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ECG: Junctional escape rhythm • Junctional escape rhythm describes an abnormal heart rhythm that arises within the AV node or from an adjacent area. • There is a slow, regular pulse rate. • Common after a pause in the underline rhythm • ECG shows absent P waves, narrow QRS complexes, and a heart rate of 40 to 60 bpm. • Retrograde P waves, which appear immediately before or after the QRS complex may be seen.

Cardiac amyloidosis Amyloid • Low-voltage ECG • Speckled pattern on echo Features • most commonly presents as restrictive cardiomyopathy. • clinical findings are those of right heart failure, i.e. jugular venous distension and peripheral oedema, • orthopnoea and paroxysmal nocturnal dyspnoea are typically absent. • systolic dysfunction (In more advanced stages,) • Postural hypotension can occur as a result of poor ventricular filling or associated autonomic neuropathy. Investigations • ECG The combination of low-voltage ECG and thickened ventricular walls is one of the characteristic features of cardiac amyloidosis. • Echo echocardiographic abnormalities include atrial dilatation, thickened interatrial septum, diastolic dysfunction and small-volume ventricles. The most distinctive feature of cardiac amyloidosis is a sparkling, granular appearance of the myocardium, but this is a relatively insensitive feature occurring only in about 25% of cases. 'global speckled' pattern on echo. • The history of rheumatoid arthritis and the echocardiographic finding of bi-atrial dilatation, ventricular hypertrophy and a speckled appearance to the myocardium make amyloidosis the most likely underlying cause. • Digoxin is contraindicated in amyloid patients as the digoxin binds irreversibly to the amyloid fibrils. The ECG typically shows low-voltage complexes with poor R wave progression in the chest leads (a pseudo-infarction pattern).

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ECG: Wrong leads • They are normally labelled red (right arm) and yellow (left arm). The other leads are green (left leg) and black (right leg). • If the wires to the right and left arms have been

accidentally swapped over It gives the appearance of abnormal T wave inversion in the lateral leads I and aVL. • The clue to recognising it is the inverted P waves in lead I and the upright aVR which are both highly unusual for a 12-lead ECG. The correct course of action Repeat the ECG again

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Early repolarization variant Mechanism • It is expresses as an early uptake of the ST segment before the descending limb of the R wave has reached the baseline. Features • benign but often alarming ST segment elevation Classically the ST segment elevation during early exercise returns to normal as heart rate increases further • It is common in black males • Clinical evaluation is entirely normal • ST elevation is usually seen in the precordial leads

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ECG: U wave Causes of prominent U waves are: • Hypokalaemia • Cardiovascular drugs, e.g. digitalis, quinidine, amiodarone • Psychotropic drugs, e.g. phenothiazines, tricyclic antidepressants.

Cardiac catheterisation and oxygen saturation levels • Questions regarding cardiac catheterisation and oxygen saturation levels can seem daunting at first but a few simple rules combined with logical deduction can usual produce the answer. Let's start with the basics: • deoxygenated blood returns to the right side of the heart via the superior vena cava (SVC) and inferior vena cava (IVC). It has an oxygen saturation level of around 70%. The right atrium (RA), right ventricle (RV) and pulmonary artery (PA) normally have oxygen saturation levels of around 70% • the lungs oxygenate the blood to a level of around 98-100%. The left atrium (LA), left ventricle (LV) and aorta should all therefore have oxygen saturation levels of 98-100% Some examples: Diagnosis & notes RA RV PA LA LV Aorta Normal 70% 70% 70% 100% 100% 100% Atrial septal defect (ASD) The oxygenated blood in the LA mixes with the deoxygenated blood in the RA, resulting in intermediate levels of oxygenation from the RA onwards Ventricular septal defect (VSD) The oxygenated blood in the LV mixes with the deoxygenated blood in the RV, resulting in intermediate levels of oxygenation from the RV onwards. The RA blood remains deoxygenated Patent ductus arteriosus (PDA) Remember, a PDA connects the higher pressure aorta with the lower pressure PA. This results in only the PDA having intermediate oxygenation levels VSD with Eisenmenger's 70% 70% 70% 100% 85% 85% PDA with Eisenmenger's 70% 70% 70% 100% 100% 85% ASD with Eisenmenger's 70% 70% 70% 85% 85% 85% Notes & Notes for MRCP
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Guidelines for the Interpretation of Cardiac Catheter Data • Right-heart saturations do not exceed 75%. Saturations more than this are suggestive of a left-to-right shunt. • Atrial septal defect (ASD) : The oxygen saturation in the RA and SVC should be the same. But in ASD there is a step-up in oxygen saturation at the level of the RA. This can only result from the addition of oxygenated blood

to the deoxygenated blood in the right heart circulation, that is, an abnormal connection between the right and left sides of the heart. Primum ASD: The location of the step-up is suggestive of a primum defect since these lesions occur low down in the A-V septum, lying immediately above the atrioventricular valves. These lesions can affect the function of the anterior leaflet of the mitral valve, causing mitral regurgitation. high pressures of Right ventricular are more likely to occur with primum ASDs. • Patent ductus arteriosus (PDA) unexpected step-up in oxygen saturation between the RV and PA. high pulmonary artery pressures high wedge pressure.

 The change in O2 saturation between the ascending and descending aorta strongly suggests the presence of a patent ductus. Unfortunately the extremely elevated right sided pressures are indicative of advanced disease, not amenable to surgical correction. In late disease the machinery murmur said to be characteristic of the disease may well not be audible. • Left-heart saturations vary from 96–98%. Saturations less than this are suggestive of a right-to-left shunt. • In right-to-left shunts, the arterial saturations do not change with inspired high-concentration oxygen. • Ventricular septal defect (VSD) There is a step-up in the oxygen saturation between the RA and RV. This can only occur when there is an abnormal connection between these two chambers, that is, via a VSD. This is confirmed by the raised right ventricular pressures. VSD with Eisenmenger's syndrome the pressures in the RV and PA are markedly elevated, but RA pressure is normal. The left ventricular oxygen saturation is low, which raises the possiblity of a right to left cardiac shunt mixing desaturated RV blood with LV saturated blood (due to right ventricular pressures exceeding left ventricular pressure). post-MI VSD and papillary rupture are difficult to distinguish clinically. The diagnosis is established by demonstration of a left to right shunt. if there is a step-up in the oxygen saturation between the RA and PA VSD if there is no step-up, papillary muscle rupture. • Fallot's tetralogy

1. VSD: step-down in oxygen saturation between LA and LV, indicating right to left shunt at the level of the ventricles.
2. Pulmonary stenosis: there is ↑mmHg gradient across the pulmonary valve (RV systolic - PA systolic).
3. RVH: Right ventricular pressures are high and there is a right to left shunt, which indicated by the oxygen saturations.
4. Over-riding aorta: there is a further step-down in oxygen saturation between the LV and aorta. This could occur in either Fallot's or with a patent ductus arteriosus with right to left shunting. However, given the other features of Fallot's, this is most likely to be caused by an over-riding aorta with reduced saturations due to a mixture of deoxygenated blood from the RV entering the left heart circulation. The over-riding aorta recieves a mixture of blood from the left and right ventricles as is formed above a VSD. • Pulmonary hypertension does not occur in Fallot's tetralogy due to narrowing of the right ventricular outflow tract/ subpulmonary valve stenosis. • A VSD with a right-to-left shunt and pulmonary stenosis can be differentiated from Fallot’s tetralogy by examining the oxygen saturation in the left ventricle and the ascending aorta. In the case of a VSD, the saturations in the left ventricle and the aorta will both be low and very similar.

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 In the case of Fallot’s tetralogy, the aortic oxygen saturation will be much lower than the oxygen saturation in the left ventricle because the right ventricle pumps most of the deoxygenated blood into the overriding aorta. • A pulmonary artery pressure exceeding 35 mmHg is suggestive of pulmonary hypertension. • A pressure drop of more than 10 mmHg across the aortic or pulmonary valve is suggestive of aortic or pulmonary stenosis, respectively. • The diagnosis of mitral regurgitation cannot be made unless you are given the PCWP ‘vwave’. A v-wave higher than 20 mmHg is highly suggestive of mitral regurgitation. • The right and LVEDP and the left and right atrial pressures are roughly equal in pericardial constriction • When interpreting right heart catheter data, remember the saturation should decrease gradually as the venous blood reaches the pulmonary capillary wedge saturation, which should be equal to arterial blood. • In Ebsteins anomaly there should be elevated RA pressure due to significant tricuspid regurgitation. • Hypertrophic cardiomyopathy Left ventricular pressures are high with a steep drop-off between the LV and aortic systolic pressures. • Anomalous pulmonary venous drainage to SVC normally oxygenation in the superior vena cava should always be lower than the inferior vena cava, due to the high oxygen demands from the brain. If SVC sats is markedly higher than the IVC, sugest a diagnosis of anomalous pulmonary venous drainage of more highly oxygenated blood into the SVC (left to right shunt). What is meaning of “valve gradient”? • The valve’s gradient describes the severity of the narrowing of the valve by the increase in pressure behind it. • It helps to measure the amount of blood that is able to pass through the valve. • It also indicates whether the “velocity” (or speed of movement) of the blood flow is increased because of the increased pressure behind the narrowed valve. Diagnosis of tricuspid stenosis • mean gradient by echocardiogram or cardiac catheterisation of 2 mmHg or greater, but is usually found to be >7 to 10 mmHg in severe TS Diagnosis of pulmonary hypertension • If the pulmonary arterial pressure is greater than the normal one-fifth of systolic measurements pulmonary hypertension is present. Diagnosis of right ventricular failure • The right atrial pressure is grossly elevated, with a normal wedge pressure. Normal right atrial pressure = (4–8) mmHg. Normal indirect left atrial mean pressure (wedge) = (5–10) mmHg. normal wedge pressure excludes acute left ventricular failure or acute mitral regurgitation. Diagnosis of aortic stenosis • a greater than 25mmHg gradient across the aorta valve, demonstrating moderate aortic stenosis. • systolic gradient of ↑mmHg across the aortic valve (LV systolic pressure - aortic systolic pressure), indicating critical aortic stenosis. • Hypertrophic cardiomyopathy may result in similar pressure differences, but given the clinical information, aortic stenosis is far more likely than hypertrophic obstructive cardiomyopathy (HOCM) in an old patient. • A guide to determining the severity of aortic stenosis is given below:

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Severity of aortic stenosis Severity Valve area (cm2) Mean gradient (mmHg) Mild

> 1.5 <25 Moderate 1.0-1.5 25-50 Severe <1.0 50 Critical <0.7 80 Diagnosis of mitral stenosis • A normal mitral valve expects less than 5mmHg pressure difference. • Using these inferences, the mitral valve gradient is calculated by the capillary wedge pressure of mmHg (same as the left atrial pressure) minus the diastolic left ventricular pressure of mmHg: the mmHg difference more than 5 demonstrates mitral stenosis. • The PCWP is equal to the LVEDP. When the PCWP exceeds the LVEDP, the diagnosis of mitral stenosis should be considered. • The gradient across the mitral valve (LA pressure - LV end diastolic pressure); it is usual to use the PCWP as a surrogate for LA pressure. • There is also evidence of right ventricular hypertrophy, with markedly elevated RV pressures due to secondary pulmonary hypertension. • The severity of mitral stenosis can be graded: Severity of mitral stenosis Severity Valve area (cm2) Gradient (mmHg) Mild 1.6-2.0 <5 Moderate 1.0-1.5 5-10 Severe <1.0 10 Aortic incompetence • wide pulse pressure in the aorta • high left ventricular end-diastolic pressure (LVEDP). LVEDP greater than 20 mmHg is suggestive of irreversible LV dysfunction. • All left heart valve diseases can ultimately cause elevated right heart pressures Coarctation of the aorta • There is a steep systolic gradient between the left ventricle and the femoral artery Pulmonary artery floatation catheter findings: • if the pulmonary artery occlusion pressure is low with a relatively low cardiac index, suggesting the patient is hypovolaemic, even in spite of high right atrial pressure. A fluid challenge should be performed, and values re-measured to assess response. In a fluid replete patient, the occlusion pressure would be higher (usually >13 mmHg) • if the Pulmonary artery occlusion pressure is high and cardiac index low (i.e. <2.5 L/min/m2) this would be more suggestive of cardiogenic shock. Pulmonary artery floatation catheter findings: • Low pulmonary artery occlusion pressure + low cardiac index hypovolaemia • High pulmonary artery occlusion pressure + low cardiac index cardiogenic shock

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Hyperthyroidism and cardiac catheterisation: • Cardiac catheterisation requires the use of an iodine-containing contrast. • This may worsen hyperthyroidism caused by toxic multinodular goitre, whereas it may improve the symptoms in patients with Grave’s disease (Wolff–Chaikoff effect). • The most reliable diagnostic method is a radionuclide (99Tcm, 123I or 131I) scan of the thyroid, which will distinguish the diffuse, high uptake of Grave’s disease from nodular thyroid disease. • If a toxic multinodular goitre or toxic adenoma is detected, the patient should receive an antithyroid drug before undergoing catheterisation. • The antithyroid medication must be continued for at least 2 weeks after the procedure.

Pulmonary capillary wedge pressure • Pulmonary capillary wedge pressure (PCWP) is measured using a balloon tipped SwanGanz catheter which is inserted into the pulmonary artery. • The pressure measured is similar to that of the left atrium (normally 6-12 mmHg). • The PCWP provides an indirect measurement of the left atrial pressure, and since the left atrial pressure is increased, the PCWP will also be increased. • One of the main uses of measuring the PCWP is determining whether pulmonary oedema is caused by either heart failure or acute respiratory distress syndrome. • In many modern ITU departments PCWP measurement has been replaced by non-invasive techniques. Which method is an appropriate of measuring adequate intravascular filling? • PiCCO (pulse contour cardiac output) PiCCO gives indications of cardiac output, extravascular lung water, intravascular filling and only requires a central line and a PiCCO femoral arterial line and as such is relatively simple to use.

Cardiac imaging: non-invasive techniques excluding echocardiography Nuclear imaging • These techniques use radiotracers which are extracted by normal myocardium. • Examples include: Thallium Nuclear isotopes are picked up by the Na/K ATPase of normal myocardium. If cardiac tissue is alive and perfused, it will pick up the nuclear isotope. To the myocardium, thallium looks like potassium. Decreased uptake = Damage technetium (99mTc) sestamibi: a coordination complex of the radioisotope technetium-99m with the ligand methoxy-iso-butyl isonitrile (MIBI), used in 'MIBI' or cardiac Single Photon Emission Computed Tomography (SPECT) scans fluorodeoxyglucose (FDG): used in Positron Emission Tomography (PET) scans Cardiac PET is predominately a research tool at the current time

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SPECT • The primary role of SPECT is to assess myocardial perfusion and myocardial viability. • Two sets of images are usually acquired. First the myocardium at rest followed by images of the myocardium during stress (either exercise or following adenosine / dipyridamole). • By comparing the rest with stress images any areas of ischaemia can classified as reversible or fixed (e.g. Following a myocardial infarction). MUGA • Multi Gated Acquisition Scan, also known as radionuclide angiography • radionuclide (technetium-99m) is injected intravenously • the patient is placed under a gamma camera • may be performed as a stress test • can accurately measure left ventricular ejection fraction. • Typically used before and after cardiotoxic drugs are used Cardiac Computed Tomography (CT) • Cardiac CT is useful for assessing suspected ischaemic heart disease, using two main methods: calcium score: there is known to be a correlation between the amount of atherosclerotic plaque calcium and the risk of future ischaemic events. Cardiac CT can quantify the amount of calcium producing a 'calcium score' contrast enhanced CT: allows visualisation of the coronary artery lumen • If these two techniques are combined cardiac CT has a very high negative predictive value for ischaemic heart disease. • The updated NICE guidelines recommends that cardiac CT is the first-line investigation for patients presenting with new-onset chest pain due to suspected CAD. Cardiac MRI • Cardiac MRI (commonly termed CMR) has become the gold standard for providing structural images of the heart. • It is particularly useful in: assessing congenital heart disease, determining right and left ventricular mass and differentiating forms of cardiomyopathy. Myocardial perfusion can also be assessed following the administration of gadolinium. • Currently CMR provides limited data on the extent of coronary

artery disease.

Mitral stenosis (MS) Pathophysiology • MS → mechanical obstruction of blood flow into the left ventricle (LV) → limited diastolic filling of the LV (↓ end-diastolic LV volume) → decreased stroke volume → decreased cardiac output (forward heart failure) • MS → ↑left atrial pressure → backup of blood into lungs → ↑pulmonary capillary pressure → cardiogenic pulmonary edema → pulmonary hypertension → backward heart failure and right ventricular hypertrophy

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Cardiology Causes • Common →Rheumatic fever Rheumatic valve disease is increasing uncommon in the UK, but can still be seen in other parts of the world. The physiological stress of pregnancy can exacerbate the features of rheumatic mitral stenosis. • Rare Calcification of the mitral valve annulus Autoimmune diseases: systemic lupus erythematosus, rheumatoid arthritis Congenital Mucopolysaccharidoses carcinoid Features • Malar flush: Mauve discoloration of the cheeks due to low cardiac output and systemic vasoconstriction • Dyspnea • Low volume pulse • Tapping apex beat • Auscultation Loud first heart sound (S1) Mid-late diastolic murmur (with pre-systolic accentuation) heard best at the 5th left intercostal space at the midclavicular line (the apex) in expiration. Opening snap A high frequency, early to mid-diastolic sound, heard after S2 suggests that the mitral valve is mobile opening snap is not heard when the mitral valve is heavily calcified the high left atrial pressure → rapid reversal of anterior mitral valve leaflet towards the left ventricle in early diastole lead to early diastole sound. Complications • Compression by the enlarged left atrium Compression of the esophagus → Dysphagia Compression of the recurrent laryngeal nerve → Hoarseness (known as Ortner syndrome.) • Atrial fibrillation Embolic disease (e.g., stroke, mesenteric ischemia) Patients with mitral stenosis often develop acute heart failure following the onset of atrial fibrillation. • Leads to left atrial enlargement, but the left ventricle is usually small. • Right heart failure (paroxysmal nocturnal dyspnea, orthopnea, lower limb pitting edema , bibasilar rales) • Hemoptysis Mechanism of opening snap earlier in worsening MS • The mitral valve opens when LA pressure > LV pressure. Worse MS = Higher LA pressure. Higher LA pressure pushes the mitral valve open earlier. Features of severe MS • length of murmur increases • opening snap becomes closer to S2. (shorter interval between S2 and opening snap) opening snap is characteristically lost with heavy valvular calcification • high transvalvular pressure gradient and high blood flow velocity.

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Investigations • Transthoracic echocardiography (TTE) TTE is the most important test for diagnosing and guiding the treatment of mitral stenosis. Characteristic findings include: Reduced mitral valve area (MVA): ≤ 1.5 cm2 is considered to be severe MS Thickened, calcified leaflets with commissural fusion RV dilation LA enlargement Evidence of pulmonary hypertension • Chest x-ray Left atrial enlargement may be seen The main bronchi appear elevated and have > 90% angulation (splayed). Straightening or convexity of the left cardiac border Double density sign (the silhouette of the enlarged left atrium appears near that of the

right atrium.) Cardiomegaly Pulmonary congestion • ECG Often normal Characteristic findings include: Left atrial enlargement/P mitrale Atrial fibrillation
 Right ventricular hypertrophy (e.g., right axis deviation, dominant R wave in lead V1) Chest x-ray from a patient with mitral stenosis. This patient has had a sternotomy and a prosthetic mitral valve. There is splaying of the carina with elevation of the left main bronchus, a double right heart border and cardiomegaly. The features are those of left atrial enlargement. Although the entire heart is enlarged, a double contour is seen through the right side of the heart. The more medial line is the enlarged left atrium (white dotted line) and the heart border is more lateral (blue dotted line).

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Management • Asymptomatic: echocardiography follow-up every 3 to 5 years if the mitral valve area (MVA) is >1.5 cm2 every 1 to 2 years if the MVA is 1.0 to 1.5 cm2 once per year if the MVA is <1.0 cm2. • Symptomatic with severe MS 1st line: transcatheter valvotomy : in patients with favorable valve morphology 2nd line: surgical mitral valve replacement: if transcatheter valvotomy is unsuitable. Indications for surgical mitral valve replacement • Unfavorable anatomy for transcatheter valvotomy (Percutaneous mitral valve balloon commissurotomy) • Presence of thrombus in the left atrium • Mixed valvular disease (e.g., severe MR, tricuspid disease) Mitral stenosis in pregnancy • Overview MS is poorly tolerated in pregnancy due to volume overload. Pregnancy can unmask previously undiagnosed obstructive valvular heart disease. The symptoms may developed in the second trimester, when the demand for cardiac output increases by around 70%. • Treatment Medical therapy for mild symptoms (beta blockers and/or diuretics) Percutaneous mitral balloon valvuloplasty (PMBV) should be carried out for severe mitral stenosis in patients who remain symptomatic despite medical therapy. Symptomatic patients with moderate to severe MS (mitral valve area ≤1.5 cm2) should undergo intervention, preferably percutaneous balloon mitral valvotomy, before pregnancy. Vaginal delivery with assisted second stage is the preferred mode of delivery with caesarian delivery generally reserved for obstetric reasons.

Mitral regurgitation (MR) Valvular anatomy • left atrial enlargement can result in mitral regurgitation by affecting which leaflet? posterior leaflet anterior leaflet is not affected, because of its attachment to the root of the aorta. Pathology
• Myxomatous degeneration (the most common cause of MR in UK). Risk factors and aetiology • MR associated with Marfan syndrome and Ehlers-Danlos syndrome. • cardiac complication seen 3-14 days post-myocardial infarction that occurs due to papillary muscle rupture. Features • Symptoms dyspnoea, usually on exertion, decreased exercise tolerance. palpitations,

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• Signs soft S1, split S2 pan-systolic murmur Typically presents as a holosystolic blowing murmur at the apex, radiating to axilla. intensified by isometric exercise and thus helps to differentiate it from other systolic murmurs. Sudden standing and amyl nitrite decrease the

murmur. Diagnosis • Transthoracic echo is the diagnostic test of choice Which feature suggests more severe mitral regurgitation? • As mitral regurgitation becomes more severe, the left ventricle enlarges, and the apex beat displaces, and a systolic thrill can develop. Management • asymptomatic chronic MR: left ventricular ejection fraction >60% and/or left ventricular end-systolic diameter <45 mm (ACE) inhibitors + beta-blockers left ventricular ejection fraction 60% or less and/or left ventricular end-systolic diameter 45 mm or more surgery • symptomatic chronic MR left ventricular ejection fraction 30% or more surgery + medical treatment (ACE inhibitors, beta-blockers, and diuretics.) left ventricular ejection fraction <30% medical treatment intra-aortic balloon counterpulsation in severe acute cases

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Mitral valve prolapse (MVP) Epidemiology • common, occurring in around 5-10 % of the population. • the most common valvular defect in the United States • more common in females. Causes • usually idiopathic • inherited in an autosomal dominant fashion. • may be associated with: congenital heart disease: PDA, ASD cardiomyopathy Turner's syndrome Marfan's syndrome, Fragile X osteogenesis imperfecta pseudoxanthoma elasticum Wolff-Parkinson White syndrome long-QT syndrome Ehlers-Danlos Syndrome polycystic kidney disease 15-40% of people with panic disorder have associated mitral valve prolapse.

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Features The late systolic murmur with mid systolic click is indicative of mitral valve prolapse where the posterior leaflets bulge during systole. • atypical chest pain (the most common symptom) • palpitations • dyspnea, exercise intolerance, • dizziness or syncope, • panic and anxiety disorders. • mid-systolic click (occurs later if patient squatting) • late systolic murmur (longer if patient standing) heard best at the apex Complications • mitral regurgitation, • arrhythmias (including long QT), • emboli, • sudden death Treatment • Mild to moderate mitral regurgitation follow-up in clinic with repeat echocardiograms to monitor progression. • Mitral valve replacement is only indicated in: severe mitral regurgitation or if there are signs of concomitant LV compromise (reduced ejection fraction or new dilatation of the LV). • If a surgical mitral valve replacement are indicated, coronary angiogram should be part of the pre-op work-up for potential concomitant coronary artery bypass grafting.

Aortic dissection • It is most common between the ages of 50-70, being rare below the age of 40. Stanford classification • type A - Ascending aorta, (immediately above of the aortic valve) 2/3 of cases • type B - descending aorta, (after the aorta arch) distal to left subclavian origin, 1/3 of cases DeBakey classification • type I - originates in ascending aorta, propagates to at least the aortic arch and possibly beyond it distally • type II - originates in and is confined to the ascending aorta • type III - originates in descending aorta, rarely extends proximally but will extend distally Associations • hypertension (The most common risk factor) • trauma (direct blunt chest trauma) • collagens: Marfan's syndrome, EhlersDanlos syndrome • bicuspid aortic valve • Turner's and Noonan's syndrome • pregnancy • syphilis • Drugs (such as cocaine) Complications of backward tear • aortic incompetence/regurgitation • MI: inferior pattern often seen due to right coronary

involvement

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Complications of forward tear • unequal arm pulses and BP • stroke • renal failure Stanford type A / DeBakey type I Stanford type A / DeBakey type II Stanford type B / DeBakey type III Investigations • The best investigation is a CT chest with IV contrast (CT aortogram) because the IV contrast will be able to best demonstrate the size and extent of the false lumen. • Chest X-ray: is a useful first line investigation because its readily available it is, and useful for ruling out many other conditions. The chest X-ray may show a widened mediastinum (greater than 8 cm) but unfortunately, it is not a sensitive or specific investigation as 20% of patients present with normal chest X-ray and there are many causes of a widened mediastinum.

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 Looking for a separation of the intimal calcification from the outer aortic soft tissue border by 10 mm is an indication of the presence of a dissection. • In a man with low blood pressure and vague abdominal pain, always be mindful of the possibility of dissection or aneurysmal rupture. • Occasionally, there is involvement of the right coronary artery in the dissection process giving rise to the acute electrocardiographic changes. • MRI has the best sensitivity (98%) and specificity (98%) for aortic dissection. • Whilst an echocardiogram might identify disruption of the aortic root in a backwards tear, it would not identify more distal aortic pathology. This chest X-ray shows a widened mediastinum

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This computerised tomography (CT) scan demonstrates an obvious flap in the thoracic aorta indicating aortic dissection. The flap is in the middle of the descending aorta (the dark line) which separates the true lumen anteriorly from the intimal flap posteriorly. The aortic regurgitant murmur would alert the examiner to this and mediastinal widening may be seen on x ray. Differential diagnosis • Myocardial infarction and aortic dissection: an important differential diagnosis The ECG changes of inferior myocardial infarct suggest that the aneurysm has dissected the right coronary artery at its ascending aortic ostium. An inferior myocardial infarct is high in the differential; however thrombolysis will kill a patient with an aortic dissection. (delayed diagnosis and surgical treatment) up to 85% of patients with dissections may not receive appropriate medical treatment in the first hours of treatment due to an incorrect diagnosis pain onset pain in aortic dissection is abrupt in onset and maximal at the time of onset. pain associated with MI starts slowly and gains in intensity with time. Pain character In dissection although tearing is the classical description, the pain is described as sharp more often than tearing, ripping, or stabbing. In MI it is usually more oppressive and dull. Pain site with distal dissections the pain location is between the scapulae and in the back. • Oesophageal rupture Features that favor

oesophageal rupture over aortic dissection include: The history of onset while eating Blood pressure equal in both arms No diastolic murmur Good peripheral pulses, and Presence of a pleural effusion. the history of chest pain radiating to the back is concerning., early diastolic murmur suggesting aortic valve regurgitation, ECG changes in the inferior territory and indicating occlusion of the right coronary artery. These features combined suggest that the aortic dissection has tracked back to the heart itself. The enlarged heart on chest X-ray may suggest a haemopericardium, and the patient should be assessed for cardiac tamponade given his low blood pressure. This patient is highly unstable and requires urgent cardiothoracic involvement . the most appropriate next step in the management Bedside echocardiogram and urgent cardiothoracic review

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Management • Type A surgical management, but blood pressure should be controlled to a target systolic of 100-120 mmHg whilst awaiting intervention The most appropriate management strategy is to provide adequate analgesia and urgently reduce the blood pressure with intravenous antihypertensives: beta-blockers first line, and then nitroprusside. Then the cardiothoracic surgeons should be contacted. perioperative management of patients undergoing high risk vascular surgery prophylactic beta blockers for high risk vascular surgery (including those patients with COPD). Bisoprolol is the best clinical choice Atenolol is next best choice; it is cardioselective and long acting, reducing risk of postoperative myocardial ischaemia and tachycardia. • Type B conservative management bed rest reduce blood pressure IV labetalol to prevent progression endovascular repair of type B aortic dissection may have a role in the future Complication • haemopericardium and cardiac tamponade If the dissection (involving the ascending aorta (Stanford type A) results in a tear of the tunica externa, aortic blood can leak into the pericardium. Management of aortic dissection complicated by haemopericardium and cardiac tamponade acute type A aortic dissection complicated by haemopericardium and cardiac tamponade: Relatively stable patient immediate surgical repair and surgical evacuation of haemopericardium. Pericardiocentesis in these patients can increase the intra-aortic pressure and reopen the closed communication between false lumen and pericardium. This can lead to recurrent cardiac tamponade that may be lethal. marked hypotension or electromechanical dissociation pericardiocentesis Prevention • The management of patients with predisposing inherited diseases such as Marfan's syndrome and Ehlers-Danlos syndrome should include: Periodic aortic diameter screening. Lifelong beta-blockade. Consideration of prophylactic replacement of the aortic root if dilated. Moderate restriction of physical activity. Prognosis • Mortality for untreated aortic dissection is 25–30% at 24 h and 65–70% at 2 weeks • dissections confined to the descending aorta are associated with better survival (80%).

Aortic aneurysms Aortic aneurysms • Most common cause of aneurysms atherosclerosis • The nice guidelines state that an aortic aneurysm of greater than 5.5 cm in diameter should be treated. Below this size, the risk of dissection is outweighed by the risk of surgery. Definition • Localized dilation of all three layers of the abdominal aortic wall (intima, media, and adventitia) to ≥ 3 cm Epidemiology • Sex: ♂ > ♀: ~2:1 Risk factors • Advanced age • Smoking (most important risk factor) • Atherosclerosis • Hypercholesterolemia and arterial hypertension • Positive family history Localization • Infrarenal: below the renal arteries : Most common location • Suprarenal: above the renal arteries Features • Aortic aneurysms are usually asymptomatic or have nonspecific symptoms. • Lower back pain • Pulsatile abdominal mass • Bruit on auscultation

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Cardiology Abdominal vs. thoracic aortic aneurysm Characteristics Abdominal aortic aneurysm Thoracic aortic aneurysm Location • Below the renal arteries (most common) Epidemiology • Advanced age • Predominantly men • More common than TAA Etiology • Smoking (most important risk factor) • Atherosclerosis • Hypercholesterolemia and arterial hypertension • Arterial hypertension • Bicuspid aortic valve • Tertiary syphilis [10] • Connective tissue diseases (e.g., Marfan syndrome, Ehlers-Danlos syndrome) • Trauma • Smoking Clinical features • Pulsatile abdominal mass • Bruit on auscultation • Lower back pain Diagnostics • Abdominal ultrasound (best initial and confirmatory test) Therapy • Indications for repair Diameter: ≥ 5.5 cm Expansion rate: ≥ 1 cm/year Symptomatic aneurysm Complications (e.g., rupture)

Aortic regurgitation (AR) Causes • due to valve disease bicuspid aortic valve the most common cause of chronic AR in a young patient is a congenital bicuspid valve. Bicuspid valve is also a common cause of early-onset aortic stenosis. infective endocarditis the vegetations prevent the valve from creating a proper seal to prevent backflow during diastole. rheumatic fever connective tissue diseases e.g. RA/SLE • due to aortic root disease Notes & Notes for MRCP By Dr. Yousif Abdallah Hamad

• Ascending aorta (most common) • Advanced age • Predominantly men • Feeling of pressure in the chest • Thoracic back pain • Chest x-ray and CTA of chest • Indications for repair Diameter: ascending aneurysm ≥ 5.5 cm; descending aneurysm ≥ 6.5 cm Expansion rate: ≥ 1 cm/year Symptomatic aneurysm Complications (e.g., rupture)

Notes & Notes for MRCP
By Dr. Yousif Abdallah Hamad

 aortic dissection Spondyloarthropathies (e.g. ankylosing spondylitis) Ankylosing spondylitis is strongly associated with aortic regurgitation (occurs in 4% of cases). An aortitis leads to aortic root dilatation with subsequent failure of leaflet coaptation. hypertension syphilis Marfan's, Ehler-Danlos syndrome Causes of acute aortic regurgitation: • ascending aortic dissection, • infective endocarditis, • collagen vascular disorders such as Marfan’s • trauma, • dehiscence of a prosthetic valve. Features • early diastolic murmur heard along the left sternal border heard best while the patient is leaning forward on deep expiration. • collapsing pulse • wide pulse pressure • mid-diastolic Austin-Flint murmur It is a low frequency mid/late diastolic murmur due to partial closure of the anterior mitral valve cusps caused by the regurgitation streams.

There is no correlation between the presence of murmur and severity of AR, or aetiology. • Note that there is often an aortic systolic flow murmur because there is an increased volume of blood in the LV due to the regurgitation. • Isolated LV dilatation (other chambers are normal) on ECHO due to volume overload (AS, HOCM & ↑ BP hypertrophy and a smaller LV cavity) • Pulsus bisferiens; increased pulse pressure; visible, forceful, and bounding peripheral pulses (water hammer) • Corrigan's pulse - visible and vigorous arterial pulsations in neck • Musset's sign - Bobbing of the head, due to the arterial pulsations in the neck • Quincke's sign - Capillary pulsations of the nail bed • Muller's sign - Pulsations of the uvula • Traube's sign - Loud systolic sound over femoral arteries ('pistol-shot' femorals) • Duroziez sign - diastolic murmur proximal to femoral artery compression (due to flow reversal). • Hill’s sign (Higher systolic in leg than arm) Signs of severity of AR • Soft S2 • S3 • Austin Flint murmur (functional mdm at the apex due to regurgitant jet striking the anterior leaflet of the MV, therefore obstructing flow from the LA into the LV) • characteristic of the murmur. (Duration and loudness) (cf with AS) As the lesion becomes more severe, the murmur shortens. • Apex beat displaced and thrusting

Cardiology

• CCF (pulmonary oedema) • Wide pulse pressure • collapsing pulse, • Hill’s sign (Higher systolic in leg than arm) Investigations • Echocardiogram (the most important test) Echocardiographic markers of severe AR Width of AR jet on colour flow > 65 % of LVOT regurgitant fraction (RF) > 50 % left ventricular end-diastolic diameter (LVEDD) > 70mm left ventricular end-systolic diameter (LVESD) > 50mm • Cardiac catheterisation may be performed if there is doubt over the severity of the regurgitation; severity is estimated by the degree of contrast that fills the ventricles after injection into the aortic root. Treatment • Asymptomatic: Asymptomatic without signs of sever AR: ACEI improve the prognosis in asymptomatic left ventricular dysfunction. Beta blockers should be avoided as these prolong diastole and therefore would increase the regurgitant fraction. Asymptomatic with signs of sever AR: surgery (Indications for surgery in asymptomatic): signs of sever AR (echo criteria): LV ejection fraction under 50% LV end diastolic diameter greater than 7 cm LV end systolic diameter greater than 5 cm. Patient has moderate AR and is undergoing coronary artery bypass surgery or other surgery involving the ascending aorta = surgery • Symptomatic: Surgical Symptomatic (CCF, angina) deteriorating exercise tolerance, or abnormal hemodynamic responses to exercise, such as inability to augment blood pressure during a treadmill study

Aortic stenosis (AS) Epidemiology • Aortic stenosis (AS) is the most common valve problem in the United Kingdom. Risk factors • age >60 years • congenitally bicuspid aortic valve • rheumatic heart disease • chronic kidney disease Causes • degenerative calcification (tricuspid aortic valve calcification) most common cause in older patients > 65 years • congenital bicuspid aortic valve (BAV) most common cause in younger patients < 65 years
 BAV is the most common form of congenital heart disease in adults (1-2% of population). The

European Society of Cardiology states that there is an estimated 10% chance of a first degree relative being affected, which increases to 20-30% if you consider aortopathy. NOTCH1 gene mutations may be responsible. It is possible that up to a third of relatives of patients with a bicuspid valve have valve or aortic abnormalities (often a dilated aorta). NOTCH1 gene mutations may be responsible. most helpful in establishing a diagnosis of congenital bicuspid valve as the aetiology is Systolic ejection click (best heard at the apex) aortic valve replacement is eventually likely to be required Only 15% of patients with a bicuspid aortic valve will have a normally functioning valve in the fifth decade, and this often continues to deteriorate with age. • William's syndrome (supravalvular aortic stenosis) • post-rheumatic disease fibrosis Commissural fusion on ECHO • subvalvular: HOCM Pathophysiology • Pathophysiological response in aortic stenosis The LV hypertrophies increase (in the size of myocytes) in a concentric - rather than an eccentric (asymmetric) - manner in response to the increase in afterload. Aortic stenosis - most common cause: • younger patients < 65 years: bicuspid aortic valve • older patients > 65 years: calcification Aortic stenosis - S4 is a marker of severity

 There is also an increase in interstitial collagen and little fibrosis The triad of angina, left ventricular failure and syncope is classical to aortic stenosis. Features Narrow pulse pressure and new murmur aortic stenosis • Symptoms heart failure SAD Syncope (40%) Angina or chest pain (50%) Dyspnea (60%) Exertional dyspnea is the most common initial complaint • Physical exam pulse narrow pulse pressure slow rising pulse pulsus parvus et tardus weak pulses with a delayed peak Displaced apex beat thrill ejection systolic murmur (ESM) crescendo-decrescendo murmur typically, a mid-systolic ejection murmur heard best with the diaphragm of the stethoscope in the 2nd intercostal space in a patient who is sitting upright leaning forward. in the elderly the more high frequency components of aortic stenosis may be heard best at the apex, the so called (Gallavardin phenomenon) may have ejection click radiates to carotid arteries (left often louder than right). radiate to the right neck decreases with standing, Valsalva, or handgrip increases with amyl nitrate, squat, or leg raise The intensity of the systolic murmur does not correspond to the severity of aortic stenosis; As LV contractility decreases in critical AS, the murmur becomes softer and shorter. The intensity of the murmur may therefore be misleading in these circumstances. the timing of the peak and the duration of the murmur correspond to the severity of aortic stenosis. The more severe the stenosis, the longer the duration of the murmur and the more likely it peaks at late systole. S4 heart sound from stiff or hypertrophic ventricle S2 (Character of S2) soft/absent S2 paradoxical splitting of S2 heard on expiration rather than inspiration

Associated conditions • hemolytic anemia • predisposes to bleeding due to an acquired von Willebrand deficiency caused by turbulent flow across the stenotic valve. • chronic gastrointestinal bleeding that is associated with angiodysplasia. Severity of aortic stenosis • The severity of (AS)

can be accurately assessed with echocardiography. • the severity of AS is difficult to assess with echocardiography when cardiac output is low. • Catheterization to determine the severity of AS is reserved for patients in whom echocardiography is nondiagnostic • The volume of the murmur has NO relationship to the severity of the stenosis In a patient with aortic stenosis, what will lead to an overestimation of the severity of the problem when assessed by echocardiography? Aortic regurgitation due to large volumes of blood passing over the valve at high velocities Which condition is most associated with quietening of the aortic stenotic murmur? Left ventricular systolic dysfunction decreased flow-rate across the aortic valve and hence a quieter murmur. Atrial fibrillation Where the R-R interval is particularly short, such as in atrial fibrillation, flow across the valve is reduced, as such the intensity of the murmur is variable and may be significantly reduced. Aortic regurgitation has no effect on the intensity of the murmur, such that in patients with mixed aortic valve disease, the stenotic murmur is still clearly audible. Conditions which leads to accentuation of the murmur increased flow across the murmur. • High output cardiac failure • severe thyrotoxicosis The predominant component of mixed aortic valve disease is determined by the murmur that is louder (ejection systolic murmur in aortic stenosis and mid diastolic murmur for aortic regurgitation). Evaluation • Severe AS is defined by a valve area of less than 1.0 cm2. • distinguish patients with true severe (AS) with secondary LV dysfunction from those who have a falsely low calculated aortic valve area because of low cardiac output. Features of severe aortic stenosis

1. narrow pulse pressure
2. slow rising pulse
3. delayed ESM
4. soft/absent S2
5. S4
6. thrill
7. duration of murmur
8. left ventricular hypertrophy or failure

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