

# Muscles and Tendons

- function in normal, aging, & pathological foot
- evidence that foot muscles adapt quickly to stimuli.

## Introduction

- leg (shank) has 3 compartments: anterior, posterior, lateral
- + anterior: tib ant (TA), peroneus tertius (PT), extensor hallucis longus (EHL), extensor digitorum longus (EDL)
- \* all dorsiflex, TA invert too, PT evert too, & toe extension  
EPL, EOL
- + lateral: peroneus brevis (PB) & longus (PL)
- \* plantar flex, evert, stabilize lateral arch & longitudinal arch
- + posterior (plantar flexion): Triceps Surae (gastroc + soleus) & plantaris if present
- \* gastroc produces most force of all in leg
- \* deepa: flexor digitorum longus (FDL) flexor digitorum longus (FDL): flex toes & plantar flex. Tib post (PT) adduct & invert
- intrinsic foot muscles: 2 layers
- + superficial: FDB, ABH, ABDM
- + 2nd layer: QP: adjust line of pull of FDL & flex 2-5, lumbricals

+3<sup>rd</sup>: FDM, FHB, ADH  
4<sup>th</sup>: interossei → different pennate structure plantar/dorsal  
+dorsal: FDB/EHB

- Achilles tendon strongest in body, triceps surae attached through it to calcaneus
- TB attaches underside of foot, tendon wraps around medial malleolus, supports arch, inverts foot
- specific intrinsic foot muscle function difficult, but improved via MRI and ultrasound
- + ultrasound reliable for muscle volume, thickness, and CSA & plantar fascia thickness
- + Anatomical & physiological CSA of FDB correlates with toe flexion strength

## Biomechanical Function

### Normal Foot

- Toe flexion strength pivotal in gait & sports
- Foot Stability
- + Historically, PF assumed most important for arch support, acting as a truss in weight bearing
- + Quiet stance, intrinsic muscles matter a lot
- \* E.g. ABH dynamic elevation of arch, simultaneous flexion/inversion of 1<sup>st</sup> metatarsal, inversion of calcaneus, external rotation of tibia
- \* EMG shows 20-8% drop in ABH activity + 3mm navicular drop after nerve block + TB
- \* Conversely, stimulation of ABH, FDB, GP opposes medial arch compression
- \* ABH fatigue alone sees navicular drop up to 5mm.

- + FHL & FDL help too, resisting dorsiflexion
- Balance
- + Antero/posterior sway in normal stance dictated by plantar/dorsiflexions in ankle strategy
- + Tapered (narrow) stance requires dominant eversion/inversion ankle strategy for lateral sway
- + Size of ABH correlated better open loop stability & energy transfer
- + Chronic Ankle Stability  $\rightarrow$  smaller total extrinsic foot + ankle muscle volume
- \* Smaller superficial posterior group esp. soleus
- \* Lateral ankle sprain? smaller CSA of PL
- \* Smaller oblique head ADH & FHS
- + Toes play huge role in balance, esp. ADH, FDS, & QP activation
- + simple foot strengthening protocol works well for older folk
- Locomotion
- + Support phase, PF elongates, spring-like
- + with toes dorsiflexed, PF significantly thinner; tension, elevates arch + shortens foot
- + Muscles also help modulate energy function in foot
- \* FDS isometrically contracts under load to facilitate energy storage
- \* ADH, FDS, QP also change length to support arch by slowing arch deformation
- + TA eccentrically contracts to allow gradual plantar flexion; dorsiflexion foot for clearance in swing
- + FHL couples hindfoot & 1st MTP joint kinematics

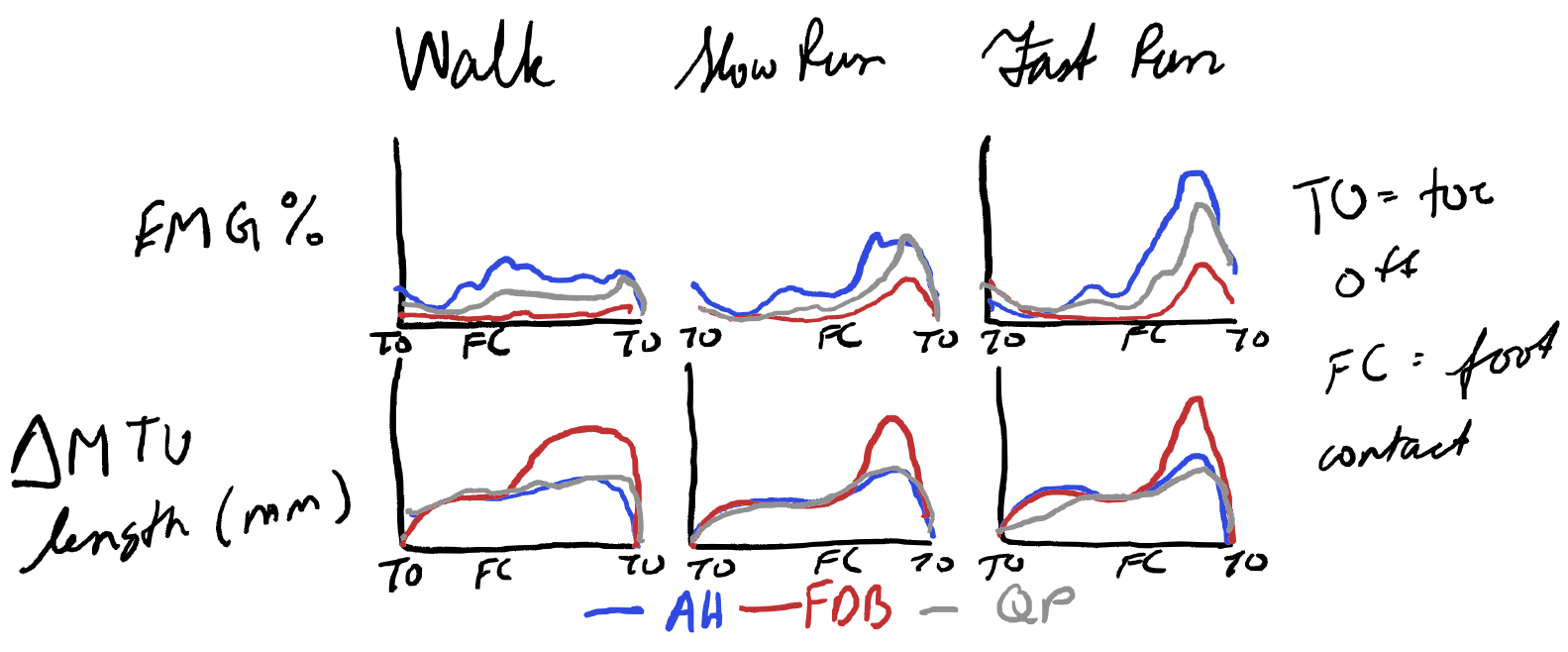
+ MTU length and peak EMG ABH, FDB, GP increase with higher gait velocity

Refer to textbook for EMG graphs of ABH, FDB, and GP in double versus single leg stance

+ see activation in late swing & stance, preparing for high deformation force

+ Running, FDB active in stance, large burst at foot contact, midstance peak, then deactivation

+ intrinsic muscles activate to absorb force and prevent excessive deformation



- + position of foot dictates muscle influence on force
- \* rearfoot strikers often rely on properties of shoe, highly cushioned
- \* forefoot, heavy reliance on intrinsic muscles to handle GRF
- + RFS → FFS, large burst of activation in ABH & FDB at midstance to absorb force & at toe off
- \* unsure about long term adaptations since peak activations in study were equal
- + Sprinters often have more developed foot muscles, but size alone does not correlate to speed
- + RFS long distance vs FFS saw no difference in CSA, thickness or strength in foot muscles

Refer to textbook for  
ultrasonography graph relating  
muscle thickness for sprinters,  
controls, and RFS vs FFS in  
both leg and foot muscles

- + Ultrasonography measured thickness in leg & foot muscles longer in sprinters than non, no diff in LDR to FF/RF
- \* study had highly trained runners well adapted to load
- \* everyone was still wearing cushioned shoes, not barefoot for forefoot strikes

## Aging

- decline in strength occurs around 6<sup>th</sup> decade
- toe muscles particularly affected, both thickness & CSA

Refer to Figure 6.4 for  
CSA diff in ankle and  
muscles between young and old.

- toe flexor strength reduction greatly correlates with fall risk
- + quad and ankle strength does not differ compared to hallux strength

## Pathologies

- many exist, discus common over here
- Plantar Fasciitis
- + MSK condition of pain in inferomedial aspect of heel exacerbated often by periods of non-weight bearing
- + Sonography reveals  $> 4$  mm thickness, or 1.2-1.3 mm more than nonaffected side is positive for PF.

- + Diagram of sonography shows hypoechoic area of plantar fascia
- + cause multifactorial, intrinsic foot muscle weaker by problem
- + toe flexors show 80% weaker
- + unsure about arch supporting muscles
- \* e.g. no discernible diff in TP
- \* Some discrepancy in forefoot to hindfoot muscle
- + Chronic PF shows smaller hindfoot muscles, but difficult to draw conclusions

### - Pes Planus

+ low medial arch, increased dorsiflexion, eversion, & external rotation  $\Rightarrow$  possible  $\uparrow$  in lower limb issues

+ focus on arch supporting muscles: TP, TA, FHL, FDL

\* TP atrophy  $\Rightarrow$  FDL hypertrophy to compensate

+ flat foot: altered activation pattern

\*  $\uparrow$  TA at contact,  $\uparrow$  TP in midfoot, propulsion,  $\downarrow$  PL & PD in early stance

\* Strong invertors

\* PL looks like it could plantar flex, but trying to prevent arm.



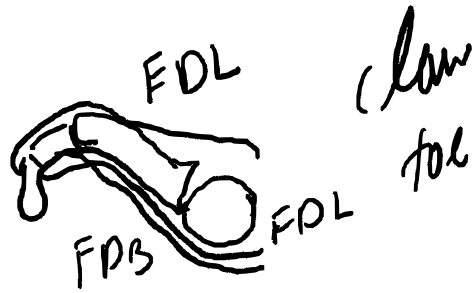
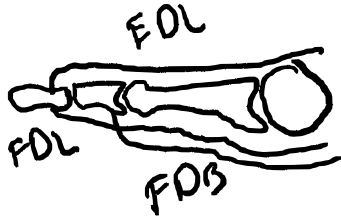
- + Wacker et al. confirm ↑ CSA of FDL in runners with planus feet
- + with pes planus, larger cohort, found: larger extrinsic invertors (FDL, FHL), in CSA & thick
- + peroneals significantly reduced CSA
- \* more recent study saw thicker peroneals in flat feet
- \* theory: weaker invertors (peroneals) advantage invertors (TP) and force FHL & FDL to produce higher moments for sagittal plane function

- + under intrinsic plantar fatigue, reduced arch height
- \* planus: ↑ ABH ↑ FDB ↓ ABDM in runners CSA
- \* pes planus: ↓ ABH ↓ FHB
- + less thick FHB → navicular drop when starting
- + weak plantar intrinsic can result in faster arch drop
- \* focus on keeping ABH and FHB strong to reduce load on FDL & FHL

+ tendons in pes planus: larger TA, thinner Achilles, thinner middle & anterior PF, PTTD in adult acquired flat foot.

- Toe deformities
- + affect 60-74% adults due to atrophy in plantar + flexor / extensor imbalance
- ↓ cadaver studies of claw / hammer toes reveal dorsal shift in tendons / muscles

Normal toe



\* intertarsal & metatarsal shift by large amount w.r.t. metatarsal shaft.

\* joint alignment alters axis of toe flexors, less biomechanically efficient.

+ Sonographic studies reveal smaller ABH & FHL in valgus

\* Association is unknown. Does excessive extension decrease advantage of ABH, making it gradually weaker? Or does disuse of ABH against ADH allow for joint deformity

+ Older folk with toe deformities have smaller ABH, FHL, ABH, FHL.

\* Extrinsic FDL does NOT differ

\* Intrinsic stiffen joints & flatten toes, Extrinsic plantarflex + control gait.

- Diabetic Neuropathy (diabetic polyneuropathy, DPN)
- + motor neuropathy detrimental to muscle strength  $\rightarrow$  atrophy
- + DPN progresses distal to proximal, attacking foot & ankle
- + MRI: foot muscle volume is  $\frac{1}{2}$  that of normal
- \* later stage sensory & motor dysfunction occurs
- \* 41% reduction in strength of ankle dorsiflexion/plantar flexion
- \* Foot muscles are smaller, decline faster in strength per year

## Footwear & Orthoses

- habitual use of orthoses induces structural & functional changes

Refer to Figure 6.7 for MRI's for progression of neuropathic diabetes for lower leg & midfoot over 10 years

- long term adoption (or lack thereof) and how it changes the foot is still relatively unknown
- + Cross culture study of populations in Mexico vs U.S showed unshod foot environment = higher arch, stronger ABH, ABDM
- hypothesis that unshod running shoes atrophic foot is conjunctural r.o.
- + Some studies do show changes in CSA + strength in ABH when using minimal shoes over unshod (+ thinner proximal fascia, thicker Achilles)
- \* Risk of higher metatarsal bone marrow edema
- perpetuated high heels shortens calf MTU
- + shorten gastrocnemius, thicker & stiffer Achilles
- orthoses intervention reduces TP activity in gait of both normal & flat-footed.
- + most only agree on above outcome, not sure if good.
- \* and not sure if that's good or not??
- + too much variation in orthoses design make conclusions and research difficult

## Interventions

- foot muscles respond well to training

- toe flexor strength considerably improved.
- four fold improvement after 6 weeks of strength training
- sway / balance significantly improved after toe strengthening
- Do people with DPN respond to toe training

similarly?

+ yes, even under condition of baseline weakness, targeted strengthening still improves outcomes

Refer to text Figure 6.9 for  
Hallux and toe strength changes in  
training, home, & control groups

- short foot improve hallux flexor strength & ABW CSA
- also use toe spread out for maximal MVC effort on ABW & ABDH
- + Toe spread out created  $3.41^\circ$  diff in hallux valgus after 8 weeks
- benefits in already healthy needs research
- + 22% volume increase in healthy runners
- \* not statistically sig diff in strength
- (\*) my note: does volume correlate with power-endurance??

## Areas of Future Biomechanical Research

- individual contributions of specific structures to normal/abnormal foot function.

- most study is done on static measures.
- + Dynamics would greatly improve understanding
- Studies are mostly based on CSA, no proper cause & effect.
- + does atrophy cause the condition, or does atrophy occur because of the condition?
- + more longitudinal studies needed, recording motor loss, change in physical activity, etc, etc.
- Specificity of foot strengthening
- + focus on flexion of proximal interphalangeal & metatarsophalangeal ROM while restricting distal interphalangeal joint.
- + look at TP in pes planus and orthotic intervention