

Muscles and Tendons

- function is normal, aging, & pathological foot
- evidence that foot muscles adapt quickly to stimulus.

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 inverts too, PT evorts too, & toe extension EPL, EDL
- + lateral: peroneus brevis (PB) & longus (PL)
 - * plantarflex, invert, stabilize lateral ankle & longitudinal arch
- + posterior (plantar flexion): Triceps Surae (gastroc + soleus)
 - & plantaris if present
- * gastroc produces most force of all in leg
- * deeper: flexor digitorum longus (FDL) flexor digitorum longus (FDL): flex toes & plantarflex. fib pos² (PT) added to invert
- intrinsic foot muscles: 21 layers
- + superficial: FDB, AB^{1,2}, ABOM
- + 2nd layer: QP: adjust line of pull of FDL & flex 2-5, lumbricals

+3^{c1}: FDM, FHB, ADH

CMP: interossei → different pennate structure plantar/dorsal

+ dorsal: FDB/FHB

- Achilles tendon stronger in body, triceps surae attached through L to calcaneus
- TB attacks 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 FDD correlates with toe flexion strength

Bio mechanical Function

Normal Foot

- Toe flexor 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 make a lot
 - * E.g. ABH dynamic elevation of arch, simultaneous flexion/inversion of 1st metatarsal, inversion of calcaneus, external rotation of tibia
 - (*) EMG shows 26.8% drop in ABH activity + 3mm navicular drop after nerve block to TB
 - (*) Conversely, stimulation of ABH, FDD, GP opposes medial arch compression
 - (*) ABH fatigue alone sees navicular drop up to 5mm.

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

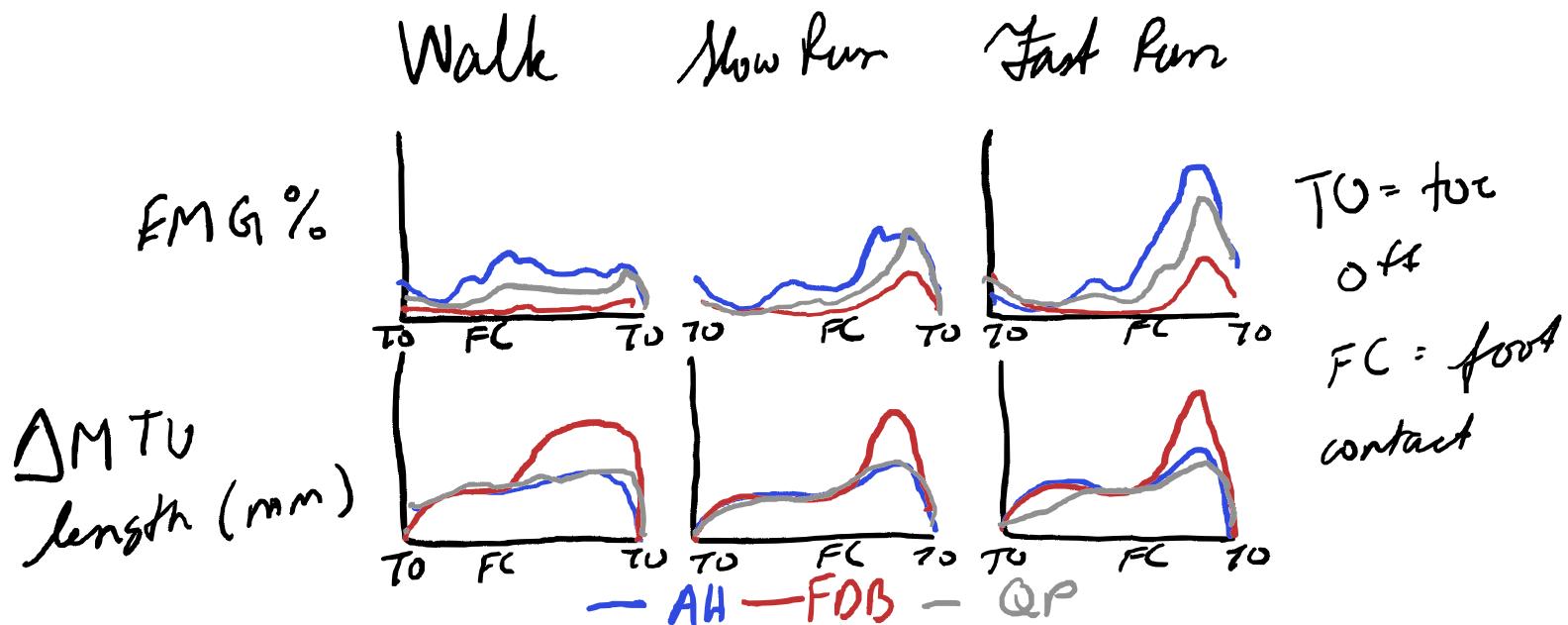
+ MTU length and peak EMG_{AH}, FDB, QP increase with higher gait velocity

Refer to textbook for EMG_{AH}
graphs of AH, FDB, and QP
in double versus single leg

+ see activation in late swing & stance, prepares 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



$TO = \text{toe off}$
 $FC = \text{foot contact}$

- + 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 \rightarrow FFS, large burst of activation in AHS & FDBs 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
ultrasoundography graph relating
muscle thickness for sprinters,
controls, and RFS vs FFS in
both leg and foot muscles

- + Ultrasoundography measured thickness in leg & foot muscles larger in sprinters than non, no diff in LDR to FF/RE
- * 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 6th decade
- toe muscles particularly affected, both thickness & CSA

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

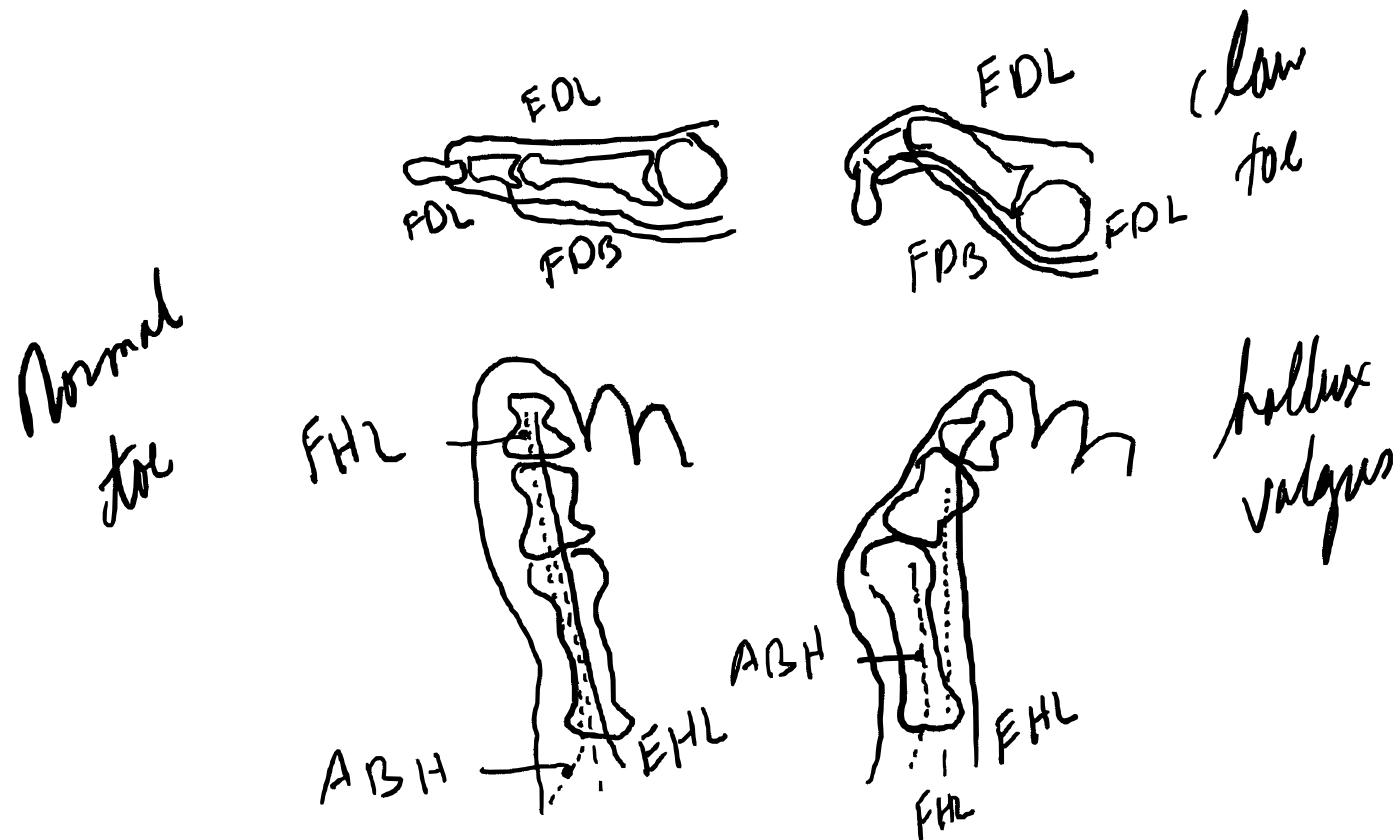
- toe flexor strength reduction greatly correlates with fall risk
- + quadr and ankle strength does not differ compared to hallux strength

Pathologies

- many exist, discuss common ones here
- Plantar Fascitis
 - + MSK condition of pain in inferomedial aspect of heel exacerbated after periods of non-weight bearing
 - + Sonography reveals ↑ heel thickness, or 1.2-1.3 mm more than unaffected side is positive for PF.

- + Diagram of sonography shows hypoechoic area of plantar fascia
- + cause multifactorial, intrinsic foot muscle weakness big problem
- + toe flexors show 80% weaker
- + unsure about arch supporting muscles
- * e.g. no discernable diff in TP
- * Some discrepancy in forefoot to hindfoot muscle
- + Chronic PF shows smaller hindfoot muscles, but difficult to draw conclusion
- Per Planes
- + lowered medial arch, increased dorsiflexion, eversion, & external rotation \Rightarrow possible ↑ in lower limb issues
- + focus on arch supporting muscles: TP, TA, FHL, FDL
- * TP atrophy \rightarrow FDL hypertrophy to compensate
- + flat feet - altered activation pattern
- * ↑ TA at contact, * TP in midfoot, propulsion, ↓ PL & PP in early stance
- * Strong invertors
- * PL looks like it could plantarflex, but tiny moment arm.

- + Wacker et.al. Confirm ↑ CSA of FDL in runners with planus feet
- + with pes planus, larger cohort, found:
 - large extrinsic invertors (FDL, FHL), in CSA & thick plantar fascia significantly reduced CSA
- + paraverts significantly reduced CSA
- * more recent study saw thicker plantar fascia in flat feet
- * theory: weaker invertors (paraverts) 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 (SA)
- * pes planus: ↓ ABH ↓ FDB
- + less shock FDB → malleolar drop when standing
- + Weak plantar intrinsic can result in faster arch drop
- * focus on keeping ABH and FDB 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/hammertoes reveal dorsal shift in tendons / muscles



- * interossei & lumbrical shift by large amount w.r.t. metatarsal shaft.
- * joint alignment alters axis of toe flexors, less biomechanically efficient.
- + Sonographic studies reveal smaller ABH & FHB in valgus
- * Association is unknown. Does excessive extension decrease advantage of ABH, making it gradually weaker? Or does disease of ABH against ABH allow for joint deformity?
- + older folk with toe deformities have smaller QP, FDB, ABH, FHB.
- * 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 → 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
- * ~11% reduction in strength of ankle dorsiflexion/plantarflexion
- * 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 lower leg & midfoot
of neuropathic diabetic 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 AB1^H, ABDM
- hypothesis that unshod running shoes atrophic foot is conjectural r.r.
- + some studies do show changes in CST + strength in AB1^H when using minimal shoes over cushioned (+ thicker proximal fascia, thicker Achilles)
- * Risk of higher metatarsal bone marrow edema
- perchance high heels shorten calf MTU
- + shorter gastrocnemius, thicker & stiffer Achilles
- orthoses intervention reduces TP activity in gait of both normal & flatfooted.
- + 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 improves vert.
- four fold improvement after 6 weeks of strength training
- sway / balance significantly improve after toe strengthening
- Do people with DPN respond to toe training similarly?
 - + Yes, even under conditions of baseline weakening, targeted strengthening still improves outcomes

Refer to text figure b9 for changes in
Haller and lever for strength in
control group
(training)

- short foot improve hallux flexor strength & ABDH CSA
 - also use toe spread out for maximal MVC effect on ABD & ABDH
 - + Toe spread out created 3.41° 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
- ④ my note: does volume correlate with power-endurance??

Area 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