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
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
- 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.

Intrinsic Injury

Michael Owen ‘pulls’ a hamstring

Aspiration of a haemotoma
**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.

**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.

**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

**Tissue Repair Processes**

The Cellular Level

**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

**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

**First Degree Strains**

**First degree (MILD)**

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

**Strains**

**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)
**Second Degree (MODERATE)**

- Partial tear, large spectrum of injury
- Significant early function loss
- Increasing severity of strain
- Increased fibre death
- Increased bleeding, haemotoma
- Eventually more scarring

**Second Degree Strains**

**Signs & Symptoms**

- Sudden onset sharp pain
- Pain on active movement
- Tender on palpation
- Swelling with haemotoma
- Impairment of function

**Treatment**

- Immediate immobilisation
- Compression bandage
- Ice and elevation of limb
- US 48 hours post-injury
- (Deep) transverse frictions
- Strapping/taping
- Gentle stretching

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**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**

- Sudden onset sharp pain
- No active movement
- Tender on palpation
- Immediate swelling
- Immediate haemotoma
- Patient’s hear a ‘crack’
- Depression in muscle bulk

**Treatment**

- Immediate immobilisation
- Compression bandage
- Ice and elevation of limb
- Potential for surgery
- Gentle contractions
- US 48 hours post-injury
- Avoidance of scar tissue

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**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

**References**

Ligament Injuries

- 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.

References

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

**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 phagecytosis.
- 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.

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**Achilles Tendon Injuries**

- 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)
- ‘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.

**Classification of Injuries**

- The achilles has no true synovial sheath and so tenosynovitis is a misdiagnosis.
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

- Slow move, no resistance
  - Painful?
    - Yes: Stay at same level
    - No: Increase speed (moderate)
- Increase speed (moderate)
  - Painful?
    - Yes: Stay at same level
    - No: Increase speed (fast)
- Increase speed (fast)
  - Painful?
    - Yes: Stay at same level
    - No: Increase resistance
- Increase resistance
  - Painful?
    - Yes: Stay at same level
    - No: Increase resistance