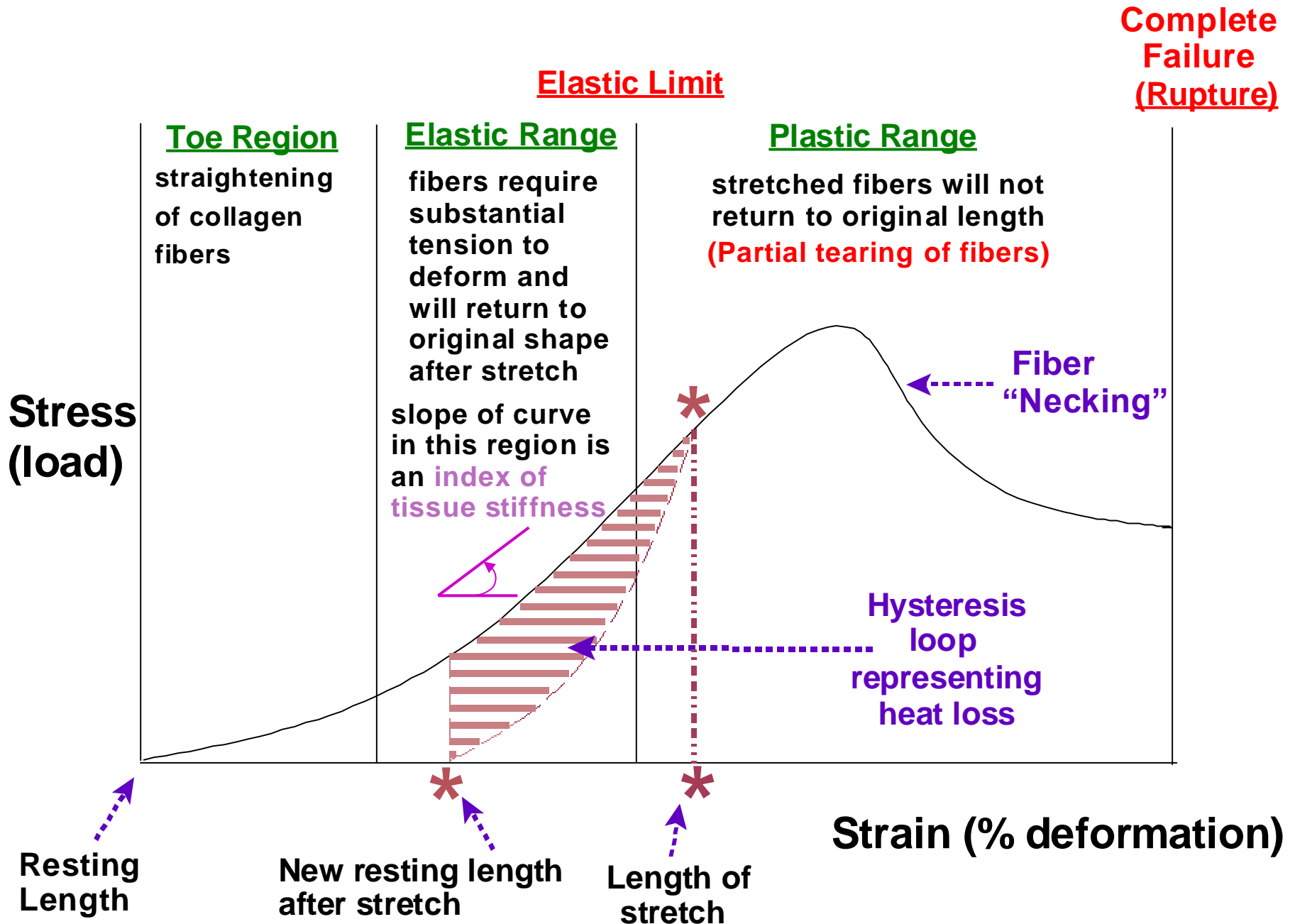


STRESS - STRAIN CURVE OF MUSCULOTENDINOUS TISSUE



Microtrauma

- Microtrauma (“overuse syndromes” “cumulative cell-matrix adaptive response”)
- Repetitive maximal or submaximal movement in the “elastic range” causes:
 - Pathological microscopic tears or lesions → inflammation and or tissue malformation
 - Overwhelming of cell metabolism → can’t maintain structural integrity by ↑ tissue synthesis
 - **Tissues that are formed in response to this overload stimulus are of inferior quality**
- Responsible for 30% - 50% of sports injuries (Herring & Nilson, 1987)
- These types of injuries are very “individualistic” in nature
- Structures most often affected in microtraumatic syndromes:
 - Bursa - synovial fluid sacs that ↓ friction between: skin-bone, tendon-bone, muscle-muscle
 - Tendon sheath (paratenon - fat & areolar tissue - “extended bursa” surrounding tendon)
 - Tendon (tendonopathies)
 - Bone (stress fractures)
 - Muscle (delayed onset muscle soreness)
- Symptomology progression of microtraumatic overuse syndromes:
 - Phase 1: pain after activity (immediate - 12 hours) which is palpable at injury site
 - Phase 2: pain during & after activity - no significant impairment - eventually resolves
 - Phase 3: pain during & after activity - significant impairment - eventually resolves
 - Phase 4: pain all the time accompanied by significant impairment - no resolution

Currently Accepted Theory as to the Cause of DOMS

Dr. Bob Armstrong - TAMU muscle biology laboratory - 1991

- Eccentric contractions → sarcomere inhomogeneities + sarcolemma (membrane) disruption
 - Disturbance in Ca^{++} homeostasis reduces ATP production → ↓ [ATP]
 - Intracellular [Ca^{++}] ↑ faster than Calcium extrusion mechanisms can pump it out
 - Cellular membrane destruction → cell death
- Prostaglandin & histamine → edema and inflammation → stimulation of free nerve endings
- ↑ protease & phospholipase activity begin to degrade the myofibril & associated membrane
- Phagocytes & macrophages invade cell 2-6 hours after the injury - continues for 2-3 days
- Cells adapt → future bouts of the same exercise causes less injury

Armstrong et al. (1991) Sports Medicine 12 (3) 184-207

MRI of Delayed Onset Muscle Soreness

- Note ↑ signal intensity in entire bicep compared to triceps



Tendinitis or Tendinosis ????

- **Tendinitis** - inflammation of tendon (rare) – inflammation of sheath is more common
 - the correct terms for inflammation of the tendon sheath are "tenosynovitis" & "paratenonitis"
- **Tendinosis** - degeneration & deleterious changes in tendon without inflammation
- **What most clinicians in the past have termed "tendonitis" is actually "tendinosis" or more generally, "tendinopathy"**
 - **Both tendinitis & tendonopathy can occur together!**
- **Regardless, the following observations for the malady may be made:**
 - The problem is most often accompanied by :
 - tissue degeneration (necrosis) and vascular abnormalities (disorganized vascularization)
 - collagen disorganization & fiber separation by mucoïd or lipoid ground substance (→ inferior tissue)
 - The problem may lead or contribute to complete or partial tendon rupture
 - Chronic tendonopathy → fibrinous adhesions → diminished tissue strength & function
 - **Although cause & etiology are debatable, recent opinions purport causes to be related to:**
 - The sliding of tendon over other structures (compressive forces)
 - Subjecting the tendon to loads close to tensile strength & exceeding its anabolic capabilities
 - Eccentric contractions
 - Negative direction on force velocity curve → what about plyometric training??????
 - Chronic anabolic steroid use → ↓ tissue quality and tensile strength → ↑ incidence of tendon rupture
 - Malnutrition influences: ↓ vitamins A & C, ↓ copper → ↓ collagen synthesis and crosslinking
 - Menopause → ↓ [E₂] → ↓ connective tissue elasticity → ↑ tendinitis & other overuse injuries
 - Note: Collagen production is impaired in smokers → Tendinopathies heal slower

General Steps in the Healing of Tendon Microtrauma Pathology

<u>Stage</u>	<u>Pathology, Healing, & Objectives</u>	<u>Treatment Implications</u>
Inflammation (if present) (days 1 - 6) Some authors: (day 1 or 2 only)	<ul style="list-style-type: none">- ↑ GAG & collagen synthesis- Prevent prolonged course of inflammation (if present)- Prevent injury to developing collagen	<ul style="list-style-type: none">- REST (activity cessation)- NSAID's, oral corticosteroids- Low level ROM exercises- Cryogenic therapy- Load reducing devices: casting, bracing, heel lifts, etc.
Fibroplastic Proliferation (days 5 - 21)	<ul style="list-style-type: none">- ↑ rate of collagen synthesis by fibroblasts- Synthesized collagen fibers need to be aligned	<ul style="list-style-type: none">- Low level / low duration exercise- Stretching → align collagen- Cryogenic-thermogenic therapy- Ultrasonography - Laser- Electricity → ↑ fibroblast activity
Remodeling Maturation (20 days – 6 months)	<ul style="list-style-type: none">- ↑ rate of crosslinkage formation & fibril size- Replacement of initial and / or inferior tissues	<ul style="list-style-type: none">- Strengthening exercises- ↑ intensity & duration- Stretching exercises to further stimulate collagen alignment



Note: almost all body tissues follow this paradigm of healing only with a different timeframe

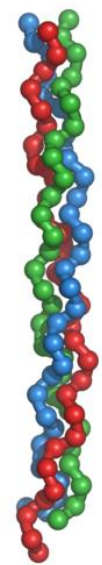
Immobilization vs. Mobilization: A Fine Line

• Effects of immobilization on injured tendinous tissue

- Protein degradation exceeds protein synthesis → net ↓ in collagen quantity
- Reduction in the number of collagen crosslink bonds
- Atrophy of tissues at myotendinous (muscle - tendon) junction
 - **2% loss of total collagen mass at 9 weeks.....27% loss at 12 weeks.**

• Benefits of mobilization (movement) on injured tendinous tissue

- The cross-sectional area of the healed tendon is greater
- Improvement in collagen fiber type and fiber arrangement in the repair
 - Type I – most abundant type humans – highest tensile strength of the 3 types
 - In tendons, muscle endomysium, fibrocartilage, bone, “final” scars
 - Type II - found in hyaline cartilage
 - Type III – found in granulation tissue, reticular tissue, & basement membranes
- Greater number of crosslink bonds
- Better quality of ground substance in the tendon



Common Therapies for Tendon Microtrauma

NSAID drugs

- In the 80'S and 90'S, approximately 73% of studies show NSAID's to be effective in:
 - ↓ healing time ↓ inflammation
- Other, more recent studies show **no measurable benefit of NSAID's**
 - Note: Approximately 50% of those using NSAID drugs will have adverse side effects

Corticosteroids

- **Oral Corticosteroids:** Short term use of such drugs as (**MEDROL DOSEPACK, PREDNISONE**) appear helpful for some ailments including:
 - Neuritis
 - Paratenonitis (Conditions where inflammation is indeed present)
 - Bursitis
- **Corticosteroid (Cortisone) Injections** [Injections:http://orthopedics.about.com/cs/paindrugs/a/cortisone.htm](http://orthopedics.about.com/cs/paindrugs/a/cortisone.htm)
 - **Will reduce pain but usefulness in ↓ inflammation & ↓ healing time has not been proven**
 - Side effects: collagen disarray, tendon rupture, skin depigmentation, atrophy at injection site
 - Used only after a 6-week trial of rest then re-conditioning
 - Used only when site of pain is palpable - avoid injection directly into tendon (inject in sheath only)
 - Allow 2 - 6 weeks after injection before re-conditioning
 - Avoid more than 3 injections
 - Avoid injections just prior to competition (↓ pain → ↑ likelihood of injury exacerbation)

Surgery - excision / debridement of damaged tissue - release & repair

- Replace “bad scar” with “good scar” - not always successful (success rate 70% - 90%)
- Does not remove microtraumatic injury stimulus – same recurrent stimulus → problems can re-appear

Chondromalacia – “Runner’s Knee” – “Patellofemoral syndrome”

Chondromalacia – degeneration & inflammation of articular cartilage:

- Usually affects the underside of the patella (most common)
- Often involves bottom of femur and top of tibia
- More common in women (greater Q-angle → ↑ lateral forces on kneecap)

Causes

- Excessive running with tight hamstrings, tight calf muscles, pronation from ITB syndrome
- Muscle imbalance results in a tracking abnormality of the tibia in the femoral groove
 - Vastus lateralis tends to be more powerful than the vastus medialis

Risk Markers & Clinical Signs

- Q-angle > 15 degrees (associated with Genu Valgum – “knock knees”)
- Pain with contraction of quadriceps while patella is held in groove

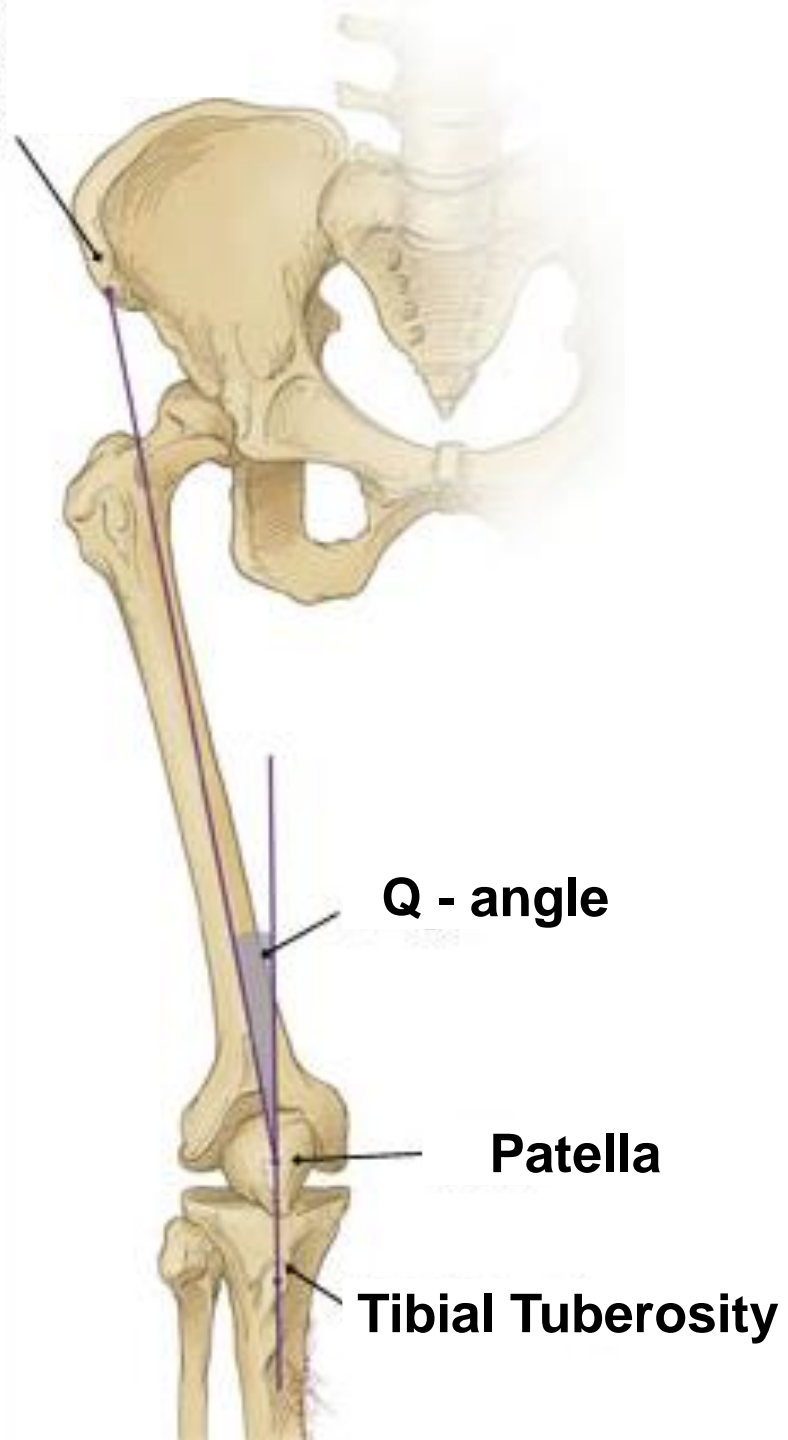
http://www.return2fitness.co.uk/injury_advice/knee_injuries/cmp.php

Treatment – 1st: removal of inflammatory stimulus

- **RICE** – NSAID’s
- Orthotics to correct pronation
- Stretching to ↑ flexibility in hamstrings, calf muscles, & ITB
- Strengthen quads, hamstrings, hip flexors, hip adductors, and hip abductors
- Avoid downhill running (eccentric contractions) when returning to activity
- Surgery
 - 1. Lateral release (cutting lateral patellar retinaculum → better patella tracking)
 - 2. Smoothing down the underside cartilage of the patella
 - Successful about in 90% of cases

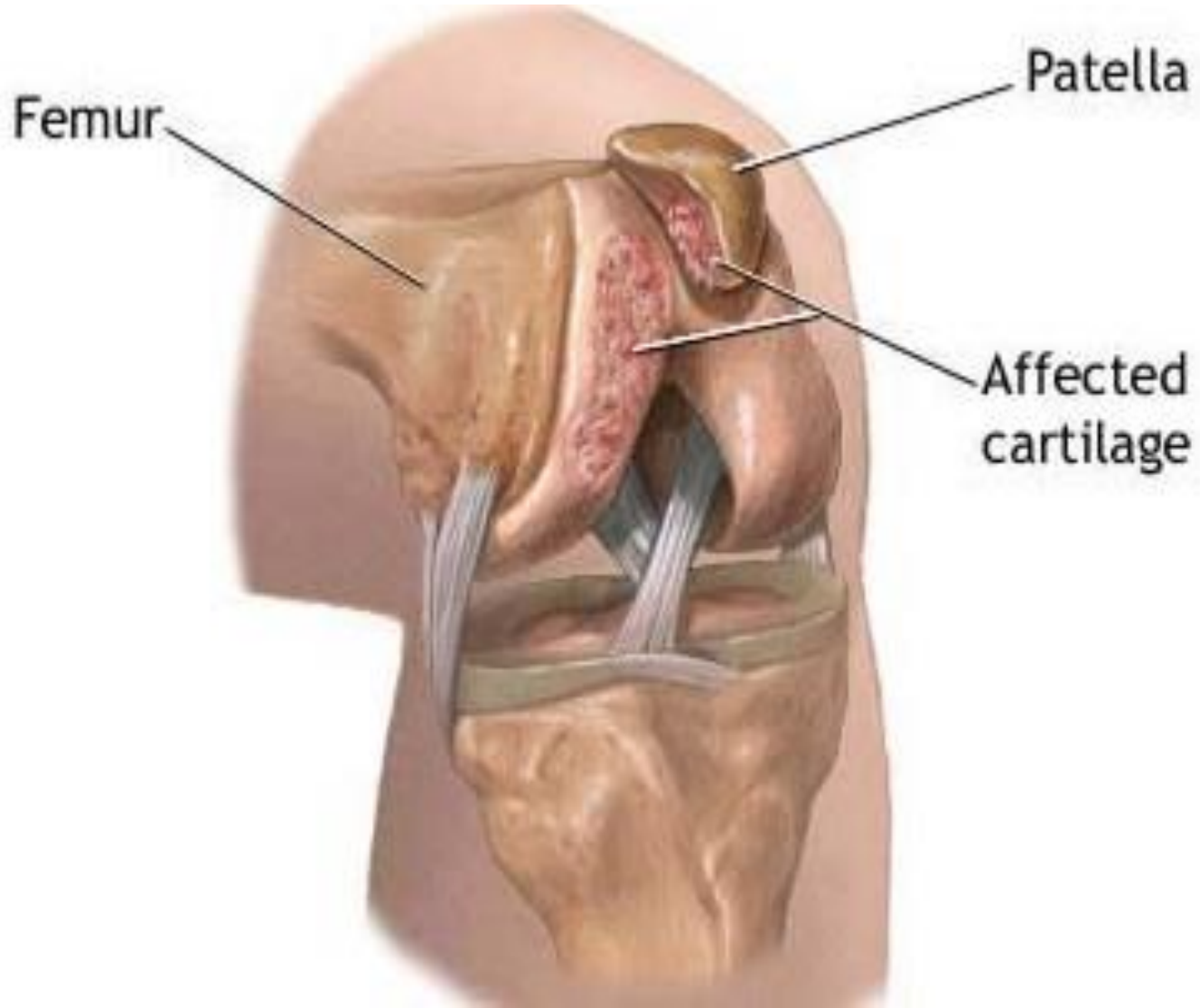
**Anterior
Superior Iliac
Spine**

The Q - Angle



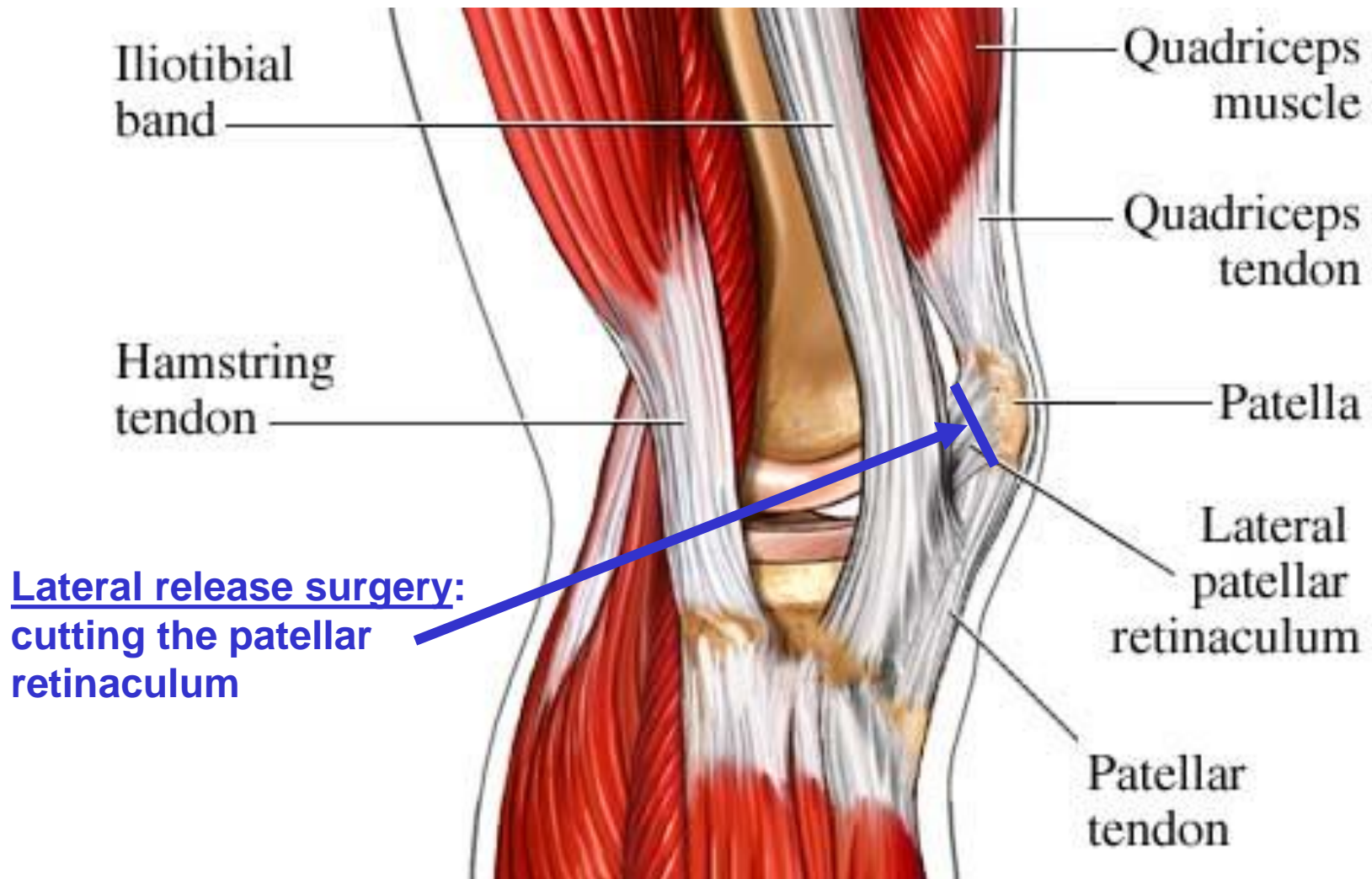
Chondromalacia – “Runner’s Knee” – “Patellofemoral syndrome”

Left Knee



Chondromalacia – “Runner’s Knee” – “Patellofemoral syndrome”

Right Knee



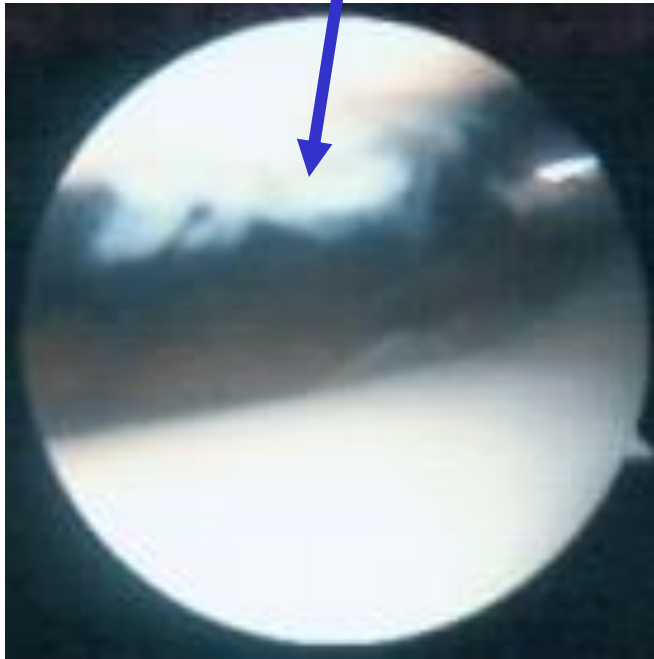
Lateral release Using Thermal Cauterizing Scalpel



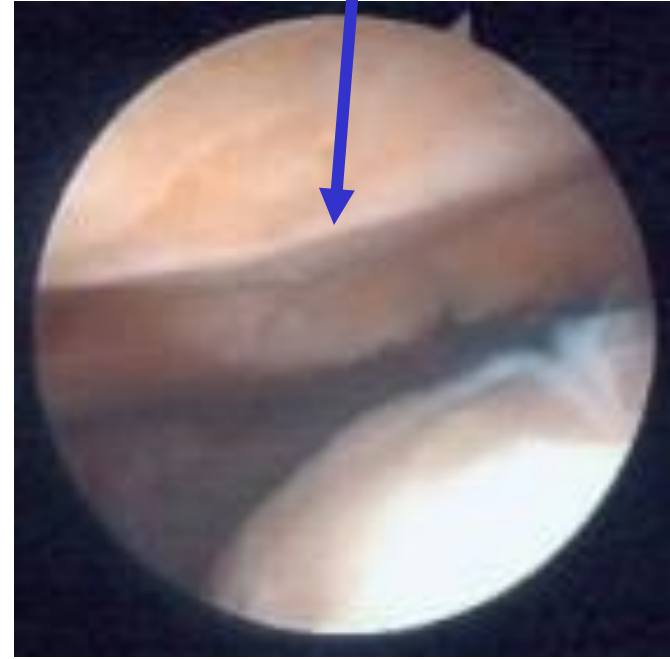
Chondromalacia – “Runner’s Knee” – “Patellofemoral syndrome”

Arthroscopic / Laser Correction of Chondromalacia

White “whispy” material is roughening of the cartilage of the underside of the patella



New smooth surface after laser removes damaged cartilage



Patellar Tendinitis (Tendinosis / Tendinopathy) – “Jumper’s Knee”

Patellar tendinitis – degeneration and or inflammation of patellar tendon or tendon sheath

- Usually affects participants of “explosive” sports involving jumping or quick movements
 - Basketball & volleyball players most affected (frequent “stops”, “starts”, & “jumps”)
- Once again, the controversy – tendinitis vs. tendinosis (tendinopathy)
 - Most now agree that most tendon overuse injuries involve little inflammation
 - Mucoïd deposits (soft greenish brown disorganized tissues) are present
 - Patellar tendonopathy may be accompanied by micro-ruptures and necrosis
 - It may predisposes the tendon to rupture

http://www.return2fitness.co.uk/injury_advice/knee_injuries/jumpers_knee.php

Causes

- Excessive activity (especially a rapid increase in the frequency or intensity of training)
- Improper mechanics of training
- Excessive weight on person doing a lot of weight bearing exercises

Symptoms

- Pain / tenderness in the patellar tendon below the knee when jumping, running, or walking
- Pain or "tightness" in the knee when bending, squatting, or straightening the leg

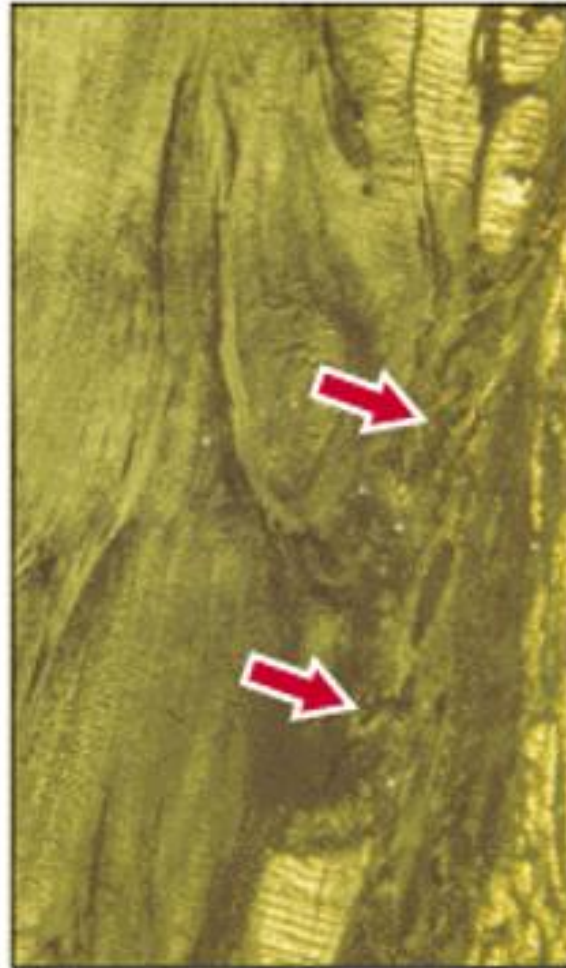
Treatment- first, removal of injury stimulus

- **RICE** – emphasis on rest followed by quadriceps strengthening exercises
- Ultrasound & laser photostimulation → increase collagen synthesis
- NSAID’s & corticosteroids have, for the most part, found to be ineffective
- Surgery
 - Excision of the damaged portion of tendon

Patellar Tendinosis



Normal Patellar Tendon



Patellar Tendinosis – note greenish-brown mucoid appearance with tissue degeneration (red arrows)

Achilles Tendinosis

- [1] ↑ signal intensity → fluid + tissue degeneration
- [2] Calcaneus

Histopathology of Achilles Tendinosis

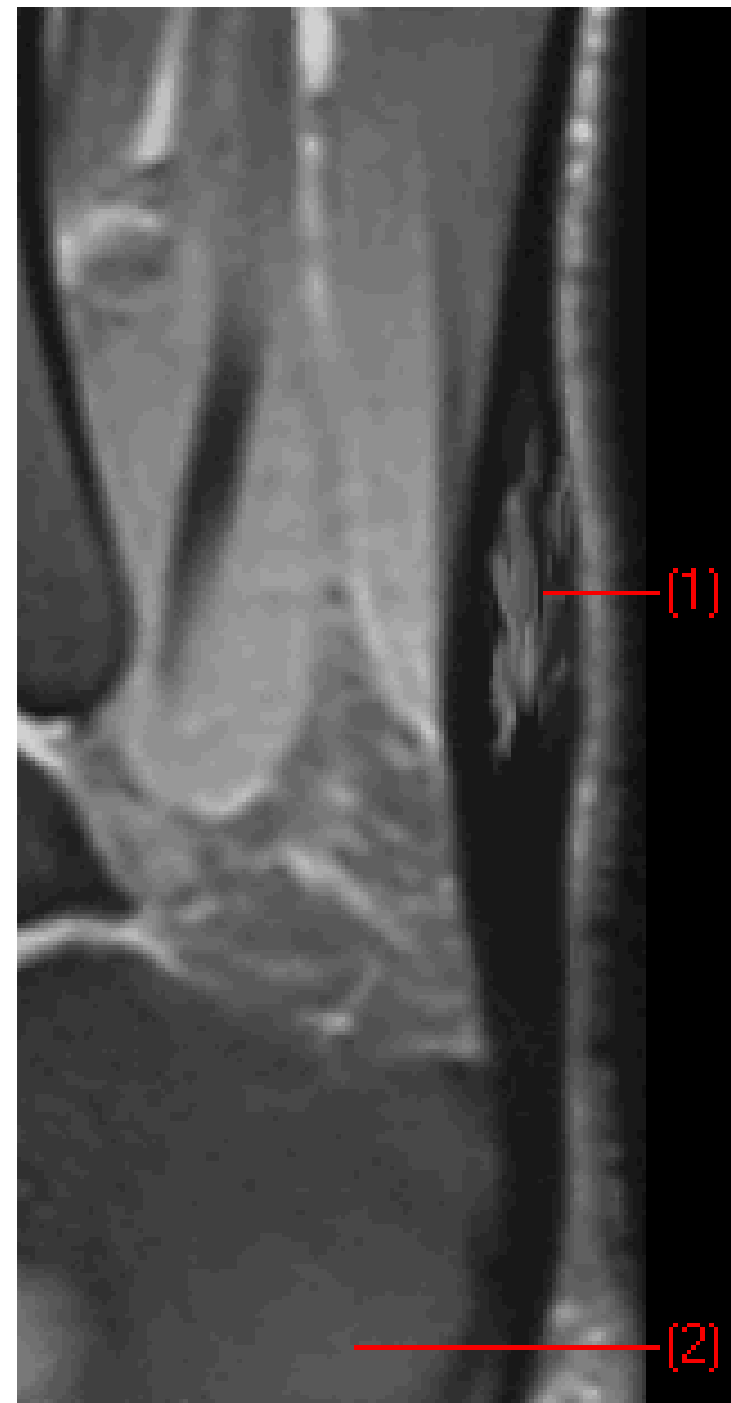
- ↑ vascularity (neovascularization)
- Collagen fiber disorganization
- Thinning of tendon fibers
- Mucoid or lipid deposits between fibers
- Inflammatory cells at sites of micro-rupture (rare)

Symptoms & Diagnosis

- Painful push-off when walking or running
- Palpable nodule on achilles tendon
- Tendon may be swollen or thickened

Treatment

- Cessation of activity and / or immobilization
- Stretching exercises
- Modalities
- Heel lift orthotics
- Surgery in extreme cases
- Gradual return to activity



MRI of Achilles Tendinosis



Here again is an MRI of a chronic degenerative tendinopathy. Note the the thickening and swelling (↑ fluid) which is visible as the “whispy” light gray color (red line) on the regular black Achilles Tendon “tube” (blue line)

Achilles Tendinosis Surgery



Achilles tendon is exposed revealing a split in the tendon



The necrotic portion of the tendon is removed

The ends of the tendon are brought together and sutured

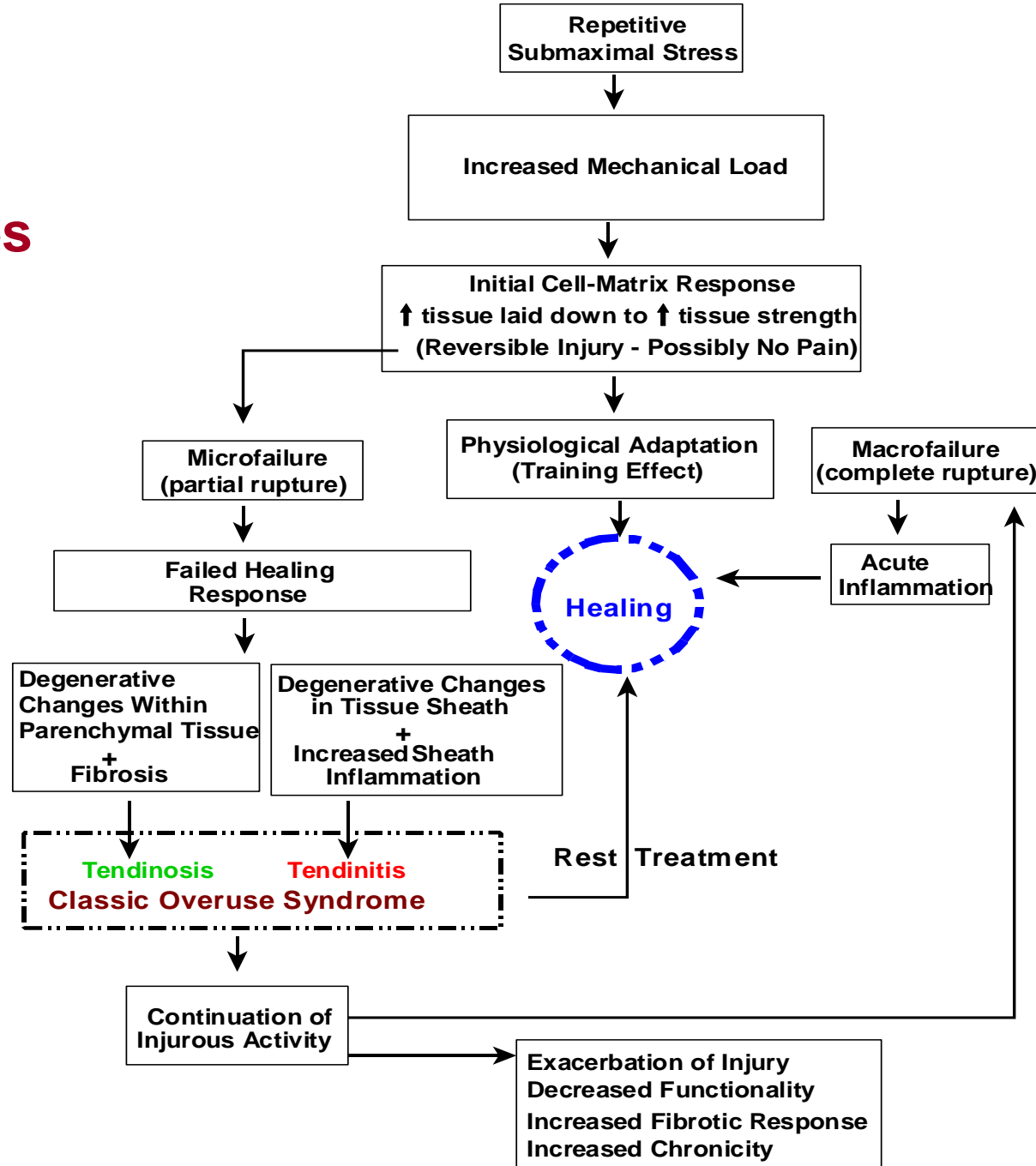


True Achilles Tendonitis (Inflammation of Paratenon)

- Diagnosed by pain syndrome and palpating the tendon where it is slightly thickened and tender along its inner edge, about four-centimeters above the attachment to the heel bone
- May be caused by excessive pronation in runners
- Treated with cessation of activity / **RICE**, physical therapy, orthotics to ↓ pronation, NSAIDS, heel lifts to reduce stress on tendon
- Physical therapy usually has the patient back to activity in 2 – 3 months
- Severe cases: surgery may be needed to remove inflamed paratenon

<http://emedicine.medscape.com/article/85115-overview>

Healing of Microtraumatic Tendinous Injuries



Stress Fractures (Hairline Fractures)

Bone Stress Continuum:

Stress Reaction  **Stress Fracture**

- **Stress Reaction:**
 - Accelerated adaptive bone remodeling
 - Most often asymptomatic
 - Occurs most often in tarsals, metatarsals, femur, & tibia
 - Sometimes seen on X-ray as increased callus (“bone”) deposits
- **Stress Fracture:**
 - Complete defect or “crack” in the bone
 - Easily seen on X-ray

Stress Fractures

- **Occurs only in race horses, greyhounds, & man**
 - Intensity and / or volume of activity is not natural
- **Most stress fractures heal by themselves without clinical manifestations**
- **Causes:**
 - Repetitive torque across a bone <http://orthoinfo.aaos.org/topic.cfm?topic=A00112>
 - Weight bearing impact forces
 - Bone does not adapt as well as muscle to impact forces
 - Inability of weak musculature to facilitate shock absorption
 - Low bone mineral density
 - Structural malalignments (leg length discrepancy)
- **Stress fractures ↑ risk of complete fracture at the stress fracture site**
- **Early stage stress fractures (stress reactions) don't show up well on X-rays**
 - Better detected by bone scan - shows up as “hot spots” (areas of increased remodeling)
- **Symptoms:**
 - Tenderness & pain over the fracture site and pain during activity
 - Early on, pain is light and occurs at end of activity....later....pain is more severe and occurs earlier
 - Pain relieved by rest but becomes progressively more frequent & persistent
 - Overlying soft tissue may exhibit swelling
 - Ultrasound or struck tuning fork placed over the fracture site will elicit pain
 - Muscle atrophy may occur at or near the stress fracture site

Stress Fractures

- **Types of stress fractures:**

- Oblique / Transverse
 - Angled to / perpendicular to the long axis of the bone
 - Most common
 - Dangerous due to the chance of complete fracture & bone “displacement”
- Longitudinal (parallel to the long axis of the bone)
- Compression

- **Two Common Examples of Stress Fractures:**

- Tibial stress fracture in runners
 - Weight bearing impact related
- Humerus stress fracture in a “thrower” (pitcher, javelin thrower, etc.)
 - Related to muscle trying to accelerate a “resisting” bone (torque)

Stress Reaction of Left Tibia

Bone Scan

"Hot Spot" may indicate fracture or stress reaction



Stress Reaction Site



X-ray Corresponding to Bone Scan

Stress Fracture of Tibia in a Runner

Note clearly defined **“Crack”**
in addition to **Extra Callus**

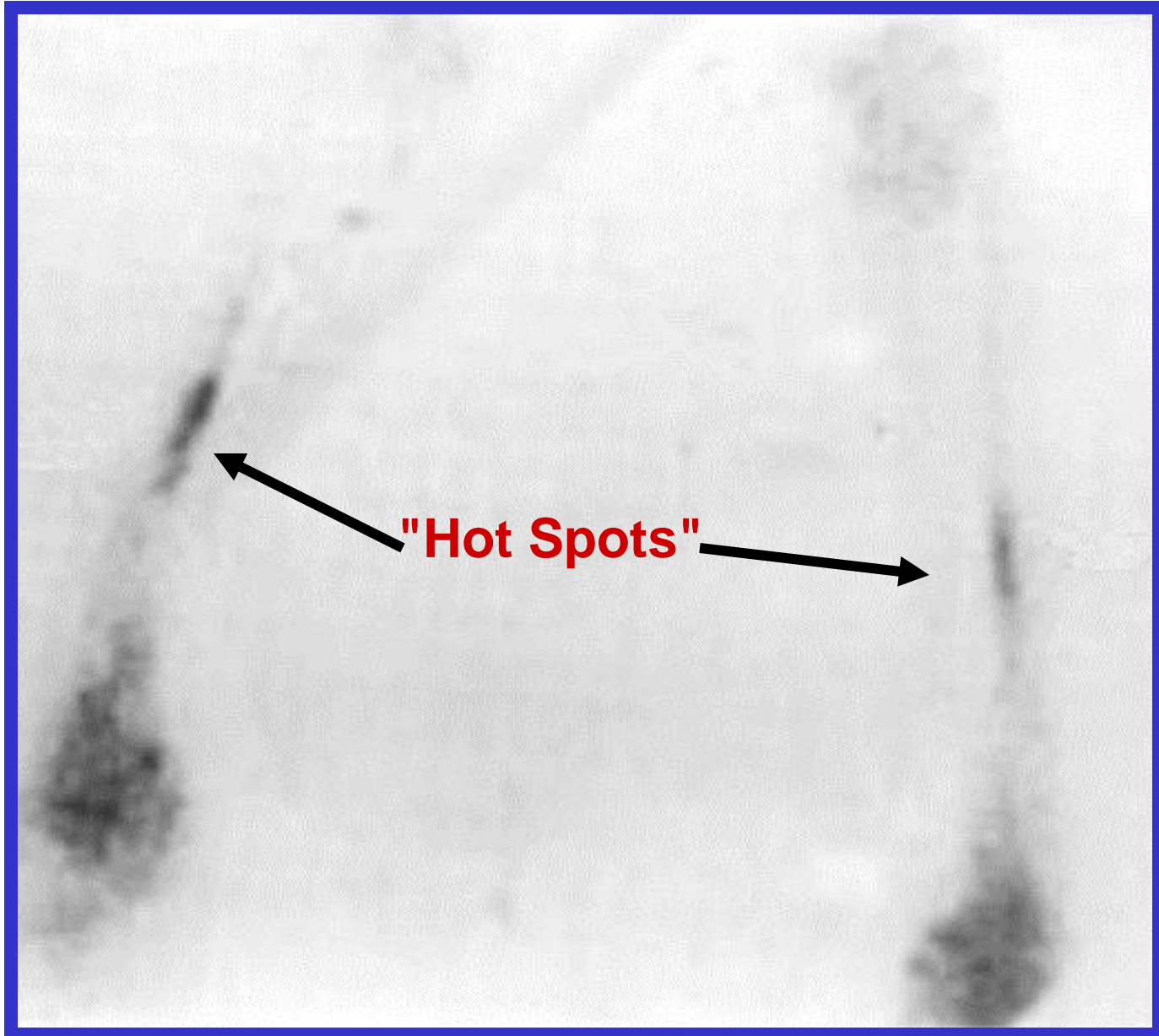


“Crack”

MRI of Stress Fracture of the Calcaneus



Bilateral Stress Fractures of the Ulna in a Weight Lifter



Stress Fractures

Stress Fracture of 3rd Metatarsal in a Runner

Note the extra callus increasing the girth of the bone

