<table>
<thead>
<tr>
<th>Topic</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bone Basics</td>
<td>2</td>
</tr>
<tr>
<td>Bone Metabolism</td>
<td>4</td>
</tr>
<tr>
<td>Osteoporosis</td>
<td>6</td>
</tr>
<tr>
<td>Osteomalacia</td>
<td>9</td>
</tr>
<tr>
<td>Biomechanics Definitions</td>
<td>11</td>
</tr>
<tr>
<td>Trauma Response</td>
<td>13</td>
</tr>
<tr>
<td>Peripheral Nerve Injuries</td>
<td>17</td>
</tr>
<tr>
<td>Neurophysiology for the Orthopaedic Surgeon</td>
<td>22</td>
</tr>
<tr>
<td>Orthopaedic Oncology</td>
<td>26</td>
</tr>
<tr>
<td>Key Features of Selected Tumours</td>
<td>28</td>
</tr>
<tr>
<td>Meniscus</td>
<td>30</td>
</tr>
<tr>
<td>Articular Cartilage</td>
<td>31</td>
</tr>
<tr>
<td>Infection &amp; Osteomyelitis</td>
<td>32</td>
</tr>
<tr>
<td>Theatre Design</td>
<td>35</td>
</tr>
<tr>
<td>Intervertebral Disc</td>
<td>36</td>
</tr>
<tr>
<td>Imaging Techniques</td>
<td>38</td>
</tr>
<tr>
<td>Ligaments &amp; Tendons</td>
<td>42</td>
</tr>
<tr>
<td>Gait Analysis</td>
<td>44</td>
</tr>
</tbody>
</table>
Bone Basics

Functions of bone:
- Reservoir of calcium
- Haematopoietic marrow – supplies erythrocytes, leukocytes and platelets
- Mechanical role – attachment of muscles and locomotion
- Protection of viscera

Structure:
- Woven / immature bone – isotropic because of random orientation of collagen fibres. Found in metaphyseal growth, fracture callus and pathological bone (tumours and Paget’s – where there is accelerated turnover)
- Lamellar / mature bone
  - Anisotropic with collagen laid down in parallel sheets / lamella, aligned to the mechanical axis of force
  - Adjacent lamellae are arranged slightly oblique to each other in a herring-bone pattern
- Cortical / compact bone
  - Lamellae arranged in concentric rings are called osteons, around a neurovascular Haversian canal – osteons are 50 microns across
  - Canaliculi run radially in these osteons and are processes from osteocytes that allow for cell signalling by gap junctions.
  - Volkman's canals run perpendicular to the long axis, and connect the outer periosteal blood supply with the Haversian system.
- Trabecular / cancellous bone
  - Less dense, less elastic and less strong
  - No Haversian systems / osteons
  - Lattice of trabeculae bounded by lamellae again arranged along the axis of force upon the bone, and containing bone marrow elements for haematopoiesis.

Components:
- Cellular phase:
  - Osteoblasts – undifferentiated mesenchymal stem cells with high ALP activity and synthetic function. They have 3 fates: become inactive bone lining cells, osteocytes or undergo apoptosis.
  - Osteocytes – 90% of bone cell population and control Ca / PO₄ metabolism, and therefore regulate bone healing and turnover
  - Bone lining cells – inactive osteoblasts recruited for new bone formation. PTH binding results in cAMP-mediated shape change that will expose bone surface allowing osteoclasts to start resorption
  - Osteoclasts – arise from haematopoietic macrophage, and will fuse together to form multi-nucleate giant cells. Lie in Howship lacunae (pits) on the endosteal and periosteal surface of bone. Have a brush
border for surface area, and contain carbonic anhydrase system (dissolves inorganic hydroxyapatite) and proteolytic enzymes (e.g. TRAP and cathepsin to dissolve organic matrix).

- **Matrix phase:**
  - Inorganic – calcium phosphate in the form of hydroxyapatite (Ca₁₀(PO₄)₆(OH)₂) and osteocalcium phosphate (brushite). These mineralise into crystals in the hole and pore regions of collagen fibrils. Inorganic matrix is a reservoir for 99% of body calcium and 80% of body phosphorous.
  - **Organic phase** –
    - Collagen type 1 (tensile strength)
    - Bone specific proteoglycans (compressive strength)
    - Non-collagenous matrix proteins – osteocalcin released by osteoblasts to control osteoclasts; osteonecstin released by osteoblasts to regulate mineralisation; osteopontin anchor osteoclasts.
    - Growth factors – BMP (member of TGF-beta family), insulin-like growth factor (IGF I & II), IL1 & 6

- **Growth plate:**
  - resting zone – pleuripotent stem cells;
  - proliferation zone – differentiation of stem cells into chondrocytes for matrix production, with columnar/palisading alignment; achondroplasia
  - hypertropic zone
    - maturation - Morquio’s syndrome and other mucopolysaccharoidosis affect this area
    - degeneration – cells die by apoptosis
  - zone of provisional calcification – affected in rickets
  - Primary and secondary spongiosum – loops of blood vessels enter this area; osteogenesis imperfect and scurvy affects these areas
  - Allows longitudinal growth by endochondral ossification, in long bones.
  - A perichondral ring surrounds the physis to prevent slippage, but also allows circumferential growth
    - Groove on Ranvier for circumferential growth; affected in bony exostoses.
  - Regulated by local factors and systemic factors (nutrition, genetics, growth hormone
    - Volkman-Heuter law – slow groth by compressing physis, and enhance growth by distraction.

- **Growth plate injuries:**
  - 20% of fractures in children will affect growth plate
  - <5% of these will suffer a growth arrest
  - Usually physis fracture in zone of hypertrophy – where there is less matrix.
Salter Harris – types 1 to 5; Rand described type 6 which is injury to the perichondral ring.

Bone Metabolism

- Calcium – absorbed by the duodenum via active transport and controlled by calcium binding protein and 1-25-Vit D3, as well as by passive diffusion in the jejunum.
  - 99% of filtered calcium is re-absorbed, with 60% from the proximal convoluted tubule.
  - Recommended daily intake:
    - children: 600 mg/day
    - 10-25: 1400 mg/day
    - 25-65: 750 mg/day
    - lactation: 2000 mg/day
    - postmenopause: 1500 mg/day
    - fracture healing: 1500 mg/day

- Phosphate – as well as being a key compound in inorganic bone matrix, it also functions as a metabolite and as a buffer in the body enzyme systems.
  - Daily requirement = 1000 – 1500 mg/day

- Vitamin D – ingested as naturally occurring steroid in fish oils and plants, or generated in the skin when UV-rays convert 7-dehydrocholesterol into cholecalciferol.
  - Average Caucasian needs 1 hour of sunlight exposure for adequate daily generation.
  - Activated initially in the liver (25-OH-cholecalciferol), and then in the kidney (1-25(OH)2-cholecalciferol by 1α-hydroxylase.
  - Enhances GI calcium and phosphorous absorption
  - Enhances osteoclastic resorption
  - Inhibits PTH release
  - Inactivated by high Ca-levels by conversion into 24-25(OH)2-cholecalciferol

- Parathyroid hormone – 84 amino-acid peptide secreted by chief cells of parathyroid gland, secreted in response to low Ca-levels.
  - stimulates 1α-hydroxylase to activate vitamin D3
  - increases reabsorption of calcium in the kidney
  - promotes urinary excretion of phosphate
  - stimulates osteoclasts - works by activating RANKL (receptor activator of nuclear factor kappa-B ligand) receptor on osteoblasts, which then secrete RANKL and stimulate fusion of osteoclasts into multi-nucleate cells within cutting cones
  - overall will increase calcium levels, and decrease phosphate.

- Calcitonin – 32 amino acid peptide secreted by para-follicular C-cells in the thyroid gland, in response to raised calcium levels.
- Directly inhibits osteoclasts, by reducing cellular motility, retraction of cytoplasmic extensions and reduction in brush border size.

- Oestrogen – inhibits bone resorption

- Corticosteroids – reduces GI absorption and increases renal excretion of calcium, resulting in secondary hyperparathyroidism.

- Thyroid hormone – increases bone turnover, favouring resorption.

- Growth hormone – increases GI calcium absorption

- Insulin – type 1 diabetes if poorly controlled may lead to bone loss

- Growth factors:
  - IL1, IL6, TNFα - stimulate osteoclast precursors
  - IGF – activated osteoblasts and produced by osteoblasts
  - TGFβ – activates osteoblasts

- Physiological changes in bone turnover:
  - Bone mass increases up to a peak between age 16 and 25
  - After which there is normal loss of bone mass at a rate of 0.3% per year
  - Women have an increase in bone loss up to 3% per year for the first decade after the menopause, before it reverts to the same rate of loss in both men and women.
**Osteoporosis**

- Significant decrease in bone mass per unit volume (or density), that is accompanied by increased fragility. Mineralisation is intact.
  - Loss of trabecular connectivity results in structural loss of strength out of proportion to the loss in mass
- Osteoporosis is defined in terms of bone mineral density (BMD) < -2.5 standard deviations away from age- and sex-matched medians in the population
- Regional osteoporosis may be due to disuse, but generalised is more common and subdivided into primary and secondary

**Primary Osteoporosis**

**Type 1 Post-menopausal = high turnover**

- Rapid bone loss in the early post-menopausal period characterised by increased osteoclast activity
- Women lose bone at an accelerated rate (3% per year) around the menopause and for the first 10 years. 10 times higher than the rate the preceding decade (0.3% per year)

- Risk factors:
  - Caucasian or Asian
  - Family history
  - Anorrexia or amenorrhoea
  - Early menopause
  - Early hysterectomy
  - Dietary insufficiencies, malnutrition
  - Smoking
  - Alcohol abuse
  - Chronic lack of exercise

- Classical symptoms/signs
  - Back pain and thoracic kyphosis from thoracic spine wedging
  - Low-energy fractures of the distal radius or other bone

- NICE guidelines state that in women older than 75 with one fragility fracture, secondary prevention may be started immediately using calcium supplements and bisphosphonates if not contra-indicated. Below the age of 75, DEXA-scans should be performed initially and secondary causes ruled-out.

- Conservative management:
  - Calcium and vitamin D supplements (Chapuy 1994)
  - Maintain activity level
  - Avoid smoking and excessive alcohol
Medical management:
- **HRT**
  - Encouraged in women with risk factors and low BMD on DEXA-scan
  - Contra-indicated if risk factors for breast cancer
  - If no hysterectomy, the risk of uterine carcinoma is offset by use of combined oestrogen and progestogen
- **Bisphosphonates**
  - Risedronate and Alendronate preparations can be given weekly
  - Reduce rate of vertebral and hip fractures (Black 1996)
  - Can have GI side-effects
- **Strontium Ranelate or Raloxifene (SERM)** may be used if cannot comply or intolerant or contra-indicated to bisphosphonates, and have an indicative T-score.
- **Tetrapareptide** may be used if SERMs and bisphosphonates not tolerated or contra-indicated, or if further fragility fractures occur despite treatment, and patient has an indicative T-score for his age.
- **Calcitonin** – available in spray form
- **Slow-release fluoride** may be combined with calcium supplements

**Type 2 Involutional (Senile) = low turnover**
- Ill-defined syndrome which emerges in very elderly people and is due to a gradual slow-down in osteoblast activity, along with dietary insufficiencies and chronic ill-health
- 15 years after menopause (or men age 70+) there is a steady loss in bone mass of 0.5% per year
  - One in three Caucasian women (33%) by age 70, will have one vertebral insufficiency fracture
- Serum and urinary biochemistry is usually normal unless there is co-existing osteomalacia

**Treatment:**
- Manage any presenting fractures
- Address risk factors:
  - Dietary deficiencies
  - Exposure to sunlight
  - Bisphosphonates may slow further bone loss, but will not restore bone density

**Secondary Osteoporosis**
- **Hypercortisonism:**
  - Suppression of osteoblasts
  - Reduced calcium absorption
  - PTH stimulation
- **Gonadal insufficiency**
  - Present in young girls with ovarian agenesis and primary amenorrhoea (Turner’s syndrome)
  - Can be iatrogenic in female athletes and women with anorexia nervosa
  - Can occur in men with overt hypogonadism, and require testosterone treatment
Hyperthyroidism will increase overall bone turnover with resorption exceeding formation – fractures usually occur after the menopause due to the cumulative climactic changes and metabolic insult
  ➢ Often a rise in serum ALP, calcium + hypercalciuria

In myeloma there is bone loss due to overproduction of local osteoclast-activating factors

Alcohol abuse
  ➢ decrease calcium absorption and increase urinary excretion (diuresis)
  ➢ often accompanied by malnutrition
  ➢ liver failure and toxic effect on osteoblast
  ➢ mild glucocorticoid effect
  ➢ propensity to falls and trauma
Osteomalacia

- inadequate mineralisation of bones leads to softening (osteomalacia) and deformity (rickets) due to effects of physeal growth and ossification

- Causes:
  - **Congenital:**
    - Familial X-linked hypophosphataemia (dominant)
    - Mutated hormone blocks renal phosphate tubular reabsorption
    - Requires combined vitamin D and phosphate rx.
    - Fanconi’s syndrome
    - Rare hereditary hypophosphotasia
    - Vitamin D-dependent Rickets Type 2
      - peripheral resistance to vitamin D from a receptor mutation
  - **Traumatic** – nil
  - **Infective** – hepatitis
  - **Neoplastic** – paraneoplastic effect of giant cell tumours or PVNS
  - **Circulatory** – nil
  - **Autoimmune** – primary biliary cirrhosis
  - **Pulmonary** – nil
  - **Metabolic:**
    - Gall bladder & liver disease (abnormal absorption of fat soluble Vit D)
    - Crohn’s disease
    - Renal osteodystrophy (lack of 1α hydroxylase)
  - **Endocrine** - Pancreatic insufficiency, hypoparathyroidism
  - **Degenerative** – elderly with reduced exposure to sunlight
  - **Drugs:**
    - Sodium fluoride
    - Bisphosphonate excess
    - Tetracycline excess
    - Phenytoin or rifampicin block metabolism of Vitamin D
  - **Iatrogenic**
    - Dietary lack of vitamin D (vegetarians, < 100 iu/day)
    - Complications of thyroid surgery with hypoparathyroidism
    - Bowel resection
  - **Psychiatric** – sequelae of paracetamol overdose

- Symptoms in children are those of rickets:
  - Failure to thrive
  - Infants may present with tetani or convulsions
  - Deformity of skull (craniotabes)
  - Thickening of knee, ankles and wrists from physeal overgrowth
  - Enlargement of costochondral junctions – rickety rosary
  - Lateral indentation of chest wall – Harrison’s sulcus
  - Bowing of tibia from sitting cross-legged
  - Bow legs, knock knees or disturbed gait

- Symptoms in adults are those of osteomalacia
  - Bone pain
  - Back ache and proximal muscle weakness before diagnosis is made
  - Mild kyphosis from vertebral collapse
  - Unexplained pain in hip or long bone, may herald a stress fracture
X-ray signs
- Rickets: thick widened growth plate, with bowing of diaphysis
- Looser zone = thin transverse band of rarefaction in otherwise normal-looking bone
  - Represent stress fractures, caused by pulsating arteries, and which heal with callus lacking calcium
- Codfish vertebra – biconcave
- Spontaneous fractures in pubic rami, femoral neck and below knee
- Trefoil pelvis (champagne glass pelvis) caused by indentation of acetabulum
- Peri-osteal erosions from secondary hyperparathyroidism (medial proximal humerus, femoral neck, lateral distal forearm bones)
- Brown tumours (osteitis fibrosa)
  - Osteoclast resorption with holes filling with fibrous tissue
  - Become confluent

Biochemistry:
- Low calcium and phosphate from vitamin D abnormalities
- High PTH (secondary hyperparathyroidism)
- High ALP (from osteoclast activity)
- Low urinary excretion of calcium
- \([\text{Ca}^{2+}] \times [\text{PO}_4^{3-}] < 2.4\)

Bone biopsy will give definitive diagnosis with tetracycline staining showing defective mineralisation.

Management:
- Conservative – dietary advice and modification, sun exposure
- Medical – Adcal D3; may need 400 – 1000 IU per day of Vitamin D
- Surgical – fracture management, osteotomies to treat bowing
**Biomechanics Definitions**

- **Viscous Material** – resist strain linearly with time
- **Elastic material** deforms immediately and returns to original shape once stress removed.
- **Viscoelastic Material** shows time dependent strain, where the strain rate can be linear (Newtonian) or non-linear (non-Neutonian), and exhibit specific properties:
  - Hysteresis – energy released from material (as heat) during cyclic loading and unloading.
  - Stress relaxation – where the stress within the material declines with time under a constant strain
  - Creep – strain increases over time as stress maintained constant
  - Rate-dependency – the rate at which a stress is applied will alter the rate of strain. As the rate of stress increases the gradient of the stress-strain curve will also increase, and less strain possible before failure. Thus increasing strain rate will increase the material stiffness but also make it more brittle.
- **Elastic Limit vs Yield point (strength)**
  - On a stress-strain graph an object deforms elastically and proportionally with stress.
  - The gradient depicts the material's Young's Modulus
  - The elastic limit is the limit of this proportionality
  - The yield point is the limit of elastic deformation, beyond which the material deforms plastically with further stress
  - Ultimate tensile stress – maximum stress before rupture
- **Ultimate tensile stress vs breaking stress**
  - As stress is applied beyond the UTS, the material will rupture.
  - Energy is lost through heat and noise, which means the stress within the material after rupture is less. Therefore breaking strength less than yield strength
  - In this phase, the cross-sectional area also reduces rapidly before failure = “necking”
- **Ductility**
  - Here as more stress is applied, plastic strain occurs by reduction in the material’s cross-sectional area.
  - It is a measure of how much plastic strain can occur in the material before failure.
- **Brittle** – a brittle material undergoes little further plastic strain, and the ultimate tensile strength is close to the yield point.
- **Toughness** – the amount of energy a material can absorb before fracture/rupture. It is equivalent to the area under a stress-strain curve up to the breaking point.
- **Fatigue strength** – the stress a material can absorb despite a large number of cycles of load.
  - The ultimate tensile strength reduces with the number of cycles of loading, tending towards an asymptote
  - The asymptote represents the Fatigue strength, and can be represented either as an absolute value or as a ratio of the ultimate tensile strength.
As long as the stress applied to a material is less than the fatigue strength, the material can undergo an infinite number of cycles.

However, if a higher stress is applied, the number of cycles needs to be estimated to ensure failure does not occur before projected life expectancy.

Usually 2.5 to 5 million cycles equates to 1-2 years of continuous physiological loading and unloading, and in a trauma circumstance should have allowed bony union to take place and convert the implant for a load bearing device to a load sharing device.

Stiffness is the limit of an elastic body (not material) to deformity, and is defined by the force applied to the body divided by the deformity it undergoes.

Rigidity is a property of a construct or prosthesis, as opposed to a material. It incorporates properties of stiffness from the material as well as properties associated with geometry (shape).

2nd Moment of an object relates to an object’s resistance to bending based on the distribution of matter around the axis of bending from a force. It relates to the geometry of an object, where more material away from the axis means more resistance.

For example a ruler will bend easily when flat as the thickness is small. However it will not bend on its side as the thickness is much greater.

This is why H-bars are used in construction, as they maximise resistance to deformity in one direction, while allowing for a lighter construct.

Amount of curvature K is inversely proportional to the radius of curvature of a beam in response to a deforming force. \( K = \frac{1}{R} = \frac{M}{E \cdot I} \)

Where M is the bending moment applied by the force, E is the Young’s Modulus, and I is the 2nd moment of area.

The maximum stress on the bending beam is located furthest from the neutral axis (i.e. on the tension or compressed surface), and is calculated as \( S = \frac{M \cdot y}{I} \) (where y is the perpendicular distance from the neutral axis).

Poisson’s ratio is the ratio of transverse strain to axial strain of an object undergoing an axial load.

For example when a rubber band is stretched it lengths and narrows.

- Rubber 0.5
- Steel 0.3
- Biological tissue 0.5
Trauma Response

SIRS

<table>
<thead>
<tr>
<th>Temperature</th>
<th>&lt;36 or &gt;38</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tachycardia</td>
<td>&gt;90</td>
</tr>
<tr>
<td>Tachypnoea</td>
<td>&gt;20 /min or PaCO2&lt;4.3 kPa</td>
</tr>
<tr>
<td>WCC</td>
<td>&lt;4 or &gt; 12 (×10⁹/l)</td>
</tr>
</tbody>
</table>

Sepsis Syndrome
Features of SIRS + evident focus of infection or positive blood cultures

Septic Shock
Features of Sepsis Syndrome in the presence of either:
- hypovolaemia (systolic < 90 mmHg)
- oliguria (<30 ml/hour)
- inadequate tissue perfusion (acidosis or lactate > 2.5)

2 Hit Hypothesis
- The first hit is initiated by tissue injury, hypoxia or acidosis.
- Rise in levels of IL-6, IL-8, PAF → priming of Polymorphonuclear neutrophils (PMN)
- PMNs lie marginally in blood vessel circulation during resting phase, once activated have a greater tendency to roll on endothelial cells and then firmly adhere.
- During days 2-5, a second “hit” can lead to excessive activation and transmembrane migration. Consequent superoxide generation and proteolytic enzyme release by secretory granule degradation result in local tissue injury.
- This occurs most frequently in the lungs, where the intrinsic architecture of the lungs allows for narrow tortuous capillaries and increased PMN contact with endothelial cells.
Following the initial inflammatory response and once the markers of SIRS have peaked, there is monocyte infiltration and anti-inflammatory cytokine release (Counter-regulatory Anti-inflammatory response syndrome = CARS).

However injury can result in reduced HLA-DR expression which results in reduced monocyte and PMN responsiveness → this can result in late sepsis / ARDS / MOF.

**Activated Protein C (APC)**

- Tissue hypoxia can drive APC production.
- APC blocks Factor-Va and VIIIa in the clotting cascade, resulting in a “coagulopathy of trauma”.
- Coagulopathy in trauma can worsen hypovolaemia through internal and external haemorrhage, increasing mortality and multi-organ failure (MOF)

**Damage Control Orthopaedics**

_DUNCAN N & MORAN S, INITIAL RESUSCITATION OF THE TRAUMA VICTIM, ORTHOPAEDICS & TRAUMA 2009, 24 (1); 1-8_

- Initial haemorrhage control and life-saving surgery, with a view to temporarily stabilise fractures (externally or percutaneously), in patients with ISS>15 or in the presence of severe abdominal trauma.
Tourniquets used safely (average time 70 minutes) without distal neurological complications.

Permissive hypotension now shown more effective at stabilising clots and avoiding secondary haemorrhage – achieved with 250ml bolus fluid challenges to make radial pulse palpable (approx. 70mmHg systolic); **only if no head injuries suspected.**

Avoid “springing” the pelvis as this can disrupt evolving clot leading to more bleeding. Apply a binder on all trauma victims with major blunt trauma with a systolic BP <110 mmHg – release within 24-48 hours to avoid pressure sores.

Followed by a period of resuscitation to restore physiological parameters such as ventilation, perfusion, coagulation and core temperature.

Definitive surgery only after period of SIRS has elapsed to avoid the “2nd hit”, which can otherwise augment the SIRS and overcome CARS, resulting in MOF and ARDS

The unstable patient:
- Polytrauma + ISS >20 + thoracic trauma (AIS>2)
- Polytrauma + abdominal/pelvic trauma + haemodynamic shock (systolic BP < 90mmHg)
- ISS >40
- Bilateral lung contusions
- Occult hypoperfusion = serum lactate >2.5 mmol/l despite haemodynamic stability

**Lethal Triad** in trauma = hypothermia, acute coagulopathy and acidosis – but actually inter-linked.

**Acute coagulopathy in trauma:**
- **Tissue trauma** – leads to endothelial injury which initiates both clotting cascade, and generalised tissue plasminogen activator (tPA) release.
- **Shock** – tissue hypo-perfusion results in acidosis, and ischaemia results in tPA release
- **Haemodilution** – from aggressive fluid resuscitation leads to dilution of clotting factors
- **Hypothermia** – exposure, cold fluids and reduced endogenous heat production (via anaerobic metabolism) \(\rightarrow\) platelet function reduced at <34°C, and zero function <30°C.
- **Acidosis** – clotting derangement is not reversed by using buffer; mechanism not known
- **SIRS** – crossover in cytokines involved in clotting cascade initiation, and inflammation.
Adequacy of resuscitation gauged by presence of *occult hypo-perfusion* (serum lactate > 2.5 mmol/l).

- Studies show that fixation of femoral fractures in trauma before reversal of occult hypoperfusion, results in twice the risk of complications (ARDS, infection, DVT, MOF)
- Correction of occult hypoperfusion within 24 hours associated 0% mortality, compared with 43% mortality if continues >24 hours.

Massive transfusion protocols reserved for patients predicted to require >10 units blood, and relies on the principal of replacing lost whole blood with whole blood in the form of a packed red cells, platelets and FFP (1:1:1 ratio).

- Attempts to reduce risk of acute coagulopathy and haemodilution
- All blood products warmed to 37°C before administration to avoid hypothermia
- Risks include: TRALI, anaphylaxis, infection (bacterial and CMV, as platelets stored at 22°C).

Areas of further research:

- Transfusion of young red cells (14 days storage), as beyond this time there is an *increase* in glycolytic metabolism by-products and *decrease* in 2,3 diphosphoglycerate (DPG). This leads to reduced pliability and structural change in the red cell with ensuing reduced end-organ capillary perfusion. These levels of 2,3-DPG can take up to 3 days to recover.
- Maintaining an ionized calcium concentration >0.9 mmol/l in an attempt to reduce the risk of exacerbation of coagulopathy. Remember blood products contain citrate which chelates calcium.
- Recombinant Factor VIIa to reduce bleeding, but also associated (but not significant) with increased thromboembolic disease. Not currently recommended pending further data.
Peripheral Nerve Injuries

- Endoneurium is connective tissue covering axons and their wrapped Schwann cells (myelin sheath)
- Perineurium separates axons into individual bundles or fasicles
- Epineurium is the outer coat of the nerve trunk and its constituent fasicles
- Ischaemia from acute nerve compression results in reversible endoneurial anoxia and can cause numbness in 15 minutes
  - Loss of pain after 30 minutes
  - Weakness after 45 minutes
  - On release of pressure, sensation is restored in 30 seconds with accompanying paraesthesias which can last 15 minutes. Motor power returns within 10 minutes.

- Axonotmesis = more severe loss of conduction with preserved neuronal continuity, usually occurring after closed injuries.
  - Accompanied by Wallerian degeneration of axons distally, with Schwann cell proliferation.
  - Regeneration commences proximally at a speed of 1 to 3 mm/day.
  - If end organs fail to be reinervated by two years, the condition is irreversible.

- Neurontmesis = division of nerve trunk, or found in crush/traction injuries where Wallerian degeneration is accompanied by destruction of the endoneurium tubules.
  - Regeneration may occur but with fibres mingling with each other and proliferating Schwann cells, without finding the correct intact distal segment, despite surgical apposition.

- Clinical features:
  - Muscle atrophy
  - Cool, shiny skin
  - Trophic ulcers
  - Nail changes
  - Palpable neuroma

- Operative tips:
  - 10/0 suture without tension on suture line
  - If tourniquet used, release and control bleeding before closure
  - Splint limb in position with least tension on nerve, for 3 to 6 weeks
  - Conservative paring of edges needed if transaction is ragged.

- Factors affecting prognosis on nerve regeneration/repair:
  - Higher level of lesion
  - Pure motor/sensory nerves vs. mixed
  - Larger gap
  - Delay in suture
  - Extent of other soft tissue injuries

Brachial Plexus Birth Injuries

- Risks:
  - Difficult cephalic delivery of larger babies or use of forceps
  - Breech delivery during extraction of head
\begin{itemize}
  \item Tend to be more common on right side
  \item Erb’s Palsy
    \begin{itemize}
      \item Damage to C5 and C6
      \item Paralysis of abductors and external rotators of shoulder, as well supinators of arm
      \item Waiter’s tip position
      \item Sensation intact
    \end{itemize}
  \item Klumpke’s Palsy
    \begin{itemize}
      \item C8 and T1 lesion
      \item Sensory loss with paralysis of intrinsic hand muscles
      \item Unilateral Horner’s syndrome
    \end{itemize}
  \item Natural history:
    \begin{itemize}
      \item Complete recovery spontaneously
      \item Partial spontaneous recovery
      \item Permanent especially if a unilateral Horner’s syndrome is present
    \end{itemize}
  \item Conservative rx
    \begin{itemize}
      \item Passive physiotherapy while waiting for limbs to recover
      \item Operative rx – if no recovery by 3 months
        \begin{itemize}
          \item Bridge any gaps with free sural nerve transplants
          \item Neurotization by transferring spare nerves (accessory, long thoracic and intercostal) to distal segments of avulsed roots.
        \end{itemize}
    \end{itemize}
    \begin{itemize}
      \item Osteotomies to correct any fixed deformities (e.g. derotation osteotomy of humeral head)
    \end{itemize}
\end{itemize}

**Brachial Plexus Birth Late**

\begin{itemize}
  \item Upper plexus injuries
  \item Lower plexis injuries
  \item Features suggesting root avulsion:
    \begin{itemize}
      \item Severe pain
      \item Paralysis of scapular muscles
      \item Horner’s syndrome
      \item Severe vascular injury
      \item C-spine fracture
    \end{itemize}
  \item Treatment:
    \begin{itemize}
      \item Initial in stab injuries or open fractures, if clean cut.
      \item Delayed tratment if too ill, or other more pressing injuries need stabilisation acutely.
      \item Not attempted if clear signs of root avulsion
    \end{itemize}
\end{itemize}

**Long Thoracic Nerve**

\begin{itemize}
  \item Arises from C5, C6 and C7, to pass through apex of axilla behind other structures of brachial plexus, and supply serratus anterior
  \item Injured during shoulder and neck injuries
    \begin{itemize}
      \item As well as 1\textsuperscript{st} rib resection, radical mastectomy, or carrying heavy items on shoulder
    \end{itemize}
  \item Features = winging of scapula or aching in arm
  \item Usually spontaneous recovery within 12 months
\end{itemize}
Operative stabilisation by transferring pectoralis minor tendon onto lower part of scapula.

**Spinal Accessory Nerve**

- Arises from C3 and C4, to supply sternocleidomastoid muscle, before traversing superficially across posterior triangle of the neck to supply the upper part of trapezius.
- Can be insured during lymph node biopsies or accompany brachial plexus traction injuries.
- Features: shoulder pain and weakness in abduction.
  - Mild winging of scapula or resisted abduction, that disappears with forceful forward thrusting.
- Explore all stab injuries, but otherwise wait 4-6 weeks if uncertain about nerve integrity (holding arm in sling to prevent further drag/traction)

**Suprascapular Nerve**

- Arises from C3 and C4, from the superior trunk of the brachial plexus, passing laterally across the posterior triangle of the neck and through the scapular notch. It supplies supraspinatus and infraspinatus.
- Damage by scapular fractures, carrying heavy loads on the shoulder or direct traction.
- Clinically – look for weakness initiating abduction, along with external rotation.
- Usually an axonotmesis with spontaneous recovery in 2-3 months.
  - In the absence of trauma may be caused by nerve entrapment, relieved by releasing the suprascapular ligament.

**Axillary Nerve**

- Arises from C5 and C6, and is a terminal branch of the posterior cord of the brachial plexus.
  - It traverses the quadrilateral space to enter the back of the arm, and supplies teres minor, before its medial branch supplies the posterior deltoid and the regimental patch area (upper lateral cutaneous brachial nerve).
  - The anterior branch then winds around the surgical neck of humerus to supply the anterior deltoid.
  - Skin landmark for this is 5 cm below acromion tip.
- Nerve bruising (axonotmesis) occurs during shoulder dislocations, fractures of surgical neck of humerus.
- Spontaneous recovery usually occurs by 3 months, beyond which the area should be explored.
  - If no recovery, limited abduction can be achieved with glenohumeral arthrodesis and residual scapular-thoracic movements.
**Radial Nerve**

- Arises from C5 to T1 via the main terminal branch of the posterior cord of the brachial plexus, runs in the spiral groove on the posterior aspect of the humerus.
  - Supplies elbow and forearm extensors, running anteriorly over lateral epicondyle of humerus
  - At the elbow, it divides into a superficial branch, and a deep branch (muscular only).
  - Deep branch winds around the lateral edge of the radius into the posterior fascial compartment of forearm, to give many muscular branches including the posterior interosseous nerve.
  - Variable skin distribution along extensor aspects of arm, but always supplies the 1st dorsal web space in the hand.

- Low lesions:
  - Found in fractures or dislocations of the elbow, or during surgical approaches to the proximal radius
  - Weak extension of MCP-joints

- High lesions:
  - Fractures of humerus, or after prolonged tourniquet use.
  - Wrist drop and sensory loss around anatomical snuff box.

- Very high lesions:
  - Shoulder trauma, or Saturday night palsy or crutch palsy.
  - Wrist drop along with triceps weakness.

- Most injuries are a result of axonotmesis and will spontaneously improve by 3 months. The hand wrist should be splinted with the MCP-joints and wrist straight, and the thumb straight and abducted, but still allowing active use of hand.
  - Exploration or salage using tendon transfers if this fails.

**Ulnar Nerve**

- Arises from C8 and T1, as a terminal branch of the medial cord of the brachial plexus
  - Runs down the anterior aspect of triceps medial to the brachial artery
  - Runs behind the medial epicondyle superficially, and enters the forearm between the medial epicondyle and the olecranon between the two heads of FCU.
  - Accompanies the ulnar artery beneath FDP. At the wrist it runs superficial to the carpal tunnel, between the pisiform and hook of hammate in Guyon’s canal.

- Low lesions:
  - Lacerations by shards of glass
  - Numbness to medial 1 ½ digits, and partial claw hand , with hypothenar and interosseous wasting
  - Long distance cyclists pressing their wrist on handlebars can suffer from ulnar nerve entrapment in Guyon’s canal
  - Unexplained motor lesions of Ulnar nerve can be caused by a deep carpal ganglion
High lesions:
- Fractures and dislocations of the elbow
- Ulnar paradox = no clawing from loss of medial half of FDP.
- Ulnar neuritis from cubital funnel syndrome, or pressure in bed bound patients.

Treatment:
- If conservative, he skin should be guarded against friction and thermal burns
- Surgical exploration
- Release of the aponeurosis over the 2 heads of FCU.
- Anterior transposition can close gaps up to 5 cm

Median Nerve

- Arises from C5-T1 from the medial root of the medial cord, and the lateral root of the lateral cord of the brachial plexus.
- Runs down the arm lateral to the brachial artery, and enters the elbow atop of brachialis.
- Enters the forearm between the heads of Pronator Teres, and runs in between FDS and FDP.
- In the carpal tunnel it lies between the tendons of FDS and FCR, with the superficial palmar branch given off proximal to the transverse carpal ligament, and running superficial to the carpal tunnel to supply the skin over the the thenar eminence.
- The anterior interosseous branch supplies the lateral half of FDP, FPL and pronator quadratus.

Low lesions caused by carpal dislocation or lacerations of the wrist, resulting in loss in sensation over radial 3½ digits and thenar eminence wasting

High lesions caused by forearm fractures or elbow dislocation, with loss of radial forearm fingers (pointing sign) and pronation.
- Anterior interosseous syndrome is rare entrapment beneath humeral head of pronator teres, with weak pinch grip (FPL and FDP), with no sensory involvement.
- After operative repair of transections, the arm should be splinted in flexion, and when later mobilised, wrist extension avoided.
Neurophysiology for the Orthopaedic Surgeon

Taken from Woodruff M (2000), Current Orthopaedics, 14; 347-355

Nerve conduction studies

- A measure of distal peripheral nerve conduction, assessing:
  - Distal latency – time from stimulation to onset of action potential
  - Amplitude of evoked response
  - Conduction velocity

- Measured by stimulating a motor action potential (MAP) at 2 sites, with a supra-maximal stimulus. The calculation involves the latency as the time for the impulse to travel along the nerve and cross the neuromuscular junction (slower chemical transmission), where it can be detected in the muscle
  - The distance between the 2 sites measured divided by the difference in latencies gives the nerve conduction velocity
  - \( \text{ncv} = \frac{d}{(pl-dl)} \)

- The amplitude of the MAP (millivolts) is a measure of the active motor units within that muscle

- Sensory nerve action potentials can be measured using orthodromic pure sensory nerves (SNAP), or antidromic mixed nerves (compound, CNAP)
  - CNAPs allow larger amplitudes which are more reliably recorded
  - But, produce movement artefact and can be uncomfortable.
  - Sensory NCV is simply the distance between the stimulus and detection electrodes, divided by the latency between them.
  - SNAP amplitude is measured in microvolts (much smaller than motor APs) – often require needle electrodes positioned close to the nerve instead of surface electrodes.

- Normal values

<table>
<thead>
<tr>
<th></th>
<th>Arm</th>
<th>Leg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor NCV</td>
<td>&lt;45 ms(^{-1})</td>
<td>&lt;40 ms(^{-1})</td>
</tr>
<tr>
<td>Sensory NCV</td>
<td>&lt;45 ms(^{-1})</td>
<td>&lt;35 ms(^{-1})</td>
</tr>
</tbody>
</table>

- Sensory NCV is a more sensitive measure of a peripheral nerve lesion

- A 50% reduction in amplitude when comparing sides is considered significant – usually representing axonal loss. A 75% axonal loss is needed before NCV is affected

- Temporal dispersion occurs when there is reduction in conduction velocity by myelin pathology, where there is loss in fibre synchronicity leading to reduced amplitude with increased duration (>4 ms).
The area under the curve remains the same, showing the pathology is in conduction and not axonal loss.
Conduction velocity drop <60% and terminal latency rise >150%; (vice-versa in axonal loss)

Factors affecting NCV measurement:
- Specific nerve
- Patient age
- Temperature – NCV slows by 2 ms⁻¹ for every degree of cooling

Late Response

Whereas standard NCS assess the distal third of a peripheral nerve, late responses assess the proximal portion of a peripheral nerve along with the nerve roots.

H-reflex is the motor response produced by sub-maximal stimulation of 1a-afferent fibres from stretch receptors within muscle, that are involved in a monosynaptic reflex arc in the dorsal horn of the spinal cord, where they synapse onto alpha motor neurons
- Elicited from soleus muscle on stimulation of the posterior tibial nerve in the popliteal fossa
- Or from the FCR muscle
- Delayed or absent in polyneuropathies or radiculopathy

F-wave represents the small muscle response from retrograde conduction along motor nerves from a point of supra-maximal stimulation to the anterior horn cells, with secondary recurrent transmission back to the muscle
- Normally an F-wave is 5% of the motor action potential.
- Therefore prolongation of the F-wave latency with normal peripheral NCS implies a lesion causing slowing over the proximal motor fibres at the plexus or root level

Nerve Injuries

Neuropraxia – a local myelin injury that manifests as a conduction block, with likely full recovery over a period of days to weeks

Axonotmesis – axonal disruption with an intact endoneurium
- Wallerian degeneration distal to point of injury
- Antegrade re-inervation occurs slowly (1 mm/day or 1 inch/month) along the intact endoneural pathways.

Neurotmesis – total disruption of the axons and endoneurium
- Wallerian degeneration distal to injury, with no recovery
- Requires anastomosis or autogenous nerve grafting to bypass disrupted segment

- In all three injury types there is a conduction block when measured proximal to the point of injury.
  - But distal conduction is possible in the acute stage in axonotmesis and neurotmesis, which is blocked once Wallerian degeneration has occurred
  - Distal conduction remains throughout in cases of neuropraxia
  - Axonotmesis distinguished from neurotmesis by return of partial motor or sensory function
  - (Complete absence of somatosensory evoked potentials usually indicates neurotmesis).

**Electromyography**

- Study of both voluntary and spontaneous electrical activity of muscle to identify neurogenic and myopathic conditions

- Motor unit = single motor nerve + neuromuscular end plate + muscle fibres it innervates

- Grounding is required to remove background noise and artefact. Surface electrodes used to stimulate motor nerve, and recording needles to assess muscle fibre response
  - Monopolar needles are narrower and better tolerated, but require surface reference electrodes which are prone to noise. Teflon coated apart from distal 0.5 mm of tip
  - Concentric needles are wider with insulated wires down the centre – active and reference needles can be close together allowing better noise reduction

- Motor unit potential (MUP) morphology:
  - Amplitude → muscle fibre density per axon at needle tip
  - Duration → indicatory of motor unit territory
  - Phases → normally triphasic, but 20% of normal muscle have > 5 phases
  - Interference pattern occurs as a synchronous indistinguishable increase in amplitude of MUP as more separate motor units are recruited and existing active motor units fire more frequently to generate a greater contractile force.

- Recording parameters:
  - *Insertional activity* – brief burst of electrical activity representing an injury potential
    - Normal duration of 300-500 msec
- Reduced after prolonged denervation, when muscle fibrosis occurs
- Increased activity: nerve entrapment, anterior horn cell disease, axonal neuropathy
  (+ early sign of denervation)

- Spontaneous activity:
  - *Fibrillation potentials* – short duration and low amplitude, seen early in partial/complete denervation at 21 days.
  - *Positive sharp waves* – [as above]
  - *Fasiculations* – spontaneous fibre contraction from loss of descending inhibition in LMN pathologies, anterior horn cell disease, neuropathies and MND
  - *Complex repetitive discharges* – earlier sign in denervation at 12 days (muscular dystrophy, MND, myositis, polyneuropathies)
  - *Myokymic discharges* – occur in chronic peripheral nerve disorders

- Other pathological patterns:
  - *Low amplitude, high frequency action potentials* – reflect increased stimulation of poorly responding muscle group (e.g. myopathies)
  - *Large amplitude, low frequency action potentials* – reflect increased response of voluntary muscle to a decreased nerve stimulus (e.g. nerve compression syndromes)
  - *MUP polyphasia* – results from loss of synchronicity of nerve fibre conduction and muscle fibre discharge (e.g. radiculopathy, nerve entrapment, polyneuropathy)
  - *Diminished interference* and recruitment pattern – seen in neurogenic disorders.

**Somatosensory Evoked Potentials**

- Used to test the proximal axons of bipolar sensory ganglion cells and integrity of dorsal column & medial lemniscal pathways, to identify plexopathies, radiculopathies and nerve root avulsions.

- A mixed peripheral nerve is stimulated (e.g. radial, medial, common peroneal, or posterior tibial nerves). Evoked potentials generated in the CNS are detected at specific sites:
  - **UL stimulation** → check Erb’s point in supraclavicular fossa, or over C2/C7
  - **LL stimulation** → check over L1, C2 or C7; or over vertex
Worrying symptoms include persistent pain, night pain or swelling in the absence of trauma. Although a history of trauma is often present as it draws the patient's attention to the area of concern.

- Paraesthesias or numbness may occur from neural compression or lesions within nerves.
- Pathological fractures can result in significant tumour contamination
- Prodromal symptoms prior to a pathological fracture.

Primary bone tumours are rare (400 new cases per year). Soft tissue sarcoma more common at 1500/year. Metastases innumerable.

Standard investigations include:
- FBC, inflammatory markers
- Serum electrophoresis
- U&E, LFTs
  - high ALP in Ewing’s, high LDH in osteosarcoma.
- PSA
- Plain x-rays
- MRI of area in question ± staging CT chest, abdomen & pelvis
  - “local and systemic staging”
- Bone scan for skips lesions of metastases
- Tissue biopsy – with later excision of biopsy tract
  - Surgeon with experience
  - Pathologist with experience
  - Breach as few compartments
  - Also send for culture as well

X-ray features:
- Narrow zone of transition for slow growing tumours, where bone has time to react to the tumour and form a sclerotic rim to demarcate the lesion
- Wide zone of transition or permeative appearance for aggressive lesions
- Codman’s triangle is an area of periosteal elevation in rapid growing lesions, before bone deposition can fill in.
- Sun-ray speculation – attempts of bone formation under involved periosteum.
- Endosteal scalloping from aggressive medullary lesions eroding the cortex

Enneking Staging System
- Grade
  - 1 = low grade with low risk of metastases, and well differentiated
  - 2 = high grade and poorly differentiated, with high cell:matrix ration and mitotic rate
- Site
  - T1 = intracompartmental
  - T2 = extracompartmental
- **Metastases**
  - M0
  - M1
- Stage 1 lesions are G1, Stage 2 lesions are G2, and Stage 3 involve metastases.

- **Treatment options:**
  - Intra-lesional – excision through pseudo-capsule (reactive zone). Only performed for benign lesions or palliation to overcome a mass effect, as the whole field is considered contaminated.
  - Marginal – lesion is excised in one piece, with plane of dissection within pseudo-capsule.
  - Wide (intracompartamental) – lesion removed with a cuff of normal tissue.
  - Radical (extracompartamental) – entire structure and origin of lesion removed.
  - Amputation – reserved for cases where perivascular or neurological invasion renders excision impossible without compromising distal function or viability. Or when resection of tumour and any biopsy tracts cannot be achieved without extensive muscle loss.
  - Reconstruction – endoprosthetic replacements or autograft (e.g. using fibula strut for diaphyseal reconstruction or osteo-articular reconstruction of distal radius).
  - Chemotherapy – adjuvant or neoadjuvant. Can shrink a tumour to allow for successful resection with an improved level of clearance.
  - Radiotherapy – used where only a marginal resection is achieved, as well as palliation of bone metastases or pathological fractures.
  - Surgical intervention considered for benign lesions that show 50% cortical thinning or if there is a mass effect. Treatment usually by intralesional resection or curettage, augmented by cytotoxic measures (e.g. local cryotherapy, phenol or cement in-filling).
Key Features of Selected Tumours

- **Chondroblastoma**
  - Usually arise in immature skeleton epiphyseal region
  - Aggressive but benign, with a high local recurrence rate
  - Recurrence reduced by curettage augmented with cryotherapy.

- **Giant Cell Tumour (GCT)**
  - 80% in mature skeleton - benign
  - Arise on the metaphyseal side of the physis, but cross it to appear as an eccentric lytic lesion in the epiphysis
  - Present as an expanding mass or pathological fracture
  - Aggressive with high local recurrence
  - Malignant transformation after radiotherapy for local recurrence
  - Usually treated by curettage and cement filling.

- **Simple bone cyst**
  - Arise in **immature** / growing skeleton
  - Proximal **humerus** (66%), **proximal femur** (15%)
  - Well demarcated; cold on bone-scanning
  - Sclerotic lines within the cavity represents **falling leaf sign**
  - Observe for spontaneous ossification, or treat larger lesions by aspiration and cement/bone marrow instillation
  - Pathological fractures can be treated in plaster, with invasive treatment reserved for fractures showing no signs of healing.

- **Osteochondroma**
  - Most common benign tumour
  - Usually solitary except in hereditary multiple exostoses
Pedunculated lesions in the juxta-physeal region
- Malignant change in the cartilaginous cap heralded by sudden growth or pain – 1 in 1000

- **Enchondroma**
  - Solitary
  - Multiple in Ollier’s disease (30% malignant change)
  - Lesions in hands or feet are benign, but when in the shoulders of pelvis should prompt investigation of malignancy

- **Osteoid osteoma**
  - Night pain relieved by salicylates
  - Femur, tibia and spine most common bones
  - Usually intra-cortical lytic lesion with a sclerotic rim
  - Increased uptake on bone scan
  - Treated by surgical excision or percutaneous thermal ablation

- **Osteosarcoma**
  - Bimodal distribution – childhood/adolescence and elderly
  - Knee (50%), humerus (25%)
  - Permeative destruction with intramedullary sclerosis, but can be medullary with lytic appearance!
  - Calification in tissues from extra-osseous component
  - High grade spindle-cell tumour
  - Association with retinal blastoma, Paget’s disease and Li Fraumeni
  - Neoadjuvant chemotherapy increases survival up to 70%, with long-term limb salvage up to 89%

- **Chondrosarcoma**
  - Peak in middle life; 50% around hip.
  - Patchy calcification or “popcorn” appearance
  - Medullary with endosteal scalloping and narrow zone of transition
    - Beware being mistaken this is a benign lesion,
  - Adjuvant chemotherapy or radiotherapy of little use

- **Ewing’s Sarcoma or Primitive Neuroectodermal tumour (PNET)**
  - Small round cell tumour (blue cells on microscope slide)
  - Large soft tissue component
  - Chromosome 11-22 translocation
  - Children and adolescents (80% under-20)
  - Femoral diaphysis with permeative appearance
  - Male to female ratio is 3:2
  - Periosteal reaction and new bone formation → onion peel appearance
  - Neoadjuvant chemotherapy reduces tumour mass
  - Can present with temperature and raised ESR – so include infection in the differential.

- Mirel’s score (1989) used for prophylactic stabilisation of secondary metastases:
**TABLE I. Mirel’s Scoring System for Risk of Pathological Fracture.**

<table>
<thead>
<tr>
<th>Score (points)</th>
<th>Site</th>
<th>Radiographic Appearance</th>
<th>Bone Width Involved</th>
<th>Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Upper extremity</td>
<td>Blastic</td>
<td>Less than 1/3</td>
<td>Mild</td>
</tr>
<tr>
<td>2</td>
<td>Lower extremity</td>
<td>Mixed</td>
<td>1/3 to 2/3</td>
<td>Moderate</td>
</tr>
<tr>
<td></td>
<td>(non-peritrochanteric)</td>
<td>(blastic-lytic)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Peritrochanteric</td>
<td>Lytic</td>
<td>More than 2/3</td>
<td>Aggravated</td>
</tr>
</tbody>
</table>

**TABLE II. Mirel’s Scoring-Based Treatment Recommendations.**

<table>
<thead>
<tr>
<th>Total Score</th>
<th>Risk of Fracture</th>
<th>Recommended Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>9 or greater</td>
<td>Impending</td>
<td>Prophylactic fixation</td>
</tr>
<tr>
<td>8</td>
<td>Borderline</td>
<td>Consideration of fixation</td>
</tr>
<tr>
<td>7 or less</td>
<td>Not impending</td>
<td>Nonoperative treatment</td>
</tr>
</tbody>
</table>

---

**Meniscus**

- Menisci are 2 crescentic fibro-cartilaginous structures
  - Thick peripheral border, convex and attached to joint capsule - vascular
  - Thin free edge – avascular
  - Formed in utero at day 45, and completely vascular at birth from medial and lateral genicular arteries via the peripherally placed pre-meniscal capillary plexus
  - Gradual regression, so by adulthood, only 10-30% is vascularised

- Medial meniscus:
  - Semi-circular
  - Wider posterior horn attached to posterior intercondylar fossa, just anterior to PCL
  - Anterior horn attached 7mm anterior to ACL
  - Peripherally attached to capsule and deep medial collateral ligament.
  - Less mobile and so tear more easily

- Lateral meniscus:
  - More circular – with a larger surface area
  - Anterior horn attaches to intercondylar fossa adjacent to ACL
  - Posterior horn attaches adjacent and anterior to posterior horn of MM
  - Peripheral attachment interrupted by popliteus tendon
  - Additional re-enforcement of posterior horn by anterior meniscofemoral ligament of Humhrey and posterior meniscofemoral ligament of Wrisberg
  - More mobile

- Composition:
  - Matrix – water (70%), type 1 collagen, elastin, proteoglycans, glycoproteins
    - Collagen majority lies in circumferential fibres
    - Few small radial fibres to provide compressive strength and resist splitting longitudinally
  - Cellular phase – fibrochondrocytes (anaerobic)
    - Fusiform – superficial zone
Ovoid – throughout
Both synthesise and maintain the matrix

Function:
- **Load transmission**
  - 50% of load in extension, and 85% in 90° flexion
  - Partial meniscectomy reduces contact area by 50%, and increases contact pressure by more than 350%
  - Lateral meniscus takes more load than medial
- **Articular conformity** – move in an AP direction and deform during flexion; also is a block to preventing soft tissue impingement and interposition
- **Joint lubrication** – better articular conformity promotes viscous hydrodynamic action needed for fluid-film lubrication
- **Joint stability** – in AP plane
- **Proprioception** – type 1 & 2 nerve endings in anterior and posterior horns

Tear pattern:
- **Vertical**
  - Longitudinal & bucket handle (long and displaced)
    - Younger patients with considerable force
    - Starts posterior
    - Associated with ACL injury
    - Causes pain at the apex of the tear anteriorly near the fat pad
    - Locking due to spasm from free fragment.
  - Radial – asymptomatic if small
- **Horizontal**
  - Cleavage
  - Partial cleavage / flap tear = most common
- **Complex** – associated with degenerate meniscal tissue

Grading of tear on MRI
- 1 = patchy degeneration which does not touch the surface
- 2 = degeneration touches the surface
- 3 = penetrates surface and is a true tear

Repair up to 85% successful if follow strict criteria:
- In the red zone (0-3 mm from periphery)
- Vertical longitudinal tears > 1cm long
- Radial tears extending into red zone
- Good tissue quality.

**Articular Cartilage**
- When planning treatment of a knee with an articular cartilage lesion:
  - Is it stable?
  - Is the meniscus intact
  - Is it well aligned?
Infection & Osteomyelitis

- Cardinal signs of acute inflammation are: rubor, calor, dolor, tumor and function laesa.

- Initial vasoconstriction, followed by vasodilation and increased vascular permeability – allows leukocyte margination and emigration with phagocytosis of foreign material and intra-cellular degradation, as well as extra-cellular release of lysosomal enzymes.

- Macrophages are activated by gamma-interferon (from T-cells) or exotoxins, and release:
  - Proteases and free radicals – tissue destruction
  - Growth factors – neovascularisation and fibroblast proliferation
  - IL1 and TNFα - connective tissue accumulation

- Three outcomes – resolution, healing by scarring/fibrosis or, chronic inflammation.
  - Chronic infection set up by persistent infection by microbes that resist intra-cellular killing (e.g. TB bacilli) or prolonged exposure to non-degradeable exogenous substances (e.g. silicosis, asbestos)

- Bacteria are pro-karyotic cells with aggregation of genetic material in a nucleoid instead of a nucleus, and the presence of a cell wall.
  - Gram +ve = cell wall retains crystals of indium dye after alcohol rinse, and appear blue under microscope.
  - Gram –ve = cell wall does not retains the counter-stain safranin-O, and appears pink.

<table>
<thead>
<tr>
<th>Gram +ve cocci</th>
<th>Gram -ve cocci</th>
<th>Gram +ve bacilli</th>
<th>Gram -ve bacilli</th>
</tr>
</thead>
<tbody>
<tr>
<td>Staph aureus</td>
<td>B catarrhalis</td>
<td>Clostridium sp</td>
<td>Pseudomonas</td>
</tr>
<tr>
<td>Enterococcus</td>
<td>Neisseria sp</td>
<td>Bacillus anthracis</td>
<td>Haemophilus</td>
</tr>
<tr>
<td>Streptococcus</td>
<td>Actinomyces</td>
<td>Corynebacterium</td>
<td>Salmonella typhi</td>
</tr>
<tr>
<td>(β-haemolytic)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Klebsiella</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Bacteroides</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Infants (&lt;1)</th>
<th>Children (1-16)</th>
<th>Adults (&gt;16)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group B strep</td>
<td>Staph aureus</td>
<td>Staph epidermidis</td>
</tr>
<tr>
<td>Staph aureus</td>
<td>Strept pyogenes</td>
<td>Staph aureus</td>
</tr>
<tr>
<td>E coli</td>
<td>Haemophilus influenza</td>
<td>Pseudomonas aeruginosa</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>E coli</td>
</tr>
</tbody>
</table>
Septic arthritis is infection of the synovium and joint, while osteomyelitis is an acute or chronic inflammatory process of the bone medulla and cortex.

- Tends to be either from haematogenous spread, congruent/adjacent metaphyseal spread, or primary seeding (open injury or surgery)
- Rapid destruction of articular cartilage
- Loss of proteoglycans by day 5, and loss of collagen by day 9.
- Acute onset with painful limp
- Non weight bear with resistance even to passive motion.
- Blood cultures positive in only 30-50%
- X-rays can show subluxation, dislocation or soft tissue swelling
- Joint destruction or ankylosis is a late feature
- Aspirate can reveal organisms on gram stain and be sent for culture
  - White cell count > 50000 mm$^{-3}$ or 75% polymorphs usually seen
  - Synovial protein > 40 mg/dl and less than serum protein
  - Elevated lactate, and reduced glucose.
- Treatment by debridement and lavage of joint, with targeted antibiotic treatment intravenous (2 weeks) and oral (4-6 weeks)

**Antibiotics:**
- **Cell wall inhibitors**
  - Penicillins (normal, broad-spectrum, extended spectrum)
  - Cephalosporins (1$^{\text{st}}$ to 4$^{\text{th}}$ generation)
  - Carbapenems
  - Monobactams
- **Protein synthesis inhibitors**
  - Aminoglycosides
  - Macrolides
  - Tetracyclines (bacteriostatic)
  - Oxazolidones (linezolid) – beware myelosuppression
- **Cell wall disruption**
  - Glycopeptides (vancomycin and teicoplanin)
- **DNA replication inhibitor**
  - Quinolones
  - Rifampicin

**Antibiotic resistance:**
- **Intrinsic:**
  - Enzyme production to denature antibiotic
  - Change in cell wall permeability
  - Alterations in structural target (e.g. ribosome subunits or change to penicillin-binding protein PBP2a)
  - Mutations in efflux mechanisms
  - Bypass of metabolic pathway
- **Extrinsic – acquisition of genes from other resistant cells via plasmids.**
MRSA = methicillin resistant Staphylococcus aureus
- Resistance via acquired PBP2a (encoded by mecA gene)
- Risk factors include old age, prolonged hospitalisation, open skin lesions, long-term indwelling devices, and chronic medical illness.
- Screening and isolation needed to prevent spread
- Identified carriers treated with nasal mupirocin and bathing in antiseptic detergent (4% chlorhexidine)

TB (mycoplasma tuberculosis)
- Chronic granulomatous condition
- On the rise in inner cities with MDR-strains, and amongst immunocompromised.
- Obligate, aerobic, acid-fast rods.
- Primary TB leads to a bronchopneumonia with a persistent Ghon focus
- Secondary spread to viscera, bone and joints – miliary TB
- Diagnosis:
  - 3 early morning sputum samples
  - acid-fast bacteria under Ziehl-Neelsen stain
  - 6 week extended culture on Lowenstein-Jensen medium
  - biopsy to look for granuloma with caseating central necrosis
  - PCR
  - Skin tests (delayed hypersensitivity) confirm either previous BCG-vaccination or active infection.
- Treated by combination antibiotics: 4 for 2 months and 2 for 4 months.
  - Rifampicin, isoniazid, pyrazinamide and ethambutol.
Theatre Design

- 4 conventional zones: outer zone, clean zone, aseptic zone, disposal zone.
- Hypothermia caused by large wounds, cool IV fluids and paralysis. Avoided by creating a warm micro-climate for the patient with warming blankets and airflow mattresses.
  - Ideal temperature 24 to 26°C, with humidity 40-60%
- Lights aim to provide 40,000 lux without shadows and be easily adjustable.
- Surgical contamination causes:
  - Airborne contamination
  - Instruments
  - The patient
  - Direct contact from surgical team
- Ventilation systems:
  - Measure bacterial airborne load using microbiological volumetric slit sampler.
  - Checked on 3 month basis in ultra-clean theatres
  - An empty Plenum theatre has <35 CFU/m³, but rises to <180 CFU/m³ when in use.
  - Ultra clean theatres have <20 CFU/m³ at the periphery of the enclosure and <10 CFU/m³ at the centre.
  - **Plenum** – air from ceiling directed to floor level vents, with positive pressure and 15-25 air changes per hour.
  - **Laminar flow** – combined with HEPA filter
  - **Ex-flow (Howorth)** – inverted trumpet of air flow moving air down and outwards to avoid peripheral entrainment.
- Clothing options:
  - Standard cotton – open weave with 80µm pores, allowing moist bacteria strike-through.
  - Ventile – close weave with 20 µm pores with a front pad impermeable to moist strike through.
  - Gore-Tex – woven polyester laminated to a film of PTFE, allowing air flow but with a pore size of 0.2 µm; uncomfortable
  - Disposable non-woven – bacterial get trapped in random fibre arrangement; expensive but cost-effective overall if infection reduced.
  - Body exhaust suits – maintain a negative pressure within the gown to prevent bacterial shedding. Cooling but claustrophobic.
- Lidwell MRC paper (1982)
  - Multi-centred RCT with three arms: positive pressure theatre (n=4133), ultra-clean (n=1789) theatre, and ultra-clean with body-exhaust suits (n=2133). Ultra-clean defined as laminar flow where <10 particles containing bacteria per cubic meter. Follow-up median 2.5 years. Also looked at prophylactic antibiotics (at induction or in cement) in a non-randomised manner.
Overall septic joints in ultra-clean vs. positive pressure theatres was 2.6 : 1. Subgroup analysis: with body exhaust suite this ratio rose to 2 : 1, while with conventional clothing reduced to 2 : 1. Overall use of antibiotics reduced incidence of septic joint by 5 times. Concurrent use of ultra-clean theatre, body suits and prophylactic antibiotics reduced deep infection rate by 14-fold!

<table>
<thead>
<tr>
<th>Prophylactic measure</th>
<th>Factor by which deep sepsis reduced</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antibiotic loaded cement</td>
<td>11</td>
</tr>
<tr>
<td>Systemic antibiotics</td>
<td>4.8</td>
</tr>
<tr>
<td>Ultra-clean air</td>
<td>2.6</td>
</tr>
<tr>
<td>Plastic isolators</td>
<td>2.2</td>
</tr>
<tr>
<td>Body exhaust suits</td>
<td>2.2</td>
</tr>
</tbody>
</table>

Other measures:
- Body drapes and incisional drapes (no evidence that they reduced infection rates)
- Masks – should be changed between cases
- BOA guidelines recommend masks be worn by ALL theatre personnel during orthopaedic surgery.
- Double gloving – because of surface colonisation and frequent glove perforation. Advice to change gloves between draping and skin incision due to contamination of tips.

**Intervertebral Disc**

- Embryological development begins at 4 weeks – mesodermal cells condense into sclerotomes.
  - The caudal and cranial parts of 2 adjacent sclerotomes form the pre-cartilaginous vertebral body
  - Mesenchymal cells within the sclerotome, form the intervertebral symphysis – and the notochord cells (endothelium germ layer) which regresses in the vertebral bodies, will enlarge in this region to form the nucleus pulposus.

- Vertebral discs are thicker anteriorly in the cervical and lumbar regions in line with natural lordosis, and more uniform in the thoracic spine.
  - They become larger / thicker caudally, with the largest disc at the L4/L5 level also being the most avascular.

- Annulus fibrosus
  - Attached to the anterior longitudinal ligament (ALL) and posterior longitudinal ligament (PLL)
  - Composed of type 1 collagen in densely packed lamellae – each arranged obliquely at 30° in alternate directions (herring-bone pattern) to allow ability to resist shear and distraction
Nucleus pulposus
- Composed on type 2 collagen and a proteoglycan matrix.
- Proteoglycan consists of hyaluronan filament with multiple aggrecan molecules, stabilised by a link protein
- The proteoglycan will draw and interact with water, to give strength versus compression

End plate
- The upper and lower surface divided into an outer 1/3 and inner 2/3
- The outer third anchors the disc to the adjacent vertebral bodies by Sharpey fibres – forming a ring apophysis
- The inner two-thirds curve into the adjacent bodies to form the fibro-cartilaginous end plate.
- End plate is hyaline cartilage in children, and gradually calcifies with age. It has no fibrillar connection with the collagen of the vertebral subchondral bone, and therefore is susceptible to shear.

Nerve supply:
- Present in the outer annular rings only
- Dorsally – from branches of sinuvertebral nerve (directly from spinal nerve as it emerges from intervertebral foramen)
- Ventrally – from sympathetic chain

Blood supply only reaches the annulus. Most of the disc is sustained via diffusion through the porous endplates and convection within the matrix.
- The L4/5 vertebra being the largest and most avascular is therefore most at risk of damage propagation and pathology

Age-related changes
- Gradual loss of cells and proteoglycan content, leading to dehydration within the nucleus pulposus
- Results in gradual loss in disc height / thickness
- Annulus becomes more fibrous, and the distinction between annulus and nucleus is less apparent
- Therefore between age 30-40, acute prolapses more common as a result of tears in the stiffer annulus, whereas in the 60+ population the pathology is more of stenosis as a result of loss of the nucleus, but flaval thickening and facet-OA as a result of abnormal loading mechanics.

Function
- Redistribute compression load uniformly even during flexion and extension
- Resists tensile, rotational and shear stress
- Achieved this via the biphasic theory – which relates to hoop stresses generated during compression in the outer layers of the annulus in comparison with the inner layer which allows deformation as a shock absorber.
- During prolonged compression during daytime activity, water is squeezed out of the discs resulting in loss of height and the annulus bulging.
During rest (supine sleep period), there is in-flow of water back into the disc, and this explains why most acute herniations occur in the morning when the full disc is loaded on upright posturing.

**Injury and Healing:**
- Annular tears can cause local back pain due to irritation of innervation in outer annulus.
- Herniation leads to pressure on the nerve roots or theca resulting in radicular pain, radiculopathy or myelopathy.
- 90% of herniations resolve within 3 months of rest, but the annular tear has limited healing potential and is a life-long weak point.

**Gradual degeneration**
- Result of natural age-related changes along with decline in the blood supply of the periphery, and compounded by calcification of the end plates.
- Interference with diffusion and convection of nutrients.
- Worsened by diabetes and smoking.
- Loss of disc function leads to reduced deformation and visco-elastic property. It acts more like a solid structure with altered biomechanics leading to ligament and facet hypertrophy to accommodate increased stress → stenosis and facet joint pain.

### Imaging Techniques

**Plain Radiograph (X-ray)**

- X-rays pass through the body with differential absorption, and fall on a fluorescent film-screen combination plate, resulting in formation of black silver crystals (film blackening).
- Calcium in bone absorbs more x-rays and therefore there is less blackening and a white appearance on the x-ray picture.

- In practice, x-rays are generated by heating a fine filament (tungsten) in a vacuum to about 2200 °C.
  - Results in thermionic emission of electrons.
  - These hit a focal spot on a metal plate cathode, also made of tungsten, at half the speed of light.
  - Generate heat when they interact with outer electrons of the target nucleus.
  - Knock out inner electrons from their orbit, and generate x-rays.
  - Hit the nucleus and deflect, slowing down → generates braking x-rays (80% of x-rays generated in the x-ray tube).
  - Quantity of x-rays proportional to number of electrons flowing from cathode to anode (measured in milliampere, mA).
  - Quality of x-rays (penetrance) determined by the energy of electrons striking the plate – proportional to the kilovoltage (kV).

- X-ray cassette contains:
- A carbon-fibre or aluminium front to filter out low-energy x-rays
- A lead sheet back to prevent back scatter
- Film – with silver iodobromide
- Intensifying screen on the front and back of the film that contain phosphor crystals, which absorb x-rays and convert them to visible that exposes the film.

**Ultrasound**

- Ultrasound waves produced by a transducer made from a piezoelectric crystal – expansion and contraction occurs at its surface when an AC current passed, to produce a compression wave
- Transmitted through the body and reflected back by tissue structures – upon hitting the crystal cause tiny distortions in shape that in turn generate a voltage.
  - The more waves reflected, the greater the voltage and the whiter the image generated.
  - The depth to echo-producing structures assessed by timing the period from ultrasound wave emission to detection.
- Can be combined with Doppler phenomenon to observe vascularity and flow.

**Uses:**
- Static and dynamic assessment of tendons – fibrillar, oval structure in cross-section
- Assess masses and distinguish between cystic (hypoechoic) and solid structures
- Confirm joint effusions and allow targeted aspiration
- Screening and evaluation of DDH without using ionising radiation

**Advantages** – no ionising radiation, inexpensive and portable, dynamic testing, non-invasive and fast. **Disadvantage** – very much operator dependent.

**Computed Tomography (CT)**

- Comprised of the scanning gantry (generator and curvilinear detector), couch, CPU and display system.
  - Each rotation of the scanning gantry produces one axial slice.

**Tissue assigned an attenuation coefficient (Hounsfield unit):**
- Bone 1000
- Liver 40-60
- Brain (white) 46
- Brain (grey) 43
- Blood 40
- Muscle 10-40
- Kidney 30
- CSF 15
- Water 0
- Fat -50 to -100
- Air -1000

- Pixels are actually 3D – therefore called voxels – with a selectable thickness
- Contrast is usually iodine based – IV, intrathecal, or intra-articular
- Advantages – useful for assessment of bone, but not soft tissues, and particularly cortical bone, with 3D reconstruction capabilities. Disadvantages include the larger radiation dose and inferior soft-tissue resolution.

**Magnetic Resonance Imaging**

- Protons naturally spin around an axis (nuclear spin), which is randomly aligned. A strong magnetic field will align all these spin axes along the long axis of the magnet.
- Precession is the normal “wobble” of each proton around its axis.
- First a strong magnet pulls nuclear spin into alignment, followed by an RF-pulse which causes them to re-align at an angle to the scanner, and pulls the precession into phase.
  - The protons act like rotating magnets in a dynamo
  - Induce currents in the receiver coils
  - When the pulse stops, the nuclear spin returns to be parallel with the scanner - longitudinal magnetisation vector increases to maximum and the time taken for it to recover to 63% is defined as T1.
  - Precession falls out of phase, and the time taken for the transverse magnetisation vector to fall to 37% of maximal is defined as T2.
- Advantages include: multi-planar imaging, high soft tissue contrast, and no ionizing radiation.
- Disadvantages: pacemakers/defibrillators, vascular clips (<2 weeks) and cerebral clips are contra-indications. Claustrophobia and the long scanning times are also issues.

**Radionuclide Bone Scanning**

- Radionuclides are unstable nuclei that disintegrate into other atoms, releasing alpha, beta or gamma radiation.
- 1 disintegration per second = 1 becquerel (Bq)
- Technetium-99 is a pure gamma emitter with a half-life of 6 hours, and within 24 hours, 70% is excreted via the kidneys and therefore bladder exposure needs to be minimised by good hydration and frequent micturition.
  - formed from its parent nuclide Molybdenum-99
  - couples with methylene diphosphonate for IV injection (99Tc-MDP)
  - absorbed onto hydroxy-apatite in bone by the phosphorous component
Photo-emission recorded from local sites or the whole body using a scintillation camera.

3 phases:
- flow or dynamic images taken immediately (1-2 minutes) to show arterial flow and hypo-perfusion
- blood pool or equilibrium images taken at 3-5 minutes, showing extracellular fluid and extent of bone and soft tissue hyperaemia
- static or delayed images taken after 4 hours when soft tissue activity has cleared, but skeletal activity persists – anterior and posterior scans.

Normally there is symmetric increased activity in the bladder, kidneys, ends of the long bones, SI-joints, tips of scapulae, nasal cavity and epiphyseal growth plates.

Advantages: useful general survey to highlight areas of pathology for further imaging.

Disadvantages: poor spatial resolution, non-specific for increased bone turnover, false negatives in myeloma and lytic metastases (reduced blood flow), and a high marrow radiation dose.

SPECT scans – combine single photo emission scan with CT technology. Provides 3D reconstructions and cross-sectional images to help localise hot-spots.

**Bone Densitometry**

Dual-energy x-ray absorptiometry (DEXA) – 2 different energies are used that absorbed differently by bone and soft tissue, to assess bone density.
- Useful in osteoporosis and assessing effect of treatments
- Also used to measure peri-prosthetic bone loss.

Measured at two sites: lumbar vertebra and femoral neck.

T score = (patient BMD) – (population peak BMD) / Std Dev of population BMD

Z score = (patient BMD) – (population age-related BMD) / (Std Dev of population age-related BMD)

Osteopaenia is T between -1 and -2.5
Osteoporosis is T < -2.5
A low Z-score implies aetiology other than age-related bone loss.

Lateral views of the lumbar spine preferred as degenerative arthritis can give a false increased value. Similarly falsely raised values can be seen after an old fracture with subsequent healing and calcification. Conversely, false low values can occur after surgical resection of the posterior elements.
Ligaments & Tendons

- Ligaments connect bone to bone:
  - Augment static mechanical stability
  - Prevent excessive / abnormal motion
  - Sensory source for proprioceptive reflexes mediating dynamic stability

- Tendons attach muscle to bone:
  - Transmit tensile forces
  - Optimise the moment of a force generated by a muscle belly away from a joint
  - Store energy (analogous to a spring)

- Structure:
  - Cellular component (20%; fibroblasts)
    - Extracellular matrix (80%)
      - Collagen (tendons > ligaments)
        - 90% type 1
        - molecules aggregate in a quarter-staggered array (= microfibrils)
        - microfibrils (0.2µm) aggregate into fibres (20µm)
        - fibres arranged in bundles – parallel in tendons, but wavy (crimp pattern) in ligaments
      - Elastin (ligaments > tendons – except ligamenta nuchae and flavum)
        - Hydrophobic non-glycosylated proteins
        - Form sheets and filaments that coil & stretch (up to 200%)
        - Allows recovery of tissue after loading
      - Ground substance
        - Proteoglycans – composed of sulphated GAGs bound to a core protein, which in turn is bound to a hyaluronic chain via a link protein.
        - Binds water molecules to create a gel which acts like a cement between collagen microfibrils to improve strength
    - Surrounding connective tissue
      - Paratenon – loose areolar tissue forms a sheath around tendons, allows gliding and is a source of healing as it is rich in cells and vessels
      - Epitenon – present underneath the paratenon in tendons which are subjected to high frictional forces (e.g. in wrist), and produce synovial fluid to aid gliding
      - Endotenon – binds together fascicles or groups of collagen bundles

- Direct Insertion sites onto bone divided into 4 zones:
  - 1 = parallel collagen fibres
  - 2 = collagen fibres inter-mesh with unmineralised fibrocartilage
  - 3 = gradual mineralisation of fibrocartilage
  - 4 = mineralised fibrocartilage merges with cortical bone

- Indirect insertion more a blending with Sharpey fibres, as seen in MCL.
Blood supply mainly at insertion sites from the peri-osteum. However, tendons with a paratenon heal better due to a better blood supply from adjacent tissue, compared with a sheathed avascular tendon that only has a vincula containing a single vessel to only a segment of the tendon.

Both contain afferent nerve endings which take part in myotactic reflexes and proprioception which limits excessive tension during muscle contraction.

Stress-strain or load elongation curves have distinct regions:
- Initial non-linear toe-in region – where crimped fibres begin to straighten
- Resistance to elongation increases as more fibres become taut
- Linear region up to the yield point
- Further elongation requires non-linear stress, with dips representing early sequential failure up until complete failure
- The strain to failure is only a few percent of the original length, as in vivo tendons rarely stretch significantly.
- The exception being the ligamentum flavum, which has a strain to failure of up to 70%.

Tendons and ligaments exhibit features of visco-elasticity: hysteresis, stress relaxation and creep.

Ageing effect:
- Collagen diameter and content increases up to age 20, and thereafter falls.
- Up to puberty the weakest link is the developing bone, and therefore failure occurs at the ligament-bone complex or with tendon avulsion injuries.
- Beyond puberty, failure is usually mid-substance in ligaments and at the musculo-tendinous junction in tendons.

Injury mechanisms:
- Repetitive micro-trauma – results in micro-tears followed by an inflammatory reaction ± calcification, which alters strength
- Macro-trauma – acute failure due to forces above the ultimate tensile strength.
- At a low loading rate, the bony insertion is weakest → avulsion injuries (± avulsion fractures).
- At high loading rates, the tendon or ligament is the weak point resulting in mid-substance tears.
- The ratio of muscle : tendon cross-sectional areas – large ratios result in tendon injuries, while small ratios result in muscle tears.
- At maximal muscle contraction, eccentric contraction results in greater tensile stress on the tendons than concentric contraction.

Injuries can be categorised by severity:
- 1 (mild) – microfailure of collagen with pain but no joint laxity
- 2 (moderate) – partial rupture with progressive collagen failure, resulting in pain and some joint laxity that is often compensated by muscle activity (assess on MUA)

Injury mechanisms:
- Repetitive micro-trauma – results in micro-tears followed by an inflammatory reaction ± calcification, which alters strength
- Macro-trauma – acute failure due to forces above the ultimate tensile strength.
- At a low loading rate, the bony insertion is weakest → avulsion injuries (± avulsion fractures).
- At high loading rates, the tendon or ligament is the weak point resulting in mid-substance tears.
- The ratio of muscle : tendon cross-sectional areas – large ratios result in tendon injuries, while small ratios result in muscle tears.
- At maximal muscle contraction, eccentric contraction results in greater tensile stress on the tendons than concentric contraction.

Injuries can be categorised by severity:
- 1 (mild) – microfailure of collagen with pain but no joint laxity
- 2 (moderate) – partial rupture with progressive collagen failure, resulting in pain and some joint laxity that is often compensated by muscle activity (assess on MUA)
- 3 (severe) – most collagen fibres ruptured. Though there may be some continuity in the tissue, there is gross instability. Pain is severe at injury but declines afterwards.

- Tendons and ligaments are less well vascularised and innervated, and therefore the healing response is less.

- Haemorrhagic / inflammatory phase:
  - Haematoma and rapid inflammatory response
  - Monocytes remove debris
  - Fibroblasts invade
  - Hours – days
  - Intra-articular injury (e.g. ACL) results in dilution of fibrin clot by synovial fluid, resulting in failure of healing.

- Proliferative phase:
  - New blood vessels
  - Fibroblasts produce new matrix material
  - Type III collagen predominates
  - Weeks

- Remodelling phase:
  - Progressive maturation and conversion of collagen fibres to type I
  - Alignment of fibres along axis of stress
  - Surgical tendon repairs are weakest in the first 2 weeks, regaining most of their original strength by 3-4 weeks
  - Maximum strength by 6 months.

- Rehabilitation:
  - Controlled mobilisation improves healing, by promoting remodelling
  - Immobilisation results in stiffness, decreased strength and increased cross-linking making more stiff.
  - Steroids and hyaluronate reduce adhesions, but slow healing and increase infection rates
  - Instability should be addressed as it reduces healing.
  - Mechanica property of ligament takes only 9 weeks to recover, but bone insertion takes up to 1 year to recover.

**Gait Analysis**

- Normal gait has 5 re-quisites (Gage 1991)
  - Stance phase stability
  - Adequate step length
  - Sufficient foot clearance during swing
  - Swing phase pre-positioning of foot
  - Energy conservation

- Gait cycle is defined by the events and the time interval between heel strikes of the *same* foot.
- Stance (60%)
  - Initial contact
  - Loading response
  - mid-stance
  - terminal stance
  - pre-swing
- Swing phase (40%)
  - Initial swing
  - mid swing
  - terminal swing

- Common terms and definitions:
  - Step length (metres) – horizontal distance along plane of progression from one heel strike the contralateral heel strike.
  - Stride length (metres) – horizontal distance from one heel strike to the next ipsilateral heel strike. Adults 1.5 metres, and in children $0.9 \times$ height.
  - Cadence – steps per minute
  - Double support phase – both feet in contact with ground
  - Float phase – neither foot in contact with ground
  - Velocity – stride length / cycle time
  - Walking base (mm) – side-to-side distance between feet from points of heel contact
  - Foot progression angle (deg) – angle between direction of progression and midline of foot. External is -ve; internal is +ve

- 97% of children walk independently by 18 months with a wide-based, stiff-knee, non-reciprocating gait. Reciprocating gait starts by 3.5 years, and an adult pattern achieved usually by age 7.

- Gait velocity remains constant until age 70; thereafter declines at 15% per decade. Step length is sacrificed with increased double support phase to reduce velocity but gain stability.

- Visual gait assessment:
  - Start with body habitus, symmetry, limb position, scars, wasting, and walking aids / prosthetics
  - Observe each leg in coronal plane and sagittal plane.

  - Pelvis – should rotate internally at heel strike, and externally at toe off. Reduces transverse plane movements in centre of gravity during gait, and makes gait more efficient.

  - Hip – is flexed by 35° at initial contact, and extends until midway through stance. Flexors start contracting at terminal stance, with maximal hip flexion at terminal swing.

  - Knee – flexors contract eccentrically prior to heel strike to prepare for energy absorption, and with subsequent knee flexion as part of loading response. This knee flexion during mid-stance also reduces change in
vertical centre of gravity, and improves energy conservation. Knee extension during stance phase, but flexion again at heel rise, and continues up to mid-swing to allow adequate foot clearance.

- Ankle – decelerating plantar flexion with eccentric contraction of dorsiflexors to prevent foot slapping the floor (1<sup>st</sup> rocker). Eccentric contraction of plantar-flexors to allow smooth ankle dorsiflexion in mid-stance phase (2<sup>nd</sup> rocker) which also stops the joint reaction force from acting as a flexion moment at the knee (that would increase work of ambulation by requiring quadriceps contraction to then maintain knee in extension. Contraction of gastroc-soleal complex causing heel rise and power for toe-off (3<sup>rd</sup> rocker)

- Trendelenberg gait (ipselateral lean)
  - Loss of motor power – hip abductor weakness from pain inhibition, postsurgical or polio.
  - Reduced lever arm – short femoral neck (e.g. coxa vara)
  - Change in fulcrum – hinge ABduction in DDH

- Anterior trunk bending
  - Forward flexion early in stance phase
  - Usually compensating for weak quadriceps, by shifting centre of gravity over the knee to create the required extension momentum (to straighten the knee after the loading response).

- Posterior trunk bending
  - Can occur early in stance phase to compensate for weak hip extensors (altering the centre of gravity moment arm behind the hip)
  - Can also occur in mid-swing phase to propel the leg forwards in the presence of weak flexors or spastic hip extensors.
  - Also evident in patients with ankylosis or arthrodesis of the hip.

- Increased lumbar lordosis
  - Similar to posterior trunk bending in terms of mechanical advantage gained, except upper trunk remains balanced over the pelvis.
  - Usually seen in patients with fixed flexion hip deformities.

- Functional limb length discrepancy (true or apparent) compensated for by:
  - Circumduction of hip
  - Hip hitching – using abdominal and paraspinal muscles
  - Steppage – excess hip and knee flexion to allow leg to swing through
  - Vaulting - shorter leg is plantar-flexed onto tip-toes during stance phase.

- Weakness of ankle dorsiflexion
  - Slapping of foot during 1<sup>st</sup> rocker
  - Also results in toe drag during swing phase, but usually compensated for by a high stepping gait.