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Chapter 5

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

Cardiology

 calculated valve area in patients with severe left ventricular (LV) dysfunction can be falsely low because low cardiac output reduces the valve opening forces. It is important to 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. An important method of distinguishing between the two conditions is to assess the haemodynamics after increasing the cardiac output by dobutamine infusion during echocardiography or cardiac catheterisation. Patients with truly severe AS manifest an increase in trans-aortic pressure gradient while the valve surface area remains the same during dobutamine infusion; those with falsely low calculated valve area manifest an increase in calculated valve surface area. Dobutamine echocardiography is also important to assess LV contractile reserve. Patients who have 20% or more increase in stroke volume after dobutamine infusion have a much better prognosis after surgery compared to those who do not have LV contractile reserve. What is the difference between aortic stenosis and aortic sclerosis? • Both aortic stenosis and aortic sclerosis are : senile degeneration of the valve there is an ejection systolic murmur, • Unlike aortic stenosis, aortic sclerosis have: Occur in > 25% of > 65 year of age Aortic stenosis occur in > 2% of > 65 year of age Absence of stenosis no carotid radiation, normal pulse (character and volume) normal S2. Investigations • Echocardiography transthoracic echocardiogram (TTE) initially transesophageal echocardiogram (TEE) is more accurate Although echocardiography will aid in diagnosis, gradient across the aortic valve may be underestimated because of the possibility of multiple echo signals and coexistent left ventricular dysfunction. • Left heart catheterization most accurate diagnostic test (the definitive investigation of choice) to assess pressure gradient across the valve only indicated to confirm the diagnosis if echocardiography is unclear findings elevated pressure gradient (> 30 mmHg) In the context of poor LV function, the aortic valve gradient may be normal or only mildly raised in the presence of a severely narrowed aortic valve area. • The next step in management after diagnosis Coronary angiography Coronary artery disease (CAD) is common in patients with AS Progressing straight to aortic valve replacement is not advised; significant coronary artery disease should be ruled out first, as CABG may be required at the same time as valve replacement.

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Patients undergoing open surgical valve replacement should first undergo coronary angiography to exclude any coronary stenosis that could simultaneously be treated with bypass grafting. Management • if asymptomatic then observe the patient is general rule • if symptomatic then valve replacement The patient's symptomatology is the most important determinant in terms of the decision to operate There are three important factors to consider regarding management of aortic stenosis: •

1. Presence of symptoms •
2. The gradient across the valve on echocardiogram •
3. Evidence of left ventricular dysfunction. • Symptomatic patient Fit for surgery →aortic valve replacement the best treatment option in an older person who can undergo the surgery. Not fit for aortic valve replacement Transcatheter aortic valve implantation (TAVI) The catheter-delivered device produces similar one-year survival as aortic valve replacement but a higher risk of stroke, TIAs and vascular complications. Balloon valvuloplasty Balloon aortic valvuloplasty is a palliative procedure prone to restenosis for patients unsuitable for other interventions. • Asymptomatic patient with severe stenosis (transvalvular gradient > 50 mmHg, valve area <1 cm^2) but has an ejection fraction of less than 50%. should be referred for aortic valve replacement or TAVI if unsuitable. with severe stenosis but has an ejection fraction is greater than 50%. Exercise testing would be recommended If pass exercise testing, then reviewed in six months. echo follow-up asymptomatic with mild stenosis every 3 to 5 years asymptomatic with moderate stenosis every 1 to 2 years asymptomatic with severe stenosis every 6 to 12 months. Indicator of poor prognosis • Clinical features of left ventricular failure deteriorating LV function (ejection fraction less than 40%) • Symptomatology exertional breathlessness or presyncope/syncope • Increasing gradient across the valve (above 70 mmHg) • Age of patient Aortic stenosis management: AVR if symptomatic, otherwise cut-off is gradient of 50 mmHg

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Cardiology Heyde’s syndrome • association between microcytic anaemia and calcific aortic stenosis. • Heyde syndrome refers to a triad of

1. aortic stenosis,
2. acquired coagulopathy (von Willebrand syndrome type 2A) and
3. anaemia due to bleeding from intestinal angiodysplasia or from an idiopathic site. Angiodysplasia most commonly occur in the ascending colon, particularly the caecum. • Pathophysiology destruction of von Willebrand’s factor as the platelets traverse the stenosed valve resulting in bleeding per rectum. • Investigation The investigation of choice after valve replacement is mesenteric angiography as the bleeding vessels are poorly visualised on colonoscopy. This would look for the presence of angiodysplasia, which may be associated with aortic stenosis. All patients with aortic stenosis should be screened for iron deficiency anaemia. • Treatment replace the valve Resection of the diseased bowel has also been described as a treatment. • There is an association with jaundice and aortic stenosis; this is thought to be due to microangiopathic haemolysis.

Williams syndrome • Supra-valvar AS is one of the characteristic findings of Williams syndrome along with: unusual elfin facies, excellent verbal skills contrasted with intellectual disability and lack of social inhibition. hypercalcemia (due to increased sensitivity to vitamin D.) • caused by a microdeletion of the elastin gene on long arm of chromosome 7

Coarctation of the aorta Definition: • congenital narrowing of the descending aorta, most commonly at the site of insertion of the ductus arteriosus Overview • more common in males (despite association with Turner's syndrome) • a bicuspid valve is found in approximately 50% of patients with coarctation of the aorta. • site of coarctation: distal to the origin of the left subclavian artery The commonest site The systolic BP in the arms exceeds that in the leg. proximal to the origin of the left subclavian artery Supravalvular aortic stenosis is the congenital cardiovascular deformity most often associated with Williams syndrome.

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 occurs in 15% of cases of coarctation if the systolic BP in the right arm is higher than that of the left arm by more than 30 mmHg, the left subclavian is involved in the coarctation (ie coarctation is proximal to the origin of the subclavian) Features • Most patients are asymptomatic • infancy: heart failure • claudication of the calf muscles. pain in calves is almost certainly due to poor distal blood supply. • Hypertension the most common presenting feature in adults • headache and nose bleeds occur due to hypertension proximal to the coarctation, • differential blood pressures between the right and left arms • radio-femoral delay • mid systolic murmur, and thrill maximal over back. continuous murmur over the thoracic spine usually originates from small, tight coarctation (< 2 mm). • apical click from the aortic valve Complications • Secondary hypertension • development of cerebral aneurysms may present with intracranial haemorrhage from a ruptured berry aneurysm • Left ventricular failure, • Bacterial endocarditis. Associations • Bicuspid aortic valve the commonest associated congenital abnormality occurs in 50% of the coarctations. • patent ductus arteriosus (PDA) • Turner's syndrome Female patients diagnosed with coarctation of the aorta should have a karyotype analysis to rule out Turner syndrome. • berry aneurysms • neurofibromatosis Investigations • Radiograph Cardiomegaly ↑ pulmonary vascular markings rib notching notching of the inferior border of the ribs (due to collateral vessels) usually manifests in adults and older children, as it takes time to develop. may demonstrate an indentation of the aortic shadow at the site of the coarctation. rib notching is not seen in young children • Echocardiography with doppler (confirmatory test): location and extent of stenosis; concurrent anomalies Treatment • Balloon angioplasty and stenting is the preferred intervention in adults. surgical correction is indicated if the pressure gradient across the coarctation is above 20 mmHg, even without associated hypertension.

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• Prostaglandin E1 should be administered to neonates with aortic coarctation to keep the ductus arteriosus open. Differences in blood pressure between arms: • up to 10 mmHg difference Normal variant (physiological) • difference of greater than 10 mmHg: abnormal: + radio-radial or radio-femoral delay (NO Leg claudication) proximal coarctation of the aorta (involves the left subclavian artery origin) + arm claudication, intermittent vertigo, ataxia or diplopia, or facial sensory symptoms (NO Leg claudication) Subclavian steal syndrome + Leg claudication (chronic intermittent leg pain, exacerbated by exercise and relieved by rest) Peripheral vascular disease

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Bicuspid aortic valve Overview • occurs in 1-2% of the population • Bicuspid aortic valve tends to be sporadic although there is a reported familial incidence of approximately 9%. • usually asymptomatic in childhood • the majority eventually develop aortic stenosis or regurgitation associated with: • left dominant coronary circulation (the posterior descending artery arises from the circumflex instead of the right coronary artery) • Turner's syndrome • coarctation of the aorta (around 5% of patients) Complications • aortic stenosis/regurgitation as above • higher risk for aortic dissection and aneurysm formation of the ascending aorta

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Tricuspid regurgitation Signs • pan-systolic murmur • giant V waves in JVP • pulsatile hepatomegaly • left parasternal heave Causes • pulmonary hypertension e.g. COPD (The most common cause) • right ventricular dilation • rheumatic heart disease • infective endocarditis (especially intravenous drug users) • Ebstein's anomaly • carcinoid syndrome

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Prosthetic valves • The most common valves which need replacing are the aortic and mitral valve. • There are two main options for replacement: biological (bioprosthetic) or mechanical.

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Biological (bioprosthetic) valves Mechanical valves Usually bovine or porcine in origin The most common type now implanted is the bileaflet valve. Ball-and-cage valves are rarely used nowadays Advantages : not requiring Long-term anticoagulation Warfarin may be given for the first 3 months depending on patient factors. Low-dose aspirin is given long-term. Disadvantages calcification over time. must be replaced within 5 to 10 years. Most older patients (> 65 years for aortic valves and > 70 years for mitral valves) receive a bioprosthetic valve Following the 2008 NICE guidelines for prophylaxis of endocarditis antibiotics are no longer recommended for common procedures such as dental work. Which pathological findings in the bioprosthesis has most likely led to the need for replacement? Calcification with stenosis

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Supraventricular tachycardia (SVT) Definition • The term ‘SVT’ literally indicates tachycardia [atrial rates >100 beats per minute at rest, the mechanism of which involves tissue from the His bundle or above. Traditionally, SVT has been used to describe all kinds of tachycardias apart from ventricular tachycardias (VTs) and AF. Causes • Atrioventricular nodal re-entry tachycardia (AVNRT). the most common supraventricular tachycardia, twice as common in females as in males the incidence is 1–3 per 1000 Small elevations in troponin are occasionally seen in this situation, but there are no ECG changes to suggest a myocardial infarction. • Atrioventricular re-entry tachycardias (AVRT) • Junctional tachycardias. Notes & Notes for MRCP
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Advantages : have a low failure rate Disadvantages ↑ risk of thrombosis meaning long-term anticoagulation is needed. Aspirin is normally given in addition unless there is a contraindication. Target INR • aortic: 2.0-3.0 • mitral: 2.5-3.5

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Differential diagnosis Paroxysmal SVT would start and stop suddenly, not gradually. Panic attacks breathlessness and palpitations start and stop gradually. Management Vagal manoeuvres and adenosine are the treatments of choice for the acute therapy of SVT, and may also provide important diagnostic information. • Acute management haemodynamically stable patient: 1st line : vagal manoeuvres : e.g. Valsalva manoeuvre Carotid sinus massage is contraindicated in patients with carotid vascular disease 2nd line: intravenous adenosine 6mg → 12mg → 12mg Adenosine can cause flushing, chest pain, and dizziness. contraindicated in asthmatics - verapamil is a preferable option 3rd line: Verapamil or diltiazem i.v. or Beta-blockers (i.v. esmolol or metoprolol) should be considered if vagal manoeuvres and adenosine fail. 4th line: Synchronized DC cardioversion haemodynamically unstable patient: Synchronized DC cardioversion: start with 70-120 J biphasic (100 J monophasic). • Prevention of episodes 1st line: beta-blockers or 2nd line: radio-frequency ablation • Do not use flecainide or propafenone in patients with LBBB, or ischaemic or structural heart disease. • Verapamil is not recommended in wide QRS-complex tachycardia of unknown aetiology. • Flecainide and propafenone are not recommended as first-line antiarrhythmic drugs in patients with ventricular dysfunction and severe fibrosis. SVT in pregnancy • Tachyarrhythmias may increase during pregnancy although the causes are not entirely clear. • Termination of acute SVT: haemodynamic stable: Vagal manoeuvres and, if these fail, adenosine (adenosine appears to be safe in pregnancy). An i.v. beta-1 selective blocker (except atenolol) should be considered for acute conversion or rate control of SVT.

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• Prevention of recurrent SVT in patients without WPW syndrome : If possible, avoid all antiarrhythmic drugs during the first trimester of pregnancy. 1st line : beta-1 selective agents (but not atenolol) beta-blockers. The cardio-selective beta-1-blockers include atenolol, betaxolol, bisoprolol, esmolol, acebutolol, metoprolol, and nebivolol. Metoprolol is the preferred and safest

Beta-blocker in prophylaxis for SVT in pregnancy (it is a short acting β blocker and a TDS regimen is required). 2nd line: verapamil 3rd line: Fluoroless catheter ablation Prevention of recurrent SVT in patients with WPW syndrome : 1st line : Flecainide or propafenone 2nd line: Fluoroless catheter ablation

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Sinus arrhythmia • The (ECG) shows normal P wave, PR interval, QRS complex and each P wave conducted to ventricles. • There is a gradual decrease in R–R interval and then an increase again. This slight beat-to-beat variation (rhythmic and cyclical variation) is termed as sinus arrhythmia. • the most common cause is respiration. Respiratory sinus arrhythmia is thus heart rate variability in synchrony with respiration, and is normal in children and young adults. The R–R interval decreases with inspiration and increases with expiration. • Anxiety reassured.

Premature ventricular ectopic (PVEs) The first line management of supraventricular ectopics is generally reassurance and lifestyle modifications (eg: reduce alcohol and caffeine intake). If symptoms persisted, then a beta blocker would be first line.

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• usually seen in normal hearts; • palpitations are described as an early beat with a pause followed by an unusually strong or 'pounding' beat, or simply as a 'flip-flop'; Symptoms are usually worse at rest and may disappear with exercise. Symptoms which increase on exercise are more worrying and significant. • may be associated with caffeine intake • Investigations baseline ECG without symptoms: typically normal ambulatory ECG: isolated wide QRS complexes If symptoms are short-lived but frequent (>2-3 times per week), use a 24-hour Holter monitor If symptoms are short-lived and infrequent (<1 per week), use an event monitor or transtelephonic recorder Exercise stress testing the relation of extrasystoles to exercise may have prognostic importance. Echocardiography - to assess LV function and heart structure. • For PVE to be significant they have to meet the following criteria: Occurring frequently (6 or more beats/min) PVE in bigeminal rhythm PVE in short runs of ventricular tachycardia PVE exhibiting R-on-T phenomenon PVE associated with serious organic heart disease and left ventricular decompensation. • Treatment Not significant PVE Reassurance Significant PVE beta-blockers Radiofrequency catheter ablation of the ectopic focus Curative with good outcome Ventricular extrasystoles are the most common type of arrhythmia that occurs after myocardial infarction. Management of symptomatic atrial extrasystoles • beta-blockers (atenolol or metoprolol). • Atrial extrasystoles arising from the pulmonary veins may be treatable by the procedure of pulmonary vein isolation.

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Arrhythmogenic right ventricular cardiomyopathy(ARVC) Overview • Arrhythmogenic right ventricular cardiomyopathy (ARVC, also known as arrhythmogenic right ventricular dysplasia or ARVD) is a form of inherited cardiovascular disease which may present with syncope or sudden cardiac death. • It is generally regarded as the second most common cause of sudden cardiac death in the young after hypertrophic cardiomyopathy. • Although ARVC was initially described in the right ventricle, most patients have biventricular involvement. Pathophysiology • inherited in an autosomal dominant pattern with variable expression • the right ventricular myocardium is replaced by fatty and fibrofatty tissue • around 50% of patients have a mutation of one of the several genes which encode components of desmosome Presentation • palpitations • syncope • sudden cardiac death Investigation epsilon potential is seen on the ECG of patients with Right ventricular dysplasia • ECG abnormalities in V1-3: Typically, T wave inversion. An epsilon wave is found in about 50% of those with ARV - this is best described as a terminal notch in the QRS complex • echo changes are often subtle in the early stages but may show an enlarged, hypokinetic right ventricle with a thin free wall • magnetic resonance imaging is useful to show fibrofatty tissue Management • drugs: sotalol is the most widely used antiarrhythmic • catheter ablation to prevent ventricular tachycardia • implantable cardioverter-defibrillator Naxos disease • an autosomal recessive variant of ARVC • a triad of ARVC, palmoplantar keratosis, and woolly hair

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Atrial fibrillation (AF) (NICE guideline April 2021) Overview • AF is the most commonly encountered cardiac arrhythmia. • Hypertension is the most common risk factor for AF. • In 15% of cases, AF is idiopathic • AF most commonly originates from the roots of the pulmonary veins. (longitudinal smooth muscle fibers in the pulmonary vein) classification Classification of atrial fibrillation (AF): AF classified into 3 patterns:

1. first detected episode (irrespective of whether it is symptomatic or self-terminating)
2. recurrent episodes, when a patient has 2 or more episodes of AF: paroxysmal AF: episodes of AF terminate spontaneously. episodes last less than 7 days (typically < 24 hours). persistent AF the arrhythmia is not self-terminating. episodes usually last greater than 7 days
3. permanent AF there is continuous atrial fibrillation which cannot be cardioverted or if attempts to do so are deemed inappropriate. Treatment goals are therefore rate control and anticoagulation if appropriate Symptoms and signs • Symptoms Palpitations Dyspnea chest pain • Signs irregularly irregular pulse Complications • AF is poorly tolerated in elderly and often leads to pulmonary oedema even in the presence of a relatively normal left ventricle (LV). Diagnosis • if an irregular pulse is detected Perform a 12-lead electrocardiogram (ECG) • In people with suspected paroxysmal AF undetected by ECG: if asymptomatic episodes are suspected or symptomatic episodes are less than 24 hours apart use a 24- hour ambulatory ECG monitor if symptomatic episodes are more than 24 hours use an ambulatory ECG monitor, event recorder or other ECG technology.

Cardiology Assessment • Assessment of stroke and bleeding risks Assess stroke risk by using the CHA2DS2-VASc score Assess the bleeding risk when considering starting anticoagulation by using the ORBIT bleeding risk score modify risk factors for bleeding: uncontrolled hypertension poor control of international normalised ratio (INR) in patients on vitamin K antagonists concurrent medication, including antiplatelets, selective serotonin reuptake inhibitors (SSRIs) and non- steroidal anti- inflammatory drugs (NSAIDs) harmful alcohol consumption reversible causes of anaemia. • Assessment of cardiac function by transthoracic echocardiography (TTE) as a baseline and to look for underlying structural or functional heart disease . Management • Anticoagulation for stroke prevention 1st line: direct-acting oral anticoagulant (e.g. Apixaban, dabigatran, edoxaban, rivaroxaban), if CHA2DS2-VASc score ≥ 1 for men or ≥ 2 for women. 2nd line: If DOAC are contraindicated or not tolerated vitamin K antagonist. 3rd line: If anticoagulation is contraindicated or not tolerated consider left atrial appendage occlusion (LAAO). • Rate and rhythm control Rate control: the first-line treatment for AF except in:

1. AF due to reversible cause
2. heart failure caused by AF
3. new- onset AF
4. atrial flutter which considered suitable for an ablation strategy to restore sinus rhythm
5. if rhythm- control strategy would be more suitable based on clinical judgement. Use beta-blocker (other than sotalol) or a rate-limiting calcium-channel blocker (diltiazem or verapamil) Consider digoxin monotherapy for initial rate control if the person does no or very little physical exercise or other rate- limiting drug options are ruled out because of comorbidities. Rhythm control: Do not offer class 1c antiarrhythmic drugs such as flecainide or propafenone to people with known ischaemic or structural heart disease. If drug treatment for long-term rhythm control after successful cardioversion is needed: 1st line: beta-blocker 2nd line: dronedarone Amiodarone for people with left ventricular impairment or heart failure. In people with infrequent paroxysms and few symptoms, or if symptoms are induced by known precipitants (such as alcohol, caffeine), a 'no drug treatment' strategy or a 'pill-in-the-pocket' strategy (in which antiarrhythmic drugs are taken only when an episode starts) should be considered a 'pill-in-the-pocket' strategy: In people with paroxysmal AF if:

 infrequent symptomatic episodes + no left ventricular dysfunction, or valvular or ischaemic heart disease + systolic BP ˃100 mmHg and a resting heart rate ˃ 70 bpm + able to understand how to, and when to take the medication. try to get the patient back into, and maintain, normal sinus rhythm. This is termed cardioversion. Drugs (pharmacological cardioversion) and synchronised DC electrical shocks (electrical cardioversion) may be used for this purpose indications of Rhythm control : coexistent heart failure, first onset AF or where there is an obvious reversible cause. Do not use rhythm control therapy in asymptomatic AF patients, nor in patients with permanent AF. Rhythm control has no survival benefit over a rate control strategy Reducing

stroke risk Anticoagulation Young man with AF, no TIA or risk factors, no treatment is now preferred to aspirin (NO treatment) Do not use antiplatelet therapy for stroke prevention in AF • Some patients with AF are at a very low risk of stroke whilst others are at a very significant risk. • NICE in 2014 suggest using the CHA2DS2-VASc score to determine the most appropriate anticoagulation strategy

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Cardiology Risk factor Points C Congestive heart failure

H Hypertension (or treated hypertension)

A2 Age ≥75 years

Age 65-74 years

D Diabetes

S2 Prior Stroke or TIA

V Vascular disease (including ischaemic heart disease and peripheral arterial disease)

S Sex (female)

The table below shows a suggested anticoagulation strategy based on the score: Score Anticoagulation

No treatment

Males: Consider anticoagulation Females: No treatment (this is because their score of 1 is only reached due to their gender) 2 or more Offer anticoagulation Atrail fibrilation related to mitral stenosis • atrial fibrillation related to valvular heart disease Warfarin In patients with non-valvular atrial fibrillation, novel oral anticoagulants have the same efficacy as warfarin in preventing stroke. • NICE guidelines suggest that valvular disease have high risk for thromboembolic events, and would benefit from anticoagulation. • Mitral stenosis patients were excluded from the studies developing the CHADS-VASC score. • None of the ‘novel’ anticoagulants currently available (rivaroxaban, apixaban, dabigatran) are indicated or licensed for atrial fibrillation related to valvular heart disease. CHADS2-VASc scoring is generally used as a tool to assess need to anticoagulate a patient with AF. However, the following are conditions that, if present, may trump the decision to anticoagulate:

1. valvular heart disease
2. prior peripheral embolism, and
3. intracardiac thrombus.

Bleeding risk assessment (using the HASBLED scoring system) • NICE recommend that we offer patients a choice of anticoagulation, including warfarin and the novel oral anticoagulants (NOACs). • Aspirin is no longer recommended for reducing stroke risk in patients with AF • Doctors have always thought carefully about the risk/benefit profile of starting someone on warfarin. • A history of falls, old age, alcohol excess and a history of previous bleeding are common things that make us consider whether warfarinisation is in the best interests of the patient. • NICE now recommend we formalise this risk assessment using the HASBLED scoring system. Risk factor Points H Hypertension, uncontrolled, systolic BP > 160 mmHg A Abnormal renal function (dialysis or creatinine > 200) Or Abnormal liver function (cirrhosis, bilirubin > 2 times normal, ALT/AST/ALP > 3 times normal S Stroke, history of

B Bleeding, history of bleeding or tendency to bleed

L Labile INRs (unstable/high INRs, time in therapeutic range < 60%) E Elderly (> 65 years)

D Drugs Predisposing to Bleeding (Antiplatelet agents, NSAIDs) Or Alcohol Use (>8 drinks/week) • There are no formal rules on how we act on the HAS-BLED score although a score of >= 3 indicates a 'high risk' of bleeding, defined as intracranial haemorrhage, hospitalisation, haemoglobin decrease >2 g/L, and/or transfusion. Atrial fibrillation: cardioversion Atrial fibrillation - cardioversion: • if no structural heart disease flecainide • With structural heart disease amiodarone offer rate or rhythm control if the onset of the arrhythmia is less than 48 hours, and start rate control if it is more than 48 hours or is uncertain • Cardioversion indications Haemodynamically unstable patient electrical cardioversion (DC cardioversion 200J → 360J → 360J) Adverse signs necessitating DC cardioversion are: Blood pressure (BP) ≤90 mmHg Notes & Notes for MRCP
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1 for any renal abnormalities 1 for any liver abnormalities

1 for drugs 1 for alcohol

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 Chest pain Heart failure Impaired consciousness, and Heart rate ≥ 200 bpm. Elective procedure where a rhythm control strategy is preferred electrical or pharmacological cardioversion Onset < 48 hours Anticoagulation patients should be heparinised. Patients who have risk factors for ischaemic stroke should be put on lifelong oral anticoagulation. Cardioversion method: electrical - 'DC cardioversion' pharmacology: amiodarone if structural heart disease, flecainide or amiodarone in those without structural heart disease Post-cardioversion: further anticoagulation is unnecessary Onset > 48 hours prior to cardioversion: anticoagulation for at least 3 weeks prior to cardioversion. OR exclude a left atrial appendage (LAA) thrombus by transoesophageal echo (TOE). If excluded patients may be heparinised and cardioverted immediately. If there is a high risk of cardioversion failure (e.g. Previous failure or AF recurrence) then it is recommend to have at least 4 weeks amiodarone or

sotalol prior to electrical cardioversion If the patient has a slow ventricular response of AF in the absence of anti-arrhythmic drugs, cardioversion should be performed after the insertion of a temporary transvenouspacing catheter Cardioversion method: NICE recommend electrical cardioversion, rather than pharmacological. The initial shock strength should be 100 J, followed by a second 200-J shock and a third 360-J shock If AF persists, a second 360-J shock with the paddles in the anteroposterior position can be attempted Post-cardioversion: Following electrical cardioversion patients should be anticoagulated for at least 4 weeks. After this time decisions about anticoagulation should be taken on an individual basis depending on the risk of recurrence • Catheter AF ablation Radiofrequency pulmonary vein isolation with ablation the treatment of choice for patients who remain poorly controlled despite medical therapy, in selected patients as first-line therapy for symptomatic paroxysmal AF Anticoagulation for stroke prevention should be continued indefinitely in patients at high risk of stroke, even after apparently successful ablation of AF.

• Surgical AF ablation Ablation can be performed in symptomatic patients during cardiac surgery for other reasons, or by stand-alone surgery either using open-chest techniques or by thoracoscopy. Anticoagulation for stroke prevention should be continued indefinitely in patients at high risk of stroke, even after apparently successful ablation of AF. The enlarged left atrial size suggests that a repeat DC cardioversion is unlikely to work for a sustained period. H/O AF + enlarged left atrial size with previous DC cardioversions. the best long term treatment option Refer for consideration of atrial fibrillation ablation longer term good result. AV node ablation: • AV node ablation is reserved for those patients where pharmacological rate control is unsuccessful or not tolerated. • The procedure is invasive and requires permanent pacemaker implantation. • Patients who are candidates for this therapy include those with tachycardia induced cardiomyopathy despite pharmacologic efforts at rate control and intolerable symptoms despite aggressive attempts at pharmacologic therapy (in some cases, much of the symptom burden is due to medications rather than AF itself). Do not perform cardioversion or catheter ablation without anticoagulation, unless an atrial thrombus has been ruled out by transoesophageal echocardiogram. Amiodarone or vernakalant have been efficient in converting post-operative AF to sinus rhythm. Vernakalant • A Novel agent for the Termination of Atrial Fibrillation • blocks sodium channels • more prominent in vernakalant's mechanism of action is its ability to block certain potassium channels. • Specifically, it blocks the atrial-selective potassium current, IKur, which is involved in atrial repolarization. Atrial fibrillation: pharmacological cardioversion • Agents with proven efficacy in the pharmacological cardioversion of atrial fibrillation amiodarone flecainide (if no structural heart disease) with large doses of oral agents or with intravenous agents. Large single doses of flecainide (300 mg) or propafenone (450-600 mg) given orally have been shown to convert patients to sinus rhythm. Flecainide and propafenone are not used in people with : known or suspected ischaemic heart disease, individuals who are already on antiarrhythmic therapy,

 those with a prolonged QT interval because these agents may have pro-arrhythmic effects (torsade de pointes). others (less commonly used in UK): quinidine, dofetilide, ibutilide, propafenone • Less effective agents beta-blockers (including sotalol) calcium channel blockers digoxin disopyramide procainamide 1. Atrial fibrillation: rate control and maintenance of sinus rhythm The patient with very recent onset of atrial fibrillation is more likely to stay in sinus rhythm • Agents used to control rate in patients with atrial fibrillation Beta-blockers should be used first line for rate control. cardioselective beta-blockers should be tried in patients with left ventricular systolic dysfunction even if they have a diagnosis of: Chronic obstructive pulmonary disease (COPD) Peripheral vascular disease Diabetes Erectile dysfunction, or Interstitial pulmonary disease. Beta-blockers should not be commenced in the setting of acute exacerbations of COPD or cardiac failure If one drug does not control the rate adequately NICE recommend combination therapy with diltiazem or digoxin calcium channel blockers (diltiazem) digoxin: not considered first-line anymore as they are less effective at controlling the heart rate during exercise. they are the preferred choice if the patient has coexistent heart failure with borderline hypotension (eg: 95/70), COPD and AF, rate control without the possibility of worsening hypotension is the aim of intervention. Digoxin is therefore the optimal intervention. It will both slow the ventricular rate and support the blood pressure. If the duration of AF is unknown caution should be used when considering the use of drugs which may cardiovert the patient - amiodarone and flecainide. • Agents used to maintain sinus rhythm in patients with a history of atrial fibrillation sotalol amiodarone flecainide others (less commonly used in UK): disopyramide, dofetilide, procainamide, propafenone, quinidine • The table below indicates some of the factors which may be considered when considering either a rate control or rhythm control strategy

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Factors favouring rate control Factors favouring rhythm control • Older than 65 years • History of ischaemic heart disease • Younger than 65 years • Symptomatic • First presentation • Lone AF or AF secondary to a corrected precipitant (e.g. Alcohol) • Congestive heart failure MRCPUK-part-2-march-2018: H/O borderline hypotension (BP: 95/70), COPD and AF. What is the most appropriate intervention? Digoxin 500 mg IV loading B-blockers and verapamil are best avoided because of the potential for worsening hypotension here.

Atrial flutter Overview • Atrial flutter is a form of supraventricular tachycardia characterised by a succession of rapid atrial depolarisation waves. • usually caused by a single macroreentrant rhythm within the atria. • What is the differences between atrial flutter and focal atrial tachycardia? Atrial flutter is caused mechanistically by macro- reentry and has atrial rate (P wave/flutter morphology) usually >250 bpm. Focal atrial tachycardia is caused mechanistically by micro-reentry or increased automaticity and has atrial rates of 100-250 bpm. Epidemiology • Sex: ♂ > ♀ (5:2) • Peak incidence: risk of atrial flutter increases with age Etiology: • similar to atrial fibrillation ECG findings • Regular, narrow QRS complexes • flutter waves, which are a saw-tooth pattern of atrial activation most prominent in leads II, III, aVF, and V1. • as the underlying atrial rate is often

around 300/min the ventricular or heart rate is dependent on the degree of AV block. For example if there is 2:1 block the ventricular rate will be 150/min • flutter waves may be visible following carotid sinus massage or adenosine Management • is similar to that of atrial fibrillation although medication may be less effective • atrial flutter is more sensitive to cardioversion however so lower energy levels may be used • Anticoagulate patients with atrial flutter similar to AF. • Catheter ablation is the definitive treatment for atrial flutter. radiofrequency ablation of the tricuspid valve isthmus is curative for most patients

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Multifocal atrial tachycardia (MAT) Multifocal atrial tachycardia has ≥ 3 P-wave morphologies on ECG Definition • it is an irregular cardiac rhythm caused by at least three different sites in the atria, which may be demonstrated by morphologically distinctive P waves. • It is more common in elderly patients with chronic lung disease, for example COPD Management • correction of hypoxia and electrolyte disturbances • rate-limiting calcium channel blockers are often used first-line • cardioversion and digoxin are not useful in the management of MAT

Atrial myxoma Overview • Benign cardiac tumor • the most common primary cardiac tumors in adults. (rhabdomyoma is the most common primary cardiac tumor in pediatric patients and strongly associated with tuberous sclerosis). • 75% occur in left atrium , arising from a pedicle on the fossa ovalis. • more common in females Three-quarters of cases of atrial myxoma occur in females • Although most cases of atrial myxoma are sporadic, an autosomal dominant variety may also exist within families. • 10% are inherited Features • One third present with emboli • One third with systemic inflammation (ESR ↑↑in 1/3) • One third are asymptomatic when detected. • There are 3 groups of manifestations:

1. Obstructive features: like MS, signs vary with posture. Occasionally, there is a lowpitched sound called tumor plop. Dyspnoea, (Exertional dyspnoea is present in three-quarters of patients). Dizziness or syncope results from the atrial myxoma obstructing the mitral valve. Mitral valve obstruction is the most likely complication Myxomas are more likely to have a stalk and be freely mobile. atrial fibrillation mid-diastolic murmur, 'tumour plop' (low-pitched sound) murmur change with posture. Elevated left atrial pressures cause dilatation.
2. Embolic features: either systemic or pulmonary embolism.
3. Constitutional features: such as fever, malaise, weakness, loss of weight, myalgia, arthralgia, clubbing, skin rash, Raynaud’s phenomenon. Investigations • echo: pedunculated heterogeneous mass typically attached to the fossa ovalis region of the interatrial septum

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• on histology gelatinous appearance abundant ground substance. Treatment • surgical removal by median sternotomy. Prognosis • sudden death may occur in 15% of patients. Carney's complex is a familial multiple neoplasia and lentiginosis syndrome, associated with

1. Primary adrenal hypercortisolism
2. Lentigines and naevi of the skin
3. Various tumours including myxoma.

Heart block Types of heart block First-degree heart block • PR interval > 0.2 seconds (> five small squares in the ECG.) • Causes: Increased vagal tone (such as in trained athletes) Ischaemic heart disease Rheumatic fever Hyperkalaemia Hypokalaemia, and Drug therapy such as digoxin or beta-blockers. • A long PR interval on the ECG may also be caused by structural abnormalities such as an atrial septal defect. • No treatment is usually required.

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Second-degree heart block • Type 1 (Mobitz I, Wenckebach): progressive prolongation of the PR interval until a dropped beat occurs Mobitz Type I with symptoms is a relative indication for a permanent pacemaker Asymptomatic NO treatment Discharge him from the clinic The risk of progression to complete heart block with Mobitz type I in an asymptomatic man is very low, unlike in Mobitz type II. • Type 2 (Mobitz II): PR interval is constant, but the P wave is often not followed by a QRS complex the most appropriate next management step Transvenous cardiac pacing Mobitz type II or complete heart block does not respond to atropine. Atropine may be useful for sinus or junctional bradycardia. In patients with Mobitz type II AV block, or complete heart block, a DDD or DDDR pacemaker is indicated. DDD pacemaker will sense and pace both atria and ventricles. DDD pacemakers ensure that the atrial and ventricles beat in synchrony thus preventing pacemaker syndrome. • Second-degree heart block with RBBB implies that this patient has a significantly increased risk of complete heart block. prior to committing to pacemaker insertion, repeat tape is the most likely next step, with an electronic patient diary to see if the recorded arrhythmia corresponds to her symptoms.

Third degree (complete) heart block Third degree (complete) heart block • there is no association between the P waves and QRS complexes • Complete heart block (whether symptomatic or not) is an absolute indication for a permanent pacemaker

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Causes • myocardial ischemia The most common cause of third degree atrioventricular block is myocardial ischemia. Complete heart block is related most to right coronary artery occlusion because this commonly involves both the AV nodal artery and the right superior descending artery.

 Prognosis is favourable, and revascularisation normally leads to restoration of sinus rhythm. As the AV nodal artery arises proximally from the right ventricular artery, distal right coronary artery occlusion is not commonly associated with complete heart block. the artery most likely to be affected Proximal right coronary Left coronary artery occlusion leads to anterior myocardial infarction. As it is less commonly associated with complete heart block, when it does occur, the prognosis is very poor. Third degree atrioventricular block that is resulting from obstruction of the left anterior descending coronary artery is usually irreversible. • Lyme disease • Drugs eg: B. blockers • Congenital third degree atrioventricular block might be due to maternal lupus. Features • Syncope • heart failure • regular bradycardia (30-50 bpm) that does not vary with exercise • wide pulse pressure • JVP: irregular cannon waves in neck • variable intensity of S1 • compensatory increase in stroke volume with a large-volume pulse and systolic flow • The escape rhythm of third-degree atrioventricular block resulting from obstruction of the right coronary artery is usually narrow-complex. • the atrial rhythm is usually regular • The bizarre, wide, inverted T-waves can be seen in Stokes-Adams attacks and do not necessarily imply new ischaemia. ECG showing third degree (complete) heart block

Treatment In patients with Mobitz type II AV block, or complete heart block, a DDD or DDDR pacemaker is indicated • Asymptomatic or mild symptoms (stable) Condition-specific management • Symptomatic (unstable): (syncope, ventricular rate is significantly low (<40 to 45 bpm) or low BP (mean arterial pressure <65 mmHg)) Whilst arrangements are being made for temporary pacing, the options to be considered, prior to temporary transvenous pacing, in this context are:

1. Atropine 0.5-1.0 mg intravenous bolus repeated as required up to 3mg .
2. Isoprenaline, intravenous infusion at 2-10 microg/min. it is a non-selective β agonist that is analog of epinephrine (adrenaline)
3. External cardiac pacing. temporary (transcutaneous or transvenous) pacing Transvenous pacing is much more reliable than transcutaneous pacing • Condition-specific management includes: treating acute coronary syndrome (i.e., antiplatelet medications, urgent revascularisation) medication toxicity (e.g., glucagon for beta-blocker toxicity, calcium for calciumchannel toxicity, or digoxin antibody for digitalis toxicity). • Intravenous aminophylline is useful in complete heart block, as the heart block is often mediated by adenosine which aminophylline inhibits • Post- MI: Following anterior MI pace-maker insertion Following posterior MI and patient is haemodynamically stable observation Often spontaneously resolved

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Pacemakers Definition • A permanent pacemaker is an implanted device that provides electrical stimuli, thereby causing cardiac contraction when intrinsic myocardial electrical activity is inappropriately slow or absent. Conditions definitely needs a permanent pacemaker • Symptomatic bradycardia due to sinus node dysfunction (sick sinus syndrome) • Third-degree heart block • second-degree (AV) block associated with any of the following: symptomatic bradycardia

documented periods of asystole of 3 s or more any escape rate less than 40 bpm in awake, asymptomatic patients type II second-degree AV block and a ventricular rate of 45 bpm when awake and asymptomatic asymptomatic sinus rhythm resulting in periods of asystole longer than 3.0 seconds asystolic pause causing syncope. dual chamber permanent pacemaker (DDDR). The R in this code stands for responsive, and in an otherwise fit and well 76-year-old, he should have a responsive element to his PPM (that is, increases his heart rate with exercise). Type II second-degree AV block has a high chance of progressing to asystole (35%) each year • Generally, permanent pacing can be justified for any degree of heart block associated with symptoms of bradycardia.