Week 1 - Cardiovascular Disorders (1)

Atherosclerosis – chronic arterial disease characterised by formation of raised plaques in arterial wall that cause narrowing of a blood vessel and impairment of blood flow (ischaemia). Affects medium to large sized arteries.
In short atherosclerosis = lipid plaques in intima

(Arteriosclerosis is the broad term for a group of diseases characterised by thickening, hardening and loss of elasticity of arterial blood vessels. Most important form of this = **atherosclerosis)**

- **Risk factors**
  - Hyperlipidaemia – elevated blood lipids
  - Hypertension – High blood pressure, MAJOR RISK FACTOR
  - Low-density lipoproteins – plasma cholesterol, shows links with higher severity of atherosclerosis
  - High-density lipoproteins – collect excess cholesterol from peripheral tissue and transport it to the liver for processing, links with lower severity atherosclerosis
  - Smoking – leads to oxidation of LDL, reduces levels of HDL, causes damage to endothelium (blood vessel lining)
  - Diabetes Mellitus
  - Advancing age
  - Male Gender – Oestrogen leads to higher levels of HDL aiding in protection of women, males are more prone before women enter menopause
  - Other factors – reduced PA, obesity, stress, oral contraceptives, high carbohydrate diet, hyperhomocysteinaemia
- **Atherosclerosis process**
  - Response to Injury
  - Damaged endothelium
  - Oxidative modification
  - Oxidised LDL toxic
  - Monocytes turn to macrophages
  - Foam cells created
  - Foam cells turn to fatty streaks
  - Raised fatty streaks
  - Fibrofatty atheromas
- **Complications**
  - Calcification – Deposits of calcium develop in advanced stage, further hardening vessel
  - Superimposed thrombosis – rupture of plaque rood leads to clotting
- **Significance of Atherosclerosis**
  - Coronary atherosclerosis – underlies ischaemic heart disease (angina, A-MI, chronic heart failure)
  - Atherosclerosis of cerebral vessels – major cause in cerebrovascular accidents (strokes)
  - Atherosclerosis in lower extremities – can cause intermittent claudication, skin necrosis and gangrene

Arteriosclerosis – Characterised by thickening, hardening and loss of elasticity on arterial blood vessels. Affects smaller sized arteries (also known as arterial sclerosis and vascular sclerosis)

Ischaemic heart disease – also referred to as coronary heart disease or coronary artery disease caused by atherosclerotic narrowing of coronary arteries. Leads to myocardial ischaemia of cardiac muscle and irreversible hypoxic injury of myocytes
***Acronyms* = Ischaemic Heart Disease IHD = Coronary Heart Disease (CHD) = Coronary Artery Disease (CAD)**

- **Pathophysiology**
  - Oxygen supply – limited by atherosclerosis, hypotension and hypoxaemia
    - Oxygen supply is critical in IHD, when supply can't meet demand.

- Oxygen demand – is increased whenever cardiac contractility and/or rate increased (PA, stress illness increased blood viscosity)
    - Blood can become thicker for various reasons (eg. athletes taking performance enhancing drugs "blood doping" to develop higher RBC count. Higher RBC = thicker blood = more difficult to carry blood around body.
- Coronary atherosclerosis – causes stable IHD (typical angina)
- Superimposed acute thrombosis – leads to further acute narrowing and worsened ischaemia – causing acute coronary syndrome (unstable angina and myocardial infarction)

Angina – IHD

- **Angina pectoris (stable) –** Lack of blood flow to heart – caused by narrowing of vessels
    - Transient chest discomfort/pain – pressure, heaviness, tightness. Squeezing, burning or choking sensation
    - Pain may be experienced in – epigastrium, back, neck, lower jaw, shoulders and arms
    - May be accompanied by – breathlessness, sweating
    - Typically precipitated by exertion but also emotional stress, large meals, cold temps.
    - Relieved by – rest and organic nitrates such as nitroglycerine
- **Angina (unstable) –** angina that changes or worsens with the following (3) features
    - New onset (no previous history), especially if severe
    - Occurs at rest (minimal exertion), lasting >10 mins
    - Occurs with crescendo pattern (distinctly more severe and prolonged or frequent than before)
    - UA is developed due to plaque rupture and superimposed acute thrombosis
    - UA is a serious indicator of impending myocardial infarction
    - When angina (stable or unstable) resolves (spontaneously or w/treatment), no cardiac muscle injury is left behind
- **Diagnosis**
    - ECG is generally normal without symptomatology (in between episodes)
    - Cardiac stress test – during exercise – Looks at T wave and ST segment; characteristic changes are *ST segment depression* and sometimes T wave flattening/inversion
    - Radionuclide stress test w/ technetium – *MIBI scan* - in patients who cannot exercise enough (eg. Due to asthma or arthritis) looking for area of insufficient blood flow
    - Coronary angiography with X ray or CT
- **Management Options**
    - Main goals – symptom relief, preventions of heart attacks
    - Nitroglycerine – delivered sublingually – main treatment
    - Beta-blockers – Long term prevention
    - Low dose aspirin – reduces risk of coronary thrombosis

Acute Myocardial Infarction (AMI) – Heart attack. Prolonged ischaemia causes damage to heart muscle tissue

- **Pathophysiology**
    - Necrosis (**irreversible Injury**) – of heart muscle section caused by prolonged critical ischaemia (typically > 20 mins)
    - Almost always caused by Atherosclerotic plaque rupture with thrombus formation in a coronary vessel – happens in the coronary artery, causing AMI
    - Ischaemia - Prolonged ischaemia (reduction of blood flow)
    - Inflammatory process – follows necrosis of the heart muscle, during this process replaces it with connective tissue within 6 weeks (scarring)
    - Left ventricle – Is involved in almost all acute myocardial infarction. (isolated MI of the right ventricle is very rare due to much less work carried out here)

- **Clinical features – Symptoms**
  - Moderate to severe chest crushing pain
  - Pain radiates into neck, lower jaw, through to the back and along the arms (more commonly to the left arm)
  - NOT relieved by rest and nitroglycerine – MAIN DISTINCTION FROM ANGINA
  - Additional symptomology – breathlessness, tiredness, fainting, nausea, pale skin, sweating, tachycardia and hypotension
  - Approx. ¼ of all myocardial infarctions are silent and without symptoms; these can be discovered later on ECGs.
- **Complications**
  - Acute heart failure – occurs in large infarctions
  - Arrhythmia – ventricular ectopic beats, ventricular tachycardia, ventricular fibrillation (especially dangerous)
  - Papillary muscle dysfunction – with mitral insufficiency and regurgitation
  - Myocardial wall rupture – accumulation of blood in pericardial cavity (fatal)
  - Development of ventricular aneurism with mural thrombosis
    - Risk of systemic embolism
  - Reactive pericarditis – can develop several days after myocardial infarction
- **Diagnosis**
  - Chest X ray – looks at pulmonary circulation
  - ECG – looks for changes in elevation of ST (acute myocardial injury), followed by the inversion of the T wave, development of Q wave is used to signal previous myocardial infarction – *STEMI (ST-segment Elevation Myocardial Infarction)*
  - Cardiac Markers – Elevated levels of;
    - Cardiac contractile proteins:
      - Troponin - T – Main indicator of impending Myocardial infarction (T = the main one!)
      - Troponin - I
    - Cardiac creatine kinase type MB (CK-MB)
- **STEMI – short for: *ST-segment Elevation Myocardial Infarction***
  - Accounts for 70% of myocardial infarctions
  - More severe symptoms, poorer prognosis
  - Complete coronary occlusion
  - Refers to ST ELEVATION in the ECG
  - Pathologic deep Q-wave
  - ST-segment elevation
  - T-wave inversion
- **NSTEMI - short for: *NON-ST-segment Elevation Myocardial Infarction***
  - Accounts for 30% of myocardial infarctions
  - Refers to NO ST ELEVATION in the ECG
  - No pathologic Q-wave
  - ST-segment depression
  - Sometimes T-wave inversion
- **Management –** Focused on salvaging as muscle tissue as possible
  - Medications
    - Opioids – Pain relief
    - Nitroglycerine – coronary vasodilation
    - Treatment of cardiac Arrhythmias if they occur
  - Reperfusion Therapy
    - Thrombolytics for thrombus breakdown
    - Coronary artery bypass grafting

Week 6 – Respiratory Disorders (2)

Cystic Fibrosis – autosomal recessive genetic disease characterised by abnormal transport of chloride (and indirectly sodium and water) across epithelium, leading to thick, viscous secretion in internal organs

Increases NaCl content in sweat – due to reabsorption problems of NaCl from sweat in sweat glands

- **Effects on organs**
  - Pulmonary – stasis of thick secretions and bronchial obstruction → chronic infection with structural changes in airways (fibrosis, dilation) → obstruction of airflow combined with restriction to lung expansion
    - Reduced ciliary clearance means airways are heavily colonised by bacteria causing persistent infections
    - Also involves possibility of fungi, with bronchiectasis occurring in 50% of patients
  - Heart – pulmonary hypertension, RV strain leading o RHF
  - Pancreas – duct obstruction, cyst formation, fibrosis, degeneration of secretory tissue -> shortage of pancreatic enzymes in duodenum → malabsorption
  - Liver – small bile duct obstruction with usually thick bile → stasis of bike In the liver → toxic effect on hepatocytes with thecir necrosis → cirrhosis (widespread heavy scarring) → liver failure
  - Reproductive (Males) – obstruction of vas deferens by thick fluid → sterility (sperm production not impaired)
  - Reproduction (females) – increased viscosity of cervical mucous → blockage of sperm penetration → sterility
  - Sweat glands – high sodium chloride content in sweat (but no excessive sweating) → electrolyte depletion
- **Diagnosis**
  - Preconception genetic testing – identifies mutations in the CFTR of both or one of the partners if the family history is positive
  - Prenatal genetic testing – by chorionic villous (placental tissue) for sampling at 10-12 weeks of pregnancy or amniotic fluid sampling from 16 weeks
  - Sweat test - collected sweat is analysed for abnormal amounts of sodium and chloride

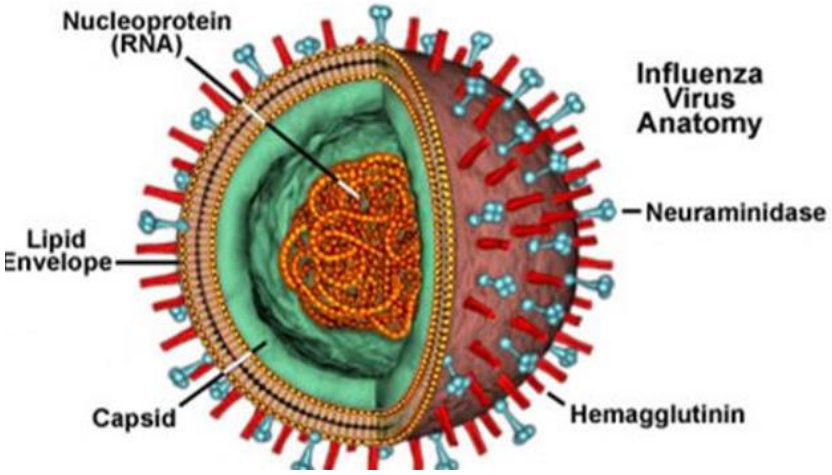
Restrictive lung disease - restriction of lung expansion (decreased lung compliance) resulting in increased work of breathing, inadequate ventilation and impairment of gas exchange - main symptomology = SOB

- **Diagnosis**
  - Pulmonary function tests – Decreased FVC (80% and less than expected); FEV1 is also reduced leading to normal FEV1/FVC ratio PEFR is typically decreased in restrictive disease but can be normal
- **Extrinsic (extrapulmonary) causes**
  - Chest wall deformities
    - Kyphosis, scoliosis, pectus carinatum, pectus excavatum
    - Obesity
  - Neuromuscular dysfunction
    - Poliomyelitis (acute infection and later complications)
    - Muscular dystrophy syndromes (Duchenne and Becker)
    - Guillain-Barre syndrome (acute polyneuropathy)
    - Myasthenia gravis
  - Pleural space disorders
    - Pleurisy and pleural effusion (fluid in the pleural cavity)
    - Pneumothorax (air in the pleural cavity)
- **Intrinsic causes (disease affecting functional lung tissue)**
  - Exposure to various organic dusts (hypersensitivity, pneumonitis, allergic alveolitis)
  - Exposure to inorganic dusts (coal, silica, asbestos, beryllium, iron
  - Infection (pneumonia, extensive TB)

Lecture 7 – Respiratory Disorders (3)

Influenza – highly contagious viral infection of the upper and lower respiratory tract, spreading around the world in seasonal epidemics

- Influenza A - many subtypes exist based on different types of 2 surface proteins (haemaglutinin and neuraminidase)
- Influenza B – only one type
- Haemaglutinin mediates binding of the virus to target cells, while neuraminidase is involved in the release of newly formed viruses from infected cells



- **Manifestations**
  - fever, chills, sore throat, muscle aches, often severe headache, anorexia, weakness/fatigue, stuffed/runny nose and unproductive cough with substernal burning ache (from tracheitis)
  - Flu can be complicated by bacterial bronchitis and bacterial or viral (influenza virus) pneumonia; mortality rate is usually around 0.25% but can go as high as 50-60% (avian flu)
- **Modes of spreading**
  - airborne route (inhalation of droplets produced by the infected person through coughing or sneezing)
  - hand-to-eye, hand-to-nose or hand-to-mouth transmission (from contaminated surfaces or direct personal contact)

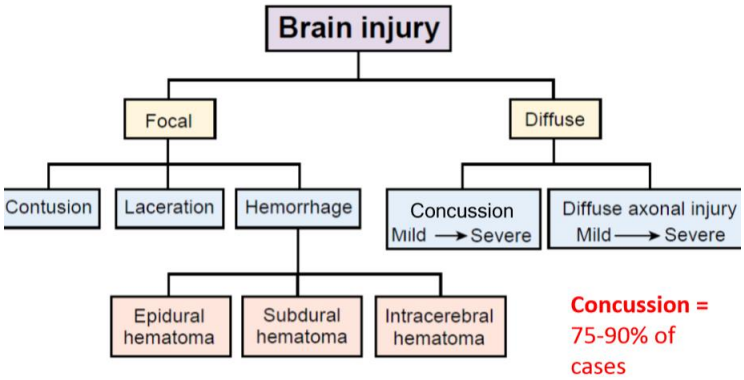
Tuberculosis - Chronic infection caused by bacterium Mycobacterium tuberculosis transmitted from individuals with active pulmonary disease by inhalation of bacteria in aerosols (droplets) generated by coughing

- **Primary TB – form of disease that develops in a previously unexposed person through close contact with the active case**
  - develops in the lower part of the upper lobe or the upper part of the lower lobe in one or both lungs, usually close to the pleura
  - Pathologically there is consolidation and necrosis in 1-3 cm diameter known as Ghon focus
  - In time there is involvement of regional lymph nodes (hilus area) which may also undergo necrosis
  - Combination of initial lung lesion and lymph node involvement = Ghon complex
  - In approximately 95% of cases there is no further progression; scarring develops but living bacteria survive inside for years/decades (latent TB infection - LTBI)
  - During latent TB infection chest X rays are typically normal, there are no symptoms, sputum cultures are negative and the person is not infectious
  - Rarely in malnourished children, elderly or immunocompromised, primary infection may develop into so-called progressive primary TB
  - Progressive primary TB causes extensive local lung tissue destruction, extension into the pleural space (pleuritis) and common spreading to other organ
- **Secondary TB – develops in a person who previously had primary TB, from reactivation of dormant bacteria when hist resistance is weakened**
  - Precipitating factors: diabetes mellitus, serious physical or emotional stress, treatment with glucocorticoids or other immunosuppressants, HIV infection
  - Only around 5% of patients with primary disease subsequently develop secondary tuberculosis!
  - Secondary pulmonary tuberculosis is classically localised to the apex of one or both lungs where it causes variable degree of necrosis and cavity formation (after expectoration of necrotic material)

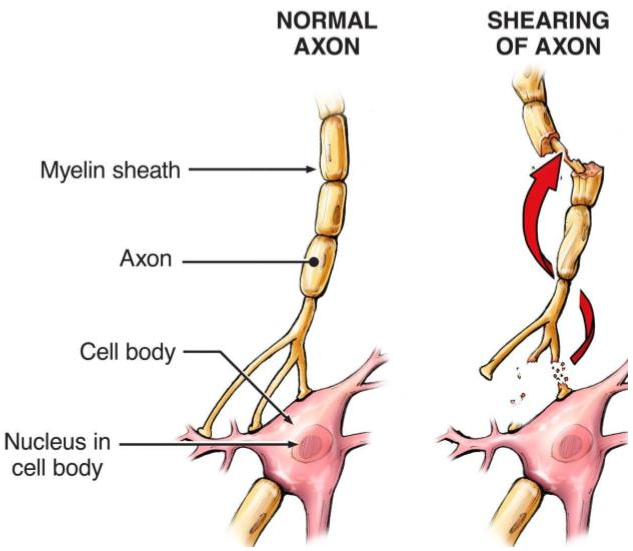
Week 8 – Neurologic Disorders (1)

Brain trauma – Injury to the head/brain by external mechanical force often resulting in loss of consciousness and various neurological impairments

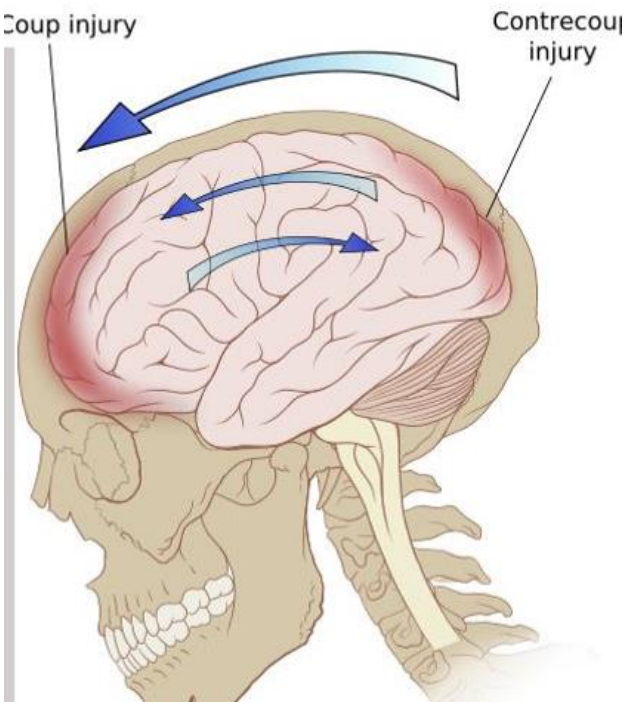
- **Types**
  - Closed/blunt (most common) – head striking hard surface or rapidly moving object striking head; dura matter intact and brain tissues unexposed
  - Open/penetrating (less common) – break in skull/dura matter continuity with exposure of train tissues to environment



- **Concussion**
  - Occurs when a blow to the head disrupts the normal functioning of the brain (temporary axonal disturbance) with some damage to neuronal cell membranes known as – Mild traumatic brain injury *MTBU), mild brain injury, mild head injury (MHI) and minor head trauma
  - Grade 1 – transient confusion, no loss of consciousness, symptoms <15 min
  - Grade 2 – transient confusion, no loss of consciousness, symptoms > 15 min
  - Grade 3 – any loss of consciousness, brief or prolonged
  - Concussed PT is confused (unaware of time, date and place), may repeatedly ask the same questions, be slow to respond to questions or directions, have a vacant stare or slurred/incoherent speech, show difficulty with reasoning or concentrating
  - Transient amnesia (the person cannot remember events leading up to the injury or immediately after it, or both) is a hallmark of concussion
  - There may be gross observable incoordination (stumbling, inability to walk in straight line, clumsiness)
  - The patient may complain of drowsiness, dizziness, headache, nausea, blurred vision and tinnitus
  - Post-concussion syndrome – symptoms do not resolve for weeks/months/years after a concussion and may be permanent
    - Persistent headaches, dizziness, fatigue, anxiety, memory recall/retention problems, attention problems, sleeping difficulty and irritability
- **Diffuse axonal injury (DAI)**
  - The result of traumatic shearing forces that occur when the head is rapidly accelerated or decelerated (whiplash injury), as may occur in vehicle accidents, falls and assaults
  - Major mechanism = disruption and disintegration of axons in white matter without visible macroscopic brain lesion
  - Produces – traumatic coma lasting more than 6 hours
  - Differs in severity – 90% with severe DAI never regain consciousness, those who wake up often remain significantly neurologically impaired



- **Contusion**
    - Focal brain injury caused by blows to the heard or the head hitting a hard surface
    - Compression/crush injury with multiple small intracerebral haemorrhages
    - Normally occur in coup (blow) or coutrecoup (counter-blow) injuries
        - Coup injuries – brain is injured directly under the area of impact force
        - Contrecoup injuries – brain is injured on the opposite side of the impact
        - Inertia involved in both injuries as the brain keeps moving after the skull is stopped by a fixed object
    - Common in anterior frontal/temporal lobes and posterior parietal occipital lobe and cerebellum
    - Contusions associated with <u>oedema</u> – affected tissue and surrounding area cause clinically relevant increase in intercranial pressure



    - Symptoms – headache, confusion, lethargy, vomiting, vision (blurred, diplopia), hearing impairment, coordination and movement issues, seizures and loss of consciousness
    - Associated with – epidural, subdural, subarachnoid and intracerebral haematomas
- **Laceration**
    - Lacerations occur when the tissue of the brain is mechanically cut or torn
    - They usually occur following penetration of the brain by a sharp object, projectile or skull bony fragments after skull fracture
    - Cerebral lacerations usually accompany other brain injuries such as contusions, and are particularly common in inferior frontal lobes and the poles of the temporal lobes (injury on bony prominences of skull base)
    - Lacerations lead to bleeding into the brain tissue (intracerebral haematoma) and into subarachnoid space (traumatic subarachnoid haemorrhage

Week 11 – Musculoskeletal disorder (1)

Rheumatoid arthritis – RA is an autoimmune symmetrical, destructive and deforming polyarthritis affecting large and small peripheral joints with associated systemic disturbances

- **Risk factors**
  - Genetic deficit in immune response (after infection with some viruses (parvovirus B19, Epstein-Barr virus and Human herpes Virus 6)
  - Lack of vitamin C
  - Smoking
- **Rheumatoid factor**
  - IgM autoantibodies (rheumatoid factor) against normal IgG antibodies are found in around 75% of patients
  - They form immune complexes in joints and extra-articular tissues leading to activation of inflammation
  - RF is not specific to RA but an increase in RF is more destructive in the disease
  - Accumulation of activated T lymphocytes, macrophages and neutrophil leukocytes
- **Pathology**
  - The main articular pathologic feature is chronic inflammation in the synovial membrane (synovitis)
  - Thickened and swollen synovial membrane called pannus spreads over and under the articular cartilage, which is progressively eroded and destroyed
  - Pannus is a feature of RA that differentiates it from other forms of inflammatory arthritis
  - Damage of joint tissues appears to be the result of action of released lysosomal enzymes, other protease enzymes and toxic oxygen radicals from macrophages, neutrophils and synovial cell
  - Fibrous adhesions eventually form across the joint space substantially reducing joint mobility (fibrous ankylosis)
  - Such adhesions can undergo partial calcification eventually forming bony ankylosis (complete joint immobility)
  - Ligament/tendon and joint destruction result in limitation of joint motion with consequent muscle atrophy, instability, subluxation (misalignment) and joint deformities
- **Clinical features**
  - Insidious onset with joint pain, stiffness and symmetrical swelling of a number of peripheral joints, usually first small joints of the fingers and toes
  - Swelling of proximal, but not distal, interphalangeal joints gives the fingers a characteristic 'spindled' appearance
  - Rest pain in the joints and especially early morning stiffness; the pain seems to improve on joint movement (exercise or mild work) In time the disease spreads to involve the wrists, elbows, shoulders, other foot joints, ankles, knees, cervical spine (with neck pain and stiffness
- **Characteristic deformities**
  - ulnar drift (deviation) of the fingers (subluxation in metacarophalangeal joints)
  - 'swan neck' deformity is due to hyperextension of the proximal interphalangeal joint with fixed flexion at the distal interphalangeal joint
  - 'button-hole' (boutonniere) deformity is due to fixed flexion of the proximal interphalangeal joint and hyper extension of the distal interphalangeal joint
    - mechanism: tear in the central section (slip) of the extensor tendon through which a head of the proximal phalanx pops through like a finger through a button hole
  - Non-tender subcutaneous rheumatoid nodules (unique to RA) develop in around 30% of patients at sites of pressure or friction such as dorsal side of the hands and elbow area
- **Diagnosis**
  - Positive RF can also be found in other connective tissue autoimmune conditions such as SLE and Sjögren's syndrome (sicca syndrome)
  - Some patients with RA also have positive anti-nuclear antibodies commonly associated with SLE

    - Anti-CCP (cyclic citrullinated peptide) antibodies in blood have high specificity (90%) and sensitivity (96%)
    - Markers in blood
        - Increased erythrocyte sedimentation rate (ESR)
        - elevated C reactive protein (CRP is more sensitive than ESR)
        - thrombocytosis
        - anaemia of chronic disease (not iron deficient)
    - Joint imaging
        - *plain X-ray* is limited to visualisation of the bony structures and their deformities and inferences about the state of the articular cartilage based on the amount of narrowing of the joint space
        - *MRI* offers the greatest sensitivity for detecting synovitis and joint effusions, as well as early bone and bone marrow changes; these soft tissue abnormalities typically occur before osseous changes are noted on X-ray
        - *ultrasound, including colour Doppler,* can detect more erosions than plain radiography, especially in easily accessible joints; additionally it can detect including increased joint vascularity indicative of inflammation
- **Management**
    - NSAIDS (non-steroidal anti-inflammatory drugs)
    - Glucocorticoids
    - Disease-modifying antirheumatic drugs (DMARDs)
    - Physical activity and physiotherapy – prevents ankylosis
    - Surgery – joint replacement
- **Prognosis**
    - 25% will have almost complete remission of symptoms and remain fit for all normal activities
    - 40% will have only moderate impairment of function despite exacerbations and remissions of disease
    - 25% will be more severely disabled
    - 10% will be severely crippled
    - Poor prognosis = high concentrations to RF, poor response to treatment

Seronegative Spondyloarthropathies – group of inflammatory disorders that primarily affect the axial skeleton, particularly the spine, *characterised by the absence of RF*

Ankylosing spondylitis – inflammation (chronic) of the intervertebral joints – affecting mainly the sacroiliac joints and spine leading to progressive stiffening and fusing of the axial skeleton

- **Pathology**
    - Inflammatory enthesopathy (enthesitis) and new bone formation
    - Seen at junction of the vertebral bodies, intervertebral discs and anterior longitudinal ligament
    - Bone ankylosis affecting mobility (reduction)
- **Clinical manifestations**
    - Slow onset, recurring episodes of low back pain, stiffness in sacroiliac area worsening during night and early morning – improves with physical activity
    - Movement restriction in normal lumbar curve becoming flattened and thoracic curve becoming more marked with a forward tilt (kyphosis)
    - Chest pain aggravated by breathing and diminished chest expansion
    - Systemic features – weight loss, low grade fever and fatigue
- **Diagnosis**
    - One or more of the following criteria
        - low back pain of at least 3 months duration, alleviated by exercise, not relieved by rest
        - restricted lumbar spinal motion
        - decreased chest expansion relative to normal values for age and sex

Cardiovascular disorders	
Problem / Disorder	**Description**
Atherosclerosis	chronic arterial disease characterised by formation of raised plaques in arterial wall that cause narrowing of a blood vessel and impairment of blood flow (ischaemia).
Arteriosclerosis	Thickening, hardening and loss of elasticity of arterial blood vessels
Ischaemic heart disease	(coronary heart disease) – atherosclerotic narrowing of coronary arteries
Angina pectoris (stable)	Lack of blood flow to the heart - caused by narrowing of vessels
Acute myocardial infarction	Prolonged ischaemia causes damage to heart muscle
Endocarditis	Endocardial inflammation (mostly infectious)
Valvular stenosis	Narrowed valves (valve leaflets fused)
Valvular insufficiency (regurgitation)	Valve leaflets fail to shut completely
Rheumatic fever	Post-streptococcal immune – inflammatory disease that affects heart and joints
Pericarditis	Inflammatory process of pericardium with or without exudation (inflammatory fluid)
Myocarditis	Inflammation of the heart muscle (caused by virus)
Cardiomyopathies	Diverse group of heart muscle disease that cause deterioration of myocardial function
Heart failure	Inability to maintain adequate CO (a state of the heart – not a disease)
Varicose veins	Distended, enlarge and torturous veins
Deep vein thrombosis	Formation of a blood clot (thrombus) in a deep vein – mainly lower extremities
Aneurysms	Localised of permanent dilation or outpouching of a specific section of a vessel or cardiac chamber wall
Hypertension	Elevation of systolic and/or diastolic blood pressure above normal level of 140/90 mmHh
Hyperaemia	Increased volume of blood in a particular tissue resulting from increased inflow of arterial blood after arterial dilation (active process)
Congestion	(passive hyperaemia) is also increased amount of blood in the tissue but due to a passive process resulting from impaired outflow of venous blood
Oedema	(swelling) is increased amount of fluid in the interstitial tissue spaces (extravascular and extracellular
Haemorrhage	AKA bleeding – indicates leakage of blood due to rupture/injury of blood vessels
Shock	failure of CV system to perfuse tissues (systemic hypoperfusion) – resulting in impairment of cellular metabolism

Pathophysiology: Definitions

Agnosia – inability to interpret sensations or recognise

Ankylosis – abnormal stiffening and immobility of a joint due to fusion of the bones

Aphasia – impaired comprehension and expression of language

Apraxia – inability to perform movement or sequence despite intact sensation, motor output and understanding

Auscultation - the action of listening to sounds from the heart, lungs, or other organs

Arrhythmia – abnormal heart rhythm

Arthralgia – joint aches

Atrophy – shrinkage in size of cell through loss of structural material

Ataxia - lack of voluntary coordination of muscle movements that includes gait abnormality

Bradycardia – heart rate is slower than normal range

Benign – localised tumor

Calcification – deposits of calcium

Cephalgia – headache

Cyanosis – a bluish cast to the skin and mucous membranes

Demyelination – any disease of the nervous system in which the myelin sheath of neurons is damaged

Diplopia – double vision

Dysphagia – pain on swallowing

Dysplasia – changes in size, shape and organisation of cells ****

Dyspnoea – difficult or laboured breathing

Dystharia – difficulty speaking

Haemoptysis – coughing up blood

Hemiplegia – paralysis on one side of body

Hemianopia – blindness over half the field vision

Hypertension – high blood pressure

Hypotension – low blood pressure

Hyperlipidaemia – elevated blood lipids