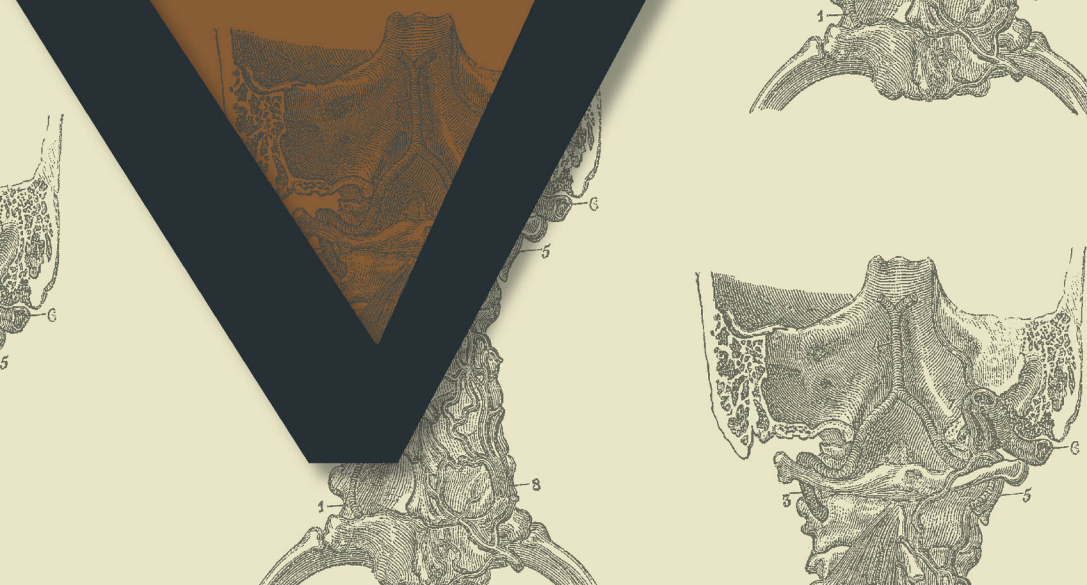
MasterPass

Essential Lists of Differential Diagnoses for MRCP

with diagnostic hints

Fazal-I-Akbar Danish



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with diagnostic hints

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Preface

This book is distinct from other books on the same subject in the sense that I have tried to refrain from burdening my readers with long and exhausting lists of (rare) causes. Instead, I have tried to restrict most of the lists to a maximum of five items that constitute the most common causes in terms of disease prevalence. This strategy is intended to help candidates not only pass the postgraduate exams but also to become competent clinicians (patient has metabolic acidosis, think of uraemia or DKA instead of paraldehyde toxicity).

Additionally, I have included what I call 'Diagnostic hints'. These refer to clinical or lab clues that if present in a clinical scenario point towards a specific diagnosis (e.g. mouth and genital ulcers, think of Behçet's syndrome, etc).

I have also included a chapter on ECG – something that I believe is unique. It contains lists of causes of ECG changes (e.g. causes of T wave inversion, hyperacute T waves, prolonged PR interval, etc.) and, conversely, key ECG features of common diagnoses (e.g. atrial fibrillation, MI, AV block, etc).

The book is primarily intended for MRCP (UK) (both Part I and Part II) and FCPS (Pakistan) (Part II) candidates, although it may also be helpful for undergraduate students preparing for their finals.

Fazal-I-Akbar Danish
January 2010

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About the author

Dr Fazal graduated from Army Medical College, Rawalpindi, Pakistan in 1999. After working in his home country for a few years in various capacities, he shifted to the UK in 2005 and has worked as Clinical Research Fellow in the Universities of Southampton and Bristol, and as a Medical SHO in various NHS trusts. Although a junior doctor, Dr Fazal has contributed appreciably in medical literature. He is the first and corresponding author of eight research papers published in different peer-reviewed journals. He has contributed a 28-web-page section namely 'Phenotyping' in an online encyclopaedia entitled 'Online Encyclopedia for Genetic Epidemiology Studies', www.oege.org. This section links and describes standardised research protocols and related information for clinical phenotyping of common diseases and risk traits. It is primarily of relevance to researchers and PhD students. Dr Fazal has three medical books to his credit – the book in your hand, *Hospital Dermatology* (a 226-page book for final year medical students and postgraduate trainees) and *Pharmacology in 7 Days for Medical Students* (a 166-page book for third year medical students). He is currently working as a CT2 in Medicine at the Princess of Wales Hospital in Bridgend.

1 General physical examination

Thin, wasted, cachectic:

1. Low calorie intake (d/t anorexia nervosa; alcoholism; drug abuse; prolonged systemic illness, e.g. COPD).
2. Tuberculosis.
3. Malignancy.
4. Thyrotoxicosis.
5. AIDS.

Fingernail abnormalities:

1. Clubbing. Acute/chronic paronychia.
2. Nail fold infarcts (d/t vasculitis).
3. Lines (Beau's; longitudinal; Muehrcke's; Mee's; Terry's).
4. Splinter haemorrhages.
5. Yellow nails.
6. Nail pitting.
7. Koilonychia.
8. Onycholysis.
9. Onychomadesis (shedding of nail).

Clubbing:

1. Respiratory disease (malignancy; infection [lung abscess, empyema]; bronchiectasis; fibrosing alveolitis).
2. Cardiac disease (congenital cyanotic heart disease [Fallot's tetralogy, transposition of the great arteries]; acyanotic heart disease, i.e. PDA with 'reversal of shunt' [clubbing only in the toes]; infective endocarditis).
3. Subclavian artery aneurysm (clubbing is unilateral).
4. GIT disease (cirrhosis; PBC; IBD; malabsorption – coeliac disease, Whipple's disease).

Cyanosis (non-oxygenated Hb >5 g/dL):

1. Peripheral cyanosis:[1]
   a. Shock (hypovolaemic; cardiogenic; septic d/t gram-negative organisms usually).
   b. Cold weather (Raynaud's phenomenon).
   c. Arterial/venous occlusion (d/t atheroma or small-vessel disease in diabetes).

1 Fingers, hands, nose, cheeks and ears would be cold and bluish.

2 Central cyanosis:[2]
   a Respiratory failure.
   b Congenital heart disease (Fallot's tetralogy; transposition of the great arteries; PDA with reversal of shunt/Eisenmenger's syndrome [→ differential cyanosis]; tricuspid atresia; Ebstein's anomaly; pulmonary AV fistula).
   c Haemoglobin abnormalities:
      i Methaemoglobinaemia (congenital; acquired – drugs like nitrates and sulfonamides).
      ii Sulphaemoglobinaemia (drugs like nitrates and sulfonamides).
      iii Hb M disease.
      iv NADH diaphorase.

Signs of dehydration:
1 ↓ skin elasticity.
2 Rapid, low-volume (thready) pulse.
3 ↓ BP (postural or persistent hypotension).
4 Sunken eyes.
5 Dry tongue.
6 ↓ urine output.

Koilonychia:
1 Iron-deficiency anaemia.
2 Rarely d/t IHD/syphilis.

Onycholysis:
1 Psoriasis.
2 Hyperthyroidism.

Beau's lines (transverse furrows):
1 Any severe illness.

Onychomadesis (shedding of nail):
1 Any severe illness.

Longitudinal lines:
1 Lichen planus.
2 Alopecia areata.
3 Darier's disease.

Muehrcke's lines (white-coloured paired parallel transverse bands):
1 Hypoalbuminaemia.

2 Undersurface of lips, tongue and buccal mucosa *also* would be cold and bluish.

Mee's lines (single, white transverse band):
1. Organ failure (heart/renal).
2. Hodgkin's lymphoma.
3. Arsenic poisoning.

Nail pitting:
1. Psoriasis.
2. Alopecia areata.

Yellow nails:
1. Bronchiectasis.
2. Hypoalbuminaemia.
3. Lymphoedema.

Terry's nails (nail tips having dark pink or brown bands):
1. Old age (>75 yr).
2. CCF.
3. Cirrhosis of liver.
4. DM.
5. Malignancy.

Nail fold infarcts (d/t vasculitis) – dark blue-black areas in the nail folds:
1. SLE.
2. Subacute bacterial endocarditis (Osler's nodes).

Hand arthropathy (IP joints involved):
1. OA (DIP joints involved – Heberden's nodes).
2. RA (multiple abnormalities seen).
3. Psoriatic arthropathy (DIP or all IP joints involved).
4. SLE (DIP or all IP joints involved).

Hand and upper limb rashes:
1. Contact dermatitis.
2. Lichen planus.
3. Atopic eczema.
4. Psoriasis.

Pigmented creases, flexures and buccal mucosa – suggests ↑ ACTH:
1. Addison's disease.
2. Pituitary-driven Cushing's disease.
3. Ectopic ACTH secretion (from lung or other malignancy).

Swellings around the elbow joint:
1. OA.

2. RA (rheumatoid nodules).
3. Gouty tophi.
4. Xanthomatosis (pale subcutaneous plaques attached to the underlying tendons).
5. Limited range of neck movement, without stiffness: chronic cervical spondylosis with osteophytes.

Neck stiffness throughout the range of movement:

1. Meningism (d/t viral/bacterial/tuberculous meningitis; SAH).
2. Acute cervical spondylitis.
3. Posterior fossa tumour.
4. Anxiety (voluntary/semivoluntary resistance to movements).

Hair loss in a specific area:

1. Alopecia areata/totalis.
2. PCOS.
3. Testosterone-secreting ovarian tumour.

Diffuse hair loss:

1. Alopecia universalis.
2. Any severe illness.
3. Iron-deficiency anaemia.
4. Pregnancy.
5. Hypogonadism.
6. Cytotoxic drugs.

Striking facial appearance:

1. CNS pathology (parkinsonism; Huntington's chorea; bilateral UMN lesion d/t MND, myasthenia gravis, cerebrovascular disease).
2. Endocrine pathology (acromegaly; hyper- or hypothyroidism; Cushing's syndrome).

Possible hypothermia (temp <35°C/95°F) – confirm with low-reading rectal thermometer:

1. Prolonged exposure to cold weather/immersion.
2. Hypothyroidism.

Arm span is more than double the sitting height:

1. Marfan's syndrome.
2. Hypogonadism.

Short limbs with normal trunk (sitting height is more than the length of the legs):

1. Achondroplasia.

Limbs and trunk are proportionate but total height is below normal:
1. Congenital hypopituitarism (pituitary dwarf).

Short 4th metacarpal (evident on making a fist) in a female:
1. Turner's syndrome.

Short 4th/5th metacarpal:
1. Pseudohypoparathyroidism.

Dactylitis:
1. Sickle-cell anaemia.
2. Sarcoidosis.
3. Infection (TB; syphilis; sepsis).

Large and broad hands:
1. Acromegaly.

Splinter haemorrhages:
1. Normally seen in manual workers.
2. Infective endocarditis.

Dark/black lesion below the nail plate:
1. Splinter haemorrhage.
2. Subungual haemorrhage.
3. Subungual melanoma.

Leuconychia (white patches in nail plates):
1. Seen in normal persons.
2. Hypoalbuminaemia.

Osler's nodes (swellings in the pulps of terminal phalanges):
1. Infective endocarditis (d/t vasculitis).

Heberden's nodes (osteophytes involving DIP joints):
1. OA.

Bouchard's nodes (osteophytes involving PIP joints):
1. OA.

Arachnodactyly (long and thin fingers):
1. Marfan's syndrome.

Palmar erythema (redness of thenar and hypothenar eminences):
1. Normal.

2 RA.
3 Thyrotoxicosis.
4 Liver disease.
5 Pregnancy.
6 OCPs.

Excessive palmar sweating:
1 Idiopathic.
2 Anxiety (palm is cold).
3 Thyrotoxicosis (palm is warm).

Puffiness of face:
1 Periorbital oedema (renal failure; acute glomerulonephritis → nephritic syndrome; nephrotic syndrome).
2 Myxoedema.
3 Angioedema.
4 Right heart failure (uncommonly and only when patient lies flat).

Redness of the cheeks (malar flush):
1 Normal.
2 Polycythaemia.
3 SLE.
4 Mitral stenosis.
5 Cushing's syndrome.

Xanthelasma:
1 Hyperlipidaemia.
2 Old age (even with normal lipid levels).

Subconjunctival haemorrhage:
1 Idiopathic.
2 Trauma.
3 Bleeding disorder.

Destruction of the nasal septum:
1 Wegener's granulomatosis.
2 Congenital syphilis.

Hyperpyrexia (rectal temperature ≥41°C/>106°F):
1 Heat stroke.
2 Malignant hyperpyrexia.
3 Neuroleptic malignant syndrome.
4 Pontine haemorrhage.
5 Thyrotoxic crisis.

6 Infection (malaria; septicaemia; viral infection).
7 Recreational drugs (amphetamines; cocaine).

Tongue enlargement:
1 Amyloidosis.
2 Acromegaly.
3 Thyroid pathology (myxoedema; cretinism).

Parotid swelling:
1 Parotid duct obstruction (d/t stone).
2 Parotid infection (suppurative parotitis; non-suppurative parotitis from ascending infection along parotid duct; mumps).
3 Parotid tumour.
4 Autoimmune disease (Sjögren's syndrome; sarcoidosis).

Lump in the face:
1 Parotid swelling.
2 Preauricular lymph node enlargement (infective/malignant).
3 Abscess/cyst (subcutaneous abscess; dental abscess; sebaceous cyst).
4 Malignancy (preauricular lymphoma; squamous/basal cell ca.; skin melanoma).

Hirsutism in female (hirsute upper lip, sideburns and chin; upwards extension of pubic hair):
1 Racial.
2 Ovarian pathology (PCOS; ovarian ca.).
3 Adrenal pathology (Cushing's syndrome; adrenal ca.).
4 Drugs (androgen analogues like Danazol, androgenic progestogens; phenytoin).

Hypertrichosis:
1 Hypothyroidism.
2 Malnutrition.
3 Porphyria.
4 Underlying malignancy (Hodgkin's lymphoma).
5 Drug-induced (minoxidil; corticosteroids; cyclosporin; penicillamine).

Submandibular lump (no movement with tongue protrusion or swallowing):
Submandibular gland enlargement:
1 Submandibular duct obstruction (d/t stone).
2 Submandibular infection (suppurative sialitis; non-suppurative sialitis from ascending infection along submandibular duct; mumps).
3 Submandibular tumour (adenocarcinoma; squamous cell ca., etc).
4 Autoimmune disease (Sjögren's syndrome; sarcoidosis).

Swellings other than submandibular gland:

1. Submandibular lymph node enlargement (infective/malignant).
2. Ranula (transilluminable cyst lateral to midline with domed bluish discolouration in the floor of mouth lateral to frenulum).
3. Submental dermoid (midline cyst in a <20-yr-old).

Anterior neck lump moving with tongue protrusion and swallowing (suggests extrathyroid lesion):

1. Thyroglossal cyst.
2. Ectopic thyroid tissue.

Neck lump moving with swallowing but not with tongue protrusion:

1. Goitre (euthyroid goitre; hypothyroid goitre; thyrotoxic goitre).

Goitre:

1. Sporadic.
2. Endemic (iodine deficiency).
3. Pregnancy.
4. Autoimmune thyroid disease.
5. Thyroiditis.
6. Drug-induced (ATDs;[3] lithium; amiodarone).

Euthyroid goitre:

1. Simple goitre.
2. Hashimoto's thyroiditis (euthyroid or hypothyroid).
3. Non-toxic MNG.
4. Thyroid enzyme deficiency – rare (euthyroid or hypothyroid).

Hypothyroid goitre:

1. Hashimoto's thyroiditis (euthyroid or hypothyroid).
2. Thyroid enzyme deficiency – rare (euthyroid or hypothyroid).

Hyperthyroid goitre:

1. Graves' disease.
2. Toxic MNG.
3. Toxic adenoma.

Solitary thyroid nodule:

1. Cyst (clinically euthyroid).
2. Adenoma (toxic/non-toxic).
3. Ca. (clinically euthyroid).

3 ATDs: antithyroid drugs.

Lump in anterior triangle (below digastric and in front of sternomastoid muscles):

1. Lymph node enlargement (infective; malignant [Hodgkin's or non-Hodgkin's lymphoma]).
2. Abscess (acute abscess; tuberculous 'cold' abscess).
3. Branchial cyst.
4. Cystic hygroma.
5. Carotid body tumour (chemodectoma).
6. Pharyngeal pouch.

Lump in posterior triangle (between sternomastoid and trapezius muscles):

1. Lymph node enlargement (infective/malignant [Hodgkin's or non-Hodgkin's lymphoma; metastases]).
2. Abscess (acute abscess; tuberculous 'cold' abscess).
3. Cystic hygroma.

Supraclavicular lump/s:

1. Lymph node enlargement (infective/malignant [Hodgkin's or non-Hodgkin's lymphoma; metastases from stomach or lungs]).
2. Subclavian artery aneurysm.

Axillary lymphadenopathy:

1. Lymph node enlargement (infective, e.g. viral prodrome, HIV infection, etc; malignant [reticulosis or primary tumour, metastases from ca. breast]).
2. Drugs (phenytoin; retroviral drugs).

Spider naevi:

1. In normal women (increased during periods).
2. OCPs.
3. Pregnancy.
4. Liver failure.

Early signs of PVD:

1. Shiny skin with loss of hair.

Pallor:

1. Anaemia.
2. Vasoconstriction (d/t shock, exposure to cold/Raynaud's phenomenon).

Subcutaneous emphysema:

1. Damage to the air-containing viscera (lungs; potentially pneumothorax, trachea; oesophagus).
2. Gas gangrene.

Campbell de Morgan's spots:

1. Commonly develop on the chest and abdomen with advancing age (red, 1–2 mm in diameter and don't blanch on pressure).

Erythema marginatum:

1. Rheumatic fever.

Galactorrhoea:

1. Pregnancy.
2. Prolactinoma.
3. Hypothyroidism.
4. Drugs (metoclopramide; domperidone; chlorpromazine and other tranquillisers).
5. Idiopathic.

Nipple abnormality:

1. Paget's disease of nipple with underlying malignancy.
2. Chronic infection → duct ectasia.
3. Duct papilloma.
4. Mamillary fistula (→ discharge from para-areolar region).

Breast lump/s:

1. Acute/chronic abscess.
2. Fat necrosis.
3. Cyst.
4. Fibroadenoma.
5. Benign fibrous mammary dysplasia.
6. Ca. (infiltrating ductal ca. or invasive lobar ca.).

Gynaecomastia:

1. Obesity.
2. Testicular problem (immature testes; primary or secondary hypogonadism; testicular tumour).
3. Chromosomal problem (Klinefelter's syndrome).
4. Cirrhosis of liver.
5. Ca. lung.
6. Drugs (digoxin; spironolactone; high alcohol intake).

Pressure sores:

1. Bedridden patient (d/t CVA; spinal cord injury, etc).
2. Poor nutrition.

Prominent leg veins (with or without leg swelling):

1. Varicose veins.

2 Thrombophlebitis.
3 DVT.

Unilateral ankle/calf oedema:

1 DVT/post-DVT venous insufficiency.
2 Cellulitis (from infection – primary, or secondary to insect bites).
3 Compression of large vein by tumour or lymph nodes.
4 Lymphatic obstruction/lymphoedema (infection, e.g. streptococcal lymphangitis [→ acute lymphatic obstruction]; filariasis/trypanosomiasis in tropics; malignant infiltration; Milroy's disease [congenital condition] – oedema present since childhood).
5 Ruptured Baker's cyst (leg swelling within seconds, usually while walking up a step; h/o arthritic knee usually present).
6 Immobility.

Common causes of bilateral ankle oedema:

1 CCF/cor pulmonale.
2 Hypoalbuminaemia d/t liver/renal disease; malnutrition, malabsorption or protein-losing enteropathy.
3 Poor venous return (d/t abdominal or pelvic masses, venous damage – postphlebitic or thrombotic).
4 Arterial/venous/lymphatic pathology (e.g. bilateral varicose veins/DVT; IVC obstruction; impaired lymphatic drainage).
5 Bilateral cellulitis.
6 Immobility.

Less-common causes of bilateral ankle oedema:

1 Pregnancy.
2 Immobility → dependent oedema.
3 Drugs (Ca^{2+} channel blockers, NSAIDs).
4 Wet beriberi (rare in western societies but commoner in Africa).
5 Idiopathic/cyclical oedema syndrome.

Pitting oedema (mechanism: ↑ interstitial fluid):

Generalised/bilateral:

1 Congestion (right heart failure; constrictive pericarditis; pericardial effusion; IVC/SVC obstruction).
2 Hypoproteinaemia (malnutrition; malabsorption; protein-losing enteropathy; cirrhosis of liver [→ ↓ protein synthesis]; nephrotic syndrome [→ ↑ protein loss]).
3 Pre-eclampsia.

Localised/unilateral:

1 Infection (e.g. cellulitis).

2 Vascular causes (venous obstruction; angioedema).
3 Immobile bedridden patient (d/t paralysis, etc).

Non-pitting oedema (mechanism: cutaneous deposition of some substance):

1 Myxoedema (deposition of mucopolysaccharide).
2 Angioedema (↑ capillary permeability → leakage and deposition of plasma proteins).
3 Lymphoedema (defective lymphatic drainage):
   a Lymphatic obstruction (infiltration by malignant cells; filariasis).
   b Iatrogenic (lymphadenectomy; irradiation of lymph nodes).
   c Congenital obstruction of lymphatics (Milroy's syndrome).

IVC obstruction:

1 Exogenous compression (large ascites; ovarian cyst; enlarged para-aortic lymph nodes).
2 Luminal obstruction (thrombosis).

SVC obstruction:

1 Exogenous compression/infiltration:
   a Malignant lesion of the mediastinum (ca. lung; lymphoma; metastases).
   b Benign lesion of the mediastinum (retrosternal thyroid; thymoma; cyst [dermoid/hydatid]; aortic aneurysm).
2 Luminal obstruction (thrombosis).

2 Cardiology

Tachycardia:

1. Physiologic (exercise; anxiety).
2. Anaemia.
3. Fever.
4. Thyroid problem (thyrotoxicosis).
5. Heart problem (tachyarrhythmias, e.g. SVT; IHD/MI; heart failure; hypotension/hypovolaemia; drugs like adrenaline, atropine, salbutamol, caffeine, alcohol, amphetamines).
6. Lung problem (hypoxia d/t any cause, e.g. pulmonary embolism).

Bradycardia:

1. Physiologic (athletes).
2. ↑ ICP.
3. Hypothermia.
4. Thyroid problem (hypothyroidism).
5. Heart problem (sick-sinus syndrome; complete heart block; drugs like digoxin, β-blockers – including eye drops; IHD/MI).
6. Obstructive jaundice.
7. Uraemia; electrolyte abnormalities.

Relative bradycardia:

1. Bacterial infections (enteric fever; meningitis with ↑ ICP).
2. Viral infections.

Sinus arrhythmia[1] (HR faster during inspiration; slower during expiration):

1. Physiologic.

Occasional premature beats:

1. Physiologic.

(Frequent premature beats in a heart patient may be pathologic).

Regularly irregular beats:

1. Digoxin toxicity (ventricular bigeminy/trigeminy).

1 Disappears in heart failure and autonomic neuropathy.

Irregularly irregular beats:

1. AF.
2. Atrial flutter with variable heart block (secondary to IHD, etc).
3. Frequent premature beats (secondary to IHD, etc).
4. Wenckebach heart block (secondary to IHD, etc).

Pulse deficit (difference between pulse rate and heart rate – counted by auscultation):

1. AF.

Slow-rising pulse (pulsus plateau):

1. AS (narrow pulse pressure).

Collapsing pulse (water-hammer pulse):

1. AR (wide pulse pressure).
2. VSD.
3. PDA.
4. Severe anaemia.

Pulsus bisferiens (two systolic peaks are palpable in one pulse; normal is one):

1. Combined AS and AR.

Jerky pulse:

1. HOCM.

Pulsus paradoxus (pulse becomes weak or impalpable during inspiration):

1. Pericardial pathology (massive pericardial effusion/cardiac tamponade; constrictive pericarditis).
2. Acute severe bronchial asthma (pulse pressure remains unchanged; in cardiac tamponade, it is reduced).

Pulsus alternans (a strong beat alternates with a weak beat and the beat-to-beat interval remains constant):

1. LVF.
2. SVT.

Pulsus bigeminus (a strong normal beat alternates with a weak premature beat; strong and weak beats occur close together and are followed by a long pause before the next pair):

1. Digoxin toxicity.

Radio–femoral delay:

1. Coarctation of aorta.

Low-volume pulse:

1. AS.
2. Heart failure (d/t any cause).
3. Hypovolaemic shock.
4. Septic shock (→ poor vascular tone → low-volume pulse).

High-volume pulse:

1. Fever.
2. Severe anaemia.
3. Bradycardia (d/t any reason) with normal myocardium.
4. AR.
5. Hyperkinetic circulation (d/t fever; AV fistula; hypercapnia; thyrotoxicosis; Paget's disease).

Absent radial pulse:

1. Aortic dissection with subclavian involvement.
2. Trauma/surgery/catheterisation.
3. Arterial embolism.
4. Takayasu's arteritis.

Aortic dissection – predisposing conditions:

1. Essential HTN (in patients of Afro-Caribbean origin); secondary HTN (d/t coarctation of aorta).
2. Marfan's syndrome.
3. Ehlers–Danlos' syndrome.
4. Pregnancy.
5. Relapsing polychondritis.

Secondary hypertension:

1. Vascular hypertension (coarctation of aorta; subclavian artery stenosis).
2. Renal disease (glomerulonephritis; pyelonephritis; renal artery stenosis; ADPKD[2]).
3. Endocrine hypertension (Cushing's syndrome; Conn's syndrome/primary hyperaldosteronism; phaeochromocytoma; acromegaly).
4. OCPs; pregnancy (pre-eclampsia).
5. Neurogenic hypertension (post head injury; post stroke).
6. Drugs (OCPs – oestrogen containing; steroids; NSAIDs; erythropoietin).

Low blood pressure:

1. Cardiogenic shock.
2. Hypovolaemic shock.

2 USG criteria for ADPKD: age <30 yr – at least two cysts (unilateral or bilateral); 30–60 yr – at least two cysts in each kidney; >60 yr – at least four cysts in each kidney.

3 Loss of vascular tone (d/t septicaemia; adrenal failure, etc).

Postural hypotension (supine and standing BP after 1 minute – >10 mmHg fall; may be accompanied by dizziness):

1 Idiopathic (especially in elderly).
2 Autonomic neuropathy (d/t DM; tabes dorsalis – rarely).
3 Drugs: (high dose of antihypertensives; antidepressants; antipsychotics [phenothiazines]; antiparkinsonian drugs [L-dopa; carbidopa]).

BP/pulse difference between arms (R > L by 15 mmHg):

1 Aortic pathology (congenital supravalvular aortic stenosis; dissection of ascending aorta; aortic-arch syndrome/Takayasu's syndrome).
2 Subclavian steal syndrome.
3 Thoracic inlet syndrome.
4 Old or new thrombosis in an already atheromatous artery/aneurysm (complication of PVD).

BP/pulse difference between arm and legs (R > L by 15 mmHg) – wide cuff needed for thigh; arm and leg must be at same level:

1 Coarctation of aorta.
2 Dissection of descending thoracic aorta, abdominal aorta, iliac arteries (especially in a diabetic).
3 Old or new thrombosis in an already atheromatous artery/aneurysm (complication of PVD).

JVP:

1 **a wave:** right atrial contraction – end of atrial systole.
2 **x descent:** right atrial relaxation – beginning of atrial diastole.
3 **v wave:** right atrial filling – end of atrial diastole.
4 **y descent:** right atrial emptying – beginning of right ventricular diastole.

c wave is thought to be artefact seen on **x descent** (right atrial relaxation – beginning of atrial diastole) d/t carotid pulsations.

Prominent 'a' wave:

1 Right atrial and ventricular hypertrophy (d/t pulmonary hypertension; PS; TR).

Strikingly large 'a' waves (cannon waves) – seen when right atrium contracts against closed tricuspid valve:

1 Complete heart block.
2 Ventricular pacing with intact retrograde conduction.

Absent 'a' wave:

1. AF.

Prominent and palpable systolic wave on JVP:

1. TR (called ventricularisation of venous pulse).

Rapid 'y' descent:

1. Constrictive pericarditis.

Raised JVP:

1. Fluid overload.
2. Right heart failure:
   a. Secondary to left heart failure.
   b. Cor pulmonale.[3]
   c. IHD.
   d. Valvular/septal heart disease (PS; TS; TR; ASD).
   e. Cardiomyopathy.
3. Arrhythmia (AF; complete heart block).
4. Pericardial disease (pericardial effusion; constrictive pericarditis).
5. Jugular vein obstruction.

Kussmaul's sign:

1. Constrictive pericarditis.
2. Tricuspid stenosis.
3. Right ventricular infarction.
4. Restrictive cardiomyopathy.

Pulsations along the parasternal border:

1. Right ventricular hypertrophy.

Pulsations in the left 2nd ICS:

1. Dilatation of the pulmonary artery.

Pulsations in the right 2nd ICS:

1. Aortic aneurysm.

Pulsations in the suprasternal notch:

1. AR.

Pulsations in the epigastrium:

1. Physiologic in thin individuals (d/t aorta).
2. AAA.

3 D/t: COPD; interstitial lung disease; recurrent pulmonary embolism; kyphoscoliosis.

3 Pulsatile liver (d/t TR).
4 Right ventricular hypertrophy.

Whole of the precordium moves with each cardiac beat:
1 Heart is greatly enlarged.

Apex beat shift:
Cardiac causes:
1 Hypertension.
2 IHD.
3 Valvular pathology (MR; AS; AR; VSD).
4 Dilated cardiomyopathy.
5 Occasionally d/t a grossly dilated right ventricle.

Non-cardiac causes:
1 Position of the patient (left lateral or sitting forward).
2 Chest deformity.
3 Pleural/pulmonary pathology causing mediastinal push/pull.

Impalpable apex beat:
1 Obesity/thick chest wall.
2 Lung pathology: emphysema.
3 Pericardial pathology: pericardial effusion.
4 Dextrocardia (apex beat palpable on the right side).

Tapping apex beat (beat is forceful but the palpating finger is not displaced):
1 Palpable loud 1st heart sound d/t MS.

Heaving apex beat (palpating finger is lifted):
Ill-sustained heave (occurs when left ventricle has to eject large blood volume):
1 MR.
2 AR.
3 VSD.

In all these conditions, left ventricle is enlarged. Thus apex beat is always displaced, as well as heaving.

Well-sustained heave (occurs when the left ventricle has to contract against high resistance):
1 AS.
2 Hypertension.

In both these conditions, left ventricle is hypertrophied initially but not enlarged. Thus apex beat, though heaving, is not displaced.

Apex beat displaced and heaving:

1. MR.
2. AR.
3. VSD.

Apex beat not displaced but heaving:

1. AS.
2. Hypertension.

Left parasternal heave (d/t right ventricular enlargement):

1. Pulmonary hypertension (which in turn could be d/t MS/MR; COPD, CRAD; primary pulmonary hypertension; kyphoscoliosis; longstanding ASD, VSD, PDA).
2. PS.
3. Fallot's tetralogy.

Palpable 1st heart sound (called tapping apex beat):

1. MS.

Palpable P_2:

1. Pulmonary hypertension.

Palpable A_2:

1. Systemic hypertension.

Palpable thrill in the neck (carotid shudder):

1. AS.

Loud S_1:

1. Tachycardia.
2. MS.

Soft S_1:

1. MR.
2. Heart failure.
3. Rheumatic carditis.

Variable intensity of S_1:

1. AF.
2. Complete heart block.

Loud P_2:
1. Pulmonary hypertension.

Soft P_2:
1. PS.

Loud A_2:
1. Systemic hypertension.

Soft A_2:
1. AS.
2. AR.

Both S_1 and S_2 are soft:
1. Thick chest wall.
2. Lung pathology: emphysema.
3. Pericardial pathology: pericardial effusion.

Splitting of S_1:
1. Arrhythmia (RBBB; LBBB; VT).
2. Valvular/septal defect (ASD; TS).

Splitting of S_2:
Usual splitting (A_2–P_2 interval is more during inspiration than expiration):
1. Physiologic (in children and young adults).
2. Pulmonary hypertension (split is narrow).
3. PS.
4. Dilated right ventricle.
5. RBBB.

Fixed splitting (A_2–P_2 interval is constant and wide):
1. ASD.

Reverse splitting (A_2–P_2 interval is less during inspiration than expiration):
1. Severe AS.
2. HOCM
3. LBBB.

S_3:
1. Physiologic (in children and young adults).
2. Pathological:[4]

4 D/t rapid ventricular filling.

a MR.
b Heart failure.
c Cardiomyopathy.
d Constrictive pericarditis.
e Hyperdynamic states (e.g. thyrotoxicosis; pregnancy).

S_4:
1 Physiologic (in children and young adults).
2 Pathological:[5]
a Left ventricular hypertrophy (d/t hypertension, HOCM).
b IHD (following MI → scarring of the infarcted tissue).
c Amyloid heart disease.

Gallop rhythm (S_3 + S_4):
1 Severe heart failure.

Heart failure → S_3; severe heart failure → gallop rhythm (S_3 + S_4).

Pericardial knock:
1 Pericardial pathology (constrictive pericarditis).

Opening snap after S_2 medial to the apex:
1 MS (it means that the valve cusps are stenosed but mobile).

Ejection systolic clicks soon after S_1:
Aortic click (at A_1 area and apex not affected by respiration):
1 AS.
2 Bicuspid aortic valve.

Pulmonary click (at pulmonary area and increases in intensity during expiration):
1 PS.
2 Dilatation of pulmonary artery (secondary to pulmonary hypertension or idiopathic).

Mid-systolic click:
1 MVP.

Systolic murmurs:
Pansystolic:
1 MR.

5 D/t the increased atrial contraction required to fill a stiff left ventricle. It doesn't occur in AF.