Sports medicine & the athlete

“ITIS” versus “-OSIS”

CAN YOU GET THERE FROM HERE

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Disclaimers/Disclosures

- Nothing to disclose
- No monetary remuneration
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- No royalties

Learning Objectives

- Distinguish between types of tendinopathies
- Understand the pathophysiology of enthesopathy
- Apply knowledge gained to practice in the field
RISK versus REWARD

- **Success** is predicated on a precarious physiologic and psychological balance easily tipped by the slightest touch of adrenaline.

- **Failure** is manifested by under-preparation coupled with compromised conditioning imperceptibly mixed with the intangible “X-factor” of chance.

TRIUMPH

**You can't succeed unless you're willing to fail.**

- **Success**
  - Individual
    - Preparation
      - Physical
      - Emotional
      - Psychological
  - Group
    - Cooperation
    - Support
    - Sacrifice

- **Failure**
  - Individual
    - Preparation
      - Physical
      - Emotional
      - Psychological
  - Group
    - Cooperation
    - Support
    - Sacrifice

Accomplish the task

FAILURE

- **Mechanisms**
  - Psychological
  - Emotional
  - Physical

- **Mechanisms**
  - Physical
    - Anatomical
    - Functional
    - Structural
    - Mechanical
    - Elemental
Focus on a concept

- Identify an injury profile that we can:
  - Understand
  - Define
  - Deconstruct
  - Reconstruct

- All for the singular purpose of:
  - Effecting positive change...

Bright idea

- Common things are common
  - Seemingly...
    - Routine
    - Mundane
    - Anticipate positive outcome

- Difficult things are difficult
  - Sometimes...
    - Challenging
    - Provocative
    - Compromised results

Enthesopathy

Tendinopathy

- Tendinitis
  - Reactivity with inflammatory response
  - Acute (or chronic) overload
  - Intrinsic or extrinsic factors

- Tendonosis
  - Damage at cellular level
  - Microtears in connective tissue leading to increase in repair cells
  - Chronic degeneration without inflammation
ACHILLES

- Greek hero of the Trojan War
- Mother nymph Thetis, father Peleus king of the Myrmidons
- Thetis attempted to make Achilles immortal by dipping him in the river Styx, but left vulnerable by the part she held him by: his heel
- Killed by Paris at end of Trojan war who shot him in the heel with an arrow

ACHILLES' HEEL: POINT OF WEAKNESS

TENDONITIS

- Acute inflammatory response to load
  - Hallmarks
    - Pain
    - Swelling
    - Stiffness
    - Warmth
    - Limited ROM

TENDONITIS

Normal structure

- Macroscopic
- Microscopic
TENDONITIS

ABNORMAL STRUCTURE
- MACROSCOPIC
- MICROSCOPIC

MECHANISM OF INJURY

LOAD TO FAILURE
- CYCLIC LOADING
  - Force couples
  - Velocity of motion
  - Intensity of motion
  - Ambient parameters
    - Temperature
    - Stiffness
- REPETITION
  - Flexion
  - Extension
  - Tension
  - Compression
  - Stress/strain curve

MECHANISM OF INJURY

FORCE AND LENGTH (MUSCLE PHYSIOLOGY)
- ISOMETRIC
  - Generate tension without changing length
    - Tense a muscle to hold in position
- ISOTONIC
  - Constant tension with change in length
- ISOKINETIC
  - Constant velocity while force changes
**Mechanism of Injury**

**Force and Length (Muscle Physiology)**

- **Concentric**
  - Isotonic
  - Length shortens as it contracts

- **Eccentric**
  - Isotonic
  - Loading while lengthening

**Mechanism of Injury**

**Acute**

**Chronic**

**Phased Healing Response**

**Spectrum**

- Hemostasis
- Inflammation
- Proliferation
- Maturation
Phased healing response

- Hemostasis
  - Hematoma stimulates platelet aggregation, activating fibrin with stasis of injury site…

- Inflammation
  - White blood cell phagocytose debris and necrotic material…
  - Platelet-derived growth factors (PDGF’s) released, stimulating migration and division of cells…

Phased healing response

- Proliferation
  - Angiogenesis
  - Collagen deposition
  - Granulation tissue
  - Contraction

- Maturation
  - Remodeling
  - Realignment
  - Tissue strengthening

Transition

“ITIS”

“OSTE”
Tendonosis

- Chronic degenerative change without inflammation
  - Hallmarks
    - Pain
    - Limited ROM
    - Recalcitrant recovery

“The Chronic”

Normal/abnormal structure

- Macroscopic
- Microscopic

Mechanism of injury

Load to failure

- Cyclic loading
  - Abnormal tissue subjected to normal stress/strain load
  - Altered response and recovery

- Repetition
  - Compromised tissue characteristics leads to progressive structural malfunction
  - Compensatory recruitment of surrounding structures
**Phased Healing Response Spectrum**

- Hemostasis?
- Non-inflammatory cellular response
- Proliferation?
- Maturation?
- Microtears in connective tissue at the cellular level

**Healing Response Mechanism**

**Inflammatory**
- Vascular component
  - Vasodilation
  - Membrane permeability
  - Plasma cascade system
  - Plasma-derived mediators
- Cellular component
  - Leukocyte extravasation
  - Cell-derived mediators

**Non-inflammatory**
- Degenerative changes in collagenous matrix
- Disorganized hypercellularity
- Hypervasularity
- Lack of inflammatory cellular response

**Healing Response**

**Non-inflammatory**
- Blocks “normal” reparative process
  - Inhibits functional reorganization of tissue from microscopic to macroscopic regeneration
  - “Abnormal” tissue with compromised response to normal tissue recovery
Can we effect change?

Modulate or enhance the cycle

- **Proactive**
  - Attempt to “pre-treat” a condition that does not yet exist

- **Reactive**
  - “Damage control” approach in response to an insult

Propose a combination of both

What works

**Proactive**

- **Muscle-tendon unit**
  - Stretch: optimize mechanical advantage
  - Strength: maximize power through arc of motion
  - Condition: prepare for physical challenge

**Reactive**

- **Muscle-tendon unit**
  - Control zone of injury to limit extent of damage
    - Support/protect
    - Modalities
    - Rehabilitate

What doesn’t work

**Incomplete rehabilitation**

- Restricted ROM
- Persistent pain
- Swelling
- Stiffness
- Muscle atrophy
- Compromised proprioception
- Inadequate/unreasonable timeframe
- Inappropriate “ramp-up” of return to play criteria
MODALITIES

**TENDONITIS**

- **Basic**
  - RICE
  - Thermotherapy
  - Cryotherapy

**TENDONOSIS**

- **Advanced**
  - Ultrasound
  - Iontophoresis
  - Phonophoresis
  - Active Release Therapy (ART)
  - Graston technique
  - Muscle energy

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ON THE HORIZON

**“IN YOUR HANDS”**

- Foundation techniques that are effective but “more of the same”

**“ALMOST THERE”**

- Realistic techniques based on scientific principles that view things from an advanced perspective

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MODALITIES

**STATE OF THE ART**

- Eccentric loading
  - Lengthening muscle/tendon contractions under load
  - Optimize excursion of functional unit over anatomic distance

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MODALITIES

State of the Art

• Soft Tissue Mobilization

 Augmented Soft Tissue Mobilization (ASTM) is a form of manual therapy that has been shown in studies on rats to speed the healing of tendons by increasing fibroblast activity.


Gale M. Gehlsen, "Fibroblast responses to variation in soft tissue mobilization pressure," Medicine & Science in Sports & Exercise, vol. 31, no. 4, pp. 531-535.

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• Shock Wave Therapy

In rat subjects, SWT increased levels of healing hormones and proteins leading to increased cell proliferation and tissue regeneration in tendons.

   

• Vitamin E

Vitamin E has been found to increase the activity of fibroblasts, leading to increased collagen fibrils and synthesis, which seems to speed up the regeneration and increase the regenerative capacity of tendons.

**Modalities**

**State of the art**

- **Nitric oxide**

Nitric oxide (NO) appears to play a role in tendon healing and inhibition of its synthesis impairs tendon healing. The use of a nitric oxide delivery system elicits tendon-patches applied over the area of injury. Tendonitis was tested in three clinical trials for the treatment of tendinopathies and was found to significantly reduce pain and increase range of motion and strength.


- **Autologous tenocyte injection**

The autologous tenocytes were sorted and purified by real-time polymerase chain reaction and amplified by flow cytometry. The tenocytes were then injected into the injured tendinopathy, which was the origin of the extensor carpi radialis brevis tendon, under the guidance of an ultrasound. After the autologous tenocyte injection treatment, patients with chronic lateral epicondylitis showed improved clinical function and structural repair at the origin of the common extensor tendon.


- **Ultrasonic percutaneous tenotomy**

Ultrasonic percutaneous tenotomy provided continued pain relief and functional improvement for recalcitrant tennis elbow at a 3-year follow-up.

MODALITIES

STATE OF THE ART

- **ACUSTOElastography Ultrasound**

  Acoustoelastography: ultrasound technique that relates ultrasonic wave amplitude changes to the mechanical properties of a tendon. This ultrasound-mediated model can be used to evaluate tendon function.


- **Nonbulbar Dermal Sheath Cells**

  Clinical trial using fibroblasts isolated from nonbulbar dermal sheath cells of hair follicles. The tendon treatment will be tested in approximately 28 subjects. Nonbulbar dermal sheath cells used because they produce more type I collagen than fibroblasts that are derived from adipose tissue. Type I collagen is the primary collagen in tendons. Nonbulbar dermal sheath cells will be replicated, then reintroduced into wounded tendons with ultrasound.

  Safety and Efficacy of RCT-01 in Men and Women With Unilateral, Chronic Achilles Tendinosis (ReACT) In: ClinicalTrials.gov [Internet]. Last updated: March 12, 2015.

- **Tendon Bioengineering**

  The future of non-surgical care for tendinosis is likely bioengineering. Ligament reconstruction is possible using mesenchymal stem cells and a silk scaffold. These same stem cells were capable of healing repair of damaged animal tendons.


  Long JH, Qi M, Huang XY, Lei SR, Ren LC (June 2005). “Repair of rabbit tendon by autologous bone marrow mesenchymal stem cells”.

**MODALITIES**

**STATE OF THE ART**
- **Micro-RNA injection**

  A trial will put injections of microRNA—small molecules that help regulate gene expression—into the tendon to decrease the production of type 3 collagen and switch to type 1.


**Damage done...**

**Compromised tissue**
- Can we take that altered environment and reorganize, reorient, regenerate the macro & microstructure to enable "normal" function...

**Endstage**

**Rupture**
- Surgical option
...DAMAGE UNDONE

FUNCTIONAL UNIT

- Ability to apply techniques at the cellular level that may enable the seemingly impossible to become possible...

SUCCESS

RETURN OF FUNCTION

MAYBE

THESE STUDIES ARE SHOWING PROMISE

- Improved understanding of micro-structural elements
- Identifying what we can actually enhance

REAL-TIME & ROUTINE APPLICATION OF PRINCIPLES

- Applicable to on-the-field or training room environment
- Knowledge base for user competency
CONTINUUM

- WE CAN GET THERE FROM HERE
- WE JUST NEED A LITTLE DIRECTION

TAKE AWAY

THE FUNCTIONAL UNIT

RISK VERSUS REWARD

- THE VICTOR
- THE GOAT
THANK YOU