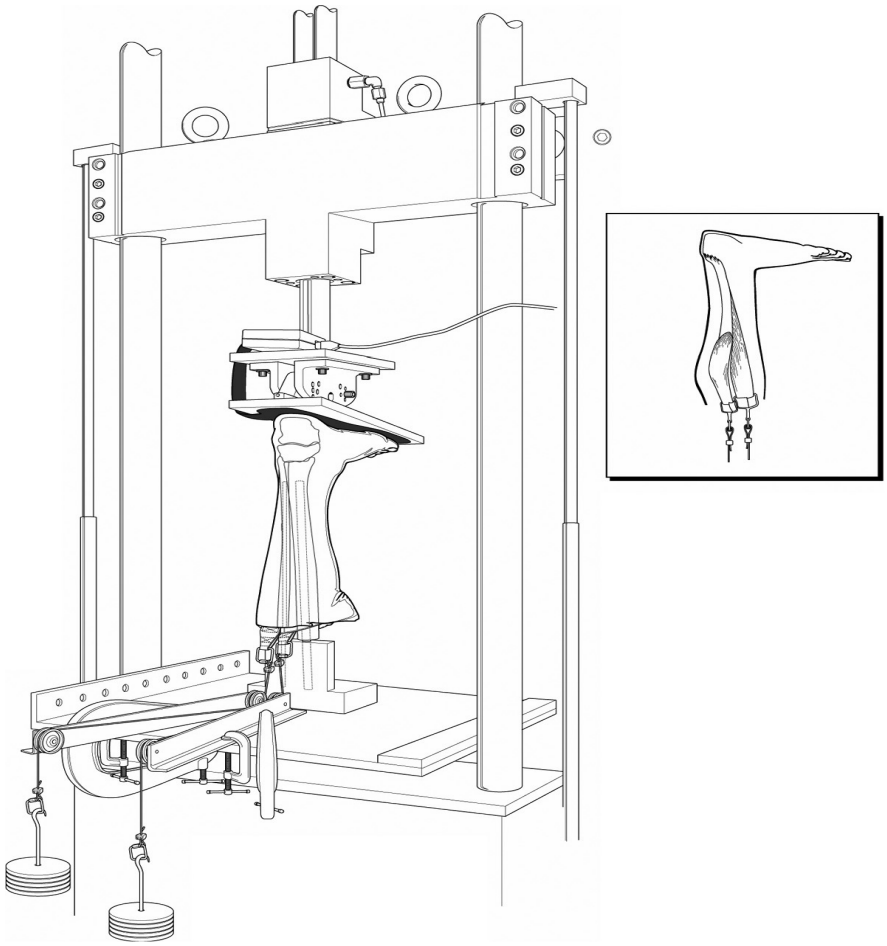


Fig. 19: The experimental set up (Aronow et al., 2006).

Visually noted a decrease in the height of the medial longitudinal arch of the specimens as triceps surae force increased with the decreased talus and first metatarsal angle and increased abduction at the talonavicular joint.

The treatment they suggest stretching exercises, serial casting, night splints, or joint stretch devices may correct the contracture, while functional orthoses may provide pressure redistribution and arch support. If none operative treatment is unsuccessful, the Achilles tendon can lengthen operatively (Aronow et al., 2006).

In other study similar method to the previous study, witch made to evaluate the effects of a medializing calcaneal osteotomy on length changes in the Achilles tendon and pressure patterns on the weight-bearing surface of the foot. For this purpose they used 14 fresh-frozen cadaver legs out of pathology abnormality or preexisting musculoskeletal disease.

Results showed that the use of calcaneal osteotomy decrease in average of the plantar foot pressure over the first and second metatarsal regions of the foot without a significant change in average pressure over the lateral forefoot and the Achilles tendon function as inverter of the foot by increasing the pressures of lateral aspect of the heel and they suggest that lengthening of Achilles tendon may not be necessary.

The fact that patients with flatfoot deformity suffer from a forefoot in varus and the calcaneus osteotomy would further increases this deformity, but it's successfully for unload the first and second metatarsal regions (Hadfield et al., 2003).

In other study to combine Achilles tendon elongation which trend toward shortening of the ventromedial aspect of the tendon with medial calcaneal osteotomy to reduce lateral forefoot pressure and shortening of the Achilles tendon while continuing to relieve medial forefoot pressure. The methods ware 28 fresh-frozen cadaver legs (age average 64 years) were axially loaded on a load-frame device.

They recommend the medial calcaneal osteotomy with 0.5cm superior translation to correct flatfoot deformity. They do not think that the Achilles tendon plays a significant role in offloading the medial forefoot as a result of the medial calcaneal osteotomy in a cadaver model (Hadfield et al., 2005).

Sometimes the gastrocnemius tightness can lead to flatfoot deformity, which need Achilles tendon lengthening correction to treat gastrocnemius muscle tightness and to correct the flatfoot deformity. In study of (Saxena & Widtfeldt, 2004) with purpose was to assess the clinical results of endoscopic gastrocnemius recession, for 2

cases study (2 females, age average 44 years) they had operative to treat gastrocnemius muscle tightness and to correct flatfoot deformity.

The measure postoperative period showed the flatfoot deformity treated and gastrocnemius tightness treated as well with increasing of the ankle dorsiflexion.

Radiographic view for flatfoot evaluation of case study of (female 55 year old) presented with left ankle pain along with progressive foot deformity (flattening of medial arch) and the “too many toes sign” positive. And are diagnosed (acquired flatfoot deformity), with PTTD and a gastrocnemius soleus tightness.

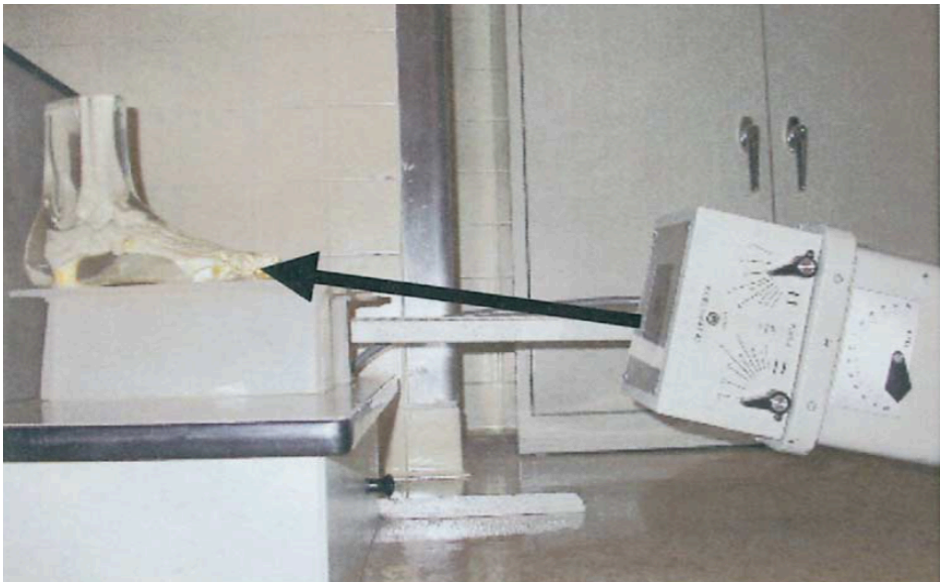


Fig. 20: The patient is positioned on the devices, standing in the base and angle of gait (Clark et al., 2004).

The treatment was medial calcaneal displacement osteotomy with Achilles tendon lengthening and flexor digitorum longus tendon transfer into the navicular. Radiographs examination (Figure 20) one year after surgery showed correction of the medial arch and complete healing of the osteotomy (Clark et al., 2004).

2.1.4. Ligaments insufficiency

The foot deformation resisted by various ligaments and the plantar aponeurosis, which support the foot and prevent the bones from moving relative to each other. When the first three metatarsals under loading they rotate relative to their corresponding cuneiforms about the axis point on dorsal side of the joint, and plantar tarsometatarsal ligaments resist this rotation. The plantar cuneonavicular ligaments resist rotation of the cuneiforms relative to the navicular, and the plantar calcaneonavicular ligament (spring ligament), which extends from the navicular to the calcaneus, resists rotation of the navicular relative to the talus about a point on the dorsal side of the talonavicular joint (Figure 21).

The Plantar tarsometatarsal ligaments resist rotation of the fourth and fifth metatarsals relative to the cuboid, and both the plantar calcaneocuboid ligament and a portion of the long plantar ligament resist the rotation of the cuboid relative to the calcaneus. While a large portion of the long plantar ligament, which originates on the calcaneus, extends only to the cuboid, there is also a portion extending from its origin on the calcaneus to the base from the second to fifth metatarsals, and that make restrains the rotation of the metatarsals relative to the cuneiforms, the cuneiforms relative to the navicular and the navicular relative to the talus for the medial arch, and of the metatarsals relative to the cuboid and the cuboid relative to the calcaneus for the lateral longitudinal arch (Arangio & Salathe, 2001).

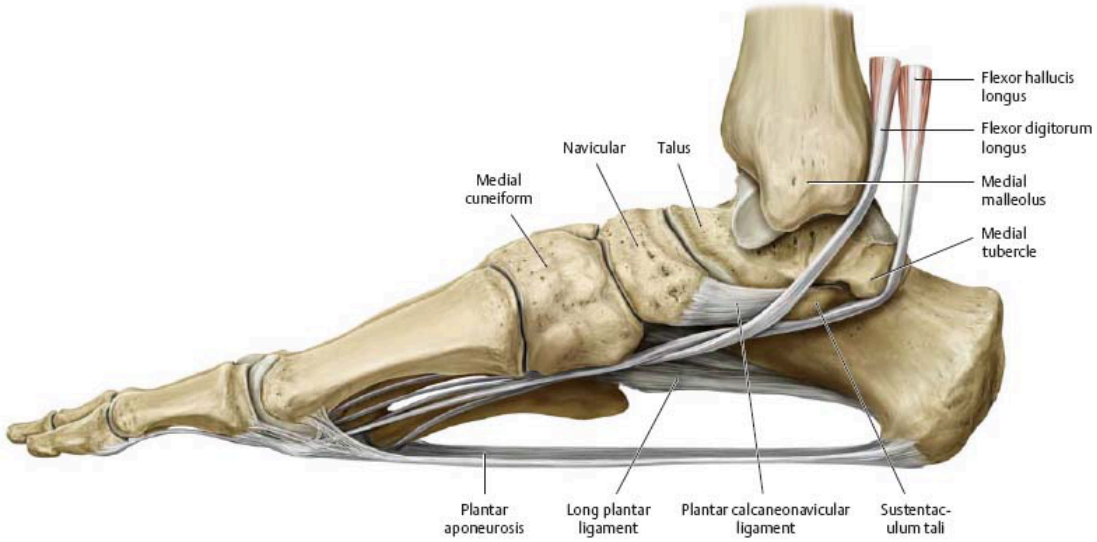


Fig. 21: Passive stabilizers of the longitudinal arch right foot, medial view (Gilroy et al., 2008).

Plantar Aponeurosis:

The biomechanical model regards the foot as a three dimensional structure consisting of rigid bones held together by flexible ligaments at their articulating surfaces. The plantar aponeurosis extends from the base of the calcaneus to each of the five rays, which one of the planter support structure. The muscles are applied additional external forces, which act at various locations on the foot. The muscle forces magnitude is determined by the requirement that they balance the body weight, preventing rotation about the subtalar joint axis and the ankle joint axis (Mann, 1992).

The plantar aponeurosis originates from the calcaneus and extends to the phalanges also resists the flattening of the foot arch and the deformation of the foot. It does so by resisting relative motion of each of the foot joints as the long plantar ligament, except in the plantar aponeurosis includes the joints of the first ray. Knowing the location of the pivot points on the dorsal side of foot joints, and the location of the points of insertion of foot ligaments and their mechanical properties, the displacements of the five metatarsal heads relative to the calcaneus can be determined in terms of the forces acting on the foot (Arangio & Salathe, 2001).

In acquired flatfoot model the tension in the plantar aponeurosis and long plantar ligaments is increasing the extending and losing their function. That was the hypothesis of (Arangio et al., 2004) by using the fresh frozen cadaveric foot specimen female 77 years old, They used data analyze the response of a normal foot, a flatfoot and a flatfoot with a 6mm subtalar arthroereisis, foot made flat by cutting medial soft tissue structures and cyclical loading. Other biomechanical evaluation and prospective controlled clinical study on subtalar arthroereisis in the flexible flatfoot will be needed. Subtalar arthroereisis is the elevation of the subtalar joint through osteotomy of the posterior facet or prosthetic insertion into the tarsal sinus.

In modeling of the flatfoot in both the uncorrected and arthroereisis corrected state, we eliminated the force in the muscle of the tibialis posterior to simulate a complete tear of the tibialis posterior tendon and reduced by 50% the strength of the spring ligament, the short plantar ligament, the portions of the long plantar ligament and the plantar aponeurosis extending to the medial rays to simulate stretching of the supporting structures over time.

The analysis of this study suggests that flattening the foot shifts the forces to and increases the moments on the medial column. The results further suggest that a 6mm subtalar arthroereisis substantially returns these forces and moments of the flatfoot back toward the values experienced in the normal foot (Figure 22).

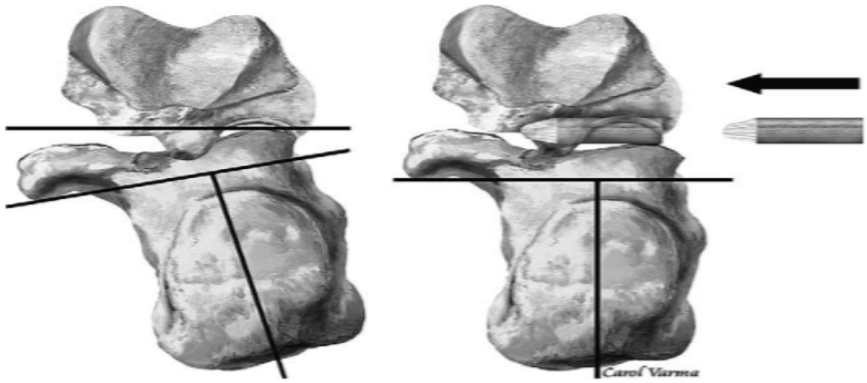


Fig. 22: Subtalar joint before and after arthroereisis (Arangio et al., 2004).

In this study the subtalar arthroereisis can return the foot geometry and the arch height to normal by X-ray criteria. However, there no correlation has been reported between clinical outcome and radiographic improvement for subtalar arthroereisis and there is no data that supports the hypothesis that the subtalar joint remodels after arthroereisis (Arangio et al., 2004).

Spring Ligament and deltoid Ligament:

The plantar calcaneonavicular (spring) ligament is a broad ligament and thick band connecting the anterior margin of the sustentaculum tali of the calcaneus; it lies below the head of the talus to the plantar surface of the navicular. It's forming part of the articular cavity for talus head and thus limits flattening of the medial longitudinal arch of the foot. The dorsal surface of the ligament is a triangular fibro cartilaginous facet on part of the talus head. The tendon of tibialis posterior supports the plantar surface of this ligament medially and laterally supported by the flexor hallucis and flexor digitorum longus. The medial border of the ligament is blended with the anterior fibres of the superficial part of the deltoid ligament of the talocrural joint (Borton & Saxby, 1997) and "deltoid ligament" consists of three parts: (tibiocalcaneal, tibiotalar and tibionavicular) (Figure 23) it is strongest ligament stabilizing the ankle joint. Sectioning the deep deltoid ligament results in greatly increased lateral talar shift and external talar rotation (Burns et al., 1993).

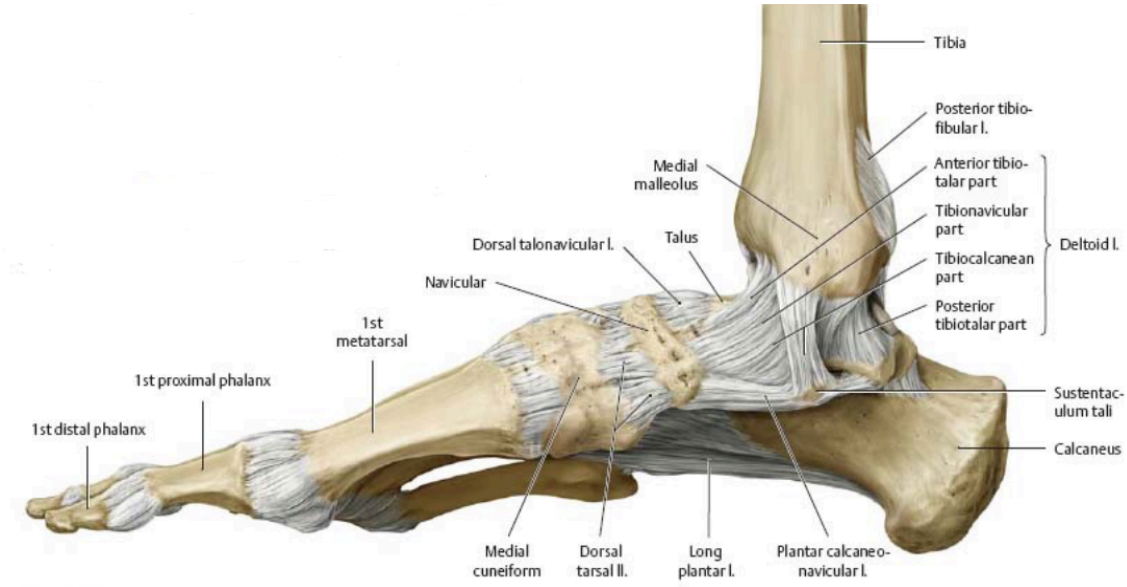


Fig. 23: Medial view of the ligaments of the ankle and foot of right foot (Gilroy et al., 2008).

The spring ligament complex traditionally has been described as containing the superomedial and inferior ligaments (Cheung & Rosenberg, 2001; Davis et al., 1996; Rule et al., 1993; Yao et al., 1999), the largest is superomedial ligament, which originates from the calcaneus (sustentaculum talus and anterior facet) and has a fanlike insertion on the navicular bone.

The average normal superomedial ligament thickness was reported as 4.8 ± 1.4mm in recent anatomic study (Taniguchi et al., 2003), because the superomedial portion is the greater contributor of stability and is the usual location of spring ligament tear and the inferior component is much smaller and is located plantar and lateral of the superomedial ligament, the original is between the anterior and middle calcaneal facets and inserting in a fanlike shape on the navicular bone. The insertion of the inferior ligament on navicular is lateral to the superomedial ligament, with interpose of fatty tissue between the two structures (Davis et al., 1996; Taniguchi et al., 2003), fatty tissue also spans the lateral border of the inferior ligament, adjacent to the bifurcate ligament (Rule et al., 1993).

When spring ligament fails, the talus rotates in a plantar fashion, and the calcaneus undergoes valgus angulation, the result adult acquired flatfoot deformity. Superomedial spring ligament, a rent in this portion of the spring ligament will be visible with abduction stress, a complete tear will allow a probe to pass directly into the talonavicular joint (Davis et al., 1996; Yao et al., 1999).

In a recent anatomic study, the third ligament of the spring ligament complex was described as being interposed in the fatty tissue between the superomedial and inferior ligament components (Taniguchi et al., 2003), and this ligament was likely regarded as part of the inferior ligament. The larger superomedial component is thought to play the most significant role in the spring ligament's contribution to ankle stability and is the component most often torn (Chen & Allen, 1997; Cheung & Rosenberg 2001; Davis et al., 1996).

In one case study of spring ligament rupture, 19 years old college triple jumper. Examination showed flatfoot deformity of the left foot with a positive "too many toes" sign. The medial arch of the foot was tender, especially at talonavicular joint appeared to be deformed.

The plantar fascia, deltoid ligament were intact and the tibialis posterior showed grade 5 power. The heel raise test was repetitive at left heel and remained in a valgus position.

Surgical exploration confirmed that there tear of the spring ligament, which repaired surgically. The patient returned to competitive sport six months after operation. Four years later he remained free from symptoms, had a normal medial longitudinal arch without valgus deformity (Borton & Saxby, 1997).

Spring ligament complex with superficial deltoid ligament stabilize the talonavicular joint (Kitaoka et al., 1997). While the superomedial ligament is a thicker band that is partly comprised of fibrocartilage, is directly medial of the posterior tibialis tendon insertion, and blends into the deltoid complex (Deland, 2001).

In study of (Imhauser et al., 2002) evaluation of the efficacy of external stabilizers in the conservative treatment of acquired flatfoot deformity, experiments ware conducted on 6 fresh-frozen cadaveric feet. Six different braces were tested including 2 in-shoe-orthoses, 3 ankle braces, and 1 molded ankle-foot orthotic. The experimental system consisted of a loading frame interfaced to a tensile testing machine, which applied an axial load equal to half body weight through the tibia-fibula. Furthermore, in-shoe-orthoses act to stabilize the medial longitudinal arch.

The orthoses act to restore the calcaneus angle and first metatarsal, the talus height and medial cuneiform. Both of them acting partially restore several parameters used to describe the geometry of the hindfoot and of the medial longitudinal arch in a flatfoot. These devices are passively supporting the plantar aspect of the foot by restoring the parameters of a non-pathologic foot. Ankle braces minimally restored the geometry of the hindfoot and the arch in a flatfoot.

In this study flatfoot sectioning the following ligaments created deformity: the long plantar ligament, the anterior portion of the deltoid ligament, the plantar aponeurosis, and the superomedial calcaneonavicular ligament (spring ligament complex), showed that the kinematic changes at the hindfoot and the medial longitudinal arch, as well as changes in plantar pressure patterns were consistent with a progressive flexible flatfoot deformity (Imhauser et al., 2002).

A spring ligament tear is typically a chronic degenerative process that occurs in conjunction with posterior tibialis tendon insufficiency (Chen & Allen, 1997; Cheung & Rosenberg 2001; Yao et al., 1999), a few case reports of acute isolated spring tears but these cases are rare (Borton & Saxby, 1997; Chen & Allen, 1997), the typical patient with posterior tibialis tendonopathy and a torn spring ligament is a middle-aged woman (Chen & Allen, 1997).

The superficial deltoid ligament spans the talonavicular and ankle joints providing coronal plane stability. With Adult acquired flatfoot deformity is the direct manifestation of diminished function of multiple ligaments including the spring ligament complex, the superficial deltoid ligament and interosseous ligament of the subtalar joint, these ligaments are often compromised. Although the spring ligament complex is the most frequently deficient ligament in flatfoot deformity, flatfoot deformity was achieved by sectioning the spring ligament complex, medial talonavicular joint capsule, superficial deltoid ligament and interosseous subtalar ligament, all of which are tissues commonly deficient in patients with adult acquired flatfoot deformity (Deland et al., 2005).

The purpose of (Baxter et al., 2015) study was to test the efficacy of several reconstruction techniques of the medial longitudinal arch, under hypothesize that an anatomic reconstruction of the spring ligament complex would correct the deformity better than other techniques tested. By using twelve fresh frozen cadaveric lower limbs of an average donor age of 55 years (range, 24-68 years).

They used three reconstruction techniques for each of them.

1- Anatomic spring ligament reconstruction.
2- Talonavicular ligament reconstruction.
3- Tibio-navicular ligament reconstruction.

The foot was held in plantar flexion (15 degrees) and moderate adduction, which resulted in a varus hindfoot and cavus midfoot.

They used a biomechanical model to investigate the effects of reconstructing different aspects of the medial longitudinal arch. They found that reconstructing the superior medial aspect of the medial longitudinal arch (Figure 24) corrected the talonavicular and hindfoot deformities that were created in the flatfoot model. The surgical techniques that reconstruct the other soft-tissue constraints of the medial longitudinal arch also corrected the abduction and dorsiflexion deformities but failed to correct coronal plane alignment of the hindfoot and midfoot.

Contradicting hypothesis of this study, the anatomic reconstruction of the spring ligament complex corrected the slightest amount of deformity but failed to correct the everted talonavicular joint and hindfoot.

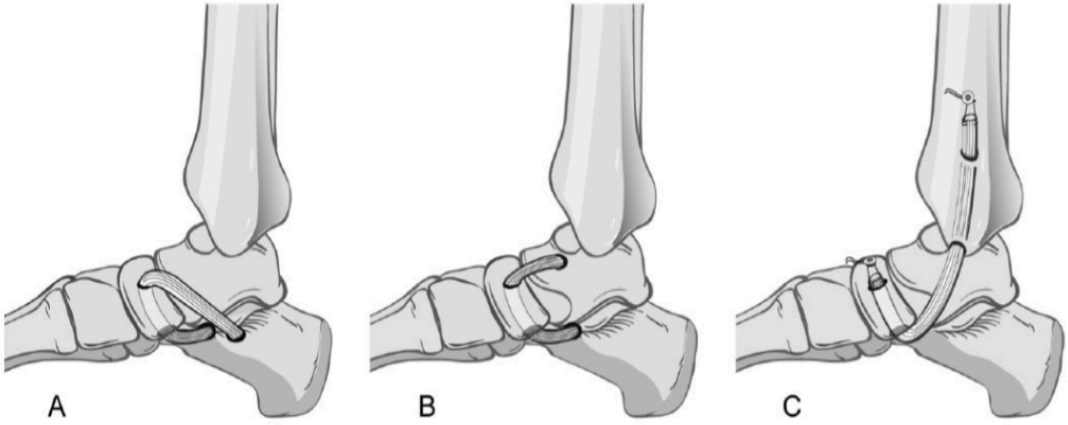


Fig. 24: Schematic of the (A) anatomic spring ligament, (B) talonavicular, and (C) tibio-navicular reconstructions. Prior to testing, 6 mm bone tunnels (dashed lines) were drilled through the navicular, tibia, talus, and calcaneus for their respective reconstructions (Baxter et al., 2015).

The conclusion, anatomic reconstruction of the spring ligament complex alone is not likely to correct all talonavicular joint ligament deformities. They find that

reconstruction of the tibial attachment of the medial talonavicular joint ligament structures can be better to correct midfoot and hindfoot misalignment in the presence of flatfoot deformity (Baxter et al., 2015).

Study (Arangio et al., 2007) was used the hypothesis that increase the loading on the medial arch in the adult flatfoot can be reduced through lateral column lengthening calcaneal osteotomy 10mm proximal from the calcaneal cuboid joint. Obtained the data by using one fresh frozen cadaver foot specimen. The foot was made flat by cutting the spring ligament, the tibionavicular portion of the deltoid ligament and the plantar aponeurosis. It was loaded manually and deformed into the flatfoot geometry and a flatfoot corrected with lateral column calculus lengthening to a load of 683n. The force of the tibialis posterior muscle was eliminated in the model, and the stiffness of the short plantar ligament, the spring ligament, and the portions of the long plantar ligament and the plantar aponeurosis extending to the medial rays was reduced by 50%.

The biomechanical analysis of the study supported the lateral calcaneal osteotomy in the treatment of adult acquired flat foot.

Research by (Gazdag & Cracchiolo, 1997) documented that PTTD may result in a stretched or ruptured deltoid ligament complex and spring ligament, the study was included 22 patients, 18 of them had a tendon transfer to treat rupture of the posterior tibialis tendon with evidence of injury to the spring ligament. The injury consisted of a complete rupture of the ligament in four patients, a longitudinal tear in the ligament in seven patients, and a lax ligament without a tear in seven patients.

The ruptured posterior tibialis tendon was treated with transfer of the flexor digitorum longus muscle in 20 of the 22 patients. Difference of methods was used to repair the ligament. It is important to determine the status of the spring ligament when patients are managed for rupture of the posterior tibialis tendon. Patients who suffers from a torn or lax spring ligament in addition to the ruptured posterior tibialis tendon more likely to have severe abnormalities of the hindfoot than those who have only a ruptured tendon (Gazdag & Cracchiolo, 1997).

Surgical repair of the spring ligament is becoming recognized as an important management of acquired flatfoot in adult, the MRI appearance of spring ligament

abnormalities by using MRI to find spring ligament tears include an abnormal spring ligament caliber, waviness, signal intensity, a full thickness gap and posterior tibialis tendonopathy. These results proved by study of (Toye et al., 2005) which done on 30 patients were selected for surgical purpose and they divided to three groups according to ligament condition. And the group with abnormality spring ligament has 14 patients, 13 of them has posterior tibialis tendonopathy, which mean there relation between the spring ligament and posterior tibialis muscle and losing function in one of them can influence on the other.

2. 1. 5. Tibialis anterior injury and foot muscles imbalance

Adult acquired flatfoot deformity can be caused by traumatic rupture of the tibialis anterior tendon associated (Aderinto & Gross, 2011), which can develop into PTTD (Frigg et al., 2006; George et al., 2009; Kolář et al., 2013; Mann et al., 1993).

Traumatic rupture of the tibialis anterior tendon can result of the weakness of ankle due to chronic tibialis anterior tendon rupture which uncommon and usually occur in patients older than 45 years, in one article of case study (Aderinto & Gross, 2011) they present the case of a 66 year old male, who underwent delayed reconstruction of left tibialis anterior tendon with 3 months after a spontaneous rupture.

They treated surgically by using an Achilles tendon allograft for treat tibialis anterior tendon rupture and it was successfully for this case.

Traumatic rupture of the tibialis anterior tendon can be associated with chronic tibialis posterior dysfunction, one case study with tibialis posterior dysfunction (68 year old female). The patient had noticed increasing flattening of the foot and lateral hindfoot pain. The clinical examination demonstrated a flatfoot on the right side valgus and obvious midfoot abduction during stance. On tiptoe test, the heel remained in valgus, was diagnosed with type II tibialis posterior dysfunction, which was treated conservatively at initially with custom orthoses and reviewed every 6 months, but was unsuccessful. The tendon dysfunction was shown on ultrasonography.

The patient underwent reconstructive surgery of both the right tibialis anterior and posterior tendons. Rupture of the tibialis anterior on a background of an already

existing PTTD can accelerate the development to acquired flatfoot deformity (George et al., 2009).

In other study similar to pervious case with tendon rupture of the tibialis anterior of patient 78 years old male. The clinical examination showed painful flatfoot deformity with hindfoot valgus and abduction of the forefoot affecting both feet. The magnetic resonance images showed a complete rupture of the tibialis anterior tendon with severe degeneration of the tibialis posterior tendon, complete rupture of spring ligament to be completely ruptured and the deltoid ligament partially ruptured.

The aim of treatment to correct the drop foot deformity, the tibialis anterior tendon was reconstructed and the deltoid ligament repaired. The clinical examination showed restore function and strength of the tibialis anterior tendon and symmetrical, hindfoot alignment corrected (Frigg et al., 2006).

Adult acquired flatfoot deformity can be caused by muscles imbalance, In one of comparative study done by (Hunt & Smith, 2004) compare walking stance phase hindfoot and forefoot motion, ankle joint moments and extrinsic foot muscle activity measured by EMG between normal and flatfeet. Study participants were 15 males with a history of musculoskeletal symptoms of flatfoot and compared with 18 males normal feet.

The extrinsic foot muscles, which are including in this study; soleus, gastrocnemius, tibialis anterior, peroneus longus and brevis muscles. The methods of this study, kinematic and kinetic data obtained from video recording of skin surface markers and a force plate, EMG recorded with surface electrodes.

They found in the flatfeet group: the forefoot was less adducted at toe-off, and total transverse plane ROM, at versus was less, the plantar flexor ankle moment at push-off was greater, the invertor moment was greater than normal feet group, for the EMG muscles activity showed higher muscle activity in tibialis anterior and lower activity in the peroneus, soleus and medial and lateral gastrocnemius muscles (Hunt & Smith, 2004).

3. Results

Q1- Is the dysfunction of the tibialis posterior muscle the main cause of acquired flatfoot?

Regarding the causes of acquired flatfoot in adult, a good base of evidence stems from a number of articles review, we found that PTTD is the most cause of acquired flatfoot in adults.

Farther details, the next common cause is Ligament insufficiency, which including the spring ligament, deltoid ligament and plantar fascia failed. In third position comes the intrinsic foot muscles dysfunction. In the fourth comes Achilles tendon tightness. The next cause is Tibialis anterior injury. In the last causes is Muscles imbalance (see Graph1).

Q2- Is the sensory motor stimulation technique the most effective therapeutic technique for the acquired flatfoot in the adults?

The sensory motor stimulation technique is not the most treatment used to treat acquired flatfoot in adults and the most used treatment is the surgical procedure (see Graph 2).

Farther details, there are multiple conservative and operative options for the treatment of acquired flatfoot in adults according to the cause, the conservative treatment different types of common treatment using (sensory motor stimulation technique, stretching exercises, strengthening exercises, orthotic devices management and medications) (see Graph 3).

Conservative Treatment in the first cause of flatfoot PTTD the common treatment using is “Orthotic devices management” is the most treatment. The Ligament insufficiency the main conservative treatment is the “Orthotic devices management”. In the Achilles tendon tightness the common conservative treatment is “Stretching exercise”. Dysfunction of plantar foot intrinsic foot muscles “Sensory motor stimulation techniques” is the most conservative treatment using to treat the acquired flatfoot. The last Muscle imbalance the common conservative treatment used is the “Stretching exercise”.

4. 1. The causes of acquired flatfoot in adults

Adult acquired flatfoot deformity and losing the medial longitudinal arch is a common disorder that can happen by many various causes. In my results, I had divided them according to the common causes of acquired flatfoot. I found the following information from a collection of authors who agreed and supported the following statements:

Statement 1. The most common cause of acquired flatfoot is the PTTD.

This statement is supported by these authors: (Alvarez et al., 2006; Arangio & Salathe, 2001; Baumhauer, 1997; Deland, 2001; Edwards et al., 2008; Ettore, 2013; Frey et al., 1990; Gambardella et al., 2014; Hinterman, 1995; Hintermann, 1996; Hintermann, 1997; Imhauser et al., 2004; Jari et al., 2002; Kitaoka et al., 1997; Kokub et al., 2012; Kolář et al., 2013; Lee et al., 2005; Mosier et al., 1999; Mueller, 1991; Myerson, 1996; Ness et al., 2008; Spratlry et al., 2013;Trnka, 2004; Vaudreuil et al., 2014) PTTD can happen by traumatic rupture of the tibialis posterior tendon (Holmes & Mann, 1992; Niki et al., 2001) or due to tibialis posterior tendonitis (Klaue, 1991; Nielsen et al., 2011), also the PTTD associated with accessory navicular bone can cause flatfoot deformity (Kiter's et al., 1999; Klaue et al., 2013).

Clinical special test for tibialis posterior muscle, which can use clinically to diagnosis PTTD, First metatarsal rise sign. The patient stands with full loading both feet, the shin of the effected side is taken with one hand externally rotated, by doing this move the heel is passively brought into a varus position because of the mechanical coupling between the tibia and calcaneus. The head of the first metatarsal remains on the ground in normal function of the tendon but is lifted in PTTD case.

Statement 2. Insufficiency of the ligaments structures is the one of causes of the acquired flatfoot deformity.

The direct manifestation spring ligament complex is the ligament most involved with PTTD. This statement is supported by these authors: (Balen & Helms, 2001; Baxter et al., 2015; Borton & Saxby, 1997; Cheung & Rosenberg, 2001; Deland et al., 2005; Jennings et al., 2008; Shibuya et al., 2008; Toye et al., 2005; Yao et al., 1999) or manifestation of diminished function of multiple ligaments including the spring ligament complex and the superficial deltoid ligament (Arangio et al., 2007; Chen & Allen, 1997; Gazdag & Cracchiolo, 1997; Imhauser et al., 2002; Kitaoka et al., 1997). Extending of plantar aponeurosis and long plantar ligaments and losing their function is cause to loss of the medial longitudinal arch, this resultantly becomes flatfoot status (Arangio et al., 1998; Arangio et al., 2004).

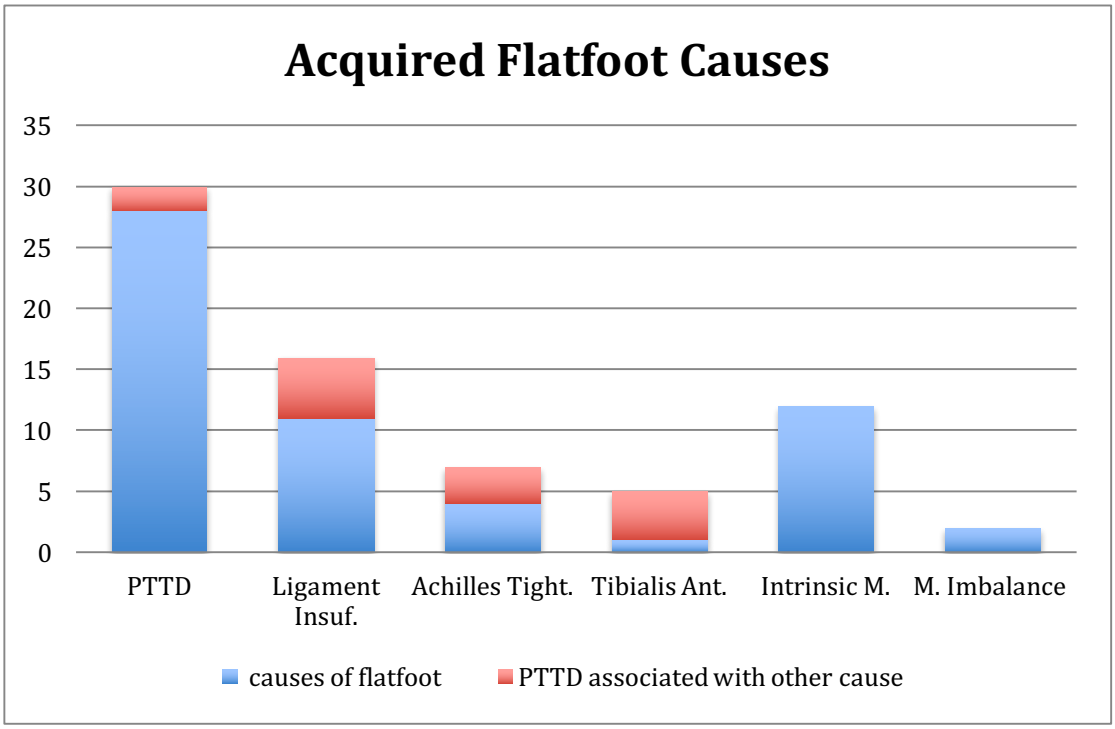
Statement 3. Achilles tendon tightness or over-pull or Achilles tendon rupture is often a cause of the acquired flatfoot deformity.

Gastrocnemius and soleus muscles tightness can lead to laxity of the ligaments which supporting the foot medial longitudinal arch of the midfoot structures, which can cause adult acquired flatfoot deformity. This statement is supported by these authors: (Aronow et al., 2006; Blackman et al., 2009; Leppilahti et al., 1998; Thordarson et al., 1995). In addition, the following authors state that acquired flatfoot can caused by gastrocnemius tightness involved with PTTD (Clark et al., 2004; Saxena & Widtfeldt, 2004; Van Boerum & Sangeorzan, 2003).

Statement 4. Adult acquired flatfoot deformity can be caused by traumatic rupture of the tibialis anterior tendon (Aderinto & Gross, 2011), which can develop into PTTD (Frigg et al., 2006; George et al., 2009; Kolář et al., 2013; Mann et al., 1993).

Statement 5. Dysfunction of the plantar foot intrinsic muscles and most common muscle is “abductor hallucis muscle”, which support the medial longitudinal arch lead to acquired flatfoot deformity (Fiolkowski et al., 2003; Fitts, 1996; Gaillet et al., 2004; Gazdag & Cracchiolo, 1997; Headlee et al., 2008; Jam, 2004; Jung et al., 2011; Kura et al., 1997; Nam et al., 2012; Saeki et al., 2015; Whittle, 2002; Wong, 2007).

Statement 6. Muscle imbalance uncommon cause of acquired flatfoot and there are including muscle strength imbalance of (gastrocnemius, soleus, tibialis posterior, tibialis anterior, flexor hallucis longus, abductor halluces, flexor digitorum longus, short plantar muscles, peroneus longus and brevis muscles) (Hunt & smith, 2004; Kolář et al., 2013). By exposition previous information on graph below, we can conclude the most cause of acquired flatfoot.



1st axis: PTTD, 2nd axis: Ligaments insufficiency, 3rd axis: Achilles tendon tightness, 4th axis: Tibialis anterior injury, 5th axis: Intrinsic muscles dysfunction, 6th axis: muscles imbalance.

Graph 1. Review of the causes of acquired flatfoot deformity in adults

Through the graph of causes it's obvious that PTTD is the most cause of acquired flatfoot according to the number of the articles. The next common cause is Ligament insufficiency, which including the spring ligament, deltoid ligament and plantar fascia failed, number of them is associated with PTTD. In the third position comes the intrinsic foot muscles dysfunction. In the fourth Achilles tendon tightness, which can also associated with PTTD. And the next cause is Tibialis anterior injury, which mostly associated with PTTD. In the last causes is Muscles imbalance (tibialis posterior, gastrocnemius, soleus, tibialis anterior, flexor digitorum longus, flexor hallucis longus, superficial short plantar muscles and peroneus longus and brevis).

4. 2. The therapeutic techniques for the acquired flatfeet in the adults

In a review of the adult acquired flatfoot treatment options, the treatment is following of the causes of flatfoot; we divided the treatment to conservative treatment and surgical treatment.

Conservative treatment:

Conservative treatment early in the course of the disease is aimed at preventing further disability and progressive deformity and avoiding future surgical intervention. There are five common types of treatment divided to (Sensory motor stimulation technique, Stretching exercises, Strengthening exercises, Orthotic devices management and Medications) for each of the causes.

- Early treatment of PTTD flatfoot deformity suggested for more effective therapy and to avoid any further progressive failure of the ligament (Deland et al., 2005; Hintermann, 1996). The Sensory motor stimulation technique can be used for PTTD flatfoot as training the foot pressure distribution, the practice of "short foot exercise" and dynamic control of pronation during gait "balance exercises" (Alvarez et al., 2006; Ettore et al., 2013; Kolář et al., 2013; Nielsen et al., 2011). Some of authors suggested foot immobilization, Orthotic devices management like removable cast or boot and footwear modifications (Alvarez et al., 2006; Edwards et al., 2008; Jari et al., 2002; Klaue et al., 2013; Lee et al., 2005; Myerson, 1996; Trnka, 2004) over and above taping technique may also be used (Nielsen et al., 2011) all these techniques used to prevent overuse and subsequent rupture of tibialis posterior to correct the hindfoot valgus. Strengthening exercises can be used as well (Alvarez et al., 2006). For the patients, who complain from PTTD caused by tibialis posterior tendonitis the treatment including Medications as anti-inflammatory drugs (Edwards et al., 2008; Lee et al., 2005; Myerson, 1996; Nielsen et al., 2011; Trnka, 2004).

- The treatment of flatfoot deformity, which caused by ligaments insufficiency (spring ligament complex and deltoid ligament) in study of (Imhauser et al., 2002) suggested treating passively by using Orthotic devices management as in-shoe-orthoses, ankle braces or ankle-foot orthotic (Deland et al., 2005).

- Treatment of the Achilles tendon tightness associated with flatfoot, by using Stretching exercises for gastrocnemius and soleus (Alvarez et al., 2006; Clark et al., 2004; Ettore et al., 2013; Hadfield et al., 2003; Saxena & Widtfeldt, 2004) or combine it with Orthotic devices; serial casting, night splints or functional orthoses to medial arch support (Aronow et al., 2006). Strengthening exercise for foot inversion and toe flexor (Ettore et al., 2013) with proprioception exercises "foot sole facilitation one of the Sensory motor stimulation techniques" (Kolář et al., 2013) and relaxation for hypertonic muscles.

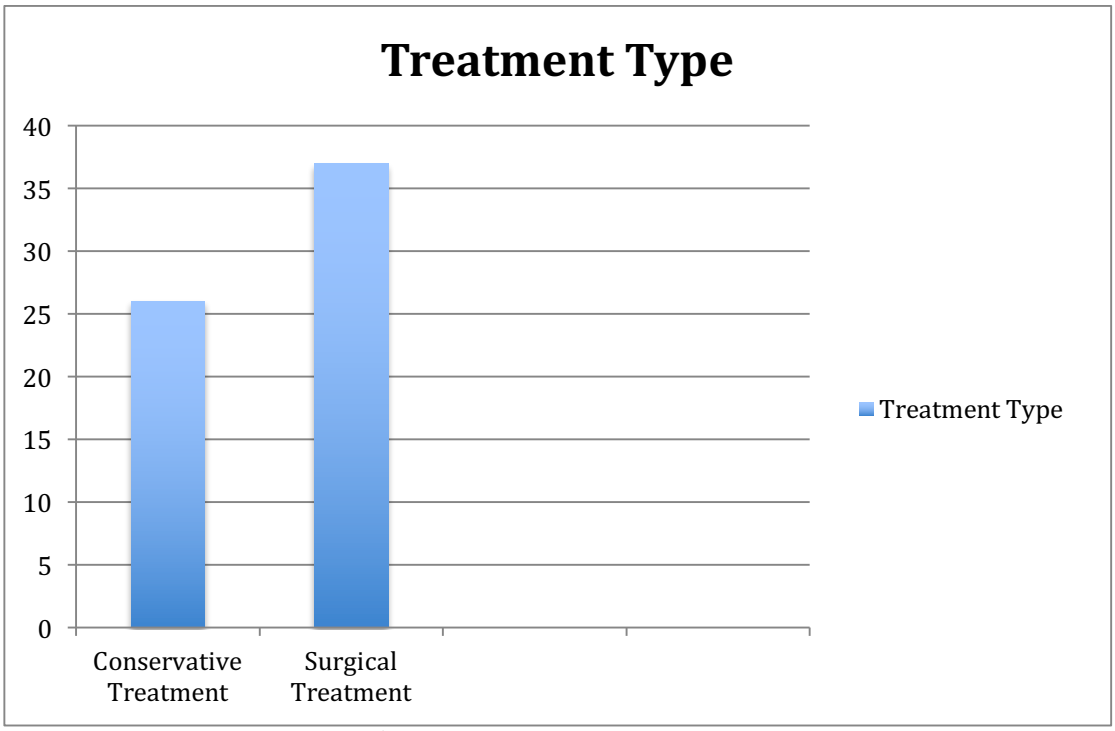
- Treatment exercises targeting the intrinsic foot muscles to strengthening and prevent flatfoot in early stages (Fiolkowski et al., 2003), and the most exercise of the Sensory motor stimulation techniques recommended is "short foot exercise" to restore the medial longitudinal arch (Gaillet et al., 2004; Gazdag & Cracchiolo, 1997; Jam, 2004; Jung et al., 2011; Wong, 2007) beside of the short foot exercise (Kolář et al., 2013) suggests foot sole facilitation (proprioception facilitation), training the foot pressure distribution, the practice of three point support (Headlee et al., 2008) which can combined with Orthotic devices and taping techniques. Other author suggests one-leg standing position, type of "balance exercise" one of Sensory motor stimulation (Nam et al., 2012). (Saeki et al., 2015) mention other exercise for toes flexors "Towel toe curl exercise" to strengthen the intrinsic foot muscles.

- Flatfoot with muscles imbalance the treatment included multiple Strengthening exercises for (gastrocnemius, soleus, tibialis posterior, tibialis anterior, flexor hallucis longus, abductor halluces, flexor digitorum longus, short plantar muscles, peroneus longus and brevis) Thera-band exercise for each muscle group included gastrocnemius and soleus muscles Stretching exercise (Alvarez et al., 2006) and also the soft tissue techniques, Stretching exercises are suggested for short and hypertonic muscles and Sensory motor exercises as sole facilitation are recommended according to (Kolář et al., 2013).

Surgery treatment:

- Moreover, patients who didn't respond after initiation of conservative treatment, surgery treatment to restore normal foot alignment is required, especially to the patients with painful flexible flatfoot deformitny (Ettore, 2013; Giannini et al., 1992; Klaue et al., 2013). Surgical repairing just for tibialis posterior is not enough for treating flexible flatfoot (Niki et al., 2001) its needs to be combined with other procedures because the ligaments, which supported the medial arch is elongated and losing its tension. The surgical procedures depend on the etiology of the flatfoot condition; techniques are including medial displacement calcaneal osteotomy (Arangio & Salathe, 2001; Kitaoka et al., 1998; Tordarson et al., 1998), arthrodesis or arthroereisis (Hintermann, 1996; Hintermann, 1997; Klaue, 1991; Lee et al., 2005; Mann et al., 1993) and the other soft tissue procedures, tibialis posterior tendon advancement and flexor digitorum longus transfer (Edwards et al., 2008; Hinterman, 1995; Hintermann, 1997; Lee et al., 2005; Myerson, 1996; Trnka, 2004; Vaudreuil et al., 2014).
- Flatfoot with gastrocnemius tightness and Achilles tendon over-pull, the treatment procedure if non-operative treatment is unsuccessful, the Achilles tendon can lengthen operatively (Aronow et al., 2006) or medial calcaneal osteotomy with lengthening of Achilles tendon (Hadfield et al., 2003; Hadfield et al., 2005), in case gastrocnemius tightness with PTTD the surgical procedure Achilles tendon lengthening correction to treat gastrocnemius muscle tightness and to correct the flatfoot deformity (Saxena & Widtfeldt, 2004) and can combined with flexor digitorum longus tendon transfer (Clark et al., 2004).
- Flatfoot with spring ligament tear or abnormalities the surgical procedure can be surgical repairing or reconstruction of the spring ligament (Baxter et al., 2015; Borton & Saxby, 1997; Chen & Allen, 1997; Deland, 2001; Jennings et al., 2008; Lee et al., 2005; Toye et al., 2005).
- Flatfoot with spring ligament tear involved with deltoid ligament tear, is treated by the surgical treatment lateral calcaneal osteotomy (Arangio et al., 2007), or by other way by transfer flexor digitorum longus muscle (Gazdag & Cracchiolo, 1997).

- In tibialis posterior dysfunction due to traumatic rupture of the tibialis anterior tendon, which is an uncommon injury, the treatment procedure is reconstructive surgery of tibialis anterior and posterior tendons (Aderinto & Gross, 2011; Frigg et al., 2006; George et al., 2009). By exposition previous information on graph below, to comber between the conservative and surgical treatment, we can conclude the most treatment used to treat acquired flatfoot.

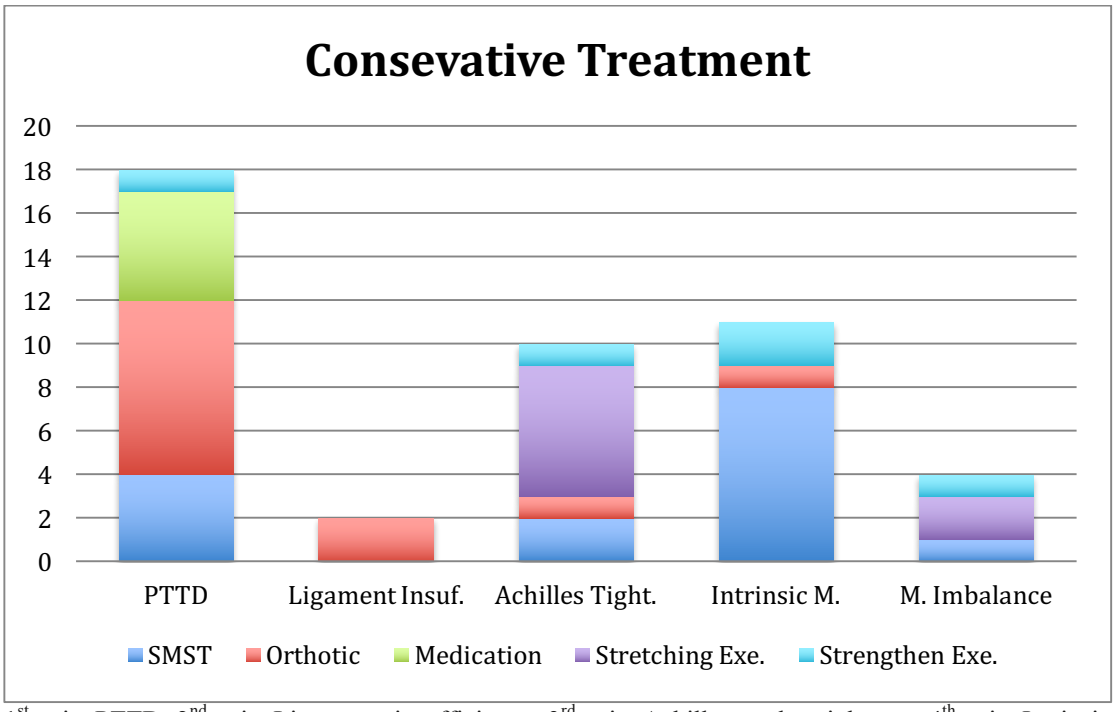


1st axis: Conservative treatment, 2nd axis: Surgical treatment.

Graph 2. The types of treatment for acquired flatfoot deformity in adults

Through the graph of "treatment type" the surgical treatment is more using than conservative treatment of acquired flatfoot and that confirm the patient comes for treatment lately after cross the first stages of acquired flatfoot and developed to farther stages, which need surgery procedure to fix the deformity.

Moreover, by exposition previous information of conservative treatment on graph below, we can conclude the most conservative treatment used to treat each type of causes of acquired flatfoot.



1st axis: PTTD, 2nd axis: Ligaments insufficiency, 3rd axis: Achilles tendon tightness, 4th axis: Intrinsic muscles dysfunction, 5th axis: muscles imbalance.

Graph 3. Review of conservative treatment for acquired flatfoot deformity in adults

Through the graph above we can see the "Conservative Treatment" in the first column the common treatment using for PTTD is "Orthotic devices management" is the most treatment. The Ligament insufficiency column the main conservative treatment is the "Orthotic devices management". In the Achilles tendon tightness column the common conservative treatment is "Stretching exercise". Dysfunction of plantar foot intrinsic foot muscles column it's clearly that "Sensory motor stimulation techniques" is the most conservative treatment using to treat the acquired flatfoot. And lately the Muscle imbalance column the common conservative treatment used is the "Stretching exercise".

5. Conclusion

Regarding the causes of adults acquired flatfoot in our results and the evidence from a number of articles review, which we divide to 6 groups (PTTD, Ligaments

insufficiency, Achilles tendon tightness, Intrinsic foot muscles dysfunction, Tibialis anterior injury and the last Muscles imbalance). We found that the most common cause of the acquired flatfoot in adults is the PTTD (posterior tibialis tendon dysfunction) and common to associate PTTD with other causes (Ligaments insufficiency: spring ligament complex and superficial deltoid ligament are often failed ligaments during acquired flatfoot. Tibialis anterior injury mostly associated with PTTD in flatfoot case. Achilles tendon tightness is partly associated with PTTD. Tibialis posterior muscle is one of the muscles, which involved in Muscles imbalance during acquired flatfoot).

It is important to establish the correct diagnosis and staging in order to choose the best method of flatfoot treatment. In our results showed that surgical procedure is more using to treat acquired flatfoot in adults, but we believe that patients should be conservatively treated before progression to late stages if possible. Early conservative treatment is often successful to preventing progression of the dysfunction and deformity.

There are multiple conservative and operative options for the treatment of acquired flatfoot in adults according to the cause, the conservative treatment, which we discus in our results are including five different types of common treatment using (sensory motor stimulation technique, stretching exercises, strengthening exercises, orthotic devices management and medications).

Through our results we found most common conservative treatment using for PTTD is “Orthotic devices management”. Dysfunction of the plantar intrinsic foot muscles the “Sensory motor stimulation techniques” is the most conservative treatment using to treat the acquired flatfoot. Ligament insufficiency the main conservative treatment is the “Orthotic devices management”. In the Achilles tendon tightness and Muscle imbalance most common conservative treatment is “stretching exercise”.

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7. Appendix

Author / year	Article Title	Causes of flatfoot	Method	Treatment
Imhauser et al., 2004	The effect of posterior tibialis tendon dysfunction on the plantar pressure characteristics and the kinematics of the arch and the hindfoot	PTTD	5 cadaveric feet, study of plantar pressure	
Niki et al., 2001	The effect of posterior tibial tendon dysfunction on hindfoot kinematics	Tibialis posterior rupture	Activity of tibialis posterior muscle by using EMG	Surgical repairing for tibialis posterior not enough for treat flexible flatfoot
Kiter's et al., 1999	Tibialis posterior tendon abnormalities in feet with accessory navicular bone and flatfoot	Accessory navicular and PTTD	MRI analyses of the 27 patients	
Imhauser et al., 2002	Biomechanical evaluation of external stabilizers in the conservative treatment of acquired flatfoot deformity	Anterior portion of the deltoid ligament and spring ligament	Changes in plantar pressure patterns by using 6 cadaveric feet	Suggest conservative treatment: In-shoe-orthoses, ankle braces and ankle-foot orthotic
Hintermann, 1997	Tibialis posterior dysfunction: a review of the problem and personal experience	Tibialis posterior rupture and I-II-III stages PTTD	Effect surgical treatment of PTTD, 38 patients	Tendon transfer, calcaneal osteotomy or arthrodesis
Hintermann, 1996	The first metatarsal rise sign: a simple, sensitive sign of tibialis posterior tendon dysfunction	PTTD	21 feet for 19 patients first metatarsal rise sign	Suggested early treatment for more effect

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Saeki et al., 2015	Relationship between navicular drop and measuring position of maximal plantar flexion torque of the first and second-fifth metatarsophalangeal joints	Intrinsic foot plantar flexor muscles	Relation between navicular drop and intrinsic plantar strength of 10 healthy men	Suggests that exercises targeting the intrinsic foot muscles
Fitts, 1996	Muscle fatigue: the cellular aspects	Fatigue of plantar intrinsic foot muscles	Medial longitudinal arch height	
Headlee et al., 2008	Fatigue of the plantar intrinsic foot muscles increases navicular drop	Plantar foot intrinsic muscles (abductor hallucis)	Navicular drop assessment and EMG of 21 healthy adults	Taping techniques and orthotic devices, dynamic control of pronation during gait through neuromuscular exercises
Gazdag & Cracchiolo, 1997	Rupture of the posterior tibial tendon	Abductor hallucis muscle	EMG of abductor hallucis for 20 subjects	Short foot exercise is more beneficial than the toe curl exercise in comparing the activity of the abductor hallucis
Fiolkowski et al., 2003	Intrinsic pedal musculature support of the medial longitudinal arch	Intrinsic foot muscles (abductor hallucis)	Navicular drop assessment and EMG of 10 subjects	Strengthening of intrinsic and extrinsic muscles may help to increase muscular support of the arch
Gaillet et al., 2004	Modifications of baropodograms after transcutaneous electric stimulation of the abductor hallucis muscle	Abductor hallucis muscle can influence on plantar pressure pattern	30 subjects assessed plantar pressure and footprint	Abductor hallucis activity can make change of plantar pressure

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Thordarson et al., 1995	Dynamic support of the human longitudinal arch. A biomechanical evaluation	Achilles tendon over-pull	10 cadaver feet, muscle activity during stance phase	
Blackman et al., 2009	Cadaveric flatfoot model: ligament attenuation and achilles tendon over pull	Achilles tendon over-pull	10 cadaver feet, assessed plantar pressure during midstance phase	
Hunt & Smith, 2004	Mechanics and control of the flat versus normal foot during the stance phase of walking	Higher muscle activity in tibialis anterior and lower activity in the peroneus, soleus and gastrocnemius	EMG study of 33 subjects	
Leppilahti et al., 1998	Ruptures of the Achilles tendon: Relationship to inequality in length of legs and to patterns in the foot and ankle	Achilles tendon rupture	101 patients measured by footprint	
Aronow et al., 2006	The effect of triceps surae contracture force on plantar foot pressure distribution	Achilles tendon contractures	Effect contracture of Achilles tendon on 10 cadaver feet	Stretching exercises, serial casting, night splints, joint stretch devices, functional orthoses to arch support. If none operative treatment is unsuccessful, the Achilles tendon can lengthen operatively
Hadfield et al., 2003	Effects of medializing calcaneal osteotomy on achilles tendon lengthening and plantar foot pressures		Plantar pressure over 1st and 2nd metatarsal of 14 cadaver legs	Medial calcaneal osteotomy with lengthening of Achilles tendon

Author / year	Article Title	Causes	Method	Treatment
Klaue et al., 2013	Central calcaneal osteotomy for correction of flexible pes planovalgus deformity	Posterior tibialis tendon pathology and accessory navicular bone	Effect surgery treatment on 26 feet in 21 patients	Surgery to restore normal foot alignment from painful flexible flatfoot than 1 year after initiation of conservative treatment like orthoses, insoles and physical therapy
Hadfield et al., 2005	The effects of a medializing calcaneal osteotomy with and without superior translation on Achilles tendon elongation and plantar foot pressures		Effect medial calcaneal osteotomy on planter pressure of 28 cadaver legs	Surgical medial calcaneal osteotomy with 0.5cm superior translation to correct flatfoot deformity
Aderinto & Gross, 2011	Delayed repair of tibialis anterior tendon rupture with Achilles tendon allograft	Weakness of ankle due to chronic tibialis anterior tendon rupture	Gait analyses of the feet for one patient	Achilles tendon allograft for treat tibialis anterior tendon rupture
Saxena & Widtfeldt, 2004	Endoscopic gastrocnemius recession: preliminary report on 18 cases	Gastrocnemius tightness with PTTD	Function test, single heel raise for 2 cases study	Achilles tendon lengthening correction to treat gastrocnemius muscle tightness and to correct the flatfoot
George et al., 2009	Traumatic rupture of the tibialis anterior tendon associated with chronic tibialis posterior dysfunction	II PTTD due to traumatic rupture of the tibialis anterior tendon	One case study assessed by tiptoe test and gait analyses	Reconstructive surgery of tibialis anterior and posterior tendons
Frigg et al., 2006	Combined anterior tibial tendon rupture and posterior tibial tendon dysfunction in advanced flatfoot	Degeneration of the tibialis posterior due to rupture of the tibialis anterior tendon	Dynamic planter pressure, one case study	Tibialis anterior tendon reconstructed and the deltoid ligament repaired

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Clark et al., 2004	The Kirby view: a radiographic view for flatfoot evaluation	PTTD with gastrocnemius soleus tightness	Radiographic flatfoot evaluation of one case study	Medial calcaneal displacement osteotomy with Achilles tendon lengthening and flexor digitorum longus tendon transfer
Arangio et al., 2004	Biomechanical model of the effect of subtalar arthroereisis on the adult flexible flatfoot	Extending of plantar aponeurosis and long plantar ligaments	One cadaveric foot, X-ray data analysis	Suggest that subtalar arthroereisis to return the forces and moments of the flatfoot back to normal foot
Arangio & Salathe, 2001	Medial displacement calcaneal osteotomy reduces the excess forces in the medial longitudinal arch of the flatfoot	PTTD	Normal foot compared with flatfoot and flatfoot with a displacement calcaneal osteotomy	Medial displacement calcaneal osteotomy
Borton & Saxby, 1997	Tear of the plantar calcaneonavicular (spring) ligament causing flatfoot	Tear of the spring ligament	One case study with positive too many toes sign	Surgical repairing of the spring ligament
Deland et al., 2005	Posterior tibial tendon insufficiency: which ligaments are involved	The spring ligament complex tear involved with PTTD	MRI of 31patients with PTTD compared group without PTTD	Suggest early treatment of PTTD to protect or prevent progressive failure of ligaments
Baxter et al., 2015	Reconstruction of the medial talonavicular joint in simulated flatfoot deformity	Spring ligament complex tear	12 cadaveric tested under loads that simulated standing	Reconstruction of the spring ligament (medial talonavicular ligament)

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Arangio et al., 2007	A biomechanical analysis of the effect of lateral column lengthening calcaneal osteotomy on the flat foot	Tear of spring ligament, the tibionavicular portion of deltoid ligament and the plantar aponeurosis	Biomechanical analysis after treatment for one cadaveric foot	Lateral calcaneal osteotomy
Gazdag & Cracchiolo, 1997	Rupture of the posterior tibial tendon	PTTD may resulted from stretched or ruptured deltoid ligament complex and spring ligament	Documental research of 22 patients with PTTD	Transfer of the flexor digitorum longus muscle
Toye et al., 2005	MRI of spring ligament tears	Spring ligament abnormalities and posterior tibialis tendonopathy	MRI finding of spring ligament tears of 30 patients	Surgical repair of the spring ligament
Nielsen et al., 2011	Nonoperative care for the treatment of adult acquired flatfoot deformity	Tibialis posterior tendonitis	Study of treatment effect on 64 patients	Medications, such as anti-inflammatory drugs, orthotics or bracing such as a foot orthoses and arch and ankle brace, low-articulating ankle-foot orthosis, cast-boot or shoe modifications
Alvarez et al., 2006	Stage I and II posterior tibial tendon dysfunction treated by structured nonoperative management protocol: an orthosis and exercise program	Stage I and II of PTTD	Study of treatment effect by using single-support heel rises for 47 patients	Foot orthotic, exercises for plantar flexor included gastrocsoleus stretching. The strengthening exercises for tibialis posterior, peroneus, tibialis anterior and gastrocsoleus muscles and included isokinetic exercises, Thera-pand exercise, heel rises, toe walking and home exercises for each muscle group

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Ettore et al., 2013	Approach and treatment of the adult acquired flatfoot deformity	PTTD	Evaluation of the treatment by using X-ray	Achilles tendon stretching exercise, inversion and toe flexor strengthening along with proprioception exercises is recommended. Surgery treatment is indicated in patients who didn't respond after 3 months or more of conservative management
Lee et al., 2005	Diagnosis and treatment of adult flatfoot	PTTD	MR, CT scan, gait analysis, EMG and force plate analysis	Initial treatment activity modifications, loss weight, orthotic devices management, immobilization and footwear modification and anti-inflammatory medications. Surgical treatment techniques are including osteotomy, arthroereisis and arthrodesis, the other soft tissue procedures, tibialis posterior tendon advancement, flexor digitorum longus transfer and reconstruction of spring ligament
Kolář et al., 2013	Clinical Rehabilitation	PTTD, dynamic stabilizer of the arch (tibialis posterior, tibialis anterior, flexor digitorum longus, flexor hallucis longus and superficial short plantar muscles)		Sensory motor exercises, sole facilitation, training the foot pressure distribution, the practice of three point support and "short foot" Treatment also incorporates soft tissue techniques; foot joint mobilization and stretching of the short muscles and relaxation witch have hyper tone

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Wong, 2007	Influence of the abductor hallucis muscle on the medial arch of the foot: A kinematic and anatomical cadaver study	Abductor hallucis muscle	8 cadaver specimens of the leg, navicular drop height	Rehabilitation regimens focused on strengthening of intrinsic foot muscles, useful to prevent and treat of early disease
Trnka, 2004	Dysfunction of the tendon of tibialis posterior	PTTD	Too many toes sign, first metatarsal rise sign, radiography, MRI, ultrasound	Medications, such as anti-inflammatory drugs, immobilization either in a below-knee cast with Cryotherapy, Modifications to footwear are indicated for severe deformity. Synovectomy, Transfer of flexor digitorum longus and medial displace-ment osteotomy, arthrodesis is indicated for a fixed hindfoot deformity with associated pain
Ness et al., 2008	Foot and ankle kinematics in patients with posterior tibial tendon dysfunction	PTTD	Motion analysis system and weight-bearing radiographs for 34 patients	
Jam, 2004	Evaluation and retraining of the intrinsic foot muscles for pain syndromes related to abnormal control of pronation	Fatigue of plantar intrinsic foot muscles	Using navicular drop Test	Short foot exercise is more beneficial than the toe curl exercise in comparing the activity of the abductor hallucis muscle and in preventing lowering of the arch

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Holmes & Mann, 1992	Possible epidemiological factors associated with rupture of the posterior tibial tendon	PTTD caused by rupture	67 patient, PTT palpation, single leg-heel rise test	
Myerson, 1996	Adult acquired flatfoot deformity	PTTD	Single leg-heel rise test	Anti-inflammatory medication, and immobilization, removable cast or boot may be used to prevent overuse and subsequent rupture. Surgical treatment including flexor digitorum longus tendon transfer for I II PTTD, calcaneal osteotomy and arthrodesis III PTTD
Edwards et al., 2008	Tibialis posterior dysfunction	PTTD	Double heel raise test, too many toes sign, radiograph and ultrasound	Anti-inflammatory medication, immobilization and orthotic to correct the hindfoot valgus. Surgical treatment including flexor digitorum longus tendon transfer for I II PTTD, calcaneal osteotomy and arthrodesis III PTTD
Mosier et al., 1999	Pathoanatomy and etiology of posterior tibialis dysfunction	PTTD	Single leg-heel rise test	
Jennings et al., 2008	The Effects of Sectioning the Spring Ligament on Rearfoot Stability and Posterior Tibial Tendon Efficiency	PTTD, spring ligament complex abnormalities	5 fresh-frozen cadaveric feet	Suggest reconstruction procedures for spring ligament

Author / year	Article Title	Causes	Method	Treatment
Vaudreuil et al., 2014	Comparison of transfer sites for flexor digitorum longus in a cadaveric adult acquired flatfoot model	Stage II of PTTD	Seven cadaveric feet, plantar pressure	Flexor digitorum longus tendon transfer
Yao et al., 1999	MR imaging findings in spring ligament insufficiency	Spring ligament insufficiency associated with PTTD	MRI study for 13 feet in 12 subjects	
Kura et al., 1997	Quantitative analysis of the intrinsic muscles of the foot	Abductor hallucis muscle	Study of 11 cadaveric feet	
Kitaoka et al., 1997	Effect of the posterior tibial tendon on the arch of the foot during simulated weight bearing: biomechanical analysis	PTTD	13 cadaveric feet, medial arch height	
Arangio et al., 1998	Effect of varying arch height with and without the plantar fascia on the mechanical properties of the foot	Plantar fascia	Cadaveric feet study, medial arch height	
Kitaoka et al., 1998	Three-dimensional analysis of flatfoot deformity: cadaver study		Average of medial arch height of11 cadaveric feet,	Suggest surgically restore appropriate alignment and function in hindfoot

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Tordarson et al., 1998	Effect of calcaneal osteotomy and plantar fasciotomy on arch configuration in a flatfoot model		Study of 7 cadaveric feet, angular between the first metatarsal and talus	Suggest medial displacement calcaneal osteotomy
Chen & Allen, 1997	MR diagnosis of traumatic tear of the spring ligament in a pole vaulter	Spring ligament and deep deltoid ligament tear	One case study by using MRI	Spring ligament and deltoid ligament surgically reconstruction
Kitaoka et al., 1997	Stability of the arch of the foot	Spring ligament and deltoid ligament	Study of 19 cadaveric feet, Metatarsal-to-talus total rotation	
Nam et al., 2012	The relationship between activity of abductor hallucis and navicular drop in the one-leg standing position	Abductor hallucis muscle	49 subjects, navicular drop in the one-leg standing position	One-leg standing position could be used to correct the navicular drop
Spratlry et al., 2013	Validation of a population of patient specific adult acquired flatfoot deformity models	PTTD II stage	6 subjects, MRI, X-ray during single-leg stance	

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Mann et al., 1993	Pes cavus. Surgery of the foot and ankle	PTTD associated tibialis anterior weakness	Height of medial longitudinal arch in standing position	Arthrodesis
Giannini et al., 1992	Kinematic and isokentic evaluation of patients with flatfoot		Gait analysis	Suggest surgical treatment to avoid the pain, arthritis or deformities for forefoot
Baumhauer, 1997	Adult flatfoot: posterior tibial tendon dysfunction pathologic anatomy	PTTD	Gait analysis, plantar pressure	
Hinterman, 1995	Dysfunction of the posterior tibial muscle due to tendon insufficiency	PTTD		Reconstruction of tibialis posterior tendon, tendon transfer if the deformity progressive to painful flatfoot
Klaue, 1991	Rupture of the degenerated posterior tibial tendon symptoms and therapy	PTTD duo to rupture or tendon degenerative		Suggest tendon reconstruction and arthrodesis
Whittle, 2002	Gait analysis an introduction	Abductor hallucis muscle	Gait analysis	
Cheung & Rosenberg, 2001	MR imaging of ligamentous abnormalities of the ankle and foot	Spring ligament tear associated with PTTD	MRI examination	
Deland, 2001	The adult acquired flatfoot and spring ligament complex	II stage of PTTD		Repair spring ligament complex

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Van Boerum & Sangeorzan, 2003	Biomechanics and pathophysiology of flatfoot	Gastrocnemius tightness with PTTD		
Frey et al., 1990	Vascularity of the posterior tibial tendon	PTTD	Study of 28 cadaveric limbs	
Jung et al., 2011	Comparison in the muscle activity of the abductor hallucis and the medial longitudinal arch angle during toe curl and short foot exercises	Abductor hallucis muscle	20 subjects, EMG of abductor hallucis during short foot exercise	Suggest short foot exercise for intrinsic foot muscles
Mueller, 1991	Acquired flatfoot secondary to tibialis posterior dysfunction: biomechanical aspects	PTTD		
Kokub et al., 2012	Effect of the posterior tibial and peroneal longus on the mechanical properties of the foot arch	PTTD	8 fresh frozen cadaveric feet	
Gambardella et al., 2014	Isolated Dislocation of the Posterior Tibial Tendon in an Amateur Snowboarder: A Case Report	PTTD	Case study, MRI	

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Balen & Helms, 2001	Association of posterior tibial tendon injury with spring ligament injury, sinus tarsi abnormality, and plantar fascitis on MRI imaging	Spring ligament abnormality associated with PTTD	MRI images study of 25 patients	
Jari et al., 2002	Non-surgical management of tibialis posterior insufficiency	PTTD	38 patients, MRI	Orthotics and custom shoe-wear as required
Shibuya et al., 2008	Association of tibialis posterior tendon pathology with other radiographic findings in the foot: A case-control study	Spring ligament abnormality associated with PTTD	MRI study of 24 patients with PTTD	