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Chapter 5

Notes & Notes for MRCP
By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology

Pericardial effusion Causes • infectious pericarditis: viral, tuberculosis, pyogenic spread from septicaemia and pneumonia • uraemia • idiopathic • post myocardial infarction (including Dressler's syndrome) • malignancy • heart failure • nephrotic syndrome • hypothyroidism • trauma CT is the most appropriate investigation provide more information than Echo quicker to obtain than (MRI). Investigations • ECG of pericardial effusion ECG reveals electrical alternans, which is caused by a "swinging" movement of the heart in a large effusion

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Constrictive pericarditis The right sided failure, ascites and pericardial calcification on x ray suggest a diagnosis of constrictive pericarditis. Pathophysiology • Inflammation of the pericardium fibrosis and constriction Risk factors • previous cardiac surgery • previous pericarditis, • radiotherapy • connective tissue disease Causes • Mediastinal irradiation • TB :Tuberculous pericarditis is the commonest cause of constrictive pericarditis worldwide. • any cause of purulent pericarditis

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Features • dyspnoea • right heart failure: elevated JVP, ascites, oedema, hepatomegaly • JVP shows prominent x and y descent • pericardial knock - loud S3 • Kussmaul's sign is positive (rise in JVP on inspiration) Investigations • CXR pericardial calcification can detect effusions only if larger than 250 mL. • Echocardiography Indication to assess for pericardial effusion and cardiac tamponade the best diagnostic tool for diagnosing pericardial effusion. shows no increase in the venous return with inspiration. The key differences between constrictive pericarditis and cardiac tamponade are summarized in the table below: Cardiac tamponade Constrictive pericarditis JVP Absent Y descent X + Y present Pulsus paradoxus Present Absent Kussmaul's sign* Rare Present Characteristic features Pericardial calcification on CXR • Kussmaul's sign* a paradoxical rise in jugular venous pressure (JVP) on inspiration • Kussmaul's sign (a rise in the JVP on inspiration) is more likely to be seen in constrictive pericarditis than cardiac tamponade. Treatment • The first line of treatment of symptomatic constrictive pericarditis is pericardiotomy.

Cardiac tamponade Cardiac tamponade is characterised by Beck's triad of: • hypotension • raised JVP (with absent Y descent), and • muffled heart sounds. Definition • an accumulation of pericardial fluid under pressure, leading to impaired cardiac filling and hemodynamic compromise Features • dyspnoea • raised JVP, with an absent Y descent - this is due to the limited right ventricular filling • tachycardia • Hypotension the best clinical features that distinguishes cardiac tamponade from constrictive pericarditis

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 hypotension is a late feature in constrictive pericarditis. • muffled heart sounds • pulsus paradoxus an exaggerated inspiratory decrease in systolic blood pressure • Kussmaul's sign Rare Most common in constrictive pericarditis • impalpable apex beat Investigations • ECG : tachycardia, low voltage, electrical alternans, (due to the swinging movement of the heart). beat-to-beat variation in QRS-axis and amplitude. • chest x-ray (enlarged cardiac silhouette with clear lung fields), • echocardiogram (chamber collapses, abnormal venous flows, exaggerated respiratory variation of cardiac and venous flows). Treatment • pericardiocentesis. Hypotension is the best clinical features that distinguishes cardiac tamponade from constrictive pericarditis hypotension is a late feature in constrictive pericarditis.

Hypertension (NICE guidelines 2019) Definition • Essential hypertension is defined as blood pressure (BP) ≥140/90 mmHg, with no secondary cause identified. Causes • Essential hypertension (95% of patients) No specific cause known. Multifactorial etiology including genetic and environmental factors • Secondary hypertension (5% of patients) RECENT: Renal (e.g., renal artery stenosis, glomerulonephritis), Endocrine (e.g., Cushing syndrome, hyperthyroidism, Conn syndrome), Coarctation of the aorta, Estrogen (oral contraceptives), Neurologic (raised intracranial pressure, psychostimulants use), Treatment (e.g., glucocorticoids, NSAIDs) are the causes of secondary hypertension. When a question says: ‘What is the most likely diagnosis?’ think about what is epidemiologically the most common cause of hypertension? Therefore the answer is essential hypertension. The most likely cause of hypertension in an obese is still essential hypertension.

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Diagnosis Hypertension - NICE now recommend ambulatory blood pressure monitoring to aid diagnosis Confirm diagnosis of hypertension in people with a: clinic blood pressure of 140/90 mmHg or higher and ABPM daytime average or HBPM average of 135/85 mmHg or higher. Measuring blood pressure • Palpate the radial or brachial pulse before measuring blood pressure with automated devices . If pulse irregularity is present, measure BP manually using direct auscultation over the brachial artery, because automated devices may not measure BP accurately if there is pulse irregularity (for example, due to atrial fibrillation). • Measure BP in both arms If the difference between arms ˃ 15 mmHg repeat BP . If the difference remains ˃ 15 mmHg: Subsequent BP should be recorded from the arm with the higher reading. Look for cases of

unequal BP from the arms, e.g. supravalvular aortic stenosis. • If BP in the clinic ≥140/90 mmHg: Take a second measurement during the consultation. If the second measurement is substantially different from the first, take a third measurement. Record the lower of the last 2 measurements as the clinic blood pressure. • If clinic BP is between 140/90 mmHg and 180/120 mmHg, offer ambulatory blood pressure monitoring (ABPM) to confirm the diagnosis of hypertension. If ABPM is unsuitable or not tolerated, offer home blood pressure monitoring (HBPM). • In people with symptoms of postural hypotension (falls or postural dizziness): measure BP with the person either supine or seated and with the person standing for at least 1 minute before measurement. If the systolic BP falls by 20 mmHg or more when the person is standing: review medication measure subsequent BP with the person standing consider referral to specialist care if symptoms of postural hypotension persist. • Ambulatory blood pressure monitoring (ABPM) The use of ambulatory blood pressure monitoring (ABPM) aims to: prevent diagnosing 'white coat hypertension' as having hypertension in patients whose blood pressure climbs 20 mmHg whenever they enter a clinical setting. ABPM has been shown to be a more accurate predictor of cardiovascular events than clinic readings. at least 2 measurements per hour during the person's usual waking hours (for example, between 08:00 and 22:00) use the average value of at least 14 measurements If ABPM is not tolerated or declined HBPM should be offered.

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• Home blood pressure monitoring (HBPM) for each BP recording, two consecutive measurements need to be taken, at least 1 minute apart and with the person seated BP should be recorded twice daily, ideally in the morning and evening BP should be recorded for at least 4 days, ideally for 7 days discard the measurements taken on the first day and use the average value of all the remaining measurements. Hypertension terms used in NICE guidelines 2019 Term Definition Hypertension clinic BP of ≥140/90 mmHg or higher and ABPM daytime average or HBPM average of ≥135/85 mmHg. White-coat hypertension A discrepancy of more than 20/10 mmHg between clinic and average daytime ABPM or average HBPM measurements at the time of diagnosis. Masked hypertension Clinic BP measurements are normal (˂140/90 mmHg), but higher when taken outside the clinic using average daytime ABPM or average HBPM BP measurements. Persistent hypertension High blood pressure at repeated clinical encounters. severe hypertension Stage 3 hypertension: Clinic systolic BP ≥ 180 mmHg or clinic diastolic BP ≥ 120 mmHg. Accelerated hypertension (malignant hypertension) A severe increase in BP to ≥180/120 mmHg (and often over 220/120 mmHg) with signs of retinal haemorrhage and/or papilloedema (swelling of the optic nerve). It is usually associated with new or progressive target organ damage and is also known as malignant hypertension. Hypertension Stages (NICE guidelines 2019) Stage Criteria Stage 1 hypertension Clinic BP ≥140/90 mmHg and subsequent ABPM daytime average or HBPM average BP ≥135/85 mmHg Stage 2 hypertension Clinic BP ≥160/100 mmHg and subsequent ABPM daytime average or HBPM average BP ≥ 150/95 mmHg Stage 3 or severe hypertension Clinic systolic BP ≥180 mmHg, or clinic diastolic BP ≥120 mmHg Management (NICE guidelines 2019) Non-pharmacological management • Lifestyle advice is the first line in hypertension management weight reduction: Of all the lifestyle modifications, weight reduction produces the greatest reduction in BP (A 10 kg weight loss is expected to decrease BP by 15–20 mmHg) low salt diet,

aiming for less than 6g/day, ideally 3g/day. (reducing salt intake by 6g/day can lower systolic blood pressure by 10mmHg) low caffeine intake.

 stop smoking, drink less alcohol If a patient on antihypertensive and drink alcohol Reduction of alcohol intake is the next step in treatment. Starting antihypertensive drug treatment • any age with persistent stage 2 hypertension. • age ˂ 60 years with stage 1 hypertension and an estimated 10-year cardiovascular risk below 10%. • age ˂ 80 years with stage 1 hypertension who have 1 or more of the following: target organ damage established cardiovascular disease renal disease diabetes an estimated 10-year risk of cardiovascular disease of 10% or more. • age ˃ 80 years with stage 1 hypertension if their clinic BP ˃150/90 mmHg • For patients ˂ 40 years consider specialist referral to exclude secondary causes. Pharmacological management : Steps of hypertension treatment • Step 1 treatment Age ≤ 55 OR any age, with T2DM with no black African origin ACEi or ARB Age ≤ 55 OR any age, with T2DM with black African origin ARB Age ≥ 55 without T2DM CCB black African origin of any age without T2DM CCB With heart failure thiazide-like diuretic, such as indapamide in preference to a conventional thiazide diuretic such as bendroflumethiazide or hydrochlorothiazide. • Step 2 treatment If BP not controlled on ACEi or ARB Add CCB or thiazide-like diuretic. If BP not controlled on CCB Add ACEi or ARB or thiazide-like diuretic. If BP not controlled on CCB in a black African Add ARB (in preference to an ACEi) • Step 3 treatment If BP not controlled with step 2 treatment ACEi or ARB and CCB and thiazide-like diuretic. • Step 4 treatment BP not controlled with the optimal tolerated doses of ACEi or ARB and CCB and thiazide-like diuretic Resistant hypertension Before considering further treatment for a person with resistant hypertension:

1. Confirm elevated clinic blood pressure measurements using ambulatory or home blood pressure recordings.
2. Assess for postural hypotension.
3. Discuss adherence Confirmed resistant hypertension: blood potassium ≤ 4.5 mmol/l further diuretic therapy with low-dose spironolactone monitor blood sodium and potassium and renal function within 1 month of starting treatment and repeat as needed thereafter. blood potassium ˃ 4.5 mmol/l alpha-blocker or beta-blocker

Cardiology Drug choice • hypertensive with benign prostatic hyperplasia alpha-blockers • hypertensive with heart failure or angina beta-blockers • hypertensive post myocardial infarction either a beta blocker or ACE inhibitor would be the agent of choice. • calcium channel blockers are now considered superior to thiazides • bendroflumethiazide is no longer the thiazide of choice Use of multiple anti-hypertensives at low doses is preferable to having fewer tablets at higher doses, in view of the synergistic effectiveness of targeting several mechanisms of hypertension. Blood pressure targets Clinic BP ABPM / HBPM Age < 80 years 140/90 mmHg 135/85 mmHg Age > 80 years 150/90 mmHg 145/85 mmHg Recommendations for BP target • British Hypertension Society Guidelines for Hypertension Management (BHS-IV) recommend a goal BP of less than 130/80

mmHg for patients with diabetes, renal impairment and established cardiovascular disease;

Hypertensive emergency Definition Hypertensive emergency: systolic BP ≥ 180 or diastolic BP ≥ 110 + end organ damage Presentation • The most common clinical presentations of hypertensive emergencies are: cerebral infarction (24.5%) pulmonary edema (22.5%), hypertensive encephalopathy (16.3%), congestive heart failure (12%). Other presentations include intracranial hemorrhage, aortic dissection, and eclampsia as well as acute myocardial infarction. Management Labetalol has both alpha- and beta-adrenoreceptor antagonistic activity and is the first choice for hypertensive crises where the aetiology is initially unclear. • Gradual blood pressure lowering over the first 24 hours in the first hour : reduce mean arterial pressure (MAP) by 10 – 20 % in the next 23 hours: 5% to 15%, so that the final BP is reduced by 25% compared with baseline. • IV antihypertensive : e.g. Labetolol The major risk of any oral agent used for hypertensive emergencies is ischaemic symptoms (for example myocardial infarction, angina pectoris or stroke) due to an excessive and uncontrolled hypotensive response usually due to lowering of BP to below the autoregulatory threshold. Therefore the use of oral agents should generally be avoided in the treatment of hypertensive emergencies if parenteral drugs are available. • The exceptions to gradual BP lowering over the first 24 hours are: Acute ischemic stroke – The BP should not lowered unless it is ≥185/110 mmHg in patients who are candidates for reperfusion therapy or ≥220/120 mmHg in patients who are not candidates for reperfusion therapy. Acute aortic dissection – The systolic BP should rapidly lowered to a target of 100 to 120 mmHg (to be attained in 20 minutes). Spontaneous hemorrhagic stroke – The systolic BP can be rapidly reduced if no contraindications exist. MAP = diastolic blood pressure + [(systolic blood pressure - diastolic blood pressure)/3] Or MAP = (2x diastolic + systolic)/3

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Parenteral drugs for treatment of hypertensive emergencies Side effects (SE) Notes Labetalol Nausea/vomiting, paresthesias (eg, scalp tingling), bronchospasm, dizziness, nausea, heart block Avoid in acute decompensated heart failure. Use cautiously in obstructive or reactive airway. Beta-blocker should not be withdrawn abruptly, but gradually tapered to avoid acute tachycardia, hypertension, and/or ischemia. Nitroglycerin (glyceryl trinitrate) (Adrenergic inhibitor) Beta-Blocker With Alpha-Blocking Activity Hypoxemia, tachycardia (reflex sympathetic activation), headache, vomiting, flushing, methemoglobinemia, tolerance with prolonged use (Vasodilators) Nicardipine Tachycardia, headache, dizziness, nausea, flushing, local phlebitis, edema (Vasodilators) Calcium Channel Blocker, Dihydropyridine Clevidipine Atrial fibrillation (most common SE), nausea, lipid formulation contains potential allergens (eg, soy, egg) Avoid in patients with defective lipid metabolism (hypertriglyceridemia is an expected SE). Patients who develop hypertriglyceridemia (eg, >500 mg/dL) are at risk of developing pancreatitis. Dihydropyridine calcium channel blockers may cause negative inotropic effects and exacerbate HF. Hydralazine (Vasodilators) Calcium Channel Blocker, Dihydropyridine Sudden precipitous drop in blood pressure, tachycardia, flushing,

headache, vomiting, aggravation of angina (Vasodilators) Direct vasodilation of arterioles Nitroprusside (Vasodilators) Elevated intracranial pressure, decreased cerebral blood flow, reduced coronary blood flow in CAD, cyanide and thiocyanate toxicity, nausea, vomiting, muscle spasm, flushing, sweating Phentolamine Tachycardia, flushing, headache, nausea/vomiting (Adrenergic inhibitor) Alpha 1 Blocker Notes & Notes for MRCP
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used as adjunctive therapy for patients with acute coronary syndrome or acute pulmonary edema. contraindicated in patients with increased intracranial pressure (eg, intracranial hemorrhage) Avoid use in acute heart failure. Caution with coronary ischemia. In general, hydralazine should be avoided due to its prolonged and unpredictable hypotensive effect. Contraindicated in coronary artery disease; mitral valve rheumatic heart disease and SLE. In general, nitroprusside should be avoided due to its toxicity. avoid in AMI, CAD, CVA, elevated intracranial pressure, renal or hepatic impairment. Alternative option for catecholamine excess (eg, adrenergic crisis secondary to pheochromocytoma or cocaine overdose).

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Hypertensive urgency Definition Hypertensive urgency: systolic BP ≥ 180 or diastolic BP ≥ 110 + NO end organ damage Presentation • Asymptomatic patient with a BP in the "severe" range (ie, ≥180/≥120 mmHg) • Often a mild headache, but no signs or symptoms of acute end-organ damage. Management • All patients should be provided a quiet room in which to rest. This may produce a fall in blood pressure ≥20/10 mmHg in approximately one-third of adults. If this is not effective, antihypertensive drugs may be given. • Gradual lowering of the BP over a period of hours to days to <160/<100 mmHg or no more than 25 to 30% of baseline BP. • The risk of adverse events (eg, stroke or myocardial infarction) that may occur if the BP is lowered too rapidly or to a level below the ability for autoregulation to maintain adequate tissue perfusion. • Can often be safely managed in the clinician's office • Add or modify oral antihypertensive

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Malignant hypertension (Accelerated hypertension) A patient with malignant hypertension always has retinal papilledema Definition • BP ≥180/120 mmHg (often over 220/120 mmHg) with signs of retinal haemorrhage and/or papilloedema (swelling of the optic nerve). • It is usually associated with new or progressive target organ damage Pathophysiology • The pathologic hallmark of malignant hypertension is fibrinoid necrosis of the arterioles which occurs systemically, but specifically in the kidneys. Management : as hypertensive emergency

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Secondary hypertension General indicators of secondary hypertension • Young age (< 40 years) at onset of hypertension • Onset of diastolic hypertension at an older age (> 55 years) • Abrupt onset of hypertension • End-organ damage that is disproportionate to the degree of hypertension • Recurrent hypertensive crises • Resistant hypertension: hypertension that is resistant to treatment

with at least three antihypertensives of different classes including a diuretic Causes • Primary hyperaldosteronism, including Conn's syndrome (5-10% of hypertensive patients) the single most common cause of secondary hypertension ↑BP + ↓K+ + ↑ Aldosterone CT or MRI of the abdomen identifies a secretory adrenal adenoma • Renal diseases: include glomerulonephritis pyelonephritis Reflux-associated scarring is the commonest renal disease. This will cause abnormalities on dimercaptosuccinic acid (DMSA) scan. adult polycystic kidney disease renal artery stenosis • Coarctation of the aorta (the commonest non-renal cause) • Endocrine disorders (other than primary hyperaldosteronism): phaeochromocytoma Cushing's syndrome Liddle's syndrome (↑BP + ↓K+ + ↑Na+) hypokalaemic hypertension metabolic alkalosis low plasma renin and aldosterone (called pseudo-hyperaldosteronism).

 congenital adrenal hyperplasia (11-beta hydroxylase deficiency) acromegaly • Fibromuscular dysplasia, a rare cause of hypertension and hypokalaemia, more common in women. It causes hyperreninaemic hyperaldosteronism. • Pregnancy (PIH, pre-eclampsia , eclampsia) • Drugs Liquorice ingestion causes a primary aldosterone type picture. It is caused by glycyrrhizic acid contained in liquorice, blocking the enzyme 11b hydroxysteroid dehydrogenase. This prevents the inactivation of cortisol, which in turn activates mineralocorticoid receptors in the kidney. driving hypokalaemic metabolic alkalosis with hypertension. NSAIDs, combined oral contraceptive pill, steroids, MAOI Different diagnostics for causes of secondary hypertension Diagnostic findings Underlying condition Hypokalaemia Conn syndrome Renal artery stenosis Metabolic alkalosis and ↑ aldosterone-to-renin ratio Difference in blood pressure in both arms Takayasu arteritis Aortic dissection Aortic arch syndrome Subclavian steal syndrome Of upper and lower limbs Coarctation of the aorta distal to the left subclavian artery Daytime sleepiness (Epworth scale, Berlin questionnaire) Nondipping in 24-hour blood pressure monitoring (the failure of BP to fall by ≥10% during sleep.) Increased 24-hour urinary metanephrines Pheochromocytoma ↑ Serum calcium, ↑ PTH level, ↓ serum phosphates ↑ Serum cortisol Excess of glucocorticoids (e.g., Cushing syndrome) ↓TSH, ↑ free T4 Hyperthyroidism MRCPUK- part 2- March 2017 : A 28-year-old woman of Afro-Caribbean ethnic origin c/o difficultto-manage hypertension, despite taking maximal-dose amlodipine and indapamide. The GP trialled an ACE inhibitor, but this was discontinued due to a rise in serum creatinine. Renin and aldosterone are both Elevated. K is 3.1 mmol. Which of the following is the most likely diagnosis? Fibromuscular renal artery dysplasia This patient’s age and ethnicity suggest that her hypertension is related to fibromuscular dysplasia rather than to atherosclerotic renal artery stenosis. The renin and aldosterone elevation, coupled with hypokalaemia and deterioration in renal function on starting ACE inhibitors, are consistent with the diagnosis. Notes & Notes for MRCP By Dr. Yousif Abdallah Hamad

 Conn syndrome Obstructive sleep apnoea Hyperparathyroidism

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Hypokalaemia and hypertension Hypokalaemia with hypertension Hypokalaemia without hypertension Cushing's syndrome

 Conn's syndrome (primary hyperaldosteronism) Liddle's syndrome (autosomal dominant disorder that mimics hyperaldosteronism) • renal artery stenosis • 11-beta hydroxylase deficiency 21-hydroxylase deficiency, which accounts for 90% of congenital adrenal hyperplasia cases, is not associated with hypertension • Carbenoxolone, an anti-ulcer drug, and liquorice excess • The first step in case of (↑ BP + ↓ K+) should be further simple investigations Plasma renin and aldosterone levels Cushing's & Conn's high aldosterone and a low renin, Renal artery stenosis high renin and aldosterone Liddle's syndrome low renin and aldosterone. Notes & Notes for MRCP
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 Diuretics GI loss (e.g. Diarrhoea, vomiting) renal tubular acidosis (type 1 and 2) type 4 renal tubular acidosis is associated with hyperkalaemia. Bartter's syndrome Gitelman syndrome

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Hypertension in pregnancy Physiology • The blood pressure in normal pregnancy: usually falls in the first trimester (particularly the diastolic), and continues to fall until 20-24 weeks after this time the blood pressure usually increases to pre-pregnancy levels by term Definition • Hypertension in pregnancy in usually defined as: systolic > 140 mmHg or diastolic > 90 mmHg or an increase above booking readings of > 30 mmHg systolic or > 15 mmHg diastolic Classification Pre-existing hypertension Pregnancy-induced hypertension (PIH, also known as gestational hypertension) A history of hypertension before pregnancy or BP > 140/90 mmHg before 20 weeks gestation Hypertension (as defined above) occurring in the second half of pregnancy (i.e. after 20 weeks) No proteinuria, no oedema No proteinuria, no oedema Oedema may occur but is now less commonly used as a criterion Occurs in 3-5% of pregnancies and is more common in older women Occurs in around 5-7% of pregnancies Resolves following birth (typically after one month). Women with PIH are at increased risk of future pre-eclampsia or hypertension later in life Notes & Notes for MRCP
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Pre-eclampsia Pregnancy-induced hypertension in association with proteinuria (> 0.3g / 24 hours) Occurs in around 5% of pregnancies

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Treatment of chronic hypertension with pregnancy • Pre-pregnancy advice: If they are taking ACE inhibitors or ARBs, thiazide or thiazide-like diuretics and planning for pregnancy discuss an alternative antihypertensive treatment, stop it if they become pregnant. the limited evidence available has not shown an increased risk of congenital malformation with any other antihypertensive. • Best antihypertensive: 1st line : labetalol 2nd line: nifedipine (if labetalol is not suitable) 3rd line: methyldopa (if both labetalol and nifedipine are not suitable) • Target BP: 135/85 mmHg • Aspirin 75–150 mg once daily from 12 weeks. • Offer placental growth factor (PlGF)-based testing to help rule out pre-eclampsia between 20 weeks and up to 35 weeks of pregnancy, if women with chronic hypertension or PIH are suspected of developing pre-eclampsia.

Treatment of hypertension in the postnatal period • If women not planning to breastfeed →treat as hypertension in general • If women planning to breastfeed: 1st line: non-black African or Caribbean women: enalapril black African or Caribbean women: nifedipine or amlodipine if the woman has previously used this to successfully control her BP. 2nd line: combination of nifedipine (or amlodipine) and enalapril 3rd line: add atenolol or labetalol to the combination treatment or swapping 1 of the medicines already being used for atenolol or labetalol. • avoid using diuretics or angiotensin receptor blockers for women who are breastfeeding. Treatment of hypertension in the postnatal period (NICE guidelines June 2019) • If women not planning to breastfeed treat as hypertension in general • If women planning to breastfeed: 1st line: non-black African or Caribbean women: enalapril black African or Caribbean women: nifedipine or amlodipine if the woman has previously used this to successfully control her BP. 2nd line: combination of nifedipine (or amlodipine) and enalapril 3rd line: add atenolol or labetalol to the combination treatment or swapping 1 of the medicines already being used for atenolol or labetalol. • Avoid using diuretics or angiotensin receptor blockers for women who are breastfeeding.

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Pre-eclampsia/Eclampsia Definitions • Pre-eclampsia: is a condition seen after 20 weeks gestation characterised by pregnancyinduced hypertension in association with proteinuria. use albumin: creatinine ratio (8 mg/mmol) or protein: creatinine ratio (≥30 mg/mmol) to confirm significant proteinuria (Do not use 24-hour proteinuria or first morning urine void). • Eclampsia: development of seizures in association pre-eclampsia. Risk factors •

> 40 years old • nulliparity (or new partner) • multiple pregnancy • body mass index > 30 kg/m^2 • diabetes mellitus • pregnancy interval of more than 10 years • family history of pre-eclampsia • previous history of pre-eclampsia • pre-existing vascular disease such as hypertension or renal disease • There is some evidence to suggest that pre-eclampsia is actually less common in smokers Features of pre-eclampsia • severe headache • problems with vision, such as blurring or flashing before the eyes • severe pain just below the ribs • vomiting • sudden swelling of the face, hands or feet. Prevention of pre-eclampsia • Aspirin 75–150 mg of aspirin daily from 12 weeks until the birth of the baby is indicated for pregnant with: One of the following high risk factor for pre-eclampsia: hypertensive disease during previous pregnancies chronic kidney disease autoimmune disorders such as SLE or antiphospholipid syndrome type 1 or 2 diabetes mellitus chronic hypertension. More than one of the following moderate risk factor for pre-eclampsia: first pregnancy age 40 years or older

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 pregnancy interval of more than 10 years body mass index (BMI) of 35 kg/m2 or more at first visit family history of pre-eclampsia multi-fetal pregnancy. Treatment • Pre-eclampsia Target blood pressure: BP of 135/85 mmHg or less Best antihypertensive: 1st line : labetalol 2nd line: nifedipine (if labetalol is not suitable) 3rd line: methyldopa (if both labetalol and nifedipine are not suitable) Consider magnesium sulfate treatment, if 1 or more of the following features of severe pre-eclampsia is present: ongoing or recurring severe headaches visual scotomata nausea or vomiting epigastric pain oliguria and severe hypertension progressive deterioration in laboratory blood tests (such as rising creatinine or liver transaminases or falling platelet count). • Eclampsia Magnesium sulphate is used to both prevent seizures in patients with severe preeclampsia and treat seizures once they develop. IV bolus of 4g over 5-15 minutes followed by an infusion of 1g / hour for 24 hours. Recurrent fits should be treated with a further dose of 2–4 g given intravenously over 5 to 15 minutes. urine output, reflexes, respiratory rate and oxygen saturations should be monitored during treatment treatment should continue for 24 hours after last seizure or delivery (around 40% of seizures occur post-partum) • Other important aspects of treating severe pre-eclampsia/eclampsia include fluid restriction to avoid the potentially serious consequences of fluid overload (limit maintenance fluids to 80 ml/hour) • delivery of the baby is the most important and definitive management step. The timing depends on the individual clinical scenario.

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Pulmonary arterial hypertension (PAH) Definition • Sustained elevation in mean pulmonary arterial pressure of greater than 25 mmHg at rest or 30 mmHg after exercise. Epidemiology • More common in females • Typically presents at 20-40 years old WHO Classification & causes

1. Group 1: idiopathic pulmonary arterial hypertension (IPAH) Idiopathic (previously termed primary pulmonary hypertension (PPH) 10% are familial (autosomal dominant) Diagnosed when no underlying cause can be found Endothelin thought to play a key role in pathogenesis
2. Group 2: Pulmonary hypertension with left heart disease Congenital heart disease with systemic to pulmonary shunts Left-sided atrial, ventricular or valvular disease such as left ventricular systolic and diastolic dysfunction, mitral stenosis and mitral regurgitation
3. Group 3: Pulmonary hypertension secondary to lung disease/hypoxia COPD Interstitial lung disease Sleep apnoea High altitude
4. Group 4: Pulmonary hypertension due to thromboembolic disease
5. Group 5: Miscellaneous conditions Lymphangiomatosis e.g. secondary to carcinomatosis or sarcoidosis Collagen vascular disease HIV (the mechanism by which HIV infection produces pulmonary hypertension remains unknown) Sickle cell disease Haemoglobinopathies (eg: sickle cell anemia, thalassemia) intravascular hemolysis → ↓ nitric oxide (NO) → pulmonary vasoconstriction Drugs and toxins: cocaine and anorexigens (e.g. fenfluramine) Increased pressure in pulmonary circuit → elevated right ventricular afterload → dilatation and/or hypertrophy of the right heart →right heart failure

and arrhythmias Lung disease can cause pulmonary hypertension by hypoxic vasoconstriction, whereas the heart can cause pulmonary hypertension by pump failure and subsequent fluid backup and stasis. • Pulmonary arterial hypertension is caused by an intrinsic increase in the resistance of the pulmonary vasculature, while pulmonary hypertension can be caused by secondary aetiologies such as lung disease and heart failure. • The most common cause of pulmonary arterial hypertension is idiopathic, while the most common overall cause of pulmonary hypertension is left-sided heart failure.

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Cardiology Features • Symptoms exertional dyspnoea is the most frequent symptom progressive SOB chest pain and syncope may also occur • On examination: cyanosis Nail clubbing raised JVP with prominent 'a' waves, left parasternal heave (due to right ventricular hypertrophy) loud P2 tricuspid regurgitation Investigation • Doppler echocardiography the initial investigation of choice the jet associated with tricuspid regurgitation can be visualised adequately (tricuspid regurgitant jet velocity) • Right heart catheterization confirmatory test the gold standard for the diagnosis World Health Organization (WHO) functional classification for pulmonary hypertension Class WHO functional classification for pulmonary hypertension I No limitations of physical activity. Ordinary physical activity does not cause undue fatigue or dyspnea, chest pain, or heart syncope. II Slight limitation of physical activity. Ordinary physical activity results in undue fatigue or dyspnea, chest pain, or heart syncope. Comfortable at rest. III Marked limitation of physical activity. Less than ordinary physical activity causes undue fatigue or dyspnea, chest pain, or heart syncope. Comfortable at rest IV Inability to carry on any physical activity without symptoms. Dyspnea and/or fatigue may be present even at rest. Management • Treatment of the underlying cause for example: Anticoagulants for PE Bronchodilators and inhalation corticosteroids for COPD, CPAP for patients with obstructive sleep apnea • Acute vasodilator testing is central to deciding on the appropriate management strategy.

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 Acute vasodilator testing aims to decide which patients show a significant fall in pulmonary arterial pressure following the administration of vasodilators such as intravenous epoprostenol or inhaled nitric oxide If there is a positive response →oral calcium channel blockers If there is a negative response: prostacyclin analogues: treprostinil, iloprost endothelin receptor antagonists: bosentan phosphodiesterase inhibitors: sildenafil • Diuretics if right heart failure • Heart-lung transplant Whilst only 10-15% of patients appear to have a pulmonary vascular tree responsive to calcium antagonism, these agents still constitute the initial therapy of choice according to guidelines, but only in those patients who show a response to vasodilator testing. Complication • Cor pulmonale

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Angina pectoris Non-atherosclerotic angina would be associated with conditions such as • Thyrotoxicosis • Aortic regurgitation • Aortic stenosis • Hypertrophic cardiomyopathy • Anaemia Anginal pain is:

1. constricting discomfort in the front of the chest, or in the neck, shoulders, jaw, or arms
2. precipitated by physical exertion
3. relieved by rest or GTN within about 5 minutes. Three of the features above are defined as typical angina. Two of the three features above are defined as atypical angina. One or none of the features above are defined as non-anginal chest pain. Features which make a diagnosis of stable angina unlikely are when the chest pain is: • continuous or very prolonged and/or • unrelated to activity and/or • brought on by breathing in and/or • associated with symptoms such as dizziness, palpitations, tingling or difficulty swallowing. Consider causes of chest pain other than angina (such as gastrointestinal or musculoskeletal pain).

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Cardiology Investigations for stable chest pain • First-line: 64-slice CT coronary angiography (CTCA) • Second-line: non-invasive functional testing (if CTCA is non-diagnostic.) Myocardial perfusion scan (MPS) with single photon emission computed tomography (SPECT) (MPS with SPECT) or stress echocardiography or first-pass contrast-enhanced magnetic resonance perfusion or MRI for stress-induced wall motion abnormalities. • Third-line: invasive coronary angiography (when the results of non-invasive functional imaging are inconclusive) In the context of risk factors for ischaemic heart disease (hypertension, hypercholesterolaemia, smoking), the clinical diagnosis should be confirmed with non-invasive functional scanning such as myocardial perfusion scanning with SPECT. • High-risk patients with classic angina symptoms should proceed directly to coronary angiography. • Offer 64- slice (or above) CT coronary angiography if:

1. clinical assessment indicates typical or atypical angina or
2. clinical assessment indicates non-anginal chest pain but 12- lead resting ECG has been done and indicates ST- T changes or Q waves. • Low-risk patients can be evaluated with non-invasive stress imaging. • Offer non-invasive functional imaging for myocardial ischaemia if 64-slice (or above) CT coronary angiography has shown CAD of uncertain functional significance or is nondiagnostic. non-invasive functional testing for myocardial ischaemia
3. myocardial perfusion scintigraphy with single photon emission computed tomography (MPS with SPECT) Use adenosine, dipyridamole or dobutamine as stress agents
4. stress echocardiography Use exercise or dobutamine for stress echocardiography or MR imaging for stress-induced wall motion abnormalities.
5. first-pass contrast-enhanced magnetic resonance (MR) perfusion use adenosine or dipyridamole as stress agents
6. MR imaging for stress-induced wall motion abnormalities Take account of locally available technology and any contraindications (for example, disabilities, frailty, limited ability to exercise) when deciding on the imaging method. • Offer invasive coronary angiography as a third-line investigation when the results of noninvasive functional imaging are inconclusive. • Treadmill exercise is no longer recommended in the work-up of new-onset chest pain. Definition of significant coronary artery disease (CAD) • CT coronary angiography is: ≥70% diameter stenosis of at least one major epicardial artery segment or ≥50% diameter stenosis in the left main coronary artery Factors

intensifying ischaemia • Such factors allow less severe lesions (for example, ≥50%) to produce angina: reduced oxygen delivery: anaemia, coronary spasm increased oxygen demand: tachycardia, left ventricular hypertrophy large mass of ischaemic myocardium: proximally located lesions longer lesion length. Factors reducing ischaemia which may render severe lesions (≥70%) asymptomatic:

• Well-developed collateral supply. • Small mass of ischaemic myocardium: distally located lesions, old infarction in the territory of coronary supply. ESC guidelines 2017 • A reduction in chest pain after nitroglycerin (glyceryl trinitrate) administration can be misleading and is not recommended as a diagnostic manoeuvre • In cases of symptom relief after nitroglycerin administration, another 12-lead ECG must be obtained. • A complete normalization of the ST-segment elevation after nitroglycerin administration, along with complete relief of symptoms, is suggestive of coronary spasm, with or without associated MI. In these cases, an early coronary angiography (within 24 h) is recommended. • In cases of recurrent episodes of ST-segment elevation or chest pain, immediate angiography is required. Drug management You should still use bisoprolol in patients with COPD and IHD, because it carries an important outcome benefit Medication • all patients should receive aspirin and a statin in the absence of any contraindication • sublingual glyceryl trinitrate to abort angina attacks • NICE recommend using either a beta-blocker or a calcium channel blocker first-line based on 'comorbidities, contraindications and the person's preference' • if a calcium channel blocker is used as monotherapy a rate-limiting one such as verapamil or diltiazem should be used. If used in combination with a beta-blocker then use a longacting dihydropyridine calcium-channel blocker (e.g. modified-release nifedipine). Remember that beta-blockers should not be prescribed concurrently with verapamil (risk of complete heart block) • if there is a poor response to initial treatment then medication should be increased to the maximum tolerated dose (e.g. for atenolol 100mg od) • if a patient is still symptomatic after monotherapy with a beta-blocker add a calcium channel blocker and vice versa • if a patient is on monotherapy and cannot tolerate the addition of a calcium channel blocker or a beta-blocker then consider one of the following drugs: a long-acting nitrate, ivabradine, nicorandil or ranolazine A beta-blocker or a calcium channel blocker is used first-line to prevent angina attacks

• if a patient is taking both a beta-blocker and a calcium-channel blocker then only add a third drug whilst a patient is awaiting assessment for PCI or CABG • The FREEDOM trial demonstrated that in diabetic patients CABG was superior to PCI in that it significantly reduced rates of death and myocardial infarction. • Cardioselective calcium antagonists such as verapamil and diltiazem do not affect prognosis in angina although they may impact on symptoms by reducing heart rate. • If a patients don’t tolerate beta-blockade, ivabradine may be a more appropriate intervention. Nitrate tolerance • many patients who take nitrates develop tolerance and experience reduced efficacy • the BNF advises that patients who develop tolerance should take the second dose of isosorbide

mononitrate after 8 hours, rather than after 12 hours. This allows blood-nitrate levels to fall for 4 hours and maintains effectiveness • this effect is not seen in patients who take modified release isosorbide mononitrate • the explanation for nitrate tolerance generation of reactive oxygen species chronic nitrate therapy ↑vascular oxidative stress ↑ degradation of nitric oxide (NO) reduced bioavailability Ivabradine • action (If ('funny' ion) channel inhibitor which is highly expressed in the sinoatrial node) reducing the heart rate • Indications a new class of anti-anginal drug there is no evidence currently of superiority over existing treatments of stable angina heart failure: with (NYHA) class II–IV stable chronic heart failure with systolic dysfunction and who are in sinus rhythm with a heart rate of 75 bpm or more and who are given ivabradine in combination with standard therapy including β-blocker therapy, angiotensin-converting enzyme (ACE) inhibitors and aldosterone antagonists, or when β-blocker therapy is contraindicated or not tolerated and with a left ventricular ejection fraction of 35% or less. • adverse effects: visual effects, particular luminous phenomena, are common. sensations of enhanced brightness in a fully maintained visual field due to blockage of Ih ion channels in the retina, which are very similar to cardiac If. mild, transient, and fully reversible. Bradycardia, due to the mechanism of action, Ulceration of an atheromatous plaque of the abdominal aorta is the most common source of emboli in old man presented with acute pain, pallor and absent pulses in his leg. MRCPUK-part-1-January 2018 exam: Which cell type is most implicated in the development of coronary artery plaques? Macrophages

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Coronary artery bypass graft (CABG) • There are two main approaches.

1. In one, the left internal thoracic artery (internal mammary artery) is diverted to the left anterior descending branch of the left coronary artery.
2. In the other, a great saphenous vein is removed from a leg; one end is attached to the aorta or one of its major branches, and the other end is attached to the obstructed artery. • CABG is superior to PCI in multivessel coronary disease. • indicated when coronary arteries have a 50% to 99% obstruction. • CABG guidelines state CABG is the preferred treatment for: Disease of the left main coronary artery (LMCA). Disease of all three coronary arteries (LAD, LCX and RCA). Diffuse disease not amenable to treatment with a PCI. high-risk patients such as those with severe ventricular dysfunction (i.e. low ejection fraction), or diabetes mellitus. • Benefits relief of angina no survival benefit with bypass surgery vs. medical therapy in stable angina Bypass surgery does not prevent future myocardial infarctions. • Complications The incidence of acute coronary syndrome within 30 days of CABG is high, at around 17.5%. Aneurysms are a rare and late complication of CABG.

Cardiac syndrome X • consist of: angina-like chest pain during exertion characteristic ECG changes during exercise testing normal coronary arteries on cardiac catheterisation no inducible coronary artery spasm during catheterisation

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Acute coronary syndrome Poor prognostic factors • age • development (or history) of heart failure • peripheral vascular disease • reduced systolic blood pressure • Killip class* • initial serum creatinine concentration • elevated initial cardiac markers • cardiac arrest on admission • ST segment deviation Clinical factors which are good indicators of ACS: • typical pain lasting at least 15 minutes, associated nausea, and sweating. • Response to GTN should not be used as indicator of ACS

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Cardiology

ACS referral Chest pain Referral guidelines: • current chest pain or chest pain in the last 12 hours with an abnormal ECG: emergency admission • chest pain 12-72 hours ago: refer to hospital the same-day for assessment • chest pain > 72 hours ago: perform full assessment with ECG and troponin measurement before deciding upon further action

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Myocardial infarction • The most specific feature, which suggests that the pain is myocardial ischaemia, is the radiation to the jaw, which is relatively specific for pain of myocardial ischaemia. • The clinical classification of MI includes: (NICE 2010) Type 1: ischaemia due to a primary coronary event such as plaque, fissuring or dissection. Type 2: ischaemia due to either increased oxygen demand or decreased supply, such as coronary spasm, coronary embolism, anaemia, arrhythmias, hypertension, or hypotension. The diurnal variation of myocardial ischaemia • There is a diurnal variation in presentation of myocardial ischaemia. • Which physiological process is responsible for this? Vasospasm • The peak incidence of STEMI and the peak incidence of death due to ischaemic heart disease both coincide at around 8-9 am. The early morning is associated with several physiological and haematological factors which predispose to vasospasm, infarction and death. There is ↑adrenergic activity ↑plasma fibrinogen levels ↑inhibition of fibrinolysis and ↑platelet adhesiveness. • Interestingly, NSTEMIs are not associated with this degree of diurnal rhythm. • Precipitating factors for an infarct include: physical exertion Rest , Sleep Surgical procedure Emotional stressors.