

CARDIOLOGY NOTES

CARDIOVASCULAR EXAM

The **second heart sound** comprises of aortic (A2) and pulmonary (P2) component. In LBBB, the aortic closure is delayed because the left ventricle contracts later. This then causes reversed splitting (A2P2 → P2A2) if the second heart sound.

LBBB and left heart strain in HCM and aortic stenosis can cause **reversal of A₂P₂** second heart sounds. Also, in type B wolf parkinson white syndrome, early activation of the right ventricle through an accessory pathway can cause P2 to close prematurely. Patent ductus arteriosus is another cause.

The **third heart sound** is caused by early diastolic filling due to ventricular relaxation, shortly after closure of the aortic valve (corresponds to Y descent in JVP).

It may be normal in children and young /middle aged adults.

Causes of an **abnormal third heart sound** are:

Left ventricular failure

Severe MR and TR

VSD, PDA

Constrictive pericarditis

Hypertrophic cardiomyopathy

Dilated cardiomyopathy

AV fistula

thyrotoxicosis

Causes of an abnormal fourth heart sound are:

Causes of raised JVP are:

Congestive cardiac failure

SVC obstruction

Constrictive pericarditis

Anaemia

Tricuspid regurgitation

Pulsus paradoxus is defined as an inspiratory systolic fall in arterial pressure of 10mmHg. It not only occurs in cardiac tamponade, but also in massive PE, severe COPD and hypotension/shock.

Canon a waves occur when the atria and ventricles contract at the same time. The causes are complete AV block, ventricular tachycardia and AV nodal reentry tachycardia

CARDIAC ANATOMY

The **left internal mammary artery** supplies the anterior chest wall. It has been shown to be superior to saphenous vein grafts (from aorta to LAD) in staying patent and hence is now the choice artery (LIMA to LAD) graft. Although circumflex and right coronary arteries are usually grafted with veins, the right internal mammary arteries (RIMA) are sometimes used to graft the RCA.

The **circumflex artery** gives off obtuse marginal branches and the LAD gives off diagonal branches.

The **intermediate artery** is not always present, it is a variant artery which is between the LAD and circumflex artery, and occasionally dominant instead of the circumflex.

The **coronary sinus** predominantly drains venous blood from the left ventricle and receives approximately 85 percent of coronary venous blood. It receives blood from the the marginal, posterior left ventricular, anterior interventricular veins and the great cardiac vein. The blood finally drains into the right atrium.

The **posterior descending artery** is often (85%) a branch of the right coronary artery.

The **sinus node artery** is a branch of the right coronary artery in 60% of cases.

The **AV node** is supplied by the posterior descending coronary artery.

ARRHYTHMIAS

Atrial flutter most commonly presents with 2:1 block, which means atrial rate of 300 but ventricular rate of 150. Ischaemic chest pain may occur due to the tachycardia. Carotid sinus massage can sometimes terminate or slow the tachycardia.

DC cardioversion should be performed if the patient is cardiovascularly unstable.

Atrial flutter is commoner in patients with dilated left atrium or in congenital/structural heart disease.

Differentiating SVT from VT

Features that favour VT are :

QRS of > 140ms,
cannon a waves on JVP
fusion and/or capture beats
dissociated p waves,
history of ischaemic heart disease,
right bundle branch block with left axis deviation,
concordance of the QRS complexes in the chest leads
HR >170 beats per minute.

In a patient who is stable with **sustained ventricular tachycardia**, the options are intravenous lignocaine, intravenous amiodarone. IV magnesium sulphate (18 / 20 mmols or 5g) is often helpful in helping to cardiovert. If the patient were unstable then he needs to be DC cardioverted immediately (with or without general anaesthetic).

The criteria for **ICD insertion** are:

1) patients with LVEF <40% with non sustained VT

- 2) patients with sustained VT
- 3) patients who have had any VT or VF leading to cardiac arrest
- 4) cardiomyopathy and ventricular arrhythmias
- 5) patients with previous MI and ejection fraction <30%

The MADIT II trial showed that in patients with a previous MI and reduced left ventricular ejection fraction (<30%), the prophylactic use of an ICD, in addition to medications, significantly reduced the risk of death.

The first form of second degree **heart block**, **Mobitz** type I (Wenkebach) is due to progressive prolongation of PR interval and then missing a beat. **Mobitz** type II second degree heart block can occur with 2:1 (only 1 QRS is conducted for 2 p waves) or 3:1.

RBBB (not LBBB) with left anterior hemiblock (or left axis deviation) is called **bifascicular block**. If first degree heart block was also present, then it is known as trifascicular block.

PROLONGED QT

A QT interval of >0.45 is prolonged.

DRUGS causing PROLONGED QT

tricyclic antidepressants (eg. amitriptyline)
 quinidine,
 erythromycin,
 amiodarone,
 phenothiazines (chlorpromazine),
 antihistamines (terfenadine)
 grapefruit juice
 sotalol

METABOLIC causes of PROLONGED QT

Hypokalaemia
 Hypocalcaemia
 Hypomagnesaemia
 Hypothermia
 Hypothyroidism

A **dominant R in lead V1 on the ECG** is associated with :

- primary pulmonary HT
- Right bundle branch block (RBBB) (including Ebstein's anomaly)
- Wolf-Parkinson-White syndrome Type A
- Dextrocardia
- Posterior MI
- Duchene muscular dystrophy

MYOCARDIAL INFARCTION

Absolute contraindications to thrombolysis include:

previous haemorrhagic stroke

ischaemic CVA within 1 year
suspected aortic dissection
active bleeding <10 days
BP>180/110
neurosurgical procedure < 6 months ago

Relative contraindications include
major surgery or bleeding within 6 weeks
pregnancy
known bleeding diathesis
severe uncontrolled hypertension
Proliferative diabetic retinopathy

Troponins tend to be elevated for up to 14 days. **CK-MB** comes down to normal level within 48-72 hours, and is the most specific of the CK enzymes. Pulmonary embolus, arrhythmias, myocarditis and right heart disorders are also known to elevate **troponins**.

HYPERLIPIDAEMIAS

The characteristics of **familial hypercholesterolaemia** are:
autosomal dominant condition
increased LDL concentrations
reduced HDL concentrations
reduced numbers of LDL receptor
cardiovascular disease
tendon xanthomas

In **familial hypercholesterolaemia** characteristically total cholesterol is > 7.5mmol/L, LDL-cholesterol is > 5.0 mmol/L and triglyceride is < 2.3 mmol/L. It is due to an LDL receptor defect.

THE PRIMARY HYPERLIPIDAEMIAS: THE FREDERICKSON CLASSIFICATION

Type II hyperlipidaemia the most common primary hyperlipidaemia. The picture is similar to familial hypercholesterolaemia but milder. It is characterised by increased levels of LDL-cholesterol (> 4.0 mmol/L). Triglyceride levels are < 2.3 mmol/L.

Type IIa hypercholesterolaemia causes heart disease as there is predominantly *raised cholesterol and high LDL*. There is also increased triglycerides cause eruptive skin xanthomas, lipaemia retinalis (white TG deposits in the retina) and pancreatitis. Cholesterol has an affinity for deposition around the tendons, tendon xanthoma is characteristic of hypercholesterolaemia.

Type II b hypercholesterolaemia also causes elevated cholesterol and triglycerides.

Type III hyperlipidemia is when cholesterol and triglyceride are both increased, and is associated with atherogenesis. Homozygosity for the E2 genotype (E2/E2) is found in most patients with **type III hyperlipidemia**. The palmar striae (palmar xanthomata) are considered pathognomonic for the disorder and occur in less than 50% of patients but tubero-eruptive xanthomata, typically on the elbows and knees, as

well as xanthelasma have been described. The underlying biochemical defect is one of a reduced clearance of chylomicron and VLDL remnants.

Type IV hyperlipidemia causes an isolated hypertriglyceridaemia. There is normal or slightly raised plasma cholesterol. There are increased triglyceride levels due to increased VLDLs

Type I and V hyperlipidaemia have lipoprotein lipase defects, and lead to raised chylomicrons and triglyceride levels.

Eruptive xanthomas in the skin are typical of triglyceridaemia. Alcohol abuse, pancreatitis and hypertriglyceridaemia are associated. Both *type I* and *type IV* are triglyceridaemic states. Triglycerides cause turbid serum.

List of causes of raised **triglycerides** are:

- nephrotic syndrome
- hypothyroidism
- biliary obstruction
- steroids
- diabetes mellitus
- renal failure
- thiazide diuretics
- oral contraceptive pill
- lipodystrophies
- and glycogen storage disease (Von Gierke's)

Chylomicrons are triglyceride-rich lipoproteins (75%-95% of their core lipid is triglyceride) made in the small intestine from **ApoB-48** (apolipoprotein).

VLDL are made in the liver from ApoB-100.

Ventricular septal defect (VSD) is the commonest form of congenital heart disease. *Maladie de Roger* is a loud systolic murmur despite a small VSD. Antibiotic prophylaxis should be used at all times. The systolic murmur does become softer as right heart pressures increase and a right to left shunt may develop in Eisenmenger's syndrome (leading to cyanosis).

PERICARDIAL DISEASE

Acute Pericarditis causes: saddle shaped ST elevation on ECG, raised Troponin
Coxsackie B is the commonest cause. Other causes are HIV, diphtheria, Chagas disease, Lyme disease, SLE and arsenic poisoning.

In **constrictive pericarditis**, because the atria are equilibrated with the ventricles in early diastole, the jugular venous waveform and right and left atrial waveforms show a prominent and *deep y descent*. The systolic *x descent* is usually also present. The main feature in **chronic constrictive pericarditis** is restriction of diastolic filling of ventricles. This may lead to the pericardial knock during ventricular filling. Ascites can occur due to chronic venous congestion and liver cirrhosis. Poor ventricular filling can lead to a low pulse pressure.

Summary signs of constrictive pericarditis are:

- rapid x and y descent
- ascites
- hepatomegaly
- ankle oedema
- pulsus paradoxus can occur but is less common especially in chronic constrictive pericarditis

Cardiac tamponade

This pattern of venous return in chronic constrictive pericarditis contrasts with that in **cardiac tamponade**, in which the venous pressure tracing shows absence of diastolic y descent (i.e. there is a deep x descent but y descent is absent) . Cardiac tamponade also causes a raised JVP, Kussmaul's sign and pulsus paradoxus.

Summary signs of cardiac tamponade are:

- rapid x descent
- Kussmaul's sign
- pulsus paradoxus

CARDIOMYOPATHY

In **hypertrophic cardiomyopathy (HCM)**, there is decreased compliance due to the disorientation of myosin fibres. 40% of patients carry mutations of cardiac beta Myosin heavy chain on chromosome 14.

Manoeuvres which decrease venous return will increase the intensity of the murmur (Valsalva – baroreceptor mediated fall in cardiac output, standing, nitrates).

Manoeuvres which increase venous return will decrease the murmur as this overcomes the obstruction (squatting, long term beta blockers).

In summary, the murmur in HCM:

increases in Valsalva Manoeuvre
increases with standing
decreases with exercise
decreases with squatting

Nitrates are best avoided because they are vasodilators which may cause hypotension with the condition. *Beta blockers and calcium channel blockers* help increase end diastolic filling and treat symptoms but have no effect on risk reduction of sudden death. Amiodarone improves survival.

Restrictive cardiomyopathy: Echocardiography showing no pericardial effusion and stiffness suggests restrictive rather than constrictive cardiomyopathy. The transmitral dopplers on the echo may show E/A wave reversal and high velocities which may suggest restrictive picture. This may be due to infiltration due to haemochromatosis, endomyocardial fibrosis, sarcoidosis, myeloma, lymphoma or connective tissue disease.

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Over a hundred different mutations in at least nine different genes, all encoding sarcomeric polypeptides, have been associated with **hypertrophic cardiomyopathy** (HCM).

ENDOCARDITIS

Osler's nodes, which are small reddish tender areas on the pulp of fingers or toes. **Janeway lesions** are reddish spots on fingers or toes, but are painless.

A definite diagnosis of endocarditis (**Duke's criteria**) is achieved when *2 major criteria* are present, or *1 major and 3 minor criterias*.

Major criteria: blood culture positive for typical organisms
persistent bacteremia
positive ECHO for vegetations
abscess or valve dehiscence

Minor criteria: valvular heart disease or IV drug user
fever greater than 38°C
vasculitis
skin lesions
suggestive ECHO (but not definite)
positive blood culture

In a patient with *prosthetic valve*, **staph epidermidis** (one of the coagulase negative staph) and **Strep viridans** are most common. Strep bovis, and enterococcus are the other organisms which commonly cause endocarditis.

One of the major dangers with aortic valve endocarditis is an *aortic root abscess*. This can lead to prolonging of the PR interval by erosion into the adjacent AV node, hence daily ECGs are useful for monitoring.

Marantic endocarditis: this disorder results in the deposition of fibrin on the valve leaflets on either side of the heart, but especially the left side.

The vegetations are sterile and small (1-5 mm). This form of noninfective endocarditis can be seen in persons who are very debilitated or in those who have a hypercoagulable state.

Major criteria for **Rheumatic fever** are:

Chorea
carditis
polyarthritis
erythema marginatum
nodules (subcutaneous)

Minor criteria are:

fever
arthralgia
raised inflammatory markers
previous rheumatic fever
prolonged PR on ECG

2 major or 1 major and 2 minor satisfy criteria for Rheumatic Fever.

The **HACEK** organisms are:

Haemophilus species (H. aphrophilus, H. paraphrophilus, H. parainfluenzae)
Actinobacillus actinomycetemcomitans
Cardiobacterium hominis
Eikenella corrodens
Kingella

These are *gram negative bacilli* which are responsible for 5-10% of cases of infective endocarditis (IE) involving native valves and are the most common cause of gram-negative endocarditis among persons who do not abuse intravenous drugs.

HYPERTENSION

Treatment is recommended for a blood pressure of > 160/100 in patients without end organ damage, by the **British Hypertension Society**.

The British Hypertensive Society guidelines suggests either a Diuretic(D) or Calcium channel blocker(C) for an older or black patient

Malignant hypertension can present with pulmonary oedema, chest pain, headache, visual disturbance and also seizures. A fundoscopic examination may reveal flame-shaped retinal hemorrhages, soft exudates, or papilledema.

Sodium Nitroprusside has an immediate onset of action and short half-life. It acts by causing relaxation of vascular smooth muscle, resulting in vasodilation and inotropy. The dose is 0.25-10 mcg/kg/min IV. IV hydralazine, beta blockers and calcium channel blockers can also be used.

Methyldopa is the safest agent to use in the first and second trimester of **pregnancy**. Beta blockers may cause intrauterine growth retardation. ACE inhibitors have also been suggested to have teratogenic effects.

Acute hypertensive crisis of phaeochromocytoma : IV administration of *sodium nitroprusside*, *nitroglycerine*, or *phentolamine* can be used.

Preoperatively, phenoxybenzamine preoperative adrenergic-blockade of α_1 and α_2 receptors with phenoxybenzamine (10-30 mg twice daily), or α_1 receptors with prazosin (starting with 1 to 2 mg three times daily).

Beta blockers can be useful for arrhythmias, but should not be commenced before alpha blockers because β -blockade alone can cause marked hypertension.

Significant outflow tract obstruction, advanced heart block, hypertension > 180 mmHg systolic, and unstable angina are all contraindications towards **Exercise Testing**.

DRUGS

Digoxin toxicity can occur especially with renal impairment. It typically causes nausea & vomiting. ST depression occurs along with bradycardia on the ECG. The patient may also get xanthopsia (yellow vision).

Aspirin blocks the synthesis of COX1 and COX2 enzymes. This leads to a reduction in the prostaglandin PGG₂, PGH₂ \rightarrow thromboxane TXA₂ synthesis \downarrow . This leads to \downarrow platelet aggregation. Aspirin does reduce urate secretion in the renal tubules and can increase risk of gout.

Brain or B type natriuretic peptide is increasingly used as a marker of congestive heart failure. It has high specificity for the condition. It is released by ventricular cardiomyocytes due to stretch as part of the pathophysiology of cardiac failure.

GIIB/IIIA inhibitors such as Reopro or Tirofiban have a treatment role in NSTEMI

Heart failure trials

Bisoprolol (CIBIS II trial), spironolactone (RALES trial), enalapril (CONSENSUS) and nitrates and hydralazine (V-HEFT) have all been shown to improve mortality in chronic heart failure.

Flecainide is a class Ic antiarrhythmic drug. It helps restoration of sinus rhythm and prevent recurrence of AF. Flecainide may prolong QT interval. The drug was used in the Cardiac Arrhythmia Suppression Trial (CAST) trial, and was associated with increased mortality in patients with ischaemic heart disease.

Sotalol has class II (beta-blocker) and class III antiarrhythmic actions.

Nifedipine is a dihydropyridine. The mechanism of action is smooth muscle relaxation and reduction in peripheral vascular resistance.

MISCELLANEOUS

Aortic Dissection

Hypertension should be treated with an infusion such as labetalol. Aortic regurgitation and pericardial effusion (haemopericardium) suggest dissection down to the aortic root. These features, or persisting chest pain suggest that the patient requires prompt surgery.

Aortic dissection can present with pain radiating to the epigastric region or back. If the dissection flap involves close to the aortic root, then the coronary flow can be affected, causing either inferior MI in right coronary artery or anterior MI in left anterior descending artery involvement. Cardiac tamponade can also be caused by dissection (blood flowing into pericardial space) and leading to a raised JVP and pulsus paradoxus.

Dissection of the aorta is associated with hypertension, cocaine abuse, trauma, pregnancy, aortitis as seen in syphilis, Marfan's, Ehlers-Danlos and other connective tissue disorders.

A **bicuspid aortic valve** may be present in up to 1% of the population. It is more common in men. 5% of bicuspid aortic valves are associated with coarctation of the aorta.

The clinical signs consistent with **severe aortic stenosis** are:

Slow rising pulse character

4th heart sound

Soft aortic 2nd heart sound

Systolic thrill

Extended length of murmur in systole

Causes of **aortic regurgitation** are bicuspid aortic valve, calcific degeneration, William's syndrome (elfin facies, congenital heart disease, cognitive impairment), endocarditis, seronegative spondyloarthritides, connective tissue diseases (Marfan's, osteogenesis imperfecta), aortitis.

Coarctation of the aorta is associated with Turner's syndrome, neurofibromatosis, bicuspid aortic valve, and more weakly with VSD and ASD. Stroke may result from hypertension or from associated berry aneurysms. It is more common in males. Notching of ribs is seen, typically between 3 to 8.

With **ASD**, a Primum defect causes RBBB and LAD, whilst Secundum causes RBBB and RAD on the ECG. Secundum ASD is associated with Holt-Oram syndrome (tri-phalangeal thumb and radial abnormalities).

Brugada Syndrome is an autosomal dominant disease with variable expression. In approximately 20% of cases, the underlying cause of Brugada syndrome is a genetic defect in the *SCN5A* gene, which encodes the sodium channel.

Brugada Syndrome manifests with syncope and cardiac arrest, typically occurring in the third and fourth decade of life. The syndrome is characterised by a typical electrocardiogram (ECG) pattern of ST segment elevation in leads V1 to V3, and incomplete or complete right bundle branch block. ICD is indicated in patients with symptoms suggestive of, or evidence of arrhythmias.

An **Atrial myxoma** can present with fevers, raised inflammatory markers and breathlessness worst lying down. There may be embolic causes of CVA or TIA. The murmur could be a diastolic 'plop' or there may be clinical signs similar to mitral stenosis.

Mitral valve prolapse is associated with a risk of endocarditis, atrial septal defect and the long QT syndrome. Squatting increases afterload and this leads to an increase in the intensity of the murmur.

Mitral valve prolapse is commonly found in patients with Marfan syndrome, Ehlers-Danlos syndrome, and adult polycystic kidney disease, and occasionally hypertrophic cardiomyopathy.

Carcinoid syndrome. The carcinoid plaque is the characteristic lesion in carcinoid heart disease. Mediator (serotonin, 5-hydroxytryptophan, histamine) release into the hepatic vein from metastatic liver disease predisposes patients to right-sided cardiac lesions.

The *pulmonary valve* shows thickening and retraction. The most important consequence of carcinoid plaques is fibrosis of the valves with resultant distortion of the valvular apparatus and ring. *Tricuspid regurgitation and pulmonic stenosis* are typically seen.

In **Ebstein's anomaly**, there is congenital malpositioning of the tricuspid valve - the posterior and septal leaflets are displaced into the right ventricular cavity. Tricuspid regurgitation and enlarged right atrium is seen. Ebstein's anomaly is associated with maternal lithium use during pregnancy

Flight Rules:

A patient should be fit to **fly** 14 days after an uncomplicated **myocardial infarct**, after normal activities have been resumed. For complicated MIs, the patient has to wait until symptoms stabilise.

Patients are only allowed to **fly** once resolution of **pneumothorax** has been demonstrated radiographically and 6 weeks after the event

Tricuspid atresia may be defined as congenital absence or agenesis of the tricuspid valve. It is the third most common cyanotic congenital heart defect; the other two more frequently observed cyanotic congenital cardiac anomalies are transposition of the great arteries and tetralogy of Fallot. Tricuspid atresia is the most common cause of cyanosis with left ventricular hypertrophy.

Electrocardiographic findings are virtually diagnostic of tricuspid atresia in a young patient with congenital heart disease. The electrocardiogram demonstrates right atrial hypertrophy, an abnormal superiorly oriented major QRS vector, the so-called left axis deviation.