Soft Tissue Injuries

Pathophysiology of damage and repair

DEFINITION

Tissue damage is defined as functional and structural defects resulting from traumatically induced biochemical, ultrastructural, microscopic and macroscopic changes.

What are the Soft Tissues?

- Connective Tissue
- Muscles
- Tendons
- Ligaments and Joint Capsules
- Cartilage
- Nervous Tissue

Connective Tissue

- Provide tensile strength, substance, elasticity and density to the body.
- Role in defence and repair.
- Fibroblasts
- Macrophages
- Mast Cells

Connective Tissue Matrix

- 80% of total tissue volume
- Collagen Fibres - ligaments, tendons, fascia, sheaths, bursae, bone and cartilage.
- Elastic Fibres
- Amorphous Ground Substance

Types of S.T.I.'s

- **TRAUMATIC: cause is identified with a specific incident or injuring force.**
- **GRADUAL OVERUSE: develop slowly and cannot be attributed to one single action.**

Injury Classification

- **ACUTE:** rapid onset, macrotraumatic event with a clearly identifiable cause.
- **CHRONIC:** slow insidious onset, gradual development of structural damage. Threshold episode usually heralded by pain or inflammation.
- **SUB-ACUTE:** period between acute and chronic, usually 4-6 weeks post-injury.

Types of Tissues Injured

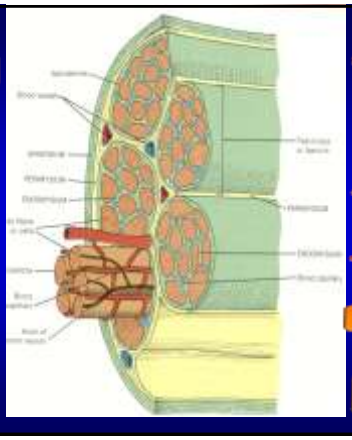
- **SKIN:** abrasions, blisters, bruising, lacerations, incisions.
- **MUSCLE:** contusions, haemotoma, strains, sprains, tears, cramps, spasms, myositis, fasciitis.
- **TENDON:** tendinitis, tenosynovitis.
- **BURSAE:** acute, chronic, compressive.

Muscle Injuries

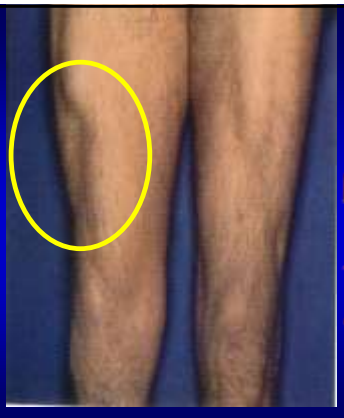
- prevention and rehabilitation

ACUTE

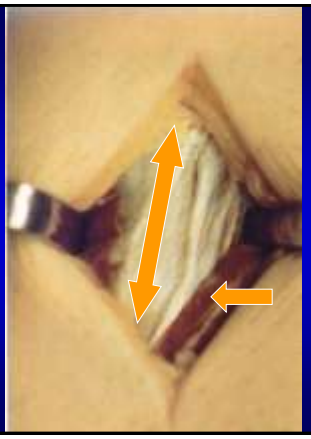
ACUTE



ACUTE DAMAGE

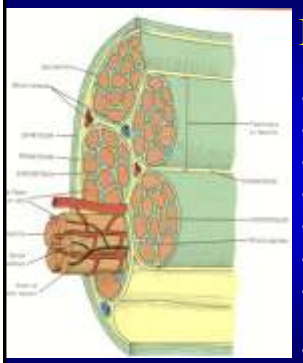


MUSCLE REPAIR



MUSCLE REHAB

Muscle Tissue



Muscle Tissue

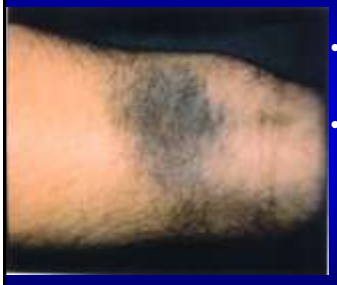
- Actin-myosin cross-bridges
- Fibres arranged in bundle
- Rich blood supply
- Rich neural supply
- Capable of contraction
- Vital for control of motion
- Muscle fibres surrounded by connective tissue
- Frequently injured

Muscle Damage

Muscle Damage

- Intrinsic Injury: Muscle damage occurs when the tensile strength of the muscle fibres is exceeded.
- Delayed Onset Muscle Soreness
- Lacerations
- Extrinsic Injury: Contusions

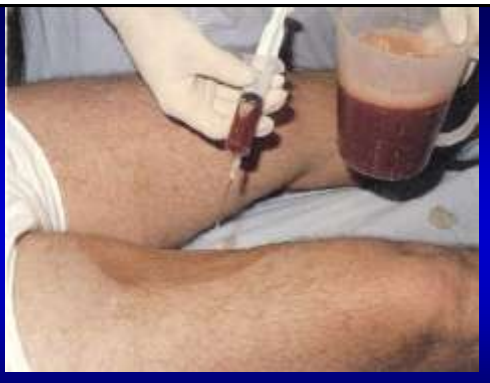
Extrinsic Injury



- Interstitial haemotoma.
- Damaged fibres and broken blood vessels have leaked blood into surrounding tissues.

Intramuscular Haemotoma

- Caused by a direct blow, i.e. a kick or knee from another player.



Aspiration of a haemotoma

Intrinsic Injury

Intrinsic Injury

Michael Owen ‘pulls’ a hamstring

T.O. Hamstring injury?

Muscle Repair

Tissue Repair Processes

The Cellular Level

Philip Evans (1980) The Healing Process at Cellular Level: a Review, Physiotherapy, 66, 8, 256-259.

What is Injury?

- Injury is the medical term for cellular damage
- Local network of blood vessels is damaged
- Lack of oxygenated blood = cell death
- Damaged vessels bleed
- Injured soft tissues consist of dead cells, extra-cellular substance and extra-vasated blood.

Stages of any Soft Tissue Injury

- Injuring force/action - initial damage
- Within 10-15mins - disrupted extra-cellular tissues, dead cells release powerful digestive enzymes
- Inflammation begins

Inflammation

- **Calor (heat):** increased blood flow to the injured area will cause an increase in temperature;
- **Rubor (redness):** with the increased perfusion comes a red/pink hue to the skin;
- **Dolor (pain):** caused by chemicals released by dying cells acting on nerve endings;
- **Tumor (swelling):** increases in blood flow and extra-cellular fluid - inflammatory exudate.

Repair and Regeneration

- Healed tissue is never the same as it was
- Fibrous connective tissues will be replaced but will not have the same structural properties
- Damaged muscles do not regenerate but heal by scar tissue
- Joint capsules thicken when healing due to the high levels of fibrin in the exudate
- Generally soft tissues heal by fibrous repair.

Healing and Repair

- Inflammatory response may last for 5 days or more
- Repair begins when nearby cells begin to actively divide
- They are surrounded by a mesh of fibrin and dead cell debris being phagocytosed by macrophages.

Factors Improving Healing

- You cannot accelerate the healing process
- Healing occurs by cell division and this cannot be forced
- We can provide optimum conditions for healing to occur
- Reduce limiting factors like ischaemia, irritation.

Fibrous Tissue Healing

- If the tissue being healed is kept completely immobile the fibrin mesh will be weak
- Natural movements should occur to increase the strength of the fibrin network
- Fibrin healing requires a good blood supply and is limited by haemotoma, buried sutures or infection.

Managing Healing Tissues

- Protection
- Rest
- Ice
- Compression
- Elevation
- ? Massage

Muscle Repair - Basic Physiology

- Phase 1
  - Inflammatory phase (Oakes, 1991)
  - Reaction phase (van der Meulin, 1982)
- Phase 2
  - Fibroblastic phase (Prentice & Bell, 1990)
  - Repair phase (Oakes, 1981)
  - Matrix and cellular proliferation phase (Oakes, 1992)
  - Regeneration phase (van der Meulin, 1982)

Muscle Repair - Basic Physiology

- Phase 3
  - Maturation phase (Oakes, 1992)
  - Remodelling phase (Oakes, 1992)

(Adapted from Sport's Physiotherapy: Applied Science and Practice, Zualaga et al (eds), 1995, pg. 19)

Scheme of Injury and Repair

- **Peritrauma period:** haemorrhage, myofibrillar retraction, cell disruption, oedema, chemotaxis
- **Intense inflammation:** inflammation fully established, mononuclear cell (macrophage) invasion
- **Phagocytosis:** intense phagocytic activity, mechanical weakening of the muscle, significant oedema

- **Early healing:** fibroblast proliferation, collagen formation, satellite cells and muscle regeneration
- **Established healing:** complete muscle fibre bridging, contractile ability returning (but still inhibited due to oedema and pain), tensile strength still low
- **Restoration of function:** collagen maturation, increased tensile strength

(Reid, 1992)

Classification of Muscle Injuries

Exercise Induced Muscle Injury (DOMS)

↑ increased activity
↑unaccustomed activity
↑excessive eccentric exercise
↑secondary muscle cell damage
↑onset some 24-48 hours post exercise

Strains

First degree (MILD)

↑Minimal structural damage
↑Minimal haemorrhage
↑early resolution
↑sudden overstretch, contraction
↑lack of flexibility, lack of warm-up

First Degree Strains

Signs & Symptoms	Treatment
× sudden onset sharp pain	× Immediate immobilisation
× pain on active movement	× Compression bandage
× tender on palpation	× Ice and elevation of limb
× little swelling	× US 48 hours post-injury
× late developing haemotoma	× Control inflammation
	× Restore motion
	× Develop muscular strength

Second Degree (MODERATE)

- ↑Partial tear, large spectrum of injury
- ↑significant early function loss
- ↑increasing severity of strain
- ↑increased fibre death
- ↑increase bleeding, haemotoma
- ↑eventually more scarring

Second Degree Strains

Signs & Symptoms	Treatment
× sudden onset sharp pain	× Immediate immobilisation
× pain on active movement	× Compression bandage
× tender on palpation	× Ice and elevation of limb
× swelling with haemotoma	× US 48 hours post-injury
× impairment of function	× (Deep) transverse frictions
	× Strapping/taping
	× Gentle stretching

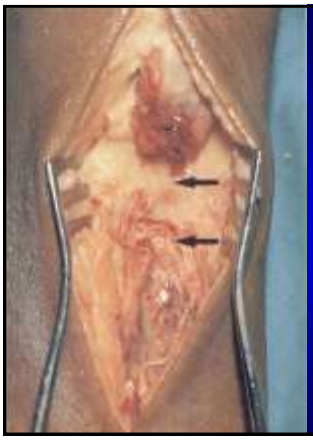
Third Degree (SEVERE)

- ↑complete tear of muscle fibres
- ↑may require aspiration and surgery
- ↑caused by steroid use or abuse
- ↑may follow previous injury
- ↑may be associated with collagen disease

Third Degree Strains

Signs & Symptoms	Treatment
× sudden onset sharp pain	× Immediate immobilisation
× no active movement	× Compression bandage
× tender on palpation	× Ice and elevation of limb
× immediate swelling	× Potential for surgery
× immediate haemotoma	× Gentle contractions
× patient's hear a 'crack'	× US 48 hours post-injury
× depression in muscle bulk	× Avoidance of scar tissue

Bilateral Rupture of the Biceps



Achilles tendon rupture

Injury due to Contusion

- Repair to the damaged muscle fibres and intramuscular connective tissue.
- Early mobilisation increases early inflammatory response.
- Subsides and leaves extensive scarring.
- Scarring leads to increases in stiffness however this is better than immobilisation of the part.

Management Principles

- There should be a mix of immobilisation and mobilising of the scar tissue.
- Oakes (1992) suggests immobilising the part for 4-5 days and then gentle mobilising.
- Best compromise is to protect the part in a stretched position by strapping/splinting.
- Semi-active management of R.I.C.E.

Injury due to Strain

- Damage is of tensile failure, often at a transition site.
- Initial immobilisation combined with gentle stretching to avoid connective tissue shortening.
- Oakes (1992) suggests 30mins per day of passive stretching.
- Isometric exercise to maintain muscle strength (1/day).
- PUS, DTF's are also effective.

Contusions

↑Mild, moderate or severe

↑Intermuscular versus intramuscular

↑results from direct blow

↑increasing muscle trauma and tearing of fibres proportionate to severity of contusion

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Ligament Injuries

Anatomy

- A ligament is a bundle of fibres connecting two bones.
- It controls relative motion of two adjacent bones.
- It inhibits separation of adjacent bones.
- It functions passively, it is not contractile.

Physiology

- Basic building block of ligaments is collagen.
- Collagen arranges itself along long protofibrils.
- These group together to form fibrils.
- Groups of fibrils form the ligament.
- Few elastic fibres in ligaments

- Ligaments are thickenings of the capsule.
- Ligaments are relatively avascular.
- Ligaments usually have an extensive nerve supply.
- Ligaments possess a ‘collagen crimp’.
- May lose proprioceptive qualities when injured.

Biomechanics of Injury

The Load-Deformation Curve

"Toe Region"

- ✦'toe' region represents normal range of strain up to 3-4% of initial length
- ✦This is due to flattening of the 'crimp'.
- ✦Repeated movement in this range does not result in damage (micro or macroscopically).

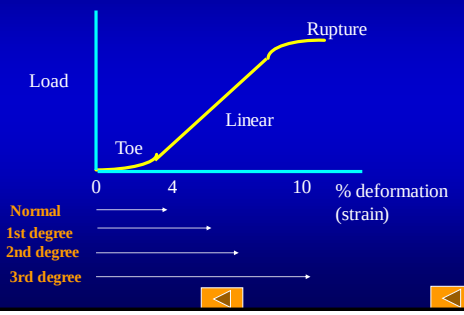
"Linear Region"

- ✦Pathological irreversible ligament elongation occurs.
- ✦Partial rupture of molecular cross-links.
- ✦Early part correlates with mild tears (0-50% fibre disruption).
- ✦Late part correlates to moderate tears (50-80% fibre disruption).

"Rupture Region"

- ★With continued loading, the linear region flattens.
- ★Failure point is reached at 10-20% strain dependent on fibre bundle macro-organisation.
- ★Complete rupture occurs at 'maximum breaking load'.

Load-Deformation Curve



Ligament Healing

Reaction Phase

- ☒ vasodilatation
- ☒ exudation of tissue fluid
- ☒ Extra-vasation of blood
- ☒ 2nd reactive oedema
- ☒ stim. of pain fibres
- ☒ chemotaxis of phagocytic cells
- ☒ gearing up of immune response
- ☒ initiation of cell division
- ☒ production of necessary elements for early healing

Regeneration Phase

- **Elimination of debris**
- ↓ granulocytes carry out phagocytosis for the initial few days and are replaced by macrophages from day 5 onwards.
- ↓ macrophages ingest macromolecules and assist in the activation of endothelial cells and fibroblasts.
- ↓ dependent on Vitamin A levels, inhibited by corticosteroids.

Regeneration Phase

- ☺ Revascularization
- ☺ new growth of damaged capillaries is necessary to maintain adequate blood supply.
- ☺ relatively avascular prior to damage so revascularization is a slow process.

Regeneration Phase

Fibroblastic proliferation

- ↓ fibroblasts begin collagen production 4 days after injury.
- ↓ this occurs after the initial intense inflammatory response to injury.

Remodelling Phase

- ◎ usually lasts up to 6 months.
- ◎ contraction of scar with subsequent maturation of collagen.
- ◎ slow increase in tensile strength as collagen fibres become orientated.
- ◎ scar may contract and shrink.
- ◎ passive stretching vital to maintain ROM.

Classification of Ligament Injuries

First Degree (mild)

Signs
- Min. loss of structural integrity.
- No abnormal motion.
- Little or no swelling.
- Localised tenderness.
- Minimal bruising.

Symptoms
- Min. function loss.
- Early return to training.
- Some protection may be necessary (10-14 days)

Second Degree (moderate)

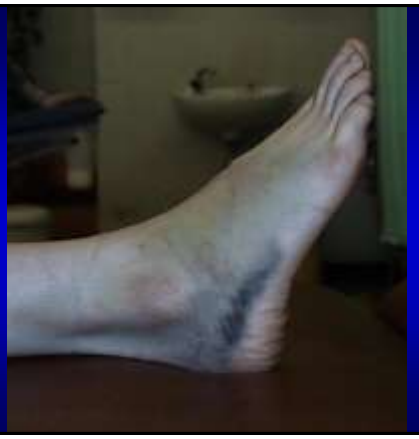
Signs
- Sig. structural weakness.
- Abnormal movement
- Solid end feel to stress
- More bruise and swelling
- Ass. haemarthrosis and effusion

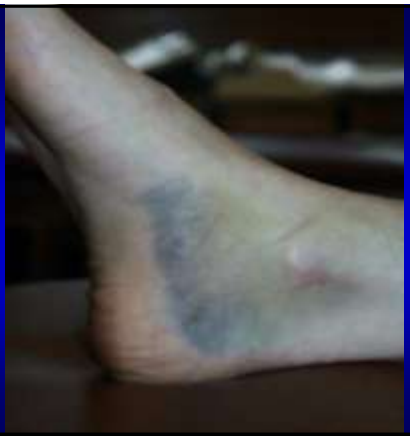
Symptoms
- Tendancy to recur
- Needs protection from risk of further injury
- Modify immobilisation
- May stretch further in time

Third Degree (severe)

Signs
- Loss of structural integrity.
- Marked abnormal motion.
- Sig. bruising.
- Haemarthrosis.

Symptoms
- Prolonged protection needed
- Surgery implicated.
- Often permanent functional instability.





Physiotherapy Management

- PRICE - initially
- Support strapping or bracing
- Surgical repair may be indicated
- NSAID's - 3 days 2400mg
- DTF's, PUS, gentle mobilising

Tendon Injuries

Anatomy

- Tendons connect muscle to bone.
- Composed of large diameter Type I collagen fibrils tightly packed together.
- Small amounts of Type III collagen dispersed in an aqueous gel containing a small amount of elastic fibres.
- Similar composition to ligaments.

Tendon-muscle Interface

- Mechanical behaviour is governed by the type of fibres present and the percentage of those fibres.
- Interface between tendon and muscle consists of interdigitations between terminal muscle fibres and collagen fibrils.
- This interface can be a site of stress due to the differences in the biological tissues involved.

Tendon-Bone Interface

Complex insertions classified as Direct or Indirect.

- Direct Insertions
  - ☑Few superficial fibres blending with the periosteum.
  - ☑Deep fibres approach bone at right angles.
  - ☑Insert into bone via 4 zones:
    - tendon proper
    - fibrocartilage
    - mineralised fibrocartilage
    - bone

- Indirect Insertions
  - ☑Primary fibres are the superficial ones which blend into the periosteum.
  - ☑Deep fibres of indirect insertions approach the bone at an angle and do not progress through the fibrocartilaginous zone.
  - ☑Tidemark zone separates nonmineralised inserting fibres and mineralised bone.

Common Terminology Used With Tendon Injuries

Everyday Names for Injuries

- Enthesopathy: tendon fibres are torn directly off their insertion.
- Tendinosis Lesion: asymptomatic tendon degeneration caused by ageing or cumulative micro-trauma without inflammation.
- Peritendinitis: inflammation of the tendon sheath, marked by pain, swelling and tenderness.
- Tendinitis: inflammation of a tendon

Other Occasionally Heard Terms

- Paratenonitis: inflammation of the paratenon.
  - Tenosynovitis
  - Tenovaginitis
  - Peritendinitis
- Paratenonitis with tendinosis
  - Tendinitis
- Tendinosis
  - Tendinitis

Pathophysiology of Tendon Repair

Acute Inflammatory Phase

- Gap in tendon is filled with erythrocytes and leukocytes.
- Within 24 hours monocytes and macrophages are the prominent cells and begin phagcytosis.
- These are replaced by fibroblasts and so begins the deposition of a Type III collagen scar.
- Glycosaminoglycan (GAG), water, fibronectin and DNA content increase.

Proliferation

- Fibroblasts predominate.
- Water content remains increased with collagen content increasing and peaking during this phase (3-6 weeks).
- Type I collagen now dominates with GAG content high.
- Increasing scar content correlates to increase tensile strength.

Remodeling and Maturation

- May take between 6 weeks - 12 months.
- Decreasing collagen, GAG and water contents.
- Scar parameters return to normal but the matrix of the scar region may remodel over a period of months.
- Occasionally calcium crystals are deposited (rotator cuff muscles).
- Large diameter fibres are slowly replaced if ever.

Achilles Tendon Injuries

Achilles Tendon Injury

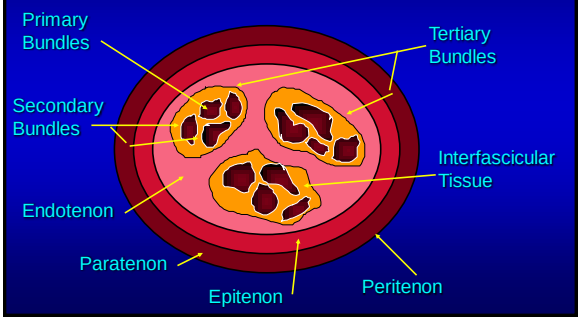
- The largest tendon in the body with a constant relative avascular portion 2-6cm above the insertion into calcaneus.
- Tendon rotates laterally as it descends beginning 12-15cm above the insertion.
- Degree of rotation depends on the amount of fusion between the gastrocnemius and soleus muscles - min. rot. is associated with greater fusion.

- Rot. produces a sawing across the fibers causing friction & damage -> degenerative changes -> rupture.
- The MT junction between the med. head of gastroc. and the tendon may also be injured.
- Three common injuries:
  - At the tendon-bone junction
  - At the musculotendinous junction
  - "True" tendinitis (2-6cm above insertion)

Classification of Injuries

- The achilles has no true synovial sheath and so tenosynovitis is a misdiagnosis.
- 'Peritendinitis' is used to describe inflammation of the peritendon.
- Inflammation, swelling and disruption of the tendon are known as 'tendinosis'.
- 'Partial rupture' should be reserved for cases of definite fiber disruption.

Cross-section of the Achilles



Achilles Tendinitis

- Caused by altered biomechanics of the forefoot and rear-foot leading to increased stress placed on the tendon.
- Calf muscle fatigue following exercise may preclude tendinitis - repetitive eccentric load-induced microtrauma.
- Poor footwear design and/or fitting - high, inflexible achilles tabs on sports shoes or flat shoes -> stretching of the tendon.

History of Complaint

- Progressively worsening symptoms usually following a traumatic event.
- Partial tears likely to have a sudden onset.
- Patients often complain of being struck in the back of the leg or feeling like they have been shot in the back of the leg.
- Past history and progression of symptoms should be noted.

Signs and Symptoms

- Pain is dominant symptom accompanied by inflammation.
- Morning stiffness is common.
- PT may feel crepitus, nodules, localized pain or thickening.
- Swelling - hard lump which moves with the tendon may mean damage to the tendon - fluctuant swelling may mean damage to the paratenon.

Diagnosis

- Should be sent for medical examination to rule out partial/complete tears.
- Ultrasound scanning may indicate whether surgery is indicated or not.
- Site of pain may indicate other structures - # calcaneus, retrocalcaneal bursitis, plantar fasciitis, subcutaneous bursitis.

Management

Conservative

- Rest from aggravating activities - crutches.
- Taping to prevent excessive movement.
- Gentle stretching and strengthening programs.
- Ice and NSAID's to control inflammation.
- Transverse frictions, PUS, TENS.
- Steroid injections may have lost favor but peritendinous injections have proven effective.

Exercise Programs

- General warm-up - no jumping or running
- Gastrocnemius and soleus stretches.
- 3x10 reps. eccentric exercise followed by stretching and ice - done 1 x daily.
- Failure in the program comes from judging the pain incorrectly and moving too quickly to the next level. (mild discomfort in final 10 reps. only)
- May be no change for 2-3 weeks but should persevere.

Progression of Exercise