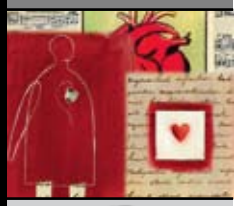
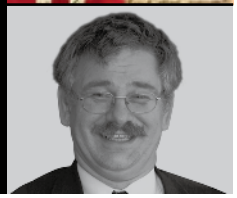
THEME

Arrhythmias





Emergency management of acute cardiac arrhythmias

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BACKGROUND

Anything other than normal sinus rhythm can be classified as an arrhythmia. However not all arrhythmias need acute intervention.

OBJECTIVE

This article reviews which arrhythmias need intervention in an acute setting, and the various options available for intervention.

DISCUSSION

The impact of an arrhythmia upon perfusion determines what intervention should be considered. Conscious level, cardiac ischaemia secondary to poor perfusion of the coronary arteries and blood pressure need to be assessed. Patients with bradycardias with adequate perfusion are treated initially with oxygen and observation. Sinus bradycardia not responding to increased oxygenation is treated with atropine. For other bradycardias the two alternatives are to drive the inherent rate with a sympathomimetic drug or to pace the patient with an external or internal pacer. Usually supraventricular tachycardias are not life threatening. Unconscious patients with wide complex tachycardia should be treated in a standard cardiac arrest approach. Conscious patients in ventricular fibrillation however, can be treated either chemically or with synchronised cardioversion. If a patient is in cardiac arrest the approach is to establish effective resuscitation and early defibrillation as per Australian Resuscitation Council guidelines.

Not all arrhythmias need acute intervention. Some are relatively benign and as such are best not addressed in the acute setting. For instance, atrial fibrillation (AF) should not be addressed in the acute setting unless of very recent origin. Other arrhythmias – while looking dramatic – are still managing to perfuse the brain and are better than the alternatives that might result from an attempt to correct them. An approach to arrhythmias that are still perfusing is to provide support with oxygen and ventilation while observing the patient. The alternative approach of more active management sometimes runs the risk of creating a situation that is worse than the current one.

The criteria for deciding that an arrhythmia needs intervention vary with the situation and the support available. Conscious level is probably the best marker of perfusion in this situation. Although blood pressure has always been considered as a key marker it is probably less a useful than conscious level, as a marker of adequate perfusion. Mean arterial pressure is the key measure of perfusion but unfortunately is not well related to measured blood pressure particularly at the extremes of heart rate. The hardest decisions regarding intervention are for patients who, while conscious, are obviously not perfusing well and for whom the consequences of intervention may include an even worse rhythm. For example, a complete heart block with a junctional escape rhythm may generate perfusion that, although not optimum, is adequate. If in doubt, an approach of maximising oxygenation, optimising preload and adopting an attitude of optimistic expectancy is the safest strategy.

Too slow

Bradycardia can be a sinus bradycardia often associated with increased vagal tone (*Figure 1*) or secondary to a failure of the sinoatrial (SA) node or atrioventricular (AV) block. Atrioventricular block is a problem of the conduction system with a block at the AV node. Complete AV block results in the failure to transmit any impulses from atria to ventricle (*Figure 2*) while the partial AV block will result in either a slower transmission or an intermittent transmission.

The degree of bradycardia and the impact upon perfusion determine whether or not intervention should be considered. Conscious level is a good guide of perfusion, as is evidence of ischaemic chest pain secondary to poor perfusion of the coronary arteries. Blood pressure and other indirect measures of perfusion (the ability to provide a pulse wave detected by a pulse oximeter) are useful adjuncts when assessing perfusion status.

Evidence of ischaemia in the right coronary artery territory (inferior leads) in a patient who is not yet bradycardic raises the possibility of bradycardia secondary to failure of the SA node or AV node. A patient with evidence of ischaemia in this area should have an intravenous (IV) inserted and preparations available for pacing or drug intervention.

The initial approach to sinus bradycardia should be to increase oxygenation and ensuring an adequate preload (*Figure 3*). Simply lying the patient down and raising the legs will improve venous return and improve the preload. If the jugular venous pulse (JVP) or pressure can be assessed and is found to be low, a little additional IV fluid judiciously given is appropriate. If however the JVP is already elevated, then increasing the preload further would be harmful as it is likely to lead to acute pulmonary oedema. Sinus bradycardia will often respond to atropine as atropine effectively removes the parasympathetic brake on the SA, and to a much lesser extent the AV node. The dose of atropine should be at least 1 mg as small doses have been known to paradoxically reduce the heart rate by interfering with the sympathetic system. In reality this means two ampoules of 600 µg of atropine for an adult.

Bradycardia that is not sinus rhythm is running on either a junctional or a ventricular pace maker (*Figure 4*). Although atropine tends to be given as an automatic response it is not logical to expect it to be very effective as there is only a slight parasympathetic innovation of the AV node. If the patient is symptomatic and oxygen and optimising the preload with posture has already been tried, the two alternatives are to drive the inherent rate with a sympathomimetic drug or to pace the patient with an external or internal pacer arrangement. Adrenaline is

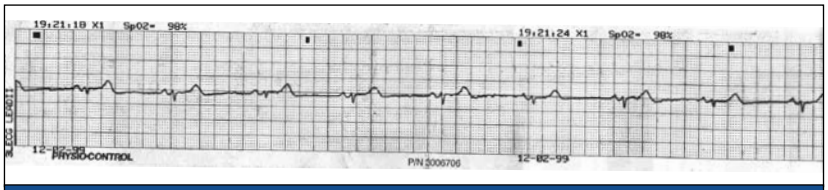

Figure 1. Sinus bradycardia

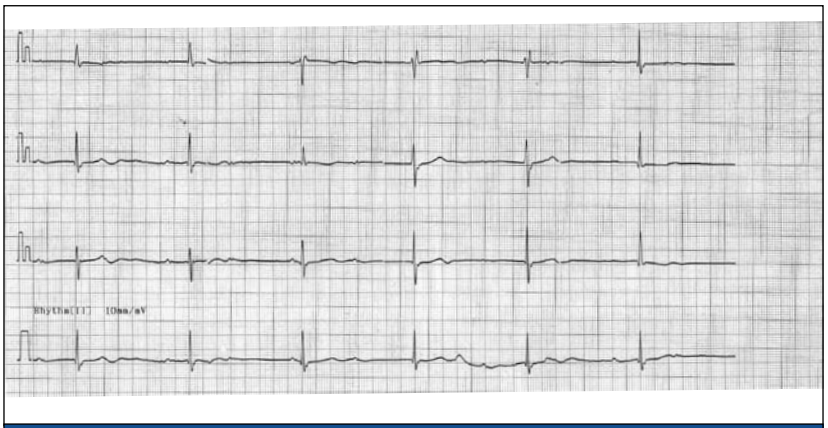

Figure 2. Bradycardia (complete block)

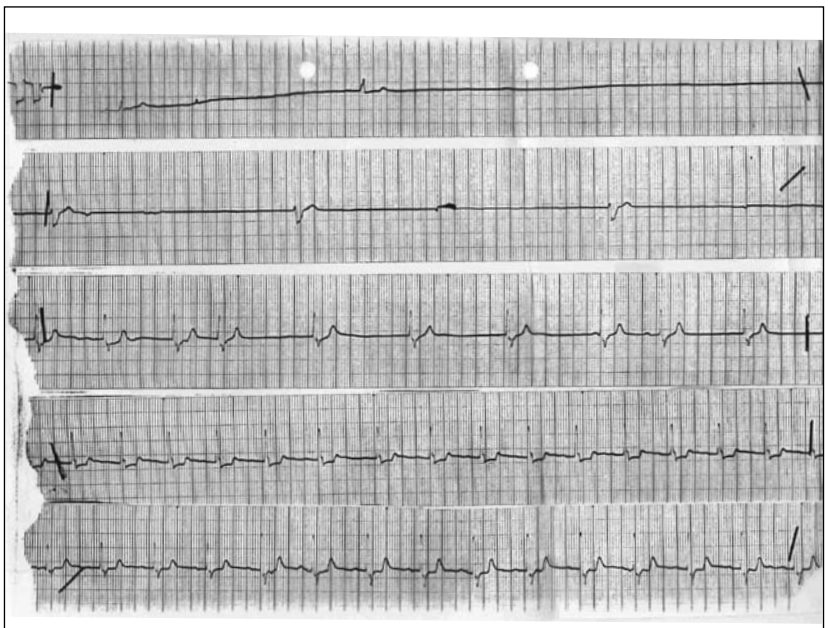

Figure 3. Bradycardia improving with oxygen

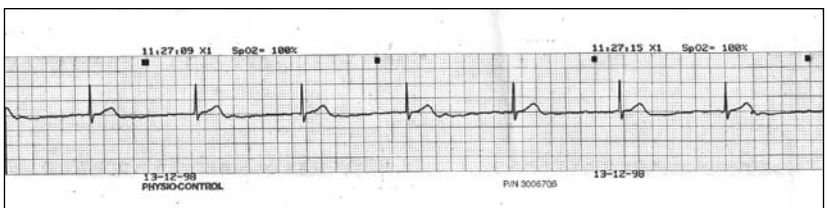

Figure 4. Bradycardia

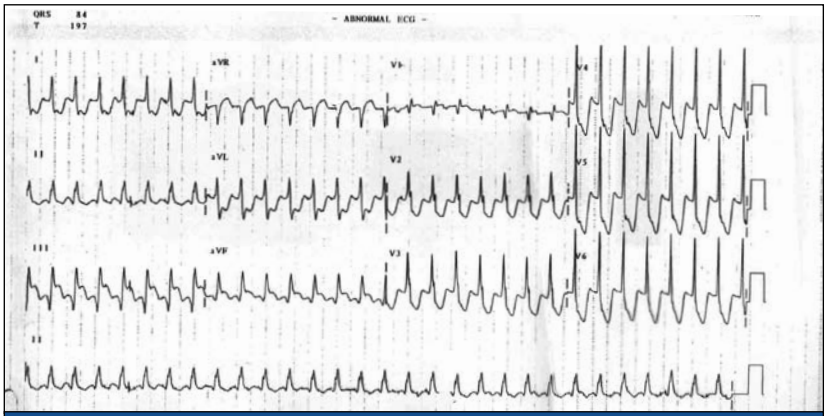

Figure 5. Supraventricular tachycardia

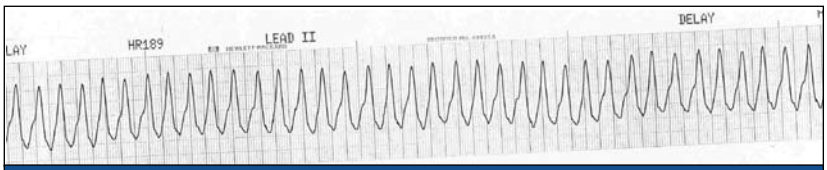

Figure 6. Ventricular tachycardia

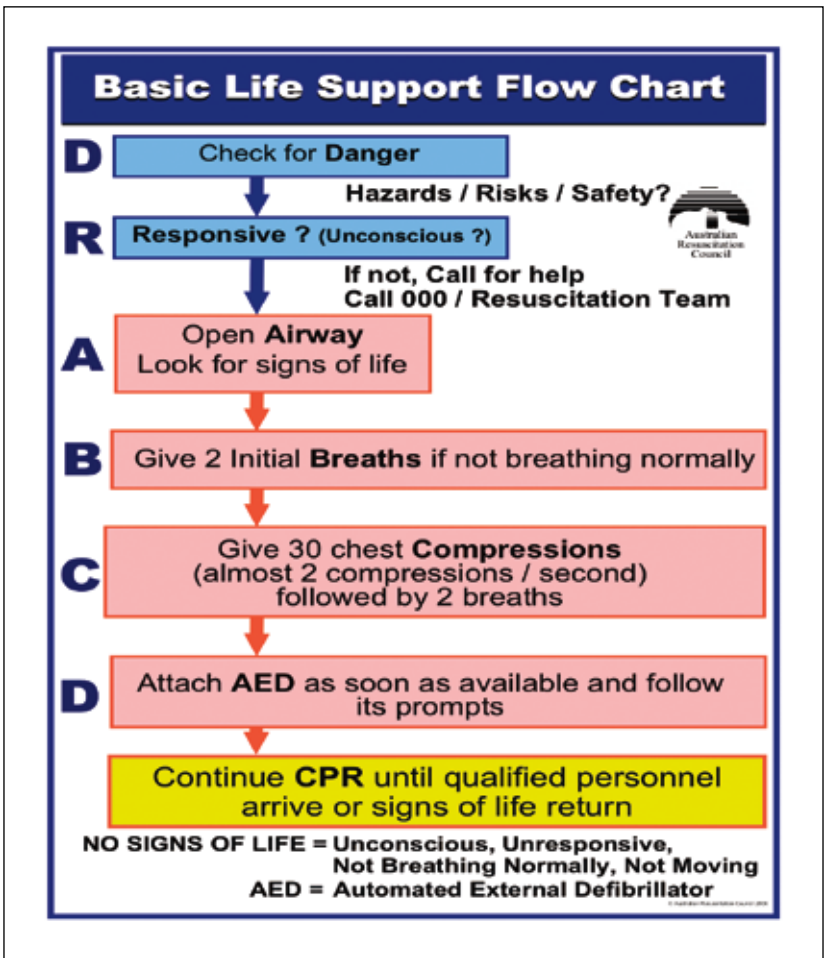

Figure 7. Basic life support flowchart
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an excellent choice of sympathomimetic drug, although isoprenaline is often the drug of choice. Whichever drug is used the route should be IV and ideally by a low dose infusion gradually increasing the infusion rate until a desired response is achieved. The issue here is balancing improved oxygenation of the coronary muscle with increased oxygen demand. Although improving the heart rate improves coronary artery perfusion pressure and thus oxygenation, oxygen demand rises as heart rate and force of contraction increase. Usually the predominant effect is beneficial from the increase in coronary artery perfusion pressure.

Externally applied pacemakers are becoming commonplace and can be easily used as an alternative. The self adhesive pads should be placed on the anterior chest wall and between the shoulder blades for optimum current transmission. The patient should be warned that this will be uncomfortable. The pacing function is selected at an initial rate of about 60 bpm. It is not a good idea to select too high a rate initially as an increase in heart rate increases the oxygen demand. The current delivered should be quickly increased to a point where capture occurs. Capture will be confirmed by the presence of a complex after each pacing spike and hopefully by the presence of a palpable pulse. At this point, the increased perfusion will be improving the patient's conscious state to the point where they will be aware of the distress caused by the repeated muscle contractions. Once perfusion has been achieved it is appropriate to give midazolam (and possibly an opiate in a small dose) to relieve the patient's discomfort and reduce their memory of the event. In a more intensive setting, formal pacing with an internal wire can be considered.

Too fast

Tachycardias can also reduce cardiac output as ventricular filling becomes ineffective; they also increase myocardial oxygen demand making ischaemia worse.

A supraventricular tachycardia (SVT) arises above the ventricles (*Figure 5*) and, with the exception of atrial fibrillation/flutter, has an atrial contraction preceding each ventricular contraction. This means that the SVT is likely to have better ventricular filling and hence better cardiac output. Usually SVTs are not life threatening and the patient remains conscious, although distressed, by palpitations. Initial management is to increase the vagal stimulus to the AV node by performing a Valsalva manoeuvre. To do this properly the patient should be supine and a forced expiration against a blocked airway needs to be maintained for at least 15 seconds. The vagal stimulus occurs after letting go. Rubbing the carotid sinus will also produce vagal stimulation, however this is considered dangerous

in anyone over 50 years of age because of the risk of disturbing atherosclerotic plaque. If a Valsalva manoeuvre has failed, the usual drug treatment is adenosine given in increasing doses of 6, 12, and if still not effective, 18 mg as sharp IV pushes into a running line (at least 18 gauge). This gives a 'chemical defibrillation' giving the heart an opportunity to restart in sinus rhythm. Some patients find this sensation distressing and so should be warned beforehand.

Rapid AF is a common supraventricular arrhythmia characterised by a rapid irregular QRS, which is often best heard by listening to the rhythm of the beat via the audible alarm. Long term AF should not be reverted in the acute setting without first excluding the possibility of mural thrombosis in the atria which might then become the source of an embolus. Management of AF is discussed in a separate article in this issue.

Ventricular tachycardia (VT) arises below the atria and is characterised by a broad complex tachycardia that often has an unusual axis (*Figure 6*). The unconscious patient should be treated as if in ventricular fibrillation (VF) with a standard cardiac arrest approach. Conscious patients can be treated either chemically or with synchronised cardioversion. The advantage of cardioversion is that there is no after effect persisting after treatment and lowering of cardiac output. Any antiarrhythmic drug used will also reduce myocardial contractility. For this reason, drug treatment of VT should be reserved for patients with a reasonable initial cardiac output despite the VT. In the past, lignocaine (1.5 mg/kg=100 mg for adults) was commonly used but is now being displaced by amiodarone (150 mg slowly in an IV infusion over 30 minutes and share the call with a cardiologist) as a drug of choice; sotalol and magnesium have also been used in this role. If the patient is not well perfused, cardioversion is preferable to drug therapy. For this procedure the patient is given a small dose of midazolam to ensure that they have little or no memory of the event, but not enough to significantly sedate them (up to 3 mg in an adult). Once the drug has circulated, which will take time in a compromised patient, a synchronised DC shock is given via the defibrillator pads or paddles. Most patients will revert at 50 J but starting at 100 J gives a high probability of first shock success. The shock is delivered 'synchronised' – this means you avoid defibrillating in the middle of the T wave which might provoke VF.

Supraventricular tachycardias in patients with a wide electrocardiogram (ECG) complex can be confused with VT. If the patient is poorly perfused or unconscious it is safer to treat them as a VT. The criteria that help to differentiate between a broad complex ST and a VT are:

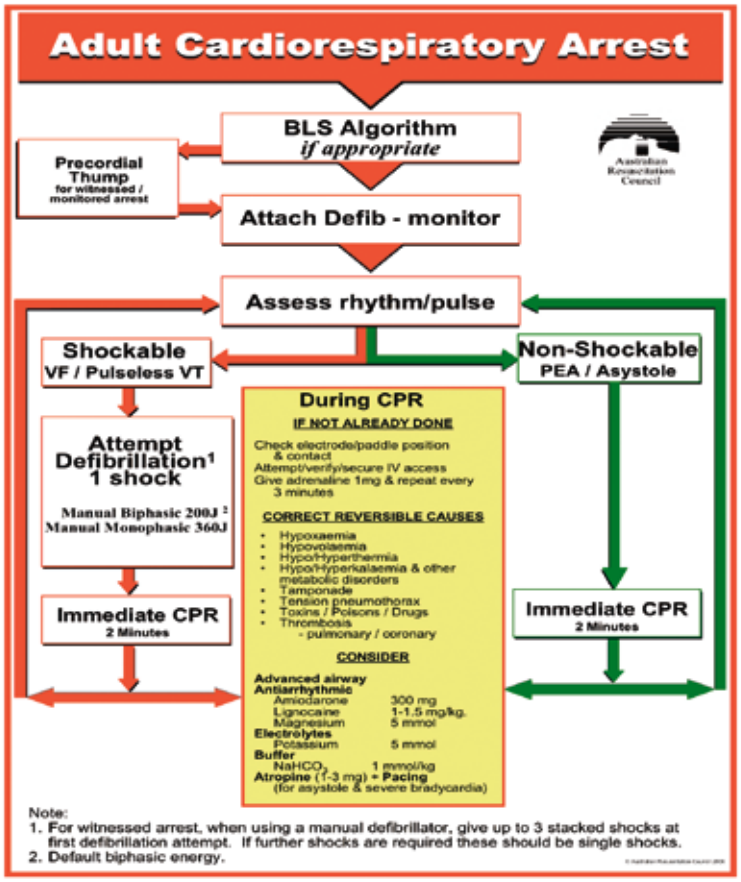

Figure 8. Adult cardiorespiratory arrest
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- the presence of P waves independently timed to the ventricular complexes in VT. These are seen as irregularities behind the QRS complexes that appear on a regular basis but are not there on all beats
- the presence of fusion beats where a P wave has transmitted through the AV node and then the ventricular depolarisation has been subsumed by the VT wave in VT
- an abnormal axis (often seen as failure of the chest, leads to progress from negative to positive; often seen in VT)
- the patient's age and perfusion status are a guide, with elderly patients with poor perfusion status being more likely to be VT.

Ventricular ectopics

The presence of more than the odd ventricular ectopic used to be a trigger to reach for an antiarrhythmic drug because of a fear of the 'R on T phenomenon'. This brought with it the consequences of decreased contractility and

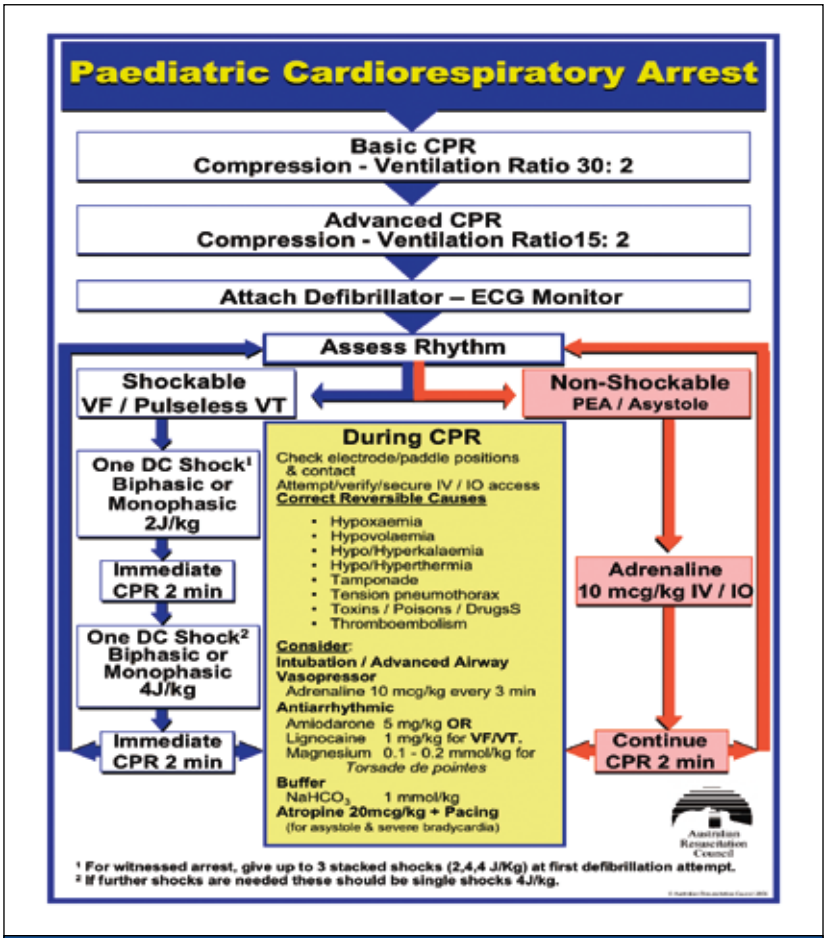

Figure 9. Child cardiorespiratory arrest
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sometimes a proarrhythmic effect of the drug. Ectopics are better treated with attention to oxygenation and perfusion and preparation to defibrillate should any VF that occur. Oxygen is a wonderful antiarrhythmic that does not decrease myocardial contractility.

Cardiac arrest

Patients presenting in cardiac arrest will have an underlying rhythm of:

- VF/VT which is treatable with defibrillation
- pulseless electrical activity (PEA) which cannot be treated with defibrillation and where an underlying cause should be sought, or
- asystole which will not respond to defibrillation but can occasionally be encouraged into a more favourable rhythm with oxygen and CPR with adrenaline.

If a patient is in cardiac arrest, the approach is to establish effective resuscitation and early defibrillation. The Australian Resuscitation Council *Basic and advanced life support guidelines* (www.resus.org.au) are described in *Figures 7–9*. The key issues are a focus on minimal gaps in the chest compression rhythm and a steadily escalating level of adjuncts.

Basic life support

Once cardiac arrest has been observed and the airway confirmed as clear with no respiratory effort, two gentle breaths are administered. Try not to overinflate the stomach as this may cause reflux and airway soiling. The pulse check is only one of the signs of life, and in a patient who has allowed two ventilations without objection, is probably not indicated. Compressions should be commenced as soon as possible at a rate of 100 per minute (ie. almost two per second) and at a depth of one-third of the chest anterior posterior measurement. A ratio of 30 compressions to two ventilations is appropriate for all adults and children regardless of the number of operators. In a specialist advanced life support setting, children can be managed with a 15:2 ratio and neonates with a 3:1 ratio. However, a ratio of 30:2 will suffice in most circumstances encountered outside a specialist unit.

Defibrillation and drugs

Defibrillation should occur as early as possible and confined to a single shock at a high energy setting. This means 360 J in an older monophasic defibrillator, and 200 J in a modern biphasic defibrillator. Children receive 2 J/kg followed by 4 J/kg rounded to the next higher setting, regardless of the defibrillator type.

Once the shock has been delivered it is important to go straight back to compressions and not waste time watching the rhythm develop. After 2 minutes of CPR (which will be 5 cycles of 30:2) it is time to see what rhythm the defibrillation produced. No harm will be done to the heart if compressions are delivered on top of a developing rhythm. 1 mg of IV adrenaline is given every 3 minutes to maintain peripheral vascular tone.

Advanced airway techniques are used as soon as possible, however if intubation is used there should not be an interruption to compressions of more than 20 seconds and a maximum of two attempts is reasonable. Failing this, a laryngeal mask airway provides an airway that is far superior to an ordinary oropharyngeal airway.

If the patient still remains in VF despite repeated cycles of CPR, defibrillation, adrenaline and improved oxygenation/ventilation then an anti-arrhythmic drug is appropriate and the preferred choice is amiodarone 300 mg IV. If amiodarone is not available, lignocaine 100 mg IV is an acceptable alternative. Persistent VF in this situation can only be treated with more CPR and oxygen once an adequate dose of antiarrhythmic has been given.

PEA and asystole

The presence of PEA should prompt a review of the reversible causes – the 'Hs and Ts' (*Figure 8*):

- hypoxia
- hypovolaemia
- hypo-/hyper-thermia
- hypo-/hyper-kalemia and other metabolic disorders
- tamponade
- tension pneumothorax
- toxins, and
- thrombosis (pulmonary or coronary).

Asystole can be treated with oxygen CPR and adrenaline. It is acceptable to give at least 1 mg of atropine in this situation as vagal tone can produce asystole.

Postresuscitation

Postresuscitation care focuses on maintaining a safe airway, adequate ventilation and oxygenation, supporting perfusion, and facilitating transport to hospital.

Conclusion

The management of arrhythmias is not as daunting as initially seems. For rhythms that are still perfusing, administration of high concentrations of oxygen, attention to optimising preload (JVP) and patience are good first responses which then allow plenty of time to share the problem with a cardiologist before embarking on drug therapy. For rhythms not perfusing, the response is as per a standard cardiac arrest following the ARC directions included in this article.

Conflict of interest: none declared.

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