036 - Pages 876-900

- 036

036

Pages 876-900

Indications for a temporary pacemaker • symptomatic/haemodynamically unstable bradycardia, not responding to atropine • post-ANTERIOR MI: type 2 or complete heart block post-INFERIOR MI complete heart block is common and can be managed conservatively if asymptomatic and haemodynamically stable • trifascicular block prior to surgery • Other indications for transvenous pacing in setting of acute MI are: asystole new bundle branch block (BBB) with first-degree heart block an old right BBB with first degree atrioventricular (AV) block and a new fascicular block Notes • All modern ICDs also function as pacemakers. • Chest pain in Ventricular pacing Pacemaker rhythm may prevent interpretation of ST-segment changes and may require urgent angiography to confirm diagnosis. Reprogramming the pacemaker—allowing an evaluation of ECG changes during intrinsic heart rhythm—may be considered in patients who are not dependent on ventricular pacing, without delaying invasive investigation Types of Pacemakers • Pacemakers are classified by the nature of their pacing mode using a code of up to five letters. • The NBG Pacemaker code was developed by the North American Society of Pacing and Electrophysiology (NASPE) and the British Pacing and Electrophysiology Group (BPEG): I II III IV V Chamber(s) Paced Chamber(s) Sensed Mode(s) of Response O = None O = None O = None O = None O = None A = Atrium A = Atrium T = Triggered R = Rate modulation V = Ventricle V = Ventricle I = Inhibited V = Ventricle D = Dual (A+V) D = Dual (A+V) D = Dual (T+I) D = Dual (A+V) Single-chamber pacemakers • utilised for patients in permanent atrial fibrillation. • VVI means there is one lead in the ventricle (pacing and sensing the ventricle, indicated by the ‘VV’). • VVI pacemaker will pace and sense the right ventricle. • VVI pacemaker is useful when we are not too concerned about atrial activity (e.g. in patients with atrial fibrillation). • In the presence or organised atrial activity, a VVI pacemaker may pace the ventricles out of synch with the atria resulting in pacemaker syndrome. Since organised atrial activity is present, a DDI pacemaker would be preferred, as this senses and paces both atria and ventricle to preserve synchrony. Notes & Notes for MRCP
By Dr. Yousif Abdallah Hamad

Rate Modulation Multisite Pacing A = Atrium

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

Cardiology

Dual-chamber pacemakers • Have pacing electrodes in both the right atrium and the right ventricle. • They allow maintenance of the physiological relationship between atrial and ventricular contraction and also allow the paced heart to follow the increase in sinus rate that occurs during

exercise. Biventricular pacemakers • Pacemaker leads are placed in the right atrium, right ventricle and left ventricle. • Useful in the management of patients with heart failure who have evidence of abnormal intraventricular conduction (most often evident as left bundle branch block (LBBB) on ECG) which causes deranged ventricular contraction or dyssynchrony. • In a patient with severe ischaemic heart failure and is on optimal medical therapy. Despite this he is still symptomatic ICD with biventricular pacing very prolonged QRS duration is indicating left dyssynchrony which is an indication for biventricular pacing according to NICE guidance. Documented VT in the context of ischaemic LV impairment necessitates the need for and a secondary prevention ICD. Pacemaker complications • Pacemaker complications are more common in the period following insertion. • can be divided into early complications (<6 weeks) or late (>6 weeks). • Most frequent complications are those related to implantation procedure, such as lead dislodgement and pneumothorax. • pneumothorax can occur up to forty-eight hours following pacemaker insertion. It occurs in 1-2% of procedures and most patients will require chest drain insertion. • The most common complication is lead dislodgement (higher rate atrial dislodgment than ventricular dislodgment). • Lead dislodgement can occur following trauma or sporadically and can be either atrial or ventricular. • Atrial dislodgment affects up to 3% of people whereas ventricular is less common affecting 1%. • If the ECG shows loss of sensing and capture around the QRS complex ventricular lead displacement in a dual chamber pacemaker. What would be the likely ECG findings in ventricular lead displacement? Loss of sensing and capture of the QRS complex • Atrial lead displacement would show an ECG with loss of atrial sensing and capture. The ECG in atrial lead displacement would show an ECG with loss of atrial sensing and capture in a dual chamber or single chamber pacemaker. • On occasion lead displacement can be seen on chest X-Ray, however, it may not be seen, in this case a lateral chest X-Ray may be of use in this scenario. • Pacemaker syndrome would show AV dyssynchronisation. • Subclavian vein obstruction is a fairly common complication over time but many patients may remain asymptomatic due to collateral vein formation. It can present with symptoms of superior vena cava (SVC) obstruction in severe cases. • Twiddler's syndrome is when the patient intentionally or accidentally turns the pacemaker on its longitudinal axis which can cause lead dislodgement. • Reel's syndrome is Twiddler's syndrome but on the horizontal axis. • Pacemaker lead fracture occurs in 1-4% of pacemakers usually following excessive exercise or direct trauma. patient will require lead extraction and replacement. • myocardial rupture: incidence is relatively small (<1%)

Notes & Notes for MRCP
By Dr. Yousif Abdallah Hamad

 can be divided into early or late rupture with respect to the time it occurs following procedure. Delayed perforations are less likely to cause such acute symptoms as well as a reduced incidence of tamponade and sudden cardiac death. Risk factors for perforation include physician technique, patient independent factor (i.e obesity or difficult anatomy) and lead design. presenting features :pericardial effusion, haemodynamically compromised following pacemaker insertion and is likely to develop cardiac tamponade and needs urgent intervention with pericardiocentesis.

Pacemaker syndrome Pacemaker syndrome • Loss of AV synchrony. • Retrograde VA conduction. • Absence of rate response to physiological need. Pacemaker syndrome (breathlessness associated with ventricular pacing in the context of normal atrial activity). VVI pacemaker will pace and sense

the right ventricle. In the presence or organised atrial activity, a VVI pacemaker may pace the ventricles out of sync with the atria resulting in pacemaker syndrome. Overview • pacemaker syndrome is related to nonphysiologic timing of atrial and ventricular contractions, which may occur in a variety of pacing modes • also named as "AV dyssynchrony syndrome," • typically associated with a VVI pacemaker that results in simultaneous atria and ventricle conduction. Risk factors • Sick sinus syndrome as have preserved AV conduction. • Single-chamber ventricular pacing. Features • Hypotension, tachycardia, tachypnoea, dizziness, syncope • Ventricular contraction against closed tricuspid and mitral valves can result in raised JVP (pulsation and fullness in the neck) cannon waves Complications of AV dyssynchrony: • Atrial fibrillation, thromboembolic events, and heart failure. What are the characteristic ECG findings associated with this syndrome? □ Small P waves with dissociation from QRS complex Management • In patients with other pacing modes, upgrading the pacemaker to a dual-chamber pacing or reprogramming the pacemaker parameters - eg, AV delay, post-ventricular atrial refractory period, sensing level, and pacing threshold voltage. DC cardioversion in patients with pacemakers (eg : in AF) • DC cardioversion is not contraindicated in patients with pacemakers • Pacemaker function should be checked after cardioversion and antiarrhythmic therapy added

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

Cardiology

Brugada syndrome Overview • Inherited cardiovascular disease with may present with sudden cardiac death. • Prevalence →1:5,000-10,000. • More common in Asians. • Autosomal dominant • A large number of variants exist • Around 20-40% of cases are caused by a mutation in the SCN5A gene which encodes the myocardial sodium ion channel protein ECG changes • Convex ST segment elevation > 2mm in > 1 of V1-V3 followed by a negative T wave • Partial right bundle branch block • Changes may be more apparent following flecainide ECG showing Brugada pattern, most marked in V1, which has an incomplete RBBB, a downsloping ST segment and an inverted T wave Management • implantable cardioverter-defibrillator

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Catecholaminergic polymorphic ventricular tachycardia (CPVT) Overview • CPVT is a form of inherited cardiac disease associated with sudden cardiac death. • Inherited in an autosomal dominant fashion • Prevalence of around 1:10,000. Pathophysiology • the most common cause is a defect in the ryanodine receptor (RYR2) which is found in the myocardial sarcoplasmic reticulum • uncontrolled calcium release from the sarcoplasmic reticulum • induced by adrenergic stress.

Notes & Notes for MRCP
By Dr. Yousif Abdallah Hamad

Features • exercise or emotion induced polymorphic ventricular tachycardia resulting in syncope • sudden cardiac death • symptoms generally develop before the age of 20 years Management • beta-blockers • There is strong evidence that flecainide is effective when prescribed in addition to beta blockers • implantable cardioverter-defibrillator • Left cervical sympathetic denervation • All first-degree relatives should be evaluated with ECG, Holter monitoring and exercise stress testing.

Ventricular tachycardia Definition • wide QRS complex (duration >120 milliseconds) at a rate greater than 100 bpm, originating from a ventricular ectopic focus. Whilst a broad complex tachycardia may result from a supraventricular rhythm with aberrant conduction, the European Resuscitation Council advise that in a peri-arrest situation it is assumed to be ventricular in origin. • It has the potential to precipitate ventricular fibrillation and hence requires urgent treatment. Pathophysiology • Among patients with prior MI or non-ischaemic cardiomyopathy, VT is usually due to reentry involving regions of slowed conduction adjacent to scar. Post MI ventricular tachycardia (VT) is most commonly due to scar tissue. The definitive investigation would be Electrophysiological study (EPS) due to the fact that if this were scar related VT, the site could be localised and even possibly ablated. If not, then an implantable cardiac defibrillator (ICD) implantation may be warranted if left ventricular (LV) dysfunction exists. MADIT-2 trial showed a 5.6% 20-month absolute survival benefit in patients with LV dysfunction (EF<30%), post MI, treated prophylactically with an ICD. • (VT) may also arise from triggered activity due to early after-depolarisations (EADs) leading to torsades de pointes, a polymorphic ventricular tachycardia seen in the setting of a prolonged QT interval, • delayed after-depolarisations (DADs), which are seen in: idiopathic right ventricular outflow tract VT or catecholaminergic polymorphic VT cellular abnormalities of calcium handling Increased intracellular calcium predispose to VT. especially during periods of sympathetic stimulation. • EADs occur during phase 2 or 3 of the action potential, whereas DADs occur during phase 4. • When an EAD or DAD reaches a 'threshold' potential, it can result in triggering of another action potential. • Ventricular tachycardia originates below the bundle of His.

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

Cardiology

Types: There are two main types of VT: • Monomorphic VT organised, single-morphology QRS arising from one of the ventricles. most commonly caused by myocardial infarction • Polymorphic VT multiple different wide QRS morphologies arising from one of the ventricles. results from abnormal myocardial repolarization. A subtype of polymorphic VT is torsades de pointes which is precipitated by prolongation of the QT interval. The causes of a long QT interval are listed below. Other classifications of VT • Sustained VT A ventricular rhythm faster than 100 bpm lasting at least 30 seconds or requiring termination due to haemodynamic instability. almost always symptomatic. • Non-sustained VT A ventricular rhythm faster than 100 bpm lasting for at least 3 consecutive beats but terminating spontaneously in less than 30 seconds, and not resulting in significant haemodynamic instability. If these do not cause any haemodynamic compromise, treatment is not needed. The most appropriate next step Check potassium and magnesium levels During the GISSI-2 trial it was observed that a serum K+ level of <3.6 mmol/l was associated with a twofold increased risk of VF. Therefore serum K+ should be maintained >4 mmol/l by oral or intravenous (IV) supplementation in patients with acute MI. Concomitant magnesium (Mg2+) deficiency is present in many patients with hypokalaemia and also makes correction of hypokalaemia difficult. Hence serum Mg2+ levels should also be checked and maintained >1 mmol/l. Feature • Patients may have a normal cardiac output or may be haemodynamically compromised • Sustained VT is usually observed in ischaemic cardiomyopathy,

but idiopathic VT may also be observed in patients without structural heart disease. • jugular veins may show cannon A waves due to atrioventricular dissociation.

Notes & Notes for MRCP
By Dr. Yousif Abdallah Hamad

Differential diagnosis • Supraventricular tachycardia with bundle-branch block may resemble ventricular tachycardia on the ECG 80% of all broad complex tachycardias are secondary to VT and the proportion is even higher in patients with structural heart disease. In all cases of doubt, the rhythm should be treated as a VT. the safest course of action is to consider a drug like adenosine, which will cause short-lived AV block in SVT but not in VT. It is the presence of aberrant conduction which can lead to diagnostic confusion. Amiodarone may be an appropriate next step for cardioversion, once the underlying rhythm has been elucidated. • Features suggesting VT rather than SVT with aberrant conduction AV dissociation fusion or capture beats positive QRS concordance in chest leads ((same polarity QRS direction in all chest leads V1 -V6) (Absence of an RS complex in all pre-cordial leads, i.e., all the leads are concordant) marked left axis deviation history of IHD lack of response to adenosine or carotid sinus massage very broad QRS > 160 ms bifid upright QRS with a taller first peak in V1 deep S wave in V6 Capture beats • intermittent narrow QRS complex owing to normal ventricular activation via the AV node • occurs when a supraventricular and a ventricular impulse coincide to produce a hybrid complex. • It indicates that there are two foci of pacemaker cells firing simultaneously: a supraventricular pacemaker (e.g. the sinus node) and a competing ventricular pacemaker (source of ventricular ectopics). • Causes: Ventricular tachycardia Accelerated idioventricular rhythm (AIVR) fusion beats (intermediate between ventricular tachycardia beat and capture beat) are seen

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

Cardiology

Management VT: cardioversion treatment VT with pulse (not respond to medical treatment) →LOW ENERGY synchronized cardioversion Synchronization avoids the delivery of a LOW ENERGY shock during cardiac repolarization (t-wave). If the shock occurs on the t-wave (during repolarization), there is a high likelihood that the shock can precipitate Ventricular Fibrillation (VF). Pulseless VT or VF HIGH ENERGY asynchronized cardioversion • If the patient has adverse signs (systolic BP < 90 mmHg, chest pain, heart failure or rate

> 150 beats/min) then immediate cardioversion is indicated. anaesthetist needs to be called to assist with direct current cardioversion (DCCV) which should be 'synchronised' to limit the risk of conversion to VF. usually at a starting energy dose of 100 J (monophasic; comparable biphasic recommendations are not currently available). If deteriorate in the meantime and become pulseless, then a precordial thump should be given, followed immediately by DCCV if not successful. In cases of pulseless VT, the electrical cardioversion should be

> unsynchronized. Amiodarone is the drug of choice for acute VT refractory to cardioversion shock. Unstable polymorphic VT is treated with immediate defibrillation. The defibrillator may have difficulty recognizing the varying QRS complexes; therefore, synchronization of shocks may not occur. • In stable patients (absence of adverse signs): stable patients stable patients with monomorphic VT and normal LV function, If LV function is impaired, amiodarone (or lidocaine) is preferred to procainamide for pharmacologic conversion because of the latter drug's potential for exacerbating heart failure. restoration of sinus rhythm is typically achieved with IV procainamide, amiodarone, or sotalol. If LV function is impaired, amiodarone (or lidocaine) is preferred to procainamide for pharmacologic conversion because of the latter drug's potential for exacerbating heart failure. In the absence of such signs antiarrhythmics may be used. If these fail, then electrical cardioversion may be needed with synchronised DC shocks If medical therapy is unsuccessful, synchronized cardioversion (50-200 J monophasic) following sedation is appropriate. prophylactic implantable cardioverter defibrillator implantation is recommended in high-risk patients.

Notes & Notes for MRCP
By Dr. Yousif Abdallah Hamad

• Polymorphic VT in stable patients typically terminates on its own. Unsynchronized Synchronized When to deliver electricity At any point in cycle Not during the T-wave Indications V-fib, pulseless VT Everything except V-fib and pulseless VT Drug therapy Verapamil is contra-indicated in VT because it can cause a catastrophic fall in blood pressure. • Amiodarone: ideally administered through a central line (i.e. given after the third shock). If amiodarone is not available lidocaine is a suitable alternative. • Lidocaine: use with caution in severe left ventricular impairment • Procainamide • Adenosine is useful diagnostically when the diagnosis of regular wide complex tachycardia is in doubt. • Verapamil should NOT be used in VT Sotalol is recommended as the first-choice drug to prevent a recurrence of ventricular tachycardia (VT) If drug therapy fails • electrophysiological study (EPS) • implant able cardioverter-defibrillator (ICD) - this is particularly indicated in patients with significantly impaired LV function C.V Resuscitation: • Guidelines from the Resuscitation Council (UK) state that if a patient has a monitored and witnessed VF/VT arrest in hospital, three quick successive (stacked) shocks should be given. Chest compressions should be started immediately after the third, with a compression to ventilation ratio of 30:2 for 2 minutes. • A precordial thump can be successful if given within seconds of the onset of a shockable rhythm. Delivery should not delay calling for help, or accessing a defibrillator, but would be indicated here whilst awaiting the defibrillator. Chest compressions should start immediately if it is unsuccessful. • Intravenous adrenaline would be given every 3-5 minutes once chest compressions had started.

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

Cardiology

QT interval • Definition QT measured from the start of the QRS complex to the end of the T wave represents the duration of activation and recovery of the ventricular myocardium • Normal duration should be between 0.33 and 0.44 seconds • Corrected QT interval (QTc) is calculated by dividing the QT interval by the square root of the preceding R - R interval. Normal = 0.42 s.

Long QT syndrome Definition • Long QT syndrome (LQTS) is an inherited condition associated with delayed repolarization of the ventricles. • A normal corrected QT interval is less than 430 ms in males and 450 ms (0.45 s) in females. One large box represents 200 ms , one small box represents 40 ms Mechanism • the usual mechanism by which drugs prolong the QT interval is blockage of potassium channels delayed repolarization of the ventricles. • Most drugs that prolong the QTc interval act by blocking hERG-encoded potassium channels, although some drugs modify sodium channels. • The most common variants of LQTS (LQT1 & LQT2) are caused by defects in the alpha subunit of the slow delayed rectifier potassium channel. Epidemiology • more common in females. Classification LQT1 LQT2 LQT3 Gene KCNQ1 KCNQH2/ hERG SCN5A Iron Ks (redifier potassium current, slow component) Pathophysiology Decreased potassium excessive sodium inward current Triger of arrhythmia outward current Exercise stress Emotional stress Rest Occurrence

> 50% 34 – 40% 10 – 15% Notes & Notes for MRCP
> By Dr. Yousif Abdallah Hamad

Kr (redifier potassium current, rapid component) Na Decreased potassium outward current

Chapter 5

Cardiology

Causes of a prolonged QT interval Antiarrhythmics Antihistamines Antiinfectives Amiodarone Disopyramide Dofetilide Astemizole Terfenadine Ibutilide Procainamide Quinidine Sotalol Antipsychotic s Gastro-intestinal drugs Opiate agonists Chlorpromazine Haloperidol Mesoridazine Pimozide Thioridazone Cisapride* Domperidone tricyclic antidepressant s, fluoxetine Arsenic trioxide Bepridil Droperidol Probuco Congenital Other conditions Jervell-LangeNielsen syndrome (includes deafness and is due to an abnormal potassium channel) Romano-Ward syndrome (no deafness) Electrolytes: hypocalcaemia hypokalaemia hypomagnesaemia acute myocardial infarction myocarditis hypothermia subarachnoid haemorrhage • *Cisapride have been withdrawn worldwide due to risk of QT prolongation • Jervell-Lange-Nielsen syndrome: includes deafness and is due to an abnormal potassium channel autosomal recessive caused by Mutations in the KCNE1 and KCNQ1 genes Mutations in the KCNE1 and KCNQ1 genes abnormal potassium channel abnormal functions of inner ear structures and cardiac muscle. • Romano-Ward syndrome: congenital long QT syndrome autosomal dominant involves only cardiac (no deafness) • The human ether-à-go-go related gene (hERG) is the gene affected by drugs that lengthen QT interval inadvertently; erythromycin, terfenadine, and ketoconazole. Notes & Notes for MRCP

Antimalarials Clarithromyci n Erythromycin Pentamidine Sparfloxacin Chloroquine Halofantrine Other drugs Levomethady l Methadone

• a non-sedating antihistamine are classic cause of prolonged QT in a patient, especially if also taking P450 enzyme inhibitor, e.g. Patient with a cold takes terfenadine and erythromycin at the same time Features • asymptomatic • may be picked up on routine ECG or following family screening • Long QT1 - usually associated with exertional syncope, often swimming • Long QT2 - often associated with syncope occurring following emotional stress, exercise or auditory stimuli • Long QT3 - events often occur at night or at rest • sudden cardiac death Diagnosis • corrected QT interval Diagnosis is based upon the QTc (corrected QT interval), QTc may be within the normal range at rest; hence Holter ECG monitoring is recommended. • genetic testing of LQTS Identification of an LQTS genetic mutation confirms the diagnosis. However, a negative result on genetic testing is of limited diagnostic value because only approximately 50% of patients with LQTS have known mutations. The remaining half of patients with LQTS may have mutations of yet unknown gene. Therefore genetic testing of LQTS has high specificity but a low sensitivity. Complications • may lead to ventricular tachycardia collapse/sudden death. Management Congenital long QT syndrome: • Beta-blockers Beta-blockers are first-line initial treatment Beta blockers alone are enough to abate collapses in up to 70% of patients. Beta blockers act by:

1. decrease sympathetic activation from the left stellate ganglion,
2. also decrease the maximal heart rate achieved during exertion and thereby prevent exercise-related arrhythmic events that occur in LQTS. should be avoided in those congenital cases in which bradycardia is a prominent feature. note sotalol may exacerbate long QT syndrome (due to blockage of K channel). This can be a particular risk in individuals with hypokalaemia. Therefore Sotalol is better to be avoided in patients with thiazide diuretics. • patients who remain symptomatic despite receiving the maximally tolerated dose of betablockers Permanent pacing and can be used in addition to beta-blockers. • patients who remain refractory to beta-blockade and pacing High left thoracic sympathectomy

Cardiology

• Implantable cardioverter-defibrillators (ICDs) are useful in rare instances when torsades still continues despite all of these treatments. • Beta-blockers should be used along with ICDs because shock can further precipitate torsades by adrenergic stimulation. • Left stellate cardiac ganglionectomy is an invasive procedure and results in Horner’s syndrome. It is performed in patients who have symptoms despite βB and have frequent shocks with ICD. Acquired long QT syndrome • avoid drugs which prolong the QT interval and other precipitants if appropriate (e.g.

Strenuous exercise) • Long-term treatment in acquired cases is usually not required because the QT interval returns to normal once the predisposing factor has been corrected. • Correction of any electrolyte disturbance Due to the pseudo-obstruction it is very likely that the patient is hypokalaemic and as such this is the first reversible aetiology for the non-sustained VT that needs to be investigated Check electrolytes Checking Magnesium would also be an appropriate step. • Beta-blockers are contra-indicated in acquired cases because bradycardia produced by these agents can precipitate torsades. They should also be avoided in those congenital cases in which bradycardia is a prominent feature. • Pacemaker implantation is effective in cases that are associated with heart block or bradycardia. • ICDs are indicated in cases that cannot be managed by avoidance of any specific precipitating factor. QT shortening: caused by: • Hypercalcaemia • Hypermagnesaemia • Digoxin • Thyrotoxicosis.

January 2019 exam: A patient develops torsades de pointes shortly after being started on sotalol. What effect does sotalol have on the cardiac cell membrane to make this more likely? Blockage of potassium channels prolonged QT interval.

Torsades de pointes (TdP) Overview • Torsades de pointes ('twisting of the points') is a rare arrhythmia associated with a long QT interval. • It may deteriorate into ventricular fibrillation and hence lead to sudden death • In its most typical form, sudden slowing of heart rate (i.e., pauses) invariably precede each burst of TdP, and the recurrent arrhythmia is referred to as “pause-dependent TdP” Risk factors • Female sex • causes of QT prolongation, • R-on-T phenomenon the R-wave, representing ventricular depolarization, occurs during the relative refractory period at the end of repolarization (represented by the latter half of the Twave).

Notes & Notes for MRCP
By Dr. Yousif Abdallah Hamad

 Long QT intervals predispose the patient to an R-on-T phenomenon, R-on-T can initiate torsades. • bradycardia, • congestive heart failure, • digitalis therapy, • severe alkalosis • recent conversion from atrial fibrillation. Management • Stop all drugs which prolong QT • Correct any electrolyte abnormalities • IV magnesium sulphate (MgSo4) the best initial drug Mode of action: MgSo4 ↓Ca influx ↓ amplitude of the VT and helping terminate runs of torsade’s. Dose : 2 gm as bolus over 10 minutes, followed by another bolus in 15 minutes if required, or continuous infusion at a rate of 5-20 mg/min. It is effective even when serum magnesium level is normal. • Temporary pacemaker/transvenous overdrive pacing (atrial or ventricular) reserved for patients with long QT-related TdP who do not respond to intravenous magnesium. • Isoproterenol usually used as a temporizing measure prior to pacing in patients who have failed to respond to magnesium and are awaiting placement of a temporary pacemaker. Action Strong beta-1 & beta-2 stimulation

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

Cardiology

Adult advanced life support Resuscitation Council (UK) 2021 guidelines Major points include: • Point-of-care ultrasound (POCUS) The guidelines recognise the increasing role of point-of-care ultrasound (POCUS) in peri-arrest care for diagnosis, but emphasises that it requires a skilled operator, and the need to minimise interruptions during chest compression. POCUS may be useful to diagnose treatable causes of cardiac arrest such as cardiac tamponade and pneumothorax. Right ventricular dilation in isolation during cardiac arrest should not be used to diagnose massive pulmonary embolism. • Immediately after the first shock (and each subsequent shock) chest compressions should be restarted immediately and pulse and rhythm reassessed after two minutes. • Chest compression Ratio of chest compressions to ventilation is 30:2 Chest compressions are now continued while a defibrillator is charged After each shock chest compressions should be restarted immediately before anything else is done. • Adrenaline should be used as soon as possible when the cardiac arrest rhythm is nonshockable after 3 defibrillation attempts for a shockable cardiac arrest rhythm. during a VF/VT cardiac arrest, adrenaline 1 mg is given once chest compressions have restarted after the third shock and then every 3-5 minutes (during alternate cycles of CPR). A 1 mg dose of adrenaline (epinephrine) would be administered with: 0.1 ml of 1 in 100, 1 ml of 1 in 1000 and 10 ml of 1 in 10,000. 10 ml of 1 in 10,000 is the recommended dose and concentration by the UK Resuscitation Council. If not able to gain any venous access within two minutes →Obtain intraosseous access (it provides adequate plasma levels of drugs and allows equivalent flow rates to IV access). Delivery of drugs via a tracheal tube is no longer recommended • Antiarrhythmic drugs (in VF/ pulseless VT) Give Amiodarone 300 mg after the third shock and 150 mg after the fifth shock. If amiodarone is not available →use Lidocaine 100 mg after the third shock and 150 mg after the fifth shock. • Atropine is no longer recommended for routine use in asystole or pulseless • Thrombolytic drugs Consider thrombolytic drug therapy when pulmonary embolus is the suspected or confirmed as the cause of cardiac arrest. Consider CPR for 60-90 minutes after administration of thrombolytic drugs. • Waveform capnography during advanced life support Use waveform capnography to confirm correct tracheal tube placement during CPR. Use waveform capnography to monitor the quality of CPR. An increase in ETCO2 during CPR may indicate that ROSC has occurred. However, chest compression should not be interrupted based on this sign alone.

• Recurrent or refractory VF Consider escalating the shock energy, after a failed shock and for patients where refibrillation occurs. For refractory VF, consider using an alternative defibrillation pad position (e.g. anterior- posterior). Magnesium sulphate IV is recommended for the treatment of refractory VF, if there is anything to suggest the patient may be hypomagnesaemic (such as on medications which might cause this, that is, thiazides). • Precordial thump Indicated only in witnessed or monitored cardiac arrect whilst awaiting the defibrillator within seconds of the onset of a shockable rhythm. It has a very low success rate for cardioversion. There is more success with pulseless VT than with VF. Chest compressions should start immediately if it is unsuccessful. The ulnar edge of a tightly clenched fist is used to deliver a sharp impact from a height of about 20 cm, then retract immediately (thereby creating an impulse-like stimulus). It delivers approximately 7-10 joules of energy. Only one thump should be delivered over the lower third of the sternum. Repeating a precordial thump is not recommended. • Electrical activity (PEA) pulseless with no respiratory effort , ECG reveals small complexes with a normal morphology →CPR

+ Adrenalin 1mg repeated every 3-5 minutes • Defibrillation Defibrillation is used to convert ventricular fibrillation to sinus rhythm Use single shocks where indicated, followed by a 2 minute cycle of chest compressions. The use of up to three-stacked shocks may be considered only if initial ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT) occurs during a witnessed, monitored cardiac arrest with a defibrillator immediately available e.g. during cardiac catheterisation or in a high-dependency area. Antero-lateral pad position is the position of choice for initial pad placement. Ensure that the apical (lateral) pad is positioned correctly (mid-axillary line, level with the V6 ECG electrode position) i.e. below the armpit. In patients with an implantable device, place the pad > 8 cm away from the device Range of the initial defibrillation energy No clear evidence so , any level from 120-360 J is acceptable followed by a fixed or escalating strategy up to maximum output of the defibrillator.

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

Cardiology

• Cardiac arrest in profound hypothermia Prolonged cardiopulmonary resuscitation with re-warming is the management of choice. Recovery with intact neurology has been reported even after very prolonged arrests, therefore resuscitation should be continued for far longer than would normally be considered. Hypothermic patients do not respond well to shocks or drugs and if there is no response to the first three shocks the patient should be rewarmed to at least 32°C before any drugs or shocks are administered. • Management of cold water drowning patients should be lifted out of the water in the prone position Re-warming such patients should be undertaken in a hospital that has extracorporeal re-warming facilities Defibrillation is ineffective if the myocardium is cold Hypothermia may render the carotid pulse impalpable so it is important to commence chest compression with firm evidence of cardiac arrest. Continuous chest compression should be applied throughout transportation, which is as effective as chest compression with expired air resuscitation Lance-Adams syndrome (Post-hypoxic myoclonus) • Definition: a rare condition that can occur following a period of cerebral hypoxia (e.g. post cardiac arrest) • Onset: occurs within days to weeks of cardiac arrest. • Characterised by intention myoclonus. • Treatment : antiepileptics (e.g. levetiracetam, valproate)

Peri-arrest arrhythmias Resuscitation Council (UK) 2021 guidelines Tachycardia • To convert atrial or ventricular tachyarrhythmias, the shock must be synchronised to occur with the R wave of the ECG. • For atrial fibrillation: An initial synchronised shock at maximum defibrillator output rather than an escalating approach is a reasonable strategy • For atrial flutter and paroxysmal supraventricular tachycardia: Give an initial shock of 70 - 120 J and stepwise increase energy for subsequent shocks • For ventricular tachycardia with a pulse: Give an initial shock of 120-150 J and stepwise increase energy for subsequent shocks • If cardioversion fails to restore sinus rhythm and the patient remains unstable: give IV amiodarone 300 mg over 10–20 minutes (or procainamide 10–15 mg kg-1 and re-attempt electrical cardioversion. The loading dose of amiodarone can be followed by an infusion of 900 mg over 24 hours.

Notes & Notes for MRCP
By Dr. Yousif Abdallah Hamad

Bradycardia • 1st line : give atropine 500 mcg IV (IO) and, if necessary, repeat every 3–5 minutes to a total of 3 mg. • 2nd line (If atropine is ineffective): isoprenaline (5 mcg min−1 starting dose), and adrenaline (2–10 mcg min−1). • For bradycardia caused by inferior myocardial infarction, cardiac transplant or spinal cord injury, consider giving aminophylline (100–200 mg slow intravenous injection). • If bradycardia caused by beta-blockers or calcium channel blockers → give glucagon • For bradycardia in patients with cardiac transplants Give aminophylline Do not give atropine, it can cause a high-degree AV block or even sinus arrest. • For bradycardia refractory to drug therapies in patients who are unstable: transcutaneous pacing If transcutaneous pacing is ineffective, consider transvenous pacing. • If atropine is ineffective and transcutaneous pacing is not immediately available, fist pacing can be attempted while waiting for pacing equipment.

Wolff-Parkinson White (WPW) Pathophysiology • Due to a congenital accessory conduction pathway, called the bundle of Kent, that connects the atria to the ventricles, bypassing the AV node and leading to ventricular preexcitation. As the accessory pathway does not slow conduction, AF can degenerate rapidly to VF Presentation • Most patients are asymptomatic. • WPW presents as SVT that can alternate with ventricular tachycardia (VT). • SVT is the most common type of tachycardia seen in a patient with WPW. often present with AV re-entrant tachycardia • The other main clue to the diagnosis is worsening of SVT after the use of calcium blockers or digoxin Possible ECG features • short PR interval • wide QRS complexes with a slurred upstroke - 'delta wave' (can be associated with negative delta waves in II, III and aVF) • ECG in sinus rhythm reveals right bundle-branch block • left axis deviation if right-sided accessory pathway* *in the majority of cases or in a question without qualification, Wolff-Parkinson-White syndrome is associated with left axis deviation • right axis deviation if left-sided accessory pathway • non-specific ST-T changes which are common in WPW and may be mistaken for ischaemia.

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

Cardiology ECG showing short PR interval associated with a slurred upstroke (delta wave). Note the non-specific ST-T changes which are common in WPW and may be mistaken for ischaemia. The left axis deviation means that this is type B WPW, implying a right-sided pathway Differentiating between type A and type B • type A (left-sided pathway): dominant R wave in V1 • type B (right-sided pathway): no dominant R wave in V1 In type B pre-excitation, the accessory pathway connects the right atrium to the right ventricle • there is a rare type C WPW, WPW in which the delta waves are upright in leads V1-V4 but negative in leads V5-V6 Associations of WPW • HOCM • mitral valve prolapse • Ebstein's anomaly • thyrotoxicosis • secundum ASD • Leber's hereditary optic neuropathy (mitochondrial disease)

Notes & Notes for MRCP
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Investigations • ECG Short PR interval ECG delta wave: a slurred upstroke at the start of the QRS complex, secondary to preexcitation

 Widened QRS • The most accurate test is electrophysiologic studies Management Acute episodes • Hemodynamically unstable: electrical cardioversion • Hemodynamically stable: assess underlying rhythm Narrow-complex tachycardia (including Afib, atrial flutter) Rhythm control measures (i.e. IV procainamide or cardioversion) are the safest treatment option. Vagal maneuvers and AV nodal blocking agents (Adenosine, B-Blockers, Calcium Channel Blockers, Digoxin) are contraindicated (may precipitate ventricular tachycardia or V-fib) Wide-complex regular or irregular tachycardia → Determine whether the rhythm is more likely to be ventricular or supraventricular in origin (e.g., Brugada criteria) VT (~80%): pharmacological cardioversion or synchronized electrical cardioversion SVT (< 20%): Determine if an accessory pathway is present. Findings suggestive of an accessory pathway: synchronized electrical cardioversion or IV procainamide HR > 200 Irregular rhythm No bundle branch block on ECG Signs of impending instability (e.g., clammy skin) Baseline ECG findings that support the diagnosis No signs of an accessory pathway: manage as SVT Undifferentiated wide-complex tachycardia: Treat as VT, with either electrical cardioversion or IV procainamide Long-term management • High-risk patients → Catheter ablation Syncope Associated atrial fibrillation, atrial flutter, or atrial tachycardia Aborted sudden cardiac death Family history of sudden cardiac death High-risk occupations (e.g., pilots, athletes, school bus driver) • Low-risk patients Asymptomatic patients: usually no treatment required symptomatic patients: First-line treatment → catheter ablation Differentiating between VT and SVT Brugada criteria ECG finding VT SVT Absence of RS in all precordial leads? Yes No R:S interval > 100 ms in one precordial lead? Yes No Signs of AV dissociation present? Yes No QRS morphology consistent with VT in leads V1-2 and V6? Yes No Interpretation • If the answer to any is yes: most likely VT • If none are present: most likely SVT

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

Cardiology WPW management • Asymptomatic : (incidentally found delta wave on ECG) →Reassurance • Asymptomatic in high-risk professions (eg pilots, school bus driver) is best managed by catheter ablation of the accessory pathway • Asymptomatic WPW in someone with a family history of sudden cardiac death is another indication for radiofrequency catheter ablation • Chronic medical therapy: flecainide, amiodarone, procainamide • Definitive treatment: radiofrequency ablation of the accessory pathway (first-line therapy) Contraindications in WPW A simple mnemonic to remember for drugs to avoid in WPW syndrome is ABCD (Adenosine, B-Blockers, Calcium Channel Blockers, Digoxin). • Digoxin • Beta-blockers • Diltiazem, verapamil • Amiodarone • This is because blocking the AV node may enhance the rate of conduction through the accessory pathway, increasing the ventricular rate and potentially deteriorating into ventricular fibrillation. If wide-complex tachycardia is present and the diagnosis of ventricular tachycardia (VT) cannot be excluded, the drugs of choice are IV procainamide or amiodarone. Lown–Ganong–Levine (LGL) syndrome: LGL syndrome is like WPW in the sense that it is a pre-excitation syndrome. However, the ECG changes present is only short PR interval without delta waves or abnormal QRS complex.

Implantable cardiac defibrillators (ICD) Indications • Congenital long QT with family history of sudden cardiac death at young age. • hypertrophic obstructive cardiomyopathy (HOCM) • previous cardiac arrest due to VT/VF • Sustained VT causing haemodynamic compromise • previous

myocardial infarction with non-sustained VT on 24 hr monitoring, inducible VT on electrophysiology testing and ejection fraction < 35% • Brugada syndrome • Arrhythmogenic right ventricular cardiomyopathy causing cardiac arrest. If it is not possible to quickly identify the underlying rhythm as SVT or VT, it is safest to treat empirically as VT with synchronized electrical cardioversion (100 J) or with IV

Acute pericarditis Overview • Acute pericarditis: inflammation of the pericardium that either occurs as an isolated process or with concurrent myocarditis (myopericarditis). • Pericarditis is one of the differentials of any patient presenting with chest pain. Features • Pleuritic chest pain Exacerbated by inspiration and lying flat, relieved by sitting up and leaning forwards • Shoulder pain (referred pain): pericarditis is innervated by phrenic nerve • Pericardial rub (present in 50% of cases.) → pathognomic feature • Other symptoms include non-productive cough, dyspnoea and flu-like symptoms Types and causes • Fibrinous pericarditis (the most common type) Causes: Viral infection is the most common cause of acute pericarditis: the most common viral cause is Coxsackie B virus Acute myocardial infarction (MI): more common than dressler syndrome friction rub is more common than pain Aspirin is the only NSAID that can be used in pericarditis complicating MI. Post MI (Dressler syndrome): rare, autoimmune-mediated phenomenon to myocardial antigens, occur 2 – 4 weeks post MI Because of the risk of hemorrhagic pericarditis, anticoagulant therapy should be stopped in patients with dressler syndrome. Radiation, trauma, severe infections Uremic pericarditis blood urea nitrogen (BUN) level is usually greater than 60 mg/dL (22 mmol/L). Hemorrhagic effusions are more common and result in part from uremiainduced platelet dysfunction. does not present with the classic diffuse ST-elevations seen on ECG as in other types of pericarditis. Uremic pericarditis is an indication for urgent hemodialysis. • Serous pericarditis Usually caused by noninfectious inflammation such as: rheumatoid arthritis (RA) systemic lupus erythematosus (SLE). Fibrous adhesions rarely occur. • Purulent or suppurative pericarditis Most commonly caused by staphylococcal and gram-negative species, high percentage of patients develop constrictive pericarditis. • Hemorrhagic pericarditis Most commonly caused by: tuberculosis, direct neoplastic invasion. Severe bacterial infections Bleeding diathesis, cardiac surgery or trauma (may cause tamponade).

• Caseous pericarditis caseation within the pericardial sac is tuberculous in origin, until proven otherwise, In tuberculous pericarditis, fever, night sweats, and weight loss are commonly noted (80%). Untreated, caseous pericarditis is the most common antecedent to chronic constrictive pericarditis of a fibrocalcific nature. Approximately 50% of affected patients develop constrictive pericarditis. ECG changes • Stage 1 (initial) Diffuse ST elevations ST depression in aVR and V1 PR segment depression (most specific ECG marker for pericarditis) • Stage 2: ST segment normalizes in ~1 week. • Stage 3: inverted T waves in all leads ~ 1 – 2 weeks • Stage 4: ECG

returns to normal baseline after weeks to months. Which ECG changes would you expect to see in the next week or two? T-wave inversion in all leads Echocardiography is often normal in patients with pericarditis but is needed to rule out pericardial tamponade and pericardial constriction Laboratory findings • Elevation of inflammatory markers may support the diagnosis of pericarditis but are not considered to be a part of the diagnostic criteria. • CBC: leukocytosis, ↑ ESR, ↑ CRP • ↑ Troponin I : suggest some degree of myocarditis. • ↑ Creatinine kinase ECG showing pericarditis. Note the widespread nature of the ST elevation and the PR depression

Notes & Notes for MRCP
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Diagnosis ESC guidelines defined the diagnosis of acute pericarditis as 2 out of 4 of the following:

1. pericarditic chest pain;
2. pericardial rub;
3. new widespread ST-elevation or PR depression; and
4. pericardial effusion (new or worsening). • Rule out other causes of acute chest pain (e.g., myocardial infarction, myocarditis) before making a diagnosis of acute pericarditis. Treatment • Pain management (analgesia, observation) NSAID therapy (Aspirin, Ibuprofen) Post-myocardial infarction pericarditis: avoid NSAIDs other than aspirin. Colchicine (in combination with NSAIDs or as a monotherapy). Useful both in acute episode and to prevent recurrence of pericarditis. • Only consider prednisone in: severe cases (not responded to NSAID and Colchicine) or in pericarditis caused by uremia, connective tissue disease, or autoreactivity. • Treat any known underlying causes • Pericardectomy is only indicated for recurrent pericarditis once medical interventions have failed. • Treatment duration: until symptoms have resolved and CRP has normalized, but normally it is for 1-2 weeks duration. • Reduce physical activity Prognosis • Recurrence Between 15 and 30% of patients with idiopathic acute pericarditis may have recurrent attacks, and this is considered to be an autoimmune phenomenon. • Poor prognostic factors include: Temperature above 38ºC Subacute disease course Presence of a large effusion or tamponade Unsuccessful therapy with nonsteroidal anti-inflammatory agents • Factors associated with complicated pericarditis include: Early administration of high-dose corticosteroids Lack of colchicine treatment Elevated levels of high-sensitivity C-reactive protein Acute pericarditis • Symptoms include sharp, severe retrosternal chest pain worse with inspiration and a supine position. • The classic physical finding is a pericardial friction rub. A low-grade fever is often present. • Diagnostic signs include new widespread diffuse concave upwards ST elevation and/or PR depression on ECG and new or worsening pericardial effusion on echocardiography; blood tests generally suggest systemic inflammation. • Treatment: All patients should be given a non-steroidal anti-inflammatory drug as first-line treatment. Colchicine should also be given unless the patient has tuberculous pericarditis. • Complications include chronic recurrent pericarditis, cardiac tamponade, and constrictive pericarditis.