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Introduction

Heart rhythms associated with cardiac arrest can be divided into two groups: ventricular fibrillation / pulseless ventricular tachycardia (VF/VT) and other rhythms. The latter includes both asystole and pulseless electrical activity (PEA), also known as electromechanical dissociation (EMD). The principle difference in the management of these two groups of arrhythmias is the need for attempted defibrillation in those patients with VF/VT. Subsequent actions, including chest compressions, airway management and ventilation, venous access, the administration of epinephrine (adrenaline) and the identification and correction of contributing factors, are common to both groups. The universal algorithm for the management of cardiac arrest can be used in conjunction with both manual and, with a minor modification, automated external defibrillators (AEDs).

Although the algorithm is applicable universally, cardiac arrest may occur as a result of special circumstances, for example: hypothermia, drug overdose and submersion. In these cases additional interventions may be required.

Ventricular Fibrillation / Pulseless Ventricular Tachycardia

In adults, the commonest rhythm at the time of cardiac arrest is VF, which may be preceded by a period of VT or even supraventricular tachycardia (SVT). The majority of those who survive a cardiac arrest come from this group. To maximise the success of resuscitation from either of these two rhythms, a shock must be delivered promptly. The chances of successful defibrillation decline by 7 - 10% for each minute that the arrhythmia persists, as myocardial
energy reserves are depleted. This process can be slowed, but not halted, by effective basic life support (BLS). Therefore, the patient’s rhythm should be determined at the earliest opportunity (using the monitoring electrodes or paddles of a manual defibrillator, an automated defibrillator, or attaching a dedicated ECG machine) and, if indicated, a shock delivered as soon as possible. Basic life support should be started if there is any delay in obtaining a defibrillator, but must not delay shock delivery. If the arrest was witnessed or monitored, and a defibrillator is not immediately to hand, a single precordial thump should be administered.

**Attempted defibrillation**

Up to three shocks are given initially with energies of 200 J, 200 J, 360 J (or their equivalent when using defibrillators with alternative waveforms). When using manual defibrillators, if more than one shock is required, the paddles are best left in position on the patient’s chest while the defibrillator is recharged, at the same time observing the ECG monitor for any changes in the rhythm. When monitoring with gel pads and defibrillator paddles, be aware of the possibility of spurious asystole. When all three shocks are required, the aim should be to administer these in less than one minute. The lone rescuer can increase the defibrillator energy level using a control on the paddles (if available), or by returning one paddle to the defibrillator and using the free hand to alter the energy setting. After each shock or sequence of three shocks is delivered, the carotid pulse should be palpated only if the waveform changes to one usually capable of providing a cardiac output (including ventricular tachycardia).

After delivering a shock there is often a delay of a few seconds before an ECG display of diagnostic quality is obtained. Successful defibrillation is followed usually by at least a few seconds of true asystole (electrical stunning). Furthermore, even when a rhythm normally compatible with a cardiac output is obtained, there is often a period of temporary impairment in myocardial contractility (myocardial stunning), resulting in a pulse that is weak and difficult to palpate. It is therefore important not to make a diagnosis of PEA automatically after successful defibrillation. For this reason the algorithm indicates only one minute of CPR before reassessing the rhythm and making a further pulse check. During this one minute epinephrine should not be administered, as this may be detrimental if a perfusing rhythm has been established.

For defibrillators using a monophasic waveform, the rationale for starting defibrillation at 200 J is that an initial shock of this energy will cause little myocardial injury and in most recoverable situations is adequate to defibrillate the patient successfully. The second shock is also at 200 J, as the first shock reduces the transthoracic impedance, thereby increasing the energy reaching the heart. Third and subsequent shocks are given at 360 J. Having restored a spontaneous circulation, if VF/pulseless VT recurs, the algorithm is applied again from the beginning, i.e., the first shock is 200 J. Some defibrillators now use alternative waveforms, most commonly biphasic, but the optimal energies for this type of waveform have yet to be determined. However, repeated biphasic shocks at less than 200 J have equivalent or higher success for defibrillation than defibrillators delivering monophasic waveforms of escalating energy (200 J, 200 J, 360 J).
Chest compressions, airway and ventilation

If ventricular fibrillation persists after the initial three shocks, the best chance of restoring a perfusing rhythm still lies with defibrillation, but myocardial and cerebral viability must be maintained with chest compressions and ventilation of the lungs (CPR). One minute of CPR (with a compression to ventilation ratio of 15:2) is undertaken during which reversible causes should be considered and, if identified, corrected. A check should be made of the electrode/defibrillating paddle positions and contacts, and the adequacy of the coupling medium (for example, gel pads).

The patient’s airway should be secured. Tracheal intubation provides the most reliable airway but only if the healthcare provider is properly trained and has adequate ongoing experience with the technique. Acceptable alternatives include insertion of a laryngeal mask airway (LMA) or a Combitube. The aim is to ventilate the patient’s lungs and deliver the highest possible concentration of oxygen, preferably 100%. Once the patient’s trachea has been intubated, chest compressions, at a rate of 100 min⁻¹, should continue uninterrupted (except for defibrillation or pulse checks when indicated), and ventilation continued at approximately 12 breaths min⁻¹. A pause in the chest compressions allows the coronary perfusion pressure to fall substantially. On resuming compressions there is some delay before the original coronary perfusion pressure is restored. Thus, chest compressions uninterrupted for ventilation result in a substantially higher mean coronary perfusion pressure. A correctly placed Combitube should also make it possible to provide asynchronous compressions and ventilation.

If a laryngeal mask airway has been inserted, attempts can be made to perform continuous chest compressions, uninterrupted during ventilation. If the seal pressure of the LMA around the larynx is high enough it should be possible to produce adequate ventilation of the lungs. Gas leaking from between the LMA and larynx will tend to pass up through the patient’s mouth rather than being forced into the stomach (with the associated risk of regurgitation). If excessive gas leakage results in inadequate ventilation of the patient’s lungs, chest compressions will have to be interrupted to allow for ventilation. Use a 15:2 compression:ventilation ratio.

Intravenous access and drugs

Intravenous access should be established if this has not been achieved already. The central veins provide the optimal route as they allow drugs to be delivered rapidly into the central circulation. However, the technique of central venous catheterisation has a variety of complications, some of which are potentially life-threatening. Peripheral venous cannulation is quicker, easier to perform, and safer. Ultimately, the route chosen will depend upon the skills and equipment available. Drugs administered by the peripheral route must be followed by a flush of at least 20 ml of 0.9% saline to assist their delivery to the central circulation. Epinephrine is administered, 1 mg by the intravenous route or 2-3 mg via the tracheal tube. Epinephrine given by the tracheal route should be diluted to at least 10 ml with sterile water. The solution in pre-filled syringes are acceptable for this purpose. Administration should be followed by 5 ventilations to disperse the drug into the peripheral bronchial tree to aid absorption. The role of epinephrine is to improve the efficacy of CPR; its alpha-adrenergic actions cause vasoconstriction, which increases myocardial and cerebral perfusion pressure. Vasopressin, in a single intravenous dose of 40
units, has been proposed as an alternative to epinephrine in cases of VF/pulseless VT refractory to three initial shocks. Further evidence is required before a firm recommendation on the use of this powerful vasopressor can be made.

The evidence supporting the use of any antiarrhythmic drugs in VF/VT is weak and no pharmacological interventions for cardiac arrest have yet been found to improve survival to hospital discharge. However, amiodarone should be considered, following epinephrine, to treat shock-refractory cardiac arrest due to VF or pulseless VT. It can be considered as early as before delivery of the fourth shock provided it does not delay delivery of this shock. Amiodarone 300 mg (made up to 20 ml with dextrose, or from a prefilled syringe) may be administered into a peripheral vein. A further dose of 150 mg may be given for recurrent or refractory VT/VF, followed by an infusion of 1 mg min⁻¹ for 6 hours and then 0.5 mg min⁻¹, to a maximum daily dose of 2 g; this maximum dose is larger than the current European datasheet recommendation of 1.2 g. Magnesium (8 mmol) should be given for refractory VF if there is any suspicion of hypomagnesaemia (e.g., patients on potassium losing diuretics).

Lidocaine should not be given if the patient has received amiodarone but may be used as an alternative if amiodarone is not available. Procainamide is another alternative to amiodarone or lidocaine for refractory VF. It is given at 30 mg min⁻¹ to a total dose of 17 mg kg⁻¹. The necessity for this relatively slow rate of infusion makes procainamide a less favourable option.

If the patient remains in VF after one minute of CPR then three further shocks, each at 360J (or biphasic equivalent) are administered, and the monitor is checked between each. The interval between the third and fourth shocks must not exceed one minute (even if the airway has not been secured and/or IV access obtained). Poor electrode or paddle application will reduce the chances of successful defibrillation.

The loop of the left hand side of the algorithm is continued with each sequence of three shocks being followed by CPR for one minute. Further attempts to secure the airway or venous access can be made if necessary. Epinephrine 1 mg is given every three minutes.

The use of bicarbonate (50 mmol) may be considered if the arterial pH is less than 7.1 (H⁺ > 80 mmol l⁻¹) and/or if the cardiac arrest is associated with a tricyclic overdose or hyperkalaemia. Further doses will depend upon the clinical condition and result of repeated blood gas analysis. In those situations where blood gas analysis is not possible, it is reasonable to consider sodium bicarbonate after 20-25 minutes, particularly if resuscitation may have been suboptimal or delayed. Bicarbonate will result in the generation of carbon dioxide, which may worsen intracellular acidosis; for this reason, an increase in minute ventilation may be necessary.

In persistent VF, the position of the paddles can also be changed to antero-posterior, and if easily available, a different defibrillator and paddles can be tried. Finally, it is important to ensure that the potentially reversible causes (4 H’s and 4 T’s) have been eliminated as any of these can reduce the chances of successful defibrillation.

The number of times the loop is repeated during any individual resuscitation attempt is a matter of clinical judgement having regard to the circumstances and the perceived prospect of a successful outcome. If it was considered appropriate to start resuscitation, it is usually considered worthwhile continuing
as long as the patient remains in identifiable VF/VT.

Non-VF/VT rhythms

The outcome from non VF/VT rhythms is relatively poor unless a reversible cause can be found and treated effectively. If apparent asystole or PEA occurs directly after delivery of a shock the rhythm and pulse should be rechecked after just one minute of CPR and before any further drugs are given. If asystole or PEA is confirmed, appropriate drugs are given and a further two minutes of CPR is given to complete the loop. A temporarily poor cardiac output, due to myocardial stunning, may result in impalpable pulses and the incorrect diagnosis of PEA. After a minute of CPR, the cardiac output might recover spontaneously and, at this stage, further epinephrine could be detrimental. The short delay before the monitor display recovers after delivering a shock could be misinterpreted as "asystole". Furthermore, after the delivery of a shock, monitoring through gel pads may result in spurious asystole being displayed. This is more likely to occur in the presence of high chest impedance and with an increasing number of shocks delivered through the same gel pads. If defibrillator paddles and gel pads have been used for "quick-look" monitoring, and "asystole" is displayed after delivery of a shock, the rhythm must be confirmed immediately with monitoring leads.

Asystole

It is essential that the correct diagnosis is made and, most importantly, that VF is not missed. Asystole must be confirmed by:

- Checking that the leads are attached correctly
- Checking the gain
- Viewing the rhythm through leads I and II

If there is any doubt, treatment for VF should be started, as the risks of not treating VF, with its greater potential for a successful outcome, are greater than for three unnecessary shocks administered to an asystolic heart.

Chest compressions and ventilation should be undertaken for three minutes with each loop (or one minute if directly after a shock), during which the airway can be secured, intravenous access obtained, and the first dose of epinephrine given. Atropine, 3 mg intravenously or 6 mg via the tracheal tube (in a volume of 10-20 ml) can be given to provide total blockade of the vagus nerve.

Whenever a diagnosis of asystole is made, the ECG should be checked carefully for the presence of P waves or slow ventricular activity because this may respond to cardiac pacing. Consideration should be given also to external cardiac percussion for patients in whom electrical pacing is to be performed, but where a delay will occur before it is achieved. Repeated precordial blows can be used to stimulate the myocardium (percussion pacing). Each blow is delivered lateral to the lower left sternal edge, using less force than a precordial thump, and at a rate of about 70 min⁻¹.

During the treatment of asystole or PEA, if the rhythm changes to VF, the left side of the algorithm is followed. Otherwise, CPR is continued and epinephrine is administered every three minutes. Any reversible or aggravating factors should be identified and treated promptly. The administration of epinephrine in single doses greater than 1 mg is no longer recommended.

Pulseless electrical activity (PEA)

This condition comprises the clinical signs of a cardiac arrest with an ECG
rhythm compatible with a cardiac output. The patient’s best chance of survival will be by prompt identification and treatment of any underlying cause. Potential causes can be categorised into two main groups, which are listed in the universal algorithm. Resuscitation should be continued while these conditions are sought. CPR is started immediately; the airway and ventilation managed as appropriate and intravenous access obtained. Epinephrine 1 mg intravenously is administered every three minutes. The administration of epinephrine in single doses greater than 1 mg is no longer recommended. If PEA is associated with a bradycardia (< 60 min⁻¹) atropine, 3 mg intravenously or 6 mg via the tracheal tube, should be given.

Potentially reversible causes

During any cardiac arrest, potential causes or aggravating factors for which specific treatment exists should be considered. For ease of memory, these are divided into two groups of four based upon their initial letter - either H or T:

The four ‘Hs’

- Hypoxia
- Hypovolaemia
- Hyperkalaemia, hypocalcaemia, acidaemia
- Hypothermia

The four ‘Ts’

- Tension pneumothorax
- Cardiac tamponade
- Toxic substances or therapeutic substances in overdose
- Thromboembolic or mechanical obstruction (e.g., pulmonary embolus)

Further reading


Wenzel V, Idris AH, Dörjes V, Nolan JP, Parr MJA, Gabrielli A, Stallinger A,

ALS algorithm

The ALS algorithm is available in Adobe PDF format.