# 033 - Chapter 5

# 033

# Chapter 5

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

Cardiology

 reduced venous return, and  reduced peripheral resistance • Sinoatrial node  has the fastest firing rate of all potential pacemakers in the heart.  Sinoatrial node impulses must occur at a rate slower than 200 impulses per minute to be considered in normal sinus rhythm. • Endothelin  preferentially constricts renal afferent arterioles.  Efferent arteriole vasoconstriction is mediated by angiotensin-II, to defend GFR in states of generalised vasoconstriction and reduced blood flow.  efferent arteriole vasodilation will occur when angiotensin-II levels fall.  Stimulates the renin-angiotensin-aldosterone system  Leads to release of atrial natriuretic peptide  Inhibits the action of vasopressin  Two types of endothelin receptor have been characterised, A and B.  Binding of endothelin to the A receptor induces vasoconstriction,  binding to the B receptor leads to nitric oxide release and hence vasodilatation. Coronary circulation physiology • The three most potent factors for vasodilation of the coronaries are:

1. Increased adenosine
2. Increased nitric oxide
3. Opening of ATP-sensitive potassium (KATP) channels by low ATP concentrations, which hyperpolarizes the vascular smooth muscle

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Physiological changes during pregnancy • Heart rate: increases by 10-20 bpm • Cardiac output and blood volume increase from the second month up to the thirtieth week to 30 - 50% above the normal levels. • The increase in cardiac output is mediated via increase in both stroke volume and to a lesser extent heart rate, along with a dramatic fall in total peripheral vascular resistance. • Venous pressure: remain the same due to a 25% reduction in systemic and pulmonary vascular resistance. • Blood pressure: drop in the first and second trimester due to vasodilatation and then climb to pre-pregnancy levels by the third trimester. • The increase in blood volume and increased cardiac output lead to all stenosic murmurs becoming more prominent (there is increased flow across the valve, with more turbulence and pressure gradient, leading to a louder sound). • Increased metabolic workload • Apex beat is displaced, because of cardiomegaly and a raised diaphragm • The increased blood flow may produce a pulmonary systolic murmur and a third heart sound. Which murmur is diminished during pregnancy?  Aortic regurgitation  The fall in diastolic blood pressure during pregnancy leads to a reduction in the murmur of aortic regurgitation.

Physiological changes during exercise Increases during exercise • cardiac output → Systemic arterial pressure • ↑venous return → ↑stroke volume • ↑heart rate • Dilatation of the blood vessels within the exercising muscles causes a fall in total peripheral resistance, resulting in a decrease in diastolic blood pressure. • Decrease in venous compliance (dilatation), caused by sympathetic stimulation, helps to maintain ventricular filling during diastole. • The pulmonary vessels undergo passive dilatation as more blood flows into the pulmonary circulation, decreasing pulmonary vascular resistance.

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Physiological changes associated with age • Decrease elasticity and compliance of the aorta →increased resistance to ejection of blood from the left ventricle →increased ventricular afterload. • Diastolic dysfunction and reduced stroke volume • ↓↓diastolic pressure (the pressure responsible for subendocardial perfusion)  subendocardial ischemia and interstitial fibrosis. (These changes are related to an increase in the magnitude of the L-type Ca++) • Higher systolic arterial pressure and increased impedance to left ventricular ejection • ↑ systolic + ↓ diastolic  ↑ pulse pressure • Increased sino-atrial conduction time  Because of the delayed LV relaxation and the stiffer left ventricle, the force of left atrial contraction increases and the contribution of the atrial contraction to LV enddiastolic volume increases • There is apoptosis of atrial pacemaker cells with a loss of 50%-75% of cells by age 50. The number of atrioventricular nodal cells is preserved and there is fibrosis and cellular loss in the His bundle • Left ventricular hypertrophy • Which physiological change associated with age during exercise?  Reduced tachycardic response

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Valsalva manoeuvre Definition • The Valsalva manoeuvre describes a forced expiration against a closed glottis. This leads to increased intrathoracic pressure which in turn has a number of effects on the cardiovascular system. Uses • to terminate an episode of supraventricular tachycardia • normalizing middle-ear pressures Notes & Notes for MRCP
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Decreases during exercise • Venous compliance • Peripheral vascular resistance • Diastolic pressure • Pulmonary vascular resistance

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Cardiology Cardiac action potential Cardiac action potential Phase Description Mechanism

Rapid depolarisation Rapid sodium influx These channels automatically deactivate after a few ms

Early repolarisation Efflux of potassium

Plateau Slow influx of calcium

Final repolarisation Efflux of potassium

Restoration of ionic concentrations Resting potential is restored by Na+/K+ ATPase There is slow entry of Na+ into the cell decreasing the potential difference until the threshold potential is reached, triggering a new action potential NB cardiac muscle remains contracted 10-15 times longer than skeletal muscle Conduction velocity Site Speed Atrial conduction Spreads along ordinary atrial myocardial fibres at 1 m/sec AV node conduction 0.05 m/sec Ventricular conduction Purkinje fibres are of large diameter and achieve velocities of 2-4 m/sec (this allows a rapid and coordinated contraction of the ventricles

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Pulses Patent ductus arteriosus - large volume, bounding, collapsing pulse Pulsus alternans - seen in left ventricular failure Pulse Causes Pulsus paradoxus (>10 mmHg fall in systolic BP on inspiration) Pulsus alternans (regular alternation of the force of the arterial pulse between strong and weak) Bisferiens pulse ('double pulse' - two systolic peaks) Collapsing aortic regurgitation, patent ductus arteriosus, hyperkinetic (anaemia, thyrotoxic, fever, exercise/pregnancy). Slow-rising/plateau aortic stenosis Jerky pulse hypertrophic obstructive cardiomyopathy Pulsus paradoxus • Definition  a greater than 10 mmHg fall in systolic BP on inspiration  → faint or absent pulse in inspiration • Mechanism  Inhalation  ↑venous return  expands right ventricle (RV)  compresses left ventricle (LV)  ↓blood pressure.  Inhale = Big RV = Smaller LV = BP drop > 10 mm Hg • Causes  cardiac tamponade (common)  constrictive pericarditis (less commonly than tamponade)  asthma,  obstructive sleep apnoea  croup. Notes & Notes for MRCP
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cardiac tamponade (common) severe LVF mixed aortic valve disease

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Heart sounds First heart sound (S1) : Closure of the mitral and tricuspid valves Changes in first heart sound (S1) Causes Loud S1 • mitral stenosis • left to right shunts • short PR interval (e.g. WPW type B), ( shortened diastole) atrial premature beats • hyperdynamic states Quiet (soft) S1 • mitral regurgitation • immobile mitral stenosis • if closure of the mitral valve is delayed e.g.:  LBBB,  long PR • hypodynamic state Split S1 • right bundle branch block, • left bundle branch block, • ventricular tachycardia, • Ebstein’s anomaly Variable intensity • Atrial fibrillation

Third heart sound (S3) Sound Origin Causes Notes Third heart sound caused by rapid ventricular filling during diastole. (S3) caused by atrial contraction against a stiff ventricle Fourth heart sound (S4) Gallops that originate from the left side of the heart (the most common) become softer with inspiration, while those that originate from the right side become louder. Heart sound and ECG correlation Notes & Notes for MRCP
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• Physiological:  young individuals • Early diastolic sound that is heard immediately after S2 • Ventricular gallop: S1 is followed by S2 and S3 . (< 40 years of age), athletes, or pregnant women • Pathological  Chronic mitral regurgitation  Aortic regurgitation  Heart failure  Dilated

cardiomyopathy • Thyrotoxicosis • Physiological: advanced age • Pathological if palpable  Ventricular hypertrophy (e.g., hypertension, aortic stenosis, cor pulmonale)  Ischemic cardiomyopathy • Acute myocardial infarction • Late diastolic (presystolic) sound heard immediately before S1 • P wave on ECG



Second heart sound (S2) Second heart sound (S2): Closure of the aortic valve (A2) (louder) and pulmonary valve (P2) (softer). Changes in S2 Causes Loud A2 arterial hypertension, coarctation of the aorta Loud P2 pulmonary hypertension Physiological split (A2 precedes P2). during inspiration → ↓intrathoracic pressure → ↑venous return to the right side of the heart → prolonged right ventricular systole → delayed closure of P2. Especially pronounced among young individuals Wide split Mechanism • Increased right ventricular afterload → prolonged right ventricular systole • Decreased left ventricular preload → shortened left ventricular systole Causes • Pulmonary hypertension • Pulmonary valve stenosis • RBBB • Massive pulmonary embolism • Severe mitral regurgitation • Wolff-Parkinson-White syndrome • Constrictive pericarditis Fixed split (Does not change with respiration and tends to be wide, i.e., the split is also audible during expiration) • Atrial septal defect (ASD)→ RV volume overload → delay in the closure of the pulmonary valve • Severe RV failure • Right bundle-branch block with heart failure (right bundle-branch block widens the split, and heart failure makes the split fixed). reversed (paradoxical) split S2 (P2 occurs before A2) • Due to delayed A2  left bundle-branch block (LBBB)  aortic stenosis (the aortic leaflets are thickened and so close slowly)  hypertrophic obstructive cardiomyopathy • Due to early P2  Early excitation of the right ventricle (e.g., RV pacing, Wolff-Parkinson-White syndrome type B) where the right-sided accessory pathway causes early RV depolarisation. Absent split (No splitting of S2) • Severe aortic stenosis (geriatric) • VSD with Eisenmenger syndrome (paediatric)



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Murmurs Most murmurs of stenosis or regurgitation are exaggerated during squatting and get softer with the Valsalva manoeuvre. The exceptions are HOCM where the opposite occurs (↑by Valsalva & ↓ by squatting) and mitral valve prolapse where the murmur gets longer. Relation between murmurs intensity and respiration: • Murmurs that increase in intensity with inspiration originate from the right side of the heart (tricuspid or pulmonary) • Murmurs that increase in intensity with expiration originate from the left side of the heart (mitral or aortic). Mnemonic: RILE (Right Inspiration, Left Expiration) Murmur Causes Ejection systolic • Aortic stenosis, HOCM • Pulmonary stenosis • ASD • Fallot's Holosystolic (pansystolic) • mitral/tricuspid regurgitation (high-pitched and 'blowing' in character) • VSD ('harsh' in character) Late systolic • Mitral valve prolapse • Coarctation of aorta Early diastolic • Aortic regurgitation (high-pitched and 'blowing' in character) • Graham-Steel murmur (pulmonary regurgitation, again high-pitched and 'blowing' in character) Mid-late diastolic • Mitral stenosis • Austin-Flint murmur (severe aortic regurgitation, indistinguishable from that of mitral stenosis). It is due to partial closure of the anterior leaflet of

the mitral valve by the regurgitant jet. Continuous machinelike murmur • patent ductus arteriosus



Murmurs in pregnancy • The intensity of Aortic regurgitation murmur diminishes during pregnancy. • Diastolic blood pressure is lower due to vasodilatation, and this is responsible for the fading of the aortic regurgitation murmur • All stenotic murmurs become more prominent Mitral murmurs are heard best during expiration and while the patients lies on the left side. All right-sided heart murmurs are intensified during deep inspiration. Isometric handgrip exercises increase blood pressure and afterload significantly. Therefore, murmurs caused by the backward flow of blood will be accentuated: • aortic regurgitation, • mitral valve regurgitation, • mitral valve prolapse and • ventricular septal defect.

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Syncope Definition • Syncope is a transient loss of consciousness due to transient global cerebral hypoperfusion, characterised by rapid onset, short duration, and spontaneous complete recovery. Cases • Syncope can be classified as  non-cardiovascular causes:  neurally-mediated (reflex syncope)  vasovagal  situational syncope: provoked by straining during micturition (usually while standing) or by coughing or swallowing.  secondary to orthostatic hypotension  cardiovascular causes (such as arrhythmias or ischaemia) • In older patients, non-cardiovascular causes are twice as common as cardiovascular causes Evaluation • The initial evaluation after T-LOC consists of:  a careful history  orthostatic BP measurements  ECG  ECG is the most useful test for classifying syncopal episodes into high risk and low risk categories:  High risk :history of heart disease or abnormal ECG.  Low risk : no underlying diseases and a normal ECG.



• The initial evaluation can define the cause of syncope in 23-50% of patients and should answer three key questions:  Is it a true syncopal episode or not?  Has the aetiological diagnosis been determined?  Are there findings suggestive of a high risk of cardiovascular events or death? • What you were doing during the episode of blackout?  during exercise : exercise-induced syncope occurred (cardiac arrhythmic cause is probable)  offer urgent (within 7 days) exercise testing, unless there is a possible contraindication (such as suspected aortic stenosis or hypertrophic cardiomyopathy requiring initial assessment by imaging).  Advise the person to refrain from exercise until informed otherwise following further assessment.  offer an ambulatory ECG and do not offer a tilt test as a first-line investigation.  TLoC at least several times a week,  offer Holter monitoring (up to 48 hours)  If no further TLoC occurs during the monitoring period,  offer external event recorder that provides continuous recording with the facility for the patient to indicate when a symptomatic event has occurred.  TLoC every 1–2 weeks  offer an external event recorder.  If the person experiences further TLoC outside the period of external event recording,  offer an implantable event recorder.  TLoC infrequently (less than once every 2 weeks)  offer an implantable event recorder. A Holter monitor should not usually be offered unless there is evidence of a conduction abnormality on the 12lead ECG.  shortly after stopping exercise

(vasovagal cause is more likely). • Unexplained syncope →offer ambulatory ECG. Do not offer a tilt test before the ambulatory ECG. • For people with suspected carotid sinus syncope and for people with unexplained syncope who are aged 60 years or older,  offer carotid sinus massage as a first-line investigation.  This should be conducted in a controlled environment, with ECG recording, and with resuscitation equipment available.  Diagnose carotid sinus syncope if carotid sinus massage reproduces syncope due to marked bradycardia/asystole and/or marked hypotension.  Do not diagnose carotid sinus syncope if carotid sinus massage causes asymptomatic transient bradycardia or hypotension • Tilt test  Do not offer a tilt test to people who have a diagnosis of vasovagal syncope on initial assessment.  For people with suspected vasovagal syncope with recurrent episodes of TLoC adversely affecting their quality of life, or representing a high risk of injury,  consider a tilt test only to assess whether the syncope is accompanied by a severe cardioinhibitory response (usually asystole). • If a person has persistent TLoC, consider psychogenic non-epileptic seizures (PNES) or psychogenic pseudosyncope if:  the nature of the events changes over time  there are multiple unexplained physical symptoms  there are unusually prolonged events. • Driving  must not drive while waiting for a specialist assessment.  Following specialist assessment  report the TLoC event to (DVLA)

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Cardiology Implantable loop recorder (ILR) • subcutaneous, single-lead, (ECG) monitoring device • used for diagnosis in patients with recurrent unexplained episodes of palpitations or syncope, • The device is typically implanted in the left parasternal region and is capable of storing ECG data automatically in response to a significant bradyarrhythmia or tachyarrhythmia or in response to patient activation. • It is particularly useful either when symptoms are infrequent (and thus not amenable to diagnosis using short-term external ECG recording techniques) or when aggregate longterm data (eg, burden of AF) are required.

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Vasovagal syncope (VVS) • Vasovagal syncope (VVS) is the most common type of syncope. Causes • features suggestive of uncomplicated vasovagal syncope (the 3 'P's):  Posture – prolonged standing, or similar episodes that have been prevented by lying down  Provoking factors (such as pain or a medical procedure)  common during dental procedures, mainly induced by pain (as the dentist started drilling).  Prodromal symptoms (such as sweating or feeling warm/hot before TLoC). Feature • VVS is usually preceded by a prodrome of symptoms such as dizziness, nausea, and diaphoresis.  The syncope lasts briefly, but nausea, warmth and sweating may persist for some time. • Twitching and jerking are often seen with vasovagal or cardiac syncope, which can be differentiated from rhythmic jerking of all the limbs in tonic-clonic seizures. • It is common to have jerking of limbs due to brain hypoxia. • Incontinence of urine can occur, but not biting of the tongue. Diagnosis • Recover very quickly supports the diagnosis of syncope. • ECG is always normal. • Tilt table test is a useful test to support the diagnosis  If structural heart disease is excluded and syncope is reproduced on tilt table testing along with fall in blood pressure and heart rate, then this is diagnostic of vasovagal syncope. Treatment • Midodrine may be indicated in patient with VVS refractory to life style management  Midodrine is a prodrug of Desglymidodrine  a sympathomimetic (alpha receptor agonist) that acts on the blood vessels to raise blood pressure.

Postural hypotension • Causes: mnemonic (HANDI)  H = Hypovolemia, Hypopituitarism (dehydration, bleeding )  A = Addison's disease  N = Neuropathy (autonomic due to diabetics, amyloidosis )  D = Drugs ( Vasodialators, TCA, antipsychotic, Diuretics etc. )  I = Idiopathic orthostatic hypotension • Management of postural hypotension  if the standing BP is clearly acceptable (110 systolic) , the most obvious first step is stopping the causative drug (eg: indapamide) and monitoring his blood pressure over the subsequent 2-4 weeks.  If he still has significant postural hypotension then the next steps would be to add elastic stockings, then fludrocortisone.  The history of pre-syncope is much more suggestive of changes in blood pressure rather than changes in blood glucose.

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Vertigo & Dizziness Clinical features of central versus peripheral vertigo Peripheral Central Nystagmus Direction Unidirectional, fast component toward the normal ear; never reverses direction Nystagmus Type Horizontal with a torsional component, never purely torsional or vertical Nystagmus Effect of visual fixation Suppressed Not suppressed Other neurologic signs Absent Often present Postural instability Unidirectional instability, walking preserved Severe instability, patient often Deafness or tinnitus May be present Absent Dix-Hallpike maneuver for positional nystagmus: Findings in central versus peripheral vertigo Peripheral disorder Central disorder Latent period before onset of positional nystagmus 2 to 20 seconds None Duration of nystagmus Less than 1 minute Greater than 1 minute Fatigability Fatiguing with repetition Non-fatiguing Direction of nystagmus Only one type, may change direction with gaze Intensity of vertigo Severe Less severe, sometimes none Notes & Notes for MRCP
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Sometimes reverses direction when patient looks in the direction of slow component Can be any direction falls when walking May change direction with a given head position

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Cardiology Algorithm for the initial evaluation of a patient with dizziness.

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The HINTS exam: (Head Impulse, Nystagmus, Test for Skew) • A three step physical exam testing oculomotor function (The HINTS exam) was able to differentiate between peripheral causes of vertigo and stroke with a sensitivity of 100%, and a specificity of 96%. • Remember, the patient needs to be currently experiencing vertigo in order to perform the HINTS exam. Head Impulse Test (HI) • Method:  Patient looks at your nose  Hold skull (not jaw) firmly  Slow movement to relax neck muscles  Quick movement about 20 degree from lateral to midline  Activate your biceps and forearm, not just wrists  Random side tested • Interpretation:  In peripheral vertigo where the vestibulo-ocular reflex (VOR) reflex is impaired, rapid head rotation toward the affected side will cause the patients eyes to slowly move away from the target and force a corrective saccade (fast) back to the target.  In central vertigo the VOR reflex remains intact. Nystagmus (N) • In peripheral vertigo:  unidirectional horizontal nystagmus with the fast phase beating away from the affected side. • In central vertigo:  vertical or rotational nystagmus, or bidirectional horizontal

nystagmus where the fast phase changes directions. Test for skew (TS) • Method  alternating covering the patients eyes while the patient fixes their gaze on a fixed target.
• Interpretation  In central vertigo:  the patients sometimes have vertical misalignment of their eyes due to impaired gravity sensing. As the cover moves back and forth between the two eyes, the uncovered eye will correct its gaze to refocus on the target. This correction should be observed repeatedly as the cover moves back and forth.  In peripheral vertigo:  no skew deviation. In summary: The HINTS exam: • Peripheral = Positive head impulse test, unidirectional nystagmus, no skew • Central = Negative head impulse test, bidirectional, vertical or rotational nystagmus,

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Sudden cardiac death • In those aged greater than 35 years:  The most common cause of sudden cardiac death is ischemic heart disease.  Up to 80% of individuals who suffer sudden cardiac death have coronary heart disease. • In those under the age of 35 years of age:  HOCM is the most common cause of sudden cardiac death, coronary artery disease being the second most common cause.  In competitive athletes <35 years of age HOCM is by far the most common cause of sudden cardiac death (prevalence is 1 in 500). • Arrhythmogenic right ventricular dysplasia (ARVD)  the second most common cause of sudden cardiac death in the young after HOCM. • Catecholaminergic polymorphic ventricular tachycardia (CPVT)  an autosomal dominant inherited cardiac disease  prevalence of around 1:10,000. • Brugada syndrome  an autosomal dominant inherited cardiovascular disease.  prevalence of 1:5,000-10,000.  more common in Asians.

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Exercise tolerance tests Indications: Exercise tolerance tests (ETT, also exercise ECG) are used for a variety of indications: • assessing patients with suspected angina - however the 2010 NICE Chest pain of recent onset guidelines do not support the use of ETTs for all patients • risk stratifying patients following a myocardial infarction  the best predictor of mortality post-STEMI  exercise capacity  Above average exercise capacity  good prognosis after a STEMI • assessing exercise tolerance • risk stratifying patients with hypertrophic cardiomyopathy Sensitivity and specificity of ETT: ( high number of false positives and false negatives) • ETT has a sensitivity of around 80% and a specificity of 70% for ischaemic heart disease. Thus, a negative test may not necessarily be true and further testing may be adviced.  Exercise ECG testing has a relatively high sensitivity but only moderate specificity for the diagnosis of CAD. • Diagnostic accuracy is poor in women and this may relate to smaller heart size. Heart rate: • maximum predicted heart rate = 220 - patient's age • the target heart rate is at least 85% of maximum predicted to allow reasonable interpretation of a test as low-risk or negative Contraindications • myocardial infarction less than 7 days ago • unstable angina • uncontrolled hypertension (systolic BP > 180 mmHg) or hypotension (systolic BP < 90 mmHg) • Any condition where left ventricular output is reduced - eg, aortic stenosis or hypertrophic obstructive cardiomyopathy (HOCM).

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• Abnormal baseline ECG (eg, bundle branch block patterns or left ventricular hypertrophy); these make interpretation of the ETT difficult. Stop if: • exhaustion / patient request • 'severe', 'limiting' chest pain •

> 3mm ST depression • 2mm ST elevation.Stop if rapid ST elevation and pain • systolic blood pressure > 230 mmHg • systolic blood pressure falling > 20 mmHg • attainment of maximum predicted heart rate • heart rate falling > 20% of starting rate • arrhythmia develops Interpreting the exercise tolerance test • The patient is normally considered to have been adequately 'stressed' if they achieve 85% or more of their maximum heart rate (calculated as 220 - age in years for men and 210 - age for women). • If ECG criteria for inducible ischaemia (chest pain is not mandatory).The next step is  Coronary angiography  this will define the coronary anatomy and give a better guide to prognosis. • If an inadequate test was performed, further non-invasive investigations may be indicated, such as myocardial perfusion scanning, cardiac MRI, or stress echocardiogram. Notes • Beta-blockers and digoxin can interfere with the results so are usually stopped before the ETT.  If ETT performed on beta blocker and there is an adequate rise in heart rate (85% of (220 − age))  so there is no indication for stopping beta blocker and repeat the test

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Cardiac enzymes and protein markers Myoglobin rises first following a myocardial infarction Key points for the exam • myoglobin is the first to rise • CK-MB is useful to look for reinfarction as it returns to normal after 2-3 days (troponin T remains elevated for up to 10 days) Begins to rise Peak value Returns to normal Myoglobin 1-2 hours 6-8 hours 1-2 days CK-MB 2-6 hours 16-20 hours 2-3 days CK 4-8 hours 16-24 hours 3-4 days Trop T 4-6 hours 12-24 hours 7-10 days AST 12-24 hours 36-48 hours 3-4 days LDH 24-48 hours 72 hours 8-10 days

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Cardiology Troponin Troponin C: Binds to calcium to activate actin: myosin interaction Troponin T: Binds to tropomyosin Troponin I: Blocks or inhibits actin: myosin interaction • Troponin is a component of thin filaments • Cardiac-specific marker with high sensitivity for myocardial necrosis • The degree of elevation correlates with the size of the infarct and risk of mortality. Levels act as a prognostic factor following an acute coronary syndrome • Other causes of an elevated troponin are:  Pulmonary embolism, Pulmonary hypertension  Hypertension, Hypotension, especially with arrhythmias  Hypertrophic obstructive cardiomyopathy, Myocarditis including Kawasaki's disease  Sepsis, Burns, Trauma, Cardioversion, Rhabdomyolysis  Subarachnoid haemorrhage and stroke  Infiltrative/autoimmune disorders including sarcoidosis, amyloidosis, haemochromatosis and scleroderma.  Drugs including: Adriamycin, Herceptin and 5-fluorouracil. CK-MB • No longer commonly used clinically; has been replaced by cardiac troponin in the diagnosis of ACS • CK-MB is more specific to cardiac tissue than total CK (but may also be due to skeletal muscle injury). • Can be helpful for evaluating reinfarction because of its short half-life but is no longer commonly used •

The degree of elevation often correlates with the size of the infarct. Serum creatine kinase • Causes of high CK  Myocardial infarction  Racial variant : serum CK activity in Afro-Caribbean people is often up to three times the upper limit of normal for white populations  Hypothyroidism  Heavy exercise  Statins Glycogen phosphorylase isoenzyme BB (GPBB) • GPBB exists in heart and brain tissue. • Rise significantly by three hours post mi. As such it is an appropriate marker for early cardiac muscle injury. • Rise earlier than myoglobin  GPBB levels increase 1–3 h after the event.  Myoglobin levels increase significantly 2 h after ischaemia.

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ECG: axis deviation Normal axis • between -30 and 90º (directed inferior and to the left) Left axis deviation (LAD) • Definition  An axis between -30º and -90º (directed superior and to the left) • Prevalence  LAD (≥- 30 degrees) is the most common "abnormality" in adults occurring in over 8%. • Causes of LAD:  left ventricular hypertrophy  left bundle branch block  left anterior hemiblock  Marked LAD (≥- 45 degrees) is called left anterior hemiblock or left anterior fascicular block  Wolff-Parkinson-White syndrome* - right-sided accessory pathway  *in the majority of cases, or in a question without qualification, WolffParkinson-White syndrome is associated with left axis deviation  congenital: ostium primum ASD, tricuspid atresia, endocardial cushion defect  Inferior wall myocardial infarction  hyperkalaemia  Normal variation (physiologic, often with age), minor LAD in obese people  Mechanical shifts, such as expiration, high diaphragm (pregnancy, ascites, abdominal tumor)  Emphysema  Ventricular ectopic rhythms • Recommendations: (If LAD is present):  Exclude hypertension. (If borderline  ambulatory BP monitoring).  check for borderline indicators of LVH (i.e., the voltage criteria and left atrial enlargement).  Note whether diagnostic inferior Q waves are present since an inferior MI can cause LAD. Right axis deviation (RAD) • Definition  An axis between 90º and 180º (directed inferior and to the right), • Causes of RAD:  right ventricular hypertrophy  right bundle branch block  left posterior hemiblock  Wolff-Parkinson-White syndrome - left-sided accessory pathway  ostium secundum ASD  chronic lung disease → cor pulmonale  pulmonary embolism  Dextrocardia  Ventricular ectopic rhythms  Lateral wall myocardial infarction  Normal variation (vertical heart with an axis of 90º ).  normal in youngsters (less than 21 years of age), tall people, thin adults and athletes

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Cardiology • If the QRS complex is positive (upright) in leads I and II, it falls between -30 and 90° and is normal, as indicated by the yellow area. • If the QRS complex is negative in I and positive in aVF, there is right axis deviation. • If the QRS complex is positive in I and negative in II, there is left axis deviation. • If the QRS complex is negative in I and aVF, there is extreme axis deviation.

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ECG: coronary territories The table below shows the correlation between ECG changes and coronary territories: Localization of myocardial infarct on ECG ECG changes Coronary artery

Anteroseptal V1-V4 Left anterior descending (LAD) Inferior II, III, aVF Right coronary Anterolateral V4-6, I, aVL Left anterior descending (LAD) or left circumflex Lateral I, aVL +/- V5-6 Left circumflex Posterior Tall R waves V1-2 Usually left circumflex, also right coronary

High lateral wall MI • ST segment elevation in leads I and aVL  High lateral wall MI • usually due to occlusion of the first diagonal branch of the left anterior descending artery, though occlusion of other arteries like branches of the left circumflex or a short left anterior descending artery may cause the same picture. Postero-lateral MI  prominent R wave in lead V1 and ST depression in V1-V3 + ST elevation in leads V5 and V6. Posterior MI (ESC guidelines 2017) • posterior wall (now termed inferobasilar), usually supplied by the posterior descending artery — a branch of the right coronary artery in 80% of individuals. • isolated ST-segment depression ≥ 0.5 mm in leads V1–V3 represents the dominant finding. These should be managed as a STEMI. • The use of additional posterior chest wall leads [elevation V7–V9 ≥ 0.5 mm (≥1 mm in men, 40 years old)] is recommended. Left main stem (LMS) • LMS occlusion typically presents dramatically with cardiogenic shock. • ECG findings include ST elevation in aVR with diffuse ST depression in other leads. • The presence of ST depression ≥ 1 mm in six or more surface leads, coupled with STsegment elevation in aVR and/or V1, suggests multivessel ischemia or left main coronary artery obstruction, particularly if the patient presents with haemodynamic compromise. (ESC guidelines 2017) Which ECG changes may be seen earlier in ischaemia ?  hyper-acute T-waves, which may precede ST-segment elevation. ECG criteria for STEMI (ESC guidelines 2017)

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• ST-segment elevation (measured at the J-point) is considered suggestive of ongoing coronary artery acute occlusion in the following cases:  Numbers of leads: at least two contiguous leads with ST-segment elevation  ST-segment elevation:  ≥ 2.5 mm in men < 40 years,  ≥2 mm in men ≥40 years, or  ≥ 1.5 mm in women in leads V2–V3 and/or  ≥1 mm in the other leads  In patients with inferior MI, it is recommended to record right precordial leads (V3R and V4R) seeking ST-segment elevation, to identify concomitant right ventricular (RV) infarction.  Likewise, ST-segment depression in leads V1–V3 suggests myocardial ischaemia, especially when the terminal T-wave is positive (ST-segment elevation equivalent), and confirmation by concomitant ST-segment elevation ≥ 0.5 mm recorded in leads V7–V9 should be considered as a means to identify posterior MI (circumflex occlusion ).

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ECG: digoxin ECG features • down-sloping ST depression ('reverse tick') • flattened/inverted T waves • short QT interval • arrhythmias e.g. AV block, bradycardia

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ECG: hypothermia The following ECG changes may be seen in hypothermia • bradycardia • 'J' wave - small hump at the end of the QRS complex • first degree heart block • long QT interval • atrial and ventricular arrhythmias

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ECG: left bundle branch block • The diagram below shows the typical features of left bundle branch block (LBBB): • The ECG would show:  broad QRS complex (>120ms),  tall R waves in the lateral leads (I, V5-6) and deep S waves in the right precordial leads (V1-3)  usually leads to left axis deviation. • One of the most common ways to remember the difference between LBBB and RBBB is WiLLiaM MaRRoW  WiLLiaM : in LBBB there is a 'W' in V1 and a 'M' in V6  MaRRoW: in RBBB there is a 'M' in V1 and a 'W' in V6

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ECG showing typical features of LBBB • Causes of LBBB  ischaemic heart disease  hypertension  aortic stenosis  cardiomyopathy  rare: idiopathic fibrosis, digoxin toxicity, hyperkalaemia

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Right bundle branch block (RBBB) • Patients with MI and right bundle branch block (RBBB) have a poor prognosis. (ESC guidelines 2017)  It may be difficult to detect transmural ischaemia in patients with chest pain and RBBB.  Therefore, persistent ischaemic symptoms occur in the presence of RBBB  primary PCI strategy (emergent coronary angiography and PCI if indicated) should be considered

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Trifascicular block The evidence of trifascicular block (RBBB, LAD and prolongation of the PR interval) in the context of dizziness and collapses. This is an indication for dual chamber (DDDR) pacing for likely complete heart block. • Trifascicular block is not strictly an ECG diagnosis but is a term used for the combination of:

1. right bundle branch block,
2. left hemiblock (typically left anterior hemiblock (LAHB)) (LAHB is diagnosed because the net QRS deflection in lead II is negative).
3. long PR interval. • the site of the lesion  AV node and Purkinje fibres

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• The most common pattern referred to as “trifascicular block” is the combination of bifascicular block with 1st degree AV block. • It implies that the bundle branches (Purkinje fibres) are blocked in the right bundle and one of the left hemibundles. • The 'third' bundle is also delayed or partially blocked hence the name. However, the delay (long PR interval) is usually at the AV node. • Clinically it means there is extensive disease of the conduction system and, in a patient such as this, would be an indication for permanent pacemaker.

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ECG: normal variants The following ECG changes are considered normal variants in an athlete: • sinus bradycardia • junctional rhythm • first degree heart block • Wenckebach phenomenon

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ECG: PR interval Causes of a prolonged PR interval • idiopathic • ischaemic heart disease • digoxin toxicity • hypokalaemia: hyperkalaemia can rarely cause a prolonged PR interval, but this is a much less common association than hypokalaemia • rheumatic fever • aortic root pathology e.g. abscess secondary to endocarditis • Lyme disease • sarcoidosis • myotonic dystrophy • A prolonged PR interval may also be seen in athletes short PR interval is seen in Wolff-Parkinson-White syndrome

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ECG: ST depression Causes of ST depression • secondary to abnormal QRS (LVH, LBBB, RBBB) • ischaemia • digoxin • hypokalaemia • syndrome X

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T wave • The T wave should be analyzed for:

1. orientation: upgoing, downgoing (inverted) or biphasic
2. concordance with QRS  Concordant: (normal) both QRS and T wave are on the same direction (upgoing or downgoing) (downgoing is common in aVR for normal ECG's)  Discordant: (abnormal) QRS is upgoing, T wave is downgoing or vice versa
3. morphology (size and shape)

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Biphasic T wave • Biphasic T waves can be "up then down", or "down, then up". • There are 2 causes of biphasic T waves:  Ischemia  Wellens’ syndrome (type II):  Two types of Wellens’ syndrome are identified:

1. Type I: The most common (75% of cases), characterised by deep negative T waves in V2–V3 and often in V4.
2. Type II: less common (one third of patients) ,present with biphasic T waves in V2–V3  pathognomonic of critical stenosis of the proximal left anterior descending coronary artery (LAD)  It is also known as the “widow maker” sign because of the high risk of an acute coronary syndrome within days/weeks if it is untreated  Hypokalaemia

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Q waves • A Q wave is any negative deflection that precedes an R wave on the ECG. • The evolution of Q waves is the most suggestive of an infarct. (more specific than ST elevation and cardiac enzyme for MI )  the most specific for a diagnosis of myocardial infarction • Small Q-waves are normal in most leads, and they can be prominent in leads III and aVR as a normal variant but should not be seen in leads V1-V3. • They are considered pathological if they are:  more than 1mm wide,  more than 2mm deep,  more than 25% of the depth of the QRS complex, or  seen in leads V1-V3. • Such pathological Q-waves usually indicate prior full thickness myocardial infarct.

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ECG: ST elevation (STE) Causes of ST elevation • myocardial infarction • pericarditis • normal variant - 'high take-off' • left ventricular aneurysm • Prinzmetal's angina (coronary artery spasm) •

rare: subarachnoid haemorrhage, part of spectrum of changes in hyperkalaemia Early repolarization • Definition

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 It appears as mild ST segment elevation (appears like an elevated “J point.”) that can be diffuse; however, it is more prominent in the precordial leads. • Causes  common finding in young, healthy individuals.  Prevalence: occurs in up to 13% of the general population • Differential diagnosis  Early repolarization (benign finding)  acute myocardial infarction (convex and not diffuse)  pericarditis  The ST elevation seen in early repolarization is very similar: diffuse and concave upward.  Three things may help to distinguish pericarditis from early repolarization:

1. The ratio of the T wave amplitude to the ST elevation should be > 4 if early repolarization is present. In other words, the T wave in early repolarization is usually 4 times the amplitude of the ST elevation. Another way to describe this would be that the ST elevation is less than 25% of the T wave amplitude in early repolarization.
2. The ST elevation in early repolarization resolves when the person exercises.
3. Early repolarization, unlike pericarditis, is a benign ECG finding that should not be associated with any symptoms. Early repolarization

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QT Interval Definition • The QT interval is the time between the onset of the QRS complex and the end of the T wave. Physiology • It represents the ventricular diastole • QRS corresponds with ventricular depolarization (when it contracts) and T wave corresponds with ventricular repolarization (when contraction stops). Which phase of the cardiac cycle shortens the most with increasing heart rate? • it is diastole.  Diastole is usually the longest portion of the cardiac cycle, and its duration diminishes the most (more than the reduction seen in the duration of systole) with increasing heart rate. Which ECG interval will show the greatest reduction during ECG stress test? • QT interval