Rheumatoid Arthritis

A

- Autoimmune disorder
- M:F is 1:2
- Genetic Component
- Onset usually in young adults
- Increased risk in: ***Smokers, stress, infection***
- Symptoms often worse in hot weather

S+S

- Symmetrical inflammatory arthropathy, causing ***DEFORMITY,*** typically affecting the small joints of the hands and feet (but also knees, hips and elbows).
- Pain and stiffness worse in morning (>20mins)
- ***Characteristic hand signs:*** *z-thumbs, butonniere's, swan-necking, ulnar deviation, muscle wasting, subluxation of MCP, sometimes carpal tunnel syndrome, anlanto-axial subluxation*
- Decreased hand function (knife and fork, doing up buttons), and ***nodules*** are common
- Extra-articular features; Sjogren'ds syndrome, Reynauds, vasculitis, ***Nodules*** (firm but usually painless)

P

- Auto-antibody production (70% IgM, 30% IgG) against joint tissues
- ***DEFOMITY*** is the key characteristic clinical sign

I

- ***Bloods:*** Rheumatoid factor is ***not diagnostic.*** Present in 70%, but also in general population. *Rheumatoid positive disease* has worse prognosis and more extensive deformity. *Anti-CCP antibodies* test similar to rheumatoid factor. ↑ESR + ↑CRP, normochromic-normocytic anaemia common
- ***X-ray:*** look for nodules, soft-tissue swelling, osteopaenia, deformity, erosions

T

- Aim to reduce long-term deformity & LoF. *Steroids* induce remission, beware SE's long term
- *DMARDs* are mainstay. All can cause *myelosuppression and rash* plus: *Sulfasalzine:* hepatic impairment, oligospermia, *methotrexate;* GI disturbance (give folic acid to reduce), mouth ulcers, hepatic impairment *gold;* medical emergency rash, photosensitivity, nephrotic syndrome; *leflunomide, chloroquine:* retinitis, tinitus, *infliximab : anti TNF-α* agent: can cause reactivation of latent diseases (e.g. TB)

Parkinson's Disease

A

•Unknown! Thought to be a mix between genetic predisposition (α-synuclein + Parkin genes) and environmental factors such as exogenous toxins.

S+S

•Bradykinesia.

•Tremor. •Rigidity.

•Tremor – "Pill-rolling" , typically but not exclusively in hands, ↓ on movement.
•Rigidity – Both Lead-Pipe and cogwheel, ↑ on movement of opposite limb.
•Bradykinesia – Difficulty in starting, stopping and changing direction.
- ↓facial expressions
•Posture – Stopped, with shuffling gait.
•↓ handwriting size and legibility.
•Brisk Reflexes

•Constipation
•Depression
•Dementia

P

•Degeneration of dopinergic neurons in substantia nigra of midbrain.
•Eosinophilic Inclusions called Lewy Bodies are found in affected areas.
•Some changes of non-dopinergic neurones -> Why l-dopa doesn't affect all symptoms.

I

•Lab tests not particularly useful
•Head imaging is usually done. (MRI or CT).

T

•MDT!! → OT, Physio, SALT and psychiatric input vital to ↓symptoms and ↑Quality of Life!!
•Pharmacological Treatment → L-dopa is the mainstay, other drugs are used to postpone it's use.
  • L-Dopa → Replaces dopamine levels. Given with peripheral decarboxylase (e.g Madopar) to ensure all dopamine goes to the brain. ↓ effect over time so left as late as possible.
  •Dopamine agonists e.g Peroglide → Directly stimulate dopamine receptors.
  •Anticholinergic agents → ↓tremor but lots of Side effects (CVS!)
  •Selegline → ↓ Breakdown of Dopamine.
•Surgery → Rare though ↑in use. Deep brain stimulation using electrodes in subthalamic region.

Neonatal Jaundice

A

- **Before 24h** = Usually either Sepsis or Blood diseases (Rhesus disease, ABO incompatibility or HS (Hereditary spherocytosis)
- **24h to 2 weeks** – most commonly Physiological or breastfeeding jaundice
- **Prolonged Jaundice** – Breastfeeding jaundice, biliary atresia, sepsis, thyroid problems (hypo), CF

S+S

- Jaundiced! Skin and sclera are orange!
- Signs of underlying disease
  - -Biliary Atresia=Pale stools
  - -Rhesus – Splenomegally.
- Signs of Kernicterus (Bilirubin >350µmol/L)
  - - Lethargic
  - - Poor feeding
  - - ↑muscle tone
  - - Fits
  - - Coma
  - - Death!

P

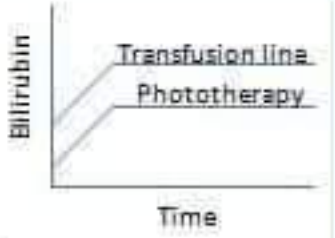
- Jaundice in general = ↑bilirubin (breakdown of haemoglobin) in blood.
- Physiological Jaundice= Fetal Hb has short life span, and the neonatal liver often has difficulty in metabolising it all. Fetal Jaundice happens in around about 60% of babies, so is very common.
- Breastfeeding Jaundice = Breastfed babies more likely to be jaundiced for longer. Multifactoral.
- Kernicterus = ↑↑bilirubin levels cause bilirubin to cross BBB and deposit in basal ganglia and brainstem. This can give long lasting neurological damage, but with good treatment this is avoided.

I

- Good history and examination.
- Bloods (Bilirubin, FBC, LFT, Blood groups, Blood film, blood culture – TORCH SCREEN, Coombs test)
- Urine
- If persistent jaundice, consider USS of biliary tree.

T

- Each centre has its own treatment chart, which look like this →→→→
- Supportive therapy (hydration status etc), if breastfeeding, keep doing so!
- Phototherapy - 450nm wavelength light converts unconjugated bilirubin into a harmless substance, baby must be completely naked but wear eye protection. Can be done via fibre-optic blanket.
- Transfusion – Either done through UVC or peripheral vein and arterial line. 2x babies blood volume of thoroughly screened blood is transfused.



Cystic Fibrosis

A

- Autosomal recessive condition. 1 in 25 are carriers (in UK) 1 in 2500 affected.
- Abnormal genes on Chromosome 7 which produce CFTR protein, most commonly defect is ΔF508

S+S

- Failure to thrive
- Malabsorption
- Recurrent Chest infections
- Harrison's Sulci
- Infertility in males
- Pneumothorax (es)
- Diabetes
- Meconium Ileus
- Bronchiectasis
- Hyperinflated Chest
- Finger Clubbing
- Steatorrhea
- Depression
- School Absences
- Haemoptysis
- Productive cough
- Cholesterol Gallstones
- Pneumonia
- 'Salty Taste'
- Nasal Polyps

P

- CFTR = Cystic Fibrosis transmembrane regulator (Na/Cl pump)
- Its absence gives ↑viscosity of secretions
- Also effects on inflammatory processes and the immune response.
- Infertility in males is due to absent vas deferens

I

- Sweat test → sweating induced by pilocarpine iontophoresis, sweat collected, +ve if NaCl concentration 60<, high false +ve and false –ve rates
- Guthrie test as neonate.
- Genetic screening.
- Bloods (FBC, LFT, U+E, Malabsorption screen, glucose)
- CXR
- Sputum culture

T

- ↑life expectancy!
- MDT!!!
- Annual review in tertiary centres
- Physiotherapy BD
- Abx (?Prophylaxis, may need central venous catheter).
- Regular nebs (saline)
- Counselling
- Gene therapy (very new)
- Diet = ↑calorie ↑protein ↓fat
- Creon (pancreatic enzymes) with meals
- Vitamin supplements
- Lung/Liver transplantation

Endometriosis

A

- Genetic Link
- Retrograde Menstruation
- Sampson's Theory (age with contraceptive/obstetric history)
- May be some blood/lymph borne spread

S+S

- Can be asymptomatic
- Chronic Pelvic pain (cyclical)
- Dysmenorrhoea
- Deep Dyspareunia
- Subfertility
- Dyschezia (Pain on defecating)
- Symptoms of other sites
  - cyclical haematuria
  - blood in stools.
  - Haemoptysis
  - Ubililcal bleeding?
- On examination, Fixed, ?retroverted uterus
- May feel masses on abdo or pv exam.

P

- Presence of endometrial tissue outside of the uterus, normally on uterosacral ligaments, ovaries pelvic wall.
- Tissue responds to cyclical hormones the same as uterine endometrial tissues.
- If in ovaries, forms chocolate cysts, which if rupture give acute abdo pain and peritonitis.
- Because of the inflammation, fibrosis occurs and adhesions form, often fixing the pelvis.

I

- Bloods → Check for anaemia
- Laparoscopy → Diagnosis by seeing active endometriosis or signs of previous endometriosis.
- Transvaginal ultrasound excludes cancers and may show cysts in the ovaries.
- MRI scan may be useful to show undetected lesions.

T

- If asymptomatic then no treatment is needed. Analgesia for pain.
- Hormonal treatment → COCP, Progesterones, IUS, GnRH analogues
- Surgical treatments → Laser/bipolar diathermy, dissection of adhesions, Hysterectomy with bilateral salpingo- oophorectomy and HRT
- Fertility help

- A – Aetiology (& Epidemiology)
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Menopause

A

- A normal physiological process for all women
- Average age is 51 – typically between ages of 45 and 55
- 5% of women will have an ***premature menopause*** (age <40). The majority of these are due to oophorectomy

- **Menopause** is said to have occurred when >12 months has passed since the last period
- **Peri-menopause** is used to refer to symptoms at the time *before* menopause has occurred

S&S

- ***Vasomotor***
  - Hot flushes – 90%
  - Night sweats – 80%
  - Palpitations – 30%
- ***Psychological***
  - *Depression*
  - *Anxiety*
  - *Irritability*
  - *Sleep disturbance*
- ***Other***
  - *Lethargy*
  - *Dizziness*
  - *Headaches / migraine*
- **Urogenital**
  - Vaginal dryness
  - Loss of libido
  - Incontinence
  - Prolapse

P

- Normal physiological response to changing hormone levels - FSH and LH will rise, oestrogen and progesterone will fall
- ***Premature menopause*** exists when menopause at <40. Usually due to oophorectomy, but can be the result of thyroid or other disease. Requires investigation. ***Early menopause*** exists when age <45. Both scenarios should be treated with HRT until age 50.
- Many patients with have symptoms <2 years duration, but up to half will have 5-7 years of symptoms
- Symptoms are very variable between patients – from minor inconvenience to very disabling

I

- Not usually useful or required, if age >45
- If early or premature menopause is suspected, request urinalysis, FBC, TSH, LFTs, iron studies and B-hCG
- In patients <45, confirm menopause by testing for FSH (↑) and oestradiol (↓)

T

- ***Address lifestyle factors*** – weight, alcohol intake, caffeine intake
- ***Vasomotor symptoms*** – consider treatment with HRT
  - Vasomotor symptoms are due to oestrogen deficiency and can be treated with oestrogen
  - Oral preparation (e.g. the COCP) are recommended in patients <50, if not contraindications
  - Transdermal patches are recommended for older patients, and DO NOT increase the risk of VTE like oral versions
  - Treatment for <5 years is NOT associated with increased risk of breast cancer
  - All patients with a remaining uterus ***MUST*** also receive progestin to reduce the risk of uterine carcinoma
- ***Urogenital symptoms*** – consider use of topical oestrogen – such as cream or pessary
- ***Irritability, lethargy, sleep disturbance*** – typically respond well to SSRI, even in the absence of depression
- ***Screen for depression / anxiety*** – and treat with SSRI, as well as psychological and lifestyle interventions

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Osteoporosis

A

- Decreased bone density – a disease of the elderly
- 50% of women over 80, 20% of men over 80
- Lifetime risk of # in osteoporosis – 60% for women, 30% men

- LOTS of risk factors: FHx of osteoporosis, smoking, alcohol, corticosteroid use, PPI use, hypothyroidism, low BMI, sedentary lifestyle, low exposure to sunlight (for vitamin D) early menopause (<45), frequent falls, diabetes, hypothyroidism + many more!

S&S

- **Often presents with a fracture** – *'minimal trauma #' – e.g. hip # at fall from standing height, or vertebral crush #*
- Back pain – from vertebral crush fracture
- Have a very low level of suspicion in any post-menopausal female, especially if multiple risk factors

P

- A gradual reduction in bone density over many years. Due to an imbalance of osteoclast vs osteoblast activity
- An oestrogen-sensitive process – which is why post-menopausal women are at greatest risk
- Also dependent on adequate calcium (dietary) and vit D (sunlight +/- supplementation)
- Sources of calcium – dairy, fish, citrus fruit

I

- Diagnosed when:
  - Minimal trauma fracture of hip or vertebral crush fracture – *consider DEXA for baseline reading, OR*
  - *DEXA shows T score < -2.5. (T score -1 to -2.5 is osteopenia)*
- X-ray any suspected fracture. Reduced bone density is visible on x-ray but only when severe (>40% bone loss)
- **DEXA scan (bone densitometry)** is the most important diagnostic test. ***Knowing when it is indicated*** is the hard part.
  - **Anyone over 70** – no formal screening programme but request opportunistically
  - **Patients <70 with** – minimal trauma fracture any other site OR with ANY risk factors (see aetiology above)

T

- **Lifestyle factors**
  - Smoking cessation - Alcohol <2 standard drinks / day - Ensure BMI >19 Kg/m²
  - Falls prevention –assessment of accommodation and referral to falls reduction programme / physio
  - Strengthening / balance exercise 30 min x 5 days per week. Aerobic exercise is not of any proven benefit
- **Supplementation** – ensure adequate calcium and vitamin D levels before starting medical management
  - Start daily calcium supplementation 1200-1300mg OD. Advise about foods high in dairy (Pathology – above)
  - Start daily vitamin D supplementation 1000-2000 IU
- **Medical management** – start all patients with confirmed diagnoses (as above) on medical management
  - **Bisphosphonates** – *e.g. alendronate 10mg OD daily.* Decrease the rate of bone reabsorption by osteoclasts. Should take on an empty stomach and remain upright for at least 30 mins. Can cause GORD. Can cause **osteonecrosis of the jaw** – especially if Ca2+ or vit D not adequate
  - **Denosumab** – a monoclonal antibody. Binds to "RANKL" - a signaller released by osteoblasts and taken up by osteoclasts. By binding to it – osteoclast activity is reduced. 60mg SC every 6 months. Lower risk of osteonecrosis.

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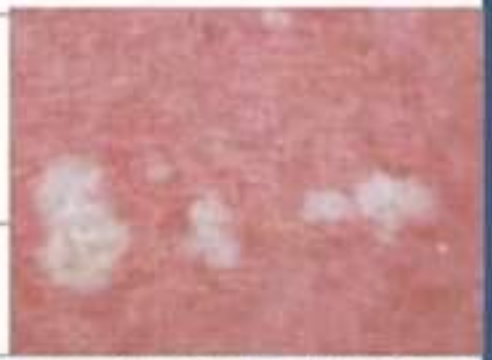
Lichen Sclerosus

A

- Typically in patients >50. Can occur at any age
- Affects 3% of women, 0.05% of men
- Caused by combo of genetic and environmental factors
- A chronic and incurable illness
- Associated with other autoimmune disease
- Associated with: increased BMI, smoking, preceding infection, skin trauma, coronary artery disease

S&S

- Small, well defined, white plaques
- Mainly on the external genitals
- **DOES NOT AFFECT INSIDE THE VAGINA**
- Itchy
- May cause dysuria and dyspareunia



P

- Poorly understood. Likely a combination of genetic and environmental factors
- High risk of squamous cell carcinoma (5% of all LS patients)
- Lesions can cause permanent scarring – including loss of hair if on the scalp

I

- **DIAGNOSIS** – is clinical. Biopsy may assist if there is uncertainty
- Differential diagnosis may include any cause of vulvovaginitis, and particularly:
  - Lichen planus – lesions DO affect the inside of the vagina, and are typically red and raised, not white and flat
  - SCC – especially if only a single patch of disease
  - Candida / thrush can cause similar vaginal discomfort

T

- **Supportive measures**
  - Loose fitting cotton clothing. Avoid synthetic fabrics.
  - Wash gently twice daily with fragrance-free soaps, or using only water to wash affected areas
  - Smoking cessation
  - Avoid certain activities such as horse riding or cycling
- **Topical Steroids**
  - **Typically a potent or ultra-potent agent – such as** mometasone fumorate 0.1% OD
  - Apply thin layer at night
  - Often for 3 months daily, and then dose can be reduced as lesions come under control
  - Controls itch well
- **Topical Oestrogen** may be of some benefit in women
- **Oral immunosuppressant** may be required in severe cases – e.g. methotrexate or ciclosporin
- **Symptomatic relief achieve in >95% of patients**

Myasthenia Gravis

A

- Exact cause unknown, though there is thought to be a strong genetic link.
- Prevalence is 1/200000, and is most commonly seen in younger women and older men.
- At ↑ risk if Family history of autoimmune diseases, or if patient has any (e.g SLE, RA etc)

S+S

- Myasthenia Gravis has a relapsing/remitting course.
- Symptoms generally best in the morning/after a rest.
- Fatigue
- Weakness of muscles → Ptosis, Diplobia, Dysphagia, Dysarthria, Dysphonia
  Weak facial muscles, Weak Shoulders/thighs,
  Weak neck/trunk muscles, weak Respiratory muscles
- Limb Reflexes → Normal or Brisk
- Muscle wasting → Only in severe/ Prolonged disease

P

- IgG autoantibodies produced against the Postsynaptic acetylcholine receptors in neuromuscular junctions.
- Associated disease of the thymus (75% get hyperplasia of thymus, 10% get a thymoma)

I

- Blood Tests → Serum acetylcholine receptor antibodies and other autoantibodies.
- Tensilon Test → Injection of Edrophionium and Atropine → Sudden symptomatic improvement.
- Electromyography → Fatigue following repeated electrical stimulation of muscle.
  → 'Jitter' found on Single Fibre Electrode.
- Thymus imaging → To check for hyperplasia.
- Spirometry → To monitor respiratory muscles.

T

- Oral acetylcholinesterase inhibitors (e.g Pyridostigmine) to ↓breakdown of acetylcholine
- Immunosupression → Corticosteroids usually, but steroid sparing agents can be used.
- Immunomodulatory agents
- In acute crises IV immunoglobulin or Plasmapheresis used.
- Thymectomy.

Chronic Asthma

A

- Asthma is Extrinsic (External Cause) or Intrinsic (no cause found)
- Extrinsic → Atopy, genetic links
- Environmental factors → e.g. hygiene hypothesis

S+S

- SOB.
- Wheezing.
- (nocturnal) productive cough.
- ↓Peak Flow (with dinural variation).
- Harrison's Sulci.
- Hyperinflated chest.
- Other signs of Atopy.
- School Absence
- ↓Exercise Tolerance

Triggers for Asthma Attack

- URTI
- Smoke
- Cold air
- Exercise
- Emotions
- Allergens (dust/pet hair etc)
- Chemical irritants

P

- Individual with genetic predisposition + Environmental factors = Bronchial hypersensitivity.
- Hypersensitivity + Trigger factors (see list) = Inflammation, Oedema, Bronchoconstriction and ↑mucosal secretions.
- Over time remodelling of the airway occurs = ↑goblet cells, ↑smooth muscle, thick membranes.

I

- Spirometry (with trial of treatment)
- Peak flow (peak flow diary)
- Exercise tests
- Skin Prick Tests
- Chest X-ray (to rule out other causes)

T

- Maintain a good QOL
- Patient and family education.
- Lifestyle advice (avoid triggers.)
- Medical treatment →→→→→→→
- Regular reviews, inhaler technique.

- Step 1 - Short acting β2-agonists (e.g salbutamol PRN)
- Step 2 – Add inhaled Steroid (e.g Beclomethasone)
- Step 3 – Add long acting β2-agonists (e.g Salmetreol) **or**
- Step 4 – Add in other agent (e.g montelukast)
- Step 5 – Refer to respiratory paediatrician
  - Add in daily oral steroids

Lymphoma

A

- Thought to be secondary to EBV infection
- M:F 1.5 : 1
- Peak incidence: 20's and 50-70's
- More common in developed countries
- Rare (incidence 3 per 100 000)

S+S

- Cervical lymphadenopathy (70%) – *rubbery, painless, may spontaneously remit*
- Mediastinal widening (CXR)
- Splenomegaly
- Other lymphadenopathy
- **Vague symptoms:**
  - *Pruritis (itch), Fatigue, Anorexia*
  - ***Alcohol induced pain at lymph nodes***
- **B Symptoms** *indicate worse prognosis*
  - *Fever, night sweats, weight loss >10%*

P

- Defect with B cell maturation in lymph nodes. B cells stop expressing correct surface antigens, and are unable to fight infection. *Reed-Sternberg cells* are a classical feature – these are essentially deformed multi nucleated B cells. ***Pathogenic process includes:***
- *Resistance to apoptosis, self- regulation, environmental factors (EBV), genetic factors*

I

- FBC - *normocytic normochromic anaemia,* ***high esoinophils, low lymphocytes***
- *ESR – raised*
- *↑LDH - lactate dehydrogenase –* ***poor prognostic indicator***
- *CXR – mediastinal widening*
- *CT scan - involvement of intrathoracic lymph nodes*
- *Lymph node biopsy - DIAGNOSTIC – Reed-Sternberg cells*

T

- Disease staged I-IV- *Ann-Arbor Classification*. Stages I + II = mild, stage III + IV = severe.
- 5yr Survival – varies, but **Stage I – 95%, Stage IV – 40%**
- **Mild** – ***Radiotherapy ± Chemotherapy.*** Chemo if more severe. Cure rate ~85%
- **Severe** – ***Chemotherapy + Radiation to bulky areas.*** Cure rate ~60%. 15yr survival = 65%
- **Common chemo** – AVBD – *Adriamycin, Bleomycin, Vinblastine, Decarbazine*
- *Initial SE's (chemo); nausea + vomiting, hair loss, myelosuppression (1% mortality), neuropathy*
- *Long term SE's (chemo + radio);* ***↑risk of: lung cancer, infertility, cardiac abnormalities, breast cancer***

Osteoarthritis

A

- Can be **Primary** (no known cause) or **secondary** (as a result of other pathology)
- There is a **genetic component** (Collagen type II genes!)
- Risk factors = **↑BMI**, Hyperparathyroidism, Manual Labourers

S+S

- Several different subsets giving different pictures.
  - Nodal (hands), Hip, Knee, Primary generalised (Hands, knees, big toe, hip)
    Erosive (hands)
- **Pain** → Ache/Burning, worse on movement, worse after inactivity.
- **Swellings**, both fluid and bony e.g Heberden's (DIP), and Bouchards (PIP)nodes
- Joint **stiffness**
- Joint line **tenderness**
- ↓range of movement.
- .Deformities (e.g Bow legs)
- Muscle wasting

P

- **↓In cartilage** so bone exposed
- This gives **↑ bone formation** inside the bone = **SCLEROSIS**
- It also gives **↑bone formation** outside the bone = **OSTEOPHYTES**
- Microfractures in the new bone cause **CYSTS** to form.
- There is also often an effusion due to the synovial membranes thickening due to damage.

I

- **Bloods** → Mainly to exclude septic/inflam/rheum arthritis
- **X-Rays** → Show 4 main changes.
  → **↓Joint space** → **Sclerosis** → **Subchondral Cysts** → **Osteophytes**
- **Arthroscopy** → Can look at the cartilage in the early stages

T

- No cure!
- **Conservative** → Lifestyle advice (Weight loss!), Analgesics, NSAIDS (careful about ulcers!), Physio, Steroid Injections, Glucosamine
- **Surgical** → Debride and washout (knees), Joint replacements, Joint fusion (small bones), Joint excision (rare), Realignment surgery

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Hyperlipidaemia

A

- Affects almost 2/3rds of the adult population
- Strongly correlated with ↑ cardiovascular risk
- Some cases have a strong genetic link – ***familial hypercholesterolaemia (FH)*** – 25x CVD risk
- Causes include: genetic factors, obesity, dietary factors, diabetes, EtOH, liver disease, kidney disease, hypothyroidism, and medications – COCP, thiazide diuretics, beta blockers, steroids
- Most commonly a combination of genetic and lifestyle factors

S&S

- **Often discovered incidentally on screening**
- Suspect if there is FHx of cardiovascular disease before the age of 55
- In severe cases (usually FH) there may be:
  - **Arcus senilis** – a ring of fatty deposit in the cornea (aka corneal arcus)
  - **Tendon Xanthomata** – from fatty deposits in the tendons – especially Achilles tendon
  - **Xanthelasma** – fatty deposits in the thin skin around the eyes

P

- Between 10-40% of the cholesterol in the body is absorbed from the diet. The rest is synthesised by the liver.
- Cholesterol can be divided into **High density lipoprotein (HDL – "good")** and **low density lipoprotein (LDL – "bad")**
- HDL is protective against cardiovascular disease, LDL causes increased risk of cardiovascular disease
- Cholesterol is a key component of the fatty build-up in arteries that form atherosclerotic plaques
- Lowering cholesterol by 10% reduces cardiovascular disease risk by 20%

I

- Ideally, total cholesterol should be <4 mmol/L, and HDL >1 mmol/L. However, when decided to treat cholesterol, there is no hard cut-off level for when to treat. The decision on when to treat is based on the overall cardiovascular disease risk score – for which cholesterol is one of multiple factors. To calculate the score, use QRISK3 or CVDCHECK.
- Screen for hypercholesterolaemia in all patients aged over 45 every 5 years. More often for those at increased risk: annually if high risk, every 2 years if moderate risk.
- Those with total cholesterol >7 should be considered for familial hypercholesterolaemia

T

- **Low cardiovascular disease risk** – lifestyle advice
- **Moderate risk** – lifestyle advice and reassess in 6 months
- **High risk** – lifestyle advice + medication
- Lifestyle advice:
  - **Diet** – low in saturated fat, carbohydrates should be complex, increase intake of plant based foods, eat fish at least x2 per week, steam and grill foods instead of frying, minimise high calorie snacks and treat foods
  - **Alcohol** – no more than 2 standard drinks on any given day, and 2 day alcohol free days per week
  - **Weight** – aim for BMI 18.5 – 24.9. **Smoking cessation**
- Medical management:
  - **Statins** – are the mainstay of treatment – e.g. atorvastatin 10-80mg daily. SE: myalgia, deranged LFTs, GI upset
  - **Ezetimibe 10mg OD** - often used if statin not tolerated, or in combination with statin if cholesterol not controlled

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Hypertension

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A

- Usually "essential" (i.e. primary) with a whole host of predisposing factors, including genetics, and lifestyle factors – smoking, obesity, lack of exercise, diet, alcohol intake, excessive salt intake

- BP rises with age
- Hypertension is present in 30-40% of the population

S+S

- Usually asymptomatic, typically discovered incidentally
- Some patient often say they can "feel" their blood pressure is elevated
- In a hypertensive crisis (RARE – about 1% of hypertensive patients), there may be:
  - Signs of heart failure – SOB, chest pain, cough
  - Signs of raised intracranial pressure – papilledema, confusion, nausea and vomiting, seizure

P

- Genetic and lifestyle factors lead to increases in peripheral vascular resistance, including vasoconstriction and hypertrophy of the arterial wall, which results in reduced arterial wall compliance
- Atheroma (plaque formation) also affects arterial wall compliance

I

- Typically in a primary care (GP) setting. After a raised reading in the GP surgery (>140/90 - either), formal diagnosis confirmed with averages of >135/85 of either:
  - 24-hour ambulatory blood pressure monitoring (ABPM), OR
  - 7 day, twice daily home blood pressure monitoring (HBPM) – ignore the first day and then average the rest of the results

T

- Results should be used in conjunction with a cardiovascular risk tool such as QRISK3 or AUSDRISK to assess the need to treat
- Address the SIX lifestyle factors mentioned in 'A' section above
- Medical management – typically start with an ACE inhibitor (e.g. Ramipril 5mg daily), or use an ARB if not tolerated. Add a diuretic (e.g. indapamide 2.5mg daily) or CCB (e.g. amlodipine 5mg daily) if remains uncontrolled.

Rheumatic Fever

A

- More commonly found in developing countries.
- Due to cross-sensitivity reaction to group A β-haemolytic strep.

S+S

- Develop 2-3 weeks after a strep URTI
- Fever
- Chest Pain
- Pericardial rub
- Pericardial effusion
- Heart Murmur, depends on valve affected.
- Cardiac failure
- Subcutaneous nodules.
- Migratory polyarthritis
- Erythema marginatum
- Chorea

P

- Exact pathogenesis not known
- Oedema and fibrinoid necrosis of collagen
- Aggregations of lymphocytes and macrophages → Aschoff's bodies
- Aschoffs bodies form MacCallum's plaques.

I

- Diagnose with Jones criteria, either 2 major, or 1 major + 2 minor + evidence of strep infection.
  Major criteria = Carditis, Polyarthritis, Erythema marginatum, Subcut Nodes, Chorea
  Minor criteria = Fever, ↑ESR/CRP, Arthralgia, ↑PR interval on ECG, +ve history.
- Throat Swabs, ECG and echo needed.

T

- Bed rest
- Procaine benzylpenicillin 0.6megaunits IM daily for 8 days.
- Aspirin at high doses (100mg/kg/day. Max 6-8g/day for 2/52 → 60mg/kg/day for 6/52
- Prednisolone 1-2mg/kg/day can be given if severe, tapered after 2/52
- Treat chorea if occurs.
- If at high risk, or have a past history of rheumatic fever → Prophylaxis
  Benzathine penicillin 1.2 megaunits, IM every 3/52, or daily phenoxymethylpenecillin 250mg PO

- A – Aetiology (& Epidemiology)
- S+S – Signs & Symptoms
- P – Pathology
- I – Investigations
- T - Treatment

Pertussis (Whooping cough)

A

- Caused by the bacteria *Bordetella Pertussis*
- Vaccination has greatly reduced incidence
- In the developed world, serious cases are rare
- Highly infectious
- Occurs in epidemics every 3-4 years
- Peak age – 3 years
- Particularly dangerous in those aged <3 months
- Also known as the "100 day cough"!

S&S

- Coryzal phase – first 1-2 weeks – runny nose, perhaps mild cough. Can't be clinically differentiated from other causes of upper respiratory tract infection
- Paroxysmal phase – week 3 onwards – the relentless cough begins. "Paroxysmal" means that the cough comes in "coughing fits". The "Whoop" occurs as the child sucks in air at the end of a coughing fit
- Also occurs in adults, but is generally much less serious. Older children and adults don't usually have the "whoop"

P

- Caused by the *Bordetella Pertussis*
- Similar to the bacteria that cause atypical pneumonia
- Complications include: pneumonia, seizures, encephalitis, apnoea
- Typically no longer infectious after 3 weeks, although symptoms remains for much longer

I

- A notifiable disease in both Australia and the UK!
- In the UK – if whooping cough is suspected – notify the local health authority – they will guide the investigations
- In Australia – the hospital or GP initiates investigation and informs the local health authority if and when it is diagnosed
- **Pertussis PCR** – a swab of the nasopharynx is taken (unpleasant for the patient), or in young children, an nasopharyngeal aspirate
- Takes 2-3 days typically for a result. Also often reported with other viral PCR results – e.g. influenza
- Not useful after day 21 – as likely no longer detectible
- **Pertussis serology** – can confirm a previous diagnosis – but often not positive until after 4 weeks – by which time, treatment is not indicated

T

- Treatment only indicated if <21 days since onset of symptoms
- Treat immediately if high clinical suspicion. Can wait for PCR if uncertain
- If positive diagnosis – treat all household contacts, and ANY contact <6 months age
- **Azithromycin** is the drug of choice
  - **Age <6 months** – 10mg/Kg daily for 5 days
  - **Age >6 months** – 10mg/Kg (max 500mg) day one, and 5mg/Kg (max 250mg) for next 4 days
  - **Adults** – 500mg day one, 250mg for further 4 days
- **Clarithromycin** is second line – 7.5mg/Kg BD max 500mg for 7 days. Co-trimoxazole for those with macrolide allergy
- Consider to be no longer infectious after 5 days of treatment or 21 days since onset of symptoms – whichever is sooner

- A – Aetiology (& Epidemiology)
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Stable Angina

A

- A manifestation of cardiovascular disease
- Affect about 4% of population
- Associated with 1.5% annual risk of mortality

- Risk factors are the same as for CVD: hypertension, hypercholesterolaemia (↑LDL, ↓HDL), age, obesity, lack of exercise, smoking, diabetes, FHx CVD, male

S&S

- **Episodic Chest pain** – *may be difficult to differentiate from acute coronary syndromes*
  - ***Typically*** – exertional, relieved by rest and / or nitrites. If doesn't resolve in <5 minutes – **treat as ACS!**
  - May radiate to neck, arm or jaw. SOB may also be present
- Some patients may have several episodes a day whilst other have only infrequent symptoms
- Frequency and severity of symptoms ***does not*** correlate to the severity of the disease

P

- The same as any other manifestation of cardiovascular disease – the build up of atherosclerotic plaques in the arteries, often exacerbated by poor arterial wall compliance ("stiff arteries")
- Associated with an increased risk of MI – clot formation on a plaque will cause ACS
- Studies show that angioplasty / stenting does not improve survival

I

Diagnosed on basis of history, exclusion of ACS & other causes of CP, and imaging – usually CTCA, MPS or stress echo.

- ECG exercise stress test has ***no*** role in the diagnosis of stable angina
- Imaging confirms narrowing of coronary arteries, or in stress echo – muscle contraction changes secondary to ischaemia
- In acute chest pain not settling within 5 minutes of rest or GTN – refer to emergency department!
- **In patient presenting to general practice with history suggestive of stable angina:**
  - X-ray and ECG – usually both will be normal.
  - Stress echo or imaging (e.g. CTCA, myocardial perfusion scan or angio).
  - In all cases of suspected or confirmed coronary artery disease – **refer to cardiologist.**

T

- Initiate treatment as soon as stable angina is suspected – e.g. whilst waiting for specialist cardiology review
- **Acute episodes** – GTN spray sublingually 400mcg – repeat every 5 minutes as requires
- **Medication** –beta-blocker (e.g. metoprolol 25mg BD), short acting nitrate – e.g. GTN spray, statin, aspirin 100mg daily
  - Use calcium channel blocker if beta-blocker not tolerated (verapamil 120mg OD or diltiazem 180mg OD)
  - Consider long acting nitrate – e.g. isosorbide mononitrate 30mg PO daily. Max 120mg daily
- **Weight** – aim for BMI in the healthy range – 18.5 – 14.9.
- **Diet** – appropriate calories, complex carbs, low in saturated fat
- **Exercise** – at least 30 minutes of "moderate intensity" (brisk walk or similar) exercise on at least 5 days a week
- **Smoking cessation**
- **Angioplasty and Stenting (PCI)** – may be considered in severe cases. Relieves symptoms but does not improve survival
- **CABG** – may be considered in severe cases. Improves mortality in severe disease. 85% are symptom free after procedure

Hyperthyroidism

A

- M:F – 1:5. prevalence 2-3% women
- 65% of cases due to **Graves Disease (GD)** - age of onset – 20-40
- 35% of cases due to **toxic multinoduoar thyroid (TMT)** – age of onset – elderly women

S+S

- **Symptoms** - agitation, 'feeling hot', palpitations, ↓weight / ↑ apetitie, diarrhoea, menorrhagia, oligomenorrhoea
- **Thyroid Eye Disease** – *only GD – dry/gritty eyes, lid retraction, lid lag, proptosis, oedema, optic nerve compression (can cause blindness)*
- ***Signs*** *– AF / arrythmia, sinus tachy, fine tremor, goitre (GD=diffuse, TMT=nodular), palmar eythema, moist palms, proximal weakness, gynaecomastia, pretibial myxoedema (GD),* ***increased reflexes with delayed relaxation.***
- ***Thyrotoxic storm*** *- fever, diarrhoea, vomiting, seizures. 30% chance of death. Often with precipitant*

P

- *GD - production of TSH receptor stimulating ABs. Genetic predisposition and environmental factors (?E. Coli). TMT - strong environmental association: high iodine intake (e.g. Dietary, or from drugs - amiodarone*
- ***Complications*** - osteoporosis, slight ↑risk of death in first 12 months (AF)

I

- **Bloods** – TFT's - **TSH(↓) + T3(↑).** T4 also raised, but T3 more sensitive in hyperthyroid. Test T4 and TSH if suspect hypothyroid.

T

- **B-blockers** - give to all patients to reduce symptoms whilst other treatments take time (typically 2-3 weeks due to long HL of T4) to have effect. **Then, Several Options:**
- **Medical** - **Carbimazole** – titrate dose until clinically euthyroid. Then, dose can be reduced over period of 6-24 months. Can cause immunosuppression – particularly a neutropaenia.
  - **Block and replace** - high dose of carbimazole, then give throxine to replace endogenous
  - *Incase of SE's* alternative thiourelenes are available.
  - **Radioiodine** - ^{131}I is given. Taken up into thyroid and destroys thyroid tissue.
- **Surgical** – thyroidectomy. Sometimes parathyroid glands are accidentally also removed, and thus calcium levels should be monitored after surgery.
- ***Problems with treatment*** – many patients become hypothyroid. Life-long monitoring is often required. **NB** – controlling hyperthyroidism in GD does not reduce development/risk of eye complications. If patient become hypothyroid, even greater risk of eye complications

Atrial Fibrillation

A

- *Heart causes:* IHD, HTN, MI, Mitral valve disease, Heart failure
- *Lung causes:* PE, pneumonia
- *Other* – alcohol, thyrotoxicosis, ↓K, ↑Mg
- 5% of over 65's, 10% of over 70's
- 15% of stroke patients

S+S

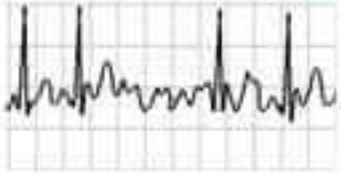
- Palpitations
- Irregularly irregular pulse
- **Pulse deficit:** HR>radial pulse – *ventricular filling does not always occur, so although the ventricles contract, sufficient blood pumping to produce a pulse does not occur.*
- ***Rarely:*** chest pain, dyspnoea

P

- ↑**Atrial rate** (300-600bpm). This cannot be conducted through the AV node, thus conduction to the ventricles is variable, hence the ***irregularly irregular pulse*** at 75-190bpm
- Cardiac output is reduced, and there is ***stasis of blood*** leading to an ***increased risk of stroke***

I

- ECG!
  - Irregular QRS
  - No discernable P waves
  - Normal shape QRS and T



- Bloods
  - ***TFT's*** – for hyperthyroidism
  - ***Cardiac enzymes*** – for MI

T

- *Acute AF* – <48hr duration :
- ***Control rate*** (see chronic AF)
- ***Give heparin*** *to prevent thrombus (CI for CV)*
- *Try mechanical cardioversion* (70% success):
  - *ITU, with O2, with anaesthetist + sedation*
  - *100J, 200J, 360J, 360J*
- ***Medical cardioversion*** *(less successful) :*
  - Amiodarone
  - *Flecanide* – 2nd line
- *Chronic AF* – >48hr – ***Ryhthmn control***
  - B-blocker OR Ca2+ antagonist
  - Add digoxin if ineffective
  - Anticoagulate - dependent on CHADS2 score (usually **WARFARIN**)
- Paroxysmal AF – *occasional spells of AF*
  - ***"Pill in the pocket"*** – *taken when symptoms occur (e.g. Palpitations).* B-blocker (elderly) OR Ca2+ antagonist (if young),. **Digoxin** 2nd line

Migraine

A

- Incidence 8-12% of the population
- Responsible for 30% of head-aches
- M:F – 1:2

- ***Migraine itself:*** 70% without aura, 30% with aura
- Precipitants: *chocolate, oral contaceptives, cheese, caffeine, alcohol exercise, trauma*
- *Associated with periods of rest (e.g. Occur at weekends)*

S+S

- **Unilateral head-ache**
- Often precede by aura – typically *visual*, but may be sensory, speech or taste related
- Episodes last 4 – 72 hours

- No neurological signs
- Heightened pain perception and sensitivity (e.g. To light)
- Associated with puberty, menarche, pregnancy and menopause

P

- Due to alterations in cerebral bloodflow. Initial vasoconstriction may cause aura, then the resulting vasodilation results in pain.

I

- Clinical Diagnosis. Perform a neurological exam to rule out other pathologies. May appear similar to TIA, but TIA will often have ***focal signs, sudden onset, and maximal symptoms at time of onset.*** *Tension headache* tends to be bilateral, no aura, associated with stress / work.
- *Diagnostic criteria* - headaches lasting 4-72 hours with aura (*classical migraine*) ***OR*** headache lasting 4-72 hours, no aura, with nausea or vomiting or photophobia, **PLUS** (2 of...) unilateral, intereferes with normal functioning, worsened with posture (e.g. Bending / walking), pulsating

T

- **Acute management of pain:**
  - *Simple analgesia* - paracetomol, aspirin
  - **5HT agonists – Triptans – e.g. Sumatriptan**
  - **Rebreathing** into a paper bag has been known to halt some attacks by increasing blood CO_2

- **Chronic Management:** *if >2 attacks / month. 65% of patients will have a 50% reduction in attacks*
  - Pizotifen - antihistamine and 5HT antagonist – causes vasoconstriction
  - Beta-bockers – e.g. Propanolol
  - Amytriptyline

Polycystic Ovarian Syndrome

A

- Genetic Link
- Smoking has also been implicated
- Insulin resistance (unproven, some think it is an effect, some think it's a cause)
- Cushing's Disease

S+S

- Amenorrhoea
- Oligomenorrhoea
- Acne
- ↑BMI
- Hirsuitism
- Subfertility
- Insulin Resistance
- ↑Cervical Secretions
- Miscarriages
- On bimanual exam, lumpy masses felt
- Signs of hyper/hypothyroidism
- Signs of diabetes
- Signs of CVS disease
- Acanothosis Nigrans , patches of darker skin caused by ↑insulins

P

- Either ↑LH or ↑insulin gives ↑androgen production by the ovaries, and sometimes the adrenals
- The androgens produced are then not converted into oestrogens as normal, so ↑in free blood androgens.
- The multiply cysts are follicular cysts that haven't ruptured.

I

- Bloods → Hormone levels → ↑LH, ↔/↓FSH, ↑Androgens ,↑ Oestrogens, ↑Prolactins,
  → Thyroid function tests
- Ultrasound ±laparoscopy → Multiple cysts on the ovaries (string of pearls)
- May need CT/MRI of adrenals with Dexamethasone suppression test

T

- Lifestyle advice → Smoking cessation, Increase exercise, weight loss,
- Treat associated conditions → Diabetes, HTN, Hyperlipidaemia
- Treat Hirsuitism (if wanted) → Local (shaving, plucking, bleaching), or systemic (Cyproterone, Spirinolactone, Finasteride)
- If trying to conceive → 1st line, Metformin ±Clomifine, 2nd Line, Other Subfertility methods.
- For irregular bleeding → COCP

Osteoarthritis

A

- Can be **Primary** (no known cause) or **secondary** (as a result of other pathology)
- There is a **genetic component** (Collagen type II genes!)
- Risk factors = **↑BMI**, Hyperparathyroidism, Manual Labourers

S+S

- Several different subsets giving different pictures.
  - Nodal (hands), Hip, Knee, Primary generalised (Hands, knees, big toe, hip)
    Erosive (hands)
- **Pain** → Ache/Burning, worse on movement, worse after inactivity.
- **Swellings**, both fluid and bony e.g Heberden's (DIP), and Bouchards (PIP)nodes
- Joint **stiffness**
- Joint line **tenderness**
- ↓range of movement.
- .Deformities (e.g Bow legs)
- Muscle wasting

P

- **↓In cartilage** so bone exposed
- This gives **↑ bone formation** inside the bone = **SCLEROSIS**
- It also gives **↑bone formation** outside the bone = **OSTEOPHYTES**
- Microfractures in the new bone cause **CYSTS** to form.
- There is also often an effusion due to the synovial membranes thickening due to damage.

I

- **Bloods** → Mainly to exclude septic/inflam/rheum arthritis
- **X-Rays** → Show 4 main changes.
  → **↓Joint space** → **Sclerosis** → **Subchondral Cysts** → **Osteophytes**
- **Arthroscopy** → Can look at the cartilage in the early stages

T

- No cure!
- **Conservative** → Lifestyle advice (Weight loss!), Analgesics, NSAIDS (careful about ulcers!), Physio, Steroid Injections, Glucosamine
- **Surgical** → Debride and washout (knees), Joint replacements, Joint fusion (small bones), Joint excision (rare), Realignment surgery

Epiglottitis

A

- Usually as a result of Haemophilus Influenzae Type B (HiB)

S+S

- Sudden Onset
- ↑temperature
- Ill looking child, sitting up straight, mouth open, drooling
- Soft Stridor and ↑respiratory effort, getting worse over minutes
- Child wont speak or swallow fluids
- Rising Sun Sign → Often present but don't look for it → Angry red epiglottis visible above tongue.

P

- Infection → Inflammation and oedema of the epiglottis.
  → Septicaemia may also be present

I

- Initially treatment is far more important than investigations, as minutes count.

T

- Medical Emergency, minutes do count.
- Don't lie the child down or examine the throat.
- Call for help → Senior Paediatricians, ENT surgeons and Anaesthetists.
- Transfer to ICU or specialist anaesthetic room.
- GA, and intubate carefully, can be extubated in 24h.
- If intubation not possible, tracheostomy is needed.
- Then take bloods for culture.
- IV antibiotics (Cefotaxime 2mg/kg/6h).
- Rifampicin prophylaxis to household contacts.

Heart Failure

A

- ***Causes:*** *Ischaemic heart disease, valvular heart disease, hypertension, arrhythmias,* thyrotoxicosis, *anaemia, pericarditis,* **myocarditis,** *cardiomyopathy, drugs.*

S+S

- SOB (especially on lying flat), peripheral oedema, **fatigue / lethargy,** weight loss, wheeze., Ascites, raised JVP, tachycardia, heave, displaced apex beat, Gallop Rhythmn (S3), ***pink frothy sputum***

- ***New York Classification (NYHA):***
- *I – no limitation of life activities*
- *II – limitation on moderate exercise*
- *III – severe limitation on exercise*
- *IV – breathless and fatigued at rest*

P

- Heart failure is the inability of the heart to pump adequate amounts of blood to meet the bodies demand. It can be due to inability of the muscles (***systolic AF***) or reduced compliance (***diastolic AF***) e.g. Due to compression in pericarditis. Most commonly it **congestive** whereby LVF and RVF co-exist, secondary to initial LVF.

I

- CXR – ***ABCDE***
- *A – alveolar oedema ("bats wing"), B - Kerley* ***B*** *lines, C - cardiomegaly, D – upper lober* ***diversion,*** *E - edema*
- ECG – for cause; arrhythmia, MI, pericarditis etc
- Echo - for vavlular defects, **definitive diagnosis**
- Echo - for vavlular defects, **definitive diagnosis** by checking ejection fraction and wall thickness
- Bloods - FBC (anaemia), LFTs (for secondary liver failure), RFT's (Cr, Ur, K+,Na+), TFTs - thyrotoxicosis.
- Angiography – check extent of IHD
- PFTs - check for another cause of SOB

T

- ***Conservative:*** *exercise, reduce alcohol intake, diet,* ***smoking cessation,*** *flu vaccination*
- ***Medical*** *- **ABCD** – ACEi + angiotension-II antagonists, ß-blockers, Calcium channel antagonists, Diuretics (loop and spironolactone) + Digoxin*
- ***Surgical*** *- revascularistaion (CABG / Angioplasty), Valve replacement, pacemaker, transplant*

- ***Acute HF:*** *"Oi Sofi" - O - oxygen, I - investigate (FBC, ECG, cardiac enzymes, ABG, CXR), S - sublingual (or oral) GTN, O - opiates (reduce anxiety), F – frusemide, I - isosorbide mononitrate (if systolic >90)*
- ***If severe*** *+ve ionotropic agents (e.g. Dobutamine, dopamine), aminophylline (bronchodilate), ventilate*
- ***Chronic AF*** *– patient often well compensated, but an acute event (e.g. MI, infection etc) sets of an 'acute-on-chronic' attack. Treatment the same as acute AF, and* ***remember to treat any underlying cause***

Varicose Veins

A

- Affect 40% men and 32% of women *but* Higher proportion of women present.
- Risk factors → ↑Age, ↑ No. Of Pregnancies, Pregnancy, Long periods of standing, +ve Family History.

S+S

- Appearance of Veins
- Heavy Legs
- Aching
- Itching
- Restless Leg syndrome
- Swelling
- Cramping (worse at night)
- Hot/Burning Feeling

P

- Primary Varicose Veins – Weak vein walls → Dilated veins → Valve cusps can't meet → ***Valve incompetence***.
- Secondary Varicose Veins – Pelvic or abdominal masses → Venous return ***obstructed***.

- Complications = Due to veins themselves → Bleeding, Thrombophlebitis
  = Due to venous hypertension → Oedema, Venous Ulceration, Varicose Eczema, Pigmentation changes, Lipodermatosclerosis.

I

- Good History and Examination
- Cough impulse (Fluid Thrill felt over incompetent vein on coughing)
- Trendelenburg Test → Use Tourniquet and position of pt to determine level of incompetent vein.
- Colour Doppler Venous Scans

T

- Often not needed → Reassure pt.
- **Supportive** (e.g compression stockings for oedema)
- **Traditional surgery** → Ligation and stripping (removal of offending vein and tying valve)
- **Endovenous Laser Therapy** (EVLT) → Catheter placed in leg and laser causes vein ablation.
- **Radiofrequency Ablation** (RFA) → Catheter placed in leg and alternating current ablates vein.
- **Sclerotherapy** → Injection of sclerosant (commonly in a foam) which promotes vein spasm.
- However all surgery has complications, and there are high relapse rates!

Croup

A

- Usually caused by viral infection → Parainfluenza, RSV, rarely measels.

S+S

- Starts with Coryzal symptoms
- Gets a hoarse voice
- Barking cough
- Harsh Stridor, often described as rasping, may reduce as infection progresses.

Signs of severe infection =

- ↑ Respiratory rate
- ↑ Restlessness
- Fatigue
- O_2 Sats <95%
- Soft Stridor

P

- Mucosal inflammation and oedema which extends to larynx, with significant inflammation around the subglottic area.
- Increased secretions

I

- Do not look in the throat, as this may precipitate total airway obstruction!
- Monitor their O_2 sats.

T

- Most cases can be managed at home, with parents advised about the warning signs of severe infection
- Some people use warm, moist air, but its clinical effects unproven.
- Prednisolne 1-2mg/kg PO Stat dose or Dexamethasone 0.15mg/kg Po Stat dose
- May need nebulised adrenaline for severe infection.
- If not improving then will need to be transferred to ICU and may need expert intubation.

Paget's disease of the bone

A

- Affects 5% of >55's. Rare under 50
- Slightly more common in men
- More common in pet owners
- Thought to be the result of environmental factors (?viral) in genetically susceptible individuals

S+S

- <30% will have symptoms
- ***Bone pain***
- Deafness – *if the skull is affected the vestibulocochlear nerve can be compressed*
- ***Pathological fractures***
- Usually unilateral, at one specific site
- Osteosarcoma (rare - <1% of cases)

P

- *Accelerated rate of bone turnover* – osteoblasts are often enlarged and abnormal. Osteoclasts are abnormal but overactive.
- A *chronic progressive disease*

I

- Bloods
  - ***ALP*** – classically raised
  - *Gamma-GT –rule out liver cause of ↑ALP*
- Bone Scan – shows increased uptake in affected bones
- Urine - often contains collagen due to high bone reabsorption
- X-ray
  - Lytic and sclerotic lesions – lots of dark blobs in the bone!
  - Widening of the bone cortex
  - Bone deformities (often bones bent)

T

- Based on symptom severity (e.g. *Pain, fracture, deformity / deafness)*
- Bisphosphonates – e.g. *Pamidronate* - mainstay of treatment ***reduce osteoclast activity*** – *typical regimen ~2 months can induce remission*
- *Analgesia*
- *Treat complications (e.g. Joint replacement)*

Fractures (GENERAL)

A

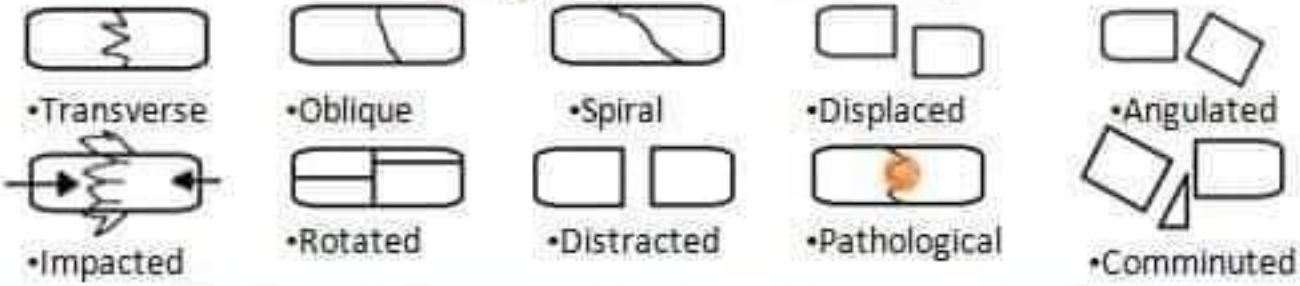
- Happen with normal bone with a lot of force, or little/no force in Weak, **pathological bone**.
- Pathological Bone – Metabolic Bone Disease (**OSTEOPOROSIS**) - Tumours (**mets**!)
  - Rheumatoid Arthritis - Infection

S+S

- VARIES DEPENDING ON THE SITE OF THE FRACTURE, BUT THERE IS USUALLY
- **Pain**
- Unless pathological there is usually history of trauma
- Usually a **tender swelling** over the site.
- Is the fracture **open** (skin broken) or **closed**?
- Visible **Deformity**
- Crepitus may be present
- Signs of nervous insufficiency.
- Signs of Vascular insufficiency.

P

- Different mechanisms of injury give you different fracture patterns.



I

- **X-ray** – Minimum of 2 (AP and lateral) → Some fractures have special views, Joints need x-ray
- **CT** – Useful for assessing complicated fractures prior to surgery.
- **MRI** – Useful if fracture is hard to spot on x-ray, or in looking for Avascular Necrosis.

T

- ABC!
- **Reduction** – Either **open** or **closed**
- **Fixation** – **Conservative** (Casts, traction (rare) or **Surgical** (Wires, Internal/external fixation, Intramedullary nails)
- **Mobilization** – MDT → Physio, OT, Social workers

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Peptic Ulcer

A

- M > F
- Duodenal ulcer 2-3x more common than gastric ulcer
- Caused by infection with *Helicobacter Pylori*
- Alcohol
- ***Smoking and NSAIDs*** – reduce prostaglandin synthesis
- *Peptic ulcer disease* – aka **PUD**

S&S

- ***Symptoms:*** epigastric pain, vomiting – especially if first thing in the morning - it may relieve pain, weight loss (patient may eat less to try and avoid pain), tiredness (secondary to anaemia)
- 50% of patients are asymptomatic
- ***Gastric ulcers*** – pain may be relieved by eating as it neutralises stomach acid
- **Red flags:** weight loss, fevers, melaena, persistent vomiting, age >55, dysphagia, iron deficiency anaemia

P

- H. pylori produces ammonia to neutralise acid around the cell, and thus enables the cell to survive in the stomach.
- *H. Pylori* tends to live in crypts just next to the acidic sensors of stomach acid, thus the normal feedback mechanisms are altered, and excess acid is produced. The bacteria causes localised inflammation, and the body's own stomach acid can worsen the effects of the inflammation. There may often be a pan-gastritis due to excess acid.

I

- **Stool test** – Tests for the presence of H. pylori, put PPI's must be stopped a week before the test
- **Urea breath test** – if H. pylori is present, any urea ingested will be converted to ammonia by *H. Pylori* and then absorbed by the body. Urea labelled with an uncommon isotope (e.g. carbon13) can then be detected in the breath of an affected individua. If H. pylori is not present, urea will pass through the GI tract undigested, cannot be detected in the breath.
- **Serum IgG** – IgG against H. pylori. Typically easier to perform than stool or breath tests, but levels do not fall for many months after eradication, thus you can't use this as a test of cure
- **Endoscopy** – should be performed in anyone aged over 55 or with red flag symptoms. Also consider endoscopy in patients who, don't respond to treatment. Enables biopsy (for cancer).
- **FBC** – to check for anaemia
- **Faecal occult blood** – not specific. May be indicated if clinically has melaena to confirm the presence of blood in the stool

T

- PPI and antibiotic combination eradication therapy - aka triple therapy is recommended for all patients with confirmed H. Pylori.
- **Triple therapy** – this is a PPI and two AB's, e.g.:
  - Omeprazole, metronidazole and clarithromycin – all given twice daily.
  - The antibiotics are taken for 7 days, and the PPI for a further 3-4 weeks
- **Complications:** haemorrhage – especially if the ulcer overlies a blood vessel, anaemia, cancer and **perforation**
- **PEROFRATION** can be life threatening. It may be the first and only sign of PUD. ***Signs:*** *sudden onset epigastric pain, may radiate to the back, peritonitis, shock, absent bowel sounds, increased RR, fever, tachycardia, CXR (gas under diaphragm)*
- ***Mortality from perforation is 25%***

- A – Aetiology (& Epidemiology)
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Haemochromatosis

A

- A genetic disorder of iron metabolism, caused by inherited defects in the HFE gene (chromosome 6)
- Two common defects are C282Y and H63D
- Usually on a clinical problem if homozygous
- Most common in Northern European ancestry – up to 20% are carriers. Much less in other races
- Variable penetrance means that even homozygous individuals may not develop significant disease

S&S

- Most heterozygous patients are asymptomatic, and only some homozygous patients will develop symptoms
- Presents late in the disease process
- Typically presents late in life – around age 30-50 in men and after menopause in women
- Presentation can include: general malaise, weakness, joint pains, erectile dysfunction
- More significant signs include: skin discolouration, diabetes, heart failure, cirrhosis, hepatocellular carcinoma

P

- Dysfunction of the HFE gene means that iron is not properly metabolised and excreted. This can lead to accumulation in organs, and organ failure – particularly significant tis that of the liver and heart.
- Complications include:
  - Liver failure, cirrhosis, hepatocellular carcinoma
  - Heart – heart failure, cardiomyopathy, arrythmia
  - Neurological dysfunction – mood swings, depression, irritability, erectile dysfunction
  - Endocrinological – diabetes
  - Joint pain

I

- Consider haemochromatosis in anyone whom presents with deranged LFTs and increased ferritin and iron saturation
- Normal ferritin effectively rules out haemochromatosis
- Iron overload diagnosed as:
  - Ferritin >1000g/L
  - Ferritin >300g/L (men) or 200g/L (women) ***AND*** transferrin saturation >45%
- Consider other causes (e.g. alcoholism) if the transferrin saturation is <45%
- In anyone who meets the criteria for iron overload – consider HFE genetic testing
- Siblings of anyone with confirmed haemochromatosis should also be tested – they have a 25% chance of being affected

T

- Mainstay of treatment is ***venesection*** – blood letting. Typically in doses of 500mls per venesection procedure
- There is an initial iron unloading phase – to quickly lower ferritin, and then a secondary maintenance phase
- ***Iron unloading phase*** – typically venesection every 1-4 weeks until ferritin <100g/L
- ***Maintenance phase*** – maintain ferritin 50-100g/L. For most patients this is about 3-4 venesection procedures / year
- Advise alcohol cessation / avoidance
- Advise to avoid iron supplements and vitamin C supplements
- Consider Hep A and B vaccination to protect the liver against other causes of cirrhosis
- Life-expectancy is essentially normal if successfully managed
- Patients with heterozygous disease should have iron studies every 2-3 years

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Cataracts

A

- A very common visual disorder
- Caused by degradation of proteins in the lens of the eye
- Affects about 65% of people over 50 and 100% of people over 80, to varying degrees

- Risk factors include: age, smoking, UV light exposure (e.g. if worked outdoors) steroid use, alcohol, previous eye trauma, diabetes
- Genetic factors important. Some cases are congenital

S&S

- Gradual reduction in visual acuity
- "Cloudy" vision
- Difficulty reading, or recognising faces
- Difficult driving – especially at night
- Painless

P

- Caused by the breakdown of proteins within the lens of the eye
- This breakdown causes the lens to opacify
- Wearing sunglasses if has large amount of sun exposure may reduce risk
- Treated surgically – the new lens often also is often tailored to correct any refractive error

I

- Assess visual acuity – considered significant if worse than 6/12
- Assess the impact on daily functioning and daily activities
- Clinical diagnosis
- Consider surgery for those with acuity worse than 6/12 or significant impact to ADLs

T

- Surgical replacement of the lens is the only effective treatment
- Usually done as a day case under local anaesthetic with or without sedation. Usually takes <10 minutes!
- The lens is broken down using ultrasound and removed. A new synthetic lens – plastic or silicone – is inserted
- Ensure patients are aware of the risk of complications:
  - **Posterior capsule opacification** – a cloudy layer of scar tissue forms at the posterior of the lens capsule after replacement.
  - Posterior retinal detachment (rare)
  - Visual disturbance
  - Bleeding
  - Infection
  - Glaucoma
- **Post surgical care:**
  - Eye patched for 24 hours
  - Can return to normal activities – including reading, watching TV etc immediately
  - Avoid bending, strenuous exercise and heavy lifting for several weeks
  - Avoid swimming and driving for 5 days
- Typically dramatic improvement in acuity in noted immediately, but it may take several months for the eye to fully adjust to the new lens

almostadoctor

SLE

A

- Autoimmune connective tissue disorder
- M:F = 1:10
- More common in Black Africans and Indian Asians than Caucasians
- Strong genetic component
- Onset at any age, peaks at 25-35 and 50-60

S+S

- Photosensitive (often malar) rash
- ***Non-specific symptoms;*** malaise, fatigue, weight loss, alopecia, mouth ulcers
- ***Arthritis*** – a symmetrical polyarthritis, usually affecting the small joints, similar to RA. Unlike RA, changes do not cause ***loss of function***
- Splinter hemorrhage and nail infarcts
- Renal impariment
- Haemaotlogical dysfunction; ***leucopaenia*** and ***lymphoma,*** as well as ***haemolytic anaemia***
- Neurological signs (e.g. Seizure)

P

- Basically unknown, but thought to e autoimmune
- Results in the production of ***ANA*** (anti-nuclear antibodies). These are highly sensitive, but not specific (can be due to. RA, medications (e.g. anti-TNFs))
- ***Complications:*** ↑risk of athersclerotic disease, infection, thrombosis

I

- ***Urine dipstick*** – for renal impairment - red cell casts, and proteinuria
- ***Bloods*** – ***↑ESR + ↑CRP,*** *anaemia, thrombocytopaenia, lymphopaenia, leucopenia*
- **ANA-testing**
- **Anti-doublestranded-DNA** – highly specific, but only present in 60% of cases

T

- Mild cases can be managed with ***NSAIDs*** and ***lifestyle changes*** (e.g. Avoid sunlight)
- Moderate to severe: treatment similar to RA; *steroids* to induce remission, long-term use:
- ***DMARDs:*** All can cause *myelosuppression and rash* plus: *Sulfasalzine:* hepatic impairment, oligospermia, *methotrexate;* GI disturbance (give folic acid to reduce), mouth ulcers, hepatic impairment *gold;* photosensitivity, nephrotic syndrome; *leflunomide, chloroquine:* retinitis, tinitus, *infliximab ; anti TNF-α* agent: can cause reactivation of latent diseases (e.g. TB)

Scleroderma

A

- Autoimmune connective tissue disorder
- M:F = 1:4
- Onset at any age, peak 30-50
- Smoking increases risk

S+S

- ***CREST*** - *C – Calcinosis – calcium deposits, usually in fingers, R – Reynaud's, E – Esophageal dymotility* causing GORD, *S – Sclerodactyly, T – Telangectasia*
- ***Other signs*** – hypopigmentation (occasionally hyper-), ulceration, necrosis and gangrene of affected tissue – usually hands and feet, Sjogren's syndrome (dry eyes + mouth), mouth ulcers, polyarthropathy
- ***Organ involvement*** – can affect any organ, but most commonly ***kidneys, GIt, heart and lungs***

P

- Autoimmune disorder, resulting in vascular damage.
- Organ damage usually due to fibrosis, secondary to vascular pathology
- ***Renal and pulmonary complications are life threatening***
- There may be periods, lasting from weeks to months, of apparent symptom regression

I

- ***Anti-centromere antibodies*** – in 40% of cases – associated with the milder *limited cutaneous scleroderma - LCS*
- ***Anti-Scl antibodies*** – in 70% of cases – associated with the more severe *diffuse cutaneous scleroderma - DCS*

T

- Hard to treat. No specifics for disease itself. Complications managed individually. *Immunosupressants* may be used in severe flare-ups (e.g. IV cyclophosphamide)
- *Renal impairment – ACE-inhibitors, Oesophageal - PPI's, Pulmonary hypertension - calcium channel blockers, annual spirometry monitoring Heart - annual ECG*
- ***Prognosis*** - *LCS* – 10yr >75%, *DCS* - 10yr-55%. Death usually from lung/heart/renal complications

Endometriosis

A

- Genetic Link
- Retrograde Menstruation
- Sampson's Theory (age with contraceptive/obstetric history)
- May be some blood/lymph borne spread

S+S

- Can be asymptomatic
- Chronic Pelvic pain (cyclical)
- Dysmenorrhoea
- Deep Dyspareunia
- Subfertility
- Dyschezia (Pain on defecating)
- Symptoms of other sites
  - cyclical haematuria
  - blood in stools.
  - Haemoptysis
  - Ubililcal bleeding?
- On examination, Fixed, ?retroverted uterus
- May feel masses on abdo or pv exam.

P

- Presence of endometrial tissue outside of the uterus, normally on uterosacral ligaments, ovaries pelvic wall.
- Tissue responds to cyclical hormones the same as uterine endometrial tissues.
- If in ovaries, forms chocolate cysts, which if rupture give acute abdo pain and peritonitis.
- Because of the inflammation, fibrosis occurs and adhesions form, often fixing the pelvis.

I

- Bloods → Check for anaemia
- Laparoscopy → Diagnosis by seeing active endometriosis or signs of previous endometriosis.
- Transvaginal ultrasound excludes cancers and may show cysts in the ovaries.
- MRI scan may be useful to show undetected lesions.

T

- If asymptomatic then no treatment is needed. Analgesia for pain.
- Hormonal treatment → COCP, Progesterones, IUS, GnRH analogues
- Surgical treatments → Laser/bipolar diathermy, dissection of adhesions, Hysterectomy with bilateral salpingo- oophorectomy and HRT
- Fertility help

Fibroids

A

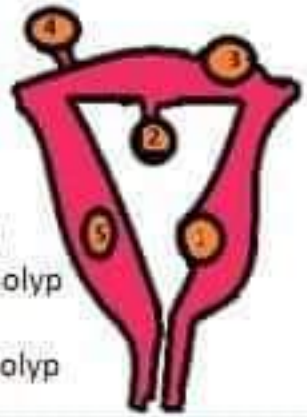
- Also known as leiomyomata, common benign tumours of myometrium.
- Enlargement due to oestrogens (sometimes progesterones too). As a result growing after the menopause, but growth may be resumed by HRT.
- Large variation in size, size may change due to the hormonal cycle.

S+S

- 50% asymptomatic
- Menorrhagia (typically if fibroid is submucosal or polypoid)
- Pain (rare unless torsion or degeneration)
- Pressure effects as fibroid grows, commonly Dysuria, hydronephrosis, constipation, sciatica
- Subfertility
- Abdominal/pelvic mass

P

- 0.1% may be malignant.
- If pedunclated, the fibroid can twist causing torsion, and acute pain.
- Fibroids can also 'degenerate' due to ↓blood supply, there is then pain, haemorrhage and sepsis.
- In Pregnancy, fibroids can give → Premature labour, malpresentation PPH and prolonged labour due to obstruction.



I

- Ultrasound → lets you know if mass
- MRI/laparoscopy → distinguish type (see picture) →→→
- Hysteroscopy/hysteroscalpingogram if in cavity.

1- submucosal
2-Intracavity polyp
3-Subseruous
4-Subserous Polyp
5 - Intramural

T

- If asymptomatic and small → no treatment!
- Tranexamic acid, NSAIDS often used, but ineffective for fibroid induced menorrhagia
- GnRH agonists work by inducing menopause, but can only be given for 6/12 then fibroids regrow.
- Surgery → Hysteroscopic (if submucosal or polyps), hysterectomy, myomectomy, Uterine artery embolisation

Hyperthyroidism

A

- M:F – 1:5. prevalence 2-3% women
- 65% of cases due to **Graves Disease (GD)** - age of onset – 20-40
- 35% of cases due to **toxic multinoduoar thyroid (TMT)** – age of onset – elderly women

S+S

- **Symptoms** - agitation, 'feeling hot', palpitations, ↓weight / ↑ apetitie, diarrhoea, menorrhagia, oligomenorrhoea
- **Thyroid Eye Disease** – *only GD – dry/gritty eyes, lid retraction, lid lag, proptosis, oedema, optic nerve compression (can cause blindness)*
- ***Signs*** *– AF / arrythmia, sinus tachy, fine tremor, goitre (GD=diffuse, TMT=nodular), palmar eythema, moist palms, proximal weakness, gynaecomastia, pretibial myxoedema (GD),* ***increased reflexes with delayed relaxation.***
- ***Thyrotoxic storm*** *- fever, diarrhoea, vomiting, seizures. 30% chance of death. Often with precipitant*

P

- *GD - production of TSH receptor stimulating ABs. Genetic predisposition and environmental factors (?E. Coli). TMT - strong environmental association: high iodine intake (e.g. Dietary, or from drugs - amiodarone*
- ***Complications*** - osteoporosis, slight ↑risk of death in first 12 months (AF)

I

- **Bloods** – TFT's - **TSH(↓) + T3(↑).** T4 also raised, but T3 more sensitive in hyperthyroid. Test T4 and TSH if suspect hypothyroid.

T

- **B-blockers** - give to all patients to reduce symptoms whilst other treatments take time (typically 2-3 weeks due to long HL of T4) to have effect. **Then, Several Options:**
- **Medical** - **Carbimazole** – titrate dose until clinically euthyroid. Then, dose can be reduced over period of 6-24 months. Can cause immunosuppression – particularly a neutropaenia.
  - **Block and replace** - high dose of carbimazole, then give throxine to replace endogenous
  - *Incase of SE's* alternative thiourelenes are available.
  - **Radioiodine** - ^{131}I is given. Taken up into thyroid and destroys thyroid tissue.
- **Surgical** – thyroidectomy. Sometimes parathyroid glands are accidentally also removed, and thus calcium levels should be monitored after surgery.
- ***Problems with treatment*** – many patients become hypothyroid. Life-long monitoring is often required. **NB** – controlling hyperthyroidism in GD does not reduce development/risk of eye complications. If patient become hypothyroid, even greater risk of eye complications

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Carpal Tunnel Syndrome

A

- Affects 5-10 of the population
- F > M
- Typical age of presentation – 30 - 50

- Risk factors include: pregnancy, hypothyroidism, diabetes, renal failure, obesity, menopause, Hx of inflammatory arthritis, overuse of wrist (extension activities) – e.g. tennis, rowing, cycling, production line workers

S&S

- ***Paraesthesia*** – 'pins and needles' in distribution of the **median nerve** – lateral 3 fingers, and half of 4th finger
- ***Pain*** – in the same distribution. Especially at night. May wake the patient from sleep
- ***Wasting of the thenar eminence*** – the muscle mass at the base of the thumb
- Can be bilateral or unilateral

P

- Compression of the median nerve as it passes through the ***carpal tunnel*** on the palmar aspect of the wrist – under the ***flexor retinaculum***
- Thought to be due to swelling from ***flexor tenosynovitis*** of the wrist. The swelling compresses the nerve

I

- Usually a clinical diagnosis. If there is significant doubt, nerve conduction studies may be performed (rarely required)
- Based of classical symptoms as above
- There are several special tests:
- **Durkan's test** – this is the most sensitive test. The examiner applies pressure over the carpal tunnel for 30s. A positive test occurs when symptoms of paraesthesia or pain are elicited in the distribution of the median nerve
- **Phalen test** – patients holds hands out, with elbows and wrists extended. Positive if symptoms elicited within 60 seconds
- **Tinel's test** – least sensitive and specific. Examiner taps repeatedly over carpal tunnel. Positive if symptoms elicited
- **Hand diagram** – draw around patients hands on piece of paper. Ask them to mark the areas that are affected. Positive test if distribution matches that of the median nerve.

T

- **First line** – *conservative management*
  - **Treat any underlying causes** – e.g. hypothyroidism, diabetes, obesity
  - Avoid any aggravating activities, e.g. desk / keyboard position at work, sporting activities
  - Try use of a wrist splint – particularly at night
  - NSAIDs for analgesia
- **Second line** – *corticosteroid injection into the flexor tendons*
  - 80% of cases respond (bad prognostic indicator if they don't). Typically effect wears off after several months
  - 20% will be pain free at 12 months.
- **Third line** – *surgical decompression*

Varicose Veins

A

- Affect 40% men and 32% of women *but* Higher proportion of women present.
- Risk factors → ↑Age, ↑ No. Of Pregnancies, Pregnancy, Long periods of standing, +ve Family History.

S+S

- Appearance of Veins
- Heavy Legs
- Aching
- Itching
- Restless Leg syndrome
- Swelling
- Cramping (worse at night)
- Hot/Burning Feeling

P

- Primary Varicose Veins – Weak vein walls → Dilated veins → Valve cusps can't meet → ***Valve incompetence***.
- Secondary Varicose Veins – Pelvic or abdominal masses → Venous return ***obstructed***.

- Complications = Due to veins themselves → Bleeding, Thrombophlebitis
  = Due to venous hypertension → Oedema, Venous Ulceration, Varicose Eczema, Pigmentation changes, Lipodermatosclerosis.

I

- Good History and Examination
- Cough impulse (Fluid Thrill felt over incompetent vein on coughing)
- Trendelenburg Test → Use Tourniquet and position of pt to determine level of incompetent vein.
- Colour Doppler Venous Scans

T

- Often not needed → Reassure pt.
- **Supportive** (e.g compression stockings for oedema)
- **Traditional surgery** → Ligation and stripping (removal of offending vein and tying valve)
- **Endovenous Laser Therapy** (EVLT) → Catheter placed in leg and laser causes vein ablation.
- **Radiofrequency Ablation** (RFA) → Catheter placed in leg and alternating current ablates vein.
- **Sclerotherapy** → Injection of sclerosant (commonly in a foam) which promotes vein spasm.
- However all surgery has complications, and there are high relapse rates!

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Rosacea

A

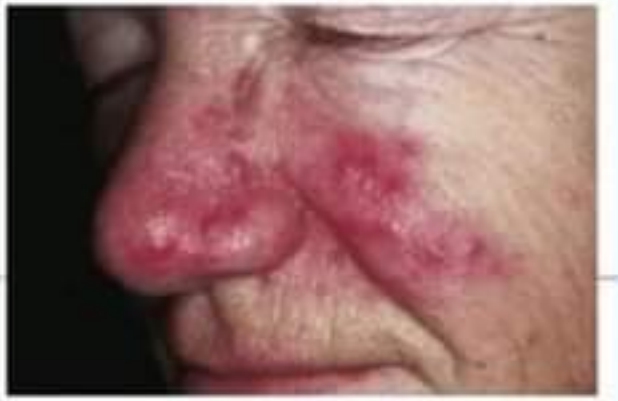
- Inflammatory skin condition of unknown aetiology
- F > M
- Tends to present between age 30-50
- May be exacerbated by alcohol, sudden changes in temperature, emotional stress, hot or spicy foods, hot baths, exercise
- Celtic" origin – fair hair, blue eyes

S&S

- ***Facial flushing***
- ***Papules, pustules and nodules*** – may be mistaken for acne
- ***Telangiectasia***
- *Typically affects the cheeks and forehead. Periorbital and periorificial areas are often spared*

P

- An inflammatory skin disorder, resulting in papules, pustules and nodules
- Over the longer-term, can result in connective tissue changes – causing complications, such as rhinophyma (a large, red bullous nose) or blepharitis
- Can be quite distressing for patients



I

- A clinical diagnosis
- Based on appearance of the lesions, in conjunction with exacerbating factors, and typically patient demographics (age 30-50, typically female)
- Usually can be clinically distinguished from acne

T

- **First line** – *topical metronidazole 0.75% for 6-12 weeks*
  - Often patient require ongoing long-term therapy
- **Second line** – *oral tetracycline antibiotics – e.g. doxycycline 100mg OD for 8 weeks*
  - Long term antibiotics can also be used – but typically at a lower dose – 50mg OD
- **Avoidance of aggravating factors**
  - Sun exposure, alcohol, sudden temperature changes, hot or spicy foods, excessive exercise, hot baths or showers, wind exposure
- **Topical skin care**
  - Wash with a soap-free product (e.g. emollient), avoid oily skin products

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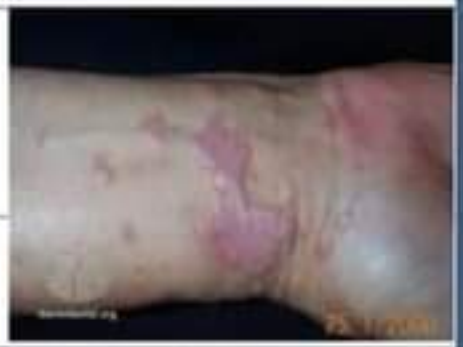
Lichen Planus

A

- Typically in patients >40
- Affects 1-4% of the population
- 50% of cases involve the oral mucosa
- More common in women
- Genetic predisposition
- 10% of cases affect the nails

S&S

- Typically a raised, red plaque-like rash
- In the mouth - often white patches.
- Itchy! Not painful
- May also affect genitals – including inside the vagina
- Longitudinal lines on the nails



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P

- Not well understood. Thought to be a T-cell mediated autoimmune disease
- Flares up at times of stress or after skin trauma
- Associated with hepatitis infections

I

- **DIAGNOSIS** – is clinical. Biopsy may assist if there is uncertainty, can can show:
  - T-cell infiltration of the dermis
  - Reduced melanocytes
  - Globular deposits of IgM (and sometimes IgG) under direct immunofluorescence
- **Differential diagnosis**
  - Lichen sclerosis – typically genital, but can affect other areas. Does NOT affect inside of vagina
  - Drug reaction
  - Eczema
  - Psoriasis
  - Candidiasis (especially if only in mouth)

T

- Most cases resolve spontaneously within a year
- Topical steroid are the mainstay of treatment
  - Can help to bring a flare-up under control – and particularly useful for controlling the itch
  - Start with moderate potency and increase if required
- Mucous membrane disease is particularly resistant to treatment
- Hyperpigmentation can occur from previous lesions
- 1% lifetime risk of oral squamous cell carcinoma – especially if smoker, high alcohol intake or associated Hep C infection

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Peripheral Vascular Disease

A

- A manifestation of cardiovascular disease (like coronary artery disease and cerebrovascular disease)
- 10% of population (maybe up to 30% - 2/3rds asymptomatic)
- 75% of patients also have coronary artery disease

- Risk factors are the same as for CVD: hypertension, hypercholesterolaemia (↑LDL, ↓HDL), age, obesity, lack of exercise, smoking, diabetes, FHx CVD, male

S&S

- **Claudication** is the classical sign – *pain in the calf, induced by activity (usually walking) and relieved by rest*
  - May radiate up the leg – as opposed to ***neurogenic claudication*** which radiates from buttock down leg
- As disease progresses, exercise tolerance ('claudication distance') decreases
- Absent peripheral pulses, poor skin condition, including ulceration
- **Acute presentation** – acute limb ischaemia – severe pain, foot pale and cold, no sensation, pulseless

P

- The same as any other manifestation of cardiovascular disease – the build up of atherosclerotic plaques in the arteries, often exacerbated by poor arterial wall compliance ("stiff arteries")
- The level of arterial occlusion directly correlates to the severity of symptoms
- Can acutely occlude and cause acute limb ischaemia

I

Diagnosed on the basis of the *Ankle-Brachial Pressure Index – ABPI*

- With patient lying supine on couch, measure the BP in the arms (bilaterally) and ankle (bilaterally)
- Take the single highest systolic reading from the arm, and the same from the ankle
- Divide the ankle reading by the arm reading. **Normal = >1.0.** **Diagnostic for PVD if <0.9**

Other investigations should include HbA1c, lipids, FBC, UEC and ECG – to check for the co-morbidities associated with PVD

CT angiogram can show the extent of disease but is mainly useful for surgical planning

T

- Many of the same factors for other cause of cardiovascular disease
- **Weight** – aim for BMI in the healthy range – 18.5 – 14.9
- **Diet** – appropriate calorie intake, complex carbohydrates, low in saturated fat
- **Exercise** – at least 30 minutes of "moderate intensity" (brisk walk or similar) exercise on at least 5 days a week
  - "Walking plan" – recommend walking to claudication and stopping for a rest when necessary
  - Difficult to achieve if exercise limited by claudication. Improves collateral circulation and arterial wall compliance
  - Walking plan can be as effective as surgery
- **Medical** – all patients with confirmed peripheral vascular disease should be started on a statin, and anti-platelet drug – e.g. aspirin 100mg daily or clopidogrel 75mg daily. Control hypertension – e.g. ramipril 5mg daily
- **No drug therapy has been proven to improve the symptoms of intermittent claudication**
- **Diabetes** – screen for and treat any diabetes.
- **Surgical intervention** – should be considered for those with severe disease who have not responded to walking therapy
- May include bypass grafting or angioplasty with stenting

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Dementia

A

- Incidence declining slightly, prevalence increasing with growing population
- Affects 5% of > 65s, 20% of > 80s and 80% of > 100s
- Female: Male ratio 2:1

- **Alzheimer's:** family history, cardiovascular risk factors, hypothyroidism, depression, prev. head injury, HIV
- **Vascular:** cardiovascular risk factors – smoking, hypertension, hyperlipidaemia, obesity, sedentary

S&S

- **Alzheimer's:** gradual onset, decline of particularly short-term memory. Autobiographical and political memory often well preserved. Poor concentration, poor sleep, low mood. Personality change – disinhibited, aggression, lack of self-care. In end stages – hallucinations, poor dentition, skin ulcers, loss of verbal communication
- **Vascular:** symptoms are similar but often may be sudden onset, and progress in a stepwise fashion. Often other signs of cardiovascular disease – e.g. coronary artery disease, peripheral vascular disease.

P

- **Alzheimer's** – poorly understood. There is ***deposition of beta-amyloid plaques*** between neurones – which is though to alter nerve transmission, and cause localised inflammation. As a result of this there is also ***build up of tau protein tangles*** within cells, which leads to apoptosis of affected cells. Atrophy of cortical and subcortical brain tissue occurs. Loss of volume of the brain. Often replaced by enlarged ventricles.
- **Vascular** – small cardiovascular events (effectively small strokes) cause death of brain tissue

I

- There is no definitive way to make a diagnosis – without a brain biopsy from autopsy!
- DSM – IV criteria for diagnosis suggest **"clear evidence of decline in memory"** (e.g. MOCA or MMSE), PLUS one of:
- Reduced language ability – reduced motor ability – reduced recognition – reduced executive function (planning)
- Investigations are mainly aimed at ruling out reversible causes
  - **CT Brain** – *may show generalised atrophy. Rules out tumours*
  - **TFTs** –*hypothyroidism*
  - **FBC** –*anaemia*
  - **U+E** – *chronic renal failure*
  - **LFTs** – *carcinoma, cirrhosis, encephalopathy*

T

- There is no cure. Some medications may help to slow progress in mild to moderate cases, but the evidence that they actually work is not very strong. They are NOT effective in severe dementia – don't prescribe if MMSE <12
- **Anticholinesterase inhibitors** such as *donepezil, galantamine, rivastigmine* may be trialled. Expected benefit – in 40% of patients they delay they decline of cognitive impairment by 3-6 months. Stop treatment if patient doesn't respond.
- **NMDA receptor antagonists** such as *memantine* may be used in conjunction with above. Are less effective.
- **Antipsychotic medications** – such as haloperidol or quetiapine are often used at night to control anxiety and agitation
- **Minimise risk factors for progression** – control hypertension, encourage regular exercise, control cholesterol, maintain healthy weight, safe alcohol consumption, promote mentally stimulating activities
- **Vascular dementia** – medication less likely to be effective but is sometimes trialled. Control cardiovascular risk factors – BP, weight cholesterol, smoking, regular exercise, Mediterranean diet

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Psoriasis

A

- Affects up to 4% of the population
- About 10-15% of cases are associated with psoriatic arthritis
- Genetic factors are important – 1/3 of patients have an affected relative

- Thought to occur in genetic susceptible individuals due to environmental triggers, such as; obesity, smoking, alcohol intake, sun exposure, and in the case of guttate psoriasis – recent streptococcal infection

S&S

- ***ITCHY!***
- **Classical psoriasis**
  - Affects the extensor surfaces – *front of knees and back of elbows*
  - May also affect the scalp, and in severe cases the torso (rare)
  - Plaques typically >3cm. Erythematous, may be raised, with a silver coloured scale
  - Nail changes – pitting, onycholysis, ridges
- ***Guttate Psoriasis***
  - *Typically occurs after streptococcal infection*
  - *Mainly affects the torso*
  - *Lesions <3cm diameter*
  - *Typically resolves in a few months, but chronic cases can be resistant to treatment*
- Other types include ***flexoral*** and ***palmopustular***

P

- Scaly thickened plaques on the skin
- Autoimmune disorder, often associated with other auto-immune disorders, such as:
  - Psoriatic arthritis, inflammatory bowel disease, Uveitis, coeliac disease, metabolic syndrome

I

- Diagnosis is clinical, typically no investigations are required
- Biopsies not usually required
- Histologically in the skin there is proliferation of keratinocytes, increased T-cells found in the dermis and thickened epidermis

T

3 factors involved in treatment – minimisation of risk factors, emollients (+/- tar) and steroids +/- vitamin D analogues).

- ***Minimise risk factors***– reduce alcohol intake, healthy BMI (18.5-25), avoidance of sun exposure, managing mental health
- ***Emollients*** are skin moisturisers. Creams (water based), lotions (oil + water) and ointments (oil based) are available, ointments are the most effective. Use liberally, 3-4x per day during flare-ups, less often may be effective between flares. Most patients under-dose. Tar is often added in psoriasis (but not in eczema) especially if the scalp is involved
- ***Steroids*** should be used **early** to control flare-ups. Typically used OD or BD. They are classed as ***mild, moderate, potent and very potent.*** Avoid the use of potent and above on the face. More effective when combined with vitamin D analogues such as ***dalvobet*** or ***Enstillar foam.*** Combination agents also help to improve compliance.
- In resistant cases, patients needs specialist dermatology referral. Immunomodulating drugs, such as methotrexate, ciclosporin, azathioprine or newer agents such as infliximab may be used. Phototherapy (UVB, PUVA) can also be effective

- A – Aetiology (& Epidemiology)
- S+S – Signs & Symptoms
- P – Pathology
- I – Investigations
- T - Treatment

Type 2 diabetes

A

- A huge and increasing problem. Largely preventable.
- Occurs in genetically susceptible individual as a result of lifestyle factors – obesity, underactivity and poor diet
- Affects 5% of population. Estimated to double in 10 years
- Other risk factors include HTN, smoking, alcohol,, increasing age, family history, Asian ethnicity
- Causes ↑risk CVD, peripheral neuropathy, retinopathy (+/- blindness) and kidney disease (nephropathy)

S&S

- May be asymptomatic and discovered on screening
- **Acute presentation:** polyuria, thirst, weight loss, and in severe cases – ketoacidosis
- **Subacute presentation:** lethargy, polyuria, frequent infections (e.g. candida, staphylococcus, UTI), and in advanced cases – onset of the complications – such as visual disturbance and peripheral neuropathy

P

- Insulin is produced by the islet ells of the pancreas. It acts on peripheral tissue to allow glucose to be taken up into cells.
- Glucose is the main fuel source used by most of the peripheral tissues in the body.
- Initially there is ***peripheral insulin resistance*** – particularly in muscle cells. As a result, the pancreas produces more insulin to overcome the resistance.. Also, the liver – probably via gluconeogenesis – produces more glucose – as a response to low ***intracellular*** glucose levels– and blood glucose levels may rise. At this point, insulin resistance is reversible with lifestyle modification and medication. However, eventually, pancreatic islet cells undergo apoptosis, and insulin levels fall. At this point, insulin injections are required.

I

Can be diagnosed by one of three methods:

- HbA1c >6.5% on two or more occasions
- Fasting glucose >7.0mmol/L
- Glucose >11mmol/L after an oral glucose tolerance test (OGTT)

Screening:

- Recommended for everyone over 40
- Involves the use of a T2DM screening tool (e.g. Diabetes risk assessment tool or AUSDRISK)
- If high risk – perform HbA1c every 1-3 years

T

- Try to keep it simple. Can get very complicated. The basic principle:
  - Lifestyle modification
  - Lifestyle + metformin
  - Lifestyle + metformin + sulphonylurea (or other second line drug) **<< start here if HbA1c >8.5% at diagnosis**
  - Lifestyle + metformin + second line drug + insulin (+/- third line drug – usually GLP1-A)
- Lifestyle modifications are:
  - **Weight:** Aim for BMI 18.5 – 24.9, AND waist circumference <80cm (f) and <94cm (m)
  - **Exercise:** at least 30mins on 5 days a week of 'moderate intensity' (brisk walk or similar) exercise
  - **Diet:** low sugar, complex carbohydrates, minimal saturated fat, low GI, calorie controlled
- Ongoing management
  - Annual review: GP, podiatrist (for peripheral neuropathy), optometrist (for retinopathy), HbA1c, U+Es and urine ACR (for nephropathy), diet assessment, BP, weight and BMI

almostadoctor

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Scabies

A

- Caused by mite sarcotobies scabei
- A widespread problem in the developing world
- Also seen commonly in general practice is developed world
- Usually cases present together – e.g. in families
- Spread by direct skin-to-skin contact – including sexual transmission. Rare to spread from inanimate objects
- Remains indefinitely if not treated

S&S

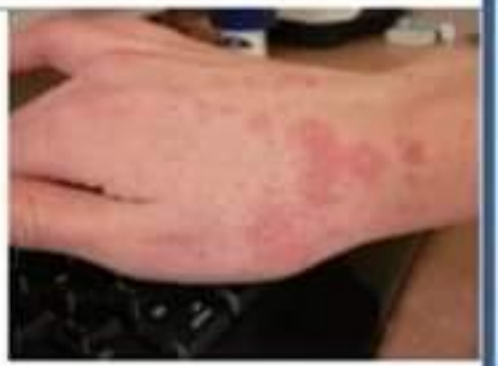
- VERY itchy red rash
- Classically on the hands – especially in the webbed spaces – and also in the groin
- Small "burrows" may be seen
- Can be secondarily infected – e.g. with *staphylococcus*
- **Norwegian ("Crusted") scabies** – a severe variation often associated with immunosuppression. Rash can affect the whole body (usually the face is spared)

P

- Mite burrow under the skin
- Life cycle is about 10-15 days from eggs to mature mites
- Mites mates on the skin, and then burrow back into the skin
- A typical infection usually only involves about 10 mites

I

- Usually a clinical diagnosis based on the location and appearance of the rash
  - Look particularly for the "burrows"
  - Be ware of secondary infection
- Crusted scabies is much more severe and often there are large ***hyperkeratotic*** areas that resemble psoriasis
- Differentials include:
  - Insect bites, pompholyx (a type of eczema), other types of dermatitis – e.g. contact dermatitis, psoriasis, folliculitis, lichen planus



T

- Topical agents are the treatment of choice. Most commonly – ***Permethrin 5% cream.*** Apply from the neck down – particularly thoroughly on the hands and in the groin
  - Consider using a brush to apply underneath the nails
  - Leave on overnight and wash off in the morning
  - Repeat every 7 days until infection resolved – usually only needs 1-2 treatments
  - Children can return the school the day after the first treatment
- Important to prevent re-infection – ***wash and or tumble dry all clothes and bedsheets on a high temp (>50°C)***
- ***Treating the itch*** (whilst waiting or inflammation to resolve) – anti-histamines, emollient cream (keep in the fridge), and in severe cases may also consider a topical steroid

Sudden Infant Death Syndrome

A

- Triple Risk Model – High Risk Infant + Environmental factors + Physiological Changes
- Risk Factors = Age (1-6m)
  - Preterm
  - Boys>Girls
  - Multiple Births
  - Family History
  - Maternal age (↑ if mum <20)
  - Low socioeconomic group
  - Single Parents
  - Smoking in household
  - Co-sleeping
  - Winter
  - Baby sleeps on front or side
  - Illness
  - Overheating

S+S

- Unfortunately only

"The sudden and unexpected death of a child under 1 for which no adequate cause is found despite thorough post mortem and case report"

P

- n/a

I

- Thorough history
- Autopsy is a legal requirement, despite how distressing it must be for the family.
- Do bloods, LP, Urine and stool cultures
- Samples of infants clothing/bedding might be tested.

T

- **PREVENT!! BACK TO SLEEP CAMPAIGN!**
- Document everything
- Notify relevant people
- Do all investigations
- Reassure family its no ones fault
- Be aware of Non-accidental injury
- Advice about the grieving process
- If wanted, suppress lactation (**Cabergoline**)
- Put family on Care of next infant scheme (CONI)