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Resource view

Resource name	Anatomy
Resource description	ANATOMY
Resource content	

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EPIDEMIOLOGY AND ANATOMY LECTURE NOTES 2013.

BLOOD SUPLY OF THE UPPER LIMB

1. On the right side:

Aorta-----brachiocephalic truncus-----subclavian artery ---> Axillary artery ---> Brachial artery---> radial and ulnar artery---> which form the palmer arch------> digital arteries

NB: The brachiocephalis truncus gives branch to common carotid artery and the subclavin artery gives branch to vertebral artery

2. On the left side direct from the aorta branches off common carotid artery and subclavian artery.

2. VENOUS SYSTEM

A. Deep veins

B. Superficial veins

DEEP VENOUS BLOOD FLOW

Palmer metacarpal --->deep palmer veins---->Radial and ulnar veins--------
Brachial vein------->Axillary vein---subclavian------->brachiocephalic

SUPERFICIAL VEINS

1. BASILIC VEIN
2. CEPHALIC VEIN

Both of which drain into brachial vein -------> subclavian vein
------→brachiocephalic ------Superior vena cava

SPECIAL NERVES:

1. **FACIAL NERVE**

Innervates the muscles of expression and anterior 2/3 of taste and oral cavity.

Branches:

1. Greater petrosal nerve : innervates the nasal glands, palates, lacrimal glands. Supplies the stapedius, chorda tympani (submandibular gland, sublingual gland and taste of the tongue.
2. Posterior auricular nerve: muscles around the ear
3. Temporal branch of facial nerve:
4. Zygomatic branch of facial nerve
5. Marginal branch of mental nerve
6. of facial nerve
7. Cervical branch of facial nerve

2. TRIGEMINAL NERVE

A. OPTHALMIC BRANCH comes out through superior orbital fissure.

-**Frontal nerve:** Innervates the scalp, forehead, upper eye lid, conjunctiva and cornea

-Nose (including the tip f the nose)

-Frontal sinuses

B. MAXILLARY BRANCH-comes out from the skull through foramen rotundum

- Infraorbital nerve -Innervetes the cheek, upper lip, upper teeth and gums.

-nasal mucosa, palate and roof of the pharynx

-the maxillary, ethmoid and sphenoid sinuses and part of the meningitis

C. MANDIBULAR BRANCH

-Comes out from foramen foramen ovale

-Innervetes the lower lip, lower teeth and gums.

-chin and jaw(but not the angle of jaw which is supplied by the C2-C3)

-

Branches:

i) **Lingual nerve** supplies the sensation of the anterior 23 of the tongue

-Inferior alveolar nerve supplies

ii) Auriculotemporal nerve=is commonly gets injured during the tempomandibular surgery leading to loss of sensation on the auricule and skin surrounding ear

iii) Buccal nerve=supplies the mucus membranes of the buccal i. e inside

iv) Mental nerve: supplies the chin and lower lip (mucus membrane) it is the branch of inferior alveolar which itself is branch of mandibular branch of trigeminal.

Specific nerve damage:

1. T4 is level of nipples
2. T10 is umbilicus
3. C4 over acromioclavicular joint
4. Diaphragmatic nerve usually irritated in peritonitis causing shoulder tip pain. Also known as phrenic nerve.
5. Claw hand is a sign if ulnar nerve damage. Usually the little finger and ring finger are affected
6. Wrist drop is a sign of radial nerve damage.
7. **Carpal tunnel syndrome** is a sign of median nerve damage and compression is at the level of the wrist. Phalen's test or tinnel test can be used to make the diagnosis of carpal tunnel syndrome.

   The nerve is compressed in the carpal tunnel and treatment can be conservative or surgical (by Incision of the flexor retinaculum)
8. Foot drop can be caused by both peroneal nerve and sciatica nerve.
9. Sciatica is a term usually used to describe the lower back pain radiating all the way down the led up to knee or below it.

ORTHOPAEDIC ANATOMY

Radial nerve→ innervates
All extensors of hand I.e. extension of wrist, fingers, elbows
Therefore radial nerve palsy causes wrist drop

Ulnar n nerve→
Innervates all intrinsic hand muscles, except the LOAF which are innervates by the median nerves.
Therefore ulnar n palsy if claw hand

Median nerve → LOAF
L- The 2 Lateral lubricals
O- Opponens pollicis
A- Abductor pollicis brevis
F- Flexor pollicis brevis

1. **Shoulder abduction:**
- Deltoid
- Axillary
- C5

1. **Elbow flexion:**
- Biceps
- Musculocutaneous
- C5, C6

1. **Elbow extension:**
- Triceps
- Radial
- C7

1. **Finger Extension:**
- Extensor Digitorium superfacialis & profundus
- Radial
- C7

1. **Finger flexion:**
- Flexer digitorium profundus & superficialis
- Median & Ulnar
- C8

1. **Finger abduction :**
- First dorsal interosseous
- Ulnar
- T1
-

1. **Thumb abductor:**
- Abductor Pollicis Brevis
- Median
- T1

1. **Finger Adduction:**
- Second Palmar interossei
- Ulnar
- T1

Shoulder Muscles:

- Pulling arm backward while the hands is on waist & move elbow backwards on resistance

Rumboid muscle

Serratus Anterior muscles:
- Imagine you are pushing the a car. In this position you are using the serratus anerior muscle

1. Supraspintus:
- Suprascpular nerve.
- Lifting arms sideways between 60 and 120 degrees.

1. Infraspinatus muscle:
- With a flexed elbow, move the arm inwards

1. Long flexors of little and finger ring:
   Flexion of distal IPJ is flexor digitorum Profundus 3 & 4
   DIP → Profundus
   PIP → Suprficialis

2. Flexor pollicis loungus:
- Flexes thumb

Hip flexion **Hip Extension**
L1, L2; Iliopsoas S1; Gluteus Muscle
; Sacral plexus ; Inferior gluteal nerve

Knee extension
L3, L4;
Femoral nerve
Quadriceps muscle

Dorsiflexion foot
L5;
Tibialis antetrior muscle
;Deep Peropneal

Knee Flexion:
- Hamstring muscle
- Sciatic nerve (foot drop)
- L5, S1
-

Plantar flexion of the foot:
- Grastrocinemeous muscle
- Posterior tibialis
- S1

Reflexes
1. Supinator→ Radial nerve , C6
2. Triceps → Radial nerve, C7, Biceps – C5 Musculocutaneous nerve
3. Finger → Median, Ulnar nerves
4. Knee → Femoral, L3, L4
5. Ankle → S1, S2

DERMATOME FOR LOWER LIMB:

L1 → Pocket

L2 → Inner thigh
L3 → Knee
L4 → Medial malleoli
L5 → Lateral Dorsum of the foot
S1 → Sole
S5 → Saddle

Upper limb

L4 Shoulder
L5 arm
6 thumb
7 Middle finger
8 Little finger

LYMPH NODES DRAINAGE

1. The cervix lymph drains into the para-aortic lymph nodes
2. Vulva lymph drains into superficial Inguinal lymph nodes ----->then into deep inguinal lymph nodes
3. Lower nodes then into deep inguinal lymph nodes
4. Body of the uterus drains into external ilia lymph nodes
5. Fundus of the uterus drains into para-aortic lymph nodes
6. Ovaries drain into para-aortic lymph nodes
7. Superior half of the rectum drains into pararectal lymph nodes --->then into inferior mesenteric lymph nodes
8. Lower half of the rectum drains into internal iliac and sacral group of LN.
9. Testes drain into paraaortic lymph nodes
10. Superficial Inguinal Lymph nodes drains fro penis, scrotum, perineum, buttock, vulva and abdominal wall below the umbilicus.

11. Usually the **superficial lymph nodes** drain into **deep inguinal** then into **external iliac** and then into **para-aortic**

12. Ovaries drain into para-aortic lymph nodes

13. Prostate drains into into external iliac

14. **EXTERNAL ILIAC LYMPH NODES:** drains from the glans of the penis, prostate, upper vagina, fundus of the bladder.

15. **INTERNAL ILIAC NODES**: drains from deeper perineum, urethra, buttock and back of the thigh.

16. **PARAAORTIC LYMPH NODES** drains from ovaries, testes and superior rectum

17. **SUPERFICIAL CERVICAL LYMPH NODES:** nodes: lower part of auricular and parotid region.

18. **ANTERIOR CERVICAL LYMPH NODES:** lower part of the larynx, thyroid gland and upper part of the trachea.

LYMPH NODES OF THE FACE

SUBMENTAL LYMPH NODES: drains from the floor of the mouth, apex of the tongue and lower lip then goes to deep cervical lymph nodes..

LYMPHATIC VESSELS FO THE TONGUE

-Apical of the tongue or tip =submental

-Lateral margin of the tongue= submaxilary lymph nodes

-basal of the tongue =superior deep cervical LN

SUBMAXILARY OR SUBMANDIBULAR LYMPH NODES= nasal cavity and gums, cheek, upper lip, lateral part of the lip, medial palpabrae commissure, lateral part of the lower lip.

SUBMENTAL LYMP NODES: lower lip and floor of the mouth and apex of the tongue.

BREAST: mainly drain into axillary lymph nodes

TERMINOLOGIES IN EPIDEMIOLOGY

1. **INCIDENCE:** Is the number of new cases divided by the total population per year who are at risk of becoming a case
2. **Prevalence:** The proportion of people in a given population at a given point/time who had a disease
3. **Mode**: is the value that occurs most frequent
4. **Median** : is the middle value when the values are ranked.
5. **Sensitivity**: Is the proportion of true positives correctly identified by a test.
6. **Specificity:** Is the proportion of the true negatives correctly identified by the tests.

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Cardiology lecture notes

Chest Pain:

1. MI (ST elevation-STEMI)
2. Stable Angina
3. Pneumonia
4. PE
5. Pneumothorax
6. Aortic dissection
7. GERD
8. Musculoskeletal pain
9. Pericarditis
10. Acute coronary syndrome
11. Unstable Angina

Acute coronary syndrome (ACS) consists of: -

- STEMI
- NSTEMI
- Unstable Angina

Unstable angina is angina which is occurs at rest or it has increased in frequency or duration or it is occurring with less effort than it used to happen before. It is therefore difficult to differentiate from Myocardial infarction.

NSTEMI stands for Non ST elevation myocardial infarction. This simply means that one can have myocardial infarction without ST segment elevation on an ECG i.e. normal ECG or ST depression. It is therefore difficult to differentiate from unstable angina.

STEMI stands for ST elevation myocardial infarction. This simply means that the patient will have elevation of ST segment on an ECG. Hence before you do an ECG, this could still be difficult to differentiate from unstable angina and NSTEMI.

NB: It is difficult to differentiate between unstable angina and NSTEMI using an ECG because in both cases ECG can be normal or show ST segment or T wave abnormalities may be seen, therefore you need to do **cardiac enzymes** to differentiate them.

The terminology **Acute Coronary Syndrome (ACS)** is used to describe the way these 3 conditions present. So before you can differentiate between these 3 conditions, this is the way to treat them.

TREATMENT OF ACS

1.O2

2.GTN

3.Aspirin 300mg

4. Clopidogrel 300mg

5.Morphine and Metoclopramide

6.LMWH(Enoxaparin and Dalteparin)

7.Beta-blockers – as anti angina

MYOCARDIAL INFARCTION

1. NSTEMI (NON ST ELEVATION MYOCARDIAL INFARCTION)

- Sudden onset central, crushing chest pain radiating to throat/ left arm
- Lasting > 20 min
- Associated with nausea & vomiting, sweating in the palms

ECG: ST depression or T wave inversion ,Troponin is raised.

If no changes are seen on ECG but the cardiac enzymes are raised then it is NSTEMI

We need cardiac enzymes to make the diagnosis

Symptoms

I. Central chest pain, crushing in nature, radiating to Left arm lasting greater than 20 min
II. Complaining of nausea, vomiting, sweating
III. Silent MI or a typical chest pain usually in diabetic patient.
IV. May lead to LVF leading to Pul.oedema, which presents with Tachycardia, low BP and SOB.

2. STEMI (ST ELEVATION MYOCARDIAL INFARCTION)

- Sudden onset of chest pain which is central, crushing, retrosternal
- Radiating to left arm or neck
- Associated with nausea, vomiting, sweating
- ECG: ST elevation or new LBBB
- Cardiac enzymes are raised
- when there is STEMI cardiac enzymes are not needed to make the diagnosis. **Diagnosis is from the ECG**

CLASSIFICATION OF ST ELEVATION MYOCARDIAL INFARCTION

I. ST elevation in lead II, III and AVF - indicates inferior MI
II. ST elevation in V_1-V_2 - indicates septal MI
III. ST elevation in V_3-V_4 - indicates ant. MI
IV. ST elevation in I, aVL, V_5, V_6 - indicates Lateral MI

Other ECG changes involve, T wave inversion & Q waves-always MI

CXR may show Pulmonary Oedema in the form of bilateral fluffy opacities or it may show cardiomegaly or it may be normal

MANAGEMENT OF STEMI OR SIMPLY MYOCARDIAL INFARCTION

1. O_2

2. Aspirin 300 mg then 75 mg everyday
3. Morphine iv and metoclopromide (anti sickness/ nausea)
4. GTN sublingual
5. Percutaneous coronary intervention (angioplasty) or Thrombolysis if PCA is not available

When the patient Is stable

1. Continue to give ACE- I even with normal BP
2. Beta- BLOCKERS(anti angina)

Indication for thrombolysis

1. Chest pain <12 hrs from onset
2. ST elevation in limb leads ≥ 1mm in 2 or more contiguous leads
3. ST elevation in chest leads ≥ 2mm in or 2 more contiguous leads
4. New LBBB

Contra indications for thrombolysis

1. Active peptic ulcer
2. Aortic dissection
3. Brain tumour
4. Recent head injury
5. Recent haemorrhagic stroke
6. Acute pancreatitis
7. Active lung cavitation
8. Recent Surgery or trauma
9. Internal PV bleed
10. Oesophageal varices
11. Previous allergy
12. Pregnancy or < 18 weeks post natal

Drug of choice for thrombolysis is Streptokinase. **Do not repeat within 4 days of 1st dose.**

- If there is history of previous use of streptokinase, then use TPA (Alteplase or tenecteplase)-tissue plasminogen activator

Complications of MI

1. Cardiac arrest/ cardiogenic shock will present with increased HR, decreased BP
2. Bradycardia: - (i) sinus bradycardia is treated with Rx with atropine or if there is no response to atroping, then do cardiac pacing.
    1. First degree heart block→ prolonged PR interval
    2. Second degree heart block , of which there are 2 types:

    a) Morbitz 1 (Wenckebach): PR interval is not fixed, the PR interval becomes progressively prolonged until a P wave is not transmitted and there is a missed QRS.

    b) Morbitz II : has frequent missing QRS complexes and requires cardiac pacing. Eg. 3:1. This can be regular or irregular. It requires cardiac pacing.

    3. Complete heart block requires temporary pacing - if not resolving then a permanent pacemaker should be inserted.

    4. Bundle branch block. This can either be LBBB or RBBB.

3. VT (ventricular tachycardia)

- Palpitations or arrythmias within 48 hrs post MI is always VT until proven otherwise

4. Left Ventricular Failure

- Shortness of breath
- Increased HR, decreased BP
- Pulmonary oedema may lead to haemoptysis
- Treatment:IV furosemide

5.Pericarditis→ Chest pain relieved by sitting forward, saddle shaped widespread ST elevation, pleuritic chest pain with pericardial rub, with or without fever

Treatment: NSAIDS

6.DVT/PE due to immobility - Please see pulmonary embolism.

7.Cardiac tamponade Triad of (i) muffled heart sound

(ii) decreased BP

(iii) High heart rate

(iv) Engorged neck veins i.e raised JVP

(v)chest x-ray shows globular heart

Investigation - echocardiogram

Treament: - Pericardiocentesis

8.Mitral regurgitation

- Due to papillary muscle rupture or chorda rupture secondary to ischemia
- Presents with Pul. Oedema due to LVF
- Mitral regurgitation - pansystolic murmur at apex radiating to axilla

9.Ventricular septal defect

- Harsh Pan systolic murmurs-left sternal edge
- Increased JVP
- Cardiac failure

Investigation: - echocardiogram

Treatment: - Surgery

10.Dressler's Syndrome

- Recurrent Pericarditis, pleural effusion, fever, anaemia
- ESR high
- 1 – 3 weeks post MI

11.Left Ventricular Aneurysm : occurs 4 – 6 weeks post MI

- Presents with LVF , recurrent VT
- Persistent ST elevation 4-6 weeks
- Systemic emboli

3. Unstable Angina

- Angina at rest or angina with increased frequency severity or duration
- Associated with sweating and nausea.
- ECG: shows ST depression or T wave inversion
- Cardiac enzymes are normal

4.Stable Angina

- Exertional pain (pain on exercise when you walk a certain distance eg. 100 meters)
- Radiates to the left arm lasting < 20mins
- Transient ECG changes (usually ST depression or T-wave inversion)
- Cardiac enzymes are normal

Investigation:

- +ve exercise tolerance test (exercise ECG +ve)
- +ve coronary angiography (arteriosclerosis)

5. **Pericarditis**

- History of viral like illness (URTI) or coryza symptoms(running nose,cough,sneezing)
- Pleuritic chest pain i.e. chest pain on inspiration / gets worse on inspiration.
- Chest pain worse on lying flat, but relieved on leaning forward.
- ECG: Saddle shaped ST elevation in all leads (widespread)
- On examination: Pericardial rub
- Treatment: NSAIDS

6. Dissecting thoracic aneurysm

- Sudden onset of central tearing chest pain radiating to the back
- With or without history of trauma
- Sometimes pain radiates between the inter scapular region
- patient is in shock (increased HR, low BP)
- Different pulses & BP in each arm
- History of intermittent claudication and hypertension
- The cause of aneurysm is arteriosclerosis
- Chest X-Ray: shows widened mediastinum
- HYPERTENSION causes atherosclerosis and this causes aneurysm
- Investigation:CT scan, USS, Transesophegeal echocardiogram
- Treatment: surgery

7. Pulmonary Embolism (PE)

- Patient complains of shortness of breath or sudden onset of pleuritic chest pain (worse on inspiration)
- Usually in a young female
- May have history of OCP use, long flight, pelvic surgery e.g hip replacement or hysterectomy
- Possible signs of DVT i.e, swollen legs, easy tenderness or erythema
- ECG:

1. T wave inversion (V_1- V_4)
2. RBBB($S_1Q_{111}T_{111}$ syndrome) with Deep S wave, Pathological Q wave, Inverted T wave
3. Sinus tachycardia
4. Atrial fibrillation

8. Pneumothorax

- Young, thin, tall male patients
- Also common in patients with COPD
- Sudden onset of pleuritic chest pain
- Commonly starting on exercise(playing football or riding bicycle)
- If trachea is shifted it is Tension Pneumothorax
- If trachea is shifted to the left, the pneumothorax is on the right and vice versa
- If the trachea is not shifted or it is central the it is only Simple pneumothorax
- Always hyper- resonance on percussion on the same side of Pneumothorax but opposite side of trachea shift

9. Pneumonia

- Fever, cough, sputum, pleuritic chest pain
- Chest X-Ray: shows consolidation
- Dullness to percussion, decreased air entry, crackles on auscultation unilateral.

10. Musculoskeletal pain

- Often follows strenuous exercise or lifting things or being in the gym
- Chest X-Ray, ECG, cardiac enzymes normal .
- Tenderness on the chest wall on palpation.

11. GERD

- History of indigestion, hiatus hernia or reflux symptoms
- Retrosternal chest pain, burning in nature
- Worst at night or when lying flat
- Chest pain relieved with antacids
- Sour taste in the mouth

PALPITATIONS

CAUSES

1. AF-atrial fibrillation
2. SVT - supraventricular tachycardia
3. VT –ventricular tachycardia
4. VF - ventricular fibrillation
5. Anxiety

6. Pheochromocytoma
7. Atrial flutters
8. Ectopic beats-usually ventricular ectopic beats.
9. Atrial myxoma

SUPRAVENTRICULAR TACHYCARDIA = This is any tachyarrhythmia originating above the ventricles. It can either be

a. **Regular or**
b. **Irregular**

1. Causes of Regular supra ventricular tachycardia

- Sinus tachycardia
- Atrial flutter - suspect this if the heart rate-is 15O
- AVRT (i.e. with accessory pathway e.g. WPW)-atrial ventricular re-entrant tachycardia(Wolf Parkinson White)
- AVN RT-atrial ventricular node re-entrant tachycardia
- Intra-Atrial re-entry tachycardia

NB: if you see ECG with heart rate 150 think of atrial flutter.

2. Irregular supra ventricular tachycardia is usually atrial fibrillation

ATRIAL FIBRILLATION (AF): presents as palpitations(300-400)

On examination: - irregularly irregular pulse

ECG - shows no P waves and irregular QRS complexes

Classification: acute, chronic and paroxysmal.

Management of AF:

1. *Chronic AF*

Rate control drugs

(i) Beta- blocker (metoprolol)
(ii) Calcium channel antagonist (diltiazem/ verapamil)
(iii) Digoxin

NB: If there is AF + heart failure the drug of choice- Digoxin

The mainstay of treatment in AF is rate control and not cardioversion.

AF needs anticoagulants

a. Warfarin (maintain INR 2-3)-to prevent embolism
b. Aspirin if <65 yrs of age and no HTN, no DM, no LV dysfunction, no valvular heart disease, no MI/TIA.

NB: use the CHAD 2 score to decide whether patient needs to be on aspirin or warfarin.

2.*Paroxysmal AF*: Flecanide (pill in the pocket),sotalol. Anticoagulation is also needed

The appropriate investigation for paroxysmal AF is 24 hour ECG (ambulatory ECG)

3. Acute AF-less than or equal to 48 hrs duration

- If very ill or haemodynamically unstable do electrical cardioversion

Rate control medication for Acute AF:

1) calcium channel blocker (verapamil & diltiazem)

2)beta blocker metoprolol 3) Digoxin

Arrhythmias

There are 2 types of arrhythmias:

a. Tachy-arrhythmias (HR > 100/min)
b. Brady-arrhythmias (HR < 60/min)

Management of tachy-arrhythmias:

First look for **signs of instability** which are

- Reduced consciousness
- Systolic BP < 90 mmHg
- Chest pain (for VT only)
- Heart failure
- Heart rate > 150

1) If **signs of instability are present** then give DC shock and if DC shock not helping then give IV Amiodarone.

NB. DC shock is the same as electrical cardioversion. Amiodarone is used for chemical cardioversion.

2) If patient with tachycardia is **stable** then check if it is broad complex tachycardia (eg VT or VF) or narrow complex tachycardia.

- **Narrow complex tachycardia** (QRS < 0.12 secs on ECG)
- If narrow complex tachycardia and **irregular** then most likely it is AF.
- If narrow complex tachycardia and **regular** then it can be sinus tachycardia, Atrial flutter, WPW syndrome or atrial tachycardia. In this case perform vasovagal manoeuvre or carotid massage, if that does not help then give Adenosine.
- If vasovagal manoeuvre/carotid massage or adenosine terminates the arrhythmia then it is AVRT (Wolf Parkinson White syndrome).
- If vasaval manoeuvre and adenosine do not terminate the arrhythmia but just slows the rate down, then it is likely to be atrial flutter, atrial tachycardia or atrial fibrillation.
- **SVT (Supra-ventricular tachycardia)** is terminology which suggests any arrhythmia arising above the ventricles.
- It is usually difficult to differentiate between AF, Atrial flutter, Atrial tachycardia and WPW syndrome. That is why we perform vasovagal manoeuvre/carotid massage or give adenosine to slow down the heart rate so that we can read and interpret the ECG properly. If it terminates with above methods then it is WPW.
- **Broad complex tachycardia** (QRS > 0.12 secs on ECG)
- It can also be either regular or irregular.
- If it is **irregular** then it is either **VF (Ventricular fibrillation) or torsades de pointe**. Give IV Magnesium (**Mg 2+**) for torsades de pointe and **DC shock for VF**.
- Ventricular fibrillation usually does not present with palpitations. It presents with collapse.
- If it is **regular** then it is more likely to be **VT (Ventricular tachycardia)**. Treat patient with **amiodarone** unless patient is unstable in which case he needs DC shock. Initial dose for amiodarone is 300 mg.

NB: If patient with arrhythmias is unstable give DC shock no matter what type of arrhythmia it is, narrow or broad complex tachycardia.

- If there is AVRT which involves accessory pathway

  E.g. **WPW: digoxin is CI** because they block AV node not accessory pathway & make symptoms worse

- ***Management of SVT: -***most of the time they mean WPW.

1. Valsalva manoeuvre (carotid sinus massage)

   No help

2) Give adenosine 6 mg

No help

9 mg 12 mg 18 mg

NB: - 1. Or 2. Terminates AVRT (Atrial ventricular re-entrant tachycardia) and AVNRT (Atrioventricular node re-entrant tachycardia)– will bring it to normal rhythm

-Therefore if arrhythmias is controlled by 1) or 2) it means it's WPW.

1. Or 2. will cause transient block and review whether it is AF, Atrial flutter, Atrial Tachycardia (but it will not terminate these, slow them down)

NB: - Atrial flutters medication is B- blockers i.e. metoprolol treat just like Atrial fibrillation.

- Ventricular Fibrillation does not present with palpitations. It presents with collapse and is incompatible with life.

- A patient with VF is usually unstable.

2. AF: There is irregular pulse on examination

Patient experiences palpitations and this is usually a common complaint.

Symptoms: SOB, chest pain, collapse, palpitations

Causes: -

A. IHD (MI, Angina)
B. Mitral valve disease esp. Mitral Stenosis
C. HTN

D. Heart failure
E. Pneumonia
F. PE
G. Atrial myxoma - tumor of heart muscle
H. Thyrotoxicosis
I. Alcohol
J. Cardiomyopathy

3. Ectopic beats – usually ventricular ectopics

Causes: - coffee, alcohol,smoking

Management: - 1. If infrequent ectopics, manage with life style modification e.g. stop smoking,reduce caffeine,reduce alcohol.

NB: Patients usually complain of missing beats or pounding in the chest

4. Atrial flutter

- Commonly secondary to valvular heart disease or IHD
- HR 300 with 2:1 block

NB: if HR= 150 think of Atrial Flutter.

NB: - VF Never presents with palpitations, it presents with collapse

-Patient cannot tolerate VF

-Always haemodynamically unstable

-Cardioversion is the only option

5. Anxiety

- Young female
- Palpitation, sweating, perioral paresthesia, SOB
- CO_2 is low ($PaCO_2$ is decreased) due to hyperventilation
- PaO_2 is normal or high

6. Thyrotoxicosis/ hyperthyroidism

- Presents with AF, tachycardia, sweating, diarrhoea, palpitation
- Wt. Loss despite good appetite, oligomenorrhoea

7. Atrial myxoma

- Benign cardiac tumour
- May present with palpitations which resolve when sitting up but worse when lying flat
- Weight loss, fever, may cause AF

8. Pheochromocytoma

- Catecholamine producing tumours from adrenal glands
- Episodic (on & off) headaches, sweating, tachycardia, hypertension
- Anxiety attacks: - tremor, palpitation

Investigations: - urinary catecholamines (which are adrenaline, Nora- adrenaline and dopamine)

Management of Brady-arrhythmias:

Patients with brady-arrhythmias can also be either stable or unstable.

Signs of instability:

- Systolic BP < 90 mmHg
- HR < 40/min
- Heart failure
- Reduced consciousness

- If patient is unstable give atropine and if atropine not effective then do cardiac pacing.
- If patient is stable then check for the risk factors of asystole (chance that heart will stop at any time) e.g. Mobitz type II block, complete heart block, recent systole or ventricular pause.
- If any of the above risk factors present then give atropine and subsequent cardiac pacing if did not respond to atropine.

TYPES OF ANGINA

I. Stable angina - Angina which comes with certain amount of exercise and relieved by rest. The patient can predict it. The pain usually lasts less than 20 minutes and responds to GTN.

II. Unstable angina

III. Decubitis angina - the patient experiences pain on lying flat

IV. Prinzmetal/ variant angina is due to coronary artery spasm, ST elevation for a short period of time which resolves quickly.

INVESTIGATION OF STABLE ANGINA

- Resting ECG
- If resting ECG is normal then observe, If abnormal or shows blockage then do angiography

MANAGEMENT-

1. Aspirin-LOWER DOSE
2. Beta- blockers e.g- atenolol
3. GTN sub lingual (Nitroglycerine)
4. Calcium channel blockers (amlodipine or diltaizem)
5. Statin if cholesterol> 4 mmol/ L
6. If symptoms not controlled then add Nicorandil (k^+ channel activator)
7. Life style modification-stop smoking, reduce alcohol intake, wt.loss.
8. Prinzmetal Angina: Treat with Calcium channel blocker (1st choice) i.e. amlodipine or diltiazem

Heart Failure:

Types:

1. **Left ventricular failure (LVF)**
2. **Right ventricular failure (RVF)**
3. **Congestive heart failure (CHF) - a combination of both left and right ventricular failure**

Left ventricular failure (LVF)

- Dyspnoea
- Orthopneoea
  - PND (paroxysmal nocturnal dyspnea)
  - Pinky frothy sputum
  - (Pulmonary Oedema)
  - Cardiac wheeze

Right Ventricular Failure R(VF)

- Peripheral oedema
- JVP/ engorged neck veins
- Ascites
- Peripheral edema
- hepatomegaly

LVF+ RVF = CCF (congestive cardiac failure)

Investigation:echocardiogram - to see LV function

Chest X-Ray→ cardiomegaly, bilateral fluffy opacities on the chest x-ray studies indicates pulmonary oedema

Heart failure can also be classified into Acute and Chronic Heart Failure

1. Acute HF

1. Commonly due to Left ventricular failure leading to pulmonary oedema

Treatment of Pulmonary Oedema

1. Sit patient up in bed
2. O_2
3. IV Diamorphine+ metaclopromide

4. IV Furosemide (main Rx)

5. GTN if BP greater than 100

2. Chronic HF

Treatment of chronic HF

1. diuretics → Give loop diuretics e.g. → furosemide

-If not helping add thiazide diuretic (e.g bendroflumethiazide)

NB: spironolactone if k^+ < 3.2mmol k

1. ACE- I eg lisinopril,enalapril esp if there is LV dysfunction

Side Effect: Dry cough

If cough problematic then consider ARBs (Angiotension receptor blocker) e.g. → condesatan or losartan

1. Beta- blockers → e.g carvedilol
2. Spironolactone → if still symptomatic despite all the above Rx.
3. Digoxin→ if AF with HF then add digoxin
4. Isosorbide mononitrate to reduce the preload of the heart (vasodilation)

HYPERTENSION

CAUSES:

1. Essential hypertension-common in 95%, usually elderly patients
2. Coarctation of Aorta - Hypertension, young patient, chronic headache, radiofemoral delay
3. Polycystic kidney disease-bilateral loin pains, haematuria, hypertension, renal failure, family history.
4. Renal artery stenosis- Hypertension, abdominal bruit. ACE-I contraindicated.
5. Pheochromocytoma- episodic headaches, episodic hypertension, flushing palpitations all intermittent
6. Conns Syndrome- Hypertension, low potassium, due to over production of aldosterone, high sodium
7. Cushing Syndrome - Typical features are an obese patient usually central obesity, hypertension, excessive facial hair, abdominal striae

Essential Hypertension

- 95% of all cases of hypertension
- Usually asymptomatic until end organ damage eg hypertensive retinopathy, nephropathy
- When to treat?

Only when BP is greater than 160/100 mmHg

Classification of Essential HTN

	Systolic	Diastolic
Normal	<140	<90
Mild HTN	140-159	90-99
Moderate HTN	160-179	100-109
Severe HTN	≥ 180	≥ 110

NB: - HTN is BP ≥ 140/90 mmHg

Management of hypertension

BP less than 160/90 -Only lifestyle modifications

BP more than 160/100 - lifestyle modification and medication

MEDICATIONS:

A=ACE-i B=B-BLOCKER C=CALCIUM CHANNEL BLOCKER D=DIURETICS

LIFE STYLE: stop smoking, reduce alcohol, weight loss, do exercise.

Age Less than or equal to 55 give following medications:

1.**A** or B

2.A+C

3.A+C+D

4.A+C+D+B

Age greater than 55 or black people (afro-caribbean) give following medications:

1.**C** or D

2.A+C

3.A+C+D

4.A+C+D+B

HEART MURMURS

1.Mitral Stenosis

-Mid diastolic murmur at the apex

-Apex beat is tapping in nature

-Commonly causes AF and pulmonary oedema

2.**Mitral Regurgitation**

-Pansystolic murmur at the apex

-Radiates to the axilla

-Apex beat is displaced

-Also causes LVF and pulmonary oedema

3.Ventricular Septal Defect

-Harsh pansystolic murmur at left sternal edge

-If it's a child it's congenital

-In an adult it usually common after MI

4.Tricuspid regurgitation

-Soft pansystolic murmur at left sternal edge or 4th rib

-Common in intravenous drug abusers

-Causes RVF(high JVP,peripheral edema,enlarged liver)

5.Aortic regurgitation

-Early diastolic murmur at left lower sternal edge

-Collapsing pulse or water hammer pulse or bounding pulse

6.Aortic stenosis

-Ejection systolic murmur in the right second intercostal space

-Radiaties to carotids

-Presents with dizziness or syncope on exercise

-Slow rising pulse

7. Hypertrophic Obstructive Cardiomyopathy (HOCM)

-History of sudden death

-Jerky pulse

-Mid systolic murmur

-Loudest at left sternal edge

-Common in young sports man esp. footballers

8. Patent Ductus Arteriosus

-Machinery like murmur

-Throughout systole and diastole

9.Atrial septal defect

-Congenital abnormalities

-Aystolic murmur in upper left sternal edge

-Usually in young children it causes cyanosis

10.Coarctation of the aorta

-Any palpable femoral pulses

-Hypertension

-Radiofemoral delay

NB:Investigation of choice for all murmurs is echocardiogram.

CONGENITAL HEART DISEASE

CLASSIFICATION

1. Cyanotic
2. Non-cyanotic

CYANOTIC:

1. Transposition of great vessel(aorta and pulmonary trunks)
2. Truncus arteriosus
3. Tetralogy of Fallot-(right ventricular hypertrophy,pulmonary stenosis,VSD,over- riding aorta.chest x-ray shows boot shaped heart)
4. Pulmonary atresia
5. Tricuspid atresia

NON CYANOTIC

1.-ASD (Atrial septal defect)

2.VSD

3.-Patent ductus arteriosus

4.-coarctation of aorta

5.-aortic stenosis

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Resource view

Resource name	Dermatology
Resource description	Dermatology
Resource content	

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DERMATOLOGY LECTURE NOTES

TYPES OF SKIN LESION:

- Ulcers & skin cancers
- Plaques / scales
- Rashes
- Blisters
- Eczema
- Erythema

ULCERS & SKIN MALIGNANCIES :

1. BASAL CELL CARCINOMA (BCC), NASOLABIAL FOLDING . A Pearly white umbilicated ulcers anywhere in the face with rolled edges /inverted edge.

2. SQUAMOUS CELL CARCINOMA

Ulcers with everted edges which bleeds easily on touch on touch found anywhere on the neck or arm.

NB: Any ulcer which is located above the neck is always basal cell carcinoma until proven otherwise.

3. MELANOMA. This is a skin cancer of Black or brown, darkly pigmented mole or ulcers which grows rapidly usually on the shin or back. Its usually located on sun exposed areas.

Investigation: Biopsy of the lesion.

ULCERS & SKIN PRE-MALIGNANCIES

1. Black / brown /darkly pigmented mole or ulcers found under the nail = LENTIGO MALIGNA. It is due to prolonged sun exposure over many years.
2. Ulcer or skin lesion on sun exposed area = SOLAR KERATOSIS
3. Ulcers or skin lesion on the sun exposed area which grows slowly for many years= ACTINIC KERATOSIS
4. Ulcer or skin lesion with a central hardcore / horny plug or thorn = KERATOACATHOMA

GENITAL ULCERS:

1. Single painless ulcer = SYPHYLLiS (CHANCRE)
2. Multiple painful ulcers = HERPES SIMPLEX TYPE 2
3. Painful ulcers + lymphadenopathy with dysuria = HAEMOPHILLUS DUCREYI (CHANCROID)
4. Beefy ulcers with the presence of Donnovan bodies = CALYMMATOBACTERIUM GRANULOMATIS (LYMPHOGRANULOMA VENERUM / DONNOVIAN DISEASE)
5. Genital ulcers with femoral & inguinal lymph node enlargement forming a groove between them called the " GROOVE SIGN" = CHLAMYDIA TRACHOMATIS (GRANULOMA INGUINALE)

ULCERS IN SYSTEMIC DISEASES:

1. Shiny painful ulcer on the leg with decreased hair distribution & tight skin around, usually on the medial malleoli = VENOUS ULCER

1. Yellowish /whitish waxy ulcer induration on the leg which might be painful with high blood sugar levels (diabetes) = NECROBIOSIS LIPOIDICA
2. Greyish ulcer in patients with inflammatory bowel disease (crohn's disease / ulcerative colitis) = PYODERMA GANGRENOSUM
3. Pearly or pink umblicated ulcers with rolled off edges + central depression /punctum + risk of HIV (Iv drug abuser or homosexual) =MOLLUSCUM CONTAGIOSUM

Treated with fluconazole

INVESTIGATION:

For any suspicion of malignancy = BIOPSY

TREATMENT :

Malignancies = EXCISION

PLAQUES /SCALES:

1. **PSORIASIS** =Silvery scales on the extensor surfaces like knees , elbow , head which bleeds on scratching + onyclolysis + psoriatic arthiritis + with presence of family history .

Psoriasis is a common condition where there is inflammation of the skin. It typically develops as patches (plaques) of red, scaly skin. Once you develop psoriasis it tends to come and go throughout life. A flare-up can occur at any time.

Treatment:

Management options for the treatment of psoriasis include:

Topical treatment: Regular emollients to soften the plaques

- First-line therapy which includes traditional topical therapies - e. g corticosteroids, vitamin D analogues, dithranol and tar preparations.
- Second-line therapy which includes phototherapy, broad-band or narrow-band ultraviolet B light.
- Third-line therapy which refers to systemic biological therapies that use molecules designed to block specific molecular steps important in the development of psoriasis, such as the TNF antagonists adalimumab, etanercept and infliximab, and ustekinumab, anti-IL12-23 monoclonal antibody.

2. LICHEN PLANUS = white or purple polygonal lacy or wavy lines or scales or plaques on flexor surface usually on the limbs.

Treatment = steroids

3. LICHEN SCLEROSIS = white or purple polygonal lacy or wavy lines or scales or plaques usually located in the genital area

Treatment = steroids

4. **BOWEN'S DISEASE: This is squamous cell carcinoma in situ. It is a** slow growing scales /plaques on lower leg .It is a pre – malignant condition which may progress into **squamous cell carcinoma**

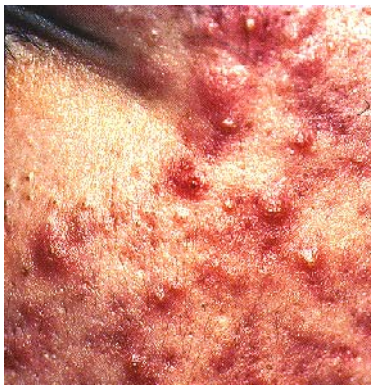
5. **MYCOSIS FUNGOIDES** (SKIN CD4 T – cell LYMPHOMA)= red scaly patches /tumours on trunk /limbs

6. ORAL HAIRY LEUKOPLAKIA (ESTEIN BAR VIRUS) = hairy plaques on oral mucosa of HIV patients

7. KAPOSI'S SARCOMA = multicentric skin nodules / plaques involving the mucus membrane common in HIV patients caused by human hominis virus 8 (HHV8)

RASHES:

1. **ACNE VULGARIS** = pimples , inflamed follicle especially in teenagers forming comedones. It is common in teenagrs but it also be found in condition like cushing disease, polycystic ovarian syndrome e.t.c



Treatment: Salicylic acid 10%

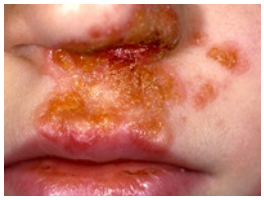
2. **ACNE ROSACEA** (chlamydia pneumonia) = chronic recurring condition causing flushes of face after taking alcohol or spicy food

Treatment = zelaic acid , metronidazole , doxycycline , azithromycin

3. **IMPETIGO** = infection due to staphylococcus aureus , usually on face with honey coloured fluid in a erythematous base (usually around the mouth and nose and common in children)

Impetigo can develop on unaffected skin as well as on the affected skin e.g on top of eczematous skin.

- Children should be kept off school or nursery until there is no more blistering or crusting, or until 48 hours after antibiotic treatment has been started.



Treatment = Flucloxacillin

4. **ERYSIPELAS** = infection due to streptococcus pyogenes , usually on the face associated with high temperature

Treatment = benzyl penicillin + Flucloxacillin (if super infected with staph aureus)

5**. GENITAL WARTS**= Are caused by HUMAN PAPILLOMA VIRUS (HPV) infection. They are usually transmitted during sexual intercourse.

Treatment = wait & see , salicylic acid, cryotherapy

6. **HERPES SIMPLEX VIRUS: CAUSES** gingivostomatitis which is the infection of the mouth causing very painful ulcers which makes it difficulty to eat and may cause dribbling of saliva

Treatment = acyclovir

7. **HERPES ZOSTER (shingles)** = affects dermatomes as it remains dormant on the nerve roots caused by varicella zoster) 2 common presentation is ophthalmic shingles or between the ribs usually starting from the back.

Treatment = acyclovir

Treatment for herpetic neuralgia = Amitriptylin / gabapentin / carbamazepine (anti-epileptic or anti-depressant)

8. PITYRIASIS ROSEA = caused by herpes hominis Virus (HHV 6&7) , this rash is preceded by a "**herald patch**" which is scaly ovoid patch which is larger than the rash that comes later

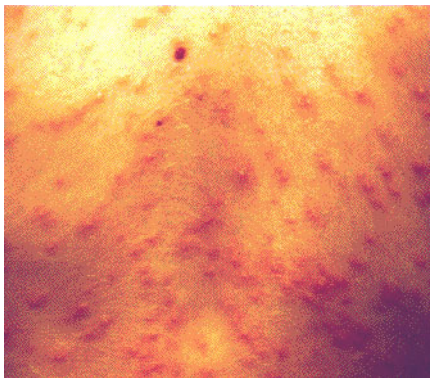
Treatment = self – limiting , oral erythromycin in increased severity

9. PITYRIASIS VERSICOLOR = areas of hyperpigmentation on fair skinned people / areas of hypopigmentation in dark people caused by malassezia furfur which is a fungal infection

Treatment = ketoconazole cream

10. Chicken pox:

- The first feature is often pyrexia - temperature of around 38-39°C is usual for up to four days.
- Headache, malaise and abdominal pain may be reported.
- Crops of vesicles appear over the course of 3-5 days - mostly on the head, neck and trunk and very sparse on the limbs



Treatment: In children between 1 year and 12 years no treatment is required. In adult treat with oral acyclovir. Treatment is also recommended in patient who are immunocompromised.

BLISTERS :

1. **PEMPHIGOID** = Tense thick walled bullae in an **old patient. Bullae** which is difficult to break and there **antibodies to the basement membrane**

Bullous pemphigoid is a skin disease that causes blisters. It mainly affects elderly people aged over 70 years.

The blisters are quite firm and dome-shaped which are difficulty to break

- Tense bullae can form anywhere but commonly around flexural areas. They can appear both on normal and erythematous skin.
- Mucosal involvement is rare and not clinically significant when it does occur

Investigation: 1) Skin biopsy 2) antibodies to basement membrane.

Treatment is usually with steroid creams or oral steroids

2. **PEMPHIGOUS VULGARIS** = thin walled bullae + **young patient** + easily breakable & prone to infections + antibodies to the desmosomal components.

The blisters are flaccid (loose) and fragile so they easily burst.
Patients are usually above 50 years of age.

Investigation: skin biopsy & immunofluorescence

Treatment = Corticosteroids e.g prednisolone

Investigation: skin biopsy & immunofluorescence

ECZEMA :

ATOPIC ECZEMA = Allergic reaction (atopy) which is usually associated with asthma/hay fever

- The skin usually feels dry.
- Some areas of the skin become red and inflamed. The most common areas affected are flexor areas of the elbows ,wrists, knees, and around the neck.

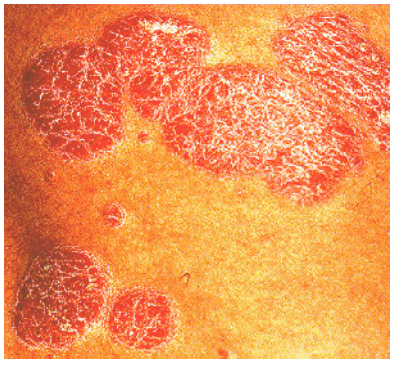
However, any areas of skin may be affected.

- Inflamed skin is itchy. If you scratch a lot it may cause patches of skin to become thickened.
- Sometimes the inflamed areas of skin become blistered and weepy.
- Sometimes inflamed areas of skin become infected.

Treatment = 1) Avoid irritants

2) constant use of emollients, do not stop even if the skin look normal

2)1% hydrocortisone cream first. If skin condition not improving then add 0.05% clobetasone or 0.1% betamethasone cream



NB: When using the emollients and steroid together, apply the emollient first. Wait 10-15 minutes after applying an emollient before applying a topical steroid.

That is, the emollient should be allowed to absorb before a topical steroid is applied (the skin should be moist which means its better to take it after shower. But the skin should not be slippery.)

Characteristics of infected eczema include weeping blisters, infected skin lumps (pustules), crusts, failure to respond to normal treatment, and rapidly worsening eczema.

IRRITANT DERMATITIS = when comes in contact with irritant like soap , detergent etc. + hands are usually involved + affected area becomes red ± weeping + dry fissuring

Treatment = avoiding irritants , using gloves

ALLERGIC CONTACT DERMATITIS = allergic reaction to metals ,latex etc

Investigation = patch test

Treatment = irritant avoidance , topical steroids

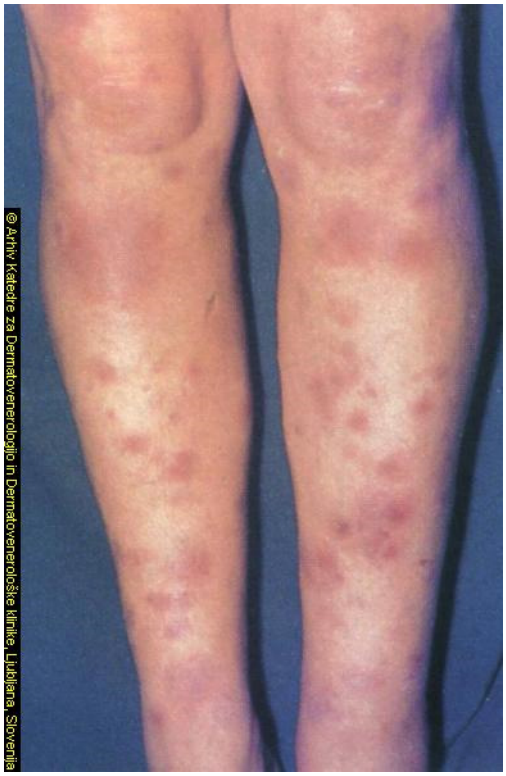
ADULT SEBORRHOEIC DERMATITIS = red scaly rash affecting the scalp causing dandruff

Treatment = ketoconazole , metronidazole

ERYTHEMA:

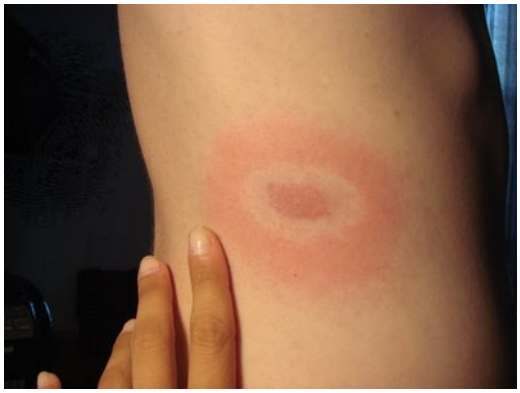
1. ERYTHEMA MARGINATUM – seen in rheumatic fever

2. ERYTHEMA NODOSUM (Painful nodules on limbs)– seen in rheumatoid arthritis , inflammatory bowel disease , TB , **sarcoidosis** ,SLE



3. ERYTHEMA CHRONICUM MIGRANS (round/ ovoid rash with central clearing) = seen in lyme 's disease caused by tick bite in forests or mountain area , the causative agent is "BORRELIA BURGDORFERI".

Treatment = **doxycycline**



4. **ERYTHEMA MULTIFORME** = drug eruption especially due to NSAIDS and antibiotics usually "without" involvement of mucous membrane

6. **STEVEN JOHNSON SYNDROME** = drug eruption "with" involvement of mucous membrane

Symptoms

- It often starts with a nonspecific upper respiratory tract infection, which may be associated with fever, sore throat, chills, headache, arthralgia, vomiting and diarrhoea, and malaise.
- Mucocutaneous lesions develop suddenly and clusters of outbreaks last from 2-4 weeks. The lesions are usually not pruritic.
- Mouth: severe oromucosal ulceration.
- Respiratory involvement may cause a cough productive of a thick purulent sputum.
- Patients with genitourinary involvement may complain of dysuria or an inability to pass urine.
- Ocular symptoms: painful red eye, purulent conjunctivitis, photophobia, blepharitis

NB: If after sore throat , patient is treated with amoxicillin or ampicillin and develops generalised rash, it means the sore throat was caused by infectious mononucleosis. Therefore it is not recommended to prescribe the above mentioned antibiotics in any patient who is suspected of having infectious mononucleosis.

SKIN CONDITIONS IN SYSTEMIC DISEASES:

1. Inflammatory bowel disease = **pyoderma gangrenosum , erythema nodosum**
1. SLE = **malar rash or butterfly rash on face (cheeks) & discoid rash**
1. Rheumatoid arthritis = **erythema nodosum**
1. Dermatomyositis = **purple heliotrope rash on eyelids , itchy rash over shoulder & back called "shawl sign"** , dermatomyositis occurs in the presence of cancers
1. Diabetic mellitus = **necrobiosis lipoidica , granuloma annularae**
1. Glucagonoma = **necrolytic migratory erythema**
1. Polyarteritis nodosa (PAN) = **livedo reticularis (** red /blue mottling of skin) , **erythema nodosum**
1. Hypo/Hyper – thyroidism = **pre-tibial myxoedema**
1. Neurofibromatosis = **café-au-lait spots. Autosomal dominant disease(1:2 chance transmission)**
1. Coeliac disease = **dermatitis herptiformis** (severe blistering itchy rash)
1. T- cell lymphoma = **erythroderma**
1. Asthma = associated with **eczema**
1. HIV = **kaposi's sarcoma , molluscum contagiosum , oral hairy leukoplakia , oral thrush ,herpes simplex infection ,varicella zoster infection or simply shingles**
1. **Sarcoidosis =erytherma nodosum**

SCABIES :

Caused - sarcoptes scabei

Spreads – from direct person to person contact

Typically affected individuals – old people in care homes , children

Symptoms – severely itchy vesicles / rash in between the fingers , groin area ,waistband area or in between the toes. Itching is allergy to toxin released by the mites.

Investigation – inspection of hands & feet , skin scrapping

Treatment - 1) 5% permethrin

2) malathion

TINEA INFECTION (fungal infection):

TINEA CAPITIS = fungal infection in the head

TINEA CORPORIS = fungal infection on the body

TINEA UNGUIUM = fungal infection of nail

TINEA CRURIS = fungal infection in the groin

Treatment :

Topical anti-fungal application

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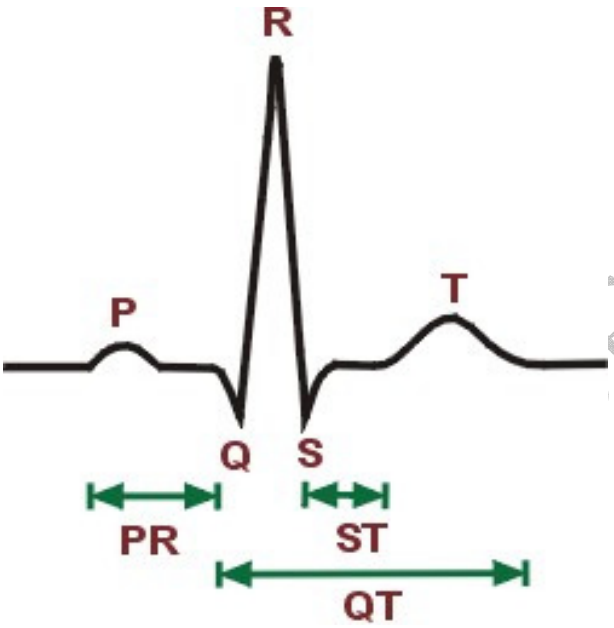
ECG with Dr. SAMSON

NORMAL QRS COMPLEXES:



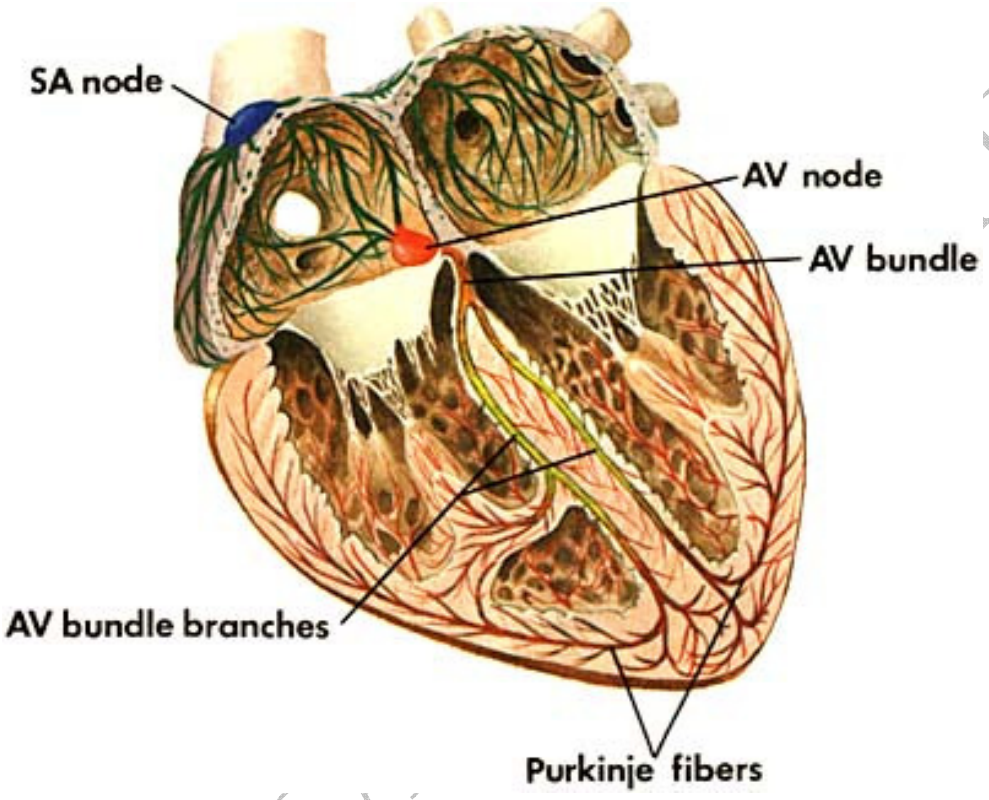
ECG LEADS

- **Limb Leads**
  - **I**
  - **II**
  - **III**
  - **AVR**
  - **AVL**
  - **AVF**

- Chest Leads
  - V_1
  - V_2
  - V_3
  - V_4
  - V_5
  - V_6

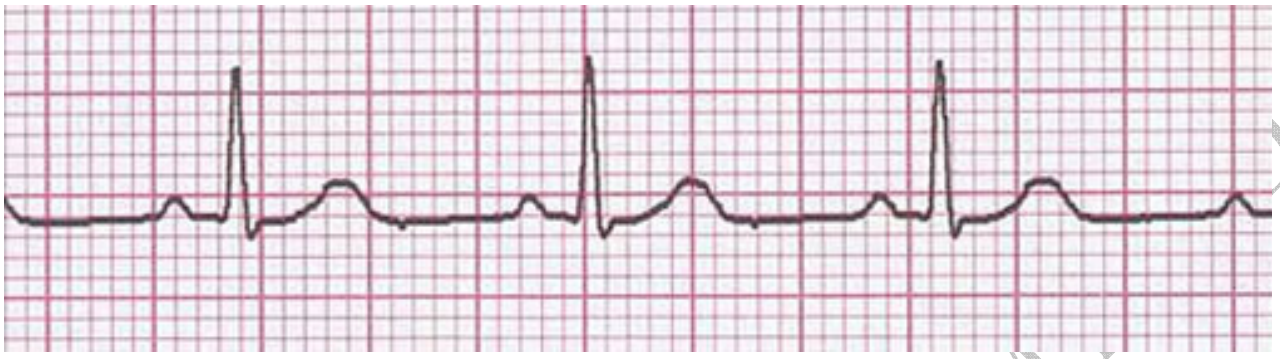
1. RHYTHM.

   If similar P-wave before each QRS complex and regular =normal sinus rhythm



Automaticity Foci

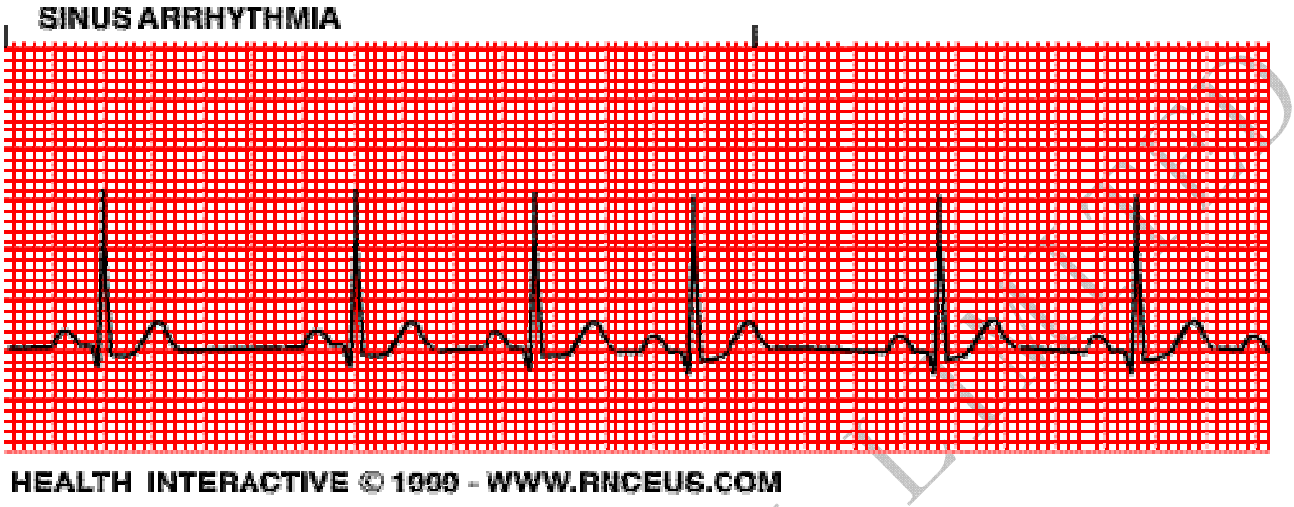
• Level	Rate
– Atria	60-80/min
– AV Junction	40-60
– Ventricles	20-40

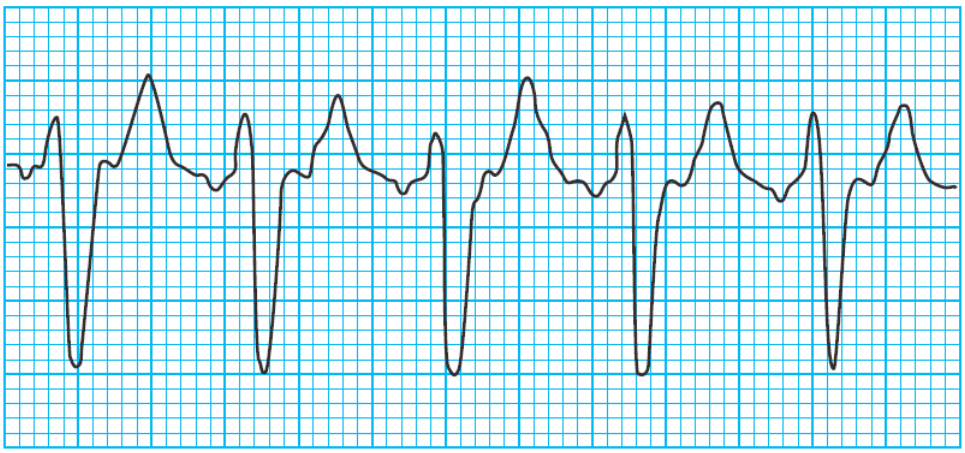


<u>If sinus rhythm & irregular</u> :

i. Extra systole

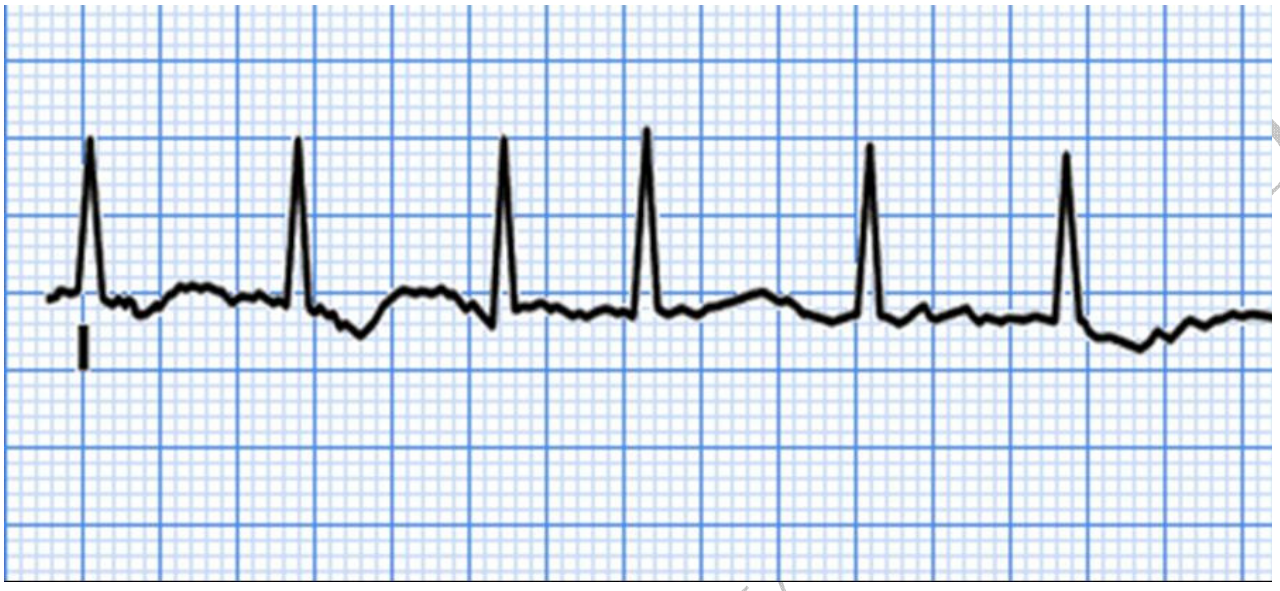
ii. Sinus arrhythmia





Atrial tachycardia with 2:1 block (note the inverted P waves)

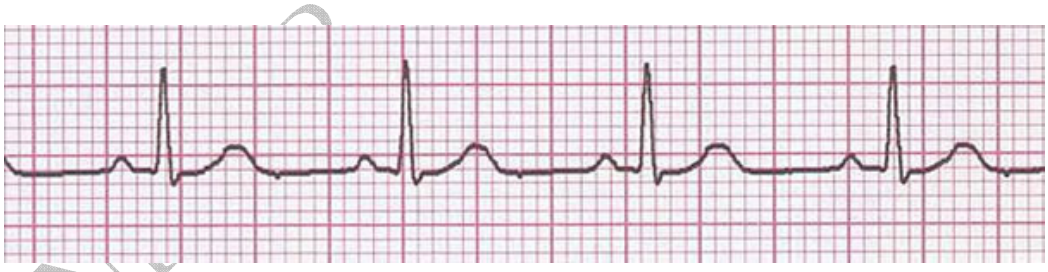
- If no P-wave
    i. Atrial Fibrillation (AF)



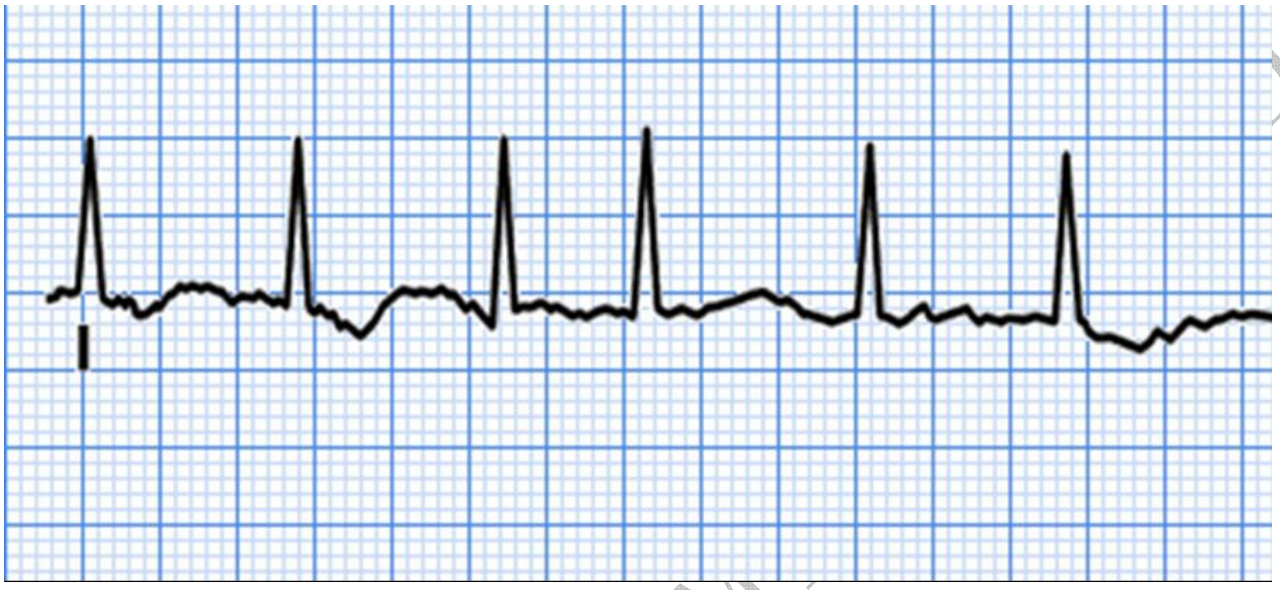
ii. Junctional rhythm from AV node.

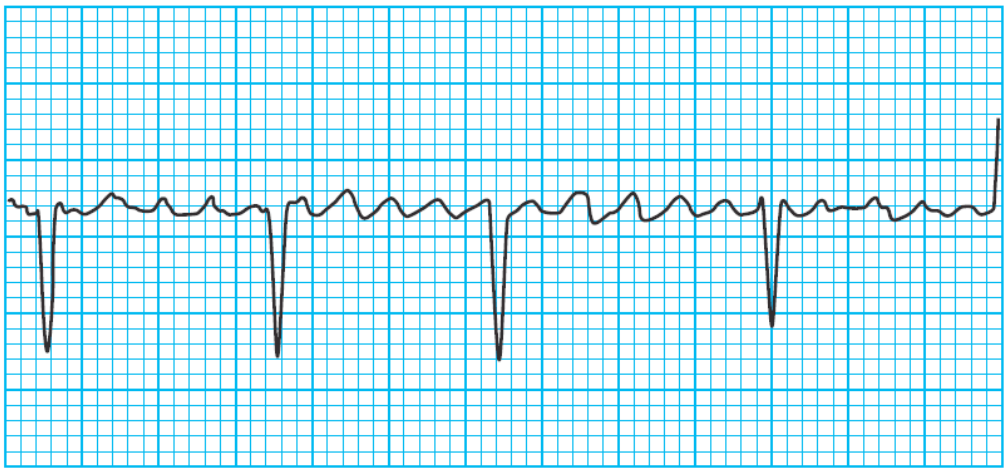
2. REGULARITY

- Regular=if the distance between each complex is the same.



- Irregular=if the distance between each compex is different
  i. AF (variably irregular)



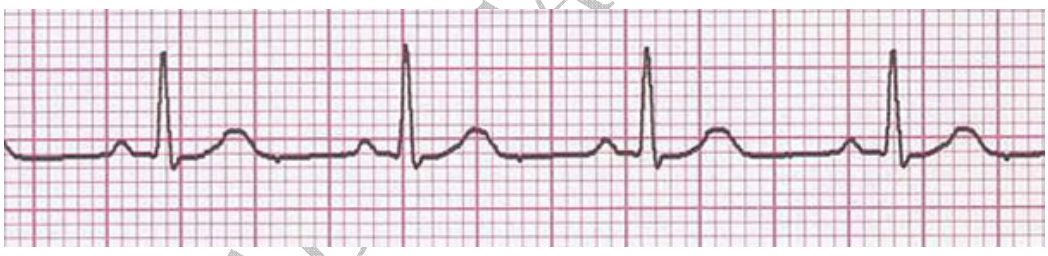


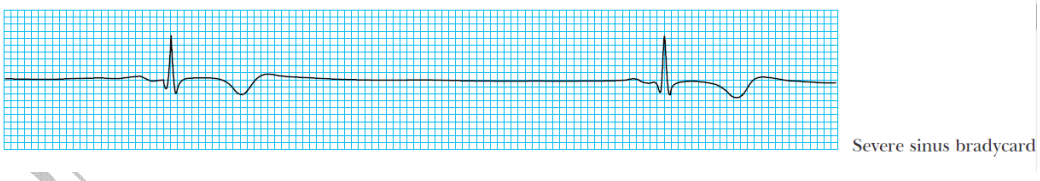
Atrial fibrillation waves seen in lead V1

ii. Extra-systole(regular rhythm except for 1 complex)

3. RATE (normal 60-100)

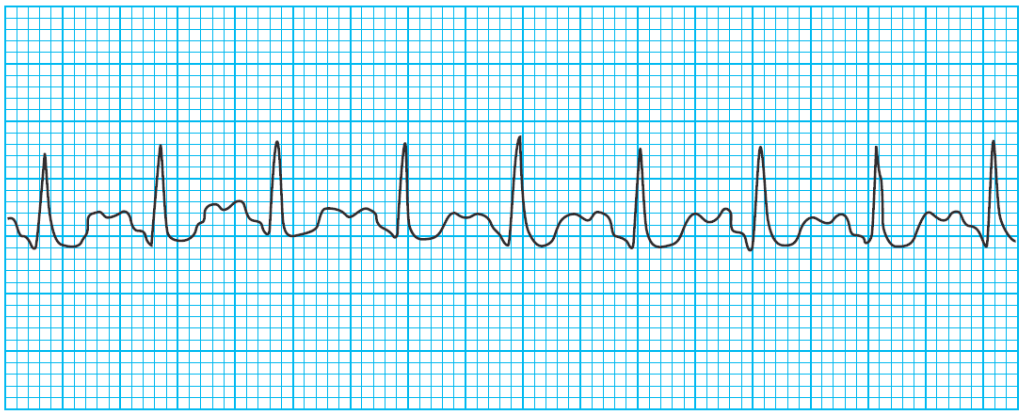
- 300/number of big squares between R-R





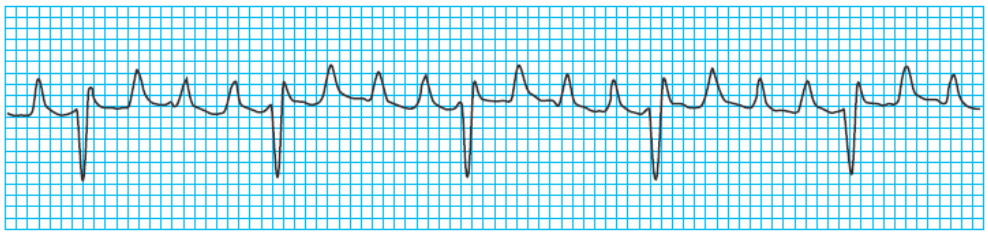
Severe sinus bradycard

HR > 100 TACHYCARDIA

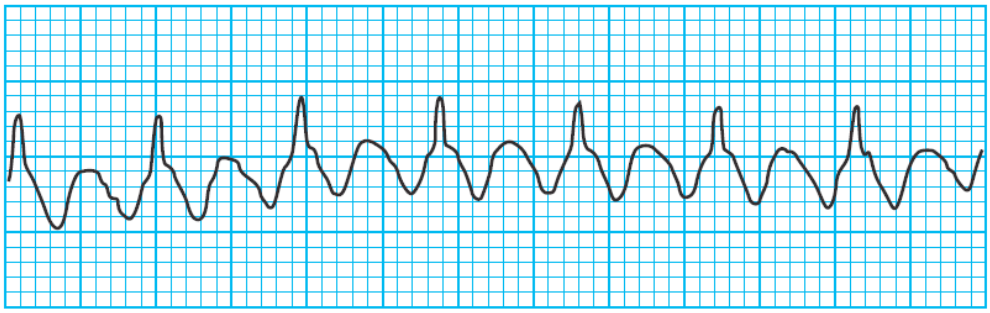


Sinus tachycardia

- If HR rate = 150



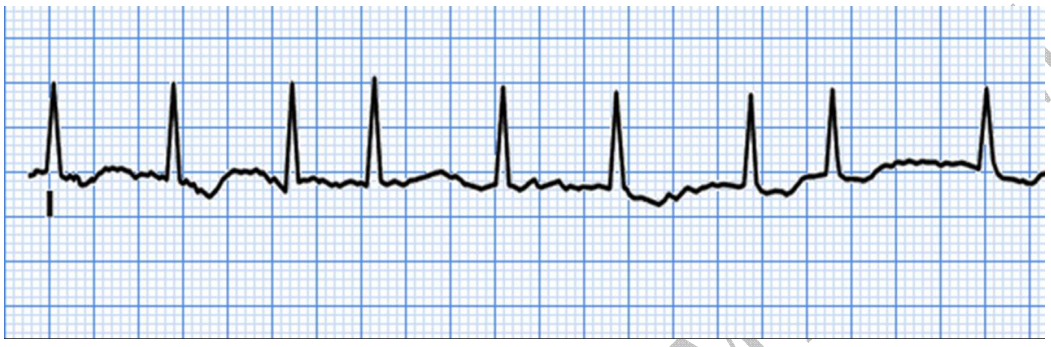
Atrial flutter is usually the result of a single re-entrant circuit in the right atrium (top); atrial flutter showing obvious flutter waves (bottom)



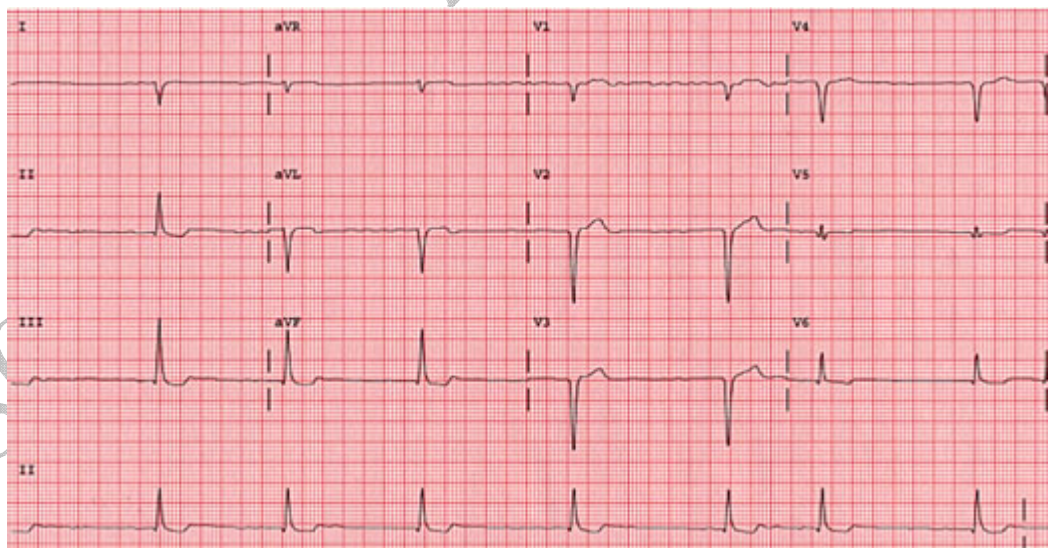
Rhythm strip in atrial flutter (rate 150 beats/min)

Ask your self is it Atrial flutter? (i.e. rate is 300/min with 2:1 block)

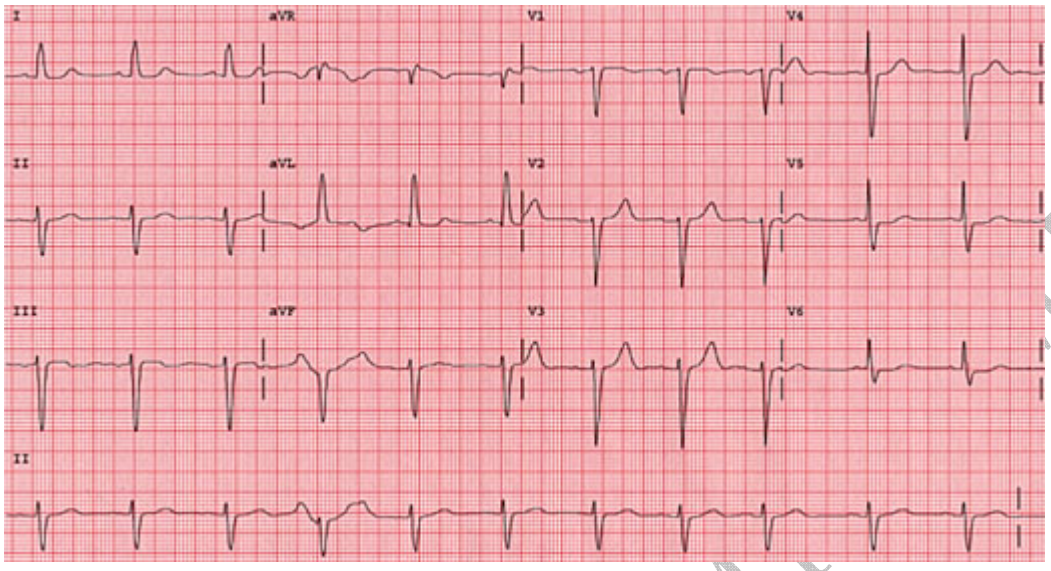
- If Irregular rhythm then rate = No. of QRS complexes within 30 big square X 10



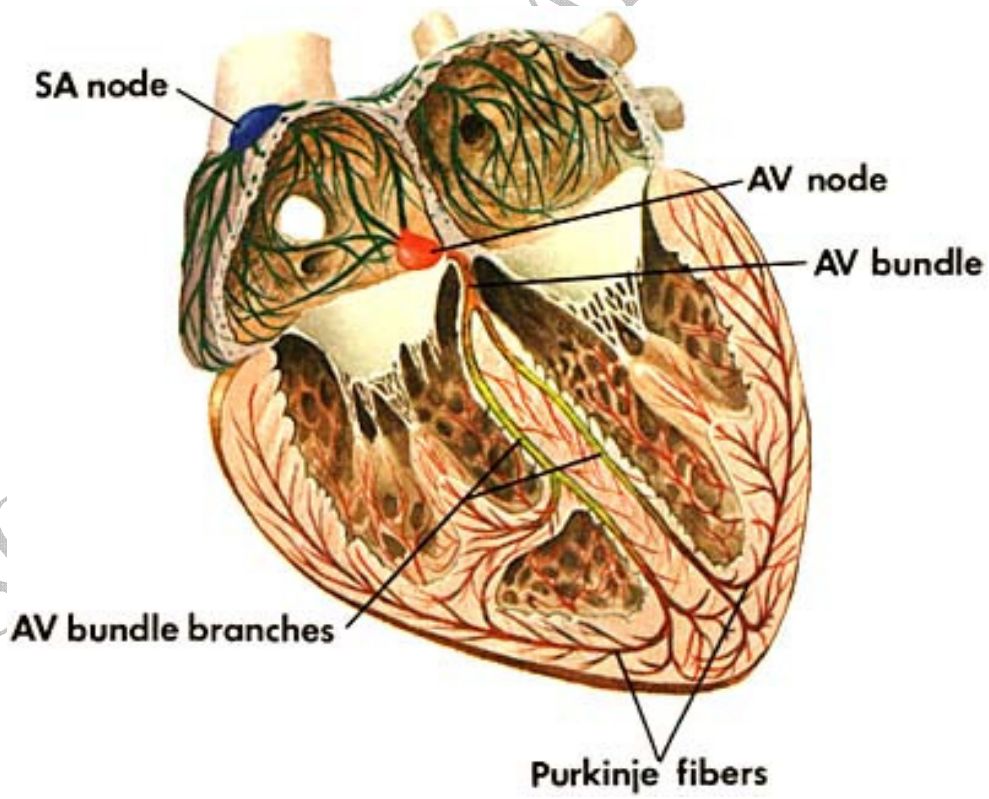
4. AXIS
    - Tallest R-wave II = Normal
    - III or aVF = Right Axis Deviation (RAD)



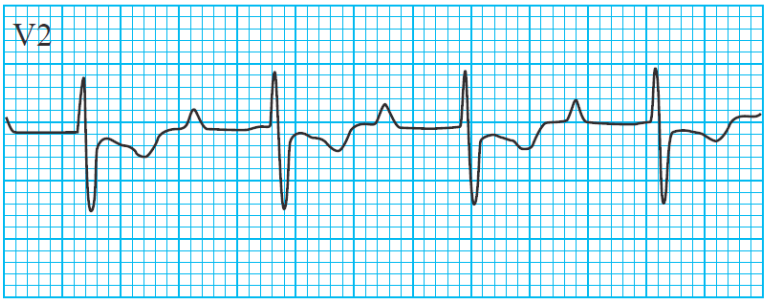
- I or aVL with equidistant R & S in II or Negative = Left Axis Deviation (LAD)



5. P-R interval (normal =<200ms or 5 small boxes)

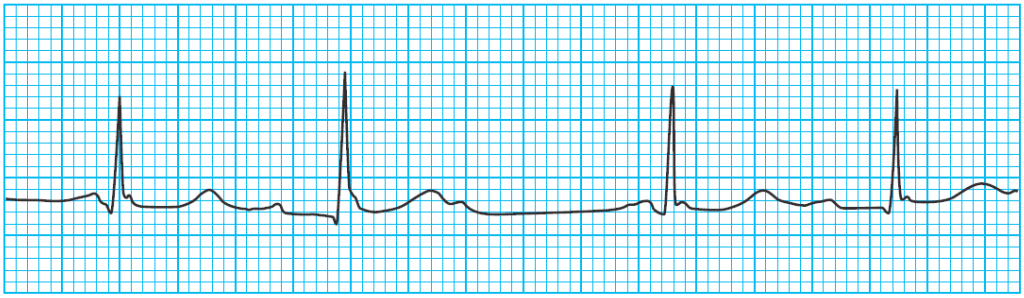


- >200ms = 1st Heart Block

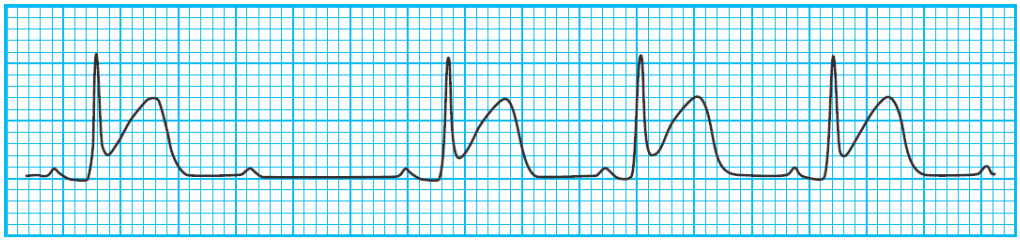


First degree heart (atrioventricular) block

- If certain QRS complex missing then 2nd Heart Block

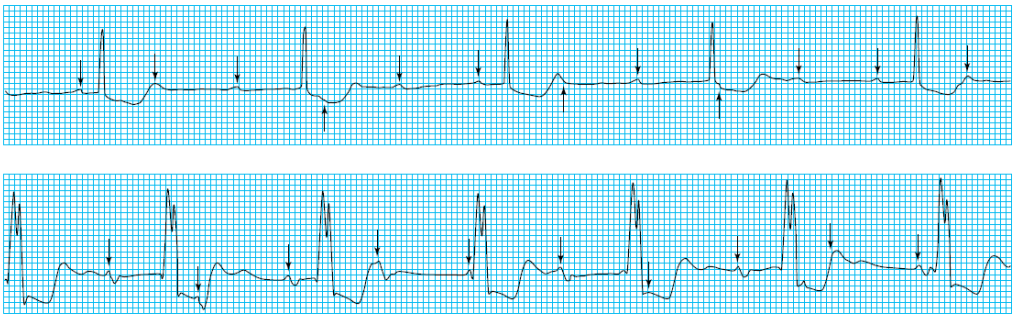


Mobitz type I block (Wenckebach phenomenon)



Mobitz type II block—a complication of an inferior myocardial infarction. The PR interval is identical before and after the P wave that is not conducted

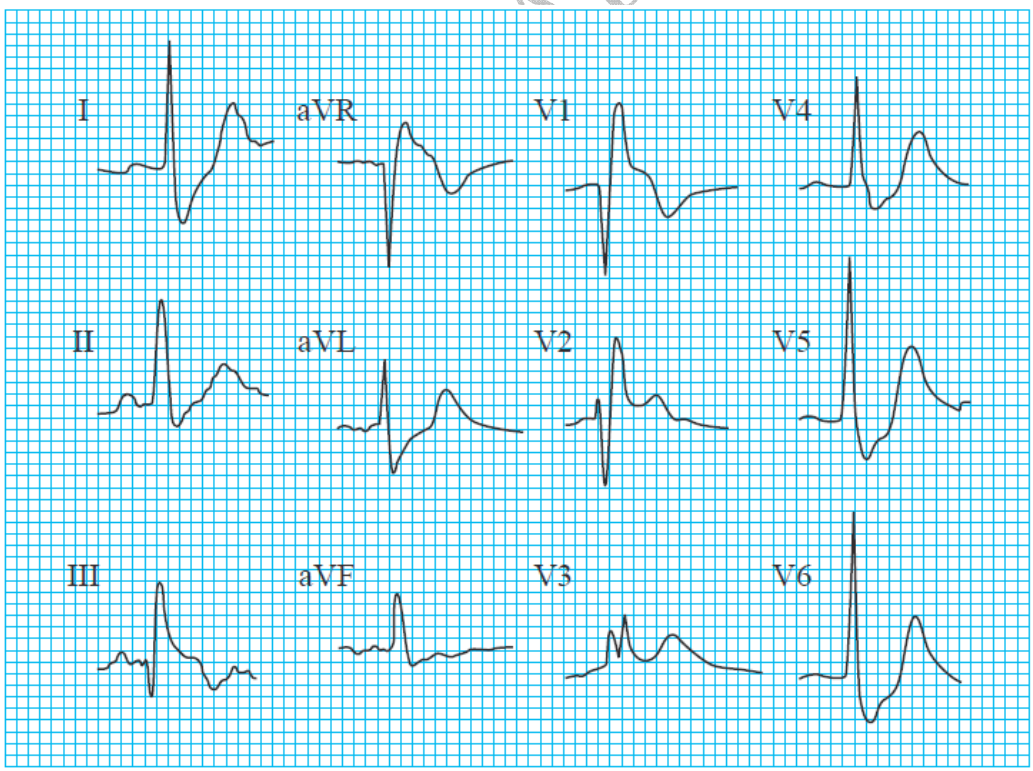
if Heart Rate <40 likely complete heart block. There is complete dissociation of the between P waves and QRS complexes.



Third degree heart block. A pacemaker in the bundle of His produces a narrow QRS complex (top), whereas more distal pacemakers tend to produce broader complexes (bottom). Arrows show P waves

- If <0.12s = Supra Ventricular Tachycardia (SVT), Wolf Parkinson White (WPW) syndrome or Accessory pathway.

6. Q-wave. Normal = less than one quater of the height of R wave.



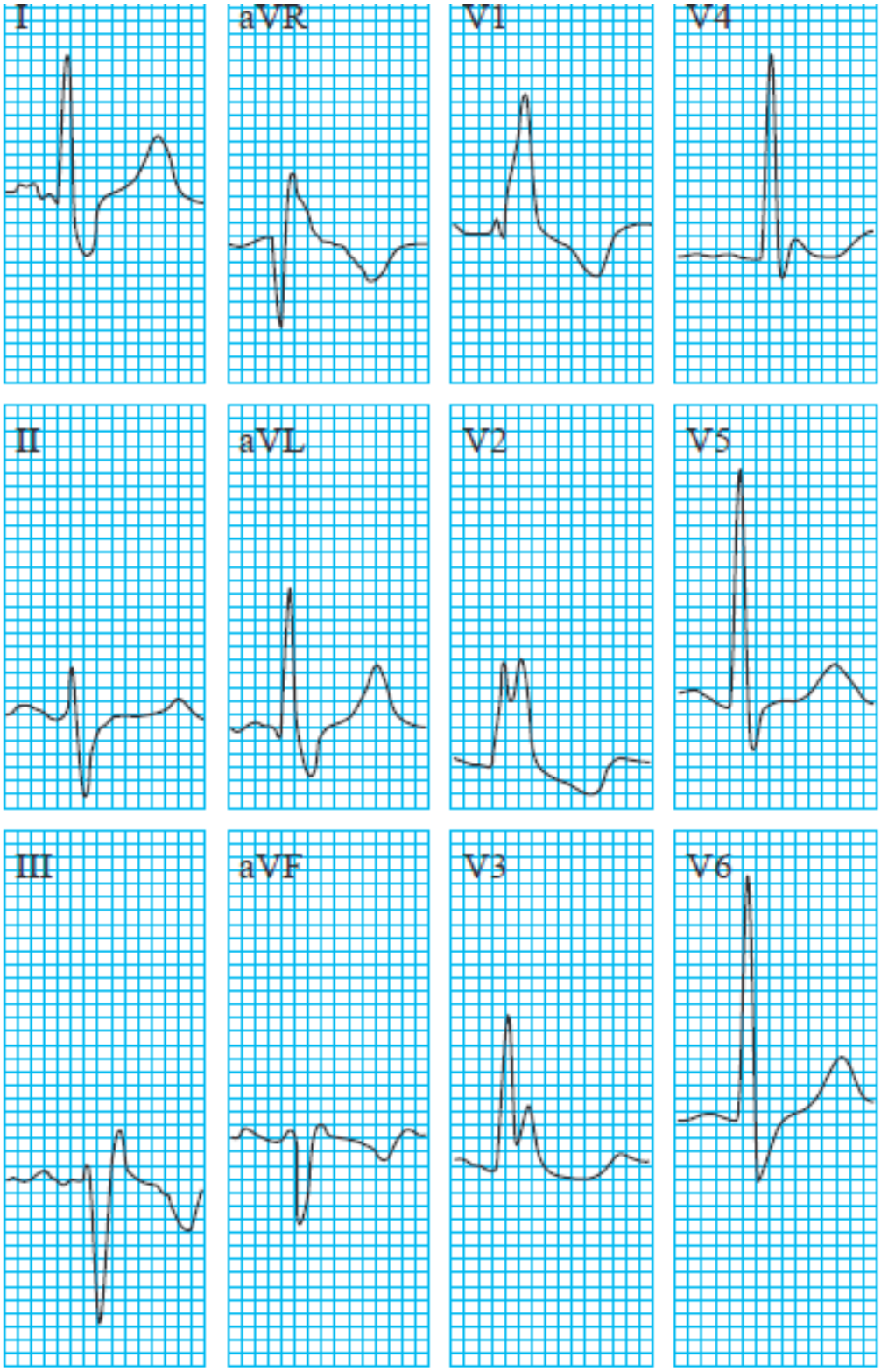
S1, Q3, T3 pattern and right bundle branch block in patient with pulmonary embolus

7. QRS normal 0.12sec= 3 small boxes=120ms

8. BUNDLE BRANCH BLOCK (BBB) – V1*

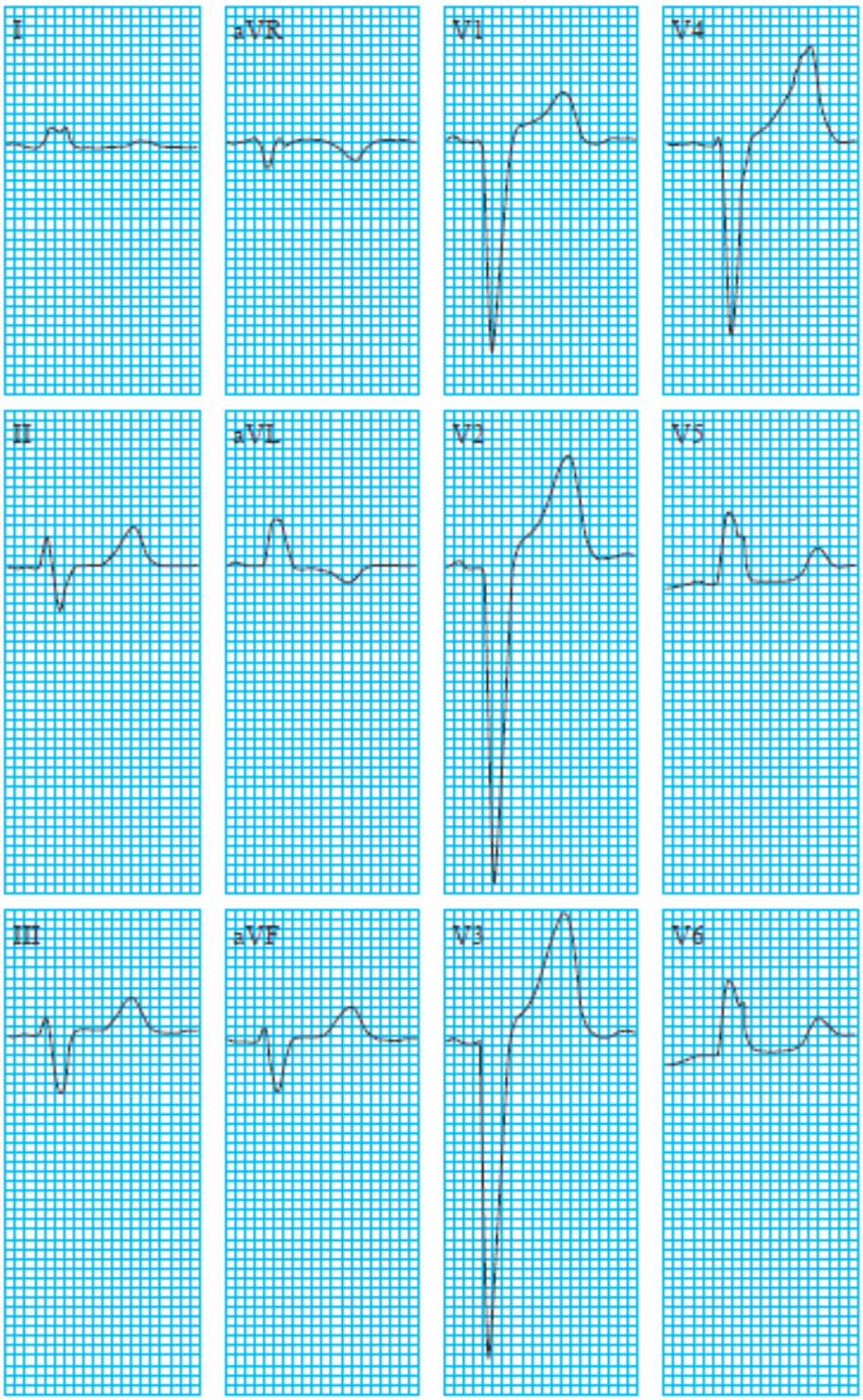
- QRS must be wide i.e. >0.12s or >3 small boxes

- If QRS
  i. Positive = RBBB



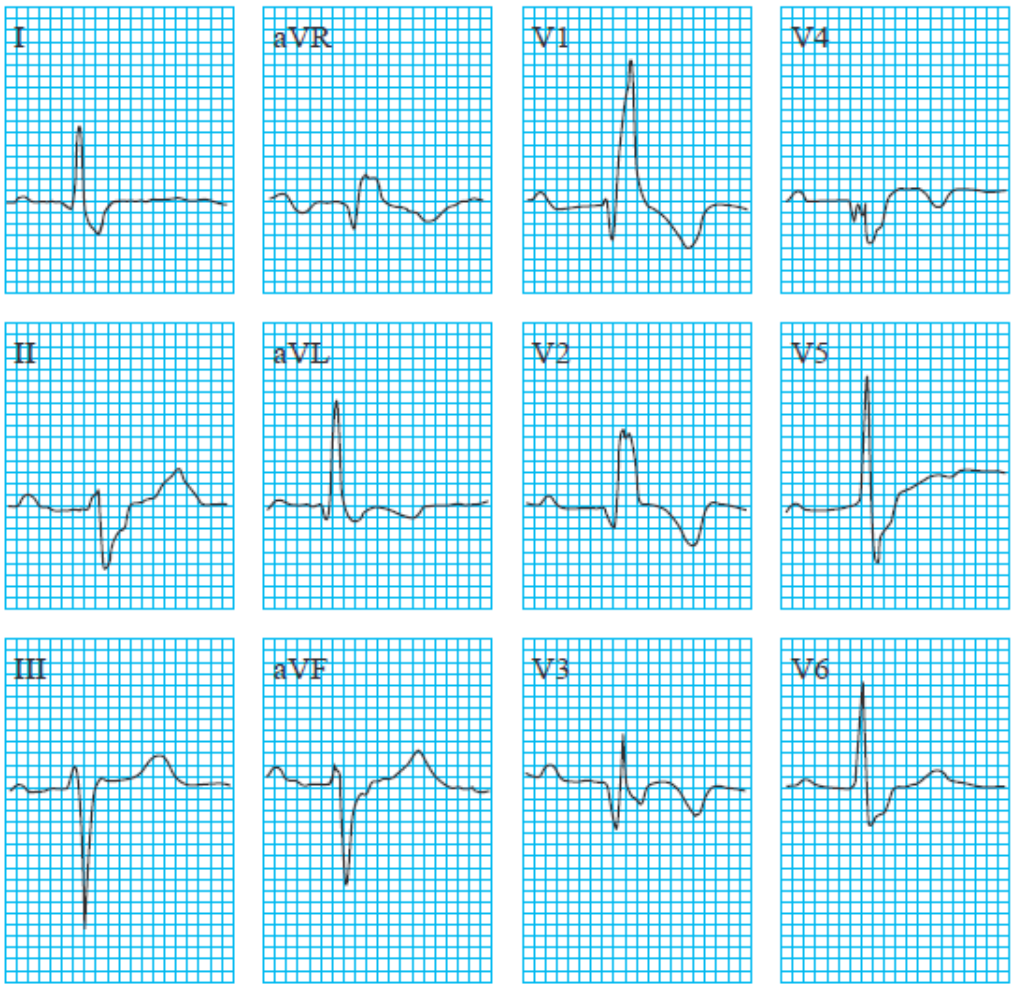
Right bundle branch block

ii. Negative = LBBB



Left bundle branch block

- RBBB + LAD + first degree heart block = Trifascicular block



Trifascicular block (right bundle branch block, left anterior hemiblock, and first degree heart block)

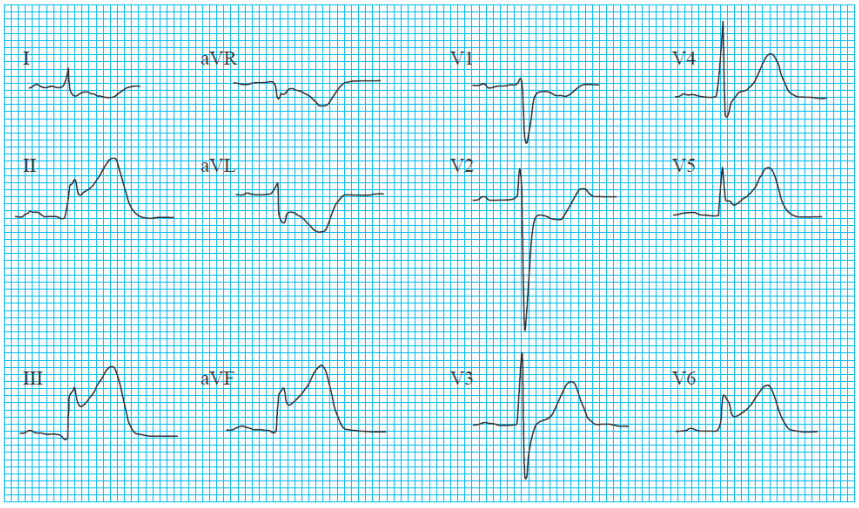
- RBBB + LAD = Bifascicular (i.e. R + posterior branch affected)

- New LBBB plus chest pain= MI

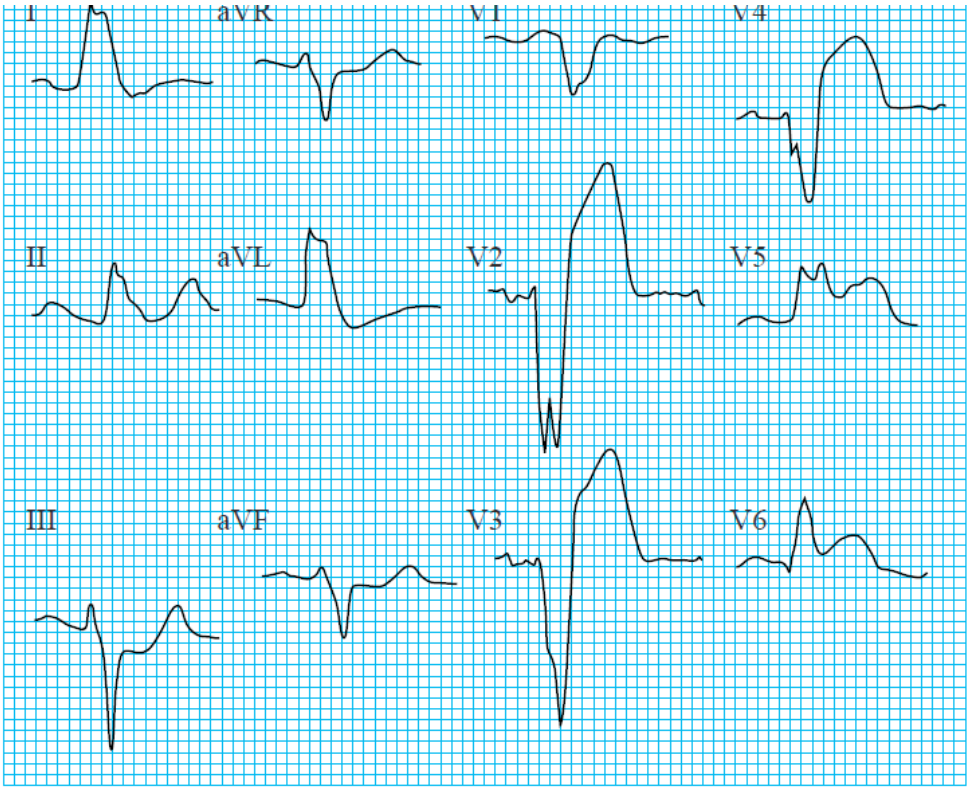
- If LBBB = No further interpretation of an ECG

9. ISCHAEMIA

- Q Wave = Old /Evolving MI
- Elevated ST = acute MI or Pericarditis(wide spread ST elevation)

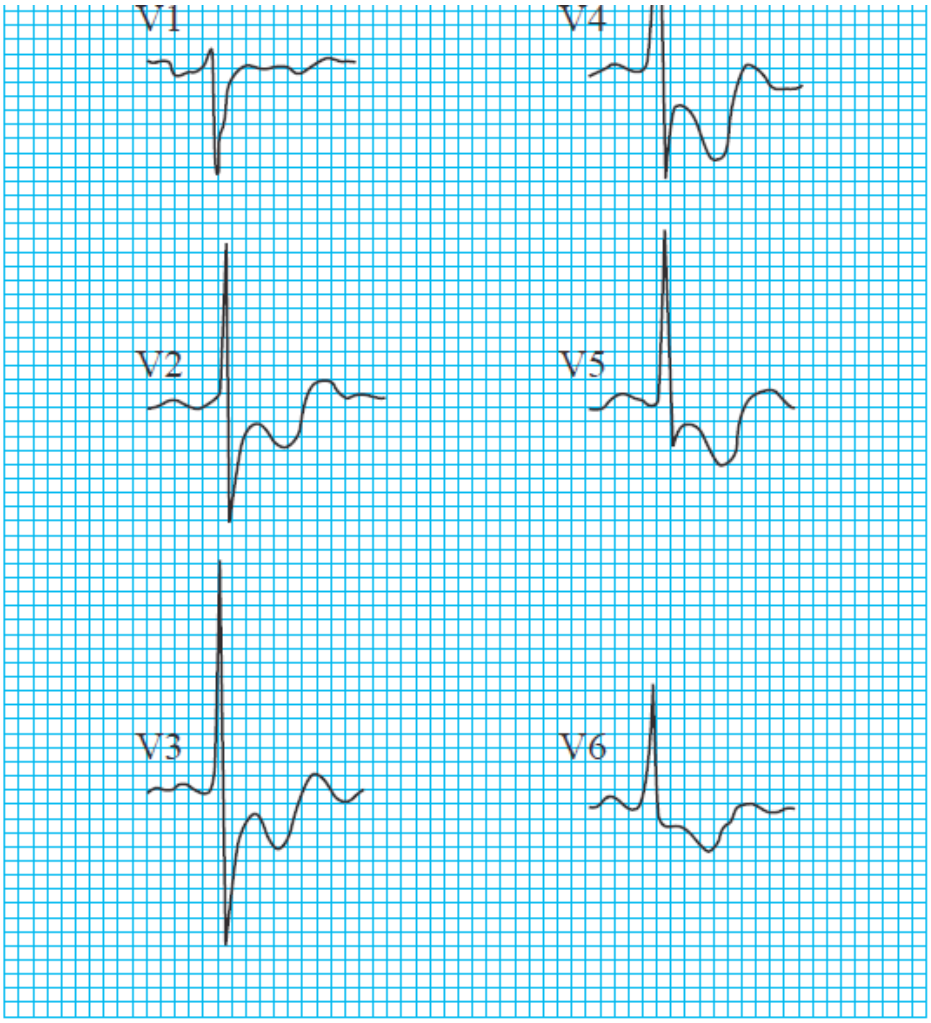


An inferolateral myo
reciprocal changes i



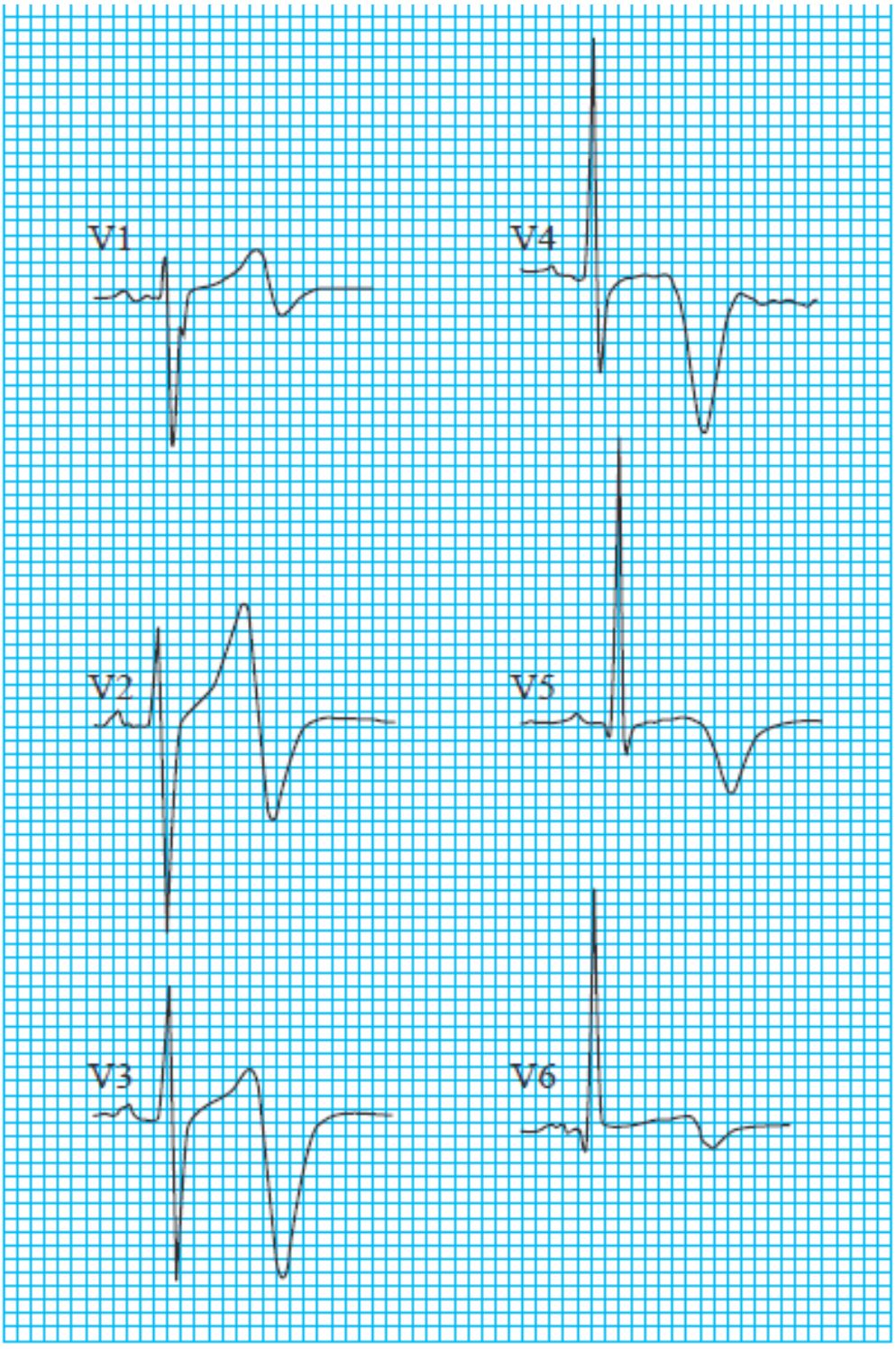
Acute myocardial infarction and left bundle branch block. Note that the ST segments are elevated in leads V5 and V6 (inappropriate concordance) and grossly elevated (> 5 mm) in leads V2, V3, and V4; note also the ST segment

- ST depression
  i. Digoxin toxicity
  ii. Ischaemia



Widespread ST segment depression in patient with unstable angina

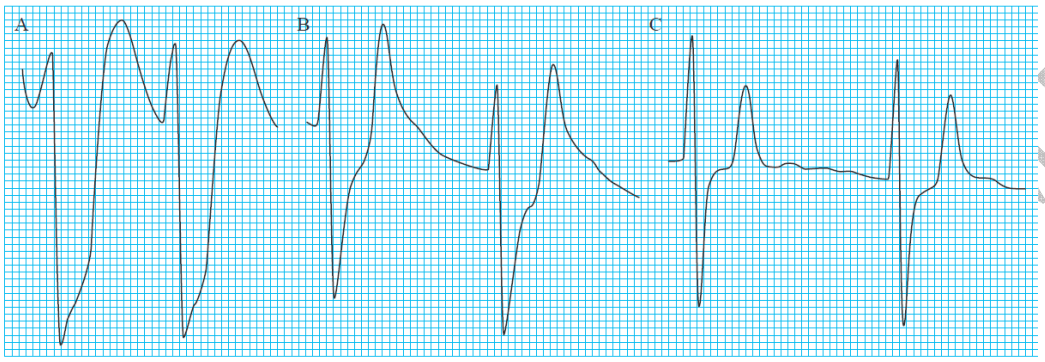
- **T-wave inversion**
  i. **Acute ischaemia**



Biphasic T waves in man aged 26 with unstable angina

ii. Old infarct *(only if exists with Q-wave)*

- Tall T-wave = HYPERKALAEMIA



Serial changes in patient with renal failure receiving treatment for hyperkalaemia. As potassium concentration drops, the electrocardiogram changes: 9.3 mmol/l, very broad QRS complexes (A); 7.9 mmol/l, wide QRS complexes with peaked T waves an absent P waves (B); 7.2 mmol/l, QRS complex continues to narrow and T waves diminish in size (C)

10. Walls:

aVF, II and III =inferior MI,

I, AVL, V5, V6=lateral MI

V1-V6, aVL, I= anterior lateral MI

V1-V4 =Anterior MI

V1-V2=septal MI

11. HYPERTROPHY (USE CHEST LEADS)

2. R + S ≥ 35mm = Left Ventricular Hypertrophy (LVH)
3. Tallest R in (V4 or V5) plus deepest S (V1 or V2)

4. Peaked P-wave = P. Pulmonale = ↑ Right atria (R atrial strain)

5. Toothed (biphasic) P-wave = P Mitrale = ↑ Left atria (Left atrial strain)

6. Dizziness, syncope, fainting on exercise = aortic stenosis

7. New LBBB plus chest pain= acute MI until proven otherwise (*check previous ECG & compare the two)*

8. If murmur or new AF = ECHO

9. If dizziness on exercise = ECHO

10. LAD is associated with LBBB & LVH

11. Common cause of P Mitrale = Mitral Stenosis

12. If LAD & No LVH or LBBB or RBBB → think of *L anterior hemiblock*

13. WPW

    1. if wide QRS is in V1 → then the *accessory pathway is on the left side*)
    2. if wide QRS in V6 → then the *accessory pathway is on the Right*

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+44 0794 0433 068

Resource view

Resource name	Endocrinology PLAB 1 Notes
Resource description	Endocrinology
Resource content	

ENDOCRINOLOGY

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Contents:

c. Conn's Disease
d. Pheochromocytoma

6. Acromegaly

7. Syndrome of In Appropriate Anti Diuretic Hormone

8. Hyperprolactinemia

9. Diabetes Insipidus

1. DIABETES MELLITUS – This is high serum blood glucose

DM results from lack or reduced effectiveness of endogenous insulin.

It is imperative that a diabetic person having hypertension should have it well controlled.

TYPE 1 DIABETES

- Usually juvenile onset
- Common in young patients
- It is due to absolute deficiency of insulin
- An autoimmune condition in which there is destruction of the B cells of the pancreas
- **Symptoms:** Polyuria, Polydypsia, Weight loss, Diabetic Ketoacidosis
- First presentation can be Diabetic Ketoacidosis
- There could be history of other autoimmune conditions like Addison's disease, Thyroid disease and Pernicious Anaemia
- **Antibodies** e.g. anti-glutamic acid decarboxylase (GAD) antibodies and islet cell antibodies

TYPE 2 DIABETES

- Usually occurs in the adults mostly in Asian men and above the age of 40 years, most are obese
- Is due to insulin resistance and relative insulin deficiency
- Often it is asymptomatic and may first present with complications like diabetic retinopathy, nephropathy and neuropathy
- **RISK FACTORS:** Pregnancy, obesity, polycystic ovarian syndrome (PCOS), renal failure, lack of exercise

DIAGNOSIS: To make the diagnosis of diabetes, you need to consider the following:

1. If the patient is asymptomatic do the blood tests twice, either:
- Fasting glucose >7.0 OR
- Random blood glucose> 11.0 confirms the diagnosis

Fasting glucose <6.1 is normal
Fasting glucose 6.1-7 is Impaired fasting glucose
Fasting glucose ≥ 7.1 is Diabetes Mellitus

2. If the patient is symptomatic, perform one of the following tests once.

- Fasting glucose ≥ 7.0 or
- Random blood glucose ≥ 11.1

Do the Oral Glucose Tolerance Test (OGGT) if there is impaired fasting glucose. This is done with 75mg of sugar and blood glucose is measured after 2 hrs.

- Glucose <7.7 is normal
- Glucose > 7.8—11 is Impaired oral glucose tolerance
- Glucose ≥ 11.1 confirms Diabetes

NB.
- **Only venous blood glucose is used for making the diagnosis.**
- **Capillary blood glucose is only used for monitoring.**
- **For monitoring blood glucose control you monitor glycosylated haemoglobin (Hba1c). Normal levels should be less than or equal to 6.1.**

Treatment:

Impaired Glucose Tolerance Test

This is treated with diet and exercise.

Type 1 Diabetes Mellitus

In type 1 diabetes Insulin is always used but in different regimes e.g.
1. **Twice a day if patient has a regular life style**
2. **Four times a day plus long acting at bedtime if a patient has variable activity e.g. exercising.**
3. **Once a day in the morning long–acting when switching from oral hypoglycaemic to Insulin**

Type 2 Diabetes Mellitus

Stage 1 (Newly diagnosed):
- **Diet and exercise**

Stage 2 (Not controlled by diet and exercise alone):
- **Diet and exercise +**
- **Oral hypoglycaemic**

Stage 3 (Not controlled by above treatment):
- **Diet and exercise +**
- **Oral hypoglycaemic +**

- **Insulin**

Oral hypoglycaemic medication

1. **Biguanides** e.g. Metformin
- Especially good for patients who are obese. It is always the first choice. Increases insulin sensitivity
- Weight loss
- Causes lactic acidosis
- Does not cause hypoglycaemia
- If glucose is not controlled add Sulfonylurea.

SE: nausea, vomiting, lactic acidosis.

1. **Sulfonylureas** e.g. Glibenclamide or Gliclazide
- They increase insulin sensitivity

SE: Hypoglycaemia

1. **Thiazolidines** e.g. Glitazones- Poiglitazone and Rosiglitazone
- They are used if patient not tolerant to either metformin or Sulfonylurea.
- They cause weight gain
- Do not cause hypoglycaemia

COMPLICATIONS OF DIABETES MELLITUS

A. **Acute complications**

a. Hypoglycaemia
b. Diabetic ketoacidosis (DKA)
c. Hyperglycaemic Hyper-osmolar non ketotic coma (HONK)

A. **Chronic complications**

a. Microvascular = diabetic retinopathy, diabetic nephropathy, diabetic neuropathy, autonomic neuropathy and somatic neuropathy.
b. Macrovascular = Stroke, IHD, Intermittent Claudication, (Peripheral vascular disease) as a result of atherosclerosis

ACUTE COMPLICATIONS

1. **Hypoglycaemia** is glucose less than 3 mmol/l

Symptoms:

Pale, sweating, tremor, jittering, aggressive, tachycardia, seizure, coma.

Loss of consciousness and sweating = hypoglycaemia until proven otherwise.

Treatment

1. **If the patient is conscious give an oral sugary drink.**
2. **If the patient is unconscious then treat as follows:**
- **1st choice is 10% glucose/dextrose**
- **2nd choice 50% glucose/dextrose**
- **3rd choice is Glucagon; disadvantage is that it does not work if there is alcohol in the blood or in patients with anorexia nervosa. This is because glucagon acts by converting glycogen into glucose. Glycogen is the storage form of glucose in the liver.**

CAUSES OF HYPOGLYCAEMIA

1. Insulin overdose especially in type 1 Diabetes
2. Glibenclamide and gliclazide in type 2 Diabetes
3. Insulinoma = it is a benign tumour of the pancreas which produces insulin and it causes hypoglycaemia. Usually every time a patient misses meal he loses consciousness or he will have fit. It occurs as part of MEN-1.

Investigation: C-peptides and Insulin level in the blood.

2. Diabetic Ketoacidosis (usually it has gradual onset)

- Occurs only in type 1 Diabetes
- The criteria is pH < 7.3, HCO3 <15, ketones in urine or capillary
- ABGs will show metabolic acidosis
- **Precipitating factors:** infection, surgery, MI, Sepsis, UTI, Gastroenteritis and Pneumonia

Symptoms/Clinical features:

Young patient, weight loss, polydipsia, dehydration, lethargy, anorexia, vomiting, abdominal pain, coma, usually there is progressive drowsiness.

On examination patient will have Kussmaul respiration, which is deep and sighing breathing. Fast respiratory rate e.g. 40/min

Treatment:

- **The initial treatment is always IV fluids.**

- **Use normal saline until capillary blood glucose is 10 mmol/L then switch to dextrose 5%.**
- **Give insulin 0.1 units per kg**
- **Give K+ depending on the level. If K+ is >5 mmol/L don't give any.**

3. Hyperglycaemic Hyper-osmolar Non-Ketotic coma (HONK)

Occurs only in type 2 Diabetes
Usually elderly patient and this could be the first presentation.

Criteria for diagnosis:

1. No acidosis
2. Glucose> 35 mmol/l
3. Plasma osmolarity >350 mosm/kg
4. Ph is normal
5. No ketones in the urine

Symptoms:

1. Dehydration
2. Lethargy, weakness, polyuria, polydipsia, progressive loss of consciousness over days

Treatment: IV fluids (normal saline) an Insulin

CHRONIC COMPLICATIONS OF DIABETES

1. MACROVASCULAR COMPLICATIONS

a. Heart - Myocardial Infarction: Usually patient has silent Myocardial Infarction (MI) or atypical chest pain due to neuropathy.

b. Brain - TIA/Stroke due to atherosclerosis.

c. Peripheral vascular disease - atherosclerosis leads to intermittent claudication. Pain comes on walking and after a short rest it goes away.

2. MICROVASCULAR COMPLICATIONS

Diabetic retinopathy

A. **Background -** Micro-aneurysms, dot and blot haemorrhages and hard exudates
B. **Pre-Proliferative -** Micro-aneurysms, dot and blot haemorrhages, hard exudates and soft exudates (cotton wool spots)
C. **Proliferative -** Micro-aneurysms, dot and blot haemorrhages, soft and hard exudates, new vessel formation (neovascularization)
- New vessel formation leads to bleeding which may cause retinal detachment which comes as a sudden loss of vision and the patient complains as a curtain coming down. Cataract formation is earlier.

Diabetic Maculopathy - This is when changes develop in the macula

Vitreous haemorrhage - This is when there is bleeding in the vitreous. The patient usually complains of floaters.

Diabetic Nephropathy

- Micro-albuminuria- loss of >300mg/day of protein.
- It is a big risk factor of IHD and stroke therefore it needs treatment.
- In people who are diabetic, the target BP is ≤ 130/80 and if there is micro-albuminuria then target BP ≤125/75.

Micro-albuminuria leads to diabetic nephropathy and eventually renal failure if not treated. In renal failure insulin sensitivity increases and insulin metabolism decreases therefore insulin needs to be reduced to avoid hypoglycaemic attacks.

Diabetic Neuropathy

1. Peripheral neuropathy (somatic neuropathy) is usually symmetrical in a form of gloves and socks.
2. Mono neuropathy e.g. 3rd, 4th and 6th nerve palsy.
3. Autonomic neuropathy will cause vasovagal syncope, diarrhoea, postural hypotension or urinary retention.
4. Amyotrophy – progressive wasting and weakness of muscles especially the quadriceps muscles.

2. PITUITARY GLAND

Anterior Pituitary produces Growth hormone (GH), Gonadotropins: Follicle stimulation hormone (FSH) & Leutenizing hormone (LH), Prolactin (PRL), Thyroid stimulating hormone (TSH), Adrenocorticotrophic hormone (ACTH)

Posterior Pituitary stores Oxytocin and ADH (Anti-diuretic hormone)
- Oxytocin acts on the uterus and causes contraction.
- ADH acts on the kidneys and cause urine retention.

NB: The ANTERIOR PITUITARY PRODUCES hormones and the POSTERIOR PITUITARY STORES hormones.

Hypopituitarism
Hormones are affected in this order: GH, FSH & LH, PRL, TSH, ACTH

Causes are at 3 levels:

1. **Hypothalamus:** Kallman's syndrome (isolated FSH LH deficiency with anosmia and colour blindness), tumour, inflammation, infection

1. **Pituitary stalk:** Trauma, surgery, compression by a mass lesion (eg. due to a craniopharyngioma), carotid artery aneurysm

1. **Pituitary:** Tumour, irradiation, inflammation, autoimmunity, ischaemia (eg. Sheehan's syndrome due to post partum haemorrhage)

Clinical features: depends on the hormone that is deficient and the underlying cause.

Investigations: Check for the specific hormones and look for the underlying cause eg. MRI for pituitary tumour

Treatment: Hormone replacement and treatment of the underlying cause.

3. HYPERTHYROIDISM

Hypothalamus
↓
Thyroid Releasing Hormone (TRH)
↓
Pituitary
↓
Thyroid Stimulating Hormone (TSH)
↓
Thyroid gland releases: T3 and T4

SYMPTOMS OF HYPERTHYROIDISM

1. Weight loss, tachycardia, diarrhoea, oligomenorrhoea, irritability, heat intolerance, tremors, sweating and weight loss despite increased appetite, atrial fibrillation/sinus tachycardia
2. Typical signs of **Graves disease** are exophthalmus, ptosis, diplopia, lid retraction, lid lag

CAUSES:

1. **GRAVES DISEASE**

It is an autoimmune disease. Antibodies resembling TSH are formed and act on the thyroid and stimulate production of T3 & T4. It is associated with other autoimmune disease like type 1 diabetes, Addison's disease, Vitiligo.
There is diffuse enlargement of the thyroid gland. There is bruit and eye signs e.g. diplopia, exophthalmus.

Treatment: Carbimazole. In pregnancy use propylthiouracil. Give beta blockers if no contraindications like asthma.

1. **TOXIC ADENOMA**

It is a benign tumour of the thyroid gland and it produces thyroxine. It is a solitary adenoma, which means there will be a lump in the thyroid which moves on swallowing.

Treatment is radio-iodine.

3. TOXIC MULTINODULAR GOITER

There are multiple nodules.

Treatment is Carbimazole and radiotherapy

1. **SUBACUTE THYROIDITIS** - This is also known as De-quervain's thyroiditis

The cause is viral infection ie. Upper Respiratory Tract Infection. The thyroid is usually painful and enlarged.

Treatment is analgesia or observation

1. **MEDICATIONS:** Amiodarone, thyroxine & lithium

- For **amiodarone**, the patient will be on treatment for arrhythmia (SVT and VT)
- For **thyroxine**, it is usually patients with hypothyroidism and on replacement therapy with levothyroxine
- For **Lithium** these are usually patients being treated for bipolar mood disorder

1. **ECTOPIC TISSUE**- this is thyroxine produced by anywhere else other than the thyroid gland.

INVESTIGATIONS:

1. T3, T4, TSH
2. TSH Receptor antibodies

3. If there is a mass in the neck then USS: if the mass is solid then do FNAC and if the mass is cyst then do surgical removal.
4. Isotope scan=to decide if it is a hot nodule or cold nodule.

Hot nodule - usually indicates a benign adenoma. It accumulates iodine as it manufactures thyroxine.

Cold nodule is usually cancer. It does not take up the contrast.

SUBCLINICAL HYPERTHYROIDISM
This hyperparathyroidism with low TSH or symptoms but normal T3 & T4.

Treatment is observation
Medical treatment is needed only if TSH 0.1 or symptoms of AF, weight loss
Treat with carbimazole if treatment required

4. HYPOTHYROIDISM

CAUSES:

1. **Hashimoto's Disease**
   (Thyroid is diffusely enlarged)
   It's an autoimmune disease and is associated with pernicious anaemia, Diabetes mellitus type 1, Addison disease. Antibodies: anti-peroxidase, Anti-thyroglobulin, anti-microsomal antibodies (thyroid gland is small)

1. **Primary Atrophic Hypothyroidism**
   Diffuse infiltrate, which leads to atrophy of the thyroid. It is an autoimmune disease. There is no goitre.

1. **Iodine Deficiency**
   Common in Africa where water is not iodized.

1. **Thyroidectomy**

1. **Radio-iodine Therapy**

1. **Medications**
   Carbimazole, Lithium (do TFTs and U& E), Amiodarone

SYMPTOMS OF CLINICAL HYPOTHYROIDISM

1. Weight gain, bradycardia, constipation
2. Cold intolerance, Menorrhagia, tiredness, hoarseness, dementia
3. Toad like face, dry skin
4. Goitre
5. Cholesterol raised.

SECONDARY HYPOTHYROIDISM

The cause is low TSH due to problems in the pituitary.

TERTIARY HYPOTHYROIDISM This is due to low TRH in the hypothalamus.
Treatment: Levothyroxine

SUBCLINICAL HYPOTHYROIDISM
High TSH, normal T3 & T4.

Treatment is observation, only treat if TSH > 10 or previous disease or other associations like vitiligo, DM type 1, pernicious anaemia or if there are positive antibodies. Use thyroxine if treatment is required.

SUBCLINICAL THYROID DISEASE

TSH High	T3 and T4 Normal	Subclinical Hypothyroidism
TSH Low	T3 and T4 Normal	Subclinical Hyperthyroidism
TSH High	T3 and T4 Low	Clinical Hypothyroidism
TSH Low	T3 and T4 High	Clinical Hyperthyroidism

5. PARATHYROID GLAND

1. Produces Parathormone, the main function of which is to increase calcium in the blood.
2. Parathormone increases the production of active vitamin D and in turn vitamin D does the following actions.

- Increases reabsorption of calcium from the kidney
- Increases absorption of calcium from the gut
- Increases release of calcium in from the bones.

THE OVERAL EFFECT OF PARATHORMONE IS TO INCREASE CALCIUM IN THE BLOOD.

HYPERPARATHYROIDISM

1. PRIMARY HYPERPARATHYROIDISM

- The commonest cause of hyperparathyroidism is the adenoma of the parathyroid, usually solitary adenoma.
- The second cause is hyperplasia of the parathyroid.

SYMPTOMS OF HYPERPARATHYROIDISM are mainly due to **hypercalcaemia**. These are weakness, tiredness, depression, polyuria, polydipsia, confusion, thirst and abdominal pain and constipation.

Parathyroid adenoma is usually associated with MEN1 (Multiple Endocrine Neoplasia). MEN syndrome consists of

MEN 1

Pancreas tumour =gastrinoma
Parathyroid adenoma
Pituitary adenoma

MEN 2a

Thyroid tumour
Adrenal adenoma
Parathyroid adenoma

MEN 2b

Thyroid
Adrenal
Parathyroid
Mucosal neuromas

Zollinger-Ellison disease is multiple ulcers in the stomach, duodenum and small intestine, which are poorly responsive to PPI and caused by Gastrinomas occurring as MEN 1.

INVESTIGATIONS FOR HYPERPARATHYROIDISM

1. Serum Calcium
2. Parathyroid level
3. Bone scan for osteoporosis
4. USS of the parathyroid and thyroid

Treatment: SURGERY

2. SECONDARY HYPERPARATHYROIDISM

Causes:

1. Deficiency of vitamin D
2. Chronic renal failure→ Active vitamin D is formed in the kidney.
3. Malabsorption

Treatment: Active vitamin D and calcium.

HYPOPARATHYROIDISM

CAUSES:

1. Thyroidectomy: Usually during thyroidectomy the parathyroid glands are removed as well.
2. Symptoms are those of hypocalcaemia ie. tetany and peri-oral parasthesia

Chovestek sign- when tapping on the angle of the jaw there is twitching of the muscles of the face.
Trousseau's sign- when you tie the BP cuff on the arm there is flexion of the forearm and fingers. This sign is also called carpal pedal sign.

Treatment:

1. **Calcium Gluconate intravenously if severe**
2. **Calcium supplements if mild (Oral Ca tablets)**

1. **ADRENAL GLANDS**

HYPOTHALAMUS
↓
Corticotropin Releasing Hormone (CRH)
↓
PITUITARY
↓
ACTH
↓
ADRNAL GLAND

{Glucocorticoid (CORTISOL)
Mineralocorticoid (ALDOSTERONE)
Androgens}

Catecholamine's (adrenaline, noradrenaline)

DYSFUNCTION OF ADRENAL GLANDS

A. HYPO FUNCTION OF THE ADRENAL GLANDS

Addison's Disease - low production of cortisol mainly due to autoimmune disease and infection e.g. tuberculosis

A. HYPER FUNCTION OF THE ADRENAL GLANDS

1. **Pheochromocytoma**- tumour of the adrenal glands from the medulla.
2. **Conn's disease-** adenoma of the adrenal cortex producing aldosterone
3. **Cushing's syndrome**- see below
4. **Virilization**- increased production of the androgens

CUSHING'S SYNDROME

This is excess of cortisol from any cause

CAUSES:

1. ***Cushing disease***
   This is high cortisol due to pituitary adenoma
   This is the commonest cause
   Cushing disease is when the tumor is located in the pituitary and produces high ACTH which stimulates the adrenal gland to produce high cortisol

1. ***Adenoma of the hypothalamus***
   This leads to high production of corticotrophin releasing hormone- high production of ACTH leads to high production of cortisol in the adrenal glands.

1. ACTH produced by lung cancer usually caused by **small cell lung cancer**

1. **Iatrogenic** i.e. patient on treatment for Addison's disease or asthma or COPD.

1. **Adrenal adenoma** - this tumour of the adrenal glands.

Symptoms of Cushing Syndrome:

1. Weight gain
2. Mood changes
3. Central obesity
4. Acne
5. Amenorrhea or irregular menstrual
6. Hirsutism
7. Moon face
8. Buffalo hump
9. Impaired glucose tolerance test
10. Hypertension
11. Abdominal striae
12. Acanthosis Nigricans

DIAGNOSIS

- 1st LINE INVESTIGATIONS (SCREENING TEST)

  Overnight Dexamethasone suppression test or 24 hour urinary free cortisol.

- 2nd LINE INVESTIGATION (CONFIRMATION TEST)

  If any of 1st line test is positive go for 2nd line.
  Which are 48hr Dexamethasone suppression test OR Midnight cortisol/diurnal cortisol

- 3rd LINE INVESTIGATION (LOCALIZATION TEST): To find where is the lesion.

  Plasma ACTH: It is usually not detectable in blood.

A. **If it is increased or detectable**- (May be Ectopic or may be Pituitary cause). Perform high dose Dexamethasone suppression test.

1. If cortisol is suppressed, the diagnosis is Cushing disease. Do MRI of the pituitary because the most likely locations the pituitary gland.
2. If cortisol is not suppressed, the diagnosis is likely to be due to an ectopic tumour. Do CT scan to locate the carcinoid tumour.

B. **Decreased or undetectable** - do CT Scan of adrenal glands. If no mass is visible on the CT scan then perform Adrenal Vein Sampling.

TREATMENT

- Surgery
- If iatrogenic - Remove the cause.

ADDISON'S DISEASE- Low Cortisol

Causes

1. TB (Most common in developing world)
2. Autoimmune (Commonest Cause)
3. Metastasis
4. Steroid
5. HIV
6. Waterhouse Friderichsen Syndrome (Haemorrhage in the adrenal gland if patient has meningococcemia)

Symptoms

1. Fatigue
2. Abdominal Pain
3. Nausea
4. Vomiting
5. Hypotension (hyperkalemic hypotension)
6. Weight loss
7. Anorexia
8. Diarrhoea
9. Constipation
10. Hyperpigmentation
11. Vitiligo

INVESTIGATIONS

Short ACTH Stimulation test (Synacthen test) - Definitive Investigation.
Other investigations: U&E = K+ increased, Na+ decreased, Glucose decreased.

RISK FACTORS

1. Surgery
2. Infection
3. Sepsis
4. Trauma

TREATMENT

- **Replace steroids**
- **Hydrocortisone**
- **If Postural Hypotension- Fludrocortisone**

CONGENITAL ADRENAL HYPERPLASIA

Congenital autosomal recessive disease characterised by cortisol deficiency, with or without aldosterone deficiency and androgen excess.
It has 2 types: ***classic*** **and** ***non-classic***.

CLASSIC: severe form. It's either salt losing or non-salt losing

Symptoms:

FEMALES: Classically presents with ambiguous genitalia with enlarged clitoris and one combined sinus instead of a separate urethra and vagina. May experience salt-losing adrenal crisis.

MALES: classically present with no signs at birth.

- Those with salt-losing form typically present at 7-14 days with vomiting, weight loss, lethargy, dehydration, hyponatraemia and hyperkalaemia.
- Those with non-salt-losing form present with virilisation at age 2-3

NON-CLASSIC: mild or late-onset form, they present with hyperandrogenism in later childhood and early pubarche, infertility, hirsutism, amenorrhoea, polycystic ovaries.

Investigations:

Renal function, electrolytes, blood glucose, serum 17-hydroxyprogesterone, corticotropin stimulation test, pelvic ultrasound, bone age

Treatment:

Classic: **Standard hormone replacement, these include glucocorticoids, mineralocorticoids**

Non-classic: **Treatment only symptomatic.**

CONN'S DISEASE

This is excess aldosterone, which causes Na+ retention

CAUSES:

1. Adrenal adenoma
2. Bilateral hyperplasia

SYMPTOMS:

1. K+ decreased

2. Na+ Increased or normal
3. Weakness
4. Cramps
5. Polyuria
6. Polydipsia
7. Parasthesias
8. HTN

INVESTIGATIONS

1. Aldosterone/ Renin ratio altered or altered Serum Aldosterone
2. CT adrenal

TREATMENT

1. Hyperplasia- Medicine (Spironolactone/Amiloride)
2. Adenoma- Surgery (Spironolactone given 4wks pre-op

PHEOCHROMOCYTOMA

This is due to increased effect of catecholamines, usually due to adrenal tumour.

Rule of 10

10% Malignant
10% Bilateral
10% Extra-adrenal
10% Children
10% Familial
Increased Catecholamines, associated with MEN-2.

SYMPTOMS:

1. Episodic Hypertension and headaches
2. Anxiety
3. Sweating
4. Palpitation
5. Flushing
6. Nausea
7. Vomiting
8. Abdominal pain

Episodes or intermittent symptoms

INVESTIGATIONS:

- 24 hours urinary collection for Catecholamines/Metanephrines

TREATMENT:

A. **CRISIS:**
i. **Phentolamine**
ii. **Labetalol**

A. **STABLE PATIENT:**
i. **Alpha- blocker (Phenoxybenzamine) followed by**
ii. **Beta- blocker (Propranolol)**

Surgery is done after 2 weeks of BP control.

Treatment. Surgical removal of adenoma

7. ACROMEGALY

1. Increased growth hormone (GH)
2. Pituitary tumour (tumour compressing on the optic chiasma)

Hypothalamus
↓
Growth Hormone Releasing Hormone (GHRH)
↓
Pituitary
↓
Growth Hormone
↓
Promotes muscle and bone growth

SYMPTOMS

1. Increase in ring & shoes size
2. Spade like hands
3. Widespread teeth
4. Hoarse voice
5. Carpel tunnel syndrome
6. Excessive sweating
7. Visual field defect→Bi-temporal Hemianopia

8. Coarsening of facies
9. Prognathism
10. Macroglossia

COMPLICATIONS

1. Impaired Glucose Tolerance Test
2. Increase BP
3. Cardiomegaly, Hypertrophy
4. Increase IHD

INVESTIGATIONS:

1. Definitive- OGTT (Oral glucose tolerance test)
2. MRI of the pituitary gland
3. Serum insulin like growth hormone

TREATMENT: Surgery.

8. SIADH -Syndrome of Inappropriate Anti Diuretic Hormone

This is due to overproduction of ADH which leads to reduced production of urine.

Symptoms:

- Water Retention leading to hyponatraemia and hypertension
- Confusion, nausea, and seizure.

CAUSES:

1. Lung cancer- Small cell lung cancer
2. Pancreas Cancer
3. Prostate Cancer
4. As a complication of Meningitis and Head Injury

DIAGNOSIS:

Urine Osmolarity over than 500 mosmol/kg
Plasma Na+< 125 mmol/kg, plasma osmolality <260 mosmol/kg

9. HYPERPROLACTINAEMIA

Hypothalamus
↓
Decreased Prolactin Inhibitory Factor (PIF)
↓
Pituitary
↓
Prolactin
↓
Lactation

This is the commonest hormonal disturbance of the pituitary gland. Raised level of Prolactin (PRL) leads to hypogonadism, infertility, and osteoporosis.

- Normal PRL level is <400 mU/L
- If the PRL is mildy elevated (400-1000 mU/L) then repeat before referral consider look for causes other than a prolactinoma.
- Very high PRL >5000 mU/L usually means that a prolactinoma is present.

CAUSES OF RAISED PLASMA PROLACTIN:

1. Excess production from the pituitary gland by a Prolactinoma
2. Disinhibition, by compression of the pituitary stalk, reducing local dopamine levels.
3. Use of dopamine antagonist

Physiological: Pregnancy, breastfeeding

Drugs: Metaclopramide, haloperidol, antipsychotics

SYMPTOMS:

1. Amenorrhea
2. Infertility
3. Galactorrhea (milk discharge from the nipples)
4. Reduced libido
5. Weight gain
6. Dry vagina
7. Erectile dysfunction in men

INVESTIGATION

- Serum prolactin levels

TREATMENT

- Dopamine agonist e.g.; Bromocriptine.
- Surgery for adenoma.

10. DIABETES INSIPIDUS

This is passage of large volume, greater than 3 litres per day of dilute urine, due to impaired water reabsorption by the kidney

There are two types

1. **Neurogenic/Cranial Diabetes Insipidus:** Reduced ADH secretion from the Posterior pituitary
2. **Nephrogenic Diabetes Insipidus:** Impaired response of the kidney to ADH

SYMPTOMS:

1. Polyuria
2. Polydipsia
3. Dehydration (In dehydration Na+ is high)
4. Hypernatraemia

CAUSES OF NEUROGENIC DI:

1. Idiopathic
2. Congenital
3. Tumor
4. Trauma
5. Hypophysectomy
6. Autoimmune hypophysitis
7. Infiltration → Sarcoidosis
8. Vascular
9. Infection

CAUSES OF NEPHROGENIC DI:

1. Inherited
2. Metabolic
3. Drugs e.g. lithium
4. Chronic renal disease
5. Post-obstructive Uropathy

DIAGNOSIS:- The water deprivation test.

TREATMENT:

-For Cranial DI- Find the cause (MRI- head)
-For Nephrogenic- Treat the cause
-Desmopressin for therapeutic trail

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Resource content	

Ear Nose Throat Lecture notes

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ENT

1. Presenting Complaints

1. Hoarse voice
2. Epistaxis (nose bleed)
3. Dysphagia (swallowing difficulty)
4. A feeling of a lump in the throat
5. Lump in the neck
6. Mouth or tongue ulcers

7. Stridor
8. Facial nerve palsy
9. Nasal obstruction
10. Discharging ear
11. Dizziness and vertigo
12. Otalgia (ear ache)
13. Hearing loss (deafness)
14. Tinnitus=ringing in the ear
15. Facial pain

2. Investigations in ENT

1. Pure tone Audiometry =this is the most commonly performed investigation in ENT. It is done to test for hearing loss.
2. Tympanometry=this is a way of measuring pressure in the middle ear. It used to find the cause of hearing loss, screen for glue ear (Acute Otitis Media) and checking for patency of grommet.
3. Dix-Hallpike manoeuvre is used to check for benign positional vertigo.
4. Caloric tests: they irrigate warm and cold water in each ear and they look for movement of the eyes. It used to check for the integrity of the brainstem-cerebellar pathways and central vestibule-ocular pathways.
5. CT scan is used to assess for the temporal bone and the inner ear, traumatic head injury, in suspected mastoiditis and assessment of mandibular involvement in mouth tumour.
6. MRI: To check for acoustic neuroma MRI is Gold standard, to assess the vascular structure in the neck and head and assessment of spread into cranial of sinonasal tumour
7. RAST (radioallergosorbent testing): to find the cause of allergy in a patent
8. Hearing tests or tuning fork test (use tuning folk 256 or 512 hz)

1. <u>Rinne test</u>
   - AC>BC= positive test = means normal or sensory neural deafness
   - BC>AC= negative test→ conductive deafness
   - Rinne positive on both sides and weber equal on both sides mean normal.
   - Rinne positive on both sides but weber laterizes to the other side mean sensory neural deafness on the opposite sides.
   - Rinne negative on one side and weber laterizes to the same side means conductive deafness on that side.
2. <u>Weber test</u>
   - In the middle→ normal (or equal on both sides)
   - Localizes to affected ear => conductive deafness on that side
   - Lateralizes to one side means sensory neural deafness on the opposite side.

NB: Rinne's test suggets and weber's test confirms.

Neaonatal hearing screening test: 2 tests are used n the UK to screen all the neonates.

1) Automated oto-acustic emissions (AOAE) and

2) Automated auditory brainstem response (AABR).

These test are done between day 10 and 14. For babies who did not require special care usually uses Automated oto-acustic emissions. Babies not passing this test are given the

AABR. And if the test is not passed the n the baby is referred for audiological assessment.

Pneumonic: *CSSO: Conductive deafness same side, Sensory neural Opposite side. (Which means for conductive deafness Weber's goes on the same side and for sensory neural Weber's goes on the opposite.*

3. Infection of the Pinna

Localised infection of the pinna treat with antibiotics such as co-amoxiclav but if there is cellulitis treat with cefuroxime and metronidazole

4. Otitis Externa

This is a common infection in ENT. There is discharge, redness and swelling of the ear but the tympanic membrane is normal.

Risk factors: Swimming, Diabetics and Psoriasis). History of travel abroad for a holiday where possibly patient was swimming. If there is history of travel for a holiday it means there is chance that he could have been swimming in the rivers

Investigation: swab of discharge

Treatment: Gentamicin eardrops and hydrocortisone drops. If you have an option with only Gentamicin drops you an also choose it.

5. Acute Otitis media

This is a very common condition. Almost everyone will suffer with acute otitis media during their lifetime. Signs and symptoms may include a preceding URTI, severe and progressive otalgia, or a discharge this is usually associated with a resolution of the otalgia.

A diagnosis is made by taking a history, examining the tympanic membrane (which is red), and taking the patient's temperature.

In viral acute otitis media the tympanic membrane is pink. In this case antibiotics are not needed just give analgesia.

Treatment for acute otitis media is controversial. Systematic review suggests treatment with analgesia only. However, these reviews may have included a high proportion of viral ear infections where antibiotics would not be expected to be useful.

Management

1) Give analgesia in all cases.
2) Give oral antibiotics for one week such as co-amoxiclav PO or simply amoxicillin.
3) Warn the patient that the discharge may continue for 1 week
4) When the infection has resolved always check that the tympanic membrane has returned to normal.
5) If viral give analgesia.

Recurrent infections of the middle ear

These must be differentiated from one persisting infection. Treat any acute infections actively as above.

If the patient has more than 5 infections in 6 months, then consider alternative treatment such as grommet insertion or a prolonged course of antibiotics.

Treatment

Medical – consider prophylaxis with Trimethoprim (TMP) / Sulfamethoxazole (SMX) syrup – 2mg/kg TMP and 10mg/kg SMX as a single nightly dose PO for 3 months.

Surgical – if there is an effusion or glue ear has been present for longer than 3 months consider grommet insertion especially if there is effusion. AOM + Effusion = Glue Ears and the treatment is grommet.

All treatment needs monitoring – use an infection diary to record episodes of infection pre- and post-treatment.

Caution

Acute otitis media is often misdiagnosed. Children with nocturnal earache often have glue ear/ Eustachian tube dysfunction. The tympanic membrane may be red or infected but there is no discharge and the pain resolves very quickly upon walking.

Complications of Acute Otitis Media

1. Chronic infection

An infection may persist and become chronic. This may be due to resistant bacteria. Use a broad-spectrum antibiotic such as ciprofloxacin.

Consider myringotomy (making small hole in the TM) for the relief of symptoms or to obtain microbiological information.

2. Facial nerve palsy (7^{th} Nerve palsy)

10% of people have a dehiscent facial nerve. This may result in a facial nerve Palsy - when the bone covering is absent over the nerve. This may result in a facial nerve irritation and palsy secondary to the middle ear inflammation.

The patient must be admitted to hospital and given IV antibiotics, e.g. Cefuroxime. Also consider steroid therapy, Prednisolone 1mg/kg per day, if there is total facial paralysis, to be continued for a week.

Consider Myringotomy and grommet insertion if the condition fails in 24 hours.

3. Acute Mastoiditis

This is an infection of the mastoid air cells, which will lead to a severe earache with tenderness, swelling and redness behind the pinna. The pinna also be pushed forwards (displaced) making it look more prominent.

Investigation: CT scan

Treatment: Admit and Intravenous antibiotics (Co- amoxiclav)

4. Chronic perforation of the tympanic membrane

Repeated infections, which perforate the tympanic membrane, can lead to chronic perforation. Usually there is ear, which is followed by purulent discharge, and then the pain disappears.

Discharge = Resolving Earache

5. Sensorineural hearing loss (SNHL)

Rarely, toxins can spread to the inner ear (vestibulocochlear nerve) to produce a sensorineural hearing loss (vestibulocochlear nerve)

6. Vertigo

Infection near the lateral semicircular canal can produce a para –labyrinthitis. This can cause a spectrum of vestibular disturbance ranging from mild unsteadiness to disabling vertigo.

7. Glue ear/otitis media with effusion-this is common in children.

Glue ear is caused by a combination of exposure to infection and a non-functioning Eustachian tube. Almost 8 out of 10 children will have glue ear at some time during childhood. The incidence of glue ear decreases with age as the immune system develops and the Eustachian tube function improves.

The signs and symptoms of glue ear can include: decreased hearing, recurrent ear infection, poor speech development, failing performance at school and sometimes, antisocial behaviour. It causes conductive hearing loss.

Risk factors

1) Smoking parents
2) Bottle feeding

3) Day-care nursery
4) Cleft palate
5) Atopy (eczema, asthma, hay fever)
6) Down syndrome

Investigations

1) Full history and examination (including the palate)

2) Age appropriate audiometry (conductive hearing loss) and tympanometry (to check pressure).

Management

1) Hearing disability – how the child is coping with their hearing problem socially and at school is more important than the actual level of hearing loss.
2) Appearance of tympanic membranes – if there is gross retraction, intervention may be needed to avoid retraction pocket formation.
3) Grommet (tympano Tube)

Treatment

There are three options:

1) Watchful waiting- this should apply to all patients for 3 months as glue ear will resolve in 50 % of cases.
2) Hearing aid – there is a window of opportunity at 4-8 years of age. It is non-invasive, but may lead to teasing at school
3) Insertion of grommets

8. Chronic suppurative otitis media without cholesteatoma

This common condition is associated with Eustachian tube dysfunction with or without an infection in the mastoid.

As with other ear disease, its prevalence continues despite antibiotics.

The sighs and symptoms of chronic suppurative otitis media may include persistent recurrent otorrhoea, perforation in the tympanic membrane (usually central), and no cholestetoma present.

Risk factors

1) Smoking patient
2) Smoking parents
3) Acute otitis media
4) Decreased immunity

Investigation

1) Full history and ENT examination
2) Microscopy of the eardrum with aural toile (washing)
3) Swab for microbiology

Management

1) Give appropriate topical and system antibiotics based on the swab result. The condition may settle with antibiotics and water precautions.
2) Perform regular cleaning of the ear using microsuction –aural toilet.
3) Persistent infections may need surgery

4) Myringoplasty-repair of the perforated tympanic membrane.

5) Cortical mastoidectomy (debridement)

9. Chronic suppurative otitis media with cholesteatoma

This is often divided into congenital and acquired forms of the condition:

Congenital cholesteatoma results from an abnormal focus of squamous epithelium in the middle ear space, i.e. a dermoid.

Acquired cholesteatoma most often results from chronic Eustachian tube dysfunction.

It was hoped that the incidence of this condition would have changed with the advent of antibiotics. Unfortunately, the disease continues and can present at any age. Signs and symptoms may include recurrent otitis media with a mucopurulent discharge, hearing loss, facial nerve palsy, and vertigo.

Development of a cholesteatoma

Initially squamous epithelium migrates out of the sac with ease, but as it enlarges the squamous epithelium builds up and can no longer escape. If infection supervenes on the impacted squamous epithelium/keratin, then lytic enzymes are released causing destruction of local structures. It is normally in the attic of the ear.

Investigation:

CT scan of the temporal bone to look for pneumatisation of mastoid or erosion of scutum.

Management: usually surgical treatment

6. HEARING LOSS=DEAFNESS

The aetiology of hearing loss can be determined by careful consideration of the patient‘s history, a clinical examination, and the findings of special investigations.

The age of onset of the patient’s hearing loss is important, as is any family history of hearing loss.

Classification of patients presenting with hearing loss

Acquired	Congenital
1. Prebyacusis	Syndromic
2. Noise-included hearing loss	Non-syndromic
3. Idiopathic sudden hearing loss	
4. Autoimmune hearing loss	
5. Vascular causes	
6. Ototoxity	
7. Non-organic hearing loss	
8. Otosclerosis	

1. Otosclerosis

Here new bone is *formed around the stapes footplates*, which leads to its fixation and consequent *conductive hearing loss.*

This condition is usually common in pregnancy.

It manifests as slowly *progressive hearing loss,* usually beginning in the patient’s twenties. There is usually a family history of the condition. It is bilateral, common in pregnancy

The patient may have difficulty hearing when chewing and may have problems with quiet conversation. Some 69-80% of patients have tinnitus.

Accelerated progression is often seen during pregnancy.

Bilateral conductive hearing loss. It usually begins in the twenties.

Incidence

- The female to male ratio is 2:1
- Temporal bones have evidence of otosclerosis.
- The population have a clinical manifestation of the disease.
- The condition is bilateral in 70% of patients.
- 50% of patients with otosclerosis have a family history.

Investigations

- Check for a normal mobile intact tympanic membrane.
- Consider a CT scans – this may help to exclude other bony abnormalities of the middle ear causing ossicular fixation.

Differential diagnosis

Paget's disease - this is the only other bony lesion which involves the middle ear. Here there is increased alkaline phosphatase and a mixed hearing loss (conductive or sensory neural sensory loss)

Osteogenesis imperfecta – (also known as Van der Hoeve syndrome) leads to mixed hearing loss with **blue sclera.** There is frequently a history of multiple bony fractures with no history of trauma.

Treatment

The options are:

- No treatment
- Hearing aid as an initial treatment.
- Surgery- stapedectomy after a 3–month trial of hearing aid

2. Presbyacusis

This term describes a decreased peripheral auditory sensitivity. It is usually age-related, and affects men more than women.

Usually starts at the age of 40.

Signs and symptoms

This condition shows itself as bilateral, progressive, symmetrical sensorineural hearing loss, with no history of noise exposure. Decreasing central auditory discrimination leads to phonemic regression.

Investigations

1) Otoscopy
2) Pure tone audiogram

Management

The patient may be given counselling and advice about hearing loss, and given a hearing aid where the symptoms are troublesome

3. Noise-induced hearing loss

This is defined as damage to the inner ear caused by exposure to loud noise. There is a relationship between the volume of sound and its duration, which causes damage.

Signs and symptoms

The patient will usually present with bilateral and symmetrical hearing loss.

There may be a noise-induced temporary threshold shift (TTS) – for example. Hearing may improve over the weekend if the problem is noise at work.

The patient may have difficulty hearing in background noise or they may have tinnitus.

Bilateral sensory hearing loss (SNHL)

Investigation

- Audiometry

Treatment: Hearing aid

Prophylaxis: ear defenders

4. RIFFLE SHOOTING

This usually results in unilateral sensory neural loss, depending on how a person is holding the gun

NB: Acoustic trauma's when sudden very loud noise causes perforation of the eardrum. Sometimes someone can experience if slapped on the ear strongly. There is usually bleeding from the ear.

5. OTOTOXICITY =Drug induced especially by Gentamicin.

Caused by medication such as gentamicin which an aminoglycoside

6. Autoimmune associated with SLE, RA

7. Idiopathic hearing los where there is no cause found.

8. AUTOIMMUNE causes like SLE or RA

9. SYNDROMIC HEARING LOSS

I. Goldenhar syndrome

Either conductive hearing loss or sensory neural hearing loss

Associated with skeletal abnormalities like cervical spine or skull.

Usually a child

Associated with mental retardation or cleft lip or palate

II. Alport syndrome

Sensory neural hearing loss associated with glomerulonephritis

It is progressive in nature. It presents with renal symptoms and hearing loss

Family history of Haematuria (Glomerulonephritis) and hearing loss

NB: syndromic hearing loss is associated with other abnormalities**.**

10. NON-SYNDROMIC HEARING LOSS

This is when a patient is complaining of hearing loss but actually he/she has no hearing loss or when a patient exaggerate the hearing loss

11. Barotrauma=acoustic trauma: Especially when on flight can lead to perforation of the tympanic membrane. This will cause conductive deafness.

12. Acoustic neuroma=tour of the 8th nerve also called swhanoma.

13. Acute otitis media with effusion will cause conductive hearing loss.

7. VERTIGO

Peripheral

1. Meniers disease
2. Benign positional (BPV
3. Vestibular neuritis (labyrinthitis)

4. Vertebrobasilar insufficiency (artheroscerosis)

Drugs due to ototoxicity

1. Gentamicin due to ototoxicity.
2. Diuretics due to postural hypotension
3. Cotrimoxazole
4. Metronidazole

Peripheral vertigo usually has severe vertigo + nausea, vomiting + hearing loss + tinnitus + nystagmus (horizontal)

While in central vertigo hearing loss and tinnitus are less common

1. Benign positioned vertigo: Sudden onset of dizzines lasting> 30 sec. Exacebated by head movement epecially when turning in bed.

Investigation: Dix-Hallpike manoeuvre

Px 1. Self-limiting within months

1. Counselling and reassurance
2. Alcohol
3. Betahistine, prochlorperazine
4. Main treatment is manoeuvre called Epley manoeuvre

2. Meniers disease: (DVT) deafness, vertigo and tinitus

Also nausea and vomiting ,usually recurent episoes . Intermittent symptoms and deafness resolves but dizziness persists

The main thing is that symptoms occiu in episodes.

Treatment: 1. Cyclinzine,

2. Prochlorperazine (phenothiazines works by blocking dopamine)
3. Operative decompression of saccus endolyphaticus

3. vestibular neurectomy/ labrynthectomy if persistent.

Prophylaxis: betahistamine.

8. VESTIBULAR NEURITIS OR LYBIRINTIS: - Symptoms usually come after viral URTI

Sudden vertigo

Vomiting

The main feature is onset of symptoms after viral ilness.

Treatment: cyclinize, improvement occurs in days, full recovery occurs within 2-3 weeks

9. Acoustic Neuroma or verstibular schwanoma: Originates from schwan cell. It is the tumour of the 8th Nerve. It is usually located at the cerebellopontine angle

Vestibular schawanoma test S.N- deafness by compressing cochlear N

- Ipsilateral cerebellar signs
- signs of reaised intracranial pressure

- Dizziness
- Affect other CN 5,6,7,9,10 facial pain or numbness
- Usually there is family history.

Invx : MRI

Treatment: surgery

10. CHRONIC NASAL OBSTRUCTION

Child

1. Large adenoids
2. Rhinitis
3. Postnasal space tumour
4. Foreign body

Adult:

1. Deflected nasal septum
2. Rhinitis (allergic vasomotor)
3. Polyps
4. Sinusitis
5. Granuloma (TB, sphyllis)
6. Topical vasoconstrictor

1. Vasomotor rhinitis: 1. Bilateral nasal obst.
   2. Rhinorrhoea
   3. swollen oedematous turbinate's

TREATMENT:

No definite

Ipratropium nasal spray for Rhinorrhea

Cautery or surgery to reduce ^ info turbinate

2. Allergic Rhinitis: 1. May be seasonal (hay fever) or continuous
   2. Exposure to allergens like pollens, house dusting
   3. Sneezing, pruritis, Rhinorrhoea
   4. swollen turbinate's

Px

1. Desensitizing injury
2. Antihistamine
3. Systematic decongestants

Epistaxis: This is bleeding from the nose the cause is unknown 80%

common in elderly and the commonest cause is hypertension(always check BP)

- More in winter
- After trauma (nose picking)
- Blood dyscaryosis , alcohol

Mx. *Anterior Epistaxis* little's area

1. Sitting position, the head downward
2. Apply pressure for 10-15 minutes on soft part if the nose
3. Insert ribbon gauze with xylometazolinet lidocas
4. Cautery with silver nitrate sticks
5. If persists => anterior packing with Vaseline
6. If persistent do posterir packing

Posterior Epistaxis

1. If bleeding site is seen → bipolar cautery
2. If cannot be seen → posterior packing
3. If still bleeding → examination under GA + diathermy on ligation of syhenopalati ant.

Sinusitis: 1. Fever, facial pain on sinuses which is worse when you bend forward.

4. Nasal discharge, posterior. Nasal drip
5. Nasal obstruction
6. Anosmia

Causes: - bacterial to viral

- Swimming in infected water
- Septal deviation, polyps (predisposing factors)
- Immunodeficiency

Investigation: CT scan + rigid endoscopy

Treatment: Acute: Bed rest, decongestant, analgesia amoxiclav , if no response → drainage with lavage(washout)

STRIDOR

1. Laryngomalacia
2. Laryngitis
3. Epiglotitis
4. Laryngo-tracheo -bronchitis
5. Anaphylaxis
6. Haemangioma, papillomas
7. Trauma (thermal / chemical), intubation

Laryngomalacia

1. Present hours after birth
2. Stridor most noticeable in certain position e.g during sleep or when child is excited;
3. Symptoms are worse when child is in lying positing and sympotms improve when child is sat up.

Treatment : no treatment need, spontaneous resolution within 2 years is usally the course of the disease.

Epiglottitis:

1. Toxic child (child is ill with hugh fever and drooling of saliva)
2. Acute onset
3. Febrile, drooling of saliva
4. Sitting position

5. Voice muffled
6. Continuous stridor
7. The causative organism is Haemophillus influenza)

Rx- do not examine throat, call anaesthetist to intubate the child.

- Laryngocopy to see cherry red swollen epiglottis
- Blood culture
- Cefotaxime

Croup: - This is the same as laryngobronchiolitis , it is commoner the epiglottitis

- Viral cause (parainfluenza)
- Slow onset
- Barking cough
- No drooling of saliva (unlike epiglottitis)
- Stridor only when child is upset

In severe case => cyanosis

Treatment: 1. Steroid: dexamethasone oral or budesonide 2. Nebulised adrenaline. 3. Humidified oxygen.

HOARSENESS OF VOICE

ACUTE

1. Laryngitis this is upper rspiratory tract infection (fever, running nose, sneezing or symply called coryza symptoms)
2. CA larynx (in smokers, elderly patient, weight loss, anaemia, anorexia)
3. Trauma (especially after prolonged intubation)
4. Singing=singer 's nodes
5. Shouting=voice abuse (e.g peolple after a football match)
6. Laryngeal abscess

CHRONIC

1. Laryngitis
2. Vocal cord paralysis due to recurrent laryngeal N. Palsy
3. Functional disorders
4. Laryngeal carcinoma.

11. ENT TUMOURS:

1. SINONASAL MALIGNANCY

This is a group of malignancy affecting the nose and the sinus system

Squamous cell carcinoma account for 70% of all sinonasal tumour

Nickel workers are predisposed to SCC (squamous cell carcinoma)

Wood workers like carpenters are predisposed to adenocarcinoma

Adenocarcinoma accounts for 10% of all tumours

LOCATION OF TUMOURS

1. Maxillary
2. Nasal
3. Ethmoid bone

Symptoms:

Nasal obstruction

Sinusitis

Maxillary symptoms (loose teeth, painful ulcers on the palate, cheek swelling)

Ethmoid bone symptoms (diplopia, nasal obstruction, headache)

2. NASOPHARYNGEAL CARCINOMA

Symptoms:

Epistaxis

Nasal obstruction

Headache

Middle ear effusion

NB: everyone presenting with unilateral middle ear effusion must have postnasal space visualisation to exclude tumour

Rx: radiotherapy and surgery

3. TONSILLAR TUMOUR

1. The commonest is Squamous Cell Carcinoma (SCC)

Common in elderly and middle aged

Pain in the throat, otalgia, ulcer on the tonsils, lump in the cheek

Investigation: FNAC (biopsy)

Rx: surgery with/without radiotherapy

4. LYPHOMA

Second commonest tumour

Enlarged tonsils

Lympadenopathy

-Usually there is no mucosa ulceration. (no ulcer on the tonsils)

Inx: FNAC (biopsy)

Treatment: Radiotherapy

12. OBSTRUCTIVE SLEEP APNOEA

This is usually in obese people. They snore a lot during the night and feel tired most of the time during the day. Also they are sleepy during the day. They are usually hypoxic during the night. They wake up very frequently during the night. This condition is also associated with hypertension

Investigation: Polysomnography or pulse oxymetry during sleep.

13. COMMON OPERATIONS IN ENT SURGERY

1. TONSILLECTOMY

INDICATIONS

1. Recurrent acute tonsillitis
2. Chronic tonsillitis
3. Obstructive sleep apnoea
4. Oral pharyngeal obstruction

COMPLICATION:

Haemorrhage (if between 5-12 days its like infection the cause-this is called secondary haemorrhage.

Treat it with antibiotics.

Bleeding within 24hrs is called primary haemorrhage (usually its due to bleeding from the operation site)

2. ADENOIDECTOMY

INDICATION is adenoids causing obstructive sleep apnoea or adenoid causing glue ear.

This condition is usually in children because in adults the tonsils and adenoid they regress.

3.GROMMET INSERTION: Usually done for glue ear

4. **ANTRAL WASHOUT**: Acute sinusitis not responding to antibiotics in order to obtain microbiology sample

14. FACIAL NERVE PALSY OR 7TH NERVE PALSY

CAUSES

1. BELL PALSY: Idiopathic cause of 7 nerve palsy. Excluding other causes makes diagnosis. The commonest cause is a virus.

Rx: Prednisolone or simply steroid. Usually there is good recovery within 2 weeks

Refer to ophthalmologist due to risk of exposure Keratitis

2. RAMSAY HUNT SYNDROME

7 nerve palsy due to herpes zoster

Vesicles in the ear

Usually in immunocompromised e.g. diabetes, on steroid, HIV, elderly or cancer.

Treatment: Acyclovir oral but if patient immunocompromised then intravenous.

14. **TONSILLITIS:**

Infection of the tonsils

Common causes include B-haemolytic streptococcus, pneumococcal, viral. Symptoms: sore throat, fever, enlarged tonsils, white pus or exudates on the throat it means the cause is bacterial and you must treat with antibiotics.

Treatment: penicillin V. If patient not able to swallow then admit for intravenous antibiotics

Avoid Ampicillin or amoxicillin due to risk of infectious mononucleosis (EBV). If a patient is treated with amoxicillin and the cause was EBV patient may develop rash all over the body.

GLANDULAR FEVER =ALSO CALLED INFECTIOUS MONONUCLEOSIS

Caused by EBV

Symptoms are sore throat with Coryza symptoms

Investigation: monospot or Paul bunnel blood test

Treatment: analgesia, if you give amoxicillin patient will develop rash all over the body.

COMPLICATIONS OF TONSILLITIS:

1. Airway obstruction.

2. Quinsy: peri-tonsillar abscess. There swelling of the soft palate and the uvula is displaced to one side, drooling of saliva and trismus (failure to open the mouth)

Treatment: Incision and drainage

3. Parapharyngeal abscess

Diffuse swelling of the neck

Admit and do incision and drainage.

15. FOREIGN BODIES (FB) IN ENT

FB IN THE EAR

Symptoms include: pain in the ear, deafness, or no symptoms.

Management:

Children need it removed under general anaesthesia

Insect use olive oil

Soft foreign body e.g. cotton wool use crocodile or Tilley's forceps

Solid FB such as a bead uses a wax hook or probes or sometimes suction. DO NOT USE FORCEPS

Refer to ENT if failed attempt or uncooperative child or suspected trauma.

FB IN THE NOSE

Unilateral purulent foul smelling discharge

Nasal obstruction

Epistaxis

Usually in a child

Management:

Ask child to blow the nose

Solid FB like a bead uses a wax hook or probe

Avoid using a pair of forceps as you may push the object forward

Soft FB e.g. cotton wool use crocodile forceps or Tilley's forceps.

FB IN THE THROAT

ACUTE ONSET OF SYMPTOMS

Pricking sensation in the throat

Dysphagia

Pain in the throat

Management: use anaesthetic spray

X-ray of the neck

Use Tilley' forceps

Refer to endoscope if failed attempt, airway obstruction (urgently) or of good history but can not see FB or if visible FB on X-ray

FB OESOPHAGUS

IMMEDIATE ONSET OF SYMPTOMS

Early presentation

Retrosternal pain

Drooling of saliva

Pain

Management: X-ray neck and chest

Endoscope to remove under GA but if GB below oesophagus expectant management that the FB will pass with stool. Warn patient to come back if FB not passed in 48hrs or sign of intestinal obstruction.

NB: sharp objects needs to be removed regardless of the position.

NB: sharp objects need to be removed endoscopically as they can cause a cut to the intestines and lead to peritonitis

16. TRAUMA IN ENT

FRACTURED NOSE=clinical diagnosis, X-ray not required, refer to ENT for follow if there is a lot of swelling which is making examination difficulty.

BASAK SKUL FRACTURE: Rhinorrhhoea or otorrhoea or raccoon eye or battle sign (this is bruises on the mastoid bone)

BONES FRACTURED ARE: EITHIMOID BONE (rhinorrhoea or trauma to the forehead) AND TEMPORAL BONE (otorrhoea or fracture to the mastoid)

Raccoon eyes =bruises around the eye.

Battle sign =mastoid bruising.

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Resource view

Resource name	Epidemiology
Resource description	Epidemiology
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EPIDEMIOLOGY PLAB 1 NOTES:

Absolute risk of a disease is your risk of developing the disease over a time period.

We all have absolute risks of developing various diseases such as heart disease, cancer, stroke, etc. The same absolute risk can be expressed in different ways. For example, say you have a 1 in 10 risk of developing a certain disease in your life. This can also be said to be a 10% risk, or a 0.1 risk - depending if you use percentages or decimals.

Relative risk is used to compare the risk in two different groups of people. For example, the groups could be smokers and non-smokers. Relative risk can either be an increase or a reduced absolute risk.

In general, risk factors increase the absolute risk, and treatment reduces the absolute risk.

All sorts of groups are compared to others in medical research to see if belonging to a group increases or decreases your risk of developing certain diseases. For example, research has shown that smokers have a higher risk of developing heart disease compared to (relative to) non-smokers.

QUESTIONS:

A. In a city in the Bermuda triangle, the absolute risk of developing a disease is 4 in 100 in non-smokers. This risk is increased by 50% in smokers. What is the relative risk of developing a disease in someone who smokes in the Bermuda Triangle?

a. 10%
b. 1 in 10
c. 2%
d. 6%
e. 1 in 4

Say the absolute risk of developing a disease is 4 in 100 in **non-smokers**. Say the relative risk of the disease is increased by 50% in **smokers**. The 50% relates to the 4 - so the absolute increase in the risk is 50% of 4, which is 2. So, the absolute risk of smokers developing this disease is 6 in 100, which is 6%.

B. In Mozambique, 2 in 20 men develop prostate cancer by the age of 60. New research suggests that taking medication X would reduce the risk of developing this disease by 50%. What is the risk of developing prostate cancer in someone who is taking medication X?

a. Relative Risk 10%
b. Absolute Risk 10%
c. Relative Risk 5%
d. Absolute Risk 5%
e. Relative Risk 2%

These men have a 2 in 20 risk (which is 10%) of developing prostate cancer by the time they reach the age of 60. Research shows that a new treatment reduces the relative risk of getting this disease by 50%. The 50% is the relative risk reduction, and is referring to the effect on the 2. 50% of 2 is 1. So this means that the absolute risk is reduced from 2 in 20, to 1 in 20 (which is 5%).

NUMBER NEEDED TO TREAT:

This is the number of people who need to take the treatment for one person to benefit from the treatment.

In Toronto, the absolute risk of developing a myocardial infarction is 4 in 100. A pharmaceutical company reported that medicine X reduced the relative risk of developing this disease by 25%. What is the number needed to treat (NNT)?

a. 100
b. 50
c. 4
d. 3
e. 1

If the absolute risk of developing the disease was 4 in 100 (which is 4%) then this 25% reduction in relative risk would reduce the absolute risk to 3 in 100 (which is 3%). Therefore, the reduction of the absolute risk after treatment is by 1%. In other words, 100 people need to be treated to save 1 person.

SUMMARY:

$$NNT = \frac{100}{\text{Absolute Risk Reduction}}$$

Absolute risk reduction = Absolute Risk – Relative Risk

Absolute risk = the risk of developing a certain disease in a given period of time/life time.

Relative risk = an increase in the absolute risk or a decreased absolute risk.

i.e. The risk can increase from point A to point R **(A = absolute risk, R= relative risk)**

Treatment can reduce the risk from point A to point R.

Therefore:

$$NNT = \frac{100}{\text{A - R (Absolute reduction in risk)}}$$

EXAMPLE:

The absolute risk of developing complications from a certain disease is 4 in 20. Research shows that medicine X reduces the relative risk of getting these complications by 50%. What is the number needed to treat?

a. 20
b. 10
c. 5
d. 2
e. 1

The absolute risk is reduced from 4 in 20, to 2 in 20. In percentage terms, 4 in 20 is 20%, and, 2 in 20 is 10%.

Therefore, the reduction in absolute risk in taking this medicine is from 20% to 10% - a reduction of 10%. The NNT would be 100 divided by 10. That is, 10 people would need to take the medicine for one to benefit.

$$NNT = \frac{100}{(20\% - 10\%)} = \frac{100}{10\%} = 10$$

TYPES OF TRIALS:

There are many types of studies that explore causal connections.

A. **Case-control Study:**

The frequency of the risk factor (e.g. smoking) is compared between 2 groups, those with the disease (e.g. lung cancer) and those without the disease (control group). This study is ***retrospective***, in that it starts after the onset of the disease.

A. **Cohort Study**:

The study group consists of subjects exposed to the risk factor (e.g. smoking) and the control group comprises of unexposed people. The incidence of the disease is then compared between the groups over time, ***prospectively***.

BLINDING:

If the subject doesn't know which of the 2 trial treatments he/she is having, this trial is known as **single blind**.

To further reduce the risk of bias, the experimenter should also not know, then this trial is known as **double blind**.

In a good trial, the blind lead the blind.

RANDOMISED CONTROL TRIAL:

A study in which a number of similar people are randomly assigned to two (or more) groups to test a specific drug or treatment.

One group (the experimental group) receives the treatment being tested, the other (the comparison or *control group*) receives an alternative treatment, a dummy treatment (*placebo*) or no treatment at all. The groups are followed up to see how effective the *experimental treatment* was. *Outcomes* are measured at specific times and any difference in response between the groups is assessed statistically. This method is also used to reduce *bias*.

CONFIDENCE INTERVAL:

There is always some uncertainty in research. This is because a small group of patients is studied to predict the effects of a treatment on the wider *population*. The confidence interval is a way of expressing how certain we are about the findings from a study, using statistics. It gives a range of results that is likely to include the 'true' value for the population.

The CI is usually stated as '95% CI', which means that the range of values has a 95 in a 100 chance of including the 'true' value. For example, a study may state that 'based on our *sample* findings, we are 95% certain that the 'true' population blood pressure is not higher than 150 and not lower than 110'. In such a case the 95% CI would be 110 to 150.

A wide confidence interval indicates a lack of certainty about the true effect of the test or treatment - often because a small group of patients has been studied. A narrow confidence interval indicates a more precise estimate (for example, if a large number of patients have been studied).

CONTROL GROUP:

A group of people in a study who do not receive the treatment or test being studied. Instead, they may receive the standard treatment (sometimes called 'usual care') or a dummy treatment (*placebo*). The results for the *control group* are compared with those for a group receiving the treatment being tested. The aim is to check for any differences.

Ideally, the people in the control group should be as similar as possible to those in the treatment group, to make it as easy as possible to detect any effects due to the treatment.

PLACEBO AND PLACEBO EFFECT:

A fake (or dummy) treatment given to participants in the *control group* of a *clinical trial*. It is indistinguishable from the actual treatment (which is given to participants in the *experimental group*). The aim is to determine what effect the *experimental treatment* has had - over and above any *placebo effect* caused because someone has received (or thinks they have received) care or attention).

RETROSPECTIVE STUDY:

A research study that focuses on the past and present. The study examines past exposure to suspected *risk factors* for the disease or condition. Unlike *prospective studies*, it does not cover events that occur after the study group is selected.

PROSPECTIVE STUDY:

A research study in which the health or other characteristic of participants is monitored (or 'followed up') for a period of time, with events recorded as they happen. This contrasts with *retrospective* studies.

PREVALANCE:

Used to describe the proportion of people in a *population* who have a particular habit, a particular disease or another characteristic. For example, smoking prevalence relates to the proportion of people who smoke in a given population. Prevalence may be expressed in relation to a range of factors including age, sex, socioeconomic and ethnic group. See also *incidence*.

ABSOLUTE RISK REDUCTION (ARR):

A reduction in the likelihood of an event or outcome occurring as a result of a treatment or another intervention. For example, if a treatment reduces the *absolute risk of death* from 0.25 (25%) to 0.10 (10%), the ARR is 0.15 (15%), that is, 0.25 minus 0.10 equals 0.15.

The estimate of absolute risk reduction often comes from clinical trials. The percentage of people taking part who are receiving treatment (treatment group) and experience a specific outcome is compared with the percentage of people taking part but not receiving treatment (*control group*) who experience the same outcome.

MEAN:

Mean is the average of a group of values.

MEDIAN:

Median is the middle value when the values are ranked

MODE:

Mode is the value that occurs most frequently

SENSITIVITY:

Sensitivity is the proportion of true positives correctly identified by a test

SPECIFICITY:

Specificity is the proportion of the true negatives correctly identified by the tests.

INCIDENCE:

Incidence is the number of new cases divided by the total population per year who are at risk of becoming a case/diseased.

POSITIVITY:

- **True positive**
  - The individual has the condition and tests positive for the condition
  - The individual does not satisfy the null hypothesis and the test rejects the null hypothesis
- **True negative**
  - The individual does not have the condition and tests negative for the condition
  - The individual satisfies the null hypothesis and the test accepts the null hypothesis
- **False positive**
  - The individual does not have the condition but tests positive for the condition
  - The individual satisfies the null hypothesis but the test rejects the null hypothesis
- **False negative**
  - The individual has the condition but tests negative for the condition
  - The individual does not satisfy the null hypothesis but the test accepts the null hypothesis

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Resource name	Genetics PLAB 1
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GENETICS LECTURE NOTES 2014:

Genetic disease = are those diseases which are inherited from the parents and can be transmitted to the next generation.

Genetic counseling is the process by which individuals or relatives are at risk for disorder that may be hereditary are advised of the consequences of the disorder, the probability of transmitting it and ways of in which this may be prevented, avoided or ameliorated.

COMMON CHROMOSOMAL DISEASES

1. **Down Syndrome**

- Chromosomal abnormality,
- Trisomy 21
- Usually present at birth
- **Clinical features** (small low set ears, up-slanting eyes, prominent epicanthic folds, flat facial profile, protruding tongue, short neck, mild short stature, short broad hands

Associated conditions:

- Congenital heart disease e.g. VSD< ASD, fallot's tetralogy
- Deafness
- Dementia
- Cataract

Investigation: chromosomal analysis showing additional chromosome 21

Management: Patient needs cardiac investigations and testing for hearing.

1. **Klinefelter's syndrome**

- Boys with klinefelter syndrome enter puberty normally but by mid puberty the testes become small
- Infertility due to azoospermia, Gynaecomastia. They have decreased testosterone.

Investigation: Chromosomal analysis.

Test to prove the azospermia is testosterone.

1. **Patau syndrome**

- Trisomy 13
- Microcephaly, Microphthalmia
- Cleft lip and palate

Investigation: chromosomal analysis

1. **Edwards Syndrome:**

- Trisomy 18
- Features: Congenital heart disease
- Low birth weight
- Overriding fingers
- Usually they die within 4 days. Rarely they can live several months.

1. **Turner's syndrome**

- Most girls have single chromosomal (45, XO)
- Features: Short stature, broad neck, ptosis, widely spaced nipples, congenital heart disease,

GENETIC DISORDERS WITH CARDIAC FEATURES

1. Marfan syndrome

- Autosomal dominant multisystem disorders caused by mutation in the in the chromosome 15.
- Features: Include, tall and slim body build with long limbs scoliosis, long fingers, aortic aneurysm

1. Di George syndrome

- Short stature
- Congenital heart abnormality e.g. tetrology fallot
- Prominent nasal bridge

1. Williams syndrome

- Autosomal dominant
- Peri-orbital fullness, short stature, congenital heart abnormality chest deformity.

GENETIC DISORDERS WITH LEARNING DISABILITY

1. **Fragile X syndrome**

- It is commonest inherited disease of mental retardation
- Boys with Fragile X syndrome usually have global developmental delay
- Can affect both girls and boys
- They boys have stereotyped repetitive behavior such as hand flapping and resistance to change of routine.

1. **Prader-Willi Syndrome**

- Babies are frail and may fail to thrive
- Older children have learning difficulty and short stature.

Genetic disorders with neuromuscular features:

1. **Congenital Myotonic Dystrophy**

- It is an autosomal dominant with onset usually in adult life
- At birth the baby is floppy
- During pregnancy there is polyhydrominous

- Usually the affected children are from women who are also have myotonic dystrophy

1. **Duchene muscular dystrophy**

- Presents with development delay
- Child climb up his thigh when standing up (Gower's sign)
- Diagnosis is by genetic testing
- X-linked recessive
- Mean age of onset is 5 years.

1. **Spinal muscular atrophy**

- Autosomal recessive disorders
- Symmetrical proximal weakness as a consequence of degeneration of anterior horn cells of spinal cord

-

Investigation: genetic molecular testing

GENETIC DISORDERS WITH DERMATOLOGICAL FEATURES

1. **Ehlers Danlos syndrome**

- Autosomal dominant
- Hypermobility of small and large joint with soft skin
- Soft skin which hyper-extensive

1. **Neurofibromatosis**

- NF1 has a autosomal dominant disease
- Café au lait spots
- Neurofibroma
- Chance of transmitting it to children is 1:2

1. **X-linked hypohydrotic ectodermal dysplasia**

- It is X linked recessive
- Boys have reduced sweating which may cause dangerous hyperpyrexia in infancy

1. **Tuberous sclerosis**

- Autosomal dominant
- Characterized by hamartoma's on the skin, brain and other organs
- Commonly presents with infantile spasm, seizures and mental retardation

OTHER INHERITED DISEASES

1. Polycystic kidney disease:

An autosomal dominant disease is associated with vascular abnormality like berry aneurysm. The kidney is usually palpable bilaterally and patient has hypertension.

1. Cystic fibrosis:

This is an autosomal recessive disease: meaning there is 1:4 chance of transmitting to another child. Child presents with failure to thrive and pancreatic insufficiency and recurrent chest infection.
Investigation: sweat test

1. Glycogen storage disease:

Specific enzyme defects preventing mobilization of glucose from glycogen and resulting in abnormal storage in liver and muscle. Patient usually presents with hypoglycemia in the morning.
This is autosomal recessive condition usually presenting with hypoglycemia, lactic acidosis, poor growth, mental retardation.

1. Haemophilia:

This is a X-linked recessive disease affecting male children and presents with bleeding into the muscles and joints.

1. Achondrioplasia:

Short limbs, autosomal dominant lumbar lordosis, large head.

1. Von Willebrand's:

An autosomal dominant disease causes high bleeding time and bruises and bleeding.

POLYGENIC INHERITANCE DISEASE:

These are disease which can run in the family but there needs to be interaction with external environment which include the following:

A. Diabetes
B. Multiple sclerosis

AUTOSOMAL RECESSIVE DISEASES:

1. Sickle Cell Disease
2. Thalassemia
3. Cystic Fibrosis

AUTOSOMAL DOMINANT DISEASES:

1. Polycystic Kidney Disease
2. Huntington Disease
3. Neurofibromatosis
4. Von Willibrand Disease

X-LINKED DISEASES:

1. Duchene Muscular Dystrophy
2. Haemophilia

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GASTRO- ENTEROLOGY

OVERVIEW OF TOPICS

1. **Dysphagia**
2. **Dyspepsia**
3. **Peptic Ulcer**
4. **Zollinger Ellison Syndrome**
5. **Gastro-oesophageal reflux disease**
6. **Inflammatory Bowel Disease**
7. **Irritable Bowel Syndrome**
8. **Coeliac Disease**
9. **Jaundice & Liver Diseases**
10. **Primary Biliary Sclerosis**
11. **Primary Biliary Cirrhosis**
12. **Sclerosing Cholangitis**

13. **Primary Sclerosing Cholangitis**
14. **Alpha 1- antitrypsin deficiency**
15. **Hereditary Haemochromatosis**
16. **Pancreatic Diseases**
17. **Pancreatitis**
18. **Pancreatic Cancer**
19. **Diarrhoea**
20. **Constipation**
21. **Colorectal Carcinoma**

A. DYSPHAGIA

CAUSES	SIGNS & SYMPTOMS	INVESTIGATION	MANAGEMENT
OESOPHAGEAL CANCER	Progressive dysphagia (dysphagia of solids then liquids), old age , weight loss, anemia (pallor, fatigue, palpitations), anorexia	1. Barium swallow (initial investigation) 2. Endoscopy & biopsy as definitive diagnosis	**Surgery** resection if no metastasis **Radiotherapy** if with metastasis
ACHALASIA Pathology in myenteric pleuxs. Failure of the lower oesophageal sphincter to relax, due to decreased in ganglion cells in the oesophageal wall	Dysphagia of both fluids and solids Regurgitation Weight loss	Barium swallow	Dilatation or Heller's myotomy
DIFFUSE OESOPHAGEAL SPASM –abnormal oesophageal motility	Intermittent dysphagia +/- chest pain due to reflux which may happen with this condition	Barium swallow	Dilatation
SCLERODERMA – replacement of the smooth surface with fibrosis leading to decreased oesophageal sphincter function	Reflux		
PHARYNGEAL POUCH	Regurgitation of undigested food Sensation of lump in the throat Halitosis Bulging of neck on drinking Aspiration into lungs	If presents as dysphagia à Barium swallow If presents as neck mass à ultrasound scan	Surgical excision
FOREIGN BODY	If child then while playing	Barium swallow	

	with toys & usually child is normal & healthy. If adults then during eating chips, meat, fish etc.		Removal of the foreign body by endoscopy
OESOPHAGEAL STRICTURE	Due to ingestion of corrosives e.g. acid and due to GERD		Dilatation of the stricture
MOTOR NEURONE DISEASE (Bulbar palsy)	Dysphagia + weakness of the lower limbs, usually male patient, middle aged, no sensory loss / no sphincter abnormalities, eye muscles not affected		
PLUMMER VINSON SYNDROME/ PATERSON BROWN KELLY SYNDROME – there is post – cricoid web. This is due to spasm of upper oesophageal sphincter	Iron deficiency anaemia usually female patient, koilonychia		Treating anaemia by iron tablets
LOWER OESOPHAGEAL RING: Schazki ring	Narrowing of the lower end of the oesophageal sphincter, the mucosa forms a ring	Barium swallow	Reassurance
STROKE	Sudden + symptoms like hemiplegia, visual defects, usually in old patients	CT scan	
GLOBUS HYSTERICUS	Feels lump in the throat which need to be swallowed. Associated with anxiety, this can be complication of GERD.		
OESOPHAGEAL CANDIDA	White plaques or ulceration in the mouth, in immune-compromised patient e.g. HIV		Anti – fungal (fluconazole)
OESOPHAGITIS			

	Complication of GERD with symptoms of reflux		PPI or H2 antagonist
MYASTHENIA GRAVIS	Dysphagia + other symptoms like diplopia, fatigue by the end of the day, weakness of the eye muscles, tiredness + diplopia at the end of the day, Fatigue + dysphagia		
CHAGAS DISEASE	After many years of the disease can cause dilatation of the oesophagus (mega – oesophagus) leading to dysphagia		
EXTRINSIC PRESSURE (LUNG CANCER), RETROSTERNAL GOITRE, AORTIC ANEURYSUM, LEFT ATRIUM ENLARGEMENT	General symptoms of cancer Symptoms of thyroid Different blood pressure in each arm, pain radiating to the back History of mitral valve disease		Treating the cause
PARKINSONISM	Shuffling gait Dizziness Ataxia Nystagmus, resting tremors, cog wheel/lead pipe rigidity		

DYSPEPSIA

This is epigastric pain or discomfort with or without nausea and vomiting.

ALARMS 55

A - Anorexia

L - Loss of weight

A - Anaemia

R - Recent onset of progressive symptoms

M - Melena or haematemesis

S - Swallowing difficulties

55 - Patient 55 or more years old

STEPS OF MANAGEMENT FOR NEW DYSPEPSIA

1. If age **> 55** years or **ALARM S**igns present then perform **urgent endoscopy.**
2. If age **< 55** years and no **ALARM S**igns then

   Treat with lifestyle modifications, simple antacids +/- anti-reflux for **4 weeks.**

- **If symptoms resolve** then **no further action** required.
- If symptoms **persist** then do **test for H.Pylori.**

1. If **H. Pylori test is positive** then **eradicate H. Pylori** and review after 4 weeks.

- If symptoms resolve then no further action required.
- If symptoms persist then do a **urea breath test**.
- If this is positive then continue H. Pylori eradication.
- If this is negative then consider **UGI endoscopy**.

1. If **H. Pylori test negative** then give **PPI's or H2 blockers** x 4 weeks e.g. **Omeprazole 20 mg/24hrs or Ranitidine 150 mg/12 hrs.**

- If there is improvement then no further action required.
- If there is no improvement then give longer term, low dose treatment of the same medications and consider UGI endoscopy.

Triple therapy for eradication of Helicobacter pylori:

1. PPI
2. Clarithromycin
3. Amoxicillin or Metronidazole

DIFFERENTIAL DIAGNOSIS

1. Peptic ulcer
2. GERD
3. Mallory-Weiss tear
4. Oesophagitis
5. Gastric carcinoma
6. Gastric erosions due to (NSAIDS, steroids, gastrinoma)

PEPTIC ULCER

- **Gastric ulcer**: Patients usually present with epigastric pain after meals +/- relieved by antacids, patients usually lose weight due to fear of pain when they eat.
- **Duodenal ulcer:** Patients usually experience pain at night or before a meal. They are also called hunger ulcers.

Pathogenesis of Peptic Ulcer:

Aggravating Factors	Protective Factors
Helicobacter pylori HCl Pepsin	HCO3 Mucus Prostaglandins

Investigations in Peptic Ulcer Disease:

H. pylori test can be performed with different techniques:

1. **Urea Breath Test**
   a. **Pre-treatment test (before performing endoscopy): best test option is the carbon-13 urea breath test or stool antigen test for H. pylori.**
   b. **Post – eradication testing: best performed using carbon-13 urea breath test**

N.B: Before performing H. pylori test, stop PPI and antibiotics to reduce the chance of false positive.

1. **Endoscopy – gold standard investigation but not practical to be done in all patients.**
   a. **Done to visualize ulcers which are usually clean and smooth with sharp margin**
   b. **Has the benefit of taking biopsy for suspicious lesions and test for H. pylori**

1. **Barium studies – may be used if patient is refusing endoscopy.**

Zollinger – Ellison Syndrome

- It is due to a gastrinoma in the pancreas which causes increased HCl leading to multiple ulcers in the stomach, duodenum & small intestines (it's an unusual ulcer and doesn't respond to antacids)
- About 20 - 25 % of gastrinoma patient have MEN-1 syndrome

Investigations: High fasting serum gastrin or pancreas tumour

Management:

- **Medical therapy → Proton Pump Inhibitor**
- **Surgical therapy → Removal of tumours**

Gastro-oesophageal reflux disease:

- Dysfunction of the lower oesophageal sphincter predisposes to the reflux of acid from the stomach to the oesophagus
- Associated with smoking, obesity, alcohol, rolling/sliding hiatus hernia – displacement of the part of the stomach into the thorax (rolling hernia can cause strangulation), pregnancy, obesity, big meal

Investigations: **endoscopic** or **clinical diagnosis**

Endoscopic evidence: Oesophagitis Clinical evidence: Heart burn

There are two types of symptoms: Typical and Atypical

Typical symptoms

1. Heartburn/ retrosternal burning or discomfort related to meals or lying down therefore pain at night, pain is relieved by antacids
2. Water brush = excessive salivation
3. Odynophagia (painful swallowing) if there is oesophagitis

Atypical symptoms

1. Globus hystericus
2. Recurrent pneumonitis
3. Angina like chest pain

Complications:

- Oesophagitis
- Oesophageal stricture
- Barrett's oesophagus/CLO (Columnar line oesophagus) - transformation of squamous cells into columnar cells in the oesophagus, **pre- malignant stage** – needs regular endoscope surveillance
- Oesophageal adenocarcinoma

Medication:

1. Simple antacids (Magnesium Trisilicate)
2. If oesophagitis confirmed → Proton Pump Inhibitor
3. Prokinetics like metoclopramide, they help emptying of the stomach if there is gastric paresis

Main Points In Managing GERD

1. If age **> 55** years or **ALARM S**igns present then perform Urgent endoscopy

1. If age < 55 years and no **ALARM S**igns then lifestyle modifications plus antacids for 4 weeks are the first line steps of management in patient with typical GERD symptoms. Eg.

- Lose weight
- Stop smoking
- Small regular meals
- Raise the head end of the bed
- Avoid hot drinks, alcohol
- Avoid eating < 3 hours before bed

1. If lifestyle modifications not helpful, offer Proton Pump Inhibitor for 4 weeks.

- If symptoms resolve then no further action
- If symptoms persist, perform an **endoscopy**

A. **If endoscopy is positive** ie. shows oesophagitis then prescribe PPI/H2 antagonist

- If symptoms resolve with PPI/H2 antagonist then no further action
- If symptoms persist even with PPI/H2 antagonist then perform Fundoplication

A. **If endoscopy is negative** (ie. normal) then do 24 hour pH monitoring

- If 24 hour pH monitoring is +ve prescribe PPI/H2 antagonist (if symptoms persist even with PPI, do fundoplication)
- If 24 hour pH monitoring is –ve then check for alternative diagnosis

1. If symptoms persist after fundoplication, do endoscopy first and then pH monitoring (if required) to check if there is still ongoing reflux.

INFLAMMATORY BOWEL DISEASE

	Ulcerative Colitis	Crohn's Disease
Associations	Affects mucosa and sub-mucosa, Formation of ulcers Mucosa is friable and bleed easily No skip lesions Rectosigmoid is commonly involved with 50% Never spreads proximally to the ileo-caecal valve Common in NON SMOKERS	Affects mouth to anus Transmural inflammation Granulomatous lesion Terminal ileum is 50% Skip lesions with rectal sparing (usually) Cobblestone ulceration as a result of aphthous ulceration progression to oedema and nodular thickening Rose thorn appearance Associated with smoking & low fibre diet intake
Signs & Symptoms	Gradual onset Chronic diarrhea with blood & mucus ¯ weight Urgency & tenesmus occur with rectal disease Extra-intestinal symptoms are clubbing, aphthous ulcers, erythema nodosum, pyoderma gangrenosum, conjunctivitis, arthritis, ankylosing spondylitis, cholangio-carcinoma Associated with primary sclerosing cholangitis	Chronic diarrhea ¯ weight Aphthous ulcer Abdominal tenderness Right iliac fossa pain Perianal abscess / fistula / skin tags Fistulas elsewhere Granulomata Strictures and ulcers SAME extra intestinal symptoms as UC
Investigations	Sigmoidoscopy – granular inflammation, ulcers, crypt abscesses	Sigmoidoscopy Barium enema Colonoscopy + Biopsy
Treatment	**Mild**: < 4 motion/day & patient is well = prednisolone + mesalazine (amino salicylates) **Moderate**: 4-6 motion/day & patient is well = oral prednisolone + mesalazine + twice daily steroid enema **Severe**: IV hydrocortisone + twice daily enemas **Prevention of relapse**: aminosalicylates (mesalazine, sulphasalazine)	**Mild**: prednisolone **Severe**: hydrocortisone + metronidazole The role of aminosalicylates is prevention of relapse in Crohn's is less effective than in UC
Complications	1. **Perforation** - do erect Chest X-Ray 2. **Colonic carcinoma -** risk is 15% 3. **Toxic megacolon** (dilatation of colon) can lead to perforation	

IRRITABLE BOWEL SYNDROME:

Group of abdominal symptoms with "NO ORGANIC CAUSE" which can be found.

Signs & Symptoms:

- Patient is usually 20 – 40 years old
- Central abdominal pain relieved by defecation
- Abdominal bloating
- Altered bowel habits < constipation alternating with diarrhoea
- Chronic > 6 months
- Exacerbated by stress or eating

Investigations: **all investigations and examinations are normal therefore it is diagnosis by exclusion**

NB. Clues to rule out other causes:

- **No weight loss**
- **No bleeding**

- **No night symptoms i.e. diarrhoea or abdominal pain at night**
- **All investigations are normal**

Treatment:

Symptomatic e.g. for pain mebeverine hydrochloride (anti- spasmodics)

COELIAC DISEASE

This is a T – cell mediated autoimmune disease of the small bowel in which protamine causes villous atrophy and malabsorption

Signs & Symptoms:

- Steatorrhoea
- Abdominal pain
- Weight loss / failure to thrive in children
- Weakness
- Folic acid or iron deficiency anaemia

Investigations:

Antibodies

- **Anti - endomyseal**
- **Anti - α gliadin**
- **Anti – transglutaminase**
- **Ig A anti body duodenal biopsy**

Treatment:

Life long gluten free diet

Complications:

1. Iron deficiency anaemia
2. Folate deficiency anaemia
3. Osteoporosis
4. Lymphoma
5. Secondary lactose intolerance

JAUNDICE & LIVER DISEASES

General Approach

A: OBSTRUCTIVE JAUNDICE: ALP and/or GGT is higher than AST and ALT. Bilirubin is high as well.

B. HEPATIC PICTURE: ALT and/or AST raised higher than ALP or GGT. Bilirubin is high as well.

C. ISOLATED RISE IN BILIRUBIN:

Unconjugated bilirubin: Hemolysis, Gilbert syndrome (congenital disease)

Conjugated bilirubin: Dubin -Johnson syndrome (congenital disease)

D. ISOLATED RISE IN GGT: Suggests alcohol abuse

E. ISOLATED RISE IN ALP: Usually not due to liver disease but to a bone disease like bony metastasis, Paget's disease, osteomalacia

1. **PRE-HEPATIC JAUNDICE**

Causes:

- Hereditary haemolytic anaemia
- Acquired haemolytic anaemia, malaria

Signs & Symptoms: anaemia + jaundice

Investigations:

1.↑ unconjugated serum bilirubin

2. Evidence of haemolysis: ↓haemoglobin,↑reticulocylosis

3. ↓haptoglobin

4. ⊥ FT (N)

2. HEPATIC JAUNDICE

Causes:

a. **VIRAL HEPATITIS** A,B,C,D,E

- If acute → A, E (Faeco - oral route)

- If chronic → B, C, D (Body – fluids)

Risk Factors for Hepatitis B & C: IV drug abuser, Blood transfusion, Homosexual

b. ALCOHOL HEPATITIS

Strong history of alcohol drinking

Sign's: presence of spider naevi + stigmata of chronic liver disease, ↑GGT

c. DRUG INDUCED – eg. paracetemol

d. GILBERT'S SYNDROME - impaired conjugation

3. POST – HEPATIC JAUNDICE (OBSTRUCTIVE JAUNDICE)

Causes:

1. Common bile duct stones
2. Tumours of head of pancreas ⟶ Courvoisier's sign (presence of palpable painless gall bladder + jaundice unlikely due to gallstones)
3. Primary biliary cirrhosis
4. Primary biliary sclerosis
5. Sclerosing cholangitis
6. Primary Sclerosing Cholangitis
7. Alpha 1 –Antitrypsin Deficiency
8. Hereditary haemochromatosis

Signs & Symptoms:

- Pale stools + dark urine
- ↑ Serum conjugated bilirubin
- No urobilinogen in urine
- ↑↑ Alkaline phosphatase

PRIMARY BILIARY CIRRHOSIS

Chronic inflammation of autoimmune origin which leads to destruction of interlobular bile ducts in the liver causing progressive cholestasis, cirrhosis & portal hypertension

Signs & Symptoms:

It is usually asymptomatic but can have the following symptoms:

1. Pruritis - causing scratch marks on the body
2. Jaundice
3. Skin pigmentation which is progressive in nature & starts in an early age
4. Hepatomegaly
5. Splenomegaly
6. Osteoporosis
7. Usually young female patients

Investigations:

1. **↑ALP,↑GGT & mildly raised AST AND ALT**
2. **Ultrasound scan to rule out bile stones**
3. **Anti-mitochondrial antibodies**

Treatment:

Cholestyramine for pruritis
Urodeoxy - cholic acid improves cholestasis

PRIMARY SCLEROSING CHOLANGITIS:

Cause unknown, there is an immunological theory, there is fibrosis & stricture of the intra & extra hepatic bile ducts.

Signs & Symptoms: Pruritus with or without fatigue. In advanced cases there may be ascending cholangitis, cirrhosis and end-stage hepatic failure

Investigations:

- **↑ ALP**
- **ERCP shows multiple strictures**

Treatment:

- **Liver transplant is the mainstay for end-stage disease**
- **Cholestyramine for pruritus**
- **Ursodeoxy – cholic acid**
- **Antibiotics if there is cholangitis**
- **Endoscopic stenting helps relieve strictures**
- **Yearly ultrasound scans to check for cholangiocarcinoma**

Complications: Cancer may occur in the bile duct and gall bladder. Cancer of the liver and colon cancer are more common.

Monitoring: Regular LFT, ultrasound and AFP. It is also advisable to perform yearly colonoscopy.

α 1 –ANTITRYPSIN DEFICIENCY

The glycoprotein α1 – anti trypsin is one of the family of serine protease inhibitors.

1. The defect means it cannot be released from the liver where it synthesized, therefore accumulation leads to **liver disease as a child.**
2. Its function is to oppose protease therefore without it protease activity is unopposed and it causes to destruction of the alveoli leading to **emphysema as an adult.**

Investigation:

- **↓ serum anti-trypsin level**
- **Liver biopsy**

Treatment: Supportive

HEREDITARY HAEMOCHROMATOSIS:

This is an inherited disorder of iron metabolism in which there is ↑ intestinal iron absorption, which tends to deposition in all organs

Signs & Symptoms:

Asymptomatic in early stages then leads to

1. Weakness
2. Fatigue
3. Joint pains
4. Abdominal pain
5. Arthralgia
6. Grey/bronze skin pigmentation
7. Diabetes mellitus
8. Erectile dysfunction
9. Signs of chronic liver disease
10. Hepatomegaly
11. Cirrhosis
12. Cardiomyopathy
13. Neurological or psychiatric symptoms

Investigations:

- **↑ LFT**
- **↑serum ferritin**
- **↑ serum iron**
- **Joint x-ray = chondrocalcinosis**

Treatment: Venesection every week to every two weeks

Complications: ↑ risk of hepatocellular carcinoma due to high ferritin
Hemosiderin

HEPATOCELLULAR CARCINOMA

The commonest (90%) liver tumours are secondary (metastatic) tumours eg. from breast cancer, bronchial cancer, GI tract cancers.

Strongly associated with liver cirrhosis
High risk factors

- Family history
- Hepatitis B and C
- Inherited haemochromotosis (high ferritin)
- Primary biliary cirrhosis

Signs and Symptoms

- Fever and malaise
- Anorexia, weight loss
- Right upper quadrant pain
- Hepatomegaly
- Jaundice
- Ascites

Investigations:

- **CT scan is the investigation of choice**
- **AFP (Alpha-fetoprotein) is high**

Treatment:

1. **Surgical resection if there is a single lesion and no liver metastasis**
2. **Liver transplant gives 5 years survival rate**

Prevention: Hepatitis B vaccination

PANCREATIC DISEASES

1. ACUTE PANCREATITIS

Causes can be remembered as GET SMASHED:

1. **G**allstones
2. **E**thanol
3. **T**rauma
4. **S**teroids
5. **M**umps
6. **A**utoimmune
7. **S**corpion sting
8. **H**yperlipidemia/ Haemochromatosis
9. **E**RCP
10. **D**rugs

Signs & Symptoms:

- Severe epigastric pain which radiates to the back relieved by sitting forward
- Often precipitated by recent alcohol abuse
- **Acute:** Acute generalized tenderness with symptoms of peritonitis and paralytic ileum
- **Chronic:** weight loss, steatorrhoea (↑ fat content in the stool) causing pale foul smelling stools, difficult to flush.

GLASGOW CRITERIA FOR PREDICTING SEVERITY OF PANCREATITIS

- **P**aO2 < 8kpa (do ABG)
- **A**ge > 55 years
- **N**eutrophil WCC > 15 x 10*9
- **C**alcium < 2 mmol/L
- **R**enal function Urea > 16 mmol/L
- **E**nzymes LDH >600 IU/L, AST >200 IU/L
- **A**lbumin <32 g/L
- **S**ugar Blood glucose >10 mmol/L

Investigations:

1. **Investigation of choice is serum amylase to confirm diagnosis**
2. **Plasma lipase can also be used and is more sensitive than amylase**
3. **Ultrasound scan to look for gallstones**
4. **CT abdomen is the gold standard if diagnosis is not clear after checking amylase and lipase**

Treatment:

1. **IV fluids and Nasogastric tube if vomiting**
2. **Prophylactic antibiotics**
3. **Gallstone-related pancreatitis needs urgent ERCP and laparoscopic cholecystectomy should be performed within 2 weeks.**

1. **PANCREATITIC CANCER**

It is common in elderly patients and usually causes painless obstructive jaundice.

Risk Factors:

1. Smoking
2. Chronic pancreatitis
3. Diabetes Mellitus
4. Family history of pancreatic cancer

Signs & Symptoms:

- Head of the pancreas tumours usually cause obstructive jaundice whereas tumours of the tail of the pancreas cause obstruction of the inferior vena cava.
- Weight loss
- Anorexia
- Abdominal pain
- Jaundice
- Back pain
- Migrating thrombophlebitis
- Courvoiser's signs – distended non tender palpable gall bladder

Investigations:

1. **Abdominal ultrasound is the initial investigation**
2. **CT scan is the gold standard but is only indicated in patients with highly suspicious signs of pancreatic tumour**
3. **Serum marker: CA 19-9**
4. **ERCP: indicated when other modalities are inconclusive and suspicion of malignancy is still high. May be used to take biopsy and insert a stent at the same time.**
5. **Biopsy: should be done during endoscopic procedure.**

Treatment:

1. **Resection with intent to cure: for patients with localized tumours and fit enough to tolerate major surgery (e.g. whipple's procedure)**
2. **Palliative therapy: if there is metastasis**

DIARRHOEA

This is increased frequency of defaecation and watery or loose stools.

ACUTE DIARRHOEA: <2 WEEKS

Causes:

1. **Gastritis:** fever, vomiting, epigastric pain
2. **Gastroenteritis:** fever, vomiting, diarrhoea
3. **Gastroenterocolitis:** fever, vomiting, bloody and mucoid diarrhoea

CHRONIC DIARRHOEA: >2 WEEKS

Causes:

1. **Colorectal cancer:** altered bowel habits, weight loss, blood in stools, anaemia
2. **Irritable Bowel Syndrome:** chronic diarrhoea alternating with constipation, pain relieved on defecation, no other pathology
3. **Diabetic Autonomic Neuropathy**
4. **Hyperthyroidism**
5. **Addison's Disease**
6. **Carcinoid syndrome**

CAUSES OF BLOODY DIARRHOEA:

1. **E-coli**
2. **Camphylobacter**
3. **Shigella**
4. **Salmonella**
5. **Amoebiosis**
6. **Cronh's**
7. **Colorectal Cancer**
8. **Polyps**
9. **Ischaemic colitis**
10. **Pseudomembranous colitis**

- Travel history present → E-coli, Camphylobacter, Shigella, Salmonella, Amoebiasis
- History of long term antibiotics can cause Pseudomembranous colitis (caused by Clostridium difficile)
- History of Myocardial Infarction/Intermittent claudication → Mesenteric ischaemia (s&s = post prandial pain)
- Tenesmus→ Suggests rectal involvement

Treatment:

- **Oral Rehydration**
- **Loperamide**

CONSTIPATION

Pass stool < 3 times per week

Clinical Features:

- A mass can be felt inside the rectum due to faecal impaction
- Sigmoid colon may be palpable
- Typically in elderly & children
- May present with confusion in elderly
- May also present with overflow diarrhoea
- Usually constipation leads to faecal impaction causing obstruction of the urinary tract leading to UTI which may cause confusion in the elderly

Management:

- **Life style modifications (advise exercise/high fibre diet /high fluid intake)**
- **If the above fails then give medications**

1. **Bulking Agents e.g. Methylcellulose, Ispaghula**
   They ↑ faecal mass, therefore stimulating peristalsis. They must be taken with plenty of fluids & they take days to act.
   Contraindications: Intestinal obstruction
   Colonic atony
   Faecal impaction

1. **Stimulant Laxatives e.g. Bisacodyl, Senna – these are tablets, Glycerol – acts like a stimulant, used as rectal suppository**
   They increase intestinal motility.
   Contraindications: Intestinal obstruction
   Acute colitis (fever + diarrhoea with blood)
   NB. Stimulant laxatives are the first choice when treating constipation caused by opiates

1. **Faecal Softeners e.g. Arachis oil – as an enema, Liquid paraffin (should not be used for prolonged period)**
   Lubricates & softens stools, very useful when managing constipation with anal fissures.

1. **Osmotic Laxatives e.g. Lactulose**
   They retain fluid in the bowel.
   Contraindication: Intestinal obstruction

1. **Enemas: Useful when there is a need of rapid evacuation of stony dull faecal impaction, manual evacuation may be performed**

COLORECTAL CARCINOMA

Risk factors

- Age
- Smoking
- Polyps
- Inflammatory Bowel Disease
- Low fibre diet
- Family History
- Familial Adenomatosis Polyposis
- Diabetes Mellitus Type 2

NB. Most cancer develops from polyps in the colon, which developed at least a decade before the cancer develops

Presentation

1. **RECTAL CANCER**

- Fresh bleeding per rectum
- Sensation of incomplete evacuation
- Tenesmus
- Painful defecation
- Weight loss

1. **COLONIC CANCER**

   - **RIGHT COLON TUMOURS**
     - Usually become fairly large before any obstructive symptoms occur
     - Change in bowel habits it usually a late sign
     - Patients usually present with iron deficiency anaemia
     - Crampy abdominal pain intermittently

   - **TRANSVERSE AND LEFT COLON TUMOURS**
     - Iron deficiency anaemia is uncommon
     - Commonly present with intermittent rectal bleed
     - Crampy abdominal pain intermittently

Investigations:

- Rectal Cancer → sigmoidoscopy
- Sigmoid colon Cancer → sigmoidoscopy
- Colonic Cancer → colonoscopy

N.B: CEA is a colorectal tumour marker

Treatment: Surgery

Resource start date	2013-06-30 06:13
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Resource view

Resource name	Haematology PLAB 1 Notes
Resource description	Haematology
Resource content	

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HAEMATOLOGY LECTURE NOTES 2014

TOPICS:

1. **Anaemia**
2. **Microcytic Anaemia**
3. **Iron Deficiency Anaemia**
4. **Thalassemia**
5. **Normocytic Anaemia**
6. **Congenital Spherocytosis**
7. **Glucose-6-phosphate Dehydrogenase Deficiency**
8. **Sickle Cell Disease**
9. **Autoimmune Haemolytic Anaemia**
10. **Macrocytic Anaemia**
11. **Pernicious Anaemia**
12. **Bleeding Disorders**
13. **Abnormal Coagulation**
14. **Haemophilia A**
15. **Haemophilia B**
16. **Anti-coagulant Therapy**
17. **Bleeding in liver disease**
18. **Massive transfusion/cardiopulmonary bypass**
19. **Abnormal Platelets**
20. **Thrombocytopenia (ITP)**
21. **Vascular Defect**
22. **Malignancies in Haematology**

23. **Pancytopenia**
24. **Leukaemias**
25. **Acute Lymphoid Leukaemia**
26. **Acute Myeloid Leukaemia**
27. **Chronic Myeloid Leukaemia**
28. **Chronic Lymphoid Leukaemia**
29. **Hodgkin's Lymphoma**
30. **Non-Hodgkin's Lymphoma**
31. **Myeloma**
32. **Myeloproliferative Disorders**
33. **Polycythemia Rubra Vera**
34. **Essential Thrombocytopenia**

ANAEMIA

This is low haemoglobin levels.

Parameters for anaemia:

Hb <13.5 g/dl in men
Hb <11.5 g/dl in women

Classification:

1. Microcytic anaemia - MCV <76
2. Normocytic anaemia - MCV 76-96
3. Macrocytic anaemia - MCV >96

Classification depends on the value of **MCV**. Each type has different causes.

Signs & Symptoms:

1. Light headedness
2. Tinnitus (ringing in the ear)
3. Pallor
4. Fatigue
5. Weakness
6. Dyspnoea
7. Palpitation
8. Headache
9. Angina

Complications of anaemia:

1. Tachycardia
2. Murmurs (harsh systolic murmur)
3. Heart failure
4. Hyperdynamic circulation
5. Cardiomegaly

CLASSIFICATIONS OF ANAEMIA AND COMMON CAUSES

1. **LOW MCV <76 (microcytic hypochromic)**

A. Iron deficiency
B. Thalassemia
C. Congenital sideroblastosis

1. **NORMAL MCV 76-96 (normocytic normochromic)**

A. Anaemia of chronic disease (e.g. RA, SLE)
B. Bone marrow failure e.g. aplastic anaemia
C. Renal failure

D. Pregnancy
E. Haemolysis
F. Acute blood loss

1. **HIGH MCV >96 (macrocytic)**

A. B12 & folate deficiency
B. Alcohol
C. Liver disease
D. Hypothyroidism
E. Anti-folate therapy (e.g. Phenytoin or Trimethoprim)
F. Leukaemia

A. MICROCYTIC ANAEMIAS

1. IRON DEFICIENCY ANAEMIA

Causes:

1. Chronic blood loss e.g. in bleeding: GIT bleed from use of aspirin/NSAIDs or peptic ulcer or menorrhagia, GIT malignancy
2. Hookworm infection - caused by Ancylostoma duodenale and Necator americanus. They attach to the walls of the small intestines causing blood loss which is occult and presents as anaemia. Investigations: FBC for eosinophilia and stool analysis for ova. **Treatment: Albendazole**
3. Diet e.g. vegetarian/vegan
4. Malabsorption e.g. Coeliac disease which causes both iron and folate deficiency
5. Inflammatory bowel disease (crohn's disease)

Signs:

1. Koilonychias
2. Atrophic glossitis (sore tongue)

Investigation results:

1. Decreased MCV, hypochromia, anisocytosis, poikilocytosis
2. Decreased serum iron
3. Decreased serum ferritin (transport affected)
4. Decreased TIBC (total iron binding capacity) / (no iron)

Treatment:

- **Oral iron → ferrous sulphate for at least 3 months**
- **Any case with Hb < 8 → blood transfusion**
- **Any case with Ischemic Heart Disease and Hb <5 → blood transfusion**

2. THALASSEMIA

This is common in Asian continent like Sri Lanka and Sub Mediterranean continent like Italy, Malta

There is underproduction or no production of Hb β peptide chain

β Thalassemia major = Hb is usually <9:

There is mutation in β - globin genes → decreased or absent β chain product

Signs & Symptoms:

1. Severe anaemia
2. Failure to thrive
3. Usually splenomegaly (because haemolysis usually occurs in the spleen)

Investigations:

- Hypochromic, microcytic anaemia (MCV <76)
- Target cells
- HbF raised
- HbA2 variable
- HbA absent

Treatment:

- **Transfusion depending on haemoglobin levels (if Hb <8)**
- **Desferrioxime to prevent iron overload**
- **Splenectomy**
- **Folate supplements**
- **Bone marrow transplants**

- If no transfusion → death may occur.
- With transfusion → normal development but increased risk of iron overload.
- If blood transfusion is not adequate in children → anaemia→ decreased growth → skeletal deformity (bossing of the skull)
- After 10 years of repeated transfusions it can lead to endocrine failure, liver failure, heart toxicity (siderosis) due to accumulation of iron.

β-Thalassemia minor: Hb usually >9

- MCV< 75 microcytosis
- Hb> 9 mild – moderate anaemia
- Hb A2 > 3.5 %
- Hb F slightly increased

Treatment: No intervention just observe if asymptomatic

B. NORMOCYTIC ANAEMIA

This is anaemia with normal MCV 76-96

CAUSES:

1. **Anaemia of chronic disease** e.g. polymyalgia rheumatica, rheumatoid arthritis, SLE etc.
2. **Bone marrow failure** e.g. usually autoimmune (aplastic anaemia)
3. **Renal failure** is due to reduced production of erythropoietin. In this case you treat anaemia with erythropoietin IM.
4. **Pregnancy** due to increased demand of iron and folate. Anaemia in pregnancy treat with iron sulphate.
5. **Haemolysis** usually causes normocytic anaemia but chronic repeated haemolysis can cause microcytic anaemia.

HAEMOLYTIC ANAEMIA

CAUSES:

1. Genetic/Congenital causes (usually young patients or a child)
    a. RBC Membranopathies e.g. Spherocytosis, elliptocytosis
    b. Haemoglobinopathies e.g. Sickle cell anaemia, thalassemia
    c. Enzyme defects eg. G6PD Deficiency

1. Acquired Causes

a. Immune e.g. Haemolytic disease of the newborn, blood transfusion reactions, autoimmune haemolytics anaemia, drug induced (penicillin, L-dopa)

b. Non-immune eg. Trauma (microangiopathic haemolytic anaemia), infection (septicaemia, malaria), paroxysmal nocturnal haemoglobinuria

CLASSIFICATION

1. Intravascular haemolysis (takes place in the vessels): free plasma haemoglobin, haemoglobinuria, ↓ haptoglobins, haemosiderinuria.

2. Extravascular haemolysis (takes place in the spleen): There is splenomegaly because the red blood cells are destroyed in the spleen.

Signs & Symptoms:

1. Jaundice

2. Haematuria
3. History of drug intake
4. Previous anaemia
5. Family history
6. Hepatosplenomagaly
7. Leg ulcers (sickle cell anaemia)

Investigations:

- Increased bilirubin (unconjugated)
- Reticulocytosis (<1% is normal) N.B reticulocytes are young RBC.
- Increased urinary urobilinogen → polychromasia
- Increased haptoglobins (binds free Hb)
- **Direct Coomb's test** → positive in immune type haemolysis

1. CONGENITAL SPHEROCYTOSIS: This is an autosomal dominant condition.

   Strong family history, inheritance pattern is 1:2.

There is splenomegaly due to extravascular haemolysis. In this condition the red blood cells fail to change their shape as they pass through the spleen. As a result they get haemolysed by the spleen. There is ↑ risk of gallstones.

Investigations:

1. Osmotic fragility test
2. Spherocytes in blood film

Treatment: folate, splenectomy

NB: Spherocytosis can either be congenital or autoimmune. To differentiate you need to do direct coombs test. If it is positive then it's an autoimmune.

2. G6PD: X- linked Seen commonly in males

Precipitated by: Primaquine, sulfonamides, ciprofloxacin, **broad beans**

Signs & Symptoms:

1. Rapid anaemia
2. Jaundice
3. Heinz bodies (characteristic in microscopy, common in men)
4. Usually episodic depending on the precipitating factor.

Investigation: Enzyme assay after several weeks after a crisis

Treatment: avoidance of precipitating factors, transfusion if severe anemia

3. SICKLE CELL DISEASE

This is a congenital haemoglobinopathy which is common in Afro-Caribbeans.

Haemoglobin S when deoxygenated it causes sickling so the red blood cells are more fragile which leads to haemolysis in the spleen.

Signs & Symptoms:

1. Painful crisis e.g. chest pain, bone pain (bone infarction), etc.
2. Infection e.g. chest infection, urinary tract infection
3. Anaemia which may require recurrent blood transfusion
4. Jaundice which indicates haemolysis
5. Crisis can be precipitated by menstrual period

SICKLE CELL CRISIS:

1. **Thrombotic crisis:** precipitated by cold, infection, ischemia, severe pain.
2. **Aplastic crisis:** due to parvovirus
3. **Sequestration crisis:** hepatosplenomegaly → RUQ pain, increased LFT , decreased Hb

Management of crisis:

1. **Prompt analgesia (IV opiates) e.g. morphine**
2. **Give oxygen**
3. **Cross match blood, Full Blood Count, reticulocytes**
4. **Check for signs of infection: Blood culture, Chest X-Ray , Mid-Stream Urine**
5. **Rehydrate by giving normal saline and keep warm**
6. **Antibiotics if febrile**
7. **Blood transfusion as required**

Investigations:

- Hb 6-8g/dl
- Reticulocytes 10 – 20 % (normal reticulocytes is <1%, high reticulocyte count generally means haemolysis of any type)
- Increased Bilirubin
- Electrophoresis to check the sickle cells

Management of chronic disease:

- **Chemotherapy (hydroxyurea) if frequent crisis (increase the level of fetal haemoglobin)**
- **Chronic blood transfusion keep HbS < 30 %**
- **Bone marrow transplant**
- **Splenic infarct → hyposplenism → patient will need antibiotics + immunization for streptococcus and pneumococcal pneumonia, N. meningitides, H. Influenzae**

4. AUTOIMMUNE HAEMOLYTIC ANAEMIA (AHA)

1. Primary (idiopathic meaning cause not known)
2. Secondary (secondary to lymphoma or SLE or CLL)

Warm AHA: presents as acute or chronic anaemia. Haemolysis occurs at 37 degrees and above.

Treatment:

1. **Steroids**
2. **Splenectomy**

Cold AHA: chronic anaemia gets worse with cold often associated with Raynauds phenomenon

Treatment: keep warm, blood transfusion, chemotherapy, chlorambucil

C. MACROCYTIC ANAEMIA

This is anaemia with high MCV > 96 fl

Causes:

1. Decreased B12
2. Decreased Folate
3. Alcohol
4. Liver disease
5. Pregnancy
6. Haemolysis
7. Hypothyroidism
8. Anti-folate drugs (e.g. phenytoin, methotrexate)

Investigations:

- Hypersegmented polymorphs/neutrophils usually found in B12 deficiency.
- Target cells (liver disease)
- LFT (increased GGT in alcoholism)
- Serum B12
- Serum folate
- Bone marrow biopsy → if the cause can't be found by any of the above tests
- If the B12 is↓ then schilling test → malabsorption from terminal ileum
  → intrinsic factor deficiency (pernicious anaemia)
- Oral radioactive B12 → check for the amount present in urine

CAUSES OF LOW B12:

1. Pernicious anaemia (an autoimmune disease)
2. Post-gastrectomy: deficiency in parietal cells lead to deficiency in intrinsic factor which means that B12 will not be absorbed in the GI tract.
3. Diet: Common in Vegans and Vegetarians

CAUSES OF LOW FOLATE:

1. Poor diet common in alcoholism
2. Increased need pregnancy
3. Haemolysis
4. Malignancies
5. Long term haemodialysis
6. Malabsorption like in celiac disease
7. Anti-folate medication like phenytoin, trimethoprim

NB: alcohol causes high MCV but there is usually no anaemia. Then you can check GGT. It is commonly raised in alcoholism.

1. **PERNICIOUS ANAEMIA:** This is an autoimmune disease with antibodies to parietal cells and to intrinsic factor. Parietal cells are in the stomach and they produce intrinsic factor.

Common features:

1. Tiredness
2. Weakness
3. Shortness of breath
4. Paraesthesia/weakness in the limbs due to spinal cord degeneration
5. Atrophic glossitis (sore red tongue)
6. Diarrhoea

Associations:

1. Thyroid disorders
2. Vitiligo
3. Addison 's disease
4. Carcinoma stomach
5. Associated with atrophic gastritis which is usually shown by absence of mucosal folding and this usually leads to stomach cancer

Investigation results:

1. Decreased Hb, MCV > 110, decreased B12, decreased WCC, decreased platelets
2. Hypersegmented polymorphs/ neutrophils
3. Megaloblasts in bone marrow
4. Antibody to parietal cells in 90 % (positive results are **not diagnostic**)
5. Antibody to intrinsic factor in 60% (are **diagnostic** if present)

Treatment:

- **Hydroxycobalamine (B12) IM for 2 weeks every other day**
- **Maintenance → IM every 2 months for life**

Intrinsic factor binds to B12 in the stomach which protects B12 from being ingested by the hookworms in the intestine.

COMPLICATIONS:

Sub-acute combined degeneration of spinal cord:

Posterior lateral columns are affected

Triad: extensor plantars + brisk knee jerk + hyper-reflex at ankle

Also there is parasthesia and weakness or ataxia of the limbs (lower limbs)

1. BLEEDING DISORDERS

General approach

After injuries these occur in order to stop bleeding:

1. Vasoconstriction
2. Platelet aggregation
3. Coagulation cascade (fibrin)

Vascular & Platelet disorders ⇒ prolonged bleeding + purpura + bleeding from mucous membrane

Coagulation disorder ⇒ Usually there is bleeding into joints, muscles GI & GU

Therefore normal haemostasis requires the interaction of

a. **Platelets**
b. **Fibrin from clotting cascade**
c. **Normal microvasculature**

Bleeding could be due to:

a. **Platelets = too few or dysfunction**
b. **Coagulation abnormality**
c. **Microvasculature abnormalities**

A. ABNORMAL COAGULATION

COAGULATION TESTS

1. PT → test for extrinsic system 10, 7, 2, 1 (10 – 14 sec)
2. INR → 0.9 – 1.2 (PT control), increased INR in warfarin, vitamin K deficiency & liver disease
3. APTT → intrinsic system 12, 11, 9, 8 (35 – 45 sec), increased PTT (heparin, haemophilia (factor 8 affected)
4. Thrombin time → 10 – 15 sec, increased in heparin, increased in DIC
5. Bleeding time (normal 7 min) → commonest cause is Von Willebrand's disease.

NORMAL VALUES

Prothrombin Time (PT): 10-14 seconds
APTT: 35-45 seconds
Bleeding Time (BT): < 7 min
Thrombin Time (TT): 10-15 seconds

In the GMC exam make sure you use their values as standard normal as sometimes there are variations e.g. an APTT of 42 may be regarded as high and if you do not use their values you may get a question wrong.

Abnormality	Type of defect	Causes
High PT	Extrinsic pathway defect	warfarin, liver disease, vitamin K deficiency
High APTT	Intrinsic pathway defect	Heparin, haemophilia, Von Willenbrand's disease, lupus anti-coagulant (anti-phospholipid syndrome
High PT & APTT	Multiple defect	Liver disease, DIC, warfarin
High TT	Abnormal fibrinogen production	Fibrinogen defect, excess fibrinogen degradation products
Low fibrinogen	Excess consumption of clotting factors and fibrinogen	Consumption coagulopathy e.g DIC or Liver disease
High Bleeding time	Abnormal platelet function	Von Willenbrand disease (Also causes high APTT), or acquired platelets dysfunction, Perform platelets studies if you suspect this.
High TT, APTT & PT	Multiple (aquired) defects	Deficiency or abnormal fibrinogen or heparin

Common causes of abnormal coagulation

1. Haemophilia A & B
2. Anti-coagulants e.g. warfarin
3. Liver (usually oesophageal varices)
4. Massive blood transfusion (due to dilution thrombocytopenia)
5. DIC (precipitated by sepsis, or severe bleeding e.g. placental abruption)
6. Von Willebrand's disease
7. Vitamin K deficiency (Obstructive jaundice, small bowel disease due to malabsorption)

General Management:

1. **Fresh Frozen Plasma - indicated for treatment of**
   1) Acute DIC with bleeding
   2) To improve haemostasis in decompensated liver disease
   3) Emergency reversal of warfarin therapy if no prothrombin complex concentrate (PCC) available
1. **Vitamin K: phytomenadione used for reversal warfarin overdose**
2. **Protamine sulphate: used to reverse heparin**
3. **Cryoprecipitate /fibrinogen concentrate used if fibrinogen is less than500g/L**
4. **Anti-fibrinolytic (e.g. tranexamic acid) is used if there used sometimes for treatment of life threatening bleeds following thrombolytic therapy.**

1. HAEMOPHILIA A: Deficiency of Factor VIII, X- linked recessive (only males are affected)

Signs & Symptoms:

- Depends on severity
- After trauma ⇒ bleeding into joints (haemarthrosis) & muscles (haematoma)
- Haematoma usually in buttocks

Investigation: Increased APTT, decreased factor VIII assay

Management:

- Avoid I.M injection
- In major bleeding → give factor VIII

1. **HAEMOPHILIA B:** This is deficiency of Factor IX. It is also called Christmas disease.

Treatment: Give factor IX

1. **ANTI-COAGULANT THERAPY:**

Indications:

- Deep vein thrombosis
- Pulmonary embolism

- Atrial fibrillation
- Stroke prevention
- Prosthetic heart valve

WARFARIN:

- Oral
- Narrow therapeutic range
- Inhibits reductase which is responsible for generating active vitamin K

INR targets: (NORMAL INR: 0.9-1.2)

1. Prosthetic heart valves → 3 – 4.9
2. Atrial fibrillation → 2- 3
3. Pulmonary embolism → 2- 3 (3.5 if recurrent)
4. Deep vein thrombosis → 2-3

Warfarin overdose causes increased INR and therefore increased PT

Management:

- **INR 5-8 and asymptomatic: does not usually require specific treatment. Only withhold warfarin until INR less than 5.**
- **INR >8 and asymptomatic: give vitamin K**
- **If there is minor bleeding: give vitamin K**
- **If there is severe bleeding: Requires urgent correction. Prothrombin complex concentrate (II, VII, IX, and X) is the preferred treatment for life threatening bleeding.**

NB. Fresh frozen plasma should only be used if prothrombin complex concentrate not available.

Heparin: Can be standard heparin or low molecular weight heparin

Treatment for Heparin toxicity → stop heparin, give protamine sulphate

4. BLEEDING IN LIVER DISEASE

The liver is involved in synthesis of clotting factors II, VII, IX, X which are Vitamin K dependent factors and also vitamin K

Obstructive jaundice: prolonged PT due to vitamin K deficiency

Liver cirrhosis: Increased PT, APTT, and TT; low fibrinogen

Management: Give vitamin K (Fresh Frozen Plasma is more effective as it contains the clotting factors)

5. MASSIVE TRANSFUSION/cardiopulmonary bypass

This causes **dilutional** thrombocytopenia. Therefore if a patient starts bleeding after massive blood transfusion, for example 10 units of blood, think of dilutional thrombocytopenia.

Treatment: Transfuse platelets.

ABNORMAL PLATELETS

1. **THROMBOCYTOPENIA - This is low platelets**

Normal Platelet count is 150-400 x 10/9 /L

Causes

1. **Increased platelet consumption**

Immune

- Idiopathic (ITP = Idiopathic Thrombocytopenic Purpura)
- Drug induced
- SLE

Non –Immune

- Massive blood transfusion leads to dilutional thrombocytopenia
- Hypersplenism (platelets are destroyed in the spleen)
- DIC due to increased consumption of platelets
- TTP

2. Reduced platelet production

- Myelosuppressant (drugs, alcohol, viral illness) e.g. aplastic anaemia
- Bone marrow infiltration/failure
- B12 or folate deficiency (they take part in the production of platelets)

3. Abnormal platelet function

- The common disease of this type of **Von Willebrand disease**.
- In this case **bleeding time is increased**.
- In the exam if you see bleeding time increased think of Von Willebrand first.
- The next investigation would be to do platelet function studies

GENERAL MANAGEMENT

1. **For immune mediated thrombocytopenia use steroid e.g. prednisolone with or without immunoglobulin**
2. **In DIC/massive transfusion = transfuse platelets to keep platelets above 75**

Idiopathic Thrombocytopenic Purpura ⇒ marrow failure, virus, DIC, lymphoma, hypersplenism

Signs & Symptoms:

- ITP usually follows an upper respiratory tract infection, common in children, patient present with bleeding from nose but patient is generally well.
- Bleeding
- Purpura (dependent pressure areas)
- Epistaxis
- No splenomegaly
- Everything is normal (Patient is clinically well)

Investigations:

- Anti – platelet Ig G +VE

Treatment: Steroids e.g. Prednisolone.

VASCULAR DEFECTS

Usually this is vasculitis

Congenital:

- Osler – Weber – Rendeu syndrome which is also known as Hereditary haemorrhagic telangectasia (nose bleeds and mucosal bleeds in GMC scenario)

Acquired:

1. Trauma
2. Vasculitis e.g. in Henoch – Schonlein Purpura
3. Scurvy (vitamin C deficiency causing bleeding from gums and usually peri-follicular bleeding)

1. MALIGNANCIES IN HAEMATOLOGY

a. **Leukaemia**
   i. **Acute/Chronic**
   ii. **Lymphoid/Myeloid**
b. **Lymphoma**
   i. **Hodgkin**
   ii. **Non-Hodgkin**

c. **Myeloma**
d. **Myeloproliferative disease**

A. LEUKAEMIAS

CLASSIFICATION

Acute or Chronic: LOOK AT THE AGE

If less than 40 years old it is likely to be acute leukaemia

If more than 40 years old it is likely to be chronic leukaemia

Lymphoid or Myeloid: LOOK FOR LYMPHADENOPATHY

If there is lymphadenopathy it is likely to be lymphoid leukaemia otherwise it is myeloid leukaemia.

N.B. splenomegaly alone means myeloid

LOOK FOR BLOOD FINDINGS: If neutrophils are in the blood it's myeloid, if lymphocytes then it's lymphoid leukaemia.

1. ACUTE LYMPHOID LEUKAEMIA:

- Patient is less than 40 years
- Anaemia
- Infection → zoster, CMV, measles, candidiasis, pneumocytosis
- Bleeding
- Splenomegaly (hepatosplenomegaly)
- Lymphadenopathy
- Orchidomegaly
- CNS involvement eg. meningitis

Investigations:

1. Blood film shows lymphocytosis
2. Bone marrow biopsy

Complication: Commonly lymphadenopathy

Treatment:

1) Supportive care → blood & platelet transfusion, I.V antibiotics

2) Chemotherapy (main treatment)

Poor prognosis if adult, male, presence of Philadelphia chromosome 9:22, CNS signs, WCC > 100*10*9

2. ACUTE MYELOID LEUKAEMIA (AML):

- Anaemia
- Infection
- Bleeding
- Bone pain
- CNS sign (cord compression)
- Hepatomegaly
- Splenomegaly (very common)
- Lymphadenopathy
- Weakness and malaise
- Fever
- Gum hypertrophy

Investigations:

- WCC variable/high
- Blast cell in blood
- Bone marrow biopsy

Complications:

- Infection
- **TUMOUR LYSIS SYNDROME** due to chemotherapy
- Rapid cell death on starting chemotherapy, which causes a rise in serum urate, potassium and phosphate.
- **Treatment: Initial step is IV fluids, then allopurinol and high fluid intake**
- Big nodes may cause mass effect: Shortness of breath by compression of trachea
- Splenomegaly

Treatment: same as for ALL (chemotherapy)

3. CHRONIC MYELOID LEUKAEMIA (CML):

- Middle aged people, associated with Philadelphia chromosome 9:22

Signs & Symptoms:

- Chronic
- Weight loss
- Tiredness
- Gout
- Fever
- Bleeding
- Abdominal pain
- Massive splenomegaly
- Hepatomegaly
- Anaemia
- Bruising

Investigations:

- Raised WBC >100 x 10^9
- Haemoglobin levels are low or normal
- Platelets variable
- Increased urate (causing renal stones or gout) & alkaline phosphatase

Natural history:

- Chronic
- Accelerated phase → increased symptoms, spleen size
- Blast transformation with features of leukaemia

Treatment: Chemotherapy (Imatinib mesylate, Hydroxyurea)

4. CHRONIC LYMPHOID LEUKAEMIA:

- > 40 years
- More common in men
- Bleeding + low weight + infection + anorexia
- Enlarged rubbery lymph nodes
- Late stage hepatosplenomegaly

Investigations:

- Lymphocytosis
- Normocytic normochromic anaemia
- Low or normal platelets

Complications:

- Auto immune haemolytic anaemia
- Infection
- Bone marrow failure
- Lymphadenopathy

Treatment:

- **Chemotherapy**
- **Radiotherapy**
- **Supportive Care e.g. transfusion or antibiotics**

HODGKIN LYMPHOMA:

Associated with Ebstein Barr Virus

Common in young black patients

Malignant proliferation of lymphocytes (Reed Sternberg cells)

Signs & Symptoms:

- **Enlarged painless rubbery lymph nodes, especially cervical**
- Fever, weight loss, lethargy, night sweats
- Hepatosplenomegaly
- Anaemia

Investigations: Lymph node biopsy

Staging:

- I→ single lymph node
- II→ 2 or more regions on same side of the diaphragm
- III→ both sides of diaphragm
- IV —> beyond lymph node metastasis

Needs to be differentiated from TB which has tender matted cervical lymphadenopathy and night sweats.

Treatment:

- **Radiotherapy for I A & II A**
- **Chemotherapy for IIA – IV B**

NON HODGKIN LYMPHOMA:

- Weight loss, fever, anorexia, night sweats
- **No Sternberg cells**
- Rapid extranodal spread (skin nodes)
- **SKIN NODULES**

MYELOMA:

Malignant proliferation of plasma B-cell

Elderly >70 years of age (typical symptoms include chronic back pain, hypercalcemia, anaemia, proteinuria and raised ESR)

Signs & Symptoms:

- Bone pain in back, ribs, long bones
- Pathological fracture e.g. vertebral collapse
- Fatigue (from anaemia), bleeding
- Pyogenic infection
- Hyperviscosity (visual disturbance, headache)
- Proteinuria

Investigations:

- Serum electrophoresis
- Urine (Bence Jones protein with paraproteinuria)
- Bone marrow (plasma B cell)
- X-ray → punched out lesion (pepper pot skull)
- High Urea and hypercalcemia

Treatment:

- **Chemotherapy is the main treatment**

- **Analgesia**
- **Transfusion**
- **High fluid intake**
- **Bisphosphonates (control the increase in calcium, & bone pain)**
- **Radiotherapy locally, it decreases the pain if the pain is secondary to bone metastasis.**

1. PANCYTOPENIA

This is when all 3 types of cells are reduced, i.e. low haemoglobin,white cell count and platelets

CAUSES

1. Bone marrow failure e.g. aplastic anaemia
2. Hypersplenism
3. SLE
4. Megaloblastic anaemia

CAUSES OF BONE MARROW FAILURE

1. **Aplastic anaemia:** In this one all the 3 cell types are reduced i.e. low red blood cell (low Hb), low white cell count and low platelets. This is what differentiates aplastic anaemia from leukaemia in that WCC is increased in leukaemia.

   Aplastic anaemia can be caused by drugs like cytotoxic cyclophosphamide, radiation, autoimmune, drugs (gold), viral hepatitis etc.

1. **Infiltration** (malignancy, Tuberculosis)
2. **Myelofibrosis**

Signs & Symptoms:

- Anaemia (from low haemoglobin)
- Infection (from low white cell count)
- Bleeding (from low platelets)

Treatment:

Supportive to increase blood count while undertaking definitive which is bone marrow transplant.

5. MYELOPROLIFERATIVE DISORDERS

Classification:

1. Increased RBC → **Polycythaemia Rubra Vera**
2. Increased WBC → **Chronic Myeloid Leukaemia**
3. Increased Platelets → **Essential thrombocythaemia**
4. Increased fibroblasts → **Primary myelofibrosis**

A. POLYCYTHERMIA RUBRA VERA

Signs & symptoms:

- Itch after hot bath + plethoric face (red appearance of face)
- Angina - may cause electric shock like pain
- CNS signs
- Raynauds
- Gout (Increased turnover or cells = ↑uric acid)
- Enlarged spleen (60%)

Investigations:

- Increased PCV (packed cell volume)
- Increased WBC Blood film
- Increased RBC
- Increased Platelets

- Bone marrow biopsy

Treatment: Venesection, chemotherapy e.g. hydroxyuria

B. ESSENTIAL THROMBOCYTHAEMIA:

- Raised platelets
- Abnormal function & shape
- Bleeding / thrombosis

Treatment: Chemotherapy

***MYELOFIBROSIS**

- De-arranged haematopoesis in spleen
- Systemic upset
- Bone marrow failure

Investigation: Myeloblasts and teardrop RBCs in blood film

Resource start date	2013-06-26 08:40
Resource end date	2023-06-27 08:40

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SAMSONPLAB ACADEMY
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Resource view

Resource name	Infectious Diseases PLAB 1 Notes
Resource description	Infectious Diseases
Resource content	

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INFECTIOUS DISEASES LECTURE NOTE 2014

TOPICS:

1. Causative Agents
2. Hepatitis
3. HIV
4. AIDS
5. Epstein Barr Virus
6. Herpes Virus
7. Varicella Zoster
8. Cytomegalovirus
9. Brucellosis
10. Listeria Monocytogenes
11. Pneumonia
12. Tuberculosis
13. Lyme Disease
14. Typhoid Fever
15. Streptococcal infections
16. Staphylococcal infections
17. Scabies
18. Leishmaniasis
19. Schistosomiasis
20. Malaria
21. Toxoplasmosis

CAUSATIVE AGENTS

1. **Viruses**
2. **DNA viruses** – papova/ adeno/ human herpes/ pox/ hepatitis B
3. **RNA viruses** – reo / picorna / toga / rhabdo / other hepatitis viruses

1. **Bacteria**

1. **Cocci:**

   i. ***gram +ve***: staphylococcus / streptococcus / enterococccus
   ii. ***gram –ve:*** neisseria meningitis / moraxella catarrhalis

1. **Rods:**

a. ***gram +ve anaerobes***: clostridium / actinomyces

b. ***gram +ve aerobes***: bacillus anthracis / cornebacterium diptheria / listeria monocytoges / nocardia
c. ***gram –ve***: enterobacterias (e.coli , shigella , salmonella , klebsiella) / pseudomonas aeruginosa / haemophillus influenzae / brucella/vibrio cholerae / campylobacterium jejuni / bordetella pertusis

VIRAL INFECTIONS

1. HEPATITIS (A, B, C, D, E)
All are RNA viruses except B which is a DNA virus

Used for follow up:
PCR: quantitative & qualitative
Viral load, viral titre, viral count

Hepatitis A:

- Faeco– oral route
- Self-limiting illness, one of the causes of traveller's diarrhoea
- Incubation period = 2-6 wks

Investigations:

- IgM antibody to Hepatitis A suggests acute infection
- IgG antibody to Hepatitis A persists for many years

- **Prevention:** active immunisation – vaccination for travellers, give three months protection passive immunization IgG. (Exposed to infection)

- **Treatment: supportive / avoid alcohol or any hepatotoxic drug / interferon ∝ for fulminant hepatitis (peglated ∝ 2a)**

Hepatitis B:

- Spreads through blood products, IV drug abusers, and homosexuals

Serological Markers:

Antigens:

- **The presence of HBeAg implies high infectivity.**
- **Persistence of HBsAg for >6 months defines carrier status**

Antibodies
- **Anti-HBc IgM indicates acute infection**
- **Antibodies to hepatitis B core antigen (HBcAg) - ie anti-HBc - imply past infection**
- **Antibodies to HBsAg - ie anti-HBs - alone imply vaccination.**

- **Complications:**

- Fulminant hepatic failure (rare)
- Chronic hepatitis
- Cirrhosis (5-10 yrs)
- Hepato -cellular carcinoma (HCC) → 10 fold ↑ if HBs Ag is +ve;
  60 fold ↑ if HBeAg & sAg is +ve

- **Vaccination:**
  Active: Live attenuated virus
  Passive: Anti-HBV Ig to non-immune contacts after high-risk exposure (scratch, bite, needle injury, baby of HBV +ve mother)

- **Treatment:**
  Supportive - avoid alcohol
  In chronic HBV – Antivirals (Lamivudine / ribavirin / adefovir), interferon's

Hepatitis C:

- Incubation period – variable
- Spread: IV drug abuse, sex, blood transfusions
- Early infection: mild or asymptomatic
- 35%: chronic infection
- 20 %- 30 %: cirrhosis in 20 years time
- Few – HCC
-

Investigation:

- HCV RNA to check for infectivity
- Anti–HCV only positive after 3 months

Hepatitis D:

- Only coinfects with Hepatitis B infection
- **Investigation:** Anti-HDV antibody

Hepatitis E:

- Faeco- oral

- Causes acute hepatitis similar to Hepatitis A
- Increased mortality in pregnancy

HUMAN IMMUNODEFICIENCY VIRUS (HIV)

- HIV type 1 is responsible for most cases
- HIV type 2 seems to have longer latent period
- In the UK, more commonly heterosexually acquired than homosexually

- **Transmission:** Sexual 75 %, infected blood, IV drug abuser , perinatal (vertical) route

- **Immunology:**
  HIV binds to CD4 receptors on:

- T- lymphocytes (via gp 120 enveloped glycoprotein)
- Monocytes
- Macrophages
- Neural cells

CD4 +ve cells migrate to the lymphoid tissues where the virus replicates → producing new virions → infect new CD4+cell→ impaired function CD4+ve cell→ immune dysfunction
The number of circulating viruses are referred to as viral load → predicts onset of AIDS and also used for monitoring treatment

Stages of HIV:

1. **Acute Infection:** often this stage shows no symptoms
2. **Sero–conversion:** transient illness (2- 6 weeks after exposure)
   Fever, malaise, rash, pharyngitis, myalgia, meningo – encephalitis (rare)
3. **Stage of Lympadenopathy:** Lymphodenopathy >1cm in diameter, >/- 2cm in extra inguinal region
4. **Persistent generalised lymphadenopathy**
5. **AIDS**

ACQUIRED IMMUNODEFICIENCY SYNDROME

CD4 count usually <350*10/6 per L, presence of indicator disease
(Normal CD4 count = 600 – 1200)

Prodrome of AIDS:

Related complex – constitutional symptoms

1. Pyrexia
2. Night sweats
3. Weight loss
4. Diarrhoea

+/- Minor opportunistic infection, oral candida, oral hairy leukoplakia, herpes zoster, recurrent HSV, seborrhoeic dermatitis, tinea infections.

Diagnosis:
HIV- antibodies by ELISA, usually confirmed by western blot
(Window period 1-3 weeks after exposure)
P24 Ag (earliest but not routine nearly 2 weeks)
HIV RNA (viral load) – by PCR done to monitor treatment

Prognosis - about 2 years if untreated

All patients who have newly diagnosed with HIV should have:

1. Tuberculin test
2. Toxoplasmosis test
3. CMV test
4. Hepatitis B and C
5. Syphilis serology

Tests 1,2&3 are used to identify past or current infection that may develop as immunosuppression progresses. Tests 4&5 are to test for co-infections of HIV (because of same mode of transmission).

***COMPLICATIONS OF AIDS:**

1. **Cryptosporidium**
   Signs & Symptoms: profuse cholera-like diarrhoea, RUQ pain, vomiting
   Investigation: Stool analysis
   Treatment: No specific therapy, usually responds spontaneously.
   Treat the underlying immunosupression with highly active antiretroviral therapy (HAART)

1. **Cryptococcus Meningitis**
   Signs & Symptoms: Headache +/- meningism signs, seizures, confusion
   Investigation: India ink stain of CSF, serum antigen.
   Treatment: Amphotericin IV + Flucytosine

1. **Candida**

Oral candidiasis
Signs & Symptoms: Curd-like white patches in the mouth. May also have marked erythema and soreness.
Treatment: Nystatin suspension. Second choice is oral fluconazole.

Oesophageal candadiasis
Signs & Symptoms: Dysphagia +/- retrosternal discomfort
Treatment: Oral fluconazole is first line. Second choice is IV fluconazole.

1. **Pneumocystis Jerovocii /Carinii**
   HIV patient with dry cough is always Pneumocystis Jerovocii until proven otherwise.

   Signs & Symptoms: Dry cough, exertional dyspnea, tachypnea,fever, chest pain
   Investigation: PCR, chest x-ray (perihilar fluffy shadows)
   Treatment: High dose co-trimoxazole (trimethoprim/sulfamethoxazole)
   If CD 4 count <200, start prophylactic co-trimoxazole

1. **Toxoplasmosis**
   Signs & Symptoms: Presents like a space occupying lesion, progressive headache, seizures, confusion
   Investigation: CT (ring enhancing lesion)
   Treatment: Sulfadiazine + pyrimethamine

1. **Human herpes virus 8** → Kaposi sarcoma
   Skin lesions may be nodular, papular or blotchy; they may be red, purple, brown or black.

1. **CMV**
   Causes Encephalitis and retinitis
   Investigation: PCR
   Treatment: Ganciclovir

1. **Tuberculosis (TB)**
   HIV patient with productive cough is always TB until proven otherwise

EPSTEIN-BARR VIRUS:

Can cause:

- Glandular Fever
- Lymphoma
- Nasopharyngeal Carcinoma
- Oral hairy leukoplakia
- Leiomyosarcoma

***Infectious Mononucleosis:** **(Glandular Fever)**

- Common in young adults
- Also known as **Kissing disease**
- Incubation period 4-5 weeks
- Self limiting infection

Symptoms:

- Sore throat
- Fever
- Anorexia
- Lymphadenopathy (especially cervical)
- Palatal petechiae
- Splenomegaly
- Hepatomegaly
- Haemolysis

Investigations:

- Lymphocytosis - nearly 20 % of White Cell Count (WCC)
- Heterophills antibody tests (Monospot: Paul – Bunnel)
- Serology

Treatment: Supportive
Analgesia (NSAIDS), steroids if tonsillitis is severe and causing breathing difficulties.
NB. If you give amoxicillin in a patient with infectious mononucleosis, patients will develop generalised rash.

Complications:

- Meningitis
- Encephalitis
- Ataxia
- Cranial Nerve Lesion (VII)
- Guillian-Barre Syndrome
- Rupture spleen
- Erythema multiforme

NB. URTI with cervical lymphadenopathy is infectious mononucleosis until proven otherwise.

HERPES VIRUS INFECTIONS

Herpes Simplex Virus Type 2 – causes multiple painful genital ulcers (usually HSV 2)

Herpes Simplex Type 1:

- **Gingivostomatitis :** mouth ulcer
- **Herpes Labialis:** cold sores. **Treatment: Topical aciclovir**

- **Herpetic Whitlow:** vesicle formation on the fingers

- **Herpes Simplex Virus Keratitis:** corneal dendritic ulcer (do not give steroid drops because it causes blindness.

Treatment: topical aciclovir

- **Herpes Simplex Virus Encephalitis:** fever, fits, headaches, odd behaviour, dysphasia, hemiparesis & spread centripetally)
  Investigation: PCR on CSF (urgent)

Investigation: Swab the lesions

VARICELLA ZOSTER (CHICKEN POX)

Incubation period: 11-21 days

Symptoms:

- Fever
- Crops of blisters of different ages starting on the back then to chest

Infectivity: 4days before the rash until all lesions are scabbed (crust)
Dormant: Remains dormant in the dorsal root ganglia → reactivation due to illness or immunosuppresion → **Shingles** (pain in dermatomal distribution which is unilateral and painful)
NB. Shingles cannot be transmitted unless the person who has been exposed has never had chicken pox before.

TREATMENT FOR VARICELLA ZOSTER (CHICKENPOX):

- **Refer to specialist if it affects special system (ex. eye) or if pregnant (Aciclovir should be prescribed by a specialist)**

- **No treatment if >28 days, <12 years and healthy**
- **Oral acyclovir 800mg 5 times per day within 14-24 hours if >12 years, immunocompetent to reduce duration and severity of symptoms**

- **IV acyclovir if immunocompromised, <28 days, or >80 years**

PROPHYLAXIS FOR VARICELLA ZOSTER (CHICKENPOX)

- **Give varicella IgG if**
  - **Neonates whose mothers develop chicken pox in the last seven days or soon after delivery**
  - **Women exposed to chickenpox during pregnancy especially in the first 20 weeks and those near term**
  - **Immunocompromised individual**

TREATMENT FOR HERPES ZOSTER (SHINGLES):

- **Acyclovir oral within 3 days of onset of symptoms can reduce severity and duration of pain**
- **Pain – Paracetemol**
- **Post Herpetic Neuralgia – Antidepressants (amitryptylline) or antiepileptics (gabapentin)**
- **Conjunctivitis – beware iritis – regular acuity check, acyclovir 3% ointment. Refer to ophthalmologist.**

CYTOMEGALOVIRUS

Spread through direct contact, blood transfusion, organ transplantation
Causes infections in immunocompromised patients eg. HIV patients, patients after organ transplant

Investigations: Serology, PCR, culture

Treatment: Immuno-compromised → Ganciylovir IV

Complications of CMV

- Retinitis
- Pneumonia
- Hepatitis
- Colitis

Prevention: Use antiviral medications eg. Ganciclovir in patients undergoing organ transplant

BACTERIAL INFECTIONS

BRUCELLOSIS

It is a zoonosis meaning it is acquired from animals through animal contact, droplet inhalation, unpasteurized goat milk especially in the Middle East, Far East, Bosnia
Typical history is a patient who traveled or worked in a farm areas.

- B. meletensis⟶ sheep / goat
- B. suis ⟶ pigs
- B.canis⟶dogs
- B.abortis ⟶ cattle

Symptoms: indolent & last for years

- Pyrexia of Unknown Origin
- Sweat, malaise, anorexia, weight loss
- Hepatosplenomegaly
- Rash

- Constipation
- Myalgia, arthritis, spinal tenderness, bone pain

Investigations

- Blood culture
- Serology (anti – O polysaccharides Abs)
- Rose Bengal test → screening

Complication: Osteomyelitis

Treatment: Doxycycline + Gentamycin + Rifampicin

LISTERIA MONOCYTOGENES

Source: pates, raw vegetables, unpasteurized milk & soft cheese

Symptoms:

- Flu–like illness
- Pneumonia
- Meningo–encephalitis
- Ataxia
- Rash → especially in immuno-compromised
- Pregnant→ miscarriage or still birth

Investigation: Blood culture, CSF, amniotic fluid (neonatal sepsis)

Treatment: Ampicillin + gentamycin (if allergic, give erythromycin)

NB. Listeria monocytogenes commonly causes meningitis in patients above 50 years of age.

TUBERCULOSIS:

Caused by Mycobacterium tuberculosis

History: Asia, Africa. Tuberculosis is more common in Africa.

Symptoms:

- Weight loss
- Night sweat
- Cough with copious sputum (long standing > 3 months)
- Haemoptysis

Screening:

- **Chest X-ray**→ capsule looking for dormant foci
- **Sputum analysis** for Acid Fast Bacilli

Diagnosis:

- **Microscopy:** Acid Fast Bacilli in clinical samples like sputum, pus, urine (PCR), CSF (PCR), ascites,
- **Culture:** use the Lowein-Stein Jenson medium
- **Histopathology (biopsy)** – Caseating granuloma
- **Radiology:** Chest X-Ray – consolidation, cavitation, fibrosis, calcification
- **Immunological:** Tuberculin skin test (not in UK)
  Should be read between 48 and 72 hours after administration
  Interpretation: Depends on measurement in millimeters of the induration, and person's risk of being infected with TB and of progression to disease if infected

Treatment of Pulmonary TB:

Initial phase (2 months):
Rifampicin, isoniazide, pyrazinamide, ethambutol

Continuation phase: (4 months)
Rifampicin & isoniazide

Done as Direct Observation of Treatment (DOT)

Isolation
Done in patients with suspicious chest x-ray, past history of MDR-TB (multi drug resistant TB)

Termination of isolation:
After 3 or more –ve sputum samples on 2 stains for MDR- TB

Other Types of Tuberculosis:

Haematogenous spread → miliary TB
TB spine → Pott's disease
TB skin → lupus vulgaris (face / neck jelly like nodule)

6. LYME DISEASE

Scenario gives a bite in the forest/on a hiking trip
Caused by the a tick borne spirochete Borrelia burgdoferi

Symptoms:

- Erythema migrans → site of tick bite → after 3 – 30 days, redness gradually expands with central clearing
- Fatigue
- Chills
- Fever

- Headache
- Muscle & joint pain

Complications

- Cranial nerve palsy
- Lymph adenopathy
- Myocarditis

Investigation: Blood tests are not necessary, it is a clinical diagnosis. But if the question asks for investigation, do serology. If –ve do PCR.

Treatment

- **Doxycycline is the first choice.**
- **Cefuroxime if both are contra-indicated and no allergy to penicillins**
- **If pregnant or <12 years: Amoxicillin or cefuroxime**

10. TYPHOID FEVER

Caused by salmonella

Symptoms →

- Fever
- Relative bradycardia (increase in temperature without corresponding increase in heart rate)
- Rose spots on the abdomen

Investigation:

- Vidal test
- Culture from blood or urine sample

Treatment: Ciprofloxacin

STREPTOCOCCAL INFECTIONS

a) CELLULITIS

Infection of the skin. Commonest cause is Streptoccus, occasionally caused by Staphylococcus

Signs & Symptoms:

- Fever
- Affected area is red, warm to touch
- POORLY demarcated borders

Treatment:

1. **If localized limb infection and no evidence of systemic upset give oral antibiotics. Flucloxacillin + penicillin**
2. **If systemically unwell, admit and give IV antibiotics: flucloxacillin+ penicillin OR co-amoxiclav**

b) ERYSIPELAS

Streptococcal infection limited to more superficial parts of the skin

Signs & Symptoms:

- Affected area is red, warm to touch
- CLEARLY demarcated borders

Treatment: Oral phenoxymethylpenicillin

C) TONSILLITIS

Commonest cause is infectious mononucleosis. If bacterial infection will have pus.

Signs & Symptoms:

- Sore throat
- Pain on swallowing
- Fever
- Enlarged hyperaemic tonsils with **white exudates/pus** (bacterial)

Treatment: Penicillin V

D) SCARLET FEVER

Streptococcal infection causing diffuse scarlet rash usually neck, chest, axillae and groin.

Signs & Symptoms:

- Rough sandpaper like skin (due to occlusion of sweat glands)
- White strawberry tongue

Treatment: Penicillin. If allergic give Erythromycin.

F) NECROTIZING FASCIITIS

Serious condition requiring prompt surgical attention. Rapidly progressing infection, spreads along fascial planes. Patient is usually septic and there is often multiorgan failure if treatment is delayed.

Cause: Commonest B haemolytic streptococcus

Clinical Features:

- Could be recent trauma or surgery
- It may complicate chickenpox
- Erythema/redness, necrosis, pus discharge
- Fever, tachycardia,

Investigations:

- Swab and blood culture
- X-ray shows gas in the soft tissue, but may be normal

Treatment: Urgent surgical debridement is the main treatment.
Resuscitation with IV fluids, antibiotics (penicillin & clindamycin)

***Fournier's gangrene** - Necrotizing fasciitis of the scrotum.

STAPHYLOCOCCAL INFECTIONS

S. aureus (coagulase –ve)

a. **IMPETIGO**

Highly infectious superficial skin infection caused by Staphylococcus (may also be caused by Streptococcus). Common in children.

Clinical Features

- Lesions usually start around mouth and nose spreading rapidly on the face and other parts of the body.
- Usually there are irregular golden-yellow crusted lesions in streptococcal infections
- Staphylococcus may cause bullous impetigo with a bulla-containing pus

Treatment: Oral flucloxacillin or topical fusidic acid

a. **STAPHYLOCOCCAL SCALDED SKIN SYNDROME**

Caused by Staph aureaus. Causes separation of the outer layers of the epidermis which slide off with minimal pressure leaving large raw areas resembling a severe scald. Common in children.

a. **OSTEOMYELITIS**

Infection of the bone, common in children and immunocompromised patients.
Signs & Symptoms: Bone tenderness, fever, redness, swelling

Treatment: IV clindamycin

a. **INFECTIVE ENDOCARDITIS**

May develop on previously normal heart valves as well as on diseased or prosthetic valves.

Signs & Symptoms: Fever + new murmur is infective endocarditis until proven otherwise

Treatment: Benzyl penicillin

a. **STAPHYLOCOCCAL SEPTICAEMIA**

Occurs particularly in immunocompromised patients and in IV drug abusers.
Signs & Symptoms: High fever

Treatment: IV Flucloxacillin

a. **TOXIC SHOCK SYNDROME**

Caused by S. Aureus. Common in women who use tampons, during menstrual period. May occur after surgical operations, burns, trauma, or local infections

Signs & Symptoms:

- High fever
- Generalised rash
- Confusion
- Diarrhea
- Renal failure
- Hypotension

Treatment: IV Flucloxacillin, refer to ICU

MRSA (Methicillin resistant Staphylococcal Aureus)

Typically a hospital-acquired infection causing pneumonia, septicaemia, wound infection and death.

Prevention

- **Isolate patient with suspected MRSA**
- **Wash hands before seeing patients**
- **Screen patients for MRSA**
- **Use gown and gloves when dealing with infected or colonized patients**
- **Take surveillance swabs of patients and staff during MRSA outbreaks**

Treatment: Vancomycin IV

CLOSTRIDIUM INFECTIONS

Gram +ve anaerobic infections

a) TETANUS
Caused by Clostridium tetani (gram +ve). Spores are usually found in the soil and animal faeces and are transmitted when they contaminate the wound. The spores produces an exotoxin (tetanospasmin) which blocks the nerves in the CNS causing spasm and rigidity.

Acute fatal disease common in Asia, Africa and South America. Very rare in developed countries.

Clinical features

- Stiffness of masseter muscles causing difficulty opening mouth (trismus or lockjaw)
- Spasm of chest muscles (may restrict breathing)
- Abdominal rigidity
- Stiffness of limbs
- Forced extension of the back (opisthotonus)
- Fever is common

Treatment

- **Refer to ICU**
- **Penicillin + metronidazole**
- **Diazepam for spasms**
- **Tetanus immunoglobulin (Ig)**

Prophylaxis
Depends on the 1) wound and 2) tetanus status of patient

- Wound may be classified as **CLEAN** or **TETANUS PRONE**

Tetanus prone:

- Heavy contamination especially with soil or faeces
- Wounds >6 old (patient presents after 6 hours)
- Puncture wounds and animal bites
- Devitalized tissue
- NB. PREGNANCY IS NOT A CONTRAINDICATION TO GIVE TETANUS PROPHYLAXIS
- DO NOT GIVE IF PATIENT HAD PREVIOUS SEVERE REACTION
  ****GIVE IgG IN <u>ANY</u> TETANUS PRONE WOUND REGARDLESS OF IMMUNISATION STATUS**

Full immunisation course:

- Initial course (3 doses in infancy)
- 2 boosters (4 years and 14 years)

When to give the vaccine after a wound:

a. Fully immunised patient (5 doses): No vaccine
b. Initial course incomplete or boosters not up to date: Give vaccine now and refer to GP to complete full course of vaccination
c. Not immunized or immunisation status unknown or uncertain: Give vaccine now refer to GP to complete full course of vaccination

Antibiotic Prophylaxis

- Not required for most wounds.
- Give antibiotics in open fractures (penicillin + flucloxacillin), hand wounds, mouth wounds, human bites, animal bites, contaminated wound, puncture wounds, patients present after 6 hours.

b) BOTULISM

Exotoxin paralyses the autonomic and motor nerves by blocking Acetylcholine at the neuromuscular junction.

Infection from eating tinned or preserved food contaminated with C. Botulinum spores.

Clinical Features:

- Dry mouth
- Cranial nerve palsies (ptosis, squint, fixed pupils, weakness of tongue)
  Coma
- Limb weakness

Treatment: Botulinum anti-toxin. Refer to ICU.

c) GAS GANGRENE
Caused by C. Perfringens. Rapidly spreading infection of muscles. Fatal if untreated. May involve wounds of the buttocks, amputations for vascular disease or gunshot wounds.

Clinical Features:

- Sudden severe pain at wound site
- Sweating
- Fever
- Swelling and discolouration around wound

Investigations:

- X-ray shows soft tissue gas
- Swab of wound discharge

Treatment: IV Penicillin + IV clindamycin

c) PSEUDOMEMBRANOUS COLITIS

Caused by C. Difficile.
Also known as Clostridium difficile-associated diarrhoea/disease (CDAD), CD-positive diarrhoea, antibiotic-associated colitis.

Risk factors

- Prolonged courses of antibiotics or multiple antibiotic usage
- Increasing age, severe comorbidity
- Nonsurgical invasive gastrointestinal procedures eg. NG tube.
- Increasing duration of hospital stay, patients in long-term care facilities.
- Immunocompromised patient

Clinical Features:

- Symptoms usually between 5 and 10 days after antibiotic therapy. May occur up to10 weeks after.
- Watery diarrhoea ± blood-stained stools, abdominal cramps, fever
- Severe cases: rigors ± septicaemia.
- Severe abdominal pain is uncommon but may mimic an acute abdomen.
- Frank rectal bleeding suggests other causes (eg. inflammatory bowel disease).

Investigations: C. difficile toxin in stool (Stool cytotoxin test)

Treatment:

- **Stop offending antibiotic if possible**
- **Give oral metronidazole**
- **Use oral vancomycin if severe**

TRAVELLERS DIARRHEA

- Most common cause is E. coli
- Illness lasts 3-5 days with nausea, abdominal cramps, watery diarrhea with no blood
  Treatment: Oral hydration

Giardiasis

- **Causes chronic diarrhea (>14 days)**
- Suspect Giardiasis in any diarrhea lasting more than 14 days
- Caused by Giardia lamblia
- Spread by feco-oral route.
- Investigation: Stool microscopy for cyst
- **Treatment: Metronidazole**

Cholera

- **Profuse rice-water diarrhea**, vomiting, fever, abdominal
- Caused by Vibrio cholera
- Uncommon in Western nation, usually after ingestion of raw seafood
- **Treatment: Self limiting disease but tetracycline may be used**

- **If diarrhoea presents with blood**, possible causes are amoeba dysentery, shigella, campylobacter

Bacterial Dysentery

- Usually caused by Shigella
- Feco-oral transmission
- **Usually causes bloody diarrhea**
- **Treatment: Amoxicillin if severe**

Amoebic Dysentery

- Caused by Entamoeba histolytica
- Causes intermittent diarrhea +/- blood in stool
- **Risk factors: Homosexuals, and patients with recent travel to third world countries**
- Investigations: Stool culture and microscopy for amoebic cyst
- **Treatment: Fluid replacement, metronidazole**

OTHER INFECTIONS

1. **SCABIES**

Caused by the mite Sarcoptes scabei.

Clinical Features

- Most often found in fingerwebs and flexor aspect of the wrist.

- **Intense itching (allergy to toxin produced by the mite)**
- May form burrows in between fingers
- Commonly found in nursing homes
- Usually spreads in families

Treatment:
1. Permethrin or malathione.
2. Oral antihistamine for severe itching.

2. LEISHMANIASIS

- Granulomatous disease caused by the protozoa *Leishmania*
- Transmitted by the sand fly in Africa, India Middle East, Mediterranean, Latin America and Southern USA

Types:

1. Cutaneous Leishmaniasis
2. Mucocutaneous Leishmaniasis
3. Visceral Leishmaniasis

1. **Cutaneous Leishmaniasis** - lesions develop at the bite from an itchy papule which crusts and falls off to leave an ulcer (Chiclero's ulcer). Heals with a scar.

Investigation: Culture and microscopy of aspiration from ulcer
Treatment: Topical paromomycin

1. **Mucocutaneous Leishmaniasis** - may spread to mucosa of nose (called espundia), pharynx, plate, larynx and upper lip. Causes severe scarring.

Investigation: Leishmanin skin test, antibodies or PCR
Treatment: Sodium stibogluconate

1. **Visceral Leishmaniasis also called Black Sickness** - spreads via the lymphatics from the skin and multiplies in the reticuloendothelial system. Presents with dry warty hypo pigmented skin lesions, pyrexia, sweats, arthralgia, abdominal pain, hepatosplenomegaly, lymphadenopathy.

Investigation: Microscopy of lymph nodes, bone marrow or spleen
Treatment: Liposomal amphotericin B is the first line. Can also use miltefosine.

3. SCHISTOSOMIASIS

Causative organisms:

- Schistosoma japonicum
- *Schistosoma mansoni*
- ***Schistosoma haematobium*** *- Causes urinary tract infection which increases risk for bladder carcinoma*

Usually there is history of travel to tropical countries and swimming in rivers.

Signs & Symptoms: Earliest symptom may be swimmer's itch
Complications: Increased risk of bladder cancer with S. haem

- **Treatment: Praziquantel**

4. MALARIA

Causes

- **Plasmodium falciparum** →

- Responsible for severe disease and malaria-related deaths.
- Incubation 7-14 days (up to 1 year if semi-immune); most travellers present within 8 weeks.
- Classical tertian and subtertian periodicity (paroxysms at 48- and 36-hour intervals) are now rare,

- **Plasmodium Vivax**
  - Causes benign tertian malaria - fever every third day.
  - Incubation period of 12-17 days.
  - Relapse due to dormant parasites in the liver.
- **Plasmodium ovale**

- Relapsing course as with P. vivax.
- Incubation period of 15-18 days.

- **Plasmodium malaria**

- Causes benign quartan malaria - fever every 4th day, (not common in early infection)
- Long incubation period (18-40 days) Parasites can remain dormant in the blood. 5-10% present over a year after infection

Signs & Symptoms

- Fever, often recurring
- Chills & rigors
- Headache, cough, myalgia
- GI upset
- Splenomegaly & hepatomegaly
- Jaundice
- +/- abdominal tenderness

Signs of severe disease (usually *P. falciparum*)

- Impaired consciousness
- Shortness of breath
- Bleeding

- Fits
- Hypovolaemia
- Hypoglycaemia
- Renal failure
- Nephrotic syndrome

Complication: Cerebral malaria – 20% mortality

Investigation: Thick and thin blood film

TREATMENT OF NON-FALCIPARUM MALARIA:

- **P. Malaria: Chloroquine**
- **P. Vivax and ovale: Chloroquine and primaquine**
- **Prevention of relapse: primaquine**

TREATMENT FOR UNCOMPLICATED FALCIPARUM MALARIA

- **Oral quinine + doxycycline. Give sugar because quinine will lead to hypogylcemia.**

TREATMENT OF SEVERE OR COMPLICATED FALCIPARUM MALARIA

- **IV quinine is first-line. ECG monitoring is required.**
- **Switch to oral quinine once patient is well enough to complete a 5- to 7-day course in total.**
- **Give Doxycycline (clindamycin for pregnant women) for a total of 7 days from when the patient can swallow.**

PROPHYLAXIS

- Take 1 week before travel up until 4 weeks after return
- Not a guarantee against infection
- Medicine depends on the area of travel
- N**o chloroquine resistance areas,** give Chloroquine
- **Moderate chloroquine resistance areas** give Chloroquine plus proguanil (include folate in pregnancy)
- **High choloroquine resistance areas** give Quinine + doxycycline
- In **pregnant patients** give Quinine + clindamycin

5. TOXOPLASMOSIS
The cause is Toxoplasmosis gondii and is usually acquired from cat faeces through the feco-oral route.

50% in UK are affected (presents only when immunocompromised especially with HIV)

CT head shows ring enhancing lesions

Treatment: Pyrimethamine + sulfadiazine

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Resource description	Nephrology
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NEPHROLOGY
LECTURE NOTES 2014

FUNCTION OF THE KIDNEYS:

1. To excrete the end products of the metabolic processes in the body like urea
2. To maintain the acid balance in the body.
3. It produces hormones like:

- Renin
- Erythropoietin
- Prostaglandins
- Vitamin D

1. Some hormones act on the kidney:
2. Antidiuretic hormone (ADH or vasopressin)
3. Aldosterone
4. Atrial natriuretic peptide
5. Parathyroid hormone

INVESTIGATION IN NEPHROLOGY

1. **URINE DIPSTIX OF MID-STREAM URINE:**

HAEMATURIA

A. **Painful haematuria**
B. **Painless haematuria**

Causes of Painful Haematuria

1. **Urinary Tract Infection**

- Dysuria
- Frequency
- Fever
- ± abdominal pain
- Positive nitrates on urine dipstix

1. **Renal Calculi**

- No fever
- Right or left iliac fossa pain radiating to the groin is ureteric colic and if loin pain then it's renal stones.
- Haematuria

1. **Urological Trauma:** e.g Urethral injury

There will be history trauma and signs of trauma like bruises in the perineum, ± blood in the external meatus.

1. **Honeymoon cystitis**

History of painful sexual intercourse and recent onset of sexual intercourse or recently marrried.

It presents like UTI i.e dysuria, frequency and haematuria but MSU and urine dipstix are normal.

Causes of Painless Haematuria

1. Renal Tumour

- Palpable mass in the renal angle
- Signs of malignancy like weight loss, anaemia and anorexia.
- Confirmed by ultrasound scan or CT scan

1. **Bladder Tumour:** *Painless haematuria in an adult is bladder cancer until proven otherwise*

- Pelvic mass
- Pain in suprapubic area
- Haematuria
- Signs of malignancy (weight loss, anaemia and anorexia)
- Confirmed by cystoscopy

1. **Bleeding Diathesis**

- Bruises on examination
- History of being on anticoagulant therapy

1. **Prostate Cancer**

- Back pain
- PSA is raised (normal PSA: 0 - 4ng)
- Haematuria
- Nodular prostate on per rectal examination

1. **Rhabdomyolysis**

- History of crush syndrome ie. history of lying on the floor for several hours under a heavy object
- Myoglobin colours urine red.

1. **Haemolysis**

- Haemoglobulinuria
- Jaundice
- Anaemia

FBC = Shows ↓ Hb (anaemia)

1. **Polycystic Kidney Disease:** Autosomal dominant (1:2)

- Hypertension
- Family history of polycystic kidney disease
- Haematuria

PROTEINURIA (Normal protein excretion < 150 mg/day)

Causes:

1. Urinary Tract Infection (please see haematuria)
1. Orthostatic proteinuria (after standing for a long time – occupations like security guard)
1. Glomerulonephritis (proteinuria, hypertension and haematuria)
1. Hypertensive nephropathy (history of longstanding high blood pressure)
1. Diabetic nephropathy (history of diabetes mellitus)
1. Myeloma (elderly age >60 years, ESR ↑, back pain, anaemia and high ESR)

GLUCOSURIA

Usually due to diabetes mellitus, there might be ketones in the urine as well.

↑ WHITE CELL COUNT

Usually due to infection.

KETONES

Usually in diabetic ketoacidosis, starvation or dehydration. There might be glucosusia.

NITRATE

A sign of infection, the commonest causes are E.coli, Proteus and Klebsiella. So there will be fever.

MICROSCOPY OF MID-STREAM URINE:

1. ↑ **Leukocytes:** common cause is urinary tract infection
2. ↑ **Red cells:** means haematuria
3. **Casts:** these are cylindrical aggregates formed in the distal tubules or collecting ducts.

There are different types:

a. Hyaline casts and fine granular casts are normal findings. Hyaline casts are mainly protein.
b. Red cell casts indicate glomerulus bleeding, usually due to glomerulonephritis.
c. White cell casts suggest acute infection, usually bacterial (pyelonephritis).
d. Fatty casts can occur in nephrotic syndrome.
e. Tubular cell casts occur in acute tubular necrosis (ATN).

4. **Crystals**: OXALATE - indicate predisposition to form calculi
CYSTINE - diagnostic of cystinuria

5. **Glomerular Filtration Rate:**

Normal is 120 ml/min, if it falls below 30 ml/min then it causes urea and creatinine to accumulate in the body. High levels of creatinine indicates renal failure.

6. **Immunological Investigation:**

a. Anti-Neutrophil Cytoplasmic Antibodies (ANCA) - suggests vasculitis
b. Anti-Glomerular Basement Membrane Antibodies - suggest Goodpasture's Syndrome.
c. Anti-Nuclear antibodies and Double Stranded DNA for antibodies and low Complement levels -suggests SLE.

7. **Ultrasound Scan**: Good for evaluation of renal masses, hydronephrosis and for any renal masses e.g. tumour or polycystic kidney disease.

8. **KUB (x-ray Kidney Ureter and Bladder):** the initial investigation for all renal stones and it can detect 99% of all radio-lucent stones. Radio-opaque stones like urate stones are not visible on KUB.

9. **Intravenous Urography (IVU) or Excretory Urograph:** the investigastion of choice for all renal stones.

10. **Renal Biopsy:** is used for chronic and acute renal failure, nephrotic syndrome and glomerulonephritis if the cause is not known.

11. **Retrograde Pyelography:** is used to determine the lower level of obstruction after IVU or ultrasound scan has shown obstruction.

12. **Cystoscopy:** is the investigation of choice in bladdar tumour.

13. **CT KUB**: can be used instead of IVU for evaluation of renal stones. NICE guidelines recommend IVU. So if you have both of them in the options choose IVU.

14. **Antegrade Pyelography:** Percutaneous injection of contract into the pelvi-calyceal system and ureter. It is used when ultrasound scan has shown dilated pelvi-calyceal system in a patient with suspected obstruction. It is used when a draining catheter is need to be placed i.e nephrostomy. Also, if a stent needs to be put if there is a Ureter Stricture.

15. **Micturating Cystourethrography (MCU):** This investigation is done to look for vesicoureteric reflux, It is an investigation which is done in children only to look for vesicoureter reflux. If reflux is found in children surgery is usually done.

16. **Renal Arteriography:** is used in renal artery stenosis

17. **Dynamic Scintigraphy:** Uses Technium 99 (Tc99). Contrast is inserted into the veins and it is followed by a gamma camera. It allows to detect renal perfusion e.g. in renal stenosis.

Indications:

a. Renal artery stenosis is suspected as a cause of high blood pressure.
b. In severe oliguria e.g. post-trauma, post aortic surgery or after kidney transplant.

18. **Static Scintigraphy:** Tc99 is used to check for renal function. This susbtance is taken up by tubular cells according to their function. This is done to identify ectopic kidneys and pseudo-tumours.

N.B. Dynamic and Static Scintigraphy are also called Isotope Scans. They are used for investigating kidney damage caused by urinary tract infection in children.

URINARY TRACT INFECTION (UTI):

1. Bacteriuria: presence of bacteria in the urine. It can be symptomatic and asymptomatic.
2. Urethritis: infection of the urethra
3. Cystitis: bladder infection
4. Pylonephritis: infection in the kidney
5. Prostatitis: infection in the prostate
6. Epididymo-orchitis: infection of the testes and epididymis

RISK FACTORS

1. Female
2. Sexual intercourse
3. Pregnancy
4. Menopause
5. Immunosuppression
6. Diabetes Mellitus
7. Urinary obstruction due to stones or tumour, faecal impaction, malformation and catherization.

A recurrent urinary tract infection is a furthur infection with a new organism. Common in females. Usually 2 or more infection in a year with different organisms.

A relapse is a further infection with the same organism. Usually 2 or more infection within a year.

Uncomplicated UTI: is when you develop urinary tract infection with normal renal function and normal GU tract anatomy.

Complicated UTI: This is UTI in the presence of abnormal GU tract or abnormal renal function e.g. urinary obstruction.

N.B: Assume all UTI's in men and children (whether boy or girl) without any risk factors as complicated.

UTI in a catheterised patient: The urine may turn **purple** in the catheter bag due to breakdown of urine substances into pigments by bacterial enzymes

Common causes of UTI are as follows and in this order:

1. E.Coli
2. Proteus
3. Klebsiella
4. Pseudomonas.

Symptoms:

- Cystitis: frequency, dysuria, urgency, haematuria, suprapubic pain
- Acute Pyelonephritis: high fever, vomiting, loin pain
- Prostatitis: flu-like symptoms, lower backache, swollen or tender prostate on per rectal examination.
- Epididymo-orchitis: swollen, painful testes.

Investigation:

1. Urine dipstix will show nitrates, white cells or there is presence of blood
2. Mid stream urine for microscopy, culture and sensitivity
3. Cystoscopy if bladder cancer is suspected eg. in an elder patient with recurrent UTI
4. Ultrasound scan for UTI in children, men and if recurrent in women in order to look for predisposing factors.

- In young men and women, the most likely cause is renal stones so investigate with IVU
- If IVU is normal perform an ultrasound scan and vice versa
- In children preform an ultrasound scan first
- Suspect bladder cancer in elderly patients with recurrent UTI so perform Cystoscopy
- In children, UTI can cause kidney damage and lead to kidney fibrosis which may cause hypertension whey they grow up so perform an isotope scan to check for any damage.

Treatment:

1. Bacterial UTI in women: Trimethoprim, nitrofurantoin or cefalexin. 2nd line is co-amoxiclav.
2. Acute Pyelonephritis: Co-amoxiclav
3. Bacterial UTI in men: Levofloxacin

NB. In pregnant women with UTI, use amoxicillin and cefalexin

DIFFERENTIATING SEXUALLY TRANSMITTED INFECTIONS FROM UTI

1. Gonorrhea: purulent discharge, symptoms usually appear 2-3 days after unprotected sexual intercourse. Short incubation period of gonorrhea (<1 week).

2. Chlamydia: mucoid discharge and is the commonest sexually transmitted disease in the UK. There might be travel history for a holiday e.g Thailand or change of sexual partners (PID).

3. Hemophilus Ducreyi: there will be intense dysuria and on examination there are multiple ulcers and tender inguinal lymphadenopathy.

4. Syphilis: transient painless ulcer, there might be history of unprotected sex over several months. It can be as long as 3 months, due to long incubation period.

PROPHYLAXIS:

1. **Antibiotic prophylaxis:** if given continuously or post-coital it decreases infection rates in women with recurrent UTI or immunocompromised patients with recurrent UTI.
2. **Local oestrogen** in post-menopausal women.
3. **Hygiene and double voiding** in honeymoon cystitis.

NEPHROTIC SYNDROME (Heavy Proteinuria):

Nephrotic syndrome is a triad of Proteinuria (>3.5g/24hr, Hypoalbuminaemia <25g/L and Oedema.

Nephrotic syndrome is not a diagnosis. >80% are due to Glomerulonephritis, DM, Amyloidosis, SLE and Drugs.

Complications:

1. Increased risk of infections due to loss of immunoglobulins.
2. Thromboembolism e.g. deep vein thrombosis/pulmonary embolism, renal vein thrombosis - due to increased clotting factors.
3. Hyperlipidaemia- increase cholesterol and triglycerides production by the liver in response to low oncotic pressure.

Investigation: Renal Biopsy to find out what type of glomerulonephritis it is.

Treatment:

1. Reduce oedema with loop diuretic e.g. Furosemide
2. Reduce proteinuria ACE inhibitor or Angiotension receptor blocker (ARB) should be started.
3. Treat the underlying cause

GLOMERULONEPHRITIS: (inflammation of the glomeruli)

Is a common cause of End Stage Renal Failure

Presentation:

pastedGraphic.png

Investigation:

1. ANA
2. ANCA
3. ASOT
4. Anti-ds DNA
5. Anti-aBM
6. Urine for protinuria and heamaturia
7. Renal Biopsy

TYPES OF GLOMERULONEPHRITIS

1.**Thin basement membrane nephropathy**: It is an autosomal dominant disease. It is benign persistant microscopic heamaturis in children. Reassure.

2.**Minimal change disease:** common cause of nephrotic in children. On light microscopy looks normal but on electron microscope shows Fusion of Podocytes.
Treatment: Cyclophosphamide

3.**Membranous Nephropathy**: presentation usually is Nephrotic Syndrome. It is associated with SLE, Malignancy, Gold, Penicillin, Rheumatoid Arthritis. Renal Biopsy- shows thickened basement membrane, Rx: Steroids + Cyclophosphmide

4.**Ig A Nephropathy (Berger's Disease):** typically presents as Macro or Micro-Heamaturia and is typically in a young male child with hematuria 1-2 days after URTI, Treatment: Cyclophosphamide

5.**Focal Segmental Glomerulosclerosis:** associated with HIV, Hepatitis, Ig A Nephropathy and it can be Idiopathic. Renal Biopsy shows some glomeruli have scarring of certain segments.
Treatment: Steroids, cyclophosphamide, cyclosporin.

6.**Proliferative Glomerulonephritis**: the chief cause is Post- Strep glomerulonephritis. Typically 1-12 weeks after sore throat (URTI). Presentation is Nephritic Syndrome which consists of Hematuria, Proteinuria and Hypertension. It is the same as acute glomerulonephritis. Treatment: Supportive

7.**Rapidly Progressive Glomerulonephritis:** There are many causes, especially anti-glomerular basement membrane (Goodpasture's, SLE, IgA) Renal biopsy shows Cresent (macrophage) in bowman's Capsule. Treatment: high dose steroids and Cylophosphamide.

8.**Mesangiocapillary Glomerulonephritis**- Biopsy shows proliferation of the mesengial cells and presents as Nephrotic Syndrome, Rx- Steroids

9.**Henoch-Schonlein Purpura**- is a systemic variant of IgA nephropathy causing small vessel vasculitis. Smptoms are Purpuric rash on extensor surface typically on legs, abdominal colic, polyarthritis and GN.

ACUTE KIDNEY INJURY

The term acute renal failure has now changed due to acute kidney injury.

It is defined as deterioration in renal function occurring over hours or days. Biochemically is detected by raising Urea and Creatinine.

Causes:
Commonest causes are ischaemia, sepsis and nephrotoxins. The causes can be classified as:

1. Pre-Renal: Renal hypoperfusion due to hypovolaemia (vomiting, dehydration, diarrhea or bleeding), sepsis (causing systemic vasodilatation), liver failure, renal artery stenosis.

2. **Intrinsic renal**: ATN damage to tubular cells due to ischaemia or nephrotoxins such as haemoglobin, drugs (antibiotics, NSAIDS or ACEI), Myoglobin.

3. **Post-Renal**: Caused by urinary tract obstruction. Common causes of obstruction are benign prostatic hypertrophy, stones or stricture.

MANAGEMENT: Treat the underlying cause

Prerenal: Correct volume depletion, treat sepsis if present

Intrinsic Renal: Refer early to nephrology.

Post-renal: Catheterise and consider CT of renal tract. Refer to urology if obstruction is likely.

When deciding treatment of renal injury find out if it is chronic or acute as the treatment is different.

Is the injury acute or chronic?
Suspect chronic injury if the following are present:

- The kidneys are small (<9cm) on ultrasound
- Anaemia
- Low Ca
- High PO4

NB. The only definite sign of chronic disease is previous blood results showing high creatinine/low GFR

TREATMENT OF COMPLICATIONS OF ACUTE RENAL FAILURE

1. **Hyperkalemia**: may cause arrythmia or cardiac arrest

Treatment:

1. 10ml of 10% Calcium Gluconate is cardio protective since it stabilizes myocytes
2. I.V. Insulin with 50ml of 50% glucose. (shifts potassium into the cells)
3. Nebulized Salbutamol (shifts potassium into the cells)

2.**Pulmonary Oedema**:

- High flow O2 is the inital treatment.
- Furosemide is the main treatment (Intravenously)
- Vasodilators like Morphine
- If not responding to furosemide then dialysis

3.**Bleeding**: There can be impaired heamostasis due to increased urea.

- If there is renal failure and active bleeding then give fresh frozen plasma and platelets
- Blood Transfusion to maintain Hb >10
- Desmopressin to increase Factor VIII activity

INDICATION OF DIALYSIS IN ACUTE RENAL FAILURE:

1. Refractory Pulmonary Oedema (not responding to IV furosemide)
2. Persistant Hyperkalemia >7mmol/l (not responding to insulin or salbutamol)
3. Severe Metabolic Acidosis pH <7.2
4. Ureamic Encephalopathy (headache, confusion)
5. Uraemic Pericarditis, if there is Pericardial Rub
6. Drug overdose eg. Lithium, salicylates, etc.

CHRONIC KIDNEY DISEASE (CKD): is a long standing and irreversible reduction in GFR. It is caused by DM, Hypertension, chronic urinary retention, glomerulonephritis, pyelonephritis, polycystic kidney disease and vasculitis.

Causes: Diabetes, hypertension, glomerulonephritis, pyelonephritis, cause may be unknown

Symptoms: fatigue, weakness, anorexia, vomiting, anaemia and ankle swelling.

Investigations:

- FBC will show anaemia due to deficiency of erthropoetin.
- Ultrasound scan will show small kidneys and will exclude obstruction.
- Renal biopsy if the cause is unclear.

Treatment:

1. If Hypertension - ACEi is the first choice drug (not to be given if creatinine is >200)
2. Hyperlipidemia – Statins
3. Pulmonary oedema - Furosemide
4. Anemia – Erythropoeitin
5. Acidosis - consider sodium bicarbonate

Urea and creatinine will be increasing as number of glomeruli decreases so treat hyperkalemia.

If hyperkalemia in chronic renal failure then treat with insulin and glucose.

Long term treatment of chronic renal failure is different from treatment of acute renal failure. In chronic renal failure urea and creatinine will be increasing and it is not a major concern as we know it will be increasing.

1. Long term dialysis (in end stage renal failure)
2. Kidney transplant.

URINARY TRACT STONES

Causes:

1. Hyperparathyroidism: Increased parathyroid hormone leading to increased serum calcium levels leading to formation of bilateral renal stones or recurrent renal stones. Investigation: serum calcium and parathyroid hormone
2. Idiopathic Hypercalcaemia
3. Sarcoidosis there is increased serum calcium and increased ACE
4. Increased vitamin D leads to hypercalcaemia
5. Gout: Increased uric acid forms urate stones which are radiolucent on KUB. Urate stones are common conditions like gout, myeloma and tumour lysis syndrome
6. Familial metabolic causes e.g cystinuria, hyperoxaluria, hyperuricuria
7. Infection predisposes to stones and stones predisposes to infection.
8. Obstruction of urinary tract e.g. stricture on ureter
9. Diet: tea, spinach, and rhubarb predisposes to oxalate stones if all biochemical and imaging in normal check dietary history.
10. Dehydration in long distance runners and also in people who work in hot climate.

TYPE OF RENAL STONES

1. Calcium stones usually combined with phosphates or oxalate.

1. Triple phosphates stones (struvite) consists of magnesium, ammonium and calcium. They are also called Staghorn.

1. Urate stones normally associated with gout and myeloma or after Tumour Lysis Syndrome.

Tumour Lysis Syndrome: Radiotherapy/chemotherapy to break down the cancer cells, causes increased urate, forming stones.

Gout Symptoms: red, swollen, painful joints especially the big toe.

Myeloma usually present with back pain, increased ESR, proteinuria and anaemia.

Renal stone symptoms:

1. Renal stones: renal colic which is pain in the loin, +/- Heamturia.
2. Ureteric stones: ureteric colic which is right /left iliac fossa pain radiates to the groin +/- haematuria
3. Bladder stones: pain in the suprapubic area plus haematuria.

INVESTIGATIONS IN RENAL STONES:

1. Initial is KUB X-ray (can visualise 99% of stones, except urate stones which are radio-lucent)

2. IVU is the investigation of choice in all renal stones whether it is in the ureter or kidney.

2. Mid-stream urine if there is fever because that means there is infection caused by pre-existing stone.

3. If recurrent stones or bilateral stone's and pancreatitis think of hyperparathyroidism Investigation is serum calcium and then parathyroid hormone.

4.If symptoms of obstruction then ultrasound scan to check for hydronephrosis.

URINARY RETENTION: (common in men)

1. **BENIGN PROSTATE HYPERPLASIA**: is the commonest cause (due to poor stream of urine) Symptoms: frequency, nocturia and dribbling. But there is haematuria in BPH.

2. **CLOT RETENTION:** Usually elderly or middle aged male. The patient is usually with bladder cancer. The tumour bleeds and clots block the urethra. Remember, painless haematuria in an elderly patient is always bladder cancer until proven otherwise.

3.**SPINAL CORD COMPRESSION:** There is usually sudden onset of urinary retention and bowel symptoms like constipation, incontinence and lower limb weakness or sensory loss.

4.**FAECAL IMPACTION**: Typically in elderly patient or young child with history of constipation. On examination there can be palpable mass in the abdomen usually left iliac fossa. Faecal impaction presses on the Urinary Tract --> Urinary Retention --> UTI --> Confusion in elderly. There may be overflow diarrhea.

Treatment: Quick evacuation of the faecal impaction with a phosphate enema per rectal if it is causing UTI.

5. **MULTIPLE SCLEROSIS**: Typically in a patient with already diagnosed multiple sclerosis.

Treatment: Intermittent self catheterisation.

6. In children **Meningocele:** is the herniation of meninges like dura and arachnoid or meningomylocele and there is cord involvement.

Treatment: If the child is developing well and is old enough then intermittent self catheterization.

URINARY INCONTINENCE:

1. **Urge incontinence:** the cause is detrusor overactivity or instability. It is found both in nulliparous and multiparous women. Detrusor is the muscle of the bladder and it's overactivity causes a sudden desire to pass urine.

2. **Stress incontinence**: small quantities of urine escape as intra-abdominal pressure rises e.g. during sneezing or cough in the absence of bladder contraction.

3. **Mixed incontinence**: when there is incontinence of urine whether a patient laughs, sneezes or not.

4. **Overflow incontinence**: this happens when there is incomplete bladder emptying, there is dribbling of urine e.g. benign prostatic hypertrophy.

5. **Fistula:** Usually form after surgery eg. hysterectomy, or if there is an inflammatory condition eg. Crohn's disease, diverticulitis. Patients complain of constant leakage of urine. There may also be air or faecal matter in the urine.

N.B: In children incontinence is normal, all you need to use is incontinent pants

STRESS INCONTINENCE:

Continence in women is maintained by the proximal and distal sphincter which maintains the pressure in the urethra to be higher than that of the bladder. There are also other structures like pelvic muscles which supports the urethra and helps to maintain this higher pressure. This pressure is also higher than that of the intra-abdominal.

Factors that may contribute to incontinence:

1. Pregnancy: raises intra-abdominal pressure
2. Delivery: causes weakness of the pelvic muscles therefore lowers the pressure in the urethra.
3. Menopause: lack of oestrogen also weakens the closure of the sphincter, causing decreased pressure in the urethra.
4. Obesity: increases intra-abdominal pressure, therefore losing weight might help
5. Uterine prolapse

Cysotocele: upper front wall of the vagina and bladder attached to it bulges into the vagina.

Urethrocele: lower anterior of the vagina wall bulges, this will displace sphincter and impaired sphincter mechanism causes decreased urethra pressure which leads to stress incontinence.

Enterocele: small intestine bulges into the posterior wall of the vagina may contain intestine in the pouch of douglas.

Uterine prolapse: there is a dragging sensation or feeling of something coming down which gets worse by day. Cystitis, frequency, stress incontinence and difficulty in defecation along with feeling of pressure in the perineum.

Rectocele: part of the rectum bulges into the posterior wall of the vagina.

Treatment of uterine prolapse:

1. Ring pessaries for temporary treatment or for very frail women who cannot undergo surgery.
2. Surgery

INVESTIGATIONS IN URINARY INCONTINENCE:

1. Mid-Stream Urine: if infection (dysuria, frequency, fever)
2. Filling urodynamic assessment
3. Voiding urodynamic assessment - The bladder is filled with normal saline and at the same time pressure in the bladder and intra-abdominal pressure is measured.

Bladder pressure will always be high during incontinence. The abdominal pressure during incontinence will differentiate between stress incontinence and urge incontinence.

- If the abdominal pressure is also high, then the diagnosis is stress incontinence.
- If the abdominal pressure is normal, then the diagnosis is urge incontinence or detrusor instability.

Voiding Urodynamic - is assessed during voiding. If the bladder is filled with 500ml saline and the patient urinated only 350ml it is regarded as abnormal. If the voiding speed is less than 15 ml/sec it is considered abnormal.

Treatment of stress incontinence:

1. Pelvic floor exercises
2. Oestrogen if post menopausal
3. Physiotherapy e.g.vaginal cones
4. Drugs e.g. duloxetine
5. Surgery for severe stress symptoms e.g. colposuspension or bladder neck surgery.

Treatment of detrusor instability:

1. Pelvic exercise
2. Avoid caffeine
3. Bladder training to increase time between voiding
4. Pelvic floor physiotherapy
5. Oxybutin

Fistula

Causes:

- Inflammatory conditions - usually diverticulitis and crohn's disease (inflammatory bowel disease)
- Malignancy - usually rectal carcinoma
- Iatrogenic - post surgery and radiotherapy

Types of fistulas:

- Enterovesical - e.g. colovesical usually presents with pneumaturia (gas in the urine) and faecaluria (fecal matter in the urine)
- Enterovaginal - eg colovaginal. Passage of stool or flatus via the vagina is pathognomonic of a colovaginal fistula. It may also present with frequent vaginal infections or copious vaginal discharge.

NB. Symptoms of the chronic disease causing the fistula may be present. These symptoms will help to determine the cause of the fistula.

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Resource view

Resource name	Neurology PLAB 1 Notes
Resource description	Neurology
Resource content	

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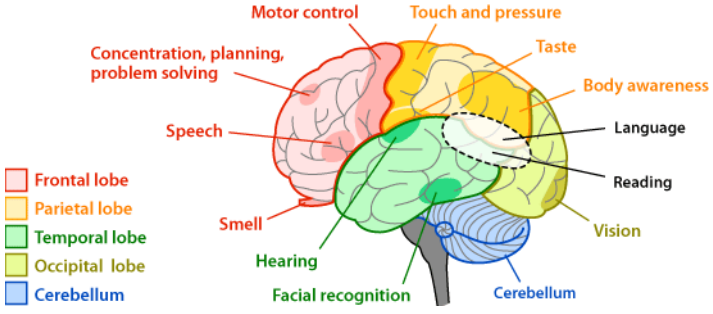
Lecture notes: Neurology

The brain is comprised of the left and right hemisphere. Each hemisphere has four lobes.

Lobes

1. Frontal lobes
2. Temporal lobes
3. Parietal lobes
4. Occipital lobes



Left hemisphere:

- Is usually the dominant hemisphere
- 70% of all left handed people have the left hemisphere as dominant
- Almost all right handed people have the left hemisphere as dominant
- The dominant hemisphere contain areas of speech

Right hemisphere:

- Usually it is non-dominant, meaning no areas of speech

There are 2 areas of speech:

Broca's Area

- Broca's area is located in frontal lobe
- If affected, patient will have expressive aphasia (Broca's aphasia)
- Difficulty to find right words
- Speech is slow
- Comprehension is intact but Coherence is lost
- Writing and reading are impaired
- Difficulty in naming objects

Wernicke's Area

- Reading + writing relatively maintained
- Wernicke's area is located in the temporal and parietal region
- If affected, it causes Receptive Aphasia / Dysphasia
- Fast, fluent speech, full of empty words
- Comprehension is lost but coherence is maintained
- Difficulty in understanding
- Comprehension is lost, coherence is maintained

Dysphasia:

It is a disorder of speech. There are two types:

1. Nominal aphasia: difficulty in naming objects (Broca's aphasia)
2. Global aphasia: Broca's aphasia and Wernicke's aphasia where patient cannot understand and express himself.

Agraphia is:

Disorder of writing

Alexia is:

Disorder of reading

Dysarthria: (slurred speech)

Disorder of articulation e.g. in alcoholics or cerebellar problems due to loss of coordination of the muscles of the tongue

Dysphonia:

Disorder of speech volume e.g. vocal cord lesion(e.g. laryngitis), recurrent laryngeal nerve palsy

Dyspraxia:

Inability to do complex movements. There are different types:

1. Gait dyspraxia
2. Dressing dyspraxia
3. Constitutional dyspraxia (building)

Ataxia is:

Unsteadiness or lack of balance

Brain lesion

Effects of Lesion on the brain: A lesion can do 2 things to the brain

1. Causes destruction/compression on the brain: there will be focal neurological signs like weakness of the legs, visual symptoms, dysphasia sensory loss
2. Can cause irritation of the brain and cause functional problems like epilepsy or hallucinations and delusions.

Focal Neurological Signs:

1. Frontal lobe:

- Personality change
- Intellectual impairment
- Broca's aphasia (if dominant hemisphere)
- Mono or hemiparesis
- Urinary Incontinence

2. Temporal lobe:

- Memory loss (amnesia)
- Déjà vu (a feeling everything is familiar)
- Je'mais vu (failure to recognise situations which have been encountered before)
- Wernicke's aphasia if dominant hemisphere
- Upper quadrant anopia
- Agnosia (loss of perception)

3. Parietal lobe:

- Sensory loss
- Astereognosis (failure to recognise objects by touch)
- Loss of 2 point discrimination
- Wernicke's aphasia (if dominant hemisphere)
- Lower quadrant anopia

4. Occipital lobe:

- Homonymous hemianopia
- Cortical blindness due to bilateral occipital lobe infarction (patient is blind but does not understand that he is blind. He has no insight to his blindness)

5. Cerebellum (is mainly responsible for coordination):

  - Diplopia
  - Dizziness
  - Nystagmus
  - Dysarthria (slurred speech)
  - Hypotonia
  - Past pointing
  - Dysdiadokinesis
  - Heel-shin test positive
  - Ataxia (cerebellar ataxia)

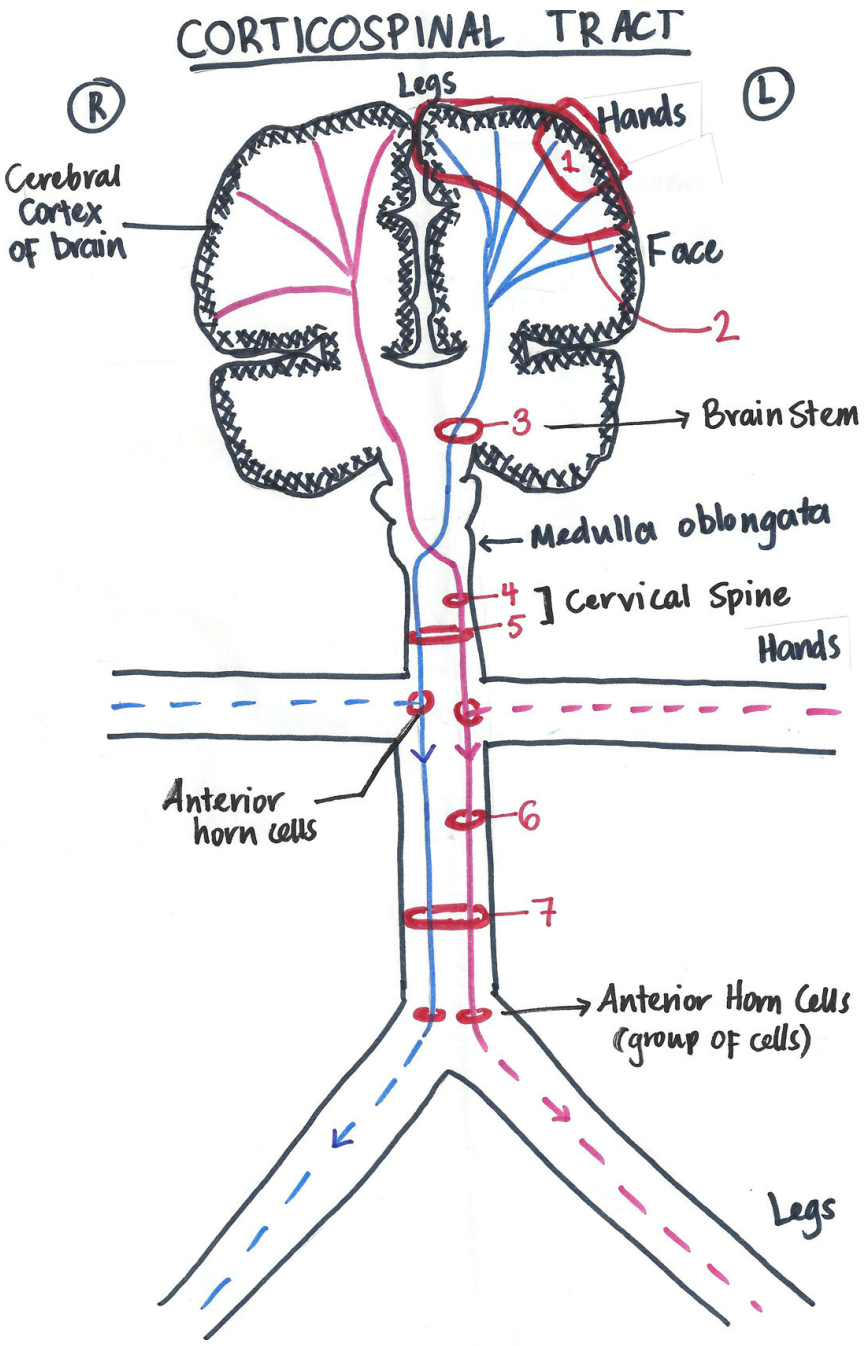
6. Cerebellopontine angle

- There will be cerebellar signs
- Cranial nerves V, VII and VIII are affected together, because they pass through the cerebellopontine angle e.g. acoustic neuroma ((schwanoma) tumour of the VIII nerve)

CORTICO-SPINAL TRACT (Pyramidal system - motor system):

Nerve fibres start in the cortex of the brain and end in the anterior horn cells of the spinal cord. The anterior horn cells are a group of nuclei.

Each part of the brain cortex is responsible for one part of body



1. Cortical Ischaemia - Contralateral monoparesis
2. Cortical Ischaemia - Contralateral hemiparesis
3. Brain stem - Contralateral hemiparsis (cranial nerves will be affected aswell)
4. Cervical spine lesion - Ipsilateral hemiparesis
5. Cervical spine lesion - Tetraplegia or quadriplegia
6. Ipsilateral monoparesis – Left leg for example
7. Complete section of the spinal cord – paraplegia

Upper Motor Neuron signs (UMN): (lesion is above anterior horn cell)

1. Hyperreflexia
2. Hypertonia
3. Upgoing /extensor plantar reflexes (Babinski Sign)

Lower Motor Neuron signs (LMN): (lesion is on the anterior horn cell)

1. Hypotonia
2. Hyporeflexia
3. Flexor plantar
4. Muscle wasting
5. Muscle fasciculations

Motor Neurone Disease (MND):

This is damage to anterior horn cells and nuclei. Only the motor system is affected.

Symptoms:

1. Weakness in the limbs
2. Cranial nerve damage causing dysphagia and speech problems.

Common in middle aged men aged between 35-55 years.

Bulbar Palsy: (Cranial Nerves IX-XII)

LMN lesions of muscles of mastication, swallowing and talking

Signs:

1. Muscle fasciculations
2. Tongue fasciculations
3. Jaw jerk normal or absent
4. Speech is quiet
5. Donald duck speech (nasal speech)

Pseudobulbar palsy:

Lesion is above the mid-pons. UMN lesion of muscles of tongue, chewing, swallowing and facial muscles.

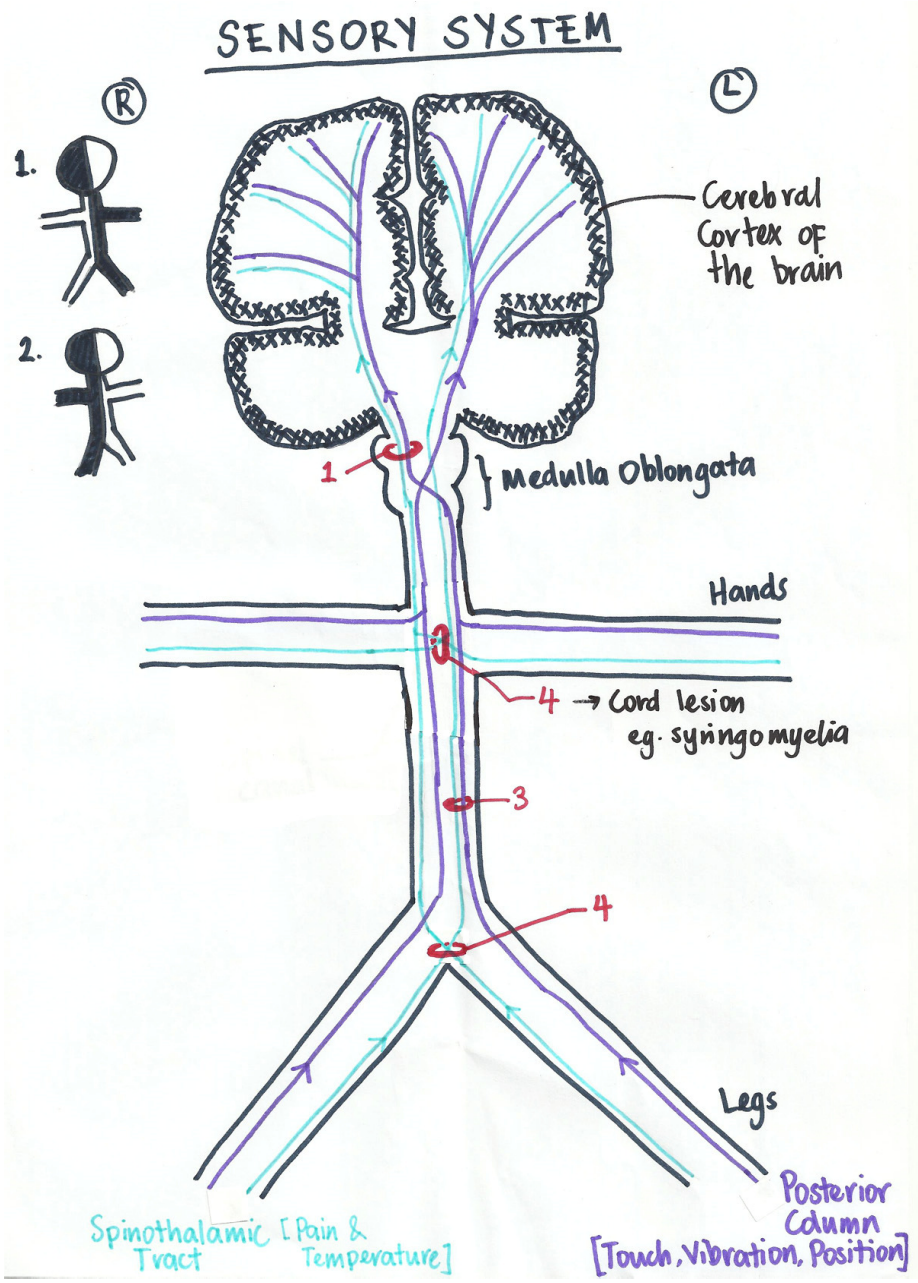
Signs:

1. Increased jaw jerk
2. Unprovoked sorrow
3. Mood incongruence

N.B: In MND there is:

- NO sensory loss only Motor loss
- NO sphincter disturbances
- Eye muscles are NOT affected, which differentiates it from myasthenia gravis.

SENSORY SYSTEM:



N.B: The posterior column carries the light touch, position and vibration. It enters the spinal cord and remains on the same side until the fibres reach the medulla oblongata where they cross to the opposite side.

Spinothalamic tract carries the pain and temperature fibres. It crosses the spinal cord within the first 2 segments.

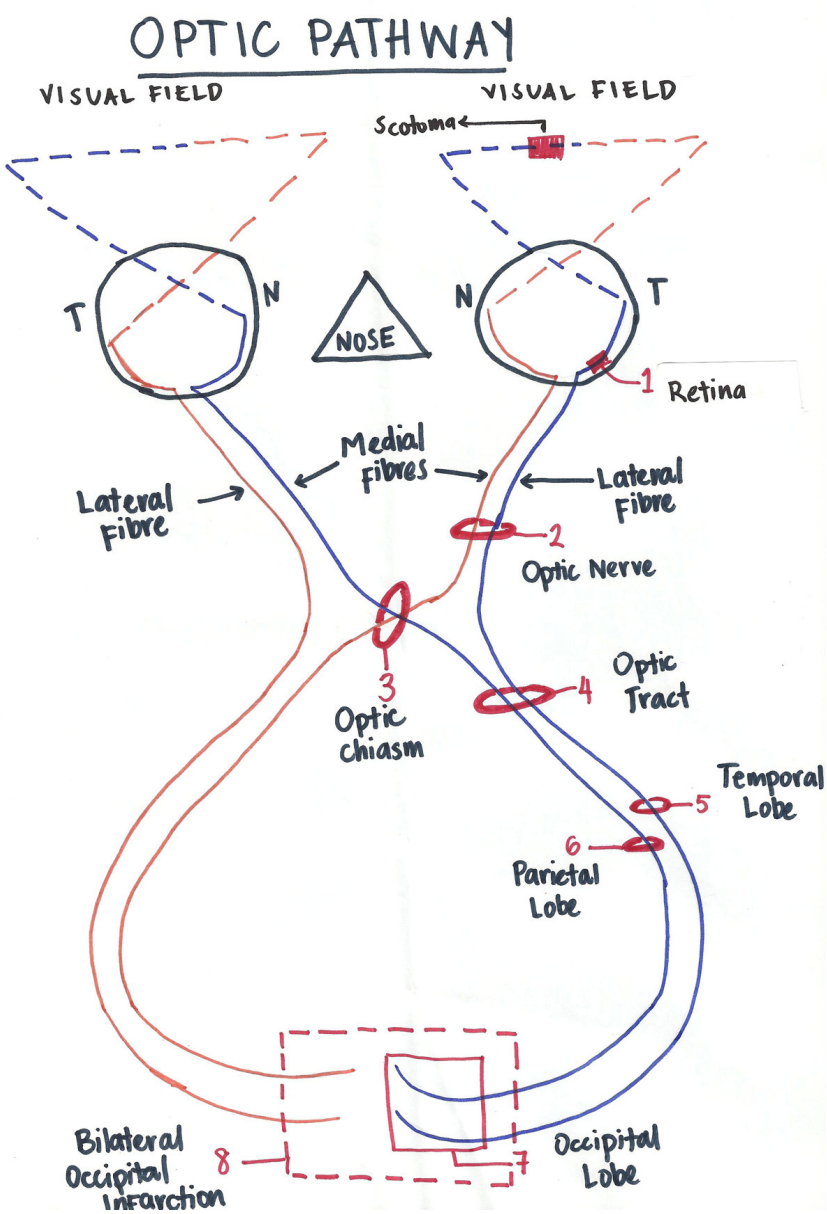
1. Lesion in the ***brainstem*** will cause sensory loss on the **same side** of the face and **contralateral** sensory loss in rest of the body below the face.

2. If the lesion in the ***thalamus***, sensory loss occurs on the **same side** of the face as on the rest of the body.

3. **Brown Sequard syndrome:** Hemi-section of the spinal cord will cause sensory loss on the **same side d**ue to damage in posterior column and loss of pain and temperature on the **contralateral** side due to damage of spinothalamic tract. Causes include trauma, tumours etc.

4. ***Cord lesion*** will cause loss of pain and temperature only and usually in a segmental pattern. Common cause of cord lesion is syringomyelia.

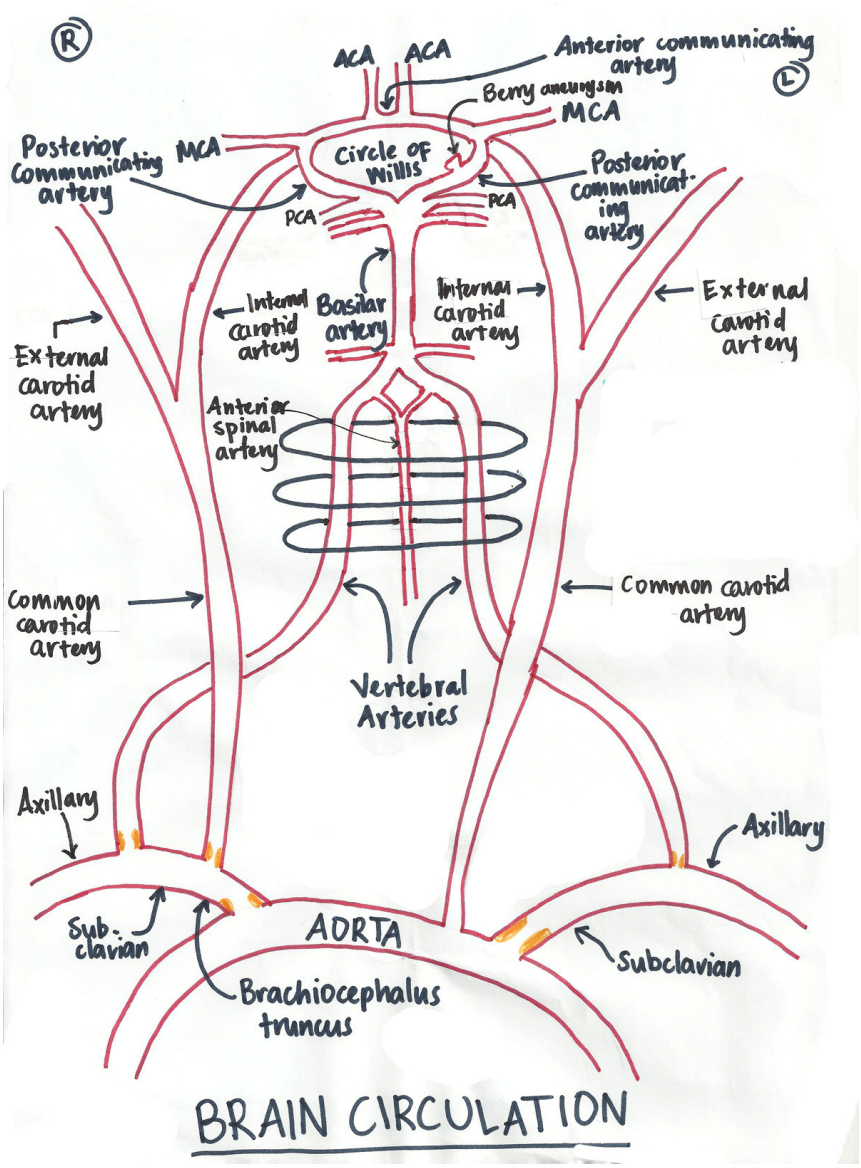
II CRANIAL NERVE: Optic nerve



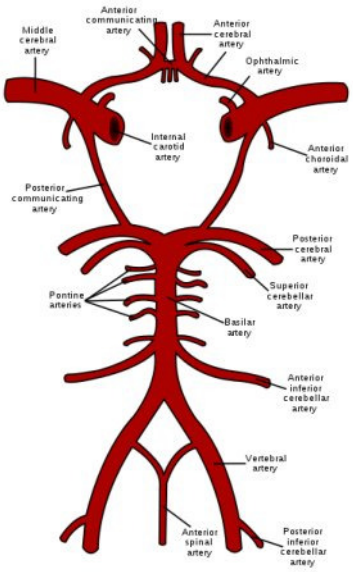
NB: The optic nerve consists of medial and lateral fibres. The media fibres cross and the lateral fibres remain on their side.

1. Lesion on the ***retina***: causes include retinal detachment or haemorrhage. A lesion will cause scotoma. A scotoma is a small area in the patient's vision where he/she cannot see.
2. Lesion on the ***optic nerve***: common cause is multiple sclerosis, which causes optic neuritis. Symptoms include sudden loss of vision in one eye and there is usually dull pain when moving the eye. It will cause monocular blindness.
3. Lesion on the ***optic chiasma***: common cause is pituitary tumour, which compresses the optic chiasma. Other causes include craniopharyngioma and meningioma. Visual field defect is bitemporal hemianopia.
4. Lesion on the ***optic tract***: common causes include stroke and tumour. Visual field defect is non-congruous homonymous hemianopia. The visual field defect is always on the opposite side to where the lesion is.
5. Lesion in the ***temporal lobe***: causes include stroke and tumour. Visual defect is homonymous upper quadrantanopia. The visual field defect is always on the opposite side to where the lesion is.
6. Lesion in the ***parietal lobe***: causes include stroke and tumour. Visual field defect is homonymous lower quadrantanopia.
7. Lesion on the ***occipital lobe***: causes include stroke and tumour. Visual field defect is congruous homonymous hemianopia. Can be with macula sparing or non-macula sparing. If it is macula sparing then the cause is posterior cerebral artery. The visual field defect is always on the opposite side to where the lesion is.
8. ***Bilateral lobe lesion*** e.g. bilateral stroke causing cortical blindness.

BLOOD SUPPLY OF THE BRAIN:



ACA = Anterior cerebral artery
MCA = Middle cerebral artery
PCA = Posterior Cerebral Artery



A. Vertebro-basilar artery supplies:

1. Posterior Cerebral Artery which supplies the occipital lobe
2. Cerebellum
3. Brainstem

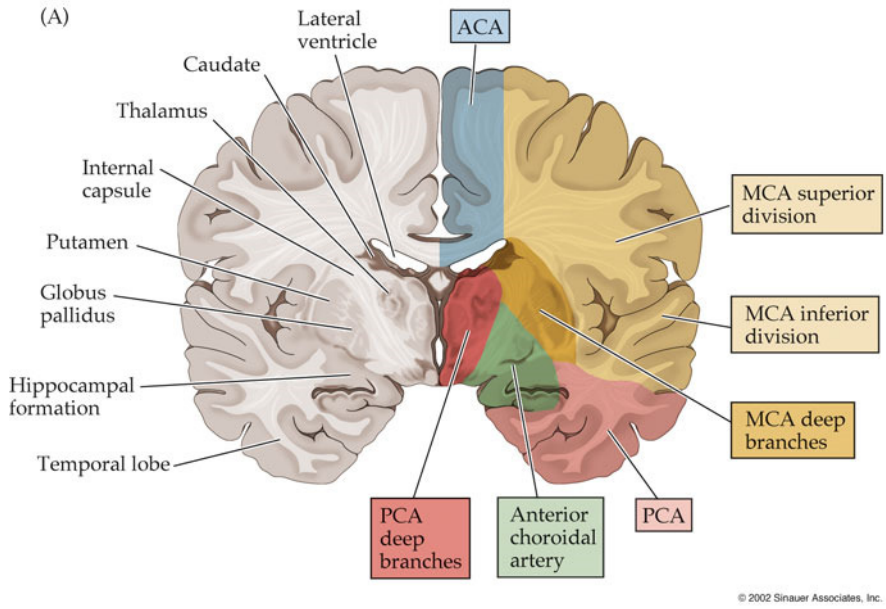
(Vertebro-basilar insufficiency/atherosclerosis)

B. Internal Carotid artery supplies:

1. Anterior Cerebral Artery which supplies the frontal lobe and inner aspect of the hemisphere
2. Middle Cerebral Artery which supplies the outer aspect of the hemisphere

(Carotid artery disease)

C. External Carotid: supplies the eye. It causes amaurosis fugax.



Areas of the brain supplied by the cerebral arteries.

Vertebro-basilar Atherosclerosis:

Causes hypoperfusion of the cerebellum leading to the following symptoms:

1. Dysarthria
2. Diplopia
3. Nystagmus

4. Hypotonia
5. Ataxia
6. Finger nose past-pointing
7. Dysdiadokinesia
8. Vertigo

Atherosclerosis of the **posterior-inferior cerebellar artery or vertebral artery** causes lateral medullary syndrome which is ischaemia to the brain stem and cerebellum leading to Horner's syndrome and cerebellar signs.

Horner's Syndrome:

1. Ptosis (incomplete)
2. Miosis
3. Enopthalmus
4. Anhydrosis (loss of sweating on the ipsilateral side of the face)
   This is due to damage to sympathetic fibbers

Subclavian Steal Syndrome

Subclavian artery stenosis proximal to the vertebral artery, causing blood to be 'stolen' down the axillary artery down the arm, leading to brain ischaemia.

Suspected if you have a different blood pressure in each arm, usually >20mmHg.

N.B: Thoracic aortic aneurysm will cause different blood pressure in each arm aswell.

DEMENTIA:

Main feature is memory loss. Impaired cognition with intact consciousness causes:

1. **Alzheimer's Disease:**

- Commonest cause of dementia in the UK
- Gradual onset
- Visuo-spatial dysfunction e.g. getting lost when taking a walk in the park or when driving.
- Progressive memory loss and cognition
- Normally in old age
- Pathologically: **Neurofibrillary tangles and senile plaques**
- Patient may be previously fit and well
- There are behaviour changes: aggressive, emotionally liable, depression
- May have seizures in the late phase.

Investigations: Rule out the following reversible causes (dementia screen)

a. B12/Folate deficiency
b. TSH (hypothyroidism)
c. HIV test
d. Thiamine deficiency
e. Syphilis serology

CT scan: to rule out vascular dementia, subdural haematoma or brain tumour.

Diagnosis is made by mini-mental state examination or mental state examination

Treatment: Donepazil

2. **Vascular Dementia/ Mult-infarct Dementia:**

- Second commonest cause of dementia in the UK
- Sudden onset
- Due to multiple TIA's or Stroke
- History of hypertension or diabetes mellitus
- History of ischemic heart disease
- History of atherosclerosis (intermittent claudication)
- There is **STEPWISE DETERIORATION** in cognition and memory

Investigation: CT scan brain (which will show brain infarction)

3. **Fronto-temporal lobe Dementia:**

- Early personality change
- Visual spatial functions are relatively preserved from the beginning
- Early intellectual impairment

4. **Lewy Body Dementia:**

- Hallmark of Lewy Body dementia is **FLUCTUATING** loss of memory
- It is associated with Parkinsonism (bradykinesia, tremors, rigidity)
- Pathological finding = Lewy Bodies

5. Huntington's Disease:

- There is a strong family history
- It is autosomal dominant (1:2 inheritance)
- Usually in a young or middle aged patient
- Chorea

6. **Pseudo-dementia:**

- Due to depression
- Symptoms of depression like low mood, poor sleep, loss of appetite, loss of interest in daily activities
- Responds to antidepressants

7. **Pick's Dementia:**

- Mainly cognition impaired
- Memory is usually normal (they score very well on the memory test)
- Unusual sexual adventures and talking about their sexual life openly
- It may lead to stealing

1. **Wilson's Disease:**

- This is accumulation of copper in different organs due to deficiency of a protein called ceruloplasmin which transports copper in the body
- Copper accumulates in different organs of the body:

a. Copper deposition in the brain leads to dementia
b. Copper deposition in the liver leads to liver disease
c. Copper deposition in the joints leads to joint pain
d. Copper deposition in the eye will cause Kaiser Fischer rings

- Investigation: Serum copper

1. **Wernicke's Korsakov Syndrome:**

- This is brain damage due to deficiency of Vitamin B1, also called thiamine
- It is common in alcoholics
- The patient always has liver disease
- Consists of Wernicke's encephalopathy: which is a triad of confusion, ataxia and opthalmoplegia. Patient will also have flapping tremor and headache.
- Korsakov's Psychosis: confabulation due to short-term memory loss. Confabulation is making up stories due to short-term memory loss.
- Treatment: Thiamine

11. **Creutzfeldt-Jacob Disease (Prion Disease/Mad Cow Disease):**

- Prion is an altered protein which can be transmitted through blood transfusion or surgical instruments from one patient to another
- It can transform other proteins to become abnormal therefore it is infective
- Common in haemophilics with history of previous blood transfusion
- Accumulation of these prions in the brain causes dementia
- No treatment available

1. **HIV dementia:**

- Must be an HIV patient

- History of blood transfusion or IVDU or homosexuals
- HIV patients are usually immune-compromised and usually also they have tuberculosis
- Dementia present with confusion but this is progressive confusion for weeks and months

1. **Age-related forgetfulness:**

- Patient has insight to problem
- It is normal forgetfulness which everyone develops with age
- The patient is usually concerned and not happy about it

N.B: Patients with true dementia have no insight in to the problem.

Cranial Nerves

i. **Olfactory**: if it is affected patient will have anosmia (loss of sense of smell)

i. **Optic nerve**: please see above

i. **Oculomotor:** Damage to this nerve will cause diplopia in different directions. There is also dilated pupil and complete ptosis. Diplopia on looking up means third nerve palsy but it can cause diplopia in other directions as well as it innervates 4 muscles of the eye:

1. Superior Rectus
2. Inferior Rectus
3. Medial rectus
4. Inferior oblique

The eye deviates downwards and outwards.

i. **Trochlear**: Diplopia on looking down i.e. walking down the stairs or reading. But dilated pupils will mean 3rd nerve palsy and 4th nerve palsy does not cause dilated pupils. The 4th nerve innervates the superior oblique (SO4), which pulls the eye down and medially.

i. **Trigeminal nerve:** Sensory loss on face of ophthalmic, maxillary or mandibular division i.e. loss of sensation on the face. It innervates the involuntary muscles and muscles of mastication (masseter muscles and temporalis). It also innervates the cornea, so can cause corneal anaesthesia or loss of the corneal reflex and jaw jerk reflex.

i. Trigeminal Neuralgia is electric, stabbing, knife like, shooting painon the face. Treatment: carbamazepine.

i. **Abducent nerve**: It innervates the lateral rectus muscles (LR6) and if it is affected there will be diplopia on looking sideways. Dilated pupils would indicate 3rd nerve palsy.

i. **Facial nerve:** Failure to close the eye, loss of nasio-labial folding, deviation/dropping of angle of mouth, loss of taste in anterior 2/3rd of the tongue.
    i. *Lower motor lesion- Both face and forehead are affected*
    ii. *Upper motor lesion- Forehead is spared (not affected)*

N.B: It innervates the voluntary muscles of the face (muscles of expression)

i. **Vestibulocochlear nerve**: Damage will cause deafness and balance problems (sensory ataxia – dizziness and vertigo). It has connection with the posterior column.

i. **Glossopharyngeal**: Innervates Tongue and Pharynx
    i. Loss of taste on Post 1/3rd of tongue
    ii. Deviation of palate to one side

i. **Vagus**: Loss of gag reflex plus it innervates a lot of visceral organs including the heart.

i. **Accessory nerve**: There will be failure to shrug shoulders on resistance because it innervates trapezius and also failure to turn head sideways on resistance because it innervates sternocleidomastoid muscle (SCM).

i. **Hypoglossal nerve**: Deviation of tongue one side. The lesion is on the same side where tongue deviates

MENINGITIS:

Inflammation of the meninges of the brain

Causes:

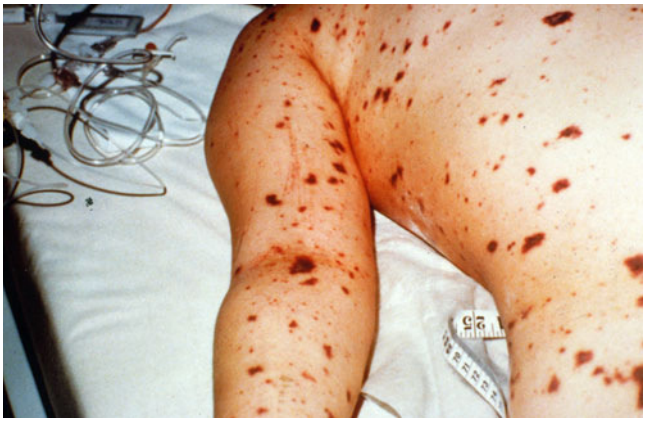
a. Bacterial

1. Streptococcal pneumonia is the commonest in adult
2. Neisseria meningitides usually if there is rash
3. Tuberculosis in patients from endemic areas like Africa and Asia
4. Listeria monocytogenes should be considered if more than 50 years of age

a. Viral
b. Protozoa
c. Leukaemic Infiltration if there is history of leukaemia
d. Malaria in patients with recent history of travel to endemic areas like Africa

Symptoms:

1. Headache
2. Fever
3. Photophobia
4. Vomiting
5. Rash - suggests meningococcemia and the cause is Neisseria meningitides



Signs:

1. Brudzinki's Signs
2. Kernig's Sign
3. Neck Stiffness
4. photobia

Management of Meningitis

1. If you suspect meningitis and you are outside the hospital (commonly in the GP) you should give 2.4 g intravenous benzylpenicillin and the patient should then be sent to hospital

1. In hospital give intravenous third generation cephalosporin antibiotics (ceftriaxone or cefotaxime)

1. Now look for rash. If patient has got rash then do blood culture as the diagnosis is meningococcal septicaemia. The causative organism is Neisseria meningitides.

1. If no rash then we need to do a lumbar puncture, but check that there are no signs of raised intracranial pressure (drowsiness, papilloedema, focal neurological signs); Headache and vomiting are not very reliable signs of raised ICP in meningitis as most of the time meningitis presents with vomiting and headache.

1. If there are signs of raised intracranial pressure then do CT scan of the head in order to rule out raised ICP. If CT proves there is raised ICP do not do lumbar puncture as there is risk of herniation of the brain.

1. If there are no signs of raised ICP then do lumbar puncture straight away.

If patient more than 50 years of age add cotrimoxazole to cover listeria, but if listeria has been identified then give ampicillin/amoxicillin and gentamicin.

Key Points of Management:

1. GPs should give benzylpenicillin or a 3rd generation cephalosporin (cefotaxime and ceftriaxone) before urgent transfer to hospital.
2. Give chloramphenicol if there is a history of anaphylaxis to penicillin or cephalosporins.
3. **Meningococci:** Benzylpenicillin or 3rd generation cephalosporin for at least 5 days.
4. **Pneumococci:** 3rd generation cephalosporin or benzylpenicillin for 10-14 days. If resistant, add vancomycin.
5. **Haemophilus Influenza:** 3rd generation cephalosporin for 10 days.
6. **Listeria:** Amoxicillin and gentamycin

Signs of raised ICP in general

1. Papilloedema
2. Drowsiness
3. Headache
4. Vomiting

Lumbar Puncture

	VIRAL	T.B.	Bacterial
Cells	Lymphocytes	Lymphocytes	Neutrophils and Polymorphs
Glucose	←→ or Slightly↓	↓	↓
Proteins	↑	↑	↑

Normal CSF:

- Glucose is 2.5 - 4.5 mmol/L (1/2 – 2/3 of blood glucose)
- Protein 0.2 - 0.5g/L (200-500mg/dl)

Prophylaxis of Meningitis

Rifampicin BD (twice daily) for 2 days for all contacts including doctor and nurse e.g. nursery, classrooms.

NB: prophylactic antibiotics are given only to people who have come in contact with a patient or those who are more likely to have been in contact.

Complications of meningitis:

1. Deafness (especially in children – 10% of child cases) - arrange hearing test after treatment of meningitis in children.
2. Failure to thrive in children.
3. SIADH - causes low sodium. Treat with fluid restriction.
4. Raised ICP - treat with mannitol and monitoring.
5. Seizures - treat with lorazepam and then phenytoin or treat as status epilepticus.
6. The majority of patients do not develop complications.

CNS infection in HIV:

Toxoplasmosis Gondii

Presents with mass like effect i.e. progressive headache and focal neurological signs

Investigation is CT scan which shows ring enhancing lesion

If signs of raised intracranial pressure give dexamethasone

Treatment is pyrimethamine and sulphadiazine

Crytococcus Meningitis

Presents with altered consciousness or confusion with meningism and headache

Investigation is Lumbar Puncture for CSF

Treatment is amphotericin in acute state and fluconazole for maintenance

Parkinson's Disease

The hallmark of Parkinson's disease is presence of lewy bodies and neural cell death in the pars campus in the substantial nigra.

Parkinson disease does not develop until the level of dopamine falls below 20% of normal amount.

Symptoms:

1. Mask like face
2. Shuffling gait
3. Bradykinesia: slowness of initiation of movement
4. Rigidity
5. Tremor (unilateral)

Symptoms are usually unilateral and there is good response to levodopa.

Symptoms are progressive

Investigation: no diagnostic test. Diagnosis is made by clinical symptoms.

Treatment:

1. Levodopa
2. Dopamine agonist cabergoline, ropirinole,
3. Apomorphine
4. Amantidine
5. MAOI like selegiline
6. Entacapone a COMT drug
7. anticholinergic agent like benzhexol

Differential Diagnosis:

1. Lewy Body Dementia – does not respond very well to levodopa. Parkinson's disease does respond well to levodopa.
2. Drug induced Parkinsonism – Typical antipsychotics (haloperidol)

EPILEPSY (Abnormal electrical discharge in the brain)

CLASSIFICATION:

1. Partial
2. Primary Generalised

PARTIAL:

this is when **e**lectrical discharge from 1 hemisphere

a. Focal (remains in 1 hemisphere)
b. Secondary Generalised (spreads to both hemispheres) e.g. Jacksonian attack (jerking starts from the thumb -> hands -> body.

PRIMARY GENERALISED:

Electrical discharge from both hemispheres

a. Infantile spasms (salaam attack) common in infants. This child jerks back and forth like the way muslims pray. It can also cause severe developmental delay
b. Absent seizures -> in children. A period the child stops for about 10 seconds and then resumes what he is doing
c. Tonic clonic
d. Atonic (floppy)
e. Myoclonic (twitching of the muscles of the face and the whole body)

Epilepsy:

1. Simple (no loss of consciousness)
2. Complex (loss of consciousness)

Symptoms:

1. Jerking (fit)
2. Urinary or faecal incontinence
3. Tongue biting
4. Loss of consciousness
5. Post-ictal status

Investigations:

1. EEG: Investigation of choice is EEG
2. CT Scan (to rule out brain tumour)

a. If seizure of new onset at night (during sleep)
b. If seizure is associated with prolong headaches
c. New onset focal seizures in adults (epilepsy is usually develops in a child)

Treatment:

1. Primary Generalised epilepsy - Sodium valproate
2. Absence seizure - Ethosuximide
3. Partial seizure - Carbamazapine
4. Infantile Spasm - Vigabatrin

If seizures without any cause -> send to 1st fit clinic and advise the patient not to drive and inform DVLA.

General advice to give patient:

1. Do not drive and advice patient to inform DVLA
2. Avoid unsafe activities e.g. swimming alone, mountain climbing, riding a bicycle
3. Take showers rather than bath
4. Avoid precipitants like night club, watching TV with strobe lights on

Status Epilepticus:

This is seizure lasting more than 30 minutes or repeated attacks of seizures without gaining consciousness in between.

Treatment of Status Epilepticus:

IV lorazepam
↓
IV lorazepam
(repeat)
↓
Phenytoin IV
↓

Phenobarbiturate IV
↓
Anaesthetise and Intubate

If after PR Diazepam if patient is still fitting and you have gained an intravenous line give IV Lorazepam

Drug Side Effects

1. Carbamezapine: Rash (in short term), Renal failure, diplopia, hyponatraemia, SIADH, neutropenia

1. Sodium Valproate: weight gain, liver failure, tremor, sedation, rash, low platelets, hair loss

1. Phenytoin: gum hypertrophy, cerebellar signs and depression.
↓
i. Nystagmus
ii. Diplopia
iii. Ataxia
iv. Dysarthria
v. Dizziness

1. Ethosuximide: bone marrow suppression, headache, lethargy, ataxia, agranulocytosis, GIT irritation.

1. Benzodiazepines: IM injection may cause cold abscess

INTRACRANIAL BLEED:

1. Subarachnoid Haemorrhage:

- Common in young patients 30-50 years
- Cause is Berry aneurysm - located at posterior or anterior communication artery.
- May present with collapse while exercising
- It is associated with Polycystic Kidney Disease, coarctation of aorta, Ehler-Danlos syndrome.

Signs and symptoms

1. Sudden onset headache at the back of the head (occipital)
2. Projectile vomiting
3. Neck stiffness
4. Photophobia
5. There is usually family history

N.B: Sudden onset of headache is always subarachnoid haemorrhage until proven otherwise. Severe headache but no history of head injury.

Investigation:

1. CT scan head
2. If CT scan head does not show bleeding, do lumbar puncture at least 12 hours after onset of headache. Usually you look for bilirubin in the CSF (xanthochromia).

Treatment:

1. Nimodipine for the pain
2. Refer to neurosurgeon
3. Treatment is usually surgery (clipping of the aneurysm)

2. Subdural Haematoma:

- Common in elderly with recurrent falls
- Also in alcoholics and boxers due to recurrent head injury
- changing level of consciousness (progressive drowsiness)
- Trauma may happen long time and patient may forget about it e.g. 2 weeks ago
- Signs of raised ICP which are headache, papilloedema and vomiting
- Focal neurological signs i.e. weakness of the legs or sensory loss
- Can present with cognitive impairment

Investigation: CT scan

Treatment:

1. Refer to neurosurgeon for evacuation of haematoma
2. Surgery - Burr hole (done as an emergency to relieve increased intracranial pressure)

3. Extradural Haematoma:

- Rapid deterioration of consciousness
- There is lucid interval i.e. in minutes to hours
- Almost/always there is history of head injury
- Signs of raised ICP (headache, vomiting and papillaoedema)
- Fits
- Focal neurological signs
- Up going plantar reflexes (Babinski sign)
- Monoparesis

Investigation: CT scan

Treatment: 1. Surgery for evacuation of haematoma
2.Burr hole may be used in emergency to relieve the pressure

4. Intracerebral bleeding (haemorrhagic stroke)

- Elderly patient with history of hypertension, usually uncontrolled or untreated hypertension.
- Sudden loss of consciousness, preceeded by headache
- History of ischaemic heart disease or diabetes mellitus

- Normally focal neurological signs
- Up going plantar reflex
- Dilated pupils
- UMN signs in limbs
- Intracerebral bleed is same as haemorrhagic stroke

Investigation: CT scan head.

Treatment: Refer to neurosurgeon to evacuate haematoma

Differential Diagnosis for Focal Neurological Signs:

Focal neurological signs are impairments of nerve or brain function that affects a specific region of the body. For example: weakness of the arm, or the leg etc.

Differential diagnosis:

1. Intracranial bleeding
2. Meningitis
3. Multiple Sclerosis
4. Brain or spinal tumour
5. Syringomyelia
6. Trauma
7. Migraine
8. Stroke
9. Motor Neurone Disease
10. Myasthenia Gravis

FOCAL NEUROLOGICAL SIGNS:

1. Multiple sclerosis: Autoimmune condition. It is due to demyelination of the nerve fibres in the central nervous system (brain and spinal cord). It only affects CNS not peripheral nervous system. Patients are usually young females, typical age 18-30 years old.

There is mono-symptomatic presentation depending on the location of the lesion and lesions are scattered in neuro-anatomy.

a. **Optic Nerve -** Optic neuritis, sudden loss of vision with dull pain in the eye, then symptoms will resolve and then come back
b. **Spinal Cord** - Weakness in the limbs, urinary retention/incontinence
c. **Optic Disc** - Papilloedema

d. **Brainstem** - Weakness in the leg or sensory loss with cranial nerves palsy.
e. **Cerebellum** - Dysarthria, nystagmus, ataxia, etc.

Symptoms appear on by one and each time the lesion is in a different location.

Classification:

1. Remitting/Relapsing MS: symptoms comes and go (symptoms stay for weeks-months, then disappear completely). This is commonest form
2. Primary Progressive MS: from the beginning the symptoms keep progressing without remission.
3. Secondary Progressive MS: Initially it was remitting/relapsing MS then symptoms started progressing.

Investigation:

a. MRI scan of the brain or spinal cord depending on the symptoms. This is the investigation of choice

a. Lumbar Puncture - look for monoclonal bands in CSF

Diagnosis is made by MRI and clinical signs

Treatment:

During attack	During remission
↓	↓
IV Methylprednisolone	Interferon α +b (disease modifying drugs)

CEREBROVASCULAR ACCIDENT:

Consists of stroke and transient ischaemic attack (TIA). The cause for both are the same.

Brain ischemia: results in brain infarct

Causes:

1. Thrombosis
2. Emboli
3. Thromboembolism
4. Haemorrhage

Patients are elderly with sudden onset of symptoms.

Cardiovascular Accident

1. **Stroke**: symptoms >24 hours
2. **TIA:** symptoms <24 hours

STROKE:

1. Ischaemic
2. Haemorrhagic -> intracerebral haemorrhage (associated with increased blood pressure, uncontrolled or untreated hypertension)

Symptoms:

1. Carotid atherosclerosis or carotid artery disease:

a. Frontal lobe - hemiparesis, monoparesis, dysphasia if dominant
b. Temporal lobe - memory loss
c. Parietal lobe - sensory loss
d. Eye - amarousis fugax
e. If dominant hemisphere aphasia/dysphasia

2. Vertebrobasilar artherosclerosis or vertebrobasilar insufficiency:

a. Dysarthria
b. Vertigo
c. Nystagmus
d. Cerebellar ataxia
e. Hypotonia

3. Brainstem: Cranial nerves are involved and causes difficulty swallowing
(UMN sign lesions in limbs)

A. Investigations to find the cause:

- If Atrial Fibrillation then Echocardiogram to see intra-cardiac emboli
- If recurrent Myocardial Infarction then Echocardiogram to see mural thrombi
- If Carotid bruit then Doppler ultrasound of the neck
- If history of ischaemic heart disease, it means atherosclerosis is present then Doppler ultrasound (carotid artery and vertebrobasilar artery) needs to be done
- If diabetic patient then Blood glucose
- Blood pressure measurement to rule out Hypertension
- Heart murmur it means there is vulvular heart disease which can cause emboli Echocardiogram to see an emboli
- Thoracic Aortic Aneurysm then CT scan chest

B. Investigation to make diagnosis:

CT Scan of head

1. Ischaemic
2. Haemorrhagic

General Management of cerebral vascular accident

- If patient has symptoms of stroke do CT scan head to exclude haemorrhagic stroke.
- If no haemorrhagic stroke then give Aspirin 300mg stat
- If patient presents within 3 hours and no haemorrhagic stroke, arrange thrombolysis.
- If patient had symptoms of stroke, which have now resolved it means he had TIA - give aspirin 300mg orally, even before doing a CT scan because symptoms have resolved which suggests it is TIA.

Prophylaxis: Aspirin + dipyridamole

If aspirin allergy substitute with clopidogrel

SPACE OCCUPYING LESION:

Causes:

1. Tumour
2. Abscess
3. Haematoma
4. Brain metastasis
5. Aneurysm
6. Granuloma

Symptoms:

- Usually in elderly patients
- Weight loss, anorexia
- Anaemia, tiredness
- Increased ICP: headache, vomiting and papilloedema
- Focal neurological signs (weakness in the limbs)
- Seizures

Investigations: CT scan head

BRAIN TUMOUR:

- Usually in elderly patients
- Symptoms are usually gradual onset
- Progressive worsening headache usually bilateral
- Adult onset seizure is always brain tumour until proven otherwise
- Headaches worse in the morning and on bending forward due to raised intracranial pressure

Treatment: 1.Dexamethasone if headaches or raised ICP to reduce oedema around tumour
2. surgery if localised tumour
3. Radiotherapy if metastasis but appropriate in that patient

MIGRAINE

- Young, female patient typically
- Recurrent headaches: throbbing, pulsatile, unilateral
- Associated with nausea and vomiting
- Aura – can be sensory or visual

a. Sensory aura usually tingling or numbness in upper limb
b. Visual aura usually patient sees zig-zag lines but it can be any other form of visual loss)

- Aura usually lasts seconds to minutes and within 1 hour of aura headache follows
- there is usually family history

Investigations: routine investigations, if diagnosis is not clear, to rule out other causes

Treatment: ACUTE = high dose aspirin 900mg or ketoprofin

Prophylaxis:

1. Beta-Blocker (Propranolol)
2. Topiramate
3. Amitryptalline

MYASTHENIA GRAVIS

It is due to reduction in the number of nicotine AChR at neuromuscular junction.

This is due to acetylcholine receptor antibodies formed against the acetylcholine receptor.

There is abnormality of the thymus gland in 75% of the cases either in a form of hyperplasia or thymoma.

It is an autoimmune condition.

It is associated with other autoimmune conditions like SLE, Pernicious Anaemia, Grave's disease, Rheumatoid Arthritis

Symptoms:

1. Painless muscle weakness which increases with exercise
2. Generalised weakness
3. Dysphagia, dysphonia, dysarthria, limb weakness
4. There is fatiguability and weakness which worsens by the end of the day or whenever patient works hard.
5. Eye muscle weakness may present with diplopia

Investigation:

1. Serum AChR antibodies are diagnostic of myasthenia gravis
2. Tensilon (edrophonium) test diagnostic
3. CT mediastinum to look for thymus gland

Treatment:

1. Cholinesterase inhibitor e.g. Pyridostigmine
2. Steroid can be used if symptoms not adequately controlled by cholinesterase inhibitors
3. Azathioprine if steroid is contraindicated
4. Other immune suppressant e.g. ciclosporin, methotrexate may also be used
5. Plasma exchange and immunoglobulin are used in patients with myasthenia crisis.
6. Thymomectomy may be used in patients with AChR antibodies and under 45 years of age.

BENIGN POSITIONAL VERTIGO

This is characterized by recurrent episodes of dizziness provoked by quick change in position.

Precipitating factors: Trauma and viral illness.

This is a mechanical disorder due to movement of debris within the endolymph to the most dependent part of the canal during head movement.

Typical symptoms are vertigo on turning over in bed, lying down or sitting up from supine position.

Investigation: Dix - Hallpike manoeuvre

Treatment: Epiley manoeuvre

Peripheral versus Central Nystagmus

Peripheral Nystagmus:

- Has fixed direction
- Last less than 60 second
- There is fatiguability i.e. lessing of symptoms with repetition
- Severe vertigo with nystagmus
- Inconsistent when trying to reproduce it

Central Nystagmus:

- Consistent when trying to reproduce it
- Mild symptoms
- Marked nystagmus
- No fatiguability
- Symptoms are constant

HEADACHES

1. **CLUSTER HEADACHE:**

- Common in young middle aged men
- Usually unilateral severe headache, which radiates to the forehead
- Associated with redness of the eye and lacrimation.
- Severe headache which makes patient cry
- Headache occurs in clusters (comes and goes in periods e.g. 2 months of headaches at the same time and then 8 months of free headache and followed up by another period of headaches) this is why it is called cluster headache
- Treatment is with high 100% oxygen and sumatriptan.

1. **GIANT CELL ARTERITIS:**

- Typical age above 50 or elderly patients
- Common in women
- Unilateral headache on the temple areas
- Headache worse with combing hair, chewing (jaw claudication)
- Usually associated with weight loss, anorexia and weakness of the upper limbs
- In 25% it is associated with polymyalgia rheumatic which is an autoimmune condition mainly affecting the muscles especially those of the upper limb which make patient difficulty to stand up from the chair.

Investigation:

1. Initial is ESR
2. Definitive is temporal artery biopsy

Management:

If symptoms of GCA and ESR is raised then next step is treatment with IV methylprednisolone for 3 days followed by high dose oral prednisolone for 2 - 3 years i.e. long term.

Definitive diagnosis of GCA is made by temporal artery biopsy within 3 days of provisional diagnosis.

1. **TRIGEMINAL NEURALGIA:**

- Usually electric shock like or knife like or stabbing pain in the face
- Usually in the distribution of the trigeminal nerve branch e.g. mandibular, maxillary or ophthalmic
- Pain can be triggered by shaving or chewing
- Facial pain runs up and down the face

Treatment: Anti-epileptic medication e.g. Carbamazepine, gabapentin

1. **MIGRAINE:**

- Common in young women.
- Unilateral pulsatile or throbbing headache
- Usually preceeded by visual or sensory aura
- Visual auras commonly are simply visual fortification or zig zag
- Sensory aura usually tingling and numbness in the hands
- Auras are followed by headache within 1 hour
- Headache usually associated with nausea and vomiting

1. **ACUTE CLOSED ANGLE GLAUCOMA:**

- Typically pain is in the eye with redness and lacrimation
- Usually associated with nausea, vomiting and loss of vision
- Unilateral headache
- Severe ocular pain
- Previous intermittent headache
- Usually there is family history
- Common in females
- Haloes around the light
- On examination there is corneal oedema (fine ground glass) and fixed dilated pupil oval shaped
- Raised intraocular pressure usually more than 40mmHg (normal IOP <22mmHg)

1. **SINUSITIS:**

- Usually pain between the eyes or forehead on the sinuses
- Usually there is coryza symptoms: sneezing, running nose and cough
- Facial pain which is worse on bending forward

Investigation: CT Scan

Treatment: Antibiotics +/- washout of the sinuses

1. **BENIGN RAISED INTRACRANIAL PRESSURE (pseudotumour cerebri)**

- This is common in young obese women
- Morning headaches
- Papilloedema
- 6^{th} nerve palsy
- Plus or minus diplopia
- Signs of raised intracranial pressure i.e. headache, vomiting, papilloedema

Investigation: CT head to rule out brain tumour

Treatment: weight loss, acetazolamide

1. **BRAIN TUMOUR:**

- Usually progressive headache
- Usually elderly patient
- Signs of raised intracranial pressure (papilloedema, vomiting, headache)
- Plus or minus focal neurological symptoms (e.g. weakness or sensory loss in the limbs)

Investigation: CT brain

Treatment:

1. Dexamethasone to reduce raised ICP or as treatment of headaches
2. Surgery if no metastasis
3. Radiotherapy if metastasis

1. **TENSION HEADACHE:**

- Bilateral band-like headache, usually comes when patient is stressed

Treatment: Paracetamol/ aspirin/ ibuprofen or stronger painkillers

1. **SUBARACHNOID HAEMORRHAGE:** please see above

1. **INTRACRANIAL HAEMORRHAGE:** please see above

1. **MENINGITIS:** please see above

DIZZINESS OR LOSS OF CONSCIOUSNESS

1. **CARBON MONOXIDE POISONING:**

- This is usually due to leaking gas in the house therefore other family members will be affected, there is history of problems with boilers or gas in the house.
- Carbon monoxide poisoning can also occur whilst painting, as it is contained in paints.
- Carbon monoxide poisoning can also occur in house fire incidents. Usually there is singed nasal hair, or coughing black sputum

Investigation: serum carbon monoxide levels

Treatment:

1. 100% oxygen (1st choice)
2. Hyperbaric 100 % oxygen
3. if patient is unconscious then intubate and ventilate with 100% oxygen

1. **HYPOGLYCAEMIA: this is serum glucose less than 3mmol/l**

- Loss of consciousness with sweating is always hypoglycaemia until proven otherwise
- There is history of diabetes or alcohol abuse
- Repeated loss of consciousness after missing meals suggests insulinoma

Investigation: serum blood glucose levels or capillary blood glucose

Treatment**:** IV 50% glucose 50ml or 10% glucose

N.B: other causes could be insulinoma in which there is a tumour of the pancreas, which produces insulin (usually presents with loss of consciousness every time a patient misses her meals). Investigation for this is serum insulin levels followed up by CT scan pancreas to localise the tumour

Treatment: Surgery

1. **VASOVAGAL SYNCOPE: it is due to overactivity of the vagus nerve which causes severe bradycardia leading to hypo-perfusion of the brain and collapse. There is a short-lived loss of consciousness**

- This is usually in young girls
- Loss of consciousness usually less than 2 minutes
- Precipitated by long standing and unpleasant situation like pain, seeing blood or observing an operation.
- Patient usually go pale before falling down
- No investigation required simply reassure the patient

1. **STOKE ADAMS:**

- There is transient arrhythmia. It is transient complete heart block which leads to hypoperfusion of the brain cause confusion or TIA.
- Any elderly patient with repeated collapse or syncope without warning is always Stoke Adams until proven otherwise
- Patient usually go pale before falling

Investigation: 24 hour ECG (ambulatory ECG)

Treatment: may need permanent pace maker

1. **EPILEPSY:**

- This is usually associated with jerking of the limbs with urinary or faecal incontinence or tongue biting
- Usually preceded by aura
- There is post ictal status

1. **MENINGITIS:**

- Any patient with loss of consciousness and rash is always meningitis until proven otherwise
- Please see under section of meningitis for more details.

1. **POSTURAL HYPOTENSION;**

Any patient with hypertension and on treatment with some anti-hypertensive medication especially bendroflumethiazide and has repeated falls the cause is always postural hypotension until proven otherwise.

Investigation: Standing and lying blood pressure the difference between the two should be more than 20 mmHG

Treatment: Review antihypertensive medication

1. **SITUATIONAL SYNCOPE:**

This is when people lose consciousness in situations like when opening bowels, laughing, or sneezing.

1. **MENIERES DISEASE: it does not cause loss of consciousness but causes dizziness**

Patient usually has DVT (Deafness, Vertigo and Dizziness)
Patient could be on anti-emetics like prochlorperazine

1. **ANAEMIA:**

- Anaemia causes light headedness. When you see the symptom of light headedness think of anaemia first as very few things actually cause light headedness.
- Patient usually on aspirin or NSAIDS which lead to GI bleed and anaemia

Investigation: FBC for haemoglobin and then gastrointestinal endoscope

1. **HYPERTROPHIC OBSTUCTIVE CARDIOMTOPATHY (HOCM)**

- This is a congenital abnormality in which there is hypertrophy of the muscles of the heart.
- Common in young males
- Autosomal dominant
- There is usually family history of sudden death

Investigation: Echocardiogram
HOCM is part of long Q-T syndrome

Treatment: B-blockers for symptomatic relief

1. **AORTIC STENOSIS:**

- This can be either congenital or acquired
- If congenital it will be in a young patient or child

- Acquired will be in elderly
- Common presentation is dizziness or syncope on exercise e.g. while in the gym
- Therefore dizziness or syncope on exercise is always aortic stenosis until proven otherwise

Ix: Echo

TREATMENT: Surgery: Aortic valve replacement

1. **SUBARACHNOID HAEMORRHAGE:**

Sudden onset of headache is always subarachnoid haemorrhage until proven otherwise.

1. **SUBDURAL HAEMATOMA:**

This is usually common in alcoholics and elderly with recurrent falls.

1. **ALCOHOL INTOXICATION:**

Where there is alcohol there are 3 possible causes of loss of consciousness:

a. Subdural haematoma
b. Hypoglycaemia
c. Intoxication

Always start the assessment by check blood glucose.

In intoxication they usually wet themselves and smell of alcohol and they have slurred speech.

1. **DRUG TOXICITY:**

These are usually young patients who present with delusion or haemodynamic instability

1. **ANXIETY:**

- Common in young women.
- Difficulty in breathing and generalised chest pain
- Also tingling and numbness in the hands and lips
- feeling of impending doom
- feeling they have catastrophic disease eg stroke or heart attack

Treatment: Re-breathing bag during an acute attack

1. **HYPOXIA:**

Usually this is associated with shortness of breath and there is cyanosis.
there is different causes e.g COPD, Asthma or pneumonia

1. **OPIATE OVERDOSE:**

- This will cause pin point pupils and respiratory depression. So the respiratory rate will be less than 12.
- Puncture marks on the arms which suggests IV drug abuse

Treatment: Naloxone

1. **HYOTHERMIA:**

- Patient are usually shaking because they are feeling cold
- Low temperature usually less than 35 degrees
- ECG shows J waves

TREATMENT: 1. give warm IV fluid
2. give blankets

1. **STROKE:**

- Usually sudden onset of symptoms with dysphasia or weakness on the limbs
- Facial asymmetry

1. **INTRACEREBRAL BLEED:**

- This is common in patients with untreated or uncontrolled hypertension.
- Usually there is sudden headache as well or it can simply present with loss of consciousness.

1. **DIABETES:**

- This will cause loss of consciousness due to either hypoglycaemia or hyperglycaemia.
- Hypoglycaemia is usually sudden onset and common in diabetic patient or medication like sulfanyluria (Gliclazide and glibenclamide) or insulin.
- Hyperglycaemia can either be diabetic ketoacidosis or hyperglycaemic hyperosmolar non-ketotic (HONK) coma.
- Diabetic ketoacidosis and HONK is gradual onset and there is increasing drowsiness and dehydration.
- Also there is kaussmaul breathing in which there is deep, fast and sighing respiratory.

Investigation:

a. Serum or capillary blood glucose is initial
b. Arterial blood gas is diagnostic.

1. **PULMONARY EMBOLISM:**

- Young woman with sudden onset of chest pain or shortness of breath with risk factors of pulmonary embolism/deep vein thrombosis
- Please see respiratory medicine notes

1. **DISSECTING AORTIC ANEURYSM:**

- Chest pain or abdominal pain radiating to the back.
- For thoracic aneurysm there different blood pressure and pulses in each arm.
- For abdominal aneurysm there radial femoral delay or absent femoral pulses and pulsatile mass in the abdomen.

N.B: Abdominal or chest pain radiating to the back is always aortic aneurysm until proven otherwise.

1. **HEPATIC OR WERNICKE'S ENCEPHALPATHY:**

- There is usually history of alcohol abuse and stigmata of liver disease.
- Hepatic or Wernicke encephalopathy will present with progressive drowsiness.
- Sudden loss of consciousness in a patient with liver disease suggests hypoglycaemia, but gradual onset of loss of consciousness may suggest hepatic encephalopathy.

1. **ATRIAL FIBRILLATION:**

Irregularly irregular pulse is always Atrial fibrillation until proven otherwise.

1. **BRADYCARDIA:**

- There is slow pounding heart beat and pulse is less than 60.
- If heart rate less than 40 then its complete heart block.

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Resource view

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Resource description	Obstetrics and Gynaecology
Resource content	

OBSTETRICS & GYNAECOLOGY PLAB 1 NOTES 2014

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Contents

16. Dyspareunia
17. Infertility

5. Alarm Bells in Obs and Gyne

BEFORE PREGNANCY

PHYSIOLOGY OF PREGNANCY

- Plasma volume is increased by 10 – 15 %
- Total WCC increases
- Platelets decrease in pregnancy
- Clotting factors increase, making pregnancy a hyper-coagulation state.
- Cardiac output increases from 5L to 6.5 L /Min

PREPARING FOR PREGNANCY

1. Folic acid
Give 0.4 mg (low dose) oral - from before conception till 13 week of pregnancy to prevent neural tube defects and cleft lip.
This is offered to all women in the UK as soon as they start preparing to get pregnant.

2. Smoking
Advise to stop smoking because it is associated with preterm labour & perinatal death.

3. Alcohol
Advise to stop it because it is associated with alcohol fetal syndrome (features of which are microcephaly, hypoplastic upper lip, small eyes, low IQ, short palpebral fissure).

4. Spontaneous Miscarriage
Risk of miscarriage is about 9%, for women between 20 – 40 years, 75% for women >45 years of age, after 3 miscarriages the risk of failure of the next pregnancy is 45%.
(If patient has one miscarriage simply reassure).

5. Recurrent spontaneous miscarriage
This is 3 or more consecutive miscarriage.

Anti phospholipid syndrome: suspect if any of the blood tests are positive.

- *Anti-phospholipid Antibodies,*
- *Anti-cardiolipin Antibodies,*
- *Lupus-anticoagulant antibodies*

Most women with these antibodies have miscarriage in the 1st trimester (usually between 10 to 12 weeks).

Treatment if any antibodies are positive:

Aspirin 75 mg daily from the day of positive pregnancy test AND LMWH (enoxaparin/deltaparin) 40mg daily as soon as the fetal heart sound is heard (usually from the 5th week)

6. If Diabetic:
Aim to control blood glucose. If on any other medication for diabetes it should be replaced by insulin and maintained throughout pregnancy (e.g. gliclazide, glibenclamide and metformin must be changed to insulin).

DIAGNOSIS OF PREGNANCY

1. **Common Symptoms:**

   Abdominal discomfort, nausea, vomiting and fatigue.

1. **Pregnancy test:** Urine pregnancy test

1. **Dating of Pregnancy**

   1. From the LMP (last menstrual period)
   2. Dating USS usually done at 12 weeks.

PRE-NATAL DIAGNOSIS

Routine Pre-Natal Screening:

Maternal serum screening is done in 15-18 weeks. It is usually offered to all women.

i) Serum blood test
1. AFP (alpha fetal protein)
2. Beta HCG (human chorionic gonadotrophin)
3. Estriol

Types of tests

1. *Double test* = AFP + HCG
2. *Triple test* = AFP + HCG + Estriol
3. *Quadruple* = AFP + HCG + Estriol + Inhibin

Open neural tube defects (spina bifida)

- Alpha-fetal protein (AFP) - highly increased
- Human Chorionic Gonadotropin (HCG) - Normal
- Estriol - Normal

Trisomy 21 (Down Syndrome)

- AFP - Decreased
- HCG - Increased
- Estriol - Decreased

Trisomy 18 (Edward syndrome)

- AFP - Decreased
- HCG - Decreased
- Estriol - Decreased

ii) Fetal Nuchal Scan

Done at 8 – 12 weeks in which we look for nuchal fold thickness i.e. (accumulation of fluid in the neck of the baby)

Abnormal Values for nuchal fold thickness

- **8-12 weeks: >2.5mm**
- **12-16 weeks:** ≥4mm
- **>16 weeks:** ≥6mm

iii) USG

Done at 18 – 22 weeks (It is an anomaly scan in which we usually look for structure abnormality)

Definitive Tests

1. Pre-Implantation diagnosis
2. Chorionic villus sampling
3. Amniocentesis

Indications for Definitive Tests:

- Age > 35years
- Prior child with neural tube defect / chromosome defect
- Chromosomal abnormality in either of the parents
- Family history of abnormality
- Abnormal maternal serum tests
- Teratogen exposure
- Abnormal nuchal fold thickness
- Abnormal fetal structure survey (anomaly scan usually done at 12 weeks)

i. Pre-implantation genetic diagnosis

  - Pre-implantation genetic diagnosis (PGD) is available to couples that are at risk of having a child with a specific genetic or chromosome disorder, such as cystic fibrosis, sickle cell disease or Huntington's disease etc.
- Fertilisation is done in the laboratory. The embryos are tested for genetic abnormalities, 1 or 2 unaffected embryos are implanted into the uterus.

ii. Chorionic villous sampling (CVS)
This is done at 10- 13 weeks)
It is used if the screening test suggests aneuploidy (trisomies, cystic fibrosis, thalassemia, sickle cell disease). Other indications as above.

Advantage: It is done early in pregnancy therefore it enables early termination of pregnancy if the woman decides not to continue with the pregnancy.

Disadvantage: It has 1% risk of miscarriage and risk of transmitting infections like HIV and hepatitis to the child.

iii. Amniocentesis
This is done at 15-18 weeks
Indications

- Screening test suggests aneuploidy
- Or it can be done for enzyme assays looking for inborn error metabolism (G6PD)
- DNA analysis for Cystic Fibrosis and Thalassemia
- Diagnosis of fetal infection (Cytomegalovirus, Toxoplasmosis)

Advantage Carries low risk of miscarriage 0.1%

Disadvantage Late identification of the affected fetus leads to late termination.

ANTE-NATAL CARE
Consist of 1st, 2nd and 3rd trimester visits.
Antenatal care starts once the pregnancy is confirmed.

Routine tests (these tests are offered to all women in the UK)

- FBC
- Blood Group
- Antibody Screen (ABO & Rh) to assess the risk of rhesus incompatibility
- Routine infection screen (rubella, syphilis, hepatitis, HIV)

Specific blood tests:

- Afro-Caribbean origin = sickle cell test
- Screening for Thalassaemia (Cyprus, Eastern Mediterranean, Middle-East, South-East Asia, Indian subcontinent).

PREGNANCY

FIRST TRIMESTER COMPLICATIONS

a) Hyper-emesis Gravidarum: Excessive vomiting or severe morning sickness 1ST Trimester

- Vomiting, can cause weight loss
- Muscle wasting
- Dehydration
- Inability to swallow saliva
- Thirst
- Tired due to dehydration

Investigations
U&E (Na, Urea and Creatinine will be increased
LFT, FBC, USGto exclude multiple pregnancy & molar pregnancy .

Treatment
1) Admit + IV fluids (if cannot tolerate oral fluids) + check blood + keep NBM for 24 hrs then introduce some light diet.
2) Antiemetic eg. IM Cyclizine, metoclopramide, prochlorperazine, promethazine, chlorpromazine, domperidone and ondansetron
3) Thiamine should be prescribed routinely either orally or IV
4) If vomiting doesn't stop with antiemetics then give steroids.

b) Recurrent Miscarriages: This is 3 or more consecutive miscarriage

Causes

1. **Anti-Phospholipid syndrome**

Treatment for anti phosphoslipid syndrome:

- Aspirin 75 mg from day of +ve pregnancy test
- LMWH like enoxaparin as soon as fetal heart sound heard or from 5 weeks.

2. **Genetic causes like chromosome translocation or Fetal Chromosomal Abnormality**

Treatment if family Hx. positive: Refer for genetic test and counseling

3. **Fibroids**: Common in Afro-Caribbean women. It can be so big that it can distort the uterine and a myomectomy may be needed.
4. **Thrombophilia** is inherited and causes recurrent miscarriage. Rx is with LMWH (enoxaparin). Patient usuallypresents with thrombosis, DVT and PE.
5. **Uterine abnormalities** e.g. that of uterine septum usually causes miscarriages in the 2nd trimester.
6. **Infection** like bacterial vaginosis
7. **Endocrine causes** like PCOS

c) Infection

Group B Streptococcal infection (GBS)

- Asymptomatic in pregnant women.
- Diagnosis is culture from endocervical swab. If that option is not available then choose lower vaginal swab, if that is not available choose high vaginal swab or perianal swab
- Up to 70% of all children born from infected GBS mothers are also colonized at delivery but only 1% develop infection
- GBS can cause Pneumonia, Septicaemia , Meningitis

Prevention: Antibiotics are given to mothers during delivery if they are colonized with GBS or if mothers have previous Hx. of GBS infection

Treatments: Intravenous Benzyl penicillin

a. **Abortion (Termination of pregnancy)**

Under UK laws no one has to have abortion and no one has to do one, unless there are medical or social reasons like risk to mothers life if the pregnancy continues.

Methods of abortion:

- < 7 wks – medical
- 7-15wks – medical or surgical
- >15 wks – medical

Medical Treatment

- **Mifepristone orally and then gemeprost per vagina 36-48 hours later**

OR

- **Mifeprestone orally followed by misoprostol per vagina 36-48 hours later**

NB. Add second medication only if abortion not completed

Surgical Treatment is by D& C (dilatation and curettage)

e) Bleeding in 1st trimester of pregnancy

Commonly associated with

1. Miscarriage
2. Ectopic pregnancy
3. Gestational trophoblastic disease

Miscarriage: This is a spontaneous abortion. It is defined as expulsion or removal of the embryo or fetus at a stage of pregnancy when it is incapable of independent survival.

NB: This is loss of pregnancy BEFORE 24 weeks (AFTER 24 weeks it is called STILL BIRTH)

Majority of miscarriages occur between 10-12 weeks

Management:

- **Send to early pregnancy assessment unit (EPAU)**
- **Transvaginal Ultrasound scan**
- **Serum HCG**
- **Anti D prophylaxis**

1. **Non-sensitised with rhesus –ve factor and<12 weeks.**
2. **All women >12 weeks gestation who are bleeding**
3. **If any medical or surgical intervention has been used.**

TYPES OF MISCARRIAGE

i) Threatened miscarriage

- Bleeding per vagina
- With or without abdominal pain
- **Closed cervical os**
- USS shows intrauterine gestation sac, fetal poles, fetal heart activity

Management: give anti D if > 12 weeks gestation or if having heavy bleeding.

ii) Complete abortion

- Bleeding per vaginal
- Abdominal pain
- Closed cervix
- USS shows empty uterus
- Endometrial thickening < 15 mm

Management: Anti-D if >12 weeks or heavy bleeding or if Rh-ve. Monitor serum HCG (human chorionic gonadotropin)

iii) Incomplete abortion

- Bleeding per vaginal - usually there is passing of large clots
- With or without abdominal pain
- Cervix open on PV examination
- USS shows heterogeneous tissue +/- gestational sac
- No fetal activity

Management: Medical or expectant management , Anti D if > 12 weeks

iv) Missed miscarriage (Fetus dies but is retained in the uterus)

- +/- bleeding
- +/- abdominal pain
- +/- loss of pregnancy symptoms
- USS shows no fetal activity (but the fetus is in the uterus)
- No fetal pole, yolk sac
- Os closed

Management: Surgical or medical, Anti – D if > 12 weeks.

v) Inevitable abortion (miscarriage)

- Bleeding per vaginal
- +/- abdominal pain
- Open cervix
- USS shows +/- gestational sac , +/- fetal pole
- Positive fetal heart activity

Management: Surgical/medical/expectant
Most appropriate treatment is expectant management
Give Anti-D if >12 weeks

Expectant Management:

- **Highly effective in incomplete miscarriage**
- **If expectant management is unsuccessful then offer surgery**
- **Warn about pain and increased bleeding**

Medical Management:

- **Prostaglandin analogues**

  e.g. misoprostol or gemeprost either P/O or P/V.

- **Bleeding can take up to 3 weeks**
- **24hrs telephone advice should be available admission**

Surgical Management:

- **Evacuation of retained product of conception**
- **Suction and curettage may be used**
- **Indications: Excessive bleeding, inevitable abortion, missed miscarriage, patient request**

Complications of Surgical Management

1. Infection
2. Haemorrhage
3. Uterine perforation
4. Cervical tears
5. Intra uterine adhesions (Asherman's syndrome) This can lead to infertility.

Ectopic Pregnancy: This is implantation of the products of conception outside the uterus

Symptoms

- Amenorrhea of typically **6-8weeks**
- Lower abdominal pain
- Shoulder tip pain
- Adnexal tenderness
- Cervical excitation and tenderness
- PV bleeding

Investigations

- Urine Pregnancy Test
- TV USS scan
- Serum pregnancy test (beta HCG)
- Diagnostic laparoscopy

Management
Depends on clinical picture

1. If patient collapsed / in shock = do pregnancy test. If positive, perform **URGENT LAPAROTOMY**.

2. If pregnancy test positive with abdominal signs of ectopic pregnancy (shoulder tip pain, pelvic tenderness, cervical excitation test) , do Transvaginal ultrasound scan- If it shows an empty uterus perform **Diagnostic laparoscopy**.

3. If pregnancy test positive but Uterus empty on TV scan and no abdominal signs **do quantitative serum HCG**

A. If serum HCG > 1000 IU following a TV Ultrasound scan or if > 6500 IU following transabdominal scan perform **Diagnostic laparoscopy**

A. If less than the above figures, then **recheck beta HCG in 48hrs**.

- Beta HCG should fall to less than half the baseline value every 48 hours.
- If beta HCG not less than half the baseline value or steady or only slightly reduced perform **Diagnostic laparoscopy**.
- If falling rapidly it means the pregnancy is aborting. If patient is well then only expectant management is needed. Repeat HCG to ensure levels are falling.

NB : IF SYMPTOMS DEVELOP AT ANYTIME THEN DO LAPAROSCOPY

Management:

- Laparoscopy is preferred
- Laparotomy is preferred only if patient is in shock or collapsed

Procedure:

Ectopic in fallopian tube is treated either by Salpingotomy or Salpingectomy.

Gestational Trophoblastic Disease

a. Hydatidiform mole (premalignant)

- Large for dates
- Exaggerated symptoms of pregnancy
- Hyperemesis Gravidarum due to markedly increased beta HCG
- Hyperthyroidism
- "Snowstorm" appearance on ultrasound

Management:

- **Suction curettage is the method of choice of evacuation**
- **Give Anti-D prophylaxis**
- **Monitor Beta HCG after evacuation every 2 weeks until normal. After Beta HCG is normal, monitor monthly for up to 6 months.**
- **Measure Beta HCG 6-8 weeks after any future pregnancy regardless of outcome**

a. Gestational trophoblastic neoplasia/Choriocarcinoma (malignant)

- **Usually** follows a molar pregnancy but can follow a normal pregnancy, ectopic pregnancy or abortion
- Should always be considered when a patient has continued vaginal bleeding after the end of a pregnancy.
- Should also be considered in any woman developing acute respiratory or neurological symptoms after any pregnancy (due to metastasis)

Management: Chemotherapy

2. SECOND TRIMESTER COMPLICATIONS

a. **Pregnancy Induced Hypertension (PIH)**

- Defined as hypertension in the 2nd trimester of pregnancy in the absence of proteinuria or other markers of pre-eclampsia.
- PIH includes risk of developing pre-eclampsia
- Delivery should be aimed at the time of expected date of delivery.

Treatment: i) Methyldopa ii) Labetalol iii) Nifedipine

a. **Pre-Eclampsia**

- BP ≥140/90 and 300 mg proteinuria in 24 hour urine collection
- In women who are already hypertensive a rise of BP ≥ 30 mmhg systolic or ≥ 15 mmhg of diastolic is used

Risk factors

- Previous severe or early pre-eclampsia
- Age > 40 years
- Family history of pre-eclampsia
- DM , HTN , Renal diseases

Signs & Symptoms

- Headache
- Visual disturbances
- Epigastric or RUQ pain
- Nausea & vomiting
- Rapid oedema especially of the face
- BP ≥ 140 /90 or
- In severe pre-eclampsia BP is ≥ 170 /110

- Proteinuria > 300 mg / 24hrs
- Confusion

Investigation: To rule out HELLP Syndrome

- FBC - Thrombocytopenia & Anaemia
- Coagulation profile – PT & APTT are prolonged
- Biochemistry – increased Urea & Creatinine.

1. Mild to Moderate Pre-Eclampsia
Definition: BP < 160 systolic and < 110 diastolic with significant proteinuria and no maternal complication

Management

- If significant proteinuria then ADMIT {i.e + + proteinuria or > 300 mg proteinuria /24hrs}
- 4 hourly BP
- Cardiotocography to monitor for fetal distress
- Daily urinalysis to check for proteinuria
- 24hr urine collection for proteinuria
- Regular USG assessment every 2 weeks to monitor for fetal retardation
- **IF > 160 SYSTOLIC OR >110 DIASTOLIC START ANTI –HYPERTENSIVE**

2. Severe Pre-Eclampsia
Definition: BP ≥ 160 systolic or ≥ 110 diastolic in the presence of significant proteinuria (≥ 1g /24hrs or >++ on dipstick) or if maternal complications occur .

Management

- Anti hypertensive to bring BP down to < 160 systolic and < 110 diastolic.
- Hydralazine intravenously is the 1st choice
- Labetalol
- Give MgSO4 to prevent eclampsia
- CTG & USG to monitor to baby
- If less than 34 weeks gestation give steroids to help production of surfactant.

Complications of Pre-eclampsia

1. **Eclampsia**
2. **HELLP syndrome – Haemolysis, Elevated Liver enzymes , Low Platelets**
3. **DIC**
4. **Renal failure**
5. **Placental abruption**

Indications for Immediate Delivery in Pre Eclampsia:

1. Worsening thrombocytopenia or coagulation profile
2. Worsening renal or liver functions
3. HELLP syndrome
4. Eclampsia
5. Fetal distress-as shown by CTG
6. Severe maternal symptoms

A. **Eclampsia**

Definition: Symptoms of Pre eclampsia + seizure = eclampsia
HELLP syndrome is regarded as a variant of severe pre – eclampsia. **NB. If a woman has a fit a few days after delivery, it is always eclampsia until proven otherwise because eclampsia can happen in the post partum period.**

Management:

- **ABC**
- **MgS04 intravenous bolus 4g, then 1 g intravenous infusion for 24 hours and if seizure recurs give intravenous bolus.**
- **Monitor BP, pulse , RR & O2 saturation every 15 minutes**
- **If BP > 160/110 give anti hypertensive (Hydralazine , labetalol , nifedipine)**
- **CTG to monitor the baby**
- **Deliver the baby once the patience is stable, delivery is by caesarean section usually but if appropriate then Per Vaginal.**

NB. If patient has been given Magnesium sulphate and suffered another fit, repeat Magnesium sulphate.

3. THIRD TRIMESTER COMPLICATIONS

Antepartum Haemorrhage in the 3rd trimester

Common Causes
i. Placenta Praevia
ii. Placenta Abruption

i.Placenta Previa
The placenta is inserted wholly or in part into the lower segment of the uterus.

Types:

- **Placenta major** - the placenta completely covers the cervical os
- **Placenta minor -** the placenta is located close to the cervical os or cover it partially

Presentation: Bright red painless bleeding per vagina.

Investigation: Transvaginal USS is more accurate than transabdominal

Management

1. **If major Placenta Praevia and bleeding – Admit patient**
2. **If placenta edge is < 2cm from the internal Os then elective caesarean is the mode of delivery**

ii. **Placenta Abruption**

This is when the placenta separates from the uterus before delivery of the fetus. Blood accumulates behind the placenta in the uterine cavity or is lost through the cervix.

Presentation: Dark coloured blood per vagina with sudden onset, severe, constant abdominal pain with rigid abdomen.

Investigation: Diagnosis is clinical but ultrasound is done to exclude PP and to check the baby.

Management

1. **Admit**
2. **Check the wellbeing of the baby with cardiotography (CTG) and USS.**
3. **If fetal distress or maternal compromise resuscitate and deliver now (caesarean).**
4. **If no fetal distress deliver by term.**

Distinguishing Placental Abruption from Placental Praevia

Placental abruption

- Shock is out of proportion from visible blood loss
- Constant pain
- Tender tense uterus
- Fetal heart sounds absent/distressed
- Coagulation problems like DIC

Placenta Praevia

- Shock in proportion with visible blood loss
- No pain
- Non tender uterus
- Normal fetal heart sounds
- Coagulation problems are rare

MEDICAL PROBLEMS IN PREGNANCY

a. Diabetes

- Avoid unplanned pregnancy
- Monitor Hba , c </= 6.1% Glycosalted Hb is an indication of the general control of diabetes.
- Give folic acid 0.4mg po od.
- Stop all oral hypoglycaemic medications and use insulin
- Stop Statin, ACE-i , A2A
- Use other anti HTN- e.g. Methyl-dopa.
- Glucosuria in pregnancy is common and it does not mean diabetes.

Complications of Diabetes in Pregnancy

- Hypoglycemia of the newborn.
- Stillbirth
- Large Baby
- Intrauterine Death

b. Epilepsy

- Use anti – convulsant that controls seizures
- Give high dose folic acid 5 mg/day
- Give Vitamin K from 36 weeks

Complications

- Fetal Valproate syndrome is associated with major systemic anomaly
- Neural tube defects are common with Valproate
- Neural developmental delay.
- Anemia = Hb less than 11.5 Rx. FeSO4 200 mg once a day.
- Thyroid Disease
- SLE= Common in Pregnancy and post partum

a. **Thyroid disease in pregnancy**

- **Hypothyroidism is common, especially in the post partum period due to Sheehan's syndrome. Sheehan's syndrome is pituitary infarction due to excessive post partum bleeding.**
- **Grave's Disease** improves in pregnancy.
- **Treatment: Propylthiouracil is the first choice anti-thyroid medication in pregnancy and breastfeeding instead of carbimazole.**

d. **Anaemia in Pregnancy: Hb <11.5 g/dl**
Common problem, occurs in 1/3 of women in 3rd trimester.

Causes: 85% caused by Iron deficiency anaemia
Less commonly caused by: Folic acid deficiency, sickle cell disease, thalassemia, B12 deficiency, hemolysis, PNH, leukemia, GI bleed, occult celiac disease

Investigations:

- Hb
- MCV: if <76 most probably due to iron deficiency anaemia. If normal it is typical of anaemia in pregnancy (dilutional anaemia)
- Serum ferritin: 10-50 ug/L needs monitoring, <10 ug/L requires treatment

Management:

- **Routine iron replacement is not recommended in the UK. If given routinely it can cause iron overload and lead to haemochromatosis.**
- **Treat with oral ferrous sulphate if Hb <11.5 g/dl**

e. SLE - common in pregnancy and post partum period. If the patient already has SLE, it usually flares up in pregnancy.

f. Carpal tunnel syndrome - common in pregnancy. Treatment of this condition in pregnancy is usually conservative

LABOUR

There are 3 stages of labour:
1st Stage: Starts from regular contractions to full dilatation of the cervix i.e. 3cm to 10 cm.

2nd Stage: This is from full dilatation of the cervix to the delivery of the baby i.e. from 10 cm to the birth of the baby.

3rd Stage: From the delivery of the baby to the delivery of Placenta (takes approximately 30 mins).

Indications for Induction of Labour:

- Prolonged pregnancy >42 week.
- Uteroplacental insufficiency
- Intrauterine growth retardation
- Oligohydramnious
- Abnormal CTG
- Pre labour rupture of membranes
- Chorioamnionitis
- Intra- uterine death
- Severe pre – eclampsia/eclampsia after maternal stabilization

Medical Indications for labour Induction

- Severe hypertension
- Uncontrolled DM
- Renal diseases + deterioration of the renal function
- Malignancy

Monitoring in Labour

1. Regular PV examination to monitor the progression of dilatation. It should dilate by 2cm every 4 hours.
2. CTG - To check for fetal distress. If there is fetal distress then do fetal blood sampling.
3. Fetal blood sampling (FBS) - this is ABGs from the scalp of the baby. If it shows hypoxia then do C-section
4. If there is Meconium Stained Liquor, perform CTG to check for fetal distress. And similarly if there is fetal distress, do FBS.
5. BP, Pulse and Temperature to check for chorioamnionitis.

MECONIUM ASPIRATION SYNDROME

This is when the fetus aspirates the meconium which leads to mechanical blocking of the airway. It acts as a chemical irritant and causes Pneumonitis.

Management:
Meconium stained liquor and PROM is associated with high risk of infection therefore it requires immediate induction using erometrine and oxytocin.

PRE-LABOUR RUPTURE OF MEMBRANE (PROM)
This is leakage of the amniotic fluid in the absence of the uterine activity at term (after 37 weeks)

Complications:

- Neonatal infection
- Chorioamnionitis
- Post partum endometritis

Chorioamnionitis:
Symptoms

- Fetal tachycardia
- Maternal tachycardia
- Maternal pyrexia

- Increased WBC
- Tender uterus

Treatment: Metronidazole + amoxicillin

Management of PROM

- **Expectant until 24 hrs**
- **If after 24 hrs labour has not started, then induce labour**
- |
- **If PROM and the mother is GBS positive do immediate induction of labour. Give the mother Benzyl penicillin IV in labour and screen the baby for GBS infection after birth.**
- **PROM+Meconium stained liquor needs immediate induction of labour.**

OBSTETRIC EMERGENCIES

1) Sudden maternal collapse

- There are multiple causes – MI, PE

Management: Use the ABCD approach to manage these types of patients.

2) Cord prolapsed

This is when the umbilical cord protrudes below the presenting part; this may lead to cord compression (lead to hypoxia + cut of blood supply)

Management: Urgent delivery by Caesarian section or instrumental delivery.

3) Shoulder distocia = shoulder Impaction

Defined as any delivery that requires additional obstetric maneuvers in order to deliver the shoulder.

Management: Episiotomy

4) Uterine inversion

Severe symptoms like hemorrhage, shock, mass in the vagina on per vaginal examination

Management: – ABC
Johnson maneuver (push up fundus through the cervix with the palm)

CAESARIAN SECTION

Indications

- If repeat c-section
- Fetal compromise
- Failure to progress in labour
- Eclampsia

NB. Once a caesarian section, almost always a caesarian section.
This means that women who have had one caesarian section are likely to give birth by caesarian section in the future due to increased risk of uterine rupture if vaginal delivery is attempted.

PAIN RELIEF IN LABOUR

- **NB. Usually if a woman is not tolerating pain in labour, she should be given entonox as an initial analgesia**
- **If Entonox is not effective, then offer pethidine IM.**
- **If she is still not tolerating pain, then offer epidural anaesthesia.**

- **Epidural anaesthesia is the most commonly used anaesthesia for C-section**

1) Non-pharmacological

- Education,
- Trusted partner
- Warm bath
- Acupuncture
- Hypnosis
- TENS-Trans Electrical Nerve Stimulation, stimulates central opiate receptors and non – pain fibers

2) Pharmacological

a. **Entonox** – 50% nitrous oxide + 50% Oxygen. It can be inhaled throughout labour. It is safe for the baby.

a. **Pethidine** (IM injection) It can be given until 2 hrs before delivery as it can cause respiratory depression of fetus if given < 2 hrs before delivery.

a. **Diamorphine** – can be used but can cause respiratory depression if given 4hrs from delivery time

a. **Pudendal Nerve Block** – used for operative vaginal delivery (episiotomy) or instrumental delivery dilated (Episiotomy).

3) Epidural- provides adequate analgesia, it is the most effective type

- Set up only once labour has begun i.e. .cervix 3 cm or more.
- It is appropriate especially for breech & multiple pregnancy, because usually mode of delivery is caesarian section.

Side Effects

1) Motor-neuro inhibition, which causes weakness of the lower limbs, making it difficult to walk around during labour.
2) Postural Hypotension

Contra- indications to epidural anaesthesia

- Septicaemia
- Infection at the site
- Thrombocytopenia <75
- Coagulopathy
- Allergy to lidocaine
- Severe aortic stenosis

4) Spinal anesthesia

- Enables mother – child bonding earlier
- Little motor blockage , allowing mothers to stand and walk around during labour especially if it's combined with epidural.

Instrumental Delivery
(operative - Forceps/Vacuum -)

Indications

- Delay in 2nd stage of labour
- Prolapsed cord or fetal distress in 2nd stage of labour

Retained Placenta

It's the undelivered placenta within 1 hr of physiological labour or 30 min of active labour

Treatment:

1) oxytocin or syntometrin
2) Maneouvers - eg. controlled cord traction and uterine Massage
3) Manual evacuation of the placenta requires general anaesthesia

Abnormal Placental attachment

- ***Placenta accrete*** – villa are attached to myometrium
- ***Placenta increta*** – villi invade into myometrium
- ***Placenta percreta*** – villi passes through myometrium into serosa membrane,which may cause heavy bleeding in delivery and hysterectomy may be required.

NB. In normal circumstances, the placenta is attached to the endometrium.

POST PARTUM HAEMORRHAGE

Types

a. Primary This is loss of > 500ml of blood in 24hrs
b. Secondary This is excessive loss of blood between 24hrs & 6 weeks

Causes

1. Uterine Atony This is due to prolonged labour. The uterus becomes tired and fails to contract after delivery. Failure of the uterus to contract leads to continuous bleeding.
Treatment: Oxytocin

2. Genital tract trauma – This usually occurs after episiotomy or any other instrumental delivery.

3. Infection of the Uterus (Endometritis) This usually occurs 5-7 after delivery. Presents with fever and PV bleeding.
Treatment: Metronidazole + amoxicillin.

4. Retained Product of conception can cause bleeding on its own.
Treatment is D&C (dilatation and curettage)

5. Coagulation Disorder like disseminated intravascular coagulopathy (DIC). This usually follows severe bleeding eg. placenta abruption or uterine rupture.
NB. Uterine rupture is common in women with previous caesarean section. There is usually serosanguinous discharge from the scar.

6. Large Placenta Site eg. from large babies like in Diabetic mothers, or multiple pregnancies

GYNAECOLOGICAL CONDITIONS

CONTRACEPTION

a. Routine Contraception

- **Barrier method:** Condoms, caps, cervical sponge , spermicide , female condoms.
- **Male condoms** are the most widely used.

- **Natural methods:** These are done by monitoring the fertile and non-fertile days in the menstrual cycle and timing sexual intercourse with non-fertile days.

- **IUCD (Intrauterine Contraceptive Device):** Insert after 4 weeks post partum as long as not pregnant, and 48 hrs after termination of pregnancy (TOP)

Mechanism

- It prevents pregnancy by inhibiting implantation in the uterus.

Complications

- Infection
- Perforation
- Irregular per vaginal bleeding
- Dysmennorrhea
- **Ectopic pregnancy -** risk for ectopic pregnancy is increased because if the pregnancy is not implanted in the uterus, it will be implanted outside the uterus.

Contraindications

- Pregnancy
- Undiagnosed per vaginal bleeding
- Current urinary tract infection
- History of pelvic inflammatory disease
- Large fibroids that make insertion difficult
- Copper allergy

Advantage
Provides long term contraception lasting for up to 3 yrs maximum.

- **COCP:** Combined Oral Contraceptive Pill

Contains **estrogen** + **progesterone**
Ideally started on 1st day of menstrual period for 21 days followed by 7 pill-free days.

Advantages

- Decrease in menstrual pain and bleeding (can be used for dysmenorrhea and menorrhagia)
- Lower incidence of functional ovarian cysts.
- Decreased risk of ovarian and endometrial cancer.
- Decreased risk of colorectal cancer.
- Improvement in acne vulgaris

Disadvantages: Increases the risk of DVT/PE, ovarian & breast carcinoma), increases blood pressure.

Interactions:

- Affected by enzyme inducing drugs.

- **Enzyme inducing antibiotics** are **rifabutin and rifampicin** and they require extra precautions eg. condoms.
- Other antibiotics do not reduce the efficacy of COCP so extra precautions are not needed.
- When using enzyme inducing antibiotics, appropriate contraceptive measures are required up to 8 weeks after stopping the enzyme inducing drugs.

- **Enzyme inducing anti-epileptics** are carbamazabine, phenytoin, primidone, topiramate, oxcarbazepine, and phenobarbital
- If taking COCP with enzyme inducing antieileptics, take an increased dose of COCP but **alternative contraception is the best advice.**

- **Non-enzyme inducing anti-epileptics:** Sodium valproate, lamotrigine, ethosuximide

Contraindications

- Pregnancy
- Smoking >20/day AND above 35 years of age
- History of DVT or PE
- Migraine with aura
- Migraine without aura but above 35 years
- Liver disease
- BMI >39

- **Transdermal Patch - Contains the same hormones as the COCP. It is changed once a week for three weeks followed by one patch-free week.**

Mechanism: Inhibits ovulation

NB. The transdermal patch is not recommended for use when taking an enzyme inducing anti epileptic medication.

- **POP**– Progesterone only pill

Disadvantage: Less effective compared to COCP, can be used if COCP is contra-indicated.

Advantage: Does not cause DVT / PE.

NB. Not affected by any type of antibiotics.
POP is not recommended for use when taking an enzyme inducing anti epileptic medication.

- **Depo- provera**– Injectable progesterone only contraception which is injected every 3 months.

Advantages

- Very effective
- Used in women unwilling or unable to take the pill or unable due to various reasons e.g. – mental retardation

NB. Not affected by enzyme inducers like certain anti-epileptics.

- **Contraceptive Implant** - Progesterone only implant, it is implanted under upper arm. It lasts for 3 years.

Disadvantages: menstrual disturbances and weight gain

- **Mirena Coil** - also called the IntraUterine System (IUS)
  - It is a Levonorgestrel containing coil which is inserted into the uterus. It lasts for 5 years with failure rate of 1-2%.
- IUS is the preferred contraception if the woman needs long term contraception.
- It is also beneficial in women with menorrhagia who desire long term contraception.
- May be used to treat menorrhagia in women with fibroids unless the fibroids are very big and prevent insertion of the device.

- **Female laparoscopic sterilization** – Regarded as a permanent procedure because it is difficult to reverse. It is done by occluding the fallopian tube by clips or by ligation and tie. Failure rate is 1 in 200.

- **Male sterilization** – easier, quicker, safer day case procedure, with less complications. The procedure is done under local anaesthesia.

b. EMERGENCY CONTRACEPTION

1. **IUCD**= COPPER COIL or simply Coil, can be used for up to 5 days (120 hours) after un-protected sex. It provides long-term contraception as well.

1. **Emergency Contraception Pill (Morning pill)** (Levonorgestrol) can be used up to 3 days (72 hours). Next period may be late or early.

NB. If taking emergency contraception with enzyme inducing antiepileptics, the dose of emergency contraception must be doubled.

NB. All hormonal contraceptive pills prevent pregnancy by inhibiting ovulation.
All IUCDs including Mirena coil prevent implantation of fertilised egg in the uterus there there is a risk of ectopic pregnancy.

MENOPAUSE

- Average age is 52 years but is usually between 45-55 years.
- This is permanent cessation of menstruation for a minimum of 12 months with no other cause of amenorrhea

- **Peri-menopause** is a period beginning with the first clinical signs and endocrine signs of the approaching menopause. There are usually irregular periods around this time.
- **Pre-menopause** is 1-2 yrs immediately before the menopause.
- **Post – menopause** period is from the last period.
- **Climacteric** is the phase compassing the transition from the reproductive state to non-reproductive state.

Complications of Menopause:
Short term:

- Vasomotor symptoms (hot flushes , night sweats , start before menopause , irritability)

- Treatment for hot flushes is hormonal replacement therapy (HRT)
- There are two types of hormonal replacement therapy: Estrogen only HRT and combined HRT.
- Estrogen only HRT is suitable for women who had hysterectomy.
- Combined HRT contains progesterone and estrogen and is suitable for women with a uterus. This is because progesterone protects proliferation of the endometrium which may lead endometrial carcinoma. It therefore protects against endometrial carcinoma.
- Sexual dysfunction (vaginal dryness, atrophic vaginitis leading to dyspareunia. Treat with local oestrogen.
- Psychological dysfunction: depression , anxiety , mood swings, irritability , lack of energy

Long term:

- Osteoporosis
- Cardiovascular disease
- Urogenital atrophy- Atrophic Vaginitis

OSTEOPOROSIS

- This is a progressive systemic skeletal disease characterised by reduced bone mass/density.
- This leads to an increased bone fragility and susceptibility to fractures.

Risk factors for Osteoporosis

- Ankylosing spodylitis
- Family hx of hip fracture
- Low Ca intake eg.: lactose intolerance
- Prolonged steroid use
- Rheumatoid arthritis
- Malabsorption
- Hyperthyroidism
- Hyperparathyroidism
- Alcohol abuse
- Early menopause (<45yrs, low BMI)
- Premature ovarian failure (<40yrs causes of which are chemotherapy and ovary removal)

Investigations:
Bone Mineral Density
(Measured using DEXA - Dual Energy X-ray Absorptiometry)
Normal: +1 to -1
Osteopaenia: -1 to -2.5
Osteoporosis: less than -2.5

Management :

- **Prescribe Calcium & vitamin D supplements**
- **Biphosphonates (Alendronic acid) are the 1st choice treatment as well as for the prevention of osteoporosis.**
- **Risedronates and etidronates are indicated if Alendronic acid is CI, Raloxifen is not recommended for treating post-menopausal females for primary prevention of osteoporosis unless increased risk of breast & endometrial cancer.**

URINARY INCONTINENCE

1. **Stress Incontinence**

Leakage of urine while coughing,sneezing, laughing and running.

Treatment:

- **Weight loss**
- **Physiotherapy**
- **Duloxetin**
- **Bladder neck surgery**

1. **Urge Incontinence**

Detrusor muscle instability, presents with sudden desire to urinate.

Treatment:
Avoid caffeine. Bladder training and pelvic floor exercises are good.

1. **Mixed incontinence (Urge and Stress)**

Treatment: Oxybutamine or Toterodine can be tried.
If there is enuresis then Desmopressin can be tried.

1. **True incontinence**

This is due to fistula formation. There is constant leaking of urine. Usually there is history of recent surgery.

Urometry/Urodynamics Assessment/Urodynamic Studies

1. **Filling urodynamic studies**

- During this process the bladder is filled with normal saline and at the same time, the bladder and abdominal pressures are measured simultanously
- If the patient has incontinence of urine during filling of the bladder, you check the 2 pressures.
- If the bladder pressure increased but the abdominal pressure was normal, it means the bladder contracted which lead to the increased bladder pressure. This confirms the diagnosis of **urge incontinence.**
- If both the bladder pressure and the abdominal pressure increase simultaneously, it suggests **stress incontinence**.

2. Voiding urodynamics studies

- During this phase, the woman is asked to urinate and the speed of urination and residual urine is measured.
- If the speed is <15 ml/sec this is abnormal.
- Residual urine of >150 ml is abnormal.

PROLAPSE

1. **Uterine prolapse** - This is usually in an elderly woman. There is a dragging sensation or feeling of pressure in the perineum or something is coming down.

   Treatment: Surgical or conservative if not possible to do surgery due to comorbidities eg. severe heart failure.

1. **Urethrocele** - This is prolapse of the lower anterior vaginal wall involving the urethra into vagina

1. **Cystocele** - This is prolapse of the upper anterior vaginal wall involving the bladder into the vagina

1. **Enterocele** - This is prolapse of the upper posterior wall of the vagina along with the small intestine

1. **Rectocele** - This is prolapse of the lower posterior vaginal wall along with rectum

CERVICAL CARCINOMA

- Cervical Intraepithelial Neoplasia CIN is a precursor of cervical cancer
- Columnar cells change into Squamous cells, this is termed as Columnar Metaplasia
- Caused by Human Papilloma Virus (HPV)

Risk Factors

- Persistent HPV
- Multiple partners
- Smoking
- Immunocompromised Eg HIV
- Use of COCP

N.B. Vaccination against HPV helps to reduce incidence of cervical carcinoma.

Cervical Smear
Routine screening is done using cervical smear every 3 years between ages 25-50, then every 5 years until aged 65.

- If **Normal smear:** Repeat every 3 years for women between 25 to 50 or every 5 years for women aged 50-65.

- **Inflammation:** Repeat smear after 6 months.

  Do Colposcopy after 3 abnormal results

- **Atypical Cells:** Repeat smear after 4 months.

  Do Colposcopy after 2 abnormal results

- **Mild/moderate/severe Dyskariosis:** Refer for Colposcopy.
- Mild Dyskariosis= CIN 1 (30%)
- Moderate Dyskariosis= CIN 2 (50-70%)
- Severe Dyskariosis= CIN 3 (90%)
- If **invasive**, suspect carcinoma à Urgent Colposcopy
- If **abnormal glandular cells** à Urgent Colposcopy

When to refer for Colposcopy:

- Any smear showing mild, moderate, severe Dyskariosis
- Any suggestion of malignancy
- Glandular abnormal cells
- 3 consecutive inflammatory smears
- 2 consecutive atypical smear
- 3 consecutive borderline smear
- 3 consecutive inadequate smear
- Post coital bleeding.

Management of CIN
Large loop excision of transformation zone.

Symptoms of Cervical Cancer:

- Weight loss, anaemia, tiredness
- Post-coital bleeding is alarming. It means cervical cancer until proven otherwise.
- Post- menopausal bleed

Investigation: Colposcopy and Biopsy

Stages of Cervical Cancer:

- **<** 4cm diam. = 1
- Parametrial involved = 2
- Extension to pelvic wall = 3
- Distant Mets. = 4

Treatment for Cervical Cancer:

- **1st stage = local excision +/- radiotherapy +/- hysterectomy**
- **From 2nd stage onwards = radiotherapy +/- chemotherapy**

VAGINAL DISCHARGE

Classification

- **Infective (Non-sexually transmitted)**
  - **Candida**
  - **Bacterial vaginosis**

- **Infective (Sexually transmitted)**
  - **Chlamydia trachomatis**
  - **Neisseria gonorrhoeae**
  - **Trichomonas vaginalis**
  - **Herpes simplex virus**

- **Non-Infective**
  - **Foreign bodies**
  - **Cervical polyps and ectopy**
  - **Genital tract malignancy**
  - **Fistula**
  - **Allergy**

A. Infective (Non-sexually transmitted)

1. **Vaginal Candida/Thrush**

Causes by Candida albicans in 70-90%

Risk factors:

- Estrogen exposure (reproductive years, pregnancy)
- Recent use of antibiotics
- Especially seen in immunocompromised patients
- Patients with DM

Signs & Symptoms:

- White, thick, curdy, **itchy** discharge with no odour.
- Soreness, superficial dyspareunia and dysuria
- Vulva may appear normal or may have vulval erythema, oedema, fissuring and satellite lesions

Investigation: Endocervical swab if not given then high vaginal swab. Vaginal pH ≤4.5.

Treatment: Vaginal and oral azoles are equally effective
Topical: Clotrimazole topical cream
Oral: Fluconazole capsule
NB. Use only topical antifungals in pregnant women. ORAL ANTIFUNGALS SHOULD BE AVOIDED.

2. Bacterial Vaginosis (mixed growth)
Caused by over growth of Gardnerella vaginalis, one of the organisms that is part of the normal flora of the vagina.

Signs & Symptoms:

- Thin offensive/fishy smelling discharge.
- No itch
- No vulval inflammation
- Discharge may coats the vagina and vestibule

Investigation: Endocervical swab if not given then high vaginal swab. Clue cells on microscopy. Vaginal pH >4.5

Treatment:

- **Oral Metronidazole is first line. Can be used in pregnancy.**
- **Clindamycin if there are side effects with Metronidazole (metallic taste, GI symptoms)**

B. Infective (Sexually transmitted)

1. **Chlamydia (STI)**
Most common bacterial STI in the UK. Asymptomatic in 70% of women.
> 1 week incubation period after unprotected sexual intercourse, can be up to 3 weeks.

Signs & Symptoms

- Mucoid purulent discharge
- Lower abdominal pain
- Abnormal bleeding (postcoital or intermenstrual) due to cervicitis or endometritis
- Dysparenuia or dysuria

Investigation: Endocervical swab / urethral swab

Treatment: Doxycycline. Use erythromycin in pregnancy.

Complications:

- Pelvic inflammatory disease (PID)
- Perihepatitis
- Reiter's Syndrome (Arthritis, urethritis, conjunctivitis)

2. **Trichomonas Vaginitis (STI)**

Signs & Symptoms:

- Scanty to profuse, offensive smelling, frothy, itchy discharge
- Dysuria, lower abdominal pain
- Inflamed vulva
- Strawberry cervix
-

Investigation – Endocervical smear. Vaginal pH >4.5

Treatment: Metronidazole. Can be used in pregnancy.

3. **Gonococcal infection**
Asymptomatic in up to 50%. < 1week incubation period. Symptoms will come few days after un protected sexual intercourse

Signs & Symptoms

- Increased or purulent vaginal discharge
- Lower abdominal pain
- Rare cause of heavy menstrual, postcoital or intermenstrual bleeding due to cervicitis or endometritis

Treatment:

- **Ciprofloxacin if uncomplicated.**
- **Ceftriaxone or cefotaxime if complicated.**

4. **Pelvic Inflammatory disease** – partner might have urethritis, hx of change of sexual partners. Usually the cause is PID. Common causatives agents are Gonococcus and Chlamydia therefore it is important that the broad spectrum antibiotics cover those 2 micoorganism.

Investigation: Endocervical swab is the investigation of choice. High vaginal swab can also be done.

Treatment: Broad spectrum antibiotics

- **Metronidazole + Doxycycline + Ceftriaxone**
- **Metronidazole + Ofloxacin**

5. **Human Simplex Virus**(HSV2) –
Occasionally presents with discharge

Signs & Symptoms

- Multiple painful vesicles and ulcers on vulva
- History of flu like illness

C. Non-infective

1. **Cervical Ectropion or Erosion**
Can be a normal finding in women of reproductive age. Common in pregnancy and in women using COCP. There is increased physiologic discharge, and the cervix bleeds easily on touch.

Investigation: Speculum examination.

Treatment: If symptomatic use acidic gel, silver nitrate cauterization, laser or cold coagulation

2**. Foreign Body**

Blood stained discharge with history of use of ring pessary, tampons, or IUCD. It is also common in young girls due to hygiene issues.

Investigation = speculum examination.
For IUCD do USS. If it not visible on ultrasound then perform pelvic x-ray. Since it is metal it will be visible.

Treatment: Removal of the foreign body

3**. Cervical Carcinoma**

Usually there is foul smelling blood stained discharge

Investigation: Colposcopy & biopsy

4**. Fistula**

Causes:

- **Inflammatory conditions - usually diverticulitis and crohn's disease (inflammatory bowel disease)**
- **Malignancy - usually rectal carcinoma**
- **Iatrogenic - post surgery and radiotherapy**

Types of fistulas:

- **Enterovesical - e.g. colovesical usually presents with pneumaturia (gas in the urine) and faecaluria (fecal matter in the urine)**
- **Enterovaginal - eg colovaginal. Passage of stool or flatus via the vagina is pathognomonic of a colovaginal fistula. It may also present with frequent vaginal infections or copious vaginal discharge.**

NB. Symptoms of the chronic disease causing the fistula may be present. These symptoms will help to determine the cause of the fistula.

VULVAR ULCERS

a. **Herpes simplex**

Caused by HSV Type 2
Acquired from infectious secretions on oral, genital, or anal mucosal surfaces during sexual intercourse and contact with lesions from other anatomical sites, eg eyes, skin or herpetic whitlow.

Clinical features:

- Multiple painful ulcers
- Systemic symptoms like headache, joint pains, generally feeling unwell (this differentiates it from chancroid)

a. **Chancroid**

Caused by Hemophilus ducreyi

Clinical features:

- Usually multiple ulcers
- Not indurated (soft core)
- Painful inguinal lymphadenopathy
- No systemic symptoms

Investigation: Swab from ulcer

Treatment: Azithromycin or ceftriaxone

a. **Granuloma inguinale (Donovanosis)**

Caused by Klebsiella granulomatis

Signs & Symptoms

- Beefy red ulcer

Investigations: Donovan bodies (intracellular inclusions in macrophages)

Treatment:
Doxycycline or Co-trimoxazole (Trimethoprim/sulfamethoxazole)

a. **Lymphogranuloma venereum (LGV)**

Caused by Chlamydia trachomatis. Common in Africa, India, South America, Caribbean.

Clinical Features:

- Painless papule or shallow ulcer

- Arthritis
- Buboes (grossly enlarged tender nodes in inguinal/pelvic/perirectal nodes
- Groove sign - separation of the enlarged inguinal and femoral lymph nodes by the inguinal ligament

Treatment: Doxycycline

a. **Syphilis**

Caused by Treponema pallidum which is transmitted during sex.
Primary syphilis - very infectious transient hard ulcer (chancre). Typically it is a single ulcer.

Secondary syphilis – 6 weeks-6 months after infection. Usually present with fever, lymphadenopathy, and rash on trunk, face, hands, and soles.

Tertiary syphilis – develops 2 years or more after initial infection. Forms granulomas in the skin, mucous, bones, joints and visceral organs.

Quaternary syphilis

1. Vascular syphilis: Affects ascending aorta causing aneurysm and aortic regurgitation
2. Neurosyphilis: Cranial nerve palsy, stroke, dementia

Investigations: Treponema specific antibodies / Veneral disease research lab slide test (VDRL)

Treatment: Benzylpenicillin or doxycylcine
For neurosyphilis: Ceftriaxone

NB. Any anogenital ulcer or sore is syphilis until proven otherwise.

Malignancies
Elderly patients with vulvar ulcer is always vulval carcinoma until proven otherwise.

VULVAR LESIONS

1. **Allergy** - worse after contact with eg : nylon , underwear , soap , chemicals.

1. **Lichen sclerosis** – white, flat, shiny vulva ; intense itch , bruised red purpura or erosions. Usually after middle age.

1. **Leukoplakia** – itchy white vulval patches due to skin thickening and hypertrophy

1. **Cancer of vulva** – indurated ulcer or any ulcer on the vulva.

1. **HSV**- Vesicles, very painful! Usually there are multiple ulcers with vesicles

Hyperprolactinaemia
Causes

- Prolactinoma (commonest cause)
- Antipsychotic medications eg. Haloperidol
- Pregnancy
- Head injury
- Hypothyroidism
- PCOS
- Cushing's

Signs & Symptoms

- Galactorrhea
- Amenorrhea/Oligomenorrhea
- Infertility
- Reduced libido
- Weight gain
- Dry vagina
- Facial hair

Prolactinoma
This is an adenoma in the pituitary gland.

Signs & Symptoms:

- **Same symptoms as above**
- Bumping into objects (bilateral hemianopia due to compression of the tumour on the Optic Chiasma)
- Headache

Investigation: 1) serum prolactin
2) MRI pituitary (for prolactinoma)

Treatment: Surgery (for prolactinoma) otherwise treat underlying cause

MENORRHAGIA

1. Dysfunctional Uterine Bleeding – DUB – heavy irregular bleeding in the absence of pathology.

Treatment:

1st Line: Mirena coil - If long term contraception desired otherwise offer antifibrinolytics eg. tranexamic acid
2nd line: Tranexamic acid
3rd Line: COCP (if contraception required), injectable progesterone
4th line: Endometrial or hysterectomy (If no desire to conceive)

2. Fibroids (Uterine Myoma, Fibroma, Leiomyoma)
Common in Afro-caribbean females
Majority are asymptomatic and do not require treatment
Responsive to oestrogen therefore it may increase in size during pregnancy, may decrease in size after menopause

Signs & Symptoms:

- Pelvic pain (compression onto adjacent structures)
- Infertility/ecurrent miscarriage
- Pelvic mass
- Menorrhagia

Investigation: USG

Management: Mirena coil is the first choice if the fibroid does not cause distortion of the uterus. Big fibroids make it difficult to insert.

1. If <3cm

   Trial of **pharmacologic treatment** first eg. Tranexamic acid
   ↓
   If that fails and uterus no bigger than 10-week pregnancy do **endometrial ablation**
   ↓
   If that fails do **hysterectomy**.

1. If >3cm and desires to retain uterus, and/or wants to avoid surgery do **uterine artery embolisation (UAE)**

1. If >3cm and desires to retain uterus do **hysteroscopic myomectomy** or **myomectomy**

3. Endometrial Carcinoma

Risk Factors:

- HRT
- Early menarche
- Late menopause
- Nulliparity

Signs & Symptoms:

- Per vaginal bleed
- weight loss
- Anaemia
- Tiredness
- Weakness
- Abdominal mass

NB. POST-MENOPAUSAL BLEEDING IS ALWAYS ENDOMETRIAL CANCER UNTIL PROVEN OTHERWISE

Investigation:

- Transvaginal Ultrasound
- Hysteroscopy and biopsy
- Endometrial sampling

Treatment:
Localized: Hysterectomy
Locally invasive or with metastasis: Chemo/Radiotherapy

PRIMARY DYSNMENORRHEA
These are painful periods in the absence of any structural abnormalities.

Treatment – Mefenamic Acid (NSAIDS). Paracetamol is usually not effective.

POLYCYSTIC OVARIAN SYNDROME

Two of the three following criteria are diagnostic (Rotterdam criteria)

- Polycystic ovaries (12 or more peripheral follicles)
- Oligo-ovulation or anovulation
- Clinical and/or biochemical signs of hyperandrogenism

Signs & Symptoms

- Oligomenorrhoea (defined as <9 periods per year)
- Infertility
- Acne
- Hirsutism in 60% (often on the upper lip, chin, around the nipples and in a line beneath the umbilicus)
- Male pattern balding, alopecia
- Obesity (usually central distribution) or difficulty losing weight
- Psychological symptoms - mood swings, depression, anxiety, poor self-esteem
- Acanthosis nigricans (may be present, sign of insulin resistance)

Investigations:

- USS/Laparoscopy
- Fasting sugar may be increased
- May have normal to reversed LH:FSH ratio
- Free testosterone may be increased

Treatment:
No treatment reverses the hormonal disturbances of PCOS and treats all clinical features, so medical management is targeted at individual symptoms, and only in association with lifestyle changes.

1. Weight loss and exercise, consider **Orlistat**
2. **Co-cyprindrol/ Eflornithine** for hirsutism
3. **COCP** /**POP** to control menstrual irregularities
4. **Metformin** for increased insulin resistance, menstrual irregularity, hirsutism, acne

For treatment of infertility:

1. **Clomifene** - stimulates ovulation
2. **Metformin** – can be used with clomifene
3. **Laparoscopic ovarian drilling or gonadotrophins (LH/FSH) –** 2nd line treatment to clomifene

Complications

- **Endometrial hyperplasia and endometrial cancer** in untreated cases
- **Increased risk of type 2 diabetes** especially if obese, >40 year old, strong family history of DM Type 2
- **Complications in pregnancy**: there is a higher risk of gestational diabetes, pre-term birth and pre-eclampsia.

ENDOMETRIOSIS
Chronic oestrogen-dependent condition characterised by growth of endometrial tissue in sites other than the uterine cavity, most commonly in the pelvic cavity (including the ovaries)

Signs & Symptoms:
The appearance or worsening of symptoms at the time of menstruation, or just prior to it, suggests endometriosis.

- **Dysmenorrhoea**
- **Deep dyspareunia**
- **Cyclical pelvic pain**
- **Infertility**
- Examination is often normal but can have: Fixed retroverted uterus, enlarged boggy tender uterus (typical for Adenomyosis), posterior fornix or adnexal tenderness, palpable nodules in the posterior fornix or adnexal masses

Investigation – Laparoscopy is the gold standard to visualize the endometrial deposits in the pelvic cavity

Treatment

1. **Medical treatment.** COCP, medroxyprogesterone acetate, GnRH agonists, Mirena coil.

Medical treatment reduces symptoms in majority of patients, symptoms recur once treatment has stopped.

1. **Surgical Treatments**
   a. Excision of deeply infiltrating lesions (may reduce pain)
   b. Adhesiolysis
   c. Bilateral oophorectomy (often with a hysterectomy)

OVARIAN TUMOUR
Risk Factors:

1. Nulliparity
2. Infertility
3. Early Menache
4. Hx. of Ovarian Cancer
5. Past use of contraceptive pills

Types of Ovarian Tumours

1. **Benign cystic tumours** eg. Teratoma (dermoid cyst) - may contain hair and teeth.

1. **Benign neoplastic solid tumour** eg. Fibroma - small solid tumours which are associated with Meg's syndrome and ascites

Meg's Syndrome:
3 Cardinal symptoms:

1. Benign ovarian tumour
2. Ascites
3. Pleural effusion

NB. When encountered with pleural effusion, remember Meg's syndrome.

Investigations:

- CA 125
- CT abdomen and pelvis

Treatment: Surgery

DEEP DYSPAREUNIA (Pain during sexual intercourse)

Causes

1. Endometriosis – accompanied by cyclic pelvic pain, infertility, hemoptysis (if spread to lungs)
2. PID – young sexually active patient, +/- history of recent change in sexual partner
3. Atrophic Vaginitis – post menopausal women. can be treated with local estrogen cream or local lubricant

INFERTILITY

Failure to conceive after one year of unprotected sexual intercourse (2-3 times a week). Investigations should be started after one year of trying.

Causes:

The main causes of infertility in the UK are:

- **Unexplained infertility (no identified male or female cause) (25%)**
- **Ovulatory disorders (25%)**
- **Tubal damage (20%)**
- **Factors in the male causing infertility (30%)**
- **Uterine or peritoneal disorders (10%)**

Abnormalities of the tubes, uterus or cervix

- Pelvic inflammatory disorders
- Reversed female sterilization
- Uterine abnormality eg. septate uterus
- Damage to cervix due to cone biopsy
- Endometriosis

Male factors: Azoospermia- poorly motile or low sperm count on semen analysis

Investigations for females:

1. Mid-luteal progesterone level to assess ovulation (done 7 days before last day of menstrual cycle eg. day 21 of a 28 days cycle)
2. Gonadotropins (LH/FSH) if with menstrual irregularity because it is difficult to determine the duration of the menstrual cycle

NB. Basal body temperature charts are not recommended to predict ovulation, as they are unreliable.
In irregular menstruation, mid-luteal progesterone levels cannot be used.

Investigations for males:

1. Semen analysis
2. FSH if 2 unsatisfactory results from semen analysis
3. Test for complete azoospermia is testosterone levels

ALARM BELLS IN OBSTETRICS AND GYNAECOLOGY

1. Postmenopausal bleeding is always endometrial cancer until proven otherwise.

1. Unscheduled bleeding on HRT is always endometrial cancer until proven otherwise.

1. Postcoital bleeding is always cervical cancer until proven otherwise even with a recent normal smear.

1. Pelvic discomfort, abdominal distention and dyspepsia could be symptoms of ovarian cancer.

1. All women of reproductive age with amenorrhea are pregnant until proven otherwise.

1. Abdominal or pelvic pain in a woman of reproductive years is always ectopic pregnancy until proven otherwise.

1. Abdominal pain(intermittent) with a negative pregnancy test could be ovarian cyst rupture.

1. "Being wet all the time" may signify urogenital fistula.

1. Vulval ulceration or bleeding may be neoplasm.

1. Exclude cancer in enlarging fibroids.

In a Pregnant Woman

1. Headache, flashing lights and epigastric pain means pre-eclampsia until proven otherwise.

1. Painless vaginal bleeding in the late pregnancy is always placenta previa until proven otherwise.

1. Haemoptysis, shortness of breath or chest pain is pulmonary embolism until proven otherwise.

1. Calf pain or swelling is always DVT until proven otherwise.

1. Watery vaginal discharge may signal pre term rupture of membranes.

1. Severe itching especially of palms and soles may be due to obstetrics cholestasis.

1. When a woman says that her baby is not moving as usual, take her seriously it is an indication of foetal distress.

1. Continuous worsening lower abdominal pain may indicate placental abruption.

1. Multiple changing, trivial complaints or missed appointments may flag up psychological or social problems including domestic violence.

Resource start date	2013-06-26 09:20
Resource end date	2023-06-27 09:20

Back

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Resource view

Resource name	Oncology
Resource description	Oncology
Resource content	

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ONCOLOGY LECTURE NOTES 2013

TUMOUR MARKERS

1. Alpha feto protein hepatocellular carcinoma, neural tube defect, germ cell carcinoma
2. CA 125 = Ovarian cancer
3. CA 153 + Breast cancer
4. CA 19-9 pancreatic cancer
5. CEA or carcino-embryonic antigen = colorectal carcinoma
6. HCG (human chorionic gonadotrophin) = choriocarcinoma
7. Prostate specific antigen (PSA) Prostate cancer

COMPLICATIONS IN METASTATIC CARCINOMA

TUMOUR LYSIS SYNDROME

1. This is breakage of cancer cells in patients who are being treated with radiotherapy or chemotherapy. Breaking down of cancer cells releases urea, potassium and phosphates. Urea is so high that it leads to formation of renal stones or gout.

Investigation: 1. 24 hour urinary urate or serum uric acid if there is renal stones.

Otherwise you need to check urea and electrolytes

Treatment: rehydration with intravenous fluids

2. **Superior Vena Cava (SVC obstruction** in metastasis:
This is compression of the SVC by the tumour leading to swelling of the face and arms. Patients have plethoric face. Also patient may present with shortness of breath as trachea may be compressed.

Investigation: Chest x-ray

Treatment: Dexamethasone orally in emergency

3. **CEREBRAL METASTASIS:**

Signs: signs of raised intracranial pressure

a. Headache
b. Vomiting
c. Papilloedema
d. With or without focal neurological deficits, fits or seizures

Investigation: CT scan head

Treatment: Dexamethasone or simply steroid.

4. SPINAL CORD COMPRESSION

This is usually due to metastasis to the spinal cord e.g from prostate or breast or lung cancer.

Symptoms: constipation, urinary symptoms and weakness or sensory loss in the lower limbs.

Investigation: MRI scan

Treatment: Dexamethasone in emergency, definitive treatment is decompression of spinal cord.

NB: Back pain does not mean spinal cord metastasis, it only suggests possible vertebral bone metastasis and the investigation of choice is nuclear medicine bone scan.

NB: BACK PAIN IS NOT A SIGN OF SPINAL CORD COMPRESSION, IT ONLY SUGGESTs POSSIBLE METASTASIS TO THE VERTEBRAL BUT AGAIN BACK PAIN IS COMMON IN PROSTATE CANCER AND MYELOMA

If there is back or bone pain, bone metastasis is more likely therefore investigate with bone scan.

5. HYPERCALCAEMIA

This is common in malignancy e.g breast cancer or prostate or lung cancer. Hypercalcaemia is common in squamous cell carcinoma.

Symptoms: thirst, polyuria, abdominal pain, constipation and confusion

Investigation: serum calcium.

Treatment: Rehydration or simply intravenous fluids, if not responding then biphosphanate e.g pamindronates.

SYMPTOMATIC TREATMENT IN TERMINALLY ILL PATIENT:

1. **PAIN MANAGEMENT:**

Use the pain ladder which contain 3 steps:

1 step: simple analgesia like paracetamol. Aspirin, NSAIDs
+/- adjuvant therapy

2 step: weak opiates: e.g codeine, tramadol, dihydrocodeine
+/- adjuvant therapy

3. Strong opiates: morphine, fentanyl patches, diamorphine, oxycodone.
+/- adjuvant therapy.

Long term analgesia is usually given orally e.g morphine orally — if not helping you keep increasing analgesia toghy doses e.g 400-500mg of morphine ——> if high oral doses not controlling the pain ——> then use patient controlled analgesia (PCA) which is usually given subcutaneously. Or you can give as an infusion if patient has constant pain.

NB: sometimes patient controlled analgesia is called morphine pump or simply morphine subcutaneous.

Morphine intravenous bolus is used in acute situation and is not an appropriate route of administration of morphine in terminally ill patient.

YOU CAN START PAIN LADDER FROM ANYWHERE BUT ONCE YOU START MAKE SURE YOU FOLLOW IT.

ADJUVANT THERAPY: Is specific type of treatment for a specific type of pain.

For example:

i. Nerve pain is better treated with steroid or anti-epileptic or anti-depressant like amitriptyline.
ii. Bone pain secondary to metastasis can be treated with radiotherapy
iii. Viscera pain is better treated with anti-spasmodics e.g mebeverine.

For example trigeminal neuralgia is treated with anti-epileptic like trigeminal neuralgia, post hepertic neuralgia is treated with anti-depressant like amitriptyline.

2. Intractable hiccough due to metastatic carcinoma use Haloperidol

3. Malodourous Fungating cancers use metronidazole

4. Severe bronchosecretions which is distressing to patient use hyoscine injection

5. Constipation secondary to opiate use stimulant laxatives e.g senna

6. Vomiting secondary to morphine use metoclopramide which is centrally acting anti-emetics

7. Severe anorexia may be treated with steroid.

8. Itching due to jaundice in metastasis is treated with cholestyramine

1. **GASTROSTOMY** may be used if there high risk of aspiration and there is need of permanent solution

1. **Stenting** can be used in certain situation like stricture of the oesophagus for symptomatic relief.

1. **Aspiration of pleural fluid or pleurodesis** may be used in recurrent pleural effusion if patient is terminally ill.

Starting patients on strong pain killers:

MORPHINE:

- Start with oral morphine 5-10mg every 4 hours orally, with equal amounts of break through dose as often as required (which means 5-10mg as required)
- If this is not effective, increase by 30-50% of the initial dose e.g. 5mg - 10mg - 20mg - 30mg - 40mg
- Once you have established the required dose of morphine, change to MST (morphine sulphate tablets) which is modified release morphine.

BASIC PRINCIPLES:

1. Morphine is usually prescribed twice a day i.e. 12 hourly every day once the dose is established.
2. Break through dose i.e. as required medication usually equals 1/6 of the total dose and is given every 4 hours.
3. Break through pain is the pain which come in between the doses of morphine, for example if you prescribe morphine twice a day at 08:00 and 22:00. If the patient experiences pain at 16:00. This is called break through pain.

To cater for break through pain, every time you prescribe morphine you have to prescribe as required dose for break through pain.

The break through dose is 1/6 of the total required dose in 24 hours.

When starting someone on morphine the regular dose of morphine equals the break through dose.
This is because the required amount of morphine in 24 hours is not yet known since you are just starting the patient on morphine.

The break through morphine is prescribed as an oral solution.

The common side effects of morphine are:

1. Drowsiness
2. Nausea and vomiting
3. Constipation
4. Dry mouth

If the oral route is not available try morphine/diamorphine IV/SC

If patient can not tolerate morphine/diamorphine due to side effects try oxycodone PO/IV/SC/PR.

Start oxycodone at an equivalent dose to the amount of morphine the patient was on.

e.g. If patient was on 100mg of morphine and is not tolerating it due to side effects, change it to oxycodone but find out what is the equivalent dose of oxycodone to the 100mg of morphine.

Oxycodone is as effective as morphine and is 2nd line opiate to morphine.

Oxynorm is an oral liquid form.

Morphine can be given as a liquid, tablets or IV form.

For break through pain, the liquid form of either morphine or oxycodone is prescribed.

Regular doses are prescribed either as tablets or IV or SC.

When just starting patient on morphine remember you start with oral solutions.

Fentanyl transdermal patches are used if it is not possible to take orally but can only be used if the required dose per 24 hours is known.

This is because the patches are changed every 72 hours and therefore it is not possible to change the dose in between.

Therefore it is important to use fentanyl patches in patients whose morphine requirement is established.

Treatment dose of morphine does not usually cause addiction.

Also, respiratory depression is also unlikely with the treatment dose of morphine.

There is no maximum doses of morphine or opiates, each individual is different.

Use pain ladder

If patient has severe pain, you can skip the first 2 steps and start with oral morphine straight away.

Once you start the patient on one step of the pain ladder and the pain is not controlled, move to the next step. Do not try another pain killer that is in the same step.

Do not skip steps i.e. if you started patient on paracetamol and pain is not controlled, start codeine and do not jump to morphine.

Do not use pain ladder for acute pain e.g. pain due to fractures.

Pain ladder is only for chronic pain management.

For acute pain choose analgesia according to severity of pain.

CANCER THERAPIES:

1. CHEMOTHERAPY:

These are cytotoxic drugs. They may be used as main treatment e.g. in haematological malignancies or may be used as an add on.

Side Effects:

- Vomiting (use metoclopromide)
- Alopecia
- Neutropenia
- Can cause damage to nerves

1. RADIOTHERAPY:

Can be used to cure the cancer or only to relieve the symptoms.

3. SYRINGE DRIVER:

This allows continuous subcutaneous infusion of drugs when oral medication is no longer feasible, and avoid repeated cannulation attempts.

Many drugs can be mixed together in the syringe.

e.g. midazolam for agitation
cyclizine for vomiting
hyoscine hydrobromide for respiratory secretions etc.

RISK FACTORS OF CANCER:

1. Familial adenomatosis polyposis is due to mutations in the APC genes. It is a risk factor for colorectal cancer.
2. Family history of colorectal cancer
3. Family history of prostate cancer
4. Hereditary non-polyposis colorectal cancer (HNPCC) predisposes to a colorectal cancer plus uterus, ovary, stomach, renal pelvis, small gut or pancreatic cancer.

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OPHTHALMOLOGY

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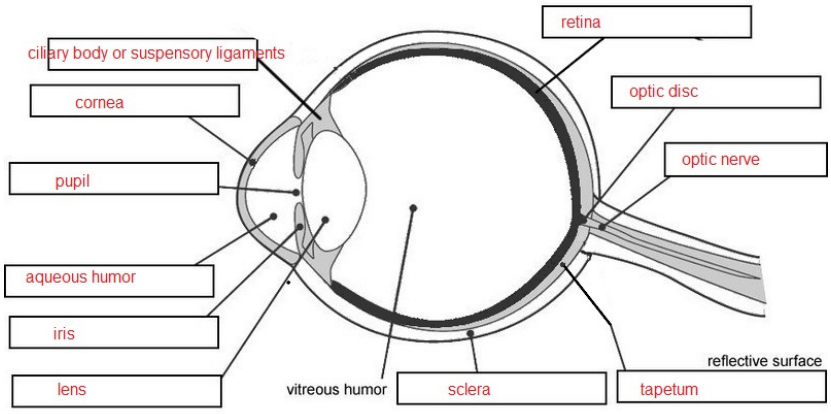
1. ANATOMY OF THE EYE

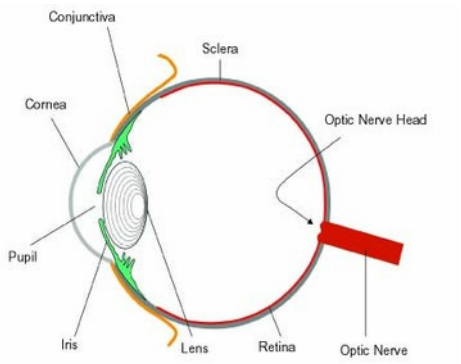
Sclera, conjunctiva, cornea, aqueous (iris, ciliary body are anterior uvea and choroid is posterior uvea)

Lens with suspensory ligaments, vitreous

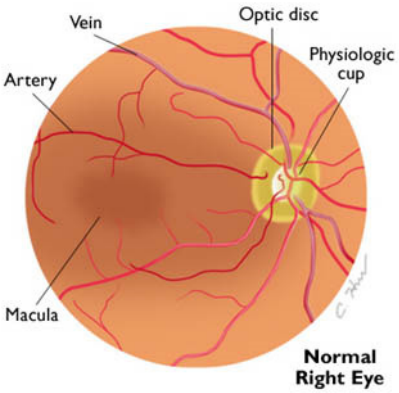
Coats of the Eyeball

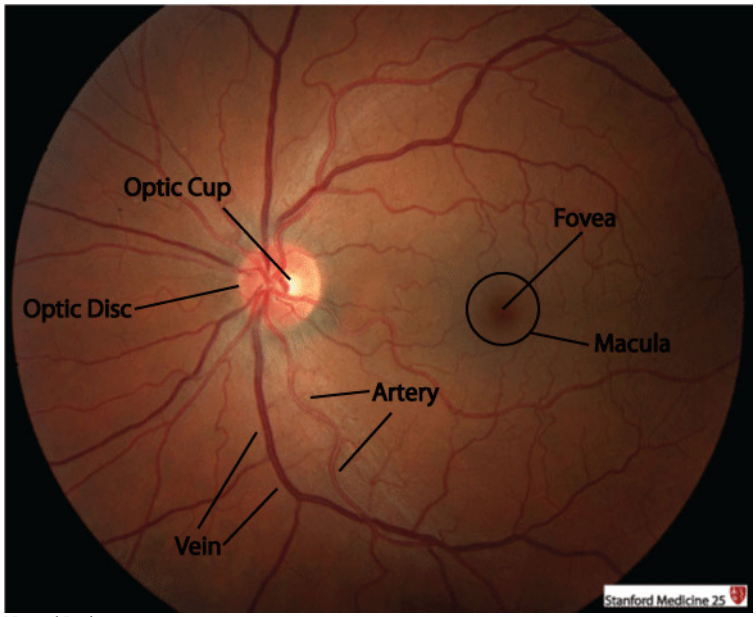
1. Sclera
2. Choroid (Uvea)
3. Retina





- Fovea centralis (rich with cones)
- Optic discs is part of optic nerve that can be seen by ophthalmoscope
- Optic cup is depression in centre of optic disc.





Normal Retina

2. LOSS OF VISION

Causes:

1. CENTRAL RETINAL ARTERY OCCLUSION (Severe visual loss to finger counting or hand movement)

- Usually elderly patient
- Sudden onset of visual loss
- Associated with hypertension and diabetes

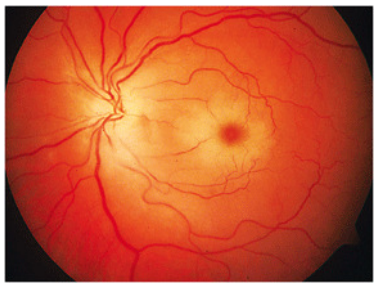
On examination: plus or minus carotid bruit

Fundoscopy: Optic disc is pale due to ischaemia. Cherry red spots on macula. Sometimes cholesterol is visible in the retinal arteries

Causes: Arteriosclerosis, Emboli

Investigations:

- Slit lamp examination
- Fundoscopy (Pale Optic Disc)
- Carotid Doppler- If carotid bruit



T/M:

-Macular massage

-Anterior chamber paracentesis

-I/V Acetazolamide if raised intraocular pressure

2. CENTRAL RETINAL VEIN OCCLUSION

Common in Obese men

-Also associated with HTN and diabetes with gradual onset

-It can have a sudden onset

-Usually patient wakes up in the morning with loss of vision due to decreased blood flow during night.

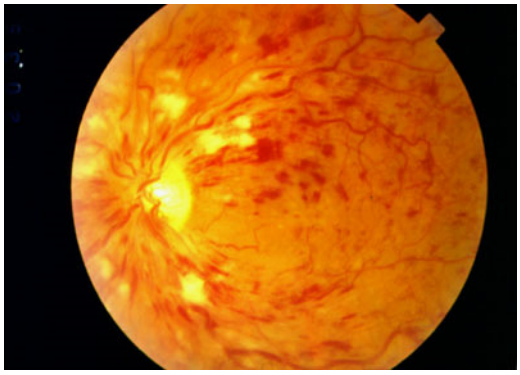
Risk Factors: Polycythemia Rubra Vera

-Cancer

Fundoscopy: flamed shaped haemorrhages and hard exudates. Stormy sunset or tomato splash appearance

Inx: -Slit Lamp

-Fundoscopy



Treatment: Reduce risk factors (DM, HTN, Smoking)

-If increased intraocular pressure –acetazolamide 500 mg IV

3. RETINAL DETACHMENT

Risk Factor: Myopia (or simply short sighted)

-May be caused by trauma

-Sudden onset

-Like a curtain coming down or like a flashing light like a camera

Investigations:

-Slit lamp investigation

-Fundoscopy

Treatment: Surgery (It will not resolve on its own unless surgery is done).

4. ACUTE CLOSED ANGLE GLAUCOMA

-Sudden loss of vision

-Acute red eye with pain

-Acute loss of vision with nausea and vomiting

- Halos on looking at light

-Plus or minus tunnel vision

-Nausea and vomiting indicate closed angle,

On examination: corneal oedema fixed and dilated pupil, IOP greater than 40mmHg, shallow anterior chamber

Risk factors: family history, common in female, increased age

Investigations:

-Measurement of intraocular pressure

Treatment:

-Acetazolamide intravenously 500mg

-Pilocarpine drops, dexamethasone drops (Steroid drops),

-Beta blockers- betoxalol, Timolol

If IOP pressure still persistently high then use Mannitol.

Dilating eye drops are contraindicated.

5. Optic Neuritis

- Common in multiple sclerosis
- Optic disc is pale
- Dull pain in the eye
- Usually young female patient (18 to 40 years in MS)
- Loss of red colour vision initially
- Sudden loss of vision, which is painless with possible similar symptoms in the past, which resolved completely (weeks or months).

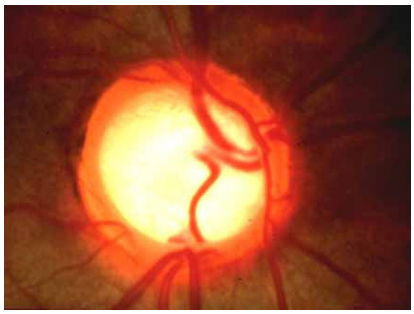
3. CHRONIC GLAUCOMA

-No vomiting/nausea

-No headache or red eye

-Usually gradual loss of vision

-Fundoscopy shows disc cupping or simply increased cup: disc ratio



-Most people see halos around the light

- Can cause tunnel vision

Investigation: Measurement of IOP

Treatment -Eye drops - pilocarpine

-Beta-blocker eye drops

4. GIANT CELL ARTERITIS aka TEMPORAL ARTERITIS aka CRANIAL ARTERITIS

-Age >50 years

-Common in females

-Unilateral headache

-Sudden loss of vision usually unilateral, worse with combing hair

-Temporal Arteritis, cranial arteritis

-Weight loss, unilateral headache worse on combing hair, weakness in upper limbs

Investigations:

-ESR is raised- this is **initial** investigation

-Temporal artery biopsy is **definitive**

NB: If you don't treat one eye you will lose vision in the other eye.

Treatment: Admit, Initially intravenous Methyl prednisolone is given for three days, followed by oral steroid (Prednisolone) for 2-3 years high dose. Then reduce the dose gradually.

SIDE EFFECTS of STEROIDS

1) GIT Bleeding- give PPI to prevent

2) Osteoporosis- Give Bisphosphonates to prevent

3) DM- If ↑ glucose give short acting Insulin

4) HTN- Treat and monitor

5) Cataract- Regular check ups and surgery

6) Suppressed Immune System- watch out for fever

7) Cushing Syndrome

8) Addison's disease

5. MACULAR DEGENERATION:

DRY WET

1. DRY AGE-RLATED MACULAR DEGENERATION

Risk factors: Increasing age, smoking, alcohol and female sex.

Symptoms:

-Gradual onset of decreased vision, initially to read and recognise faces due to loss of central field of vision.

-Central Scotoma (walking into desks)

-It is due to photoreceptors

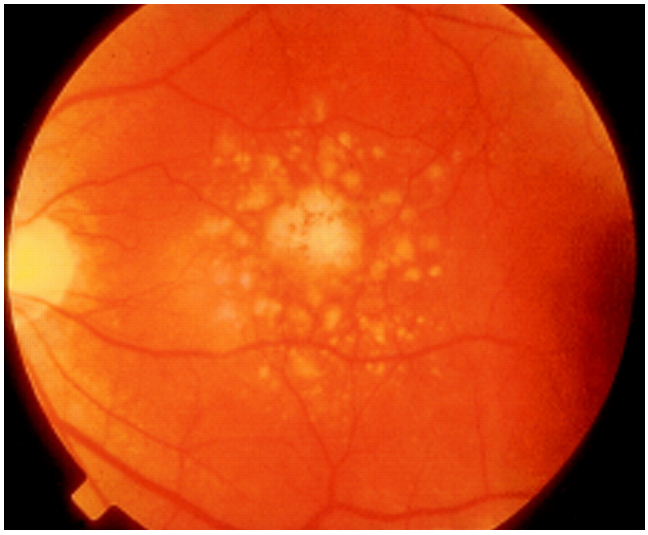
-Patient sees wavy lines -

-Macula has pigmentation geographically

-Bumps into objects

-30% inheritance

Investigation: Fundoscopy or slit lump examination you see a large area of geographical atrophy at the macula and pigmentation.



Treatment: No treatment, just reduce the risk factors and supportive treatment.

2. WET AGE RELATED MACULAR DEGENERATION

-Less common than dry macular degeneration

-There is accumulation of fluid, which decreases the membranes of the macula

-There sudden loss of vision

-On examination of the fundus you see grey or yellow plaque like membrane.

6. Transient Ischemic Attack (TIA)

-Sudden onset of symptoms such as visual loss, dysphasia, or weakness in the limbs. Symptoms resolve quickly within minutes or hours (< 24hrs).

-Risk factors include: diabetes, HTN, AF, valvular heart disease

-Amaurosis fugax-loss of vision like curtain coming down. It resolves on its own.

7. CATARACT

-This is opacity in the lens

-Usually in elderly patient

-Usually causes blurred vision

-Bilateral cataract causes gradual visual loss plus or minus frequent change of glasses

Investigation: 1) Slit lump 2) Fundoscopy

Treatment: Surgery

8. OPTIC ATROPHY (MS=Optic Neuritis, GCA)

- Optic disc is pale

- Secondary to glaucoma or retinal damage

- or due to ischemia (retinal artery occlusion)

- Toxic causes: tobacco, methanol, lead arsenic they cause amblyopic

- M.S Syphilis, external pressure on ^ nerve

9. KERATITIS

9.1 BACTERIAL KERATITIS

-Inflammation of cornea

-This is the commonest cause of Infectious Keratitis

-Bacterial Keratitis covers all organisms, which causes Keratitis in a group of bacteria.

CAUSES: The commonest cause is Pseudomonas Aeruginosa especially in those wearing contact lenses, staphylococcus aureus/epidermidis, and streptococcus.-Pseudomonas causes purulent discharge

SYMPTOMS: Increasing foreign body sensation, pain, red eyes, photophobia and reduced vision (which is not the case in conjunctivitis)

Investigation: eye corneal swab and culture.

Treatment: Antibiotics Cefuroxime eye drops

9.2 Acanthamoeba Keratitis

This is sight threatening caused by free living amoeba (a protozoa) which is found in tap water, swimming pools, fresh water and soil

-Also common in contact lens wear

-Especially those that swim with contact lenses or washing them under tap water.

Investigation: Swab and culture

Treatment: anti-septic e.g. eye drops Chlorhexidine

9.3 FUNGAL KERATITIS

-Common in contact lens wear, diabetes, immunocompromised, agricultural trauma (farmers).

-Cause is fungus aspergillus or fusarium

INVESTIGATION: swab and culture

TREATMENT: Topical Amphotericin.

9.4 VIRAL KERATITIS

- Usually follows an upper respiratory tract infection.

-Commonest cause is herpes simplex virus

-Also called herpes simplex keratits

-Presents with red eye, watering, photophobia and foreign body sensation.

-Herpes simplex keratitis causes **dendritic ulcer**

On examination of the cornea there is 1 or more linear branching dendritic ulcers with terminal bulb appearance at the ends.

-**If topical steroid are used the dendritic ulcer enlarges into large geographical ulcers which can lead to total blindness.**

-Very painful keratitis

-Therefore steroid drops are contraindicated

INVESTIGATION: Florescence examination of the cornea.

-Diagnosis is clinical.

TREATMENT: Topical Acyclovir for 2 weeks

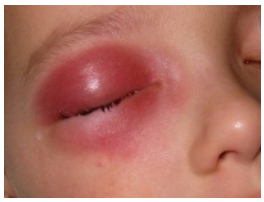
9.5 EXPOSURE KERATITIS

-This is due to inability to close the eye and the cornea is constantly exposed to air and becomes dry.

-Common with facial nerve palsy and sometimes seen after drinking a lot of alcohol

-Treatment: eye lubricant/artificial tears

Orbtial Cellulitis



10. FOREIGN BODY:

METALS OTHERS (wood, cotton, sand and grass etc.)

METALS

-Welders

-In cutting metals

Can cause penetrating trauma- Intra-ocular Foreign Body

Investigations: X-ray orbit

OTHER FOREIGN BODIES: wood especially when working in the garden, cotton, grass

Examine with florescence and remove the foreign body

11. ANTERIOR UVEITIS (Iris + Ciliary Body)

-Inflammation of the iris is called Iritis, which is part of anterior Uveitis.

-Inflammation of ciliary body and iris (anterior uvea)

-Associated with Ankylosing Spondylitis (Young male with back pain)

-Rheumatoid Arthritis, Sarcoidosis, SLE, Bechet's disease, Juvenile idiopathic arthritis and inflammatory bowel disease.

Symptoms: pain, photophobia, blurred vision, red eye,

Investigations:

-Slit lamp examination shows cells in anterior chamber with flare, pupils are small (Miosis) and irregular

-Human Leucocyte antigen HLA B27 association could be a cause (sero-negative arthritis)

Treatment:

-0.5% Prednisolone drops

-Cyclopentolate (dilating drops)

12. RED EYE:

CAUSES:

1.Viral conjunctivitis

2.Bacterial conjunctivitis

3.Foreign body

4.Closed angle glaucoma

5.Anterior Uveitis

6.Subconjunctival haemorrhage

7.Cluster headache

8.Corneal abrasion

9.Trauma

10. Scleritis

11. Episcleritis

12. Keratitis

12.1 Sub conjunctival Haemorrhage:

-No loss of vision

- Spontaneous painless bleed.

-Usually in patients with HTN or Warfarin (raises INR)

Investigations: Check for coagulation and blood pressure

Treatment: Reassurance if bloods are normal. Usually resolves in 10 to 14 days.

12.2 Viral conjunctivitis:

-Acute red eye with lacrimation

-**Watery discharge**

-This is the commonest cause of conjunctivitis

-Matting of eye lids in morning

-Photophobia and FB sensation in the eye.

Treatment: -Chloramphenicol drops to prevent bacterial infection or simply

-Topical antibiotics

12.3 Bacterial Conjunctivitis:

Acute red eye, lacrimation, foreign body sensation in the eye

-**Purulent discharge**

-Matting of lids in the morning

-Easily spreads in the family and hence members of same family may also have similar symptoms

Treatment -Chloramphenical drops or simply topical antibiotics

12.4 Corneal Abrasion:

Common in adults (usually mothers) after fingernail scratch by a baby

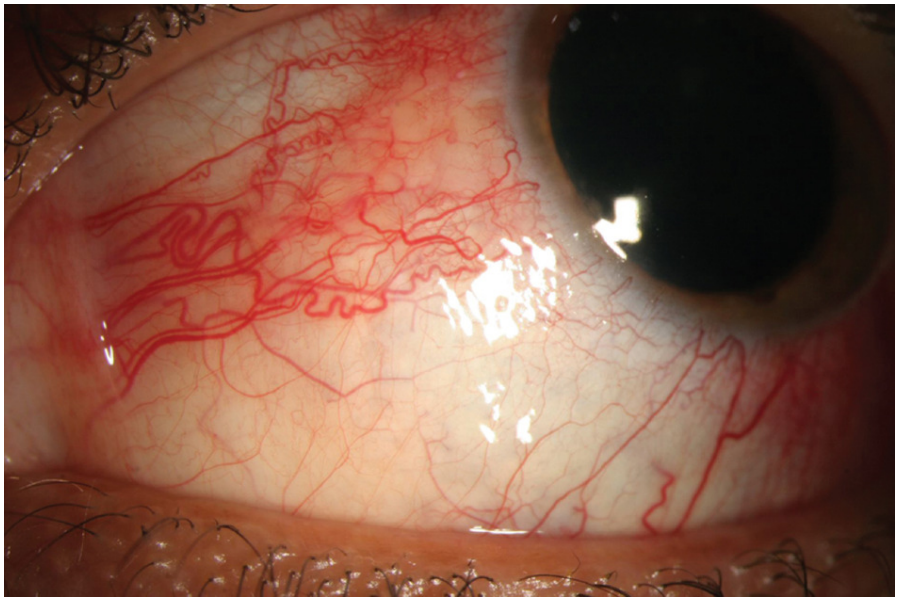
Treatment**:**

- Usually heals on its own in 2 to 3 days.
- Topical antibiotics i.e. Chloramphenical eye drops to prevent infection.

12.5 Episcleritis

- Inflammation below the conjunctiva in the Episclera, is often seen with an **inflammatory nodule.**
- Sclera looks blue below engorged vessels
- Dull eye aches with tenderness over inflamed area
- It might complicate Rheumatic fever, PAN and SLE

Rx: Topical or Systemic NSAIDS.



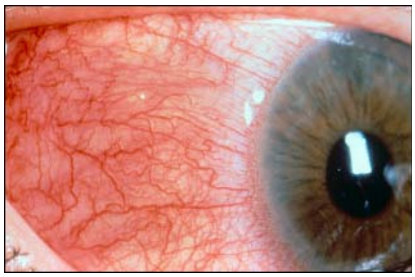
12.6 SCLERITIS

More significant pain when associated with connective tissue disease

Scleral thinning

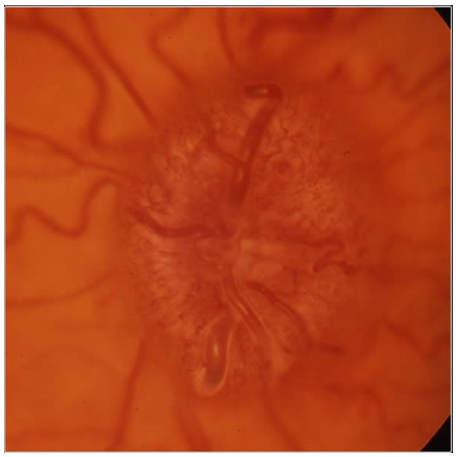
Rx Refer to ophthalmologist

Most will need oral steroid drops or immunosuppressive therapy



FUNDOSCOPY:

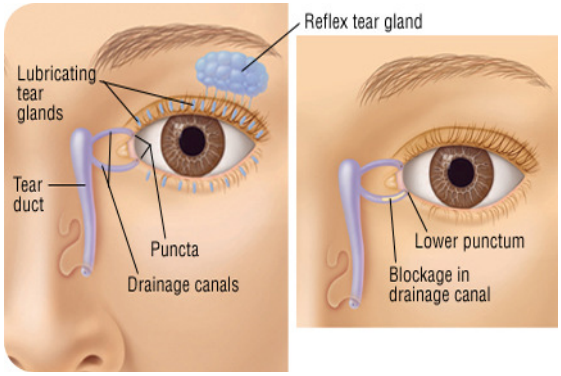
1. Optic neuritis=The optic disc is pale and patient has loss of vision
2. Retrobulbar Neuritis=optic disc is normal and patient has loss of vision
3. Papilloedema (pictured below) =Optic disc is oedematous and congested but patient has no loss of vision.

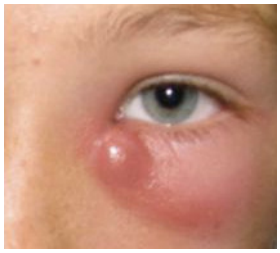


13. ACUTE DACROCYCTITIS

Inflammation of the lacrimal gland and tear ducts leading to formation of **mucocele** i.e. enlarged lacrimal gland with pus. Usually located on the nasal side of eye. When you press the swelling pus is discharged.

Treatment: Oral antibiotics





14. DRY EYES:

Sjogren's syndrome is a common disease which causes dry eyes, dry mouth and dry vagina

- Dry vagina causes dyspareunia (Pain during sexual intercourse)
- Dry mouth causes difficulty in swallowing

Inx: **Schimmers test** positive

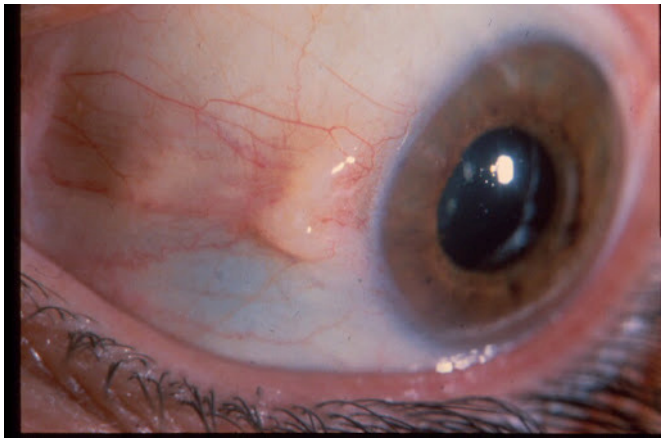
Treatment: Artificial tears (Visco tears)

15. EXTERNAL EYE PATHOLOGIES

15.1 Pinguecula

Degenerative yellow nodules on conjunctiva on either sides of cornea (typically nasal sides)

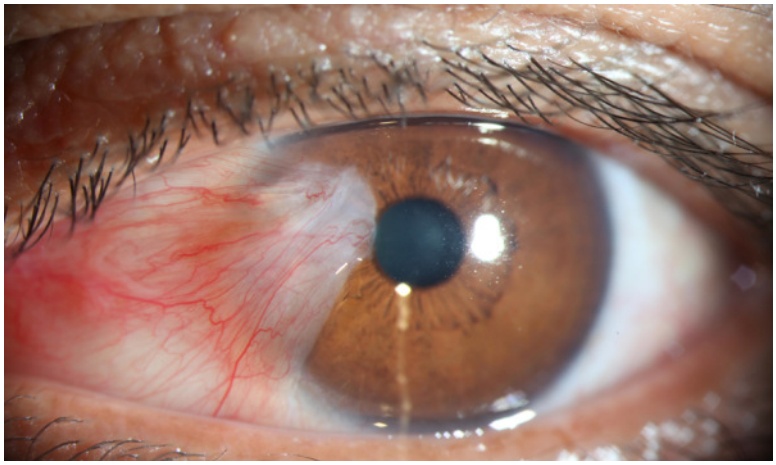
Rx: Topical steroid



15.2 Pterygium:

Degenerative wing shaped white/yellow nodules that encroach on to the corneal corners

Rx: Steroid can help but surgery may be needed



15.3 Ophthalmic shingles

This is the herpes zoster infection of the ophthalmic branch of 5th cranial nerve i.e. the trigeminal nerve. It is common in elderly due to immunocompromised state.

S/S: Pain, tingling around the eye and burning sensation on the scalp. There is a visible blistering rash.

May cause:

- Mucopurulent conjunctivitis
- Scleritis
- Epscleritis
- Visual loss
- V nerve palsy – which presents as loss of sensation on the cornea
- Keratitis
- Iritis
- Optic Atrophy

Rx: Aciclovir Po for 14 days

15.4 Retinoblastoma:

Most common primary intra ocular tumour in children.

S/S:

- Strabismus
- White pupil
- Absent red reflex

Rx: Enucleation (to take the eye out) with radiotherapy (Not usually used nowadays)

Focal procedures to preserve eye

16. PUPILS

Muscles of the Eye: Superior Oblique is supplied by the 4th cranial nerve and the lateral rectus by the sixth. The rest are innervated by the 3rd Nerve

LIGHT REFLEX (Pupillary Reflex)- Direct and Consensual

Afferent – Optic nerve injury – absent direct reflex

Efferent – Occulomotor Nerve injury

Causes of fixed dilated pupils:

- 3rd CN injury
- Mydriatics (dilating eye drops)
- Trauma to iris
- Acute Glaucoma

1. Adie Pupil

→ Large Pupil

→ Poor accommodation

→ Degeneration of the parasympathetic ganglion

2. Argyll Robertson Pupil

→ Small and irregular pupils poorly react to light but good accommodation.

→ Commonly caused by Syphilis.

Pupils of different sizes = anisocoria

17. LENS

ERRORS OF REFRACTION

1) Myopia (short sightedness)

Concave lenses for correction

2) Astigmatism: defect in the curvature of lens and the cornea

Cylindrical lenses for correction

3) Hypermetropia: (long sightedness)

Convex lenses for correction

4) Presbyopia: It usually starts at the age of 40. In elderly lens becomes stiffer – long sightedness

Loss of accommodation.

18. The EYE in DM

Structural changes: Accelerates the formation of cataract

Retinopathy:

- Background Retinopathy: Micro aneurysms, dot & blot haemorrhage and hard exudates
- Pre-Proliferative: Micro aneurysms, dot & blot haemorrhages plus soft exudates (aka cotton wool spots)
- Proliferative Retinopathy: new vessel formation
- Maculopathy: leakage close to haemorrhage exudates^ macula

Rx:

1. Good control of DM, Heart disease, Renal disease, Increased lipids (cholesterol)
2. Photo coagulation for maculopathy & proliferative retina

19. BLEPHRITIS:

Inflammation on the lid margins. Very common

- This is chronic lid inflammation.

Symptoms

- Burns, itching, FB sensation
- Hard + brittle scales

- An external style may develop if follicles infected

20. Tears and Lacrimation

- Keratoconjunctivitis Sicca due to decreased tear production
- Sjogren's syndrome
- Mumps
- Sarcoidosis
- Lymphoma
- Leukaemia
- SLE
- Scleroderma

Investigation: Shimmer's test

Treatment: artificial

21. CHEMICAL INJURY

The main treatment is irrigation of the eye with normal saline.

Resource start date	2013-06-26 09:46
Resource end date	2023-06-27 09:46

Back

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Resource view

Resource name	Trauma and orthopaedics
Resource description	Trauma and orthopaedics
Resource content	

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ORTHOPAEDICS & TRAUMA

OVERVIEW OF TOPICS IN ORTHOPAEDICS:

A. **Upper Limb Injuries**
   1. **Shoulder Dislocation**
   2. **Clavicle Fracture**
   3. **Acromio-clavicular Dislocation**
   4. **Sterno-clavicular Dislocation**
   5. **Fracture neck of Humerus**
   6. **Fracture shaft of Humerus**
   7. **Supracondylar Fracture**
   8. **Elbow Dislocation**
   9. **Monteggi Fracture-Dislocation**
   10. **Galeazzi Fracture- Dislocation**
   11. **Nightstick Fracture**
   12. **Colle's Fracture**
   13. **Smith's Fracture**
   14. **Barton's Fracture**
   15. **Greenstick Fracture**
   16. **Scaphoid Fracture**

17. **Perilunate Dislocation**
18. **Mallet Finger**
19. **Bennett's Fracture**
20. **Game Keeper's Thumb**

B. **Lower Limb Injuries**

1. **Dislocated Hip**
2. **Neck of Femur Fracture**
3. **Fracture shaft of Femur**
4. **Patella injury or fracture**
5. **Dislocation of Patella**
6. **Dislocation of Knee**
7. **Ligamental Injury**
8. **Meniscal Injury**
9. **Tibia or Fibula Fracture**
10. **Ankle Dislocation**
11. **Achille's Tendon Rupture**
12. **Quadriceps Tendon Rupture**

C. **Limping Child**

1. **Perth's Disease**
2. **Slipped Upper Femoral Epiphysis**
3. **Septic Arthritis**

D. **Sprains & Soft Tissue Injuries**

GENERAL APPROACH TO ORTHOPAEDICS:

1. **Mechanism** (FOOSH=fall onto outstretched hand, Epilepsy, direct trauma, electrical shock)
1. **Age** Some fractures are more common in children and others are more common in adults
1. **Clinical presentation**: pain, swelling, deformity or open wound and loss of function
1. **Which Nerve is damaged**
1. **Which Artery is damaged**
1. **Investigation**: X-ray
1. **Treatment:**

- **Conservative:** Includes Plaster of paris(POP), sling, splint,
- **Operation**: Open reduction and internal fixation (ORIF)
- **Dislocation:** Manipulation or reduction

A. UPPER LIMB INJURIES

1. Shoulder dislocation It refers to acute dislocation of glenohumeral joint. The shoulder joint is a ball and socket joint

Classification & Mechanism:

1. **Anterior shoulder dislocation**. More than 95% are anterior dislocation. often follows a fall or other mechanism where there is force rotation in abduction.
2. **Posterior shoulder dislocation:** Uncommon. Usually associated with electric shock or Epileptic seizure.

Signs & Symptoms:

Anterior dislocation:

- Pain & absence of active movement
- Abnormal shoulder contour

- Absence of sensation over the badge area indicates axillary nerve damage/compression.
- Absence of hand pulses suggest compression of the axillary artery and need for urgent reduction.

Investigations: Shoulders X-ray

Management: 1. Reduce immediately in ED

2. Immobilize in a collar & cuff

2. **Clavicle fracture:**Commonly it's a mid clavicular fracture and most often treated conservatively.

Mechanism:

- Fall on to outstretched hand (FOOSH)
- Fall directly onto the point of the clavicle or shoulder

Signs & Symptoms:

- Pain, deformity, occasional bruising
- Subclavian artery can be damaged by bone fragments resulting in an ischemic Stroke
- Neurological sign- Brachial plexus damage

Investigations: X-ray of clavicle

Management: 1.>95% are treated conservatively

2. A broad arm sling to support the arm.

3. Acromio – clavicular dislocation (ACJ joint)

Mechanism: Often follows rolling onto the shoulders impact on to anterior shoulder

Signs & Symptoms:

1. Pain and deformity at lateral end of clavicle.
2. Outer end of affected side is more prominent then normal side.

Investigations: X-ray of ACJ

Management: Broad arm sling for 3 weeks.

4. Sterno- clavicular dislocation(SCJ joint)

Mechanism: Fall or blow to the front shoulder; e.g: rugby players. Occasionally may be spontaneous.

Signs and symptoms: Localized tenderness and asymmetry of the inner ends of the clavicle.

Investigations: X-ray of SCJ

Management:

1. **Acute vascular problems/ airway obstruction require immediate intervention.**
2. **A broad arm sling for 2- 3 weeks.**

5. Proximal Humerus fracture: e.g neck of humerus fracture

Common in elderly/ osteoporotic patients.

Mechanism:

- Fall on to outstretched hand or face directly onto shoulder
- Often occur in osteoporotic bone
- High energy impact in young

Signs & Symptoms:

1. Pain, deformity and swelling
2. Unable to move the shoulder
3. Axillary nerve and artery may be damaged

Investigations: X-Ray. Check for pathological lesions

Management: Depends on the type of fractures.

1. **Non displaced/ minimal displaced fracture, treat with collar & cuff and mobilize**
2. **Displaced anatomical neck fracture, treat treat collar & cuff**

6. Fracture of Shaft of Humerus Common in adults.

Fracture are considered and described as being of the proximal, middle or distal third.

Mechanism:

1. FOOSH
2. Direct blow to the arm

**Low energy injury may suggest pathological fracture like in osteoporosis and bone metastasis.

Signs & Symptoms:

1. Pain, deformity & swelling
2. Wrist drop & sensory loss over the 1st dorsal web space (Radial Nerve damage)

Complications: Non-union

Investigations: X-ray of the humerus

Management:

1. **Displaced, comminuted/ angulated fractures require ORIF (open reduction & internal fixation or if options include plates & screws & intramedullary mails.**
2. **If not displaced or minimally displaced then conservative Management.**

7. Supracondylar fracture

Transverse fractures of the distal third of the humerus.

Commonly seen in children, less frequently in adults.

Need to be assessed & addressed promptly to prevent serious complication.

Mechanism: FOOSH in a child is the typical presentation

Signs & Symptoms:

1. Pain & swelling
2. The child tends to hold the affected arm with the other.
3. **Check for pulses & nerve!**

Arteries: Presence & quality of radial, ulnar & brachial arteries. Absence/ diminished findings suggest compression of the brachial artery may leads to ischemia. **It is an emergency.**
Sensation & Motor Function: Check all components of the median & radial nerve.

Investigations: X-ray of the elbow

Management: 1. Undisplaced: Backslab plaster

2. # > 50% displacement : Theatre

8. ELBOW DISLOCATION Occurs in children and adults

Mechanism:

1. FOOSH
2. direct fall on to the elbow.
3. Common in young children who after being swung by parents or the child falls down while being held by the hand by a parent.

Signs & Symptoms:

1. Pain & absence of movements
2. Abnormal elbow contour

Investigations: X-ray of the elbow

Management: Reduction

9. Monteggia fracture-disclocation:

This is a fracture of the proximal ulnar with dislocation of the radial head.

Mechanism: FOOSH with forced pronation

Signs & Symptoms:

1. Elbow pain and swelling
2. Elbow flexion and forearm rotation are limited and painful
3. Motor branch of radial nerve commonly damaged, sensory branch not commonly damaged but should also be checked

Investigations: X-ray of the entire forearm (elbow to wrist)

Management: ORIF

10. Galeazzi fracture-dislocation Solitary fractures of the distal one third of the radius with accompanying subluxation or dislocation of the distal radioulnar joint (DRUJ). Sometimes called reverse Monteggia fracture.

Mechanism: Fall on to an extended, pronated wrist

Investigations: X-ray of the entire forearm (elbow to wrist)

Management:

1. **Admit and place in a above elbow back slab plaster, elbow at 90 degrees and elevate the limb on pillows**
2. **ORIF**

11. Nightstick Fracture Isolated mid shaft ulnar fracture

Mechanism: usually caused by a direct blow to the ulnar bone, classically if someone receives a blow from an object whilst raising their arm in defence

Signs & Symptoms: point tenderness over the ulnar shaft, and forearm swelling

Investigations: X-ray the entire forearm (elbow to wrist)

Management:

- **Non-displaced or minimally-displaced fractures Conservative (posterior splint)**
- **Marked displacement or angulation ORIF**

12. Distal Radius Fracture (Colle's fracture) Occurs most commonly in osteoporotic bones. It refers to dorsal displacement, radial displacement and impaction occurring within 2.5cm of the wrist joint.

Mechanism:

1. FOOSH: forced dorsiflexion of the wrist may be bilateral
2. In the young it is usually caused by high energy injuries
3. Check for other upper & lower limb injuries

Signs & Symptoms:

1. Dinner fork deformity with pain on attempted wrist movement.
2. Check for median nerve compression (carpal tunnel syndrome)
3. Look for signs of new onset carpal tunnel syndrome

Investigations: X-ray of the wrists (shows dorsal displacement, dorsal angulation and radial shortening)

Management:

1. **Uncomplicatied fracture do Closed manipulation**
2. **Complicated fracture, especially if there is nerve injury do ORIF**

13. Distal Radius Fracture (Smith's fracture) Often described as reverse of colle's #.

Mechanism: Fall on to the back of the and with wrist flexed.

Investigations: X-ray of the wrist (shows volar/ palm displacement)

Management:

1. **Uncomplicated fracture Close manipulation under LA**
2. **Complicated fracture ORIF**

14. Barton's Fracture Volar distal radial fracture which extends into radio carpal joint.

Investigations: X-Ray

Management: ORIF

15. Distal Radial fracture in children/ Greenstick Fracture

Very common fracture of childhood. Greenstick fracture is an incomplete fracture.

Mechanism:

- Usually an indirect injury following FOOSH
- Occasionally caused by a direct trauma

Signs & Symptoms:

1. Pain & dinner fork deformity
2. Check for median nerve compression
3. Look for signs of carpal tunnel syndrome
4. Examine the elbow & fingers fully

Investigations: 1. X-ray wrist 2. Obtain full length and forearm & elbow if in doubt.

Management:

1. **Non displaced/ non angulated fractures, put in plaster cast for 3-4 weeks (backslab for 1st 2-3 days)**
2. **If symptomatic or angulation do MUA (manipulation under anaesthesia.)**

16. Scaphoid fracture: Meta carpal bone fracture often occurring in young adults.

Mechanism: FOOSH

Signs & Symptoms:

1. Tender scaphoid tubercle
2. Tender anatomical snuff box
3. Pain on movement of the thumb
4. Pain on deviation of wrist over the scaphoid region
5. Check pulses from distal to proximal for Radial artery damage

Investigations:

1. **Scaphoid X-ray (7% identified on 1st X-Ray, 20% are identified on X-Ray at 10-14 days,)**
2. **Bone isotope scan/MRI if not seen on X-ray**

Management:

1. **Severely displaced fracture - immediate reduction and ORIF**

2. **Non displaced fracture - Scaphoid cast for 8-10 weeks**
3. **Non visible fracture but significant clinical presentation - put scaphoid cast & review in 7 – 10 days**

1. **Still not visible - MRI/CT scan**

Complications:

1. Non- union (5-10% undergo non union leading to Avascular necrosis)
2. AVN (Avascular necrosis)
3. Osteoarthritis

17. Peri-lunate dislocation Lunate is m/c dislocated carpal bone

Mechanism: FOOSH

Signs & Symptoms:

1. Pain at the wrist
2. Signs of acute median nerve compression caused by the bone protruding into carpal tunnel.

Investigations: X-ray wrist

Management: 1) MUA 2) K-wire reduction

18. Mallet Finger

This is rupture of the extensor digitorumprofundus resulting into failure to extend the distal interphangial joint.

Common in hotel workers, sustained when tucking in bed sheets

Investigations: X-ray to r/o avulsion fracture

Management: Mallet splint

19. Bennett's fracture: Fracture of the base of the thumb or 1st metacarpal bone due to thumb hyperextension

Investigations: X-ray

Management:ORIF

20. Gamekeeper's thumb

Tear of the ulnar collateral ligament of the thumb at MCP joint due to forced abduction of the thumb

Management: Surgery

B. LOWER LIMB INJURIES

1. Dislocated Hip

a. Normal Hip

Causes: Requires high energy impact e.g. road traffic accident

b. Total Hip Replacement

Causes:

1. Problem with prosthesis (loose components)
2. Problem with patient (poor compliance not following instruction

Mechanism:

- Occurs in elderly after fall, twist or low energy injury
- Pain & inability to bear weight
- On/ examination the limb is typically shortened

Investigations: Pelvis X-ray

Management: Admit for reduction in theatre under general anaesthesia

2. Neck of Femur Fracture Patients with NOF commonly present with co-existing multiple co-morbidities

1. Majority due to osteoporosis
2. Metastatic deposits
3. In young due to high energy impacts e.g. road traffic accidents

Mechanism: Follows a fall

Signs & Symptoms:

1. Pain over the hip & groin
2. Affected limb is often shortened & externally rotated
3. Inability to raise straightened leg
4. Check for sensation in the foot & adequate pulses
5. Circumferential artery and sciatica nerve are commonly damaged.

6. Full examination is mandatory

Investigations: X-ray pelvis & femur
Management: Operative fixation (ORIF) or hip replacement

3. Femur Shaft Fracture

Mechanism: Usually due to a fall or road traffic accident

Signs & Symptoms:

- Swelling deformity, pain
- Femoral artery and femoral nerve are commonly damaged.

Investigations: X-ray

Management: Thomas splint

In children commonly this is the only treatment.

In adults - Thomas splint followed by ORIF and Intramedullary Nail

4. Patella Injury or Fracture

Mechanism:

1. Typically occurs after direct blow e.g – fall on to the knee
2. Avulsion fracture (from muscle contraction)

Signs & Symptoms:

- Pain over the patella
- Inability to raise straightened leg/ extend knew
- A palpable gap felt at the level of the fracture

Investigations:

1. **X-ray of patella (knee joint)**
2. **Patella fracture can be: vertical,; non displaced horizontal; comminuted**

Management:

1. **Hold in cylinder cast or cricket cast**
2. **Vertical & non displaced-horizontal fracture -> cylinder cast for 6 weeks**
3. **Displaced horizontal —> ORIF**

5. Dislocation of Patella

Mechanism:

- Typically in young females as a congenital anomaly
- Also direct trauma

Signs & Symptoms:

- The patella typically displaces laterally
- Patients may present with dislocated patella or after it has reduced

Management:

1. **Reduce by pushing the patella medially while the leg is straight (extended knee) with Entonox analgesia**
2. **Cylinder plaster cast for 3 weeks followed by physiotherapy.**

6. Dislocation of the knee

Significant injuries. May occur from high/low energy mechanism

1. **Anterior dislocation:** Due to severe hyperextension
2. **Posterior dislocation:** Direct injury to the front of the tibia Check for presence of distal foot pulses (difficult to feel popliteal artery pulse)

Management:

1. **Knee should be reduced asap**
2. **Give Entonox for initial pain relief**
3. **Morphine IV**
4. **Always check neurovascular deficit (both before & after reduce)**
5. **Treat in a loose above knee back slab and admit**

*Ligamental & Meniscal Injuries:

- Common in sportsmen, also in older less active peo
- Extremely painful, even in absence of fracture

7. Ligamental Injuries

Mechanism: Twisting, or foot got caught while running

Types of Ligament Injury:

i) Anterior Cruciate Ligament: Anterior draw test positive

- Associated with medial collateral ligament or medial meniscus injury

ii) Posterior Cruciate ligament: Posterior drawer test positive

- Associated with middle/ lateral collateral ligament injury

iii) Lateral collateral ligament: Varus stress test positive

- Fracture of head of fibula

iv) Medial collateral ligament: Valgus stress test positive

- Associated with medial meniscal and ACL injuries
- 'unhappy triad' = ACL, MM, MCL

Signs & Symptoms:

- Popping sensation, cracking sound, immediate pain or no swelling
- May/may not able to bear weight
- Effusion – detectable, mild, undetectable
- Intense pain

Investigations: X-ray to exclude fracture.

Management: Analgesia & elevation
If minor (fully weight bearing) —> mobilize
If major (non weight bearing) —> use crutches & surgical repair.

8. Meniscal injuries

Mechanism:

- Result of twisting injuries with flexed knee
- Common in Footballers, typically in men
- Often history of leg giving away

Signs & Symptoms:

- Pain and difficulty in bearing weight
- Presence of locked knee – not fully extended
- Effusion
- Joint line tenderness

Investigations: X-rays, MRI or Arthroscopy (especially if there is locked knee)

Management:

- **Knee strapping ± crutches**
- **Locked knee —> admission & analgesia**

9. Tibia & Fibula Fracture

Mechanism: Twisting force or direct force to the leg, or any mechanism that presses lower ligament

Signs & Symptoms: Pain, swelling, deformity.

Management: ORIF

10. Ankle Dislocation

- This is an **Orthopaedic Emergency**
- Always associated with ankle fracture

Mechanism: Often follows serious traumatic injuries e.g heavy fall, direct blow
Joint is displaced in some degree

Signs & Symptoms:

- Foot is cold and pale
- Impalpable pulses
- Diagnosis is **clinical**

Management: Reduction before X-ray (reduction can take place in A&E)

11. Achilles Tendon Rupture

- Lower limb tendon problem
- Common injury

Mechanism:

- Follows sudden muscular contraction e.g jumping, pushing off
- Patients reports feeling of clicking back of leg or heard a crack as tendon ruptures.

Signs & Symptoms:

- Pain and swelling
- Poor walking with inability to stand on toes
- Visible gap palpable at tendon.
- Simmond's Test Positive – patients lies prone on table with feet hanging off edge. Positive if no movement of foot on squeezing corresponding calf.

Investigations:

- **Plain X-ray ankle to rule out an avulsion fracture**
- **USG shows extent of injury**

Management:

- **Conservative** - **Cast in plantar flexion**
- **Invasive** - **Surgical repair**

12. Quadriceps Tendon Rupture

- Rupture of quadriceps tendon
- Usually 60 – 70 yrs
- Associated with hyperparathyroidism, diabetes, renal fx, arthritis, gout
- Also common in patients on steroids or those who abuse steroid especially sportsmen.

Mechanism: Strong contraction of quadriceps muscle

Signs & Symptoms:

- Associated with intense pain ± haemarthrosis
- Loss of extension of knee
- Walking impossible
- Gap superior to patella is palpable

Investigations: Plain knee X-ray

Management: Admit for early open repair

C. THE LIMPING CHILD

1. Perth's Disease

- Form of aseptic necrosis of femoral head
- Often due to disruption of blood supply to femoral epiphysis
- Common 3-8 years
- M:F 5:1
- Mostly unilateral, bilateral in 10%,

Signs & Symptoms:

1. Limp and painful gait
2. Pain referred to knee, thigh (middle side) on exam
3. Hip abducted &internal rotation are limited
4. One leg may be shorter than the other
5. Muscle wasting

Investigations:

- **X-ray hip**
  - **Fermoral epiphysis appears smaller on affected side**
  - **Widening of joint space**
  - **Femoral head sclerosis**
- **X-ray is not always enough, can do CT**

Management: Refer to Orthopaedics

Initial Management: Bed Rest & Analgesia

2. Slipped upper femoral epiphysis (SUFE):

- Commonest hip disorder in adolescents
- Boys: 10-13 years old ; Girls: 11-14 years
- M:F 3:1;
- More common in obese boys

Cause: Unknown

- Seen during rapid growth (during this time plates are rapidly growing so prone to injuries as it is soft)
- Hx of trauma in 50%
- 60% bilateral

Signs & Symptoms:

- Pain and limping
- Not localised to the hip
- Shortening of limb & external rotation.

- Pain & limited internal rotation on examination

Investigations: X-raysshow

- **Widening of epiphysis**
- **Displacement of femoral head**
- **Epiphysis appears smaller due to post slippage.**

Management: Prevent further slippage with conservative treatment and maintain function

Definitive management is surgical pinning

3. Septic Arthritis

- Common in children less than 2 years
- Acute pain in the joint, fever and limping
- Joint is hot swollen
- Reduced joint movement and unable to weight bear

Investigations:

- **X-ray**
- **Joint aspiration with microscopy, culture & sensitivy**
- **Blood culture if fever.**

Management of Sprains and Soft Tissue Injuries

1. **Hand sprains -** immobilize in high arm sling for 2-3 days to reduce the swelling.
2. For **ankle sprain -** give crutches due to pain and adviSe to elevate the leg.
3. For **whiplash injury -** physiotherapy. Nowadays they do not use neck collar

OVERVIEW OF TOPICS IN TRAUMA:

A. **Chest Trauma**
B. **Abdominal Trauma**
C. **Urological Trauma**
D. **Head Injury**
E. **Wound Management**
F. **Burn Management**

CHEST TRAUMA

1. **Traumatic Diaphragmatic Rupture**

Mechanism: May occur after blunt or penetrating trauma

Presentation:

Abdominal content usually moves into the thoracic cavity. This may be apparent on insertion of NG tube, which is usually coiled in the chest.

Investigations: Chest x-ray - will show elevated diaphragm. CT is more definitive.

Management: NG tube to decompress stomach followed by surgical repair.

1. **Oesophageal rupture**

Causes:

- Post endoscopy (usually in difficult endoscopy) - Localized neck pain/retrosternal chest pain
- Trauma

Presentation:

- **Boerhaave's syndrome - Mackler's triad**: vomiting, followed by severe chest pain (usually retrosternal) and surgical emphysema
- Low grade pyrexia
- Pale clammy tachycardia
- Hypotension
- Pleural effusion
- Subcutaneous (surgical) emphysema in the neck or chest

Investigations: Chest x-ray - shows free gas in the mediastinum

CT Scan with oral contrast

Management:

- **Take care of ABC's, keep NBM, give antibiotics (cefotaxime + metronidazole)**
- **Definitive management can be conservative or surgical**

1. **Massive Haemothorax**

Haemothorax and pneumothorax often coexist together (haemopneumothorax)

Massive haemothorax will cause trachea shift, hypoxia, shock, shortness of breath and chest pain

Presentation:

- Trachea shift, hypoxia, shock, shortness of breath, and chest pain
- Tachycardia, tachypnoea, reduced chest expansion, dullness to percussiom, decreased breath sounds, shock

Investigations: Chest x-ray initially. CT scan is the investigation of choice.

Management: Take care of ABCs and insert chest drain. If there is massive ongoing bleeding after insertion of chest drain

1. **Simple Pneumothorax**

Air in pleural space without aggressive increase in intrathoracic pressure

Presentation:

- Chest pain
- Shortness of breath, tachycardia
- Affected side has reduced chest expansion, hyperresonance, reduced to absent breath sounds
- Trachea is central

Investigations: Chest x-ray

Management: Traumatic simple pneumothorax regardless of size requires chest drain insertion.

1. **Open pneumothorax**

Open thoracic wound with breach of parietal pleura, leads to air in pleural space

Presentation:

- Ipsilateral chest pain
- Open chest wound
- Same as simple pneumothorax

Investigations: Chest x-ray

Management:

Initial treatment: application of sterile occlusive dressing to cover whole wound. Tape is secured on three sides to make a one way valve.

Then insert chest drain.

1. **Tension pneumothorax**

Presentation:

- Chest pain
- Distressed patient, tachypnoea with cyanosis, profuse sweating, tachycardia and hypotension
- Affected side has reduced chest expansion, hyper-resonant on percussion, reduced to absent breath sounds
- Trachea is deviated to the contralateral side
- Distended neck veins

Management: Do IMMEDIATE needle thoracocentesis, followed by chest drain insertion as soon as possible

NB. This is a clinical diagnosis, no need for investigations to confirm before management.

1. **Rib Fractures**

A. **Single rib fracture**

Isolated fracture of a single rib after trauma is uncommon, suspect multiple fractures and exclude injuries to the underlying structures

A. **Multiple Rib Fractures**

- Fracture to the lower ribs (10-12) should raise suspicion of injuries to the spleen or liver
- Fracture to the middle ribs (4-9) are commonly fractured in blunt chest trauma and usually associated with pneumothorax, haemothorax and pulmonary contusion

NB: The presence of subcutaneous emphysema suggests pneumothorax

Clinical Features: Visible deformity, tenderness, bruises

Investigation: Chest x-ray

Management:

- **If uncomplicated rib fractures, only conservative management is needed, just prescribe oral analgesia**

1. **Flail chest**

Occurs when two or more ribs are fractured in two or more places. The flail segment is paradoxically drawn in during inspiration and drawn out during expiration. This causes inadequate ventilation.

Presentation:

- Severe chest pain
- Paradoxical chest movement of the flail segment
- Hypoxia

Investigations: Chest x-ray, CT of chest

Management: Refer to cardiothoracic surgery for operative fixation. Ensure adequate oxygenation – patient may require CPAP or require mechanical ventilation.

1. **Cardiac tamponade**

Mechanism: Caused by either penetrating or blunt chest trauma

Presentation:

- **Beck's triad:** Muffled heart sounds, distended neck veins, hypotension
- Tachycardia
- Shock

Investigations: Echocardiogram

Management: Pericardiocentesis

1. **Thoracic Aortic Dissection**

Presentation:

- Sudden of severe tearing chest pain radiating to interscapular area
- Stroke or syncope may occur
- Abdominal pain due to mesenteric ischaemia
- Paraplegia due to spinal ischaemia
- Collapse
- On examination: shock, hypotension, tachycardia
- Different blood pressure and pulses in each arm

NB: Abdominal pain radiating to back is always abdominal aortic dissection until proven otherwise.

Investigations: Chest x-ray shows widened mediastinum. Contrast enhanced CT chest is investigation of choice.

Management: Refer to cardiothoracic surgeon immediately. Transfer to ITU. Definitive treatment is open repair of aneurysm.

B. ABDOMINAL TRAUMA

Causes:

1. Penetrating knife/ gunshot wound
2. Blunt blows/ RTA due to deceleration

Organs affected:

Solid Viscera:

1. Liver
2. Spleen
3. Mesentary artery
4. Duodenum/Small Bowels
5. Pancreas

1. Liver

- Occurs in penetrating or blunt injuries
- Common in major rib fracture especially lower ribs on the right side.

Presentation:

- Unresponsive hypotension
- Abdominal distention
- Pain & peritonitis in conscious patient

Investigations: CT is investigation of choice. USS if not CT scan in the options.

Management: Emergency laparotomy

Definitive: Transplant

2. Splenic Rupture

In penetrating or blunt injury. It is also associated with fractures of the left lower ribs.

Presentation:

- Unresponsive hypotension, abdominal distention

- Pain & Peritonitis
- **Left flank bruising** (most important)

Investigations:

- CT scan, Ultrasound of the abdomen
- Definitive: Splenectomy

3. Major Vessel Laceration:

Mechanism: Commonest in deceleration injuries. Mesentary arteries are commonly damaged by the belt.

Presentation:

- Unresponsive Hypotension
- Abdominal distention
- Back & Flank pain
- Flank Bruising

Investigations: CT scan is diagnostic
Management: Emergency laparotomy

4. Duodenal Rupture: mesenteric vascular injury
Mechanism: Compression seat belt injury
Presentation:

- Positive DPL = diagnostic peritoneal lavage (bile stained)
- Features of Peritonitis
- Associated with pancreatic or lumbar injury

Investigations: CT scan diagnostic

Management: If part of multiple injuries then put surgical staples
Definitive Management is Primary repair

5. Small Bowel Laceration:
Mechanism: Caused by sharp penetrating trauma
Presentation:

- Features of peritonitis
- Features of sepsis

Management: Same as duodenal rupture

6. Pancreatic Disruption:
Mechanism: Deceleration injury
Presentation: Back pain, flank bruising,
Investigations: CT scan
Management: Analgesia & drainage of pancreatic collection

7. Abdominal Aortic Dissection
Presentation:

- Sudden of severe tearing abdominal pain radiating to back
- Stroke or syncope may occur
- Abdominal pain due to mesenteric ischaemia
- Paraplegia due to spinal ischaemia
- Collapse
- On examination: shock, hypotension, tachycardia
- Radiofemoral delay
- Weak or unpalpable femoral pulses

NB: Abdominal pain radiating to back is always abdominal aortic dissection until proven otherwise.

Investigations: Chest x-ray shows widened mediastinum. Contrast enhanced CT chest is investigation of choice.

Management: Refer to cardiothoracic surgeon immediately. Transfer to ITU. Definitive treatment is open repair of aneurysm.

INDICATIONS FOR EMERGENCY LAPARATOMY:

1. Unexplained shock
2. Clinical peritonitis
3. Positive DPL
4. Evisceration
5. Gun shot wounds
6. Bleeding PR, penetrating trauma, stomach.

INDICATION FOR CT ABDOMEN.

1. Abdominal pain and vomiting with hypotension.
2. Signs of peritonism
3. Severe abdominal pain after road traffic accidents

**For abdominal trauma It's to do mandatory chest X-ray & Pelvic X-ray

C. UROLOGICAL TRAUMA:

Causes: Penetrating or blunt injury

Most Common Causes:

1. Restrained passengers in RTA high speed (Compression of kidneys)
2. Crush injuries
3. Vehicle vs pedestrian injuries
4. Abdominal stab wounds
5. Blunt assault Renal contusion

Investigations:

Mandatory

- **Chest & pelvic X-ray**
- **Urinalysis for blood (looking for hematuria)**
- **PR Exam for prostate**

Optional

- **CT, Abdomen & Pelvis is diagnostic method of choice. Therefore, CT scan for diagnosis**
- **Intravenous Urography (IVU) is definitive for kidney function**

1. Renal Injuries

- 5-10% of all abdominal injuries affect kidney
- Most common causes in the urinary tract are RTA, sports injuries, falls, assaults

Presentation:

- Microscopic haematuria on urine dipstix
- Loin & back pain
- Hypotension

Delayed Presentation: Flank Pain, fever due to infected hematoma

Investigations: CT scan is diagnostic Contrast extravasation on CT or IVI

Management:

May be conservative or surgical depending on severity of the injuries

Laparotomy indicated if:

1. **There are penetrating injuries with signs of shock or peritonitis**
2. **Blunt injuries with haemodynamic compromise despite fluid resuscitation**

Complications of Renal injuries:

1. Secondary Heamorrhage
2. Perinephric abscess
3. Fistula development
4. Ureteric injury

2. Urethral Injuries

Mechanism: Major pelvic fracture from RTA

Presentation:

- Supra pubic pain
- Blood at urethral meatus
- Inability to void urine
- Perineal swelling/bruising
- High riding prostate on Per Rectal examination

Investigations: Retrograde urethrogram – shows extravasation of contrast

Management:

****In emergency do NOT attempt Urethral Catherisation****

Do supra pubic catherisation

Definitive Management:

- **Incomplete laceration** - **conservative Management with urethral catheters**
- **Complete transection** - **Primary repair with catheter**

D. HEAD INJURY

Common causes:

1. Non accidental injury
2. Shake baby syndrome especially in premature babies
3. Epilepsy
4. Falls
5. RTA
6. Sports – rugby

NB. All can cause intracranial bleed which can cause dilated pupils due to 3rd nerve palsy

1. Skull Fracture

Base skull fracture - Rhinorrhea, otorrhea, hemotympanum, battle sign (mastoid bruising), raccoon eyes/panda eyes (bruising around eyes)

Depressed skull fracture - Indication for CT

Vault skull fracture - Crack, needs a CT scan

2. Cerebral Contusion

Focal intraparenchymal oedema

Located at the site of impact

3. Extradural Hematoma

Head injury & immediate LOC = Extradural hematoma

- Hx of trauma
- Commonest injury causes **immediate LOC**
- Due to disruption of **middle meningeal artery**
- Lucid interval
- Bruise in the temple area
- Rapid deterioration in consciousness
- Lucid interval is usally minutes to hours., and not days or weeks.

If weeks or days then subdural haematoma is the diagnosis.

Investigations: CT scan

Management: Burr hole ~ Emergency evacuation of hematoma to relieve increased intracranial pressure

4. Subdural hematoma

- Common in alcoholics, recurrent falls
- May be no history of trauma because trauma may have happened weeks ago and patient has forgotten about it
- Fluctuating LOC
- ± cognitive impairment
- Progressive confusion
- Subdural Hematoma – acute < 24 hours

Chronic > 24 hours

Management: Evacuation of hematoma

INDICATIONS FOR CT SCAN HEAD IN HEAD INJURY

1. GCS < 13 at initial assess
2. GCS 14 if 2 hours after injury
3. Any neurological deficit
4. Depressed skull fracture
5. Base skull fracture
6. Post traumatic seizures
7. Vomiting post trauma ≥3 times
8. Amnesia

CLASSIFICATION OF HEAD INJURY:

GCS 13 -15 = Mild (observe 24 hours)

9 – 12 = Mod (CT scan)

≤ 8 = Severe = **call anaesthetist and intubate**

NB:

1. **Alcoholic with head injury, even of GCS 15/15 observe for 24 hours**

2. **If no supervision at home, admit**

E. WOUND MANAGEMENT

Full course of Tetanus Vaccination:
3 Vaccines in infancy + 2 boosters

Scenarios of Wound Management:

1a) Fully immunized & wound is clean: No tetanus vaccine or immunoglobulin

b) Fully immunized & wound is tetanus prone (meaning dirty).
Involves manure ((garden) or extensive necrosis

- Give only Tetanus-specific Ig (TIG)

2) Not immunized or immunization status unknown/ uncertain &
a) Wound is dirty:
- Give Tetanus Vaccine and Ig
- Arrange for full Vaccination in different arms via GP

b) Wound is clean: Arrange vaccination only

3) Primary immunisation incomplete or booster not up to date
a) Wound is dirty
- Give Immuniglobulin and vaccination

b) Wound is clean
- Give vaccine

Antibiotics NOT required
Unless in human or animal bite or hand wound

F. BURNS

Causes:

1. Fire
2. Hot water
3. Chemicals
4. Electrical
5. Irradiation

Calculating total body surface area (TBSA) of burn:
Use rule of 9
Leg: 18% each
Arm: 9% each
Trunk back: 18%
Trunk back: 18%
Head: 9%
Perineum: 1%
Just erythema is not counted as an area of burn.

Classification of burn:

1. Partial thickness
2. Full thickness burns

Management:

1. If it's just erythema in some part of body, no treatment is required just reassure
2. For all other serious burns
    a. **Check the airway**, if signs of **inhalation injury**, e.g soot singed nasal hair, burns to the oropharynx, hoarseness of voice, black sputum
        Management: anaesthetize and intubate.
    a. Give analgesia
    b. Give IV fluids
    c. Transfer to special burns units if indicated

CRITERIA FOR TRANSFER TO BURNS UNIT:

1. Partial thickness burn: > 10% TBSA in adults, > 5% TBSA in children / elderly
2. Full thickness burns: >5% any age group
3. Burns on face, hands, feet, perineum, genelatia, major joints, chest
4. Any size of electrical burns
5. Any size of chemical burns

6. Any burn with inhalation injury

INTRAVENOUS FLUIDS:

1. 10% in children and 15% in adult of partial thickness burns
2. 10% or more in anyone of full thickness burns

*Use Hartmans solution/Ringer's Lactate or Normal Saline

Spinal Cord Compression:

1. Constipation
2. Lower limb wakness& Sensory loss
3. Urinary retention

- Peri-anal anaesthesia & reduced anal tone on PR examination
-

Investigation : MRI

Treatment: 1) Dexamethasone as an emergency
2) Surgical decompression is definitive

Disc Prolapse:

- Back pain radiating posterior aspect of the thigh down to the knee. All the way up to below knee
- After heavy lifting
- If Sensory loss is on L5 dermatome then disc prolapse is at L4/ S1
- If sensory loss is on L4 dermatome then disc prolapse is at L3/L4
- If sensory loss is on the S1 dermatome then disc prolapse is at L5/ S1

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Resource name	Paediatrics plab 1 notes
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PAEDIATRIC LECTURE NOTES 2014

PRESENTING COMPLAINTS:

1. BRUISES/PURPURA/PETECHIAE

Causes:

1. Non Accidental Injury (NAI)
2. Leukaemia
3. Lymphoma
4. Haemophilia
5. Idiopathic Thrombocytopenic Purpura (ITP)
6. Henoch-Schönlein purpura (HSP)
7. Haemolytic Uraemic Syndrome (HUS)/
8. Meningococcal Septicaemia

NON ACCIDENTAL INJURY (NAI) - This is the same as child abuse.

Features of non accidental injury are as follows:

1. Late presentation - The child is usually brought to hospital late for example 2-3 days after the fall.
2. Bruises of different ages / shape: which suggest that injuries were sustained at different times.
3. Old stories, lacking congruence with injuries – e.g. child fell from sofa and break shaft of femur
4. Accompanying adult may not be the parent. This could be step father or boy friend or girl friend or foster parents. This group od parents are more likely to abuse as he/she is not their biological parent.
5. Unplanned pregnancy
6. Differential diagnosis is osteogenesis imperfecta which is a congenital abnormality with unexplained fracture. In this case there is usually no history of trauma. Also child may have blue sclera.

7. Young parents usually teenager parents

CATEGORIES OF CHILD ABUSE
Four categories of child abuse are generally recognized - a child may suffer more than one type at a time:

- **Physical abuse**: involves physical harm such as hitting, shaking, burning, poisoning or causing suffocation

- **Emotional abuse**: persistent emotional ill-treatment or neglect causing adverse effects on the child's emotional development. For example: making the child feel worthless; unrealistic expectations; preventing normal social activity; serious bullying; seeing the ill-treatment of another person; making a child often frightened; exploitation or corruption.

- **Sexual abuse**: forcing or enticing a child into sexual activity (this includes both penetrative and non-penetrative acts). It also includes 'non-contact' activities – e.g. involvement in pornography; the child looking at sexual activities or pornographic material; or encouraging inappropriate sexual behaviour in a child.

Usually children who suffer from child abuse are girls. Signs include blood stained under pants and change of behavior of a child.
In this case a child may need to be examined under general anaesthesia.

Neglect: the persistent failure to meet a child's basic physical or psychological needs, in a way likely to impair the child's health or development seriously. For example: not providing food or shelter; inadequate protection from danger; not enabling adequate medical care; emotional neglect.

Management:

1. **Check FBC to rule out ITP**
2. **Skeletal survey to rule out other pre-existing fracture if child has presented with a fracture.**
3. **Admit child under paediatrician**
4. **Give analgesia**
5. **Refer to orthopaedics if there is fracture.**
6. **Involve consultant or seniors, check child `s name on protection registrar involve the social services**

LEUKEMIA: Common leukemia is acute leukemia in children.

In Acute myeloid leukemia you will find splenomegaly, anaemia and bruises.

In acute lymphoid leukemia there is lymphadenopathy, anaemia and bruises.

LYMPHOMA: lymphadenopathy, night sweats, weight loss, lethargy, fever, splenomegaly, hepatomegaly

HAEMOPHILIA: Usually a male child, in early in life, bleeding in to joints and muscles (haematoma) bleeding may be after trauma or surgery. APTT and PT are prolonged. In GMC you have to use their values as the normal values as they may be slightly different to those in OCHM.

ITP (IDIOPATHIC THROMBOCYTOPENIC PURPURA): causes bruising/ purpura /petechial after upper respiratory tract infection. There is usually bleeding from nose, gum etc.

No lymphadenopathy, hepatosplenomegaly, or pancytopenia. Platelets are low

The patients is well, meaning that not ill, no fever.

HSP (HENOCH SCHOLNLEIN PURPURA: Purpura (purple spots/nodules not dispensance) over buttocks, extensor surfaces, arthralgia, abdominal pain, renal involvement.

No history of trauma

Patient is well

Platelet count normal

It is vasculitis

Non-blanching purpura

Immunoglobulin A (Ig A)

MENINGOCCOCAL SEPTICAEMIA = patient is ill, fever purpura, drowsy, photophobia, vomiting (platelets) Any Rash suggest meningococcal septicaemia

HAEMOLYTIC URAEMIC SYNDROME Microangiopathic haemolytic anaemia, purpura, renal failure and endothelial damage to glomerular capillaries

Typical age = 3 months to 3 years

Investigations:

FBC = Fragmented red blood cells, oliguria, patient ill platelet low

CHILDHOOD INFECTIONS

1. **VARICELLA**

Morphology of rash: Clear vesicles on erythematous base (5-12mm) evolves into pustules that burst and crust

Distribution: Lesions occur in crops, starts on the trunk and spreads peripherally, mucosal involvement is common

Incubation period 10-21 days

Associated features: Pyrexia

Complications: Bacterial infection (commonly staphylococcus), encephalitis, pneumonia, reactivates as herpes zoster later in life when patient is ill or immunocompromised

Treatment:

If >1 month + <12 years + immunocompetent: No treatment

If <1 month OR immunocompromised: IV Aciclovir

If >12 years old + immunocompetent: Oral aciclovir

Preventing Spread:

- A person with chickenpox is infectious from 2 days before the rash first appears until all the spots have crusted over.
- Children should not be allowed to go back to school until all the rash has crusted.

1. **MEASLES (also known as 3rd Day Disease)**

Morphology of rash: Maculopapular rash, which appears on the 3rd day of illness

Distribution: Starts on the head and neck, and spreads peripherally

Incubation period: 10-14 days

Associated features: coryza, conjunctivitis, cough, lympadenopathy, koplik spots in mouth

Complications: Otitis media, pneumonia, meningitis, encephalitis

Treatment: Symptomatic unless with complications.

1. **RUBELLA (GERMAN MEASLES)**

Morphology of rash: Pink macular rash

Distribution: Starts from trunk

Associated features: Lymphadenoapthy, especially sub-occiptal lymphadenopathy

Complications: Arthritis in adults, encephalitis

Treatment: Generally symptomatic

1. **PARVOVIRUS** (Slapped cheek, erythema infectiosum, 5th disease)

Morphology of rash: Facial erythema in children, macular or macular papular rash

Distribution: Facial rash in children (slapped cheek appearance)

Associated features: Lymphadenopathy, arthralgia

Complications: Arthritis in adults, foetal loss in pregnancy (hydrops, anaemia in patients with haemoglobinopathies

Treatment: Symptomatic

5. **MUMPS**:

This is a viral infection spread by saliva and respiratory droplets

Typical features:

- Fever with pain and swelling in one or both parotid glands. Aseptic meningitis may occur
- Orchitis develops in 10-15% post-pubertal males.
  Pain relief: analgesia or steroids.

NB. Orchitis is uncommon before puberty. CONSIDER TESTICULAR TORSION.

Incubation period: 14-18 days

Treatment: Symptomatic

STRIDOR IN A CHILD

STRIDOR – is a musical sound produced during inspiration.

Causes:

1. Foreign body
2. Croup
3. Anaphylaxis
4. Epiglotittis
5. Diphtheria
6. Anaphylaxis

I. CROUP = This is defined as an acute clinical syndrome with inspiratory distress = acute viral laryngotracheobronchitis

Causative organism = parainfluenza 1,2,3

Age group = 5 months to 6 years

Clinical features = barking cough, harsh stridor, hoarseness, fever, coryza symptoms often start at night.

Treatment: at home = Steroids: Dexamethasone oral or budesonide if symptoms not settled then nebulized adrenaline with oxygen.

II. FOREIGN BODY = previously well child was playing with a toys/coins – "Choking child". This is usually in children who have been lef t unsupervised.

Treatment:

1. **Infant < 1 year left unsupervised. Back blows with chest thrusts. Use 2 fingers i.e index and middle fingers.**
2. **In young children: above 1 year child should be on the lap. Back blows with chest thrust (compression) with a child on the lap.**
3. **In older children or adults = helmlich manoeuvre (just like in adult)**

4. If the manoeuvre has failed then laryngoscopy

5. If a child is choked and is coughing = encourage coughing

III. EPIGLOTTITIS

Cause: Haemophillus influenza

Symptoms: acute onset of illness with fever, lethargy, inspiratory stridor, no cough drooling of salvia

Investigation = clinical diagnosis (cherry red swollen epiglottis)

Management:

1. **Call anaethetist to intubate the child, do not examine the throat**
2. **Medical = I.V cefotaxime or ceftriaxone**

IV. ANAPHYLAXIS = is potentially life threatening, immunologically mediated syndrome in which laryngeal oedema can develop over minutes.

Symptoms: Itching flushing, stridor, wheeze, facial swelling, shock

Management of Anaphylaxis

1. **First remove allergen**
2. **Give IM Adrenaline if there are indications**
3. **Intubate if there is complete airway obstruction**
4. **Chlorpheniramine (Anti-histamine) must always be given to prevent delayed onset of allergic reaction.**

- **Rash secondary to allergy is called urticarial and if this is the only symptom then adrenaline is not indicated. Use only antihistamine orally.**
- **If there is a big localised swelling then use local antihistamine cream especially after bee sting.**

Indications of Adrenaline in Anaphylaxis

1. **Hoarseness of voice**
2. **Wheeze**
3. **Shortness of breath**
4. **Shock**
5. **Stridor**
6. **Swelling of the tongue and cheek**
7. **Facial swelling**

ADRENALINE DOSE:

Always use 1:1000 concentration and administer intramuscular (IM)

1. Age > 12 give 0.5ml
2. Age 6 years-12 years give 0.3ml
3. Age <6 years give 0.15ml

V. DIPHTHERIA = In children who have not been immunized against the disease **cause carnebacterium diphtheria**

It usually starts with tonsillitis + false membrane over fauces.

Symptoms:

- polyneusitis (cranial nerves)
- myocarditis
- bradycardia
- dysphagia
- brassy cough

Investigation: Swab culture below the membrane

Management:

1. **Diphtheria antitoxin**
2. **Plus erythromycin**

Lower Respiratory Tract Infections

1. **Acute bronchiolitis** = lung inflammation in infants i.e. < 1 year.

- Coryza proceed cough
- Low fever
- Wheeze
- Difficulty in feeding
- Apnoea/ shortness of breath
- Intercostal recession + cyanosis
- Hyperinflated lungs on CXR

Cause: typically respiratory syncytial virus (RSV)

Investigation = Nose + throat swab

CXR shows hyperinflated lungs.

Treatment:

I. **Oxygen**
II. **Nebulized salbutamol**
III. **Dexamethasone**

1. **Pneumonia** = sign increase in temperature, poor feeding, cyanosis, cough, sputum

Investigation: Chest X- Ray. Consolidation = infection, cavitation = T.B (upper lobe) or staph

Treatment: Erythromycin – if allergic to amoxicillin ± co – amoxiclav, which is used in severe infection

3. **Whooping cough** = Bordetella pertussis

Signs = apnoea, bouts of coughing ending with vomiting

- Fever always < 38.4
- No wheeze

Investigation: PCR, culture unsatisfactory

Treatment: Erythromycin, Salbutamol nebulised + steroid if acute shortness of breath

Prevention: Vaccination

FAILURE TO THRIVE

1. **Cystic fibrosis** – autosomal recessive diseases. It is a chloride channel defect. Meaning that the chance to for parents to transfer to their children is 1:4 or simply 25%

Symptoms:

1. Recurrent chest infection is due to the thick mucus, which is difficulty to clear. As a result it causes blockage and leads to recurrent infection.
2. Rectal prolapse
3. Pancreatic insufficiency

- Endocrine
- Exocrine
- Steatorrhoea
- Decrease in weight

1. Meconium ileus
2. Failure to thrive due to the recurrent chest infections

Investigation: Sweat test: Cloride < 40 mmol/L normal

>60 – diagnostic.

Complications: Common complication is bronchiectasis which is dilatation of the bronchi due to recurrent chest infection. Usually develops but the age of 20 years.

Management:

1. **Symptomatic management**
2. **Physiotherapy**
3. **Antibiotics if there is chest infection**

Asthma

Chronic allergic reaction characterised by reversible airway, obstruction, wheeze, cough and dyspnoea.

Triggers: pollen, dust, feather, fur, exercise, infection.

Prophylaxis

1. Avoid triggers
2. Use sodium chromoglycate in exercise induced asthma or pre-exercise bronchodilator (salbutamol)

Management:

1. **Stable patient:** Goes to GP or outpatient department (salbutamol)
2. **Unstable patient:** Patients come to A&E and have severe symptoms

A. **Stable patient**

Step 1: Occasional short acting B agonist inhaler e.g salbutamol as required. If needed > 2/week or (night symptoms) or if getting exacebationmove to next step.

Step 2: Add regular Inhaled Steroid: Therefore at this stage child will be taking salbutamol as required and inhaled steroid e.g. beclomethsone 400 mg. If not effective then increase up to 800 mg.

Step 3: Check diagnosis, check technique (use the spacer with a mask). Add 1 dose montelucast (leucotriene antagonist) in the evening. If 2-5 years add leukotriene antagonist (e. g. montelucast), fulmeterolol. If <2 years refer to the paediatrician.

If child > 5 years add Long acting – agonist e.g. salmeterolol. At this stage if not getting any benefit from long acting beta-agonist them discontinue it. But if it has added some benefit then continue it.

Step 4: Increased inhaled steroid up to maximum dose (e.g Beclomethasone 800 µg --- 1000 µg). If child develops oral candida reduce the dose.

At this stage also consider leukotriene antagonist if not already used or modified long acting beta agonist or aminophylline

Step 5: Add prednisolone – oral

A. **Unstable patient = Acute exacerbation of asthma.**

Patients with acute problems usually go the accident and emergency department

a. **Mild to moderate asthma (able to talk, Pulse < 125, PEFR > 35% of the predicted value. Oxygen > 92%.**

Treatment:

1. Oxygen
2. Nebulised salbutamol or terbutaline
3. Prednisolone.

a. **Severe asthma (can not speak in complete sentences in one breathor child too breathless to speak, PEFR 35%-50%, Oxygen saturation < 92%)**

Treatment:

1. Oxygen - high flow 15L/min or 100%
2. Neb salbutamol ± IV salbutamol
3. Oral Prednisolone or IV hydrocortisone
4. IV $MgSO_4$
5. Aminophyline

a. **Life threatening asthma:** (Silent chest, cyanosis, hypotension, bradycardia, agitation, reduced consciousness, saturations < 92%)

1. Oxygen - high flow 15L/min or 100% oxygen
2. Nebulised salbutamol ± IV salbutamol
3. Oral prednisolone or IV hydrocortisone
4. IV $MgSO_4$
5. Aminophylline

N.B: Intravenous Salbutamol can be used if patient is not able to nebulise for e.g. if someone is vomiting.

Congenital Heart Disease

A. Cyanotic

- **Tetralogy of Fallot**
- Right ventricular hypertrophy
- Pulmonary stenosis
- Ventricular Septal Defect (VSD)
- Overriding aorta
- Chest X-Ray shows booth shaped heart
- Transposition of the great arteries, Chest X-Ray shows egg shaped heart

B. Non Cyanotic

1. VSD = loud pan systolic murmur (harsh)
2. Atrial Septal Defect (ASD) = systolic murmur in the upper left sternal edge
3. Patent ductus arteriosus (PDA) = systolic continuous machinery murmur on the pulmonary area
4. Coarctation of aorta = unpalpable or weak femoral pulses
5. Aortic Stenosis = crescendo-decrescendo systolic ejection murmur

Convulsing child

Causes

1. Epilepsy
2. Febrile convulsion (Fits caused by high temperature)
3. Brain tumours

Management:

1. **The most appropriate treatment is IV Lorazepam.**
2. **If the patient is not responding, give another dose of IV Lorazepam.**
3. **If the patient is still having seizures, load with phenytoin**
4. **If the patient is still convulsing, give phenobarbital**
5. **If the seizure is still ongoing, put patient under general anaesthesia and intubate.**

NB. If no IV access, give per rectal diazepam

Vomiting in infancy

1. **Meningitis** = rash, photophobia, fever, drowsy headache
2. **Pyloric stenosis** = projectile vomiting

- No bile in vomiting (Investigation: Ultrasound Scan)
- No diarrhoea
- Dehydrated and hungry child [6 week old baby] – olive shaped mass in epigastrium

1. **GERD** = vomiting usually occurs after feeding, it is usually reduced when the child sits up
2. **Overfeeding** = also after feeding and child is described as greedy baby
3. **UTI** = fever, vomiting, failure to thrive

N.B. In both GERD & overfeeding, vomiting occurs after feeding.

1. **Gastroenteritis**: Commonest cause of diarrhoea and vomiting is Rotavirus infection

a. Viral
b. Bacterial: Fever, bloody diarrhoea, abdominal pain, vomiting

Investigations: = U&E to check for dehydration

Management: **Rehydration with Oral Rehydration Salts (ORS) 60 mmol/L of Sodium**

Childhood jaundice

Differential Diagnosis

1. Physiological: 24hours - 14 days
2. Breast milk jaundice
3. Haemolytic, sepsis
4. Urinary Tract Infection
5. Hypothyroidism
6. Biliary atresia
7. Galactosemia
8. Hepatitis A
9. α- antitrypsin
10. Prematurity
11. Rhesus incompatibility
12. ABO incompatibility
13. Bruising

Physiological jaundice

- Jaundice in the neonatal period is very common and is usually due to a physiological immaturity. It is self-limiting as the liver matures over the first week. Nearly all preterm infants become jaundiced in the first few days of life, due to the immature hepatocytes.
- Low liver enzyme activity
- Breakdown of fetal haemoglobin

Breast milk jaundice

- This is persistent jaundice in an otherwise well, breast-fed infant. This is due to the inhibition of liver conjugation enzymes. Split bilirubin should be measured (conjugated & unconjugated) to exclude conjugated hyperbilirubinaemia. Normally manifests itself by day 4-7.
- Well baby who is breast-fed.
- Jaundice develops in second week.

Hypothyroidism

- May be associated with pituitary disease

Biliary atresia

- Present 4-6 weeks after birth
- Present in obstructive jaundice
- A conjugated hyperbilirubinaemia develops over a period of weeks. Stools become clay coloured i.e pale stools and dark urine
- Persistent jaundice with rising conjugated fraction
- Pale, chalky stools
- Requires urgent referral for assessment, diagnostic isotope scan and surgical correction.

Prematurity – Pre-term born babies

- Immature liver enzymes

Rhesus incompatibilty

- **Usually develops on the first day of life**
- If mother is Rh negative and baby Rh positive, the maternal IgG can cause haemolysis
- Sensitization occurs in earlier pregnancies
- If severe can cause hydrops in utero
- Coombs' test positive, high unconjugated bilirubin

ABO incompatibility – Autoimmune – Coombs' positive

- Usually milder than rhesus

Bruising

- Skin or scalp bruising from traumatic delivery is broken down into bilirubin

Key points

- Mild jaundice is extremely common in newborn infants, especially preterm ones.
- Jaundice within the first 24 hours or lasting beyond 2 weeks needs investigation.
- Phototherapy and occasionally exchange transfusion are used to treat significant jaundice.
- Biliary atresia causes an obstructive persistent jaundice with pale stools. Early treatment is essential.
- Conjugated hyperbilirubinaemia is always abnormal. Exclude biliary atresaia.

N.B.

- < 24 hours = pathological
- > 24 hours = usually physiological

Investigations – Serum bilirubin

Routine Paediatric Immunisations

- **2 months - 5 in 1 (DTaP/IPV/Hib), PCV, Rotavirus (by mouth)**
- **3 months - 5 in 1 (DTaP/IPV/Hib), MenC, Rotavirus**
- **4 months - 5 in 1 (DTaP/IPV/Hib), PCV**
- **12 - 13 months - Hib/MenC, PCV, MMR**
- **2 and 3 years - Flu nasal spray**
- **Pre school (3 years 4 months) - DTaP/IPV, MMR**
- **Girls 12-13 years - HPV**
- **Around 14 years - Td/iPV, MenC**

Key:

- **DTaP - Diphtheria, Tetanus, acellular Pertussis (in thigh)**
- **IPV - Inactivated Polio Virus**
- **Hib - Haemophilus influenza type b**
- **PCV - Pneumococcal Conjugate Vaccine (in thigh)**
- **Men C - Meningococcal group C**
- **MMR - Measles, Mumps, Rubella (upper arm/thigh)**
- **HPV - Human Papillomavirus (upper arm)**
- **Td - Tetanus, diptheria (upper arm)**

Developmental Milestones

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Resource view

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Resource description	Psychiatry
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PSYCHIATRY NOTES

MENTAL STATE EXAMINATION

I. APPEARANCE AND BEHAVIOUR

- State and colour of clothes
- Eye contact (Poor eye contact in depression)
- Tearful (Depression)
- Down cast gaze
- Agitation(seen in depressive illness, anxiety, psychosis)
- Tremor, Fidgeting
- Distracted
- Visual scanning (for danger)

II. MOOD AND AFFECT

The patient has an emotion and feeling, tells the doctor their mood and the doctor observes the patient affect

Affect is the mood by appearance of the patient as judged by the doctor

Mood Alteration

Persistent change in mood

Fluctuating or labile mood

Inconsistent or incongruent mood

A persistent change in mood

1. ***Depression***:

- Low mood
- Feeling sad
- Tearful & low in spirits
- Feeling guilty & low confidence level
- Anhedonia – loss of interest in daily activities
- Loss of appetite & weight
- Loss of libido

2. ***Anxiety:*** A feeling of constant inappropriate or excessive worry, fear or tension. Common in young women

3. ***Irritability.*** Temper or impatient

4. ***Elation***: Feeling of high spirits e.g. mania

5. ***Blunting of affect:*** Total absence of emotion, seen more commonly in chronic schizophrenia

Fluctuating or labile mood

This is when different emotions rapidly follow one another so that a patient is crying one moment and laughing the next.

Inconsistent or incongruous mood

This occurs when emotional expression fails to match thoughts and actions

e.g. Patient may laugh when describing the death of a relative

SPEECH

Disorders of stream of thought

1. *Pressure of speech*:

Can be recognized by loudness, rapidity and difficulty in interrupting speech e.g. mania

2. *Poverty of speech*:

Absence of any thought and patient report their mind to be empty e.g. in schizophrenia

3. *Thought block:*

Speech is normal initially and then it stops suddenly

Occurs in schizophrenia

i.e. there is an abrupt(sudden) and complete interruption of the stream of thought, so the mind goes blank

Disorders of form of thought

1. Flight of ideas

Patient's thoughts rapidly jump from one topic to the next, such that one train of thought is not completed

This is characteristic of mania and is often accompanied by pressure of speech

2. *Preservation*: persistent and inappropriate repetition of same thoughts or action.

Occurs in frontal lobe lesions e.g. If you ask patient to say No Ifs ands or buts, he will say No ifs ifs ifs……

3. *Loosening of associations*: loss of normal structure of thinking

It is usually difficult to understand what the patient is speaking

4. *Thought broadcasting*

A feeling as if other people are hearing or understanding their thoughts despite not telling people what they are thinking. E.g. schizophrenia

5. *Thought insertion*

Feeling that someone is putting thoughts into their mind. Patient feels that the thoughts in his mind are not his/her. E.g. schizophrenia

6. *Thought withdrawal*

Feeling that someone is taking my thoughts away or stealing of thoughts

4, 5 and 6 are the 3 factor's which indicate schizophrenia

IV. THOUGHT CONTENT

1. An obsessional rumination: is a recurrent, persistent thought, impulse, image or musical theme that enters the mind despite individual's effort to resist it. E.g. Obsessive Compulsive Disorders

2. Compulsion: is a repetitive and seemingly purposeful action performed in a stereotyped way which is called a compulsive ritual. E.g. Obsessive Compulsive Disorder

V. INSIGHT

Is the awareness of the patient that he or she is unwell or has a problem.

ABNORMAL BELIEFS **these are** called delusions

A delusion is an abnormal belief that is held with absolute conviction

Not amenable to reason or modifiable by experience

Usually false

Not shared by those of common social or cultural background

ABNORMAL PERCEPTION

1. *Illusion*: This is the distortion or misinterpretation of external stimuli or real things e.g. see a tree as if it's a person

2. *Hallucination*: hearing or seeing things which are not there and without any stimuli. It is a false perception and not distortion.

- Hypnogogic Hallucinations: Episodes of seeing or hearing things as one is falling asleep. These dreams can be frightening and one can often cause a sudden jerk and arousal just before the sleep. It is associated with narcolepsy.
- Hypnopompic Hallucinations: Episodes of seeing, hearing or feeling things while getting up from sleep. E.g. Narcolepsy
- Auditory Hallucinations
- Visual Hallucinations e.g. schizophrenia, substance abuse- PCP or cocaine
- Tactile Hallucinations e.g. small insects crawling on skin in chronic alcoholics or alcohol abuse
- Gustatory Hallucinations
- Olfactory Hallucinations
- Third Person Hallucination (Usually auditory-Running commentary that he is an evil person and ants to kill me)

3. *Pseudo or False hallucinations*: usually auditory e.g. hear a voice in my head speaking to me. Patient have insight to the problem

4. *Derealization*: unpleasant feeling that he external environment has become unreal i.e. patient feel like that they are in a dream like state

5. *Depersonalization*: change in self awareness such that a person feels unreal or detached from their body

6. Déjà vu: a sudden familiarity with the situation or event as having been encountered before when it is in fact new.

7. Jamais vu: Failure to recognise an event or situation which was encountered before.

FUNCTIONAL DISORDERS

1. CHRONIC FATIGUE SYNDROME

- This is when a person feels tired, fatigued all the time
- All investigations are normal
- Fatigue and tiredness does not improve with rest
- No structural abnormalities found

Management: Education and reassurance, Education about appropriate rest and activity, CBT, Graded Exercise Program, relaxation therapies- yoga, meditation.

Referral to psychiatrist and psychologist

2. FIBROMYALGIA (Chronic widespread pain)

- This a functional widespread pain all over the body
- All investigations are normal

3. IRRITABLE BOWEL SYNDROME

- Functional disorder
- Presents as abdominal pain which is usually relieved by passing flatus or stools
- Diarrhea, bloating, constipation
- All investigations are normal
- It is a diagnosis of exclusion
- No PR bleed
- No night symptoms
- No weight loss

Management: Education and symptomatic treatment

4. PREMENSTRUAL SYNDROME

- Physical and psychological symptoms that regularly occur during the premenstrual phase and diminishes soon after the period starts
- Cause is likely to be hormonal
- Abdominal pain, Irritability, Low mood, Bloating

SOMATOFORM DISORDERS

1. SOMATIZATION DISORDER

- This is multiple, recurrent, medically unexplained symptoms, usually starting early in life. Usually patient presents with one symptom at a time.
- Nausea, Vomiting, Abdomen pain
- Neck pain, Back pain, Headache
- Etiology is unknown
- Investigations are normal

2. HYPOCHONDRIASIS

- Preoccupation with assumed serious diseases.
- Commonly patient believes they are suffering from cancer or HIV even after repeated reassurance with normal investigations for symptoms and they repeatedly request investigations.

NB: Management of somatoform disorders is reassurance and education, Referral to psychologist.

DISSOCIATIVE DISORDERS (Conversion)

- Also known as hysteria.
- It is a condition where there is a profound loss of awareness or cognitive ability.
- E.g. Amnesia- loss of memory
- E.g. Pseudo-seizure (Psychogenic non epileptic seizures): Seizure like activity resembling epileptic fit but without any electrical discharges associated with epilepsy and the patient is not hurt during this seizure and usually tries to resist any attempt to change his posture by other people. Long-term video EEG & Serum prolactin is usually done to distinguish between true seizure and pseudo-seizure.

MOOD (Affective disorders)

Depressive disorders

A. Unipolar: Depression occurring on it's own.
B. Bipolar: Depression alternating with mania.

Bipolar affective disorder

A. DEPRESSION

1. Low mood
2. Low energy level
3. Feels sad
4. Anhedonia – loss of interest in daily activities
5. Early morning waking
6. Loss of appetite
7. Feeling guilty
8. Reduced self esteem
9. Thoughts of self-harm
10. Fatigability – feeling tired
11. Loss of libido

B. DYSTHYMIA

It is characterized by the mild depressive illness that lasts intermittently more than 2 years.

C. SEASONAL AFFECTIVE DISORDER

It is characterized by the recurrent episodes of depressive illness occurring during the winter months. It occurs annually usually in winters.

- Atypical symptoms
- Low mood, anhedonia
- Excessive sleep
- Increased appetite and weight gain

Management: Light therapy, Pscho-therapy and antidepressants.

***D.* POST NATAL DEPRESSION**

- Occurs after delivery
- Poor sleep, low confidence, anhedonia
- Loss of appetite & weight
- Feeling that she is not capable of looking after her child
- Guilt feeling that she is not a good mother
- Tearful, Anxiety
- Occurs in the first 3 months after delivery
- Mother feels as if someone/partner wants to harm her baby

***E.* BABY BLUES**

- Occurs in 50% women after giving birth
- Normal phenomena and resolves within a few days usually 3-4 days
- Poor sleep, anxiety, irritability, tearful, crying for no reason

Management: Family support & Reassurance

***F.* POST NATAL PSYCHOSIS**

- Usually occurs within 2 weeks after delivery
- Usually starts with post-natal depression
- Delusional ideas that the baby is deformed, evil or otherwise affected in some way and she has intent to kill the baby, evils or self harm

G. BIPOLAR AFFECTIVE DISORDER

Mood swings of mania and depression

Treatment of depressive illness

1. SSRI: Selective Serotonin Re-uptake Inhibitors: First choice of treatment

- Citalopram- Preferred in IHD
- Escitalopram
- Fluoxetine
- Paroxetine

2. Tricyclic Antidepressant (TCA)

- Amitryptilline
- Dosulepin
- Lofepramine, Trazodone

Contraindication: in Ischaemic Heart Disease (IHD) and Glaucoma

Side Effect's:

- Arrythmia's
- Dry Mouth
- Constipation
- Raised Intra-ocular pressure- leads to Glaucoma

3. Other Anti depressants

- Mirtazapine
- Venlafaxine
- Reboxetine

4. Monoamine oxidase inhibitors (MAOI)

- Phenelzine

NB: 1. Depression with obesity=fluoxetine (It helps without weight loss)
2. Depression with sexual dysfunction=mirtazapine
3. Post stroke depression use nortriptyline (TCA)
4. Depression with obsessive compulsive disorder=clomipramine (TCA)
5. Depression with ischemic heart disease=SSRI e. g citalopram

NON-MEDICAL TREATMENT

a. **Electroconvulsive therapy (ECT)**

Indications:

1. Refusing to eat and drink and their weight is dangerously going low
2. Dangerously suicidal (Patient's looking for every opportunity to kill themselves)
3. Psychotic symptoms
4. Depression not responding to anti-depressants
5. Depression with Psychosis

b. **Cognitive Behavior therapy**

Good for mild to moderate depression. It is as effective as medical treatment.

Good for anxiety disorder

It involves identification of abnormal thinking that keeps triggering depressive, anxiety or any other symptoms and tries to fix or change it. E.g. Depression, OCD, PTSD

c. **Behavior therapy**

Based on learning theory

Works as desensitization e.g. in OCD, phobia (arachnophobia- fear of spiders, agoraphobia- fear of open spaces, claustrophobia- fear of closed spaces)

d. **Interpersonal psychotherapy**

Used for depression and eating disorder especially depression triggered by personal relationship

e. **Couple therapy**

When a patient with depression is having problems in relationship like sexual, emotional etc.

f. **Family therapy**

If family is supportive – educate the family about the condition and the members of the family

g. **Supportive therapy**

It is similar to counseling. E.g. Bereavement, baby blues

h. **Group therapy**

Where a problem involves a group of people e.g. drug abuse

I. Exposure and Response Prevention Therapy

Used in OCD & phobia's

SEROTONIN SYNDROME

- Toxic hyper-serotonergic state
- Occurs when SSRI and MAOIs are used together or overdose of SSRI's or when 2 SSRI's are started together. Symptoms:
- Agitation
- Confusion
- Tremor
- Diarrhea
- Tachycardia
- Hypertension

II. MANIA AND HYPOMANIA

Elevated mood: characterized by euphoria, overactivity and disinhibition

Hypomania: is mild form of mania, it lasts shorter time and less severe

Mania: almost always occurs as bipolar affective disorder

Clinical features of Mania

- Elevated mood
- Fast pressurized speech, flight of ideas
- Excessive energy, anhedonia, self confident, Over-spending, delusion of wealth
- Delusion of Grandiose
- Delusion of Control
- Dis-inhibition
- Hallucination

Treatment of MANIA

ACUTE TREATMENT

1. Lithium is the first choice
2. Antipsychotic is 2nd line eg. Halperidol or chlorpromazine

PROPHYLAXIS

1. Lithium is the first choice
2. If not responding to lithium then use anti convulsants e.g. Carbamazepine

Monitor patients on Lithium with Thyroid functions tests and U & Es

***Lithium adverse affects*:**

- Can cause nephrogenic diabetes insipidus which presents with polyuria, polydipsia.
- Hypothyroidism
- Nausea
- Tremor
- Weight gain

At therapeutic levels lithium has the following side effects

ANXIETY DISORDERS

1. ***Generalized anxiety disorder***

Patient is worried about different number of events every day. Almost everything triggers the anxiety.

2. ***Mixed anxiety and depressive disorders***

There is equal amount of anxiety and depression

- Palpitation & chest pain
- Excessive worries
- Low mood
- Poor sleep & guilt feeling
- Reduced appetite

3. ***Panic disorder or Anxiety attack***

- There is hyperventilation (SOB) difficult to take a deep breath
- Chest pain (all over the chest)
- Choking sensation
- Palpitation, sweating
- Perioral paresthesia & Tingling and numbness in the hands due to hyperventilation and CO2 washout leading to low ionic calcium.
- Patient has belief of catastrophic illness e.g. MI or stroke
- Feeling of Impending doom or feeling as if having a heart attack or going to die.
- Feeling of butterfly's in tummy

Management

- Rebreathing bag
- Reassurance
- Relaxation therapy
- Beta-blocker can be used for upcoming stressful event e.g. an exam or a job interview but is not used acute attack of panic attack. In acute attack use re-breathing bag.
- Anxiolytics: E.g. Diazepam- If having severe symptoms

PHOBIA DISORDERS

1. Agarophobia: translated as fear of the market place, fear of going out in open places. As soon as patient is out, starts to have panic attack. e. g shops, markets

2. Social phobia: Fear to socialize with other e.g. Meetings, Parties, Crowds and with normal life.

Simple phobias

Arachnophobia: Fear of spiders particularly in women. Treatment: Exposure and response prevention therapy, Desensitization, Behavioral therapy.

Treatment of anxiety disorder

1. Relaxation therapy: If symptoms have resolved

2. Behavior therapy: Graded exposure called systemic desensitization – first choice treatment for phobias.

3. CBT: Best Rx for panic disorder, endogenous phobia and anxiety disorder

4. Drugs: Anxiolytic: e.g. Benzodiazepine

ACUTE STRESS AND ADJUSTMENT DISORDERS

1. ***Acute stress disorder***

- Occur in individual without psychiatric illness, in response to exceptional physical and/or psychological stress
- Symptoms are severe but they subside within hours or days include – sudden changes in some circumstances (accidents, rape)

2. ***Adjustment disorder***

- This follows acute stress disorder usually in hospital
- It is prolonged lasting up to 6 months
- It is reaction to bad news or a significant event

3. ***Pathological or abnormal grief***

- This is a type of adjustment disorder
- There is excessive and prolonged grief or denial of the bereavement
- Repeated dreams of a dead person
- Patients have anger at doctors or even the person who died him/ herself.

4. ***Post traumatic stress disorder***

This is delayed or prolonged response to stressful situation

e. g: Sexual abuse

War, Road Traffic Accident

Human disaster

Clinical features

1. Flashbacks: relieving the event

2. Insomnia

3. Avoidance of activities – avoid similar circumstances

4. Hyper-vigilant and hyper-arousal

***Treatment*: CBT**

Obsessive Compulsive Disorder

They are repetitive and intrusive (interfere with personal life and activities)

Common examples:

1. Checking doors locked all the time expectedly
2. Walking back and forth again and again
3. Cleaning the toilet repeatedly
4. Washing hands excessively

Treatment:

1. Cognitive Behavior therapy
2. Anxiolytics – Benzodiazepine

NB: Benzodiazepines:

They are used for alcohol withdrawal

Cause respiratory depression

Lorazepam is short acting

Diazepam & Chlordiazepoxide is long acting

NB: Treatment of anxiety or alcohol withdrawal use long -acting benzodiazepine.

ALCOHOL ABUSE

1. ***Alcohol withdrawal:***

- Delirium tremens
- Agitation
- Aggression
- Tremor (Usually these symptoms appears in a patient admitted for an operation & 2-3 days after operation or hospitalization due to any other medical reason)
- Confusion, Tachycardia, Hypotension
- Visual hallucinations are very common in alcohol withdrawal and they are very suggestive of alcohol abuse. E.g. insects crawling in beds, tactile hallucinations.

Management of alcohol withdrawal -detoxification program use Long acting benzodiazepines

1st choice: Chlordiazepoxide

2nd choice: Diazepam

2. ***Wernicke's encephalopathy***

- Headache
- Confusion
- Flapping tremor, ataxia
- Opthalmoplegia.

Management: IV high potent vitamins, IV Thiamine (Vitamin B1) followed by IM thiamine

General Management of alcohol abuse:

1. Detoxification program: This is during withdrawal period use long acting benzodiazepine e.g. chlordiazepoxide

Maintenance of abstinence:

1. Acamprosate: Decreases craving, decreases relapse
2. Disulfiram: Causes unpleasant symptoms if alcohol is consumed
3. Naltrexone: Not licensed in the UK for this purpose (but decreases craving and and relapses)

DRUG ABUSE

1. Opiate e. g heroin/morphine/methadone
Detoxification (during withdrawal)= use methadone
Symptoms of withdrawal

- Flu like symptoms
- Muscle cramps
- Running nose
- Occurs 7-10 after stopping use of Opiates
- Agitation, restlessness
- Diarrhea, abdominal pain
- Yawning & sweating, difficulty to sleep

Maintainance use methadone as well.

Opiate overdose is treated with naloxone, usually there is decreased respiratory rate less than 12 and also pin point pupils or IV marks/ puncture marks on arms or legs.

NB: Cocaine is not an opiate its an amphetamine analogue.

HALLUCINOGENIC DRUGS

LSD-LYSERGIC ACID DIETHYLAMIDE

- Causes distortion of sensory perception and visual hallucination e.g. an orange tie of the teacher is speaking to me.

COCAINE:

- Usually sniffed
- Dilatation of pupils
- Tachycardia
- Hypertension
- Causes nasal perforation leading to the whistling in the nose.
- Overdose can lead to Sub-arachnoid hemorrhage, myocardial ischemia and acute MI.

Withdrawal symptoms of cocaine:

Tremor

Depression

Muscle pain

Investigation: Creatinine phosphokinase is elevated.

SCHIZOPHRENIA

Symptoms: The 3 main symptoms are:

1. Hallucinations
2. Delusion
3. Thought disorder

- Thought Insertion
- Thought With-drawl
- Thought Broadcasting
- Thought Blocking
- Passivity Phenomenon e.g. there is a device in the brain trying to control patient.
- Blunt Effect
- Incongruence Mood
- No Insight

Management: Anti psychotics - Can be classified into typical and atypical

Typical

Eg. Haloperidol or Chlorpromazine

Side effects: Neuroleptic syndrome, fever, hypothermia, tachycardia, fluctuating consciousness, increased WCC, abnormal LFT, hyperprolactinaemia

Atypical

Olanzapine, risperidone, clozapine, quetiapine, amisulpiride

SIDE EFFECT OF TYPICAL ANTI-PSYCHOTIC: Dopamine receptor blockage (Haloperidol and chlorpromazine)

Extra-pyramidal side effect:

- Parkinsonism: Brady-kinesia, tremor & rigidity
- Hyper-prolactinaemia causing Galactorrhoea, Amenorrhea, Infertility, Oligomenorrhoea.

If Extra-pyramidal side effects please switch to Quetiapine.

SIDE EFFECTS OF ATYPICAL ANTI-PSYCHOTICS

1. Clozapine causes Agranulocytosis

2. Risperidone & Olanzapine can cause extra-pyramidal side effect and hyperprolactinaemia in higher doses.

3. All atypical anti-psychotic can cause sexual dysfunction e.g erectile dysfunction, low libido, low arousal, anorgasmia, sexual dysfunction, weight gain.

EATING DISORDERS

Anorexia nervosa

Commonly young female

Mobid fear of being fat & distorted body shape

Weight loss

Amenorrhea

BMI <17.5

Binge eating

Lack of sexual interest

Treatment: CBT
If severe weight loss, BMI <15.5,
Dizziness, weakness, Admit and
Assess suicidal risk

Bulimia nervosa
Morbid fear of being fat
Craves for food
History of misuse of laxatives
Fluctuating body weight
Self induced vomiting
Treatment: CBT

ANOREXIA NERVOSA

Mild Anorexia Nervosa:

- BMI > 17.5

Management: Educate, Refer to self help groups and make a food diary.

Moderate Anorexia Nervosa:

- BMI 15-17.5
- Evidence of organ failure

Management: Referral to Eating disorder Clinics, Adolescent mental health team.

Severe Anorexia Nervosa:

- BMI <13
- Rapid weight loss
- Evidence of systemic failure
- Arrythmias
- Electrolyte imbalance
- Amennorhea

Management: Urgent referral to Eating Disorder Unit or Medical Unit or Pediatric Ward

PERSONALITY DISORDERS

CLUSTER A: Odd or Eccentric

- Paranoid
- Schizoid
- Schizotypal

CLUSTER B: Dramatic, Emotional or Erratic

- Histrionic
- Anti- social
- Borderline
- Narcissistic

CLUSTER C: Anxious or Fearful

- Avoidant
- Dependent
- Obsessive Compulsive

1. ***Borderline (emotionally unstable)***: Act impulsively and develop intense but short-lived emotional attachment to others. They are usually attention seekers but not suicidal.

2. ***Paranoid***:

- Extreme sensitivity
- Suspiciousness and a tendency to excessive self importance
- Suspicious of others to do harm to them or other
- Preoccupied with thoughts that others will harm them

3. **Schizoid (social withdrawal)**

- Lack of capacity to express emotions
- Little interest in sex
- Do not make friends
- Try to work at places where people do not come
- May precede depression

4. **Schizotypal**

- Idea of reference
- Magical thinking
- Unusual perception

5. **Histrionic**

- Attention seeking
- Excessive shallow emotion
- Self dramatization
- Shallow + labile emotion

6. **Antisocial personality disorder**

- Involved in criminal offences
- Aggressive and rude behavior
- Involved in dangerous acts
- Lack of capacity to maintain enduring relationship
- Low tolerance of frustration
- Inability to experience guilt

7**. Anankastic personality disorder (Similar to obsession disorder)**

- Perfectionism
- Feeling of excessive doubts

8. **Dependent personality disorder**

- Encourage other to make decisions for them
- Feel unable to care for themselves
- Constant fear of being abandoned
- Usually in women

9. **Avoidant (anxious) personality disorder**

- Feeling of tension and inadequacy
- Social inhibition
- They only interact with people if they know if they will be liked

UNUSUAL PSYCHIATRIC SYNDROMES

1. **Othello syndrome:**

- Pathological jealousy
- Patient is delusional convinced that their partner is being unfaithful (cheating)
- They try to prove the fact that the partner is cheating on them
- Common in men

2**. Cotard's syndrome**

Is characterized by nihilistic delusion in which patient believes that parts of their body are decaying or rotting or have ceased to exist. Patient may believe they are dead and they ask people to burry them.

3. **Folie-a-deux**

It is an induced or shared delusional disorder. It a delusional belief that is shared by 2 or more people of whom only one of them has features of psychiatric illness

4. **Erotomania**

Patient has a belief that someone is in love with him/her

Normally the object of there affection is someone from high society. E.g. Queen is in love with him or Angelina jolie is sending messages to him.

5. **Couvade syndrome**

Experience symptoms of pregnancy in men

Abdominal swelling, nausea, vomiting

Commonly in expecting father

6. **Munchausen's syndrome**

This is deliberately creating medical symptoms

- Usually these people have medical back ground
- Abdominal pain, sexual abuse, hallucination
- Multiple abdominal scars which suggests multiple managements.

Types of delusion

1**. Persecution**: Someone or something is interfering with the person

Worried that people are against him/her and trying to harm him/her

2. **Grandiose:** Being famous, having supernatural power or enormous wealth. Believe that they have exceptional abilities or talent and keep praising themselves. E.g. usually seen in high society figures- actors, mania.

3. **Delusion of reference:** Other people, event, media are referring to the person or communicating a message. E.g. someone is giving them special messages through Newspaper, TV and radio.

4. **Passivity**: Action, feeling or impulses can be controlled or interfered with by outside influence. Feeling another person is controlling what you are doing. E.g. Schizophrenia

5. **Paranoid Delusion:** Feeling that people are trying to kill them.

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Resource description	Respiratory Medicine
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RESPIRATORY PLAB 1 NOTES 2014

Presenting symptoms

1. Cough
2. Shortness of breath = Dyspnea = breathlessness
3. Stridor
4. Wheeze
5. Hemoptysis
6. Hoarseness of voice
7. Sputum
8. Apnea (cessation of breath)
9. Orthopnea (shortness of breath when lying flat, sign of LVF)
10. Chest pain

COUGH

A. Dry cough = no sputum

B. Productive cough = with sputum

Causes of dry cough

1. Atypical pneumonias (chlamydia psittaci, mycoplasma, legionella, pneumocystis jiroveci or carinii)

2. Interstitial lung disease
3. Asthma
4. Bronchogenic carcinoma

Causes of productive cough (This is cough with sputum)

1. Pneumonia (commonest)
2. Bronchiectasis
3. Lung abscess
4. Tuberculosis
5. Pulmonary edema
6. Infective exacerbation of COPD

Cough

A. Acute
B. Chronic

ACUTE COUGH

1. Asthma
2. Allergy
3. Drug side effect e.g. ACE inhibitors
5. Foreign body inhalation or aspiration-usually sudden onset.

CHRONIC COUGH

1. Tuberculosis
2. Lung cancer
3. COPD
4. Bronchiectasis
5. Interstitial lung disease

CAUSES OF DRY COUGH

1. Atypical pneumonia=all have dry cough

i) Legionella: history of travel abroad for a business trip or holiday or staying in a hotel, patchy consolidation on the chest x-ray. Legionella colonizes water tanks in hotel at 60 degrees.

ii) Mycoplasma Pneumonia: flu-like illness, dry cough and patchy consolidation on the chest X-ray

iii) Pneumocystis jiroveci/carinii: dry cough, HIV patient or homosexual or IV drug abuser or from Africa, low CD count cell.

iv) Chlamydia psittaci: dry cough, contact with birds or works in a parrot shop

*** If unsure about atypical pneumonia- choose MYCOPLASMA.**

2. Asthma

- **Dry cough at night**
- **History or family history of atopy i.e. eczema, hay fever, asthma or history of allergy to other things.**
- **Wheeze**

3. Interstitial lung disease

- **History of working in a coal mine, pharmaceutical companies or exposure to dust usually due to occupation.**

4. Lung carcinoma (bronchogenic carcinoma)

- **Usually elderly patient**
- **Weight loss, anemia, tiredness, anorexia, fatigue**
- **Smoking history**
- **Hemoptysis**

PRODUCTIVE COUGH (COUGH WITH SPUTUM or PHLEGM)

1. Pneumonia

- **Cough, fever, shortness of breath**
- **Pleuritic chest pain- Chest pain which is worse on inspiration**
- **Rusty brown sputum**
- **Chest X-Ray shows consolidation**

2. Infective exacerbation of COPD

- **Fever, cough with sputum**
- **Chronic shortness of breath**
- **Long-term history of smoking history**
- **Usually middle aged man between 35-55 years old**

***There is usually long standing history of smoking in a middle-aged man**

3. Bronchiectasis

- **Productive cough with green/yellow sputum more in the morning**
- **History of recurrent chest infection**
- **History of cystic fibrosis (failure to thrive as a child, rectal prolapse, recurrent chest infection as child)**
- **Caused by recurrent chest infections leading to localized and irreversible dilation of airways**

4. Lung abscess

- **Purulent sputum**
- **Fever usually swinging fever or intermittent fever)**
- **History of alcohol abuse suggest aspiration**

*** Swinging fever always means ABSCESS.**

5. Tuberculosis

- **Patient from Africa or Asia or if within the UK its homeless alcoholic**
- **There can be history of intravenous drug us e or homosexual which suggest that HIV more likely.**
- **Weight loss, night sweats, haemoptysis, chest pain**
- **Lymphadenopathy (enlarged lymph nodes)**

NB: In an HIV patient weight loss with purulent sputum is TB, but weight loss with dry cough its Pneumocystis carinii pneumonia (PCP).

SPUTUM

1. Rusty brown means pneumonia

2. Pinky frothy is clear sputum with tinge of blood means pulmonary edema or left ventricular failure

3. Mucoid sputum means pneumonia

4. Purulent sputum usually in lung abscess, bronchiectasis or pneumonia caused by staphylococcus aureus.

HAEMOPTYSIS - this is coughing up of blood

1. Pulmonary embolism

- **Usually young patient**
- **Risk factors (COCP, pregnancy, long flights e.g. from South Africa, America or Australia. If patient from Africa it can be PE as well as it is also a long flight from Africa but make sure it not TB or Pneumocystis jiroveci.**
- **Post-operative usually 7-10 days post operatively or after fracture of the long bones.**
- **ECG findings:**

a. **Sinus tachycardia**
b. **RBBB**
c. **T wave inversion V1-V4**
d. **SIQIIIT III syndrome**
e. **AF**

NB: fat embolism usually causes confusion and rashes all over the body.

2. Tuberculosis causes haemoptysis - please see above

3. Pulmonary oedema

- **Usually pinky frothy sputum**
- **Cough**
- **History of valvular heart disease especially mitral stenosis or LVF**
- **Chest X-Ray shows bilateral fluffy opacities or or enlarged hear heart if there is heart failure.**

4. Bronchogenic carcinoma please see above

5. Good pasture's syndrome - its an autoimmune disease in which antibodies are formed against basement membrane of the glomerulus (kidney) and basement membrane of the alveoli (lung). Therefore you have haemoptysis and renal symptoms.

Symptoms

- **Young patient**
- **Proteinuria**
- **Haematuria**
- **Haemoptysis**

6. Lung abscess especially if there is cavitation, history of swinging fever, purulent sputum, chronic alcoholism and aspiration.

7. Anti-coagulant e.g. warfarin

SHORTNESS OF BREATH OR DYSPNOEA OR BREATHLESSNESS

A. Acute

B. Chronic

ACUTE

1. Asthma

2. Pulmonary Embolism

3. Myocardial Infarction

4. Pneumonia

5. Pneumothorax

6. Pulmonary oedema (acute left ventricular failure)

7. Anaphylaxis

8. Foreign body

9. Exacerbation of COPD

10. Anxiety

11. Carbon Monoxide poisoning

CHRONIC

1. COPD

2. Tuberculosis

3. Bronchogenic carcinoma

4. Chronic Heart failure

5. Mesothelioma

6. Sarcoidosis

7. Chronic anemia

8. Cor pulmonale –right ventricular failure due lung problem.

ACUTE SHORTNESS OF BREATH

1. Myocardial Infarction

- **Elderly or middle aged patient**
- **Central crushing central chest pain radiating to the neck or left arm**
- **Nausea and vomiting**
- **Sweating usually in the palms**
- **Shortness of breath only if there is underlying pulmonary edema secondary to LVF**

2. Spontaneous Pneumothorax

- **Usually young, tall, thin man**
- **Sudden onset of shortness of breath**
- **Sudden onset of chest pain**
- **There is no history of trauma but sudden chest pain and shortness of breath usually start while doing exercise e.g. playing football or riding a bicycle.**

3. Anxiety or Panic attack

- **Young female with previous episodes of panic attack**
- **Peri-oral paraesthesia, tingling and numbness in the hands due to low ionic Calcium secondary to hyperventilation and CO2 wash down.**
- **Shortness of breath with difficulty to take deep breath**
- **Palpitations, feeling of impeding doom**
- **Feeling that they are having heart attack or going to die**

Treatment: re-breathing through a paper bag (during the attack)

4. Anaphylaxis

- **Urticaria(allergic rash) e.g. after playing football or after playing in the grass or after eating peanuts or eating in the restaurant**
- **Acute facial swelling**
- **Acute shortness of breath due to laryngeal edema**
- **Acute shock i.e. low BP and tachycardia**
- **May present with collapse**
- **Hoarseness of voice, wheeze, tongue swelling.**

Treatment: IM adrenaline 1:1000 in anterolateral aspect of mid thigh

5. Foreign body ingestion

- **Usually in children usually after playing with toys/coins and left unsupervised. Child previously fit and well.**
- **In adult while eating**

Treatment: if patient is having shortness of breath now, then needs urgent laryngoscopy, but if no symptoms of air obstruction then do chest x-ray to check for foreign body in the GIT.

If foreign body below diaphragm then observe for the foreign body to pass with stool in 48 hours.

6. Carbon monoxide poisoning

- **Usually due to leaking gas and several members of the family are effected**
- **It can be after house fire, black soot in nose, singed nasal hairs, black sputum**
- **Airway obstruction is due to laryngeal edema or inhalational burn leading to SOB.**

Treatment: if any signs of inhalation injury (i.e. black sputum, singed nasal hair, soot in the mouth, hoarseness of voice) then needs general anaesthesia and intubation.

CHRONIC SHORTNESS OF BREATH

1. Congestive heart failure (CCF): This is both right and left ventricular heart failure

Signs of right heart failure=peripheral edema, enlarged liver, raised to jugular venous pressure or engorged neck veins, shortness of breath

Signs of left ventricular failure=pulmonary edema, shortness of breath

2. Mesothelioma

- **Exposure to asbestosis e.g. shipyard worker**
- **Pleural effusion**
- **Pleural plaques**

Investigation: Pleural biopsy is the best investigation
CT scan

3. Sarcoidosis

- **Chronic shortness of breath**
- **Erythema Nodosum (a rash on the leg) on the lower limbs**
- **Chest x-ray shows bilateral hilar lymphadenopathy**
- **Raised calcium and raised ACE**
- **CT scan will show pleural effusion and pleural thickening.**

4. Chronic anaemia

- **Light headedness**
- **History of heavy periods (menorrhagia, prolonged periods or passing clots, normal periods usually last 3-5 days)**
- **History of per rectal bleed**
- **History of long term of non steroidal anti-inflammatory drugs e.g. ibuprofen, aspirin, naproxen**
- **History of long use of aspirin for heart problems or prophylaxis of stroke**
- **Shortness of breath and weakness**
- **Palpitations**

Investigation: FBC to check for haemoglobin.

5. Cryptogenic Fibrosing Alveolitis

- **This is an idiopathic disease meaning cause is not known**
- **Chronic fibrosis of the lungs, usually bilateral and its progressive**
- **It causes shortness of breath on exercise , the patient is usually hypoxic on exercise.**

Investigation: Chest X-Ray shows reticulo-nodular shadowing, honey combing(late stage)

Treatment: Steroid

6. Extrinisic Allergic Alveolitis

- **Shortness of breath which is intermittent depending on the exposure to the causative organism which is aspergillus fungus**
- **Common in farmers.**

7. Cor Pulmonale

- **Right ventricular failure secondary to lung problem commonly due to COPD or PE or pulmonary hypertension**
- **Peripheral edema, high JVP, enlarged liver, shortness of breath, ascites.**

WHEEZE

1. ASTHMA

- **Usually young patient or child**
- **Dry cough at night**
- **Wheeze and shortness of breath**
- **Precipitating factors like exercise, smoking, pets, dust**
- **There is usually history of atopy i.e. eczema, hay fever, asthma.**
- **Family history of asthma**

2. COPD - please see above.

- **Usually presents during an exacerbation**
- **Long standing history of smoking**
- **Middle aged man**

3. BRONCHIOLITIS

- **Child less than 1 year**
- **Bilateral wheeze with hyper-inflated lungs**
- **Fever, cough, running nose, cough, vomiting, sneezing, feeding difficulty**
- **Common in winters**
- **Caused by respiratory syncytial virus (RSV)**

Investigation: Nose and throat swab.

STRIDOR

1. EPIGLOTITIS

- **Usually a child**
- **Child is unwell and sick**
- **High grade fever e.g. 39 degrees**
- **Drooling of saliva**
- **Following people in room with eyes not moving their head**

NB: there is no cough in Epiglottitis.

Management: Call anaesthetist and intubate

2. FOREIGN BODY - please see above

3. LARYNGEAL CARCINOMA

- **Elderly patient with long standing history of smoking**
- **Pain in the ear**
- **Weight loss, anemia, anorexia**
- **Hoarseness of voice**

4. LARYNGEAL OEDEMA

- **Could be after house fire or anaphylaxis**
- **Please see above**

HOARSENESS OF VOICE

1. Laryngeal carcinoma - please see above

2. Laryngitis

- **Coryza symptoms (running nose, sneezing, cough)**
- **Fever**

3. Functional dysphonia

- **Usually in anxious people, can be triggered by emotions.**
- **Young female**
- **Sudden onset**
- **Can be exacerbated by laryngitis**
- **Triggered by emotions**

4. Endocrine causes:

- **Hypothyroidism (constipation, weight gain, bradycardia, cold intolerance, menorrhagia)**
- **Acromegaly (increased ring size or shoe size, weight gain, spaced teeth, protruding jaw-prognathism)**

5. Singers nodule

- **Usually in professional singers or teacher's**

Investigation: Laryngoscope.

6. Voice abuse e.g. shouting especially at the football match/ cricket match or teachers

Treatment: reassure, no investigations required

7. Vocal cord paresis

- **Trauma especially after endoscopy**

8. Recurrent laryngeal nerve palsy.

- **Usually after thyroid surgery**
- **Thyroid carcinoma especially anaplastic carcinoma (rapidly enlarging mass in neck)**
- **Aneurysm**
- **Enlarged Heart**
- **After long-term intubation**

Treatment: usually resolves after some time, only needs reassurance

APNOEA - cessation of breathing

1. Opiate overdose:

- **Small Pin-point pupils**
- **Puncture marks on the arm**
- **Slow respiratory rate <12 per minute.**

Treatment: naloxone - short acting antagonist of opiates therefore may need to be repeated as the duration of action of opiates is longer than that of naloxone.

2. Breath holding spells in children

- **Usually when they are upset and can be precipitated by trauma or when separated from the parents. Basically anything which may upset a child including falling down**
- **Usually, these children stop breathing for some time, they may turn blue or have little jerks of the limbs. Then they spontaneously start breathing after a short period of time and within 1 hour usually they completely fine.**
- **Usually there is a previous history**

Treatment: Reassure

ORTHOPNOEA

This is shortness of breath when lying flat.

This is a sign of heart failure.

Patients usually use 3-4 pillows to help elevate the bedside and prevent shortness of breath.

Usually this is at night.

CHEST PAIN

A. RESPIRATORY

B. CARDIOVASCULAR

C. MISCELLANEOUS

RESPIRATORY CAUSES

1. Pleurisy - Chest pain on inspiration, usually after pneumonia or after upper respiratory tract infection.

2. Bronchogenic carcinoma

3. Tension pneumothorax

4. Pneumonia

5. Pulmonary Embolism

CARDIOVASCULAR CAUSES

1. Myocardial infarction

2. Acute coronary syndrome

3. Ruptured Thoracic aortic aneurysm-excruciating pain usually radiating to the back.

4. Pulmonary embolism

5. Pericarditis - also chest pain worse during inspiration but relived by leaning forward. ECG shows Saddle shaped ST elevation.

6. Stable angina-pain on exercise or exertion and resolves when you rest.

7. Unstable angina – chest pain at rest and as well as exercise. The chest pain not relieving by rest and have increased in severity or duration.

MISCELLANEOUS

1. Muscular skeletal pain - especially after strenuous exercise, usually there is tenderness on the chest wall.

2. Trauma - especially rib fracture

3. Costochondritis

4. Shingles usually between the ribs as it follows the intercostal nerves.

5. Gastro-oesophageal reflux disease: There is retrosternal chest pain when lying flat.

EXAMINATION IN RESPIRATORY MEDICINE

1. INSPECTION

2. PALPATION

3. PERCUSSION

4.AUSCULTATION

INSPECTION:

1. **Central cyanosis means hypoxia:**
   - **This can be in any condition like Pneumonia, COPD or Asthma.**
   - **These patients are usually breathless and are using accessory muscle.**
   - **There is intercostal recession and sternal tug.**
   - **Investigations - Pulse oximetry and ABG.**

2. PALPATION - WHAT TO PALPATE FOR?

- **Trachea**
- **Chest tenderness**
- **Chest expansion**

1. TRACHEA

- **Central which is normal**
- **Deviated which is abnormal, but the question: Is the trachea being pushed or pulled?**

Trachea can be pushed by:

- **Tension pneumothorax**
- **Massive haemothorax**
- **Massive pleural effusion**

Trachea can be pulled by:

- **Lung collapse (Common)**
- **Pneumonectomy**
- **Lung fibrosis**

TENSION PNEUMOTHORAX

- **Common in young, thin males**
- **This is usually due to rupture of the large bullae and accumulation of air into the pleural cavity.**

- **Another precipitating factor is COPD**
- **Usually, there is hyper-resonance and reduced air entry on one side and trachea shifted on the other side.**
- **Chest expansion reduced on the same side as hyper-resonant and reduced air entry**

COLLAPSED LUNG

- **This will create an empty space**
- **There is reduced air entry on the same side where the trachea has been shifted.**
- **The trachea is being pulled by an empty space created by a collapsed lung**
- **Reduced Chest expansion on the same side, reduced air entry**
- **There is hyper-resonant on opposite to the side where there is reduced air entry, also reduced air entry on the same side.**
- **Could be due to foreign body, lung carcinoma, as a complication of major operation.**

REDUCED CHEST EXPANSION

1. Should be equal on both side which is normal

2. Reduced unilaterally

3. Reduced bilaterally

CAUSES OF REDUCED AIR ENTRY UNILATERALLY

1. Pneumothorax

2. Pneumonia

3. Haemothorax

4. Pleural effusion

CAUSES OF REDUCED AIR ENTRY BILATERALLY

1. Pulmonary oedema secondary left ventricular failure (common)

2. Lung fibrosis especially cryptogenic fibrosing alveolitis

CHEST PALPATION:

Tenderness e.g.

- **After trauma usually there is localized tenderness**
- **Shingles in which you will also find rash which run around the trunk following the nerves, usually shingles starts from back moving to the front of the trunk. Shingles is common in immune-compromised patients like elderly, patient on steroid, diabetic patient, HIV patient.**
- **Musculoskeletal pain usually after strenuous exercise in gym, especially on muscles**

PERCUSSION - can be any of the following:

1. Resonant - which is normal

2. Hyper-resonant - which can be on one side or both side

3. Dullness either or one side or both side

4. Stony dull means pleural effusion.

CAUSES OF UNILATERAL HYPER-RESONANCE

- **Pneumothorax is the commonest**
- **Large bullae**

CAUSES OF BILATERAL HYPER-RESONANT

- **COPD**
- **Bronchiolitis**

CAUSES OF UNILATERAL DULLNESS

- **Pneumonia**
- **Haemothorax**
- **Unilateral pleural effusion**
- **Lung collapse**
- **Lung abscess**

CAUSES OF BILATERAL DULLNESS

- **Left ventricular failure due to pulmonary edema**

AUSCULTATION

1. Vesicular: normal heart sounds
2. Crackles: which can be either on one side or on both sides
3. Reduced air entry (reduced breath sounds) which can be either one side or both sides

REDUCED OF BREATH SOUNDS UNILATERLLY

1. Pneumonia
2. Lung abscess
3. Pneumothorax
4. Pleural effusion

REDUCED AIR ENTRY BILATERALLY

1. Left ventricular failure due to pulmonary edema, usually on the lung bases.
2. Congestive heart failure again due to left ventricular failure

BILATERAL CREPITATIONS

Pulmonary edema due to left ventricular failure especially at lung bases

UNILATERAL CREPITATION/ CRACKLES

1. Pneumonia (Commonest)
2. Lung abscess

INVESTIGATIONS IN RESPIRATORY MEDICINE

CHEST X-RAY FINDINGS

1. Consolidation means pneumonia. Consolidation could be different.

- **Patchy consolidation can be either in mycoplasma or legionella**
- **Upper lobe consolidation usually in tuberculosis, rarely in klebsiella**
- **Bilateral interstitial shadowing usually in pneumocystis jiroveci**
- **Bilateral cavitation usually due staphylococcal pneumonia**
- **Upper lobe cavitation usually tuberculosis**

2. Enlarged heart means heart failure

3. Widened mediastinum means dissecting aortic aneurysm (Thoracic)

4. Free gas in the mediastinum means ruptured/ perforated esophagus

5. Surgical emphysema means perforation of an organ e.g. esophagus

6. Gas under the diaphragm means perforated gastrointestinal tract (perforated peptic ulcer, perforation secondary to diverticulitis)

7. Bilateral fluffy opacities means pulmonary edema due to left ventricular failure.

8. Bilateral hilar lymphadenopathy in Sarcoidosis

ARTERIAL BLOOD GAS

pH.....7.35-7.45

Pa02..... >10

PaC02.....4.5-6

HC03.....22-28

C02 is an acid and its controlled by the lungs therefore its respiratory problem

HC03 is an alkali or base, its controlled by the kidneys its therefore metabolic problem

Low PH means acidosis caused by high C02 and Low HCO3

High PH means alkalosis caused by high, HC03 and Low C02

If base excess is negative it means acidosis, if it is positive it means alkalosis.

Please follow the following steps in the interpretation of blood results;

1. Look at the pH - if it is low it is acidosis and if it is high it is alkalosis. Therefore, this step is to decide either this is acidosis or alkalosis.

2. Look at the PaC02 - if it can explain the pH then it is respiratory, if CO2 cannot explain it then it is metabolic. Therefore, this step is to decide if this is respiratory or metabolic.

3. Look at the HCO3 to confirm your findings to step 2 and also to check if there is compensation.

COMMON ACID BASE BALANCE ABNORMALITIES

1. Vomiting - you lose HCI, so patient will have metabolic alkalosis and hypokalemia

2. Diarrhea e.g. gastroenteritis - loose salts, therefore patient will have metabolic acidosis. NB: diarrhea also results in hypokalemia and hyponatremia.

3. In Villous adenoma- you lose potassium, therefore patient will have hypokalemia

4. Diabetic acidosis cause metabolic acidosis

5. Metformin causes lactic acidosis

6. Salicylates overdose metabolic acidosis.

7. Alcohol causes metabolic acidosis

From the arterial blood gas you can also work out the type of respiratory failure.

Respiratory failure is oxygen PaO2 <8.

Respiratory failure type 1 is oxygen less than 8 with normal or low PaCO2, this can be caused by PE, pneumonia.

Respiratory failure type 2 is oxygen < 8 with high CO2, this can be caused by COPD.

In type 1 respiratory failure give 100% oxygen or highest available % of oxygen or 15 L/min.

In type 2 give 24% oxygen via venture mask. The commonest cause of type 2 respiratory failure is COPD.

PNEUMONIA

Lower respiratory tract infection, also called chest infection.

Symptoms:

- **Fever, cough, shortness of breath, sputum, chest pain.**

Investigation is chest X-ray showing consolidation, indicating pneumonia.

Sputum culture is rarely done in the UK for common pneumonia, most of chest infection are treated empirically.

Treatment is antibiotics commonly empirical treatment

SPECIFIC PNEUMONIA'S

1. COMMUNITY ACQUIRED PNEUMONIA

- **Commonest cause is streptococcal pneumonia, also called pneumococcal.**
- **Commonly after recovering from streptococcal pneumonia people usually develops herpes labialis, therefore if you see herpes labialis or cold sore, its likely to be streptococcal pneumonia**
- **Chest x-ray simply shows consolidation**

Treatment is amoxicillin or co-amoxiclav or benzylpenicillin

2. Staphylococcal pneumonia

- **Usually develops after viral illness i.e. upper respiratory tract infection, influenza.**
- **Chest x-ray shows consolidation and sometimes bilateral basal cavitation**
- **Therefore, if someone has pneumonia after a common cold means he has staphylococcal pneumonia.**

Investigation: Chest x-ray

Treatment: Flucloxacillin, if MRSA resistant use vancomycin.

3. Aspiration pneumonia:

Usually in alcoholics after binge drinking and they aspirate the gastric content, sometimes after swallowing problems like stroke, parkinsonism, motor neuron disease (i.e. swallowing difficulties).

4. Haemophilus Influenza (Gram positive bacilli)

- **Usually causes infection in bronchiectasis and COPD.**
- **For bronchiectasis there might be history failure to thrive, recurrent chest infection as a child suggesting cystic fibrosis, which is a common cause of bronchiectasis due to recurrent infection.**

Investigation: Chest x-ray

Treatment: 1. Clarithromycin or 2. Erythromycin or 3. Tetracycline

5. Pseudomonas Aeruginosa

- **Commonly causes infection in COPD and Bronchiectasis**

Investigation: Sputum culture

Treatment: Ciprofloxacin, anti-pseudomonas, meropenem

6. Tuberculosis

- **Patient from African or Asia**
- **Weight loss, haemoptysis, night sweats, productive cough.**

- **Cervical lymphadenopathy**
- **If patient is not from Africa or Asia then there will be history of alcoholism and he is homeless.**

Investigation: Sputum culture and microscopy for TB. Or test for Acid fast bacilli or ZN stain.

7. Klebsiella

- **Causes infection in elderly, alcoholic and diabetics.**
- **Usually causes upper lobe cavitation.**

Investigation: Chest x-ray - upper lobe cavitation.

Treatment: Cefotaxime or imipenem

8. Hospital acquired pneumonia

Is usually caused by gram negative bacilli (Klebsiella, E. Coli, Proteus) or pseudomonas or staphylococcal.

9. ATYPICAL PNEMONIAS: all of them have got dry cough.

i) Pneumocystis Jiroveci

- **HIV patient, or homosexual or IV drug abuser or from Africa**
- **Weight loss, lymphadenopathy**
- **Low CD count,**
- **Prophylaxis for pneumocystis carinii is recommended if the CD 4 count falls below 200.**
- **Bilateral perihilar interstitial shadowing**

Treatment is cotrimoxazole

ii) Legionella Pneumophila

- **Dry cough, history of travel abroad or staying in a hotel**
- **Confusion and diarrhea is common in legionella**
- **Legionella causes hyponatremia.**

Investigation: Chest x-ray = patchy consolidation. Also you can do urine antigen for legionella.

Treatment is clarithromycin or erythromycin

iii) Mycoplasma Pneumonia

- **Flu-like illness**
- **Dry cough**

Investigation: Chest x-ray will show patch consolidation. Also cold agglutinin test is positive (clotting of RBC's)

Treatment: clarithromycin or erythromycin or tetracycline

iv) Chlamydia psittacci

- **Dry cough**
- **Contact with birds or works in a parrot shop**

Investigation: Chest x-ray

Treatment: Tetracycline or Clarithromycin

v) Chlamydia Pneumophilia is also treated with tetracycline.

ASTHMA

Asthma is an allergic inflammatory airway reaction characterized by reversible airway obstruction, bronchoconstriction leading to dry cough, shortness of breath and wheeze.

Symptoms:

- **Dry cough especially at night**
- **Wheeze**
- **Shortness of breath**
- **Usually there is family history of asthma or history of atopy i.e. asthma, eczema and hay fever.**

Precipitating factors:

smoking, pets, dust, viral illness, exercise, aspirin, NSAIDs.

Management of asthma depend so on the probability.

Low probability of asthma is symptoms associated with cold. If there is low probability then patient needs to be investigated to make sure the diagnosis is clear.

High probability of asthma is symptoms associated with exercise, or 3 or more symptoms or if family history of atopy or if past history of atopy. If high probability you give trial treatment before you treat even without investigations.

Prophylaxis

1. **Avoid triggers**
2. **Use sodium chromoglycate in exercise induced asthma or pre-exercise bronchodilator (salbutamol)**

Treatment:

1. **Stable patient: Goes to GP or outpatient department (salbutamol)**
2. **Unstable patient: Patients come to A&E and have severe symptoms**

A. **Stable patient**

Step 1: Occasional short acting beta 2 agonist inhaler e.g. salbutamol as required. If needed > 2/week or (night symptoms) or if getting exacerbation move to next step.

Step 2: Add regular Inhaled Steroid: Therefore at this stage child will be taking salbutamol as required and inhaled steroid e.g. beclomethasone 200 micrograms, if not helping go to 800 micrograms, if still not resolving then go to step 3.

Step 3: Check diagnosis, check technique (use the spacer with a mask). Add 1 dose monteleukast (leucotriene antagonist) in the evening. If 2-5 years, add leukotriene antagonist (e. g. monteleukast), formeterol. If <2 years refer to the paediatrician.

If child > 5 years add Long acting – agonist e.g. salmeterol. At this stage if not getting any benefit from long acting beta-agonist them discontinue it. But if it has added some benefit then continue it.

Step 4: Increased inhaled steroid up to maximum dose (e.g. Beclomethasone 800 µg --- 2000 µg). If child develops oral candida reduce the dose.

At this stage also consider leukotriene antagonist if not already used or modified long acting beta agonist or aminophylline

Step 5: Add prednisolone – oral

A. **Unstable patient = Acute exacerbation of asthma**

a. **Mild to moderate asthma (able to talk, Pulse < 125, PEFR > 35% of the predicted value. Oxygen > 92%.**

Treatment:

1. **Oxygen**
2. **Nebulised salbutamol 5mg every 15-30 minutes or terbutaline**
3. **Prednisolone**

a. **Severe asthma (can not speak in complete sentences in one breath or child too breathless to speak, PEFR 35%-50%, Oxygen saturation < 92%)**

Treatment:

1. **Oxygen**
2. **Nebulised Salbutamol 5mg every 15-30 minutes ± IV salbutamol**
3. **Oral Prednisolone 40-50 mg or hydrocortisone 100 mg IV**
4. **MgSO4**
5. **Aminophylline after talking to ITU**

a. **Life threatening asthma: (Silent chest, cyanosis, hypotension, bradycardia, agitation, reduced consciousness, saturations < 92%)**

1. **Oxygen**
2. **Nebulised Salbutamol ± IV salbutamol**
3. **Prednisolone or hydrocortisone**
4. **MgSO4**
5. **Aminophylline**

PREVENTION OF ASTHMA

1. Stop smoking

2. Avoid allergens (pets, dust)

3. Use sodium cromoglycate for exercise induced asthma or pre-exercise bronchodilator. If you have both options in the question choose sodium cromoglycate.

4. Avoid infection

PULMONARY EMBOLISM

- **Usually young female with risk factor e.g. history of long flight or on combined contraception.**
- **Sudden shortness of Breath, sudden chest pain.**
- **Haemoptysis**
- **Shortness of breath**

RISK FACTORS OF PE:

Malignancy, pregnancy, post operative especially hip operation and hysterectomy, combined oral contraception pills, long flight

Management of Pulmonary embolism

You need wells score.

Well's score

1. Entire leg swelling ---------- +1

2. Calf swelling more than 3 cm------- +1

3. Active malignancy---------- +1

4. I*mmobilization more than 3 days---- +1*

5. Pitting edema---------- +1

6. Collateral superficial veins---------- +1

7. Calf circumference more than 3 cm compared to other leg--- +1

8. Other diagnosis likely---------- -2

Low probability=0 or less points

Intermediate probability 1-2 point

High probability 3 or more

Management:

1. Low probability --- do D-dimer first

If D-dimer negative then PE has been ruled out

If D-dimer positive ---> start low molecular weight heparin ---> investigate with V/Q scan ---> if confirmed continue treatment and add warfarin. If V/Q scan negative stop the low molecular weight heparin.

2. If intermediate or probability ---> start treatment with low molecular weight heparin ---> investigate with V/Q scan if confirmed continue treatment with low molecular weight heparin and add warfarin, if negative then stop the treatment.

If confirmed PE then continue both low molecular weight heparing and warfarin and stop heparin when INR reaches 2. You then continue warfarin maintaining INR 2-3.

NB: In pregnancy, you use low molecular weight heparin, as warfarin is teratogenic.

Investigations in Pulmonary embolism

1. V/Q scan this is the most appropriate investigation as it expose patients to less radiation but it can only be done if CXR is normal. So if the question says CXR is normal then definitely choose V/Q scan

2. Pulmonary angiogram is the most definitive investigation and the gold standards.

3. CTPA is better than V/Q and if chest X-ray is abnormal it is always preferred.

4. D-dimer choose only if there is low probability of PE, do not choose if intermediate or high risk. Also if patient is post-operative D-dimer is not used.

NB: Maintain INR between 2-3.

TENSION PNEUMOTHORAX

As discussed earlier develops in young tall, thin men.

Please see above under the section of examination.

Investigation is Chest x-ray

Treatment: Wide bore needle or cannula in the intercostal space. This is also called needle decompression.

If none of the above are in the options, then you can choose chest drain.

CYSTIC FIBROSIS

- **This is congenital disease affecting new born babies. it is an autosomal recessive condition and there is 1:4 or 25% chance that the other next child will be affected.**
- **It is chloride channel defect**
- **As a baby there is history of recurrent chest infection, meconium ileus, failure to thrive.**
- **Usually at the age between 18-30 the patients develop bronchiectasis as a complication. Therefore watch for the age of the patient.**

Investigation: sweat test.

Treatment is symptomatic: physiotherapy, antibiotics for infection.

COPD - Chronic Obstructive Pulmonary Disease

There are 2 types COPD: emphysema type and chronic bronchitis.

This is due to smoking, therefore there is long standing history of smoking.

- **Usually middle aged men 35-50.**
- **Look at the age of the patient because long history of smoking, if elderly patient malignancy is more likely than COPD.**

Investigation: Respiratory function test.

Treatment: steroid inhalers and bronchodilator

Patient with COPD usually develops type 2 respiratory failure and therefore do not give 100% oxygen. Give 24% oxygen by venturi mask and do an ABG.

If the amount of CO_2 on 28% is increasing, then reduce to 24%.

Type 2 respiratory failure is when oxygen is less than 8 and CO_2 is high (above 6).

Type 1 respiratory failure is when oxygen is less than 8 and CO2 is normal or low.

ASPERGILLUS

Causes extrinsic allergic alveolitis -

- **There is intermittent shortness of breath depending on the exposure.**
- **It is due to exposure to aspergillus clavatus - a fungus.**

Treatment: anti-fungal e.g. amphotericin

Aspergillus can cause asthma as allergic reaction due to exposure and as well it can as a complication in Asthma.

LUNG CANCER

- **Usually elderly patient with chronic history of smoking**
- **Weight loss, anorexia, anaemia**
- **Usually this is bronchogenic carcinoma**
- **Progressive or worsening shortness of breath, haemoptysis**
- **Small cell lung cancer can cause SIADH with low Na and high ADH. Also, cushing's syndrome can be cause by small cell cancer.**
- **Squamous cell carcinoma causes hypercalcaemia**

Investigations: Bronchoscopy for bronchogenic carcinoma. CT-scan of the chest for carcinoid.

BRONCHIECTASIS

This is permanent dilatation of the bronchi due to recurrent chest infection.

Causes: cystic fibrosis, recurrent infection.

Symptoms: chronic cough, copious purulent sputum, intermittent hemoptysis

Investigation is high resolution CT scan

Treatment: postural drainage, physiotherapy.

Give antibiotics if chest infection.

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Resource view

Resource name	Rheumatology PLAB 1 Notes
Resource description	Rheumatology
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RHEUMATOLOGY NOTES

Table of Contents:

1. **SYNOVIAL FLUID ANALYSIS**

a. **Normal fluid** is clear/colourless, with </=200 WBC mm3.

a. **Non-inflammatory and Degenerative Diseases** fluid is clear/colourless, with </=5000 WBC mm3, Neutrophils </= 25%, increased viscosity.

a. **Haemorrhage Tumours, Trauma, Haemophilia** fluid is bloody/ xanthochromatic with </=10000 WBC mm3, Neutrophils </= 50%, varied viscosity.

a. **Acutely Inflamed fluid** is turbid yellow with near 1-50000 WBC mm3, Neutrophils > 60%, decreased viscosity.

a. **Septic/Infection** fluid is turbid yellow with 10-10000 WBC mm3, Neutrophils </= 90%, decreased viscosity.

a. **Osteoarthritis** fluid is clear/straw coloured with </=1000 WBC mm3, Neutrophils </= 50%, increased viscosity.

ARTHRITIS

This is inflammation of the joint. Features are pain, stiffness, redness, swelling, warmth and loss of function.

Classification of Arthritis

1. **Monoarthritis-** which means it usually affects one joint

- Septic Arthritis
- Gout
- Osteoarthritis
- Trauma (Haemoarthrosis)

2. **Polyarthritis**- affects multiple joints

- Rheumatoid Arthritis
- Psoriatic Arthritis
- Viral Arthritis (Hepatitis A, B and C)
- Reactive Arthritis
- Osteoarthritis
- SLE

3. **Oligoarthritis**

- Crystal Arthritis
- Psoriatic Arthritis
- Reactive Arthritis
- Ankylosing Spondylitis
- Osteoarthritis

RHEUMATOID ARTHRITIS

Chronic inflammatory disease affecting the peripheral joints. Usually it affects the small joints, it is symmetrical and causes stiffness, which improves as the day goes on.

Rheumatoid Arthritis

Persistent, symmetrical, deforming peripheral arthropathy usually in the 5th decade.

Female:male ratio is 2:1

Articular Features

1. Painful swollen hands and feet especially small joints but larger joints can also be involved
2. Ulnar deviation
3. Dorsal wrist subluxation
4. Swan neck deformity
5. Boutonniere deformity
6. Z-deformity of the thumb
7. Wasting of intrinsic muscles and accentuation of extensor tendon

Extra-Articular features

1. Anaemia
2. Vasculitis
3. Episcleritis, scleritis, keratoconjunctivitis sicca
4. Pleurisy & pericarditis
5. Pulmonary fibrosis

Investigations

1. X- ray will show increased soft tissue swelling, decreased joint space narrowing, bony erosions in the late stages, with or without subluxation
2. Blood test - increased ESR, CRP & platelets, decreased Hb, WCC.
3. Rheumatoid factor is +ve in 70%
4. ANA is +ve in 30 %

Treatment of Rheumatoid Arthritis:

1. Physiotherapy
2. Intra articular steroid
3. Surgery to relieve increased pain (joint replacement)
4. Oral drugs: NSAIDS (avoid on Warfarin)
5. Oral steroid with proton pump inhibitor for gastric protection
6. Disease Modifying Drugs (Sulfasalazine, Methotrexate, Gold, Cyclosporine, Azathioprine, Penicillaine, Hydroxychloroquine)

OSTEOARTHRITIS:

This is the commonest joint condition. It usually occurs in those aged more than 50 years of age and is usually monoarthritis.

Bouchard's nodes and Herbeden's nodes are usually present.

Female:male ratio is 3:1 - usually affects 50 years & above

The Hip and Knee joints are commonly affected. Usually affects big joints like hip and knee.

Symptoms

- Single joint usually affected, with pain on movement worsening by the end of the day accompanied by stiffness and joint instability
- In polyarthritis Osteoarthritis herbeden's nodes are seen in the DIP, involvement of cervical and lumbar vertebrae, knee , thumb and MCP joints

Investigation

- X-ray
- Subchondral sclerosis of cysts
- Marginal osteophytes

Treatment

- Do exercise and keep active and physiotherapy
- Regular paracetamol
- Topical NSAIDS
- Intra-articular joint steroids
- Low dose Tricyclic anti-depressant medications for night pain
- Weight reduction
- Physiotherapy and walking aids
- Joint replacement in end stage osteoarthritis

CRYSTAL ARTHROPATHIES

1. Gout
2. Pseudogout or Calcium Pyrophostate Dihydrate Arthropathy (CPPD)

1. **GOUT**

- This is acute inflammation of the joint
- It usually presents with intermittent joint pain.
- It is caused by high urate (uric acid) in the blood, which gets into the joint and cause crystals.
- Usually monoarthritis
- High Serum uric acid can occur from tumour lysis syndrome (break down of cancer cells while on treatment with chemotherapy or radiotherapy.

- Gout can also occur in the absence of high serum uric acid.
- High uric acid can be hereditary: This is when certain people generally have increased uric acid production - female:male ratio is 1:5

- Gout is usually precipitated by trauma, surgery, starvation, infection, diuretics, polycythermia rubra vera, leukaemia, cytotoxics and alcohol abuse

Symptoms

- Usually severe sudden onset of joint pain with redness, and swelling.
- Usually single joint
- Common affected joint is metacarpal phalangeal joint of the big toe or ear pinna
- Other wise any other joint may be affected
- Urate deposit called "tophi" are seen in pinna, tendons & joints
- Gout may present with renal stones due to accumulation of uric acid in the urinary tract.

Investigation

- Joint aspiration for microscopy, culture and sensitivity, which shows shows negatively birefringent crystals.

Treatment

- NSAIDS e.g. Endomethacin in acute gout
- If NSAID are contraindicated then give colchicine
- If there is renal failure both NSAIDs and colchicine are problematic so use steroids

Prevention

- Avoid prolonged fasting
- Avoid excess alcohol
- Avoid dehydration
- Lose Weight
- Use Allopurinol for prophylaxis if there are recurrent attacks.
- Avoid certain foods which contain a lot of uric acid like tomatoes, red meat etc.

PSEUDO-GOUT

- Can present as acute or chronic.
- Usually it presents like gout with acute inflammation of the joint.
- Risk factors are old age, osteoarthritis, hyperparathyroidism, dehydration, surgery or illness.

Acute

- It presents similar to gout as monoarthropathy.
- It affects different joints like knee, hip and wrist.
- It is self-limited.

Chronic

- Can present as polyarthritis like rheumatoid arthritis
- Causes destruction of the joints like osteoarthritis.

Investigation: Joint aspiration (positively birefringent crystals ion microscopy)
N.B: In Gout there is negatively birefringent crystals.

Treatment:
Acute attack: 1. NSAIDS 2. Steroids
Chronic: Methotrexate and hydroxychlolorquine may prevent attacks.

SPONDYLOARTHRITIDES

Chronic inflammatory disease of the spine and sacroiliac joints. The cause is unknown.

CLASSIFICATION OF SPONDYLOARTHRITIDES

1. Ankylosing Spondylitis
2. Enteropathic Spondyloarthropathy
3. Psoriatic Arthritis
4. Reactive Arthritis

1. **Ankylosing Spondylitis**

Typical patient is a young man of < 30 years of age with chronic back associated with morning stiffness.

Ankylosing Spondylitis is usually associated with HLA B27 genes.
Usually patients suffer from recurrent anterior uveitis (iritis)

Symptoms

- Low back pain, morning stiffness
- Progressive loss of special movements, later patient usually develop kyphosis
- Iritis (anterior uveitis), carditis, IBD
- Hip & knee involvement
- There may be a history of Inflammatory bowel disease

Investigation

- Diagnosis is made by clinical and radiological signs
- X-ray usually show irregularities, erosions or sclerosis, later squaring of the vertebrae (bamboo spine).

Treatment

- NSAIDs are usually very effective
- Exercise (encourage to keep moving)
- Autoimmune suppressant drugs like sulfasalazine, cyclosporin and

  methotrexate may be used.

1. **Enteric arthropathy:**

This is joint inflammation secondary to inflammatory bowel disease. There is usually history of Crohn's disease or Ulcerative colitis.

Usually arthritis resolves with treatment of the inflammatory disease.

1. **Reactive Arthritis**

- This is joint inflammation which usually follows either sexually treatment infection (chlamydia arthritis) or gastroenteritis (shigella, salmonella).
- There is no inflammation in the joint, its usually simply a reaction.
- It can take 1-4 weeks after gastroenteritis or urethritis before patient can develop arthritis.
- Reactive arthritis is a sterile arthritis typically affects 1 in 4
- It can occur as part of Reiter's syndrome i.e. urethritis, conjunctivitis and arthritis

Risk Factors

a. Secondary to Chlamydia trachomatis (sexually transmitted infection)
b. Camphylobacter
c. Salmonella
d. Shigella
e. Yersinia

Symptoms

- Affects large joints especially lower limbs
- Iritis

Investigation

- Increased ESR & CRP
- Stool culture if diarrhoea
- Serology
- X-ray shows periostitis with effusion

Treatment:

NSAIDs are the first choice.

- If symptoms are lasting more than 6 months then consider using steroids, sulfasalazine & methotrexate

1. **Psoriatic Arthritis**

It is a manifestation of psoriasis. It can present before skin manifestation of psoriasis.

- Polyarthritis
- Asymmetrical
- Affects the spine
- It is associated with nail changes in 80% of cases = Anychilosis
- Affects the extensor aspects of the joints
- Can effects multiple joints like rheumatoid arthritis but rheumatoid factor is negative which differentiate it from rheumatoid arthritis.
- **Sausage shaped fingers** are classical of psoriatic arthritis

1. **Viral Arthritis**

Usually happens after a viral illness. Patient recently may have suffered from cold e.g. sneezing, running nose, temperature.

There may be rashes on the body due to viral illness.

6. **Septic Arthritis**

Acute painful joint with swelling and fever.

This is an infection of the joint unlike reactive arthritis, which is simply inflammation of the joints.

N.B: Any acutely swollen joint is septic arthritis until proven otherwise.

Symptoms

- Acute onset of joint pain
- Fever
- Redness around affected joint
- Swelling
- Loss of movement in the joint and unable to bear weights

Risk Factors

- Common in immune-compromised patients e.g. those on steroids and in those with HIV and cancer.
- Patient with pre-existing infection somewhere else e.g. UTI, pneumonia.
- Pre-existing joint disease e.g. Rheumatoid Arthritis, Diabetes Mellitus, Chronic renal failure (CRF)

Investigation:

- Joint aspiration for microscopy, culture and sensitivity
- Blood culture if there is high fever

Treatment:

Intravenous antibiotics: Flucloxacillin

AUTOIMMUNE CONNECTIVE TISSUE DISEASES:

1. SLE
2. Scleroderma
3. Sjogren's Disease
4. Behcet's Disease
5. Idiopathic Inflammatory Myopathies
6. Polychondritis

Systemic Lupus Erythematosus

Multi systemic autoimmune antibodies are produced against the variety of antigen

Female:male ratio is 9:1

Seen usually in pregnancy, in Afro-Caribbeans and Asians around the age of 30-40, associated with HLA B8, DR2, DR3, has remitting & relapsing course

SYMPTOMS

- Malar rash
- Discoid rash,
- Butterfly rash on the face
- Photosensitivity
- Oral ulcer
- Arthritis
- Serositis, pleuritis, pericarditis
- Renal disorders: proteinuria
- CNS: seizures, psychosis
- Haematological disorder: haemolytic anemia, leukopenia, lymphopenia, increased platelets
- D**NA antibody 60% is specific for SLE**
- **Anti-Sm antibodies 20-30%**
- Anti-nuclear antibody (+ve in 95%)
- FBC to reveal anaemia and decreased other blood cells

TREATMENT

- Refer to rheumatologist
- NSAIDs and sun creams
- Hydroxychloroquine
- High dose prednisolone
- Cyclophosphamide if there is renal involvement
- Azathioprine for steroid sparing

SCLERODERMA

The name *scleroderma* is derived from the Greek for 'hard skin' and emphasises the dermatological component of the disease.

Types of systemic sclerosis:

SSc is classified into two main types, according to the extent of skin involvement

A. **Limited cutaneous systemic sclerosis (lcSSc), or limited scleroderma**

- 70% of SSc cases.
- Affects only the face, forearms and lower legs up to the knee.
- The older term for limited scleroderma is CREST syndrome

Calcinosis
Raynaud's disease
Esophageal dysmotility
Sclerodactyly
Telangiectasia

A. **Diffuse cutaneous systemic sclerosis (dcSSc), or diffuse scleroderma**

- 30% of SSc cases.
- Involves also the upper arms, thighs or trunk.

Aetiology

The cardinal features of SSc are excessive collagen production and deposition, vascular damage, and inflammation or autoimmunity.

Clinical features:

Common presenting symptoms are raynauds phenomenon, skin hardening in hands or face, and oesophageal symptoms.

Early symptoms can also be non-specific – e.g. fatigue, musculoskeletal pains and hand swelling.

Both limited and diffuse scleroderma can involve internal organs, and the severity of skin changes does not necessarily reflect the severity of internal organ involvement.

LcSSc

- Generally a milder disease, with less skin involvement, slow onset and slow progression.
- The slow onset may mean that symptoms are relatively unnoticed until internal complications occur.

DcSSc

- Usually a more rapid onset, with skin thickening and Raynaud's phenomenon occurring together or within a short interval. The skin changes may spread rapidly, within a few months of disease onset.
- Skin changes can remit after several years, with softening of the skin and significant improvement in mobility.
- Internal organ involvement is more common.

General features

- Fatigue
- Weight loss

Skin features

Signs in the hand:

- Swelling (non-pitting oedema) of fingers and toes - a common early sign; digits may look sausage-like; hand movement may be limited.
- Skin becomes hard and thickened - this may limit joint movement or cause joint contractures; in the fingers, this is known as sclerodactyly.
- Swelling and sclerosis reduce hand movements, so patients may be unable to make a fist, or to place the palmar surfaces together - the 'prayer sign'.
- Fingertips may have pitting, ulcers or loss of bulk from finger pads.
- Raynaud's phenomenon. This is the most common symptom and is present at some point in 90% of cases.
- Raynaud's phenomenon with puffy fingers is thought to be a cardinal sign of likely SSc.
- Calcinosis - nodules or lumps of chalky material which may break through the skin.
- Face and mouth:
- Tightening of facial skin.
- Tight lips (microstomia)
- Telangiectasia.
- 'Salt and pepper' appearance of skin, due to areas of hypopigmentation and hyperpigmentation.
- Dry or itchy skin; reduced hair over affected skin areas.

Musculoskeletal features

- Joint pain and swelling.
- Myalgia (due to inflammatory myopathy).
- Restriction of joint movement, contractures and muscle atrophy due to skin sclerosis.
- Tendon friction rubs - palpable/audible over the flexor/extensor tendons of the hands, knees and ankles.

GI features

- Heartburn and reflux oesophagitis
- Oesophageal scarring and dysphagia
- Delayed gastric emptying – e.g. fullness after meals
- Reduced small bowel motility - can cause bacterial overgrowth, with bloating, malabsorption, diarrhoea and malnutrition
- Constipation due to reduced colonic motility

Pulmonary features

Pulmonary fibrosis (interstitial lung disease):

- Occurs in as many as 75% of scleroderma patients, but only a few develop end-stage disease
- Causes restrictive lung disease
- Symptoms and signs: exertional dyspnoea, cough, coarse basal crackles

Pulmonary hypertension:

- Occurs in about 10-15% of patients with scleroderma.

Investigations

Auto-antibodies:

- **Antinuclear antibody** - positive in 90-95% but not specific to SSc.
- Other autoantibodies in SSc
- **Anti-topoisomerase 1** (Scl 70) antibody - strongly associated with lung fibrosis and with renal disease.
- **Anti-centromere antibody (ACA)** - seen almost only in patients with lcSSc, and is associated with increased risk of pulmonary hypertension
- **Anti-RNA polymerase III antibody** - associated with dcSSc and especially with kidney involvement.
- **Anti-fibrillarin (anti-U3RNP) antibody** - associated with heart involvement, pulmonary hypertension, kidney involvement and myositis.
- **Anti-PM-Scl antibody** - strongly associated with the combination of myositis and scleroderma.
- **Anti-U1RNP (anti-nRNP) antibody** - associated with joint involvement and overlap syndromes.

Skin biopsy

Treatment:

1. Cyclophosphamide
2. Oral steroids e.g. prednisolone
3. Nifedipine for Raynauds phenomenon

POLYMYOSITIS & DERMATOMYOSITIS

Both of these conditions are characterized with symmetrical proximal muscle weakness associated with dysphagia, facial oedema, and respiratory muscle weakness may develop.

Skin signs: (esp. for dermatomyositis)

- Macular rash over back & shoulder (shawl sign)
- Red papules over extensor surface of phalanges
- Rough cracked skin on lateral palmer surface of fingers and hands
- Patient may fail to stand up from chair

Systemic signs: - fever - Raynaud's phenomena

- Lung involvement
- Polyarthritis
- Calcification
- Retinitis
- Myocarditis
- Arrhythmia
- Dysphagia
- Gut dysmotility

Investigation:

1. Muscle enzyme (ALT, CK Creatinine Phosphokinase). Creatinine phosphokinase is usually raised in myositis and dermatomyositis which differentiate them from polymyagia rheumatic or giant cell arteritis
2. Muscle biopsy
3. Antibodies = anti MI-2, anti JO

Treatment:

1. Rest
2. Prednisolone
3. Immunosuppressive can be used (e.g. Azathioprine, Methotrexate, Cylophosphamide, Cyclosporin)

VASCULITIS

Inflammatory disorder of the blood vessel walla and the vessels of any organ.

Classification

1. Large Vessels Vasculitis e.g. GCA or Takayasu disease
2. Mediums Vessels Vasculitis

PAN
Kawasaki Disease
Small Vessels Vasculitis

ANCA + ve

- GN
- Wegener's Granulomatosis
- Microscpic Polyangitis
- Churg Straus Syndrome

ANCA –ve

- Henoch Schonlein Purpura HSP
- Good Pasture Syndrome

Features of Systemic Vasculitis

→ Fever, malaise, weight loss, muscle pains
→ can affect any age

It can affect any organ

1. **Cardiac** - loss of pulse, cardiac failure, angina & myocardial infarction
2. **Pulmonary** - dyspneoa, haemoptysis
3. **GIT**- abdominal pain, malabsorption
4. **ENT & EYE** – epistaxis, deafness, episcleritis, visual loss
5. **Renal** – hypertension, haematuria, proteinuria, acute/chronic renal failure
6. **Skin** – purpura, ulcer, digital gangrene
7. **CNS** – fits, hemiplegia, psychosis, confusion & decreased cognition
8. **General** – fever, malaise, weight loss, myalgia, arthralgia

DIAGNOSIS: CLINICAL + HISTOLOGICAL + ANGIOGRAPHIC

TREAMENT:

1. High dose Prednisolone
2. Cyclophosphamide, methotrexate or azathioprine in major organ involvement

GIANT CELL ARTERITIS

- Large vessel vasculitis
- It is associated with Polymyalgia Rheumatica in 25%
- Common in elderly
- Rare <55 years (usually patient above 50 years)

Symptoms:

1. Unilateral headache worse when the patient combs hair
2. Weight loss
3. Temporal artery and scalp tenderness
4. Jaw claudication
5. Sudden loss of vision (usually irreversible)

If not treated patient may loss the other eye

Investigations: Initial = ESR

Definitive: Temporal Artery Biopsy

Management:

If ESR is increased, start the patient on intravenous methylprednisolone and then arrange temporal artery biopsy.

You need to start treatment as soon as possible and not wait for temporal artery biopsy.

Once diagnosis of giant cell arthritis has been confirmed start patient on long term high dose oral prednisolone for 2-3 years:

SIDE EFFECTS OF LONG STEROID USE:

1. **Hypertension:** patient needs regular follow up
2. **Hyperglycaemia:** If patient on steroids develops hyperglycaemia or diabetes treat with insulin. Usually short acting insulin once in the morning if the patient is being given steroid once in morning or twice a day optimal insulin if patient is being given steroid more than once a day.
3. **Weight:** Patient be informed and advised to control weight using exercise and diet.
4. **Peptic ulceration:** start patient on prophylactic proton pump inhibitor
5. **Osteoporosis**: In elderly patients, prophylactic biphosphanates may be prescribed.
6. **Cataract:** Regular eye check; if significant cataract the only treatment would be cataract surgery.
7. **Immunosuppression:** Patients are prone to infection and they may develop infection. Patients should be warned that if they develop a temperature they need to seek medical help.

TAKAYASU'S ARTERITIS (Aortic Arch Syndrome)

This is large vessel vasculitis.

Idiopathic narrowing carotid, subclavian and renal arteries.

Usually affects females around 20 - 40 years

SYMPTOMS

- Visual impairment, cataract, atrophy of iris, hemiplegia, vertigo, syncope, fit, systolic murmurs, it can affect any vessels above and below the clavicle, hypertension
- General symptoms like weight loss, fatigue

INVESTIGATION

- ESR more than 40mm/hour
- Aortography

TREATMENT:

Prednisolone, Methotrexate

POLYARTERITIS NODOSA

PAN is a necrotising medium vessel vasculitis that causes aneurysms and thrombosis in the medium size vessels leading to infarction of the effected organ.

Symptoms:

- General features are fever, weight loss, and joint pain
- Skin changes: livedo reticularis, urticaria
- Renal: glomerulonephritis
- Heart: Myocardial Infarction, Angina
- CNS: fits, stroke, psychosis

Investigation:

1. ESR
2. ANC
3. Angiography

Treatment: Steroids, Cyclophosphamide

POLYMYALGIA RHEUMATICA

- Common in patient over 70 years, usually females

SYMPTOMS

- Symmetrical joint pains, morning stiffness of shoulders and proximal limb muscles of more than 1 month
- Patient usually find it difficulty to stand up from the chair due to proximal limb muscle weakness
- Fever, fatigue, weight loss
- It is usually associated with giant cell arteritis

TREATMENT

- Prednisolone

PLASMA ANTIBODIES AND THEIR COMMON CONDITIONS

1. **Rheumatoid Factor**

- Scleroderma
- Sjogren Syndrome
- Rheumatoid arthritis
- Juvenile Arthritis

- SLE

1. **ANA (Anti Nuclear Antibodies)**

- SLE
- Sjogren
- Scleroderma

1. **Anti Histone**: Drug induced SLE

1. **Anti- Double Stranded DNA**→ very specific for SLE

1. **Anti- Phospholipid Antibodie**s: Anti-phospholipid Syndrome and SLE

1. **Anti-Centromere Abs**: Limited Systemic Sclerosis

1. **Anti- Ro:** SLE, Sjogren, Systemic Sclerosis

1. **Anti-SM**: SLE

1. **Anti-Jo and Anti MI-2**: Polymyositis and Dermatomyositis

1. **Anti SLE 70**: Diffuse Systemic Sclerosis

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SURGERY LECTURE NOTES

OVERVIEW OF TOPICS

I. **Pre-operative Considerations**
II. **Post-operative Complications**
III. **Neck Lumps**
IV. **Breast**
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XIV. **Upper Gastrointestinal Bleeding**
XV. **Acute Abdomen**
XVI. **Procedures And Relevant Anatomy In Surgery**

I. PRE-OPERATIVE CONSIDERATIONS

PRE-OPERATIVE ASSESSMENT
These are investigations that are done before patient goes to theatre.

All patients who are fit and well must have at least full blood count done before they go to theatre. This is standard procedure.

INVESTIGATIONS

1. **Full blood count:** even fit and well patients must have at least FBC done

   This is to check for Haemoglobin levels

   There are 2 types of operations:

A. Elective or planned operation
B. Emergency operation

Normal haemoglobin levels:

- >11.3g/dl in women
- >13.5g/dl in men

For emergency operations:

- Even if Hb is low, always proceed with **emergency** operations.

- If Hb <8 g/dl then transfuse and stabilise first before proceeding.

For elective operations:

- Only proceed if Hb >10 g/dl.
- If Hb <10 g/dl then defer the operation and investigate first.
- If Hb <8 g/dl you must also transfuse.

N.B: Hb <8g/dl = do blood transfusion

1. **ECG** if you suspect atrial fibrillation or if irregular pulse (atrial fibrillation) or history of atrial fibrillation.

1. **Echocardiogram**: do it if there are signs of valvular disease or left ventricular failure

1. **Refer for specialist opinion** if severe problem e.g. recurrent angina attacks (just doing an ECG is not enough)

1. **Blood Glucose** in diabetes

1. **Blood pressure monitoring** if hypertensive

1. **Respiratory function test** if respiratory problem e.g. asthma (do PEFR) or COPD

1. All Afro-Caribbean do **sickle cell test** (sickle cell disease is common in Afro-Caribbean race)

1. IV drug abuser or homosexual: do an **HIV test** (always take consent before doing it)

1. **Chest X-Ray**: do it if cardiorespiratory function or age >65

1. **Clotting Screen**: if with history of easy bleeding after minor procedures like dental extraction.

PRE-OPERATIVE BOWEL PREPARATION

1. **Oesophago-duodensoscopy, ERCP, closure (reversal) ileostomy:** do not require any bowel preparation.

1. **Colonscopy, rectoplexy, right or left hemicolectomy, pancolectomy, sigmoidectomy, anterior resection, abdominoperineal resection, Hartman's reversal** – all require full bowel preparation.
    a. Give 1 sachet of Picolax a day before surgery at 08:00 and 14:00. Picolax = sodium picosulphate. You can also use magnesium citrate.
    b. Patient can eat low residue foods whilst taking bowel preparation

1. **Haemorrhoidectomy, examination under general anaesthesia, flexible sigmoidoscopy, proctoscopy, anal** fissure – all require phosphate enema on the day of surgery

PRE-OPERATIVE MEDICATION

1. Aspirin can continue until operation
2. Target pre-operative INR for patients on Warfarin is INR <2.5. Warfarin must be stopped 5 days before the operation and be given heparin pre-operatively
3. In diabetic patients start insulin on the morning of surgery (sliding scale)
4. Antibiotics should not be given routinely to people undergoing a dental or surgical procedure even if they are high risk
5. Antibiotics for prevention of infective endocarditis should only be given if the operation is infected and the risk of bacteraemia is high

ANTIBIOTIC PROPHLAXIS IN SURGERY
Type of Surgery: Gastric/oesophageal surgery
Common Pathogens: Enteric gram –ve bacilli, gram +ve cocci
Antibiotic of choice: Single dose gentamicin IV **OR** cefuroxime IV

Type of Surgery: Colorectal surgery
Common Pathogens: Enteric gram –ve bacilli, enterococci, anaerobes
Antibiotic of choice: Single dose gentamicin IV + metronidazole IV/PR **OR**
cefuroxime IV + metronidazole IV **OR**
co-amoxiclav IV alone

Type of Surgery: Appendicectomy
Common Pathogens: Enteric gram –ve bacilli, enterococci, anaerobes
Antibiotic of choice: Single dose gentamicin IV + metronidazole IV/PR **OR**
cefuroxime IV + metronidazole IV **OR**
co-amoxiclav IV alone

Type of Surgery: Biliary surgery (open)
Common Pathogens: Enteric gram –ve bacilli, enterococci, clostridia

Antibiotic of choice: Single dose cefuroxime IV + metronidazole IV/PR **OR**
gentamicin IV + metronidazole IV/PR

Type of Surgery: ERCP
Common Pathogens: Enteric gram –ve bacilli, enterococci, clostridia
Antibiotic of choice: Single dose gentamicin IV **OR**
ciprofloxacin IV/PO

Type of Surgery: Vascular surgery
Common Pathogens: S. aureus, S. epidermidis, anaerobes in diabetes, gangrene, or undergoing amputation
Antibiotic of choice: Single dose cefuroxime IV **OR** ciprofloxacin IV. Add Metronidazole for suspected anaerobe infection

Type of Surgery: Lower limb amputation/major trauma
Antibiotic of choice: Benzylpenicillin 300-600 mg qds for 5d **OR (**for penicillin allergy) metronidazole 400-500 mg tds

TYPE OF OPERATIONS

A. Day case surgery (patient is not admitted)
B. Inpatient operation

All patients can have a day case surgery except in the following situation:

1. Mentally retarded or learning disability (may not be able to recognize the complications)
2. Those who live alone
3. Infection at the site of an operation
4. People with severe heart diseases

II. POST-OPERATIVE COMPLICATIONS

A. General complications
B. Specific complications
C. Wound problems

GENERAL COMPLICATIONS

1. **Fever (pyrexia)** causes could be:

a. **Pneumonia**: (cough, fever, shortness of breath) Usually 48 hours onwards. **Investigation: CXR**, **Rx: antibiotics (amoxicillin)**

a. **Urinary Tract Infection**: commonly due to catheterization. Causes confusion in elderly, dysuria, frequency, **Investigation: MSU**, **Rx: antibiotics (trimethoprim)**

a. **Atelectasis**: Lung collapse, common in people who smoke. Chest pain and mild fever. Usually within first 48 hours after an operation which is close to the lungs especially splenectomy, **Investigation: CXR to rule out pneumonia**, **Rx: physiotherapy**

a. **Would infection**: Usually after 5 days post op. Presents with discharge from the wound, redness and tenderness around the wound, Investigation: wound swab, **Investigation: Microscopy, culture and sensitivity. Rx: Antibiotics**

a. **Intraabdominal abscess -** (Subphrenic abscess or subhepatic abscess) Usually after 5 days post op. Presents with swinging fever. **Investigation: CT abdomen, USS abdomen**. **Rx: Incision and drainage**

a. **Anastomotic leak**: usually after bowel resection. Any physiological change after bowel resection is always anastomotic leak until proven otherwise. Does not immediately occur after the operation. Develops after patient has started eating and drinking, usually on day 3-7 post op.

Investigation: CT abdomen. **Rx: Antibiotics broad spectrum IV immediately (cefuroxime + metronidazole), Laparotomy**

2. Intraabdominal Bleed - usually few hours after surgery
Management: Laparotomy

3. Confusion

1. **Infection** – UTI & pneumonia (especially in elderly) Usually after 5 days post op.

1. **Medication** e.g opiate usually used during an operation.

4. Hypoxia

1. **Alcohol withdrawal** = **delirium tremens.** Usually develops 10-72 hrs after last alcohol intake, usually there is high MCV. Symptoms: agitated, aggressive, confused, shaky, tremors, visual hallucinations usually insects crawling in blanket.

1. **Shortness of breath or dyspnea**

a. **Pulmonary embolism** = chest pain, haemoptysis, 5 days onwards after an operation

a. **Pulmonary oedema** = post operative pulmonary oedema almost always due to fluid overload

a. **Pneumonia**

a. **Pneumothorax** due to ventilation pressure especially if there was a small pneumothorax. Therefore if a patient has a small pneumothorax a chest drain must be inserted.

a. **Myocardial infarction** = chest pain radiating to the left arm.

1. **Hypotension**

a. Post operative hypotension almost always is due to **bleeding** so give **intravenous fluids**.

a. Medical conditions like Myocardial infarction, pulmonary embolism, sepsis

8. Oliguria
Post operative oliguria is almost always due to **inadequate fluid replacement** so **give fluids.**

9. Anuria
Post operative anuria almost always is due to **blocked catheter** , so **check catheter**

10. Post operative hyponatraemia
1. SIADH, especially after brain surgery
2. Over hydration, especially with colloids

11. Deep venous thromboembolism
Symptoms: unilateral calf swelling, pitting oedema, calf pain
In post operative patients we do not use D-dimer as an investigation. The investigation of choice is always **compression ultrasound scan**.

SPECIFIC POST OPERATIVE COMPLICATIONS

1. **Mastectomy**

Common complication is lymphoedema (arm becomes swollen)
Management: physiotherapy and arm exercise.

1. **Throidectomy**

a. **Recurrent Laryngeal Nerve Palsy**, presents with hoarseness of voice, usually resolves after sometime.

Management: Reassurance

a. **Tracheal obstruction by haematoma** usually presents with acute shortness of breath and stridor immediately after the operation, commonly when the patient is still in the recovery room.

Management: release the stitches on bedside

a. **Hypocalcaemia** usually due to hypoparathyroidism which causes hypocalcaemia.

Patients presents with tetani, Checkvostek's sign and Trouseau's sign (carpopedal spasm), muscle irritability
Checkvostek sign = on tapping on the angle on the jaw there is twitching of the muscles of the face.
Trouseau's sign = on occluding the brachial artery with an inflated BP cuff the wrist and fingers flex and draw together (carpopedal spasm)

4. POST TURP (Transurethral resection of the prostate) SYNDROME
The irrigation fluid used to visualise and distend the urethra and bladder gains intravascular access through the venous circulation causing dilutional hyponatremia.
Management: Fluid restriction

5. APPENDICECTOMY
Common complication is **abdominal or pelvic abscess** especially if it's perforated or gangrene appendicitis. There is usually swinging fever. **Investigation: CT scan abdomen, or abdominal ultrasound scan. Management: Incision and drainage.**

6. SPLENECTOMY
Spleen takes part in immune system so splenectomy causes low immune function and patients have recurrent infections. Prophylaxis vaccination is required against the following infections:

i. Pneumaccocal
ii. Meningococcal
iii. Haemophilus influenza

Patients also require long term antibiotics prophylaxis.

7. ABDOMINAL SURGERY

a) Paralytic ileus usually presents with abdominal distention, constipation, vomiting and reduced bowel sounds. Occurs few days after the operation. No abdominal pain.
Investigation: Plain abdominal X-ray (dilated bowel loops)
Mgt: Nasal gastric tube and Intravenous fluid.

a. **Obstruction secondary to adhesions**. This will occur after weeks, months or years. Cardinal symptoms: abdominal pain, vomiting, constipation, abdominal distension, increased bowel sounds.

1. **ERCP**

a. **Acute pancreatitis** – abdominal pain
b. **Cholangitis**

C. WOUND PROBLEMS

Wound swelling, bleeding or discharge needs inspection/exploration of the wound.

1. **Laparotomy wound dehiscence**

Dehiscence of laparotomy wound is spontaneous opening of deep suture layers with or without superficial layer
Clinical features:

1. Serosanguinous discharge from wound
2. Usually 7-10 days post op

Treatment: Resuture wound

1. **Wound bleeding**

Usually bleeding is minor and settles spontaneously.
Clinical features:

1. Wound oozing
2. Wound hematoma on palpation

Treatment: If minor bleeding, try gentle pressure for 5 minutes. If ongoing large amount of patient, patient may need to go to theatre

1. **Superficial wound Infection and Abscess**

Clinical features:
1. Wound pain
2. Pyrexia
3. Pus like discharge
Treatment: Inspection/exploration of wound.
If temperature >37.5, take blood cultures, CRP, FBC, U&E.
If abscess wound drainage, take swab for microscopy, culture and sensitivity

III. NECK LUMPS

Neck lumps are located either in the **anterior triangle, posterior triangle** or **midline**

Anterior triangle lumps

1. Branchial cyst
2. Carotid body tumour
3. Parotid tumour

Midline lumps

1. Thyroglossal cyst
2. Thyroid lump
3. Dermoid cyst

Posterior triangle lumps

1. Cervical rib
2. Cystic hygroma
3. Pharyngeal pouch
4. Subclavian aneurysm

1. BRANCHIAL CYST

Lump containing cholesterol cyrystals located in the anterior triangle. Usually before age of 30 years. It emerges under the anterior border of the sternocleidomastoid muscle where the upper 1/3 meets the lower 2/3.
Investigation: USS, FNAC
Treatment: Surgical removal

2. Carotid body tumour aka Chemodectoma

Located in anterior triangle. It moves side to side but not up and down.
It may be pulsatile but usually does not cause bruit. It is located just anterior to the upper 1/3 of sternocleidomatoid muscle.

Investigation: Doppler USS, arteriography
Treatment: Surgical extirpation.

3. Parotid tumour

Located in the anterior triangle at the upper posterior region at the angle of the jaw.
Usually patient age >40 years
Investigation: USS, mumps test will be negative

Treatment: Surgical

4. Thyroglossal cyst
- Transillimunating midline lump which moves on tongue protrusion but not on swallowing
Investigation: USS
Treatment: surgical removal

5. Thyroid lump
- Midline lump which moves on swallowing but not on tongue protrusion
Investigation:

1. **All patients with thyroid nodules must have TSH measurement. If low, then measure T4 and T3.**
2. **USS recommended in patients with atypical solitary nodules and multiple goiter**
    a. **If it is a CYST then treatment is surgical removal**
    b. **If it's SOLID then FNAC (Fine needle aspiration cytology). FNAC is recommended in all patients with solitary nodules.**

THYROID CANCER
Risk factors:

- Pre-existing goiter
- Radiation of neck in childhood

Types including Frequency & Clinical Features

- **Papillary** (60%) – solitary thyroid nodule
- **Follicular** (25%) – Slow-growing thyroid mass, symptoms are usually from distant metastases
- **Anaplastic** (10%) **-** rapidly growing thyroid mass causing tracheal and oesophageal compression
- **Medullary** (5%) – Thyroid lump, may have MEN II A (medullary thyroid carcinoma, pheochromocytoma, hyperparathyroidism) or MEN ii B (medullary thyroid carcinoma, phaeochromocytoma, multiple mucosal neuromas, Marfanoid habitus) syndrome

Management:

- **Papillary**

  Surgery: total thyroidectomy & removal of involved lymph nodes
  Adjunctive tx: L-thyroxine to suppress TSH (it stimulates papillary tumour growth)
  Prognosis: Excellent

- **Follicular**

  Surgery: thyroid lobectomy or total thyroidectomy if metastasis are present
  Adjunctive tx: radioactive iodine for distant metastases and L-thyroxine for replacement therapy to suppress TSH

- **Anaplastic**

  Surgery: only palliative to relieve pressure symptoms
  No radiotherapy/chemotherapy
  Prognosis: Very poor

- **Medullary**

  Exclude phaeochromotyoma before treating
  Surgery: total thyroidectomy & excision of regional lymph nodes

6. Dermoid cyst
Midline lump that does not move on swallowing or tongue protrusion.
If patient is less than 20 years the dermoid cyst is likely.
Investigation: USS
Treatment: Surgical removal

7. Cervical rib =Thoracic outlet syndrome
Located in the posterior triangle.
It is an extension of C-7
It can cause compression of upper arm vein or nerves therefore it can cause tingling and numbness or swelling of the arm.
Symptoms depend on the compressed structure.
Investigation: Cervical spine X-ray
Treatment: Surgical removal

1. **Cystic hygroma**

Located on the posterior triangle. These are massively distended lymphatic vessels. that can cause compression of airway.
They present at birth and transilluminate brightly.
Investigation: USS
Management: Surgical removal

1. **Pharyngeal pouch**

Located in the posterior triangle. It is a diverticulum of the esophagus which comes out between the inferior pharyngeal constrictor muscles.
Symptoms: regurgitation of undigested food particles, halitosis, , swelling in the neck, bulging in the neck after drinking

Investigation: If it presents as a **mass** in the neck then investigation is **USS**
If it presents as a **dysphagia** then investigation is **barium meal**

Treatment: surgical

1. **Subclavian artery aneurysm**

It is a pulsatile mass located n the posterior triangle at the base of sternocleidomastoid muscle

Investigation: Doppler USS
Treatment: Surgical repair of the aneurysm

IV. BREAST

Symptoms of the breast;

1. Pain
2. Lump
3. Breast cancer
4. Nipple or skin changes
5. Discharge

1. PAIN

Pain in the breast is called mastalgia
Mastalgia can either be cyclical or non cyclical

Cyclical mastalgia - pain occurs every month before periods.
Mx: reassure patient.

Non Cyclical mastalgia - needs to be investigated just like a lump n the breast.

1. LUMP IN THE BREAST

All patients with breast lump must undergo triple assessments.

First assessment: Clinical examination of the breast including axillary lymph nodes.

Second assessment: Imaging
If a woman is **< 35** years then perform **USS** scan only
If she is **35 or above,** do **mammography first and then USS**

Third assessment: Cytology. In either case you have done an ultrasound scan which will show whether the lump is cyst or solid.

If it's a **cyst** perform **FNAC**. Further management depends on the type of fluid aspirated:

i. if **clear fluid** just **aspirate** and **reassure the patient**.
ii. If **blood stained, aspirate** send to lab for **cytology**
iii. If **clear fluid but residual mass** perform **core biopsy**.

iv) If the **lump** is solid perform **core biopsy**.

EXAMINATION
If a lump is **mobile**, not attached to underlying structure, firm in consistence, smooth surface = it's likely to be **fibroadenoma**, especially in a young patient.

If **non-mobile** lump, **hard** in consistency, **attached to underlying structure**, **irregular** surface = it's likely **carcinoma**.

If there are **lumps in the axilla** it means **carcinoma** because that is a sign of metastasis.

If there are **no palpable masses** the investigation of choice is **stereotactic biopsy**.

Lumpiness of the breast especially in the upper outer quadrant but **no dominant mass = Benign Breast Change**.
Women usually in 30's presenting with multiple cysts which may be associated with pain or green brownish discharge from the nipple.
Also called **BENIGN BREAST DISEASE, FIBROADENOSIS, FIBROCYSTIC CHANGE, FIBROCYSTIC BREAST DISEASE,**
Management: Triple assessment

Family history=if patient has got **no symptoms** but just present because she has got family history of breast cancer then do **genetic testing and counseling**.

1. BREAST CANCER

ALL BREAST LUMPS REQUIRE TRIPLE ASSESSMENT.

Risk factors

- Strong family history of breast cancer (genetic factors – BRCA 2 gene)
- Early menarche and late menopause
- Nulliparity

Clinical Features

- Palpable, hard, irregular, fixed breast lump, usually painless
- Nipple retraction and skin dimpling
- Nipple eczema in Paget's disease
- Peau d'orange (cutaneous oedema secondary to lymphatic obstruction)
- Palpable axillary nodes

Investigations: Triple assessment

Treatment: Early breast cancer treatment is aimed at local control with wide local excision, lymph node treatment and prevention of systemic relapse.
Treatment of late breast cancer is usually palliative and mostly medical

1. **SKIN CHANGES**

1. If nipple skin changes and areola area e.g eczematous changes or inflammatory changes then it's likely to be Paget's disease, especially if unilateral.

Investigation: open biopsy or punch biopsy.

1. If **nipple retraction or peu de orange** or ulcer then **breast cancer** is the diagnosis.

1. If there is an **ulcer** on the breast do biopsy of the ulcer. Ulcer means **cancer**.

5. DISCHARGE FROM THE NIPPLE

1. Blood stained discharge can be caused by :
   a. **Paget's disease**
   b. **Duct papilloma**, esp. if discharge is from the duct, usually single duct.

Investigation: Ductography/ductogram

   a. **Breast cancer**

1. Clear discharge is caused by **intraduct papilloma** (discharge from the duct).

Investigation: Ductography/ductogram

1. Orange, yellow creamy, green discharge is caused by **ductasia**, discharge from multiple ducts.

Investigation: Ductogram

1. Purulent discharge is caused by **breast abscess**, common in breast feeding mother. The causative organism is staphylococcus aureus. **Rx: flucloxacillin**.

1. Milky discharge is caused by **galactorrhoea.** Causes include prolactinoma, side effect of anti-psychotic medications, and physiologic in lactating mothers.

MESENTERIC ISCHAEMIA

Acute Mesenteric Ischaemia
Symptoms:

- Sudden onset of severe abdominal pain, with soft abdomen is soft, no findings on examination of the abdomen.
- Also per rectal bleed
- Severe hypovolaemia

Risk factors: AF, MI (mural thrombus), aortic aneurysm, valvular heart disease
Cause: Emboli
Investigation: Arteriography.
Management: Intravenous fluids, heparin, gentamicin and metronidazole.

Chronic Mesenteric Ischaemia
Symptoms: Post prandial pain i.e pain after eating. Patients lose weight due to fear of pain after eating.
Risk factors: HTN, DM, high cholesterol
Cause: Artherosclerosis
Investigation: Arteriography
Treatment: reduce the risk of artherosclerosis.

LIMB ISCHAEMIA

Acute limb ischaemia
Symptoms: 6 P's - painful, paralysis, pale, perishing cold, parasthesiae, pulseless
Risk factors: AF, MI (mural thrombus), aortic aneurysm, valvular heart disease
Cause: Emboli
Investigation: Doppler USS or arteriography.
Management: Immediate referral to vascular surgeon for embolectomy, unfractionated heparin IV

Chronic Limb Ischaemia (Peripheral vascular disease)
Symptoms: intermittent claudication
Risk factors: HTN, DM, high cholesterol
Cause: artherosclerosis
Investigation: i) Ankle Brachial Pressure Index (if <0.5 = critical limb ischemia) ii) Doppler USS iii) arteriography
Treatment:

- **Reduce the risk of artherosclerosis. E.g reduce cholesterol and hypertension**
- **Also bypass graft if severe symptoms**
- **Exercise to improve symptoms of claudication.**

Differential diagnosis
Thromboangiitis obliterans (Burger's disease) =usually in young men around 40 years with strong smoking history.

PER RECTAL BLEED

Causes

1. Haemorrhoids (aka Piles)
2. Anal fissure
3. Acute mesenteric ischaemia
4. Colonic cancer
5. Rectal cancer
6. Diverticulitis
7. Angiodysplasia
8. Inflammatory bowel disease
9. Trauma
10. Bleeding diathesis

1. **HAEMORRHOIDS (AKA PILES)**

- History of constipation
- Also common in liver cirrhosis
- Fresh blood per rectal which splashes in the toilet pan.
- No pain
- Itching is usually present

STAGES OF HAEMORRHOIDS

- 1st degree – Remains in the rectum
- 2nd degree – Prolapses during defaecation but reduces spontaneously
- 3rd degree – Prolapses during defecation but requires digital reduction
- 4th degree – Remains persistently prolapsed, cannot be reduced

Management:

1. **Conservative management:**
   - **First line treatment of choice**
   - **Lifestyle modifications: high fibre diet, topical anaesthetics, behaviour modifications incl. weight loss, no reading while in the toilet**
2. **Non-surgical management:**
   - **Rubber band ligation – good choice for first and second degree haemorrhoids**
   - **Injection sclerotherapy – an alternative treatment for first and second degree haemorrhoids**
3. **Surgical Haemmorrhoidectomy**
   - **Used if minor procedures not effective and in external haemorroids (3rd degree haemorroids)**

NB. Painful peri-anal haematoma must be treated with incision and drainage

2. ANAL FISSURE

- Tear on the anus
- History of constipation
- Intense pain in the anus
- Fresh blood per rectal
- Per rectal examination may be impossible due to severe pain

Treatment:

1. **First try conservative treatment i.e. laxatives, ↑fluid intake, topical lubricants.**
2. **Topical glyceryl trinitrate (GTN) ointment is the first line treatment of chronic anal fissure.**
3. **Topical diltiazem**
4. **Botulin toxin used if failed response to GTN**

3. DIVERTICULITIS

- Inflammation of diverticulum (outpocketing of weak area of intestinal wall)
- Presents with fever and left iliac fossa pain relieved by defecation
- Usually patients are 60 years and above
- Profuse bleeding per rectal but there is no rectal pain

Investigation:

- **In the acute phase, CT scan is investigation of choice. Do not do colonoscopy during acute phase as it can cause perforation.**
- **Colonoscopy is best for diverticular disease, can be used in diverticular bleeding both for diagnosis and treatment.**

Management: Antibiotics are 1st choice for acute diverticulitis (co-amoxiclav or ciprofloxacin and metronidazole)

4. COLONIC CANCER

- Usually elderly patient
- Symptoms of malignancy i.e weight loss, anorexia, fatique, anaemia.
- Left colonic cancer usually presents with per rectal blood mixed with stool
- Right colonic cancer usually presents with anaemia
- Change in bowel habits i.e alternating diarrhea and constipation

Investigation: Colonoscopy and biopsy

5. RECTAL CARCINOMA

- Elderly patient
- Symptoms of malignancy i.e. weight loss, anaemia, anorexia, fatigue, tiredness.
- Fresh per rectal bleed
- Tenesmus which is a feeling of incomplete evacuation.

- Ulcer in the rectum means cancer

Investigation: Sigmoidoscopy (for lesions in rectum up to sigmoid colon) and biopsy

6. ANGIODYSPLASIA

- Congenital arterio-venous malformation
- Presents in elderly patient with unexplained spontanenous bleeding per rectum with no other possible cause of bleeding

Investigation: colonoscopy or barium enema which may show no abnormality. Capsule endoscopy may also be used.

7. PERI-ANAL HAEMATOMA

- This is a thrombosed haemorrhoids
- There is severe pain
- It is locate at the anal verge
- It is purple blue lump.

Treatment: Incision and drainage of hematoma

8. INTUSSUSCEPTION

- This condition is common in **children**.
- The typical age is 5-12 months
- Intermittent abdominal pain
- Child crying while pulling the legs towards the abdominal
- Per rectal fresh bleed, currant jelly like stools
- There is sausage shaped mass in the abdomen
- Shock

Investigation: Air enema/barium enema
Management: Pneumatic reduction which is the air enema.

INFLAMMATORY BOWEL DISEASE

1. Ulcerative colitis
2. Chrohn's disease

ULCERATIVE COLITIS

- Usually young patient (20-30 years) with chronic history of bloody diarrhea
- Fever
- Usually does not go beyond ileo-cecal valve
- Granular inflammation of mucosa
- Extra intestinal manifestation e,g arthritis, conjuctivivtis, pyoderma gangrenosum-ulcer on the leg

CROHN'S DISEASE

- Young patient between 20-30 years
- Chronic diarrhoea
- -/+ blood per rectal
- Can affect any part of the GI tract from the mouth to the anus
- Transmural granulomatous inflammation of the intestinal mucosa
- Extra intestinal manifestations can be conjunctivitis, pyodema gangrenosum, arthritis mouth ulcers

NB: Typical signs of crohns disease are: fistula in ano, peri-anal abscess, skin tags, skip lesion pattern, granuloma formation, cobblestone appearance on colonoscopy, rose thorn appearance, colonic stricture. These signs suggest Crohn's disease whether there is per rectal bleed or not.

VARICOSE VEINS

Signs: Eczematous changes, oedema, pigmentation, tortuous veins

Pathophysiology:

- There are 2 types of veins in the lower limb: Superficial veins and Deep veins.
- The superficial vein drains into deep veins.
- The superficial veins and deep veins are connected by perforating veins.
- The perforating veins have valves which allow blood flow only in one direction i.e from superficial to deep veins.

- If the perforating veins become incompetent they begin to allow flow in opposite direction. i.e from deep into superficial.

Superficial veins

1. **Long saphenous vein** runs on the medial aspect of the leg all the away up to the sapheno-femoral junction.
2. **Short saphenous vein** runs on the lateral aspect of the leg into the sapheno-popliteal vein at the posterior aspect of the leg.

Deep veins

1. **Popliteal vein**
2. **Femoral vein**

Management

1. **Lifestyle modifications (avoid prolonged standing and elevate legs)**
2. **Minimally invasive therapies:**
    a. **Radiofrequency ablation - uses radio frequency energy to seal the lumen of the long saphenous vein**
    b. **Endovenous laser therapy - uses high-intensity laser**
    c. **Foam sclerotherapy - seals the vein using foam**
3. **Surgery:**
    a. **Phlebectomy - removal of vein in parts**
    b. **Stripping - removal of the entire vein**
    c. **Sclerotherapy - seals the vein using sclerosing agent**
4. **Compression stockings if interventional therapy not appropriate. Always exclude peripheral arterial disease before prescribing.**

LUMPS IN THE GROIN

1. EPIDIDYMAL CYSTS =also called **spermatocele**

- Usually located on the upper pole of the testes.
- It is above and behind the testes, palpable separately from the testes.
- It fluctuates and transilluminates

Investigation: USS
Management: Surgical removal if symptomatic otherwise leave it

2. HYDROCOELE This is accumulation of fluid within the tunica mucosa.

- Whole scrotum is enlarged, it can be of very big size e.g 10cm.
- No mass palpable in the testes rather the testes are enlarged as a whole
- Testes are not palpable
- It fluctuates and transilluminates

Investigation: USS

Management: Aspiration. If asymptomatic then reassure.

3. **TESTICULAR TUMOUR**

- Long standing history of a mass in the testes.
- Mass is firm in consistency , attached to the testes.
- If age between 20-30 its teratoma
- If age between 30-40 its seminoma.

Investigation: Initial investigation is USS and blood tests for markers

Definitive: biopsy by doing orchidectomy.

4. TESTICULAR TORSION

- **Sudden onset of severe testicular pain is always testicular torsion until proven otherwise.**
- Common in young patients especially adolescents
- May have vomiting as well
- Pain may start while riding a bicycle or playing football or any other sports but there usually no history of trauma

Investigation and Treatment: Exploratory surgery.

5. HERNIA

- The mass is usually above and medial to pubic tubercle
- -/+ cough impulse
- On examination you cannot get above the mass.

6. VARICOCELE This is due to dilated veins of the scrotum

- Feels like a bag of worms
- Bluish in colour
- Disappears when patient lies flat
- Can be itchy and have an aching pain.

Investigation: USS
Management: If asymptomatic then reassure
If symptomatic then perform surgery.

7. EPIDIDYMO-ORCHITIS

- Fever, dysuria, frequency of micturition.
- Swelling and redness on the testes

Investigation: MSU
Management: antibiotics

INGUINAL HERNIAS

CLASSIFICATION

1. **Reducible hernia**
    - Contents can be replaced completely into the peritoneal cavity
    - Presentation: Painless lump that disappears on lying flat and with cough impulse

2. **Irreducible hernia**
   - Due to adhesions if its contents to the inner wall of the sac
   - Presentation: Painless lump, no cough impulse and the lump is not reducible on examination
3. **Strangulated hernia**
   - Contents of the hernia are constricted by the neck of the sac to such an extent that their circulation is cut off.
   - Unless relieved, gangrene is inevitable and perforation will eventually occur
   - Presentation: Often with signs of intestinal obstruction i.e vomiting, constipation and distended abdomen plus the lump is tender, not reducible hernia and bowel sounds are increased.

TYPES:

1. **INDIRECT INGUINAL HERNIA**

Enters the internal inguinal ring, transverses the inguinal canal. If large enough it emerges through the external ring and descends into the scrotum. The hernia can be controlled by pressure with one finger over the internal inguinal ring.

1. **DIRECT INGUINAL HERNIA**

Pushes through the posterior wall of the inguinal canal media to the internal ring. It is not controlled by digital pressure over the internal ring.

Treatment

1. **In children below 12 years of age you do herniotomy**
2. **If presentation is below 1 year, wait until 1 year to do herniotomy.**
3. **In adults you do herniorrhaphy or also called hernia repair.**
4. **If it is REDUCIBLE OR IRREDUCIBLE HERNIA then you do elective (planned) hernia repair**
5. **If it is STRANGULATED HERNIA then you do immediate hernia reair.**

1. **FEMORAL HERNIA:**

More common in women and it commonly strangulates.
Treatment: Because of high risk of strangulation, all must be treated surgically as soon as possible.

ANATOMY

- The inguinal canal is 4 cm long.
- It passes downward and medially from deep to superficial from the internal to the external ring.
- It lies parallel to and immediately above the inguinal ligament.
- The internal ring represents the point at which the spermatic cord pushes through the transversalis fascia. The internal ring lies above and lateral to pubic tubercle and it is 1-2 cm above the femoral pulse.
- The external inguinal ring is a defect in the external oblique aponeurosis and lies immediate above and medial to pubic tubercle
- The inguinal canal contains the spermatic cord and the ilio-inguinal nerve.

LEG ULCERS

1. **DIABETIC ULCERS**

- Painless ulcer on the base of heal or base of the metatarsal
- History of diabetes or history of polyuria, thirst and weight loss

Investigation: Blood glucose

1. **VENOUS ULCER**

- Ulcer on the medial malleoli
- On examination there are varicose veins
- History of standing for long time due to venous stasis, e.g. people who work as waiters or guards.

1. **PYODERMA GANGRENOSUM**

- History of inflammatory bowel disease

Investigation: Biopsy for pyoderma gangrenosum

1. **MELANOMA**

- Usually middle aged or elderly patients with an ulcer over the shin or any other exposed area.
- The ulcer is pigmented, increasing in size with irregular margins and changing in shape.

Investigation: biopsy for melanoma

1. **ARTERIAL ULCER**

- History of intermittent claudication
- Painful ulcer

DEEP VENOUS THROMBOSIS

Signs

- Calf tenderness
- Calf warm to touch
- Swelling of the calf
- Mild fever
- Pitting oedema

WELL'S SCORE

- Active cancer (treatment within last 6 months or palliative): +1 point
- Calf swelling ≥ 3 cm compared to asymptomatic calf (measured 10 cm below tibial tuberosity): +1 point
- Swollen unilateral superficial veins (non-varicose, in symptomatic leg): +1 point
- Unilateral pitting edema (in symptomatic leg): +1 point
- Previous documented DVT: +1 point
- Swelling of entire leg: +1 point
- Localized tenderness along the deep venous system: +1 point
- Paralysis, paresis, or recent cast immobilization of lower extremities: +1 point
- Recently bedridden ≥ 3 days, or major surgery requiring regional or general anesthetic in the past 12 weeks: +1 point
- Alternative diagnosis at least as likely: −2 points[4]

1. 0 or less than zero points is low probability
2. 1-2 points is intermediate probability
3. 3 or more points is high probability

Management of Deep venous thrombosis

1. **IF LOW PROBABILITY do D-dimer as initial investigation**
   a. **If D-dimer –ve it means DVT has been ruled out.**
   b. **If D-dimer +ve then start treatment with heparin and investigate with compression US. If confirmed DVT then add warfarin and continue both warfarin and heparin until INR is 2. When INR is 2 stop heparin and continue warfarin. Maintain INR between 2-3.**

1. **IF INTERMEDIATE OR HIGH PROBABILITY, DO NOT PERFORM D-DIMER. START TREATMENT with low molecular weight heparin then Investigate with compression ultrasound scan**
   a. **If +ve then add warfarin and continue both warfarin and heparin until INR is 2 then stop heparin and continue warfarin maintaining INR 2-3.**

UPPER GASTROINTESTINAL BLEEDING

CAUSES:

1. **Peptic ulcer disease**
2. **Mallory weiss tear**
3. **Oesophageal carcinoma**
4. **Gastric carcinoma**
5. **Gastric erosions due to medications (NSAIDs, Aspirin, steroid, biphophanates)**
6. **Curling ulcers**
7. **Oesophageal varices**
8. **Renal failure**

Symptoms: the two main symptoms of upper GI bleeding are haematemesis and melena(black stool)

1. PEPTIC ULCER:
-There are 2 peptic ulcers we need to know: gastric and duodenal ulcers.

Gastric Ulcers
-stomach ulcers
-epigastric pain worse with meals
-relieved with anti-acids
-history of indigestion
-patient may loose weight due to fear of eating.

Duodenal Ulcer
-epigastric pain relieved with food and anti-acids
-worse at night due to fasting
-also called hungry ulcers.

2. GASTRIC CARCINOMA

- Elderly patient
- Symptoms of malignancy i.e weight loss, anorexia, tiredness, fatique.
- Early satiety
- Epigastric pain or discomfort which may radiate to the back.
- Metastasis to the spraclavicular lymp nodes called virchow's nodes.
- Common in Japanese.

Investigation: Gastroscopy and biopsy
Management: Surgery if no metastasis

3. MALLORY WEISS TEAR

- Usually young patient after binge drinking
- Patient needs not to be a alcoholic. Alcoholics usually suffer from oesophageal varices
- Usually they vomit small amount of blood after retching.
- Usually haemodynamically stable

Investigation: monitor vital signs and FBC
Management: Check full blood count 24 hrs after

4. OESOPHAGEAL CARCINOMA

- Old age of the patient
- Dysphagia for solid initially then liquids.
- Weight loss, anaemia, anorexia, fatique

Investigation: Oesophago-gastroscopy and biopsy

Management:

- **If there is no metastasis then treatment is resection of the oesophagus**
- **If there is metastasis treat with radiotherapy.**

5. OESOPHAGEAL VARICES

- Usually in alcoholics or patients with long standing liver disease e.g primary bilary cirrhosis or chronic viral hepatitis
- Massive bleeding
- Patient is in shock
- Repeated haematemesis
- On examination there can be stigmata of liver disease(spider naevi)
- Patient may smell of alcohol

Primary Management of Oesophageal Varices:

1. **Oesophago-duodensoscopy (OGD) is recommended in all patients when diagnosis of cirrhosis is made.**
2. **If patient has liver cirrhosis but no oesophageal varices the ne needs OGD every 3 years.**
3. **Cirrhotic patients with small varices need repeat OGD every 1-2 years. Use beta blockers for prophylaxis of bleeding. Use both beta blockers or endoscopic variceal ligation.**
4. **Nitrates may be used together with beta blockers.**
5. **Sclerotherapy has no role in the primary prophylaxis of varcieal bleeding.**

Management of Active Oesophageal Bleeding:

1. **Take care of the ABC's of the patient**
2. **Get IV lines and send bloods including Group and Save**
3. **Cross match 6 units of blood**
4. **Emergency endoscopy (OGD)**
    a. **Treatment of choice is band ligation**
    b. **Sclerotherapy should be used if ligation is technically difficult.**
5. **Short term (<1 week) antibiotic prophylaxis should be prescribed e.g. ciprofloxacin, in any pateitn with cirrhosis and GI bleeding.**
6. **Somatostatin or its analogue must be prescribed e.g. octreotide**
7. **If bleeding does not stop with above measures, the balloon Sengstaken tube tamponade**
8. **If all fails consider Transjugular Intrahepatic Porto-systemic Shunt (TIPS)**

Prevention of Secondary Bleeding (Rebleeding)

1. **Use of band ligation (or sclerotherapy) plus beta blockers.**
2. **Band ligation is the first method.**
3. **Sclerotherapy should only be used if band ligation is technically difficult**
4. **TIPS is more effective than endoscopic procedures but does not increase survival**

6. GASTRIC EROSIONS

- Common in patient who are long term use of non steroidal ani-nflammaor drugs e.g ibuprofen, naproxen,aspirin, a
- Also steroid especially if patient is allergy to NSAIDs then is more likey to have been using steroid since he/she can not use NSAIDs.
- Also biphosphanate. E.g alendronic acid.
- There can be history of back or joint pain or rheumatoid arthritis or osteoarthritis which indicate that patient has been taking NSAIDs

7. CURLING ULCERS
These is usually after burns.
Treatment: Treat with proton pump inhibitors (PPI) if ulcers are severe

8. CHRONIC RENAL FAILURE
Can also cause ulcers due to reduced excretion of gastrin

MANAGEMENT OF UPPER GI BLEEDING

1. **FBC to check for Hb**
2. **If you suspect patient of having oesophageal varices then needs urgent upper GI endoscope**
3. **Usually all patient with upper GI bleeding will need UGI endoscope except in cases of Mallory Weiss tear where there small amount of bleed.**
4. **Banding and sclerotherapy may be performed during endoscopy.**

ACUTE ABDOMEN

RIGHT UPPER QUADRANT

1. **ACUTE CHOLECYSTITIS**

Inflammation of the gall bladder usually with pre-existing gallstones.

Signs & Symptoms:

- Fever
- Pain in the right quadrant radiating to the right scapula worse with fatty foods.
- Nausea and vomiting
- Murphy's sign is positive

Investigation:

1. **USS is the investigation of choice (look for gallstones)**
2. **MRCP (Magnetic resonance cholangiopancreatography) is indicated if no evidence of gallstones found despite classical biliary pain.**
3. **ERCP should not be used as a routine investigation but in those patients who are likely to require intervention.**

Management:

1. **Symptomatic gallstones are most effectively treated with laparoscopic cholecystectomy.**
2. **Asymptomatic gallstones should be managed conservatively unless in the following situations when laparascopic cholecystectomy is recommended**
   a. **Big stones in the gall bladder**
   b. **Small stones in the gall bladder but in very young patients**
   c. **High risk of complications like in a diabetic patient**

1. CHOLANGIITIS - This is inflammation of the common bile duct.

Signs & Symptoms:
Charcot's triad of cholangitis: right upper quadrant pain, fever and jaundice
Reynold's pentad of ascending cholangitis: Charcot's triad + shock and altered mental status

Investigation: USS
Management: cefuroxime and metronidazole.

1. BILIARY COLIC - Usually presents with intermittent pain in the right upper quadrant due to pain caused by the stones in the gallbladder.

Signs & Symptoms

- Pain in the RUQ
- Radiating to the right shoulder
- Jaundice but there is no fever.

Investigation:
1) USS
2) ERCP-if obstructive jaundice and worsening LFT or if there is a stone in the common bile duct.

Management: Conservative treatment is first line treatment for small stones.

1. **PYELONEPHRITIS**

Signs & Symptoms

- Loin pain
- Fever
- Dysuria and frequency
- Rigors and vomiting

Investigation: MSU
Management: Antibiotics: cefuroxime or cefotaxime

1. **LOWER LOBE PNEUMONIA**

Signs & Symptoms:

- Fever
- Cough
- Shortness of breath
- Sputum
- Chest pain

Investigation: CXR
Management: Antibiotics

1. **RENAL COLIC**

Signs & Symptoms

- Intermittent loin pain
- Haematuria

Investigation: IVU
Management: rehydration or stone removal.

1. **AMOEBIC HEPATIC ABSCESS**

- History of travel to tropical areas and diarrhea
- Right upper quadrant pain and jaundice

Investigations: USS, Stool antigen
Treatment: Metronidazole +/- Incision and Drainage

1. **ACUTE VIRAL HEPATITIS A (HAV)**

- History of travel e.g to India
- Upper quadrant pain
- Jaundice
- Diarrhoea

Investigation: Hepatitis serology
Management: Interferon

LEFT UPPER QUADRANT

1. **SPLENIC RUPTURE**

- Usually is due to trauma
- On examination there are bruises on the abdomen

Investigation: USS abdomen
Management: Laparotomy.

1. **PYELONEPHRITIS**=same as in RUQ.
2. **RENAL STONE**=same as in RUQ
3. **LOWER LOBE PNEUMONIA**=same as in RUQ

EPIGASTRIC PAIN

1. **PERFORATED PEPTIC ULCER**

- Pain and tenderness in the epigastrium or upper abdomen
- May be history of indigestion or use of NSAIDs or history of rheumatoid arthritis or back pain because people with these conditions usually use NSAIDs.

Investigation: Erect CXR=to see gas under the diaphragm (pneumoperitoneum)
Management: Laparotomy

1. **ACUTE PANCREATITIS**

- Epigastric or upper abdominal pain which radiates to the back
- Profuse vomiting
- Abdominal pain may quickly become generalized
- Reduced bowel sounds
- Patient is in shock i.e low BP and tachycardia.

Investigations:

1. **Investigation of choice is serum amylase to confirm diagnosis.**
2. **Plasma lipase can also be used and is more sensitive than amylase**
3. **USS to look for gallstones**
4. **CT abdomen is the gold standard if diagnosis is not clear after checking amylase and lipase**

Treatment:

1. **IV fluids and NGT if vomiting**
2. **Prophylactic antibiotics**
3. **Gallstone-related pancreatitis needs urgent ERCP and laparoscopic cholecystectomy should be performed within 2 weeks.**

1. **MYOCARDIAL INFARCTION**

- Epigastric pain
- Elderly patient
- Nausea
- Sweating in the palms
- History of ischaemic heart disease.

Investigation: ECG &Cardiac enzymes.

RIGHT ILIAC FOSSA

1. **APPENDICITIS**

- Central abdominal pain which then moves to right iliac fossa
- Vomiting, fever, anorexia
- On examination there rebound and guarding
- **THIS IS A CLINICAL DIAGNOSIS**

Investigation: FBC
Management: Appendicectomy

1. SALPINGITIS This is Pelvic Inflammatory Disease

- It is due to sexually transmitted infection, commonly Chlamydia
- Per vaginal disharge

Investigation: Endocervical swab
Management: Antibiotics.

1. TUBO-OVARIAN ABSCESS Complication of pelvic inflammatory disease

- Usually there is swinging fever
- On examination there is a mass in the in the iliac fossa.

Investigation: USS
Management: Incision and drainage.

1. URETERIC COLIC Due to stones in the ureter.

- Right or left iliac fossa pain which radiates to the groin.
- Haematuria

Investigation: IVU
Management: Rehydration or depending on the size of the stone may use lithotripsy or open surgery.

LEFT ILIAC FOSSA

1. **DIVERTICULITIS** See Diverticulitis under section of **PER RECTAL BLEED**.

1. **URETERIC COLIC** - same as in RIF
2. **SALPINGITIS** – same as in RIF
3. **TUBO-OVARIAN ABSCESS** - same as in RIF

CENTRAL ABDOMEN

1. **INTESTINAL OBNSTRUCTION**

The four cardinal signs of intestinal obstruction

- Vomiting
- Abdominal pain
- Distended abdomen
- Absolute constipation

Investigation: Plain abdominal X-ray=dilated bowel loops
Management: Surgery

1. **ABDOMINAL AORTIC ANEURYSM**=

- Usually middle aged or elderly patient
- Pulsatile mass in the abdomen
- Absent femoral pulses bilaterally
- Patient is in shock i.e low BP and tachycardia
- Abdominal pain which radiates to the back

Investigation: CT abdomen
Management: Laparotomy.

3) **ACUTE MESENTERIC ISCHAEMIA**

- Sudden onset of abdominal pain, with no signs of peritonism i.e no rigidity or guarding.
- There is per rectal bleed
- History of IHD or AF or valvular heart disease
- Cause is an emboli

Investigation: Arteriography
Management: Heparin and antibiotics.

SUPRAPUBIC AREA

1. UTI - urinary tract infection.

- Dysuria

- Frequency of micturition
- Fever

Investigation: MSU
Management: Antibiotics, usually Trimethoprim

1. **BLADDER STONES**-

- Pain on urination
- Haematuria
- Usually suprapubic pain

Investigation:

- **KUB x-ray is the initial investigation**
- **IVU is the most appropriate investigation**

Management: Rehydration or stone removal.

XVI. PROCEDURES AND RELEVANT ANATOMY IN SURGERY

1. **Needle Cricothyroidotomy –** Cricothyroid membrane will be the last structure pierced before reaching the desired anatomical space, the trachea.

1. **Chest drain –** 4th to 6th intercostal space at the mid axillary line through the "safe triangle". Pierce the intercostal muscles. The structure likely to be damaged is the intercostal nerve.

1. **Lumbar puncture –** Inserted between L3 & L4, landmarks is the plane between the iliac crests. Pierces the dura mater before reaching CSF

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Resource view

Resource name	Toxicology PLAB 1 Notes
Resource description	Toxicology
Resource content	

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TOXICOLOGY AND PHARMACOLOGY LECTURE

COMMON FEATURES OF DRUG POSOINING

Tachycardia or irregular pulse (AF)	Salbutamol, tricyclic, quinine
Respiratory depression	Opiates, Benzodiazepine
Hypothermia	Phenothiazine, barbiturates
Hyperthermia	Amphetamine, cocaine, ecstasy
Coma	Benzodiazepines, alcohol, tricyclic antidepressant, barbiturates, opioids
Seizure	Hypoglycaemia, tricyclic, phenothiazine, theophylline
Dilated pupils	Amphetamines, cocaine, quinine, Tricyclic antidepressant.
Hypoglycaemia	Insulin, oral hypoglycaemia agents, alcohol, salicylates
Metabolic acidosis	Alcohol, methanol, paracetamol, carbon monoxide poisoning, salicylates
Renal failure	Salicylates, paracetamol, NSAIDs
Constricted pupils (pin point pupil)	Opiates, organophosphates

GENERAL MANAGEMENT OF DRUG POISONING

1. Maintain airway if it is compromised
1. Give oxygen to maintain oxygen saturation above 92%
1. Generally take care of the airway, breathing and circulation. If hypotensive give intravenous fluids.
1. Torsades de pointe treat with magnesium sulphate
1. Arrhythmia from tricyclic anti-depressant should be treated with 50ml of 8.4% sodium bicarbonates via a central line or large vein
1. Metabolic acidosis increases the risk of seizure it should be treated with correction of hypoxia and intravenous fluids and not settling still then 250ml of 1.26% sodium bicarbonate.
1. Treat seizures with intravenous lorazepam 2-4mg or diazepam 10-20mg in adults.
1. If patient is agitated treat with oral or IV diazepam
1. Use antidotes if required
1. Induced vomiting is not of any use and potentially dangerous
1. Give activated charcoal (50g) orally within 1 hour of ingestion
1. Medication like lithium, iron, methanol and ethylene glycol are not bound to charcoal
1. Gastric lavage is rarely needed. It should only be done if life threatening. And it should not be used in patients with reduced consciousness due to risk of aspiration.
1. Haemodialysis can improve outcome in severe cases of overdose e. g digoxin, ethylene glycol, lithium, methanol and salicylates

SPECIFIC OVERDOSE

1. **PARACETAMOL POISONING**

1 tablet of paracetamol is 500mg

150mg/kg or > 12g of paracetamol is considered as a dangerous dose

Key Points:

- 1 tablet = 500 mg
- **Dangerous dose** is >12 g (24 tablets) or >150mg/kg
- **Time since ingestion** is crucial to determine management.
- Drug levels are checked **4 hours after ingestion**
- Treatment with N-acetylcysteine is determined by the paracetamol treatment **graph**
- N-acetylcysteine is most effective when given **within 8 hours after ingestion**
- Other blood tests are INR, plasma creatinine, U&E, LFT, ABG

Management:

- **Management if time since ingestion is unknown:** Start treatment with NAC immediately.
- **Management within 1 hour of ingestion:** Give activated charcoal if >12g or 150 mg/kg. Take bloods 4 hours post ingestion and use graph to determine if NAC needed.
- **Management within 4 hours of ingestion:** Take drug levels 4 hours post ingestion and use graph.
- **Management at 4-8 hours from ingestion:** Take drug levels immediately and use graph. Wait for paracetamol levels.
- **Management 8-15 hours from ingestion:** If >12 g or 150 mg/kg start treatment immediately. If drug levels are below treatment line and patient asymptomatic, stop treatment.

- **Management 15-24 hours from ingestion:** If >12g or 150 mg/kg start treatment immediately. If drug levels and bloods at 24 hours post ingestion are normal and patient asymptomatic, stop treatment.

- **Management >24 hours since ingestion:** If >12g or 150 mg/kg or bloods abnormal or patient symptomatic, start treatment immediately. Seek expert opinion.

Staggered Overdose: overdose in a prolonged period of time.

The normogram cannot be used

If dangerous level i.e. 150mg/kg or 12g use N-acetylcysteine

Check INR or PT, U&E, LFT and FBC.

1. **BENZODIAZEPINE:**

Example of benzodiazepine: chlordiazepoxide, diazepam are long acting benzodiazepine, lorazepam is a short acting.

Causes drowsiness, hypotension, coma, respiratory depression, CNS depression, coma.

Toxicity is worse alcohol or other CNS depressant medication.

Treatment: Antidote is Flumezenil.

1. **SALICYLATE: e.g. aspirin**

Symptoms:

1. Vomiting
2. Tinnitus
3. Deafness.

In severe cases confusion, seizures, initially respiratory alkalosis then metabolic acidosis, pulmonary oedema

Toxicity >500mg/kg can cause death

Give activated charcoal if ingestion less than 1 hour ago and took more than >120mg/kg

Check 2 hours post ingestion if patient symptomatic.

If asymptomatic check at 4 hours after ingestion.

Investigation: U&E, PT or INR, Blood glucose causes hypoglycaemia), ABG can cause hypokalaemia

Treatment:

If salicylate >500mg/kg then IV 8.4%sodium bicarbonate 225ml over 1 hour

Alkalinization of urine

4. ANTIDEPRESSANT

Tricyclic antidepressant (amitriptyline, dosulepin)

Symptoms:

1. Tachycardia
2. Dilated pupils
3. Urinary retention
4. Hyperreflexia
5. Divergent squint
6. Hypotension
7. Seizures
8. Coma
9. Arrhythmias
10. Prolonged QRS complex

5. Selective serotonin reuptake inhibitors (SSRIs)
e.g. paroxetine, sertraline, fluoxetine, citalopram

Symptoms: nausea, vomiting, tremor, serotonic syndrome

6. Selective norepinephrine reuptake inhibitors
e.g. venflaxine cause same symptoms as TCA

7. Mirtazepine: vomiting, nausea and drowsiness

Management:

Activated charcoal if taken within 1 hour

If QRS >120ms after overdose give 8.4% sodium bicarbonates 50ml serotinin syndrome may occur after taking 2 or more antidepressant e.g. TCA, SSRI or MAOI

8. OPIOIDS

EXAMPLES: codeine, morphine, dihydrocodeine, fentanyl, tramadol, methadone, pethidine.

Features:

1. Reduced consciousness
2. Respiratory depression
3. Pin point pupils
4. Hypotension
5. Vomiting and nausea
6. Respiratory rate < 12
7. Patient may have puncture marks on the arms.

Management:

Naloxone IV 0.4 - 2mg.

Can also be given IM for which its effect can be more prolonged.

N.B: The plasma half life of naloxone is shorter than that of most opioid, so the repeated does are often required. This is especially true with long acting opiates e.g. Morphine sulphate tablets or methadone where naloxone infusion might be needed.

N.B: Opiates accumulates in the body in people with renal impairment. Therefore opiate toxicity should be suspected in all patients with unexplained type 2 respiratory failure.

1. **RECREATIONAL DRUGS**

FEATURES:

Stimulant drugs such as NMDA (ecstasy), amphetamines, cocaine. lysergic acid diethylamide (LSD) may cause severe agitation, tachycardia, sweating, pyrexia, dilated pupils, hypertension, arrhythmia and seizures.

Severe cases results in coma, rhabdomyolysis, renal failure and subarachnoid haemorrhage, myocardial infarction, repeated seizures and death.

SPECIFIC FEATURES

1. **COCAINE**: Coronary artery aneurysm, myocardial ischaemia and infarction and aortic dissection. Also subarachnoid aneurysm.
2. **Ecstasy:** causes severe hyponatremia and water intoxication
3. **Gamma hydroxybutyrate (GHB)** may cause bradycardia, hypotesion, reduced consciousness and coma associated with severe withdrawal symptoms;

Management:

1. Measure U&E, LFTs, Creatinine phosphokinase (CK)
2. Perform ECG and monitor cardiac rhythm
3. Hypertension settles with diazepam if still persistent then use glycerine tri-nitrate
4. Treat cocaine induced chest pain and ECG changes with diazepam, aspirin and nitrates
5. Amphetamine tachycardia can be treated with beta blockers

1. **IRON OVERDOSE**

1. Common in children of pregnant mothers, after left unsupervised and ingested mother's iron tablets. The iron tablets are red in colour and child may vomit reddish-brown materials.
2. Iron tablets=iron sulphate
3. Features: nausea and vomiting metabolic acidosis, stomach ulcers, liver failure, Brain damage. Brain damage and coma.

NB: stomach ulcers may cause stricture.

Investigation: **serum iron levels**

Treatment : Desferoxamine, if not improving then dialysis.

11. DIGOXIN

Reduced cognition, yellow-green visual haloes, nausea, vomiting, cardiac arrhythmia.

Treatment: digoxin specific anti-body fragment.

12.CYANIDE

Dizziness, headache, breathlessness, shock, odour of bitter almonds. No Cyanosis

Treatment: oxygen, sodium nitrite and sodium thiosuphate, dicobalt edetate.

12. METHANOL

Headache, breathlessness, photophobia, papilloedema, optic atrophy, blindness.

Treatment: Gastric lavage, bicarbonate infusion, ethanol plus IV calcium.

13. CARBON MONOXIDE

Pink skin, headache, vomiting, tachycardia, tachypnoea, fits, coma and cardiac arrest

Investigation: serum carbon monoxide levels, oxygen levels might be high.

Managment: 100% hyperbaric oxygen, manitol for cerebral oedema.

14. ETHYLENE GLYCOL (anti-freeze)

GI upset, neurological involvement, cardiorespiratory collapse, acute renal failure,

Treatment: gastric lavage, bicarbonate infusion, ethanol, IV calcium.

15. ORGANOPHOSPHATE:

Salivation, lacrimation, urination, diarrhea, sweating, small pupils, bradycardia, respiratory depression, muscle fasciculation

Treatment: Atropine and pralidoxime

16. PARAQUAT

Nausea, vomiting, diarrhea, painful oral ulcers, alveolitis, renal failure

Treatment: activated charcoal.

17. LITHIUM TOXICITY

Vomiting, diarrhea, coarse tremor, decreased consciousness, ataxia, increased tone, hypokalaemia

Treatment: IV fluid and haemodialysis

18. PHENYTOIN TOXICITY

Gum hypertrophy and cerebellar signs e.g. nystagmus, ataxia, diplopia, dizziness etc.

Investigation: serum drug levels

Phenytoin toxicity: renal failure and liver failure:

Check U&E, INR and drug levels

19. BETA BLOCKERS AND CALCIUM CHANNEL BLOCKERS

Bradycardia, hypotension and arrhythmia

Treatmnet: Atropine

20. ANTI-PSYCHOTICS:

Low blood pressure, ECG changes, extrapyramidal side effects: tremor and hyperprolactinaemia.

21. NSAIDs:

Renal failure and heart failure.

Investigation: check U&E

22. LSD:

Visual hallucinations, agitation, excitement, tachycardia and dilated pupils. Hypertension and pyrexia may occur.

Massive overdose may lead to coma, respiratory arrest and coagulation disturbances.

Treatment is supportive.

23. ECSTASY:
Euphoria, agitation, sweating, dilated pupils, ataxia, teeth grinding, headache, tachycardia, hypertension.

Can lead to renal failure, liver failure, cerebral haemorrhage and coma.

Treatment:

1. Consider activated charcoal if less than 1 hour since ingestion.
2. Observe for at least 4 hours
3. Monitor ECG, pulse, blood pressure and temperature

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