Anatomy
Soft tissues, nerves, blood vessels

- Soft tissues: A group of cells of common origin with some specialised structure and common function. Tendons, organs, muscle, connective tissue
- Connective tissue: Fibroblasts that secrete extracellular matrix; adipocytes store fat; defensive cells inc. Mast cells (release histamine when damaged) and macrophages; chondrocytes and osteocytes (prproduce cartilage and bone respectively)

Extracellular matrix:
- Collagen fibres (collagen): limited elasticity, strong supportive. Closely packed in tendons
- Reticular fibres (reticulin): delicate branched, supports tissues containing many cells in organs/lymph nodes and endocrine glands
- Elastic fibres (elastin): found in fibres or discontinuous sheets, essential in arteries and lungs.
- CNS (Brain Spinal cord) and PNS. Peripheral nerves in bunches called fascicles, surrounded by fibrous perineurium, all surrounded by protective Epineurium (there is also blood supply within).
- Structure neurone: large cell body with nucleus; Dendrons (further divided to dendrites) transmit signal to cell body; Axon single long extension transmits away from cell body ending in somatic knobs that stimulate effector. Most insulated with fatty myelin sheath (enables rapid conduction) made by Schwann cells separated by the nodes of Ranvier along length.
  - Motor neurone (impulses to muscle fibres at neuromuscular junctions)
  - Sensory Neurone (impulses to spinal cord and synapse with relay neurones)
  - Relay Neurone (synapse with motor and sensory neurone relaying impulses)

Blood vessels: veins, arteries
- Arteries (types include, elastic and muscular): structure – 3 main layers; TUNICA INTIMA single layer of flat cells, the endothelium, supported by a basement connective tissue layer containing collagen. TUNICA MEDIA smooth muscle, fibres of collagen and elastin (thick in large arteries and absent in blood capillaries). TUNICA ADVENTITIA collage fibres that are relatively tough
- Veins: usually surrounded by skeletal muscle whose contraction squeezes vein and gets blood moving. Septum valves in veins ensure blood is squeezed in right direction

<table>
<thead>
<tr>
<th>Feature</th>
<th>Artery</th>
<th>Vein</th>
<th>Capillary</th>
</tr>
</thead>
<tbody>
<tr>
<td>TUNICA ADVENTITIA</td>
<td>Present, with collagen fibres</td>
<td>Present with collagen fibres</td>
<td>Absent</td>
</tr>
<tr>
<td>TUNICA MEDIA</td>
<td>Present, with smooth muscle and elastic fibres</td>
<td>Present, with smooth muscle and a few elastic fibres</td>
<td>Absent</td>
</tr>
<tr>
<td>TUNICA INTIMA</td>
<td>Present</td>
<td>Present</td>
<td>present</td>
</tr>
</tbody>
</table>

Knee
- Modified synovial hinge joint (due to slight rotation in flexed position)
- Allows flexion and extension

Bones
- Distal end of femur, patella, proximal tibial plateau
- 2 joints:
  1. Patellofemoral joint – patella and surface of femur, anterior to femoral condyles → gliding of patella on anterior aspect of femoral epicondyles
  2. Tibiofemoral joint – weight-bearing joint between medial & lateral femoral condyles and the tibial plateau.

Cartilage
- All articulating surfaces covered with hyaline cartilage
- Menisci between tibial and femoral condyles
  - Circulate synovial fluid
  - Compensate for irregular shapes of bones
  - Shock absorbers
  - ↓ friction and ↓ wear and tear.
Ligaments
5 ligaments holding femur and tibia together; Patellar ligament, lateral collateral, medial collateral, anterior cruciate, posterior cruciate

Nerves
- Sciatic nerve
  - Behind the knee in popliteal fossa
  - Innervates hamstrings
- Femoral nerve
  - Runs on anterior-medial aspect of knee
  - Innervates quadriceps & skin on anterior-medial leg.

Vessels
- Popliteal artery posterior to knee (& short saphenous vein, which drains into the popliteal vein)
- Great saphenous vein runs up the leg on the anterior-medial side of the leg.
- Knee capsule supplied by superior & inferior glenicular arteries.

Hip
Synovial joint ball-and-socket joint, articulation of head of femur with acetabulum of the pelvis. Function is to support the weight of the body in both static and dynamic postures. The pelvic girdle is formed from two hip bones; sacrum and coccyx.
Stability is dependent on 2 key factors; the deep bony socket and 3 ligaments.

Bones of the hip are divided into 5 areas;
- Ilium - The top region is the iliac crest, which ends anteriorly as the anterior superior iliac spine and posteriorly as the postero-superior iliac spine.
- Ischium - The ischium forms the lower and back part of the hip bone. It is situated below the ilium. The ischial tuberosity is postero-inferior to the acetabulum and is associated mainly with the hamstring muscles of the lower posterior thigh. It is divided into upper and lower areas by a transverse line.
- Pubis - front-most area of the hip bones. It attaches to the ilium on the sides and the ischium on the bottom. It provides structural support, and serves as a place of attachment for the muscles of the inner thigh.
- Coccyx - is small vestigial bone that attaches to the base of the sacrum.
- Sacrum - It is located at the base of the vertebral column that is created by the fusion of five vertebrae. It attaches to the ilium on the sides. It also provides a point of muscle attachment for back muscles.

Muscles
Extension (15-20°) – gluteus maximus and hamstrings.
Flexion (120°) - iliopsoas, rectus femoris, tensor fascia lata and sartorius.
Abduction (45-60°) – gluteus medius and minimus.
Adduction (45°) - adductor brevis, longus and magnus, gracilis and pectineus.
Medial rotation (60-70°) – gluteus medius and minimus, adductor muscles.
Lateral rotation (60-70°) – Piriformis, quadratus femoris, obturator internus, the gemelli, sartorius.

Ligaments of the hip are designed to limit hip extension.
Iliofemoral (y-shaped) – anterior to hip joint, stem attaches to ilium and arms attach to the intertrochanteric line of femur.
Pubofemoral – antero-inferior to hip joint; arises from the pubic component of the acetabulum and attaches to the root of the femoral neck.
Ischiofemoral - passes laterally from the ischium posterior to the neck of femur to attach to the greater trochanter deep to the iliofemoral ligament.
Within the joint is the transverse acetabular ligament, which joins the ends of the acetabular labrum to form the acetabular notch.
Within the joint is also the ligament of the head of femur - ligamentum teres – passes from acetabular fossa to fovea of femoral head.

Blood supply
From the medial circumflex femoral and lateral circumflex femoral arteries, both branches of the deep thigh (profunda femoris), but may also arise directly from the femoral artery.

Nerves
Femoral nerve innervates hip joint anteriorly.
Posterior is innervated by nerve to quadratus femoris. Sciatic nerve sends out small articular branches supplying hip joint. Other nerves include; obturator nerve and superior gluteal.

**Ankle**

**Ankle Joint**

- Synovial, hinge-like with hyaline cartilage
- Synovial membrane covers articular cavity and is itself covered by fibrous membrane
- Allows dorsiflexion and plantarflexion
- Distal ends of tibia and fibula are extended to create a socket for the talus (medial and lateral malleoli)
- Medial ligament (aka deltoid, strong)
  - *Tibionavicular* – attaches to the tubercle of the navicular
  - *Tibiocalcaneal* – attached to the calcaneus
  - *Posterior Tibiotalar* – attaches to the medial tubercle of the talus
  - *Anterior Tibiotalar* – attaches to the medial aspect of the talus
- Lateral ligament
  - *Anterior Talofibular* – attaches anterior aspect of lateral malleolus to anterior aspect of talus
  - *Posterior Talofibular* – runs posteriorly from the medial aspect of the lateral malleolus to the posterior aspect of the talus
  - *Calcaneofibular* – from the lateral aspect of lateral malleolus to the lateral aspect of the calcaneus
- Dermatomes: L4 medial malleolus, L5 lateral malleolus, S1 heel.

Tibialis anterior: tibia to 1st metatarsal – extends ankle, inverts subtalar – deep peroneal nerve
Extensor digitorum longus – fibula to distal phalanges – extends ankle, toes – ditto
Extensor hallucis longus – fib to hallux – extends ankle, hallus – ditto
Peroneus tertius – fib to 5th met – extends ankle, eversion subtalar – ditto
Peroneus longus – fib to 5th met – extends ankle, everts subtalar – superficial peroneal nerve
Peroneus brevis – fib to 5th met – extends ankle, everts subtalar – ditto
Gastrocnemius – femoral condyles to calcaneus – flexes ankle, knee – tibial nerve
Soleus – tib & fib to calcaneus – flexes ankle – tibial nerve
Plantaris – fem condyle to calcaneus – no ankle action (weak knee flex) – tibial nerve
Tibialis posterior – tib n fib to navicular - flexes ankle, inverts subtalar – tibial nerve
Flex. Dig. Longus – tib to distal phalanges – flexes ankle, lat 4 toes – tibial nerve
Flex. Hallucis longus – fib to hallux – flex ankle, hallux – tibial nerve
Popliteus – tib to lat fem condyle – no ankle action, laterl rotation femur – tibial nerve.

- Arteries: anterior tibial (becomes dorsalis pedis) & posterior tibial (becomes medial and lateral plantar arteries)
- Veins: great saphenous from medial *dorsal venous arch* passes laterally and anteriorly to medial malleolus. Small saphenous from lateral *dorsal venous arch* passes laterally and posteriorly to lateral malleolus
<table>
<thead>
<tr>
<th>Muscle</th>
<th>Proximal attachment</th>
<th>Distal attachment</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serratus anterior</td>
<td>1st – 8th ribs</td>
<td>Scapula (medial)</td>
<td>Protraction</td>
</tr>
<tr>
<td>Rhomboids</td>
<td>C7-T4</td>
<td>Scapula (medial)</td>
<td>Elevation &amp; retraction = medial rotation of scapula</td>
</tr>
<tr>
<td>Levator scapulae</td>
<td>C1-C4</td>
<td>Scapula (superior)</td>
<td>Elevation of scapula</td>
</tr>
<tr>
<td>Trapezius</td>
<td>T1-T12</td>
<td>Clavicle &amp; scapula</td>
<td>Lateral rotation scapula</td>
</tr>
<tr>
<td>Teres major</td>
<td>Scapula (inferior)</td>
<td>Humerus</td>
<td>Adduction, medial rotation &amp; extension of shoulder</td>
</tr>
<tr>
<td>Latissimus dorsi</td>
<td>T7-L5</td>
<td>Humerus</td>
<td>Adduction, medial rotation and extension of shoulder</td>
</tr>
<tr>
<td>Subscapularis</td>
<td>Scapula</td>
<td>Humerus</td>
<td>Medial rotation shoulder</td>
</tr>
<tr>
<td>Supraspinatus</td>
<td>Scapula</td>
<td>Humerus</td>
<td>Initiates abduction of shoulder</td>
</tr>
<tr>
<td>Infraspinatus</td>
<td>Scapula</td>
<td>Humerus</td>
<td>Lateral rotation of humerus</td>
</tr>
<tr>
<td>Teres minor</td>
<td>Scapula</td>
<td>Humerus</td>
<td>Lateral rotation of shoulder</td>
</tr>
<tr>
<td>Pectoralis major</td>
<td>Sternum &amp; clavicle</td>
<td>Humerus</td>
<td>Shoulder flexion</td>
</tr>
<tr>
<td>Pectoralis minor</td>
<td>3rd – 5th ribs</td>
<td>Coracoid process</td>
<td>Draws scapular forwards and down</td>
</tr>
<tr>
<td>Deltoid (anterior and posterior fibres)</td>
<td>Clavicle, acromion &amp; scapula</td>
<td>Humerus</td>
<td>Shoulder abduction, flexion, medial rotation, extension &amp; lateral rotation</td>
</tr>
<tr>
<td>Coracobrachialis</td>
<td>Coracoid process</td>
<td>Humerus</td>
<td>Shoulder flexion</td>
</tr>
</tbody>
</table>
3 JOINTS

1. **Glenohumeral joint**: articulation between scapula & humerus
   - Most mobile joint in body, sacrificing stability for mobility.
   - Ball and socket synovial joint (synovial cavity extending inferiorly to allow more abduction)
   - Glenoid fossa is the socket, humeral head the ball
   - Glenoid fossa deepened by rim of fibrocartilage – glenoid labrum – to increase articular surface for humeral head.
   - Despite this, humeral head is larger than the labrum (ie, extensive movement, unstable joint).

2. **Acromioclavicular joint**: Not very mobile
   - Gliding synovial joint: pivots scapula to allow greater degree of shoulder movements.

3. **Sternoclavicular joint**
   - Gliding synovial joint separated by fibrocartilaginous disc, with fibrous cartilage on ends of sternum and clavicle at joint (instead of hyaline cartilage).

Arteries:
1. **Subclavian** – to lateral border 1st rib
2. **Axillary** – from 1st rib to lateral border teres major, branches:
   - Thoracoacromial artery serving acromion
   - Anterior and posterior circumflex humeral arteries surrounding humeral neck, supplying shoulder joint.
   - Subscapular artery serving scapular muscles
3. **Brachial** – from teres major to cubital fossa

Common Conditions Affecting the Shoulder:
- Rotator cuff syndromes
- Impingement syndrome (involving the rotator cuff and subacromial bursa)
- Adhesive Capsulitis
- Calcific tendonitis
- Bicipital tendonitis
- Rheumatoid Arthritis

Trauma:
- Rotator cuff tear
- Glenohumeral dislocation (most common is anteriorly)
- Acromioclavicular dislocation
- Fractures clavicle
- Fractured head/neck of humerus

The Brachial Plexus:
<table>
<thead>
<tr>
<th>MUSCLE</th>
<th>ORIGIN</th>
<th>INSERTION</th>
<th>INNERVATION</th>
<th>FUNCTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extensor carpi radialis longus</td>
<td>Lateral supracondylar ridge of humerus</td>
<td>Dorsal surface of base of metacarpal II</td>
<td>Radial nerve before division into superficial and deep branches</td>
<td>Extends and abducts the wrist</td>
</tr>
<tr>
<td>Extensor carpi radialis brevis</td>
<td>Lateral epicondyle</td>
<td>Dorsal surface of base of metacarpals II and III</td>
<td>Deep branch of radial nerve</td>
<td>Extends and abducts the wrist</td>
</tr>
<tr>
<td>Extensor digitorum</td>
<td>Lateral epicondyle</td>
<td>Base of middle and distal phalanges of fingers (dorsal surface)</td>
<td>Posterior interosseous nerve</td>
<td>Extend fingers and wrist</td>
</tr>
<tr>
<td>Extensor digiti minimi</td>
<td>Lateral epicondyle</td>
<td>Distal phalanx of little finger</td>
<td>Posterior interosseous nerve</td>
<td>Extends the little finger</td>
</tr>
<tr>
<td>Extensor carpi ulnaris</td>
<td>Lateral epicondyle</td>
<td>Dorsal aspect base of metacarpal V</td>
<td>Posterior interosseous nerve</td>
<td>Extends and adducts the wrist</td>
</tr>
<tr>
<td>Supinator</td>
<td>Posterolateral aspect of ulna &amp; lateral epicondyle</td>
<td>Lateral surface of the radius</td>
<td>Posterior interosseous nerve</td>
<td>Supination</td>
</tr>
<tr>
<td>Abductor pollicis longus</td>
<td>Posterior aspect of proximal ulna and radius</td>
<td>Lateral aspect base of metacarpal I</td>
<td>Posterior interosseous nerve</td>
<td>Abduction of thumb</td>
</tr>
<tr>
<td>Extensor pollicis brevis</td>
<td>Posterior aspect of distal third of radius</td>
<td>Dorsal surface of base of proximal phalanx of thumb</td>
<td>Posterior interosseous nerve</td>
<td>Extension of thumb</td>
</tr>
<tr>
<td>Extensor pollicis longus</td>
<td>Posterior surface of ulna</td>
<td>Dorsal surface of base of distal phalanx of thumb</td>
<td>Posterior interosseous nerve</td>
<td>Extension of thumb</td>
</tr>
<tr>
<td>Extensor indicis</td>
<td>Posterior surface of ulna</td>
<td>Base of index finger proximal phalanx</td>
<td>Posterior interosseous nerve</td>
<td>Extends index finger</td>
</tr>
<tr>
<td>MUSCLE</td>
<td>ORIGIN</td>
<td>INSERTION</td>
<td>INNERVATION</td>
<td>FUNCTION</td>
</tr>
<tr>
<td>-------------------</td>
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<td>----------------------------------------------------</td>
</tr>
<tr>
<td>Flexor carpi ulnaris</td>
<td>Medial epicondyle of humerus &amp; olecranon of ulna</td>
<td>Pisiform, hamate and base of metacarpal V</td>
<td>Ulnar nerve</td>
<td>Flexion and adduction of wrist joint</td>
</tr>
<tr>
<td>Palmaris longus</td>
<td>Medial epicondyle of humerus</td>
<td>Proximal phalanges of fingers via palmar aponeurosis</td>
<td>Median nerve</td>
<td>Flexes wrist joint, resists shearing force during gripping</td>
</tr>
<tr>
<td>Flexor carpi radialis</td>
<td>Medial epicondyle of humerus</td>
<td>Base of metacarpals II and III</td>
<td>Median nerve</td>
<td>Flexes and abducts the wrist</td>
</tr>
<tr>
<td>Pronator teres</td>
<td>Medial epicondyle of humerus &amp; coranoid process of ulna</td>
<td>Mid shaft of radius, lateral surface</td>
<td>Median nerve</td>
<td>Pronation</td>
</tr>
<tr>
<td>Flexor digitorum profundus</td>
<td>Anterior medial surface of ulna &amp; anterior medial half of interosseous membrane</td>
<td>Palmar surface of distal phalanges of fingers</td>
<td>Lateral half by median nerve, medial half by ulnar nerve</td>
<td>Flexes distal and metacarpophalangeal joints of fingers. Flexes wrist joint</td>
</tr>
<tr>
<td>Flexor pollicis longus</td>
<td>Anterior surface of radius and radial surface of interosseous membrane</td>
<td>Distal phalanx of thumb (palmar surface)</td>
<td>Median nerve</td>
<td>Flexes interphalangeal and metacarpophalangeal joints of the thumb.</td>
</tr>
<tr>
<td>Pronator quadratus</td>
<td>Distal anterior surface of ulna</td>
<td>Distal anterior surface of radius</td>
<td>Median nerve</td>
<td>Pronation</td>
</tr>
<tr>
<td>Flexor digitorum superficialis</td>
<td>Medial epicondyle, coranoid process of ulna, oblique line of radius</td>
<td>Base of middle phalanges of fingers (palmar surface)</td>
<td>Median nerve</td>
<td>Flexes proximal interphalangeal and metacarpophalangeal joints of fingers. Flexes the wrist joint also.</td>
</tr>
</tbody>
</table>

**Blood vessels**
- Radial and ulnar arteries traverse the wrist joint dorsally and anastomose in the hand and fingers
- Cephalic vein runs dorsolaterally through the anatomical snuff box
- Basilic vein runs dorsomedially
- These two veins anastomose in the hand

**Elbow**
- **Joint**
  - Consists of 2 joints: humeroulnar-radial allowing 150° flexion & radioulnar permitting pronation and supination. No extension beyond anatomical position is possible.
  - Hinge joint therefore 2 collateral ligaments: medial/ulnar collateral fans out distally from medial epicondyle to medial surface of olecranon and coronoid process, lateral/radial fans distally from lateral epicondyle to lateral aspect of ulna to blend with annular ligament. Annular ligament maintains proximal radioulnar joint by holding radius firmly against ulna – forms sling around neck of radius, attaches anteriorly and posteriorly to lateral aspect of ulna.
  - Proximal synovial capsule of proximal radioulnar joint encloses and communicates with capsule of main elbow joint.
<table>
<thead>
<tr>
<th>Muscle</th>
<th>Proximal attachment</th>
<th>Distal attachment</th>
<th>Action</th>
<th>Tested Clinically</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biceps</td>
<td>Long head supraglenoid tubercle, short head coracoid process</td>
<td>Via biceps aponeurosis to the ulna, main attachment to radial (biceps) tubercle</td>
<td>Flexion of elbow joint and supination of forearm</td>
<td>Flexion of elbow with hand pronated against resistance</td>
</tr>
<tr>
<td>Brachialis</td>
<td>Distal half anterior aspect of humerus</td>
<td>Coronoid process of ulna</td>
<td>Flexion of elbow joint</td>
<td>Tested with biceps</td>
</tr>
<tr>
<td>Triceps</td>
<td>Long head infraglenoid tubercle, medial and lateral heads posterior shaft of humerus either side of radial groove</td>
<td>Olecranon process</td>
<td>Extension of elbow joint</td>
<td>Extend elbow against resistance</td>
</tr>
<tr>
<td>Anconeus</td>
<td>Lateral epicondyle of humerus</td>
<td>Lateral aspect olecranon process</td>
<td>Weak extension of elbow joint</td>
<td>Tested with triceps</td>
</tr>
<tr>
<td>Brachioradialis</td>
<td>Lateral supracondylar ridge proximal aspect</td>
<td>Lateral aspect radial styloid process</td>
<td>Flexion of elbow joint and places forearm in mid-prone position</td>
<td>Place forearm in mid-prone position and flex elbow slightly</td>
</tr>
</tbody>
</table>

- Neurovascular
  - Vascular supply to the elbow joint is through an anastomotic network of vessels derived from collateral and recurrent branches of the brachial, profunda brachii, radial, and ulnar arteries.
  - The elbow joint is innervated predominantly by branches of the radial and musculocutaneous nerves, but there may be some innervation by branches of the ulnar and median nerves.

**Cervical and Thoracic Spine**
- Cervical vertebrae: Vertebral body (short in height and square shaped), transverse foramen (perforated by foramen transversarium), spinous process (short & bifid), vertebral foramen (triangular in shape)
- Key features of atlas (C1): No vertebral body, ring-shaped and composed of two lateral masses interconnected by an anterior arch and a posterior arch, lateral mass articulates with occipital condyle of skull above and superior articular surfaces of C2 (the axis) below
  - atlanto-occipital joint allows nodding of head
  - Posterior surface of anterior arch has articular facet for dens, projecting from axis. Dens is held in place by transverse ligament of atlas
- Key Features of Axis (C2): The dens is a superiorly projection from the C2 vertebra that allows for a rotational movement, the alar ligaments join the apex of the dens to the anterior aspect foramen magnum (the hole the spinal cord passes into the cranium).
- Thoracic vertebrae: Posterior facet, superior and inferior demifacets that articulate with the ribs
- Intervertebral discs: Annulus fibrosus (outer ring of cartilaginous fibres, with many layers of fibres that lie in different directions), nucleus pulposus (within the annulus and is jelly-like)
- Muscles: superficial (Iliocostalis lumborum, thoracis, cervicalis, Longissimus thoracis, cervicalis, capitis, Splenius & semispinalis capitis, Serratus posterior inferior)
- Muscles: Deep (Multifidus, semispinalis cervicis, Levator costae, intertransverse/rotator)
- Arterial supply: circumflex branches of the aorta
- Venous drainage: venous plexus

**Lumbar Spine**
Spinal column composed of 24 individual vertebrae divided into three regions: Cervical (7 vertebrae); Thoracic (12 vertebrae); Lumbar (5 vertebrae); These articulate causally with the sacrum (5 fused vertebrae), which in turn articulates with the coccyx (3-5 small fused vertebrae).
A typical vertebra consists of a vertebral body and a posterior vertebral arch. Extending from the vertebral arch is a number of processes for muscle attachment and articulation with adjacent bones. Typical features are: body, pedicles, transverse processes, laminae, articular processes, spinous processes.
<table>
<thead>
<tr>
<th>Vertebrae</th>
<th>Vertebral body</th>
<th>Spinous process</th>
<th>Vertebral foramen</th>
<th>General comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cervical vertebrae</td>
<td>Short in height and square shaped, relatively small</td>
<td>Transverse foramen, Perforated by foramen transversium</td>
<td>Short and bifid</td>
<td>Triangular in shape</td>
</tr>
</tbody>
</table>
| Atlas C1       | None                               | • Ring-shaped and composed of two lateral masses interconnected by an anterior arch and a posterior arch  
• Lateral mass articulates with occipital condyle of skull above and superior articular surfaces of C2 below |                          |                                           |
| Axis C2        | The dens is a superior peg projection that allows rotational movement | • The alar ligaments join the apex of the dens (odontoid process) to the anterior aspect foramen magnum (the hole the spinal cord passes into the cranium.  
• |
| Thoracic       | Intermediate size                  | Long slender and project inferiorly   |                          | Facets for articulation with ribs        |
| Lumbar         | Large size                         | Short blunt spines project posteriorly |                          | Do not overlap therefore good for spinal tap |
**Dermatomes, Myotomes and Sensory pathways**

(A) The main sensory pathways. (B) Spinothalamic tract. To show layering of the spinothalamic tract in the cervical region: C represents fibres from cervical segments which lie centrally; fibres from thoracic, lumbar and sacral segments (labelled T, L and S respectively) lie progressively more laterally.

**Segmental and peripheral nerve innervation and points for testing anterior cutaneous sensation of limbs.** By applying stimuli at the points marked, both the dermatomal and main peripheral nerve distributions are tested simultaneously.

<table>
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<th>Dermatomal Testing</th>
<th>Dermatome</th>
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<tr>
<td>Posterior Aspect of Head/Occiput</td>
<td>C2</td>
</tr>
<tr>
<td>Lateral Aspect of Neck</td>
<td>C3</td>
</tr>
<tr>
<td>Acromioclavicular Jt</td>
<td>C4</td>
</tr>
<tr>
<td>Over Deltoid</td>
<td>C5</td>
</tr>
<tr>
<td>Tip of Thumb/Radial Side of Wrist</td>
<td>C6</td>
</tr>
<tr>
<td>Tip of Middle Finger</td>
<td>C7</td>
</tr>
<tr>
<td>Tip of Little Finger/Ulnar Side of Wrist</td>
<td>C8</td>
</tr>
<tr>
<td>Medial Elbow</td>
<td>T1</td>
</tr>
<tr>
<td>Medial Upper Arm</td>
<td>T2</td>
</tr>
<tr>
<td>Segmental Trunk</td>
<td>T3-T12</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Dermatomal Testing</th>
<th>Dermatome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Groin (by report)</td>
<td>L1</td>
</tr>
<tr>
<td>Upper 1/3 of Anterior Thigh</td>
<td>L2</td>
</tr>
<tr>
<td>Medial Knee</td>
<td>L3</td>
</tr>
<tr>
<td>Lateral Knee to Medial Malleolus</td>
<td>L4</td>
</tr>
<tr>
<td>Lateral Lower Leg to Dorsum of Big Toe</td>
<td>L5</td>
</tr>
<tr>
<td>Heel</td>
<td>S1</td>
</tr>
<tr>
<td>Medial Aspect of Posterior Thigh</td>
<td>S2</td>
</tr>
<tr>
<td>Perianal Area (by report)</td>
<td>S3,4,5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Reflex</th>
<th>Root</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biceps</td>
<td>C5</td>
</tr>
<tr>
<td>Brachioradialis</td>
<td>C6</td>
</tr>
<tr>
<td>Triceps</td>
<td>C7</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Reflex</th>
<th>Root</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patellar Tendon</td>
<td>L4</td>
</tr>
<tr>
<td>Achilles Tendon</td>
<td>S1</td>
</tr>
<tr>
<td>Resisted Motion</td>
<td>Myotome</td>
</tr>
<tr>
<td>-----------------------------------------</td>
<td>----------</td>
</tr>
<tr>
<td>Shoulder Shrug</td>
<td>C2,C3,C4</td>
</tr>
<tr>
<td>Arm Abduction (Deltoids)</td>
<td>C5</td>
</tr>
<tr>
<td>Elbow Flexion</td>
<td>C6</td>
</tr>
<tr>
<td>Elbow Extension</td>
<td>C7</td>
</tr>
<tr>
<td>Wrist Extension</td>
<td>C6,C7</td>
</tr>
<tr>
<td>Wrist Flexion</td>
<td>C7</td>
</tr>
<tr>
<td>Thumb Extension</td>
<td>C8</td>
</tr>
<tr>
<td>Finger Abduction &amp; Adduction</td>
<td>T1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Resisted Motion</th>
<th>Myotome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip Flexion</td>
<td>L1,L2</td>
</tr>
<tr>
<td>Knee Extension</td>
<td>L3,L4</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>L4,L5</td>
</tr>
<tr>
<td>Extension of Big Toe</td>
<td>L5</td>
</tr>
<tr>
<td>Ankle Plantarflexion</td>
<td>S1</td>
</tr>
</tbody>
</table>

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Mechanisms by which joint movement occurs and anatomical structures that restrict movement

<table>
<thead>
<tr>
<th>Movement limited by</th>
<th>Shoulder</th>
<th>Shoulder</th>
<th>Hip</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flexion</td>
<td>avail. cartilage</td>
<td>avail. cartilage</td>
<td>muscle (hamstrings)</td>
</tr>
<tr>
<td>Extension</td>
<td>avail. cartilage</td>
<td>avail. cartilage</td>
<td>ligament(s)</td>
</tr>
<tr>
<td>Adduction</td>
<td>-</td>
<td>-</td>
<td>other leg</td>
</tr>
<tr>
<td>Abduction</td>
<td>avail. cartilage</td>
<td>avail. cartilage</td>
<td>muscle leverage</td>
</tr>
<tr>
<td>Rotation</td>
<td>-</td>
<td>-</td>
<td>muscle leverage</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Structure of ligaments and tendons</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ligaments and tendons are made of fascicles which consist of fibrils</td>
</tr>
<tr>
<td>Fibrils are made up of microfibrils, in which fibroblasts secrete type 1 collagen into dense, regular parallel rows</td>
</tr>
<tr>
<td>This structure gives them tensile strength but weakness in compression and twisting</td>
</tr>
<tr>
<td>Other macromolecules in the extracellular matrix are: elastin, glycoproteins, and proteoglycans</td>
</tr>
<tr>
<td>Tendons attach muscles to bone, the following tissue make up this transition: tendon, fibrocartilage, mineralised fibrocartilage, bone</td>
</tr>
</tbody>
</table>

Carpel Tunnel Syndrome

- Compression of median nerve as it passes through the carpal tunnel (formed by space between transverse carpal ligament and carpel bones) at the wrist. Idiopathic but associated with several conditions.
- More common in women (especially middle-aged and elderly)
- Conditions predisposing to carpal tunnel syndrome: diabetes mellitus, hypothyroidism, rheumatoid arthritis, pregnancy, acromegaly, trauma (e.g. wrist fracture).
- Clinical features: pain/parathesia in the median nerve distribution which can radiate to the elbow, often worse at night, often wake the patient from sleep.
- Phalen’s (wrist held in maximal palmar flexion) and Tinel’s (tap over median nerve proximal to transverse carpal ligament in the wrist) test may reproduce the symptoms.
- Investigation with nerve conduction studies shows reduced nerve conduction at the wrist.
- Definitive treatment is with surgical decompression of the carpal tunnel by division of the transverse carpal ligament (under local anaesthetic). Wrists splints may help nocturnal symptoms, corticosteroid injections may bring some pain relief.

Basic sciences

Genetic conditions in the foetus, screening, organization of genes on chromosome

Inherited disorders

1. **Autosomal recessive disorders** (expressed in homozygous people, so only one parent needs to have the Dominant Gene, e.g. cystic fibrosis and sickle cell anaemia)
2. **Autosomal dominant disorders** (expressed in heterozygous person, so both parents need to have the Gene, e.g. Huntington’s)
   - the protein produced by the one “normal” gene cannot compensate
   - disorders with high variance in manifestation; some people have the genotype but not the phenotype; parent may be mildly affected but have a seriously affected child.
3. **X-linked disorders** (Women have the disease only if gene inherited from each parent, less likely than Men, who need only receive the X chromosome from their mothers, e.g. haemophilia A)
4. **Abnormal chromosome numbers**:
- Non-disjunction:
  o Chromosome/chromatids fail to separate ('non-disjunction') in meiosis/mitosis = 1 daughter cell gets 2 copies, 1 daughter cell gets 0
  o during meiosis \(\rightarrow\) ovum/sperm with extra chromosome \(\rightarrow\) trisomic foetus/ one chromosome
  o trisomy 13, 18, and 21 survive; all other combinations spontaneously abort, Down’s = Trisomy 21

5. Abnormal chromosome structures (disruption to the DNA and gene sequences \(\rightarrow\) genetic disease)

i) Deletions = portion of a chromosome deleted is a problem if two copies of the genes are needed (e.g. Prader-Willi syndrome, deletion of long arm of chromosome 15)
ii) Duplications = portion of the chromosome is has 2 copies (e.g. Charcot-Marie-Tooth disease from small duplication of a region of chromosome 17)
iii) Inversion = reversal of a segment within a chromosome; e.g. abcdefgh becomes abcgedfh, for example, haemophilia (p. 472)
iv) Translocations = two chromosome regions join together when they shouldn’t (e.g. in somatic cells this is associated with tumorigenesis)

6. Mutations

i. Point (substitute one nucleotide for another)
ii. Splicing (sequence that guides introns from mRNA are mutated \(\rightarrow\) abnormal splicing \(\rightarrow\) translated protein may carry intron sequences \(\rightarrow\) altered amino acids incorporated into polypeptide chain)
iii. Termination (‘stop’ codons mutated)

Screening:

1. Ultrasound:
   i. nuchal translucency to exclude major chromosomal abnormalities (e.g. trisomies and Turner’s syndrome)

2. Bloods:
   i. pregnancy-associated plasma protein-A (PAPP-A from the syncytial trophoblast) for trisomy 21;
   ii. Triple test for chromosomal abnormalities.

3. Amniocentesis:
   i. cells in amniotic fluid sampled, cultured and karyotypically analysed for chromosomal abnormalities e.g. Down’s

Organisation of Genes in chromosome:

- Human cells have 46 chromosomes, 2 of each one of the 23 different types
- Chromosomes contain genes
- Gene = unit of heredity that specifies an RNA sequence
- RNA sequence determines the proteins that the body is able to synthesise
- Genes are arranged on chromosomes
- Allele genes hold the same place (locus) in the homologous chromosomes
- Heterozygote = diploid cell or organism with different alleles of a gene at one locus on homologous chromosomes
- Homozygote = diploid cell or organism with the same allele of a gene at one locus on homologous chromosomes

Polymerase Chain Reaction (PCR): how it can be used to identify, for example, chlamydia.

- PCR can be used to amplify a specific sequence of DNA from the genome of an organism, can be used to amplify known section of chlamydia genome from a swab or urine sample.
- DNA purified from sample, heated to 95° C to separate DNA strands (break up H bonds), primers added and attach to 3’ end of each strand when cooled to 55° C, nucleotides and Taq polymerase added temp increased to 70° C to allow replication, cycle repeated, products run on electrophoresis gel, positive test will show a band.
Role of enzymes and the role of liver enzymes in diagnosis

- Biological catalysts, enzyme forms complex with substrate(s) and increase likelihood of conversion into product(s), reduce the Gibbs free energy required for conversion of substrates to products (less activation energy), accelerate chemical reactions.
- At constant enzyme conc speed of conversion of substrate to products is dependent on [S]. Km is the [S] required for Vmax ½.
- \[ V = \frac{V_{\text{max}} \times [S]}{[S] + K_m} \]. Large Km means low enzyme-substrate complex affinity, low Km means high substrate affinity.
- Co enzyme assist enzyme by transferring energy (phosphate group of ATP), electrons (NAD, NADP) or functional group (Acetyl CoA).
- Enzyme inhibition: irreversible alteration of enzyme (aspirin on COX), competitive reversible blocking of active site (ibuprofen), non competitive reversible (allosteric).
- Enzyme activity affected by temp, pH and [S].
- LFTs: ALT (transfers alanine R group to produce pyruvate and glutamate, raised when liver is injured or inflamed), ALP (removes phosphate groups from nucleotides and proteins, bone derived and liver derived).
- Bone derived ALP: raised in pagets, fracture healing, secondary bone metastases.
- Liver derived ALP: raised in cholestasis, liver tumour, liver metastasis (esp if gamma GT also raised), drug induced hepatotoxicity.
- Albumin: maintains osmolarity in blood and transports drugs and many hydrophobic molecules, low levels are associated with liver disease.
- Bilirubin: breakdown product of heme before excretion into bile, raised conjugated levels associated with cholestasis, gallstones, pancreatic tumour, cirrhosis and hepatitis, raised unconjugated levels associated with excessive erythrocyte breakdown.
- Gamma GT: marker for excessive alcohol consumption.
- LFTs can be used for diagnosis and monitoring of disease/treatment.

Oncogenes: role in normal cell proliferation and in carcinogenesis. Grading and staging of tumours

- Carcinogenesis: cancer caused by altered gene expression of tumour suppressor gene or oncogene (mutation), cell grows indefinitely and blocks apoptosis (immortalisation), cell ignores normal growth restraints/signals (transformation), cell invades normal tissue (growth and metastasis).
- Carcinogens: initiators and promotors.
- Chemical modification of bases in tumour suppressor or oncogene can also cause cancer (epigenetic changes), associated with failure of DNA repair systems.
- Oncogenes: involved in pathways that stimulate cell division, gene amplification e.g. MYC family, chromosome translocation e.g. BRCA ABL, base substitution e.g. Ras family, single mutation not enough to create cancer cell, 2-7 events required, mutation in only one allele is sufficient.
- Tumour suppressor genes: involved in prevention or inactivation of cell division, also genes involved in apoptosis, extra cellular signals tightly control cell division, when these signals are absent a cell induces apoptosis, mutation on both alleles is required, p53 is a major tumour suppressor gene, mutant p53 present on 30-50% of cancers, functions (DNA repair, induce apoptosis, arrests cell cycle if DNA damage detected, allows time for damaged DNA to be repaired before cell cycle continues).
- Chronic myeloid leukaemia: transformation of the Philadelphia gene, occurs in pluripotent stem cell in bone marrow, translocation occurs between chromosomes 9 and 22, results in fusion of oncogenes BRD and cABL.
- Malignant neoplasm: group of cells that have escaped normal growth control and have invaded surrounding tissue, differentiation refers to the degree with which the tumour differs from the parent tissue.
- Grading of tumours: system used to measure the degree of progress of a tumour or neoplasm, degree of cellular abnormality under the microscope, the higher the grade the more poorly differentiated the neoplasm from the surrounding tissue.
- Low grade (well differentiated), intermediate grade (moderately differentiated), high grade (poorly differentiated).
- Staging: degree of tumour spread to other tissues, important for prognosis.
Cell death during the human life cycle

- Apoptosis: programmed cell death, safe way of dealing with cells that pose a threat e.g. virus infected, DNA damaged, immune cells, the process (cell shrinks and detaches from surroundings, chromatin condenses and DNA is fragmented, membrane buds off to form apoptotic bodies, apoptotic bodies are phagocytosed and recycled.
- Importance of apoptosis: rate of cell division in a tissue must be balanced by rate of cell death to maintain tissue size and function, turn over required to replace damaged and diseased cells, apoptosis required for sculpting of tissue and organ during development e.g. finger and toe formation, prevention of cancer, cells irreparably damaged by ionising radiation, clonal selection of B and T lymphocytes.
- Activators of apoptosis: loss of growth signals from surrounding cells, death activators (TNFα, Fas ligand), DNA damage
- Necrosis: uncontrolled cell death following damage, swelling and mitochondrial damage, lysis and release of cellular contents, inflammation oedema and damage to surrounding cells
- Causes of necrosis: ischaemia, infection, toxins, inflammation, radiation, pattern of necrosis varies depending on cause

Bone structure and how it is maintained

- Types of bone – Long (femur), Short (carpals), flat (skull), irregular bones (vertebrae)
- Spongy bone (cancellous or trabecular) usually epiphysis, light large surface area therefore remodelled more quickly, also in cortex of flat bone with interconnecting trabeculae
- Cortical bone (hard, compact or lamellar) usually diaphysis, provides strength with mechanical and protective function. Round layered lamellae surround harversian canals, with lacunae (small cavities) between. Lymph/artery and vein run in canal, which are joined horizontally by Volkman’s canal.
  - Regions: Epiphysis (prox. and dist. ends) Diaphysis (shaft), Metaphysis (shaft and growth plate/epiphysis)
  - Membranes: Endosteum – single layer cells lining inner surface of medullary cavity, Periosteum – bilayer membrane on outer layer of bone (excluding joints surfaces of long bone) contains nerve endings and pain fibres.

Bone maintenance (bone remodelling cycle):

- Bone modelling units called osteoblasts stimulate osteoclasts to resorb bone, and then the osteoblasts lay down new bone. This happens over a period of two years. ~10% undergoing remodelling in an adult and 90% quiescent.
- Resorption –Osteoclasts: Production controlled by osteoblasts, large phagocytic multiple nuclei cells. From stem cells. Resorption of bone. Release HCl and proteolytic enzymes through ‘ruffled border’ in to space ‘howships lacuna’. Osteoclasts apoptose (bisphosphonates and oestrogen actually promote this!)
- Reversal -Osteoblasts: found at bone surface, release osteoid (type I collagen) to form bone, removed or remains (differentiating to osteocytes) to become a lining, rich in alkaline phosphatase.
- Formation –matrix synthesis
- Quiescence –after mineralisation this phase occurs

Muscle: how structure relates to function

Muscle
Structure of Muscle
- Sarcomeres made from myofilaments
- Myofibrils made from end to end sarcomeres → striation

Structure of Fibril
- Sarcomeres = dark and light zones
- Dark = A bands, light = I bands
- I band has Z line in centre
- Sarcomere = space b/w 2 Z lines
- H band = pale area when muscle is relaxed where A and I bands don’t overlap
- A bands = thick myosin
- I bands = thin actin
- T tubules from sarcolemma to A and I bands
- Sarcoplasmic reticulum surrounds fibrils
- Each T tube in contact with 2 regions of sarcoplasmic reticulum

Figure 1: Muscle belly split into various component parts (from Essentials of Strength Training & Conditioning, National Strength & Conditioning Association)
To Contract
- Action potentials spread along T tubules
- Ca\(^{2+}\) channels in sarcoplasmic reticulum open = excitation: contraction coupling
- Ca\(^{2+}\) released from sarcoplasmic reticulum → ↑Ca\(^{2+}\) conc → troponin binds to Ca\(^{2+}\) → actin binding site exposed
- Myosin has 2 heads and a tail
- Breakdown of ATP → ADP + P → change in angle of the head, head moves towards actin
- Each head forms cross-bridge with 1 actin filament
- Myosin and actin slide past each other = cross-bridge cycling (shortening muscle)

To Relax
- Ca\(^{2+}\) in the sarcoplasm is pumped back into sarcoplasmic reticulum
- Troponin and tropomyosin bind to actin preventing it from binding to myosin heads
- ATP binds to myosin → actin and myosin dissociate

Function
- Skeletal muscles maintain body posture
- Create movement of limbs and internal organs

Structure and function of a neurone
Nervous system consists of 2 types of cells; neurones and neuroglia. Neurones are cells that carry nerve impulses. Neuroglia are cells which provide structural or metabolic support to the neurones and this includes Schwann cells.

There are 3 types of neurones; motor, sensory and relay neurones.
Motor neurones carry nerve impulses to muscle fibres at neuromuscular junctions.

<table>
<thead>
<tr>
<th>Feature</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Large cell body</td>
<td>This contains the nucleus and many of the other cell organelles.</td>
</tr>
<tr>
<td>Dendrons</td>
<td>These are short extensions of the cell body which transmit impulses towards the cell body.</td>
</tr>
<tr>
<td>Dendrites</td>
<td>Each dendron has smaller extensions called dendrites. These dendrites are stimulated by other neurones and are points at which impulse transmissions always start in a motor neurone.</td>
</tr>
<tr>
<td>Axon</td>
<td>It is a single extension of the cell body. It can be up to one metre long. It always transmits nerve impulses away from the cell body. It ends in a series of synaptic knobs, which are structures that stimulate the effector.</td>
</tr>
<tr>
<td>Myelin</td>
<td>In mammals many axons are surrounded by a sheath of fatty material – myelin. The myelin enables the neurones to conduct nerve impulses rapidly.</td>
</tr>
</tbody>
</table>

Sensory neurones carry impulses into the spinal cord where they have synapses with relay neurones. Relay neurones have synapses with motor neurones, which are then able to produce an effect.

When a neurone is not conducting an impulse it is in ‘resting potential’.
Resting potential is the difference in charge between the inside and outside of a neurone cell (when it is not conducting an impulse).
It is normally around -70 mV (millivolts).
The concentration of sodium ions (Na\(^{+}\)) is greater on the outside the cell than on the inside.
The concentration of potassium ions (K\(^{+}\)) is greater on the inside of the cell than on the outside.
These concentration gradients are maintained by sodium-potassium pumps.
During an action potential a stimulus causes sodium and potassium gates at a particular point in membrane of the neurone to open, allowing sodium ions to enter and potassium ions to exit.

The inside becomes positive with respect to the outside (~+40 mV). This is called membrane depolarisation. The sodium-potassium pumps then work to restore the resting potential. The membrane is said to be repolarised.

Once the polarity across the membrane has changed during an action potential, it has to return the resting potential before another action potential can occur. This is known as the refractory period.

For a stimulus to generate an action potential it needs to be of a certain magnitude. This magnitude is known as the threshold value.

An action potential will not occur if the stimulus does not reach this magnitude.

For this reason action potentials are said to be all or nothing.

When an action potential reaches the pre-synaptic membrane of a synapse, calcium channels open and calcium ions diffuse in.

These calcium ions cause the synaptic vesicles to fuse with the pre-synaptic membrane, releasing acetylcholine into the synaptic cleft.

Acetylcholine binds to receptors on the postsynaptic membrane of the adjacent neurone, causing sodium ion channels to open and releasing acetylcholinesterase.

This causes an action potential in the post-synaptic neurone.

Acetylcholinesterase acts to break down acetylcholine into its constituents which are absorbed across the pre-synaptic membrane before being used again.

Peripheral nervous system: components and how electrical impulses are transmitted

Components of peripheral nervous system
- Peripheral nervous system: 2 parts – Somatic (innervates skeletal muscle and autonomic (smooth and cardiac muscle)
- Somatic consists of a single neuron between CNS and peripheral, autonomic has a two-neurone chain (connected with synapse: Autonomic ganglion) between CNS and effector. Somatic causes muscle excitation, autonomic: either excitatory or inhibitory.
- ANS controlled by hypothalamus: sympathetic (fight or flight: activates physical exertion) & parasympathetic (counteracts physical exertion; state of rest).

How electrical impulses are transmitted
- Neurotransmitter (Acetylcholine (Ach) for neuromuscular, noradrenaline for sympathetic) transmits action potential to presynaptic membrane of neuron.
- Calcium channels open, Ca rushes in, attracts vesicle of neurotransmitter to fuse with presynaptic membrane releasing acetylcholine into the synaptic cleft.
- Ach binds with receptors on postsynaptic membrane causing sodium ion channels to open. Ach recycled by Acetylcholinesterase.
- Resting potential: difference in charge between the inside and outside of a neurone cell. ~-70mV.
- Sodium ion conc greater on outside, potassium ion conc greater on inside; gradients maintained by Na+/K+ (sodium/potassium) pump (uses ATP for active transport).
- Action potential:
  - Stimulus strong enough to cross threshold value: AP opens voltage gated Na channels, Na rushes into neuron cell body – more positive on inside now (depolarisation).
Gates quickly close whilst voltage gated K+ channels open so K+ rapidly diffuse out. Cell now more pos on outside (hyperpolarisation).

K channels close. Na ions in cell diffuse to area of lesser conc, changing polarity of membrane ahead of AP, causing voltage gated Na channels to open. Na rushes in so again AP spreads to adjacent part of neuron. K+ diffuses out again restoring neg charge inside.

Meanwhile Na/K pump pumps 3xNa out and 2xK in restoring resting potential (repolarisation).

Refractory period: The time it takes for the resting potential to be restored from depolarisation before another action potential can occur.

Neurological mechanisms involved in voluntarily contracting a muscle
1. Skeletal muscles are under voluntary/reflex control by motor neurons of the somatic motor system
2. Somatic motor neurons = efferent neurons with cell bodies located in the central nervous system (CNS)
3. A single muscle cell responds to one motor neuron (whose cell body is in the ventral horn of the spinal cord)
4. Axon of a motor neuron branches near its termination \(\rightarrow\) innervates multiple individual muscle cells
5. The group of muscle fibers innervated by all of the collateral branches of a single motor neuron is the motor unit
6. Muscles \(\rightarrow\) wide range of forces + shortening by varying the number of motor units excited within the muscle.
7. Innervation of skeletal muscle according to need for fine/crude movements: the number of muscle fibers innervated by a single motor neuron decreases with the need for fine movement (extraocular muscles need fine precision, innervation ratio \(\sim\) 3 muscle fibers per neuron; the gastrocnemius muscle, \(\sim\) 100 to \(\sim\) 1000 muscle fibers per neuron)

(I think the neurological part ends here, but for completion’s sake here you go)
8. A motor nerve axon the contacts each muscle fiber to form a synapse = neuromuscular junction
9. At this level excitation of the skeletal muscle involves chemical activation by release of acetylcholine (ACh) from the motor nerve terminal
10. ACh binds to nicotinic receptor \(\rightarrow\) depolarizing the end-plate potential
11. A potential of sufficient magnitude \(\rightarrow\) propagates an action potential in sarcolemma membrane potential to the firing threshold
12. Voltage-gated Ca\(^{2+}\) channels open, Ca\(^{2+}\) enters the pre-synaptic membrane
13. Rise in Ca\(^{2+}\) \(\rightarrow\) synaptic vessels fuse into presynaptic membrane \(\rightarrow\) Ach diffuses across synaptic cleft and binds to post-synaptic cell \(\rightarrow\) eventually Ach is taken up by pre-synaptic terminal/diffuses away from synapse

Being a doctor

Doctor’s role of certifying a patient as unfit for work and patient autonomy

Sick certificate:
- Illness for less than 7 days = self-certify (SC2 form)
- Doctors are required to issue a sick note due to illness lasting more than 7 days
  - Med 3 (most common) - examine on day, patient must be seen
  - Closed certificate issued (has return date) if pt can return to work in 14 days
  - Open certificate = up to 6 months
  - Med 4 for social security, no need to keep re-issuing Med 3’s
  - Med 5 patient not seen but written report received less than 1 month previously
  - Med 6 when on Med 3 form but GP does not wish to disclose diagnosis
  - Med 10 for inpatient
- A&E can also issue sick notes as well as GP’s

Autonomy:
- Patient has autonomy = patient in control of their own health
- Certificate could be beneficial to pt as time off work may aid recovery
- Certificate may also be harmful as pt may adopt long term sick role
- Doctor has duty of care to pt so must only give note if pt needs it
- I.e. giving sick note is similar to giving other treatment
- It can be harmful for society to give unnecessary notes as it costs money to get another employee to cover
How doctors are regulated: GMC

- GMC: hold the medical register, register newly qualified drs, issue guidance on good practice, investigate complaints against drs and issue punishment, regulates medical school and post grad curricula.
- Fitness to practice: panel consists of drs and general public, investigate cases of impaired fitness to practice (too ill to work safely, not up to date with medical knowledge and skills, abuse of role as a dr), possible results include suspension/removal from register, placing conditions of the drs registration or issuing a warning
- Complaint: if not fitness to practice related or outside of GMC remit then drs employer will do their own ix, otherwise GMC will launch ix
- ix: gathering of documents and witness statements, expert opinion, review of drs general performance and health, conclusion (no further action, issue warning, agree undertakings or refer to fitness to practice)

The process begins with a complaint being made which is then investigated. Either by the physician’s employer or the GMC in serious cases. This can then have 4 possible outcomes;

1. Conclude the case without further action
2. Issue a warning
3. Refer for Fitness to Practise
4. Undertakings on health and performance issues following a health or performance assessment.

The evidence is then reviewed so a fully informed decision can be made (adjudication).

Over the past few years a number of cases of negligence/malpractice undertaken by the GMC;

Harold Shipman – jailed for the murder of 15 patients and is suspected of killing more than a 100 others.

Rodney Ledward - gynaecologist was struck off medical register in 1998 after being found guilty of bungling 13 operations, causing the patients to be scarred and maimed.

Clifford Ayling – was a GP convicted in 2000 for sexually abusing ten of his female patients, he was sentenced to four years and struck off the medical register.

How doctors recognise signs of pressure and stress on themselves and others, the importance of self-care, stress reduction and avoidance of unhealthy practices such as alcohol misuse, what to do if concerned

- Stress costs NHS £300-400m per year in lost productivity
- Causes: demands of job e.g. long working hours, less autonomy due to increased regulation and guidelines, relationships at work, competition for posts, changes to contracts, danger to self (viral, accidental), litigation threat omnipresent.
- Symptoms: Behavioural (insomnia, anorexia, smoking, booze); physical (headaches, nausea); mental (lack of concentration); emotional (irritable).
- Booze / drugs as negative coping behaviours. Many docs referred to GMC Fitness to Practice have booze/drug problems.
- Blood alcohol concentrations of 80 mg/ 100 ml (the legal limit for driving) double the risk of an adverse incident whilst concentrations of 100 mg/ 100 ml increase the risk tenfold for docs.
- Docs supposed to be respectable and competent so fear admission of problems.
- Docs also look out for each other, fear of GMC action: average delay between onset and consulting about an alcohol or drug problem is more than six years.
- Need to confront colleagues / report to higher authorities in order to protect patients, other staff, and the person themself: duty of care.
- Never confront alone: colleague may claim you have malicious motive or may try to get someone ‘on their side’ ie denial; also give colleague choice of avenues for support.
- Probs: docs self-treat and rarely see GP.
- Self management: Make colleague aware of the signs of stress, change reactive behaviour patterns to positive ones (eg teamwork, hobbies), avoid stressors, get rest, carry around snacks, develop positive coping skills (planned exercise / time management or assertiveness courses).
- Docs’ resources:
<table>
<thead>
<tr>
<th>Agency</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>National counselling service for sick doctors</td>
<td>Confidential advice from medical advisors.</td>
</tr>
<tr>
<td>BMA 24 hour stress counselling service</td>
<td>Stress counselling</td>
</tr>
<tr>
<td>BMA: doctors for doctors</td>
<td>Speak to another doctor in confidence.</td>
</tr>
<tr>
<td>GMC’s fitness to practise division</td>
<td>Provides informal advice and guidance about invoking the GMC’s health procedures.</td>
</tr>
<tr>
<td>The Association of Anaesthetists</td>
<td>Operates its own scheme for all anaesthetists, including those in training.</td>
</tr>
<tr>
<td>The Sick Doctors’ Trust</td>
<td>24 hour advice – chemical dependence.</td>
</tr>
<tr>
<td>The British Doctors’ and Dentists’ Group</td>
<td>Ongoing group support from recovered drug and alcohol abusers, students welcome.</td>
</tr>
</tbody>
</table>

**Factors (biases and errors in memory, attention, concentration and decision making ability) that can influence interactions with patients and the diagnostic skills of the doctor. Factors that could exacerbate or ameliorate their impact.**

Causes of Biases and errors in Memory, Attention, Concentration, Decision Making:

1. **Job related**
   - Poor communication with team
   - Poor Team Work
   - Lack of support from peers and/or organisation
   - Not coping with stress
   - Overworked and under resourced
2. **Personal**
   - Lack of sleep
   - Personality (self critical)
   - Unsupportive relationships
   - Family problems, divorce
   - Addiction (alcohol and drugs to cope with stress)
   - Unhealthy lifestyle (sedentary, smoking)

What makes it better:

1. **Physical self-care** (exercise, healthy diet, snacks on hand at work, water, eating breakfast, treating underlying illness)
2. **Emotional self-care** (reduce stress in healthy ways, time off, hobbies, family, friends, counselling, peer support)

What makes it worse:

1. **Self-treating** (carry on working, self-med, addiction, denial, shame, isolation, reluctance to seek own GP)
2. **Illness** (physical or mental)
3. **Unnoticed by peers** (suggest help for doctor friend, patient-safety issues)
Chronic illness
Factors which may predispose a person to developing chronic disabling pain
- Belief that pain and activity are harmful
- Sickness behaviours such as extended time in bed
- Social withdrawal
- Emotional probs:
  - negative mood
  - depression – 40-50% chronic pain patients have depression
  - anxiety or stress – direct correlation to perception of pain and levels of anxiety
- Problems and/or dissatisfaction at work
- Problems with claims or compensation or time off work
- Overprotective family; lack of support
- Inappropriate expectations of treatment
- Loss of control
- Illness beliefs can have an effect:
  - Identity: Lack of knowledge about diagnosis
  - Cause: Incorrect perception of what is causing the pain
  - Time-line: correct expectations of length of illness
  - Consequences: understanding how the illness will effect their life
  - Cure/Control: knowing if there is a cure and how the disease can be controlled

Impact of chronic disease on patients’ psychological well being
Psychological Impact of Chronic Disease
Chronic illness results in:
- Diagnosis → sick role
  - exempted from normal responsibilities
  - not blamed for being sick
  - expected to seek help from a doctor
  - expected to comply with medical advice
- Impact on everyday life
  - unable to undertake usual day to day tasks
  - becomes more isolated
  - leading to frustration and depression
- Identify chronic illness
  - shock and denial
  - uncertainty and anxiety
  - yearning and searching
  - disorganisation and despair
  - feel like a failure
- Reorganisation
  - acceptance of frailty
  - reconstruction of new vision of self and body
- Stigma and discrimination
  - low self esteem
  - reconstruction of new vision of self and body
  - reconstruction of role in society

Connective tissues (SLE, Sjogrens, PMR, CREST, dermatomyositis, Vasculitis)
Clinical features and investigations of connective tissue disorders
1. Inflammatory Muscle Disease (Polyomyositis and Dermatomyositis)
   Autoimmune, chronic inflammation of striated muscle (polyomyositis) and sometimes skin (dermatomyositis), anti Jo1 antibodies; DM associated with malignancy, treat with steroids

Clinical Features
- Insidious (3-6 months), Progressive, Painless (usually) proximal muscle weakness, with/ without rash is the hallmark of the disease
- **Cutaneous** manifestation:
  - ✓ Rash is erythematous and scaly: (Heliotrope over eyelids, Grottron’s over finger joints, macular over face and neck, chest, shoulders)
  - ✓ Calcinosis (usually children)
  - ✓ Vasculitis (causes ulceration)
  - ✓ Telangiectasia (over cuticles)
- Extramuscular: fatigue, malaise, weight loss, fever
- Musculoskeletal: arthritis, proximal muscle weakness
- Pulmonary: lung fibrosis
- Cardiovascular: Arrhythmias, heart failure
- GI: dysphagia, regurgitation
- Malignancy: lung, oesophagus, breast, colon, ovary
- Creatine kinase (CK), Aspartate, alanine transaminases, Lactate dehydrogenase

2. **Polymyalgia Rheumatica (PMR)**
   - Clinical Features:
     - Proximal, bilateral, symmetrical and diffuse pain and stiffness in the neck, shoulder and pelvic girdles
     - Systemic features: low grade fever, fatigue, weight loss and high inflammatory markers (ESR, CRP)
     - Responds to small doses of corticosteroids (10-15 mg prednisolone/day) can be dramatic, treat up to 2 years
     - Incidence: >50s, female> male
     - Diff Diagnosis: Neoplasm, Joint disease (OA, RA, SLE), Myeloma, Leukaemia, Lymphoma, Polymiositis and other muscle disease, Infections, Bone (Paget’s), Hypothyroidism

3. **Giant Cell Arteritis**
   - Clinical Features
     - Fatigue
     - Headaches
     - Jaw claudication (muscle pain when moving it), vision disturbance/loss (emergency)
     - Scalp tenderness
     - Polymyalgia Rheumatica
     - Temporal and occipital arteries are tender with reduced or no pulse and may be visible
     - Complications: CVA, MI, Peripheral neuropathy
     - Temporal Artery biopsy: inflammatory cell infiltration, giant cells and granulomata visible, unless you get a ‘skip lesion’

4. **Systemic Lupus Erythematosus (SLE)**
   - Immune complex disease involving excess antibody formation and immune complex deposits in tissue; Antibody properties are important in determining the pathogenic potential of immune complexes; Complement deficiencies may alter immune complex handling; Genetic variations in receptors for immune complexes alter antibody binding and influence the disease process; inflammatory, multi-system, unknown aetiology with diverse manifestations, variable course and prognosis, affects more women between 15-40 years of age;
   - Clinical Features from most common to less:
     - Non-specific: fatigue, malaise, weight loss, fever
     - Arthritis: Swan Neck and Boutonniere deformities due to tenosynovitis and fibrosis, osteonecrosis
     - Skin: photosensitivity, malar butterfly rash (acute), annular (subacute), chronic (discoid); Alopecia; Ulceration in nose, mouth, vagina
     - Renal: glomerulonephritis (most common cause of death) - monitor BP, urynalysis for protein to monitor renal disease
     - Vasculitis: atherosclerosis, Pulmonary Embolism
     - CNS: seizures, stroke, movement disorder, Headache, cranial/peripheral neuropathy, psychosis
- **Lungs:** pleurisy and pleural effusions
- **CVS:** Pericarditis, myocarditis, endocarditis, coronary heart disease, Raynauds (1/3 of SLE)
- **Antiphospholipid Syndrome**
- **Sjögren’s**
- **Drug-Induced SLE by:** Carbamazepine, Chlorpromazine, Hydralazine, Isoniazid, Methyldopa, Minocycline, Penicillamine, Phenytion, Quinidine, Sulphasalazine

5. **Anti-phospholipid syndrome**
   Recurrent vascular thrombosis, fetal loss and thrombocytopenia with high levels of antiphospholipid antibodies (APA). This can complicate other diseases such as SLE.

   Clinical features:
   - Fetal complications (3rd trimester abortion usually due to placental infaction, or premature birth)
   - Venous thrombosis (DVT and Pulmonary Embolism)
   - Arterial Thrombosis (Stroke, TIA)
   - Thrombocytopenia (not severe to cause haemorrhage)
   - Livedo reticularis
   - Leg Ulcers
   - Cardiac abnormalities (aortic and mitral regurgitation)
   - Chorea
   - Epilepsy
   - Migraine
   - Haemolytic Anaemia
   Recommendations:
   - Avoid the oral pill
   - Don’t smoke
   - Treat HTN and high cholesterol or diabetes
   - Asymptomatic: low dose Aspirin
   - Warfarin lifelong, keep INR between 2.5-3.0, but stop before conception, enoxiparin S/C curing pregnancy

6. **Sjögren’s syndrome**
   Primary or secondary, slowly progressive, inflammatory autoimmune disease affecting primarily the exocrine glands; Lymphocytic infiltration causes replacement of functional epithelium and reduces secretions in eyes, mouth etc

   - **Immunopathology:** Trigger autoimmunity; Perpetuation of reactivity via normal regulatory mechanisms; Ongoing inflammatory process causes damage, fibrosis affecting glands
   - **Mucosal Dryness:** Eyes, mouth, trachea, vagina
   - **Major salivary gland enlargement** and atrophic gastritis
   - **Non-erosive polyarthritis**
   - **Skin:** Raynauds, digital ulcers
   - **Lung, kidney, vasculitis, peripheral neuropathy**
   - Increased risk of lymphoma
   - Associated with: RA, SLE, Systemic Sclerosis, PM, Primary biliary cirrhosis

   Treatment:
   - Follow for disease progression
   - Lubrication — eyes, mouth, vagina
   - DMARD — Hydroxychloroquine
   - Extra-glandular disease — steroids, further immunosuppression
   - Treat lymphoma accordingly to histological type (by haematologists)
7. **Systemic Sclerosis**
   - Autoimmune, affecting skin and internal organs
   - Characterised by fibrotic arteriosclerosis of peripheral and visceral vasculature
   - Variable degrees of collagen accumulation in skin and viscera
   - Activation of immune system, Release of cytokines (TGF-β, PDGF, IL4 etc) from platelets, macrophages, T-cells, Cytokine activation of fibroblasts to increase extracellular matrix production
   - anticentromere and anti-Scl-70 autoantibodies
   - Limited (skin) or Diffuse (skin and organs)

   **Limited form (CREST):** skin thickening over distal sites, hands, feet and neck, microstomia
   - Calcinosus
   - Raynaud’s Phenomenon (see digital pitting, ulceration, gangrene, amputation; ESR elevated and ANA positive)
   - (o)esophageal dysmotility (heartburn)
   - Sclerodactyly
   - Telangiectasia
   - Anti-centromere antibody

   **Diffuse form:**
   - **Skin:** whole body, small mouth (microstomia), ulceration, dryness and irritation, Telangiectasia, calcinosis
   - **Musculoskeletal features:** arthralgia and myalgia, & later muscle atrophy and weakness
   - **GI tract:** abnormal motility, malabsorption
   - **Lungs:** interstitial fibrosis and pulmonary hypertension
   - **Heart:** pericarditis, pulmonary hypertension leading to heart enlargement, arrhythmias, myocardial disease
   - **Renal involvement:** renal crisis, check BP
   - **SCL-70 antibody**

   **Treatment:**
   - NO STEROIDS
   - drugs for vascular ischaemia, immune modulation and fibrosis, ACE inhibitors in scleroderma renal crisis, Raynauds: keep warm, stop smoking, vasodilator drugs, treat infection, surgical procedures

8. **Vasculitides**
   - **Skin:** Rashes, palpable purpura, ulceration, ischaemia (distal end of fingers)
   - **Joints:** Arthritis, Arthralgia
   - **Kidneys:** Glomerulonephritis
   - **GI tract:** Ischaemia
   - **Nervous:** Neuropathies, stroke
   - **Lungs:** Pulmonary Haemorrhage
# Investigations for Connective Tissue Disease

<table>
<thead>
<tr>
<th>Disease</th>
<th>Tests and Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SLE</strong></td>
<td>ANA (95% of SLE) &lt;br&gt; Anti dsDNA (marker of disease activity) &lt;br&gt; FBC: low WCC, low platelets &lt;br&gt; Low C3, C4 &lt;br&gt; False positive test for syphilis &lt;br&gt; Renal / liver tests: monitor function &lt;br&gt; ESR high on flare ups &lt;br&gt; CRP usually normal unless there is infection &lt;br&gt; Skin biopsy shows deposition of IgG and</td>
</tr>
<tr>
<td><strong>Drug Induced lupus</strong></td>
<td>Anti histone</td>
</tr>
<tr>
<td><strong>Antiphospholipid Syndrome</strong></td>
<td>Diagnosis based on positive anti-cardiolipin antibodies or lupus anticoagulant assay</td>
</tr>
<tr>
<td><strong>Polyomyositis and Dermatomyositis</strong></td>
<td>Anti Jo-1 &lt;br&gt; CK raised &lt;br&gt; ESR raised &lt;br&gt; ANA positive</td>
</tr>
<tr>
<td><strong>Scleroderma</strong></td>
<td>Anti Scl-70 &lt;br&gt; Anti centromere (less severe form)</td>
</tr>
<tr>
<td><strong>Sjögren’s syndrome</strong></td>
<td>Anti Ro (SSA) &lt;br&gt; Anti La (SSB) &lt;br&gt; Normocytic Anaemia &lt;br&gt; ESR↑ &lt;br&gt; IgG↑ &lt;br&gt; Shirmer’s test (measuring tears) &lt;br&gt; Salivary Gland Biopsy &lt;br&gt; Rose Bengal Stain (to see damaged cornea)</td>
</tr>
<tr>
<td><strong>Temporal Arteritis</strong></td>
<td>Temporal Artery Biopsy: inflammatory cell infiltration, giant cells and granulomata visible, unless you get a ‘skip lesion’</td>
</tr>
</tbody>
</table>
Tendon insertion to bone and the presentation of common clinical conditions which arise

- Tendons attach muscle to bone.
- They are inelastic and transmit the power from muscle contraction to bone. There is not a clear boundary between tendon and bone; instead there are four transitional tissues:

  Tendon → Fibrocartilage → Mineralised fibrocartilage → Bone

- Tendon damage can occur from degeneration, leading to mesenchymal syndrome and calcification.
- There are three main pathologies associated with common tendon problems;

1. **Tendinopathy**

   **Shoulder – rotator cuff**
   
   This is a classic disorder of tendon insertion, associated with the supraspinatus attachment to the greater tuberosity. The place where tendon joins to bone, or the ‘critical zone’ has poor blood supply. Overtime the tendon can become avascular and tear. This tendinopathy produces a painful arc. Active abduction is not possible. The patient will not be able to hold their arm in space – this is a positive drop arm sign. Painful arc is relieved by anaesthetic injection – this is known as Neer’s test.

   **Jumpers’ knee**
   
   Pain over the inferior pole of the patella, from overuse.

   **Tennis/Golfer’s elbow**
   
   Enthesitis of the wrist extensor tendon into the lateral epicondyle is tennis elbow. Enthesitis of the wrist flexor to the medial epicondyle is golfer’s elbow; this is ten times less common. Pain is exacerbated on resisted wrist flexion/extension.

2. **Tenosynovitis**

   - Inflammation of the synovial lining of a tendon sheath, caused either by inflammatory arthritis or trauma (usually repetitive or unaccustomed movement).
   - Pain, swelling, tenderness and crepitus on palpation.
   - Common sites include the abductor pollicis longus and extensor pollicis brevis tendons (Dr Quervain’s tenosynovitis) as well as finger flexors. With finger flexor tenosynovitis a nodule can develop on the tendon. This ‘catches’ as it enters/leaves the flexor tunnel: this is trigger finger/thumb.

3. **Tendon rupture**

   - Resulting from chronic inflammation and degeneration or trauma. Achilles tendon rupture has been mentioned.
   - Rupture of the extensor tendons of the fingers is often seen in rheumatoid arthritis.
   - There is loss of movement (at the joint to which the tendon provides power), deformity, and sometimes swelling. With a ruptured Achilles tendon there is an audible pop/crack sound.
Diet and Health

Nutritional status assessment, constituents of a healthy diet and the consequences of an unbalanced diet, prolonged abnormal lipids and obesity

- Assessment: BMI, Cholesterol test, blood pressure, Waist circumference. WHO categorises a circumference of 39 inches for men and 35 inches for women as obese.
- Healthy diet: 30% fat (11% unsat, reduced trans fats), 55% carbs (11% sugar), 15% protein. 5 a day, 14-21 units of alcohol per week. 18g fibre/day.
- Fats: unsat reduce LDL and raise HDL, found in plants, provide essential FAs.
- Sat fats and trans fats: raise LDL and lower HDL, present in processed foods, fast foods and snacks. Linked to an increased risk of heart disease.
- Salt: major risk factor for HTension, max 6g per day intake. Increase in K in diet can reduce the risk.
- Diet and cancer: link with sat fat. Vitamins A, C and E protect against cancer. High meat and fat diet increased risk of colorectal cancer. Risk reduced with 5 a day.
- Type II diabetes: overweight, obese, central adiposity, high sat fat intake. Risk reduced with unsat fat and 5 a day.
- Obesity: energy intake is higher than expenditure. Social, and genetic aspect. Increased mortality. Risk factor for HTension, OA, gout, PCOS and cancer (increase levels of hormones that control cell growth).
- BMI: <18.5 (underweight), 18.5 - 24.9 (normal), 25 – 29.9 (overweight), 30 – 34.5 (class I obese), 35 – 39.9 (class II obese), 40 > (class III obese).
- Apo E: enzyme in lipid metabolism. Polymorphism E4>E3>E2 raised LDL.

Differences between a healthy and unhealthy lifestyle and the consequences of either changing or not changing ones lifestyle with regard to smoking and alcohol

<table>
<thead>
<tr>
<th>Healthy</th>
<th>Unhealthy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Exercise</strong></td>
<td>30 mins 5 x week of moderate exercise maintains health and musculoskeletal function. ↓ BP</td>
</tr>
<tr>
<td><strong>Diet</strong></td>
<td>Balanced diet = nutrients for growth/repair/function: 10-15% proteins 25-30% fats (pref. unsaturated) 50-60% carbohydrates Protein intake = essential amino acids</td>
</tr>
<tr>
<td><strong>BMI</strong></td>
<td>Ideal = between 18.5 and 24.9</td>
</tr>
<tr>
<td><strong>Smoking</strong></td>
<td>None</td>
</tr>
<tr>
<td><strong>Alcohol</strong></td>
<td>Moderate red wine = protection from CHD Limits per week: f = 14 units, m = 21 units</td>
</tr>
<tr>
<td><strong>Drugs</strong></td>
<td>Weed/cocaine as analgesics Others for short term euphoria</td>
</tr>
<tr>
<td><strong>Underweight</strong></td>
<td>Underweight = less than 18.4</td>
</tr>
<tr>
<td><strong>Overweight</strong></td>
<td>Overweight = between 25 and 29.9</td>
</tr>
<tr>
<td><strong>Obese</strong></td>
<td>Obese = between 30 and 39.9</td>
</tr>
<tr>
<td><strong>Very Obese</strong></td>
<td>Very Obese = over 40</td>
</tr>
<tr>
<td><strong>Smoking</strong></td>
<td>Smoking = Lung cancer, COPD, oesophageal cancer, ischaemic heart disease, peripheral vascular disease, bladder cancer, asthma, reduced # healing, osteoporosis</td>
</tr>
<tr>
<td><strong>Alcohol</strong></td>
<td>Any more than guidelines = loss of co-ordination dehydration toxicity tolerance dependence Liver cirrhosis, epilepsy, osteoporosis, pancreatic disease, bowel &amp; oesophageal cancer</td>
</tr>
<tr>
<td><strong>Drugs</strong></td>
<td>Cocaine = MI, depression, irritability, convulsions, paranoia</td>
</tr>
<tr>
<td><strong>Solvents</strong></td>
<td>Solvents = ataxia, dizziness, amnesia</td>
</tr>
<tr>
<td><strong>Amphetamines</strong></td>
<td>Amphetamines = fatigue, depression, paranoia</td>
</tr>
</tbody>
</table>
Environmental risks and hazards: relation to mortality and morbidity

Morbidity is the state of being ill or diseased. Mortality is the incidence of death in the population in a given period.

Radiation: UV and γ rays can generate free radicals, damage to cells and DNA, mutations, cancer, antioxidants reduce risk of damage caused by free radicals.

Toxins: Natural (plants, fungi, bacteria, animals), man made (factory waste, nuclear waste, pesticides, fertilizers, heavy metal bioaccumulation in fish).

Effects of toxins: liver/renal failure, inflammation of tissue, GI effects.

Hazard: poor housing, built and natural environment, RTAs, hazards at home (electricity, cooking fires)

CO poisoning: competes with O2 for binding to haem in erythrocytes, higher binding affinity, hypoxia, death.

CN poisoning: used in industry, reacts with metalloproteins, inhibits electron transport chain, no energy produced by mitochondria, cell death.

Lead and mercury poisoning: react with thiol groups in proteins.

P450 system: clears toxins and drugs in the body, adds on functional group to make toxin more soluble, toxin then excreted in liver (bile) or kidney (urine).

Importance of dietary intake in chronic ill health

- Over and under nutrition – obesity vs nutrient deficiency (eg diets rich in junk food, poor in vitamins)
- 3rd world, malnutrition; people too weak to fight disease.
- Obesity = higher disease burden in terms of QALYs (quality adjusted life years) and earlier death (2-7 years at 40 years old) = higher cost to society re: NHS treatment, benefits.
- In 2005, all chronic diseases account for 72% of the total global burden of disease in the population aged 30 years and older.
- ‘05: 35 million dead from heart disease, stroke, cancer and other chronic diseases worldwide, in many overweight (BMI over 25 kg/m2) is a factor. Obesity also associated with diabetes & arthritis such as gout, OA (metabolic implications of visceral fat & joint implications of extra body weight, plus too much purine/boozie intake).

Bleeze: ↑Risk liver, breast Cancer.

Essential: Total fat - maintain average total intake at 35% food energy (Saturated fat - reduce average total intake to 11 %), Fruit and vegetables - at least 5 portions/day, Fibre - 18 grams/day, sugar – 11 % of food energy, salt 6 g/day. Avoid trans fats and LDLs.

Hit food targets above to reduce cancer incidence by 19%, reduce CV deaths by 16-19% and reduce type 2 diabetes as well as stress on joints.

Macronutrient deficiencies: calcium & vit D – osteomalacia, osteoporosis. Beware: Too much fat loss can lead to amenorrhoea and osteoporosis/osteopenia.

Conc: diet as prevention as well as symptom management – public health agenda important as well as influences from doc, friends, tv ads, supermarket promos etc. Remember in conjunction with exercise and low boozie intake.

Lipid metabolism: factors affecting and management (pharmacologically and lifestyle)

Factors affecting lipid metabolism:

1. Diet:
   - Foods that increase LDL: saturated fats, trans fats, cholesterol (liver, offal); animal fats and dairy
   - Foods that decrease circulating fats: fiber (cereal, whole wheat)
   - Alcohol: worsens existing lipid disorder by increasing lipid deposition in liver

2. Exercise:
   - Raises HDL Lowers LDL

3. Smoking:
   - Raises fatty acids in blood stream

4. Genetics:
   - Familial hyperlipidaemia
   - ApoE4,3,2
   - South Asian communities in the UK

Management:

1. Lifestyle Changes:
   - Diet: substitute saturated fats for unsaturated (monounsaturated and polysaturated fats found in nuts, vegetable oils, soybean, sunflower, fish and corn oils), 5 fruit and veg a day decreases risk of heart disease
   - Exercise and weight loss
   - Quit smoking, decrease drinking to recommended doses (women: 2-3 u/d; men: 3-4 u/d)
2. Drugs:
   - Statins (inhibit HMG CoA reductase)
   - Niacin (B vitamin in foods and multi-vitamin supplements); lowers LDL cholesterol and raises HDL cholesterol
   - Ezetimibe (cholesterol absorption inhibitors)
   - bile-acid sequestrants

**Background to Answer:**

**Cholesterol synthesis**
- a sterol, synthesized mainly in the liver, intestine, adrenal cortex and skin
- also acquired through diet
- synthesised from acetyl coenzyme A by the enzyme HMG CoA reductase
- Insoluble in water, transported in blood by: chylomicrons, VLDLs, LDLs, HDLs
- Excreted in bile and reabsorbed in the small intestine

**Cholesterol Function**
- Controls membrane permeability
- insulates nerve fibres, and is an essential building block for hormones, such as the sex hormones and the hormones made and released by the adrenal glands
- Cholesterol also enables the body to produce bile salts and steroid hormones

**Terms**
- Chylomicrons: transport digestion products of fat to the liver
- Very low density lipoproteins (VLDLs) circulate in blood until its tryglycerides are hydrolysed to free fatty acids (FA) these become denser in cholesterol and make Low Density lipoproteins (LDLs)
- LDLs: Deliver cholesterol to the liver and to peripheral tissues depositing fat in tissue (adipose)
- High Density lipoproteins (HDLs) the good guys: take up cholesterol from peripheral cells and transport it to the liver, where it is broken down

**Cholesterol and diet**
- We need FA from diet, cannot make them otherwise
- We get FA from UNSATURATED FATS found in: nuts, vegetable oils, soybean, sunflower, fish and corn oils
- Deficiency of unsaturated fats affects negatively fetal development and can lead to: metal retardation, decreased mental capacity, dermatitis, impaired vision, decreased immune system, new studies are also linking it to depression and sleep apnea.
- TRANS fats are dangerous! As little as 100 mg per day! These are used in mass production because they are more solid at room temperature, therefore assisting with storage and “better looking” products.
- High serum cholesterol levels of LDL and VLDL are linked to heart disease
- Severe trends in obesity have tremendous economic impact in health services
- Impaired glucose tolerance is the highest predictor of diabetes II development
- New research showing that FAT cells are not inert, that they have some role in inflammation processes, that they affect circulating hormones, and all of these may be linked to Cancer as well because they affect cell growth processes.
- The safest way to lose weight is 0.5 - 1.9 kg per week

**Economics**
- Huge costs of prevention are worthwhile in the end as opposed to treatment

Comparison of treatments: Treating hyperlipidemia pharmacologically annually costs 11,000 pounds per person per year, as opposed to treating it with a nurse who assist the pt with nutritional counselling and behavioural changes which costs around 900 pounds per person per year, as opposed to some indirect public health strategies which cost even less and have an equally similar effect.
Disability

Chronic disability: impact of living with

1. Psychological (depression, anxiety, loss of identity, social isolation, loneliness, addiction)
2. Sociological (stigmatization, prejudices, loss of autonomy, deprived of social contact, loss of role in family and society, loss of educational opportunities, decreased overall quality of life)
3. Economics (loss of job and income, poverty, dependent on benefits or family, financial stress on the family)
4. Physical (other diseases develop due to decreased mobility, pain, obesity, malnutrition, addiction, diabetes, cardiovascular, respiratory, musculoskeletal, further deterioration of initial disease)
5. Behavioural (pain changes the person over time, anger, stress, denial, loss of motivation)
6. Family (dynamics of adjusting to disability in the family, the sick role of a parent, burden on the carers, sick spouse replacing children’s needs, financial stress)
7. Environment (world not adapted to the needs of people with disabilities, barriers to transportation if mobility is an issue, sidewalks (or whatever else you call this in England) without ramps, physical barriers to access leisure and education)
8. Stages of bereavement: Shock and denial, Yearning and searching, Disorganisation and despair, reorganization

- A possible sociological impact of living with chronic disability is oppression, whereby the disabled individual is prevented from fully participating in society
- Loss of transcendence (self is more than its body and much more than an illness) can occur whereby the disability is integrated into one’s identity
- Individual may adopt the sick role. The aim of the sick role is to minimise social disruption by returning the sick person to health ASAP and control the illness by controlling deviant (different from normal) illness behaviour.
- The sick role has 2 rights: sick are not obliged to perform normal social roles, sick not considered responsible for own state and 2 responsibilities: get well as soon as possible, consult and cooperate with medical experts
- Sick role is modified slightly in chronic illness: sufferers must attempt to maximise their ability to carry out social roles (because returning to health quickly is not possible) or risk losing the rights of the sick role

Interventions the multidisciplinary team may offer a patient with a disability

Primary care;
- Receptionist - first port of call
- Nurse - advice/information/monitoring/counselling
- GP - advice/monitoring/meds review
- Community Pharmacist/Community nurse
- Carers (voluntary or employed) - ADL assistance

Secondary care;
- OT - home assessment to assist ADL
- PT - realistic goal setting for early mobilisation/gait
- Speech and language therapist - assessing and treating speech problems e.g. stroke victims or head injury
- Podiatrist - aids/orthoses
- Pharmacist - regular review of meds
- Nutritionist - advice on lifestyle modification particularly diet
- Specialist physician e.g. Rheumatologist, Orthopaedic surgeon, Radiologist, Pathologist, Haematologist etc.
- Psychologist - counselling
- Nurse Practitioner
- Midwife
- Back care adviser - advice on back care/prevention on returning to work
- Occupational Health adviser
- Technicians
- ODP
- Porters/Cleaners
- Legal/Social services
Tertiary care;
- Care in the community - the NHS and Community Care Act 1990 dictates the working together of local authority and social services and the NHS - harmonising packages of care.
- Department of Health - education/information on 1°/2° prevention
- NICE - issuing guidelines

**General interventions**
- Continuous partnership with patient and family to establish patient negotiated agenda
- Realistic goal setting
- Common language to facilitate adequate communication
- Education and advice in a format in which the patient can understand
- Co-ordinating team responsible for care of patient

**Disability, handicap and impairment: definitions**
- Impairment: Any loss or abnormality of psychological, physiological and anatomical structure of function e.g. amputation, blindness, stroke
- Disability: any restriction or lack of ability (resulting from an impairment) to perform an activity in the manner or within the range considered normal for a human being e.g. unable to walk, unable to see, hemiplegia
- Handicap: a disadvantage for an individual, resulting from an impairment or disability, that limits or prevents the fulfilment of a role for that individual e.g. cant play football, cannot drive a car, cannot work
- Social model: disability is something imposed upon those with impairments by unnecessarily isolating and excluding them from full participation in society. Disability is the social situation encountered by people with impairments
- Importance of social model: uses the approach that barriers for people with impairments should be removed instead of putting an emphasis on curing the impairment/fault of the individual, the fault lies with society and not the disabled individuals
- Some impairments do not give rise to a disability but still result in a handicap e.g. restricted diet for type 1 diabetes social exclusion due to stigma and stereotypes about the disease

**Dr-Pt**

**Doctor patient relationship in primary care: importance of, factors affecting, how it can affect patient outcomes.**

*Importance of doctor-patient relationship in primary care...*
- Reflects and reinforces wider social relations and structural inequalities - especially those of gender, race and socio economic class. The relationship and values maintained form key dimension to social control.
- Traditionally, the view of the patient has been neglected.
- The quality of the doctor patient relationship impacts on the health outcomes. Note; the Patient’s Charter (1992 but updated in 1995) - this has formally given patients a number of rights and also affirmed what standards the public can expect from the NHS.

*Factors affecting relationship...*
- Mead and Bower (2002);
  b) The biopsychosocial perspective
  c) The patient as person
  d) Sharing power and responsibility
  e) The therapeutic alliance- common goals and agendas
  f) The doctor as person

- Paternalism: disease focussed, closed questions, little patient involvement
- *Argument for paternalism;* doctors have a duty to act in the patient’s best interests, even if the patient doesn’t know what her bests interests are
- *Argument against paternalism;* Patients are better placed than their doctors in deciding what is in their own interests. Whilst doctors may be able to make better ‘medical’ decisions, they are not necessarily able to make better ‘moral’ decisions.
Advantages of patient centred approach...

- Patient centred approach: ICE, cues and clues, active listening and summarising, illness focussed (impact on patient), mutual agreement on treatment, prevention and health promotion.
- Benefits of patient centred care: less Rx, more advice on prevention, better treatment of psychosocial problems, Better patient adherence to treatment, less repeat consultations, better clinical care of chronic illness, more pt satisfaction.
- Pt agenda (reason for consultation), Dr agenda (appropriate Tx), health promotion (diet and lifestyle) and management (services available)

Risk: defining, explaining to patients

- Risk factors affect likelihood of patient getting an illness e.g. diet, smoking, alcohol, sexual activity, exercise.
- Any Tx or Rx carries the risk of side effects
- Absolute risk: risk of developing a disease over a given time
- Relative risk: comparing risk of developing a disease between groups of people
- RR can be misleading if AR is very low anyway
- NNT: number of patients needing to be treated to improve the outcome of 1 patient, lower the better.
- Good practise in communicating risk: Information is not only communicated from patient to doctor. Rather, patient communicates acceptability of risk. This way treatment decisions can reflect the patients’ attitude towards risk. Comparing the risk to an everyday risk which can be easily understood by the general public can be useful e.g. risk of Creutzfeldt-Jakob disease was roughly the same as getting struck by lightning (~1 in 10 000 000). Aids such as charts and graphs can also be useful.

Immunisations: reasons why people choose for or against

- Side effects: local (rash, pain, swelling), systemic (fever, anaphylaxis)
- Serious adverse events: seizure, anaphylaxis, persistent crying
- Risks of using live vaccine: reversion to virulence, contraindicated in pregnancy and immunocompromised, risk of secondary spread
- MMR: Drop in vaccine coverage in 1990s, wakefield paper linking MMR to autism, rise in incidence of measles, refused consent by parents, role of media, reliance on herd immunity, lack of confidence in govt and drug companies, leo blair, 95% coverage needed for herd immunity currently 85%, commercial conflict of interest, public health vs right to autonomy
- Religious reasons: Christian groups opposed to HPV as it encourages sex before marriage

Reasons why people take time off work and support that is available

- Back pain, stress, depression, chronic illness, pregnancy, addiction, accident.
- DLA: under 65, physical/mental disability for last 3 months and likely to continue for at least 6 more months
- ESA: replaced incapacity benefit and income support, work capability assessment (what you can do), means tested, access to personal advisers and training courses to get back to work, £64 per week at highest rate, NI contributions paid for, 16-65, not eligible for SSP or SSP stopped (unemployed/self employed).
- SSP: contract employment, ill for at least 4 consecutive days, earning above £95 per week, must provide medical evidence to employer, £80 per week from employer for up to 28 weeks.
- SMP: must be working for employer 26 weeks before into 15th week before baby is due, earning over £95 per week, first 6 weeks at 90% of gross earnings, remaining 33 weeks at £123, can claim maternity allowance from the state of not eligible for SMP, same rates apply.
Dying and the elderly
Prescribing for the elderly: problems that may arise

- Causes of ADRs in the elderly: polypharmacy (many on 4 or more due to multiple comorbidities, drug interaction risk increases), iatrogenesis (over prescribing, using wrong drugs/doses), lack of clinical trials in this age group, drug interactions with OTC drugs.
- Risk of falls/fractures increased: antidepressants, antipsychotics, antihypertensives
- Age related alterations in pharmacokinetics of drugs:
  - Absorption (increased gastric pH resulting in decreased absorption),
  - Distribution (body water volume decreases and fat content increases with age, polar drugs have a more concentrated effect and lipid soluble drugs a less concentrated effect),
  - Metabolism (reduced hepatic enzyme activity, drugs have longer half life),
  - Excretion (renal efficiency and GFR decreases with age, greater risk of nephrotoxicity)
  - Sensitivity nervous system more sensitive to drugs, dose give usually starts at 50% adult dose
- Compliance issues: impaired memory and cognition and capacity to understand, impaired vision, difficulties swallowing, physical ability (e.g. arthritis). Polypharmacy and too many drugs difficult to know how many to take and when.

Ageing: normal and abnormal, how good health can be maintained in older people, implications of acute and chronic ill health.

- General effects: decrease in height, increase in girth, loss of muscle mass and strength
- Effects on muscle: reduction in mass mainly due to loss of type II fibres, loss of strength and power output, denervation due to loss of motor neurones.
- Effects on bone: peak BMD at 30, loss of BMD thereafter, accelerated by the menopause, decline in osteoblastic activity, increased risk of Oporosis and #.
- Effects on skin: loss of elasticity, reduced blood supply to the skin, poor thermoreg and wound healing
- Effects on GI system: reduced absorption, mucous secretion and motility, less hepatocytes in liver, constipation, increased risk of diverticulosis, gallstones and bowel cancer.
- Effects on CV system: arterio/atherosclerosis, cardiomegaly, reduced response to exercise (cannot increase cardiac output as efficiently), general rise in systolic BP.
- Effects on resp system: weakening of diaphragm and intercostals muscles, reduced ciliary function and lung elasticity, decreased alveolar surface area.
- Effects on kidneys: reduced GFR, excretion of Na+ and drugs becomes more difficult
- Good health can be maintained by diet, exercise and healthy lifestyle, abnormal aging related to genetic and environmental risk factors for pathology, chronic/acute ill health can increase morbidity and mortality
- Benefits of exercise: reduce muscle wastage, load bearing exercise increased peak BMD, lower BP, flexibility and mobility of joints, helps maintain balance (lower risk of falls).

Context of care: influence on the care of the dying patient and the support provided to their family

- Palliative care: pain and symptom control during terminal illness and final stages of life, Liverpool care pathway (evidence based best practice guidance for end of life care), aims to make the pt as comfortable as possible, alleviates stress on relatives caring for the pt, physical and psychosocial wellbeing, day care or in patient services available at a hospice
- Symptom control: D and V, pain, constipation, fatigue, depression, anxiety, weight loss, incontinence, symptoms can be hard to control by carer/family at home and can require in patient or day patient palliative care
- MDT role: dr, nurse, PT and OT, chaplain, social worker, psychologist, centred around the wishes of the pt and immediate family
- Contributors to pain: effects of illness and treatments, anger (delay in diagnosis, poor dr-pt communication, treatment failure), depression (loss of job, self esteem, role in family, helplessness, chronic pain), anxiety (fear of death, worry about family, fear of pain)
- As prognosis worsens level of treatment should be inversely proportional to level of palliative/supportive care, supportive care for bereaved should continue after death (bereavement services).
- Good palliative care: open communication, focus on QoL and symptom control, treat psychosocial issues also, involve relatives/carers in care process, patient autonomy for place of care and treatment options (even if against family wishes).
- Where do people die: mostly in hospital then home then hospice, rise in number of people choosing to die at home.
Cultural and religious practices associated with death and bereavement

- Death has become increasingly medicalised in western cultures, more deaths in hospital, better palliative care prolongs dying process, people live longer and so many are not exposed to bereavement of close family until adulthood.

- Orthodox Christians: terminally ill pt takes communion close to death, minister says prayers of reconciliation. Bereavement: lighting of candles at home and wearing black for 40 days, as soul of the deceased walks the earth and awaits judgment, prayers & memorial service with holy bread after 40 days, (funeral after 7 days in less orthodox churches) to help ascension into heaven.

- Islam: family gathers round the pt and extracts from the Koran are read, common for family to be present at time of death. Body should be buried as soon as possible, post mortem can cause probs, afterlife in paradise.

- Judaism: can pray to remove someone from suffering. Respectful to watch over the person at moment of death. Family cover mirrors (no vanity) and sit ‘shiva’ (sit receiving wishes for ‘long life’ from relatives etc on hard uncomfortable stools) for a week after person’s death. Funeral to happen asap, stone setting a year later.

- Hinduism: reincarnation, pt to leave this life in good mental state for good karma into next life so pt should be conscious at moment of death (re: less palliative sedation), all conflict and unfinished business should be in order before death.

- Buddhism: death as transition into new life therefore less grief-stricken outlook. Karma important so pt to avoid harm when dying.

- Social death: when family, friends and health professionals have difficulty talking to dying pt, withdraw from pt or pt withdraws from others in preparation of death, loneliness and isolation, made worse by moving pt to side room or closing bed screen, reassurance and support to pt important in final stages.

- Good death: self awareness of death and what will happen (sense of control), dignity and privacy, symptom and pain control, choice to decide where to die, spiritual and emotional support, dying in presence of loved ones, have time to say goodbye and issue advanced directives.

Death: defining, confirming, certifying and reporting to coroner

1. Definition:
   - absence of vital functions, such as cessation of heart beat
   - brain death: permanent functional death of centres in brain stem

   Law Case
   Regina v Malcherek : The "victim" was on a life support machine which was switched off by the doctors; courts said that according to medical evidence there is only one test of death and that is the irreversible death of the brain stem which controls the basic function of the body such as breathing; In the case of brain stem death, the body has died, even though, by medical means the lungs are caused to operate and some blood is in circulation.

2. Confirming:
   - Body temperature
   - Shine light into eyes (pupil fixed and dilated?)
   - Carotid pulse for 1 minute
   - Examine Chest 1 minute: move?, heart beat?, breath sounds?
   - Confirmed death: write in notes, date, time, sign, print, bleep number

   Brain Stem Testing:
   2 senior docs, registered 5 years, 2 separate occasions, not members of transplant team, minimum 6 hrs since coma onset, If post arrest, after cerebral air or fat embolism, or ill-defined period of anoxia, 24 hrs after adequate circulation re-established. Pre-conditions: there must be cause of apnoeic coma (e.g. head injury, hemorrhage), exclude effects of: hypothermia, meds, metabolic and endocrine abnormalities (e.g. electrolytes and glucose)

3. Certifying:
   - Write a legal death certificate
   - Talk to relatives: cultural/religious issues
   - Post-mortem? Seek consent from relatives
4. Reporting to Coroner:
If unsure discuss with consultant and pathologist
Refer to Coroner without writing the Death Certificate if:
   a. Death due to unnatural cause (incl. accident)
   b. Cause cannot identified
   c. Death may have been due to drugs, medicine, abortion or poison;
   d. during an operation or prior to recovery from the anaesthetic or due to an incident during
      anaesthesia or surgery;
   e. Related to the deceased’s employment or previous employments.

Death: epidemiology of the causes of and how this knowledge can be used to prevent premature death
- Mortality data can be used to determine the extent of ill health within a population.
- Advantages: death registration is a legal requirement (completeness of data), indisputable outcome measure,
  med cert of cause of death show what person died of, data available from Office of National Statistics (ONS)
- Disadvantages: not useful for chronic and non fatal illnesses, some details on death cert may be inaccurate,
  conditions inappropriately assigned to section II
- Crude death rate: deaths per 1000 per yr, simplest measure, can be used to identify trends over time, not useful
  to compare populations with varying age and sex structure
- Specific mortality rates: death rate within a sub group e.g. age, sex / population of sub group x 1000, good for
  comparison between pops
- Standardisation rates: same as specific mortality rates but figures are adjusted for confounding factors, complex
  calculations and requires a standard population
- Avoidable deaths: deaths from causes that could have been avoided with preventative measures or better
  clinical management, can point out areas that require further Ix, other factors such as disease severity may
  account for differences between pops.
- Survival rates: commonly used as a prognostic measure for those diagnosed with cancer
- Relative survival: compares survival rate in a population of cancer sufferers with that of the general population,
  takes into account mortality for reasons other than cancer
- Life expectancy: can be used to measure improvements in sanitation, healthcare and QoL
- Prevention: primary (disease incidence), secondary (disease prevalence), tertiary (minimise effects of disease
  or impairment), can implement prevention services once mortality data recognises a trend within a population.

Falls
Why the elderly are more at risk of falling than younger people
Falls: multifactorial. 1/3 >65yrs in community fall/yr, ½ >80yrs in community fall/yr

Systems affected by normal ageing:
- Vision: Reduced: acuity, contrast, dark sensitivity, depth perception
- Auditory: hearing loss
- Sensory: Reduced proprioception and touch sensitivity in the feet
- Central processing: Fewer neurons, slower reaction times, impaired integration of sensory information
- Muscles: Reduced: mass, strength and slower contraction.

Pathological changes due to diseases of old age:
- Parkinson’s: tremor/dizziness/instability
- Cardiovascular: Hypertension/hypo: risk of MI/stroke/fainting/dizziness
- Psychiatric: dementia – forgetting to use stick/unfamiliar environments
- M/S: OA/OP/RA – reduced range of motion, fragile bones/ligaments/pain
- Diabetes: peripheral neuropathy & foot ulcers
- Nutrition: lacking macronutrients, dehydration: weak/dizzy
Sum: more risk of falls: Increased sway, flexed posture, slower gait, shorter steps; less able to deal with/react to environmental obstacles

Infections
- UTIs - E coli etc (10-20 % over 80s), URTIs – Streptococcus pneumonia, influenza etc (3.4 % over 70s).
  - UTI risk in elderly: incomplete emptying of bladder (wom), prostate disease (men), diabetes neuropathy, infected catheter.

ADRs/polypharmacy
- ADRs in elderlies: reduced absorption, altered vol of distributions, reduced renal function, increased sensitivity, accessing the container, altered homeostasis
- Polypharmacy
  - >4 meds = higher fall risk
  - Sedatives/antipsychotics/hypnotics (impair insight, balance, coordination)
  - Diuretics and laxatives: dehydration & urgency
  - Drugs causing postural hypotension (decreased baroreceptors in old age)

MDT in patients with a history of falls
1. GPs
   - Identify patients with a high risk of falling based on their medical history (e.g. osteoporosis, epilepsy, stroke) or from a previous history of falls
   - Refer to falls clinic, social services
   - Mental Status Assessment, balance, gait, underlying physical issues (e.g. infections, dehydration, hypertension, diabetes, hypoglaecemia)
   - Find cause of fall and treat it—medications, fits, blood pressure, stroke, TIA, environmental hazards (i.e. pets, rugs), reduced muscle strength, pain, low mood, incontinence and frequency
   - Prophylaxis treatment (eg Bisphosphonates, decrease polypharmacy)
2. Pharmacists
   - Medication review and assess side effects and interactions (orthostatic hypotension, dehydration, confusion, fits, hypoglaecemia, electrolyte imbalance)
   - Decrease medications if possible
3. Surgery Nurse
   - Assist with finding cause of falls—medications, fits, blood pressure, stroke, TIA, physical hazards (i.e. pets, rugs)
   - Provide information and educate on risk of falls and how to prevent it
4. Occupational Therapist
   - Home assessment specifically for personal (balance, gait, vision, footwear), indoor (light, temperature, clutter, floor, stairs) and outdoor factors (access, paths, steps, transport)
   - Rehabilitate and long term support (provide equipment and home improvements)
   - Footwear assessment
   - Assist with regaining confidence and decreasing fear
5. Physiotherapist
   - Rehabilitation, assess gait, balance, muscle and nervous functioning, need for walking aids, adaptations, and home care
   - Tailored exercises to increase muscle performance
   - Assist with prevention of further falls and more serious injuries
   - Assist with regaining confidence and decreasing fear
6. Social Worker
   - Funding for social care support and home improvements, walking aids, equipment for safety
7. Public Health professionals (Doctors, Nurses, Social Workers, Researchers)
   - Health Promotion strategies (increase physical activity, improve diet and nutrition, exercise services, immunisations)
8. Carers (paid/family members)
- Washed and cleared floor and surfaces, cabinet drawers closed, notice hazards, notice patient’s deterioration status mentally and physically

9. **Orthopaedics**
   - Treat underlying musculoskeletal problems (fractures, dislocations, pain)

10. **Medicine for the Elderly Physician and Nurse**
    - Treat underlying comorbidities (osteoporosis, seizures, hypertension, diabetes, TIA)
    - Assist with regaining confidence and decreasing fear

11. **A & E Staff**
    - During the acute phase, classify, treat and provide analgesia
    - Refer to appropriate services (MFE, Orthopaedics)

12. **Pain Team**
    - Manage underlying pain pre/post fall

**Advice given to a carer of a patient with repeated falls, including prevention and what to do in the event of a fall**

*Advice to carer*

- Measures they can take to prevent further falls
- Preventable nature of some falls
- Physical and psychological benefits of modifying falls risk
- Where to seek further advice and assistance
- How to cope/access financial and emotional help

*Falls prevention*

The focus on prevention of falls is on identifying a verifiable risk and managing that risk - hence multifactorial risk assessment

**Home assessment (NICE 2004)**

- What can be done to make living in the property easier?
  - Alternative ways of carrying out basic daily tasks
  - Equipment that can assist daily living
  - Identifying what people can help e.g. relatives, friends neighbours etc.
  - What major and minor alterations can be made to the home to assist daily living
- Discussion with the patient on how best to meet their needs
- How any potential work on the property will be funded
- How the process works following the assessment

- **Personal considerations**
  - Balance and gait
  - Appropriate footwear
  - Vision and hearing
  - Pets

- **Indoor environment**
  - Heating and lighting
  - Cluttering furniture
  - Appropriate flooring
  - Stairs
  - Hand rails and door handles

- **Outdoor environment**
  - Access: home, garden, garage etc
  - Suitable and well lit paths
  - Steps/ramps
  - Transport

**Home assessment in a patient with falls**

- **Aim**: identify potential environmental hazards (hazards (rugs, stairs, pets, lighting, flooring, hand rails and door handles as well as footwear) in the home that may increase the risk of a fall
- **Assess intrinsic, extrinsic and behavioural risk factors**
• Assess patient for functional ability (e.g. climbing stairs)
• Combine observations – provide aids and techniques for patient to complete ADLs safely, and provide adaptations to the home to increase safety (e.g. stairlift).
• Identify people that can help e.g. family, friends/neighbours
• Help make action plans
• Check if patient eligible for grants if they need further care or expensive adaptations
• Arrange for MDT to provide package of care. E.g. physio: exercise programmes to improve strength and balance, get GP/pharmacist to review medications if problems are identified
• Refer to falls clinic if deemed necessary
• Discuss with patient changes they are willing to make, and potential barriers to compliance (e.g. fear of falling). Discuss ways of increasing motivation, and explain benefits of interventions proposed. Offer further advice and assistance and how to cope with a fall. Advice on lifestyle changes.

Fractures
Fracture healing: cellular process
• Fracture healing is a cascade process:
  • Requires immobildation of approximated ends.
  • All healing starts with an acute inflammatory response.
  • Healing by regeneration restores a tissue to original form, healing by repair results in a scar.

1. There is a fracture haematoma
   • Into this there is an in-growth of blood vessels stimulated by cytokines released by macrophages which also phagocytising the debris
   • Clot is replaced with precursor cells and fibroblasts which are also stimulated by the cytokines
   • This tissue is called granulation tissue and leads to callus

2. New woven bone forms
   • From the cells derived from periostium
   • Also formed to a lesser degree in the medullary cavity
   • As this process progresses, # becomes less and less mobile
   • Need to additional support reduces

3. Bone union begins
   • Collagen is laid down, and bone union (endosteal bone union) can proceed. Collagen matrix undergoes mineralisation forming callus around the 2 bone ends.
   • If bone ends are in close contact, osteoclasts tunnel across # line.
   • If displaced, fibrous tissue would have formed, this is removed by in-growth of callus

4. Remodelling takes place
   • Haversion systems are created; osteoclasts remove any bridging callus that remains.
   • Thus this is all healing by regeneration

5. Bone remodelling
   • Osteoclasts carve out tunnels in bone tissue, and then osteoblasts build it up again. In different parts of the skeleton, a full cycle of remodelling may take place in as little as 2-3 months or last much longer. Remodelling normally serves to:
     1. Renewing bone tissue before deterioration sets in and
     2. Redistribution of bone matrix along lines of mechanical stress.

6. Bone resorption
   • An osteoclast attaches to the bone surface and forms a seal, then releases protein-digesting enzymes (digest collagen + other organic substances) + several acids into the seal (dissolve bone minerals). Several osteoclasts carve out a small tunnel in the old bone. Degraded proteins and minerals (calcium + phosphorus, enter an osteoclast by endocytosis, cross the cell, and undergo exocytosis on opposite side. In the interstitial fluid, products of bone resorption diffuse into nearby blood capillaries. Once small area of bone has been resorbed, osteoclasts depart and osteoblasts move in.
Principles behind general management of a fracture

- History, examination, investigation, analgesia & fluid balance.
- Aims of # management: reduce, hold, rehabilitate.
- Reduce: bones realigned – open (surgical incision in skin) or closed (manipulation)
  - Non operative if: low energy undisplaced #, cancellous bone #, phalangeal / metacarpal / metatarsal, if no reduction needed (ie clavicle), some kid #s. Sometimes bedrest & analgesia suffices.
- Surgical: Int & ext fixation: bones anchored— Percutaneous wiring: wires passed across site, internal fixation: plate and screws, intramedullary nail in medullary cavity, external uses pins or wires inserted into the bone are held by a frame externally.
  - Int. Fixation: if displaced intra articular / periarticular (wiring / plate), lower limb long bone, # with vascular/nerve injury. Intramed nail for lower limb long bone: minimally invasive, restores full length n alignment n rotation, v strong. Plate for meta / diaphyseal upper limb: precision, less risk 2o OA, but invasive, higher com placation rate.
  - Ext. Fixation: if closed # but soft tissue trauma, some open #, juxta-articular too tricky to plate, temporary stabilisation in multiple trauma, limb lengthening, deformity correction. Less invasive, more versatile, but infection prone & poss malunion.
- Conservative vs surgery: non invasive v invasive, cheap v expensive, not always precise v precise, possible instability v stability, poss malunion v union, ongoing outpatient monitoring v quicker.
- Hold = stabilise: internal fixation / external fixation / cast / splint / sling / traction to ensure union – until bone is healed.
- MDT for rehabilitation #: Physiotherapy, occupational therapy. Mobilise as soon as # is stable, ie, even in cast/sling, weight bear if stable, move whatever joints possible, try to maintain full ROM.

Describe the different types of fractures

1. **Open or closed** (skin intact/not, open fracture - > infection)
2. **Diaphysis position**: (Simple/Wedge/Complex)
3. **Proximal and Distal Segments**: (A. Extra-Articular/ B. Partial Articular/ C. Intra-articular)
   - Generality and reliability of systems ends here (intraobserver and interobserver errors begin)
4. **Spiral** (rotational force)
5. **Oblique**
6. **Transverse** (force from the side)
7. **Comminuted #** (>2 fragments)
8. **Segmental #** (diaphysis at 2 levels with floating segment)
9. **Pathological #** in diseased bone (e.g. due to metastatic disease, osteoporosis, Paget’s, Osteomalacia, Rheumatoid Arthritis, Infection)
10. **Hairline/ avulsion #/ fatigue #**
11. **Displaced/Undisplaced**
12. **Angulation in degrees** (distal end of the distal fragment, medially/laterally, anteriorly/posteriorly)
13. **Soft tissue involvement** (nerves, arteries, vessels)

AO System of Classification:


Always note:
- Skin condition (open/closed, blisters and abrasions)
- Peripheral nerve function (weakness or numbness)
- Vascular status (peripheral pulses and perfusion by capillary refill)

Why classify fractures?
- Identify nature of injury and Energy involved
- Universal communication and research
- Planning treatment
- Relate to prognosis
- Anticipate complications (infections for open #, non-union, avascular necrosis, nerve/arterial/muscle damage)

**Radiology: principles behind diagnosing fractures**

- X rays should be taken in 2 planes at 90 degrees to each other (AP and lateral) to provide accurate information on suspected fracture bone. Failure to obtain views in 2 planes may result in missing or underestimating the extent of injuries and may significantly affect pt outcome. Additional views are occasionally required in some areas e.g. shoulder, hip, wrist and foot due to complexity of anatomy.
- X rays of joint are needed when suspected associated joint injury.
- Special views are taken for certain fracture e.g. scaphoid views.
- Ring fractures - see one fracture and look for another.
- A rigid ring must be broken in at least 2 places
- X ray the joint above and below when paired bones are injured

**Complications of fractures**

**Immediate**

- **Local**
  - Open fracture: skin tears
  - Nerve palsy: fracture fragments may press on nerves or blood vessels (resulting in ischemia). Nerve or vessels are torn completely very occasionally.
- **General**
  - Haemorrhage (blood loss): especially from femoral, pelvis, open or multiple fractures. May result in hypovolaemic shock.

**Early**

- **Local**
  - Compartment syndrome: excessive pressure developing in closed fascial muscle compartments which can cause the blood supply to be impaired. Occurs at level of small vessels so peripheral pulses are usually still present. Patient will complain of excessive pain and pain on movement of the digits. Parasthesia develops early as a result of ischaemia to the nerves. Treatment: surgical fasciotomy (release of compartments)
  - Infection: can occur early or late following operative stabilization or open fracture.
  - Complex regional pain syndrome: idiopathic. Usually upper limb affected: patient has red, swollen, shiny fingers with excessive joint stiffness.
- **General**
  - Deep vein thrombosis: can occur after any lower limb injury. Mechanical (e.g. stockings) or chemical agents (e.g. heparin) are used for prevention.
  - Fat embolus: common after long bone fractures. Fat enters the circulation and embolizes to the lungs. Medullary canal of long bones contains fat. Early stabilisation of fractures reduces risk.

**Late**

- Delayed union/non-union: fracture is slow to unite or fails to do so. More common in high energy fractures or those complicated by compartment syndrome. Treatment is further surgery to encourage the bone to heal.
- Malunion: fracture heals in abnormal position (usually due to inadequate stabilisation of the fracture. Reduces movement in associated joint, predisposes to arthritis.
- Osteoarthritis: more common after interarticular fractures.
- Growth disturbance: fracture occurs through growth plate can cause growth arrest
Femoral fractures: classification and basic surgical management of

- **Hx:** medical, social, mobility, mini mental test score
- **Examination:** leg externally rotated and shortened
- **Ix:** X-ray pelvis AP and lateral, FBC, U&E, blood culture, chest X-ray and ECG
- **Initial Tx:** analgesia, IV fluids, catheter, prep for surgery
- **Intracapsular undisplaced #: Retinacular vessels intact, cannulated screw fixation**
- **Intracapsular displaced #:**
  - under 55 reduce and screw (risk of avascular necrosis and non union, can revert to THR),
  - older and fit/active use THR (longer operation, more durable, better function and less pain)
  - if older and unfit/immobile use hemiarthroplasty (cemented takes longer but better fixation, uncemented shorter op so less risk but may loosen with time
- **Extracapsular #:** simple (dynamic hip screw), comminuted (intramedullary nail and screws)
- **Local complications (THR/hemi):** Early (infection, dislocation), late (loosening, dislocation, acetabular erosion)
- **Local complications (DHS/IMN):** Early (infection, wound), late (fixation failure, non union)
- **General complications (THR/hemi/DHS/IMN):** Early (MI, stroke, resp failure), late (DVT/PE pressure sores)
- **Post op management:** analgesia, fluids, early mobilization, heparin, antibiotics, OP management, rehab (OT, PT), falls clinic referral

Why fractures in some patients heal more slowly

- Bone type: cancellous bone heals faster than cortical
- # type: transverse takes longer than spiral
- Blood supply: poor circulation delays healing
- Diet: deficiency in Ca and Vit D will delay healing
- Age: children heal twice as fast as adults, once growth plates fuse healing rate is constant at all ages
- Smoking: adversely affects rate and quality of # union
- Causes of non union: poor fixation (disrupts callus formation), insertion of soft tissue between # fragments, severe soft tissue damage, infection, abnormal bone

Gait/limp

Gait cycle: description and control of

- **Toe off:** dorsiflexors contract to create energy for push off
- **Mid swing:** momentum of forward propulsion helps drive leg forward through the air, ankle extensors contract (tibialis anterior extensor hallucis/digitorum longus fibularis tertius), hip flexion (ilio-psoas, rectus femoris and sartorius), most activity is passive except for ankle extensors
- **Heel strike:** hip flexed, knee slightly flexed and ankle extended, controlled by glut maximus and hamstrings, quads limit further knee flexion,
- **Mid stance:** weight transfer to calcaneus and distal ends of metatarsals I and V (medial and transverse arches diffuse load), glut medius and minimus of standing hip contract to limit pelvic tilt and shift COG to stance leg, hip and knee joints extend, ankle flexion initiated by dorsiflexors (gastro, soleus, tib posterior, flexor hallucis/digitorum longus, fibularis longus and brevis)
- **Toe off:** start of new gait cycle
- 6 major determinants of gait: pelvic rotation, pelvic tilt, lateral motion of the pelvis, knee motion in stance phase, knee motion in swing phase, foot and ankle motion
- 4 things must take place to allow efficient gait cycle
  1) each leg must be able to support the body weight without collapsing
  2) Balance must be maintained during single leg stance
    1) The swinging leg must be able to advance to a position where it can take over the supporting role.
    4) Sufficient power must be provided to make the necessary limb movements and to advance the trunk.
Musculoskeletal causes of limp: differential diagnosis

DD Child

- (0-3 years), Infection (would be clinically unwell) (septic arthritis/osteomyelitis), juvenile idiopathic arthritis (JCA); Trauma, accidental, non-accidental injury, DDH
- (3-11 years) Perthes disease (boys>girls), , irritable hip, neuromuscular, slipped up per femoral epiphysis (SUF E), non-accidental injury
- (12-16 years) Slipped upper-femoral epiphysis, infection, non-accidental injury, Osgood schlatters

Fever – yes: viral/JCA is poss; no: trauma, IH, SUFE
Stiff morning – yes: JCA; no: trauma, IH, Viral, SUFE
Many joints – yes: JCA, viral; no: trauma, IH, SUFE
Sudden – yes: trauma, IH, viral, SUFE; no: JCA

DD Adult

- Trauma, Osteoarthritis (knee,hip), Vestibular ataxia (acute labyrinthitis/meniere’s disease, intermittent claudication(IC). Occasionally – foot drop (nerve), MS, nerve root, clauda equine, myasthenia gravis. Rare, tabs dorsalsis (syphilis), Dystrophia myotonica, motor neurone disease, cerebellar ataxia, hysteria

- Sudden – trauma,poss.OA,vestibular; insidious – parkinsons
- Painful unilateral limp – yes: trauma, OA, IC; no: vestibular, parkinsons
- Worse with exercise – yes: parkinsons, OA; no: trauma, vestibular, IC
- Shuffling gait – yes:parkinsons, poss. OA/otherjoint issue; no:trauma, vestibular, IC

Reasons why a child may limp with reference to age

- Infection—(clinically unwell)
- septic arthritis
- osteomyelitis (typically in child under 10)
- Trauma (history of trauma / or unexplained bruising or injuries in NAI)
- fracture: accidental or NAI (especially in children under 10)
- slipped upper femoral epiphysis (in children over 10)
- soft tissue
- Developmental (No pain and symptoms since started to walk)
- hip dysplasia in children under the age of 4
- Osteochondroses (gradual onset of hip pain in well child)
- Legg-Calvé-Perthes disease (or Perthes disease) – AVN of capital femoral epiphysis (usually in children below ten)
- Synovitis (acute onset and difficulty weight bearing)
- Irritable hip
- JIA
- Neoplasia - primary bone tumour (night pain)

<table>
<thead>
<tr>
<th>Age</th>
<th>Differential Diagnosis</th>
</tr>
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<tbody>
<tr>
<td>All Ages</td>
<td>Juvenile Idiopathic Arthritis (JIA), infection –septic arthritis or osteomyelitis, malignancy (leukaemia), trauma/# &amp; non-accidental injury (NAI)</td>
</tr>
<tr>
<td>Infant (0-3 years)</td>
<td>Developmental dysplasia of hip (DDH), neuromuscular (muscular dystrophy, cerebral palsy)</td>
</tr>
<tr>
<td>Childhood (3-11 years)</td>
<td>Perthes Disease (mostly b/w 4-8 yrs), transient synovitis (irritable hip), limb length discrepancy</td>
</tr>
<tr>
<td>Adolescence (12-16 years)</td>
<td>Slipped Upper Femoral Epiphysis (SUF E), Osgood-Schlatter's condition, tarsal coalition, ACL tear &amp; meniscus tear</td>
</tr>
</tbody>
</table>
**Immunology**

**Host defence mechanism against pathogens: innate and acquired**

- **Innate IS:** Skin, lysozyme in eyes, respiratory (mucous, cilia, cough reflex, alveolar mphages), GI (commensal bacteria, low stomach pH), vagina (low pH), flushing of urinary tract

- **Components of innate IS:** Immediate and non specific response to pathogenic threat, no memory, interacts with and potentiates adaptive IS, inflammation and nphil recruitment (chemotaxis and diapedesis), phagocytosis (phagolysosome and oxidative burst).

- **Recognition molecules:** toll like receptors (mphages), LPS receptors, mannan binding receptors, CRP, INF (respond to presence of viral DNA, inhibit viral repn, activate NK cells)

- **Complement:** Positive feedback mechanism, C3 most important (deficiency results in overwhelming bacterial infection), lysis of bacteria and infected cells (MAC with c3b c5b c6-9), recruits inflammatory cells (c3a and C5a), opsonisation for Pytosis (C3b), activates ephils

- **Adaptive IS:** Recent evolution, adaptive, memory (faster, stronger more specific response on subsequent exposure), delayed onset

- **Components of adaptive IS:** B cells (Abs, memory cells) T cells (Th cells secrete ILs and work with B cells and Mphages, Tc cells lyse infected cells), mphages, Abs, TCRs (recognise Ags presented in MHC class I (Tc) and II (Th)), HLAs, IL signalling cytokines

- **Abs:** 4 chains (2x heavy 2x light), variable and constant regions, 5 classes of Ig based on composition of heavy chain IgM (intravascular, 1st produced in infection, pentamer, activator of compliment), IgG (main Ab, major component of secondary response, Fc receptors on Mphage), IgA (dimer expressed on mucosal surfaces), IgD (on surface of B cells), IgE (bind parasitic Ag, mediate anaphylaxis, present on surface of mast cells and basophils, allergy)

- **Ab function:** activate compliment, opsonisation for phagocytosis, neutralise bacterial toxins, activate mast cells

**Acute viral infection: symptoms of and how to differentiate from more serious illnesses e.g. appendicitis**

- **Fever** – caused by prostaglandin E2 (being triggered by a pyrogen) acting on the hypothalamus that changes the temperature set point. Pyrogens can be endogenous - cytokines or exogenous – e.g. bacteria cause release of endogenous factors.

- **Autonomic nervous system (controlled by the hypothalamus)** – causes increased muscle tone, shivering, vasconstriction (feels cold), increased heart rate and increased BP.

- **Interferon release (non specific) – fever, malaise, muscle aches, fatigue**

- **Increased insensible losses from skin (sweating) and lungs (breathing fast)** – leading to decreased extracellular osmolality leading to increased Anti Diuretic Hormone (ADH) from the pituitary leading to water conservation in the kidney – decreased urinary volumes

- **Diarrhoea and vomiting leads to sodium loss** – this is made worse by replacing the lost isotonic fluid by water. Hyponatraemia leads to decreased extracellular fluid volume – this leads to decreased BP, increased pulse, dry mucus membranes, sunken eyeballs, decreased skin turgor, decreased conciousness.

- **In serious illnesses – the acute phase protein response causes the metabolic activity to increase to direct resources to the site of action, the body is in a state of catabolism, if the body is unable to eat then the body is in a state of famine leading to immnosuppression, decreased wound healing, delayed tissue repair, muscle weakness.**

**Why common pathogenic organisms are infective**

- **Bacteria, parasites, viruses, prions, fungi** : use of host resources (nutrients, shelter) to thrive.

- **Break through physical barriers and enter tissues and cells, resist/hide from IS, proliferate intra/extracellularly, damage host tissue.**

- **Pathogenicity:** ability of a microbe to causedisease (species comparison)

- **Virulence:** degree of pathogenicity of the microbe (strain comparison)

- **Contraction mechanisms:** air, food, water, contact (human, fomite), STI, blood borne, vector, iatrogenic, faecal -oral,

**Common pathogenic organisms: characteristics and how they can be identified**

- **Bacteria:** gram positive (peptidoglycal outer layer), gram negative (2 lipid bilayers with peptidoglycan inbetween, LPS on outer membrane), unicellular, cell wall, pili and flagellum, circular DNA in cytoplasm, smaller ribosomes than eukaryotes (50S and 30S), variety of shapes (rod, coccus, spiral, in pairs, chains or clusters)
- Viruses: protein capsule, nucleic acid inside capsule (DNA or RNA), no organelles, no means of self replication, small genome, cannot survive or replicate without host.
- Parasites: nucleus, multicellular, no cell wall, specialist organelles (golgi, ER, mitochondria).
- Identification: clinical Hx, examination, swab and culture (bacteria), microscopy (light and EM), PCR of viral genome, host tissue culture, ELISA, southern blot, blood culture, urinalysis, faecal culture, sputum, CSF, full blood count.
- Specific tests:
  - Bloods: Giesmas stain for malaria parasite; culture for any bacteria
  - Urine: Microscopy and culture
  - Faeces: clostridium difficile toxin
  - CSF: Microscopy and culture for meningitis; auramine stain/TB culture for TB.
  - Aspiration : culture

**Immunological response: cells and organs involved, structure of antibodies and their mode of action**

**Immune Response**
- Phagocytes → engulf pathogen → activation of immune response
- Phagocytes use fragments from pathogen to produce antigen → antigen presenting cell
- Antigen presenting cells interact with T-helper cells that have matching antigens
- Interleukin-1 is released by phagocytes → stimulates T-helper cells to secrete interleukin-2 (IL-2)
- IL-2 → proliferation of T-cells and B-cells
- Infected body cells produce anti-bodies → bind to antigens in cytotoxic T-cells
- T-cells → chemicals that kill infected cells
- T-helper cells activate B-cells → proliferate into plasma cells and memory cells
- Plasma cells → anti-bodies that blocks antigens in pathogen
- Memory cells → secondary immunity → after exposure to disease or vaccination

**Structure of antibodies**
- Antibodies are immunoglobulins
- Five major classes; IgM, IgG, Iga, IgD, IgE
- Consists of four polypeptides
  - 2 heavy chains
  - 2 light chains
  - Join to form a "Y" shaped molecule
- Variable region
  - amino acid sequence in tips of "Y"
  - varies greatly among different antibodies
  - 110-130 amino acids → specificity for binding antigen
  - includes the ends of the light and heavy chains
- Treating antibody with protease can → fragment antigen binding (Fab)
- Constant region determines mechanism used to destroy antigen

**Role of immune system in inflammation**
- **Inflammation** serves to destroy, dilute, and wall-off injurious agents.
- An inflammatory response can be **acute** or **chronic**.
- **Acute** inflammation;
  - Typically a short duration (minutes, hours or a few days)
  - Triggered by a range of insults (chemical, thermal damage or infection).
  - Infection is sensed by macrophages which release chemokines and cytokines attracting neutrophils to the site of infection.
  - In other instances, inflammation is initiated by resident mast cells, which attract eosinophils.
  - Once inflammation is initiated several changes occur in vascular endothelium to allow attachment and the 'leaking' of leucocytes – primarily neutrophils but also monocytes and lymphocytes.
This results in **vascular changes**, (vasodilatation, slowing of circulation and entry of inflammatory cells) **leukocyte extravasation** and chemical mediators of inflammation (histamine, cytokines, coagulation system etc).

There are several possible outcomes resulting from acute inflammation. These include:

- Regrowth and resolution
- Healing by collagenous scar formation
- Abscess formation
- Chronic inflammation

**Chronic inflammation arises;**

- When the causative agent **cannot be eliminated** and antigenic persistence occurs.
- Key cells of chronic inflammation are **macrophages, lymphocytes** and **plasma cells**.
- This is a contrast to acute inflammation as this is characterised by neutrophils.
- Macrophage secretory products mediate characteristic features of chronic inflammation:
  - Tissue damage via proteases and oxygen radicals
  - Revascularisation by angiogenic factors
  - Fibroblast migration
  - Collagen synthesis
  - Remodelling via collagens
  - Simulation of T-cell activity by secretion of IL-12.

Lymphocytes and plasma cells also present at the site of inflammation. In the case of chronic infection both macrophages and T-cells are required to control infection.

**Autoimmunity: consequences and recognition of self**

- **Tolerance:** immune system unresponsive to self antigens. Doesn’t work in autoimmunity.
- **T & B cells (lymphocytes)** randomly recombine genes for receptors: risk of reactivity to self-antigen.
  - **Autoimmunity:** these cells aren’t eliminated hence host not tolerant to itself.

**Central tolerance:**

- **Positive selection:** deletes T cells unable to recognise self MHC molecules so T cells can recognise MHC on antigen presenting cells.
- **Negative selection** – early clonal deletion of the most self reactive T cells in thymus (high affinity to self-MHC) & B cells in bone marrow that recognise self-antigen (lesser self reactive ones get through), generates self tolerance.
- **Receptor editing →** autoreactive B cells given 2nd chance to rearrange their gene, if they are still binding to self antigen they are deleted.

**Peripheral tolerance:**

- **Deletion:** Mature self-reactive lymphocytes that got thru may be inactivated or deleted by encounters with self-antigens, preventing autoimmunity especially if low MHC expression (MHC enables T cell recognition of antigens)
- **Immunological ignorance:** mature autoantigenic T cells in periphery never encountering appropriate antigen due to physical barriers eg testis.
- **Clonal anergy (functional inactivity):** Autoreactive T cells exposed to antigen peptides with lack of co-stimulatory molecules (e.g. B7-1 or B7-2) become anergic (non-responsive).
- **Immunological Suppression:** T regulatory (T<sub>reg</sub> cells) → produce TGF-β and IL-10 to prevent or suppress the action of other, potentially harmful, self-reactive lymphocytes
- **Autoimmunity not always associated with harmful effects and diseases.**

**Suspected cause of autoimmune diseases: multifactorial: genes, environment, immune regulation:**

- Some inherited HLA alleles don’t bind to self antigen well, so reactive T cells not deleted in thymus (eg HLA DR4 for RA).
- Some pathogens do molecular mimicry – their antigens resemble host cell components so self-immunity is triggered.
Some infections activate T & B cells non specifically – proliferation of clones mediating autoimmunity (polyclonal activation).

**Mechanisms & consequences:**
- Direct antibody mediated pathology: antibodies specific for self-antigen bind to tissues/cells: opsonisation/complement activation eg: Myasthenia Gravis.
- T cell mediated damage: recognition of autoantigen by T helper / T cytotoxic cells mediate systemic or organ specific conditions via cytokine destruction, activation of macrophages (RA, type 1 diabetes).
- Immune-complex mediated pathology: antigen/antibody complexes not cleared from circulation trigger inflammation eg of blood vessels (SLE).

**Acute and chronic inflammation: clinical and histological features**

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<thead>
<tr>
<th></th>
<th>Clinical</th>
<th>Histological</th>
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<tbody>
<tr>
<td><strong>Acute</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Redness</td>
<td>- due to dilation of small blood vessels within the damaged area</td>
<td>1. Increased vessel diameter + Leucocytes migration</td>
</tr>
<tr>
<td></td>
<td>e.g. cellulitis due to bacterial infection</td>
<td>2. Increased vascular permeability (oedema)</td>
</tr>
<tr>
<td>2. Heat</td>
<td>- due to hyperaemia through the region resulting in vascular dilation and delivery of warm blood to the area</td>
<td>3. Exudate d/t inflammatory leucocytes crossing endothelial cells into the adjacent interstitial tissue: Margination- leucocytes move into outer margin of blood flow Pavementing- leucocytes line the endothelial surface Rolling- leucocytes roll/tumble along endothelial surface</td>
</tr>
<tr>
<td>3. Swelling</td>
<td>- oedema results from accumulation of fluid in extravascular space as part of fluid exudate</td>
<td></td>
</tr>
<tr>
<td>4. Pain</td>
<td>- tissues being stretched + chemical mediators (bradykinin, prostaglandins, serotonin)</td>
<td></td>
</tr>
<tr>
<td>5. Loss of function</td>
<td>movement of an inflamed area is consciously inhibited by pain</td>
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| **Chronic** |                                                                 |                                                                              |
|             | 1. Chronic ulcer e.g. chronic peptic ulcer of stomach with breach of mucosa | 1. Cellular infiltrate = lymphocytes + plasma cells + macrophages |
|             | 2. Chronic abscess cavity e.g. osteomyelitis                          | 2. Fibrous tissue from granulation tissue                                    |
|             | 3. Granulomatous inflammation e.g. TB of lung                          | 3. Destruction, regeneration and repair of tissue keep happening             |
|             | 4. Fibrosis d/t chronic inflammation after cell infiltrate subsides e.g. chronic cholecystitis. | 4. Tissue necrosis may ensue                                                |
Summary acute

1. Initial reaction to tissue injury
2. Dilation of vessels
3. Increased permeability
4. Vascular leakage
5. Neutrophils recruited to site
6. Outcome: resolution, abscess, organization or chronic infla

Summary Chronic:

1. Usually followed by acute inflammation
2. Lymphocytes, plasma cells and macrophages predominate
3. Granulomatous inflammation
4. Granuloma = aggregate epithelioid histiocytes

Investigations/radiology

Methods of imaging bone: advantages and disadvantages

- Plain film xray: ionising radiation captured on film for development, x rays capture high density structures such as bone
  - Advantages: cheap, easy to use, portable, quick
  - Disadvantages: radiation, doesn’t detect early pathology, limited use for soft tissue pathology

- Isotope bone scan: bone metabolite with technecium tracer (radioactive), signal detected with Geiger counter
  - Advantages: detects physiological changes in bones, detects early changes covers whole body
  - Disadvantages: radiation, time consuming, limited spatial resolution

- Ultrasound: high frequency soundwaves, probe transmits and receives soundwaves
  - Advantages: real time image, no radiation, cheap, widely available, portable, good soft tissue detail
  - Disadvantages: poor resolution through adipose tissue (obese), cant see through ossified bone, user dependent with long learning curve

- CT: ionising radiation beam circles round patient, signal picked up by circular row of detectors to produce cross sectional image
  - Advantages: good bone detail, can reconstruct image in multiple planes
  - Disadvantages: massive doses of radiation, limited soft tissue detail, doesn’t pick up early bone pathology

- MRI: strong magnets align protons in magnetic field, when signal is switched off it leaves a signal
  - Advantages: Good soft tissue detail, multiple plane imaging, no radiation
  - Disadvantages: not widely available, expensive, claustrophobia, metal hazards such as pacemakers
**Inflammatory disease: blood tests**

Example of Rheumatoid Arthritis

- **RF**: Low specificity but high sensitivity
- A positive result could mean a number of other conditions such as Sjögren’s
- **FBC**: In active RA there is anaemia (normocytic—with normal haemoglobin levels) and a high platelet count due to inflammation
- Polymorphonuclear leucocytosis (increased number of neutrophils) may be a sign of inflammation
- **Thrombocytosis**: increased number of platelets often in active inflammation.
- **WBC count** should not be elevated but may be checked for exclusion of septic arthritis in some cases
- **LFT**: Elevation of alkaline phosphate in some cases in response to systemic inflammation
- Decreased albumin levels due to inflammation
- **ESR (non-specific)** and **CRP**, both usually increased in active RA. CRP usually a better indicator of acute inflammation in RA than ESR as it is independent of any other factors in the blood which interfere with the results (e.g. serum immunoglobulin levels)

**Red flag symptoms: how their presence influences the management of a patient**

- **Red flags** are those symptoms that a patient may present with that should prompt further investigation or referral as they could be the result of a serious pathology.
- In relevance to **low back pain**, the red flag symptoms are as follows;
  - Patient presents aged below 20 or above 50
  - **PMH**
    - Malignancy
    - Drug abuse
    - HIV or other immunosuppressive disorder
  - **Pain**
    - Thoracic pain
    - Night pain
    - Persistent pain that is not provoked by mechanical movement. Also rest does not relieve the pain.
  - **Physiological changes**
    - Fever, including night sweats
    - Unexplained weight loss possibly accompanied by systemic illness
    - Bowel/urinary incontinence
    - Structural deformity
  - Unusual neurological changes such as:
    - Numbness around the anus, perineum and genitals
    - Loss of strength in legs, particularly when walking
    - Cauda equina syndrome
  - Recent history of violent trauma
  - Long-term oral steroid use e.g. corticosteroids

If a patient presents with any other the above symptoms they **must** be referred to their practitioner and immediate action should be taken.

If **cauda equina syndrome** is suspected immediate consultation is required for emergency MRI or CT and definitive treatment.

If an infection, tumour or pathological fracture is suspected a full blood count including ESR and CRP levels should be taken. A urine sample, PSA (if necessary) x-rays and bone scans should be taken. MRI is appropriate for suspected epidural abscess, diskitis and/or osteomyelitis or for spinal neoplasm with potential cord or nerve compression.

For a fracture an x-ray of 2 plains will be required and maybe an MRI scan.

**Once a diagnosis is reached follow-up care will be necessary.**

Reassess diagnosis, review symptoms, physical findings and compliance with treatment plan.

Patients with neuro-motor deficits require earlier follow-up. Consider specialty referral.
Are psychosocial issues a barrier to recovery? Remain alert for non-physiologic pattern of pain or inconsistent physical findings. Determine whether modification of treatment is warranted. Provide assurance that recovery is expected. Support the patient to return to work and required daily activities as soon as possible. Encourage muscle conditioning exercises when able to tolerate.

**Law and ethics**

**Confidentiality, informed consent, individual autonomy, professional duty: definitions**

- Informed consent: Risk, benefits, side effects and alternatives. Must be voluntary and patient must be competent. Must be explained in balanced, unbiased and objective manner.
- Individual autonomy: Patient makes their own treatment decision based on informed consent. Right to refuse treatment. Opposed to paternalism
- Professional duty: duty of care to patient, behave in professional manner, unbiased, non judgmental.
- Confidentiality: private sensitive patient info must only be shared with those involved in the care of the pt, no disclosure without the consent of the pt, anonymise data where apt. Can only be broken to protect pt or others, if in public interest, to a court of law or to statutory regulatory bodies.

**Duty of Care: doctors’ legal duty, principles applied in assessing allegations of negligence**

- Duty of care: when a dr accepts a patient there is duty to treat the patient using all due caution, Dr has responsibility to use their skill and knowledge to achieve this aim, emergency treatment, patient presents at A & E, patient presents at hospital or GP surgery
- Standard of care: Bolam test, dr must act in manner that is accepted as proper practice by a responsible body of medical practitioners skilled in that area. Bolam v. Friern Hospital Management Committee [1957] 1 WLR582
- Breach of care: standard of care was not met
- Causation: breach of care is responsible for the damage suffered by the plaintiff, use the ‘but for’ test, but for the negligence of the defendant the plaintiff would not have suffered harm.
- Damages: level of compensation is related to the severity of the damage and its effects on the plaintiff
- Bolam case: plaintiff was psychiatric pt given ECT without muscle relaxant or manual restraint, sustained bilateral acetabular #s, defendant found not guilty as he treated pt in a way consistent with a reasonable body of medical practitioners at the time. This gave rise to ‘the Bolam test’

**Competency: definition, how this impacts on informed consent in older people**

Competence = ability to appreciate right from wrong & make decisions based on that

Informed consent:

- Understand there is a choice
- Making a choice will have consequences
- Choice not influenced by pressure
- Understand concepts of treatment
- Understand benefits of treatment
- Understand there are alternatives
- Understand side effects of the treatment and it’s alternatives
- Understand side effects of alternatives treatment
- Understand risks of not receiving the treatment
- Has enough time to make a decision

If pt is incompetent:

- Doctor act in best interest of patients unless pt stated otherwise (advance directive)
- Not treating is acceptable when:
  - Evidence proves that further treatment will not save life
  - Pt is irreversibly close to death
  - Pt has severe permanent brain damage & will be unable to carry out any independent activities
Informed consent and individual autonomy

Consent is a prerequisite to treatment of any person who is mentally competent. Treatment without consent can lead to the health practitioner being liable for trespass to the person, negligence or, in extreme cases, a criminal prosecution of assault or battery.

According to the GMC there are 12 key pieces of information to give patients in order to obtain informed consent; Details of the diagnosis, prognosis and the likely prognosis if left untreated. Uncertainties about the diagnosis including options for further investigation prior to treatment. Options for treatment or management of the condition, including the option not to treat. The purpose of proposed investigation – details of procedure, methods of pain relief, how patients should prepare, what they might experience etc. Explanations of the risks and benefits of the treatment. Advice about whether a proposed treatment is experimental. Side-effects and how they will be monitored. Name of the doctor who will have overall responsibility. Whether doctors in training will be involved and the extent of student involvement. Patient can change their decision at any time. Patient has the right to seek a second opinion. Where applicable, details of cost or charges which the patient may have to meet.

When presenting information to a patient it must be at an appropriate level. E.g. language, sensitivity, plenty of time etc.

For consent to be valid it must be free from coercion. Whilst patients may take into account the advice of others (family, friends, medical staff etc) they must still feel they are able to make an autonomous decision.

Treatment can only be forced upon patients in narrowly defined circumstances. Consent is not required when; The patient is unconscious and requires emergency treatment. Testing for certain infectious diseases e.g. cholera, plague, smallpox etc. The patient is incapable of giving consent e.g. child or mental disability. If a child is 16 or 17 they are treated as competent patients therefore can provide consent. Children under 16 are not normally considered competent unless they are Gillick competent.

Autonomy means ‘self-rule’. It refers to the ability; To reason and think about one’s own choices Decide how to act To act on that decision

It is more than simply being free to do what one wants to do. It implies that rational thought is involved.

Involves the process of weighing out risks and benefits of a decision.

Euthanasia and assisted suicide: ethical and moral issues

- “euthanasia” eu = good and thanasia = death. Action or omission by a medical professional that directly results in the death of a patient (e.g lethal injection).
- Euthanasia 1: Voluntary: Requested by a competent patient who is fully informed; Non-voluntary: Carried out on a patient who is not competent; Involuntary: carried out on a competent patient who has expressed a wish not to die (murder).
- Euthanasia 2 (above forms are either): Active: 1 person’s action leads to death of patient (consented or not) e.g. R vs. Cox 1992 terminally ill patient in constant pain was injected with a lethal dose of potassium chloride by Dr Cox; Passive: patient allowed to die by withholding or withdrawing treatment e.g. do not resuscitate (DNR). This is deemed acceptable if it is in the best interest of the patient.
- Autonomy. Legal & ethical dilemma: individual is demanding the right not only to escape suffering, but also to die with the aid of a third party.
• Arguments for: Suicide legal but severely disabled can’t do so without help; Passive euthanasia acceptable (DNR) but active euthanasia would cause less suffering; Life shortening painkillers & sedatives permissible in terminally ill patients to relieve pain; also speed up death.

• Against: Good palliative care; what would patient have chosen before they got ill (competence an issue at end of life?); mentally ill/disabled may be manipulated via guilt; slippery slope to non voluntary death of the vulnerable (dementia).

• Assisted suicide: individual unable to take their own life due to incapacity is provided with the means to commit suicide by a third party (eg help travel to dignitas).

• Suicide Act of 1961: suicide legal but it is a criminal offence for a third part to assist an individual in committing suicide.

• Recent high profile cases involving the issue of assisted suicide: Daniel James, 23-year-old rugby player, paralysed from the chest down (not terminally ill). Parents helped him to dignitas; Debbie Purdey, 46 year old MS sufferer, appeal now at House of Lords for clarification of law so husband isn’t prosecuted.

• Dilemas: should assisted suicide only be for terminally ill?; should someone have to travel alone to Switzerland before they get too ill?

**Informed consent**

Issues behind Informed Consent

1. Competency: Gillick competent child; patient can suffer from mental illness as long as she has the capacity to understand consequences, the greater the consequences the greater the understanding needed. If patient lacks capacity then:

2. Advanced Directives can be utilised: this document must be written and witnessed, must apply to the situation, an appointed and registered person with Lasting Power of Attorney to make decisions) exists. Otherwise, ‘Best Interests’ of the patient prevails.

   Case example:
   Malette and Shulman (1988); Jehova’s witness carried a card saying no transfusion but in A & E they gave her blood, she won because the patient’s Advanced Directives must be respected even if that means she will die.

3. Family and Friends could represent the patient’s views

4. Independent Mental Capacity Advocate (2007): independent from NHS and local authorities, role is to assist people without capacity to make decisions and to be heard while understanding measures that have to do with their life

5. Patient’s best interests and Duty of Care: Responsibilities and Rights of health care workers such as: able to legally refuse to treat a patient but must transfer over; must provide emergency treatment; ethically should not let own views affect care given; document clearly if patient agrees/refuses; if conflict arises move on to next level.

   Case example
   Bland (1993); a doctor needs only show that she is following an accepted medical practice, even if that practice is followed by a minority of medical professionals; this was in reference to Dr. Howe who could have been charged with murder, but was not, because he withdrew life support measures from Mr. Bland, who was in a persistent vegetative state.

6. Court of Protection

7. Higher Courts

**Define:** Consent is the patient’s agreement for a health professional to provide care; If a healthcare professional does something without that consent he or she will, in most cases, have acted unlawfully and be guilty of a criminal and/or civil offence and at risk of disciplinary proceedings e.g., being struck off by the GMC, Implied/Oral/Written Consent

The following are the items necessary for someone to provide fully Informed Consent:

1. Patient must be competent
2. Needs to be INFORMED
3. Needs to UNDERSTAND the information
4. Must RETAIN INFORMATION
5. Weighs up PROS and CONS
6. Gives consent FREELY
7. Must be RESPECTED

Law Cases of note
• Bolam vs Friern Hospital Management Committee [1957]: set the standard of negligence.
  o Courts determine which risks need to be mentioned to patients using the Bolam test.
  o It requires the standard of care not to fall below: ‘the standard of the ordinary skilled man exercising and professing to have that special skill’ – the standard set by the medical profession.
  o John Bolam was psychiatric patient undergoing electro-convulsive therapy (ECT).
  o He was not warned of the risk of fractures (which he sustained) due to the convulsions.
  o Judgement: other doctors testified that they would have treated Bolam in the same way, that is, a reasonable body of medical practitioners would not have done anything differently so Bolam’s doctor was not found guilty of negligence.
• Sidaway v. Board of Governors of the Bethlem Royal Hospital and the Maudsley Hospital [1985]: relates to informed consent
  o The claimant suffered from pain in her neck, right shoulder, and arms. Her neurosurgeon took her consent for cervical cord decompression, but did not include in his explanation the fact that in less than 1% of the cases, the said decompression caused paraplegia. She developed paraplegia after the spinal operation.
  o Judgement: rejecting her claim for damages, the court held that consent did not require an elaborate explanation of remote side effects. In dissent, Lord Scarman said that the Bolam test should not apply to the issue of informed consent and that a doctor should have a duty to tell the patient of the inherent and material risk of the treatment proposed.
• Bland v Airedale NHS Trust [1993]: issues surrounding withdrawal of treatment
  o Tony Bland was a football supporter who was left in a persistent vegetative state following the Hillsborough disaster in 1989.
  o He was being fed artificially and mechanically through a nasogastric tube.
  o Airedale NHS Trust applied to the courts for a declaration to the effect that: they might lawfully discontinue all life-sustaining treatment and medical support measures, including ventilation, nutrition and hydration by artificial means.
  o In the House of Lords, Lord Goff stated that where a patient lacks capacity, a treatment can be discontinued where its use is no longer considered to be in the patient’s best interests.
• Re C (Adult: refusal of treatment) [1993]: definition of capacity and the Re C test
  o C was an inpatient diagnosed with schizophrenia who believed he was an internationally renowned doctor.
  o He developed gangrene on the toes of one foot. C’s surgeon believed he should have an amputation, believing it better to die with both feet than live with one.
  o C’s solicitor applied for an injunction to prevent amputation, which was granted as the courts found that, notwithstanding his schizophrenia, C was capable.
  o 3 stages of Re C Test:
    ▪ Can patient take in and retain information
    ▪ Does the patient believe this information
    ▪ Can the patient weight that information balancing needs and risks
• Diane Pretty [2002]: euthanasia
  o Dianne pretty suffered from motor neurone disease and wanted permission for her husband to assist in her suicide once she became unable to do it herself.
  o The House of Lords ruled that, despite the patient’s competence and autonomy, she was asking for her husband to perform an act of assisted suicide, which is not legal in the UK in order to protect life and the vulnerable.
• Burke v the General Medical Council [2004] end of life decision making
  o Leslie Burke is a man with cerebellar ataxia, a degenerative brain condition which will eventually lead to loss of speech and movement; he will require treatment by the way of artificial nutrition and hydration to keep him alive.
- He is afraid that when he becomes unable to communicate, although he may still be conscious of what was happening to him, artificial feeding would be withdrawn with the effect that he would starve to death.
- Autonomy and the right of self-determination do not entitle the patient to insist on receiving a particular medical treatment regardless of the nature of the treatment.

- **The Gillick Case [1986]** (Fraser competence): relates to children and consent
  - Mrs Gillick had 10 children, a number of whom were girls under the age of 16
  - She was concerned by a Department of Health and Social Security circular that advocated the preservation of confidentiality when the patient was requesting contraception, even if the patient was less than 16.
  - Mrs Gillick objected and went to court to ensure that the Health Authority did not give contraceptive advice to her children without her consent
  - The House of Lords decided against Mrs Gillick because it was asserted that:
    - The parental right to control a child existed for the benefit of the child not the parent; it is thus only justified in the best interests of the child
    - Parental right should yield to the child’s right when the child reaches a sufficient understanding and intelligence, which may be present under the age of 16 (up to the doctor to decide when the child can understand the medical, social and moral aspects of proposed treatment)
  - The judgement allows children under 16 to consent to medical treatment, it does not allow them to refuse (which is not binding until 18)

- **Miss B [2002]**
  - A woman known as "Miss B", who was paralysed from the neck down, died peacefully in her sleep on 29 April 2002 after winning the legal right to have medical treatment withdrawn.
  - The high court that Miss B had the "necessary mental capacity to give consent or to refuse consent to life-sustaining medical treatment".
  - It was the 43-year-old former social care professional's case that it was her decision, not her doctors', whether the ventilator which kept her alive should be switched off.
  - In a landmark ruling, Dame Elizabeth gave Miss B the right to be transferred to another hospital and be treated in accordance with her wishes, including drug treatment and care to "ease her suffering and permit her life to end peacefully and with dignity".

- **Cox [1992]**
  - Dr Nigel Cox remains the only doctor ever to be convicted in the UK of attempting to perform a mercy killing.
  - A consultant rheumatologist from Hampshire, he was found guilty of attempted murder after injecting 70-year-old Lillian Boyes with a lethal drug.
  - Dr Cox's act was discovered by a nurse who read Miss Boyes medical notes. She realised that the potassium chloride he had used would not alleviate pain, but instead stop Ms Boyes' heart.
  - The charge of attempted murder was brought because it could not be proved conclusively that the injection had killed her.
  - Despite the verdict, Winchester Crown Court imposed a suspended sentence, while the General Medical Council let him off with a reprimand. He is still practising medicine in Hampshire.

### UK legal system

**Sources of law**
- **Parliament** – acts and statutes
- **Court-made** – common law

**Types of law**
- **Criminal**
  - Usually R v. Smith
  - Defendant accused of committing a crime by prosecution (ie battery assault)
  - Proof must be ‘beyond reasonable doubt’
- **Civil (tort) law** (usually medical are civil and often negligence)
  - Usually Jones v. Smith
  - Defendant sued by plaintiff
  - Proof ‘balance of probabilities’
Medical negligence
1. A duty of care
2. A standard of care
3. A breach
4. Causation
5. Damages

MDT

Shared care in patients with inflammatory arthritis
- Shared care refers to dividing care between 1° and 2° care settings
- The purpose of this is to relieve the burden of repeated outpatients appointments for patients in hospitals and allow patients who need care (e.g. flare up) to get it when they need it.

Shared care in chronic illness, differing roles of primary, secondary and intermediate care professionals
- Rheumatology dept: rheumatologist (Dx, Tx, management of care plan, research), rheumatology practitioner (pt education, monitor DMARDs and biologics, DAS scores, advice line for pt and GPs)
- Role of rheumatology dept: pt centred care, clinical assessment, lx for diagnosis and monitoring, (Bloods, Xrays, DEXA, U&E, Cultures, LFTs), re view 2/12, symptom management
- Pt education: disease process, side effects of drugs, coping strategies, benefits advice,
- OT: ADL, home assessment, walking aids, home domestic aids (hand splints, tilting kettles etc)
- PT: exercise programs, assess mobility, hydrotherapy, ultrasound
- Nurse: cannulation, injections, observe infusions
- Podiatrist: assess gait and stance, orthotic aids
- Orthopaedic surgeon: arthroscopy, arthroplasty, arthrodesis
- Dietitian: advice on healthy eating, weight loss/gain
- GP surgery: make original referral to rheumatologist (18 week target), day to day care, referrals to specialist, administer MTX injections, pt education and advice, fill in blood monitoring and DMARD dosage booklets, comm With rheum dept
- Others: pain clinic, dermatology, social services, psychologist
**Misc. Unit 2**

**Osteoporosis: epidemiology and societal costs of osteoporosis**

**Epi**
1 in 3 women, 1 in 12 men in UK, 200 million worldwide
180,000 fractures per year in the England and Wales result from osteoporosis
Lifetime risk estimates 50+ year old white women n men for OP #s: forearm: 13% wom, 2% men, vertebrae: 11% wom, 2% men, femoral neck: 14% wom, 3% men.
Risks: age (especially post menopausal women), steroid use, low body mass

**Soc costs**
£1.7 billion rough annual total UK cost.
£ Costs: # management, rehab, falls prevention, medication, side effects of OP prophylaxis (oesophageal damage if patient is forgetful about how to take bisphosphonates correctly).
OP causes > 200,000 fractures each year at a cost to the NHS of more than £940m.
Other costs: worry to patients about getting out and about after a previous #, worry to families about relative’s condition and living accommodation, less social life for pt, less independence, care home/nursing costs to family or individual, grief.

Morbidity & mortality rate: After hip # up to: 20% die within a year, 50% are incapacitated, 20% require long-term residential care.
OP treatment reduces the risk of vertebral fracture by between 30-65% and of non-vertebral fractures by between 16-70%.

**Polyarticular pain: differential diagnosis and clinical features**

**OA or Degenerative Arthritis - “wear & tear”**

- Primary
- Secondary
  - Trauma
  - Congenital Disorders
  - Metabolic
  - Endocrine
  - Neuropathic
  - Paget’s
  - Inflammatory arthritis
- Clinical features of OA - pain – activity related, transient morning/inactivity stiffness, joint enlargement, limitation of movement, muscle atrophy, crepitus
- Distribution - DIP > PIP > MCP, CMC Thumb, AC Shoulder, Hips, Knees, MTP Feet, Facet joints

**Inflammatory Polyarthritis**

- RA - additive joint involvement, Articular features= pain, stiffness (noticed on waking), swelling, small-medium joints in symmetrical fashion. Note: palindromic rheumatism. Extra articular features= rheumatoid nodules, tenosynovitis, carpal tunnel. Systemic features= general malaise, fatigue, fever. Anaemia, cardiac disease, eye disease, amyloidosis and vasculitis
- Sero-negative Spondylarthritis e.g. Ank Spond - clinical features - low back/buttock pain, early morning stiffness, enthesopathies, peripheral arthritis
- Connective Tissue Disease - non specific features e.g. fatigue, malaise, fever. Polyarthritis is non erosive, deformity occurs due to tenosynovitis and fibrosis rather than cartilage/bone erosion. Dermatological features= photosensitivity, malar rash, discoid lupus, alopecia, urticarial lesion, palpable purpura and splinter haemorrhages. CVS features= pericarditis. Pulmonary features= pleurisy and pleural effusions. Renal features= glomerulonephritis. Neuro features= headaches to psychiatric problems
Crystal Arthritis - intermittent joint involvement usually 1st MTP, chronic gout is characterised by tophus (soft tissue deposits of urate) formation and joint destruction in digits and helix of ear.

Infection Related - acute onset, fever + systemic upset, variable severity, self-limiting, 6-12 weeks duration e.g. reactive arthritis - features; aseptic arthritis following infection, 1-4 week time lag, M=F, most common in young, adult males, acute or insidious onset, fever, fatigue, weight loss, extra-articular features, HLA-B27 in 60 - 80%

Infection in bone: presentation and causative organisms

- Osteomyelitis: infection of bone by bacterial organism
- Causes: Post trauma (open fracture, requires urgent surgical debridement and lavage otherwise fracture will fail to heal), post surgery (bacteria on prostheses) acute haematogenous Osteomyelitis (most common in children)
- Pathology: Trauma followed by bacterial colonisation of metaphysis of long bone, inflammation and pus formation occurs as the metaphysis has a good blood supply and few phagocytes, pus escapes into the subperiosteal space to form an abscess, the affected area of bone starts to die leaving behind a piece of dead bone (sequestrum) that harbours infection, new periosteal bone forms ( involucrum) over the sequestrum in response to infection, infection can be contained by growth plate but if it gets past then it can go into the joint and cause septic arthritis
- Causative agents: S. aureas, group B strep, HIB, TB (potts disease of spine)
- Risk factors: Sickle cell disease, haemophilia, diabetes, renal failure, IV drug use, malnutrition, immunosupression, HIV/AIDS
- Clinical features: Pain, fever and loss of joint function (insidious onset), most common in tibia and femur, limb will be tender to palpate, erythematous and possibly swollen, sometimes caused by another focus of infection that has spread to the bone e.g. infective endocarditis, chronic osteomyelitis occurs when a sequestrum remains present in the bone and can flare up unexpectedly after many years
- Ix: FBC (raised WCC, CRP and ESR), blood culture, Xray (normal at first but will display lysis, periosteal elevation and new bone formation after 10 days)
- Complications: Chronic Osteomyelitis, damage to physis resulting in impaired growth and deformity, septic arthritis, prognosis is good if complications are avoided otherwise surgery or amputation are considered
- Management: Analgesia, splintage and IV antibiotics, for S aureas infections flucloxacillin or fusidic acid usually used first line, If abscess is present then needs to be drained surgically. Any sequestrum must also be removed, flare ups can be treated with antibiotics, damaged joints can be replaced with prosthetic joints.

Monoarticular Pain
Osteoarthritis: risk factors

- Increasing age
- Female sex (for knee disease)
- Family history (several chromosomal loci and gene variations have been identified as putting someone at increased risk for OA)
- Previous joint injury including infection, intra-articular fracture and ligament tear causing joint instability
- Joint malalignment problems such as Perthes disease, slipped upper femoral epiphysis, congenital dislocation of the hip
- Obesity
- Occupational (knee OA in elite athletes, elbow OA in people working with pneumatic drills)
- Ethnic origin (more common in white Europeans)
### Acute septic arthritis and acute crystal arthritis: presentation, investigation, pathophysiology

<table>
<thead>
<tr>
<th>Causative Organism</th>
<th>Acute Septic Arthritis</th>
<th>Acute Crystal Arthritis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Adults:</strong></td>
<td>- staphylococcus aureus</td>
<td>- Prolonged hyperuricaemia leading to uric acid crystal formation in synovium, connective tissues and kidneys</td>
</tr>
<tr>
<td></td>
<td>- streptococci</td>
<td>- Pseudo Gout is calcium pyrophosphate dehydrate CPPD</td>
</tr>
<tr>
<td></td>
<td>- Neisseria gonorrhoeae (common causative organism in young sexually active adults)</td>
<td>- Inflammation due to crystals being ingested by neutrophils and releasing inflammatory enzymes</td>
</tr>
<tr>
<td><strong>Children:</strong></td>
<td>- Haemophilus influenza (children)</td>
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<table>
<thead>
<tr>
<th>Pathological Process</th>
<th>Acute Septic Arthritis</th>
<th>Acute Crystal Arthritis</th>
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</thead>
<tbody>
<tr>
<td><strong>1. Contamination by:</strong></td>
<td></td>
<td>Causes of hyperuricaemia:</td>
</tr>
<tr>
<td>- Direct penetration through skin</td>
<td>1. reduced renal urate excretion as opposed to increased uric acid production due to:</td>
<td></td>
</tr>
<tr>
<td>- Spread from metaphysis</td>
<td>✓ Drugs (diuretics, low dose salicylates, pyrazinamide, ethanol)</td>
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</tr>
<tr>
<td>- or from an infected site (haematogenous)</td>
<td>✓ Renal disease</td>
<td></td>
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<tr>
<td>2. The infective organism reaches the synovium causing inflammation</td>
<td>✓ Hypertension</td>
<td></td>
</tr>
<tr>
<td>3. Leukocytes migrate to joint</td>
<td>✓ Increased levels of organic acids (lactic acidosis, ketoacidosis, respiratory acidosis)</td>
<td></td>
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<tr>
<td>4. Enzymes and breakdown products cause damage to the articular cartilage (within hours)</td>
<td>✓ Hyperparathyroidism</td>
<td></td>
</tr>
<tr>
<td></td>
<td>✓ Increased uric acid production</td>
<td></td>
</tr>
<tr>
<td></td>
<td>✓ Increased dietary intake of purines (red meat, offal, beer)</td>
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</tr>
<tr>
<td></td>
<td>✓ Increased turnover of purines</td>
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</tr>
<tr>
<td></td>
<td>✓ Increased purine synthesis – HGPRT(ii) deficiency</td>
<td></td>
</tr>
<tr>
<td>2. Precipitants:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>✓ Trauma</td>
<td>✓ Vision</td>
<td></td>
</tr>
<tr>
<td>✓ Illness</td>
<td>✓ Surgery</td>
<td></td>
</tr>
<tr>
<td>✓ Surgery</td>
<td>✓ Binge drinking</td>
<td></td>
</tr>
<tr>
<td>✓ Starvation</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Presentation</th>
<th>Acute Septic Arthritis</th>
<th>Acute Crystal Arthritis</th>
</tr>
</thead>
<tbody>
<tr>
<td>- sudden onset of hot, red, swollen, agonizing pain worse on movement</td>
<td>- sudden onset of hot, red, swollen, agonizing pain worse on movement</td>
<td></td>
</tr>
<tr>
<td>- Systemically unwell, fever</td>
<td>- Gout: 1st MTP</td>
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<tr>
<td></td>
<td>- Pseudogout: Knee /wrist</td>
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</table>

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Acute Septic Arthritis</th>
<th>Acute Crystal Arthritis</th>
</tr>
</thead>
<tbody>
<tr>
<td>- White Cell Count</td>
<td>- X-ray: pseudo-gout = chondrocalcinosis</td>
<td></td>
</tr>
<tr>
<td>- Joint Aspiration</td>
<td>- Synovial fluid: urate crystals = needle-shaped</td>
<td></td>
</tr>
<tr>
<td>- Gram Stain and culture of synovial fluid</td>
<td>CPPD = rod-shaped</td>
<td></td>
</tr>
<tr>
<td>- Blood cultures</td>
<td>birefringent</td>
<td></td>
</tr>
<tr>
<td>- Skin/rash/oral/urethral swabs</td>
<td></td>
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</tr>
</tbody>
</table>

<p>| Important considerations | Symptoms aren’t as severe in people with RA or those on immunosuppressants (steroids) |</p>
<table>
<thead>
<tr>
<th></th>
<th>Septic Arthritis</th>
<th>Crystal Arthritis (acute)</th>
<th>Osteoarthritis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Presentation</strong></td>
<td>Hot, red, swelling, agonizing pain, immobility, systemic illness</td>
<td>Hot, red, swelling, agonizing pain, usually in 1st MTP</td>
<td>Aching/burning pain, stiffness, swelling, loss of function</td>
</tr>
<tr>
<td><strong>Cause</strong></td>
<td>Infection in joint - Staphylococcus Aureus/Streptococcus</td>
<td>Crystal formation due to hyperuricaemia</td>
<td>Focal erosion of cartilage</td>
</tr>
<tr>
<td><strong>Predisposing factors</strong></td>
<td>Haematogenous, spread from metaphysis or penetrating trauma/surgery</td>
<td>Reduced renal excretion or increased uric acid production</td>
<td>Secondary factors: anything that caused existence of joint damage.</td>
</tr>
</tbody>
</table>

### Neck/Back Pain

**Non-pharmacological advice which may help a patient with cervical pain**
Reducing stress and learning to relax often reduces neck and back pain. There is a wide variety of psychological therapies available: behavioural management, hypnosis, simple breathing techniques. Referring patients with chronic pain to pain clinics can be beneficial.

- Neck pain is the second most common musculoskeletal disorder after lower back pain. At any one time between 5.9-13.4% of the population are affected.

**Physiotherapy**

- Passive approaches (usually short term relief): Done by PT. Manual therapy involves mobilisation, manipulation, traction and massage. Other methods include trigger point therapy, electrotherapy and acupuncture.
- Active treatments (can improved long term outcomes): Done by patient. Specific exercises, advised to partake in appropriate general exercises, self mobilisation exercises, advice on lifestyle and ergonomic changes. **Patient learns self treatment/coping strategies so will require less healthcare intervention.**

Ultrasound, heat, hydrotherapy.

Osteopathy involves (non-surgical) manipulation. Chiropractic treatment, acupuncture involves insertion of fine needles into different points on the body thought to relax muscle spasm and stimulate. Osteopathic treatment, acupuncture involves insertion of fine needles into different points on the body thought to relax muscle spasm and stim. Prod. Of endorphins.

Trancutaneous electrical nerve stimulations (TENS) and percutaneous electrical nerve stimulation (PENS) involves sending low voltage electrical impulses to nerve fibres. Thought to work by Gate theory.

### Imaging techniques for cervical spine: advantages and disadvantages

<table>
<thead>
<tr>
<th>Type of imaging</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plain film x-rays</td>
<td>Cheap, widely available, easy to use, quick, portable</td>
<td>Radiation, insensitive to early pathology, limited evaluating of soft tissue pathology</td>
</tr>
<tr>
<td>Nuclear Medicine (Isotope bone scan)</td>
<td>Highly sensitive to early bone pathology, covers whole body</td>
<td>Radiation, time consuming, cost</td>
</tr>
<tr>
<td>Ultrasound (US)</td>
<td>No ionising radiation, cheap, widely available, portable, soft tissue detail</td>
<td>Blocked by bone, requires specialist training</td>
</tr>
<tr>
<td>Computed Tomography (CT)</td>
<td>Good bone detail, can reconstruct an easy to interpret 3D image</td>
<td>Ionising radiation, insensitive to soft tissue &amp; early bone pathology, cost</td>
</tr>
<tr>
<td>Magnetic Resonance Imaging (MRI)</td>
<td>Good soft tissue detail, detects early bone pathology, image in multiple planes, no ionising radiation</td>
<td>Not widely available, expensive, time consuming, contraindications: claustrophobics, pacemakers, eye/cochlear implants</td>
</tr>
</tbody>
</table>
Therapeutic interventions for neck pain

Physiotherapy is one of the therapeutic interventions. It is divided into active and passive approaches. Passive therapies include manual therapy, electrotherapy and acupuncture. Active therapies are those that patients can do themselves e.g. exercises, lifestyle advice etc. These provide long term relief.

Physiotherapy is available on the NHS and is given to inpatients or outpatients through referral. In some parts of the country it is now possible to self-refer to a physiotherapist.

Osteopathy involves non-surgical manipulation. It is based on the notion fixing structural defects should improve function and relieve pain. A private 30-40 minute session costs between £25-50. It is regulated by The General Osteopathic Council, (GOsC), one of the 13 health and social care regulators in the UK.

Chiropractic treatment uses pressure or physical thrust and a combination of other alternative therapies. It focuses on structural and functional interactions, e.g. misaligned vertebrae.

Acupuncture involves the insertion of very fine needles into different points in the body. It helps Qi (life energy) to flow correctly. Different points in the body are related to vital organs and systems. From a more scientific view, its direct effect is to relax muscle spasms, and is thought to stimulate the production of endorphins (endogenous pain relief) and serotonin. It is still not fully understood.

Electrical stimulation therapies involve sending low voltage electrical impulses to nerve fibres. The patient can control the amount of stimulation. The mechanism of electrical stimulation is still not completely understood, though it is accepted that its analgesic effect is due to alteration of neuronal ability.

Therapeutic massages relax muscles, improve circulation, help remove metabolic waste and stimulate release of endorphins. It is also considered of psychological benefit through relaxation. It can reduce muscle spasm and have ‘indirect effect’ factors.

Work related musculoskeletal pain and the interventions available to reduce these problems

- Big prob: 1.14 million people in 2006/07 in the UK
- 2 main WMSD categories:
  - Disorders arising from soft tissues (e.g. muscle/tendons) → tenosynovitis
  - Disorders arising from bones and joints → osteoarthritis of the hip
- Spectrum of WMSDs (5 categories)
  - Lower back pain – awkward posture / lifting, twisting, forceful movement, whole body vibration
  - Neck & shoulder pain – poor posture – manual handling, repetitive movements, work above chest level (neck flexion)
  - Upper limb pain – carpal tunnel (regular prolonged vibration), wrist tenosynovitis (repetitive movements, manual handling), epicondylitis (repeated pro/supination, force), WRULD work rel. d upper limb disorder (non specific diffuse forearm pain – psychosocial factors).
  - Hip pain – OA (big loads: farming, lifting, stairs)
  - Lower limb pain – knee OA (bending, kneeling – eg carpet layers).

Interventions to reduce WMSDs

Changes for employer to introduce:

- Training → how to do the job in the safest way possible → manual handling
- Induction period → lets employees observe from colleagues how the work should be done safely
- Job rotation/variability → stop having to do same repetitive movements
- Rest breaks
- Avoid repetitive, monotonous tasks
- Task optimization
- Good job design
- Good ergonomic — see past outcome on this, making the job fit the worker!
- Improve job satisfaction to avoid psychosocial causes!


**MDT role in treating low back pain**

1. **GP** (Explain Diagnosis, pathology and proposed management)
   - Avoid loss of work by recommending early activity, rehabilitation, pain management
   - Identify urgency of referral and/or treatment (red flags)
   - Provide adequate information on referral
   - Filling out sick forms, informing of benefits
   - Assess potential for long term chronicity and disability, such as financial and social problems

2. **Physical therapists** (Physiotherapist, Occupational Therapist, Osteopath, Chiropractors)
   - Schedule of physical rehabilitation, Manipulation for pain relief, early mobilization, acupuncture

3. **Occupational Therapy** (Workplace Rehabilitation)
   - Occupational/Activity review
   - Ergonomics in the workplace

4. **Occupational Health** (Modifications in the workplace, nurse and doctor)
   - Ergonomics in the workplace, modified duties, close follow up of progress and assistance with bureaucratic paper work, recommendation

5. **Anaesthetic team** (Pain management)
   - Epidural injections, psychological support, assessment of social environment

6. **Psychotherapist/Counsellors** (Emotional support)
   - Management of distress, depression and anxiety, clarifying emotional issues due to loss of income and chronic pain

7. **Orthopaedic/Spinal Team** (Surgical options)
   - Assist patient with decision making and realistic expectations of results, i.e. pain may never go away completely

8. **Social Worker**
   - Assist with receiving Benefits, as well as identifying social barriers to returning to work (chronic illness, family support or lack of)

9. **Complementary Medicine**
   - Acupuncture, Osteopathy, Homeopathy, Chiropractor, Chinese Medicine

10. **DEA** (Disability Employment Advisor)
    - Assist with Disability Benefits and getting back to work programmes

11. **Administrative Support** (Secretarial, administrator)
    - Management of paper work, referral letters, mailing out paper work, assist with communication between involved carers

12. **Insurance personnel** (If applicable)
    - Assessment of fitness to work, physical status, pain levels, modified work, compensation & redress if work related

**Overall goal of the MDT is to:**
- Provide comprehensive care — prevention, maintenance and chronic disease management
- Assist patients with being active partners in health care plan
- Empowering patient to make informed decisions
Degenerative and age related changes in the spine Prolapsed intervertebral disc
- Occurs when part of the nucleus pulposus herniates through the annulus fibrosus and presses on a spinal nerve root.
- Herniation of disc material tends to occur posterolaterally where the annulus is thinner.
- Central disc prolapse can occur and press on nerve roots - cauda equina syndrome. Prolapse can occur without spinal root involvement- pt will have symptoms of back pain but not true sciatica.
- Most commonly at L4-L5 or L5-S1 but can occur at any level (rarely thoracic).

Spondylolisthesis
- Forward slipping of one vertebral body on the one below
- Usually occurs at the L5-S1 level
- Caused by conditions that induce instability by interfering with the posterior bony elements of the vertebra or with the posterior ligament complexes.
- May be associated kyphoscoliosis

Spinal stenosis
- Caused by degenerative changes narrowing the spinal canal and causing compression of the nerve roots.
- Thickening of soft tissues, osteophytes and posterior disc bulge encroach into spinal canal.
- In lateral recesses of spine, nerves may be compressed by overhanging facet joints so pt present with sciatica fairly similar to that produced by disc herniation. BUT, because the thick joints lie posterior to the spinal nerves, the sciatica will be worse with standing/walking but relieved by sitting.

Clinical history: mechanical back pain, inflammatory back pain and metastatic back pain, including red flags
- Mechanical back pain
  - Patients most often complain of pain in the lumbosacral area.
  - No pathological changes present in the back.
  - No neurological signs present in the legs.
  - Most people experience pain primarily in the lower back (lumbo-sacral region). The pain may spread (radiate) to the buttocks, and posterior thighs; it is often asymmetrical in distribution.
- Inflammatory back pain
  - Presents with gradual onset of lower back pain and stiffness
  - Symptoms worse early in the morning or after long periods of rest and usually improve with exercise
  - Systemic features e.g. anorexia, fever, weight loss, fatigue
- Metastatic back pain: red flags
  - Age of onset <20 or >55 years
  - History of malignancy
  - Persistent, non-mechanical pain
  - Night pain
  - Fever/ unexplained weight loss
  - Bladder/ bowel dysfunction
  - Progressive neurology, abnormal gait, saddle anaesthesia

Low back pain: epidemiology and risk factors
- 4.7% incidence from ages 25-64, accounts for 5% of all GP consultations, cost NHS £500mil in 1995, cost £1.4bil in benefits in 1995
- Major cause of work disability, costs economy £6bil per yr, the longer you are off work the less chance you have of returning
- Peak prevalence occurs between ages 20 and 55, becomes less frequent in later life men and women are equally effected.
- 50% of patients are better within 1 week and 90% within 6 weeks
- Recurrence: 60% will have a recurrence within 1 year, recurrent attacks tend to settle within 3 to 5 years
- Risk factors: Heavy physical work, lifting and handling of loads, including patients, awkward postures and movements including bending, twisting, static postures and having to lift in an awkward way, whole body vibration as with driving a large vehicle
Psychosocial reasons for failure to recover (Yellow flags): Belief that activity that causes pain is harmful, sickness behaviours such as extended rest and taking to bed, social withdrawal, emotional problems (low mood, depression, anxiety and stress), problems at work or dissatisfaction with work, pending medico-legal claim, overprotective family or lack of support, inappropriate expectations of treatment, including not appreciating the need for active participation in treatment.

Lifting and handling principles
- Manual handling: ‘any supporting or transporting of a load (including lifting, putting down, pushing, pulling, carrying or moving therof) by hand or by bodily force.
- Musculoskeletal ill health accounts for 27% of early retirements in Medical staff.
- Reasons for back pain: Poor Posture, Poor Lifestyle, Repetitive Workload, Physical & Psychological Stress, Little “down” time, Poor or Cramped Work Environment
- Avoid: overreaching, bending forward, leaning on bed to stabilise, twisting spine, continual standing, repetitive movements, little or no movement or leaning.
- Ensure Head is up, elbows in (load close to body), knees and hips unlocked, dynamic stable base and no twisting or bending.
- Workstation: ensure clear: Chair, Desk, Screen height right; adequate lighting.
- Spine – Natural S Shape ‘strong, stable, secure, safe’ – unnatural C Shape ‘caution, care’

Pain

Physiology of pain, how pain is perceived
- Physiology of pain: stimulation of nociceptors, pain gate theory, signal travels up spinal cord, brain conceptualises signal, relates signal to previous memory.
- Nociceptors stimulate by: mechanical deformation, excessive heat, and many chemicals
- Nociceptive fibres in PNS sensitised by PGs, become more sensitive to histamine, bradykinin etc, PGs also sensitise neurones in CNS spinal cord, sensitise to substance P.
- 4 basic principles involved in nociception: transduction, transmission, perception, modulation
- 2 kinds of primary afferent nociceptive cell: C fibres (unmyelinated and slow conducting, dull and not well localised) and Aδ fibres (myelinated and fast conducting, sharp and well localised)
- 3 stages of transmission: a) from site of transduction along nociceptor fibres to the dorsal horn of the spinal cord. b) from the spinal cord to the brainstem via spinothalamic and spinparabrachial pathways. C) Through connections between the thalamus, cortex and higher levels of the brain.
- Perceptions of pain: intensity (background, acute, dull, rising and falling, constant), spread (radiating down a limb, shooting pain), duration (always, precipitated, better/worse with movement, night time), nature (sharp, stabbing, dull, throbbing, aching).
- Biopsychosocial model: bio (neuropathic and nociceptive pain), psycho (distress, thoughts, understanding, memory), social (illness behaviour)
- Referred pain: incoming nociceptive afferents active interneurons which are shared by more than one nociceptive afferent (e.g. somatic and visceral share same interneuron during a heart attack leading to referred pain).

How pain is experienced: influence of physical and psychological external factors
- Transduction, transmission, evaluation and modulation (memory, hyperalgesia and allodynia)
- Nociceptive pain: pain fibres stim in skin, connective tissue, bone etc, visceral pain, aching pain, well localised
- Neuropathic pain: CNS, phantom limb pain, nerve root compression, MS, nerve lesion, release of substance P at damaged axon terminal mediates chronic inflammation
- Neuroplasticity: occurs after physical and psychological trauma, changes in neurone (new synapses, altered gene expression, altered cell surface receptors), glial cells and astrocytes (release inflammatory cytokines and form new synapses)
- The pain gate: Limited capacity channel in dorsal horn, close the gate (happiness, sleep, exercise, acupuncture, massage, control, distraction), open the gate (depression, anxiety, loss of sleep and pain becomes a vicious cycle, tiredness, bereavement, job loss, divorce)
• Influences on pain perception: sex, past experience of pain, genetics, personality, culture, society, environment (learned behavior from family)
• Altered behavior due to chronic pain: primary gain (avoidance of work, sex), secondary gain (financial gain from benefits, medicolegal claim), tertiary gain (sick role of pt and carers role of spouse)
• Definition of pain: unpleasant sensory and emotional experience related to actual or potential tissue damage, subjective experience

**Pharmacological treatments: how the modulate chronic pain pathways**

Prostaglandin production.

- Prostaglandins
  - Thromboxane A₂ - vasoconstriction
  - Prostaglandin PGI₂ - cause arteriolar vasodilation
  - Prostaglandines E₂, F₂, D₂ -
  - PGE₂ and PGI₂ have the majority of effects on pain mechanisms
- Tissue injury results in the release of mediators of the inflammatory process. Some are thought to directly activate nociceptors, others act indirectly on mast cells, macrophages, other inflammatory cells to enhance the release of bradykinin, serotonin, histamine.

Variety of receptors identified in dorsal root ganglia and more distally in peripheral nociceptive nerve fibres. Including: PGE₂, PGD₂ and PGI₂. COX is expressed in inflammatory cells, dorsal root ganglia and in the spinal cord. Animal models have shown upregulation of COX and hence increased levels of PGs following injury. NSAIDs are thought to attenuate the pain experienced with inflammatory conditions.
- NSAIDs work by inhibiting prostaglandin production from arachidonic acid by inhibiting the enzyme cyclooxygenase (COX).

There are three different ways COX inhibition can occur:
- **Irreversible inhibition** – e.g. aspirin causes acetylation of the active site
- **Competitive inhibition** – e.g. ibuprofen acts as a competitive substrate
- **Reversible (non-competitive) inhibition** – e.g. Paracetamol has a free radical trapping action that interferes with the production of hydroperoxidases, which are believed to have an essential role in COX activity

**WHO ladder of pain**
Non pharmacological pain treatments: mechanisms by which they modify the perception of pain

Mechanisms of Non-Pharmacological Pain Relief

- Non pharmacological techniques interfere with the pain gate to alter pain sensation
- Mechanism is not clearly established, suggested mechanism involves:
  - Stimulation of larger nerve fibres near area of pain
  - Causing pain gates to close, release of endogenous opiates and pain relieving hormones
  - Massage thought to work using touch to stimulation large nerve fibres
  - This overpowers sensation of pain
  - Transcutaneous nerve stimulation (TENS) emits low-voltage electrical impulses of various frequency and intensity
  - This travels through the skin and stimulates large nerve fibres
  - Acupuncture acts on Chi – energy flowing within human bodies
  - It aims to unblock/rebalance
  - It also stimulates larger nerve fibres to close pain gates and alter the perception of pain

Psychology of pain

- **Unpleasant sensory** and emotional experience associated with actual or potential tissue damage or described in terms of such damage.
- **Acute pain**: tissue damage/painful stimuli. Adaptive. Alerts us of behaviour causing pain. More positive outlook therefore patients have a better coping strategy.
- **Chronic pain**: continuation of pain (> 3 months). No protective role. Associated with functional, psychological, social problems. Powerless feeling and patients have negative outlook. Pain dominates life.
- **Negative pain cycle**: Pain – disability – distress – pain, etc. Disruption = learn adaptive behaviours; identify triggers and factors reducing pain and relaxation.
- **Lifestyle/behavioural changes (CBT)** = Biggest determinant of success in chronic pain management.

<table>
<thead>
<tr>
<th>Pain Perception</th>
<th>Pain Behaviour</th>
<th>CBT=Cognitive Behavioural Techniques</th>
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<td>Patient’s mood</td>
<td>Operant conditioning</td>
<td>Education</td>
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<tr>
<td>Motivation</td>
<td>Classical conditioning</td>
<td>Distraction</td>
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<td>Cultural/Social environment</td>
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<tr>
<td>Personality</td>
<td>Function of pain</td>
<td>Music</td>
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<tr>
<td>Duration of pain</td>
<td>Inform</td>
<td>Stress/anger management</td>
</tr>
<tr>
<td>Extent of damage</td>
<td>Learn</td>
<td>Coping skills</td>
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<tr>
<td>Validation or not of pain</td>
<td>Protect</td>
<td>Financial help</td>
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<tr>
<td>Previous pain</td>
<td>Stop causative agent</td>
<td>Acupuncture</td>
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<tr>
<td></td>
<td>Rest</td>
<td>Improving social/occupational identity</td>
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<tr>
<td></td>
<td>Recover</td>
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</tbody>
</table>

**Referred pain: musculoskeletal mechanism**

- Referred pain is pain that is felt at somewhere other than the site of nociceptive focus. It can be referred from viscera, nerve, muscle, or bones and joints.
- **Visceral**: referred pain - same spinal nerve roots supply both visceral and somatic structures, brain misinterprets direct visceral stimulus as somatic nociception. Sensory neurons branch – one nerve serves both somatic and visceral structures, so 1 nociceptor can have receptors in skin and muscle/viscera.
- Eg: heart: T1-5 reached by nociceptive stimuli travelling along afferent nerves in association with sympathetic nerves from viscera. Dermatomes with T1-5: left harm, hand & jaw
  - Sensation: dull.

- **Radicular**: pain originates from damage to/entrapment of the nerve/root (eg, fractures, herniated disc, osteophytes, tumours, or inflammation/ischaemia along the neuron itself); pain radiates to dermatomes served by that nerve/root.
  - Eg: C6 and tingling in thumb.
  - Sensation: stabbing.

- **Myofascial**: voluntary muscle tenses & shortens; pain; or stretching: pain. Trigger points: taught nodular bands - painful twitching with pressure. Mechanism unknown, perhaps deep muscle lesion or nerve lesion affecting myotome. Tender pain.
  - Sensation: stabbing.

- **Phantom**: peripheral nerve damage from trauma/amputation: neuropathic. Demyelination can cause hyperexcitability, or pain from death of nerves in dorsal ganglia. C fibres (slow: aches) can join to cutaneous sensory fibres with low thresholds causing persistent pain.
  
  NB: Possible mechanism for hip pain referred to knee: afferent neurons from different site converging at the same spinal level.

**Pharmacology**

**Ibuprofen and paracetamol: why they are effective in treating pyrexia**

- Pyrexia: increase in core temp set point in preoptic area and anterior hypothalamus, mediated by release of PGE2 in the hypothalamus, hypothalamus thinks core temp too high and tries to reduce temp, cold and shivering after fever.
- Ibuprofen: NSAID, inhibits COX1 and 2 (competitive inhibition), stops conversion of aa to prostaglandins, set point restored
  - COX1: constitutive, blood flow and GI protection, inhibition does not reduce fever.
  - COX2: induced at sites of inflammation but constitutive in brain, kidney and colon, inhibition does reduce fever.
- Paracetamol: reversible non-competitive inhibition of COX, free radical trapping action.
- Other treatments: aspirin (beware reyes in children), tepid water, cool room, and regular fluid intake.

**Antibiotics: general mechanisms at molecular, cellular and tissue level. Different groups**

- **Cell wall synthesis inhibitors**
  - Penicillins: β lactam ring, inhibit cross linking of peptidoglycan, act as structural analogues to cell wall precursors, cause cell lysis, amoxicillin is broad spectrum, flucloxacinillin for ESBL staph, hypersensitivity is main side effect
  - Cephalosporins: β lactam ring similar mode of action to penicillins, broad spectrum 2nd choice, 3 generations of drugs, inactivated by β lactamase enzyme, hypersensitivity linked to that of penicillins
  - Glycopeptides: binds cell wall precursors and prevents cross linking enzyme from binding substrate (lower resistance), mainly given IV e.g. vancomycin for MRSA and C.difficile

- **Bacterial nucleic acid inhibitors**
  - Antifolates: sulphonamides (sulphadiazine) inhibit dihydrofolate synthetase, trimethoprim inhibits dihydrofolate reductase, folate needed to make purine bases for DNA, commonly used for UTIs, teratogenic risk during pregnancy,
  - Quinolones: inhibit DNA gyrase (unwinds DNA during replication), ciprofloxacin (broad spectrum) and nalidixic acid (gram –ive),
  - Rifampicin: inhibits RNA polymerase, 1st line use for TB, can cause orange urine and hepatotoxicity

- **Protein synthesis inhibitors**
  - Aminoglycosides: bind irreversibly to 30S and cause misreading of mRNA, gentamicin and streptomycin, can induce ototoxicity and nephrotoxicity at high levels.
  - Tetracyclines: bind reversibly to 30S and inhibit binding of aa -tRNA, oral uptake inhibited by calcium (milk), can depress bone growth and permanently discolour teeth
  - Chloramphenicol: inhibits peptidyl transefease activity of 50S, toxic so only used for life threatening infections, blood monitor required for anaemia, neutropenia and thrombocytopenia
  - Macrolides: bind 50S and prevent translocation of ribosome along the mRNA, erythromycin (staph and C.difficile), clarithromycin (hib and H.pylori), azithromycin (single dose to treat chlamydia)
Proton pump inhibitors: mechanisms at a molecular, cellular, tissue, and organ level

- Proton pump: H/K ATPase pump present on the membrane surface of parietal cells on the lining of the stomach, pumps H+ out into the stomach and pumps K+ into the cell at the expense of ATP. ACh and gastrin stimulate parietal cells to secrete H+, they also stimulate secretion of histamine which binds H2 receptors on parietal cells to stimulate H+ secretion.

- PPI: irreversibly inhibit H/K ATPase pumps, inactive prodrug, converted to sulphonamide in low pH of stomach, bind irreversibly with thiol groups of ATPase (break up disulphide bridges and denature protein), inhibition is highly specific and localised, less acid secreted into stomach, less degradation of mucosal lining, peptic ulcers can heal.

- Indicated for peptic ulcers, H.pylori infection, GORD, oesophagitis and Zollinger Ellison syndrome, usually given in combination with 1 or 2 antibiotics.

Paracetamol: mechanism as analgesic at a molecular, cellular, tissue, and organ level

- Reversible non competitive inhibitor of COX1 and COX2, free radical trapping action that disrupts production of hydroperoxidases which plays an essential role in COX activity, metabolised in liver.

- COX 1 constitutive in most tissues, gastric mucosal lining, COX 2 induced at sites of inflammation, analgesic effect largely due to COX 2 inhibition.

- PLA2 converts membrane phospholipid into arachidonic acid, arachidonic acid is converted into PGs (inflammation, sensitise nociceptive fibres to bradykinin and histamine, amplification of pain message), reduced PG synth in CNS so also reduces neuropathic pain.

- Toxic doses >10g can induce fatal hepatotoxicity, conjugating enzymes becomes saturated, drug then gets converted by a mixed function oxidase to imine metabolite, imine reacts with cell proteins and causes necrosis, can be treated with cysteine or methionine if given early (increases glutathione).

Morphine: mechanisms as an analgesic at a molecular, cellular, tissue, and organ level

- Activation of nociceptors in peripheral tissue: thermal, chemical or mechanical stimuli activate afferent fibres Aδ (fast myelinated) and C (slow partially myelinated), pain signal transmitted along primary afferent neurone to dorsal horn of spinal cord.

- Dorsal horn: relay centre that transmits periphery pain signal to brain via relay neurons in the spinothalamic tract, pain signal transmitted by substance P, bradykinin and glutamate, signal is inhibited by local interneurones (release opioid peptides), descending noradrenaline and serotonin fibres from brainstem (activated by opioid peptides).

- Spinothalamic tracts: send pain signals up to the thalamus where they are processed and distributed to the relevant areas of the cerebral cortex.

- Opioid receptors: μ (responsible for most analgesic effects of opioids), δ (in periphery) κ (spinal level, don’t contribute to physical dependence), activation (inhibition of G protein coupled adenyl cyclase therefore less cAMP production, inhibition of voltage gated Ca2+ ion channel opening, activation of K+ channel opening resulting in hyperpolarisation)

- Morphine is an opioid analgesic: agonist action on opioid receptors, results in pain relief and feeling of euphoria, indications (chronic and acute pain)

- Spinal level: inhibits transmission of nociceptive impulses in the dorsal horn.

- Brain level: see opioid receptor above

- Side effects: respiratory depression (decreases sensitivity of respiratory centre to pCO2), nausea and vomiting, skin rash (local release of histamine from mast cells on skin surface near site of injection), constipation (reduced smooth muscle contraction resulting in reduced peristalsis and gastric emptying).
**NSAIDs: pharmacology and side effects**

**NSAIDs Pharmacology**
- **Effects:** Anti-pyretic, Anti-inflammatory, Analgesic, Anti-platelet
- **Reversible competitive inhibitors**
- **Inhibit hydroperoxidase → cyclooxygenase (COX1 & COX2) → arachidonic acid → no prostaglandin synthesis (PGE2, PGF2a, PGD2)**
- **Reduction in pain/inflammation/fever**
  - Damaged cells produce PGE2 → increased sensitivity of nociceptors to pain producing chemicals (bradykinnins)
  - In hypothalamus inflammatory stimuli (e.g. TNF-alpha) = ↑ COX-2 transcription and translation → ↑ production of PGE2 → ↑ temperature set point → increases body temperature
- **Action is peripheral & at site of tissue damage; don’t remove the original insult**
- **Coxibs used in high risk patients**
  - > 65 years, previous peptic ulcer, on current gastric damaging drugs
  - ↓ GI side-effects, equal analgesic effects but ↑ cardiovascular, don’t affect platelet adhesion

**NSAIDs GI Effects**
- **COX 1 important in maintenance of normal mucosal function, protection by regulating blood flow, mucus and bicarbonate secretion**
  - inhibition = ↓ protective function → promoting damaging factors such as gastric acid → ulcers
- **Direct damage → acidic in nature → a direct corrosive effect**
  - absorbed across gastric mucosa → trapped in epithelial cells
- **Uncouple mitochondrial oxidative phosphorylation → impair cell function → damage stomach epithelium**
- **Anti-platelet function → prolonged bleeding time**
Analgesics: how they work, contraindications

Analgesia = absence of pain

**NSAIDs**
- Reduce pain, stiffness, and inflammation of conditions affecting bone, muscles and joints. (OA, RA etc)
- Response to drugs from these drugs varies between individuals and they do not change progress of the disease.

**Opioids**
- Reduces pain by acting on central nervous system and gastrointestinal tract
- Treats acute pain (e.g. post-operative pain), palliative care and more recently in the management of non-malignant chronic pain.

**Paracetamol**
- mild to moderate pain, pyrexia

**Mode of action:**
- Block COX-1
- Inhibits PG production
- Reduces pain and inflammation
- Blocking COX-2 results in anti-inflammatory effect.

- Bind to opioid receptors in CNS and other tissue
- Effect depends on which receptors they bind to
- Essentially it is due to reduced perception of pain, and reaction to pain and increased tolerance to pain

**Side-effects/Contraindications:**
- Peptic ulcerations
- Gastritis
- Renal Toxicity: acute + chronic
- COX-2 contraindicated with ischaemic heart disease or stroke
- Used with caution in elderly, pregnancy, breast feeding and coagulation defects

- Constipation
- Respiratory depression
- Sedation
- Nausea
- Vomiting
- Compromise immune system – decreases proliferation of macrophages.

- Side effects are rare but rashes, thrombocytopenia
- Interactions with anticoagulants (e.g. warfarin)

**Steroids: indications and common side in patients with musculoskeletal disease**

May be taken orally or injected locally in M/S disease.

**Indications in M/S disease**
- Treatment reserved for specific indications, e.g. when other anti-inflammatory drugs (eg NSAIDS) are unsuccessful.
- Also for acute flares where symptoms are too severe to wait for DMARD therapy to take effect (12 weeks).
- Can be used in conjunction with DMARDS or when trying to gradually reduce steroid dose.
- Steroids should be given for the shortest period of time with the lowest possible dose. They should only be given if their effect outweighs their side effects.
- Steroid replacement therapy (oral): adrenocortical insufficiency from long-term steroid use (ie somebody cannot be weaned off them completely).
- Systemic inflammation (oral): RA, SLE, polymyositis. 7.5 mg daily may reduce the rate of joint destruction in moderate to severe rheumatoid arthritis of less than 2 years’ duration
- Local inflammation (injections): eg: OA, ReA.

**Common side effect in pts with M/S disease**
- Osteoporosis – this is the main one and prophylactic treatment of bisphosphonates is indicated in those on high doses, long term treatment, elderly.
• Corticosteroids decrease the amount of calcium absorbed by the intestine & increase calcium excretion through the kidneys so PTH increases, so more bone resorption.
• Also decrease oestrogen & testosterone resulting in bone loss.
• Steroid muscle weakness – inactivity – bone loss.
• Osteoblasts inhibited – loss of trabecular bone (eg in spine).

Other side effects
• Hunger, weight gain, Easy bruising (purpura), thin skin, Infections
• High doses of corticosteroids can cause Cushing’s syndrome, with moon face, striae, and acne.

Side effects: how to discuss side effects of steroids with a patient starting new treatment

Side Effects:
1. Carry an ID card at all times with the name and dose of the medication
2. Lowered ability to fight infections so avoid with people who have colds or infections even chickenpox, shingles, or measles
3. Call the doctor if you notice signs of infection like fever, sore throat, rash, or chills.
4. Do not receive any live vaccines (e.g., measles, mumps, smallpox) while you are on steroids and talk to your doctor before you receive any vaccine
5. Tell your dentist that you take steroids before you receive any medical or dental care, emergency care, or surgery
6. If you have Diabetes this medication may affect your blood sugars; check blood sugar levels closely
7. Always tell the doctor if the dose of your diabetes medicine changes
8. Blood tests from time to time, keep your appointments
9. You may experience some of the following common side effects:
   a. Acne
   b. clumsiness; dizziness
   c. facial flushing; feeling of a whirling motion
   d. general body discomfort
   e. headache; increased appetite
   f. increased sweating; nausea
   g. Nervousness; sleeplessness; upset stomach.

   Let your doctor know if these persist or get worse

10. Seek urgent medical attention if you experience
   a. a rash; hives; itching; difficulty breathing; tightness in the chest; swelling of the mouth, face, lips, or tongue
   This is a severe allergic reaction
   b. vomit that looks like coffee grounds
   c. seizures

11. Inform us as soon as possible about:
   a. black, tarry stools
   b. Unusual weight gain
   c. changes in menstrual period; changes in skin color
   d. chest pain
   e. easy bruising or bleeding
   f. increased hunger, thirst, or urination
   g. mental or mood changes (e.g., depression)
   h. muscle pain, weakness, or wasting
   i. severe nausea or vomiting
   j. shortness of breath; signs of infection (e.g., fever, chills, persistent sore throat)
   k. sudden severe dizziness or headache
   l. swelling of ankles, feet, or hands
   m. tendon or bone pain
MORE IF NEEDED:
Advice on how to take steroids:
1. with food
2. strict on directions of how often to take
3. Do not miss any doses, but if you do, take it as soon as possible
   If it is almost time for your next dose, skip the missed dose and go back to your regular dosing schedule.
   Never take 2 doses at once.
4. Avoid alcohol

Lipid metabolism: factors affecting lipid metabolism and management: both pharmacologically and with lifestyle
- FAs are broken down by oxidation to produce ATP for energy.
- Excess FAs get stored in adipose cells by lipogenesis.
- Dietary intake: sat FAs, trans fats, cholesterol. Unsat FAs lower LDL and raise HDL
- Exercise: raises HDL and lowers LDL
- Smoking raises the level of FAs in blood stream
- Alcohol: increases fat accumulation in the liver
- Genetics: ApoE4,3,2 and familial Hyperlipidaemia
- Cholesterol: transported by LDL and HDL, adipose tissue, liver makes cholesterol from Acetyl CoA and HMG CoA reductase is the rate limiting step. Is excreted in bile and reabsorbed in the small intestine through Niemann Pick cells on the epithelial lining.
- Pharmacological treatment: statins, ezetimibe, fibrates (PPAR alpha agonist), nicotinic acid, bile salts (bind bile acid in intestine, resulting reduced bile acid leads to increased conversion of cholesterol to bile), orlistat (fatty acid chelator)

Physiology
Homeostatic control
- Homeostasis is a regulatory device that maintains a constant internal physiological state in the body
- Sensor detects change, info relayed to integrating centre (eg brain or spinal cord), integrating centre evaluates info, integrating centre increases or decreases activity of effectors.
- Thermo regulation (fever) and blood glucose control (diabetes)
- Blood glucose: rise in glc, beta cells in pancreas release insulin, liver converts glc to glycogen and stops glycogen breakdown, muscles and liver take up more glc from blood, glucagons release is inhibited blood glucose drops, alpha cells in pancreas release glucagons, glycogen is broken down in liver, glc is released by liver, blood glc rises.
- Blood pressure: Increase detected by Baroreceptors in carotid sinus detect arterial wall stretch> signal impulse to cardiovascular centre of medulla oblongata in brain>impulse to smooth muscles of arteries causing vasodilation, impulses to sinoatrial node (SAN) in heart to decrease cardiac output> reduction in blood pressure.
- Body Temperature: Cooler temp. Detected by thermoreceptors> impulses sent to thermoregulatory centre in hypothalamus> impulse to skeletal muscles initiates contraction causing shivering> Causes increase in resp. Rate and an increase in rate of heat production.
Hormones: mechanisms of action including thyroxine, adrenaline and the combined contraceptive pill

- Thyroxine: TRH produced in hypothalamus travels to the pituitary, TSH produced in pituitary, TSH released into systemic circulation and travels to thyroid, iodine uptake stimulates production and release of T4 and T3, T4 and T3 exert negative feedback on hypothalamus and pituitary, T4 and T3 bind protein carrier and enter circulation, enter target cell via diffusion through cell membrane (hydrophobic in nature), all T4 converted to T3 in cytoplasm, T3 enters nucleus through pores and binds nuclear receptor proteins, binds hormone response elements on certain genes, transcription and translation of proteins.

- Adrenaline: activates G protein linked receptors (fast signal transduction), adrenaline binds receptor and induces conformational change, affinity for $\alpha_\beta_\gamma$ trimer increased and GDP dissociates from $\alpha$ subunit, GTP binds $\alpha$ subunit which then dissociates from $\beta_\gamma$ dimer, $\alpha$ subunit-GTP is active form of G protein bind Ca$^2+$ channels (effects many cellular changes), or PLC (converts PIP2 to IP3 and DAG (regulation of enzyme activity)

- Normal ovulation: hypothalamus secretes GnRH, GnRH stimulates release of LH and FSH from pituitary, LH and FSH stimulate release of oestrogen and progesterone from ovaries, negative feedback of hypothalamus and pituitary by oestrogen.

- Combined pill: binds to oestrogen and progesterone receptors, slows release of GnRH, release of FSH and LH inhibited, graafian follicle does not develop and egg is not released from ovary

Physiology of reflexes

- A reflex is an automatic response intended to protect an organism from a harmful stimulus.
- Common examples include; pupil dilation with response to light, salivating at the sight of food and the knee jerk reflex.
- Reflexes are controlled by dorsal root ganglia.
- Simple reflexes are innate but more complex reflexes can be acquired.
- Reflexes can either be monosynaptic or polysynaptic.
- Most reflexes are processed in the spinal cord (although some are processed in the hypothalamus and medulla in the brain), these are called spinal reflexes.
- The process of a spinal reflex is as follows;
  - Hand touches hot plate (stimulus)
  - Heat receptors and nociceptors detect stimulus
  - Generator potentials are produced and an action potential is initiated in the sensory neurone.
  - Sensory neurone carries impulses into the spinal cord (coordinator) via dorsal (posterior) root
  - Sensory neurone synapses with relay neurone
  - Relay neurone synapses with motor neurone
  - Motor neurone carries impulses out of spinal cord via ventral (anterior) root
  - Impulses are carried to the muscles (effector)
  - Motor neurone stimulate release of calcium ions resulting in muscle contraction
  - Hand is moved away from the hot plate

- If the response triggered by the effector causes a decrease in the magnitude of the stimulus that triggered the sequence of events, then the reflex leads to negative feedback.

Synovial fluid, how its analysis may be helpful in the management of disease

Synovial Fluid: Normal and Abnormal

Normal:
- Has a high viscosuity
- A clear plasma filtrate
- Contains
  - Hyaluronic acid
- glycoproteins
- electrolytes (same concentration as blood electrolyte)
- 95% water
- Produced into joint space by synovial membrane under pressure
  - This is a few cells thick
  - Has a smooth, non-adherent surface
  - Has type A and type B cells
    - Type A are phagocytic
    - Type B produce synovial fluid
- Under pressure SF is squeezed out and diffuses into the joint space
- Approximately 2ml in a synovial joint
- Cushions the cartilage, minimise friction
- Hyaluronic acid lubricates the joint space and provides nutrition to the avascular hyaline cartilage

### Abnormal:

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>OA</th>
<th>RA</th>
<th>Septic</th>
<th>Gout</th>
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<tbody>
<tr>
<td>Appearance</td>
<td>Clear</td>
<td>Clear</td>
<td>Turbid</td>
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<tr>
<td>WBC/ml</td>
<td>100</td>
<td>1000</td>
<td>30000</td>
<td>100 000</td>
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<td>Neutrophils</td>
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<tr>
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<td>No</td>
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<td>Viscosity</td>
<td>Normal</td>
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<td>Low</td>
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<td>Neg</td>
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<td>Glucose</td>
<td>Serum</td>
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<td>Low</td>
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### Pregnancy and Childcare

**MDT: care of the pregnant woman: hospital and community**

- Psychosocial model: pregnancy is normal until (if) pathology occurs. Biomedical model: pregnancy is only normal in retrospect and is risky.
- Community:
  - GP role: confirm pregnancy by urine test, completes a booking form that is passed to the hospital, refers pt to midwife, Rx any drugs the pt needs during pregnancy.
  - Midwife: advice on home birth, consent for antenatal screening, blood tests, weight, height, referral to obs if needed, delivery (except if complications), initial examination after birth, postnatal monitoring (4 weeks)
- Health visitor: qualified nurses experienced in child health, health promotion and education. The health visitor carries out postnatal care of the mother (10-15 days after the birth).
- Hospital:
  - Sonographer: using ultrasound to produce diagnostic images, scans and videos to monitor the growth and development of the foetus.
  - Obstetrician: assumes care for pt if any abnormalities detected, pt with past obs Hx of complications, pre eclampsia, gestational diabetes, obs cholestasis (bile build up in blood), breech births, caesarean
  - Virologist (if pt HIV positive)
  - Paediatrician: carry out the first medical examination of the newborn baby, primarily checking the heart for any cardiac abnormality.
- Conc: women are often in the community when they discover they’re pregnant. Many normal pregnancies are seen through in the community, with actual birth taking place in hospital (70 % in ‘95). Any deviations from the norm prompt hospitalisation during pregnancy as well as birth. So it’s teamwork between community AND hospital practitioners.
Tests carried out during a normal pregnancy

Nullip = Nulliparous

<table>
<thead>
<tr>
<th>WEEKS</th>
<th>10</th>
<th>16</th>
<th>18-20</th>
<th>25 Nullip</th>
<th>28</th>
<th>31 Nullip</th>
<th>34</th>
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<td>BP</td>
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<td>Weight + BMI ([kg]/[m]^2)</td>
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<td>X</td>
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<tr>
<th>Urine</th>
<th>Proteinuria</th>
<th>Bacteriuria</th>
<th>Prot</th>
<th>Prot</th>
<th>Prot</th>
<th>Prot</th>
<th>Prot</th>
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<tr>
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<td>Triple test:</td>
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<tr>
<td></td>
<td>In Down’s</td>
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<td>α foetal protein ↓</td>
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<td>estriol ↓</td>
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<td>X</td>
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<tr>
<td>Symphysis -Fundal Height</td>
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<table>
<thead>
<tr>
<th>Other</th>
<th>Diabetes screening if risk factors present</th>
<th>Amniocent</th>
<th>Diabetes check</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Offer Down Syndrome Screening b/w wk 11-13; nuchal translucency test; beta hCG; plasma protein A</td>
<td></td>
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<tr>
<td></td>
<td>Measure blood pressure and test urine for proteinuria: offer screening for asymptomatic bacteriuria</td>
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<tr>
<td></td>
<td>Offer blood tests to check blood group and rhesus D status, and screening for: anaemia, haemoglobinopathies-Thalassaemia and sickle cell, red-cell alloantibodies, hepatitis B virus, HIV, rubella susceptibility and syphilis.</td>
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<td></td>
<td>Inform women younger than 25 years about the high prevalence of Chlamydia infection in their age group, and give details of their local National Chlamydia Screening Programme.</td>
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<tr>
<td></td>
<td>Offer screening for Down's syndrome. The 'combined test' (nuchal translucency, beta-human chorionic gonadotrophin, pregnancy-associated plasma protein-A) should be offered to screen for Down's syndrome between 11 weeks 0 days and 13 weeks 6 days. For women who book later in pregnancy the most clinically and cost-effective serum screening test (triple or quadruple test) should be offered between 15 weeks 0 days and 20 weeks 0 days.</td>
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<tr>
<td></td>
<td>Offer early ultrasound scan for gestational age assessment and ultrasound screening for structural anomalies</td>
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</tbody>
</table>

25 weeks – for nulliparous women
- Measure blood pressure and test urine for proteinuria. 
- Measure and plot symphysis–fundal height.

28 weeks
- Measure blood pressure and test urine for proteinuria. 
- Offer a second screening for anaemia and atypical red-cell alloantibodies. 
- Investigate a haemoglobin level below 10.5 g/100 ml and consider 
- Iron supplements. Offer anti-D prophylaxis to women who are rhesus D-negative1. 
- Measure and plot symphysis–fundal height

31 weeks – for nulliparous women
- Measure blood pressure and test urine for proteinuria. 
- Measure and plot symphysis–fundal height.
• 34 weeks
  o Measure blood pressure and test urine for proteinuria.
  o Offer a second dose of anti-D prophylaxis to women who are rhesus D-negative.
  o Measure and plot symphysis–fundal height.

How the mother can ensure optimal development from the sperm and the ovum to the full-term baby: dietary intake, alcohol, and smoking
• Balanced diet: protein, carbs, fruit and veg, dairy, restrict foods high in fat and sugar, avoid liver (vit A)
• Folic acid 400 micrograms per day for first 12 weeks, iron supplements if anaemic, food containing vit C (absorb dietary iron) and calcium (bones development)
• Vit D: 10micrograms per day during pregnancy and breastfeeding
• Warnings: undercooked meat (toxoplasmosis and salmonella), unwashed fruit and veg, mould ripened soft cheese (listeria), liver (vit A), Tuna (mercury), peanuts (allergies), restrict caffeine to 200mg per day
• Smoking: ischaemia and increased heart rate, underweight, preterm delivery, inc risk of stillbirth, bronchitis and asthma. Give details on using smoking cessastion services.
• Alcohol: foetal alcohol syndrome (brain damage) and preterm delivery, 2 units per week no increased risk
• Drugs: paracetamol, most antibiotics, nicotine replacement usuallu safe. Illegal drugs not safe.
• Exercise: non strenuous, drink water and plenty fluids. Sports that cause abdominal trauma e.g. scuba diving should be avoided.
• Complimentary therapies: advise women that few complementary therapies have been proven as being safe and effective during pregnancy
• Sexual intercourse: reassure women that intercourse is thought to be safe during pregnancy.

Unplanned pregnancy: cultural and religious, impact on decision making
• Less home births, more women starting family in 30s, rise in rate of abortions since the abortion act in 1967, use of contraceptive pill and morning after pill, detection of foetal abnormalities since the 1970s, IVF, higher birthrates among ethnic minorities and immigrant populations, highest teenage pregnancy rates in Europe (low SE background, poor sex education)
• Hindus: soul enters baby in 7th month (ritual to mark this), burial of placenta, female friend may wash mothers breasts, jatakarma ritual performed immediately after birth, no ruling on contraception, abortion forbidden except when life saving.
• Islam: female Dr and midwife preferred, breast feeding encouraged, baby must be washed before being held, irreversible contraception and abortion forbidden (except for genetic disorders)
• Roman catholic: placenta discarded, abortion and contraception unacceptable, child christened after birth
• Judaism: males circumcised after 8 days, abortion only acceptable if danger to mother (consult with rabbi).

Discuss the concept of human life as a dynamic process that starts with fertilisation and ends with death, outlining aspects of the life cycle and discussing the concept of social gerontology.
• Infant/Child 0-2 months; rapid growth (length up by 50 % weight by 300 %), sleep cycles, first smile. 2-5 months vocalisation (moms influencing social development), 5-8 months move away from egocentric to notice people and objects. 8-18 months kids form associations ie crying = attention. 18 + months development of speech, letter & shape recognition.
• School child; beyond age 2-3, until puberty, the growth rate is steady at approx. 3-3.5kg and 6cm/year. Appropriate behaviours learnt, language & social skills.
• Young adulthood: 18-39, peak strength, marriage, careers, responsibilities for families (dependent parents & kids), marital probs, single parenthood.

• Middle age: 40-60 onset of midlife crisis for some men – last chance to achieve goals, menopause for women – loss of bone density and for some, sexuality. Decline in senses.

• 65-74: young old, 75-84 old, 85-99 old old, 100+ oldest old.

• Social gerontology: multi-disciplinary sub-field that specialises in studying or working with older adults by social workers/nurses/psychologists etc.

• Many elderlies live independent & mobile lives, some don’t.

• Myths/stereotypes by health pros can affect self esteem & independence of elderlies.

• Recall may take longer; not necessarily less intelligent!

• Sexual relationships often limited, especially for women in care homes (widowed/divorced/naughty independent men go for younger women). Also societal taboos.

• Elderlies more likely poor, too proud to claim benefits though, despite entitlement.

Social gerontology aims: educate, research and advance causes for older people.

Describe psychological barriers to behavioural change, especially when pregnant, including treatment adherence, techniques for facilitating lifestyle changes for healthier living, and treatment compliance. Discuss how physical and mental factors interact in long-term conditions.

• Barriers to change: Anxiety and paranoia, Misconception (media, family, friends), resistance to lifestyle change (trivialisation and denial), impairment (hearing, speech, visual), communication barriers (cultural, jargon), Dr Pt relationship (lack of empathy, hurried consultation), culture and religion (catholics non use of contraception)

• Assisting lifestyle changes: Health education (diet, lifestyle, benefits and consequences), social support (family, friends), psychology (cognitive behavioural therapy)

• Cognitive dissonance theory: being aware of two opposing beliefs/attitudes causes an aversive psychological states (hypocrisy condition), resulting in a change in attitude

• Central route processing: when a lengthy and detailed message is put forward to an already knowledgeable audience. This is more likely to effect a permanent change as a lengthy process of thought is required.

• Peripheral route processing: Where a simple message is put to an audience and a judgement is made based on the credibility and attractiveness of the message. This process is less likely to affect a long term change.

• Behaviour change strategies: self monitoring (diary, weighing scales), control env (avoid pub if alcoholic), set SMART goals, self reward, social support, make gradual changes

Psychology

Psychological barriers to behavioural change, techniques for facilitating lifestyle changes for healthier living

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Unexplained symptoms: patient and doctors perspective, potential for psychological factors to contribute to: illness, course of disease and success of treatment

- These disorders are important because they are common and they cause similar levels of disability as symptoms caused by disease.
- If not treated properly they can result in large amounts of resources being wasted and iatrogenic harm.
- This is a clinically, conceptually, and emotionally difficult area.
- Clinical presentations vary greatly—from people who frequently attend the general practitioner with minor symptoms to people with chronic fatigue who are bed bound. What unites them, however, is the difficulty in explaining the presenting symptoms on the basis of any known pathology.
- Strong feelings are common, with patients often referred to in pejorative terms as “frequent fliers,” “heart sink patients,” “thick folder patients,” or “somatisers.”
- Doctors may feel that their competence is challenged by their inability to explain the symptoms.
- Patients may feel that they are disbelieved and accused of fabricating their symptoms.
- Conceptually, the area is hindered by a dualism that divides causes into physical or psychological and by simplistic aetiological models that rely on a single explanatory factor.
- Medically unexplained symptoms are common. All symptoms should be treated seriously, regardless of cause.
- Explanations should integrate psychological and biological factors and provide patients and doctors with a model for managing the condition.
- Anxiety and depression often present with medically unexplained symptoms.
- Cognitive behaviour therapy is an effective treatment.
- Associated pathology is rare and rarely missed, whereas psychiatric diagnoses are common and often missed.

Unemployment, employment, long-term sickness benefit or retired: sociological and psychological impact on health

Unemployed/Long-term Sickness/Retired: Loss of work is directly correlated with poor physical health, due to decreased income, debt, stress

i) Sociological:
- self-identity and image changes as job and career are a major part of a person’s social role
- loss of social contacts
- lack of purpose
- personal satisfaction from achievement of recognition and social interactions provided at work are lost;
- forced retirement threatens self-image, self-worth and self-respect

ii) Psychological: psych stress can lead to ill health, high rates of chronic illness in those with mental illness
- Social Isolation
- Loneliness, Depression, Anxiety about the future
- Feelings of rejection
- Sedentary lifestyle, poor diet -> addiction, crime
- disorganisation, dependency
- regression (behaviour of young child, clinging, complaining, over demanding, selfish)
- withdrawal

Retirement does not have to be the end of it if you plan for it.

Employed
- Sociological: opposite of everything above
- Psychological: opposite of everything above
Psychological factors: potential to contribute to illness, course of disease & success of treatment

- Dr patient relationship, pt centred treatment, autonomy, motivation and positive mental attitude, rehabilitation, SMART objectives, compliance with treatment, support from friends and family
- Stress, anxiety and depression can result in somatization (muscle pain, stiffness, headaches, fatigue), hyperochondriasis, being a burden on others.
- Stress-coping paradigm: stressful stimulus of illness (harm, loss or threat), coping strategies (seek information, do nothing, take direct action, worry), illness stimulates a cognitive process that generates stress and anxiety, level of stress increases if the patient does not feel in control.
- Control: social networks, family, financial security, education, involvement in management strategy, SMART objectives, promotion of independence, discourage over reliance on others.
- Adaptation strategies: normalising illness to neutralise the treat (compliance problems), denial of illness (eases anxiety in early stages, prevents patient from confronting illness in log run), avoidance of situations that exacerbate symptoms (can lead to social exclusion), resignation (life revolves around the illness), accommodation of illness as part of patients life but not central to it.
- Addiction (compulsion to engage in an activity despite being aware of harmful consequences) and dependence (physical and mental withdrawal symptoms)

Grief process: stages expected, factors that influence the outcome of grief

- Kubler-Ross: denial, anger, bargaining, depression, acceptance, not necessarily involving every stage or in that order.
- Normal grief: feelings (sadness, loneliness, anxiety, shock), physical sensations (hollowness, weakness), cognition (disbelief, sense of presence of deceased), behaviours (loss of appetite/sleep, social withdrawal, crying)
- Abnormal grief reactions: delayed or absent grief (difficulty grieving freely for sake of children, delayed if body not found, can be triggered by a separate loss long after the death), chronic grief (cant move on in the grieving process, usually caused by ambivalent relationship with deceased at time of death), exaggerated grief (excessive and intense, can result in depression or anxiety).
- Risk factors for abnormal grief: relationship with deceased (dislike, over dependence), violent death, not being present before or during the death (guilt), bereaved has experienced previous losses.
- Bereavement care: family and friends, GP, clinical psychologist, bereavement counselling services.
- Surviving spouse: increased risk of all cause mortality in 1st 6 months (accident, illness, suicide, alcohol abuse)

Describe how knowledge of the psychology of illness behavior may help healthcare professionals to treat people with spinal pain

- Psychosocial reasons for failure to recover (Yellow flags): Belief that activity that causes pain is harmful, sickness behaviours such as extended rest and taking to bed, social withdrawal, emotional problems (low mood, depression, anxiety and stress), problems at work or dissatisfaction with work, pending medicolegal claim, overprotective family or lack of support, inappropriate expectations of treatment, including not appreciating the need for active participation in treatment
- Behaviours that close pain gate: Happiness, contentment, sleep, being in control, company, activity, knowledge, realistic expectations
- Pain management approach: MDT, education, cognitive/behavioural therapy, complementary therapy, reduce maladaptive thoughts (reduce anxiety, decreased arousal and tension), encourage appropriate behaviour, challenge misinformation, set SMART objectives, stress management, coping techniques, lifestyle changes, group/family therapy or support groups

Public health

NHS: structure and function

- Equitable and universal health service free at the point of entry and funded by general taxation (ideology of Aneurin Bevan).
- Primary care: frontline, gatekeepers to specialised care. GP, pharmacists, dentist etc.
- Secondary care: acute or specialised care (elective or emergency) in hospitals. Foundation trusts (greater financial freedom to generate income and can retain surpluses e.g. borrow money, buy and sell land)
• Increased privatisation of primary and secondary care
• PCT: controls majority of NHS budget and commissions services to primary and secondary care. The make sure a communities healthcare needs are being met.
• Practice Based Commissioning: allows GPs to design and commission services based on healthcare needs of the area. Report to the Strategic Health Authorities (SHA)
• SHA: 10 across the country, they implement directives and fiscal policy as set down by the department of health. Reports to the Department of Health (DoH)
• NICE: Provides guidance to healthcare professionals on current best practice based on cost efficacy and evidence based medicine
• The healthcare commission: Inspects and audits quality of healthcare services (hospitals mainly) and reports directly to parliament
• DoH: Set NHS legislation and policy, apportion budget from treasury

**Immunisation programme in UK, basis of immunization and vaccination therapy, principles of active and passive immunization**

- 2 months: DTaP/IPV/hib (Diptheria, tetanus, pertussis, polio, haemophilus influenza B) and PCV (pneumococcal conjugate vaccine)
- 3 months: DTaP/IPV/hib and menC
- 4 months: DTaP/IPV/hib and menC and PCV
- 12 months: Hib/MenC
- 13 months: MMR and PCV
- 3 yrs 4 months: DTaP/IPV and MMR
- 13-18 yrs: Td/IPV (diptheria, tetanus, polio) and HPV (females)
- non routine: BCG (TB) for high risk babies, HepB if mother HepB positive.
- Passive immunity: transfer of protective Ab e.g placental, breast milk, last as long as the Ab is present, tetanus Ab after possible exposure or to treat infection, short lived and can induce anaphylaxis
- Active immunity: controlled exposure to small level of pathogen, altered bacterial toxoid, inactivated organism (by heat or chemical alteration, given with adjuvant e.g. alum to enhance immune response), surface protein e.g. polysaccharides involved in invasion of host (don’t always produce immune response, less effective against intracellular pathogens), live vaccine (attenuated and non pathogenic, poor replication, cant escape IS)
- Live vaccine advantages: less need for booster, lifelong immunity, smaller dose, wider herd immunity, closer to normal physiology

**Environmental risks and hazards: impact on health**

- Radiation: UV and γ rays can generate free radicals, damage to cells and DNA, mutations, cancer, antioxidants reduce risk of damage caused by free radicals.
- Toxins: Natural (plants, fungi, bacteria, animals), man made (factory waste, nuclear waste, pesticides, fertilizers, heavy metal bioaccumulation in fish).
- Effects of toxins: liver/renal failure, inflammation of tissue, GI effects.
- Hazards: poor housing, built and natural environment, RTAs, hazards at home (electricity, cooking fires)
- CO poisoning: competes with O2 for binding to haem in erythrocytes, higher binding affinity, hypoxia, death.
- CN poisoning: used in industry, reacts with metalloproteins, inhibits electron transport chain, no energy produced by mitochondria, cell death
- Lead and mercury poisoning: react with thiol groups in proteins
- P450 system: clears toxins and drugs in the body, adds on functional group to make toxin more soluble, toxin then excreted in liver (bile) or kidney (urine)
Sexually transmitted diseases: impact to society, role of prevention, factors that alter the public's perceptions

- HIV/AIDS: increased morbidity and mortality, treatment expensive, contagious through sex and blood, economic impact in Africa, double stigma of ‘STD’ and ‘terminal illness’
- Can lead to infertility and ectopic pregnancy, stigma attached to STIs
- Prevention: barrier contraception, sex education at school and contraception advice at family planning clinic, chlamydia screening program, HPV vaccine program, government TV campaigns, celebrity drs.
- Altering public perception: mass media can raise health issues in public view, promote and challenge stereotypes. This can work both positively or negatively

Wider determinants of health and basic concepts in public health

Public Health
Determinants of health

- Individual
  - Age
  - Gender (women live longer)
  - Genetics (family history, predispositions)
  - Ethnicity (Afro-Caribbean ↓ risks for cancer)

- Environmental
  - Nutrition
  - Exercise
  - Smoking
  - Alcohol
  - Drug use
  - Air, chemical and noise pollution

- Social and Community
  - Access to water and sanitation
  - Access to green spaces
  - Family and social relations
  - Good working conditions
  - Access to medical care (access and use of health care & vaccination)
  - Education

- Economic
  - High financial status = good education, better job conditions, better access to medical care

Public health

- the science of preventing disease, prolonging life and promoting health through organized efforts and informed choices of society, communities and individuals
- Concerned with threats to the overall health of a community based on population health analysis
- The population in question can be as small or large
- Divided into epidemiology, biostatistics and health services
- 2 characteristics
  - Preventative through surveillance and health promotion, not curative
  - Population-level not individual-level health issues
Rheumatoid Arthritis

Long term musculoskeletal complications in patients with RA

- Articular features of RA: Joint pain, stiffness, swelling, acute synovitis moving rapidly from one region to another leading to difficulties with daily activities (combing hair etc.), ulnar deviation of fingers, boutonniere (PIP flexion and DIP hyperextension) and swan-neck deformities (MCP flexion, PIP hyperextension, DIP flexion), radial deviation of the wrist, atlantoaxial subluxation, Z-deformity of thumb, small muscle wastage
- Extra articular features of RA: Rheumatoid nodules, tenosynovitis and bursitis, carpal tunnel syndrome, systemically unwell (general malaise, fever, weight loss & lethargy), anaemia, lung disease (pleural effusions, lung nodules), cardiac disease (pericardial inflammation, pericardial effusion)

Principles behind management of patients: role of MDT, introduction of newer treatments

Management

- Non-pharmacological treatment
  - rest
  - hot baths
  - paraffin wax
  - physiotherapy
- Pain relief with NSAIDs and aspirin
- Corticosteroids - improve pain & swelling
  - prednisolone in early disease
  - can be given intra-articularly to treat local synovitis
  - given IV or IM to treat flare-ups
- Biologics
  - help prevent bone erosions
  - Infliximab
  - Adalimumab
- Surgery
  - reconstructive surgery if destructive arthropathy
- relieve joint pain
- correct deformities
- improve joint function

- **DMARDs**
  - put disease into remission
  - slow-acting (3-6 months)
  - reduce ESR, CRP, and sometime titre of RF & erosions

**Role of MDT**

- **MDT**
  - control pain
  - minimize fatigue
  - maximize functional mobility

- **Rheumatologist**
  - team leader
  - monitoring of disease activity
  - prescribe & monitor drug therapy

- **Orthopaedic surgeon**
  - replace damaged joints
  - surgical synovectomy (removal of inflamed joint tissue)

- **Physiotherapist**
  - exercises

- **Occupational Therapist**
  - provide aids & appliances to assist with activities of daily living

**Resource allocation**

**Screening programmes: explain the justification for those currently offered in the NHS**

NHS national screening is investigating healthy individuals with the object of detecting unreocgnised diseases. Screening acts on 3 levels of prevention:

- **Early detection** of disease where *prognosis is improved* by earlier treatment e.g. breast cancer screening and offering treatment.
- Detection of people at *increased risk* of developing disease where interventions will reduce the risk. E.g. screening for high cholesterol and offering dietary advice.
- Identification of people with *infectious disease* where treatment or other control measures will improve outcome for individual and prevent spread of disease. E.g. health workers for hepatitis B.

The GPs follow set guidelines set out by the **UK National Screening Committee (UK NSC)**. The NSC uses research evidence and the skills of multi-disciplinary expert groups to develop policies for screening. The guidelines for deciding when screening is appropriate drawn up by Wilson and Jugner (1968);

- The condition being screened for should be an important health problem.
- The natural history should be well understood.
- Treatment at an early stage should be of greater benefit than at later stage.
- There should be a detectable early stage.
- The test should be acceptable.
- There should be a suitable, valid test for the early stage.
- Intervals for repeating the test should be determined.
- Treatment at an early stage should be of greater benefit than at later stage.
- There should be a detectable early stage.
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- Intervals for repeating the test should be determined.
- The test should be a suitable, valid test for the early stage.
- There should be adequate health service provision for the extra clinical workload resulting from the screen.
- The costs should be balanced against the benefits.
- The costs should be balanced against the benefits.

Evaluation of a potential screening programme involves consideration of 3 main issues:

- **Feasibility** – how easy it is to organise the population to attend screening, whether the screening is acceptable, whether facilities and resources exist to carry out necessary diagnostic tests.
- **Effectiveness** – extent to which implementing screening programme affects outcomes. Screening programmes need to be tested by RCT’s to avoid bias.
Cost – do not just relate to the implementation of screening programme, but also further diagnostic tests and the cost of treatment. On the other hand absence of screening costs will be incurred by treatment in advanced stages of disease.

Ethics of screening must be taken into account as it is done to a person who is not ill and to someone who has not usually initiated the request for the test. False positives can produce stress and anxiety. Unplanned effects of a positive effect, e.g. diagnostic labelling leading to adapting to sick role. Risks associated with diagnostic tests. False negatives may give false reassurance.

Examples of important screening tests used today include;

Cervical cancer – all women aged 20-64 and whose name appears on GP lists held by health authorities are called. Every 3-5 years.

Breast cancer – women from the age of 50 up to and including 70 years, receive routine invitations for screening. A double-view mammogram is offered at 3-year intervals.

Other screening programmes being researched include those for prostate, ovarian and colorectal cancer.

It is important to remember that there is a potential injustice, in that not all PCT’s/areas of the country offer all of these screening programmes; it is up to the need of the area and the patients that form within it. In the Norwich PCT, the follow screening programmes take place:

Chlamydia Screening Programme
Bowel Cancer screening programme
Cervical Cancer Screening
Breast Cancer Screening
TB screening

Scarcity: in relation to health provision and the necessity for choice/s

- Key principle: equity (of health care, but also living conditions and equity of autonomy).
- Equal treatment of equals (geographic, ethnicity, socio-economic group): horizontal equity. “free at point of delivery, equal access for all”.
- Aim: equality of health outcome, expenditure on health care, access to and utilisation of health care.
- Usually related to need – health need, capacity to benefit, societal burden of disease.
- So set priorities (re: Acheson Report), but this means rationing resources. Not just NHS resources... reduce income inequalities, improve living conditions, educate kids.
- Our Healthier Nation (Government paper) gives guidelines to reduce inequality in health provision specifically.
- But resources don’t match need, therefore role of ethics in prioritizing: causation (risky behavior), fair-innings, capacity to benefit, medical need, triage, socially useful, those with dependants.
- Many choices to be made: National level eg. defence v. Health, sector level – primary vs 2o care; programme level: kids vs elderly, speciality level: hip replacements vs heart bypasses, patient level: screen patients or wait and see.
- Choosing involves foregoing other opportunities to do good (‘Opportunity Costs’). Minimising opportunity cost will ensure we are being efficient with society’s limited resources
- Need to think about treatments and screenings – not everyone will benefit. So economics, best evidence & cost/benefit analysis is important to help make those choices of whom to treat/screen as well as clinical judgement.
- Conc: equity as key principle but MUST consider: needs, equality (smoker vs non smoker for new lungs), cost effectiveness (effectiveness of treatment related to cost) and QALYs – quality life years gained from intervention – in order to make best choices for health provision. Choices made by government, officials, doctors, society, patients (sometimes) and postcode lottery.
Health needs assessment, how resource allocation decisions are made

Health Needs Assessment (HNA):
1. Begin by defining the population and reviewing issues (n, where, ethnic group, diseases, socio-economics)
2. Services already available (costs, effectiveness, price)
3. What the population wants (surveys)
4. Assess capacity to benefit from new services (accessibility, improved mortality and morbidity, decreased costs in the long term)
5. Assess effectiveness and cost of services (compare numbers between PCTs, research, literature review)
6. After implementation: monitor changes (collect morbidity and mortality data)

Resource allocation decided by looking at:
1. Need: What people might benefit from according to HNA (seeing a measurable change in health due to the intervention) E.g. Immunisation, Breast Cancer Screening, TB medication
2. Demand: what people ask for, but might not really need
3. Supply: what is actually provided by the NHS based on HNA

MORE STUFF IF WANTED:
Goals of HNA:
- prioritise resources to reduce health inequalities
- improve pop’s health
- monitor equity in access and type of services

USE OF HNA:
- Improve health
- Health service planning
- Collaboration b/w health authorities (PCTs, DOH, government)
- Priority setting: what to buy and how much
- Monitoring and promoting equity in services

Rationing of healthcare: pros and cons
2 levels of resource allocation
2) Micro allocation- decisions about treatment between patients
3) Macro allocation- decisions over the share of a society’s total resources which are devoted to health and the division of the health care budget between possible uses (Harris 2001)

Pros
• Medical resources are limited, cost effective treatments ensure the resources are used in the most efficient way, minimising opportunity cost
• With scarcity comes a need to decide who receives treatment and who does not
• But while resources are not infinite they are not finite either- they are indefinite. Any budget can be traded off other budget- priorities can be reassessed.
• Demand for a service is not inevitably infinite- rather ‘the amount of free service I determined at the point where customers see no additional benefits to be gained from additional resources to the service in question. This can be at quite modest levels (Harris 2001)

Cons
• How to choose between patients?
• Rationing methods: triage, medical need (specialists opinion), benefit gained, entitlement (tax contributions, war veterans), fair innings (oldest last), causation (risky behaviour, unhealthy lifestyle).
• NICE charged with issuing guidance based on cost efficacy, use of QALY (quality and quantity of life gained from an intervention), used to determine most effective treatment available according to evidence based medicine (most appropriate antihypertensive drugs) also used to determine if a treatment will be available at all (herceptin).
• QALY discriminates against age and previous morbidity/disability before the treatment is required, young and healthy preferred over old and disabled even if treatment can provide the same outcome.
Role of state in protecting and promoting health and preventing disease. Problems with priority setting in health

- DoH: Health/social care policy guidance and publications for the NHS, health improvement advice, immunisation guidance, health clearance guidance for healthcare professionals, infection control guidance (including notifiable diseases), guidance on the use of blood and blood products in the NHS.
- NICE: issue evidence based national guidance on new and existing treatments, disease management and health promotion
- Care quality commission: independent regulator of NHS and social services, monitoring and inspection, issue fines warnings and closures.
- Social services: child protection, fostering and adoption, social security benefits, care for the elderly, support for the disabled.
- NHS: government funded, provider of healthcare to the nation
- Rationing: some benefit to the detriment of others, opportunity cost, most effective use of finite resources

Sociology

Where and why individuals do or do not seek medical attention for acute illness.

Sociological factors, for example, diversity and inequality impact on their health.

Seeking medical attention...

- Symptom iceberg: over 2 weeks period 75% of population will experience 1 or more symptoms of ill health, 33% consult, 33% do nothing 33% self medicate
- Proportion of people with serious symptoms that do not consult 26%, proportion of people with minor symptoms that do consult 11%
- Symptom perception: severity of symptom, familiarity of symptom, duration and frequency of symptom.
- Anxiety about potential serious pathology: PMH, Family Hx, anxiety disorder, mothers dilemma.
- Avoidance of consultation: other things in life more important, wait and see, difficulty communicating seriousness to HCP, access difficulty, app availability at GP surgery, dr-pt relationship, lack of knowledge, perception that dr cant help (ICE), time off work especially if self employed.
- Labelling- potentially negative label and stigma- labels given in a medical diagnosis have significance beyond the medical consultation and into wider society.

Sociological factors...

- Diversity- social stratification- economic/educational deprivation may lead to health care deprivation. Fear of crime- leading to restriction of activity particularly of older people. Ethnic minorities - cultural/language barriers.
- Poverty- certain conditions e.g. respiratory may be more prevalent amongst lower socio economic classes, life expectancy reduced
- Poor housing- older housing, increased heating costs and damp, choice between food or heat- hypothermia or malnutrition results
- Poor mobility- goods (inability to drive/not owning a car), services, social contacts - lack of this leads to isolation
- Inequality- gender can affect long term health due to combined effects of biological, social and cultural influences.

Sociological approaches to the study of health and medicine in relation to class, gender and ethnicity, diversity and inequality

- Decrease in mortality rates across all social classes but increase in the disparity of mortality rates between higher and lower social classes, morbidity rates follow a similar pattern
- Black Report tried to explain reasons why: behavioural (smoking, alcohol, diet and lack of exercise in lower classes), materialist (poor housing, low pay), social selection (class structure acts as a filter and health determines social class), artefactual (health and class are artificial variables and thus any relationship is accidental).
• At all ages men have higher mortality but women higher morbidity, women consult more often than men due to differences in gender and sex, pregnancy and birth, consulting on behalf of children and elderly relatives, more socially acceptable for women to consult and less so for men (gender stereotyping), differences in pain thresholds linked to levels of testosterone and oestrogen/progesterone (raises pain threshold)
• Ethnicity: prejudice and institutional racism (stress and anxiety), language barriers, mistrust of medical profession, poor access and working conditions, preference for traditional medicine, lack of medical resources in poor areas, fear of deportation if illegal immigrant, higher unemployment rates, unequal or inappropriate provision of health services for ethnic groups.

Compare and contrast the sociological impact of a long-term condition on the individual and their family to that of a short-term condition
• Long term condition: Uncertainty (can make plans as symptoms are unpredictable), strained family relationships (role change to patient and carer, sex life, loss of social circles), impairment disability and handicap, unemployment and economic problems, reliance on social security benefits, loss of autonomy, loss of social role and identity, stigma and discrimination.
• Short term condition: self limiting or treatable, if no morbidity then problems are short term and have less of an impact on family dynamics, patient consults with ICE.
• Primary gain (long and short term conditions, illness used as excuse to avoid a problem e.g. sex or work), secondary gain (long term conditions, material benefit, compensation, social security benefits, more than when working), tertiary gain (long term conditions, role change in relationship to pt and carer), secondary losses also due to psychosocial impact of long term illness.

Basic sociological approaches to the study of health and medicine in relation to class, gender and ethnicity
• Social class model of Scambler and Blane splits witho ‘working-class’ and ‘middle-class’.
  - The registrar general splits class into 6 categories: I, Professional, eg doctor; II, Intermediated, eg teacher; IIIN, Skilled non-manual, eg shop assistant; IIIM, Skilled manual, eg bus driver; IV Semi skilled manual, eg postman; V, Unskilled manual, eg cleaner.
  - Mortality rates for all social classes have been falling over the last 100 years. However, the mortality rates for social class I have been decreasing faster than rates for social class V:
  - Social causation: men and women lead different lives, with different social expectations and stereotypes upon them.
  - Why is this? - Cultural/behavioural, Materialist or structuralist, Social selection, Artefactual
• Gender and health: Social scientists make the distinction between sex and gender, whereby ‘sex’ refers to physical and biological diffs. And ‘gender’ refers to the social definitions of how women and men should behave under certain circumstances.
  - At any age men are more likely to die, but women are more likely to be ill
  - Gender diffs. Have a greater impact on health and health care than diffs. In biological sex.
  - Women consult doctors more often than men do.
  - Medical professionals have diff. expectations of and ways of dealing with male and female patients
  - Why is this? – Artefact, Genetic/biological, Social causation
• Ethnicity: Research usually applied to immigrants as place of birth is on death certs.
  - The concept of ‘race’ does not exist in any biologically meaningful way.
  - Ethnicity is a complex concept consisting of the interplay between culture, history, language and so on. We all belong to an ‘ethnicgroup’.
  - Ethnic minority groups are at increased risk of being poorer, principally through the effects of racism and negative discrimination.
  - The major disease and health problems of most minority ethnic groups are the same as for the general population (e.g. coronary heart disease, stroke, cancer).
  - There is evidence of unequal and inappropriate provision of health services for people in ethnic minority groups.
  - Why is this? – artefact, biological, material, cultural
Diversity and inequality: how this impacts on health and disease

Inequality
- Men have a lower life expectancy than women
  - This is not merely a result of the Y chromosome
  - It is a combination of biological, cultural, and sociological factors
- The Acheson Inquiry showed that:
  - Despite the death rate falling across all social groups, mortality rates between the top and bottom of social scale have widened
  - Mortality is decreasing amongst higher social classes faster than it is decreasing amongst lower social classes
  - Men in the lowest social classes were twice as likely to be dependent on alcohol than those in the highest classes
  - Addiction in a family increase the risk of abuse
- Children from manual households are more likely to have chronic illness
- Low social class parents are more likely to be under stress be depressed → attachment issues between parent and child → behavioural problems and poor performance in school
- Poorer families less likely to have a good diet → leading to health problems
- Mothers from the low social class less likely to breastfeed (under 25% do in lowest class) → immunity problems in neonates
- Health disadvantage to newborns due to poor maternal nutrition & smoking
- Damp housing → respiratory problems

Soft tissue

Soft tissue swelling: differential diagnosis
- **Soft tissue** consists of;
  - **Tendons**: Fibrous connective tissue between muscle and bones that transmit the force of contraction during locomotion.
  - **Ligaments**: Flexible bands of connective tissue between bones that strengthen and stabilise joints
  - **Menisci**: Crescent shaped fibro-cartilage structures present in the knee and other joints. They act as shock absorbers.
  - **Bursae**: Fluid filled sac lined by synovial membrane that lies between bones in a joint and acts as a cushion.
  - **Skeletal muscle**: Striated muscle controlled by the nervous system that contract to create locomotion.

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| **Tendinopathy** –  | *Tenderness of the insertion.*  
| pain arises from strain or injury to tendons and their insertions to bone. | *An increased in pain when active movement is performed against resistance.*  
|                      | *Soft tissue swelling.*                                                          | Can be diagnosed clinically and investigations are often unremarkable. X-rays may show calcification in chronic rotator cuff disease. | *Rest*  
|                      |                                                                                  |                                                                            | *NSAID’s*  
|                      |                                                                                  |                                                                            | *Local corticosteroid injection*  
|                      |                                                                                  |                                                                            | *Ultrasound*  
|                      |                                                                                  |                                                                            | *Surgery*  |
| **Tenosynovitis** –  | *Presents with pain in the region of the affected tendon.*  
| inflammation of the synovial lining of a tendon sheath. | *Pain and swelling also present.*                                                  | On examination the tendon is swollen and tender and crepitus may be felt on palpation. | *Rest*  
|                      |                                                                                  |                                                                            | *Splinting*  
|                      |                                                                                  |                                                                            | *Local corticosteroid injection*  
|                      |                                                                                  |                                                                            | *Surgical decompression*  |
Tendon rupture – may result from chronic inflammation and degeneration or trauma.

- Loss of movement at the joint.
- Deformity
- Swelling

On examination tendon is painful and tender.

- Surgery is performed to repair the tendon and restore function.

Meniscal tears – can occur due to a traumatic or degenerative tear.

- Painful locked knee
- Gradual nagging pain
- Swelling
- Joint line tenderness

In most cases diagnosis is made solely on history and examination. X-rays may be used to exclude OA.

- RICE (rest, ice, compression, elevation)
- Physiotherapy
- Surgery

ACL tear – 1 in 3000 of the population per year.

- Excruciating pain
- Swelling
- Unable to stand

Can be diagnosed clinically.

- RICE
- Physiotherapy
- Surgery

Olecranon bursitis – precipitated by excess friction at elbow.

- Swelling at the back of the elbow
- Septic cases can cause pain on flexion and redness

History and examination

- Most cases require no treatment
- NSAID’s
- Steroid injection
- Aspiration
- Surgery
- Antibiotics

Cellulitis – bacterial soft tissue infection

Cellulitis most commonly affects one of your legs, but symptoms can develop in any area of your body. The condition affects your skin in several ways, causing it to become:
- Red
- Painful
- Hot
- Swollen
- Tender

GP will diagnose cellulitis through assessment of the symptoms.

- Antibiotics
- Self care
- Pain relief
- Hospital treatment if severe

How pain arises from different soft tissues, and common causes of soft tissue pain

- Noxious stimulus detected by nociceptors in visceral or somatic structures.
- Nociceptive pain: arising from tissue damage, nociceptors in visceral & somatic

Nociception (4 parts):

- Transduction: endings (nociceptors) of C fibres and A delta fibres of afferent neurons respond to noxious stimuli (trauma/inflammation/infection/ischemia).
  - C fibres: small diameter, unmyelinated, slow, respond to mechanical/chemical/thermal, diffuse dull ache.
  - A fibres: large diameter, myelinated, fast, mechanical stimuli over certain threshold, sharp localised pain.
- Transmission: 3 stages:
  - Nociceptor to dorsal horn – synaptic cleft at end of fibre to dorsal horn neuron – impulse transmitted via neurotransmitter (ATP/glutamate/bradykinin) binding to receptors.
  - Spinal cord to brain stem via spinothalamic and spinparabrachial pathways
  - From thalamus & cortex to higher levels of brain (no discrete pain centre).
- Perception Pain: conscious. Cortical areas involved:
- Reticular system- autonomic and motor response to pain;
- Somatosensory cortex- interpretation of senses and relates to past experiences;
- Limbic system- emotional and behavioural response to pain

**Modulation**: change/inhibit pain transmission in CNS re: gate theory (ie distraction or making it worse with stress etc). Wind-up: injury – rapid long term changes in CNS pain transmitters – hypersensitivity of spinal cord.

- Soft tissue: Tissue that connects, supports, or surrounds other structures and organs of the body. I.e. muscles, tendons, ligaments, skin etc.
- Common tendon pathologies:
  - Teniopathy: strain/injury from damage to tendons, particularly at bone insertions. Torn fibres – overuse, systemic inflammation, idiopathic.
    - EG: tennis/golfers elbow: forearm extensor/flexor origin – inflammation of lateral/medial epicondyle tendon
  - Tenosynovitis: inflammation of the synovial lining of tendon sheath – repetitive movement / inflammatory arthritis.
  - Tendon rupture: when the tendon breaks into 2 eg Achilles. Trauma, inflammation, degradation, prolonged steroids.
- Other common soft tissue injuries:
  - Bursitis: joint synovial lining inflammation – injury/systemic/infection/gout eg olecranon; ligament rupture: cruciates/collaterals/ankle sprain – trauma (eg sports); meniscal tears: usually medial, trauma (sports) / degeneration (elderlies); soft tissue infection
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