Posterior Tibial Tendon Dysfunction: Conservative and Surgical Management Strategies

Date: Friday, September 30, 2016
Time: 1:00 PM – 3:00 PM

Session ID & Location: 3A: MtgRm11
CEU Eligibility: .20

Presented by: Dr. Jason Wamack, DPM, FACFAS, Diplomat ABPS; Andy Gean, PT, DPT, OCS, COMT

Session Description: Posterior tibial tendon dysfunction (PTTD) is a progressive condition that affects both active and sedentary individuals and represents a wide continuum of clinical presentations. Impairments and functional limitations associated with PTTD range from mild soft tissue involvement to severe foot deformity. Much research and investigation has been performed by the surgical and rehabilitation communities into the potential biomechanic, tendon-specific, and activity-related factors associated with individuals who have PTTD.

This session will present a review of the clinical presentation of individuals with PTTD, clinical and radiographic considerations for individuals with PTTD, conservative management strategies, and surgical management strategies for individuals with PTTD. Andy Gean, PT, DPT, OCS, COMT, will discuss conservative management considerations to include discussions of: current research on PTTD and exercise, orthotic considerations, concepts of regional interdependence, and cortical neuroplastic changes associate with individuals with tendinopathy. Dr. Jason Wamack, DPM, will provide expert insight into conservative and surgical decision-making factors for individuals with PTTD.

Session Learning Objectives:

- Discuss the continuum of clinical presentations of individuals with PTTD
- Discuss clinical and histological differences in stages of PTTD
- Review clinical and biomechanical exam for individuals with medial ankle pain
- Provide evidence and rationale for conservative management of PTTD
• Discuss treatment of PTTD to include concepts of regional interdependence and cortical motor control changes associated with tendinopathies
• Discuss surgical approaches for management of PTTD and relevant post-operative considerations.

Presenter Bio(s):

Dr. Jason Wamack, DPM, FACFAS, Diplomat ABPS is a board certified podiatric surgeon with the Center for Sports Medicine & Orthopaedics in Chattanooga, TN. Dr. Wamack is a 2001 graduate of Barry University of Graduate Medical Sciences. He completed residency with Gulf Coast Podiatric Surgical Residency Foundation in Houston, TX where he served as chief resident. He has practiced in private practice while also participating in Erlanger Hospital residency.

As a board certified podiatric surgeon at the Center for Sports Medicine and Orthopaedics, Dr. Wamack specializes in all foot and ankle problems from the athlete to the ‘weekend warrior’. He, his wife Heather, their sons Carter and Hudson, and their daughter Kaitlyn make their home in Ooltewah.

Andy Gean, PT, DPT, OCS, COMT is a 2007 graduate of the University of Tennessee-Chattanooga DPT program and practices in an outpatient setting with the Center for Sports Medicine & Orthopaedics in Hixson, TN. Andy is a Board Certified Orthopaedic Clinical Specialist and is a Certified Orthopaedic Manual Therapist through Maitland Australian Physiotherapy Seminars. He has additional training in Dry Needling, Instrument Assisted Soft Tissue Mobilization, and the Selective Functional Movement Assessment which he utilizes with his large caseload of foot/ankle conditions. Andy is the membership chair with the Chattanooga District of the TPTA. He and his wife, Callie, have three children: Tatum (4), Noah (2), and Knox (8 months).
1. True or False: Posterior tibial tendon dysfunction is the primary cause of adult acquired flatfoot deformity.

2. Actions of the posterior tibial tendon include all of the following EXCEPT:
   a. Foot adduction
   b. Plantar flexion
   c. Assisting with foot supination
   d. Foot abduction

3. Which stage of PTTD does not include foot/ankle deformity and/or tendon attenuation?
   a. Stage 1
   b. Stage 2
   c. Stage 3

4. True or False: In strength models, concentric contractions generate 20-60% greater force than eccentric contractions.

5. True or False: Histological changes associated with tendinopathy support the primary inflammatory model of tendinopathy.

6. Which of the following are potential contributors to excessive foot/ankle pronation:
   a. Stance phase pelvic drop
   b. Rearfoot varus (open chain rearfoot position assessed prone)
   c. Forefoot varus (open chain forefoot position assessed prone)
   d. Tibial varum
   e. Stance phase knee valgus, hip internal rotation motor control impairment
   f. Limited great toe extension
   g. Limited talocrural dorsiflexion

7. True or False: Surgical gastrocnemius recession can be performed when equinus deformity is not present in an individual with PTTD undergoing a corrective procedure.
Tibialis Posterior Tendon Dysfunction
An Adult Acquired Flatfoot Deformity

C. Jason Wamack, DPM
Diplomate, American Board of Podiatric Surgery
Fellow, American College of Foot and Ankle Surgeons

Tibialis Posterior Tendon Dysfunction

- Tendon degeneration/tendonopathy
  - Tenosynovitis, Tendonitis, tendonosis
- Painful, progressive pes valgo planus deformity
- Typically middle aged or older patients
  - Women > Men
- Risk factors: DM, obesity, steroid treatment, previous surgery, or trauma

Anatomy and Biomechanics
Anatomy

**Deep posterior compartment**
- Muscular origin
  - Proximal 1/3 of leg
  - Adjacent posterior tibia and fibula
  - Interosseous membrane
  - Deep transverse intermuscular septum
- Musculotendinous junction
  - Medial side of muscle
  - Distal 1/3 of leg superior to the medial malleolus
- Within sheath, courses distal-medial deep to FDL, enters medial malleolar sulcus & the first compartment of the flexor retinaculum

**Insertions** (divides just proximal to navicular tuberosity)
- Anterior slip (main): nav tuberosity and inferior medial cuneiform
- Middle slip: inter and lat cuneiforms, cuboid, 2-5th met bases
- Posterior slip: anterior surface of sustentaculum tali

**Ligamentous complexes of tibialis posterior tendon**
- Dorsally: inf band of int extensor retinaculum & talo-nav lig
- Medially: plantar calcaneo-nav lig & tibio-nav lig, fibrous septae, medial plantar fascia, deep fascia, & fibrocartilage navicularis
- Plantarily: deep fascia, tarsometatarsal ligaments, & peroneus longus tendon fibrous slips

**Innervation:** Tibial nerve
- Primarily L5
- Some L4

**Blood supply to tendon:**
- Prox: post tibial via mesotenon & visceral layer of sheath
- Distally: long vessels from epitenon, communication with periosteal blood vessels from med plantar br of post tibial & med tarsal artery
Anatomy

- **Watershed area**: zone of hypovascularity in central portion of tendon where mesotenon is absent.
  - 40 mm from nav tuberosity and extends proximally 14 mm

Biomechanics

- Plantarflexes and inverts the foot, adducts the forefoot
  - Most powerful supinator
  - Opposes peroneus longus tendon
- Primary dynamic supporter of medial longitudinal arch
- Acts on 3 **functional joints**:
  - tibiotalar, subtalar, & midtarsal
- Functions during the **stance phase** of gait
  - **Heel contact**: (eccentric contraction) shock absorber; decelerates STJ pronation, decelerates forward momentum & int rotation of tibia
  - **Midstance**: (concentric contraction) **rigid lever**; STJ inversion locking the transverse tarsal joints supporting the medial longitudinal arch
  - **Propulsive phase**: Accelerate STJ sup, assist in heel lift

Pathomechanics of TP Tendon Dysfunction

- Excessive pronation & ligamentous stretching
  - Stimulates local proprioceptors causing reflex muscular contraction, leading to muscle fatigue and further stretching and weakening of the ligamentous complexes
  - Forefoot abduction at talonavicular joint
  - Increased hindfoot valgus
  - Lateralized gastrocsoleus complex (pronatory force on RF)
  - Decreased tarsometatarsal joint stability
  - Loss of 1st ray stability
  - Adduction and plantarflexion of talus on calcaneus & dorsolateral articulation of navicular on talar head
  - Loss of ligamentous support→decreased medial arch height (example: elongated/disadvantaged spring ligament)
  - Tension on deltoid lig→weakens deltoid→valgus talus tilt
  - Apropulsive gait (less lateral column load, delayed heel lift)
  - DJD
  - Pain
Pathology
- Pathological elongation/attenuation of TP tendon
  - As little as 1 cm significantly reduces its efficiency
- Degenerative tenosynovitis, tendinitis, tendinosis
  - Hypoxic degenerative tendinopathy
  - Disrupted collagen bundle structure & organization
  - Mucinous degeneration
  - Tendolipomatosis
  - Calcinifying tendinopathy
  - Vascular hyperplasia
  - Tendon Sheath hypertrophy
  - Fibroplastic proliferation
  - Fibrocartilage metaplasia
- Nonspecific reparative process to tissue injury
- Recent studies show lack of histological evidence of inflammation (tendinosis vs tendinitis as primary pathology)

Etiology
- Not really known in most cases, probably multi-factorial
- Post traumatic/surgical inflammation
- Previous steroid treatment → collagen degradation
- Obesity → overuse, excessive strain on tendon
- Age (over 35) → wear and tear degeneration
- Hypovascularity → hypoxic degenerative tendinopathy
- Systemic factors
  - DM, HTN → collagen derangement & vascular disease
  - Seronegative arthropathies
  - Rheumatoid arthritis
  - Ehlers Danlos syndrome
  - Humoral autoimmune response
- Xanthoma, tumor, ganglion
- Anatomic → tight flexor retinaculum, shallow sulcus
**Etiology**

- Congenital anatomic variants
  - Duplication of TP tendon within its sheath
  - Common sheath for TP and FDL
  - Accessory navicular bone
  - Prominent navicular tuberosity
  - Congenital pes planus → chronic stress on TP tendon

- Mueller's etiologic classification of TP dysfunction
  - Type I → direct injury
  - Type II → pathologic rupture associated with inflammation
  - Type III → rupture of unknown origin
  - Type IV → functional rupture w/ dysfunction w/o complete rupture

**Clinical Presentation**

- Pain and swelling along the course of the tendon
- Fatigue and aching on plantar-medial foot and ankle
- Previous feeling of a snap or “giving way”
- "My toes are pointing out"
- Decreased or collapsed medial longitudinal arch
- Difficulty walking on uneven surfaces
- Pain on WB after a period of rest
- Abnormal medial shoewear pattern
- Bunion and hammertoes
- Heel pain/Plantar fasciitis
- Hyperkeratotic midfoot lesions
- Tarsal tunnel entrapment symptoms
- Difficulty or inability to stand on toes
- Possible knee pain
- Lateral foot pain in progressed deformities
Diagnosis

- Clinical exam
  - Appearance: too many toes sign, pes valgo planus, medial swelling, talar head prominence, deformities
  - Palpation: Check for joint crepitus and passive ROM
  - Strength and ROM: put foot in plantarflexion and inversion and have patient hold that position against resistance
  - Single limb heel rise test: heel inversion normal
  - Toe raising test of Jack
  - Trunk twist test: heel inversion normal
  - Hubscher maneuver: heel inversion w/ hallux DF is normal
  - Gait evaluation: apropulsive, abducted, antalgic, hyperactivity of tibialis anterior and/or the digital flexors, rearfoot eversion, bulging of TP tendon
Diagnosis

- Radiographic (AP, Lat, Ankle mortise)
  - AP: abduction of forefoot at transverse tarsal joint (inc cuboid abduction on calcaneus), navicular lateral on talar head, inc talocalc angle
  - Lat: dec talometatarsal angle, medial column sag, sinus tarsi covered by lat process of talus, inc talocalc angle, inc talar declination, dec calc declination
  - Ankle mortise: Valgus talar tilt, ankle arthritis
  - Advanced cases: DJD (subchondral sclerosis, osteophytic lipping, & joint space narrowing)
- Tenograms: can be difficult and little diagnostic value
- Ultrasound: hypoechoic regions w/ irregular thickened margins
MRI
Good for surgical planning, excellent soft tissue detail
Evaluate tendon girth, shape, fluid accumulation, and intratendinous signal changes

Double heel rise test
Abnormal on right
Abnormal on left

Too many toes sign
Abnormal on right
**Classification & Clinical Presentation**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Tendon Condition</th>
<th>Pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Paratendoscopic transection/tendon tear</td>
<td>Degeneration</td>
</tr>
<tr>
<td>II</td>
<td>Degenerated tendon</td>
<td>Degeneration</td>
</tr>
<tr>
<td>III</td>
<td>Tendon rupture</td>
<td>Degeneration, complex rupture</td>
</tr>
<tr>
<td>IV</td>
<td>Tendon rupture</td>
<td>Degeneration, complex rupture</td>
</tr>
</tbody>
</table>

**Conservative Treatment**

- Rest, Ice
- NSAIDs
- Compression stockings and braces
- Orthotics → UCBL, AFO
- CAM Walker
- Cast immobilization → 6 to 8 wks, followed by physical therapy to strengthen leg muscles
**FDL Augmentation of TP Tendon**
- FDL passed through a 4.5mm drill hole in the navicular from inferior to superior
- Hold the forefoot inverted and suture FDL back onto itself

**FDL Tendon Transfer**
- FDL passed through a 4.5mm drill hole in the navicular from inferior to superior
- Hold the forefoot inverted and suture FDL back onto itself

**Cobb Procedure**
- Medial incision, synovectomy and shortening of TP tendon
- Identify TA tendon 5cm above ankle joint, 1cm incision
- Transect medial half, then use tendon stripper to divide tendon distally to medial cuneiform, leave medial half attached
- Tunnel free end to plantar nav-cun articulation and weave it together with the TP tendon
Koutsogiannis
- Oblique incision 2cm distal to lateral malleolus
- Calcaneal osteotomy at 45° to the sole of the foot
- Distal segment displaced medially 1cm
- Fixation by 6.5mm partially threaded, cannulated screw

Evans

Silver

Dwyer
Severe Dysfunction (Stage IV)

- Triple arthrodesis
- Pan talar fusion
Objectives

• Discuss the continuum of clinical presentations of individuals with PTTD
• Discuss clinical and histological differences in stages of PTTD
• Review clinical and biomechanical exam for individuals with medial ankle pain
• Provide evidence and rationale for conservative management of PTTD
• Discuss treatment of PTTD to include concepts of regional interdependence and cortical motor control changes associated with tendinopathies
• Discuss surgical approaches for management of PTTD and relevant post-operative considerations.

Overview of PTTD

• Primary cause of acquired flatfoot deformity in adults
• Progressive condition
  • Wide continuum: localized tenosynovitis to complex rigid foot/ankle deformity
### PTTD

#### Demographics
- Female>male
- Middle age>elderly most common
- Obese
- HTN
- DM
- Others:
  - Treatment with steroids
  - History of medial foot/ankle trauma
- Younger:
  - Higher incidence of HLA-B27 positivity

#### PTTD: A Continuum
- Source of pain and potentially significant morbidity, loss of mobility
- Loss of dynamic stabilizers→Loss of static stabilizers→structural changes

### Management of PTTD
1. Understanding of lower quarter biomechanics, arthrokinematics
2. Reliable clinical lower quarter examination skills
3. Appreciation of tendinopathy continuum
4. Appreciation of appropriate orthotic intervention
5. Knowledge of exercise prescription

### Differential Diagnosis
- FHL tendonitis
- Tarsal coalition
- Underlying RA
- Fracture of sustentaculum tali
- Stress fracture
Medial Longitudinal Arch

- Passive Support
  - Plantar fascia
  - Spring Ligament (Calcaneonavicular ligament)
  - Long & Short Plantar Ligaments
- Dynamic Stabilizers
  - Posterior Tibial Tendon
  - Intrinsic(s)

Posterior Tibialis

- **Origin**: posterior intersosseous membrane, superior 2/3 of medial posterior fibula, superior aspect of posterior tibia
- **Insertion**: splits after spring ligament → navicular, medial cuneiform, MT 2-4
- **Action**: principal invertor, adducts foot, PF ankle, control supination

Posterior Tibialis

- **Concentrically** → Propulsion, heel rise
  - Controls RF motion to allow efficient plantarflexion with gastroc/soleus activation
- **Synergistic frontal plane control with peroneals**
- **Eccentrically** → Shock Attenuation
  - Controls rearfoot and dispersion of GRFs during landing/loading
Functional Complaints Associated with PTTD

• Difficulty negotiating uneven ground
• Difficulty descending stairs
• Difficulty with dynamic balance activity
• Pain associated with these activities

Joints of Foot, Ankle

• Tib/Fib:
  • Stable syndesmosis
  • Allows frontal plane motion in DF
• Talocrural:
  • Normal ROM: 10-20 deg DF, 30-50 deg PF
  • Axis: 20-30 deg post to frontal plane and 10 deg inf

Subtalar Joint

• Normal ROM:
  • 20-30 deg Inv
  • 10-20 deg Ev
• Axis: 42 deg sup from TV plane and 16 deg med from sagittal plane
  • 1:1 ratio of TV to frontal plane movement
  • Triplanar motion
• Pronation: DF, abd, ev
• Supination: PF, add, inv
• STJ: Guides shock absorption (pronation) and torque conversion (supination) phases of gait
• PTTD: Impaired shock absorption and/or torque conversion capability/tolerance
Subtalar Joint Axis

Midfoot Locking

- Midfoot Axes:
  - Oblique Midtarsal Joint (OMTJ)
    - Calcaneocuboid Joint: AKA Little Talocalcaneal Joint
    - Action: MF PF/DF
  - Longitudinal Midtarsal Joint (LMTJ)
    - Talonavicular Joint: AKA Little Subtalar Joint
    - Action: MF Sup/Pron
  - Pronation: Midfoot Axes Parallel
    - Mobile Adapter $\rightarrow$ Shock Attenuation
  - Supination: Midfoot Axes Cross
    - Rigid Leger

Biomechanics: Coupled Motions

- HF Inv:Tib Ext Rot
- HF Ev:Tib Int Rot
- Foot structure matters:
  - High STJ Axis (>45 deg): Tib IR=Ev (Ev:Tib IR < 1.0)
  - Low STJ Axis (<45 deg): Ev=TIB (Ev:Tib IR > 1.0)
  - Avg STJ 30-35 deg: greater Ev than Tib IR

Piazza, Foot Ankle Clin N Am, 2005
Gait

http://www.therapyedu.com/orth2/orth2-02.htm

Sequelae

- Progressive medial arch collapse
- Forefoot abduction
- Lateral impingement
- Heel cord adaptive shortening
- Spring ligament disruption
- Potential for deltoid ligament involvement
- Hallux valgus?
- Gait efficiency disruption!

Impairments Associated with PTTD

- Reduced great toe ext
- Reduced ankle DF and/or gastroc soleus mobility
- Impaired motor control of related joints (i.e. hip/pelvis)

http://www.myrehabspecialists.com/blog/3-Tips-to-Improve-Great-Toe-Mobility.html
Tendinopathy Continuum: Cook, Purdam

- **Recommended Reading:**
- Inaccurate model of tendinopathy: primary inflammatory model
- **Proposed:**
  - Continuum model: Stages not distinct
  - Continuum model: Tendon capable of back/forth movement in early stages of tendinopathy

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**Tendinopathy Continuum: Cook, Purdam**

**Reactive Tendinopathy**
- Non-inflammatory proliferative response, short-term adaptation to reduce stress; tendon copes with loads through thickening and stiffening
- Reversible
- Clinically: acute, younger, overload or blunt trauma
- Imaging: US – diffuse hypoechogenicity; MRI – minimal or no increased signal

**Tendon Dysrepair**
- Similar to reactive tendinopathy but with greater matrix breakdown; possible increase in vascularity and neuronal ingrowth (neovascularization)
- Imaging: US & MRI – increased matrix disorganization and swelling
- Clinically: chronically overloaded tendon; wide range of ages and loading environments; more thickened tendon and localized changes

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**Tendinopathy Continuum: Cook, Purdam**

**Degenerative Tendinopathy**
- Progression of matrix and cell changes
- Large areas of matrix disorganization, neovascularization, and breakdown
- Considerable heterogeneity between parts of tendon
- Imaging: US & MRI – extensive compromise
- Clinically: Often in older individual or athlete; often has experienced repeated bouts of tendon pain; can lead to rupture if allowed to progress
Tendinopathy
Continuum: Cook, Purdam

Histological changes
Mosier Foot Ankle Int, 1998
- Increased mucin content
- Hypercellularity
- Neovascularization
- Chondroid metaplasia
- Increased proportion type 3 vs type 1 collagen

Tendinitis?
Mosier et al
- Absence of inflammatory infiltrates despite clinical appearance of tenosynovitis during surgery
- Noted reduction in linear organization of collagen could reduce tensile strength and promote further attenuation
Tendinositis Model – Kulig & Arya

Repetitive stress on Tendon

Inadequate Repair

Decrease in tendon stiffness and mechanical strength

Increased Vulnerability to Injury

Repetitive stress on Tendon

Adequate Repair

Stage 1 PTTD

- Medial foot, ankle pain
- Potential for localized swelling
- Tender to palpation
  - Often tender posterior and superior to medial malleolus
  - May also be tender distal to medial malleolus, navicular
- Tenosynovitis or tendinosis
- No structural deformity
- Single leg heel raise: painful, able to perform
- Imaging: normal, typically not indicated

Stage 2 PTTD

- Medial foot, ankle pain
- Tender to palpation: near distal insertion of PTT
- Hypovascular zone: 14 mm proximal to navicular tuberosity
- Swelling potentially
- PTT attenuation or rupture
- Flatfoot deformity
- Flexible, reducible
- Single leg heel raise: < full elevation, unable to lock RF
Stage 3 PTTD

- Fixed nonreducible deformity
  - Minimal in/ex Available
  - Greater abd. rearfoot valgus
- Marked calcaneal valgus and midfoot abduction
- Potential lateral ankle pain from calcaneo-fibular abutment
- Markedly + heel rise test
- Surgical consideration

Stage 4 – Myerson & Corrigan

- Rigid or flexible flatfoot deformity with associated ankle involvement.
- Valgus talar tilt due to deltoid ligament failure → lateral tib-talar degeneration

“Normal”

- OKC: Calc 0-2 deg inverted (varus)
- CKC: Both plantar condyles and metatarsal heads in the same plane
  - CKC: Calcaneus slightly everted or rectus
Rearfoot Varus

- Calcaneus inverted with respect to lower leg (OKC)
- Gray et al: Most common osseous foot deformity
- Looks like neutral foot when fully compensated
- Problem: Foot partially pronated or pronated during propulsion
  - Creates difficulty stabilizing 1st ray

Forefoot Varus

- Medial forefoot inverted in relation to lateral forefoot. Forefoot also inverted relative to rearfoot.
- McPoil: FF varus most common foot deformity
- Calcaneus everts beyond neutral into everted (valgus) position
- Significant late stance pronation

Forefoot Valgus

- Medial aspect of forefoot inverted relative to lateral aspect of forefoot
- Forefoot everted relative to plantar condyles of calcaneus
Exam: Standing Static

- Resting calcaneal stance position (RCSP)
- Half squat
- Single leg stance
- Single leg stance with half squat
- Appreciate:
  - Rearfoot position relative to vertical
  - Distal leg to vertical
  - Too many toes sign?
  - Talonavicular “bulge”
  - Single leg: self-corrective strategies

Resting Calcaneal Stance Position and Half Squat

- Resting calcaneal stance position (RCSP)
  - Provides info on self-selected foot posture
  - Patient’s most common subtalar joint position
- Half squat
  - Provides info on maximum pronated position
  - Mimics loading/early midstance

Tibial Varum

- Frontal plane curvature or bowing of tibia places rearfoot in inverted position relative to supporting surface
- Measurement: standing leg bisection relative to true vertical
  - Normal: up to 4 degrees
  - Notable: 4-7 deg
  - Pathological > 8 deg
  - Implications: Requires ST, MT joints to pronate to bring medial border of foot to supporting surface—increased and late stage pronation

http://www.sportspodiatryclinic.co.uk/running.html
Exam: Rearfoot to Leg angle

- Bisect leg and rearfoot
  - RCSP
  - Half-squat

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Exam: Single Leg Stance

Single leg stance (balance):
- Qualitative assessment of balance:
  - Medial vs lateral excursion
  - Ability to self-correct
  - Use of UE for self-correction
  - Involvement of digits
    - Flaring (great toe vs digits 2-5)
    - "Clinging to the floor" (great toe vs digits 2-5)
  - Involved vs Uninvolved

Single leg stance half-squat:
- Provides similar info as half-squat but in unilateral position
  - UE assist allowed

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Exam: Heel Rise Test

- Involved to uninvolved
- Presence/absence of pain
- Comparison of heel height/excursion
- Calcaneal inversion
- Light UE assist allowable for balance
Exam: Supination-Pronation Test

- Used to predict suspected amount of subtalar motion that is normal for the individual
- Measures frontal plane motion of calcaneus to transverse plane motion of talus
- High axis = 2-4° of suspected eversion at maximum pronation, average axis = 4-6° of eversion, low axis = 6-8° of eversion
- Clinician palpates talar neck, calcaneus
  - Average axis: 1:1 talus:calcaneus
  - High axis: 2:1 talus:calcaneus
  - Low axis: 2:1 calcaneus: talus

Exam: Functional: Single leg squat

- Provides dynamic assessment of single leg control
- Influence of lumbopelvic control on foot/ankle position

Exam: Windlass Mechanism

- Test of great toe mobility
- Assessment of windlass mechanism to create rigid foot of osseous foot structures via passive tensioning of plantar aponeurosis
- OKC: >70 deg
- CKC: >30 deg
Standing Exam

- RCSQ
  - Measure leg to rearfoot
- Half-squat
  - Measure leg to rearfoot
- Distal leg to floor
  - Measure leg bisection→vertical
- SLS
- SLS with half-squat
- Supination pronation test
  - Motion: excessive, normal, insufficient
- Heel Rise Test
  - Pain? Full height? Calcaneus inverted?
- Single leg squat:
  - Qualitative assessment of kinematic chain and influence on foot/ankle (Vice versa)
- Other(s):
  - Windlass mechanism assessment
  - Toe walking (if heel raise normal)

STJN

- Prone: palpate talar head for congruency while passively inverting/evertg foot via 4th/5th MT then DF to 1st tissue stretch
  - Normal: 0-3 deg varus

Exam: Prone – DF ROM

- Measurement: in STJN position
- Axis: 1.5 cm distal to lat malleolus
- Move to a tissue stretch end feel
- Repeat with knee flexed
  - Gross: 10-20 deg
  - DF in STJN: 0-5 deg
Exam: Prone – Inversion, Eversion

- Measurement:
  - Bisect distal leg
  - Bisect heel
  - Axis at malleoli

Exam: Prone – HF to FF

- Reliability: Poor with goniometer
- Reliability: Good with visual estimation

Exam: Prone – 1st Ray Position and Mobility

- 1st ray: 1st MT, cuneiform, navicular
- Plantar flexed 1st ray: inferior to plane of MT 2-5
- If present: Rigid? Flexible?
- Flexible: functions like pronated foot

http://www.dynamicchiropractic.ca
Exam: Forefoot Mobility on Hindfoot

- Qualitative Assessment
- Assess mobility of mid/forefoot relative to position of rear foot:
  - Assess supination/pronation of LMTJ (talonavicular joint) with HF in inversion then eversion.
  - Assess PF/DF of OMTJ (calcaneocuboid joint) with HF in inversion then eversion.
  - For both LMTJ and OMTJ, motion with HF in eversion should be greater than inversion.
  - Can also simply assess PF/DF of entire mid/forefoot with HF in both Inv/Ev positions.
- Remember: position of HF guides locking of MF!

N=24 Runners (12 controls, 12 with stage 1 PTTD)
- Measurements: arch height index, maximum voluntary isometric invertor strength, 3D walking kinematics during walking for rearfoot and medial longitudinal arch

Rabbito et al – JOSPT 2011

- Findings:
  - Strength between groups equal
  - MLA measures between groups not significantly different
  - Eversion excursion similar not significantly different (6.6 deg vs. 5.9 deg)
  - Individuals with PTTD exhibited greater peak eversion during gait (6.0 vs 2.9) and longer time to peak eversion (45.8% of stance vs 38.1%)
- Summary:
  - Stage 1: no foot deformity however individuals with PTTD however there is greater rearfoot eversion for a greater % of stance versus healthy controls
- Clinical Implications:
  - Early orthotic intervention?
Rabbito et al – JOSPT 2011

- N=47 with stage 1 or 2 PTTD
- Prospective, observational
- Median age: 50 yo
- Median weight: 163 pounds
- Treatment:
  - AFO/Orthotic
  - Multimodal clinic and home exercise

Alvarez et al. – FAI 2006

- Pain > 3 months, inability to walk 1 block, or inability to perform heel raise
- Pain < 3 months, ability to walk 1 block and perform heel raise
- Treatment:
  - Custom Articulating AFO: Pain > 3 months, inability to perform single-supported heel raise (SSHR), inability to walk >1 block
  - ¾ TPE Orthotic: Pain < 3 months, able to perform SSHR, able to walk >1 block
  - AFO→shoe orthotic when strength deficits <10-15%, pain subsided
  - Clinic based and home exercise
Rehabilitation

Phase 1
- Isokinetic testing
- Home exercise
  - Side-to-side: 25 reps 4x/day → 12x/day by day 10
  - Red exercise band in, ev, DF
- Sole-to-sole: 25 reps 4x/day → 12x/day by day 10

Phase 2 = Visits 2-6
- Isokinetic strengthening
  - 30 deg/s, 60 deg/s sets 20-30 reps
- Double → single supported heel raise progressing to 50 reps
- Toe walking progressing to 100 yards

Phase 3 = Visits 5-7
- Isokinetic testing
- SSHR (assisted to unassisted) with Assessment (goal 50)
- Toe walking assessment

Outcomes:
- Median # of visits: 10 (3-17) over 4 months
- 42 of 47 “satisfied”
- 5 required subsequent surgery

Discussion:
- Conversion of AFO → shoe orthotic
- Availability of isokinetics
- Clinically significant conservative results
- Comprehensive in nature
- Lack of control group

RCT, Stage 1 or 2 PTTD > 3 months (N=36)

Groups:
- Orthosis + stretching (OO)
- Orthosis + stretching + concentric exercise (OC)
- Orthosis + stretching + eccentric exercise (OE)

Outcomes:
- Pain
- FFI
- 5-Minute Walk Test
### Tibialis Posterior: Activation

**Kulig et al:**
- CKC foot adduction barefoot most selectively activated PTT in individuals w/ normal arch
- CKC foot add in shoes most selectively activated PTT in individuals w/ pes planus

- Eccentric exercise: Force capabilities 20-60% greater
- Benefits of eccentric exercise established with Achilles, patellar, hamstring tendinopathies but yet to be established with PTTD

### “Tib Post Loader”

**Interventions**
- OC, OE groups
- 0.9-9 kg
- Horiz add, PF
- Required to maintain pressure in PF to prevent tib ant recruitment
- OC, OE groups: 3x15 reps, 2x/day, resistance progressed weekly
- Calf stretching

### Orthotics + Concentric Vs Eccentric Exercise

**Pain:**
- Statistically significant improvements in all groups – greatest in OE group
  - OE>OC>OO

**Foot Function Index**
- Statistically significant improvement across all groups – greatest in OE group
  - OE>OC>OO

No change in 5-Minute Walk Test distance traveled

- Previous work with Achilles tendinopathy supports improvement in structure and reduction in pain associated with eccentric activity
- Improvements in all categories
  - GREATEST WITH ECCENTRIC ACTIVITY WITH ORTHOTIC
JOSPT 2011

- N=34 (17 w/ PTTD, 17 controls)
- Measurements:
  - Single leg heel raise [PF strength]
  - Frontal and sagittal plane strength and endurance measured w/ dynamometry
  - Comparisons side-to-side and between groups

Kulig et al, JOSPT 2011

Kulig et al, JOSPT 2011
Kulig et al, JOSPT 2011

- Pelvic impairments
  - Did pelvic impairments precede the problem?
  - Cause the problem?
  - A result of the problem?
- Regional Interdependence must be considered when formulating a care-plan!

PTTD: Exercise

What we know about exercise:
- Concentric and eccentric strengthening improve pain and function in individuals w/ PTTD 1 & 2
  - Ecc > Conc – Kulig PTJ 2009
  - High reps, low resistance important – Alvarez, Marini FAI 2006
  - Exercise effective with orthotics

What we don't know about exercise
- Effect of exercise on tendon remodeling
- Exercise affects healing, remodeling in other tendinopathies (hamstring, patellar tendon, Achilles)
- Effect of isometrics
  - Isometric exercise induces analgesia and motor control in patellar tendinopathy – Rio et al BJS 2015
  - Effect of exercise without orthotics
  - Effect of addition of:
    - Innovo strengthening/motor control
    - Lumbo-pelvic strengthening/stabilization/motor control

PTTD: Orthoses

- Stage 1
  - Shoe-orthotic!
  - IDC – ie Powerstep or SuperFeet
  - Custom Orthotic – correct HF varus, FF varus, and/or FF L & Ray
    - Semi-rigid: Fastech
    - Rigid: TPE
    - UCBL – greater HF control
- Stage 2
  - Custom Orthotic
  - UCBL
  - Articulating AFO – Tri-planar control
- Stage 3
  - Accommodative Orthotic – Articulating AFO (fixed deformity)
PTTD Orthoses

- Non-custom
- Custom Semi-rigid
- Custom TPE

**Beyond the distal leg.....**

**Regional Interdependence**

- How do we assess?

  - **SFMA** – Selective Functional Movement Assessment:
    - 1) Joint mobility dysfunction (JMD) or Tissue Extensibility Dysfunction
      - Ex: limited talocrural mobility (JMD) or shortened gastroc/soleus (TED)
    - 2) Stability/Motor Control Dysfunction (SMCD)
      - Ex: varus/valgus collapse

- Mike Boyle/Gray Cook

  - Body series of alternating mobile and stable joints
    - Mobile joint: moves in >1 plane of motion (ankle, hip)
    - Stable joint: moves primarily in 1 plane of motion (Great Toe)

- Necessary mobility must be present prior to restoring normal motor control
Regional Interdependence

What movement impairments potentially affect PTTD?

- JMD/TED – Joint Mobility Disorder/Tissue Extensibility Disorder
  - Many potentially
  - Limited great toe ext
  - Limited ankle DF @ TC
  - Limited HF Ev (Requires compensatory MF pronation)
  - Limited ankle DF via gastro/soleus extendibility
- SMCD
  - Many potentially
  - Impaired rearfoot control
  - Dynamic knee collapse (knee valgus/hip IR)
  - Impaired lumbopelvic control (pelvic drop during stance)

Causes of Excessive Foot Pronation

- Foot related factors
- Hip related factors
- Shoe related factors
- Activity, training related factors

Foot Related Causes of Excessive Pronation

- Limited ankle DF
  - Compensatory midfoot break
- Hypomobile STJ
  - Compensatory midfoot motion
- Hypermobile rear/midfoot
  - Excessive eversion
  - Invertor weakness or motor control deficits
- Rearfoot and/or forefoot varus
  - Increased excursion
Hip Related Causes of Excessive Pronation
- Medial Collapse
  - Components of medial collapse: knee add, tib IR
- Pelvic Drop

Shoe Related Causes of Excessive Pronation
- Shoe Related Causes of Excessive Pronation
  - Excessive lateral flare
  - More relevant for active, running population

Activity or Training Causes of Excessive Pronation
- Activity
  - Surface (Ex: concrete, unstable surface)
  - Exposure (Ex: Prolonged standing activities)
- Training/Running:
  - Toe out pattern → increased medial tensile stress
  - Midfoot/forefoot strike pattern
    - Increased Post-tib, gastro demands vs RF strike
    - Cross-over contact

Toe-out & Foot Pronation
- Causes:
  - Ext tib torsion
  - Femoral retroversion
  - Compensation for femoral anteversion
  - Limited hip IR ROM
  - Limited DF
    - Limited 2nd, 3rd rocker
Motor Control: Glute Max
The Tri-Planar Muscle

- Action: Extension, abduction, external rotation

Frontal Plane Control
Tri-planar Control

Motor Control: Lumbo/Pelvic/Hip

- Lumbopelvic, “Core” control is reflexive, subconscious.
- Reactive Neuromuscular Training

Don’t forget about me!

- Origin, insertions of intrinsics→stabilization capacity
- Shod society→underutilization of foot intrinsics
- Capacity for “re-training”
- Reference: Irene Davis, PT, PhD
Intrinsic Training

**Short Foot (Foot Doming)**
- "shorten" or dome foot keeping heel and forefoot on ground, avoid curling toes
- Seated/Standing (Unilat) Threading (Unilat) Threading with domed foot

**Toe Spreading (spreading)**

**Wall Leans (Nose Reaches)**
- Barefoot, controlled anterior trunk/body sway near wall
- Bilat ➔ Supported Unilateral ➔ Unilateral
- Heel Raises – Barefoot

Irene Davis: Unpublished Study: Promising ability to train intrinsics in adults (non-PTTD population)

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"Current tendon rehabilitation may fail to adequately address the multitude of contributing factors to altered motor control, which would include not only muscle strength and tendon capacity, but corticospinal control encompassing excitability and inhibition as well as belief systems about pain and contextual factors."

**Tendinopathy Motor Control Research:**
- Motor control changes noted bilaterally
- Implications: Changes to motor patterns

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• How does current rehabilitation address motor control deficits in individuals with tendinopathy?
• Individuals with tendinopathy (not specific to PTTD) exhibit:
  - Greater cortical inhibition than controls
  - Greater corticospinal excitability than controls


“Current tendon rehabilitation may fail to adequately address the multitude of contributing factors to altered motor control, which would include not only muscle strength and tendon capacity, but corticospinal control encompassing excitability and inhibition as well as belief systems about pain and contextual factors.”

Tendinopathy Motor Control Research:
- Motor control changes noted bilaterally
- Implications: Changes to motor patterns
Current Tendon Rehabilitation and Proposed Concept

- Jill Cook: “Once a tendon, always a tendon”
- Achilles Tendinopathy Research:
  - Evidence for heavy, slow concentric and eccentric contractions improving pain and function
- Current Tendinopathy Rehabilitation
  - Self-paced: pt guided in sets, reps, load without external auditory or visual pacing
  - Current tendinopathy rehabilitation may fail to restore corticospinal control of the muscle-tendon complex

External Pacing in Tendinopathy Rehabilitation

- Prior Research: In health individuals, external pacing (metronome or visual stimulus) capable of changing excitability and inhibition both ipsilaterally and contralaterally
- Research protocol:
  - Tendon Neuroplastic Training (TNT) → Externally paced strength training
  - Heavy, Isometric or isotonic strength training with external pacing
  - N=9 with patellar tendinopathy
  - Length of intervention: 4 wks

Tendon Neuroplastic Training
Tendon Neuroplastic Training

- Conclusion: Reduction in cortical inhibition changes for individuals with tendinopathy versus those without tendinopathy same.
- Conclusion: Significant analgesic effect of isometric contractions.

My Treatment Toolbox

Modalities:
- Acute/Rehabilitation
- Exercise/Strength Training
- Invertor, Plantarflexor High Rep, Low Resistance with regular progression (avoid Tib Ant substitution)
- Resisted Sidelying Eversion w/ eccentric emphasis (Ankle Isolator)
- Isokinetic strengthening: invertor conc, ecc; high reps, multiple speeds
- Isometric inversion, PF: long hold, low repetition for analgesic effect
- Isometric strengthening, Task Directing (Plantarflexion, Tibialis Ant.)
- Lumbopelvic strengthening: OCK, CKC (biofeedback w/ CKC)
- Metronome with HEP (bands)
- Bilateral Pain-free heel raises: Bilat w/ UE assist (biofeedback – ball between heels for calc inversion); Alter-G body weight supported heel raises
- Toe Walking

Orthotics:
- No deformity: Shoe recommendations, taping, medial felt posting, Ankle brace/ASO, OTC orthotics, OR semi-rigid orthotics if acute, activity induced PTTD
- Deformity or recurrent: Semi-rigid, ¾ TPE custom, short articulating AFO

Motor Control – Balance Training with and without shoes:
- Alter-G body weight supported single leg stance with video feedback
- Upper Extremity RNT w/ balance training – emphasis on maintaining intrinsic activation

Taping:
- Plantar flexion

IASTM:
- Never directly to tendon
- Intermediately to fascia, muscle to reduce tone, improve extensibility

Mobility:
- CKC DF Mobs
- Protected Gastroc Stretch (Toe → Opposite Heel)
- Mulligan DF Mobs (CKC)
- Talar AP mobs in STJN
- Great toe extension stretches, mobilization

PTTD: Response to Conservative Intervention

- Largely "successful" in individuals with stage 1 or 2 PTTD
- Variable reports: up to 85%
- Retrospectively: Split tear of PTT associated with failed conservative intervention
- Compliance with conservative care challenging
- Post-op communication crucial!
References


• Inman V. The Joints of the Ankle. Baltimore, MD: Williams & Wilkins; 1976.


Posterior Tibial Tendon Dysfunction

Post-Test

1. True or False: Evidence supports the effectiveness of conservative management in the majority of individuals with stages 1 and 2 PTTD.

2. Kulig et al reported the greatest reductions in pain and self-reported functional limitations in individuals with state 1 or 2 PTTD with:
   a. Orthotics only
   b. Orthotics + concentric exercise
   c. Orthotics + eccentric exercise

3. Histological changes associated with tendinopathy include all of the following EXCEPT:
   a. Increased mucin content
   b. Hypercellularity
   c. Neovascularization
   d. Chondroid metaplasia
   e. Increased proportion type 3 vs type 1 collagen
   f. Presence of significant amount of inflammatory substances within and around the tendon

4. True or False: Tendinopathy research indicates that individuals with tendinopathy exhibit increased cortical inhibition and greater corticospinal excitability in individuals versus controls suggesting neuroplastic changes associated with tendinopathy.

5. True or False: Middle age females with PTTD did not exhibit any changes in proximal hip or pelvic strength capacity or endurance as compared to controls.

6. Common changes in the foot/ankle associated with the progression of PTTD include:
   a. Lateral impingement symptoms associated valgus changes
   b. Adaptive Achilles/gastrocnemius shortening
   c. Increased midfoot mobility with compensatory forefoot abduction
   d. Altered foot pressures and resultant callous formation
   e. Disruption of the spring ligament from talar adduction and plantarflexion

7. Surgical procedures performed for PTTD include which of the following (circle all that apply):
   a. PTT tenosynovectomy
   b. PTT repair
   c. FDL transfer
   d. Lateral column lengthening
   e. Calcaneal slide osteotomy