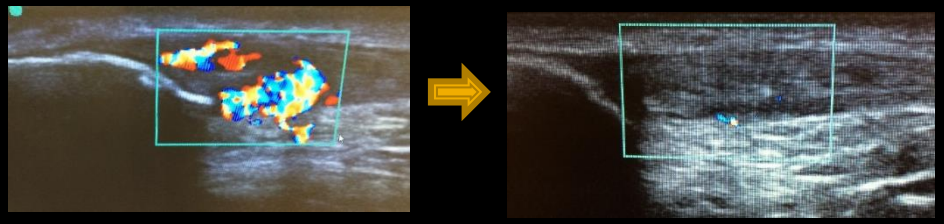
Update on Patellar Tendon Injuries: Pathophysiology and treatments



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Andrews Sports Medicine and Orthopedic Center

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Disclaimer

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- Speaker and grants from Smith & Nephew.
- Faculty at the Regenerative Medicine Training Institute

Objectives

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- Tendon Anatomy
  - Function
  - Metabolism
  - Biomechanics
- Patellar Tendinopathy
  - Overuse Theory
  - Mechanical Deficits
- Treatment options
  - Acute vs Chronic
  - Conservative and surgical options
  - Role of Orthobiologics

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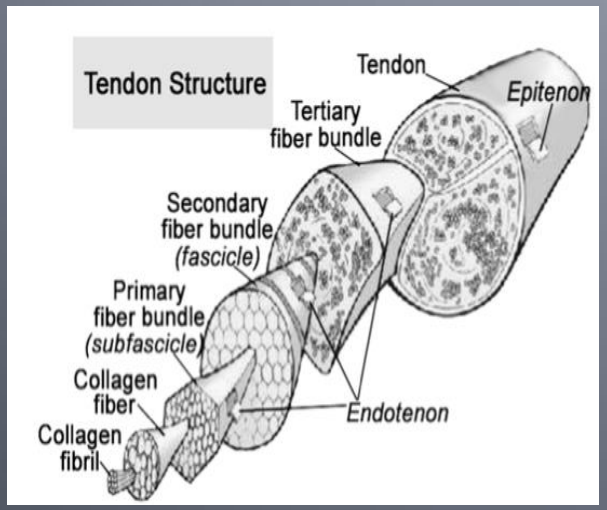
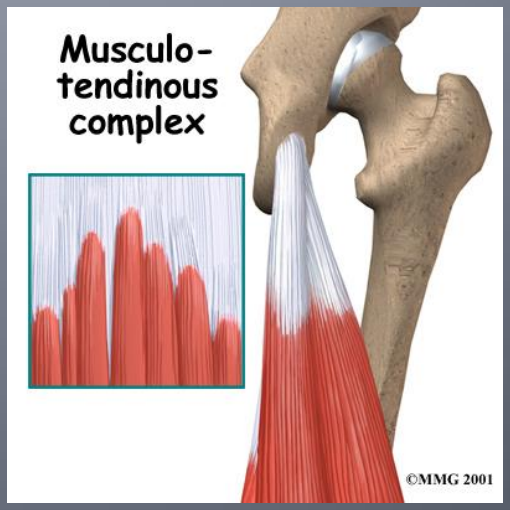
Basic Tendon Anatomy





Tendon Composition

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- Collagen (60% of the tendon)
  - Type I (95%) - Extensively cross-linked, Provides stiffness
  - Type III - less organized, Found at the insertion sites
  - In tendinopathy, the ratio of Type I:III fibers decreases, weakening the tendon.
- Non-collagenous Components (40% of the tendon)
  - Tenocytes - Fibroblast-like cells responsible for synthesis of the extracellular matrix (ECM)
  - Glycosaminoglobulins (GAGs)– proteoglycan & hyaluronan
    - Keeps ECM hydrated and Allows fibril sliding
  - Water, Elastin, Tenascin-C

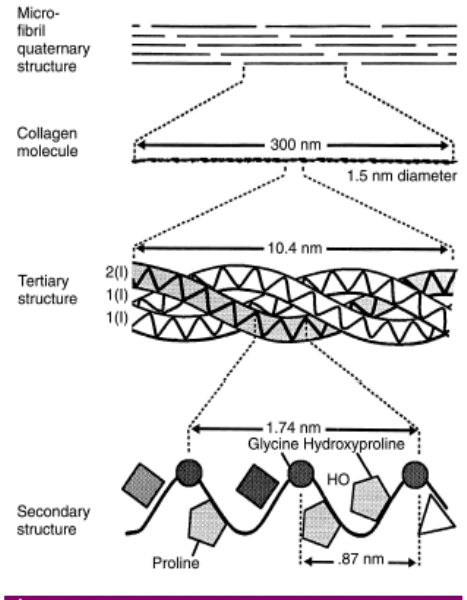
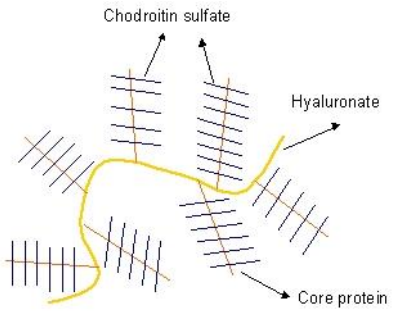


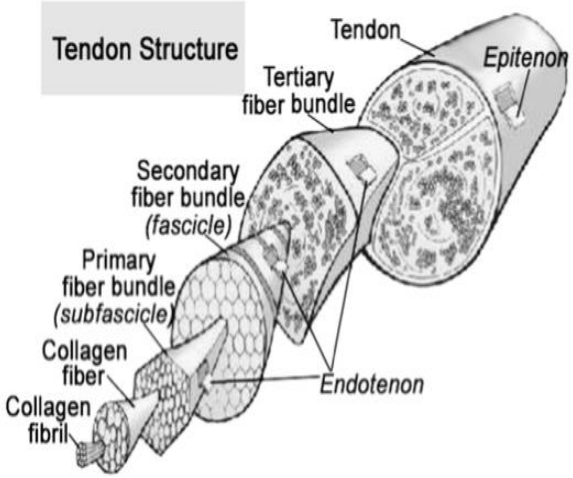
Figure 4

Schematic drawing of structural organization of collagen into the microfibril.



Tendon Structure

- Fascicles
  - closely packed, highly ordered parallel bundles of fibrils
    - Mainly Type I and III
  - Includes the tenocytes and non-collagenous substructures
- Extracellular matrix
  - Refers to the collagen fibers and the other proteins that keeps the tendon fibers and fascicles together
    - Like putting glue in a horse tail



Tendon Attachments

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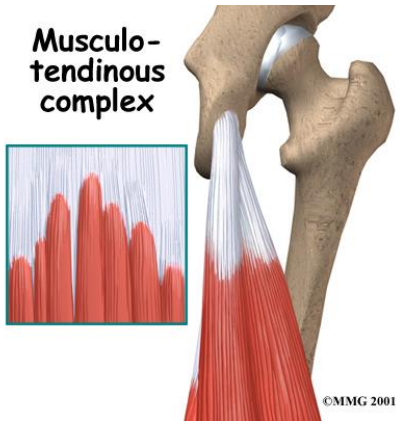
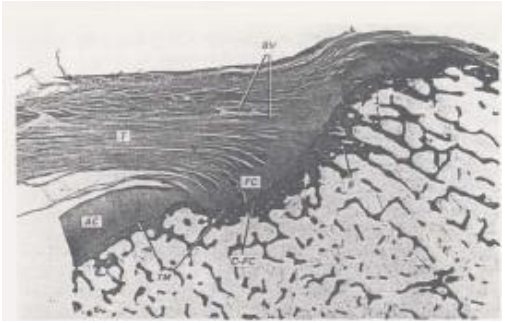
- Myotendinous Junction
  - Tendon fibers insert into deep recesses in the muscle
    - weakest point of the muscle-tendon unit
  - Site for acute tendon injuries
    - Ex. "Tennis leg"



- Enthesis
  - Tendon -> Fibrocartilage -> Bone
    - Tendon prone to compressive forces
  - Site for chronic injuries
    - First tries to heal with tendon tissue, then bone ('spur')



Blood Supply

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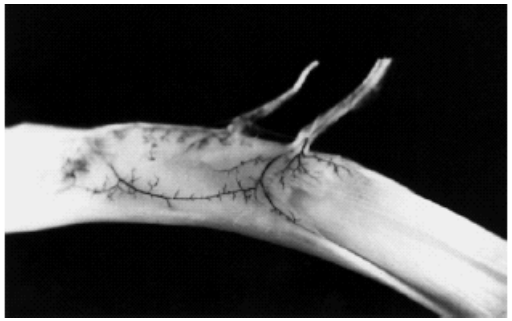
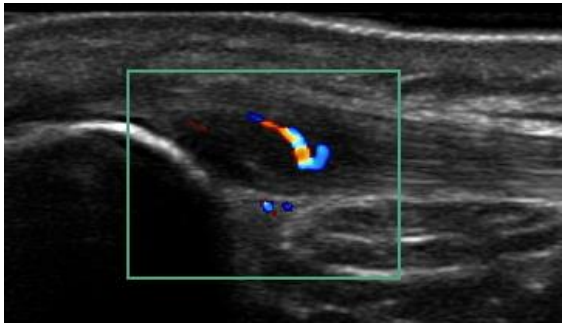
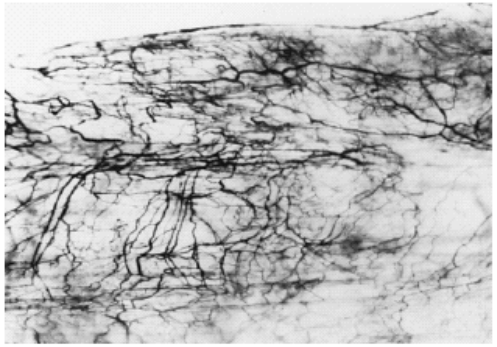
- Vascular tendons
  - Arterioles enter directly from the periphery into the tendon
    - Ex: Neo-vessels in Patellar tendinitis





- Avascular tendons
  - Circulation via perfusion
  - Found in areas subjected to increased mechanical stress
    - Ex: Peroneal tenosynovitis



Normal vs abnormal blood flow

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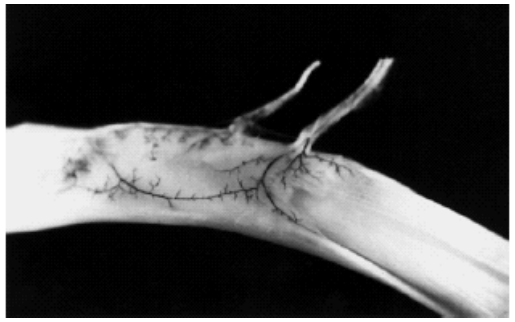
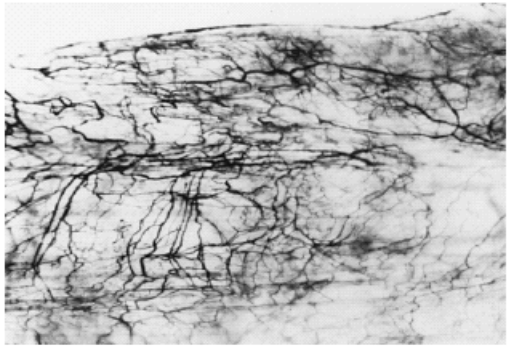
- Tendinopathy damages the ECM
  - allows arterioles in the epitenon to grow capillaries into the tendon
- Angiogenesis turns capillaries into arterioles "Neo-vessels"
  - Arterioles are innervated
    - Innervation = Pain

Normal



Pathologic



Metabolism

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- Adaptations to carry loads and maintain tension for long periods:
  - Tendon have low blood perfusion
  - Oxygen consumption of tendons is 7.5 times lower than that of skeletal muscles
- Low metabolic rate results in slow healing after injury
  - Ex. Acute shoulder strain → supraspinatus tendinitis → tendinosis → partial degenerative tendon tear.

Biomechanics of tendons

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- Tendons possess the highest tensile strength of any soft tissue in the body
  - Ex: Achilles tendon can carry up to 12 times the body weight
- Tensile strength = thickness + collagen content
  - Tendinopathy has lower Type I:III ratio
    - the tendon is at a higher risk of rupturing
- Tendons have weak resistance against repetitive shear and prolonged compressive forces
  - Examples
    - Dequervain tenosynovitis
    - Gluteus medius tendinitis due to abduction weakness

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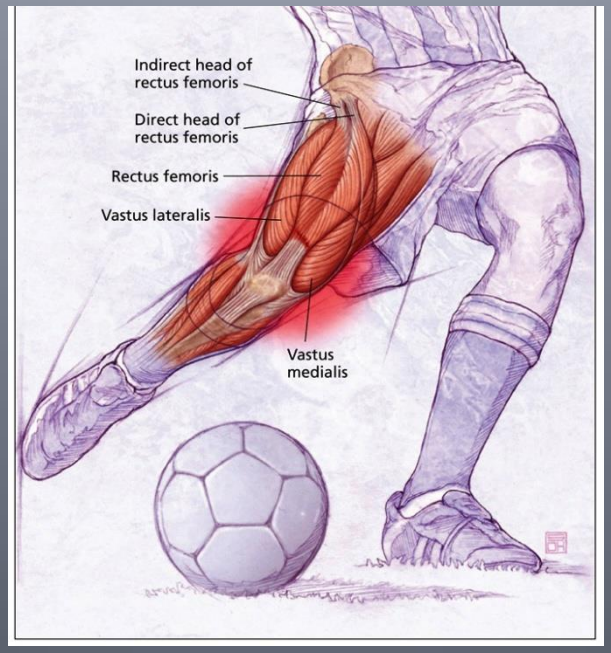
Patellar Tendinopathy



Patellar Tendon Anatomy

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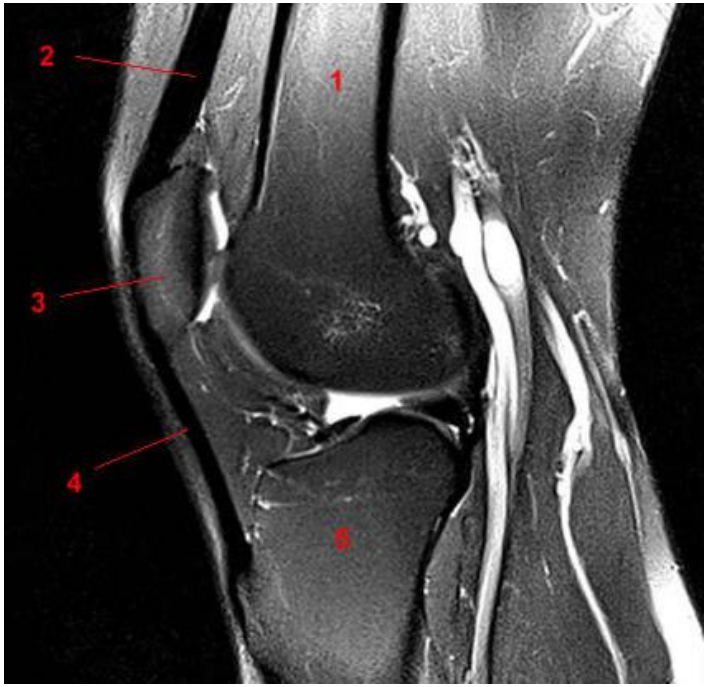
- Patellar bone is a sesamoid bone
  - acquired bone in the quadriceps tendon
    - Decreases friction in the tendon
    - Increases leverage for knee flexion/extenion
- About 15% of the quadriceps tendon continues over the bone into the Patellar tendon



"Jumper's Knee"

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- Patellar tendinopathy was first described in 1973
- Most common in sports with repetitive jumping and in heavy weight lifting
  - i.e. Basketball, volleyball, gymnastics, etc.
  - Incidence: 22% - 39% / males > females
- May occur at any of the three bony attachments
  - 25% quad tendon
  - 65% proximal patellar tendon
  - 10% distal patellar tendon

Patellar Tendinopathy in 'non-jumper' sports (baseball)

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- Dahm DL, et al: *Am J Orthop (Belle Mead NJ).* March/April 2016.

Sports Medicine Diagnostic Coding System (SMDCS)	N (%)	Mean Days Missed (Median)
1.. Knee contusion	662 (30.5)	6.0 (2.0)
2. Other knee injury	337 (15.5)	15.7 (5.0)
3. Patellar tendinopathy	222 (10.2)	14.7 (5.0)
4. Medial collateral ligament 1° sprain	120 (5.5)	18.1 (10.0)
5. Medial meniscal tear	102 (4.7)	41.2 (36.0)
6. Lateral meniscal tear	98 (4.5)	47.5 (49.0)
7. Lateral collateral ligament 1° sprain	46 (2.1)	15.1 (7.5)
8. Knee synovitis	44 (2.0)	13.0 (4.0)
9. Patellofemoral pain syndrome	44 (2.0)	12.5 (4.0)
10. Complete anterior cruciate ligament tear	41 (1.9)	156.2 (161.5)
All other SMDCS	455 (21.0)	20.9 (7.0)

Table from AJO article

Acute vs chronic

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- Acute tendon injuries: tendinitis or a full tear secondary to an excessive eccentric contraction.
  - Sudden onset of severe pain and swelling
- Chronic tendon injuries: repetitive stress of the tendon causes multiple microtears and degenerates the ECM (tendinosis).
  - Many "Acute" insertional tendon tears have been shown to have pre-existing degenerative changes. – Fenwick et al. *The vasculature and its role in the damaged and healing tendon. Arthritis Res 2002, 4:252-260.*

Why do tendons degenerate? Overuse Theory

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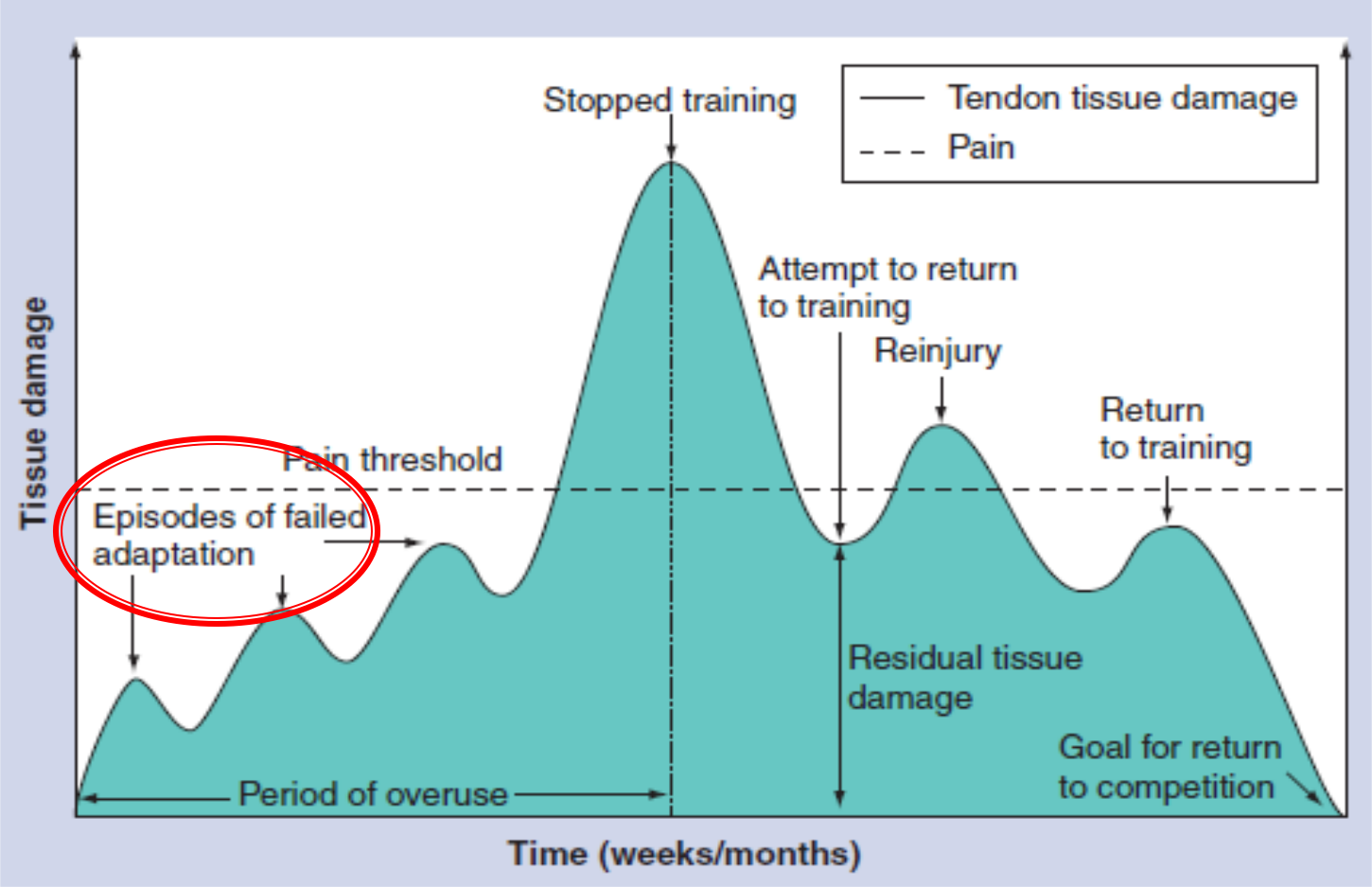

Load elongation curve

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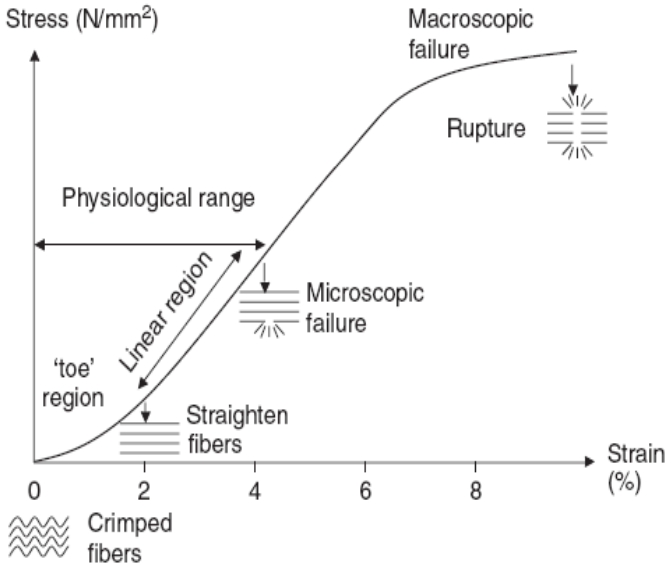
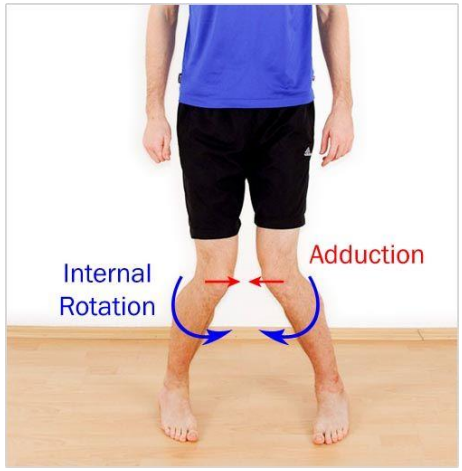
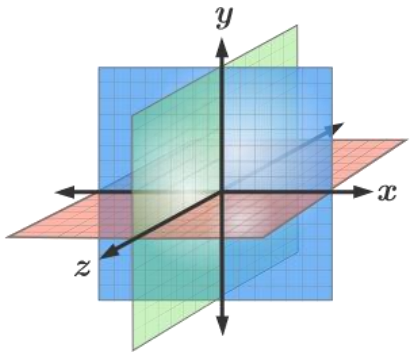
- Microscopic tears occur from:
  - Putting an extremity at a mechanical disadvantage
    - Loading past the physiologic range ("sticking point") leads to microscopic (patellar tendinitis) and macroscopic failure (pec rupture)



Mechanical Deficits

- Improper biomechanics load the tendon in a disadvantageous position
  - May be related to:
    - improper alignment
    - weakness
    - Tightness in adjacent regions





Flexion/extension deficits

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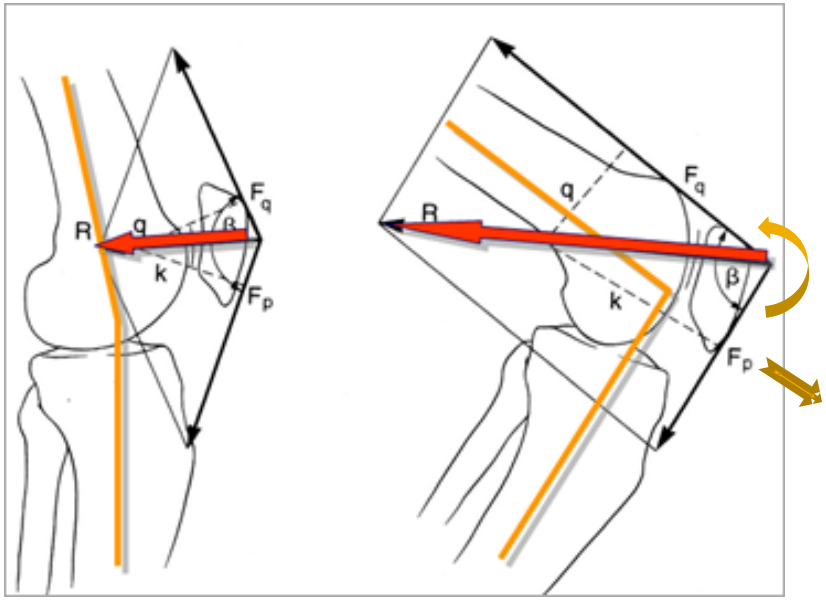
- Excessive knee flexion and anterior translation tilts the patella and stresses the proximal patellar tendon enthesis
  - Core : Lumbosacral extension weakness
  - Hip : Hip flexion contracture, glut max weakness
  - Knee : Hamstring contracture, quad/VMO weakness
  - Ankle : Achilles tendon contracture, ankle plantar flexion weakness



Medial/lateral deficits

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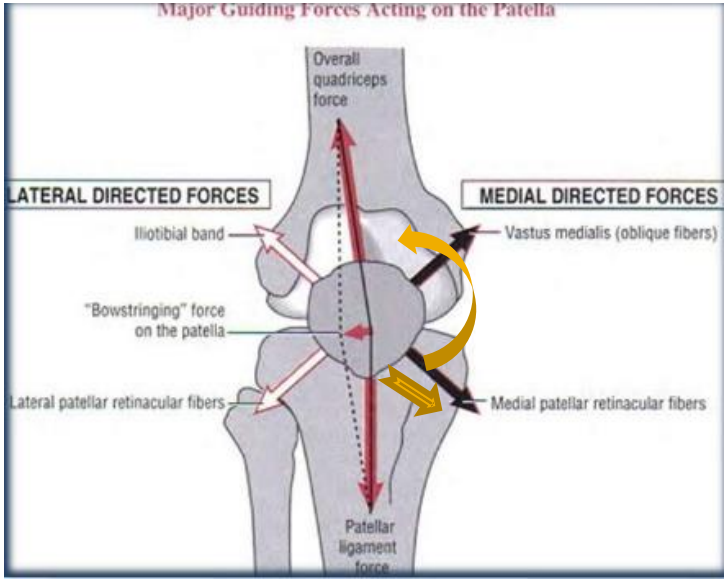
- Imbalance between the IT band and vastus medialis muscle leads to lateral tilting of the patella, creating a rotational torque that stresses the proximal patellar tendon
  - Core : Increased postural sway (obliques weakness)
  - Hip : IT band contracture, hip girdle (abduction) weakness
  - Knee: IT band contracture, VMO weakness
  - Ankle: Varus instability (ex. s/p high ankle sprain)



Rotational deficits

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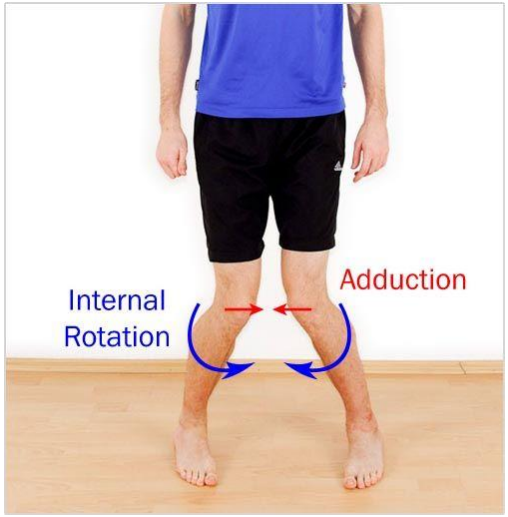
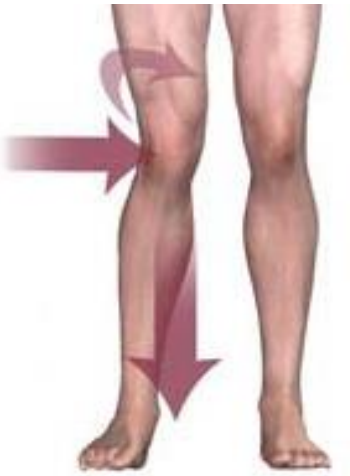
- Increased internal rotation of the femur
  - Core : Increased postural sway (rotation weakness)
  - Hip : Hip adductor contracture, External rotation weakness
  - Knee: VMO weakness (last 10^{o} of knee extension also externally rotate the tibia), MCL injury
  - Ankle: Planovalgum deformity, posterior tibialis dysfunction, talonavicular subluxation





Functional tests

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- Normal squat
- Single leg ½ squat
- Single leg deadlift
- Single leg drop from box and jump
- Jump ten times in place (watch the ankle recoil)

- https://www.youtube.com/watch?v=b2QMwvrkYUw

Squat evaluation

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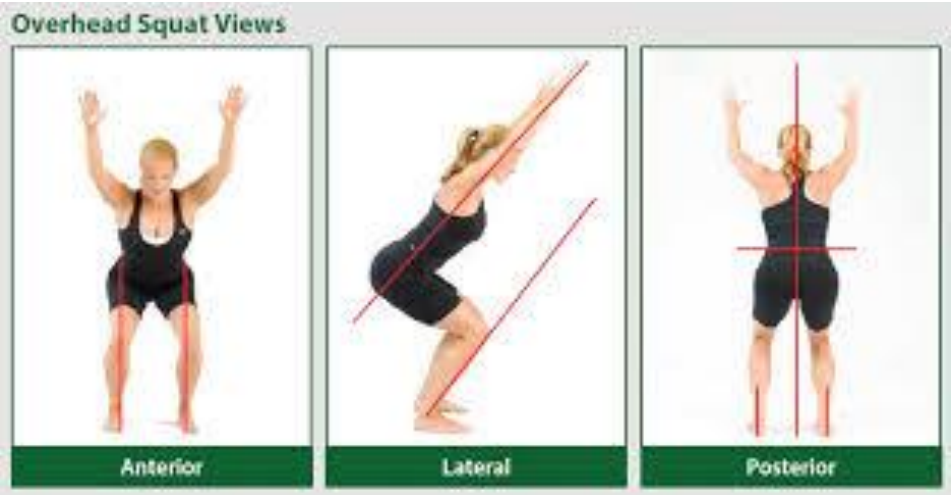

Correct

Incorrect

Factors Affecting Tendon Healing

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- Training
  - Strengthening stimulates tendon healing, but overuse weakens ECM
- Immobilization
  - ECM degenerates with prolonged immobilization.
- Age
  - Tensile strength decreases and stiffness increases after age 25
- Fluoroquinolones
  - Inhibit tenocyte metabolism and reduce collagen synthesis
- Anabolic steroids
  - Cause collagen fiber dysplasia, which decreases mechanical tensile strength and increases risk of rupture
- Anti-inflammatory medications
  - NSAIDS inhibit the proliferation of precursor cells during tissue healing
  - Corticosteroid injections have resulted in tendon rupture

Vascular deficit

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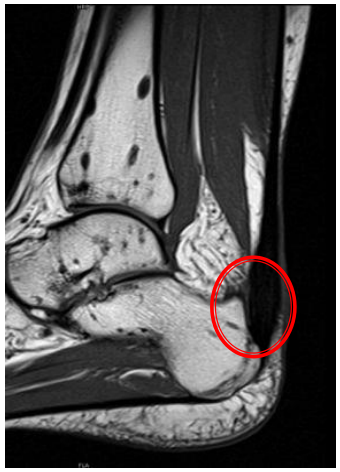
- Ischemia occurs under maximal tensile load.
- On relaxation, reperfusion occurs, generating oxygen free radicals that damage the ECM
- Since tendons are relatively avascular, they have a harder time getting the nutrients they need to heal
  - Tendinosis can take 3 to 6 months to heal



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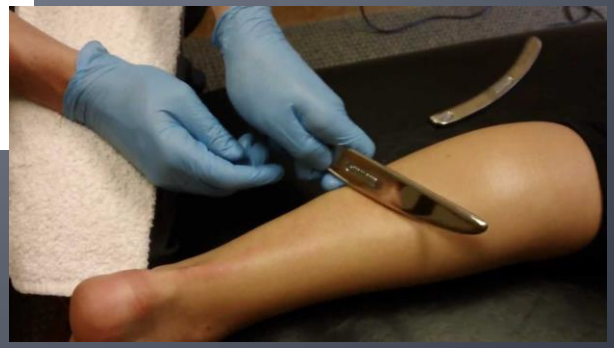
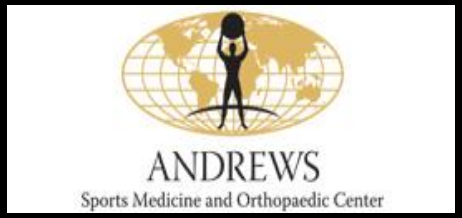
Treatment of Acute Tendon injuries



Rehabilitation Therapy



The rehabilitation of acute tendon injuries should be divided into three phases according to the phases of wound healing:

- Phase I: Inflammatory Phase
  - Tissue Protection -> First three days
- Phase II: Proliferative Phase
  - Collagen Deposition and Remodeling -> Day 3 to week 6
- Phase III: Maturation Phase
  - Collagen Strengthening -> Week 6 to 3 months, and on…

Healing potential of acute tendon injuries

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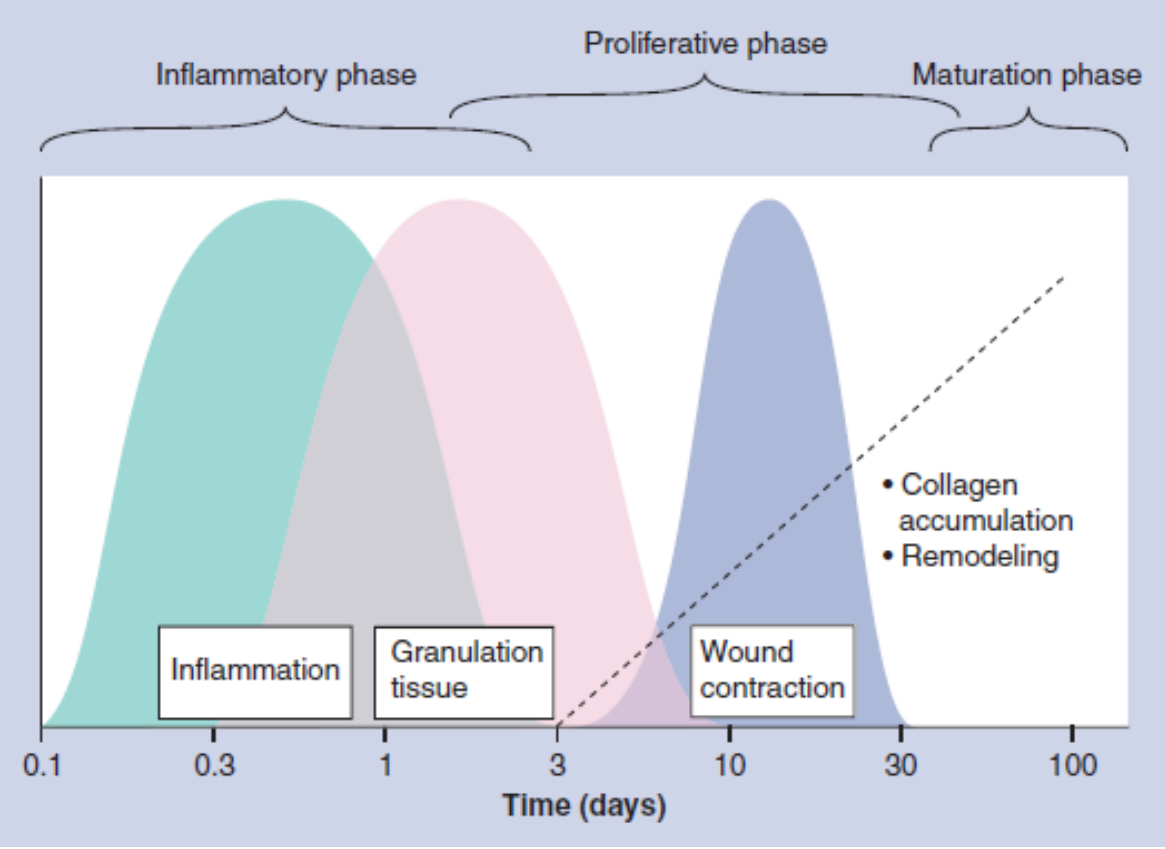
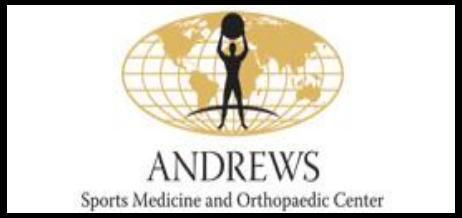
- Healing occurs in 3 phases:



Phase 1: Tissue Protection



- Immediately after acute injury
  - First three days
  - PRICE
- The objectives during this phase are pain control and tissue protection.
  - minimize excessive motion of the involved area (rest)
  - Limit NSAIDs and steroids unless profuse bruising or severe pain interfering with the patient's rehab.

NSAIDS

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- Anti-inflammatory medications are controversial for tendon injuries
- NSAIDs have an analgesic effect
  - Best results in acute tendinitis to allow early restoration of range of motion
- Controversial in chronic tendinopathy
  - Theoretically, tendinosis is not an inflammatory process

Activity Modification

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- Activity modification
  - Decrease the mechanical demand
  - Eliminate the overuse strain
- Bracing
  - Protect the injured tendon if there is a partial tear for 3 to 7 days
    - Ex. Knee immobilizer
  - Decrease tissue swelling with compression for 2 to 6 weeks
    - Avoids disruption of the ECM
    - Ex. Counterforce brace for lateral epicondylitis

Physical Therapy

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- Therapeutic modalities
  - Cryotherapy
    - Leads to vasoconstriction and decreased soft tissue swelling
  - Iontophoresis & Phonophoresis
    - Serve as a medium to deliver medication topically
  - Deep Friction Massage (ex. Graston)
    - Myofascial release of adjacent muscles
  - Therapeutic Ultrasound after 3 to 7 days
    - A form of heat that stimulates increase in blood flow into the injured site

Phase 2: Start rehab

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- After the first 3 to 7 days, start:
  - Gentle stretching and isometric strengthening of the involved tendon
  - Begin treatment on kinetic chain/adjacent regions
  - Progress to functional weight bearing
  - Progress to low intensity strengthening exercises, i.e. low weight/high repetition, to progressively increase the load placed on the tendon.

Phase 2: Collagen Deposition and Remodeling

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- Mechanical stimulation helps drive tendon healing
  - This activates mitosis in tenocytes, collagen synthesis, and stimulates converting type III to type I fibers
    - Strengthening exercises = tendon healing
    - But too much loading can causes micro-tears
  - In order to load the tendon appropriately, each athlete should respect their limits with regards to:
    - How much weight, Reps, and Sets
      - Gradually increase these parameters
    - Rest

Valgus Control

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- The Effect of Valgus Control Instruction Exercises on Pain, Strength, and Functionality for PFPS

  Sports Health, May/June 2019; vol 11, no. 3; 223-37

  - Provides 16 exercises for a 6-week patellofemoral pain rehab program that focuses of strength and conditioning to correct biomechanical deficits
    - Divided into 3 phases of progressive loading every two weeks

Phase 3: Collagen Strengthening

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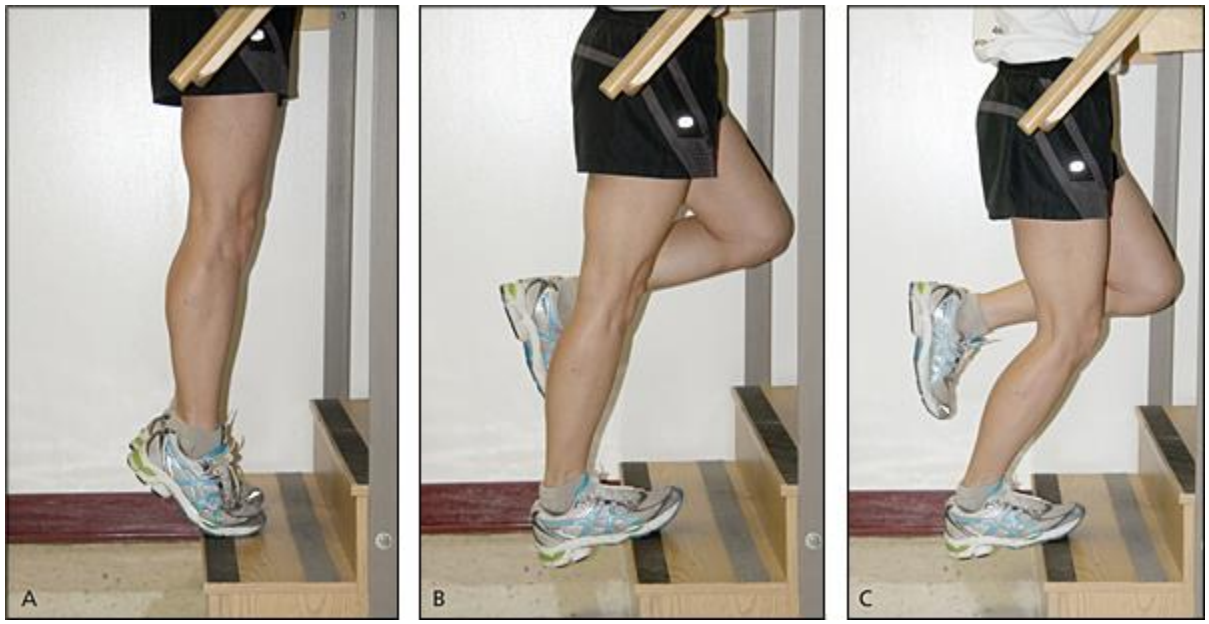
- After 2 to 6 weeks, introduce eccentrics, sports-specific drills, and eventually 'return to play'



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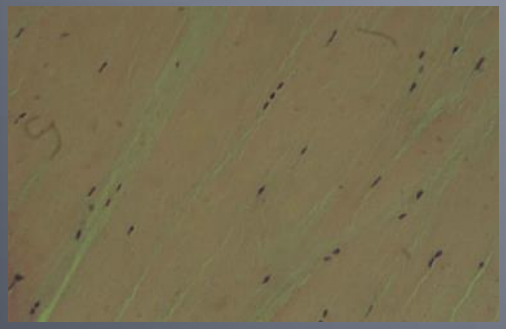
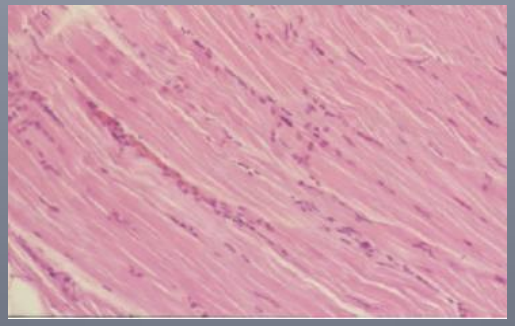
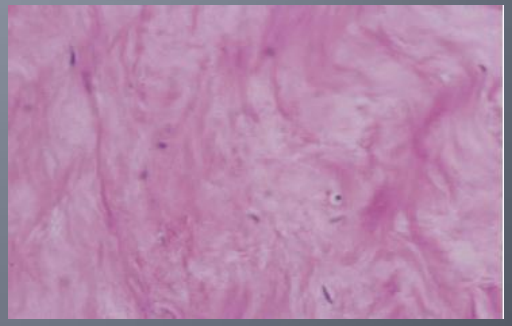
Treatment of Chronic Tendon injuries

Normal → Degenerative

Historical Approach to Tendon Pathology

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- The treatment of tendinosis used to focus on treating perceived acute inflammation
  - Not all tendon injuries are acute
  - NSAIDs, CSI's, etc. commonly offered for ALL cases
    - May weaken the tendon
- Those considered cured (because their pain lessened) often had residual structural weakness
  - Tendon tears
  - Calcifications
  - Poor collagen deposit (low Type I:III ratio)
  - Re-injury occurred with return to play

Accelerated versus Appropriate Return to Play

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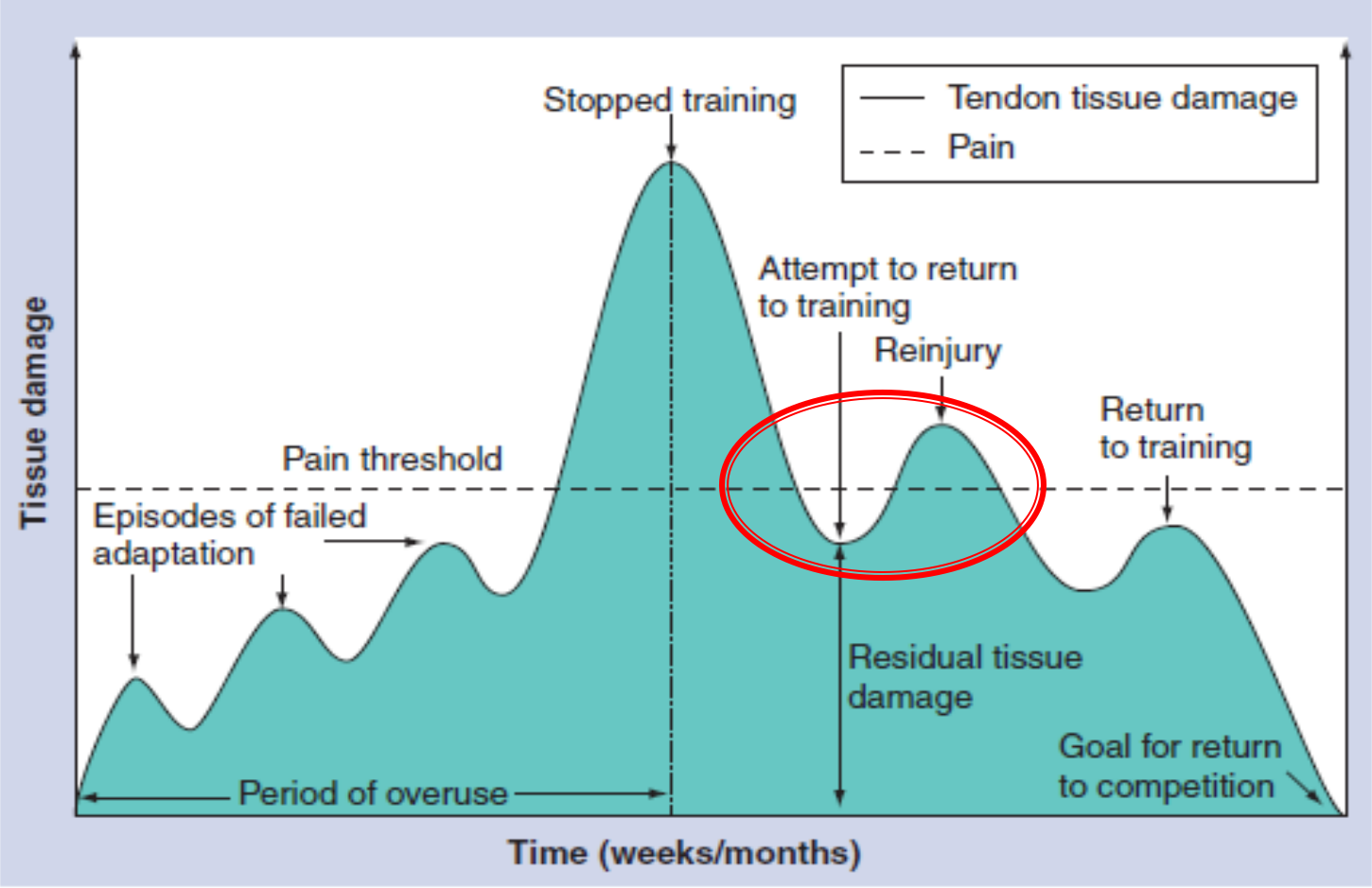

Eccentric Exercises = gold standard

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Resisted lengthening of the muscle-tendon unit ("negative sets")

- Best described in Achilles tendinopathy
- Theory:
  - Eccentric exercises occlude and terminate neovessels and its small nerve fibers.
    - Thought to be a part of the pain generator in tendinopathy
  - They stimulate collagen remodeling and strengthening[2,3]

1. Alfredson H, Pietilä T, Jonsson P, Lorentzon R. Heavy-load eccentric calf muscle training for the treatment of chronic Achilles tendinosis. *Am J Sports Med*. 1998 May-Jun;26(3):360-6.
2. Langberg H, et al. Eccentric rehabilitation exercise increases peritendinous type I collagen synthesis in humans with Achilles tendinosis. *Scand J Med Sci Sports*. 2007 Feb;17(1):61-6.
3. Rees JD, Maffulli N, Cook J. Management of tendinopathy. *Am J Sports Med*. 2009;37(9):1855-1867

Eccentric Exercises

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- Exercises are done twice a day for 12 wks
  - May present a compliance issue
- Works best w/ mid-substance tendon lesions
- Complications:
  - Patient may experience an initial worsening of pain
  - Eccentric activity too early may be detrimental to the regenerative cascade
  - Compression is detrimental to insertional tendinopathy

Any strengthening heals

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- Beyer R, et al. Heavy Slow Resistance vs Eccentric Training as treatment for Achilles Tendinopathy: A Randomized Controlled Trial. *Am J Sports Med.* 2015 Jul; 43 (7):1704-11.
  - **Evidence:** Randomized controlled trial; Level of evidence, 1.
  - **Methods**: 58 patients with chronic (>3 months) midportion Achilles tendinopathy were randomized to ECC or HSR for 12 weeks
  - **Results:** Both improved in VISA-A and VAS up to one year out. Patient satisfaction was greater at 12 weeks with HSR (100%) than with ECC (80%; P = .052) but not after 52 weeks (HSR, 96%; ECC, 76%; P = .10), and compliance rate was 78% in the ECC group and 92% in the HSR group (P < .005).
  - **Conclusion:** Equal outcomes with better compliance in the HSR group.

Extracorporeal Shock Wave Therapy

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Used for multiple soft tissue conditions

- Delivers low-energy shock waves to painful area
  - Theorized to cause nerve degeneration
  - Some evidence that tenocytes release growth factors
  - Ideal intensity/frequency/duration is unclear

- Best evidence for treatment of calcific tendinosis and plantar fasciitis

Outcomes with conservative treatments

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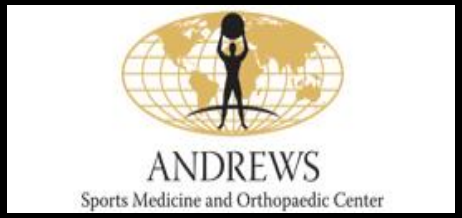
Steunebrink, M., et al. "Comparison of the Effect of 5 Different Treatment Options for Managing Patellar Tendinopathy: A Secondary Analysis." *CJSM* (2017).

- Objective: Assess which treatment option provides best outcome
  - Extracorporeal shockwave therapy (ESWT), ESWT plus eccentric training, eccentric training only, topical glyceryl trinitrate patch plus eccentric training, and placebo treatment
- Results:
  - 37.7% improved clinically after 3 months of treatment.
  - Odds ratios (ORs) for clinical improvement were significantly higher in the eccentric training group (OR 6.68, P = 0.009) and the ESWT plus eccentric training group (OR 5.42, P = 0.015)
  - High training volume, longer duration of symptoms, and older age negatively influence a treatment's clinical outcome

Corticosteroids



- Effectiveness & safety is controversial
  - depends on duration of use, amount, and site of injection
- Consensus
  - Limited oral use does not compromise integrity
  - Do not inject within the tendon
    - Case reports of local injections in the Achilles and patellar tendon causing rupture
  - Long term use may inhibit collagen biosynthesis and weaken tendons
- No good evidence to support use in chronic tendinosis

Surgery

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- Considered after prolonged conservative treatment has failed (6 to 9 months)

- Allows inspection of tendon for areas of degeneration and partial tearing

- Goal: Remove necrotic debris, suture full thickness tears, and initiate a healing response with the local tissue trauma from the surgery

Why do we do surgery?

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- It is NOT because of medical evidence
  - "Only 20% of orthopedic surgeries are estimated to be supported by at least one low-risk-of-bias randomised controlled trial showing that surgery is superior to a non-operative alternative."
    - *Lohmander LS, Roos EM. The evidence base for orthopaedics and sports medicine: scandalously poor in parts. Br J Sports Med. 2016 May 1;50(9):564-5.*
- Patients have misconceptions about surgery:
  - Surgery = "fixing it", "100% success rate", "best outcome"

Systematic Reviews on Surgery for patellar tendinopathy

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- Coleman, B. D., et al. "Studies of surgical outcome after patellar tendinopathy: clinical significance of methodological deficiencies and guidelines for future studies." *Scandinavian Journal of Medicine & Science in Sports: Review article* 10.1 (2000): 2-11.
  - Negative correlation between reported success rate and methodology
- Larsson et al. 2012: *Treatment of patellar tendinopathy–a systematic review of randomized controlled trials.*
  - *Surgery lacks reliable evidence*
- Everhart, Joshua S., et al. "Treatment options for patellar tendinopathy: a systematic review." *Arthroscopy: The Journal of Arthroscopic & Related Surgery* 33.4 (2017): 861-872.
  - *Open vs arthroscopy had similar outcomes: average 57% success rate*

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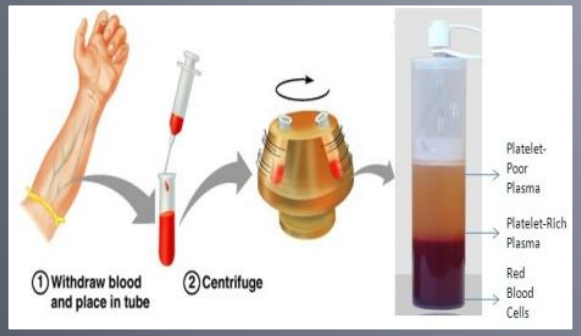
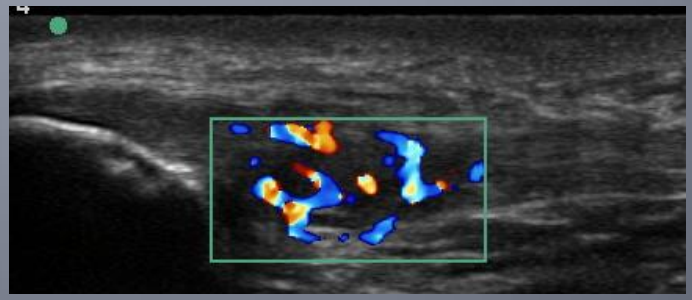
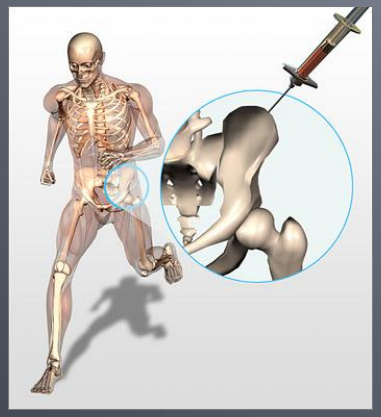
Orthobiologics:

"The holy grail of orthopedics is finding a way to effectively regenerate the injured tissue back to its original state"







Orthobiologics

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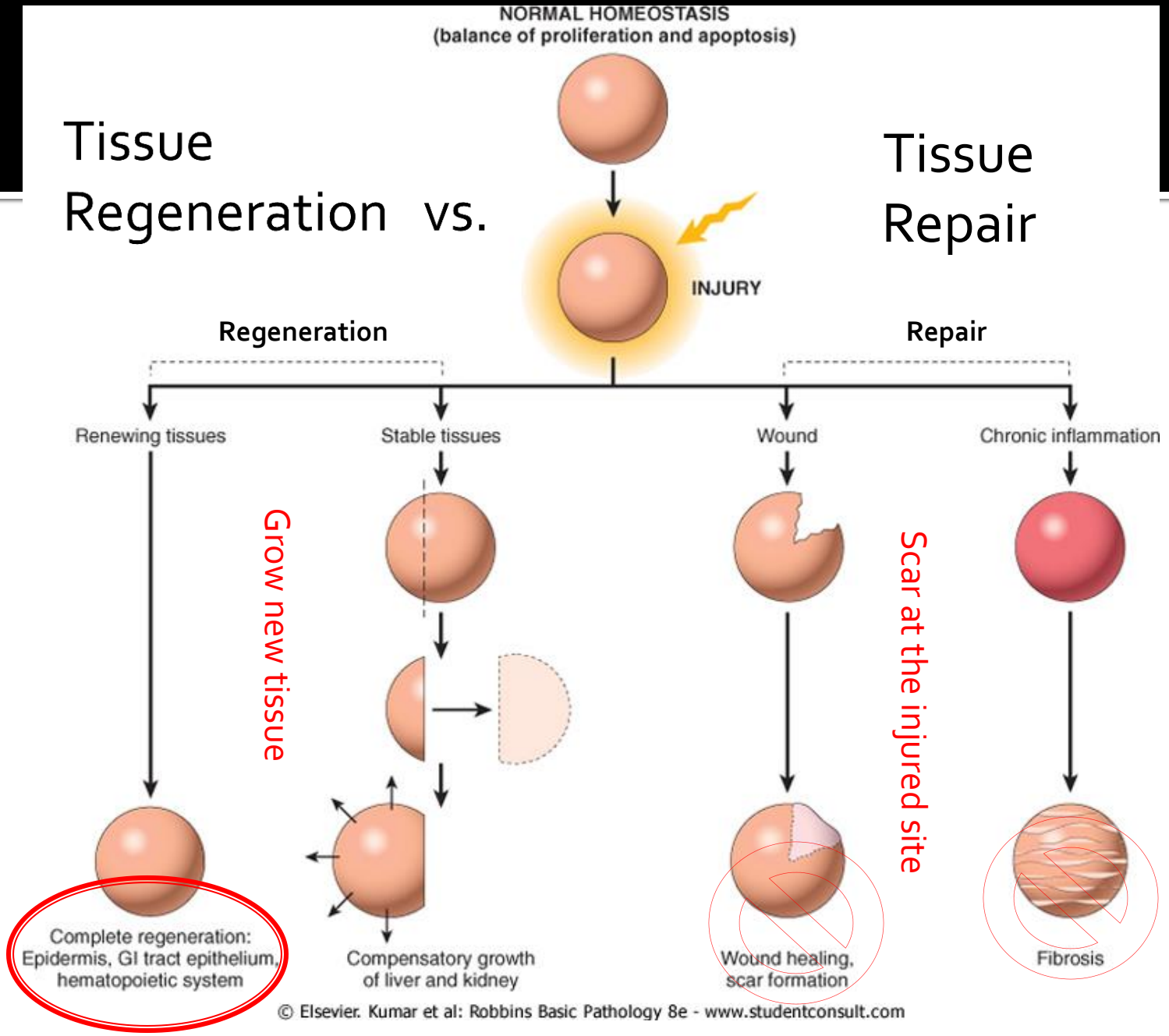
- Orthobiologics is based on using growth factors to activate the natural regenerative response of injured tissues.
- These growth factors are found inside:
  - Platelet cells harvested from the patient's own blood
    - PRP = platelet rich plasma
  - Mesenchymal 'signaling' cells harvested from the bone marrow or amniotic tissue
    - BMAC = bone marrow aspiration and concentration

Tissue Regeneration vs. Tissue Repair



PRP for Patellar tendinopathies

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- Kon et al, 2009 : case-series report on patellar tendon
  - Significant improvement for all functional and sports-specific scores, and radiographic imaging at 6 months
- Filardo, 2010 : prospective case-control study on PRP vs PT
  - Significant improvement in functional scores at 6 months
- Gosens, 2012 : Prospective case-control - PRP as 1st line Tx
  - PRP as first line therapy outscored Physical Therapy alone
- Kaux and Croisier et al, 2013 : Case-series report
  - More than 75% of athletes returned to sports, with significantly better outcomes in younger patients -> junior and collegiate-level athletes
- Filardo et al, 2013 : Prospective case series
  - Significant improvement for at least four years in the VISA-P
- Vetrano et al, 2013 : RCT on PRP vs ECSW
  - Significantly better outcome in the PRP group at 6 & 12 ms

Long-Term Outcomes on PRP

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- Mautner K, Colberg R, et al. "Outcomes after ultrasound-guided platelet-rich plasma injections for chronic tendinopathy: a multicenter, retrospective review." *PM&R* 5.3 (2013): 169-175.
  - Progressive improvement up to one year after injection
  - Improvement maintained for at least 3 years after the PRP injection

- Fralinger, David J., et al. "Biological treatments for tendon and ligament abnormalities: a critical analysis review." *JBJS reviews* 4.6 (2016).
  - Evidence supports PRP for chronic, degenerative tendinopathies such as patellar tendinopathy.
  - It does not support PRP to promote ligament-to-bone healing in anterior cruciate ligament (ACL) reconstruction.

Bone marrow-derived Cells

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- There is limited literature on BMAC for soft tissue injuries
  - May be related to the success of PRP
  - BMAC studies have focused on bone and joint pathology

- Pascual-Garrido, Cecilia, A. Rolón, and A. Makino. "Treatment of chronic patellar tendinopathy with autologous bone marrow stem cells: a 5-year-followup." *Stem cells international* 2012 (2012).
  - 8 patients, average age 24, failed conservative x 6 ms
  - Conclusions: At 5-year follow-up, statistically significant improvement was seen for most clinical scores. Seven of eight patients said they would have the procedure again if they had the same problem in the opposite knee, had excellent results, and were completely satisfied with the procedure.

What about Amniotic stem cells?

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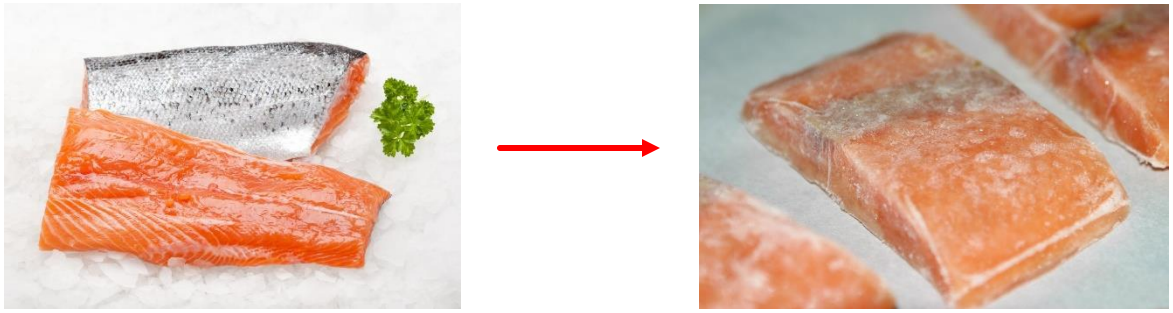
- No studies have been published on patellar tendinopathy
  - Results are promising for other pathologies, but questionable
    - Ex. Capacity to regenerate a degenerative disc
- Dilemma - these cells do not survive in a dehydrated or frozen state
  - Placebo effect?
  - Do the growth factors remain viable?



What about the neovessels?

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- Some argue that the neovessels need to be obliterated with percutaneous tenotomy in order for the tendon to heal appropriately.
  - Just injecting PRP alone does not achieve this

US guided Intra-patellar tendon debridement

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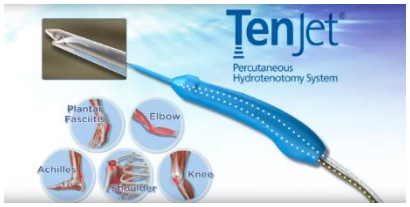
- Ultrasound allows you to target pathology, like the neovessels and degenerated tissue inside the tendon, which is not possible with arthroscopy
- Instruments used:
  - Supersonic saline debridement (Tenex and TenJet)
  - Plasma field coablation debridement (Topaz EZ microdebrider)



Studies on patellar tenotomies

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- Testa VI, et al. Ultrasound-guided percutaneous longitudinal tenotomy for the management of patellar tendinopathy. Med Sc Sp Ex. 1999.
  - 34 athletes had US-guided tenotomy, followed for 2 years, and 74% had significant improvement.
- Housner, Jeffrey A., et al. "Should ultrasound-guided needle fenestration be considered as a treatment option for recalcitrant patellar tendinopathy? A retrospective study of 47 cases." *CJSM 2010.*
  - 47 cases followed up to 45 months out, and 72% had significant improvement.
- Dragoo, Jason L., et al. "Platelet-rich plasma as a treatment for patellar tendinopathy: a double-blind, randomized controlled trial." *AJSM 2014*
  - PRP vs percutaneous tenotomy
  - PRP provided quicker pain relief at 3 months but they both had similar improvement at 6 months
- Nanos, Katherine N., and Gerard A. Malanga. "Treatment of patellar tendinopathy refractory to surgical management using percutaneous ultrasonic tenotomy and platelet-rich plasma injection: a case presentation." *PM&R*. 2015
  - a collegiate athlete with chronic proximal patellar tendinopathy who was effectively treated with percutaneous ultrasonic tenotomy and PRP after not responding to extensive nonoperative treatment, surgical debridement, and platelet-rich plasma injections.

Accelerated versus Appropriate Rehab and Return to Play

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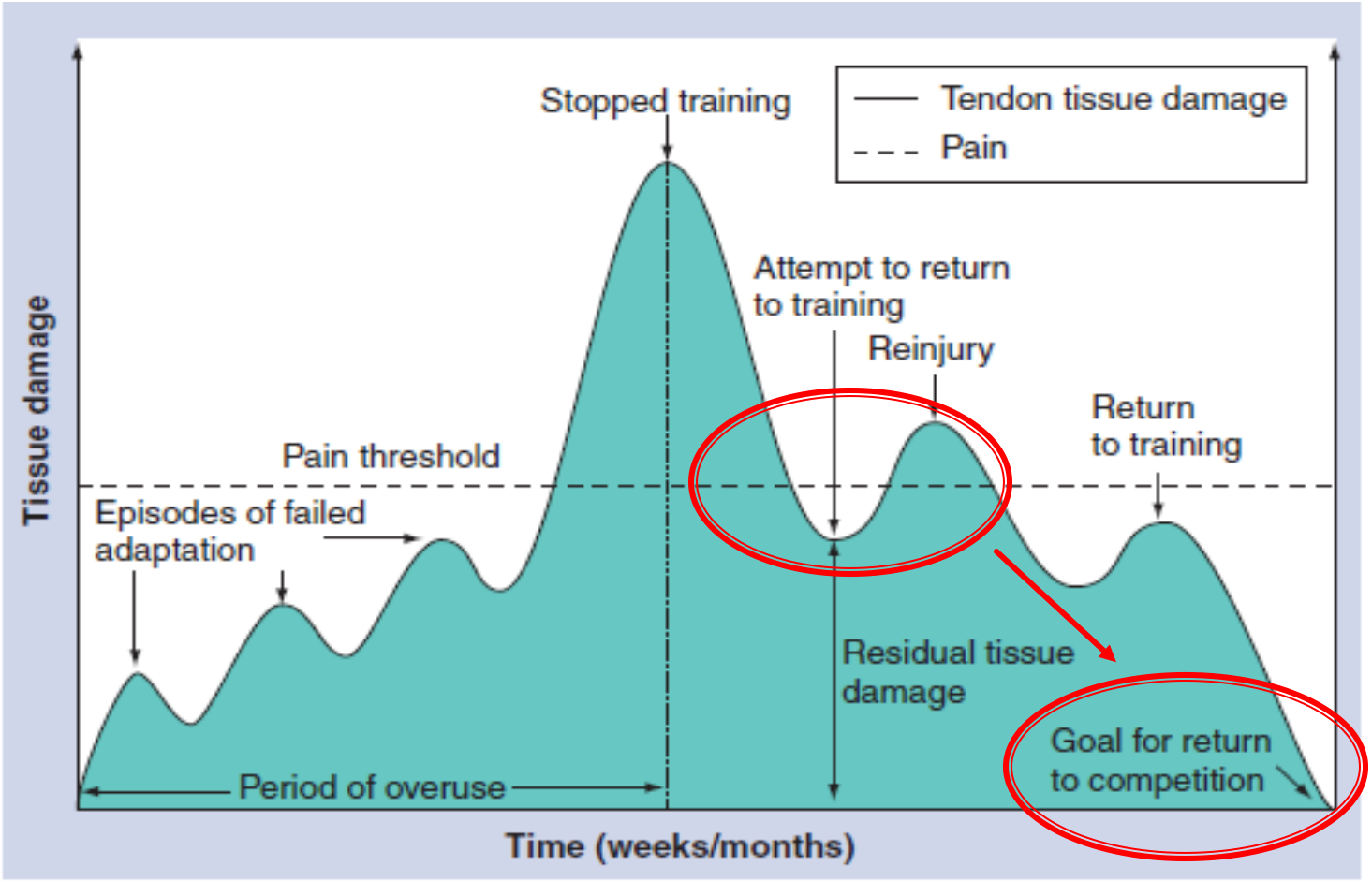

Table 3. Suggested rehabilitation protocol following platelet-rich plasma injection.

Phase	Length of time	Restrictions	Rehabilitation
Phase I: tissue protection	Days 0–3	Consider NWB or protected WB for lower extremity procedures, especially if in pain. No weight training, avoid NSAIDs and use limited ice	Relative rest. Activities as tolerated; avoiding excess loading or stress to treated area. Gentle AROM
Phase II: early tissue healing; facilitation of collagen deposition	Days 4–14	Progress to FWB without protective device. Avoid NSAIDs	Light activities to provide motion to tendon; aerobic exercise that avoids loading of the treated tendon. Gentle prolonged stretching. Begin treatment on kinetic chain/adjacent regions. Glutei strengthening and core strengthening
	Weeks 2–6	Avoid eccentric exercises. Avoid NSAIDs. Avoid ice	Progress to WB activities. Low weight, high repetition isometrics (pain scale <3/10). OKC activities. Soft tissue work to tendon with CFM, IASTM and 'dynamic' stretching
Phase III: collagen strengthening	Weeks 6–12	–	Eccentric exercises (keep pain scale <3/10). Two sets of 15 repetitions. CKC activities. Plyometrics; proprioceptive training and other sport-specific exercises. Progress to WB activities and consider return to sport if pain <3/10
	Months 3+	Reassess improvement; if not >75% improved consider repeat injection and return to Phase I	Progress back to functional sport-specific activities with increasing load on tendon as pain allows

AROM: Active range of motion; CFM: Cross-frictional massage; CKC: Closed kinetic chain; FWB: Full weight bearing; IASTM: Instrument-assisted soft tissue mobilization; NWB: Non-weight bearing; OKC: Open kinetic chain; WB: Weight bearing.

Summary

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- Tendon anatomy and biomechanics are intimately related to it's response to injury
  - Injuries occur due to overuse, improper alignment, or weakness
- Acute injuries follow the three phases of healing
  - May have a component of chronicity
- Tendinosis is degenerative in nature, not inflammatory
  - Strengthening is the gold standard for tendinosis
    - Typically more important than stretching
- Orthobiologics or surgery may be indicated if the patient fails conservative treatments.

Summary of Common Treatment Options

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Conservative

- Activity modification
- Braces

Physical Therapy

- Therapeutic ultrasound
- Cryotherapy
- Iontophoresis / Phonophoresis
- Massage
- Extracorporeal Shock Wave Therapy
- Eccentric exercises

Medications

- NSAID's
- Corticosteroid injections
- Orthobiologics

Surgery

- Open Debridement
- Tenotomy
- Repair

Thank You!

Questions?

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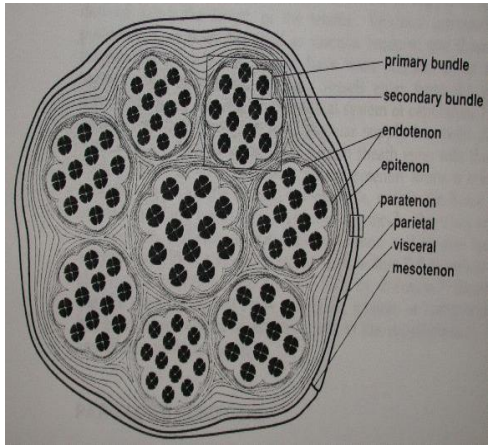
Tendon Membranes

- Endotenon
  - divides tendons into fascicles
  - contains capillaries & small nerve fibers
    - Ex. Tendinitis
- Epitenon
  - covers the outer surface of the tendon
  - contains innervated arterioles
    - Ex. Pathologic neovessels
- Paratenon
  - Membrane surrounding the epitenon
  - Has an inner synovial lining
    - Ex. Tenosynovitis



Enthesis

There are two types of entheses:

- *Fibrous*
  - The tendon, ligament or joint capsule directly attaches to the bone
    - Ankylosing Spondylitis
- *Fibrocartilaginous*
  - Divided into four zones:
    - Tendinous area
    - Fibrocartilaginous region of tenocytes/collagen and chondrocytes
    - Abrupt transition from cartilaginous to calcified fibrocartilage
      - the so-called 'tidemark' or 'blue line'
    - Bone

  Ex. Infrapatellar and Achilles calcific tendinitis (Bone spurs)

Blood Supply

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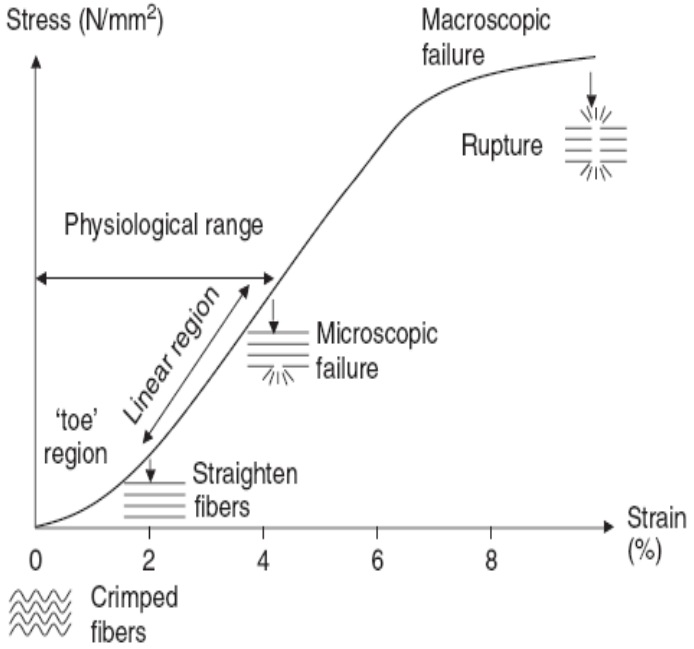
- Tendons receive their blood supply from three main sources
  - The intrinsic systems
    - myotendinous junction
    - osteotendinous junction
  - The extrinsic system through the epitenon and paratenon (synovial sheath)

- The ratio of blood supply from the intrinsic systems to that from the extrinsic system varies from tendon to tendon.
  - Tendons have watershed areas that also vary throughout the body.

Biomechanics

- Load-elongation curve
  - 'Toe' region
    - straightening of the relaxed fibers
  - Linear region
    - Physiological range
  - Failure region
    - Microscopic (patellar tendinitis) and macroscopic failure (pec rupture)



Epidemiology of Tendon Injuries

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- Incidence of sports-related tendon injuries is between 30% to 50%. *(Järvinen TA)*
- Acute tendon injuries usually present as tendinitis in the watershed area or a full tear of the myotendinous junction secondary to an excessive eccentric contraction.
- Chronic tendon injuries usually occur secondary to poor healing of tenditis or repetitive movement that causes multiple microtears near the tendon enthesis.
  - Even "Acute" insertional tendon tears have been shown to have pre-existing degenerative changes. – Fenwick et al. *The vasculature and its role in the damaged and healing tendon. Arthritis Res 2002, 4:252-260.*

Classifications

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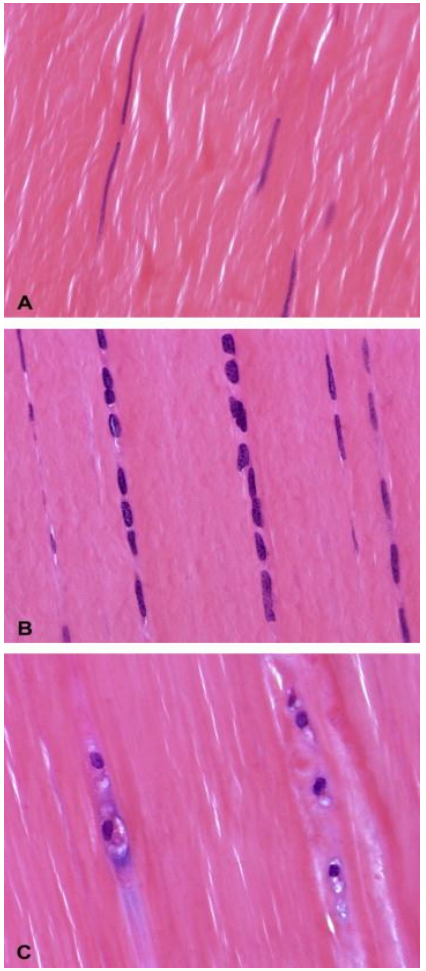
- Tendinitis - A tendon with an acute inflammatory process
- Tendinosis – An injured tendon that:
  - no longer has an active inflammatory process, evidenced by lack of inflammatory cells in the tissue
  - Has residual degenerative pathology such as abnormal tissue repair, collagen degeneration, neovascularization, and thickening of the tendon.
- Tendinopathy - Tendon pathology without implying presence or absence of inflammation.
- Enthesopathy – Tendon pathology at the bony insertion.

Tenocytes

- Tenocyte is the primary cell type
  - Poorly differentiated fibroblasts
  - Arranged in parallel rows
  - Surrounded by protective pericellular sheath containing type IV collagen, fibrillin (provide scaffolding for elastin deposition), & versican (chondroitin sulfate proteoglycan)



Chronic Tendinopathy

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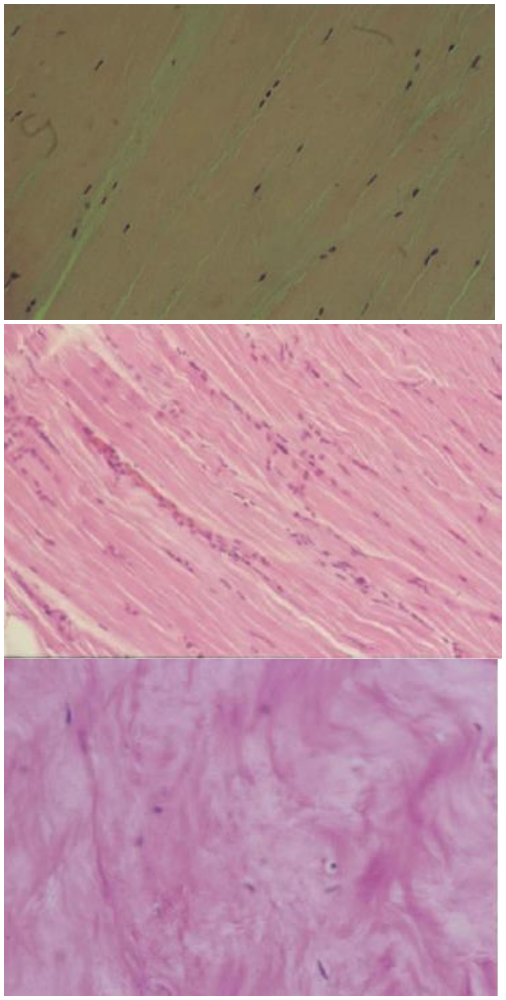
- 5 features are present
  - Collagen and ECM degeneration
  - Increased proteoglycan (more water)
  - Tenocyte abnormalities (areas of hypercellularity)
  - Altered cell populations (chondroid/synovial metaplasia)
    - Enthesopathic calcification
  - Increased presence of innervated microvessels



Normal

Degenerative

Rees et al. Rheumatology, May 2006

Biomechanics and tendon healing

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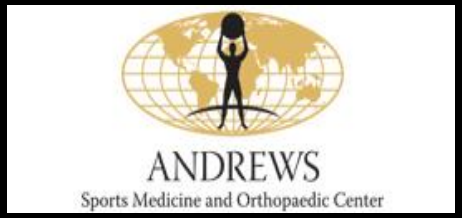
- Tendons require loading to regulate their metabolism and maintain their tensile strength
  - The mechanical loading parallel to the vector force activates mitosis in tenocytes and collagen synthesis, and stimulates converting type III to type I fibers
  - Immobilization (lack of load response) causes loss of tensile strength and ECM degeneration
    - Virchenko & Aspenberg reported tendon repair in a rat's Achilles tendon model after mechanical stimulation and no tendon repair with prolonged immobilization.

Vascular Theory



- Tendons have a low metabolic rate and are less vascular than muscles
  - Can be explained by:
    - Lack of vascular supply beyond the proximal third of the tendon - Watershed
    - This is a structural adaptation of tendons to stand phasic & tonic contractions without risk of hypoxic injury in order to maximizes the load-carrying capacity

Historical Approach to Chronic Orthopedic Injuries

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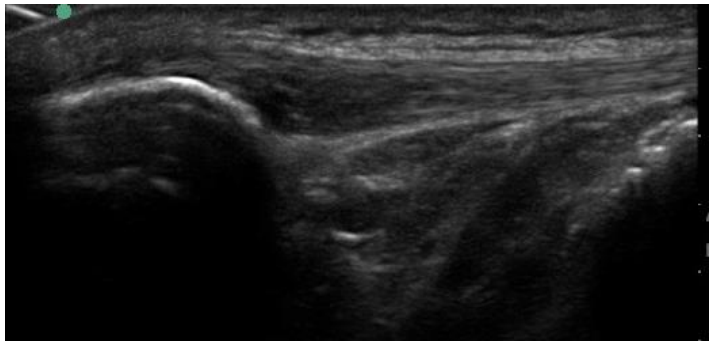
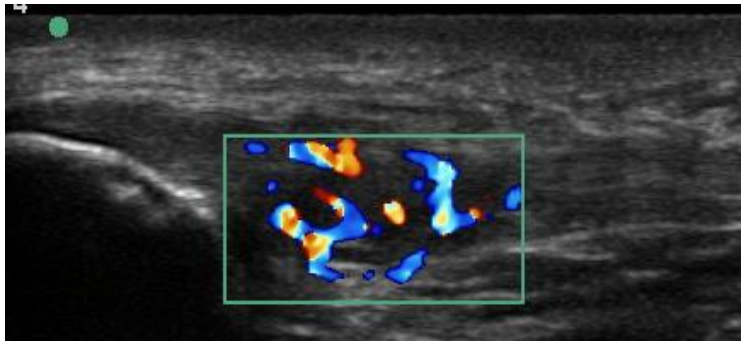
- The treatment of chronic injuries to this day still frequently focuses on treating perceived inflammation.
  - Ice, NSAIDs, CSI, etc.

    –> Provide short-term relief but may interfere with the tissue healing

- Those considered cured (because their pain lessened) often have residual structural damage
  - Reinjury occurs with attempted return to play





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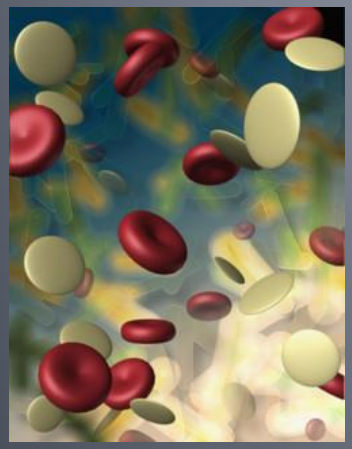
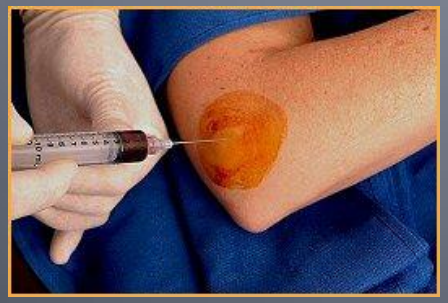
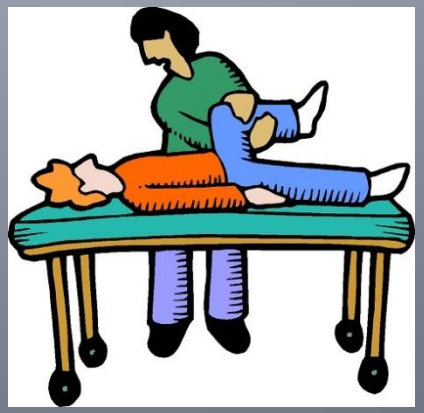
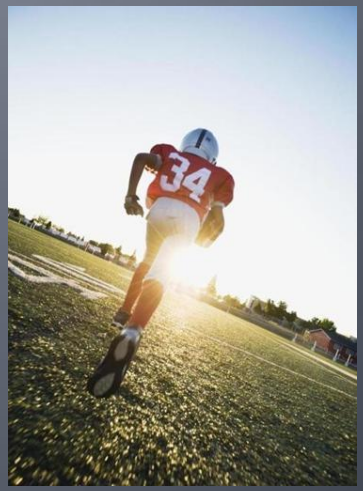
Optimizing outcomes after regenerative medicine therapy









Inflammatory Phase

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0 to 72 hours

- The tendon's injured site fills with platelets, white blood cells, and necrotic debris
  - Monocytes & macrophages predominate
    - They release proteolytic enzymes to degenerate the injured cells and tissue and convert it into necrotic debris
      - This leads to loss of tensile strength (tendon weakness)
    - Phagocytosis of necrotic debris occurs
  - Platelets release growth factors that stimulate the healing process

Leukocytes and Wound Healing

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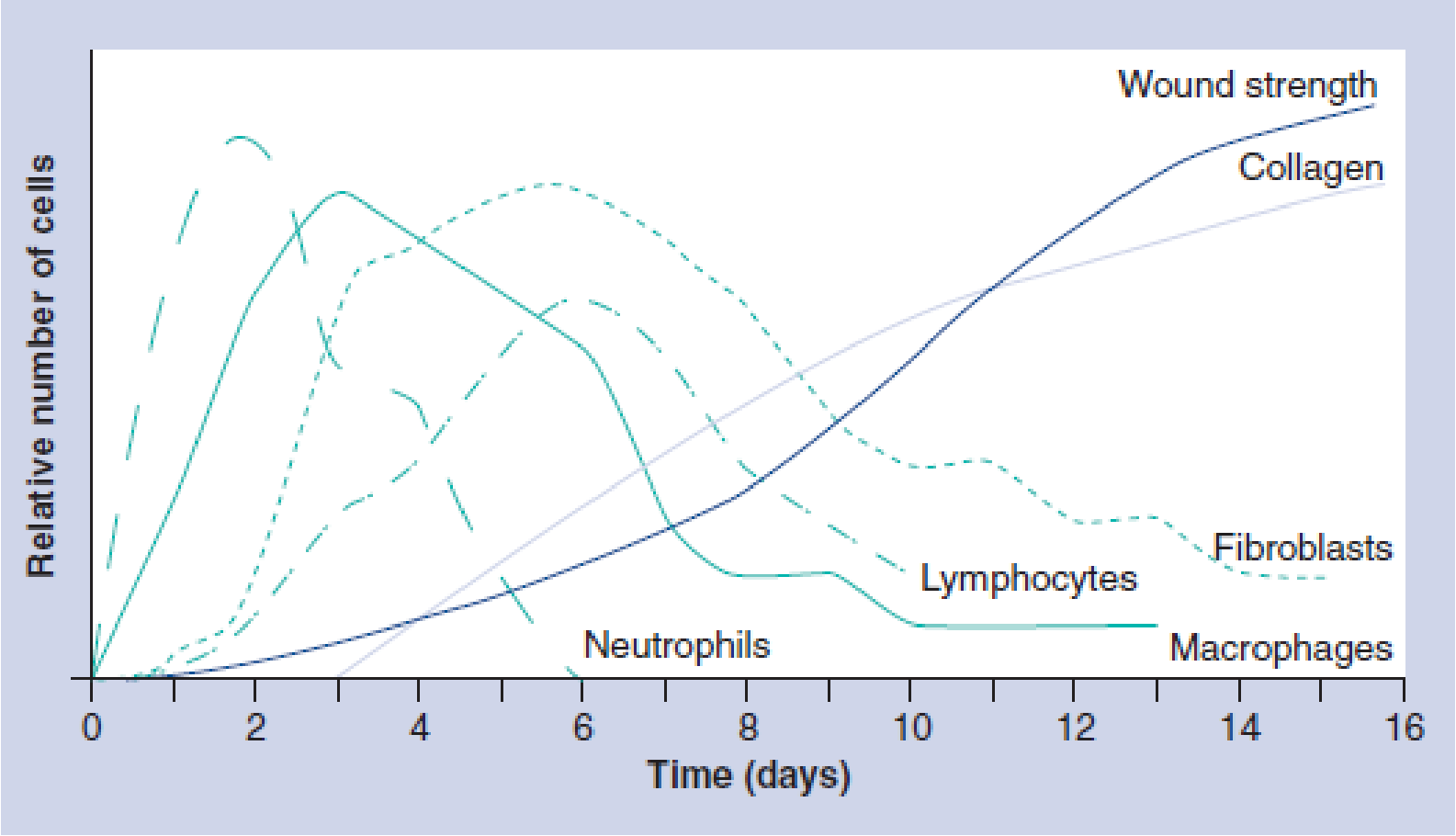

Proliferative (Repair) Phase

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Early Phase: day 2 to day 7

- Platelets attract fibroblasts to the injured site
  - Vascular & fibroblastic cells proliferate
    - Increase neovascularization to bring in nutrients
    - Start depositing Type III collagen to replace the injured tissue
  - Activate tendon cells to replicate

Late Phase: week 1 to week 6

- A new extracellular matrix of disorganized collagen fibrin forms, which leads to wound contraction
  - Ex. Think of the scab over an abrasion and skin under it

Maturation (Remodeling) Phase

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Early Phase: weeks 6 to week 12

- Fibroblasts *decrease* in size, slow down the matrix synthesis and eventually leave the site
- Collagen fibers and tenocytes orient themselves parallel to the long axis of the tendon
  - This is stimulated by mechanical tension
  - Tensile strength increases

Maturation (Remodeling) Phase

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Late Phase: Week 12 to 6 months

- Scar mass decreases
  - Type I:III collagen ratio increases
  - Tensile strength reaches its peak
- After 6 months, little histological difference is noted between healed and normal tendon.

Tendon healing potential

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- If the maturation phase is suboptimal or interrupted, the quality of the healed tendon may be vastly inferior to the original tissue
  - The tissue remains in a persistent hypervascular state
    - Myofibroblasts
  - Persistent production of cytokines and MMP leads to:
    - degeneration of the ECM
    - Increase in the hydrophylic ground substance and water
    - Thinner, disorganized, and separated collagen fibrils with low Type I:III ratio
  - Fibrosis may replace the elastin in the tendon

Optimizing outcomes after Regenerative Medicine Therapy

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Mautner K, Malanga K, Colberg R. "Optimization of ingredients, procedures and rehabilitation for platelet-rich plasma injections for chronic tendinopathy." *Pain management* 1.6 (2011): 523-532.

- Factors that influence the effectiveness of PRP:
  - High platelet concentration
  - leukocyte rich
  - Neutral pH of the injected substance
  - No need for activators
  - the method of delivery (ultrasound guidance)
  - **Post-injection Rehabilitation Protocol**
    - Tissue healing is directly related to the phases of wound healing

Patella Tendon Interstitial Tear treated with PRP

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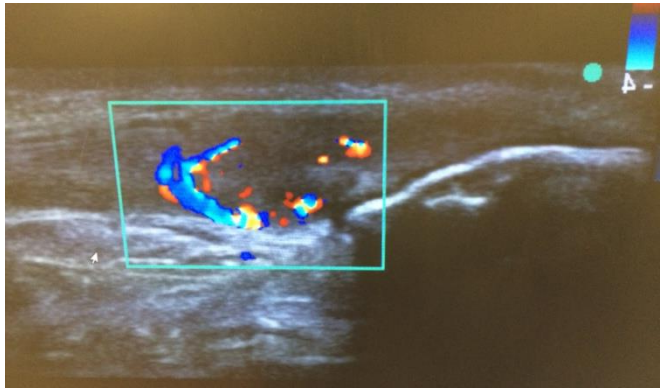
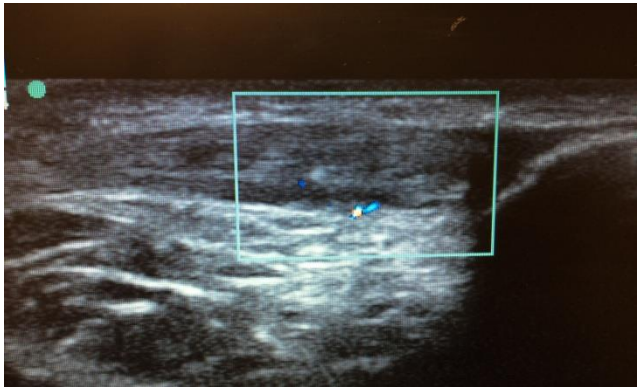
- 23 y/o male baseball pitcher with 5 year history of severe left patella tendon pain due to a high grade partial interstitial patella tendon degenerative tear treated with PRP.





3 months later

Intra-patellar tendon debridement

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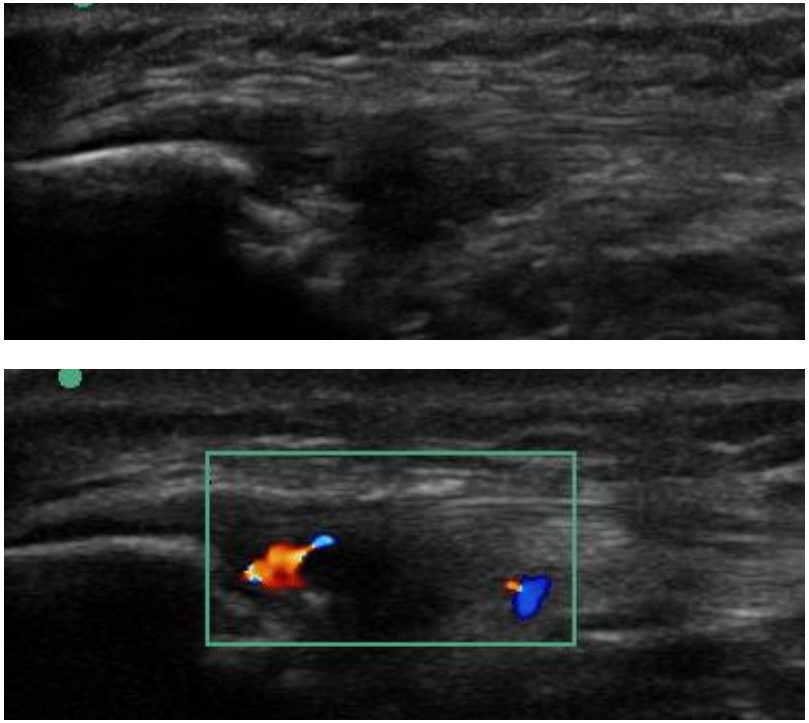
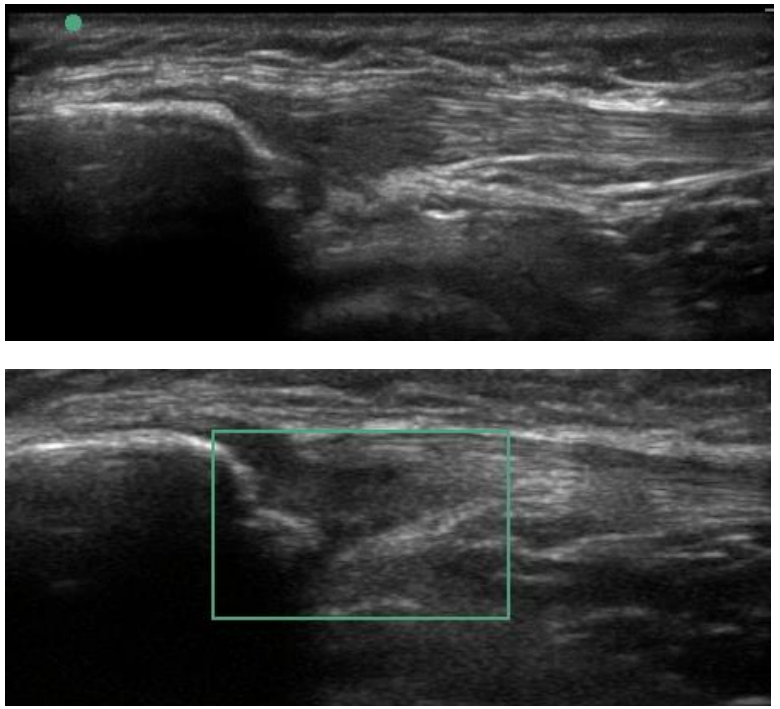
Javer A, Colberg, RE. "13 years old gymnast with chronic knee pain". Poster presentation at the AMSSM Annual Meeting, April 25 – 29, 2018.





Pre-procedure

10 weeks after procedure

(using Plasma field coablation debridement)

Sources of orthobiologics

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- Autografts (Adult cells):
  - Blood (PRP)
  - Bone marrow (BMAC and BMA)
  - Adipose (fat) tissue
  - Other soft tissue
- Allografts (embryonic cells):
  - Amniotic and chorionic membrane
  - Umbilical cord
  - Embryon (i.e. Blastocyst)

Acute vs Chronic Injury Outcomes

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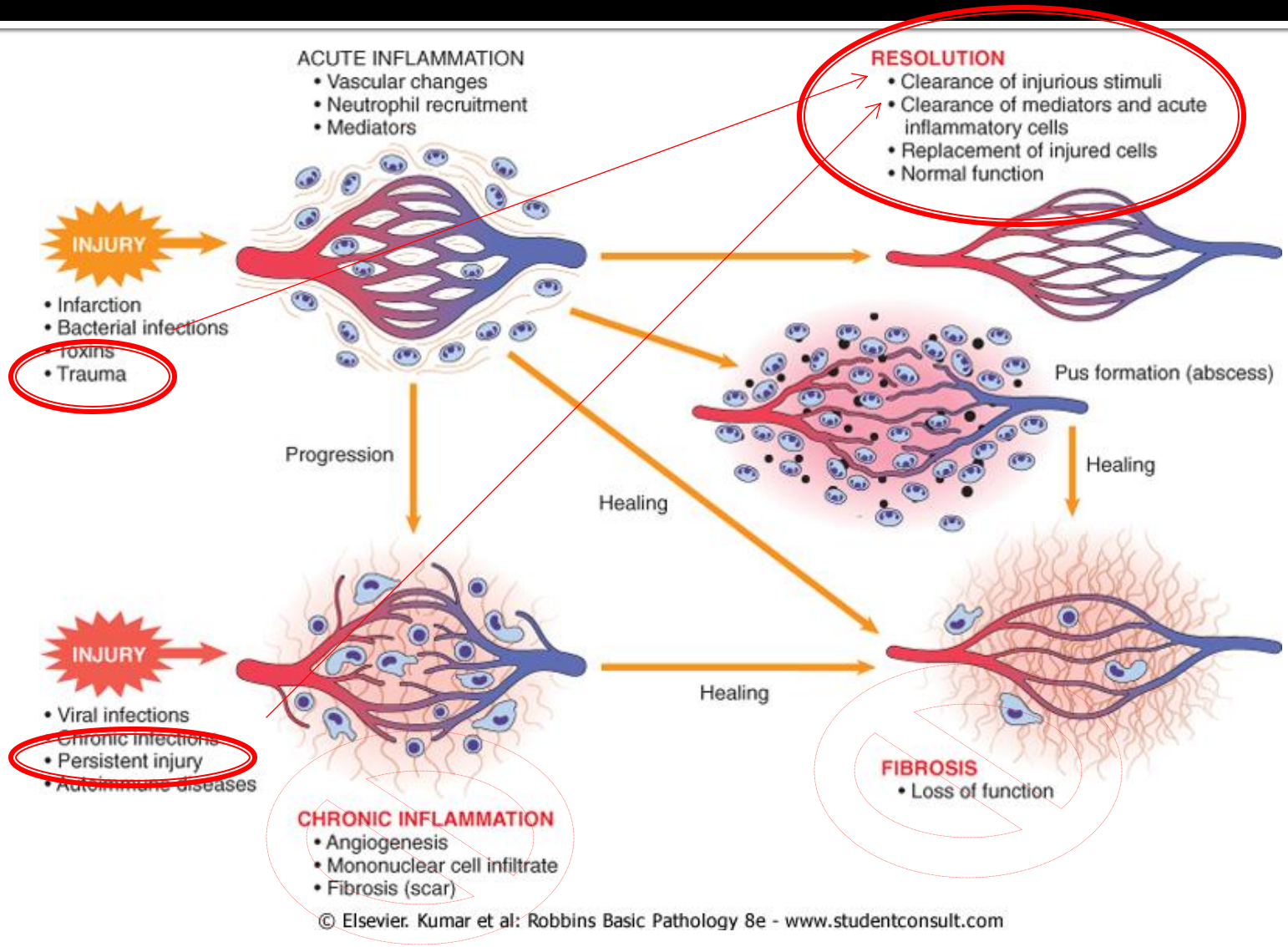

Conclusions on Regenerative Medicine Therapy

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- PRP, BMAC and percutaneous tenotomy are promising alternatives with minimal risks involved.
  - They still lack undisputable evidence that they will work
- The treatment should be individually tailored to the patient's needs and expectations.
- A rehabilitation protocol that progressively increases the load and activity in accordance to the tissue healing cascade is required for optimal outcome.

Abnormal Neovessels

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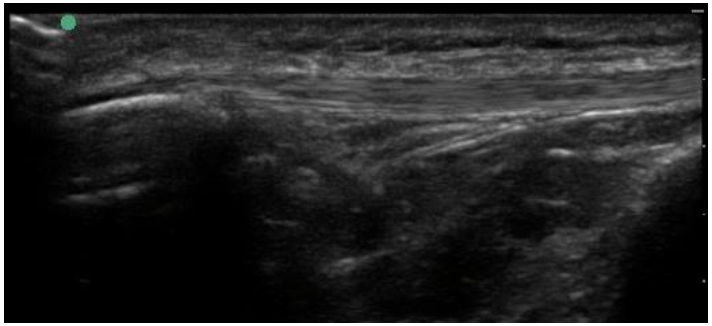

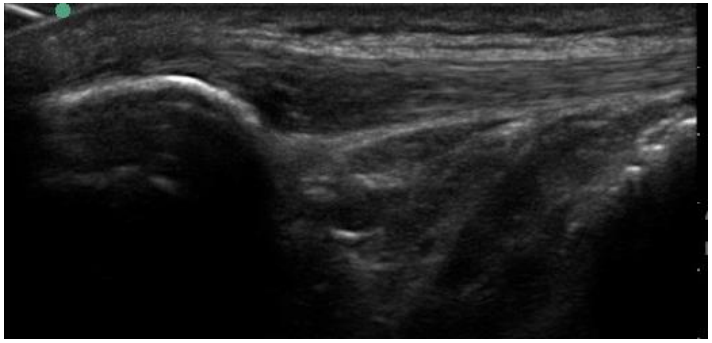

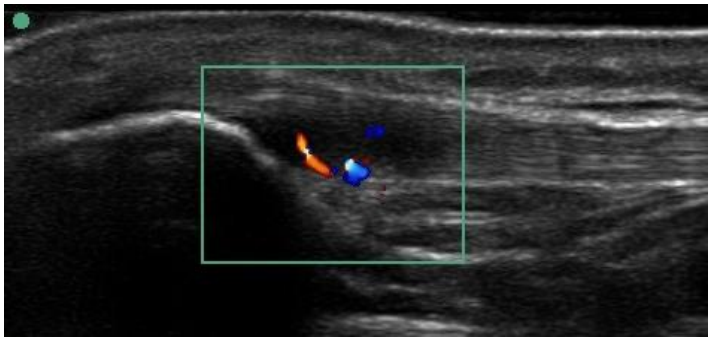

Patient with an intra-substance tear

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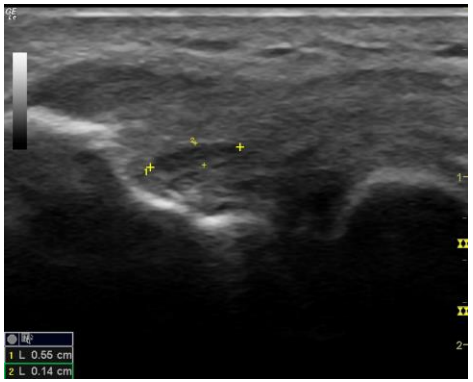
- The tear may have occurred from prior steroid injections performed or from progression of the tendon degeneration.
  - Similar cases are seen with "greater trochanter bursitis" s/p multiple cortisone injections that progress to partial gluteus medius tendon tears.
    - Was it tendinitis from the beginning and not bursitis???



Eccentrics vs concentrics

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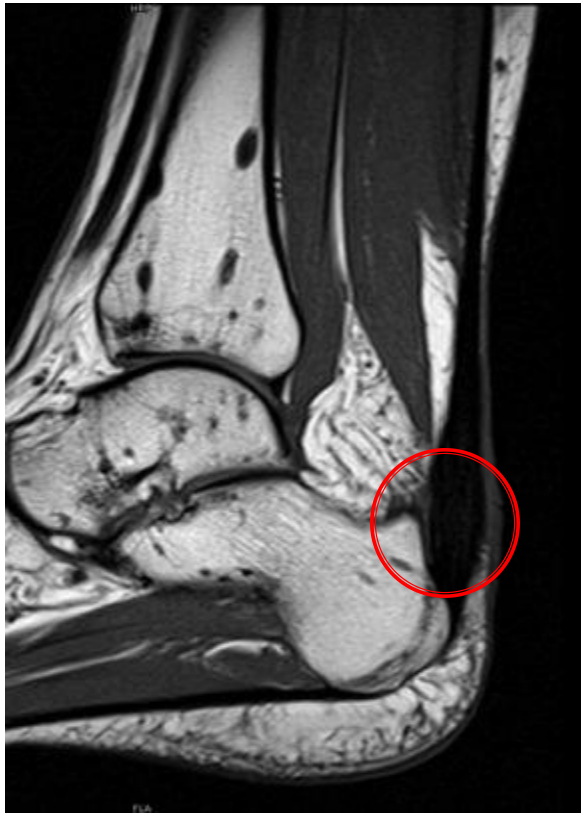
- Eccentric exercises
  - Pros: Stimulate healing and stretch the tendon to restore full ROM
  - Cons: Compressive force on the tendon insertion
  - When: Ex. Midportion Achilles Tendinopathy
- Concentric exercises
  - Pros: Stimulate tendon without compression
  - Cons: Sub-optimal for stretching
  - When: Ex. Insertional Achilles Tendinopathy



Ground-breaking study on PRP

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Peerbooms JC. et al.

- Randomized, double-blind, placebo-controlled trial of PRP versus CSI for lateral epicondylitis
  - AJSM 2010 and AJSM 2011

- Conclusion: PRP reduced pain, significantly increased function, and exceeded the effect of corticosteroid injection in lateral epicondylitis.

1 vs 3 PRP injections

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- Charousset, Christophe, et al. "Are multiple platelet-rich plasma injections useful for treatment of chronic patellar tendinopathy in athletes? a prospective study." *The American journal of sports medicine* 42.4 (2014): 906-911.
  - Methods: 28 athletes (17 professional, 11 semiprofessional) with chronic PT refractory to nonoperative management received 3 consecutive injections 1 week apart. Tendon healing was assessed with MRI at 1 and 3 months after the procedure, and functional outcomes followed for two years.
  - Results: 75% athletes returned to their pre-symptom sporting level at 3 months. Follow-up MRI showed improved structural integrity of the tendon at 3 months.

Adverse event after PRP

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- Redler, Andrea, et al. "Rupture of the Patellar Tendon After Platelet-Rich Plasma Treatment: A Case Report." *Clinical journal of sport medicine: official journal of the Canadian Academy of Sport Medicine* (2018).
  - 40 y/o male soccer player ruptured his patella tendon after 4 PRP injections

- Some questions arise:
  - Was it age related?
  - How many injections are too many?
  - Is there a degree of severity for which PRP should not be offered?

Future of Regenerative Medicine

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Many questions remain unanswered. For example:

- What is the best procedure for each injury?
  - BMAC vs PRP vs tenotomy
- What controls stem cell proliferation?
  - Can orthobiologics cause cancer?
- How much improvement is due to "placebo effect"?
  - May play a role when the patient pays out of pocket
- Is the statistically significant improvement truly clinically relevant?
- "First do no harm" - Should we offer tenotomy before surgery if they both have similar outcomes?
- Are we delaying the inevitable (i.e. surgery)?

Anatomy and Function

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- Tendons = load bearing structures that *transmit muscle forces to bone, allowing movement*

Tendon Composition

- Collagen (60% of the tendon)
  - Type I (95%)
    - Extensively cross-linked
    - Provides stiffness
  - Type III
    - less organized
    - Found at the insertion sites
  - Type V
    - regulates fibril growth
- In tendinopathy, the ratio of Type I:III fibers decreases, weakening the tendon.

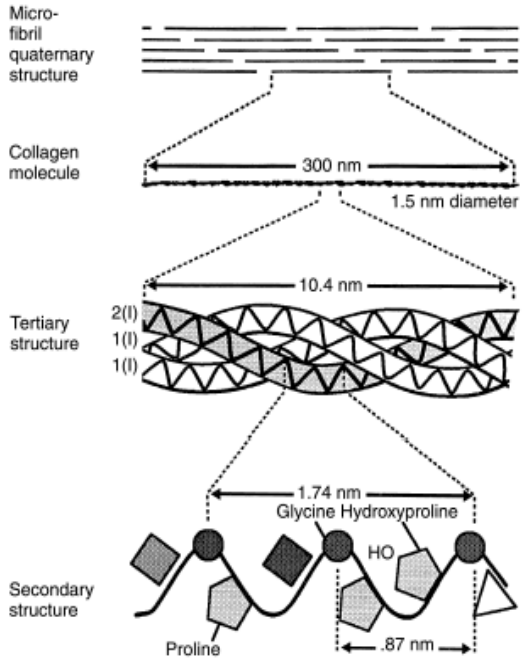

Figure 4

Schematic drawing of structural organization of collagen into the microfibril.

Tendon Composition

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- Non-collagenous Components (40% of the tendon)
  - Tenocytes
    - Fibroblast-like cells responsible for synthesis of the extracellular matrix (ECM)
  - Glycosaminoglobulins (GAGs)– proteoglycan & hyaluronan
    - Keeps ECM hydrated
    - Allows fibril sliding
  - Water
  - Elastin
  - Tenascin-C
    - May play a role in collagen fiber alignment and orientation