

## Guidelines for advanced life support

### A Statement by the Advanced Life Support Working Party of the European Resuscitation Council, 1992

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#### INTRODUCTION

The European Resuscitation Council was established officially in 1990 as a multidisciplinary group of individuals from many countries and representing wide interests. Its primary aim was to save human life by improving standards of resuscitation in Europe and by coordinating the activities of European organisations with a legitimate interest in cardiopulmonary resuscitation. Important related secondary objectives included the following 'To produce guidelines and recommendations appropriate to Europe for the practice of basic and advanced cardiopulmonary and cerebral resuscitation.'

European guidelines for basic and advanced cardiopulmonary resuscitation have not been published previously, because no organisation had the remit to do so. The European Resuscitation Council, however, receives support from the European Society of Cardiology, the European Academy of Anaesthesiology, the European Society of Intensive Care Medicine, many national societies representing the same disciplines, national resuscitation councils and also voluntary aid societies. It is able to draw on a wide range of expertise from all European countries and is the appropriate body to produce guidelines for use within our continent that are based on scientific data derived from all parts of the world. In the recent past, most European countries have used guidelines developed by the American Heart Association<sup>1</sup>, but international cooperation in resuscitation procedures began in 1986 with the publication of guidelines that had been produced jointly by the Resuscitation Council of the United Kingdom<sup>2</sup> and by a group representing five Nordic countries.

This article is concerned only with Advanced Life Support and only with the immediate period of the cardiac arrest. A nucleus of 14 experts from 11 countries was set up to prepare a draft document based on the best available information. Individual expertise and databases were supplemented from three additional sources. First, the scientific papers that were used to draw up the 1986 United Kingdom/Nordic guidelines were updated and published in 1991<sup>3–10</sup>. Secondly, a new set of

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ten manuscripts<sup>11-20</sup> have been prepared, by the nucleus group and other colleagues, complementing the 1991 publications and relevant to the scientific and ethical knowledge and concepts from which the new decisions have been derived. Thirdly, many of the nucleus group were privileged to attend by invitation the American Heart Association 1992 National Conference on Cardiopulmonary Resuscitation and Emergency Cardiac Care and were able to hear the latest views from the American continent. (Basic Life Support has attracted the same valuable transatlantic liaison and is considered elsewhere in this issue.) The new guidelines have been derived independently, but we wish to emphasise that the European Resuscitation Council enjoys excellent relations with its longer established partner in the United States and looks forward to even closer cooperation on guidelines and other matters in future years.

After the initial drafting by the nucleus group, the document was submitted to wider scrutiny. In 1991, the national societies with affiliation to the European Resuscitation Council were invited to nominate representatives to take part in the process, and many did so. Their comments were considered and — where appropriate — modifications were made, before discussion with and finally approval by the Executive Committee of the Council in September 1992.

We are presenting at this stage a document for those who have a major interest in resuscitation, as an introduction to the new guidelines. An additional document will be prepared for wider dissemination, with greater emphasis on implementation and on broader aspects of advanced life support such as post resuscitation care.

#### THE GUIDELINES: UNDERLYING PRINCIPLES FOR DECISIONS

*The first underlying principle* accepted by the nucleus group was a willingness to make important changes. In the past, much emphasis was placed on the role of drug therapy in resuscitation from cardiac arrest. Treatment with antiarrhythmic drugs such as lidocaine (lignocaine) and with alkalisng agents such as sodium bicarbonate became hallowed by tradition, even though scientific evidence for their efficacy has never been convincing. There has been a widespread perception that the status quo should remain unless conventional drug therapy can be proved to be unhelpful — rather than accepting only treatment for which there is real evidence of worth.

The scientific evidence against the value of antiarrhythmic drugs and sodium bicarbonate has been stated in papers<sup>3,14,18</sup> prepared as supporting material for the new guidelines and can be summarised briefly. Lidocaine can be of value in the prevention of ventricular fibrillation, but experimentally more energy is needed to reverse fibrillation in the presence of the drug than without it. Reliable clinical data on defibrillation thresholds do not exist. Belief in the efficacy of lidocaine for restoring coordinated rhythm is reinforced when a defibrillating shock is seen to be effective after the drug when shocks beforehand were not. But this evidence is negated by knowledge that successive shocks are — to a degree — subject to the laws of probability in determining whether or not they will restore coordinated rhythm<sup>21</sup>. Successful adjuvant measures can do no more than improve the odds of a favourable outcome, but a favourable outcome remains possible even if the odds have been somewhat reduced by an adverse intervention. Thus defibrillation after lidocaine is

not compelling evidence in favour of the drug even when successive shocks beforehand had been ineffective.

Alkalisating agents offer another example of unsafe extrapolation. In the past sodium bicarbonate has been believed to be valuable because acidosis complicates cardiac arrest and acidosis can perpetuate arrhythmias. Several problems are associated with this simplistic argument. First, acidosis — as measured by arterial blood gas measurements — develops less rapidly than had been thought during the performance of effective cardiopulmonary resuscitation, though the more rapid change of central venous blood may be of greater significance<sup>22</sup>. Secondly, the measurement of arterial, or even central venous, blood gases probably bears little relationship to myocardial intracellular values: indeed the passage of carbon dioxide across the cell membrane can lower intracellular pH whilst the alkaline residue of the metabolised bicarbonate increases the extracellular pH. Thirdly, the levels of pH at which myocardial contractility diminishes and at which arrhythmias may be provoked is not agreed. Fourthly, we have no good empirical evidence in favour of sodium bicarbonate during the early stages of cardiac arrest. Fifthly, sodium bicarbonate may induce hyperosmolarity causing a fall in aortic diastolic pressure and therefore a fall in coronary perfusion pressure. Finally the development of an iatrogenic alkalosis may be even less favourable than acidosis.

*The second principle* underlying the new guidelines is the paramount importance of minimum delay in the administration of defibrillating shocks. Thus the role of chest compression during advanced life support has also been modified to afford greater opportunity for electrical defibrillation as early as possible in the course of the arrest. This is also unconventional and calls for justification. Whilst successful defibrillation can occur for several minutes after cardiac arrest, observational data<sup>23</sup> suggest that the chances of success and also of a long-term favourable outcome are optimal for as little as 90 s, but it declines thereafter as the internal biochemical milieu of the heart deteriorates. The application of an appropriate shock should be delayed only if the internal milieu can be improved or if attention to peripheral flow — particularly to the brain — has become a critical necessity. Basic life support is unlikely to improve the internal milieu of the heart, but can be expected only to slow further deterioration. This important concept is supported by the clinical observation that defibrillation becomes more difficult with time despite effective basic life support and by the experimental observation that myocardial pH continues to fall during precordial compression<sup>24</sup>. On the other hand, the lack of cerebral flow is always a matter for concern. Thus we face a conflict of priorities: the need for prompt defibrillating shocks against the need for continuing chest compression and ventilation. But we believe that the over-riding considerations initially are that restoration of an effective spontaneous cardiac output provides the only means of reversing the metabolic sequelae of critical ischaemia and that this should be achieved as rapidly as possible.

The arguments for the initial priority of defibrillation can be summarised. First, the prospects of success decrease relatively rapidly over a few minutes after cardiac arrest. Secondly, basic life support is unlikely to improve the odds of successful defibrillation: its value is in maintaining some cerebral perfusion and in slowing myocardial deterioration. Thirdly, modern defibrillators have very rapid charge

times: three shocks of appropriate energy levels can be given within 30 seconds by a trained and well equipped team. But a balance in priorities is necessary. For resuscitation attempts with older equipment or a less skilled team, or for any rescue attempts once the likelihood of very early defibrillation has passed, the emphasis must change — with greater attention to maintaining the best achievable myocardial and cerebral perfusion as prolonged resuscitation attempts continue.

A *third principle* influenced the nucleus group. Great store was set on the value of simplicity in the algorithms and the need to reduce decision making to a minimum. An example is afforded by the precordial thump. This manoeuvre is potentially useful only very early in the course of an arrest<sup>25</sup> — but it takes only a few seconds and the pro-arrhythmic effect is both unusual and irrelevant in an arrest situation. Thus it has been included as part of the standard procedure irrespective of delays.

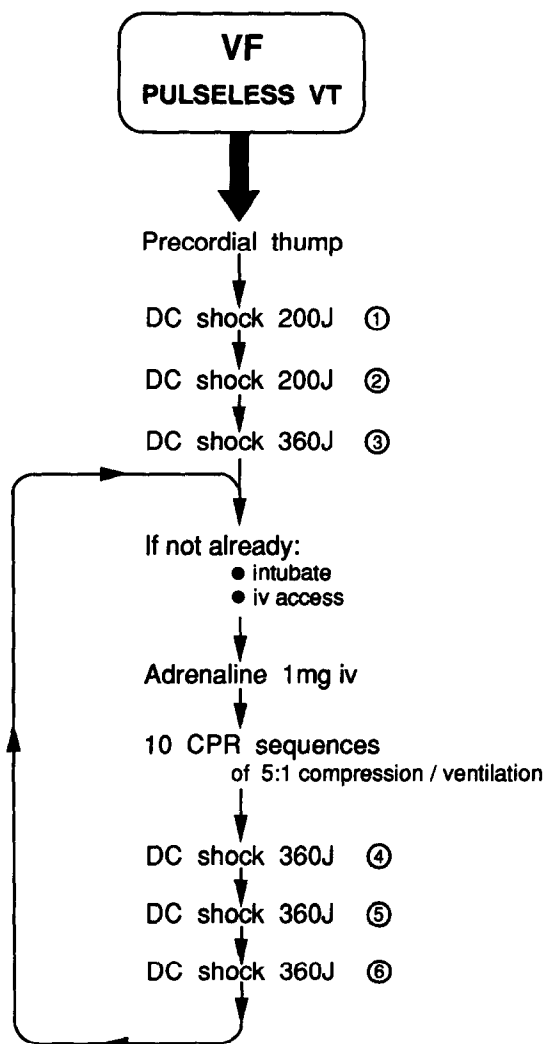
The group was influenced too by the desirability of making guidelines as similar as possible for manual and automated defibrillators. Automated defibrillators cannot perform a diagnostic test for heart rhythm during basic life support and ‘hands off’ during the early shock phase is mandatory. The group — having regard to all the considerations presented above — saw no disadvantage in recommending similar procedures for manual defibrillation, provided the initial three-shock algorithm can be completed with minimal delay. We believe that the new similarity between algorithms for ventricular fibrillation and for asystole, is an additional practical advantage, gained without compromising the need for sensible clinical decisions.

#### GUIDELINES: DETAILS OF ALGORITHMS

Cardiac arrest occurs with four underlying disorders of heart rhythm: ventricular fibrillation, pulseless ventricular tachycardia (that usually degenerates into ventricular fibrillation), asystole, and electromechanical dissociation. The first two have identical implications for treatment, so that protocols have to be considered for only three treatment schedules. Of these, the protocol (or algorithm) for ventricular fibrillation is by far the most important, since this arrhythmia is the most common immediate cause of sudden cardiac death and it is also the most amenable to treatment.

##### *Notes on algorithm for ventricular fibrillation or pulseless ventricular tachycardia (Fig. 1)*

1. In out-of-hospital cardiac arrest and in most hospital settings, basic life support will usually have been initiated before the algorithm for ventricular fibrillation or pulseless ventricular tachycardia is commenced. But if the circumstances are such that immediate defibrillation is possible — for example in a high dependency area with a rapidly charging defibrillator immediately available — only a precordial thump should be given before definitive electrical treatment. If the first three successive shocks at 200 J, 200 J and 360 J can be delivered very quickly (within 30–45 s) then the sequence should not be interrupted by basic life support. If the time to charge a manual defibrillator or to confirm that the rhythm is still ventricular fibrillation is likely to be unduly prolonged because of old equipment or inex-



- Notes:
- (i) The interval between shocks 3 and 4 should not be >2 mins.
  - (ii) Adrenaline given during loop approx every 2 - 3 mins.
  - (iii) Continue loops for as long as defibrillation is indicated.
  - (iv) After 3 loops consider:
    - an alkalisising agent
    - an antiarrhythmic agent.

**Fig. 1.** Algorithm for ventricular fibrillation or pulseless ventricular tachycardia.

perience, one or two sequences of basic life support (five chest compressions to one ventilating breath) should be administered between shocks.

2. The sequence of energies used for the first three shocks is conventional. The rationale for starting with 200 J was originally based in part on the long charge time for higher energy levels. This is no longer relevant with modern equipment, but the balance between the probability of success and the risk of damage remains pertinent<sup>17</sup>. A delivered energy level as high as 360 J improves the odds of restoring coordinated rhythm, but also causes some myocardial damage. An initial level of 200 J probably causes little damage and in most recoverable situations is adequate to achieve success. A repeat at 200 J is reasonable for two reasons: first, the original shock lowers impedance and therefore increases the energy that reaches the heart from a subsequent shock, and secondly the odds of success depend upon dynamic variables (especially moment-to-moment changes in the waveform and vectors of the fibrillatory activity) as well as on the energy level.

3. If three shocks have been unsuccessful, the prospects for recovery are poor. But they are not hopeless and attempts should continue if this strategy is appropriate on clinical grounds. As restoration of coordinated rhythm will inevitably be delayed if it occurs at all, the priority must change to preservation of cerebral function by the best possible basic life support, whilst delaying as far as possible further myocardial deterioration.

4. At this point a brief attempt should be made to intubate the patient and gain intravenous access — if this has not been achieved already. In the hospital setting these procedures will usually be attempted by two different individuals. But neither procedure should be allowed to cause undue delay either in the continuation of basic life support or in the administration of further shocks. The person who is in charge of the resuscitation procedure should allow a limited time — perhaps not more than 15 s — before chest compression and ventilation are continued, using 10 sequences of five compressions to one breath, whilst preparations are made for a new set of shocks — now all to be at 360 J. Adrenaline will be administered before the shocks (provided intravenous or endotracheal access is available), but there is no need to delay their delivery for drug treatment to become effective: the purpose of adrenaline is to increase the efficacy of basic life support<sup>15,26</sup>, not as an adjuvant to defibrillation.

5. Thereafter, the loop is repeated. If intubation and/or intravenous access have not already been achieved or if either needs revision, each loop presents an opportunity for another attempt — but always without delaying the basic life support procedures or the delivery of shocks.

6. During each loop, 1 mg adrenaline should be given intravenously. This implies a dose of up to 1 mg every 2 min if shocks are given without delay. This is not considered excessive<sup>15</sup>, having regard to the high spontaneous concentrations of adrenaline during a cardiac arrest and the need to maintain or increase these to improve peripheral blood flow during chest compression. We recognise that drugs given intravenously — even through a central line — may take several minutes to achieve an effect. But nothing is gained by deferring further shocks, because defibrillation is still the only intervention capable of restoring a spontaneous circulation: the prospects of success become progressively poorer and other interventions can do no more than slow the decline in the odds of success. If intravenous access cannot

be gained in a patient who is intubated, the endotracheal route can be considered for adrenaline or for atropine<sup>13</sup>. Doses two to three times higher than conventional intravenous doses are used, though the pharmacodynamics of the drugs cannot be predicted accurately with this route because of the many additional and uncontrollable variables that are introduced.

7. After every three loops other drugs may appropriately be used. An alkalinising agent (such as sodium bicarbonate up to 50 mmol (mequiv.)) is considered at this stage when acidosis may be a problem even with adequate ventilation and compression. Thereafter an alkalinising agent will be considered again every three loops, but ideally given only in the knowledge of the arterial or central venous pH and bicarbonate (or alternatively of base deficit). In addition to sodium bicarbonate, antiarrhythmic agents may be given every three loops within the guidelines, but — in the light of existing evidence — their use is not mandatory. Lidocaine, bretylium and amiodarone have all been advocated for this now desperate situation.

8. The use of calcium, magnesium, or potassium salts for special purposes during the management of cardiac arrest has also been advocated. The use of the algorithm is not intended specifically to deny their use either for known deficiencies in any patient or (after the first three loops) for their empirical use. But no evidence exists at present in favour of these or other agents, and calcium is implicated in ischaemic tissue injury<sup>27</sup>.

9. The number of loops to be used in any individual resuscitation is a matter of clinical judgment, having regard to the clinical situation and the perceived prospect still of a successful outcome. A resuscitation attempt may reasonably last for any time from 10 min to an hour. Resuscitation that was started appropriately should not usually be abandoned whilst the rhythm is still recognisable ventricular fibrillation. The development of persistent asystole is a useful indication that prospects for success are slight. Few situations would call for efforts continued for over 1 h, but the special situation of cardiac arrest with hypothermia should always be considered when the setting suggests the possibility of cooling.

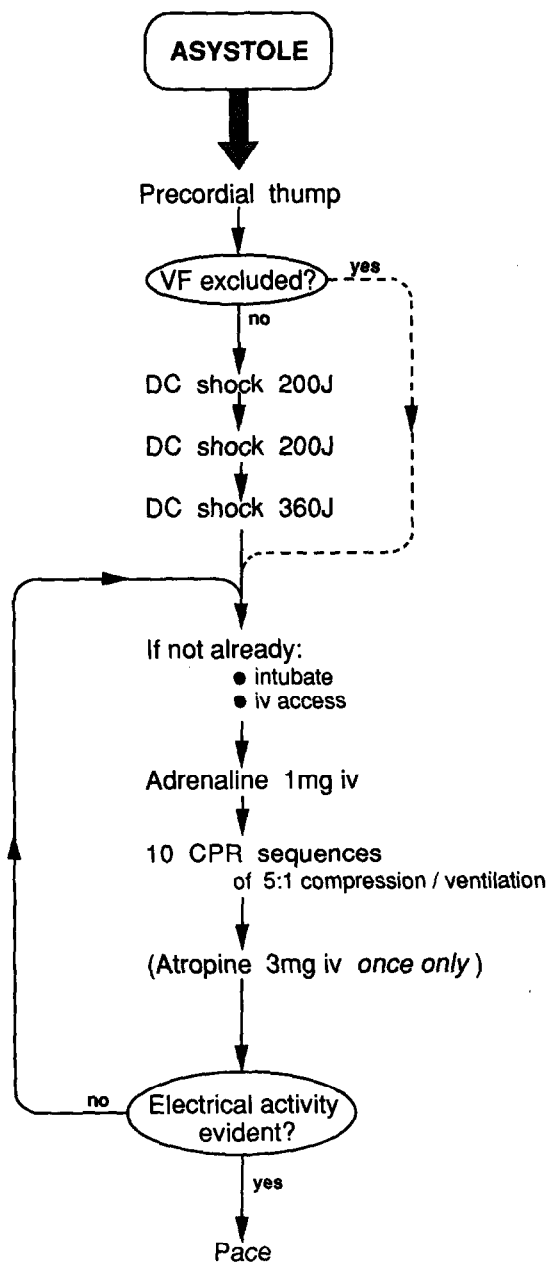
10. Before attempts at defibrillation are abandoned, the possibility of a change of paddle position and even a change of defibrillator should be considered.

11. Open chest cardiac massage is only rarely indicated in advanced life support for a patient with a 'medical' cardiac arrest<sup>10</sup>, though emergency thoracotomy in cases of trauma is a well validated procedure for which clear indications exist<sup>11</sup>. Further studies in man are warranted to delineate those groups of medical patients who are most likely to benefit from an open chest approach and to investigate the optimal timing of this intervention. Indications may exist where conventional chest compression may rapidly be seen to be ineffective as, for example, in a patient with tight aortic stenosis, or where defibrillation is intrinsically difficult as in some cases of drug toxicity or in severe hypothermia. At present, however, no firm recommendations can be made.

#### *Notes on algorithm for asystole (Fig. 2)*

1. The inclusion of a precordial thump in the algorithm for asystole is not controversial, though it is unlikely to have any relevance after a period of basic life support.

2. The prospects of recovery from asystole are poor except in cases of trifascicular



Note: If no response after 3 cycles consider high dose adrenaline: 5mg iv.

Fig. 2. Algorithm for asystole.



block (where P waves may be seen), in cases that are evolving from extreme bradycardia, or in cases where the rhythm is a transient phenomenon after defibrillation. But an important additional consideration is the possibility of mistaken diagnosis. This may not be uncommon. A waveform of ventricular fibrillation may not be seen or not recognised for a number of reasons including equipment failure, excessive artefact, uncontrollable movement as in an aircraft, or an incorrect gain setting. Even an unusually directional vector of a fibrillation waveform that happens to be perpendicular to the sensing electrode can lead to ventricular fibrillation being mistaken for asystole.

Because ventricular fibrillation is so readily treatable and because treatment is so much more likely to be successful than is the management of asystole, it is usually worth spending a short time treating fibrillation that may or may not be present. Little harm will result and the delay in the use of other measures should be slight. The possibility of a successful resuscitation that would not otherwise be possible is real.

*Warning:* If the waveform is indeed that of asystole or fine ventricular fibrillation, defibrillation in the automatic mode may be unsuccessful because the machine will determine that a shock is inappropriate. Time should not be wasted by persevering with attempts in this situation.

3. The defibrillation sequence, if used, is followed by intubation, securing intravenous access and the administration of drugs. The rationale for the adrenaline — as in the defibrillation algorithm — is to enhance basic life support. Atropine will counter any excess vagal tone although it brings no proven benefit in clinical practice. Atropine is used in a dose that blocks vagal tone fully under normal circumstances (3 mg)<sup>28</sup>, but only one dose is recommended. As in the previous algorithm, undue delay in the performance of basic life support must be avoided.

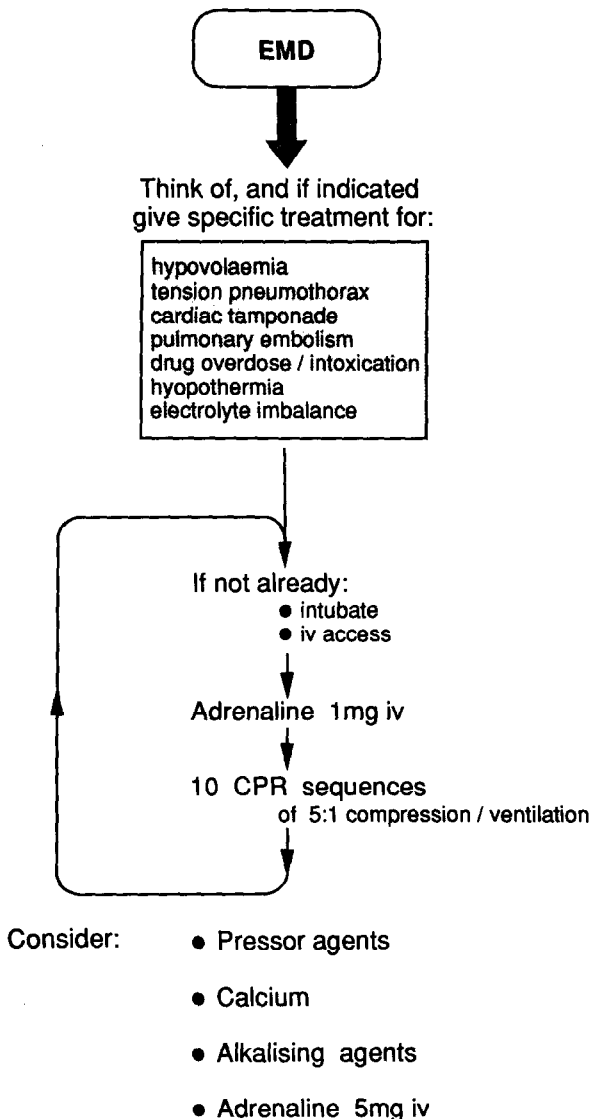
4. Once these steps have been taken, pacing should be considered, but only if electrical activity (P waves or occasional QRS complexes) has recently been present. The choice between pervenous or transcutaneous pacing as the initial strategy will depend in part on the local availability of equipment and skills.

5. In the absence of electrical activity further loops should be considered. These include an injection of 1 mg adrenaline and 10 cycles of basic life support as for the ventricular fibrillation protocol. If no response has been obtained after three cycles, the use of high dose adrenaline (5 mg) should be considered though its value is unproven<sup>15</sup>. Careful consideration should be given before undertaking prolonged resuscitation attempts in patients with asystole, having regard to the poor outcome. Recovery after 15 min of asystole is a very remote possibility, but the special situation of hypothermia must always be kept in mind.

#### *Notes on the algorithm for electromechanical dissociation (Fig. 3)*

1. Electro-mechanical dissociation implies continued electrical activity of the heart without mechanical activity. A definite diagnosis can rarely be made clinically because feeble contraction of the heart may produce no pulse, no detectable heart beat and no heart sounds. But both absence of mechanical activity and undetectable mechanical activity carry an equally poor prognosis except when they are transient phenomena during a cardiac arrest, or when there is a specific remediable cause.

2. Search for and recognition of specific and correctable causes of the clinical pic-



**Fig. 3.** Algorithm for electromechanical dissociation.

ture of electro-mechanical dissociation is therefore of prime importance. The principal ones are listed in the algorithm.

3. If no evidence exists for any of the specific causes, cardiopulmonary resuscitation should be continued with the usual associated procedures of intubation, establishing venous access, and adrenaline administration. No recommendation can be made for routine use of pressor agents, calcium chloride, alkalising agents, or high dose adrenaline though in some circumstances one or more of these measures may possibly be of value.

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