Condition Summaries

Cardiology + vascular

Heart failure

- Impaired heart function, specifically the ability of the left ventricle to pump blood around the body
- Exertional breathlessness, cough, orthopnoea, PND, peripheral oedema
- Tachycardia, tachypnoea, HTN, 3rd heart sound, bilateral basal crackles, JVP, oedema
- Ix:
    - FBC – rule out anaemia, infection. TFTs, U&Es nephrotic syndrome, LFTs, troponin, BNP, iron studies, bone profile (rule out myeloma)
    - CXR – pleural effusion, cephalization of vessels, kerley B lines, increased cardiothoracic ratio
    - Echocardiogram
    - Stress test, angiogram
- NYHA classification (1-4) – measure of symptomatic severity
- >50% EF = preserved ejection fraction – diastolic failure, dilated cardiomyopathy, obstructive etc
- <40% EF = reduced ejection fraction – systolic ventricular failure/loss of synchrony, e.g. post STEMI
- Rx:
- Conservative – smoking cessation, alcohol cessation, fluid/sodium restriction,
- Medical – avoid NSAIDs and Steroids – both cause fluid retention
    - ABAL – ace inhibitor, bisoprolol, aldosterone antagonist e.g. spironolactone, loop diuretics
    - A-E assessment and treat accordingly
    - Treat underlying cause e.g. arrhythmia, HTN, fluid balance – restrict/diuretics, **ABAL** (**ACEi, Beta blocker, Aldosterone antagonist, Loop diuretic**)
    - **Furosemide** (consider PO/IV)
    - Vasodilators e.g. **GTN**
    - Beta blockers – cardioselective e.g. **atenolol, bisoprolol**
    - Diamorphine for breathlessness
    - Resp support
    - Preserved ejection fraction vs reduced ejection fraction considerations:
        - SLGT2 inhibitors – beneficial for rEF
        - Aldosterone antagonist used especially in cases of left ventricular function – reduced EF, **eplerenone** preferred as less likely to cause gynecomastia
- Surgical
    - Revascularisation, valve repair/replacement
    - Cardiac resynchronisation therapy
    - Dual chamber pacing
    - Heart transplant

Peripheral arterial disease (PAD)

- Narrowing/occlusion of peripheral arteries – caused by atherosclerosis > stenosis
- Types: intermittent claudication, acute limb ischaemia, critical limb ischaemia
- Critical limb – burning pain, worse at night
- Pathophys:
    - Atherosclero narrows arteries > limits blood flow
    - Acute limb ischaemia = thrombus/embolus – plaque breaks off/bursts > thrombosis > **sudden** reduction in art perfusion of limb
- Risk factors: CVD, smoking,, DM, hyperchol, CKD, arrythmias
- Sx:
    - intermittent claudication
    - acute limb ischaemia: 6P's
        - Pain
        - Pallor
        - Pulseless
        - Paralysis
        - Paraesthesia
        - Perishing cold
    - critical limb: chronic pain at rest, worse at night, skin absent foot pulses, skin changes (non-healing, gangrene), sleep with leg hanging out of bed

- Leriche syndrome – occlusion in the distal aorta or proximal common iliac artery
    - Thigh/buttock claudication
    - Absent femoral pulses
    - Male impotence
- Exam: foot pulses, temp, ulceration, skin changes, elevation pallor, CRT, CV exam. **Buerger's test (blue, dark red)**
- Ix: ABPI = <0.9 = PAD / <0.5 critical limb / >1.4 could be PAD in DM ; duplex USS ; angio CT/MRI
    - Highest SBP ankle/ highest SBP arm
- Rx: **statin, clopidogrel. Surgery –** endovasc angioplasty/stenting, endarterectomy, bypass

Stable angina

- Caused by atherosclerosis of the coronary arteries, causing reduced blood flow to the myocardium
- Ischaemia → pain
- "stable" angina is when symptoms come on only with exertion, and are relieved by rest/GTN
- Ix: ECG, CT angio
- Rx: 1st line = **GTN**, **beta-blockers** OR **CCB** to reduce sx
    - Beta blocker or CCB 1st line
    - if CCB as monotherapy, rate limiting one should be used e.g. **Verapamil** or **Diltiazem**
    - If used in combination with beta-blocker longer acting should be used e.g. **amlodipine** or modified release **nifedipine**
    - 3rd line - long acting nitrate (**isosorbide mononitrate**) or **ivabradine**

ACS

- Common management for all ACS – MONA-C (morphine, oxygen, nitrates, aspirin, clopidogrel OR pCi)
    - **aspirin 300mg (loading dose) + clopidogrel (300mg)**
    - oxygen should only be given if the patient has oxygen saturations < 94%
    - morphine should only be given for patients with severe pain – IV
    - nitrates – **GTN** - IV or sublingual – caution if hypotensive
- If unstable angina / atrial fibrillation and unable to get PCI within time frame consider anticoagulating also **(LMWH)**

Unstable angina/NSTEMI

- Symptoms at rest
- Ix: ECG (normal), trop T (raised in NSTEMI)
- Rx: GRACE score
    - <3 Low risk – dual antiplatelet (aspirin + clop)
    - >3 Interm/high risk – angio +/- PCI within 72 hours + dual antiplatelet therapy + **antithrombin (LMWH) 1mg/kg BD**

STEMI

- Sx: pain (chest/abdomen)
- Ex
- Ix: ECG, trop T, new LBBB
- Rx: dual platelet therapy + PCI (if within 2 hours of admission), if PCI not available + within 12 hours → **alteplase**
    - If fibrinolysis – give **antithrombin (LMWH) 1mg/kg BD** as well + dual platelet therapy
- Complications: DREAD – DARTH VADER
    - Death, Rupture, oEdema, Arrhythmias, Dressler's
    - Death, arrhythmia, rupture, tamponade, heart failure, valve disease, aneurysm, Dressler syndrome, embolism, recurrence

ACS secondary management

- Cardiac rehab
- Cardioprotective drugs (4A's + C): **atenolol**, **ACEi**, **atorvastatin**, **aspirin** + **clopidogrel**
    - If have HF with reduced EF as well (aldosterone antagonist) – **spironolactone**

Familial hyperlipidaemia

- Autosomal dominant genetic condition causing very high cholesterol level - **LDLR** most common
- Signs: Achilles tendon thickening, xanthelasma, corneal arcus, cardiovascular disease in younger patient
- Important features for clinical diagnosis: family history, very high cholesterol >7.5mmol/L, tendon xanthoma
- Rx: genetic testing + **statins**

Hypertrophic cardiomyopathy

- Inherited. Hypertrophy of LV myocardium without stenosis
- Gene encoding sarcomere protein
- Ex: Jerky pulse, S4 (hypertrophy of the ventricles), ejection systolic murmur increasing with valsalva manoeuvre, decreases with squatting. Mitral regurg.
- Ix: ECG - deep Q waves, left sided hypertrophy (larger QRS complexes)
- Echo – diagnostic (asymm LV hypetroph with septal thickening, mitral regurgitation)

Takotsubo cardiomyopathy

- Non-ischaemic cardiomyopathy, triggered by stress
- Chest pain
- "Octopus pot" sign on XR, ST elevation on ECG

Murmurs

Diastolic (S2->S1)	Mitral stenosis - Mid diastolic - Opening click (stiff valve) - Low pitch grumbling - IE, rheum fever - Accentuated: lie on left	Aortic regurg - Soft, low pitched rumbling - Corrigan's pulse (collapsing), - Austin flint (apex, early diastolic) 3rd sound regurgent blood hitting mitral leaflets - - Nailbed pulsation – Quinke's sign - Head bobbing (de Musset's sign) - Caused by age, connective tissue dis - Leads to HF - Accentuated: lean forward, expiration
Systolic (S1->S2)	Mitral regurg - Pansystolic - High-pitched whistling (BRRR) - Reduced EF HF, age (weakening of pap muscles), IHD, IE, rheum, connective tissue dis	Aortic stenosis - Ejection systolic - Crescendo decrescendo - High-pitched - Radiates to carotids, slow rising pulse, narrow pulse pressure, exertional syncope - Age (calcification)

- RILE – right side louder on inspiration, left side louder on expiration
- Heart sounds
    - S1 = mitral and tricuspid valves closing
    - S2 = aortic and pulmonary semilunar valves closing
    - S3 (Kentucky) = normal in young, heart failure in old (stiff/tired papillary muscles)
    - S4 (Tennessee) = always pathological
- SCRIPT – to describe murmurs (site, character, radiation, intensity, grade (velocity), timing
- Grading (Levine scale) – (when in doubt 2 or 3)
    - 1 = difficult o heart ; 2 = quiet ; 3 = easy to hear ; 4 – easy to hear with palpable thrill ; 5 = hear with steth barely touching chest ; 6 hear without steth

- **Hypertrophy and dilatation**
    - Hypertrophy = muscle working harder because of stenosis
        - Left atrial hypertrophy = due to mitral stenosis (atrium working harder)
        - LVH = aortic stenosis (ventricle working harder)

    - Dilatation = blood flowing backwards, making the chamber bigger
        - Left atrial dilatation = mitral regurg
        - Left ventricular dilatation = aortic regurg
- **Rheumatic fever**: ***strep pyogenes***. Main cause of mitral stenosis (scars valve)
    - Ix: throat swab: rapid GAS antigen test
    - Rx: penicillin, NSAIDss
    - IE: IVDU = ***s aureus***. Everything else: the ***coccis*** (strep, enteroc, staph)
- **VSDs**
    - Acyanotic USUALLY (Eisenmenger's syndrome = conversion to cyanotic approx 6 weeks after birth)
    - increased risk of infective endocarditis (requires prophylactic abx for surgery). Also, pulmonary HTN → RV hypertrophy (→ eisenmenger's syndrome if right sided pressure exceeds left)
    - Rx: Transvenous catheter closure

Cardiac tamponade

- Causes = pericardial effusion, haemopericardium
- Signs (Beck's triad): muffled heart sounds, hypotension, raised JVP + pericardial rub, pulsus paradoxus (BP drops on inspiration)
- Ix: CXR big globular heart, ECG - electrical alternans (QRS alternating in height), echo
- Rx: pericardiocentesis, **NSAIDs**

Pericarditis

- Idiopathic, post-MI (Dresslers), Lupus, infective (viral: influenza, herpes, coxsackie A & B), thyrotoxicosis, malignancy e.g. bronchus
- Pericardial friction rub, relieved on leaning forward, wore on inspiration
- Ix: ECG (saddle-shaped ST elevations, global)
- Rx: **NSAIDs**, **colchicine**
- Complications: pericardial effusion > cardiac tamponade

Aortic dissection

- SURG EMERGENCY
- Stanford: Type B = descending only, Type A = asc only or asc + arch +/- desc)
- Sx: Tearing, ripping pain, can radiate to back – intrascapular.
- RF: HTN!!!!! Marfan, Ehlers-Danlos, aorta conditions (bicuspid valve, coarctation, valve replacement)
- Exam: hypotension (as dissection progresses), BP different in arms, pulse different in LLs, murmurs, syncope
- Ix: CTA – visualise intimal flap
- Rx: **beta blockers**, **morphine** (**ACEi** long-term)
    - Type A = open surgery, remove that part of the aorta (replace with synthetic), maybe replace valve
    - Type B = endovascular stent/graft repair

Atrial Fibrillation (AF)

- Condition where the electrical activity in the atria becomes disorganised, leading to fibrillation (random muscle twitching) of the atria and an **irregularly irregular** pulse
- Leads to heart failure due to impaired filling during diastole
- Turbulent blood flow causes hypercoagulability → clots → stroke
- Pathophysiology: Signal between SAN and AVN interrupted
- Common causes: **SMITH** likes to drink **alcohol** and **coffee. MOST COMMON = AGE.**
    - **S**epsis
    - **M**itral valve pathology
    - **I**schaemic heart disease
    - **T**hyrotoxicosis
    - **H**ypertension
- Sx: palpitations, shortness of breath, dizziness/syncope, stroke
- Ex: irregularly irregular pulse, tachycardia
- Ddx of irregularly irregular pulse: ventricular ectopics - this should normalise at higher HR e.g. during exercise
- Ix: ECG - absent p waves, narrow QRS, irregularly irregular. Echo - valvular disease, heart failure.
- Paroxysmal AF - episodes lasting between 30s and 48hrs - Ix: holter monitor / cardiac event recorder
- Rx: Medical: Rate or Rhythm control AND anticoagulation
    - Beta blocker - **bisoprolol/atenolol** (contraindicated in asthma)
    - or CCB - **verapamil/diltiazem** (contraindicated in heart failure)
    - **Digoxin** if fast AF
    - **Amiodarone** as rhythm control if co-existing heart failure
    - **Anticoagulation** if CHA2DS2VASc > 1 = DOAC (e.g. **Rivaroxaban**) or **Warfarin** (preferred in valve replacement)
        - Direct factor Xa inhibitor = **Rivaroxaban, apixaban** etc. Reversal agent = **Adexanet Alfa**
        - Direct thrombin inhibitor = **Dabigatran**. Reversal agent = **Idarucizumab**
- Rx: Acute presentation (e.g. within first 48hrs) → cardiovert. If presenting outside of this window must be anticoagulated for 3 weeks prior to cardioversion
- Rx: surgical - catheter ablation, pacemaker
    - catheter is fed into femoral vein under XR guidance - abnormal pathway is ablated

Supra-ventricular tachycardia – SVT

- including Wolf-parkinson white (delta waves)
- Management
    1. Vagal manoeuvres
    2. **Adenosine** – 6, 12, 18 – not to be given in asthmatics – can cause bronchospasm
    3. **Verapamil/beta blocker**
    4. DC cardioversion
- Ablation of accessory pathways = definitive treatment

Ventricular tachycardia

- if unstable → cardiovert
- If stable → amiodarone, can cons lidocaine but contraindicated in impaired LV function, procainamide
- absolute contraindication - verapamil

Atrial flutter

- Sawtoothing
- Regular – division of 300bpm (e.g. 150, 300 rate)
- Treated same as atrial fibrillation
- Management – rate/rhythm control
    - Beta blocker or if low BP **digoxin** (loading dose 500microg #trauma, maintenance dose 125microg)
        - level recorded 6-8 hours after dose
    - Implantable

Bradycardia

- Total heart block can result in bradycardia as ventricles self-pacing
- Rx: Management of unstable patients and those at risk of asystole involves:

    - Intravenous atropine (first line)
    - Inotropes (e.g., isoprenaline or adrenaline)
    - Temporary cardiac pacing
    - Permanent implantable pacemaker, when available

Heart block

- First degree:
    - Delayed conduction through the AVN
    - Every atrial pulse leads to a ventricular contraction - but PR interval > 0.2s/5 small squares
- Second degree
    - Some atrial impulses do not make it through AVN
    - Mobitz type 1 - PR interval gets progressively longer until it fails + drops a beat, then cycle reapeats
    - Mobitz type 2 - Intermittent failure of AVN, absence of QRS complexes following P waves. Typically set ratio, e.g. 3 p waves to each QRS. PR interval is normal. RISK OF ASYSTOLE.
- Third degree
    - Complete heart block
    - No relationship between P waves and QRS
    - Ventricles pace themselves - typically associated with bradycardia + RISK OF ASYSTOLE
    - Wide pulse pressure
- Rx: Acute management = **atropine**, +ve inotropes (e.g. **adrenaline**), external pacing, implantable pacemaker

Dermatology

Pigmented skin lesions – weighted 7-point checklist:

- 2ww referral to derm = >=3
- But if really strong factors present (personal/fmhx melanoma, immunosuppression, ++tanning beds) refer anyway

Major features (2 points)	Minor features (1 point)
- Change in size - Irregular shape or border - Irregular colour	- Largest diameter >=7mm - Inflamm - Oozing, crusting - Change in sensation

Pityriasis rosea

- Herald patch – smoky salmon coloured solitary patch – gets bigger over days
- Christmas tree distribution
- Self resolves 6-8w

Malignant melanoma	SCC (rhymes with p53)	BCC (just a patch)
- Pigmented skin lesion which "stands out from the crowd" - Superficial melanoma – large, flat, irregularly pigmented grows laterally first (then vert) - **Nodular melanoma** – aggressive, rapidly growing, pigmented nodule, bleeds, ulcerates BRAF mutations	- Usually raised - Keratinised - Ulcerated - Crusting - Typically grow on head, neck, back of hand - More common in immunosuppressed p53 tumour suppressor gene damaged	- Ulcer with raised rolled edge - Prominent fine blood vessels around lesion - Nodules, often waxy or pearly in appearance □ sun-exposed areas Patched (PTCH) tumour suppression gene damage

Impetigo

- Non-bullous = ***s aureus*** +/- ***strep pyogenes***
- Bullous = fluid-filled. ***s aureus***
- Can occur anywhere, but commonly on face. Honey/golden-crusted
- Rx: hydrogen peroxide cream □ Fucidin □ abx if systemically unwell e.g. flucloxacillin

Eczema

- Rx: **emollients > topical steroids (inflamed skin) > antihistamines > oral corticosteroids (severe, extensive) > abx for secondary bacterial infx** (can also use topical **calcineurin inhibitors**)

Bullous pemphigoid

- **SUBepidermal** blisters
- Autoimmune (HLA) rx, but can be caused by drugs, infx
- Attacks HEMIdesmosomes

- Tense bullae (looks like SJS)
- Rx: **topical steroids, sedation** (v. uncomfortable

Pemphigus vulgaris

- Painful **INTRAepidermal** blisters on mucous membranes – usually mouth
- Cause: autoimmune, drugs
- Pathophysiology: IgG attacks desmosomes (hold your skin together)
- ++ complications (infx)
- Ix: biopsy, direct immunofluorescence
- Rx: **systemic corticosteroids**

Dermatomyositis

- inflammatory disorder causing symmetrical proximal muscle weakness
- Sx: myalgia, proximal muscle weakness, gottron papules (back of hands), heliotrope rash on eyelids (dark purple like the flower apparently??), periorbital oedema
- Ix: Creatine kinase – elevated. Autoantibodies (**anti-Jo-1**). EMG. CTAP (paraneoplastic syndrome - breast, stomach)
- ddx for proximal muscle weakness: steroids, alcohol, polymyositis, myasthenia gravis (?)
- Rx: corticosteroids, monoclonal antibodies
- Complication - Kaposi's sarcoma (immunosuppressed)

Actinic keratosis

- Pre-malignant small crusty/scaly lesions, typically sun-exposed areas
- Topical **fluorouracil** – chemotherapy agent

Psoriasis

- Common chronic skin disorder – thought to be some genetic component, and some evidence there is T cell involvement, stimulating keratinocyte proliferation. In addition environmental triggers (infection worsens, sunlight improves)
- Koebner phenomenon - site of injury causes development of new psoriasis patch
- Sx: red/scaly patches on the skin, pitting/onycholysis, arthritis
- Subtypes:
    - Plaque psoriasis – most common subtype, well-demarcated, red/scaly patches affecting extensor surfaces, sacrum, and scalp
    - Flexural psoriasis – skin folds affected, often non-scaly
    - Guttate psoriasis – triggered by streptococcal infection – red teardrop lesions
        - Classic prodrome = 2-4 weeks sore throat
        - Trunk/limbs affected
    - Pustular psoriasis – palms and soles of feet – pustules filled with white cells, painful and erythematous emergency treatment required.
- Complications – psoriatic arthropathy cardiovascular disease, VTE, metabolic syndromes
- Rx: regular emollient, **topical corticosteroid** + **topical vitamin D analogue** for up to 4 weeks. Also phototherapy, oral **methotrexate**, **ciclosporin**, systemic retinoids, biologics (infliximab)
    - If no improvement – change steroid formulation, consider use of salicyclic acid prior to application
    - Beta-blockers can exacerbate psoriasis

Endocrine

Metabolic syndrome

- DM + HTN + obesity
- Greater risk of developing CVD

T2DM

- Increased insulin resistance, reduced insulin secretion → persistent hyperglycaemia
- Chronic exposure to high carb/glucose, liver + cells become desensitised + pancreas becomes fatigued
- Risk factors: modifiable and non-modifiable (>40y, ethn, hx GD, low birth weight, PCOS, Cushing's)
- Sx: asymptomatic (found on routine screening), mild-moderate = fatigue, vague sx, infx, poor wound healing, severe = hyperosmolar hyperglycaemic state
- Ex: acanthosis nigracans, oral thrush
- Ix: urine glucose, 2 x HbA1c (42-47 = prediabetic, >48 = diabetic)
- HbA1C can't be used in: children, pregnancy, acutely unwell, CKD, anaemia
- Rx: conservative (MDT, 4C's, DELAYS), medical, surgical (treat complications)
    - Metformin
    - + Second agent - **53 mmol/L**
        - SGLT2 inhibitor - **dapagliflozin** - especially in cardiovascular disease/heart failure or QRISK >10%
        - Sulfonylurea - **gliclazide**
        - DPP-4 inhibitor - **sitagliptin**
        - Thiazolidinedione - **pioglitazone** (reduces peripheral resistance to insulin)
    - If dual therapy doesn't improve HbA1c → Triple therapy OR
    - Insulin therapy
- Complication: macro (impaired vasodilation) and microvascular (cataracts, periph neuropathy, nephrop).
- Hyperglycaemic hyperosmolar state - Glycosuria takes water with it making serum more concentrated
- TARGETS –
    - 48mmol – GENERAL TARGET
    - On mono/dual therapy already: aim for HbA1C of 53mmol/mol
    - Measure HbA1c every 3-6 months

Gestational diabetes

- Lower threshold for diagnosis
- Fasting plasma glucose 5.6 or OGTT 7.8 mmol/L (5, 6, 7, 8) at 24-28w

T1DM

- Autoimmune – destruction of pancreatic beta cells (associated with coeliac, thyroid, HLA DR3/DR4)
- Pathophys:
    - Polyuria: hyperglyc □ exceeds renal capacity to absorb □ glycosuria □ inhibits kidney's concentrating ability
    - Polydipsia: response to dehyd + direct response to high blood glucose stimulates thirst
    - Weight loss: unopposed lipolysis, proteolysis for gluconeogenesis precursors
- Sx: polydipsia, polyuria, weight loss, fatigue
    - Prayer sign - unable to get hand flat to the table
    - Chronic manifestations:
        - peripheral neuropathy
        - diabetic nephropathy
        - diabetic retinopathy
        - necrobiosis lipoidica diabeticorum (shiny painless yellow/red skin on the shin, vascular changes)
        - infections
- Ix: random glucose ≥11.1 mmol/L, ketosis
- Dx: hypergluc, ketosis, rapid weight loss, (<50y), (BMI <25), personal or fmhx of autoimmune disease

T1 & T2DM

- Rx: MDT, monitoring (4 C's – control, competency, coping, complications), lifestyle, **insulin (basal-bolus)**, surgical (islet/pancreas transplant recurrent severe hypoglycaemia)
    - Lifestyle (DELAYS): dietary, exercise, lipids, reduce alcohol, yearly check up, smoking cessation
    - **Insulin**: rapid-acting (peaks 30-90m, lasts 5h), short-acting (peaks 2-4h, lasts 4-8h), intermediate (peak 4-14h, last 14-20h), long acting (last 20-24h)
    - Target = HbA1c 48 or lower
- Complications: microvasc (retinop, nephrop, neurop), macrovasc (cardiovasc, cerebrovasc, peripherovasc)
    - BP control, check for renal disease (ACEi, urine dip), cholest checks, eye checks, foot checks
- Sick days – take insulin normal (might need to increase due to increased met demand) – check BM more often

MODY - autosomal dominant

Pathophys of DM

- Glycation of Hb – modification of proteins
- Sorbitol (product of glucose) pathway overactivity – big, get stuck in cell membrane, osmotically activate metabolites that draw water into tissues
- Disturbance of cellular redox state
- Impaired vasodilation

Diabetic ketoacidosis

- Triad (DKA): Diabetic (BM >11.1), Ketonaemia (serum ≥3, urinary ≥3), Acidosis (pH <7.3, bicarb <=15)
- Pathophys: ketones produced as alternative energy source made from fatty acids acidaemia shock
- Ketones = acetone, acetoacetate, β-hydroxybutyrate
- Causes (5 I's + 1 P): infection, intoxication, inapprop withdrawal insulin, intercurrent illness, infarction, pancreatitis
- Sx: dehydration, signs of shock (from dehyd), high RR (blow off acid), severe abdo pain, N/V, altered mental state Kussmaul breathing
- Ix:
    - Bedside: VBG (ABG also fine, but VBG preferable), ECG (sinus tach), urine dip (ketones), bHCG in F
    - Bloods: FBC, U&E, LFT, CRP, lab gluc, amylase.
        - Every 1h: glucose, ketone monitor, lab gluose
        - Every 4h: U&E
    - Imaging: CXR (exclude other source)
    - Cultures if febrile
- Exam: A-E
- Rx: cardiac monitor, wide bore, **IV fluids** if BP <90 (1L in 1h > 1L in 2h x2 > 1L in 4h x 2), fixed rate **insulin** infusion, **K+ replacement + dextrose** (when drop below BM 14), swap to long-acting insulin
    - Resolved = ketones <0.6, pH >7.3

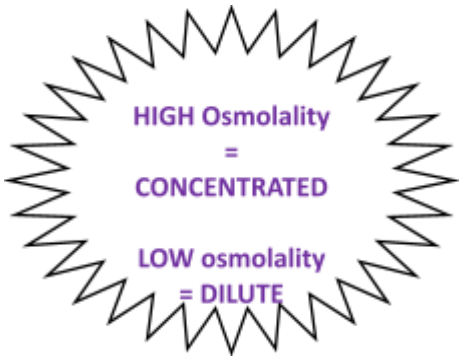
Hypoglycaemia

- Plasma gluc <4mmol/L
- Causes: reactive hypoglyc (post prand, gastric surg), drug-induced (**insulin**, **sulphonylurea**), fasting (PLAIN – pituitary failure, liver disease, Addison's, Islet cell tumours, Neoplasm)
- Sx:
    - Non-specific – N, headache
    - Autonomic – sweating, palp, tremor, hunger
    - Neuroglycopenic – confusion, clumsiness, beh changes
- Rx:
    - Conscious, oriented, able to swallow – **oral glucose** replacement (juice, glucotabs, sugary drink).
        - BM 10-15m later. Still <4 **IM/IV**. If >4 give **carbs**
    - Conscious, can swallow but aggressive/confused – **dextrogel/glucogel** teeth/gums, **IM glucacon**

- Unconscious/seizures/v. aggressive – A-E, **IM glucagon** (TRY IM FIRST AS QUICKER) or **IV glucose**

Hyperosmolar hyperglycaemic state (HHS)

- Rare complication of T2DM. Can be fatal
- Osmotic diuresis secondary to hyperglycaemia causes increase in concentration of solutes (Na, K+)
- Sx: same as TDM + altered mental state
- Ix: hypernatraemia, hyperkalaemia (because losing all the water so fast), VERY high serum osmolality
  - Low/normal ketones – why not DKA
- Rx: ITU, IV fluids, **IV insulin**



Thyroid disorders

- Primary – originating within the gland (e.g. hashimoto's, iodine deficiency)
- Secondary – originating upstream in the hormonal axis e.g. hypothalamus or pituitary
- Ix: ECG, BM, TFTs, U&E to rule out electrolyte causes of arrythmia, antibodies, imaging: Ultrasound, Radioisotope scan

Hypothyroidism

- Common causes: Hashimoto's – anti-TPO
- Iodine deficiency, Radiotherapy vestibul/radioactive iodine, medications, thyroidectomy
- Sick Euthyroid – can be predictor of how unwell someone is, general decrease in T3/4
- Sheehan's syndrome – following childbirth and PPH, pituitary ischaemia
- 1st line – **levothyroxine** - (note that iron and Ca supplements both inhibit absorption)

Hyperthyroidism

- Common causes: Grave's disease – TSH receptor antibodies. Thyroid eye disease – exophthalmos, lid lag. Pre-tibial myxoedema. STOP SMOKING. Conn's syndrome.
- Toxic multinodular goitre, toxic thyroid nodule, transient thyroiditis
- Pituitary adenoma
- 1st line **Carbimazole** – titration to control symptoms or block and replace
- 2nd line **Propylthiouracil**
- Symptomatic management e.g. **beta blockers**
- Surgical management – thyroidectomy, radioactive iodine

Thyroid cancer

- Neck lump, voice change, hoarseness, dysphagia
- Ix: Ultrasound +/- guided biopsy (FNA)
- Most common subtype = Papillary (70%) then follicular (20%)
- Medullary thyroid cancer - **calcitonin**

Prolactinoma

- most common subtype of pituitary adenoma
- triad:
  - amenorrhea in females/impotence in males
  - headache
  - bitemporal hemianopia

- also - galactorrhea (excess prolactin)
- Ix: MRI
- Rx: transphenoidal resection

Cushing's syndrome – any set of symptoms caused by excess cortisol

- Cushing's disease – pituitary adenoma secreting ACTH
- Sx: thinning of skin, proximal muscle weakness, dorsocervical fat pad, weight gain, moon face
- Causes – adrenal adenoma , pituitary adenoma, small cell lung cancer
- Ix: **dexamethasone** suppression test (low dose and high dose), bloods (WCC can be elevated), U&Es as adenomas can also secrete aldosterone
    - Pituitary adenoma – high dose suppresses ACTH AND cortisol
    - Adrenal adenoma – high dose suppresses ACTH but NOT cortisol
    - Ectopic ACTH – high dose will not suppress either – CT chest for SCC
- Treat cause

Addison's disease – adrenal insufficiency – diabetes of the adrenal gland – low aldosterone and low cortisol

- Most commonly autoimmune ao
- Sx: Hyperpigmentation of the skin, fatigue, hypotension, nausea, abdominal pain/cramps
- Signs: hyperpigmentation (because ACTH precursor is also melanocyte stimulating hormone (MST) precursor > more ACTH > more MST > more melanin stimulation >++pigmentation)
- Ix: Short synacthen – 3 blood tests, one as baseline, one 30 mins after synacthen, one 60 mins after, measure cortisol. Cortisol should at least double. **Synacthen = synthetic ACTH.**
- Management:
    - Glucocorticoids (**hydrocortisone**) and Mineralocorticoids (**fludrocortisone**).
    - Acute (crisis) only replace glucocorticoid
        - IM rescue hydrocortisone

Addisonian crisis

- Triggers – sickness, absence of steroids, trauma, also long term steroid withdrawal (in someone without Addisons).
- Presentation – reduced GCS, reduced BP, hyponatraemia, hyperkalaemia, hypoglycaemia
- Management – fluid resus, IV **hydrocortisone**, **magnesium glycinate**, **insulin dextrose infusion**

Hyperaldosteronism + Conn's Syndrome

- Hyperaldosteronism = high aldosterone
- Conn's syndrome = adrenal adenoma (producing aldosterone)
- RAAS system
    - Juxtaglomerular cells detect BP, secrete renin in response to low BP
    - Renin converts angiotensinogen into angiotensin 1
    - ACE (produced by lungs) converts angiotensin 1 → angiotensin 2
    - Angiotensin 2 increases BP, but also increases adrenal secretion of Aldosterone
    - Aldosterone increases BP
- Sx:
    - Hypertension
    - Hypokalaemia (increased excretion in distal tubule)
    - Metabolic alkalosis (increased H+ excretion in collecting duct)
- Primary hyperaldosteronism causes
    - Bilateral adrenal hyperplasia (most common)
    - Adrenal adenoma
    - Familial hyperaldosteronism
- Secondary hyperaldosteronism causes - excess renin due to:
    - Renal artery stenosis
    - Heart failure
    - Liver cirrhosis + Ascites
- Ix: **Aldosterone:Renin ratio (ARR)**, CT/MRI to look for adrenal tumour, renal artery imaging (doppler), adrenal vein sampling (identify which adrenal gland is making more aldosterone)
    - High aldosterone + **low** renin = primary

    - High aldosterone + **high** renin = secondary
- Rx: Aldosterone antagonists, e.g. **Eplerenone, spironolactone**

Pheochromocytoma

- Adrenal tumour secreting unregulated and excessive catecholamines (inc. adrenaline)
- Chromaffin cell tumour
- Typically secretes in bursts, giving intermittent episodes of symptoms
- Causes: Idiopathic, MEN2, Von hippel-lindau disease
- Sx: "fight or flight"
    - Anxiety
    - Sweating
    - Headache
    - Tremor
    - Palpitations
    - Hypertension
    - Tachycardia
- Ix: 24/hr urinary collection of metanephrines (breakdown product of catecholamines) + plasma free metanephrines, CT/MRI to look for tumour
- Rx: Alpha-blockers e.g. **Phenoxybenzamine**/**Doxazosin**, beta blockers 2nd line
    - Definitive management = surgery, but symptoms are controlled medically to reduce anaesthetic + surgery risks

Acromegaly

- Cause – almost always pituitary adenoma
- Sx: enlarged hands, increased shoe size, excessive sweating + oily skin, headaches
- Ex: bitemporal hemianopia, coarse facial features, hypertension
- Ix – Insulin-like growth factor 1 (IGF-1), oral glucose tolerance test with GH measurement (should suppress growth hormone). MRI brain, ophthalmology (visual field testing)
- Management – somatostatin analogues and dopamine agonists (**Bromocriptine**), surgery
- Complications: hypertension, diabetes, cardiomyopathy, colorectal cancer

SIADH

- Increased release of ADH increased water reabsorption from urine dilution, and **hyponatraemia**
- Causes: increased secretion from posterior pituitary OR ectopic e.g. small cell lung cancer
    - Increased secretion can be caused by: post operative post major surgery, lung infection, head injury/stroke, medications (**SSRIs** and **carbamazepine**) HIV
    - **TOP 3 = 3Ss – Surgery, SSRIs, Small cell lung cancer**
- More water reabsorption more concentrated urine **HIGH URINE OSMOLALITY + HIGH URINE SODIUM** (because concentrated!! Not because excreted)
- Sx:
    - Headache
    - Fatigue
    - Muscle aches/cramps
    - Confusion
    - Severe hyponatraemia seizures + decreased GCS
- Ix:
    - BP – normal
    - U&E – hyponatraemia
    - Low serum osmolality + high urine osmolality
    - High urine sodium
    - Exclude other causes of hyponatraemia:
        - Short synacthen test – exclude adrenal insufficiency
        - Diuretic overuse
        - Diarrhoea, vomiting, burns, fistula, sweating
        - Excessive water intake – primary polydipsia LOW URINE SODIUM AND OSMOLALITY
        - CKD/AKI, heart failure/liver disease
    - If no cause identified – malignancy needs excluding, CTAP, MRI head etc

- Rx: admission if serum Na+ <125
    - Treat cause
    - Fluid restrict e.g. 1.5L
    - ADH receptor antagonists – e.g. **tolvaptan**
    - Sodium must be corrected SLOWLY to prevent **OSMOTIC DEMYELINATION**

Osmotic demyelination syndrome

- Complication of long term severe hyponatraemia being treated too quickly e.g. >10mmol/L per hour
- Chronic hyponatraemia causes the brain cells to have a low osmolality – if a load of sodium gets added into circulation water will rapidly shift across the blood brain barrier into the blood
- Causes a 2 phase reaction:
    - Electrolyte imbalance (between brain and blood) encephalopathic + confused, headache, vomiting, seizures
    - Demyelination of neurones especially in the pons quadriparesis, pseudobulbar palsy, death
- Rx: not much can be done, very very important that you don't do this by accident trying to treat hyponatraemia (I'm looking at u alex karev)
- similarly for potassium - 10mmol/hr max infusion rate

Diabetes Insipidus – diabetes but ADH not insulin

- CRANIAL vs NEPHROGENIC
    - Lack of ADH = Cranial Diabetes Insipidus
        - Brain tumours/injury/surgery/infections
        - Genetic mutation in the ADH gene (autosomal dominant)
        - Wolfram syndrome (diabetes insipidus, optic atrophy, deafness, diabetes mellitus)
    - Lack of response to ADH = Nephrogenic diabetes insipidus
        - DRUGS – lithium especially
        - Genetic mutation in ADH receptor gene (x-linked recessive)
- In either case – kidneys are unable to reabsorb water and concentrate the urine, resulting in:
- Sx:
    - Polydipsia
    - Polyuria
    - Dehydration
    - Postural hypotension
- Ix:
    - Low urine osmolality
    - High/normal serum osmolality
    - >3L urine per 24hr
    - Water deprivation test (**desmopressin** stimulation test)
        - Patient avoids all fluids for 8 hours urine osmolality measured
        - IF LOW give **desmopressin** (synthetic ADH) measure urine osmolality
        - Cranial – urine osmolality increases in response to ADH
        - Nephrogenic – urine osmolality is unchanged as nephron is unable to respond
- Rx:
    - Treat cause e.g. stop lithium
    - Cranial DI – **Desmopressin**
    - Nephrogenic DI –**Thiazide diuretics**, **NSAIDs**, High-dose **desmopressin**

	Urine Osmolality After Water Deprivation	Urine Osmolality After Desmopressin
Primary Polydipsia	High	Not required
Cranial Diabetes Insipidus	Low	High
Nephrogenic Diabetes Insipidus	Low	Low

Multiple endocrine neoplasia

- **Type 1= 3Ps = PARATHYROID, PITUITARY, PANCREAS**
    - Most common presentation = hypercalcaemia
- **Type 2a= Medullary Thyroid + 2Ps = PARATHYROID, PHEOCHROMOCYTOMA**
- **Type 2b = Medullary Thyroid + 1P = PHEOCHROMOCYTOMA**, also marfan-oid body habitus
- Inherited + often malignant condition
- Autosomal dominant

Parotid swelling

- Benign masses
    - Pleomorphic adenoma - most common, slow growing, red flags = skin tethering, facial nerve invasion, overlying skin ulceration, previous cancer
    - Warthin's tumour - more common in males, smokers. Red flags as above.
- Parotid gland sialadenitis - acute infection of salivary gland, rapid onset pain + swelling. Commonly Mumps.
- Parotid gland sialolithiasis - accumulation of fluid caused by stone, swelling and pain at meal times.
- Acinic cell carcinoma - malignant, rapid onset + red flags

Eyes/visual

Hypertensive retinopathy "PACCHH"
- PACCHH: AV nicking / cotton wool spots / copper wiring / hard exudates / haemorrhages

Diabetic retinopathy:
- Non-proliferative: Cotton wool spots, Microaneurysms, Blot haemorrhages, hard Exudates
- Proliferative: CoMBE + Vitreous haemorrhage, Neovascularisation (new blood vessels)

Macular degeneration: Both 'DAD', wet 'NO'
- Both (wet and dry): Drusen, Atrophy of retinal epithelium, Degeneration of photoreceptors
- Wet 'NO': new vessels, oedema

Optic neuritis
- Colour desaturation, painful eye mvmnts
- Ix: MRI (MS)
- Rx: **high dose steroids**

Keratitis = inflammation of the cornea, urgent + sight threatening
- Inflammation of the cornea
- Bacterial = staph aureus / pseudomonas in contact lens wearers
- Amoebic = contaminated water, acanthamoebic keratis, extreme pain
- Red eye, photophobia, foreign body/gritty sensation
- Sx: pain, foreign body sensation
- Ex: Hypopyon, red eye
- Rx: Urgent ophthalmology referral, **quinolone** first line abx

Scleritis
- Severe pain worse on movement
- Associated with autoimmune conditions e.g. RA
- Rx: Urgent ophthalmology referral - oral **NSAIDs/Glucocorticoids**

Acute angle closure glaucoma
- RISK Fx: Hypermetropia, pupillary dilatation, lens growth associated with age
- Severe pain, ocular/headache
- Decreased visual acuity, halo
- Semi-dilated pupil
- Hazy cornea
- Rx: lie them back on back without pillow, **pilocarpine** (parasympathomimetic, contracts cilia → opens trabeucular meshwork → increased outflow of aqueous humour), **timolol** (decrease aqueous humour production), **IV acetazolamide**. Definitive Rx = Laser Peripheral

Corneal abrasion
- trauma to the corneal epithelium
- Sx: pain, lacrimation, foreign body sensation
- Ix: Fluorescein staining
- Rx: topical abx to prevent secondary infection

Conjunctivitis
- Viral: serous, unilateral
- Bacterial: gunky, bilateral, gritty, eyes get stuck together. Rx: eye drops (abx)
- Allergic: watery, itchy, gritty.
- Rx: abx and steroid (for bacterial)
- In <1 month old – think ***gonococcal***

Episcleritis	Scleritis
- Section of sclera - Mild tenderness - Unilateral - Benign, self-limiting	- VERY red - Worse on movement, deep pain - Photophobia, visual disturbance - Rx: NSAIDs, steroids, immunosuppressants,

Subconjunctival haemorrhage
- Excess pressure (coughing, sneezing)
- Resolves spont

Anterior uveitis
- Ciliary flushing (ring around iris), weird pupil, hypopyon (fluid level)
- Whole eye is red, but the 'ciliary flushing' is a VERY red ring around the eye
- Pain, red eye Blurred vision, photophobia, floaters
- Rx: **steroids**

Corneal ulcer
- Infx, dry eye syndrome, trauma (scratch, injury)
- EMERGENCY

Herpetic simplex keratitis
- Keratitis caused by HSV
- Most common cause of blindness

Retinal vein occlusion
- Tomato ketchup splash

Retinal artery occlusion
- Cherry red spot
- Treatable cause: giant cell arteritis – high dose steroids

Retinal pigmentosa
- Night blindness

Argyll Robertson - "prostitutes pupil" (accommodates but does not react + caused by syphilis)
- Classic pupillary syndrome, commonly seen in neurosyphilis
- ARP present, light reflex absent
- Sx: small irregular pupils
- Can also be caused by diabetes

Holmes-Adie
- parasympathetic
- one side dilated pupil + excess sweating
- absent achilles tendon reflex

Gastrointestinal

Gastroenteritis

- 1-6 hrs: ***Staphylococcus aureus*** (sudden onset N, V, abdo cramps), ***Bacillus cereus*** (reheating rice, <6h for V)
- 12-48 hrs: ***Salmonella*** (severe V, high fever)**,** ***Escherichia coli (most common cause of bloody diarrhoea)***
- 48-72 hrs: ***Shigella, Campylobacter*** (prodrome, bloody diarrhoea, incub period 1-6d, MOST COMMON UK)
- 7 days: **Giardiasis, Amoebiasis (cysts on microscopy)**
- **C diff** – associated with hospital
- Ix: faecal MC&S, ova/cysts/parasites, PCR for virus, C diff toxin
- **Rx** – supportive rx in first 48h (cos can be viral, self-resolves)
    - ***E coli* – ciproflox**, **azithromycin** - most common cause of traveller's diarrhoea
    - ***Salmonella, shigella, campylobacter*** (blood diarrhoeal, most common) – **ciproflox**, **azithromycin**
    - ***Clostridium toxin* – metronidazole**, **vancomycin**, **bezlotoxumab** (preventative)
- Complications: Guillain-Barre after Campylobacter, RA, septicaemia, endocarditis, arthritis

Viral	Bacterial	Protozoa
- Acute onset - Profuse D, V - Early recovery Ix: antigen detection, serology Rx supportive (oral rehydration therap) Norovirus – CLASSIC bad for 2 days, then stops Rotovirus – bad for a week	Shigella/campylo/salmonella - 3-10 days - Rx: **ciproflox, azithromycin** ***C diff*** – days to weeks after abx - Pseudomembranous colitis - Gross yellow plaques on toxic megacolon = SPECIFICALLY C DIFF - **Metronidazole, vancomycin**	Incubation: 2-4w Sx: weeks and weeks Amoebiasis, Giardia

IBS

- ABC: abdo pain, bloating, change in bowel habit
    - Abdo pain/discomfort associated with bowels/better on pooping AND 2 OF other sx (mucus, worse after eating, bloating etc)
- >6months
- Rx: **loperamide** for D, bulk laxatives for C (**ispaghula husk**), antispasmodics (**buscopan**) for cramps

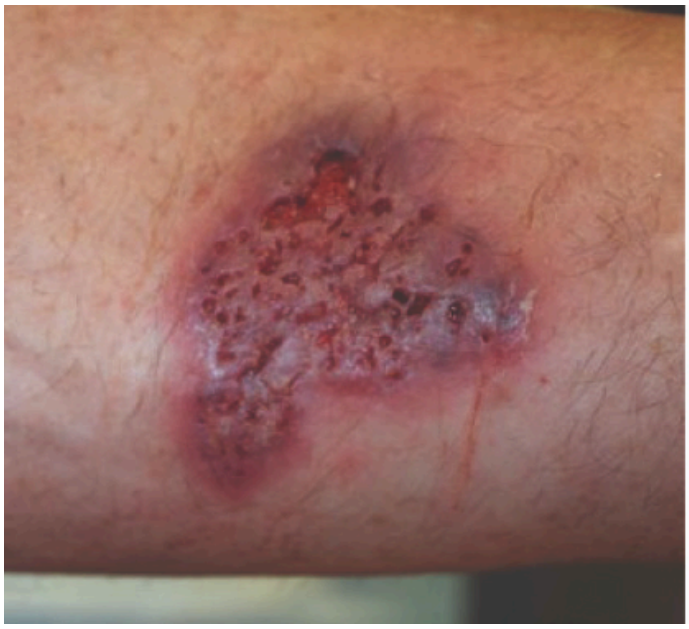
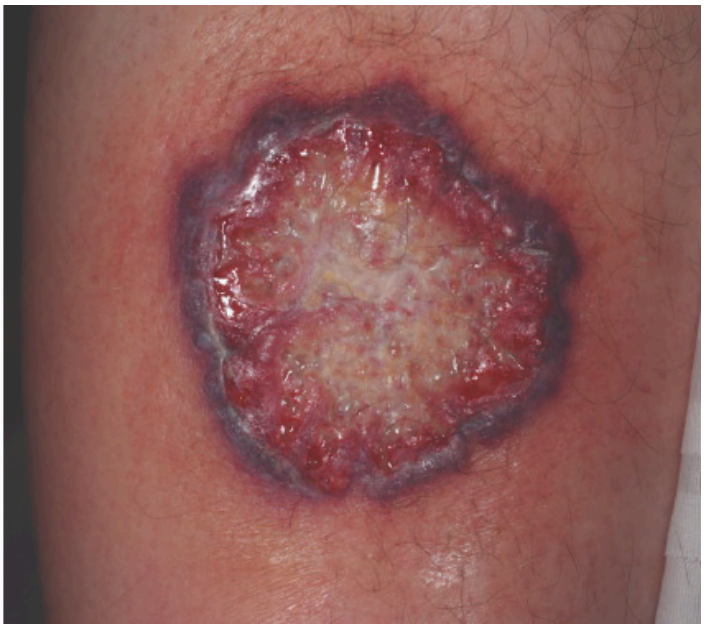
Coeliac disease

- Chronic autoimmune condition triggered by gluten
- Causes: villous atrophy & malabsorption
- Pathophys:
    - Normally – gliadin (antigen in gluten) normally binds to IgA in muscular mucosa and eliminated.
    - Coeliac – gliadin recognised by activated by TTG as a 'foreign antigen' □ autoantibodies created and immune system activated (macrophages, B, T cells) to attack gliadin
    - Ends up attacking epithelial cells of intestine □ inflammation
- HLA-DQ2
- Auto-antibodies: anti-tissue transglutaminase (anti-TTG) and ant-endomysial (anti-EMA)
- Sx: diarrhoea, weight loss, anaemia (from iron, B12, folate def), steatorrhea, abo pain, bloating, mouth, ulcers, dermatitis herpetiformis (gliadin binds to IgA in skin □ localised skin rash, intensely itchy + knees & shins)
- Ix:
    - RBC (low Hb, MCV, B12, ferritin, raised RCDW)
    - Blood film – Howell-jolly bodies and target cells (hyposplenism)
    - IgA (exclude IgA def), anti-TTG, anti-EMA
    - Endoscopy and duodenal biopsy – villous atrophy, crypt hyperplasia
- Rx: gluten-free diet

- Complications: nutrition def, anaemia, reduced bone mineral density (can't absorb calc/vit d), hyposplenism/asplenism, malignancy (Hodg, Non-Hodg, small bowel adeno, panc ca), derm herpetiformis
    - Hyposplenism – increased risk of infx from ***Strep pneum***, ***H influenza***, ***Neisseria meningitidis***

Ulcerative colitis

- Distal colon – spreads proximally. Superficial, only affecting mucosa. Abdominal pain and bloody diarrhoea more common in UC than Crohn's. Mucus – more common in UC than Chron's – pathological – stringy + looks like snot, white/green.
- XR: lead-piping (loss of haustra), **toxic megacolon** – life threatening complication of UC
- Associations: HLA B27 stuff (ankylosing spondylitis, erythema nodosum, enthesitis, episcleritis), pyoderma gangrenosum, primary sclerosing cholangitis
- Ix: faecal calprotectin (produced by degranulating white cells), flexible sigmoidoscopy, colonoscopy/endoscopy + biopsy (DIAGNOSTIC)
- Rx: 1ST line: **AMINOSALICYLATES (mesalazine, sulfasalazine (posh aspirin))**, 2nd line: **corticosteroids** 3rd line: **azathioprine** (TPMT testing before starting) and methotrexate, **4th line: biologics (infliximab anti-TNF-alpha), monoclonal antibodies.**
    - Maintain remission: **aminosalicylates, azathioprine, mercaptopurine. Budesonide** as long term steroid therapy.
    - Definitive rx: colectomy
- Complication: bowel cancer
- Flare up
    - Severity – categorised mild-severe, based on number of stools passed (>6 = severe) and signs of systemic unwellness (pyrexia, tachycardia, anaemia)
    - mild-moderate - first line = rectal mesalazine
    - severe - admit to hospital for IV corticosteroids
- Extent – proctitis left-sided colitis extensive colitis/pancolitis

Pyoderma gangrenosum - characteristic blue edge

Crohn's

- Transmural – fistulae more common, perianal, biliary
- Cramping more common – small bowel more likely to experience frequency with cramping.
- Signs of malabsorption and malnutrition (affects ileum) – B12 and iron commonly
- Associations: pyoderma gangrenosum, erythema nodosum
- Small bowel imaging – thin lines spanning entire width of bowel – valvulae conniventes
- Rx:
- Cons:
    - MDT involved – IBD nurses, gastro, surgeons, dietician
    - Modifiable lifestyle factors
- Med:
    - ACUTE: **STEROIDS (budesonide, prednisolone oral, or IV hydrocortisone)**,
    - 1st: **anti-metabolite adjunct** e.g. **azathioprine (bone marrow insufficiency), mercaptopurine**
    - 2nd: **methotrexate, infliximab, adalimumab**
    - Long term high dose steroids – PPI cover is needed, gastritis, osteoporosis are side effects of long term steroid use, diabetes, avascular necrosis of the femoral head
    - More specific e.g. targeted biologics will cause less issues with opportunistic infections
    - If giving **IV hydrocortisone** for severe flare – may also consider abx (**IV co-amoxiclav**). Preferably don't want to be giving IV steroids for more than 72hrs.
- Surg:
    - repair strictures/fistula, resection of distal ileum)
    - Anastomotic leak = big risk for peritonitis and abdominal sepsis
- Gastro-protective therapy for upper GI disease – **PPIs**
- Complication: bowel cancer, toxic megacolon
- Classified by activity – mild-severe, and where. E.g. moderate ileocecal disease

Toxic Megacolon

- Causes:
    - Infection – c diff commonly
    - Inflammation – crohn's, UC
    - Ischaemia – ischemic bowel, haemolytic uraemic syndrome

Bowel cancer

- Fourth most common cancer (breast, prostate, lung, bowel)
- RF: diet (red meat, low fibre), obesity, smoking, sedentary, genetic (FAP, HNPCC/Lynch syndrome – Amsterdam criteria), IBD
    - Familial adenomatous polyposis (FAP) and Lynch – both aut dom
- Sx: change bowel habit (increased frequency + loose stools 80%), rectal bleeding, unexplained abdo pain, iron def anaemia, abdo/rectal mass on exam, **obstruction** (20%)
- Screening – 60-74, FIT test 3 yearly. One off flexi-sig at 55 years
- 2ww
    - Over 40 years with abdominal pain **and** unexplained weight loss
    - Over 50 years with unexplained rectal bleeding
    - Over 60 years with a change in bowel habit or iron deficiency anaemia
- Ix: FIT (human Hb in stool), endoscopy (colonosc, sigmoidosc), staging CT TAP, **CEA** (predicting relapse) PETCT – liver is common area of metastasis,
- Staging: TNM
- Rx: chemo/radio/surg resection (all the different types of colectomy incl Hartmann's)
    - Hartmann's Procedure – usually as an emergency following perforation, creates colostomy that can later be reversed.
    - Loop colostomy – to rest an area of the bowel

Jaundice

- Pre – haemolytic anaemias (hereditary spherocytosis, G6PD)
- Hepatic – hepatitis, malignancy, cirrhosis, Gilbert's syndrome
- Post – PSC, PBC, ascending cholangitis, pancreatic cancer

Bowel obstruction

- 3,6,9 – diameter of parts of colon at which they are considered dilated – 3cm small, 6cm large, 9cm caecum.
- Causes: malignancy, adhesions, inflammation (diverticular disease, IBD), Peutz-Jegher's syndrome

Diverticular disease / Diverticulosis

- **Most common cause of PR bleed** in older adults
- Herniation of colonic muscle through the muscular wall of the colon, creates outpouching = diverticula
- Present where teniae coli is not present as the teniae coli support the bowel wall - rectum usually spared due to outer longitudinal muscle layer surrounding entire diameter
- Common with old age
- Risk fx: low fibre diet, obesity, use of NSAIDs, constipation
- Ix: colonoscopy, CT, barium enema
- Rx: increased fibre diet, bulk-forming laxative (e.g. **Ispaghula husk**, **methylcellulose**)
- Complications:
    - Diverticulitis
    - Haemorrhage
    - Fistula formation
    - Perforation and faecal peritonitis/abscess formation

Diverticulitis

- Inflammation of diverticulum - outpouching of the intestinal mucosa
- Sx: LIF pain, fever, diarrhoea, nausea/vomiting, rectal bleeding, palpable mass
- Ix: raised CRP + white cells, FBC (anaemia), group + save!!!
- Rx: **Co-amoxiclav** (at least 5 days), analgesia (avoiding NSAIDs and opioids if possible), clear liquids until 2-3 days from symptom resolution
- Complications: perforation, peritonitis, abscess, fistula, large haemorrhage

Gastro-oesophageal reflux disease

- Acid from the stomach flows into the oesophagus through the lower esophageal sphincter, esophageal lining becomes irritating, causing pain
- Oesophageal anatomy - 25cm fibromuscular tube from pharynx → stomach. Squamous epithelium (stomach has columnar which is not irritated by HCl).
- Triggers: greasy/spicy food, caffeine, alcohol, smoking, obesity, **NSAIDs**, hiatus hernia
- Sx: heartburn, acid reflux, bloating, retrosternal/epigastric pain, hoarse voice, nocturnal cough
- RFs: Dysphagia, age over 55, weight loss, anaemia
- Ix: Endoscopy +/- biopsy (oesophago-gastro-duodenoscopy), CXR (hiatus hernia), barium swallow, H-pylori testing (stool antigen, breath test, blood antibody, requires 2 weeks without PPI for accurate result)
- Rx: Lifestyle fx, medication review, antacid e.g. **Gaviscon/Rennie**, PPI e.g. **Omeprazole/Lansoprazole**, Surgery if indicated

Achalasia

- Failure of oesophageal peristalsis + no relaxation of the lower oesophageal sphincter due to degenerative loss of ganglia from the oesophageal plexus
- Sx: Dysphagia with solids AND liquids, heartburn, regurgitation
- Ix: barium swallow (bird beak + grossly expanded oesophagus), CXR (wide mediastinum), oesophageal manometry = diagnostic, detects patterns of muscle activity in the oesophagus
    - Oesophageal manometry - catheter inserted into stomach via nose, remains there while patient takes small sips of water, measures pressure throughout oesophagus
- Rx: **nitrates**, **calcium channel blockers**, balloon dilation, surgery if needed (heller cardiomyotomy)

Pharyngeal pouch

- Diverticulum in the wall of the pharynx
- Sx: dysphagia, regurgitation, aspiration, neck swelling which gurgles on palpitation, halitosis
- Ix: barium swallow
- Rx: surgery

Genitourinary

Bacterial vaginosis

- ***Gardnerella vaginalis***
- Sx: discharge fishy-smelling, thin/watery, grey/white, no itch/soreness, vaginal pH >4.5
- Ix: pH, Clue cells
- Rx: **metronidazole**
- Complication: late miscarriages, pre-term birth

Trichomonas vaginalis

- ***Trichomonas vaginalis***
- Sx: Discharge yellow/green, frothy, itchy/sore, can be fishy-smelling
- Exam: strawberry cervix
- Rx: **metronidazole**, contact-tracing

Chlamydia

- ***Chlamydia trachomatis*** (gram -ve, intracellular)
- inflammation of the cervix +/- urethra in women, urethra in men
  - Uncomplicated = infection hasn't spread to the upper genital tracts
  - Complicated = infection has spread to the upper genital tracts PID in women → Fitz-Hugh-Curtis, epididymo-orchitis
- Sx:
  - F: dysuria, purulent discharge, abnormal bleeding, deep dyspareunia, pelvic pain
  - Same as F, epididymo-orchritis, reactive arthritis - keratoderma blennorrhagicum
- Ix:
  - F: vulvo-vaginal/endocervical swab (can do FCU, but NICE says swab first)
  - M: FCU
- Rx: **doxycycline** & contact tracing

Gonorrhoea

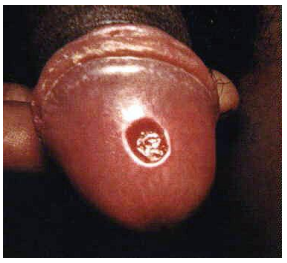
- ***Neisseria gonorrhoea***
- Can be astmptomatic
  - F – abnormal vag discharge, lower abdo pain, dusria, dyspareunia
  - M – purulent or mucupur discharge, dysuria, rectal pain (MSM)
- Complications:
  - F – PID, miscarriage, disseminated (joints)
  - M – epididiymitis, prostatitis, urethral stricture, infertility
- Ix: refer to GUM,
  - F: NAAT vulvovaginal swab in women
  - M FCU in men – NAAT
  - If NAAT positive swabs for MC&S (sensitivity and identification of resistant strains)
  - AT LEAST 3 days AFTER sex with infected person (give that shit time to grow enit)
- Rx: culture before prescribing abx (high resistance), **IM ceftriaxone** or **oral cefixime** and **azithromycin**

Candidiasis

- ***Candida albicans***
- White, curdy ("cottage cheese"), odourless, vulval itching and superficial soreness, dysuria and dyspareunia
- Rx: oral, topical or pessary antifungals (**fluconazole**)

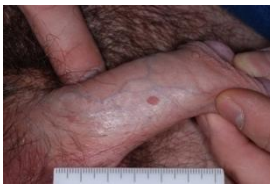
Syphilis

- ***Treponema pallidum***
- 1st: single painless ulcer (chancre), localised lymphadenopathy
- 2nd: 6-12w later, systemic sx (rash, alopecia, headaches, fatigue, fever etc)
- 3rd: neurosypihllis, gummatous, cardiovascular syphilis



- Ix: swab primary ulcer dark field microscopy or PCR, syphilis serology (+ve TPHA & +ve TPPA)
- Rx: IM **benzylpenicillin**
- Complication: Jarisch-Herxheimer reaction (flu-like symptoms)

HPV



- Transmitted sexually
- Fleshy papule genital wart
- Non-specific sx: abnormal bleeding (IMB, PCB, PMB), discharge (blood, mucus), dyspareunia
- Cryotherapy

Anterior abdominal wall hernia

- Epigastric – within the linea alba, weakening in the epigastric region
- Umbilical – skin closes, muscle doesn't. Paraumbilical can happen in pregnancy. Most common in neonates congenitally (e.g. omphalocele)
- Semilunar/spigelian hernia ultrasound is diagnostic
- Incisional

Inguinal hernia

- Landmarks
    - **Deep inguinal ring** = midpoint of inguinal ligament
    - **Superficial inguinal ring** = split in the external oblique aponeurosis, **superomedial** to the pubic tubercle
- Inguinal canal borders: MALT – roof=muscles (internal oblique, transversus abdominis), anterior=aponeurosis (internal oblique, external oblique), floor= ligaaments (inguinal ligament, lacunar ligament) posterior = transversalis fascia
- Do not cause sx of strangulation
- Direct inguinal hernia hesselbach's triangle (rectus abdominis muscle, inferior epigastric vessels, inguinal ligament)
- Indirect inguinal hernia superficial ring testes
- Exam: **SUPEROLATERAL to pubic tubercle**, cough (press on deep inguinal ring)
    - Direct: reducible, appears on cough impulse (increased intra-abdominal pressure), always acquired, 12% bilateral at presentation
    - Indirect: doesn't emerge on cough, may be present in the scrotum, most common, affecting neonates often, caused by similar mechanism of hydrocele = patent processus vaginalis
- Management – conservative = watch and wait, TRUSS. Surgery – tension free repair (mesh)/tension repair
- Groin lump differentials:
    - Saphena varix
    - Psoas abscess
    - Lymph node
        - TB ??????????? why is it always TB
    - Femoral aneurysm
    - Ectopic testes
    - Dermoid cyst
    - Lipoma
    - Arterial aneurysm

Femoral hernia

- F>M, connective tissue
- Femoral canal borders: antero-superior = inguinal ligament, posterior = pectineal ligament, medial = lacunar, lateral = femoral vein
- Sx: lump in groin, mildly painful
- Exam: non-reducible, **inferolateral to pubic tubercle**, erythema of overling skin
    - Strangulated: vomiting, bloody stools, toxic appearance (=sx of necrosis)
    - Incarcerated: non-reducible, risk of strangulation but not yet lost their blood supply
- Ix: USS

- Rx: lap surgery for decompression
- Complications: incarceration, strangulation (SURG EMERGENCY), bowel obstruction (SURG EMERGENCY) > bowel ischaemia

	Indirect inguinal hernia	**Direct inguinal hernia**	**Femoral hernia**
Anatomy	Protrudes through the inguinal ring Passes lateral to the inferior epigastric artery **Superolat** to PT	Protrudes through Hesselback triangle Passes medial to the inferior epigastric artery **Superolat** to PT	Protrudes below the inguinal ligament, **inferolateral** to the pubic tubercle
Cause	Failure of the processus vaginalis to close	Defect or weakness in the transversalis fascia area of the Hesselbach triangle	
Risk of strangulation	Low risk of strangulation	Low risk of strangulation	High risk of **strangulation**
Age	May occur in infants	Seen in adults	Seen in adults
Sex	Much more common in males	Much more common in males	More common in females (pregnancy)
		Reduce easily	

Hydrocele

- Fluid in tunica vaginalis
- Causes: congenital (paeds), acquired (adult), non-communicating (no comm between scrotum and abdo / trauma, infx, torsion, cancer etc), communicating (patent proc vaginalis, opens with intra-abdo pressure)
- Sx/exam: painless, soft, non-tender, anterior & below testicle, transilluminates, can get above nassm common in neonates
- Ix: USS in 20-30y because 10% testicular ca thought to present with hydroceles
- Rx: self-resolves, aspiration, surgery

Varicocele

- Dilated pampiniform venous plexus. L side.
- Asymptomatic, but associated with infertility.
- Pathophys: ++hydrostatic pressure L renal vein > ++ venous pressure > compressed by SMA > dilation of pampiniform >swelling
- Sign: bag of warms, painless, non-tender (dull, dragging), disappears on lying, reappears on standing,
- Ix: USS doppler
- Rx: low-grade – reassurance. High grade – lap/percut repair

Testicular cancer

- Types: seminomas (most common), teratomas
- Risk factors: undescended testis (cryptorchidism, even if repaired), infertility, mumps orchtis, infant hernia, 20-40y
- Sx: hard painless nodule on testis, gynaecomastia (increased oestrogen:androgen) (+ other cancery sx: back pain, blank pain, lymphad, weight loss etc)
- Ix: USS doppler (confirms), CXR/CT TAP (for staging/mets), excision biopsy, tumour markers
    - bHCG = seminoma
    - LDH = seminoma
    - AFP = teratoma
- Rx: orchidectomy, store sperm, (chemo/radio if mets)

Spermatocele/epididymal cyst

- Benign. Serious fluid at head of epididymis. Smooth. (Spermatocele = contains sperm)
- Associated with PKD, CF
- Middle aged. Unknown cause. Most common cause scrotal swelling seen in GP
- Sx: chronic, painless, non-tender, smooth, fluctuant, separate from body of testis, **posterior** to testicle, no transillum
- Ix: clinical, USS
- Rx: nothing. Surg excusion if large/symptomatic

Testicular torsion

- Torsion/twisting of spermatic cord. Common in 13-16y
- SURGICAL EMERGENCY. +++pain
- Risk factors: testicular tmour, testicles with horizontal lie, hx undescended, Bell-Clapper deformity (tunica vaginalis abnormal highly attached to sperm cord), trauma
- Sx: sudden onset PAIN, unilat, pain while walking, N/V
- Exam: firm, hard, non-transillum, negative Prehn sign, absent cremast reflex (L1/L2)
- Ix: no time, straight to surgery (could do USS doppler, but probs won't)
- Rx: UROLOGICAL EMERGENCY, surg expoloration of scrotum, orchiopexy (correct position of testicles, fix in ppro), orchidectomy if surg delayed or necrosis

Epididymo-orchitis

- Inflamm to both epididymis and testicle, can reduce fertility
- Usually cused by **STI (*chlam*, *gono*)**, UTI (***E coli)***, ***mumps*** or **amiodarone**
    - Pre-pubertal tends to be non-infective and self-limiting (?reflux urine into ejac ducts?)
- Risk factors: sex, immunocomp
- Sx: unilat pain/swelling over hours/days, PAINFUL, TENDER, LUTI sx, fever/sweats/N/V etc
    - Parotid swelling suggests mumps
- Exam: palpable swelling of epidid +/- testis, positive Pregn sign, tender
- Ix: FCU sample for urine MC&S, STI screen
- Rx:
    - STI – **ceftriaxone** AND **doxycycline** AND refer to GUM – doesn't matter what type STI
    - Severe (e.g. immunocomp), admit + refer to sex health specialist
    - Supportive (NSAIDs, rest, scrotal support, **steroids**)

Haematology/immunity

Microcytic anaemia	Normocytic anaemia	Macrocytic anaemia
• Iron Deficiency (chronic bleeding) – hookworm! • Chronic disease • Thalassemias • Haemoglobinopathies • Sideroblastic Anemia	• Chronic disease • Acute blood loss • Primary marrow disorders • Combined deficiencies • Haemolysis • Renal failure	• B12/folate deficiency • Liver disease/alcohol • Marrow disorders • Reticulocytosis • Drugs (azathioprine)

Haemolytic anaemia

- Haemolysis leading to anaemia
- Hereditary or acquired conditions that make RBCs more fragile and break down faster chronic haemolytic anaemia
- Ix: normocytic anaemia, **schistocytes** on blood film, positive **Direct Coombs test**, **decreased haptoglobins, elevated LDH**
- Acquired
    - Autoimmune – antibodies attack RBS.
        - Warm type – more common, occurs at normal/high temps
        - Cold – cold temps cause RBC agglutination – causes destruction, usually caused be malignancy, viral infx, or autoimmune condition (SLE)
        - Rx: **transfusions, steroids, ritxumab**, splenectomy
    - Alloimmune – **transfusion reaction, haemolytic disease of newborn**
        - Ix: normocytic anaemia, **schistocytes** on blood film, positive **Direct Coombs test**, **decreased haptoglobins, elevated LDH**
- Inherited - membrane, metabolism, Hb
    - **G6PD deficiency** –
        - x-linked recessive. African, meditarranean
        - triggers: infxs (e.g. malaria), meds (e.g. anti-malarials, quinolones), **fava (broad) beans**
        - Sx: jaundice, gallstones, anaemia, splenomegaly
        - Ix: Heinz bodies (blood film), G6PD enzyme assay
- **Autoimmune** – autosomal dominant
        - Haemolytic disease
    - **Hereditary spherocytosis - membrane**
        - Autosom dom. Sphere shaped RBCs – fragile
        - Sx: jaundice, gallstones, splenomegaly, failure to thrive,pu aplastic crisis if parvovirus
        - Ix: FBC = normocytic anaemia, raised MCHC (++ reticulocytes), raised reticulocytes
        - Rx: folate, splenectomy (maybe cholecystectomy if gallstones problem)
    - **Hereditary elliptocytosis - membrane**
        - Same as sphero. Ellipse-shaped.
- Abnormal haemoglobin
    - **Sickle cell**
    - **Thalassaemia**
- General features
    - Splenomegaly
    - Jaundice
    - Anaemia

Sickle Cell Anaemia

- Genetic condition (autosomal recessive) causing crescent shaped RBCs, abnormal shape is more fragile, causes haemolytic anaemia
- Sickle cell trait (one copy) reduces severity of malaria, selective advantage makes sickle cell trait more common in endemic areas
- Screening = newborn bloodspot, 5-7 days from birth
- Sickle cell crises

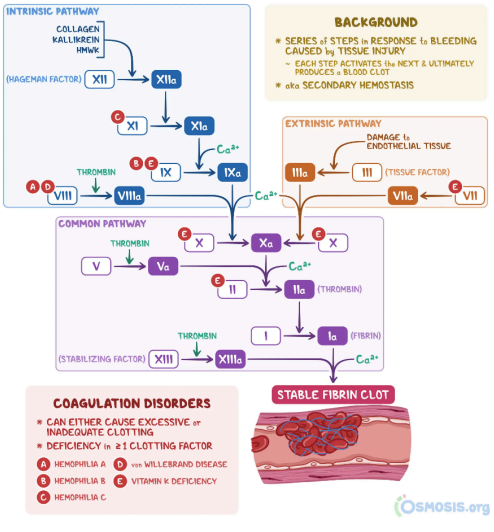
    - Vaso-occlusive - painful, usually affects peripheries, can cause priapism in males
    - Aplastic - commonly triggered by infection e.g. **Parvovirus B19**
        - Rx: supportive
    - Splenic sequestration - acutely enlarged and painful spleen, severe anaemia and hypovolemic shock
        - Rx: splenectomy
    - Acute chest syndrome - can be triggered by vaso-occlusive crisis, like a PE
        - Ix: CXR showing pulmonary infiltrates
        - Rx: analgesia, Abx/Antivirals, incentive spirometry (machine encouraging deep + effective breathing), resp support (oxygen/NIV)
- General management of sickle cell:
    - avoid triggers for crises e.g. dehydration, viral/bacterial infx
    - up to date vaccinations
    - Abx prophylaxis e.g. **Penicillin V**
    - **Hydroxycarbamide** ⟶ stimulates production of HbF
    - **Crixanlizumab** ⟶ targets P selectin, reduces adherence of RBCs to blood vessel wall ⟶ prevents vaso-occlusive crises

Thalassaemia

- Autosomal recessive, defect in globin chains ⟶ thalassemia
- Defect in alpha globin chain ⟶ alpha-thalassemia
- Defect in beta-globin chain ⟶ beta-thalassemia
- Defective red cells more susceptible to breakdown, causing haemolytic anaemia and splenomegaly
- Sx: Vary based on type, universal features:
    - Fatigue, Pallor
    - Jaundice (haemolysis)
    - Gallstones
    - Splenomegaly
    - Poor growth + development
- Ix: FBC (**microcytic anaemia**), raised ferritin (iron overload), **haemoglobin electrophoresis** = diagnostic
- Alpha-thalassaemia - **chromosome 16**
    - Carrier = asymptomatic
    - Haemoglobin H disease = moderate sx
    - Alpha-thalassaemia major = intrauterine death
- Beta-thalassaemia - **chromosome 11**
    - Thalassaemia minor - heterozygous - mild microcytic anaemia
    - Thalassaemia intermedia - heterozygous, one deletion gene, one other defective variant - microcytic anaemia, may require transfusion + iron chelation to prevent overload
    - Thalassaemia major - homozygous for deletion gene - severe anaemia + failure to thrive
        - Frontal bossing (prominent forehead)
        - Enlarged maxilla
        - Depressed nasal bridge
        - Protruding upper teeth
        - Rx: regular transfusion, iron chelation, splenectomy. Bone marrow transplant can be curative.

Normal Haemostasis

- Primary – local vasoconstriction, formation of primary platelet plug
- vWF helps with formation of initial clot formation
- Secondary – clotting cascade □ stable clot
    - Main aims =
        - Prothrombin □ thrombin
        - Fibrinogen □ fibrin
    - Extrinsic pathway (1972) □ prothrombin time
    - Intrinsic pathway □ activated partial thromboplastic time



Von Willebrand's

- Autosomal dominant. Abnormality, deficiency of vWF
- Pathophys: vWF protects factor VIII from degradation, therefore VWD less factor VIII prolonged aPTT
- Bruising, menorrhagia, nose bleeds, post-op bleeding, endomeotriosis, PPH
- Fmhx v. important
- Ix: PROLONGED aPTT (but can be normal), vWF antigen
- Rx: avoid aspirin and NSAIDs / **desmopressin** (**stimulates vWF**) or **vWF concentrate**
- Rx **menorrhagia**: **tranexamic acid**, **COCP**

Haemophilia

- X-linked recessive
- Haem A (**A** sound in 8) – fVIII, more common, more mild
- Haem B – fIX, more severe
- More MSK bleeding – bleeding in joints (but can also be excessive bruising, menorrhagia, fatigue)
- Ix: prolonged aPTT and FVIII/IX low
- Rx:
    - avoid blood thinners (NSAIDs, aspirin)
    - Haem A: **desmopressin** ; stimulates release of vWF which protects from breakdown of deficient factor
    - Tranexamic acid – when needed (e.g. trauma/surgery); prevents breakdown of fibrin (improves clotting)
- Can be acquired later in life – autoimmune disorder (antibodies to factor VIII) affecting elderly people, bleeding is often into soft tissue

Thrombophilia

- Factor V Leiden – mutation in gene for V -
- Protein C & S deficiency – degrade V and VIII stabilise clotting
- Antithrombin deficiency
- Prothrombin gene mutation

Polycythaemia vera

- Rare blood (myeloprolif) disorder
- Increases ALL blood cells esp RBCs
- Pathophys: TOO MUCH PROLIFERATION OF MARROW STEM CELL increased volume, overprod (neutrophils, platelets) increases risk of MI, stroke
- Sx: Hyperviscosity, pruritus, splenomegaly
- Ix: high Hb, high platelets, high WBCs – **JAK2 mutation**
- Rx: **low dose aspirin**, **venesection** (keep Hb in range)
- Complication → **AML**

Idiopathic thrombocytopenia purpura

- Mild-severe bleeding in otherwise normal FBC, absence of organ dysfunction
- Sx: petechiae, purpura, haemorrhagic blister/bullae = mucosal feature
- Treatment = corticosteroids

Pseudothrombocytopenia

- Due to agglutination of platelets
- Associated with use of EDTA as an anticoagulant

TTP – thrombotic thrombocytopenia purpura

- Deficiency of ADAMTS13 (a metalloprotease enzyme) which breakdowns ('cleaves') large multimers of von Willebrand's factor
- Pentad of features
    - fever
    - fluctuating neuro signs (microemboli)
    - microangiopathic haemolytic anaemia – schistocytes
    - thrombocytopenia

    - renal failure
- causes: infection, pregnancy, tumours, SLE, HIV, drugs (cyclosporins, combined pill, penicillin, clopidogrel)

Other thrombocytopenia causes:

- Moderate: Heparin induced, alcohol, liver disease, hypersplenism, viral infection, pregnancy, SLE/antiphospholipid syndrome, B12 deficiency
- Severe: DIC (sepsis), haematological malignancy, chemotherapy (causes aplastic anaemia)

Heparin induced thrombocytopenia

- 5% of patients exposed to heparin products, greater for unfractionated heparins
- Immune complex forms between platelet factor 4 and antibodies
- IgG mediated Immune response platelets aggregated and removed by spleen thrombocytopenia AND hypercoagulable state (aggregated platelet is basically a clot)
- DVT, PE, skin necrosis

Antiphospholipid syndrome

- Autoimmune condition that makes blood very clotty (**idiopathic** or **secondary to SLE**)
- Pathohys: Causes raised anti-phospholipid ANTIBODIES inhibits protein C and activates thrombin
- Sx: venous and arterial thrombosis, miscarriages, difficulty conceiving, splinter haemorrhages
- Ix: anti-beta2 glycoprotein 1, cardiolipin antibodies, lupus anticoagulant, prolonged APTT
- Rx: refer to rheum & haem, primary prevention **aspirin**, secondary prevention LT **warfarin** (indicated in APS over DOAC)

Multiple myeloma:

- Disease of plasma cells ++B lymph producing ++ antibodies → suppression of normal cell production
- plasma cells produce immunoglobulin light chains which damage kidneys → renal impairment
- Osteoclastic activation → lytic lesions, hypercalcaemia
- Sx: unrelenting pain not related to posture, fever/rigors, wt loss, under 40, night pain, t spine pain
- CRAB (hyperCalcaemia, Renal impairment, Anaemia, Bone pain)
- Ix: Bence Jones protein, serum electrophoresis (monoclonal bands – antibodies),

Common variable immunodeficiency (CVID)

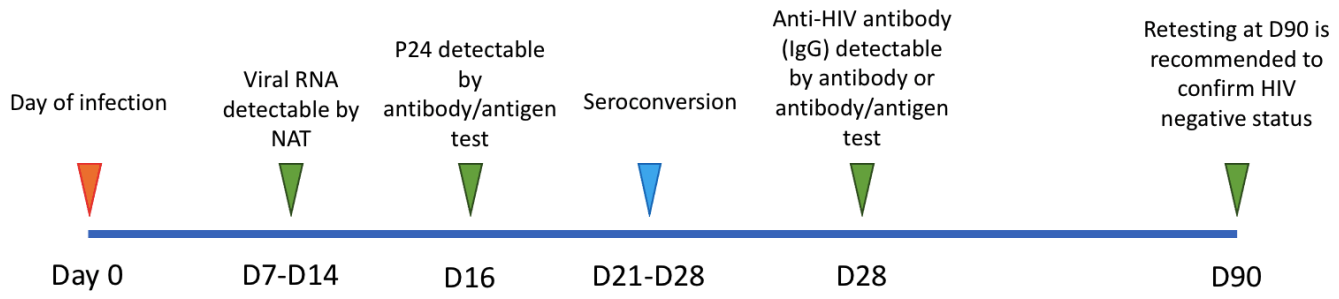
- Low immunoglobs (usually IgA)
- Recurrent infections – usually ears, URTI and LRTIs
- Ix: low Ig, fail to produce antibodies in response to vaccines

Chronic granulomatous disease

- Phagocytes (e.g. neutrophils) unable to destroy microbes (no oxidative burst)
- Persistent recurrent infections – skin infections (GRANULOMAS), abscesses, septic arthritis
- **Ix:**
    - Microscopy: nitroblue-tetrazolium (NBT) test – normal phagocytes reduce NBT to a dark pigment
    - Flow cytometry: Dihydrorhodamine (DHR) test – normal phagocytes reduce DHR to a fluorescent pigment
    - Genetic testing: identify genetic defect
- **Rx:** early rx of infx, prophylactic abx, IFN-gamma, curative = bone marrow/stem cell transplant
    - **o** Die without rx

HIV

- Cancers: Kaposi sarcoma, non-Hodgkin, cervical cancer
- Infx: TB (pulm or extra-pulm), CMV, HSV, parasitic infx (cereb toxoplasmosis, leishmaniasis), funcal (pneumocyst, candida)
    - Candida oesophagitis
    - Pneumocystis jirovecci few chest signs, desaturation on exercise
- <500 – minor infx (cold sores from HSV, fungal infx, candidiasiss)
- <200 – opportunistic infx, cancers
- Rx: 2 NRTIs and 1 other
    - PREP (for sex contact) = take 3 times (once before sex, twice after)
    - PEP (sex contact) = take within 72h of having sex, for 28 days
- HIV induced nephropathy - presents as nephrotic syndrome - focal segmental glomerulosclerosis



Neutropenic sepsis

- Temperature >38°c and/or signs of sepsis and neutrophil count of ≤0.5 x 10^9/L
- Causes
    - Congenital: Kostmann's syndrome (aut rec), Cohen syndrome (aut rec)
    - Acquired: viruses (HIV, EBV, CMV, hep B), autoimmune (destroy neutrophils Crohn's, RA, SLE), bone marrow failure (Felty (hypersplenism), leukaemia), immunosuppressive drugs (DMARDs)#
    - Chemotherapy
- Must not miss – patients have no immune response and can deteriorate rapidly
- Rx: broad spectrum abx = **meropenem**
    - High risk of MRSA = **vancomycin**

Felty's syndrome

- RA + splenomegaly + neutropenia (low WCC)
- Associated with lots of autoimmune conditions (Sjogren's, vasculitis)
- Joint pain, inflammation, decreased immune fx
- Can be life-threatening (neutropenic sepsis)

Vasculitis

- Granulomatosis with polyangitis: small vessels. **C-ANCA**, Behcet's syndrome.
    - **p-ANCA** Eosinophilic granulomatosis with polyangiitis - mononeuritis multiplex - MPO is major target within neutrophil granule
        - acquired adult onset asthma + vasculitis
- Kawasaki: medium vessels. ANCA
- Giant cell arteritis: large vessels. ANCA
- Sx: petechial rash, pupur, ulcer + peripheral necrotic changes to skin, haemoptysis

Behcet's syndrome

- Classic triad: oral ulcers, genital ulcers, uveitis
- Shin lesions - erythema nodosum
- VTE, arthritis, + abdo pain also common
- Ix: c-ANCA

Hearing

Ototoxic drugs: All Queens Love Soft Crumpets – aminoglycosides (gentamicin), Quinine, Loop diuretic, Salicylate, Cisplatin

Acute otitis media

- Strep pneum or resp virus
- Ix: bulging TM, red/yellow/cloudy TM, loss of normal landmarks, perforation/discharge
- Rx: analgesia. Abx (**amoxicillin,** 5-7 days / **clarithromycin**, **erythromycin** if pen allergic) only for people who –
    - Sx >4 days and not improving
    - Systemically very unwell
    - Signs/sx of more serious illness/conditions
    - High risk of complications
    - Otitis media + perforation +/- discharge
- Complication: perforation (otorrhoea), acute mastoiditis, meningitis, labyrinthitis, facial nerve paralysis, neur abscess
- Mastoiditis – post auricular redness/heat – managed with IV abx

Otitis media with effusion (OME) / glue ear

- NO SIGNS OF INX
- Caused by poor aeration of middle ear, low grade viral/resp infx
- Risk factors: children with deformities (cleft palate, craniofac malform), impaired immunity (DS), primary ciliary dyskinesia, smoking in house, frequent URTIs
- Pathophys: Eustachian tube gets blocked with middle ear secretions
- Sx: reduced hearing in affected ear, fullness/"popping", aural discharge
- Exam: effusion/fluid
- Rx: nothing. Maybe grommets.
- Complication: most common cause of hearing impairment, otitis media infx, S&L development probs in kids

Acute otitis externa

- Risk factors: aural irrigation with tap water, anything that fucks with immune system (DM, HIV, chemo, CKD, radiotherapy to head or neck
- Sx of acute diffuse: hearing less, fever
- Ix: swab ear only if other rx have failed
- Rx:
    - Localised: self-limiting, pain relief, warm flannel, oral abx RARELY indicated (**flucloxacillin**), drain pus if severe pain and swelling
    - Acute diffuse: clean ear, pain relief, **topical abx +/- topical corticosteroid**. Oral abx RARELY indicated.
    - Chronic diffuse: topical antifungal (**clomitrazole**), if no cause found give **corticosteroid**
- Complications: abscess, spreading infx (cellulitis, parotitis), fibrosis (stenosis of ear canal > cond deafness), myringitis, tymp mem perforation
- Malignant: facial nerve paralysis, meningitis

	Hearing loss	**No hearing loss**
Episodic	BPPV	Menieres
Persistent	Vestibular neuronitis	Labyrinthitis

BPPV

- Dix-Hallpike = exam

- Epley = treatment
- Rx: antihistamine can be used to help with nausea

Meniere's
- Fluctuating hearing loss, vertigo, tinnitus, aural fullness
- Exam: positive Romberg
- Advised not to drive
- Rx: **antiemetics**, **antihistamines**

Labyrinthitis
- Same as vestibular neuronitis PLUS SENSORINEURAL HEARING LOSS and TINNITUS
- Recent URTI

Vestibular neuronitis
- Inflamm vestibular nerve
- Recent URTI
- NO HEARING LOSS OR TINNITUS. Vertigo + associated sx (sick)
- Ex: positive head impulse test (eyes dragged off target)
- Rx: nothing. If N/V annoying – **antiemetics** or **antihistamine** – **prochlorperazine**

Acoustic neuroma
- Tumour of vestibulocochlear nerve
- RF: Neurofibromatosis type 2
- Sx: Unilat sensorineural loss, facial numbness (compresses trigeminal nerve absent corneal reflex), dizziness, difficulty honing in on voice in a crowd
- Ix: Gadolinium-enhanced MRI head
- Rx: remove it

Otosclerosis
- Stiffening of base of stapes – can't transmit sound
- Young people <40
- Low sounds go first
- Ex: normal otoscopy, Rinne positive
- Rx: hearing aids, surgery: stapedectomy (remove and replace), stapedotomy (remove part)

Glomus tumour
- ++ vascular glomus tumour in middle ear. Baroreceptor.
- Pulsatile tinnitus – hear blood (blood moves bones in ear)
- More common in toes/fingers + under nails, painful

Cholesteatoma
- Collection of squamous epithelial cells in the ME
- erodes into middle ear bones, predisposes to infx
- foul smelling, conduct hearing loss
- Ix: CT/MRI
- Rx: surgical removal

Sensorineural loss
- Alport's syndrome – collagen disorder – affects Organ of corti, eyes, kidneys hearing loss, vision problems, haematuria/proteinuria
- Viral infx (rubella, measles, mumps)
- Ototoxic drugs

Hepatobiliary

Pancreatic adenocarcinoma

- Mostly occur in head of pancreas
- Blocks duod papilla – prevents drainage from liver > **painless obstructive jaundice**
- Hx: painless obstructive jaundice, pale stools, dark urine, general itching (+ non-specific: weight loss, upper abdo/back pain, change in bowel habit, N/V, **NEW ONSET/WORSENING T2DM**)
- Ex: Courvoisier's sign (palpable mass in epigastric + jaundice unlikely to be gallstones), lymphadenopathy, Trousseau's sign = migratory thrombophlebitis (sign of malf, common in panc ca thrombus in an area that becomes inflamed)
- Ix: CT (helps staging) + biopsy / CA19-9 tumour marker / MRCP to assess obstruction in biliary system (double duct sign – dilation of pancreatic and CBD) / ERCP to put stent in + relieve obstruction + obtain biopsy / biopsy (percut under USS, CT, or endoscopy)
- Rx: MDT meeting. Surgery (remove it, Whipple), palliative
    - Whipple = pancreaticoduodenectomy
- 2ww
    - >40 with jaundice
- Direct access CT abdo
    - >60 + weight loss + one additional sx
        - (D, back pain, abdo pain, N, V, constipation, new onset DM)

Obstructive ascending cholangitis

- Reynold's Pentad = Charcot's triad + altered mental state (confusion) and shock (hypotension, tachycardia)

Acute pancreatitis

- Causes: IGETSMASHED – ethanol, gallstones, ERCP
    - Idiopathic/Ischaemia
    - Gallstones
    - Ethanol
    - Trauma
    - Steroids
    - Mumps/Malignancy
    - Autoimmune
    - Scorpion
    - Hypercalcaemia
    - ERCP
    - Drugs
- Glasgow score (PANCREAS mnemonic)
- Ix: USS (gallstones), CT abdo (looking for complications e.g. abscess), CRP, amylase 3x upper limit, Lipase MOST SENSITIVE + SPECIFIC, serum calcium = prognostic (glasgow-imrie)
- Bruising from retroperitoneal bleed - Grey turner (flank), Cullens (c shape around umbilicus)
- Rx: SUPPORTIVE. Abx **only** if abscess, necrosis. Treat complications.
- Complications: necrosis-related

Chronic pancreatitis

- Main risk factor: alcohol
- Chronic inflammation > fibrosis
- Sx same as acute, but less intense.
    - Endocrine and exocrine dsyfx: DM, loss of digestive enzymes, formation of abscesses/pseudocysts
    - Malabsorption
- Rx: stop smoking/alcohol, **CREON**, **analgesia**, stents for strictures

Primary biliary cirrhosis

- Autoimmune damage of biliary tree obstruction of bile ducts cholestasis fibrosis, cirrhosis, portal HTN
- F>M (middle aged), obese ("FFF - fat/female/fourty")
- Associated with sjogrens syndrome (80%)
- Sx: lethargy, sleepiness, pruritis / many years later: jaundice, xanthelasmata, steatorrhoea
- Ix: anti-mitochondrial antibodies, ANA, raised IgM, raised ALP, raised GGT, raised ESR / USS / biopsy
- Rx: ursodeoxycholic acid (synthetic bile acid), obeticholic acid (FXR agonist) - delay progression.
- Bilirubin >100 is indication for liver transplant
- Complications: malabsorption (ADEK) due to cholestasis, HCC

Primary sclerosing cholangitis

- Progressive cholestasis with bile duct inflammation and strictures
- Sx: pruritus, fatigue
- Associated with UC, M>F
- Associated with cancers: bile duct, GB, liver, colon
- Ix: high ALP, MRCP diagnostic

Jaundice

- Apparent when serum bilirubin >51umol/L
- Pathophs: unconjugated bilirubin binds to albumin > taken to liver > conjugated by uridine glucuronyl (UGT) enzyme > conjug bil now water soluble > converted to urobilinogen in duodenum > 80% excreted by gut and gut, 20% recycled (most goes to liver, small amount excreted by kidneys)
- Causes: pre-hep, hep, post hep:
    - Pre-hep: haemolytic anaemias (sickle cell, thalassaemia), drugs, Gilbert, Crigler-Najjar
    - Hep: viral infx, alcohol, cirrhosis, NAFLD, hepatitis, drugs, malig of biliary
    - Post-hep: gallstones, surg strictures, extra-hep malignancy (panc ca), pancreatitis, parasitic infx
- Sickle cell: sickle cells have shorter lifespan > increased heme > increased UCB
- Vit B12: ineffective erythropoiesis of bone marrow > increased bilirubin > jaundice
- SLE: impaired erythropoiesis
- **Gilbert syndrome**: genetic disorder affects metabolism – defective UGT enzyme > triggers (infx, stress, starvation, dehyd) > increased haemolysis > ++UCB in blood
- **Crigler Najjar** – no UGT > +++UCB in blood.
    - Type 1: neonate with progressive jaundice in first few days of life > untreated causes kernicterus > death. Kernicterus = collects in basal ganglia brain damage/death
    - Type 2: less severe > no brain damage
- NAFLD – excess fat accumulation in hepatocytes > can't process bilirubin > jaundice
- Drugs: **rifampicin** failure of hepatic uptake of bilirubin
- Ix: CT abdo pelvis – find out cause of jaundice

Ascites

- Contains proteins (albumin), lipids, bile acids, WBCs / serum ascites-albumin gradient (SAAG)
    - High SAAG: **T**ransuda**T**e H**T**N (= high
    - Low SAAG: Exudate ('NIMP') – **N**ephrotic syndrome, **I**nfection (TB), **M**alignancy (peritoneal, Meig syndrome), **P**ancreatitis
- Sx: abdo distension, divarication of recti, tense shiny skin, breathlessness (pulm oedema), abdo pain (tight from fluid)
- Exam: shift dullness, fullness flanks, fluid thrill, abdo striae,caput medusae, stigmata of liver disease/malignancy
- Ix:
    - thrombocytopenia, high LFTs (++ALT), prolonged PT, low albumin, amylase
    - CT abdo pelvis – to find underlying cause
    - Ascitic tap +/- culture if worried about SPONTANEOUS BACTERIAL PERITONITIS
- Rx: trans:
    - trans = keep BP down and fluid offloading – **spironolactone**, salt restriction, drain fluid + albumin infusion to maintain intravascular compartment

- exudative = **cefotaxime**
- manage any malignancy

- Complications: SBP. Hep C, alc live disease, CHF, nephrotic syndrome, pancreatitis

Stones

- **Bile ducts stones**
  - Ix: USS > MRCP > ERCP if sure need intervention
  - Complications: pancreatitis, cholangitis
  - Rx: ERCP > cholecystectomy
- **Gallstones**
  - Cholesterol or pigment
  - 4F': fat, fair, forty, female
  - Complications: acute cholecystitis, cholangitis, obstructive jaundice, pancreatitis
  - Rx: ERCP to remove gallstones if

Biliary colic

- Pain when gallstone temporarily gets stuck in neck of GB or cystic duct
- Pathophys: fat enters GI > CCK secreted from duodenum > triggers contraction of GB > biliary colic
- Sx: colicky epigastric RUQ pain (can rad go back), triggered by meals, lasts 30m-8h hours, maybe N/V
- Ix: USS (gallstones in bladder)

Cholecystitis

- Inflamm GB caused by blockage of cystic duct
- Sx: fever, N/V, tachycardia, RUQ tenderness, Murphy's sign
- Ix: raised CRP/ESR, USS (thickened GB wall, stones/sludge in GB/fluid around GB) > MRCP (visualise biliary tree) if suspect common bile duct stone but not seen on USS
- Rx: ERCP – removes stones in common bile duct OR cholecystectomy w/in 72h
- Complications: sepsis, GB empyema, gangrenous GB, perforation

Ascending Cholangitis

- Inflamm/infection of common bile duct – impacted gallstone
- Charcot triad: fever, pain, jaundice

MASLD

- Fat deposited in hepatocytes > problems with functioning > hepatitis and cirrhosis
- Stages: NAFLD > non-alco steatohepatitis > fibrosis > cirrhosis
- Risk factors (same as CVD, DM): obesity, poor diet, high chol, smoking, HTN etc
- Ix: USS liver (confirms fatty liver) > **enhanced liver fibrosis (ELF) blood test** (1st line to assess **fibrosis**) > NAFLD fibrosis score (2nd line) > fibroscan (3rd line)
  - rule out other things: hep B and C serology, autoantibodies (autoimmune hep, PBC, PSC), immunoglobulins (autoimmune hep, PBC), ceruloplasmin (Wilsons), alpha-1-anti…, ferritin & transferring (hereditary haemochromatosis)
- Rx: lifestyle (weight, exercise, smoking, DM/BP/chol control, alcohol)

Alcoholic hepatitis

- Binge drinking inflammation in hepatocytes. Long term alcoholic fatty liver and cirrhosis
- May deteriorate 1-2 weeks after abstinence
- Refeeding risk as often patients getting significant amount of their calories from ETOH
- Ix: AST:ALT = 2:1, gamma-GT elevated
- **Maddrey's discriminant functio**n = severity. 4.6 (bilirubin/17 + PT - control). >32 is severe.
- **Glasgow alc hep score**
- Rx: Supportive primarily, ETOH withdrawal, nutrition, thiamine/pabrinex, glucocorticoids if severe. **Prednisolone 40mg for 28 days** (plusssss omeprazole and adcal as always)
- Lille criteria - predicts mortality in patients not responding to steroid therapy
- ABSTINENCE - ALS service + frank discussions. **Acamprosate/Baclofen**. **Disulfiram** (CI in cirrhosis)

Chronic liver disease

- steatosis □ steatohepatitis □ fibrosis □ cirrhosis (end stage fibrosis)
- fatty-liver disease, alcohol related liver disease, hepatitis
- complications: ascites, varices (ruptured oesophageal varices must be given abx prophylaxis, **carvedilol/propranolol** long term), hepatorenal syndrome, encephalopathy, malnutrition, thrombophilia, hyponatraemia, hyperkalaemia, hepatocellular carcinoma
    - Ascites: transudative/exudative
        - Transudative – portal hypertension, HF, budd-chiari syndrome
        - Exudative – metastases, pancreatitis, biliary ascites
        - Ix: SAAG gradient (>1.1 = transudative)
        - Rx: 1st salt restriction and **spironolactone**, consider SBP □ abx. 2nd paracentesis – must give **HAS (human albumin solution)** at the same time if it's transudative
    - Varices
        - Acute management – **Terlipressin, abx**
        - Prophylaxis – **Propranolol**
    - Hepatorenal syndrome – HRS-AKI – 50% mortality at one month
        - Management = treat precipitating causes**, terlipressin,** stop nephrotoxic drugs (but not always diuretics)
        - Avoid alcohol, monitor U&Es, avoid excess use of diuretics, albumin cover for paracentesis, give abx if infection suspected, avoid non-selective beta blockers
    - Encephalopathy – build up of ammonia in the blood due to inability to excrete + excess bacteria producing urea, crosses BBB – liver flap = test for hepatic encephalopathy. **Lactulose** reduces protein load in the gut and also moves out waste products. **Rifaximin**.
- Ix: fibroscan

Decompensated liver disease

- Can be nonspecific presentation e.g. fatigue malaise
- Can be ascites/jaundice, encephalopathy, UGIB
- Commonly may have relatively normal LFTs
- Decompensated liver bundle <3 BSG + BASL
- If first admission - NON INVASIVE LIVER SCREEN
- Complications:
    - Synthetic failure - jaundice, encephalopathy - INR is useful marker of synthetic function
    - Manifestations of portal hypertension (porto-systemic anastomosis → varices, commonly intrabdominal, oesophageal, short gastric vessels, rectal, caput medusae)
    - **Variceal haemorrhage**
        - RESUS - replace bloods, support clotting + platelets, remember fibrinogen. Vit k is often no good as the issue is intrinsic coagulopathy.
        - Vasoactive drugs - for 24-48hr stabilising period - **terlipressin 1-2mg QDS** - causes splanchnic vasoconstriction (can cause gut ischaemia / angina - must be careful in diabetics/ischaemic heart disease pts)
        - Fluid status - catheterise, strict IP/OP, JVP, monitor chest + saturations as high risk oedema. Blood + crystalloid for fluid resuscitation (hartmanns preferably). Aim Hb 70-80. Remember VBG is 6 hours behind.
        - Antibiotics - sepsis often precipitant for bleed but also significant bleed, risk of aspiration, concurrent sepsis → worsening decompensation
        - INFORM ENDOSCOPIST - if the patient is haemodynamically unstable, even MORE urgent. Otherwise, within 24 hours. Ultimately treatment is banding ligation.
        - Identify precipitants - ETOH? Drugs e.g. NSAIDs? in 70% of cases - SBP.
        - Therefore -> ASCITIC TAP is crucial
        - US abdomen - to rule out PVT as cause of sudden decompensation, and identify any alternative cause e.g. hepatocellular carcinoma
        - Nonselective beta blocker for prevention of further bleed (carvedilol, propranolol)
    - **SBP -** likely due to overgrowth of gut flora + increased permeability in portal HTN state. Bacterial translocation leads to bacteria accumulation in mesenteric lymph nodes. Reduced antimicrobial activity of ascitic fluid + reduced reticuloendothelial system phagocytic activity → SBP.

        - ANYONE WITH ASCITES ADMITTED - NEEDS A TAP
        - Clinical signs may be absent, but prevalence is 10-30% in cirrhotic pts admitted to hospital, may only be presenting in AKI
        - High morbidity and mortality (20-40%) - increases by 3% every hour delay in diagnosis
        - Predominantly **polymorphic** and **>250**mm3 = +ve tap
        - Abx - cefotaxime typically, but according to trust policy
        - HAS - 1.5g/kg on day 1 (fluid status permitting), then 1g/kg 48 hours later
        - Repeat tap on day 3 ideally
        - Secondary prophylaxis - 70% will have further episode. Co-trimoxazole.
    - Hepatorenal failure - do not assume HRS if reversible cause is present. Diagnosis of exclusion. Must have no proteinuria or renal tract disease. Renal support e.g. CRRT may be appropriate if there is a prospect of transplant or recovery otherwise.
    - Encephalopathy
        - Related to accumulation of ammonia - quantification not prognostic
        - changes to BBB and direct neurotoxicity - unclear pathophysiology
        - Reduced detoxification by poorly functioning liver and portosystemic shunting, reduced lean muscle mass which contributes to handling of intestinal toxin
        - Confusion in liver disease is encephalopathy until proven otherwise - important to differentiate from ETOH withdrawal
        - Signs:
            - Grade 1 - increased somnolence
            - Grade 2 - asterixis (liver flap), hyperreflexia
            - Grade 3 - agitation
            - Grade 4 - progression to coma
        - Management = removal of precipitating factors, e.g. treat UGIB, infection, electrolyte derangement, dehydration, uraemia, constipation, drugs (sedatives!!! including chlordiazepoxide or ETOH)
        - constipation prophylaxis important **- Lactulose 15-30ml TDS**
        - Rifaximin - non absorbable antibiotic, reduces hospital readmission by regulating gut microbiome
    - ETOH withdrawal in liver patients - **lorazepam** instead of **chordiazepoxide** as shorter acting
- Management
    - Diet - low salt (dieticians pls involve selves, may need feeding, thiamine, forceval, vit b costrong etc)
    - Hyponatraemia ←fluid restrict
    - Fluid overload ←diuresis - **spironolactone 100-200mg OD + furosemide 20-80mg OD** urinary electrolytes (sodium / potassium ratio → if excreting more sodium than potassium then retaining K+, so need to add loop diuretic. If retaining sodium, increase spiro.)
    - Paracentesis - limited drain (6 hours max) and replace with HAS (100mls 20% for every 2-3L drained) to prevent AKI. In palliative patients to consider indwelling drain to reduce hospital admission frequency)
    - TIPSS (trans-jugular intrahepatic porto-systemic shunt) - rescue therapy to reduce portal HTN. Risks: severe encephalopathy, cardiac failure, occluded portal vein.
    - Transplant - consider ETOH, functional/nutritional reserve.

High output stoma

- St John's solution/double strength dioralyte

Hepatitis

- Inflamm > necrosis > liver failure
- Sx: fever, jaundice, elevated liver enzymes
- Types. All notifiable diseases.
    - A: faeco-oral (contam food/water), usually self-limiting, most common in world, rare in UK
        - N, V, anorexia, jaundice, dark urine (cholestasis), pale stools, mod hepatomegaly. Self-resolves. Rx supportive. Vaccine.
    - B: blood, semen, IVDU, vertical via mum progressive jaundice when chronic infx leads to cirrhosis. Vaccine.

        - Surface antigen (HbsAG) – active infx (HbeAg) – marker viral replication, implies high infectivity
        - Core antibodies (HbcAb) – past or current infx
        - Surface antibody (HbsAB) – vacc or past/current infx
        - Hep B virus DNA (HBV DNA) – direct count of viral load
    - C: infected blood. NO vaccine. antibody denotes exposure not infection. 6 genotypes. New treatements - direct acting anti-viral agents.
    - D & E
- Autoimmune: smooth muscle antibody, **IgG high**, ANA LKM +ve – high ALT, AST, IgG, ANA. Rx: high dose **pred**, **azathioprine** once bili normalised
- Sx: fever, malaise, RUQ pain, headache, N/V, dark urine
- Risk factor: travel, sex, IVDU, fmhx, consumption food/water etc

Wilson's

- Autosomal recessive
- Excess deposition of copper in hepatocytes liver
- Features: parkinsonism (tremor, dystonia, ataxia), hepatitis/cirrhosis, Kayser-Fleischer rings
- Sx: typical presentation = teen/young adult, hepatitis, speech/behavioural/psych problems, AKI caused by renal tubular acidosis
- Ex: blue nails, cirrhosis features (clubbing, hepatomegaly) anaemia (haemolysis), jaundice, tremor, rigidity, asterixis
- Ix: slit lamp (Kayser-Fleischer rings), **LOW** serum ceruloplasmin, reduced total serum copper, **high** urinary copper excretion
- Genetic analysis = ATP7B gene
- Rx: chelation (**penicillamine**)

Haemochromatosis

- Chromosome 6. Aut recessive.
- Genetic – iron overload. Iron deposits in liver and pancreas > cirrhosis, chronic liver disease
- Sx: hyperpigmentation (bronze colour), memory and mood disturbance, amenorrhoea, tiredness
- Ix: iron studies (raised ferritin and transferrin saturation), liver biopsy. High risk for HCC so will require surveillance.
- Rx: venesection

Liver cancer (HCC)

- Risk fx: liver cirrhosis, hepatitis B/C, alcohol, haemochromatosis, PBC, alpha-1-antitrypsin deficiency
- Sx: jaundice, ascites, RUQ pain, hepatomegaly, pruritus, splenomegaly
- Ix: **alpha-feto-protein (AFP)** elevated, CTAP, PET CT, ultrasound (screening) – thrombocytopenia

Laxative abuse - normal colonoscopy, pigment laden macrophages on biopsy

Budd-chiari syndrome

- Classic triad: abdominal pain, tender hepatomegaly, ascites
- Associated with polycythaemia rubra vera (JAK2 mutation) and procoagulant conditions
- Ix: Liver USS + Doppler - most sensitive

Hydatid cyst

- abdominal pain, itching, rashes
- ***Echinococcus granulosus*** - parasitic infection → cystic lesions. Endemic mediterranean/middle east

Zollinger Ellison Syndrome + Gastrinoma

- multiple gastric ulcers, diarrhoea, weight loss (secondary to malabsorption)
- Often as part of MEN type 1
- elevated fasting gastrin level due to gastrinoma - G cells
- most commonly located in first part duodenum
- Dx: secretin stimulation test

Carcinoid syndrome / Carcinoid tumours

- Primary lesion commonly located in ileum
- Typically occurs when mets present in liver → release serotonin into systemic circulation.
- Sx - flushing, diarrhoea, bronchospasm, hypotension, R heart valvular stenosis
- Ix: urinary 5-HIAA, plasma chromogranin A y (avoid serotonin rich foods prior e.g. banana, pineapple)
- Rx: somatostatin analogues e.g. octreotide (synthetic somatostatin - normally released from D cells in pancreas + stomach)

Peutz-Jeghers syndrome

- autosomal dominant condition characterised by hamartomatous polyps in GI tract
- sx: pigmented freckles on lips/face/palms. Commonly presents as SBO or GIB
- 10x risk of GI malignancy

Small bowel overgrowth syndrome

- Bloating, early satiety, abdominal pain, flatulence
- RFx: diabetes, congenital/neonatal gastrointestinal abnormalities, scleroderma
- Ix: first line = hydrogen breath test
- Rx: **rifaximin**

Whipple's disease

- Middle aged men, diarrhoea, weight loss, polyarthralgia, lymphadenopathy, skin hyperpigmentation
- Anaemia, raised CRP, hypoalbuminaemia
- Ix: mesenteric lymphadenopathy on CT, jujenal biopsy → deposition of macrophages and containing periodic acid-Schiff granules
- Causative organism: ***Tropheryma Whippelii***

Colonoscopy findings

- pigment laden macrophages on biopsy - Laxative abuse
- jejunal villous atrophy - coeliac, tropical sprue, hypogammaglobulinaemia, whipples disease, cows milk intolerance

MSK

Myelopathy – compression of spinal cord
Radiculopathy – root entrapment

Sero-negative Spondyloarthropathy – hips & shoulders

- Ankylosing spondylitis
- Psoriatic arthritis
- IBD related arthritis – mucosal link to arthritis ?adhesion molecules in both bowel and joints
    - crohn's/UC, mono/oligoarthritis, may improve with colectomy IBD
- Reactive arthritis

Spine pain

- T spine – bony mets, referred pain from aortic dissection, oesophageal rupture, pneumothorax, mediastinal cancer
- L spine – referred from peptic ulcer/bleed, pancreatitis, pyelonephritis

Spinal stenosis

- Sx: dull pain, pain improves on leaning forwards/sitting, lower limb weakness

Bony metastases

- Most common cancers to metastasise to bone: prostate, breast, lung, thyroid, kidney
- Most common site: spine, pelvis, ribs, skull, long bones
- Features:
    - Pathological fractures
    - Hypercalcaemia
    - Raised ALP

Examination

- Sulcus sign – groove between articulating bones subluxation
- Winging – long thoracic serratus anterior
- Special tests – Rotator cuff
    - Empty can = supraspinatus
    - Hawkins-Kennedy = supraspinatus
    - Pain on external rotation vs resistance = infraspinatus/teres minor
    - Gerber's lift-off test = subscapularis
    - Drop test

Affected joints

DIP – gout, OA (Heberden's), psoriatic arthritis
PIP – OA (Bouchard's), RA
MCPs – OA is UNUSUAL in MCP – hemochromatosis especially if diabetic

Reactive arthritis

- Within 4 weeks of precipitating infection – mucosa + joints
    - Chlamydia usually causes reactive arthritis
    - also: Campylobacter + salmonella
    - Gonorrhoea usually causes gonococcal septic arthritis but can be reactive (hence aspiration)
- Link with HLA B27 gene (50%) – can't see, can't pee, can't climb a tree (uveitis, urethritis, keratoderma blennorhagicum + arthritis)
- Sx: joint pain, enthesitis, cant see cant pee cant climb a tree, ulcers
- Ix
    - abx until rule out septic arthritis ("hot joint rule")
    - aspirate, MC&S, crystal exam
- Rx (when sept arthr excluded)
    - NSAIDs > steroid injections

- Usually resolve within 6 months
- DMARDS if recur

Septic arthritis

- In adults or children – in adults knee most common, in children hip common also. Shoulder 3rd most common
- Causative organism: *staph aureus* most common, *Neisseria gonorrhoea* in sexually active young adults
  - Consider infective endocarditis
- Can be secondary to osteomyelitis
- Septic spine disc
- Sx: pain, swelling, restricted movement
- Ex: restricted movement, warm to touch. Loose pack position – maximises intra-articular volume (flexion, abduction, ext rotation ischiofemoral ligament is lax)
- Kocher criteria
  - Fever >38.5
  - Non-weight bearing
  - Raised ESR
  - Raised WCC
- Ix: joint aspiration – prior to abx if possible, blood cultures, XR, USS
- Rx: IV abx – **flucloxacillin/clindamycin**, oral after 4-6 days. Needle aspiration to decompress joint, arthroscopic lavage if required.

Osteoarthritis

- X-Ray findings – LOSS – loss of joint space, osteophytes, subchondral cysts, subchondral sclerosis
- Wear and tear, usually affects big joints (hips, knees, sacro-iliac, DIPs, MCP at base of thumb, wrist, cervical spine, first MTP)
- Risk factors: obesity, age, occupation, F, previous joint injury, previous joint inflammation
- Sx: pain worse on mvmnt, no/limited morning stiffness
- Exam: restricted mvmnt, palpable bony swelling (osteophyte), muscle wasting (inactivity), effusion, crepitus. Hands: bouchard, heb, squaring at base of the thumb at carpo-metacarpal joint, weak grip, reduced ROM
- Rx: lifestyle, WHO pain ladder
  - **Paracetamol**, **TOPICAL NSAIDs**, **capsaicin**
  - **Oral NSAIDs** + **PPI**
  - **Opiates**
  - **Intra-art steroid inj**
  - Severe: joint replacement (hip, knee)
- OA of the hip - most common presentation = lateral aspect of the hip pain, dull ache
  - Movement exacerbates - struggle to put shoes/socks on (requires hip flexion)
  - Can radiate to the knee, but not past
  - Pain intensity - does it wake you in the night, does it stop you doing things you usually would
  - Other joint pain?
  - PMH - drugs (steroids - AVN + osteoporosis), trauma, childhood hip problems (SUFE, perthes, DDH)
  - Ex:
    - With patient standing: gait, symmetry, muscle bulk, look at joint above + below
    - trendelenburg test (ask patient to hold your hands, feel weight through each hand, test for a minute for fatigue)
- OA of the knee - most common presentation = medial compartment pain, dull ache
  - stiffness
  - giving way
  - locking - temporary loss of full extension
  - swelling
  - downstairs = more painful with patellar-femoral problems
  - SONK - spontaneous osteonecrosis of the knee
  - Ex:
    - valgus/varus deformity, hyperextension/fixed flexion
    - movement prior to palpation for slickness - patellar tracking during movement

Gaits

- Antalgic gait
- Trendelenburg gait - ddx: superior gluteal nerve palsy, DDH, gluteus minimus + medius tendinopathy
- Shuffling gait - bradykinesia + rigidity (extrapyramidal)
- Lurching gait
- Circumduction gait - hemiplegic

Osteoporosis

- Decreased bone mineral density ⍰ pathological fractures
- Risk fx: female sex (post menopause), **corticosteroids**, smoking, alcohol, low BMI (eating disorders/cystic fibrosis, family hx, trauma, **SSRIs, PPIs, anti-epileptics**
- Ix: DEXA + bone profile & PTH (typically normal!)
- FRAX score – assess 10 year risk of fragility fracture
- DEXA scanning
    - T score: based on bone mass of young reference population
    - T score of -1.0 means bone mass of one standard deviation below that of young reference population
    - Z score is adjusted for age, gender and ethnic factors
    - T score
        - -1.0 = normal
        - -1.0 to -2.5 = osteopenia
        - < -2.5 = osteoporosis
- Rx: vitamin D and calcium supplementation, bisphosphonate e.g. **alendronate** (approx. 25% can't tolerate), **raloxifene, HRT**
    - Bisphosphonate holiday – 3-5 years after commencing treatment repeat DEXA and consider stopping
        - Reason not to take: oesophageal reaction (ulcers + oesophagitis), osteonecrosis of jaw, atypical stress fracture of proximal femoral shaft
        - Tablet should be swallowed whole with plenty of water, pt should sit upright, 30 mins before food

Osteogenesis imperfecta

- Genetic condition, autosomal recessive
- Defective collagen ⟶ brittle bones, extremely prone to fractures
- Blue sclera
- Rx: **bisphosphonates + vitamin D**, MDT support

Gout

- Disorder of purine metabolism – hyperuricemia > deposit urate crystals in joints + other tissues. Attacks extremities due to temperature (low > precipitates urate from plasma)
- Pathophys:
    - Impaired renal excretion – under-excrete (90%) or overproduce
    - Secondary causes – things that increase uric acid (HTN, hyperparathyroid, sarcoidosis etc)
    - oestrogen increases urinary excretion of uric acid, male sex far more common
- Risk factors: male, age, renal disease, obesity, met syndrome, dyslipidemia, high purine diet, alcohol
- Chronic tophaceous = final phase of gout where people have nodules affecting joints
- Sx: SEVERE pain in joint, swelling , redness, warmth, tenderness. Max intensity within 24h
    - Big toe, wrist, base of thumb
- Exam: tophi (firm, white nodules)
- Ix: joint fluid aspiration & MCS (**needle shaped negatively birefringent of polarised light** / monosodium urate crystals), serum uric acid (4-6w after acute attack) / joint XR
- Rx: lifestyle,
    - Acute flare: **NSAIDs** > **colchicine** > **steroids**
        - NEVER USE NSAIDS IN RENAL OR HEART FAILURE, GIVE **COLCHICINE** STRAIGHT AWAY
    - Prophylaxis: **allopurinol**

Pseudogout

- Crystal arthropathy caused by calcium pyrophosphate crystals collecting in the joints
- associated with haemochromatosis, hyperparathyroidism, acromegaly, and Wilson's disease
- Sx: rapid onset pain + stiffness, hot, swollen, stiff & painful joint □
- Ix:
    - Joint aspiration – **rhomboid shaped, positively birefringent** of polarised light
    - **Chondrocalcinosis on XR** (calcification of the hyaline cartilage)
- Rx:
    - **NSAIDs – PPI**
    - **Colchicine**
    - **intra-articular steroid injections**
    - **oral steroids**

Paget's

- Increased bone turnover (++ osteoblasts and ++osteoclasts activity) □ expanded and disorganised bone
- Sx: bone pain, deformity, sclerotic (hyperdense) and lytic bone changes
- Ix: high ALP, NORMAL CALCIUM AND NORMAL PHOSP, isotope bone scan - widespread pattern can affect all bones
- Rx: **bisphosphonates**

Cellulitis

- S. aureus, strep pyogenes
- Risk factors: break in skin, DM, venous insufficiency, eczema, oedema
- FBC: WCC, ESR/CRP, U&Es, blood C&S
- Rx: SEPSIS-6, self-care, flucloxacillin (doxycycline if pen allergic)
- Complications: necrotising fasciitis, sepsis

Compartment syndrome

- Increased interstitial pressure in closed fascial compartments □ restricts blood flow
- Causes: acute injury (fracture, crush injury), prolonged limb compression, muscle hypertrophy (athletes)
- Risk factors: bleeding support, compression support, thermal injury, intense muscular activity, venous obstruction, bleeding disorder
- 6P's: DISPROPORTIONATE **P**ain, **P**aresthesia, (**P**ulselessness), **P**allor, **P**aralysis, **P**ressure/**P**erishingly cold
- Ix: needle manometry
- Rx: ortho reg, elevate leg to heart level, emergency fasciotomy

Thrombophlebitis

- Thrombus in superficial vein
- LL: varicose vein trauma / UL: iatrogenic
- Risk factors: varicose veins, anything bleedy or clotty
- Exam: erythema of overlying skin following path of vein
- USS doppler – lack compressibility
- Rx:
    - >5cm or WITH DVT – **anticoag**, compression stockings
    - <5cm and NO DVT **NSAID**, compression stockings

Ruptured Baker's Cyst

- Accumulation synovial fluid outside the knee joint – cyst behind knee
- Causes: trauma, things that cause gradual increase in pressure (arthritis, infection)
- Sx: sharp pain, redness, swelling, tightness, post knee bulge
- Exam: tender, pitting oedema on LL, cyst present, worse sx on knee extension
- Ix: USS duplex leg – mas ; Well's to exclude DVT

Olecranon bursitis

- Bursa = sac created by synovial membrane, contains synovial fluid. Found at bony prominence, reduces friction between bones and soft tissues during movement.
- Bursitis = inflammation of bursa

- Can arise from friction (repetitive movements), trauma, inflammatory conditions (RA/GOUT), infection
- Important ddx: septic arthritis
- Sx: pain, swelling, erythema, fluctuant mass - if reduced ROM ?septic arthritis
- Ix: Aspiration - appearance can indicate cause, MC&S + crystals definitive
    - Pus = infection
    - Straw-coloured = less likely to be infection
    - Blood-stained = trauma, inflammatory
    - Milky = gout/pseudogout
- Rx: treat cause! if infection - **flucloxacillin**

Branchial cyst

- Cyst features – smooth, regular, subcut
- Lack of movement on swallowing
- Anterior border of SCM

Frozen shoulder – adhesive capsulitis

- Most commonly middle-aged women
- Loss of passive ext rotation & passive abduction
- Associated with diabetes (diabetic amyotrophy also), trauma
- Episode lasts between 6 months and 2 years
- Sx: Pain first, then pain goes away but stiffness
- Rx: NSAIDs, physio, oral steroids + intra-articular steroids, manipulation under anaesthesia

Shoulder Impingement / supraspinatus tear / subacromial bursitis

- Pain and loss of movement on abduction
- mid arc pain
- Subacromial bursitis – full arc pain
- Terminal arc pain – osteoarthritis of the AC joint – confirm with scarf test
- Impingement – can't abduct past 90 degrees with pronated hand

Osteomyelitis

- Most common causative organism = staph aureus, staph epidermidis (commensal)
- Most commonly affects lumbar spine, but any joint
- Diabetes, HIV+, steroid use, cancer, CKD – common risk factors for osteomyelitis or discitis
- Commonly comorbid with discitis
- Sx: presents with fever, pain, symptoms of cord compression/cauda-equina, tender mass over spine – paravertebral (abscess)
- Ix: MRI scan – diagnostic
- Rx: surgical debridement and antibiotic therapy (6 weeks **flucloxacillin**)

Acute calcific tendonitis

- Calcium deposition on entheses/tendons
- Commonly achilles/biceps
- Pain, inflammation, palpable mass on the tendon
- Associated with pseudogout + hyperparathyroid

Meniscus tear

- Trauma or degen
- Rotational forces
- Rough surface can catch, buckle
- Crepitations, intermittent locking, effusions, swelling
- McMurryy's test – flex, internally/externally rotate knee pain
- Ix: XR (rule out other things), MRI diagnosis
- Limited vascularisation so doesn't repair itself
- Rx: RICE, if 1 cm or bigger arthroplasty

Osteochondritis dissecans

- Disease process affecting the subchondral bone, in which it may break away and become loose
- Common in younger patients
- Sx: Catching/locking of the joint, typically knees/elbows
- Joint effusion, tenderness on palpation

Anterior talofibular ligament tear (ATFL)

- Excessive inversion and plantar flexion
- Anterior drawer test

Achilles tendon rupture

- Simmon's-thompson test
- Simmonds triad

Foot drop – common fibular nerve palsy

- Fibula fracture
- Loss of sensation on lateral leg and dorsum foot
- Nerve conduction test
- Recovers spontaneously. Tendon transfer.

Bacterial flexor tenosynovitis

- Four features: swollen finger, flexed finger, pain along tendon sheath, pain on passive extension
- IMMEDIATE SURGERY

Transient Synovitis

- Typical age – 2-10 years
- Acute hip pain with prodromal viral infection
- MOST COMMON cause of hip pain in children
- Sx: limp/refusal to weight bear, groin/hip pain, low grade fever (high grade ?septic arthritis), worse in AM
- Rx: supportive, rest + analgesia

Perthes disease

- Risk fx: low socioeconomic class, passive smoking, short stature, coagulation abnormalities
- Self limiting. Inflamm, ischaemia and necrosis of femoral head remodelling of the femoral head. Infarction + revascularization takes about 2 years.
- Young boys (4-8y)
- Sx: episodic, thigh/hip/knee pain and LIMP
- Ex: reduced abduction and internal rotation
- Ex: all hip movements limited (esp abduction, int and ext rot)
- Ix: XR, bone scan/MRI
- Rx: cast/braces, surgical management if needed
- Complications predisposition to OA

Developmental dysplasia of the hip (DDH)

- Spectrum of abnormality
- More common on the left due to adduction against mother's lumbar spine in utero, 20% bilateral
- Risk factors: 4Fs, feet first (breech), +ve fmhx, firstborn, female, oligohydramnios, birth weight >5kg
- Ex: Barlow, Ortolani at NIPE, 6-8w baby check
    - Asymmetric skin grease, clicking on movement, limitation in hip abduction
    - Ortolani (reduce a dislocated femoral head) + Barlow (dislocate articulated femoral head) – finger on greater trochanter, thumb around the knee
    - USS 6w post-partum for: breech baby, childhood hip probs in family, baby breech in last month of pregnancy
- Rx: nothing (stabilise spontaneously by 6 weeks), or Pavlik harness, surgery in older children
- Older children who were not treated: limitations in hip abduction, limp, lumbar lordosis

SUFE

- Teens (10-15), obese – ice-cream falling off the cone analogy
- More common in males, but does happen in girls (especially 10-12)
- Thought to be result of mismatch between growth hormone and sex hormone (pubertal onset)
- Associated with hypothyroid and hypoparathyroid
- Displacement of femoral head epiphysis posteroinferiorly
- Sx: hip/groin/medial thigh pain, bilateral in 20%.
- Ex: obligate external rotation on flexion
- Ix: XR pelvis
- Rx: internal fixation
- Complications OA, AVN femoral head, chondrolysis, leg length discrepancy

JIA (still's disease)

- Arthritis occurring in someone <16 years
- Pauciarticular = >4 joints
- ANA+ve

Psoas abscess

- Causative organism – staph aureus (in post-partum consider GBS)
- Also secondary to: crohn's, diverticulitis, osteomyelitis, femoral catheter, IVDU
- Sx: fever, flank pain, limp, weight loss
- Ex: Patient in the supine position with the knee flexed and the hip mildly externally rotated. Specific tests to diagnose iliopsoas inflammation:
    - Place hand proximal to the patient's ipsilateral knee and ask patient to lift thigh against your hand. This will cause pain due to contraction of the psoas muscle.
    - Lie the patient on the normal side and hyperextend the affected hip. This should elicit pain as the psoas muscle is stretched.

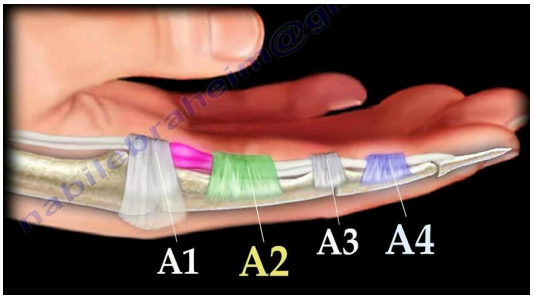
Sarcoma

- Classified based on tissue of origin: bone or soft tissue
    - Bone sarcomas: Osteosarcoma, Ewing's sarcoma, Chondrosarcoma
    - Soft tissue sarcomas: Liposarcoma, Rhabdomyosarcoma, Synovial sarcoma, Leiomyosarcoma
- Sx: pain, mass, impaired function dependent on location e.g. difficulty breathing, joint involvement
- Ex: pathological fractures, cachectic, fever
- Ix: XR, CT, MRI, PET, biopsy
    - Osteosarcoma classic sign = sunburst appearance on XR
    - Ewing's sarcoma = onion skin appearance on XR, children, pelvis + long bones
- Rx: surgery, chemo, radiotherapy

Carpal tunnel syndrome

- Compression of median nerve within the CT paresthesia + muscle weakness
- Causes: oedema/high BMI, tendinopathy, ganglion, carpal fracture (e.g. colle's)
- Ex: thenar eminence wasting, loss of sensation in lateral 3 ½ digits, Tinnels + Phalen's test, muscle weakness (MRC grading)
    - 5 = full power
    - 4 = weakness
    - 3 = can move against gravity
    - 2 = can move if gravity eliminated
    - 1 = full paralysis
- Ix: nerve conduction study (speed reduced in CTS), imaging if suspect fracture
- Rx: splint at night, **corticosteroid injection**, surgical decompression (flexor retinaculum division)
    - Surgery complications – scar sensitivity, pillar pain

Trigger digit



- Flexor digitorum tendon becomes stuck within sheath – most commonly A1 sheath
- Thumb, middle, ring finger (power grip)
- Sx: initially stiffness + snapping when extending a flexed digit, may feel nodule at base of affected finger
- Rx: **steroid injection**, splint, surgery (release A1)

Dupuytren's contracture

- Thickening of the palmar fascia – myofibroblastic deposition of collagen
- More common in older male patients, 60-70% have +ve family history
- Contraction of the ring and little finger
- Causes: manual labour, **phenytoin (+ other antiepileptics)**, alcoholic liver disease, diabetes mellitus, trauma to hand
- Rx: **collagenase injection**, surgery when unable to flatten hand – Husten's tabletop test (pen under hand)

Ganglion cyst

- Ganglion = cyst arising from a joint or tendon sheath, commonly on dorsal wrist
- More common in females, can follow an injury, kind of like an aneurysm?
- Ex: firm and well-circumscribed mass that transilluminates
- Rx: bonk? Ganglions disappear spontaneously after several months, excision for cysts with neurovascular manifestations – can reduce grip strength

Thumb cmcj arthritis – most common arthritis in hand

- Pain + stiffness
- Unilateral in women, bilateral in men
- Trapezio-metacarpal joint – saddle joint – allows opposition of thumb
- Volar oblique ligament – deterioration over time subluxation of saddle joint arthritis
- Rx: usual for OA + soft thumb wrap splint, physio to strengthen muscles around the joints

Tennis elbow – lateral epicondylitis (backhand)

- Chronic tendinopathy at common extensor origin
- Pain on passive and resisted wrist extension

Golfer's elbow – medial epicondylitis (forward swing)

- Chronic tendinopathy at common flexor origin
- Pain on passive and resisted wrist flexion

Cubital tunnel syndrome

- Compression of ulnar nerve behind medial epicondyle
- Pain worse on elbow flexion

Peripheral Nerve Supply - Hand

- Hand: all intrinsic hand muscles are **ulnar nerve** except thenar muscles and LOAF (median)
- also **ulnar**: Flexor carpi ulnaris and medial ½ flexor digitorum profundus
- Froment's test/sign - when asked to hold a piece of paper between the thumb + pointer finger, flexes DIP of thumb as adductor pollicis unable to adduct effectively
- **Ulnar paradox** - severity is greater with lower injury
    - due to paralysis of flexor carpi ulnaris in more proximal injury
- **Median** nerve innervates all other muscles in the anterior forearm, plus LOAF
    - Lumbricals (lateral 2)
    - Opponens pollicis
    - Abductor pollicis brevis
    - Flexor pollicis brevis
- **Radial** nerve innervates posterior forearm - extensor digitorum, extensor indicis, extensor pollicis (longus + brevis), abductor pollicis longus
- 3 musketeers assassinated 5 rats, 5 mice, 2 unicorns

- C5,6,7 = Musculocutaneous
- C5,6 = Axillary
- C5-T1 = Median
- C5-T1 = Radial
- C8-T1 = Ulnar

Erb

- C5/C6 palsy caused by compression of the upper trunk of the brachial plexus
- Typically result of shoulder dystocia/traumatic delivery
- Causes weakness of shoulder abduction + external rotation, elbow extension + wrist flexion (Waiter's tip)

Klumpke

- Lower trunk of brachial plexus (C8 + T1)
- Weakness of intrinsic hand muscles
- Ulnar claw

Hip #

- why operate pain relief + mobilisation
- if not bed rest + traction (only in patients close to death/already bedridden as makes it much more likely they will die)
- Hx: mechanism, medical history, mobility, accommodation
- Ex: look for reasons for fall e.g. resp exam for infection e.g. pneumonia, cardiovascular for syncope/murmur. Check NV intact.
- Ix: ECG (new arrhythmia/silent MI), urine culture, bloods
- Rx:
  - fluids, immobilise, analgesia – consider iliacus fascia block
  - opiates laxative
  - DVT prophylaxis
  - Abx – if infective cause found**, fluclox + gent** in surgery as prophylaxis
  - Surgery
    - Classification:
      - Intracapsular – risk of AVN, damage to medial/lateral collateral femoral arteries
      - Extracapsular – trochanteric or subtrochanteric (below lesser trochanter)
      - Garden system
        - I = stable with impaction in valgus
        - II = complete fracture but undisplaced
        - III = displaced fracture, rotated + angulated but still in articulation
        - IV = complete disruption
    - Fixation – younger patients (<50), older patients are already osteoporotic, and screws won't be secure
      - Risk of AVN 25%
      - Non-union 15%
      - However, preserves own hip, quick
    - Hemi-arthroplasty – older pt, poor function acetabular wear
    - Total hip arthroplasty – older pt, good function. Elective procedure for osteoarthritis
    - Dynamic (sliding) hip screw – intertrochanteric #
    - IM nail – subtrochanteric #, pre-pathological fracture (bony mets)
  - Complications of hip replacement - osteomyelitis, PE, infection, dislocation, nerve damage (sciatic), prosthesis failure, periprosthetic #

Fractures

Mid-shaft fracture humerus	Radial nerve, profunda brachial artery (travel through space and groove)
Surgical neck of humerus	Axillary nerve and posterior circumflex arteries
Downward force onto abducted arm dislocate shoulder (glenohumeral joint/ligs)	Axillary nerve
NOF intracapsular	risk of AVN, damage to medial/lateral collateral femoral arteries
NOF extracapsular	

Weber's classification ankle fractures

- class is determined based on level of fracture in relation to tibio-fibular syndesmosis
- below = A, at the level = B, above = C

Ottawa ankle rules → XR

- unable to weight bear, pain over posterior ⅓ of either malleolus OR 5th metatarsal

Scoliosis

- Curvature of the spine in the coronal plane
- Divisible into structural/non-structural
- Non-structural = more common – often postural, disappear on manoeuvres such as bending forwards

Scheuermann's disease

- Epiphysitis of vertebral joints
- Sx: back pain/stiffness, progressive kyphosis
- Ix: XR = epiphyseal plate disturbance and anterior wedging

Spondylolysis – Pars defect

- Usually L4/L5
- Commonest cause of spondylolisthesis = displacement of vertebra relative to inferior vertebral body
    - Traumatic spondylolisthesis = scotty dog appearance on XR
- Can lead to disc degeneration, radiculopathy

Causes of sclerotic bony lesions

- Renal
- Mets
- Pagets

Protrusio - femoral head translated into the pelvis through the acetabulum

AVN causes

- Trauma
- Alcohol
- Sickle cell
- Hypercholesterolaemia
- Steroids

Managing polytrauma

- A-E, ATLS protocol
- prioritisation:
    - Life-threatening bleeding wounds
    - Limb-threatening injuries
    - Fractures, dislocations, wounds at risk of complications
    - Stable fractures
- Primary Survey = C-ABCDE
    - **C**atastrophic haemorrhage (usually from neck/pericardium)
        - emergency thoracotomy for penetrating chest trauma
        - pressure over bleeding points
        - warm blood transfusion
    - **A**irway maintenance with /c-spine protection
        - Maxilla fracture, burns, inhalation, neck haematoma, GCS < 8, paralysis, vomiting/stridor
        - Airway manoeuvres
        - immobilisation until cleared by XR/CT
    - **B**reathing
        - look, feel, expansion, percuss, listen
        - oxygen/needle asp/chest drain
    - **C**irculation
        - HR, BP, peripheral pulses, heart sounds, lactate
        - Look for bleeding - blood on the floor (external) and 4 more
            - Chest
            - Abdomen
            - Pelvis
            - Long bone
        - 2x cannula, fluids/blood, pelvic binder, traction, combat application tourniquet if compression fails to control blood loss
        - Shock - classes
            - 1 - 15% blood loss
            - 2 - 15-30% blood loss, HR >100, normal BP
            - 3 - 30-40% blood loss, HR > 120, low BP
            - 4 - >40% blood loss, HR > 140, low BP
    - **D**isability
        - GCS, PEARL
    - **E**verything else
        - try not to expose if need to conserve heat unless absolutely necessary (e.g. can't get CT)
- Adjuncts to primary survey = TRAUMA CT (head, spine, chest, abdo, pelvis)
    - if not available, XR long bones, C spine, Pelvis
    - FAST scan
    - CT head
    - Catheter + monitoring
- Secondary Survey - after resus + normal vital function established
    - History - AMPLE
        - Allergies
        - Medication
        - PMH / Pregnancy
        - Last meal
        - Event/Environment related to injury
    - Adjuncts: trauma CT, specific XR, ultrasound, endoscopy/bronchoscopy, angiography
    - regular observations
- Definitive care

Paediatrics

Never given **aspirin** to kids – can cause **Reye's syndrome** (serious liver and brain damage) EXCEPT in Kawasaki

Developmental red flags

- Poor head control or floppiness at 6 months.
- Unable to sit unsupported at 9 months.
- Not weight bearing through legs at 12 months.
- Not walking at 18 months.
- Not running at 2 years.
- Not climbing stairs at 3 years.
- Persistent toe walking.
- Increased muscle tone.

Cerebral palsy

- Types: spastic (most common), dyskinetic (BG), ataxic (cerebellum), mixed
- Signs: Not sitting by 8 months / Not walking by 18 months / Hand preference before 1 year / Abnormal tone (too floppy or spastic) / weakness
- Ix: MRI brain
- Rx: MDT

Autism spectrum disorder

- Language delay or REGRESSION
- verbal and non-verbal communication impairment
- Social impairment (reduced or negative response to others e.g. rejection of cuddles)
- Repetitive, rigid, or stereotypical interests, behaviour or activities

Fragile X syndrome

- FMR1 mutation on X chromosome (always affects males)
- Delay in speech and cognition
- Macrocephaly and learning difficulties

VSV/chicken pox

- Snotty and grotty first
- Pleomorphic vesicular rash (mainly trunk). VERY ITCHY
    - 1 = red blemish, 2 = vesicular rash, 3 = crusting
- Very infectious. Droplet spread
- Incubation = 1-3w
- Infectious = 1-2 days before rash appears until crusted over
- Self-limiting. Only dangerous in pregnant, immunocompromised
- Rx: supportive only (paracetamol, fluids, topical colamine). NEVER GIVE NSAIDS – skin infx > necrotising fasciitis
- Complications:
    - Facial nerve **Ramsay-Hunt Syndrome (facial paralysis, hearing loss)**
    - **VSV meningitis**
    - Reactivates as **Shingles**

Bronchiolitis

- <2y. ***RSV***
- Pathophysiology: causes epithelial cells of small airways to slough off + mucous obstructs alveolar spaces obstruction impaired gas exchange SOB, hypoxia
- 9 day illness
    - Days 1-3: coryzal prodrome (1-3 days) + persistent cough *AND*
    - Tachypnoea and/or chest recession, *AND*
    - Day 4-5: wheeze and/or crackles on auscultation
    - Days 9-10: might still have a cough
- Ex: wheeze, crackles

- Ix: immunofluorescence
- Rx: supportive management. If hospital: humidified O2 (headbox first, CPAP second), NG tube for feeding. **Ribavirin** if severe

Slapped cheek/5th disease/erythema infectiosum

- ***Parvovirus B19***
- Pathophys: Parvovirus targets RBCs in bone marrow
- Respiratory droplets
- Sx – prodrome (mild fever, headache) > rash on cheeks (red hot burning) > pink lacy rash on limbs and trunk
- Complications: aplastic crisis in haemolytic anaemia
- Management: supportive
- Don't confuse with **scarlet fever** (which is much worse)

Scarlet fever

- Reaction caused by erythrogenic toxins produced by group A strep, e.g. **Streptococcus pyogenes**
- Peak incidence = 2-6 years, respiratory spread
- Sx: fever, malaise, headache, nausea + vomiting, sore throat
- Ex: **strawberry tongue, sandpaper rash**
- Ix: throat swab
- Rx: **Penicillin V** 10 days - children can return to school within 24hrs of starting abx
- Complications - rheumatic fever (20 days post inf), acute glomerulonephritis, meningitis, nec fasc, **otitis media = most common**

Common cold

- Rhinovirus, coronavirus, influenza
- Examination – exclude other problems and complications (sinusitis, meningism, acute otitis media, RTI)
- CENTOR score to consider abx
- Rx supportive

Roseola infantum – common 6m-2 years, fever followed by rash, febrile seizures common

Measles

- NOTIFIABLE DISEASE – local health protection team (HPT)
- Morbillivirus of paramyxovirus family
- Incubation: 10 days
- Sx:
    - Prodromal (malaise, fever, cough, **CONJUNCTIVITIS**)
    - Rash (Koplik's spots, erythematous, maculopapular) STARTS BEHIND EARS
- Ix: PCR swab (nasophar, throat)
- Rx: supportive. MMR vacc
- Complications: OTITIS MEDIA, pneumonia, encephalitis (1-2w later), MND (10yr later)

Epiglottitis *(Don't HIB me in the epiglottitis)*

- Cellulitis of epiglottis – oedema of airway, narrowing of opening
- ***HiB***
- Sx: toxic appearance (high fever, look unwell), drooling, tripod, hoarse voice, stridor
- Ix: laryngoscopy
- Rx: DO NOT TOUCH CHILD. Highly infectious. Call ENT surgeon, paediatrician, anaesthetist.
- Surgical emergency.

Tonsillitis

- Usually viral cause. Bacterial causes: ***Group a strep*** (GAS), ***strep pneumonia***
- Sore throat, fever >38, possible ANTERIOR CERVICAL lymphadenopathy
- Ix: CENTOR, FeverPAIN
- Threshold for abx lower if immunocompromised, at risk or rheum fever, living in SA/Africa, indigenous communities
- Rapid antigen test for GAS. Throat culture (if first negative but still suspect GAS)

- **FeverPAIN score** = alternative to Centor criteria. Score 4 or 5 – consider antibiotics
    - **Fever** during previous 24 hours
    - **P** – **P**urulence (pus on tonsils)
    - **A** – **A**ttended within 3 days of onset of symptoms
    - **I** – **I**nflamed tonsils
    - **N** – **N**o cough
- Rx: **Phenoxymethylpenicillin** (bact). Fluids, avoid hot drinks
    - **SIGN** criteria for Tonsillectomy: (7, 5, 3 and 1, 2, 3. 7 episodes per year in one year. 5 episodes per year for 2 years. 3 episodes per year for 3 years)
- Complications:
    - GAS: Peritonsillar abscess (quinsy), otitis media, rheum fever, scarlet fever
    - Strep pneum: Post strep glomerulonephritis, post strep reactive arthritis

Kawasaki

- Type of vasculitis – small and medium-sized (esp coronary arteries)
- Sx: CRASH and BURN: conjunctivitis (non-purulent), rash, (adenopathy) lymphadenopathy, strawberry swollen tongue, hands and feet swelling. Burn = FEVER FOR 5 DAYS (very high)
- Also peeling of hands and feet
- Rx: high dose **aspirin**, IV **Ig**
- Complication: coronary artery aneurysm

ONLY TIME YOU EVER GIVE ASPIRIN TO A CHILD

Pneumonia

- Nasal flaring, grunting, recessions, O2 sats <92%, focal crackles
- Treat as adult with lower threshold to see in hospital

UTI

- Same as adults
- Bacterial – E coli
- Might have vesicoureteric reflux pyelonephritis
- **Sx:**
    - Infants: poor feeding, vomiting, irritability
    - Younger children: abdo pain, fever, dysuria
    - Older children**:** dysuria, frequency, haematuria
    - UTI sx: temp>38, loin pain/tenderness
- Ix: urine MC&S, dipstick
- Rx:
    - Acute pyelonephritis/upper UTI: **co-amoxiclav**
    - Cystitis/lower UTI: oral **trimethoprim** or **nitrofurantoin**
    - **Recurrent: prophylactic abx**
    - **DMSA (don't mess sterile anus) – renal parenchyma defects**
- <3m – refer immediately to paeds
- >3m with upper UTI – consider hospital, abx directly
- >3 m with lower UTI – abx only

Otitis media

- Most common cause = strep pneum (virus first, then bacteria invades)
- Sx:
    - Older kids – earache
    - Younger kids = hold/tugging/rubbing of ear + non-specific sx (fever, irritability, crying etc),
- Ix: bulging tymp membrane, loss of normal landmarks, red/yellow/cloudy tymp membrane, can be perforated +/- discharge
- Rx: **amoxicillin**

Croup

- 6m-2y children
- Sx: Barking cough, sometimes stridor, hoarseness, rep distress (prodromal non-specific URTI sx)

- Parainfluenza
- Severe = stridor at rest, marked recessions, sig distress agitation, lethargy or restlessness, tachycardia
- Rx: **dexamethasone** (oral) and O2
- Complications: epiglottis swelling (CXR to rule out if suspected)

Whooping cough (pertussis)

- ***Bordetella pertussis*** (gram negative) – "100 day cough"
- Whooping = unable to take in air between coughs
- Sx: coryzal first, then >1w paroxysmal whooping cough. Can faint, vomit, pneumothorax from coughing so hard
- Ix: nasopharyngeal/nasal swab for PCR/culture, anti pertussis toxin IgG
- Rx: notifiable disease, supportive care, macrolide abx (**azithromycin**, **clarithromycin** for <1m), prophylactic abx to **close contacts**
- Complication: bronchiectasis
- Vaccine
    - 6 in 1 for babies (2, 3, 4m)
    - 4 in 1 pre-school booster (3-5y)
    - Offered to pregnant women if not already vacc-ed (16-32w)

Jaundice in the newborn

- Jaundice in first 24h always pathological
    - Rhesus haemolytic disease
    - ABO haemolytic disease
    - Hereditary spherocytosis
    - G6PD def
- Common at 2-14 days – physiological, commonly

Fragile X

- Most common cause of inherited intellectual disability
- Large low set ears, long thin face, high arched palate
- Macroorchidism
- Mitral valve prolapse

Cystic fibrosis

- Autosomal recessive, CFTR mutation, chromosome 7, ΔF508
- Pathophys: defective CFTR > Cl can't leave duct > Cl and Na stay in duct > water can't leave > thick secretions on outside > too thick for cilia > can't get rid of it > colonise > infx
- Sx: meconium ileus, FTT, sinusitis, recurrent LRTIs, pancreatitis (greasy/bulky stools), rectal prolapse, GORD
- Signs: short, DM< delayed puberty,
- Ix: sweat test, blood spot, gene testing
    - Antenatal = echogenic/bright bowel
- Complications: pneumonia (***Pseudomonas aeruginosa***), diabetes, osteoporosis, vit D deficiency, liver failure, bronchiectasis
    - Thick pancreatic, biliary secretions □ block ducts □ lack digestive enzymes
    - Thick airway secretions □ reduce airway clearance □ colonise □ LRTIs
    - Bilateral absence of vas deferens □ male infertility
    - Meconium ileus – too sticky □ obstruct bowel □ abdo distension and vomiting
- Rx:
    - Lifestyle: chest physio, exercise, high cal diet
    - Medical – **salbutamol**, prophylactic **fluclox**, **ciproflox** for pseudomonas, **nebulised hypertonic saline**, **nebulised dornase alfa** (**Dnase**), **CREON**, **vaccinations** (pneumococcal, influenza, varicella), **ADEK vitamins**
    - Surgery – lung transplant, liver transplant
- Genetics
    - A 25% chance that the child is born with two normal genes (normal)
    - A 50% chance that the child is born with one normal and one abnormal gene (carrier, without disease)

- A 25% chance that the child is born with two abnormal genes
- If both parents are healthy, one sibling has CF and a second child doesn't. What's the likelihood of the second child being a carrier? □ 2/3

Downs, Edwards, Patau's (alphabetical order) – 21, 18 (also Eighteen Edwards), 13 (goes down in number)

Down's syndrome

- Trisomy 21
- Risk factor: maternal old age
- Features: upward slanting palpebral fissures, flat occiput, protruding tongue, flat nasal bridge, epicanthal folds, ear abnormalities (ear problems) single palmar crease, sandal gap
- Internal: AVSD, VSD, ASD, Hirschprung's disease (lack of nerves in rectum), duodenal atresia, TOF, ENT problems, hypothyroidism, cataracts, early onset Alzheimer's

Edwards

- Trisomy 18
- Not usually compatible with life – 50% die within a week, 90% dead by one year
- Common cause miscarriage
- Rocker bottom feet, clenched hand, VAS/ASD

Patau

- Trisomy 13
- Polydactyly, holoprosencephaly, omphalocele + loads of other stuff

Turners

- 45 XO – only affects females (only have one X chrom)
- Webbed neck, puffy hands and feet (lymphoedema), wide spaced nipples, low set ears, short
- Internal – gonadal dysgenesis (ovaries don't work), hypothyroidism, sexual infantilism, coarctation of aorta, bicuspid aortic valve, essential HTN, horseshoe kidney, PUJ obstruction, double collecting systems

Klinefelter's syndrome

- 47 XXY – only affects males (extra X)
- In adolescents – affects secondary sex characteristics – gynaecomastia, -body hair, primary hypogonadism, microorchidism, high FSH, infertility

Williams

- 7Q11.23 deletion
- Elfin facies, IQ differences, really friendly, talking from a script
- Supravalvular aortic stenosis, DM, hypothyroidism, hypercalc

Prader-Willi

- 15Q11-Q13 deletion paternal
- Developmental probs – hypotonia, FTT, poor developed fenitals, hyperphagia (++eat), tantrums, obesity, hypogonadmi, low IQ

Intussusception

- Sx: episodic crampy abdominal pain, vomiting (green/yellow), drawing knees up, red-currant jelly stools, sausage shaped mass in RUQ
- Ix: ultrasound – target-like mass
- Rx: reduction by air insufflation under radiological control

Volvulus

- Sigmoid most common in older patients
- Caecal in all ages, Crohn's, pregnancy
- Risk fx: adhesions, IBD, malignancy, reduced motility/antikinesis (e.g. antipsychotics, parkinsons, DMD)
- Sx: constipation, pain, bleeding, nausea/vomiting
- Ix: AXR → coffee bean sign (sigmoid), small bowel obstruction (caecal)

- Rx: sigmoid = rigid sigmoidoscopy with rectal tube insertion, caecal volvulus = operative, often hemicolectomy

Duchenne's muscular dystrophy

- X-linked recessive inherited disorder in the dystrophin gene
- Sx: progressive proximal muscle weakness, typical onset 5 years
- Ex:calf pseudohypertrophy, **Gower's sign** (using arms to walk up legs to stand)
- Ix: raised creatinine kinase, genetic testing = definitive (used to be muscle biopsy)
- Poor prognosis, patients typically survive - 25-30
- Associated with dilated cardiomyopathy

Tetralogy of Fallot

- Most common cause of cyanotic heart disease
- Commonly presents at 1-2 months
- 4 Characteristic features:
    - VSD
    - Right ventricular hypertrophy
    - Pulmonary stenosis - degree of stenosis determines severity of cyanosis
    - Overriding aorta
- Sx: cyanosis in episodes, especially during crying/illness
- Ex: Ejection systolic murmur
- Ix: XR - boot shaped heart
- Rx: surgical repair, beta blockers to reduce infundibular spasm

Intestinal malrotation

- Congenital defect in the rotation of the bowel, associated with exomphalos
- Causes obstruction + can be associated with developing volvulus
- Rx: Ladd's procedure

Peutz-Jeghers syndrome

- Autosomal dominant - Polyps + mucocutaneous pigmentation of the vermillion border
- Can cause obstruction/intussusception + GI bleeding
- Predisposes to bowel cancer

Psychiatry

Depression

- Need the three major + at least one minor, most days, most of the time, for the last 2 weeks

Major (FLP)	Minor (CS SAGAS)
• Persistent sadness or low mood • Loss of interests or pleasure • Fatigue or low energy	• Poor **C**oncentration/indecisiveness • Disturbed **S**leep • Low **S**elf-confidence • **A**gitation or slowing of movements • Guilt or self-blame • Poor or increased **A**ppetite • **S**uicidal thoughts or acts

- PHQ-9 (mild 5-10-15-20 severe), HADS, BDI-II
- Types:
    - subthreshold
    - mild (a few, minor functional impairment)
    - moderate – functional impairment between mild and severe
    - severe – most sx, markedly interfere with fx +/- psychotic sx
- Ix: rule out other causes
- Management
    1. Assess/monitor, psychoeducation
    2. Subacute-mild: Low intensity (self-guided CBT)
    3. Mod-sever: High intensity (CBT) + medication (SSRI)
    4. Sev: High intensity + medication + possible ECT/crisis team/emergency admission
        i. Life threatening
- Meds: **SSRI** (SE: insomnia, GI upset/bleed)

Anxiety (GAD)

- ICD-10: anxiety must be present for most days for several months and should include elements of apprehension, motor tension and autonomic overactivity
- GAD-2 or GAD-7 to determine severity
- Sx: excessive persistent worry that is not related to particular circumstances + restlessness, poor concentration, sleep disturbance, fatigue, muscle tension, irritability, dizziness, palpitations, sweating, peripheral/peri-oral paraesthesia & abdominal discomfort. NO TIME LIMIT (DSM says at least 6m)
- RF: F, fmhx psych, childhood adversity
- Rx:
    1. Education/active monitoring
    2. Low intensity
    3. High intensity OR medication (SSRI)
    4. High intensity + medication (SSRI)

Panic disorder

- Rx
    1. Education/active monitoring
    2. CBT
    3. more than 10 sessions CBT OR medication (SSRI OR SNRI)
    4. more than 10 sessions CBT + medication

OCD

- Rx
    1. Low intensity (<10h CBT + ERP)
    2. High intensity (>10h CBT + ERP) OR medication (SSRI)
    3. High intensity + medication

Bipolar disorder

- Type 1 – worse, depression + mania (with psychotic features)
- Type 2 - hypomania
- Cyclothymia (mood swings between depression + elation)
- Rx: (BY PSYCHIATRIST ONLY) **antipsychotic (1st or 2nd gen)** + psych intervention > different antipsychotic > **lithium**

Psychosis

- Sx:
  - positive (halluc, delusion, disorganised beh/speech) – high dopamine in mesolimbic > ++ D2 in nucleus accumbens
  - negative sx (emotional blunting, reduced speech, loss motivation, self-neglect, soc withdrawal) > low DA in mesocortical > reduced activation PFC D1
- Delusions: persecutory, grandiose, nihilistic
- ICD-10: at least one first rank OR at least 2 non-first rank
  - First rank: **ABCD** (Aud hallucinations, Broadcasting thoughts, Control (delusions of control), Delusions of any kind)
- Rx
  - Acute 1st episode: ensure safety (patient and others), oral **BDZ** or rapid tranquillisation (**lorazepam**) > refer to specialist
  - Subsequent episodes: **antipsychotic drug** + psych therapy
    - **Clozapine** is **always** 3rd line
      - **CLOZAPINE** side effects – seizures, agranulocytosis, neutropenia

Delirium tremens

- Alcohol withdrawal

6-12 h	Autonomic: tremor, sweating, tachycardia, anxiety
36 h	Seizures
48-72 h	Delirium tremens: coarse tremor, confusion, delusion, halluciations (aud and vis), fever, tachycardia

- Rx: **chlordiazepoxide, oxazepam (better in liver failure)**
- **Disulfiram** – deterrent for patients with alcohol abuse history (makes them very sick when they drink)
- **Naltrexone** – blocks opioid receptor (similar to naloxone)

Respiratory

Fi02

- room air = 21%
- each additional L oxygen = approx 4%
- therefore, 2L of oxygen on nasal specs = Fi02 28%
- Fi02 - 10 is approx = to Pa02 e.g. pt on 2L should have Pa02 of 18

Asthma

- Episodic exacerbations with periods of complete recovery in between, diurnal variation, wheeze, breathlessness, atopy (IgE mediated hypersensitivity), family hx
- Airways are hyperresponsive to stimuli airway narrowing, smooth muscle contraction, inflammation/swelling, mucus production
- Occupational trigger: Isocyanates paints/varnishes in motor industry particularly
- Ix
    - 1st line:
        - FeNO 40ppb = positive result
        - Spirometry – pt explanation: pre-test stop using bronchodilators if possible, avoid smoking for 24 hours, avoid alcohol + strenuous exercise for a few hours. Test: 3 recordings will be taken, flow/volume graph will determine obstructive/restrictive pattern
            - FEV1/FVC ratio <70% = obstructive
            - Bronchodilator improvement ≥12% = positive for asthma
    - 2nd line (do if there's diagnostic uncertainty after the two tests above)
        - Diurnal variation on peak flow - >20%
        - Direct bronchial challenge test – 20% fall or less in FEV1
- Severity grading
    - Moderate – 50-75% of best PEFR – typical exacerbation regime with salbutamol, ipratropium, oral steroids (5 days)
    - Acute severe – 33-50% of best PEFR, RR>25, HR>110, inability complete sentences
    - Life-threatening – 33-92-CHEST (<33%, <92% oxygen, cyanosis, hypotension, exhaustion, silent chest, tachycardia) – if not responding escalate to ITU
    - Near-fatal – need for NIV, increased CO2 needs to be escalated to ITU
- Rx ACUTE
    - OSHIMTE (they ruined it by making magnesium above theo/aminophylline)
        - **SALBUTAMOL 5MG**
        - **HYDROCORTISONE** – IV if unable to take oral pred
        - **IPRATROPIUM 500MICROGRAMS**
        - **Magnesium sulfate** – call med reg if reaching this point
        - **Theo/aminophylline**
        - Escalate to intubation (NIV can be valuable in COPD but less in asthma as risk that airway will be lost)
    - Observation on discharge dosages for 24hrs, <75% of best PEFR, follow-up in community within 48hr
    - Personalised asthma action plan – e.g. increasing steroid dosage, taking oral steroids at certain PEFR
- Rx Chronic
    - B2 agonist – **salbutamol** – good technique/spacer, safety-netting – if needing 10 puffs in 24/hr period, should be in hospital
    - low dose ICS - e.g. **beclometasone**
    - Leukotriene receptor antagonist e.g. **montelukast**
    - LABA - **salmeterol/formoterol**
    - Combined therapy inhalers + specialist services
- Inhaler technique
    - shake inhaler
    - sit/stand up straight + tilt chin up
    - breathe out completely
    - create complete seal around mouthpiece/place spacer over face
    - breathe in slowly + squeeze the canister
    - hold breath for 10 seconds

    - wait 30-60s before taking second puff
- Inhaler side effects
    - rinse out mouth after using to remove residue + reduce side effects
    - steroid inhalers → candida, reduction in adult height for children using ICS
    - tachycardia, tremor, sore throat/cough

Bronchiectasis

- Permanent dilatation of the airways secondary to chronic inflammation or infection.
- Sx: persistent productive cough - large volumes of sputum, dyspnoea, haemoptysis
- Ex: coarse crackles, wheeze, clubbing
- Rx: inspiratory muscle training, postural drainage, abx for exacerbations, immunisations
- Most common organisms: **Haemophilus influenzae**, Pseudomonas, Klebsiella, Strep pneumoniae

COPD

- Persistent airflow limitation associated with increased chronic inflammatory response
- Encompasses chronic bronchitis and emphysema or combination of both
- Hallmark = chronic inflammation, narrowing and remodelling of airways, increased goblet cells, enlargement of mucus secreting glands pulmonary hypertension. In emphysema, breakdown of elastin reducing recoil of lung tissue
- Alpha-1-antitrypsin - impaired inhibition of protease (neutrophil elastase – degrades elastin in lung tissue, shit recoil → emphysema)
- Pulmonary hypertensionRfx: smoking, alpha-1-antitrypsin deficiency, low birth weight, recurrent infections
- Ix: sputum sample MC&S (rule out pseudomonas), obs, peak flow. ABG (retention status), CXR, spirometry
- Polycythaemia is complication of COPD (increased RCC as compensation for hypoxia)
- Acute Rx:
    - Nebulised bronchodilators (e.g. **salbutamol 5mg/4h** and **ipratropium 500mcg/6h**)
    - Steroids (e.g. **200mg hydrocortisone or 30-40mg oral prednisolone**)
    - Antibiotics if evidence of infection – typically **amoxicillin** or **doxycycline**
    - Physiotherapy can help clear sputum
    - **IV aminophylline**
    - Non-invasive ventilation (NIV) – BIPAP CPAP
- Staging - based on FEV1, stages 1-4.
    - 1 - <80% of predicted
    - 2 - 50-79%
    - 3 - 30-49%
    - 4 - <30%
- Long term Rx:
    - Rescue packs (abx and steroid)
    - SABA, then LABA/LAMA if needed
    - If asthmatic features (e.g. diurnal variability, eosinophilia) ICS + LABA
    - **Carbocysteine / Glycopyrronium**
    - Refer for specialist involvement
    - Pulmonary rehab
    - Vaccination/anti-viral therapy pneumococcal vaccine, annual flu vaccine
    - Ambulatory oxygen therapy as a last resort

pulmonary arterial HTN

- resting mean arterial pressure of >20
- sx: progressive exertional dyspnoea, syncope, peripheral oedema
- signs: R ventricular heave, loud P2, raised JVP, tricuspid regurg
- Treat underlying causes e.g. COPD, risk factors including HIV, cocaine use.
- 10% autosomal dominant inheritance
- **BOSENTAN** - endothelin receptor antagonist, decreases pulmonary vascular resistance and increase cardiac output

Lung cancer

- **Adenocarcinoma** most common. Smoking biggest cause.
  - EGFR mutations 10-40% - drive tumours growth
  - Peripheral, glandular, most prevalent cancer in non-smokers. Early metastasis, responsive to therapy
- **Small cell lung cancer** (SCLC) causes paraneoplastic (contains **neurosecretory** granules that release neuroendocrine hormones) – strongest association with smoking.
  - Metastasis early, widely. Poor prognosis and poorly differentiated.
  - Lambert-Eaton syndrome, ACTH and ADH
- **Squamous cell carcinoma**
  - Cavitating lesions most common in this type of lung cancer
  - Central, metastasis in later stage
  - Produces ectopic PTH hypercalcaemia
  - Hypertrophic pulmonary osteoarthropathy also associated - clubbing and periostitis of small hand joints
- Pathophys:
  - Spread to lymph nodes (local – hilar, mediastinal, supraclavic) > haematogenously to liver, brain, bone, adrenals
  - Changes in lung: obstruct air and mucus flow obstruction, infection
  - Pleural, pericardial effusion
- Sx:
  - Non-specific: SOB, cough, fatigue, weight loss, appetite loss, chest pain
  - Very specific: haemoptysis, clubbing, supraclavicular lymph, persistent/recurrent chest infectious, thrombocytosis, chest signs consistent with lung cancer
- Extrapulmonary signs:
  - SVC syndrome (Pemberton's sign – SCLC), facial plethora, breathlessness
  - Phrenic nerve palsy (SOB), RLN palsy (hoarse voice), Horner's, dysphagia
  - SIADH = SCLC. Ectopic ADH
  - Cushing's – SCLC. Ectopic ACTH
  - Hypercalcaemia = SCC. Ectopic PTH (PTHrP)
  - Lambert-Eaton = SCLC. VG-Ca channels on presynap tereminals, motor neurons. Prox muscle weakness, diplopa, slurred speech, dysphagia
  - Hypocalcaemia – calcitonin
- Ix: CXR, bronchoscopy with endobronchial USS (EBUS) + biopsy, sputum cytology, CTTAP contrast for staging (chest, abdo pelvis), PET CT, pleurocentesis
- Staging:
  - I – localised, small tumour <4cm, no nodes
  - II – local nodes to large tumour >4cm
  - III – extensive nodal disease
  - IV – mets present in other systems
- Rx: referral, MDT, surgery 1st line (for non-small cell cancers), radio, chemo (neo-adjuvant), palliative (analgesia, steroids), **nivolumab** (anti-tumour)
- 2ww criteria
  - If have CXR suggestive of lung cancer
  - >40 with unexplained haemoptysis
- Urgent CXR (2w) if
  - >40 with 1 very specific sx
  - >40y with 2 or more unexplained more non-specific sx
  - >40y, have ever smoked + 1 or more unexplained non-specific sx
- Management of chronic cough – **codeine linctus, carbocystine** (mucolytic), decongestant and antihistamines

Mesothelioma

- Cancer of the mesothelial cells in the pleura, strongest link to asbestos
- Sx: dyspnoea, weight loss, chest wall pain
- 30% present with painless pleural effusion
- Ix: CXR, pleural tap (MC&S), thoracoscopy
- Rx: chemo, surgery

- Poor prognosis, typical survival - 12 months

Pneumonia

- Sx: cough, sputum, fever, malaise
- Ex: reduced breath sounds, crackles + dull to percussion over affected lobe
- Ix: viral swab, sputum culture, FBC (white cells), blood cultures, ?HIV testing (pneumocystis jirovecii), CRP, **CXR**
- CURB65 – confusion, urea, resp rate, BP, age (>65) – >2 consider admission to hospital
- Rx:
    - **abx** in keeping with local guidelines
    - CXR at 6 weeks after clinical resolution

ILD

- Conditions affecting lung parenchyma causing inflammation fibrosis of the lung. Replaces normal elastic and functional lung tissue with stiff scar tissue
- Diagnosis: ground glass appearance on high res CT. Lung biopsy to confirm.
- Rx: remove/treat underlying cause, if hypoxic at rest give home O2, stop smoking, physio/pulm rehab, pneumococc and flu vaccine, advanced care planning, lung transplant
- Idiopathic –
    - SOB, dry cough >3 months
    - Bibasal fine inspiratory crackles, clubbing
- Drug induced – **cyclophosphamide, amiodarone, MTX, nitrofurantoin**
- Secondary – alpha-1-antitry def, RA, SLE, systemic scle
- Hypersensitivity pneumonitis/ extrinsic allergic alveolitis
    - Type III reaction – to environmental allergen parenchymal inflammation and destruction
    - Ix: bronchoalveolar lavage (collect cells via bronchoscopy by washing them and collecting that fluid) raised lymphocytes and mast cells
    - Birds, farmers (hay), mushrooms, malt workers (mould on barley)
- Asbestosis (fibrogenic and oncogenic) – pleural thickening, lung fibrosis, adenocarcinoma,, mesothelioma
    - Takes years/decades to present
    - Fatal
    - Need to be referred to coroners after death

TB

- Causative agent – mycobacterium tuberculosis – rod shaped bacilli, acid-fast, Zeihl-Neelsen stain turns TB bright red against blue
- Risk factors: alcoholism, immunocompromised, silicosis, apical fibrosis
- Pathophysiology
    - Inhalation of bacilli primary infection Gohn focus (calcified caseating granuloma containing bacteria forms) seen on CXR
    - Active infection primary pulmonary TB
    - Latent infection dwells within granuloma, when reactivated secondary pulmonary TB
- Presentation:
    - Cough, fever, anorexia, weight loss
    - Night sweats, pleuritic chest pain, haemoptysis
    - Erythema nodosum, crackles/bronchial breathing
    - Back pain, neuro symptoms spinal TB
- Ix:
    - Acute: three sputum samples, acid-fast bacilli smear MC&S –
        - Stains: Ziehl Neelsen (turns red when +ve), NAAT
        - Cultures: Lowstein Jensen (VERY SLOW, up to 8w)
        - HIV testing should always be done
        - Bronchoscopy + biopsy may be indicated
    - Latent: INF-gamma release assay, Tuberculin skin test (Mantoux)
    - CXR findings:
        - Primary pulmonary tuberculosis – consolidation, pleural effusion, ipsilateral hilar/mediastinal lymphadenopathy.

        - Secondary pulmonary tuberculosis – apical changes, cavitation, patchy consolidation, nodular opacities, tuberculomas in 5% of cases.
        - Disseminated Miliary TB – "millet seeds" appearance, uniformly distributed throughout lung fields, similar appearance to pulmonary fibrosis, haematogenous spread should be even
- Rx: RIPE
    - **Rifampicin** (rifamPISSin – red/orange urine) – 6 months
    - **Isoniazid** (IsoNUMBazid – peripheral neuropathy co-prescribe B6 pyridoxine) – 6 months
        - can cause drug induced lupus
    - **Pyrazinamide** (hyperuricaemia gout) – 2 months
    - **Ethambutol** (EYEthambutol - colour blindness + reduced visual acuity) – 2 months
    - **Streptomycin** – given for drug resistant TB
- Contact tracing – household + close contacts screen for latent TB

Acid-base

Resp acidosis – inadequate alveolar vent, ++ CO2 retention	Opiates/BDZ overdose – resp depression Guillain Barre (NM disorders) Life-threatening asthma COPD Obesity hypoventilation syndrome CNS depression
Resp alkalosis – excess alveolar ven, ++CO2 exhalation	PE, pneumothorax, pain, anxiety, CNS stimulation, altitude, pregnancy, salicylate poisoning (
Met acidosis	*Normal ion gap = losing bicarb (or renal tubular acidosis)* *Raised ion gap = everything else* *Increased acid production/ingestion*: ketones, alcohol, lactate, urate, acid poisoning, chronic **paracetamol** *Decreased acid excretion/HCO3 loss (renal or GI)* – diarrhoea, renal tubular acidosis, Addison's (retains H+), **acetazolamide**
Met alk	vomiting, diarrhoea, **diuretics**, nephrotic syndrome, Conn's, cirrhosis, addition of alkali

- Anion gap, base excess

Pneumothorax

- Marfans (deficient in fibrillin)
- Primary pneumothorax – no underlying lung disease
- Secondary pneumothorax – underlying disease – much lower threshold for intervention
- Tension pneumothorax – typically traumatic
- Aspiration = revised triangle safety = 4/5th ICS between ant and midaxillary fold. Chest drain = 5Ics MCL
- primary Rx: <2cm rim air and not SOB leave it, >2cm or SOB aspiration (if aspiration fails TWICE, then chest drain)
    - Avoid smoking, no flying for 2w after drain, NO scuba
- secondary rx: >2cm → chest drain

Pleural effusion

- CXR – blunting of costo-diaphragmatic recess. Consolidation may surround effusion if infective.
    - Transudative – systemic – lower protein + low protein pleural/serum ratio, lower LDH pleural/serum ratio – can be caused by hypoalbuminemia, heart failure.
    - Exudative – local – high protein + high pleural/serum ratio, higher LDH pleural/serum ratio – pneumonia, paraneoplastic effusion
- Ix: pleural tap, ultrasound guided send off sample for MC&S, protein, glucose, LDH, amylase
- **Light's criteria** - distinguish between a transudate and an exudate
    - Protein > 30g/L likely to be exudate, <30 likely to be transudate
    - If between 25-35 - Light's criteria should be applied

- Pleural fluid divided by serum protein = >0.5
- Pleural fluid LDH divided by serum LDH >0.6
- Pleural fluid LDH more than ⅔ the upper limit of normal serum LDH

Pulmonary embolism

- PERC (pulmonary embolism rule out criteria) used to negate need for further PE work up. Done in people deemed low risk.
- Wells score >2 CTPA.
    - Active cancer, paralysis or immobilisation, bedridden >3 days, localised tenderness along deep venous system, swollen leg, calf swelling >3cm, pitting oedema, collateral superficial veins, previous DVT
- Ix:
    - ECG – sinus tachycardia most common, S1, Q3, T3 – classic for exams
    - CXR – Fleisher sign
    - ABG – commonly hypoxemia and hypocapnia due to hyperventilation
    - CTPA – gold standard
    - VQ scan
    - Doppler ultrasound if ?DVT
    - Bedside echo – right side heart failure indicates “massive” PE
- Rx:
    - anticoagulation – **DOAC** or **LMWH** (especially if bridging for warfarin)
    - Thrombolysis – alteplase – first line in massive PE, saddle embolus

VTE prophylaxis

- Provoked VTE – 3 months anticoagulation THEN REASSESS RISK (e.g. chadsvasc)
- Unprovoked DVT – 6 months anticoagulation THEN REASSESS RISK – investigate for cancer (10% of unprovoked DVT will be diagnosed with cancer in 1 year, relevant red flag systems review)
- High risk patients identify from chadsvasc score, lifelong anticoagulation
- Rx: **Enoxaparin** (although contains animal products so consider using **fondaparinux** as alternative), or DOAC e.g. **Apixaban** (unless >150kg, eGFR <15, elderly)

Fat embolism

- Respiratory – hypoxia
- Neurological – agitation/confusion
- Petechial rash (tends to occur after the first 2 symptoms)

Empyema – pus collection

- Aspirate – acidic when sampled and run through ABG machine

Granulomatosis with polyangiitis

- Small-vessel vasculitis primarily affecting respiratory tract and kidneys – autoimmune
- Sx:
    - commonly affects nose/ears/sinuses – nosebleeds, hearing loss, sinusitis
    - saddle shaped nose due to nasal bridge collapse
    - cough, wheeze, haemoptysis
    - glomerulonephritis (haematuria, proteinuria)
- Ix: inflammatory markers CRP and ESR elevated, **c-ANCA**, CXR – cavitating lesions, epithelial crescents. Renal biopsy – epithelial crescents in bowman’s capsule
- Rx: high dose **prednisolone** if suspected - due to risk of retinal ischaemia
- Complication - retinal ischaemia, damage to vasa nervorom ⟶ mononeuritis multiplex - asymmetrical sensory + motor ischaemia, painful

Sarcoidosis

- Typical presentation = young adult, infectious trigger
- Multisystem disorder characterised by non-caseating granulomas (inflammatory nodule filled with macrophages)

- More common to be chronic in black patients
- Ex:
    - Erythema nodosum – inflammation of fat cells + Lupus pernio – raised purple skin lesions on cheeks/nose
    - Uveitis, optic neuritis
    - Dry cough/wheeze
- **Lofgren's syndrome: specific presentation of sarcoidosis, triad:**
    - Erythema nodosum
    - Bilateral hilar lymphadenopathy
    - Polyarthralgia
- Ddx: TB, lymphoma, hypersensitivity pneumonitis, HIV
- Ix:
    - raised angiotensin converting enzyme (ACE), raised calcium
    - CXR – hilar lymphadenopathy, CT – pulmonary nodules, PET – active inflammation
    - Histology – bronchoscopy with an ultrasound guided biopsy of mediastinal lymph nodes non-caseating granuloma
- Rx: **steroids** (+ **bisphosphonates** to prevent steroid causing osteoporosis), **methotrexate**
- prognosis: typically resolves within 2 years, some patients will progress to pulmonary fibrosis + HTN

Pulmonary fibrosis (interstitial lung disease)

- Broad group of conditions characterised by fibrosis of lung tissue – replacement of elastic tissue with non-functional scar tissue
- Most common cause = idiopathic
- Sx: shortness of breath on exertion, dry cough, fatigue, bibasal fine end-inspiratory crackles, clubbing
- Ix: high resolution CT thorax **ground glass** appearance. Spirometry. Lung biopsy. Bronchoalveolar lavage.
- Secondary pulmonary fibrosis
    - FEV1 and FVC equally reduced
    - Normal or restrictive pattern (FEV > 0.7)
- Causes:
    - Idiopathic – poor prognosis, 2-5 year life expectancy from dx
        - Pirfenidone – reduces fibrosis + inflammation
        - Nitedanib – tyrosine kinase inhibitor, reduces fibrosis and inflammation
    - Secondary to drugs
        - **Amiodarone**
        - **Cyclophosphamide**
        - **Methotrexate**
        - **Nitrofurantoin**
    - Secondary to other conditions
        - Alpha-1 antitrypsin deficiency
        - Rheumatoid arthritis
        - SLE
        - Systemic sclerosis
        - Sarcoidosis
    - Hypersensitivity pneumonitis – type 3&4 hypersensitivity reaction to allergen immune response
        - Bird fancier's lung – bird droppings
        - Farmer's lung – mouldy spores in hay
    - Asbestosis
        - Asbestos is fibrogenic + oncogenic (mesothelioma)
        - All asbestos related deaths must be referred to coroner
- Rx: (generally)
    - Treat underlying cause (i.e. stop medication, manage RA)
    - Home oxygen if hypoxic
    - Smoking cessation
    - Physiotherapy + rehabilitation
    - Pneumococcal + flu vaccine
    - Advanced care planning - palliative care where appropriate

Renal

NEPHRITIC vs NEPHROTIC syndrome

- (Glomerulo)Nephritis = generic inflammation of the kidneys with many causes, e.g. post streptococcal, IgA nephropathy, pyelonephritis
- Nephrotic syndrome – basement membrane of glomerulus becomes more permeable more protein passing into the urine than usual (in particular albumin). Again, multiple causes:
    - In adults: membranous nephropathy, focal segmental glomerulosclerosis, diabetic nephropathy
    - In **children**: MOST COMMON = **minimal change disease**
- Nephritic syndrome = group of features:
    - Haematuria
    - Oliguria
    - Proteinuria but **<3g in 24h** (normal = 150mg)
    - Fluid retention
- Nephrotic syndrome = group of features:
    - Proteinuria **>3g in 24hr** frothy urine
    - LOW serum albumin (albumin is crossing basement membrane and being excreted)
    - Peripheral oedema
    - Hypercholesterolaemia – chronic nephrotic syndrome complication idk why and nor did Dr Perkins
- Nephrotic syndrome management – typically ACEi (reduce proteinuria) steroids if that doesn't work
    - hypercoagulable state due to excretion of factor V

Membranous nephropathy

- Common cause of glomerulonephritis in adults - typically presents with nephrotic syndrome
- Causes: idiopathic, malignancy, infection, drugs (gold, penicillamine, NSAIDs), SLE, RA
- management = **ACEi/ARB, corticosteroid + cyclophosphamide**
- prognosis = rule of ⅓
    - ⅓ spontaneous remission
    - ⅓ remain proteinuric
    - ⅓ end stage renal failure
- good prognostic features: female sex, young age at presentation, asymptomatic

Acute tubular necrosis

- MOST COMMON CAUSE OF AKI
- Caused by prolonged ischaemia (hypoperfusion) or nephrotoxins
    - Ischaemia = shock, sepsis, dehydration
    - Nephrotoxins = NSAIDs, gentamicin, contrast, myoglobin (rhabdomyolysis)
- Pathophys: necrosis of renal tubular epithelial cells
- Ix: high urea, creatinine, K+, presence of **muddy brown casts**
- Rx: supportive, IV fluids, stop nephrotoxic meds, treat complications

Acute interstitial nephritis

- Triad: rash, fever, eosinophilia
- Caused by drugs (ESPECIALLY ANTIBIOTICS), systemic disease (SLE, sarcoidosis, Sjogren's), infection

    - Penicillin
    - Rifampicin
    - NSAIDs
    - Allopurinol
    - Furosemide
- Pathophys: marked interstitial oedema and interstitial infiltrate in connective tissue between renal tubules
- Ix: sterile pyuria (raised WCC in urine that doesn't have bacteria), white cell casts

Henoch-Schonlein Purpura (HSP)

- IgA vasculitis causing an IgA nephritis, typically 2-3 weeks post infection
- Pathophys: URTI or gastroenteritis IgA deposits in blood vessels inflammation in affected organs e.g. kidneys
- Features (4)
    - Purpuric rash on LLs and buttocks (usually children)
    - Joint pain
    - Abdo pain
    - Renal involvement
        - Testicular pain!
- Rash caused by inflammation and leaking of blood from small blood vessels under the skin purpura
- Dx: exclude meningococcal sepsis/leukaemia. Exclude ITP.
- Ix: FBC and blood film to rule out sepsis & leukaemia, look for thrombocytopenia. U&E for renal involvement. Serum albumin nephrotic syndrome. CRP for sepsis. Cultures, urine dip, protein:creatinine ratio
- Rx: supportive. Monitor BP and urinalysis for progressive renal involvement – weekly for 1st month, fortnightly until 3 months, review at 6 and 12.

Anti-GBM (Goodpasture's)

- Small vessel vasculitis affecting pulmonary vessels and the glomerulus
- Caused by anti-glomerular basement membrane antibodies vs. type IV collagen
- Associated w/: male sex, bimodal age distribution (20-30 & 60-70 years), HLA-DR2
- Presentation: Haemoptysis and proteinuria/haematuria, +/- rapid onset AKI
- Ix: renal biopsy (IgG deposition on basement membrane), increased transfer factor (pulmonary haemorrhages)
- Rx: plasma exchange, **steroids, cyclophosphamide**

Post-streptococcal glomerulonephritis

- Caused by deposition of IgG+IgM+C3 complex in the glomerulus post **streptococcal** infection (usually group A, e.g. **strep pyogenes**)
- Typical presentation: visible haematuria **1-2 weeks** post streptococcal infection
    - Fever, malaise, oedema, oliguria
    - Common in children
- Associated w/: alcoholic cirrhosis, coeliac disease, HSP
- Ix: proteinuria & haematuria on urine dip + low complement levels (c3). Raised anti-streptolysin O titre. Renal biopsy + immunofluorescence "granular/starry sky" appearance.

IgA nephropathy (Berger's disease)

- Typical presentation: visible/macro haematuria **1-2days** post onset of **URTI**
- Ix: histology – IgA deposits and mesangial proliferation (mesangial cells = cells at centre of glomerulus)
- Rx:
    - Proteinuria <500mg/day and normal eGFR – conservative, monitor renal function
    - If persistent proteinuria (above 500mg/day) **ACE inhibitors** (reduce proteinuria + improve prognosis).
    - If failure to respond to ACEi **steroids**

Minimal change disease

- *Presentation = nephrotic syndrome (oedema, proteinuria, low albumin) in child (although can occur in adults)*
- T-cell mediated damage to GBM increased permeability

- Associated w/: Hodgkin's lymphoma, EBV, **NSAIDs, rifampicin**
- Ix: urine dip, renal biopsy – looks normal on light microscopy, on electron microscopy **fusion of podocytes**
- Rx: 1st line = oral corticosteroids, 2nd line = cyclophosphamide

Focal segmental glomerulosclerosis

- *Presentation = nephrotic syndrome (oedema, proteinuria, low albumin) in young adult*
- Causes:
    - Secondary to other renal pathology e.g. IgA nephropathy, reflux nephropathy
    - HIV
    - Alport's syndrome
    - Sickle-cell anaemia
- Ix: renal biopsy **focal + segmental sclerosis** and **hyalinosis**
- Rx: **steroids**
- Worth considering – high rate of recurrence in renal transplant so not great candidates.

Wilm's tumour – nephroblastoma

- Childhood malignancy – 3-5 years most common – good prognosis (80% cured)
- Presentation:
    - abdominal mass (most common presenting feature)
    - painless haematuria
    - flank pain
    - other features: anorexia, fever
- metastases are found in 20% of patients (most commonly lung)
- Rx:
    - paediatric review within 48 hours
    - nephrectomy
    - chemotherapy
    - radiotherapy if advanced disease

UTI

- Causative organism typically **E.coli** (gram -ve rods)
- Sx: dysuria, polyuria, frequency, urgency, cloudy/smelly urine, low grade fever (<38), confusion in elderly pt
- Ix: urine dip + culture
- Rx:
    - Uncomplicated UTI in non-pregnant female: 3 days **nitrofurantoin** or **trimethoprim**
    - UTI in pregnant female (even if not symptomatic): 7 days nitro if early pregnancy, trimethoprim if late
    - UTI in male: 7 days nitro or trimethoprim
    - Catheterised pt: do not treat asymptomatic, if symptomatic 7 day treatment + consider changing catheter ASAP

Pyelonephritis

- Sx: fever, rigors, nausea/vomiting, loin pain, dysuria/polyuria especially if ascending infection
- Ix: MSU
- Rx: **co-amoxiclav** or **trimethoprim** (depending on MC&S)

Renal stones

- Most commonly calcium-based stones (**calcium oxalate** > **calcium phosphate**) – visible on AXR – hypercalcaemia as a cause (MM, hyperparathyroidism, cancer)
- Staghorn calculus – stones made from struvite – recent UTI, bacteria hydrolyse urea in urine ammonia which creates the solid struvite
- Other types: uric acid (hyperuricaemia/gout), struvite (infective), cystine (cystinuria – autosomal recessive)
- Complications – impaction (AKI), infection (obstructive pyelonephritis)
- Most common location for impaction
    - vesico-ureteric junction (ureterbladder)
    - pelvi-ureteric junction (renal pelvisureter)
    - crossing pelvic brim

- Sx:
    - Renal colic – fluctuating loin to groin pain
    - Haematuria
    - Nausea/vomiting
    - Anuria
    - Sepsis
- Ix: urine dip (haematuria + exclude infection), U&E (renal function), bone profile (hypercalcaemia), CTKUB within 24 hours of presentation
- Rx: **IM diclofenac, antiemetics, abx**
    - In stones <5mm – watchful waiting
    - In stones 5-10mm or smaller that did not pass with alpha-blocker (e.g. **Tamsulosin**) – surgical intervention
        - Extracorporeal shock wave lithotripsy
        - Ureteroscopy + laser lithotripsy
        - Percutaneous nephrolithotomy
    - Recurrent stones:
        - Diet + lifestyle advice
        - **Potassium citrate** (calcium based stones)
        - **Thiazide diuretics** (calcium based stones)

AKI

- KDIGO – increase in serum creatinine by >26 in 48h, or increase in serum creatinine to >1.5 x baseline
- RIFLE criteria to grade severity
    - Risk – 1.5x serum creatinine, UO <0.5ml/kg/hr 6hrs, eGFR decreased >25%
    - Injury – 2x serum creatinine , UO <0.5ml/kg/hr 12hrs, eGFR decreased >50%
    - Failure – 3x serum creatinine , UO <0.5ml/kg/hr 24hrs, eGFR decreased >75%
    - Loss – persistent ARF >4 weeks
    - End stage kidney disease - >3 months
- **Pre-renal** – hypovolaemia, dehydration, CHF, sepsis. LOW urine Na+
- **Renal** – **acute tubular necrosis** (most common cause of AKI) + acute interstitial nephritis High urine Na+
- **Post-renal** – prostatic hyperplasia, stones, tumours, obstruction of any kind

CKD

- Chronic reduction in kidney function, progressive
- Causes / risk fx:
    - Diabetes
    - Hypertension
    - Medications – nephrotoxic long term, e.g. NSAIDs, Lithium
    - Glomerulonephritis
    - Polycystic kidney disease
- Sx: - often non-specific or entirely asymptomatic
    - Fatigue
    - Pallor – anaemia in late-stage disease (less EPO – normocytic normochromic – AND less iron absorption (acute phase reactant hepcidin is released which decreases gut absorption)
    - Frothy urine (proteinuria)
    - Nausea
    - Loss of appetite
    - Prutitis (reduced bilirubin excretion)
    - Oedema
    - Hypertension
    - Peripheral neuropathy
- Ix:
    - Urine dip – proteinuria + haematuria (macroscopic might indicate infection, malignancy, stones)
    - BP – ?hypertension as cause
    - Urine albumin:creatinine ratio
    - HbA1c – diabetic nephropathy as cause
    - Lipid profile

G Stage	eGFR		A Stage	Albumin:Creatinine Ratio
G1	Over 90		A1	Under 3 mg/mmol
G2	60-89		A2	3-30 mg/mmol

    - U&Es – eGFR + urea
    - Renal ultrasound – polycystic kidney disease
- Classification – diagnosis can be made if:
    - **eGFR <60**
    - **Urine ACR >3**
    - G and A stage
- Accelerated progression – decline in eGFR of 25% or 15ml
- Rx = risk factor reduction
    - BP – **ACE/ARB** – monitor potassium carefully
    - CVD – **statin**
    - Diabetes – HBA1c monitoring, **SGLT-2 (empaglifozin/dapaglifoxin)**
    - Metabolic acidosis (shit renal function less bicarb produced) – oral **sodium bicarbonate**
    - Renal bone disease
        - kidneys convert calcidiol to calcitriol (active vit D) AND excrete phosphate, therefore calcium is not absorbed from gut, causes LOW serum calcium and HIGH serum phosphate
        - low serum calcium increased PTH increased osteoclast activity shit bone density
        - Ix: on XR – osteomalacia, osteosclerosis, rugger jersey spine
        - Rx: reduce serum phosphate and PTH
            - Phosphate binder – **sevelamer**
            - Vitamin D – alfacalcidol / calcitriol
            - Parathyroidectomy
    - Renal replacement therapy for stage 5 or evidence of uraemia
        - Haemodialysis
        - Peritoneal dialysis
    - Anaemia – treat any iron deficiency before giving EPO

Nephrotoxins

- NSAIDs
- Gentamicin
- Other abx: rifampicin, penicllins,
- Anticonvulsants:
- Contrast
- Rhabdomyolysis – myoglobin filtered by glomeruli and obstructs renal tubules

Haemolytic uraemic syndrome

- Triad: haemolytic anaemia, AKI, thrombocytopenia
- Caused by shiga toxin (produced by ***shigella*** and ***e coli-0157***) – causes haemolysis + thrombocytopenia
- Commonly affects children approx. 1 week after episode of gastroenteritis (bloody diarrhoea)
- Antibiotics + antimotility medication (e.g. **loperamide**) used as treatment can precipitate
- Sx: confusion (uraemia from breakdown of RBCs), pallor, bruising, abdo pain
- Ix: stool culture
- Rx: supportive, dialysis + blood transfusion may be required

Polycystic kidney disease

- Genetic condition where healthy renal tissue is replaced with fluid filled cysts
- Autosomal dominant = most common. Mostly **PKD1 gene** on **chromosome 16.**
- Autosomal recessive = less common but worse, picked up on antenatal scans (oligohydramnios, pulmonary hypoplasia, may require haemodialysis)
- Extra-renal manifestations = cerebral aneurysms (berry aneurysms – SAH), hepatic, splenic, pancreatic + ovarian cysts, mitral regurg, colonic diverticula
- Complications:
    - Chronic loin pain
    - Hypertension
    - Gross haematuria (cyst rupture)
    - Recurrent UTI
    - Renal stones
    - Renal failure (usually by 50)

- Rx:
    - Antihypertensives
    - **Tolvaptan** (selective vasopressin antagonist, decreases cyst formation)
    - Analgesia
    - Abx
    - Drainage of cysts
    - Dialysis
    - Renal transplant
    - Avoid NSAIDs + anticoagulants, contact sports, MR angiography

Alport's syndrome

- X-linked connective tissue disorder affecting type IV collagen
- Results in abnormal glomerular-basement membrane.
- Presents in childhood with sx:
    - Microscopic haematuria
    - Progressive renal failure
    - Bilateral sensorineural deafness
    - Lenticonus – protrusion of lens surface into anterior chamber
    - Retinitis pigmentosa
    - Renal biopsy – **splitting of lamina densa, basket weave appearance** on EM
- Ix: genetic testing
- Weird passmed thing: Alport's patient rejecting a kidney transplant = presence of anti-GBM antibodies

Rheumatology

HLA associations

- HLA-A3
  - haemochromatosis
- HLA-B51
  - Behcet's disease
- HLA-B27
  - ankylosing spondylitis
  - reactive arthritis
  - acute anterior uveitis
  - psoriatic arthritis
- HLA-DQ2/DQ8
  - coeliac disease
- HLA-DR2
  - narcolepsy
  - Goodpasture's
- HLA-DR3
  - dermatitis herpetiformis
  - Sjogren's syndrome
  - primary biliary cirrhosis
- HLA-DR4
  - type 1 diabetes mellitus*
  - rheumatoid arthritis - in particular the DRB1 gene (DRB1*04:01 and DRB1*04:04 hence the association with DR4)

SLE

- Autoimmune connective tissue condition resulting in chronic inflammation
- Pathophys: ANA start attacking nucleus, causing inflammatory response
- Multi system
- Causes lots of complications like CVD and infection, leading causes of death
- Risk factors: Fm, Asian, middle aged
- Main sx. – relapsing-remitting + flares
  - Rashes: Malar butterfly rash , subacute cutaneous, discoid
  - Lupus nephritis
  - Non-specific sx: fatigue, weight loss, arthralgia, fever, lymphad, splenomeg, SOB, pleuritic chest pain, mouth ulcers, Raynauds, hair loss
  - subluxation of MCPs
- Ix
  - ANA (sensitive)
  - Ant-dsDNA (specific)
  - Anaemia chronic disease
  - Low complement – body using them up quicker than liver can produce them
  - CRP/ESR, immunoglobs raised
  - Urine protein: creatinine + renal biopsy if suspect lup nephritis
- Rx
  - 1st line: **NSAIDS**, **prednisolone** (flares), **hydroxychloroquine**, sunscreen
  - 2nd line (more severe/resistant): **MTX, ciclosporin**
  - 3rd line: **rituximab**
- Complications: shit ton. CVD leading cause of death (chronic inflamm in blood vessels □ HTN □ CAD). Pericardial effusion, psychiatric sx, vasculitis ⟶ hypercoagulable, renal disease,

Raynaud's

- Exaggerated vasoconstrictive response of digital arteries and cutaneous arteriole to cold or emotional stress
- Maturity onset = red flag - Scleroderma/Gastric cancer
- Can be idiopathic, especially in young women
- Underlying disorders causing secondary Raynaud's:

    - Scleroderma
    - RA
    - SLE
    - Leukaemia
- Sx: Skin change: White → Blue → Red

Sjogren's

- Autoimmune damage to secretory glands
    - Primary: SICCA syndrome
    - Secondary: associated with other autoimmune (SLE)
- Pathophys: HLA/DRQ + recent infection triggers disease. Auotoantibodies (ANA, anti-Ro/anti-La) form complex with antigen > immune response in exocrine glands > glands gets obstructed > stop functioning
- Dry mucous membranes: eyes, mouth (xerostomia), vagina, cough, dental caries (tooth decay) (severe can lose nasal septum)
- Ex: Schirmer (<10mm = significant)
- Ix: anaem of chronic disease, raised IgG, raised ESR, ANA positive, Anti-Ro and Anti-La, biopsy (abnormal salivary gland, high "focus" score)
- Rx: **hydroxychloroquine** (halts progression), **pilocarpine** (stimulates saliva prod) artificial tears/saliva subs, saliva stimulants, dental care, vaginal lubricant
- Complications: lymphoma, ILD, vasculitis/periph neuropathies

Systemic sclerosis

- Limited cut = CREST
- Diffuse cut = CREST + problems with internal organs (CV, lung, renal)
- Sx: scleroderma (hardening skin, tight, shiny), sclerodactyly (hardens and restricts movement), telangiectasia, calcinosis, oesophageal dysmotility (dysphagia, acid reflux, oesophagitis), syst and pulm HTN, pulm fibrosis
- Ix:
    - ANA
    - Limit cut = anti-centromere antibodies
    - Diffuse cut = anti-Scl-70
    - Nailfold capillaroscopy – abnormal capillaries,
- Rx: MDT, lifestyle, med (no exact treatment) – **steroids** + **immunosuppressants**

Hereditary haemolytic telangiectasia

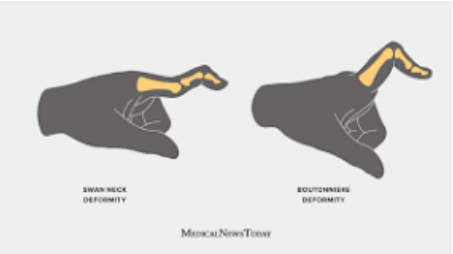
- Genetic
- Mucosal telangiectasia throughout GI tract
- AVMs - can haemorrhage

Ankylosing spondylitis - now called ankylosing spondyloarthopathy

- Axial spondyloarthritis. Sacroiliitis XR. Seronegative/ HLA-B27
- Mainly affects spine – progressive stiffness, pain. Severe: fusion bamboo spine on XR
- Risk factors: <45y, HLA-B27 (class 1), male sex, 20-30 years classic presentation
- Sx: sx gradually >3m, worse at night and in the morning, >30m in morning for stiffness to improve, sacroiliac pain in butt, flares, relief with exercise
    - Extra-articular: the As: arthritis, anterior uveitis, psoriasis, IBD, GU infection i.e. urethritis), enthesitis (plantar fasciitis), dactylitis, anaemia, aortitis, apical fibrosis, aortic regurgitation, Achilles tenonitis, AV node block, amyloidosis, Atlanto-axial disease
- Exam: Schober, modified new York criteria (radiological + ≥ 1 clinical criterion), occiput to wall distance, increased thoracic kyphosis + reduced lumbar lordosis, decreased chest expansion (more than 5cm)
    - Schob: L5, 10cm above and 5cm below, bend, <20cm = restriction
    - Clinical criteria –
        - lower back pain for >3m
        - improved by exercise but not relieved by rest
        - limitation of lumbar spine motion

        - limitation of chest expansion relative to age/sex
    - Radiological criteria
        - Sacroiliitis on XR (bone marrow oedema, fusion)
- Ix:
    - FBC (anaemia of chronic disease), CRP/ESR, U&Es
    - XR (sacroiliitis, bamboo spine, dagger spine from longitudinal ligament ossification, syndesmophytes, shiny corner)
    - MRI (bone marrow oedema early disease), HLA-B27 genetic test
    - CXR - apical fibrosis in progressed disease
- Rx:
    - Lifestyle (smoking cess, PT, exercise)
    - Pain – NSAIDs generally good response
    - Flares – **steroids, sulfasalazine, methotrexate**
    - Anti-TNF (**etanercept**, **infliximab**, **adalimumab**)
    - Bisphosphonates – for osteoporosis
    - Rx complications - e.g. OA of other joints (hips/knees), osteoporosis
    - pre-operative assessment by anaesthetist due to cervical spine involvement
    - Surgery rare (spine deformities)

Rheumatoid arthritis (RA)



- Autoimmune inflammatory condition, inflammation of synovium
- HLA DR4/DR1 (T1DM)
- Sx: symmetrical joint pain, swelling, stiffness. PIPs, MCPs. Rheumatoid nodules. Swan neck deformity, ulnar deviation. Telescoping. Boutonniere deformity. Olecranon bursitis. **Arthritis mutilans** – severe deformity “telescoping” of fingers
- Polyarthalgia = >5 joints
- Commonly hands, C-spine, shoulders
- In feet - rheumatoid nodule overlying the achilles tendon, pes planus, subtalar valgus
- Ix: RF, anti-CCP (most specific), CRP (monitor response to rx), XR (LESS), USS/MRI joints – periarticular erosions
- Rx: refer to rheum, HAQ annual review. Pregnancy – **sulfasalazine** and **hydroxychloroquine** safe
    - 1st: **DMARD monotherapy** (**MTX, leflunomide, sulfasalazine**) + short-course **pred** (hydroxychloroquine if mild) **methotrexate side effect = pneumonitis pulmonary fibrosis**
    - 2nd: **DMARD dual therapy**
    - 3rd line: **MTX + biologic (infliximab)**
    - 4th line: **MTX + rituximab**
    - Flare ups = steroid injection (**methylprednisolone acetate**)
- Complications:
    - Resp: pulm fibrosis, pleural effusion, pulmonary nodules,
    - ocular: keratoconjunctivitis SICCA, episcleritis, scleritis, corneral ulceration ,keratotitis
    - cardiac: rheumatoid nodules causing arrhythmias
    - osteoporosis
    - Skin - rheumatoid nodules, vasculitis
    - IHD
    - Neuro - carpal tunnel syndrome, radiculopathy
    - Increased risk infx
    - Depression
    - Random: Felty’s syndrome (splenomegaly, neutropenia, low WCC, raised MCV), amyloidosis (deposits of protein around organs due to chronic inflammation – esp renal)
    - Vasculitis → Mono-neuritis multiplex

Polymyalgia rheumatica

- Pain, stiffness (differentiated from polymyositis by WEAKNESS, polymyositis is weakness without pain)
- Risk factors: >60y, female, Caucasian
- Proximal muscle stiffness and raised inflammatory markers
- Associated with **temporal arteritis** (ischaemia to masseter and temporalis pain on chewing)
- Sx: bilateral aching, morning stiffness in PROXIMAL limb (shoulder, pelvic) with NO weakness
- Ix: raised ESR/CRP, normal CK and EMG (polymyositis raised CK)
- Rx: **prednisolone**

Psoriatic arthropathy

- Inflammatory polyarthritis associated with psoriasis – seronegative spondylopathy. Often precedes the development of skin lesions.
- Sx:
    - symmetric polyarthritis, similar in presentation to RA – more common
    - OR asymmetric oligoarthritis affecting hands and feet
    - Sacroiliitis
    - **Arthritis mutilans** – severe deformity "telescoping" of fingers
    - Peri-articular disease, tenosynovitis and soft tissue inflammation enthesitis, tenosynovitis, dactylitis (diffuse swelling of finger/toe)
    - Pitting, onycholysis, hyperkeratosis
- Ix: XR – pencil-in-cup appearance, coexistence of erosive changes and new bone formation
- Rx: NSAIDs methotrexate biologics (**adalimumab, etanercept**)
- Complication - increased risk of cardiovascular disease

Neuro

Alzheimer's

- Progressive degenerative disease of the brain, - majority of dementia seen in the UK
- Widespread cerebral atrophy, especially cortex + hippocampus
    - Decreased acetylcholine from damage to ascending forebrain – amyloid plaques + tau tangles
- 6CIT, GPCOG, MMSE – cognitive screening in GP
    - 4AT and CAM more delirium, done in hospital
- Ex-vacuo dilatation, hippocampal dilatation
- Rx:
    - Conservative: **MEMORY CLINIC**
    - Mild-moderate = **donepezil, galantamine & rivastigmine** (acetylcholinesterase inhibitor)
    - Severe = **memantine** (NMDA antagonist)
- Donepezil first, memantine second, yearly annual check (Dementia Care Plan)

Vascular dementia

- Stepwise deterioration
- Stroke related – having a stroke doubles risk of getting dementia
- Can be mixed with alzheimer's

Huntington's

- Autosomal dominant inheritance
- CTG repeat – anticipation
- Loss of **GABA** (inhibitory neurotransmitter) in striatum
- Chorea, eye movement, speech difficulties
- Rx: dopamine-depleting drugs (**tetrabenazine**), **antipsychotics** (2nd gen), **BDZ**

DLB

- Dementia first, parkinsonian sx second (a year between sx)
- lewy bodies deposited within the substantia nigra → parkinsonism
- Sx: progressive cognitive impairment, parkinsonism, visual hallucinations

- Clinical diagnosis, same Rx: as alzheimer's (**Donepezil** + **Memantine**)

Wernicke's encephalopathy

- Excess alcohol → thiamine deficiency (B1 is poorly absorbed in the presence of alcohol)
- Ataxia, confusion, ophthalmoplegia
- Rx: **Pabrinex** (currently national shortage of pabrinex, can use **IV thiamine** and **oral Vit C**), abstain from alcohol

Korsakoff syndrome

- Irreversible
- Amnesia - retrograde and anterograde + confabulation

Stroke

- NIHSS score – based of examination findings – grades from 0 (no stroke symptoms) – 42 (severe stroke)
- Criteria
    - unilateral hemiparesis and/or hemisensory loss of the face, arm & leg
    - homonymous hemianopia
    - higher cerebral dysfunction e.g. dysphasia
- **TACS** = 3/3
- **PACS** = 2/3
- **Lacunar** – ONE:
    - unilateral weakness (and/or sensory deficit) of face and arm, arm and leg or all three.
    - pure sensory stroke
    - ataxic hemiparesis
- **POCS:** involves vertebrobasilar arteries. ONE:
    - cerebellar or brainstem syndromes
    - loss of consciousness
    - isolated homonymous hemianopia
    - nystagmus, vertigo, dysdiadochokinesia
- **Lateral medullary syndrome (PICA)** aka Wallenberg's syndrome
    - ipsilateral: ataxia, nystagmus, dysphagia, facial numbness, cranial nerve palsy e.g. Horner's
    - contralateral: limb sensory loss
- Carotid endarterectomy 2-4 weeks post stroke: >50-99% stenosis
- **Extradural** – middle meningeal artery
- **Subdural** – bridging vein rupture (old, alcohol)
- **Subarachnoid haemorrhage** – thunderclap headache, meningism, berry aneurysm rupture (PCKD)
    - Enlarged/hyperdense ventricles.
    - ECG – t wave inversion
    - Increasing ICP – hydrocephalus
    - complication = vasospasm, prevent using **Nimodipine**
- **Cerebral venous thrombosis** – prevents drainage from venous sinus, increases ICP, headache, nausea <1% of strokes, pregnancy, antiphospholipid syndrome, malignancy
- **Posterior communicating artery** - 3rd nerve palsy that is PAINFUL, headache + eye pain, ipsilateral
- **Weber's syndrome -** midbrain stroke, ipsilateral 3rd nerve palsy, eye is not painful
- **Aphasias:**
    - Expressive aphasia - Broca's area
    - Receptive aphasia - Wernike's area
    - Conduction aphasia - Arcuate fasciculus (connection between Broca's and Wernike's area) - problems repeating back phrases, speech mostly fluent
    - Global aphasia - all of the above
- Ix: obs, ECG, blood glucose, cholesterol, clotting screen, TFTs (hyperthyroidism strokes). Head CT on arrival – rule out haemorrhage. MRI = definitive. Carotid doppler. Swallow assessment.
- ISCHAEMIC management
    - Control BP and glucose, physio and SALT, risk assessment DVT & prophylaxis (flotron)
    - 1st line: **aspirin**, **alteplase** (within 4.5h), outside 4.5hr can increase risk of haemorrhagic transformation.
    - Thrombectomy (6-24h)

    - if outside window high dose **(300mg) aspirin**
- **High dose aspirin for two weeks, then start clopidogrel**
- Secondary prevention (4 A's)
    - **Antiplatelet, atorvastatin, ACEi, anticoag**
    - If AF – **DOAC -** unless >150kg, eGFR <15, elderly
    - DVLA – **1 month** driving ban
- HAEMORRHAGIC management surgery
    - BP control, elevate head of the bed to reduce ICP, avoid endotracheal tube holders/central line dressing that increases afterload
    - Reverse anticoagulation (e.g. **vit k** for warfarin, **octaplex** more broadly)

TIA

- Resolution of symptoms within 24 hours
- Refer to **TIA clinic within 24 hours**, give **300mg aspirin**
- **Amaurosis fugax** – occlusion of central retinal artery, resolves typically in minutes.
- $ABCD^2$ – risk assessment (risk of stroke in future)

Migraines

- Common type of primary headache, unknown pathophysiology
- Sx: severe, unilateral, throbbing. <72 hours. Photophobia. Classically precipitated by "aura". Hemiplegic migraines rare.
- Triggers: tiredness/stress, alcohol, COCP, menstruation
- Acute rx: triptan + NSAID or triptan + paracetamol
- Prophylaxis: propranolol

Cluster headaches

- 15-180 mins
- Unilateral pain, autonomic symptom e.g. horner's syndrome, restlessness/agitation
- At least 5 attacks
- Acute: triptans, oxygen
- Prophylaxis – verapamil

Meningitis

- Notifiable disease
- Reservoir species = humans, lives on nasopharyngeal mucosa, break in mucosa systemic infection
- Infection and inflammation of the meninges
- Bacterial (meningococcal, pneumococcal): **Neisseria meningitidis** (gram negative diplococci), **strep pneumoniae**, **group B strep** (neonates)
- Viral (HSV1)– less common, treated with oral **acyclovir**
- Meningococcal septicaemia – non-blanching rash, DIC
- Sx: neck stiffness, photophobia, fever, headache, altered consciousness. Neonates = hypotonia, poor feeding, bulging fontanelle, papilloedema,
- Exam: Kernig (spinal pain on passive knee extension with hip fully flexed), Brudzinski (involuntary flex hip/knee when head passively flexed), glass test (meningococcal rash)
- Ix: LP (compare with serum glucose), blood culture (meningococcal PCR)
- Rx:
    - Bacterial WITH non-blanching rash +/- septicaemia: **IM benzylpenicillin** ASAP in community, **IV cefotaxime** in hospital,**+ amoxicillin** in patients <3months or >50 years to cover **listeria**
    - Bacterial without non-blanching rash: **benzylpenicillin, cefotaxime** or **chloramphenicol**
        - **Dexamethasone** to reduce risk of hearing loss
    - Contacts: **ciprofloxacin**
    - Viral: oral **acyclovir**
- Complications: HEARING LOSS, seizures, focal neuro deficit, learning difficulties, cerebral palsy

Encephalitis

- Inflammation of the brain – common causative organism = **HSV-1** (95% of cases)
- Sx: fever, headache, focal neurological signs, vomiting, seizures
- Ix: MRI, EEG, LP – lymphocytosis, elevated protein, PCR
- Rx: **IV acyclovir**

Charcot Marie tooth

- Autosomal dominant – progressive, no rx
- Dysfunction in myelin or axons > peripheral neuropathy (sensory and motor)
- Onset usually <10y but can be up to 40y
- Sx: high foot arches (pes cavus), distal muscle wasting (inverted champagne legs), LL weakness (loss ankle dorsiflex), reduced tone/reflex/sensory
- MDT, physio, OT, podiatry

Lambert-Eaton syndrome

- Autoimmune disease of the NMJ
- 40% associated with malignancy – small cell lung cancer – paraneoplastic syndrome
- Power increased with increased contraction
- Sx: insidious onset, proximal muscle weakness, ptosis, diplopia (intraocular muscle weakness), slurred speech/dysphagia, autonomic dysfunction (dry mouth, blurred vision, impotence, dizziness)
- Ex: reduced tendon reflexes**, post tetanic potentiation**
- Rx: investigate for SCLC and treat**, amifampridine**

Myasthenia gravis

- Disease of NMJ – MG
- Power decreases with increased contraction
- Auto-immune, anti-acetylcholine receptor antibodies, blocks receptor and prevents muscle contraction
- Can be caused by thymoma
- Sx: affects small muscles in head and neck, diplopia, ptosis, fatigue when chewing, progressive weakness with repetitive movements
- Ix: acetylcholine receptor antibodies + Edrophonium test (**edrophonium chloride/neostigmine** prevents breakdown of acetylcholine improvement in symptoms)
- Rx: **neostigmine/pyridostigmine**, immunosuppression, thymectomy
- Myasthenic crisis – weakness and failure of respiratory muscles
    - NIV or full intubation
    - Immunomodulatory therapy – IV immunoglobulins, plasma exchange

Syringomyelia

- Associated with chiari malformation – cerebellar tonsils descending through foramen magnum
- Classic patient has burns to hands without realising
- Lesions at C4-C5 affecting anterior white commissure, anterior horns and corticospinal tract
- LMN weakness around C4-5-6 myotome incomplete paralysis
    - Cape-like loss of pain and temp sensation due to compression of spinothalam tract fibres decussating in anterior white commissure resulting in loss of pain/temp sensory but light touch/proprioception preserved
    - Lower limb weakness
    - Neuropathic pain
    - Upgoing plantars
- Ix: full spine MRI – exclude tumour or tethered cord

Guillain-Barre

- Demyelinating polyneuropathy
- Commonly post viral infection
- Ix – nerve conduction studies and LP, elevated protein
- Management – Ig immunoglobulins, plasma

Friedrich's ataxia

- Genetic progressive neurodegen of corticospinal tracts, dorsal column., spinocerebellar tracts + hypetroph cardiomyopathy and DM
- Sx: ataxia, slurred speech, spasticity, dysphagia, limb sensory loss, vision and hearing loss
- Usually appear in teens

Multiple sclerosis

- Chronic and progressive autoimmune condition characterised by demyelinating lesions in the CNS, "disseminated in time and space"
- In early disease, oligodendrocytes will remyelinate the neurons, creating a remission phase
- Typical patient = young female with other autoimmune conditions
- Sx:
    - Most common presentation = optic neuritis
        - unilateral visual disturbance
        - central scotoma
        - pain on eye movement
        - impaired colour vision
        - Relative afferent pupillary defect (reduced response when shining light into affected eye, consensual response to shining light into unaffected eye is normal)
        - Rx: high dose **steroids**, **urgent ophthalmology referral**
    - Focal neurological symptoms - sensory or motor
        - Horner's syndrome
        - Incontinence
        - Facial nerve palsy
        - Limb paralysis
        - Trigeminal neuralgia
        - Paresthesia/numbness
    - Ataxia - +ve Rhomberg's test
- Disease patterns:
    - Clinically isolated syndrome - single initial episode. May or may not go on to develop MS. Lesions on MRI = more likely to progress.
    - Relapsing remitting = most common, can be further classified into active/inactive, or worsening/not worsening (of disability over time)
    - Primary progressive - worsening without remission
    - Secondary progressive - was relapsing/remitting, now progressive
- Dx: MRI, LP (**oligoclonal bands** in the CSF)
- Rx: High dose **steroids** during relapses, MDT approach with neuro, specialist nurses, physio + OT
- baclofen for spasticity

Motor Neurone Disease

- Group of progressive neurological diseases affecting upper and lower motor neurons, with sparing of sensory neurons
- Risk fx: Family history, smoking, heavy metal + pesticide exposure
- Typical presentation:
    - Male in his 50/60s, insidious progressive weakness throughout the body, first noticed in upper limbs most commonly
    - "Clumsiness" + slurred speech
- Ex: mixed UMN and LMN signs
    - HENS For My Farm House
        - Hyperreflexia
        - Extensor plantar
        - No wasting (disuse atrophy long term)
        - Spastic paralysis
        - Fasciculations
        - Muscle wasting
        - Flaccid paralysis
        - Hyporeflexia

- Amyotrophic lateral sclerosis - most common
- Progressive bulbar palsy - second most common - affects talking/swallowing (bulbar muscles)
    - Ex: tongue fasciculations, tongue wasting
- Dx: process of exclusion
- Rx: no effective treatment.
    - Riluzole can slow progression by several months
    - NIV when respiratory muscles weaken
    - MDT, breaking bad news
    - Symptom control
        - **Baclofen** - muscle spasticity
        - **Antimuscarinics** for extensive salivation
        - **Benzodiazepines** - anxiety
- Complications → aspiration, pneumonia, respiratory failure

Brain Mets

- Sx: headache, worse when lying down, focal neurology, seizures
- Ex: Increased ICP (fundoscopy) + focal neurology
- Ix: Imaging – CT head and MRI
- Rx: Dexamethasone, chemo, radiotherapy, surgery – typically poor outcomes for whole brain radiotherapy

Bell's Palsy

- Lower motor neuron – unilateral facial weakness without forehead sparing
- Viral induced?
- Rx: Oral **prednisolone** within 72hrs, if no improvement within 3 weeks → ENT
- Most people fully recover within 4 months

Ramsay hunt syndrome

- Skin changes (near ear typically)
- LMN unilateral face weakness (no forehead sparing)
- Varicella zoster reactivated within the geniculate ganglion of CNVII→ auricular pain, facial nerve palsy, vesicular rash around the ear
- Rx: **aciclovir** + **prednisolone**

Chickenpox → Shingles

- Varicella zoster virus
- Generalised vesicular rash
- Typically starts on trunk and spreads all over the body
- When lesions scab over no longer contagious (resp droplets or contact with lesions is typical transmission)
- Inc. period 10 days-3 weeks
- Sx: rash, itch, general fatigue/malaise, fever
- Rx: **Aciclovir** in immunocompromised pts, adults/adolescents >14 years presenting in first 24hrs, or neonates
- Complications: bacterial superinfection (**staph**), pneumonia, encephalitis (sx: ataxia)
- Shingles = reactivation of dormant VSV, sensory dorsal root ganglion cells and cranial nerves can be affected
    - Ramsay Hunt syndrome = Facial nerve - LMN palsy, rash around the ear. Rx: **aciclovir + prednisolone**
        - Facial nerve branches: **T**o **Z**anzibar **B**y **M**otor **C**ar (**T**emporal, **Z**ygomatic, **B**uccal, **M**arginal mandibular, **C**ervical)
    - Shingles = dermatomal rash + neuropathic pain

Trigeminal neuralgia

- Episodes of “shock like” pain
- Rx: **carbamazepine**

Cauda Equina

- Saddle paraesthesia, leg weakness, incontinence, sexual dysfunction, reduced anal tone, bilateral sciatica
- In younger pt – typically disc herniation
- In older ?malignancy - most common cancers metastasizing to bone:
    - Prostate
    - Renal
    - Thyroid
    - Breast
    - Lung
- **Dexamethasone** immediately if caused by malignancy – reduce swelling
- Surgical decompression as an emergency – within 24hrs

Sciatica

- Symptoms arising due to irritation of the sciatic nerve
- Nerve roots = L4-S3
- Anatomical route - exits posterior pelvis via greater sciatic foramen in the buttock area, travels posteriorly into the popliteal fossa where it divides into the common fibular and tibial nerve
- Sx: paresthesia, numbness, weakness, reflexes depending on level of affect
- MAIN CAUSES: herniated disc, spondylolisthesis, spinal stenosis, malignancy (particularly bilateral), trauma
- Ex: Localised tenderness (fracture/malignancy), neurological changes, bladder distension, reduced anal tone, sciatic stretch test
- Ix: XR, CT, MRI, inflammatory markers (ank spond)
- Rx:
    - physio, exercise, CBT.
    - **NSAIDs**, **benzodiazepines** short term for muscle spasm, amitriptyline/duloxetine, epidural **corticosteroid injection**
    - surgical decompression

Parkinsons

- Disease caused by progressive reduction of dopamine in the basal ganglia, resulting in disordered movement. Characteristically ASYMMETRICAL, older onset
- Pathonomic triad:
    - Bradykinesia – movements get slower and smaller, micrographia, hypomimia, shuffling gait
    - Rigidity – “cogwheel” (rigidity vs spasticity = extra-pyramidal vs pyramidal tracts)
    - Tremor – asymmetrical, improves on voluntary movement, no change with alcohol, worse at rest
        - Vs benign essential tremor, which is the opposite for each
- Extra-pyramidal signs
    - Akathisia – fidgety/restless
    - Dystonic reactions – oculogyric crisis, SCM muscle
    - Tardive dyskinesia – hands/face (pillrolling/lipsmacking) (can also be caused by withdrawal emergent syndrome after the medication has been stopped)
- **Parkinson’s-plus syndromes**
    - **Multiple systems atrophy** – neurons in multiple systems degenerate, including within the basal ganglia. Causes:
        - Autonomic dysfunction, e.g. postural hypotension, constipation, sweating
        - Cerebellar dysfunction, e.g. Ataxia
    - **Progressive supranuclear palsy** - postural instability + falls, stiff broad gait, trochlear nerve palsy, parkinsonism, frontal lobe dysfunction (reduction in inhibition, emotional outbursts)
- Ix: clinical diagnosis, but rule out other causes of symptoms e.g. drug induced, stroke, infection, hydrocephalus etc (bloods, head CT)
- Rx: **Levodopa** PLUS
    - Decarboxylase inhibitor e.g. **carbidopa**
    - COMT inhibitor – e.g. **entacapone**
    - MAOB inhibitor – e.g **selegiline**

- Some argument for dopamine agonists before levodopa if quality of life is not affected by motor sx.
- **Levodopa** side effects: dry mouth, anorexia, palpitations, postural hypotension, psychosis
    - Drug counselling: take it 3-4 times a day, consider on/off effect, + end dose deterioration

Normal pressure hydrocephalus

- Reversible cause of dementia seen in older patients, thought to be due to reduced CSF reabsorption by the chorionic villi. May be secondary to head injury, meningitis, SAH
- Classic triad (wet, wacky, wobbly)
    - Urinary incontinence
    - Dementia, bradyphrenia
    - Gait abnormality (similar to parkinsons)
- Symptoms may develop over several months
- Ix: CT head ventriculomegaly. (rule out acute causes of delirium – pinch me)
- Rx: ventriculoperitoneal shunting

Epilepsy

- Group of conditions which involve transient episodes of abnormal electrical activity in the brain
- Types:
    - Generalized tonic-clonic – tonic = tensing, clonic = jerking, tongue biting, incontinence
        - **Sodium valproate lamotrigine/ carbamazepine/ levetiracetam**
    - Focal seizure – temporal lobe – hallucination, flashbacks, de ja vu, lip-smacking
        - **Carbamazepine/lamotrigine sodium valproate/ levetiracetam**
    - Absence seizure – blank staring, unresponsive, common in children
        - **Sodium valproate/ ethosuximide**
    - Atonic seizures – drop attack
    - Infantile spasms – west syndrome, full body spasms, poor prognosis
    - Febrile convulsions – 6 months to 5 years old. No lasting effect.
- Kepra = levetiracetam
- Ix: EEG – children are allowed one simple seizure before being investigated. MRI should be considered in children <2, and focal seizures (space occupying lesions). ECG, electrolytes, glucose, cultures to rule out other causes of seizures.
- DRIVING – 6 months from seizure
- Drugs + side effects
    - Lamotrigine – steven johnson syndrome (DRESS), leukopenia
    - Carbamazepine – agranulocytosis, aplastic anaemia
    - Phenytoin – folate/vit D deficiency, megaloblastic anaemia, osteomalacia
- Status epilepticus – seizure lasting more than 5 minutes or 2+ seizures without gaining consciousness in between – !!airway!!
    - **IV lorazepam 2nd dose lorazepam IV phenytoin phenobarbital**

Idiopathic intracranial hypertension

- Risk fx: obesity, female sex, pregnancy, drugs (COCP, steroids, tetracyclines, retinoids, lithium)
- Sx: headache, blurred vision, papilloedema, enlarged blind spot, abducens palsy
- Rx: weight loss, carbonic anhydrase inhibitor – **acetazolamide**, **topiramate,** shunt

Subacute combined degeneration of the spinal cord

- Vitamin B12 deficiency resulting in impairment of the dorsal columns, lateral corticospinal tracts and spinocerebellar tracts
- Recreational nitrous oxide inhalation may also result in vitamin B12 deficiency → subacute combined degeneration of the spinal cord.
- Sx:
    - Dorsal column involvement
    - Distal tingling/burning/sensory loss is symmetrical and tends to affect the legs more than the arms
    - Impaired proprioception and vibration sense
    - Lateral corticospinal tract involvement
    - Muscle weakness, hyperreflexia, and spasticity

    - Upper motor neuron signs typically develop in the legs first
    - Brisk knee reflexes
    - Absent ankle jerks
    - Extensor plantars
    - Positive romberg's sign

Transverse myelitis

- Site of inflammation in the spinal cord, resulting in sensory and/or motor symptoms depending on the location of the lesion
- Rapid onset
- Commonly bilateral symptoms + pain over the area of spine affected
- Duration between 3-6 months → permanent
- Can occur independently or as complication of infection, or associated with other inflammatory conditions:
    - Multiple sclerosis
    - Systemic Lupus Erythematosus
    - Sarcoidosis
    - RA
    - Enteroviruses, HIV, Syphilis, Mycoplasma, Malaria
- Ix: rule out compressive cause (e.g. malignancy, herniation, fracture) and consider associated conditions e.g. LP oligoclonal banding (MS), ANAs (autoimmune inflammatory conditions), HIV/syphilis antibodies, blood cultures/smear
- Rx: **HIGH DOSE GLUCOCORTICOID/DEXAMETHASONE** + treat cause

Screening

Breast

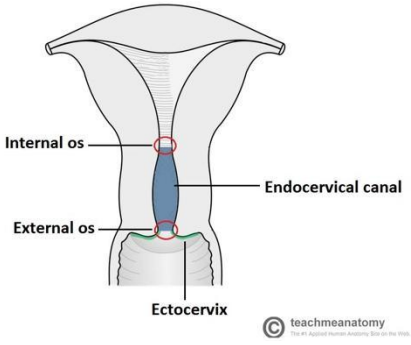
- 50-70 every 3 years
- Earlier if high risk (from 25y) – BRCA1, BRCA2
- Salpingo-oophorectomy at 40y to reduce risk
- **Triple assessment:**
    - Consultant appointment – history and exam
    - Imaging: mammogram (>40y) or USS (<40y)
    - Biopsy (FNA or core, USS-guided)

Bowel

- 55y – one off bowel scope screening (flex sig – looks for/removes polyps)
- 60-75y – home testing kit every 2 years (FIT, FOB) – looks for blood
- >75 can request it

Cervical screening/HPV

- 24.5-49 every 3 years
- 50-64 every 5 years
- > 65 if recently had abnormal cytology, or if hadn't had one in 5 years and request one
- Pregnancy:
    - Reschedule for >12w post-partum
    - If previously had abnormal test and then woman becomes pregnant – do colposcopy asap



- Schedule
    - Negative hrHPV return to normal recall
    - Positive hrHPV cytology
        - Cytology abnormal colposcopy
        - Cytology normal (i.e. hrHPV +ve, cytology normal) repeat in 12 months
            - Repeat test (12m): hrHPV -ve return to normal recall
            - Repeat test (12m): hrHPV +ve, cytology normal repeat test in 12 months.
                - Repeat test (24m): hrHPV -ve return to normal recall
                - Repeat test (24m): hrHPV +ve colposcopy
    - Sample is 'inadequate' (see bottom of document):
        - Repeat sample within 3 months
        - If 2 consecutive inadequate samples colposcopy
    - In HIV+ve patients - annual cytology following initial colposcopy
    - if post-partum delay to 3 months post delivery
- Cytology – visualise cervix with speculum, sample from transformation zone. Detects dyskaryosis (borderline changes > low grade dyskaryosis > moderate > severe > invasive SCC > glandular neoplasia
    - Dyskaryosis/koilocytosis = hyperchromic and HUGE nuclei, relative to cytoplasm.
    - Reporting results: inadequate, negative, abnormal
        - Inadequate: Was taken but the cervix was not fully visualized / taken in an inappropriate manner (for example, using an unapproved device) / Contains insufficient cells / obscuring element (for example lubricant, inflammation, or blood) / incorrectly labelled.
- Colposcopy – SEPARATE APPT
    - Acetic acid – abnormal turns white
    - Iodine – abnormal doesn't stain (normal = dark brown)
    - Biopsy abnormal areas = endocervical curettage. Swede score to interpret results.
- Colposcopy tells you about: Cervical intraepithelial neoplasia (CIN) precursor to cervical cancer
    - CIN 1 – 1/3rd thickness of cervix surface layer affected
    - CIN 2 - 2/3rd thickness affected
    - CIN 3 – full thickness affected (aka high grade, severe dysplasia, cervical carcinoma in situ)
- HPV 16 drives E6 (oncoprotein) degrades p53, HPV 18 drives E7 (oncoprotein) inhibits RB suppressor genes

AAA

- One ultrasound age 65
- <3cm = normal
- 3-4.4 = small, rescan every 12 months
- 4.5-5.4 = medium, rescan every 3 months
- >5.5 = large, refer urgently to vascular surgery
- Rapidly enlarging >1cm per year ⟶ refer
- Symptomatic ⟶ refer

Newborn screening

- NIPE (72 hours of birth)
- Heel prick
    - Sickle cell
    - Cystic fibrosis
    - Congenital hypothyroidism
    - Metabolic:
        - phenylketonuria (PKU)
        - medium-chain acyl-CoA dehydrogenase deficiency (MCADD)
        - maple syrup urine disease (MSUD)
        - isovaleric acidaemia (IVA)
        - glutaric aciduria type 1 (GA1)
        - homocystinuria (pyridoxine unresponsive) (HCU)

Obstetrics & Gynae

Menorrhagia

- ≥80 mL and/or a duration of more than 7 day
- excessive (heavy) menstrual blood loss that occurs regularly (every 24 to 35 days) which interferes with a woman's physical, emotional, social, and material quality of life.
- Ddx:
    - 50% no cause, fibroids, endometriosis, adenomyosis, PID, polyps, endometrial ca, PCOS,
    - Systemic: coag disorder, hypothyroid, DM, liver/renal disease
    - Iatrogenic: anticoag rx, chemo, IUD
- Exam: abdo, bimanual pelvic exam
- Ix: FBC, TFT, caog, screen, hysteroscopy, TV USS, vaginal/cervical swab
- Rx: same as fibroids – **progest**, **NSAIDs**, **Mefenamic acid**. If more severe – ablation, removal

Polycystic ovarian syndrome

- Emerges at puberty. Endocrine disorder.
- Rotterdam criteria
    - Ovulation disorders
    - clinical hyperandrogenism (hirsutism, oligomenorrhoea)
    - PCOS morphology (12 or more follicles in at least one ovary, or increased ovarian volume)
- Pathophysiology:
    - Insulin resistance, hyperinsulinaemia – low SHBG, high free testosterone, anovulation ; weight gain increases aromatase, increases ins resistance
    - Raised LH – increases oestrogen
    - Raised serum oestrogen – follicles don't mature properly, anovulation, hyperplastic endometrium
- Sx: abnormal mensturation, infertility, hyperandrogenism (hirsitusm, acne), obesity/weight gain
- Ix: high testosterone, low SHBG, raised LH, **raised LH:FSH ratio**, pelvic TV/USS >12 follicles in at least one ovary or increased ovarian volume)
- Rx: lifestyle, **COCP/IUS/cyclical progest**
- **Elfornithine** - for hirsutism (facial)
- Complications: endometrial ca, infertility, T2DM, CVD, preg complications, depression/anxiety

Fibroids/leiomyomas

- Benign tumours that grow in the uterus (made up of smooth muscle cells and fibroblasts)
- Form hard, round, **whorled** tumours in the myometrium
- Types
    - subserosal: outer serosal surface, extends peritoneal cavity, press on adjacent structures urinary sx
    - intramural: myometrium, interfere with constriction of blood vessels during period menorrhagiae, dysmenorrhoea
    - submucosal: inner mucosal surface of uterus, into uterine cavity. Interfere with myometrium ability to contract during period (can't stop bleeding) SIGNIFICANT menorrhagia, dysmenorrhoea, reduced fertility
- Risk factors: >30-40y, black
- Sx: usually asymptomatic. Menorrhagia, pain (pelvic, abdo), urinary tract infections
- Exam: abdo and bimanual pelvic (firm enlarged and irregularly shaped NON-TENDER uterus. Mass can be moved slightly side-to-side)
- Ix: FBC (anaemia), transabdo and TV USS
- Rx
    - F/U for asymptomatic
    - <3cm: **Progesterone therapy** (any), **NSAIDs**, **tranexamic acid**
    - If more severe: hysterectomy, myomectomy, resection of fibroids
    - If have features of cancer (unexplained bleeding, weight loss) 2ww gynae
    - If ascites or pelvic/abdo mass (not thought to be fibroids present) urgent referral (ovarian ca?)
- Complications: abnormal uterine bleeding, compression adjacent organs (urinary sx), infertility, pregnancy problems

Cervical cancer

- Squamous cell carcinoma (adenocarcinoma <10%)
- Transformation zone of cervix where cancer happens
    - Ectocervix = **stratified squamous** non-keratinised (into vagina)
    - Endocervix/endocervical canal – **simple columnar** (into uterus)
    - **Squamocolumnar junction** moves INWARDS towards uterus during puberty. This is now transformation zone because now it's squamous, but used to be columnar pre-puberty.
- Risk factors – HPV, smoking, HIV, ++sex stuff
    - Acquiring: all sex related (n sex partners, early sex, no condoms)
    - Progression HPV cervical cancer: other STI, smojing, high parity, fmhx, COCP<5y
- Pathophys:
    - HPV 16 produces E6, HPV 18 produces E7 proteins
    - E6 and E7 proteins inactivate p53 and RB (tumour suppressor genes) and act as onocogenes (promote tumour growth) > dysplastic changes at squamocolumnar junction > koilocytes > dyskaryosis/CIN > cervical cancer
        - Koilocytes = enlarged nucleus / irreg nuc membrane contour / nuc stains darker / perinuclear halo
- Sx: usually asymptomatic, IMD, PCB, PMB, blood stained vaginal discharge, pelvic pain, dyspareunia
- Ix: spec exam, colposcopy (mosaicism in transition zone, abnormal vasculiarity, lesions, white changes), biopsy, HPV screening, CT/MRI/PET to assess extent of disease
- Referral
    - 2ww cervical sx
    - Fast-track colposcopy – if visible suspicion / hrHPV+ve with abnormal cytology
- **Cystoscopy** – measure dyskaryosis extent (inadequate > none > borderline > mild > moderate > severe > glandular neoplasia). Borderline or above send to colposcopy
- CIN stages (pre-cancer) – histology from colposcopy
    - CIN 1 – 1/3rd thickness of cervix surface layer affected
    - CIN 2 - 2/3rd thickness affected
    - CIN 3 – full thickness affected
- Staging: FIGO – starts from CIN3
    - Stage 1 – confined to cervix
    - Stage 2 – extends beyond uterus to vagina, pelvic wall (not beyond lower third)
    - Stage 3 – invades lower third vagina, kidneys, para-aortic lymph nodes
    - Stage 4 – extends beyond true pelvis, rectum, bladder
- HPV: dsDNA
- Rx:
    - CIN – colposcopy with excision/ablation
    - Micro-invasive: local excision, lumph node dissection, hysterectomy/chemo
    - Early stage met disease: trachelectomy (if fertility), hysterectomy+lymphadenectomy (don't care about fertility)
    - Advanced met: chemo, palliative
- Vaccination – Childhood Immunisation programme – **Gardasil**
    - 1st dose: 12-13y girls and boys (<sexually active)
    - 2nd dose: 6-24m after 1st dose
    - MSM <45y
    - If missed 1st dose by 15y can get it for free up to 25y free on NHS. Requires 3 doses.

Endometrial cancer

- Adenocarcinoma
- Oestrogen-dependent
- Stages:
    - 1 – confined to uterus
    - 2 – invades cervix
    - 3 – invades ovaries, fallopian tubes, vagina or lymph nodes
    - 4 – invades bladder, rectum, beyond pelvis

- Pathophys:
    - PCOS – high oestrogen, lack of ovulation (lack corp luteum which usually produces progest,), insulin resistance
    - Obesity – aromatase in adipose
    - Tamoxifen
    - T2DM
- Protective factors: COCP, mirena, anything that decreases oestrogen exposure (pregnancies, late menarche, early menopause), smoking
- Risk factors: diabetes, obesity = KEY ONES. (+ PCOS + anything that increases exposure to unopposed oestrogen)
- Sx: PMB, any abnormal bleeding (timing, too much), anaemia, raised platelets
- Ix: TV USS (thickness), pipelle biopsy, hysteroscopy with endometrial biopsy
- Rx: 2ww, progesterone (slows progression), total abdo hysterectomy with bilat salpingo-oophorectomy, radio/chemo

Ovarian cancer

- Ix: **CA125**, laparotomy diagnostic
- Meig syndrome - unilateral pleural effusion

Cervical ectropion

- Can be caused by exogenous oestrogen – e.g. combined contraceptive pill

Endometriosis

- Growth of endometrial tissue outside uterus
- 2 most common gynae issue after fibroids
- Risk factors: everything that increases oestrogen exposure, white, low BMI, autoimmune, smoking
- Sx: PAIN PAIN PAIN – dysmenorrhoea, DEEP pain during/after sex, period related GI/bowel movement pain, infertility
- Exam: abdo pelvis exam
- Ix: laparoscopic visualisation of the pelvis + biopsy
- Rx:
    - 1st line: NSAIDS/paracetamol
    - 2nd line: hormonal (COCP, progesterone)
    - Surgery – fertility: lap excision or ablation ; don't care about fertility, hysterectomy
- Complications: endometriomas, fertility issues, adhesions, bowel obstruction, chronic pain

Adenomyosis

- Growth endometrium within myometrium
- Sx: dysmenorrhoea, dyspareunia, menorrhagia
- Ix: TV USS/MRI
- Rx:
    - **tranexamic/mefenamic acid**
    - **IUS, COCP, GnRH analogue**
    - Hysterectomy

Pelvic inflammatory disease (PID)

- Infx of upper genital tract caused by infx lower down
- Caused by STI (almost always)
- Risk factors: sex behaviours, instrumentation of uterus (IUD, abortion), interruption of cervical barriers
- Fitz-Hugh-Curtis syndrome – fibrosis of the liver
- Sx: lower abdo/pelvic pain, dysuria, dyspareunia, vaginal bleeding, abnormal vaginal discharge
- Exam: bilat lower abdo tenderness, bimanul vaginal exam (cervical motion tenderness, uterine tenderness), spec eam (discharge, cervicitis), fever,
- Ix: preg test, high vaginal swab
- Rx: analgesia, abx triple therapy (metronidazole, IM ceftriaxone, doxycycline), consider removing IUD/IUS
- Complications: sepsis, abscess, infertility, ectopic, Fitz-Hugh Syndrome (infx tracks to liver causing adhesion, perihepatitis).

- Never insert/offer IUD to someone with PID

Breast cancer

- Ductal cell carcinoma (most common), lobular carcinoma - e-cadhedin secreting - stickier therefore compared to lobular less likely to metastasise
- Lobular -? More aggressive
- DCIS (ductal carcinoma in situ) – premalignant, graded 1-3 based on amount of proliferation + how normal the cells look on microscopy. E.g. grade 3 abnormal cells + fast growing
- BRCA1, BRCA2 – family history diagnosis before age 40 is red flag
- HER2, oestrogen, progesterone
- Ix: FBC raised white cells + platelets, hypercalcaemia, ALP, CA 15-3 elevated, MRI, PET
- Triple assessment
    - Clinical
    - Radiological
    - Histological
- Anatomical features in surgery:
    - Long thoracic nerve – C5,6,7 – wings to heaven (serratus anterior)
    - Thoracodorsal nerve – C6,7,8 – help you bench a plate (latissimus dorsi)
    - Intercostobrachial nerve – T2, T3
    - AXILLARY VEIN - very scary and typically covered in nodes
    - Thoracodorsal bundle
    - Lateral thoracic artery
- Levels of axillary clearance
    - 1 – lateral to pec minor
    - 2 – deep to pec minor
    - 3 – medial to pec minor
- Rx:
    - Radiotherapy and adjuvant chemo
    - Anastrozole/Letrozole – post-menopausal – to target peripheral conversion of testosterone to oestrogen.
    - Tamoxifen – pre-menopausal – endometrial cancer + VTE risk
    - Dexamethasone for metastatic disease
    - Trastuzumab (Herceptin) – HER2 +ve
- Metastases to LLBB – lungs, liver, brain, bones (commonly t-spine, pelvis, long bones)
- Nottingham prognostic index – size, nodes, grading – tumour size x .2 + lymph node score + grade score
    - < 3.4 = good prognosis
    - 3.4-5.4 = moderate prognosis
    - >5.4 = poor prognosis

Breast lumps/discharge

- 2ww criteria –
- Fibroadenoma – over 5cm surgery
- Duct ectasia = green, brown discharge (ducts become blocked and secretions stagnate). Lump can occur. Common around menopause.
- Fat necrosis = common in obese. Trauma > calcification > scarring. Refer for triple assessment. No rx
- Paget's disease = eczematoid change, weepy crusty lesion, reddening and thickening of areola. Ix: triple assessment, underlying breast malignancy
- Nottingham prognostic index – PROGNOSIS OF BREAST CANCER
- Duct papilloma – watery/bloody discharge, palpable mass

Fibrocystic changes of breast

- Diffuse, symmetrical, painful, cyclical with menses
- Most common in middle aged women
- Ix: mammogram

Mastitis

- Typically associated with breastfeeding
- Sx: hot, tender, erythematous breast. Fever/malaise may be present.
- Rx: 1st line: continue breastfeeding, simple analgesia, warm compresses. IF systemically unwell and symptoms do not improve after 12-24hrs of effective milk removal abx, oral **flucloxacillin**
- Breastfeeding should continue during treatment

Breast triple assessment:

- Clinical – assessment – history and exam
- Radiological – Imaging – mammogram or USS
    - >40y have both
    - <40y have USS
- Histopathological – Biopsy – FNA (cytology) or core biopsy (histology)

Pre-eclampsia

- Shallow invasion of placenta into uterus / not as much dilation of maternal blood flow less oxygenation to foetus
- Proteinuria, HTN, oedema, end-organ dysfunction HELPP syndrome lft derangement
- Rx: delivery… but also can use **labetalol** to manage HTN or **Nifedipine** if beta-blockers are contraindicated
- Prophylactic **aspirin**: for women at moderate to high risk from 12 weeks until birth (75-150mg)
    - risk fx: first pregnancy, age > 40, BMI > 35, fhx, multiple pregnancy, CKD, HTN/previous pre-eclampsia, diabetes

Eclampsia

- **Magnesium sulphate** to treat seizures until 24hrs from last seizure
- Test reflexes, urine output, resp rate, O2
- resp rate to monitor when administering MG sulphate

Pregnancy

- Cardiovascular system:
    - Stroke volume increases + heart rate increases, therefore CO increased
    - Systolic BP should be unaffected
    - Diastolic BP reduced in early pregnancy
    - Peripheral oedema in lower limb due to reduced venous return from enlarged uterus
- Resp system:
    - Pulmonary ventilation increased, tidal volume increased due to effect of progesterone
    - Oxygen requirement increased, therefore fall in pCO2
- Blood
    - Maternal volume increased, red cells less than plasma, therefore may see anaemia
    - Hypercoagulable state
        - Increased FVII, VII, VIII, X, XII, fibrinogen
        - Decreased protein C & S
            - Normally degenerate VIIIa and Va because of negative feedback loop normally slow down clotting
        - Decreased fibrinolytic state
        - D-dimers increased
- Liver - ALP raised, albumin decreased
- Kidney - increased blood flow to kidneys, increased excretion, trace glycosuria common due to high GFR
- osteitis condensans ilii

Ectopic pregnancy

- +ve urine and blood HCG
- If ruptured, pain +++, surgical emergency
- TVU will show empty uterus – possibly adnexal mass
- Risk factors: adhesions, PCOS, endometriosis, PID
- Management options
    - Expectant management – if <35mm, asymptomatic, no heartbeat, hCG <1000

- Medical management – if <35mm, no significant pain, hCG <1500 - **methotrexate**
- Surgical management – if >35mm, ruptured, pain, fetal heartbeat, or HCG >5000
  - Salpingectomy = first line
  - Salpingotomy – can be considered for women with other risk factors for infertility in order to preserve the tube. 1/5 not successful and require further treatment.

Antenatal appts

- TIMELINE
  - BOOKING - find out about pregnancy + book with midwife, typically 8 weeks ish
    - BMI
    - BP
    - Urine dip
    - FBC, blood grouping
    - Rhesus status
    - Screening for HIV, hep B, syphilis
  - 10-14 weeks - urine dip + BP
    - Dating scan + confirm number of foetus' + viable/intrauterine
    - Combined test - Nuchal translucency, PAPP-A, BHCG - Trisomy 21, 18, 13
    - Quadruple test - only done if unable to measure Nuchal translucency, or if late booking <14 weeks, only tests for Trisomy 21 - not as accurate as combined
  - 18-20 weeks - urine dip + BP
    - ANOMALY SCAN - anatomy, sex, placental location, gross abnormality, crown-rump length, amnios (poly/oligo)
    - Amniocentesis IF YOU NEED/WANT
    - Chorionic villus sampling - blood sample from placenta - can do this earlier (from 11 weeks) as diagnostic for abnormalities from 10-14 week scan
  - 24-28 weeks - urine dip + BP
    - OGGT
  - 28 weeks - Rhesus -ve mum gets anti-D
  - 36 weeks - abdominal palpation/confirm position
  - 37 - induction for: intrahepatic cholestasis, gestational diabetes, pre-eclampsia, twins
  - 38 - normal term - if GBS status unknown, swab and send for NAAT → abx during labour
  - 41+ - wtf get out ??? induce before placenta calcifies
- Vaccines: whooping cough + flu - at booking
- Rhesus compatibility - booking bloods
  - Anti-D if mother negative (28 weeks and 34 weeks)
  - In termination – if more than 10 weeks give anti-D to rhesus -ve mother
- Gestational diabetes (24-28 weeks)
  - If fasting glucose is ever >7mmol/L **insulin**
  - If <7mmol/l, trial of diet and exercise and repeat in 2 weeks, if still >5.6mmol start **metformin**
  - OGTT - blood glucose before and 2 hours after giving 75g of oral glucose

Termination of pregnancy

- Up to 24 weeks (except special circumstances – foetal anomaly, to save the life of the mother, if there is risk of serious injury to mother)
- Choice between surgical and medical
- Medical – typically up to 10 weeks
  - If no foetal heartbeat + HCG <1000 = Mifepristone followed by misoprostol.
  - Pregnancy test required two weeks after to confirm pregnancy has ended

- Surgical – cervical priming with misoprostol often used
    - MVA – vacuum aspiration
    - D&E (D&C) – dilatation and evacuation

Labour

- Latent: painful contractions, some cervical changes (shortening, effacement, dilatation up to 4cm)
- **CTG to monitor**
    - **Contractions** – the number of uterine contractions per 10 minutes
    - **Baseline rate** – the baseline fetal heart rate – **110-160**
        - **Foetal bradycardia –** rule of 3.
            - 3 mins help
            - 6 mins theatre
            - 9 mins prepare to deliver
            - **12 mins deliver before 15 minutes.**
    - **Variability** – how the fetal heart rate varies up and down around the baseline
    - **Accelerations** – periods where the fetal heart rate spikes
    - **Decelerations** – periods where the fetal heart rate drops – more concerning, due to hypoxia.
- **DR C BRaVADO – structure for assessing CTG**
    - **DR** – Define Risk (define the risk based on the individual woman and pregnancy before assessing the CTG)
    - **C** – Contractions
    - **BRa** – Baseline Rate
    - **V** – Variability
    - **A** – Accelerations
    - **D** – Decelerations
    - **O** – Overall impression (given an overall impression of the CTG and clinical picture)
- **Stage 1** (established, active transition): dilated 10cm
    - Latent stage = established until 4cm dilated
    - Active stage = 4cm dilated until 10cm
    - **Delay in 1st stage** = <2cm of cervical dilatation in 4 hours or slowing of progress in a multiparous women
        - **Oxytocin** infusion – titrated up to reach 4-5 contractions per 10 minutes
- **Stage 2**: contractions, fully dilated – 3Ps
    - Power – length and frequency of contractions (2 in 10 – 2 contractions in 10 minutes)
    - Passage – shape and size of the pelvis and soft tissues
    - Passenger – presentation (breech, shoulder, cord, compound), position, size, multiple pregnancy, foetal abnormalities
    - **Delay in 2nd stage** = >2 hours in a nulliparous woman, >1 hour in a multiparous woman
        - Changing positions
        - Encouragement
        - Analgesia
        - **Oxytocin**
        - Episiotomy
        - Instrumental delivery
        - C-section
- **Stage 3:** birth of baby to expulsion of placenta/membranes
    - **Delay in 3rd stage** = >30 mins with active management (IM oxytocin and controlled cord traction) or >60 minutes with physiological management
- **Pethidine** – opioid, can cause foetal withdrawal sx

Post-partum Haemorrhage

- Causes = 4Ts - Tone (uterine atony - most common cause), Trauma (e.g. perineal tear), Tissue (retained placenta), Thrombin (bleeding disorder)
- Rx:
    - Primary PPH - ABCDE resus + MHP
        - Mechanical - uterine stimulation, catheterisation
        - Medical - oxytocin, ergometrine, carboprost (atony). TXA in thrombocytopenia

        - Surgical - balloon tamponade, B lynch suture, uterine artery ligation, hysterectomy
    - Secondary PPH - 24 hrs to 12 weeks postpartum
        - Usually caused by retained products of conception or infection
        - Rx depends on cause, e.g. abx for infection, surgery for retained products

Induction of labour

- **Bishop score** – score **>8** predicts successful induction.
    1. Fetal station (0-3)
    2. Cervical position (0-2)
    3. Cervical dilatation (0-3)
    4. Cervical effacement (0-3)
    5. Cervical consistency (0-2)
- Membrane sweep – inserting finger into the cervix to stimulate labour
- Vaginal prostaglandins
    - Prostin – tablet given 6hrly
    - Propess pessary – 1 pessary given for 24-32 hours. Some increase in infection.
- Artificial ROM and oxytocin infusion
- Oral mifepristone + misoprostol where intrauterine foetal death has occurred.

Contraception

- Injectable
    - (progesterone) **medroxyprogesterone acetate**
    - MoA: 1) prevents ovulation 2) thickens cerv mucus
    - IM every 12w
    - C/I = BREAST CANCER
    - Comps: sot quickly reversible, weight gain, irregular bleeding
- Implantable
    - Progestogen (Nexplanon). Most effective
    - MoA: 1) prevents ovulation 2) thickens cerv mucus
    - Lasts 3 years
    - C/I: IHD/stroke, unexplained vag bleeding, breast cancer (past/present), liver cirrhosis, liver cancer
    - Com: irregular/heavy bleeding
- IUS
    - 1st line for heavy menstrual bleeding
    - Lasts 5 years
    - MoA: 1) prevents endometrial proliferation 2) thickens mucus
    - C/I: oestrogen-sensitive breast ca, STI
    -
- IUD
    - Prevents fertilisation
    - Safe to use in breast cancer
    - Lasts 5-10y
    - C/I: Wilson, acute pelvic infx (STI)
- Emergency
    - Levonorgestrel:
        - within 72h of UPSI
        - stops ovulation and inhibits implantation
        - can be used multiple times in one cycle
    - Ullipristal (form of progesterone)
        - Within 5 days of UPSI
        - Stops ovulation
        - Once per cycle
    - IUD
        - Within 5 days of UPSI or up to 5 days after likely ovulation date
        - Inhibits fertilisation (toxic to sperm) or implantation
- Postpartum
    - COCP NEVER in <6w postpartum or while breastfeeding

- IUD, IUS - <48h after birth or >4w
- Lactational amenorrhoea method

Fibrocystic changes of breast

- Diffuse, symmetrical, painful, cyclical with menses
- Ix: mammogram

U&Es

Hypercalcaemia (>2.6 mmol/L (2.2-2.6 = normal))

- Main causes: primary hyperparathyoroidism, malignancy (bone mets, myeloma, squamous cell lung cancer, breast cancer, kidney)
  - Bony mets upregulate osteoclast activity increased serum calcium
  - Multiple myeloma **CRAB**
- Less common: multiple endocrine neoplasia (autosomal dominant), lung granulomatous diseases (sarcoidosis, TB), hyperthyroid, phaeochromocytoma, Addison's, familial hypocalciuric hypercalcaemia (autosomal dominant, resistant to calcium)
- **Drug causes: Thiazide diuretics, Lithium, Vit A/D high dose (toxicity)**
- Radiotherapy to head and neck parathyroid adenoma
- Sx: "bones, stones, groans and psychic moans"
  - bone pain, renal stones, GI disturbance (constipation, pain, nausea) and psychiatric symptoms (reduced GCS)
  - polyuria and polydipsia
  - muscle weakness
  - band keratopathy – calcification of the sclera
  - Ix: ECG – shortened QT interval, look at PTH and malignancy markers, CXR (?ca, mets, sarcoid, TB)
  - 24h urinary calcium for FHH
  - Prim hyperparathyroidism: **high ca AND high PTH**
  - Malignancy: **high ca and LOW PTH**
  - Calcium on VBG will be approx. 50% of serum calcium
- Rx: rehydration therapy (**IV 0.9% saline**) > **IV bisphosp** (or calcitonin), treat underlying cause
  - Lymphoma/sarcoidosis **glucocorticoids**
  - Malignancy **denusomab**
  - Primary/tertiary hyperparathyroidism surgery preferably, if unfit can use **cinacalcet**, CaSR agonist acts on PT gland, downregulates production of PTH

Hypocalcaemia

- Causes: malabsorption, CKD (lack alpha-hydroxylase to convert pre-vit D (usually dietary D2/3) cholecalciferol), hypoparathyroid, PTH resistance, vit D def (osteomalacia, rickets), acute pancreatitis
- CATS go numb – Convulsions, Arrythmia, Tetany/Trousseau, go numb = parasthesia
  - Paraesthesia, tetany (spasm, cramps, seizures twitching), Chvostek (tap parotid>face twitch), Trousseau (take brachial BP>carpal spasm)
- Ix: ECG (prolonged QT interval)
- Rx: **IV calcium gluconate**

Rickets

- Vitamin D deficiency reduced uptake of calcium and phosphate in the gut secondary hyperparathyroidism to correct for low calcium increased osteoclast activity weak soft bones
- Vitamin D deficiency more common in:
  - Darker skin (less vitamin D made from UV)
  - Poor diet/malabsorption disorders
  - Poor sunlight exposure

- Sx: lethargy, bone pain, swollen wrists, poor growth, dental problems, pathological fractures
- Ex: bowed legs, knock knees, rachitic rosary – lumpy chest from costochondral expansion, delayed suture closure in babies
- Ix: low serum vitamin D, low serum calcium, low serum phosphate, increased PTH, increased ALP
    - Consider anti-ttg for coeliac (malabsorption)
- Rx: preventative (diet, supplementation), treat with ergocalciferol (vitamin D) and calcium supp.

Hyponatraemia

- Cause: excess water or sodium depletion
- Sx: nausea, vomiting, headache, confusion, fatigue, seizures/reduced GCS
- Must be compared with urine sodium
    - Urine sodium >20 / normal ECF volume (renal loss of sodium)
        - Thiazides / loop diuretics
        - Addison's
        - Diuretic stage of renal failure
        - SIADH
        - Hypothyroidism
    - Urine sodium <20 / decreased ECF volume (extra-renal loss of sodium or excess fluid, dilute urine/third space shift)
        - Diarrhoea, vomiting, sweating
        - Burns, rectal adenoma
        - Secondary hyperaldosteronism (heart failure/ liver cirrhosis)
        - Nephrotic syndrome
        - IV dextrose
        - Psychogenic polydipsia
- Rx: correct slowly, no more than 10mmol per hour (risk of osmotic demyelination syndrome)
- Consider pseudohyponatremia from sample dilution in high lipid or high protein samples

Hyperkalaemia (> 5.5mmol/L)

- Causes:
    - AKI
    - Drugs - potassium sparing diuretics, ACEi, ARBs, heparin
    - Metabolic acidosis
    - Addison's disease
    - Rhabdomyolysis
- Sx: muscle weakness, fatigue, paraesthesia, cramps, bradycardia
- Ix:
    - ECG - tall tented T wave, small p wave, widened QRS complex
    - LDH - if suspecting haemolysis as cause
- Rx:
    - Stabilise cardiac membrane - **IV calcium chloride/gluconate**
    - Insulin dextrose infusion - drives K+ intracellular, **10 units ACTRAPID** + **50mls 50% dextrose**
    - **Salbutamol** nebuliser
    - IV fluids - improve urine output and BP
    - Potassium binding resin - **calcium resonium / sodium zirconium cyclosilicate**
    - Treat underlying cause
    - Consider loop diuretics, Dialysis

Hypokalaemia (<3.5mmol/L)

- Causes
    - Acidotic - diarrhoea, renal tubular acidosis
    - Alkalotic - vomiting, thiazide + loop diuretics, Cushing's syndrome, Conn's syndrome
    - Magnesium deficiency
    - Beta blockers in CKD patients
- Ix:
    - ECG - U waves

Oncology

Immunotherapy

- Checkpoint inhibitors – CTLA-4 inhibitors (Ipilimumab), PD-1 pathway inhibitors. Prevent de-activation of T cells, allows T killer cells to target cancer cells.
- Contraindications: Autoimmune diseases, already on high dose steroids
- Side effects – widespread. Every organ can be affected. Commonly – rash, pruritus, hepatitis, breathlessness, fatigue. Reduced risk of neutropenic sepsis compared to chemo. Side effects are managed with high dose steroids. Positive correlation between side effects and cancer response.
- As some side effects have high mortality (e.g. myocarditis) very low threshold for admission with patients on immunotherapy

Chemotherapy

- Side effects : Myelosuppression pancytopenia (risk of neutropenic sepsis), GI side effects, Neurotoxicity, Hair loss
- Side effects are unpredictable in onset

Neutropenic sepsis

- Very low threshold for admission/suspicion and therefore treatment – NICE guidance uses the word “unwell”.
- Initial ‘local’ t-killer and t-helper response is impaired.
- Systemic response is slowed
- Management – empirical abx **within an hour**: **Tazocin, meropenem**, local antimicrobial guidelines. May use Granulocyte colony stimulating factor (GCSF) which doesn’t change outcome but reduces inpatient stay

Lymphoma

- Cancer affecting the lymphocytes, proliferates within lymph nodes ⟶ lymphadenopathy
- Two categories:
    - Hodgkin’s lymphoma - more common, bimodal age distribution 20-25/80, good prognosis
        - risk fx: HIV, EBV, autoimmune conditions, family hx
    - Non-hodgkin’s lymphoma
        - Burkitt lymphoma - associated with EBV and HIV
        - MALT lymphoma - mucosa associated lymphoid tissue typically stomach
            - Hashimoto’s thyroiditis, H-pylori infection
        - Diffuse B cell lymphoma - rapidly growing painless mass in older pts
        - Risk fx: pesticides, trichloroethylene, + as above
- Sx: lymphadenopathy - non tender and firm/rubbery, pruritus, pain after drinking alcohol (HL), B cell symptoms (night sweats, weight loss), joint effusion
- Ix: lymph node biopsy - **reed-sternberg cells** = hodgkin’s lymphoma, large cancerous B lymphocytes with 2 nuclei and prominent nucleoli
- Rx: chemo + radiotherapy

Leukaemia

- Cancer of a particular line of stem cells originating from bone marrow - genetic mutation in a precursor cell
- Excessive production of a single type of abnormal white blood cell ⟶ suppression of other cell types ⟶ pancytopenia
- Sx: fatigue, fever, pallor, petechiae, abnormal bleeding, hepatosplenomegaly, lymphadenopathy
- ddx for petechiae:

    - Meningococcal septicaemia
    - Vasculitis
    - ITP
    - HSP
    - TTP
    - Trauma/mechanical e.g. severe vomiting
    - Non-accidental injury - choking
- Ix: FBC, blood film, LDH (non-specific marker of tissue damage), bone marrow biopsy, CT + PETCT, lymph node biopsy, genetic test + immunophenotyping
- Types:
    - ALL - most common leukaemia in children - associated w/ Down Syndrome
        - affects lymphocyte precursor → increased B cells + pancytopenia
    - CLL - warm haemolytic anaemia, **smudge cells** (ruptured WBC)
        - often asymptomatic + insidious, red flag sx
    - AML - **blast cells, auer rods**
        - can be result of transformation from myeloproliferative disorder such as polycythaemia rubra vera
    - CML - 3 phases including long chronic phase, accelerated phase, + blast phase
        - **MOST COMMON CAUSE OF MASSIVE SPLENOMEGALY IN THE UK**
        - chronic phase - often diagnosed from incidental finding of raised WCC
        - accelerated phase = first symptoms, anaemia, thrombocytopenia
        - blast phase = severe symptoms, pancytopenia, often fatal
        - Associated with **philadelphia chromosom**e (abnormal chromosome 22, codes for abnormal tyrosine kinase enzyme → drives proliferation of abnormal cells)
        - **Imatinib** = tyrosine kinase inhibitor
- Rx: MDT, chemo + target therapies e.g. monoclonals, bone marrow transplant

Complications of chemo

- Chemo doesn't work - cancer continues
- Stunts growth in children
- Immunosuppression → infection
- Neurotoxicity
- Infertility
- Secondary malignancy (radiation)
- Cardiotoxicity
- Tumour lysis syndrome → high uric acid, hyperkalaemia, hyperphosphatemia, hypocalcaemia

Prostate Ca

- can be genetic - BRCA, especially 2 with aggressive phenotype

Geriatrics

Falls (DAME ?maggie smith?)

- **D - drugs**
- **A - age related changes**
- **M - medical**
- **E - environmental**

Medical causes of falls

- V - syncope/vasovagal
- I - sepsis, UTIs, LRTIs
- T - stroke, ACS
- A - myasthenia gravis, myositis
- M - T1/2DM - hypos more common in elderly people, electrolytes + dehydration
- I - drugs drugs drugs – especially some alzheimer's meds e.g. memantine
- N - space occupying lesion, multiple myeloma

Frailty – Rockwood scale to quantify

- Frailty syndromes
    - Sarcopenia
    - Poor nutrition/weight loss – MALT
    - Falls, UTIs, LRTIs
    - Parkinson's disease
    - Cognitive impairment
    - Continence issues + loss of dignity
    - Polypharmacy (>4 meds)
    - Social isolation and loneliness
- Comprehensive geriatric assessment (CGA)
    - Pain
    - Skin integrity
    - Sensory loss
    - Cognitive impairment – worth doing 4-point AMT with every patient >65 (age, DOB, place, year)
    - SALT – safe swallow
    - Falls and mobility
    - ADLs
    - Safeguarding
    - End of life
- Age related changes – cardiovascular/resp/endocrine/MSK – basically it all gets shit

Delirium

- Sudden decline in cognitive function, acute confusional state, can fluctuate
- Can take up to 6 months to resolve completely

- Hypoactive – lethargic, reduced function, more common in women, more common in patients with BG dementia
- Hyperactive – agitation, increased vigilance, hallucinations
- Mixed delirium
- Causes: PINCHES ME
    - Pain
    - Infection
    - Nutrition
    - Constipation
    - Hydration/hypoxia
    - Environment
    - Stroke
    - Medication - !anticholinergics! - e.g. solifenacin
    - Electrolytes
- Pathophysiology - theories
    - Acute mental stress - leads to deterioration in more complex cognition. This is typically a healthy response, but if chronic and in older patients with brains already experiencing atrophy/degeneration can cause ongoing delirium
    - Recent evidence - Blood brain barrier - more permeable as you age —> more confusion
    - Sustained level stress hormone cortisol can cause psychosis - ?cause of delirium
- Ix: 4AT
    - Confusion screen (FBC, CRP, U&E, LFT, Mg2+, Bone profile, TFTs, B12 + folate)
    - CT head - low threshold for CT in new confusion
    - Review all medications
    - Bladder scan
    - PR
    - AXR/CXR for constipation/infection source
    - 3 months post delirium diagnosis will require dementia r/v
- Managed by treating the cause, meds may be required for aggressive/risk of harm to self behaviour (typically **haloperidol** - short term, low dose. Contra-indicated in parkinsons) with the aim to calm the patient
- Memory team follow-up in community to assess function when cause has been treated

Agitated patients - management

1. Do they have capacity?
    a) If no - DOLS can be completed by anyone
    b) Quick collateral from team/family
2. Are they a risk to themself/others
3. De-escalate - what are you doing / why are you doing this ?
    a) Small things - cup of tea, tv show that calms them down
4. If this does not work:
    a) Haloperidol - 500 micrograms IM
    b) Lorazepam - not preferable but may be necessary in emergency in patients where haloperidol is CI (e.g. Parkinson's)

Falls

- Screen for falls risk – how many falls in last 12 months? Have you fractured any large bones (think pelvis, femur, pathological fractures)? Was the fall provoked?
- Cause of fall?
    - Mechanical – did they stack it over a dodgy carpet or roll ankle in high heels? – OTs
    - Syncope – arrhythmias
    - BP
    - Epilepsy
    - BPPV
    - Peripheral neuropathy
    - Eyesight
    - Medication

- Take a good fall history – loss of consciousness/hit head, headache/vomiting/vision changes
- Holter monitor – portable ECG, good for investigating episodic arrythmias

Anticipatory medications

- Respiratory distress + Pain - **Morphine**
- Secretions - **Hyoscine Butylbromide**
- Agitation - **Midazolam**
- Anti-nausea - **Levomepromazine / Haloperidol**

OTHER

MRI – SOFT TISSUE – in context of MSK, good for seeing oedema within bones, rubbish at bones themselves

- T1 – fat is bright
- T2 – fat and fluid is bright

XR interpretation

- Pt details
- PA / AP
- Inspiration / expiration – 6/7 anterior ribs = inspiration
- Rotation – clavicles equidistant from the spinous process
- Penetration – can you see spinous processes through the heart
- A – tracheal deviation (tension pneumothorax (PUSH), goitre/thyroid mass)
- B – lung markings throughout, opacity, segmental pneumonias/oedema
- C – cardiomegaly
- D – diaphragm – hemiparesis, blunting of costodiaphragmatic recess
- E – clavicle/rib fractures, hiatus hernia (horizontal line behind heart shadow)

Assessment tools

- STOPP - Screening Tool of Older Person's Prescriptions - aims to identify medications where the risk outweighs the therapeutic benefit
- AUDIT/AUDIT-C – Alcohol Use Disorders Identification Test. It is comprised of 10 questions and is used to identify patients at risk of alcohol harm.
- CAGE – alcohol dependence – Cutting down, Annoyed when asked about alcohol, Guilt, Eye-opener

Antidotes

- Flumazenil – benzodiazepines
- Naloxone – opioids
- Fomepizole – methanol & ethylene glycol poisoning
- Lipid emulsion – lidocaine toxicity
- Atropine glucagon – beta blockers

Drug overdose presentations

- **Cocaine** - Rx = **benzodiazepines + GTN**
    - Coronary artery spasm ⟶ N/STEMI, can be tachy or brady
    - Hypertension
    - QRS widened, QT prolonged
    - Aortic dissection
    - Seizures, hypertonia, hyperreflexia
    - Agitation, psychosis, hallucinations
    - Hyperthermia, metabolic acidosis
- **TCAs** - Rx = **IV bicarbonate**
    - dry mouth, dilated pupils, agitation, sinus tachycardia
    - arrhythmia, seizures, metabolic acidosis, coma
    - widened QRS ⟶ IV sodium bicarbonate
- **Organophosphate insecticide poisonin**g - Rx = **Atropine**
    - Sx: SLUD (salivation, lacrimation, urination, defecation/diarrhoea
    - Also: bradycardia, miosis, fasciculations, hypotension
- **Malignant hyperthermia** - **suxamethonium** most commonly (sevoflurane) - Rx: **Dantrolene**
- **LSD -** Rx: **Lorazepam**
    - Sx: colourful hallucinations, hyperreflexia, paranoia, dry mouth, headache, nausea, tachycardia + hypertension
- **Ethylene Glycol** (anti-freeze)- **Fomepizole**
- **Warfarin - Vit K**
- **DOAC - Octaplex**

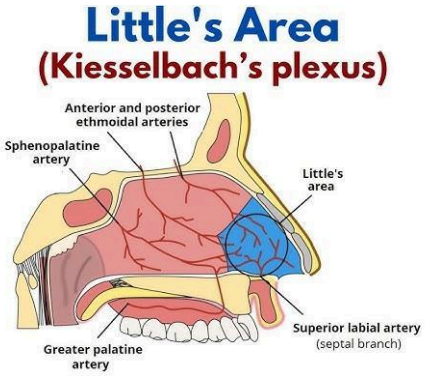
Haemoptysis

- Bronchitis (acute, chronic), TB, lung cancer, bronchiectasis, pneumonia, PE, Goodpasture's
- Haemoptysis = Bright red, frothy sputum, alkaline, Sa02 similar to peripheral arterial sats
- Blood from GI = usually darker, may have food particles, acidic, Sa02 similar to venous blood
- Blood from posterior nasal passage or nasopharynx can mimic haemoptysis without obvious epistaxis

Gingival hyperplasia

- CCBs, phenytoin, acute myeloid leukaemia

Epistaxis



- Little's area – arterio-arterial anastomosis found on the anterior nasal septum. Formed by anterior ethmoidal, posterior ethmoidal, sphenopalatine, greater palatine, septal branch of superior labial
- Causes: Trauma, inflammation, topical drugs (cocaine, nasal decongestion), vascular (HTN, granulomatosis disorders), medication (DOAC, warfarin), coagulation disorders (thrombocytopenia/VWB)
- Rx: mostly self-limiting and do not require medical treatment
    - Basic first aid – pinch soft part of nose and lean forward for 10 minutes.
    - If visible bleeding point – cautery using silver nitrate
    - If not visible – nasal packing using nasal tampons or inflatable packs
    - ENT
    - After treating acute nosebleed – naseptin nasal cream (chlorhexidine and neomycin) 4x daily for 10 days to reduce crusting/inflammation/infection. CONTRAINDICATED IN NUT ALLERGY.

Burns

- ABCDE approach with some extra things to look out for
    - A – **inhalation injury** – burns to face/oropharynx, singed nasal/facial hair, hoarse voice, stridor, swollen mouth. EARLY INTUBATION BY SENIOR ANESTHETIST
    - B – **Eschar** (tight leather dead tissue forming a circumference around a limb or the thorax) constricts and causes limb ischaemia/resp distress escharotomy
    - C – fluid resus – parkland formula – 2-4ml x bodyweight (kg) x Total Body Surface Area Affected
        - Burn percentages only include 2nd degree or greater
        - Degree = depth
            - 1st degree – superficial, looks like a sunburn, only epidermis damaged
            - 2nd degree – wet, blistered, red – epidermis and upper dermis
            - 3rd degree – dry, yellow, white – epidermis, upper, lower dermis
            - 4th degree – dry, leathery, waxy – all skin layers damaged, subcutaneous affected
        - Percentage = spread
        - RULE OF 9s / LUND AND BROWDER CHART (especially paeds)
- Rx:
    - Wound management – cool wounds with tap water if within 3 hours, clean with saline, cover with non-adherent dressing, avoid constriction. Irrigation for chemical burns.
    - Fluid resus – fluid shift occurs in the first 8-12 hours, hypovolaemia shock
    - Analgesia – pain ladder as usual but consider burns as extremely painful
    - Reconstruction – debridement, grafting
    - Specific management for specific burn types
        - Chemical – irrigation!!! – at least 30 mins with warm water (different to thermal burn, don't need to stop the burn process< want to prevent patient getting cold)
        - Electrical – arrythmia/myoglobinuria (AKI from rhabdomyolysis)

Fungal nail

- discolouration, lifting of the nail (onycholysis), thinning nail
- first line - **amorolfine nail lacquer**
- second line - **terbinafine** - check LFTs first

Lymphoedema

- Post axillary clearance - breast cancer
- Congenital - Milroy syndrome
- Turner's syndrome

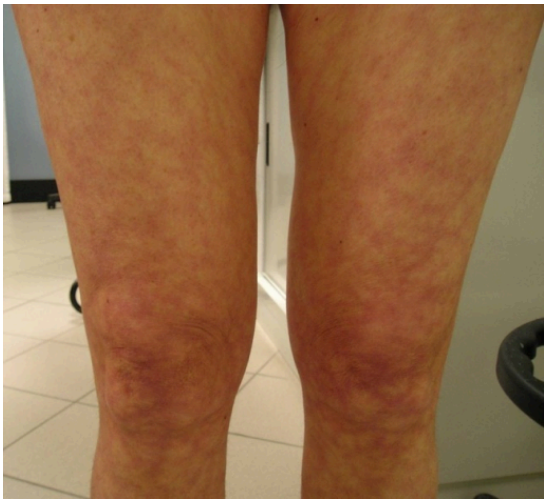
Hand signs

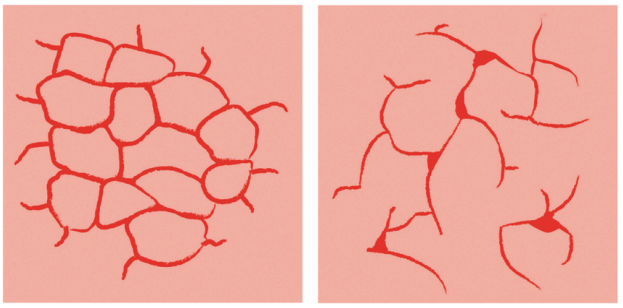
- Tar staining
- splinter haemorrhages
    - trauma
    - endocarditis
    - vasculitis
    - anti-phospholipid syndrome
- pitting - psoriasis
- onycholysis - psoriasis or fungal infection
- Palmar erythema
    - alcoholic liver disease
    - pregnancy
    - rheumatoid arthritis
    - thyrotoxicosis
- Leukonychia
    - hypoalbuminemia (nephrotic syndrome)
- Koilonychia - iron deficiency anaemia
    - malabsorption
    - dietary insufficiency
    - GI bleed
    - endometriosis
- Clubbing
    - Cardiac - endocarditis, cyanotic heart defect, atrial myxoma
    - Resp - pulmonary fibrosis, bronchiectasis, cystic fibrosis, lung malignancy, empyema
    - Gut - IBD, liver disease, coeliac
- Clinodactyly - distal phalanx turning in (deviating towards radius)

Shin lesions

- psoriasis
- erythema nodosum - pregnancy, IBD, sarcoid, TB, iatrogenic (NSAIDs, OCP, penicillin)
- panniculitis
- pyoderma gangrenosum - RA, IBD, leukaemia
- necrobiosis lipoidica
- pretibial myxoedema
- Granuloma annulare - RA

Levido reticularis - paraneoplastic, vasculitis, anti-phospholipid (unless outside in the cold)





Livedo reticularis Livedo racemosa

Livedo Racemosa - similar but more broken markings

DVLA rules

- Diabetes
    - If taking insulin or risk of hypoglycaemia must inform DVLA
    - Additional application is required for HGV licencing with insulin dependent diabetes
    - Good hypoglycaemic awareness/control required to be allowed to drive
    - No hypos in the last 12 months
    - Must monitor at least twice daily, no more than 2hrs before starting a journey and every 2hrs after journey has started
    - LEGALLY MUST NOT DRIVE UNTIL DVLA INFORMED - licence and insurance are invalidated
- Epilepsy/fits
    - Must not drive for 6 months from first fit
    - Must be fit free for 12 months if had multiple fits to apply for licence again
    - DVLA must be informed of first fit + subsequent diagnosis
- Cardiovascular disease
    - MI/ACS - 4 weeks no driving must inform DVLA
    - Post elective cardiovascular procedures e.g. angioplasty - 1 week off
- Stroke/TIA
    - 4 weeks off
    - If multiple TIAs/neurological deficit must inform DVLA

Infectious disease

SEPSIS – systemic infection leading to multi-system organ failure.

- C-RP = complement reactive protein (part of pro-inflammatory cascade) – produced by the liver along with other acute phase reactants
- Fever – hypothalamic response, improves the action of inflammatory enzymes
- Vaso distributive shock – combination of vessels becoming "leaky" and vasodilation.
    - Nitrous oxide and neutrophil margination +/- platelet adherence and thrombosis +/- DIC fat, leaky, shit vessels poor perfusion + third spacing hypoxia + organ death
    - MAP requirement is 65mm Hg or higher – fluids crucial to maintain
- Thrombocytopenia – patients in shock will eventually progress to pancytopenia but platelet productions stopped first (to prioritise white cell production)
- Rx: SEPSIS 6 (give 3 take 3) – broad spectrum antimicrobials in keeping with trust guidelines, check MRSA status if you can.
    - Blood cultures
    - Urine output
    - IV fluids
    - IV abx
    - Lactate – VBG/ABG (>2 abnormal)
    - Oxygen

Meningitis (see neurology section)

Encephalitis

- Inflammation of the brain
- Ix: **EEG**

Endocarditis

- Inflammation typically around a valve (usually tricuspid)
- Sx: fever, fatigue, night sweats, muscle aches
- Ex:
    - murmur
    - Splinter haemorrhages
    - Petechiae
    - Janeway lesions (painless red flat macules on the palms of the hands and soles of the feet)
    - Osler's nodes (tender red/purple nodules on the pads of the fingers and toes)
    - Roth spots (haemorrhages on the retina seen during fundoscopy)
    - Splenomegaly
    - Finger clubbing
- Modified Duke criteria
- Ix: Echo

Myocarditis

- Inflammation of cardiac muscle
- Cause: viral (coxsackie B, HIV), autoimmune, doxorubicin
- Sx: chest pain + dyspnoea, arrythmias
- Ix: troponins (elevated), white cells + CRP, ECG, cardiac MRI
- Rx: treat cause
- Complications: heart failure, arrhythmia, dilated cardiomyopathy

Malaria

- Protozoan infection – plasmodium falciparum
- Spread by sporozoites injected by mosquito bites
- Affects RBCs – parasite grows within red cell and causes rupture haemolytic anaemia
- Rupture + release occurs every 48 hours, this is why fever spikes are monitored (should be every other day)
- Sx: fever, malaise, myalgia, headache, nausea + vomiting

- Ex: pallor, hepatosplenomegaly, jaundice (haemolytic anaemia)
- Ix: Immunochromatographic rapid diagnostic test (not often available) – **blood film**. (to exclude malaria: 3 negative samples over 3 days.)
- Rx: refer to infectious disease team
    - Uncomplicated: Riamet (**artemether with lumefantrine**), **Quinine + doxycycline/clindamycin**
    - Complicated or severe: **Artesunate** is the usual first choice (haemolysis is a common side effect) **Quinine dihydrochloride**
    - **Primaquine** is used to prevent relapse in plasmodium vivax,
- Cannot be treated on clinical suspicion.
- Cerebral oedema – key complication, hence ICU support required with fluids

Legionella

- Pneumonia with hyponatraemia, lymphopenia, deranged LFTs legionella urinary antigen test
- Bilateral, mid-to-lower zone patchy consolidation

Schistosomiasis

- Parasitic flatworm infection S. mansoni = most common
- Endemic infection for travellers
- Risk factor for developing SCC of bladder
- Sx:
    - Acute infection"Swimmer's itch" - rash following exposure to water containing larvae, fever, cough, diarrhoea
    - Chronic infection - urinary frequency, haematuria
- Ix: Eosinophilia, schistosome antibodies in asymptomatic patients, urine/stool microscopy in symptomatic
- Rx: **Praziquantel**

Toxoplasmosis

- Protozoan infection often from cats
- Symptomatic in immuno-suppressed patients, causing cerebral lesions (single or multiple ring enhancing on CT)
- Sx: reduced GCS, confusion, non-specific fatigue + malaise, lymphadenopathy
- Ix: Serology
- Rx: **pyrimethamine** and **sulfadiazine**

Dengue fever

- Retro-orbital headache
- Fever
- Facial flushing
- Maculopapular rash
- Thrombocytopenia - petechiae, epistaxis

Typhoid fever

- Fever, headache, malaise
- rose spots
- constipation
- relative bradycardia

Yellow fever

- Prodromal flu-like phase which resolves (headache, myalgia)
- Then high fever, hepatitis, jaundice, bradycardia
- Ix: hepatocytes → councilman bodies

Prescribing (WPA + PSA)

5% dextrose = 50g in 1000ml
1 in 1000 = 1g in 1000ml

Fluid prescribing
Consider approach (3Rs) - is this Resus, Replacement, or Routine?

1. Resus - 250/500ml isotonic crystalloid, 0.9% Sodium Chloride or 5% Dextrose, over 15 mins
2. Replacement - Consider rate of infusion + if possible, oral! e.g. Sando K
    2.1. Potassium - can only be corrected 10mmol/hour, and added to 0.9% NaCl or 5% Dextrose
    2.2. E.g. **1L 0.9% Sodium Chloride/Potassium Chloride 0.15% (20mmol/L) over 2 hours**
    2.3. Potassium Chloride 0.3% = 40mmol/L
3. Routine - meet daily requirements e.g. reasonably stable patient who is NBM
    3.1. 25-30ml/kg = water
    3.2. 1mmol/kg = K+, Na+, Cl-
    3.3. 50-100g glucose
    3.4. To meet all req a minimum of two prescriptions needed, so in PSA will typically be in a patient who has already had >1L of NaCl in the last 12 hours, or adequate oral intake for glucose
    3.5. E.g. pt who has had 1L of NaCl and 40mmol potassium - **Glucose 5%/Potassium Chloride 0.3% over 12 hours**
4. Frail + Elderly infusion rate = NO FASTER THAN 1L / 10-12 hrs

Common drug interactions

- Omeprazole + Clopidogrel
- Clarithromycin + Statins
- Warfarin - literally everything. Amiodarone, Amoxicillin, Ciprofloxacin, SSRIs, Clarithromycin, even eating more green veg will send your INR off on one

Insulin prescribing

- Short vs Long acting - Long acting is not stopped in DKA, short is just for before meals
- If morning blood glucose too high - increase long acting dose PM
- Consider injection site rotation + compliance

Gentamicin/drug level monitoring

- If peak too high → decrease dose
- If trough too high → decrease frequency
- If trough too low → increase frequency
- If peak too low → increase dose

Appendix 1 in the BNF - drugs causing electrolyte abnormalities

- Wide QTc = Hypokalemia, hypocalcaemia, hypermagnesemia
- Short QTc = Hyperkalemia, Hypercalcaemia

Drug doses to just know

- Adrenaline
    - Cardiac arrest - every 3-5 mins - 1mg / 1ml 1 in 10,000
    - Anaphylaxis - 0.5ml of 1 in 1000
- Amiodarone - broad complex tachycardias - 300mg
- Salbutamol - 5mg every 20-30 mins via nebuliser
- Ipratropium bromide - 500 micrograms via nebs
- Insulin - when changing dosages, 2-4 units, or prn bolus for a hyper 2-4 units good place to start

UHCW FY1

Apps

- Accurx - has all the bleeps + phone numbers, if what you need isn't there go via switch
- Microguide - trust specific abx prescribing
- MDCalc - can pin your favourites, recommend creatinine clearance, BMI etc
- BNF/BNFc - although pharmacists prefer if you use BNF online as better cross-sensitivity checking

Bleeping people

- Can only bleep from hospital phone
- Dial "66"
- should only ring tone once then through to the directory
- input bleep no. you are trying to reach e.g. for my bleep it would be "2627"
- short pause (1-2 seconds) then input extension you are calling from (should be written on the handset you are using) e.g. "28378"
- If accepted, finished
- If "error" or nothing happens, try again
- Receiving bleeps - check number on top of bleep - this is extension to call back
- If you cannot call back right now - WRITE IT DOWN as they may not bleep again and if you get another bleep in the meantime it may not save the message history

How to not piss of the team

- Document your cannulas on EPR
- Find out how the team prefers to document - e.g. some use the clinical summary as the template and then enter exam findings/systems review. Find out how they prefer it to be saved!! e.g. on Geris, type = Elderly Care Progress Note, save as WR Consultant "Gaywood" - for morning ward round with consultant dr gaywood
- Make sure any stat doses are handed over to nursing staff, do not assume they will magically know
- If you speak to patient family document names and phone numbers clearly as free text note
- Blood transfusions - take bloody (if you will pardon the pun) ages to arrange, still on paper, blue transfusion pathway document has a proforma for consent, EPR form also needs to be completed
- If your area has a doctors/nurses jobs list, check it and if you add to it let someone know
- To check if phlebs have <u>not</u> collected bloods - under specimen collection on EPR, should still show as "not collected", sometimes has the reason they were unable to collect also
- Do not forget to prescribe oxygen!!!! Also - if an HCA is on a 1-1 with that patient just let them know what you're doing so they don't panic when they come back from a break and their patient is no longer on oxygen when they were on 3L before. Update sats target if patient found to be retaining CO_2.