

Cardiopulmonary resuscitation for sports medicine diploma

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Pharmacological treatment of significant cardiac arrhythmias

For life threatening cardiac arrhythmia, the administration of drugs should be by the intravenous route. Drugs delivered through a peripheral vein should always be followed by a 20 ml bolus of saline to aid delivery to the central circulation. Where no venous access is possible, drugs (particularly adrenaline (epinephrine)) may be delivered by the endotracheal route in double or triple doses.

Ventricular fibrillation (VF)/pulseless ventricular tachycardia (VT)

The pharmacological treatment of VF/pulseless VT is of secondary importance to early defibrillation. Administration of antiarrhythmic drugs is considered in cases of refractory VF/pulseless VT—that is, when cardioversion does not occur after 12 DC shocks with appropriate advanced life support provided. Drugs recommended by the European Resuscitation Council are those outlined for use in VT.

Ventricular tachycardia

LIGNOCAINE (LIDOCAINE)

Lignocaine is a class IB antiarrhythmic drug and is the first choice for VT. It is given intravenously in a dose of 1–3 mg/kg. For cardiac arrest, a 100 mg bolus is given, which may be repeated after 5–10 minutes. If successful cardioversion occurs, plasma levels can be maintained by an intravenous infusion of 2–4 mg/min. Lignocaine has no effect on supraventricular tachycardia (SVT). Like most antiarrhythmic drugs, lignocaine depresses myocardial contractility, and toxic levels can produce paraesthesia, drowsiness, muscular twitching, or seizure.

AMIODARONE

Amiodarone is an effective class III antiarrhythmic drug. It has a long half life, up to 100 days, and is associated with serious side effects on long term administration. Its antiarrhythmic effect may take up to 30 minutes. It is therefore not normally used as a first line treatment, unless the patient is clinically stable. It can be used for both ventricular and supraventricular arrhythmias and is usually given in a dose of 5 mg/kg (300 mg) over one hour. In life threatening situations, such as cardiac arrest, it can be given over 15 minutes and repeated after one hour. A further loading dose of 15 mg/kg (up to 900 mg) is given over the next 24 hours.

BRETYLIUM

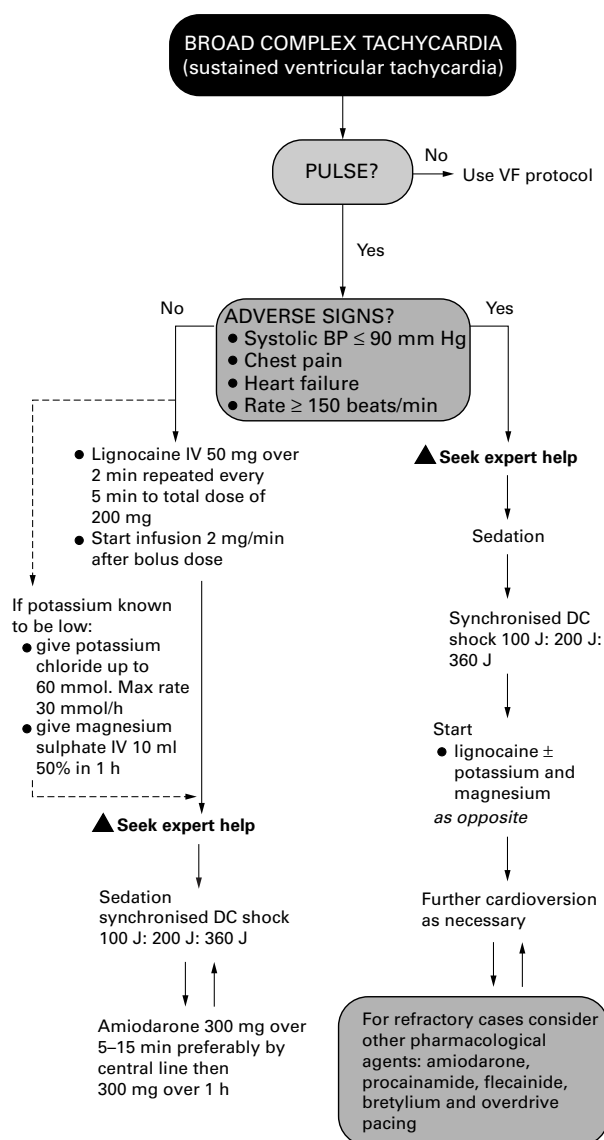
Bretylium is likely to exhibit both class II and III effects. Initially it causes release of noradrenaline (norepinephrine) from sympathetic nerve terminals and subsequently blocks further release of catecholamines from the sympathetic nervous system. It may therefore be associated with transient hypertension, but will then often cause pronounced hypotension. Its antiarrhythmic effect may not be seen for up to 20 minutes, therefore cardiopulmonary resuscitation should be continued for at least this length of time. Bretylium can be used for refractory VT that does not respond to other agents, and is administered in a dose of 5 mg/kg diluted with 100 ml dextrose. If this is unsuccessful, a further dose of 10 mg/kg can be administered.

MAGNESIUM

Magnesium may be effective for VF/VT, particularly when these rhythms are associated with acute myocardial infarction. The dose is an 8 mmol bolus injection followed by a 2.5 mmol/h infusion.

Supraventricular tachyarrhythmias

Treatment of narrow complex tachyarrhythmias (SVT) depends on the clinical condition of the patient. If the patient is decompensated—for example, hypotensive, in heart failure, experiencing angina, or has a heart rate above



Doses based on adult average body weight.
In all cases give oxygen and establish IV access.

Figure 1 Algorithm for treatment of broad complex tachycardia. Reprinted with permission from the UK Resuscitation Council.

200 beats/min—then they should be sedated and treated by DC cardioversion, with subsequent pharmacological prophylaxis, if indicated.

VAGAL MANOEUVRES

If the patient is clinically stable, then manoeuvres that increase vagal tone should be tried in the first instance. The commonest of these is the Valsalva manoeuvre in which the patient tries to exhale forcibly against a closed glottis. Alternatively, unilateral carotid artery pressure can be applied, but this should not be performed in the presence of a carotid bruit, because of the risk of stroke.

ADENOSINE

Adenosine is a naturally occurring purine nucleotide which selectively blocks AV nodal conduction. It is therefore the drug of choice for terminating AV nodal re-entrant tachyarrhythmias. It has an extremely short half life and should be given by rapid bolus injection, followed by a saline flush. Initial dosage is 3 mg, and if no effect is seen after one or two minutes, then 6 mg can be administered followed by a maximum dose of 12 mg. Adenosine will nearly always slow SVT, often allowing identification of the underlying rhythm, but will have no effect on VT. It can be given in combination with β blockers and does not cause depression of myocardial contractility. The effect of adenosine is enhanced with dipyridamole and reduced by theophylline. Side effects such as flushing and chest pain are commonly experienced with adenosine, but usually last less than 60 seconds. Bronchospasm can be precipitated in asthmatic patients, and therefore it should not be routinely used in these patients. Adenosine must be given in a monitored environment—for example, critical care unit or accident and emergency department—as it can cause transient complete heart block.

VERAPAMIL

Verapamil is a calcium channel blocker, which slows conduction through the AV node. It is also negatively inotropic. It is used in the treatment of definite SVT and is given in doses of 5–10 mg over 60 seconds. It is contraindicated if β blockers have been taken by the patient because of the risk of profound bradycardia and hypotension. Verapamil should not be used for SVTs associated with Wolff-Parkinson-White syndrome, as in this situation it may precipitate VT/VF by allowing conduction through the accessory pathway.

β BLOCKERS

β Blockers act by blocking the excitatory effects of circulating catecholamines. They can be used in the treatment of supraventricular tachyarrhythmias as they slow conduction through the AV node. All β blockers can precipitate left ventricular failure, particularly in those with depressed myocardial function. Esmolol, which has a very short half life (eight minutes) and is available as an intravenous preparation is particularly useful as any unwanted side effects will be short lived. β Blockers should also be avoided in asthmatics because of the risk of bronchospasm.

DIGOXIN

Digoxin may be useful in controlling the ventricular response rate in atrial fibrillation, but it has limited applications in emergency settings.

Bradyarrhythmias

Transient bradycardias are common in acute myocardial infarction, particularly inferior myocardial infarction. Treatment depends on the clinical condition of the patient. Bradycardia of less than 40 beats/min or associated with signs of cardiac failure or type II heart block should be treated initially with atropine. It can be given in doses of 0.5 mg up to 3 mg, titrating the dose according to the heart rate response. In patients with pauses longer than three seconds, Mobitz type II heart block, type II heart block in association with an anterior myocardial infarction, and previous asystole, preparation should be made immediately for temporary cardiac pacing.

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Further reading

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Handley AJ, Swain A. *Advanced life support manual*. 2nd edition. UK Resuscitation Council, 1996.