ELSEVIER

Resuscitation 37 (1998) 81–90

RESUSCITATION

The 1998 European Resuscitation Council guidelines for adult advanced life support

A statement from the Working Group on Advanced Life Support, and approved by the executive committee of the European Resuscitation Council

Colin Robertson (UK), Petter Steen (Norway), Jennifer Adgey (UK), Leo Bossaert (Belgium) *, Pierre Carli (France), Douglas Chamberlain (UK), Wolfgang Dick (Germany), Lars Ekstrom (Sweden), Svein A Hapnes (Norway), Stig Holmberg (Sweden), Rudolph Juchems (Germany), Fulvio Kette (Italy), Rudy Koster (Netherlands), Francisco J de Latorre (Spain), Karl Lindner (Austria), Narcisco Perales (Spain) [1]

1. Introduction

The publication of guidelines for advanced life support (ALS) by the European Resuscitation Council (ERC) in 1992 was a landmark in international co-operation and co-ordination [1]. Previously, individual countries or groups had produced guidelines [2] but for the first time an international group of experts produced consensus views based on the best available information. Since 1992, even wider international collaboration and support has occurred. In particular, the establishment of the International Liaison Committee on Resuscitation (ILCOR) has facilitated global co-operation and discussion between representatives from North America, Europe, Southern Africa, Australia, and most recently Latin America. The Advisory Statement produced in 1997 by ILCOR forms the basis for these guidelines [3].

The 1992 ERC guideline documents indicated that review would occur on a regular basis. Change is not advocated for its own sake, and is not warranted without convincing scientific or educational reasons. Education and its organisation is a process with a long latency, and it can be confusing and distracting for trainers and trainees if the message lacks consistency.

The ERC ALS working group recognised that the previous guidelines necessitated a level of rhythm recognition, interpretation and subsequent decision-making that some users found difficult. While automated external defibrillators (AED's) ease some of these problems, the 1992 guidelines were not specifically designed for these devices. These 1998 ALS guidelines are applicable to manual and automated external defibrillators. Decision making has been reduced to a minimum whenever possible. This increases clarity, while still allowing individuals with specialist knowledge to apply their expertise.

Changes in guidelines are only the first step in the process of care. Their implementation necessitates considerable effort. Training materials and methods may require modification, information must be disseminated and, perhaps most importantly, evaluation of efficacy is needed. For these purposes, reporting and publication of out-of-hospital and in-hospital cardiac arrest events using the Utstein templates [4,5] is strongly advised to provide objective outcome assessment.

The limitations of guidelines must be recognised. As always in the practice of medicine, words and flow charts must be interpreted with common sense and an appreciation of their intent. While much is known about the theory and practice of resuscitation, in many areas our ignorance is profound. Resuscitation practice remains as much an art as a science. Further, the interpretation of guidelines may differ according to the environment in which they are employed. We acknowl-

* Corresponding author.

[1] With contributions from: Raoul Alasino (CLAR), Vic Callanan (ARC), Richard Cummins (AHA), Walter Kloeck (RCSA) and Brian Connolly (HSFC)

PII S0300-9572(98)00035-5

edge that individual resuscitation councils may wish to customise the details while accepting that the guiding principles are universal. Any such changes must be approved by the ERC if they are to be regarded by this organisation as representing their official guidelines.

2. Precursors to cardiac arrest

In the so-called industrialised world, the commonest cause of adult sudden cardiac death is ischaemic heart disease [6–9]. Prevention of cardiac arrest is to be greatly preferred to post hoc treatment. The 'Management of peri-arrest arrhythmias' produced by the ERC in 1994 and updated in 1996 and 1998 provides guidance for treatment of arrhythmias which may lead to the development and recurrence of cardiac arrest in critical situations [10].

Small, but important, sub-groups of patients sustain cardiac arrest in certain special circumstances other than ischaemic heart disease. These include trauma, drug overdose, hypothermia, immersion, anaphylaxis, pregnancy, hypovolaemia. While this ALS algorithm is universally applicable, specific modifications may be required to maximise the likelihood of success in these circumstances.

3. Specific ALS interventions and their use in the ALS algorithm

3.1. Defibrillation

In adults, the commonest primary arrhythmia at the onset of cardiac arrest is ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT) [11–14]. The overwhelming majority of eventual survivors come from this group [15–18]. If the definitive therapy for these arrhythmias, defibrillation, can be implemented promptly, a perfusing cardiac rhythm may be restored and lead to long-term survival. The *only* interventions which have been shown unequivocally to improve long-term survival are basic life support and defibrillation. VF is an readily treatable rhythm, but the chances of successful defibrillation decline substantially with the passage of each minute [19,20]. The amplitude and waveform of VF deteriorate rapidly reflecting the depletion of myocardial high energy phosphate stores [21,22]. The rate of decline in success depends in part upon the provision and adequacy of BLS [23]. As a result, the priority is to minimise any delay between the onset of cardiac arrest and the administration of defibrillating shocks.

At present, the most commonly used transthoracic defibrillation waveform has a damped sinusoidal pattern. Newer techniques such as biphasic waveforms, or sequentially overlapping shocks producing a rapidly shifting electrical vector during a multi-pulse shock, may reduce the energy requirements for successful defibrillation [24–26]. Automated defibrillators that can deliver a current-based shock appropriate to the measured transthoracic impedance are available and are being evaluated. Their use may increase the efficacy of individual shocks, while reducing myocardial injury in patients with unusually high, or low, transthoracic impedance [27,28].

The use of groups of three shocks is retained, the initial sequence having energies of 200 J, 200 J and 360 J. The reasons for choosing 200 J as the energy for the first two shocks of conventional wave-form defibrillation have been presented [29]. Subsequent shocks, if required, should have energies of 360 J. If a co-ordinated rhythm has supervened for a limited interval, there is no strong scientific basis for deciding whether one should revert to 200 J or continue at 360 J. There is evidence that myocardial injury, both functionally and morphologically, is greater with increasing energies, but the comparative success rates for defibrillation attempts at this point with 200 J versus 360 J are unknown. Both strategies are therefore acceptable. Most AED's have an algorithm that does not revert to 200 J after a short period of non VF/VT. In this case, defibrillation should continue with 360 J instead of restarting the AED to allow a 200 J shock to be given. Alternative waveforms and energy levels are acceptable if demonstrated to be of equal or greater net clinical benefit in terms of safety and efficacy.

A pulse check is required after a shock (and should be prompted by an AED), only if the waveform changes to one compatible with cardiac output. Thus if VF, or VT with an identical waveform, persists after the first 200 J shock, the second shock at 200 J is given without a pulse check being carried out. If, in turn, this shock is unsuccessful, the third shock—this time at 360 J—is given. With modern defibrillators, charging times are sufficiently short for three shocks to be administered within 1 min.

Only a very small proportion of the delivered electrical energy traverses the myocardium during transthoracic defibrillation [30] and efforts to maximise this proportion are important. The commonest defects are inadequate contact with the chest wall, failure or poor use of couplants to aid the passage of current at the interface between the paddles and the chest wall, and faulty paddle positioning or size [31–34]. One paddle should be placed below the right clavicle in the mid-clavicular line and the other over the lower left ribs in the mid/anterior axillary line (just outside the position of the normal cardiac apex). In female patients the second pad/paddle should be placed firmly on the chest wall just outside the position of the normal cardiac apex avoiding the breast tissue [35].

If unsuccessful, other positions such as Apex–Posterior can be considered [36,37]. Although the polarity of the electrodes affects success with internal techniques such as implantable defibrillators, during transthoracic defibrillation the polarity of the paddles seems unimportant [38–40].

3.1.1. Airway management and ventilation

In 1996, guidelines for the advanced management of the airway and ventilation during resuscitation were published by a working group of the ERC [41]. These guidelines outline basic and advanced approaches to airway management together with their separate indications, contraindications and descriptions of the procedures. Further reviews have been published in 1997 [42,43].

While recognising that tracheal intubation remains the optimal procedure, these guidelines acknowledge that the technique can be difficult and sometimes hazardous and that regular experience and refresher training are required. The laryngeal mask airway (LMA) offers an alternative to tracheal intubation and although it does not guarantee absolutely against aspiration, the incidence in reported series is low [44,45]. The pharyngotracheal lumen airway and the oesophageal/tracheal Combitube are alternatives but require more training and have their own specific problems in use [41].

During cardiac arrest and CPR, lung characteristics change because of an increase in dead space while the development of pulmonary oedema reduces lung compliance [46,47]. Oxygenation of the patient is the primary objective of ventilation and the aim should be to provide inspired oxygen concentrations (FiO_2) of 1.0. Carbon dioxide production and delivery to the lungs is limited during the initial period of cardiac arrest. Tidal volumes of 400–600 ml are adequate to make the chest rise [48]. Adequate minute ventilation is necessary to facilitate carbon dioxide elimination and prevent the potential development of hypercarbic acidosis following the administration of carbon dioxide-producing buffers such as sodium bicarbonate.

Ventilation techniques vary from simple bag valve devices to the most sophisticated automatic ventilators which can provide an FiO_2 of 1.0, consistent tidal volumes, inspiratory flow rates and respiratory frequencies that are adjustable on demand.

3.2. CPR techniques

The only change recommended in the technique of closed chest compression is that the rate should be 100 min^{-1}. There have been and are ongoing trials of new techniques, most notably with active compression–decompression (ACD) CPR, but at present no clinical data show unequivocal improvement in outcomes [49–52]. To improve the scientific basis for future recommendations, the use of new techniques should be carefully evaluated by clinical trials before implementation into prehospital and in-hospital practice.

3.3. Drug delivery

The venous route remains the optimal method of drug administration during cardiopulmonary resuscitation. The previous guidance with regard to venous cannulation is unchanged [53]. If already in situ, central venous cannulae can deliver agents rapidly to the central circulation. If a central line is not present, the risks associated with the technique—which can themselves be life-threatening—mean that for an individual patient the decision as to peripheral versus central cannulation will depend upon the skill of the operator, the nature of the surrounding events, and available equipment. If a decision is made to attempt central venous cannulation, this must not delay defibrillation attempts, CPR, or airway security. When peripheral venous cannulation and drug delivery is carried out, a flush of 20 ml of 0.9% saline is advised to expedite entry to the circulation.

The administration of drugs via a tracheal tube remains only a second line approach because of impaired absorption and unpredictable pharmacodynamics. The agents which can be given by this route are limited to adrenaline/epinephrine, lidocaine, and atropine. Doses of 2–3 times the standard IV dose diluted up to a total volume of at least 10 ml of 0.9% saline are currently recommended. Following administration, five ventilations are given to increase dispersion to the distal bronchial tree thus maximising absorption.

3.4. Specific drug therapy

3.4.1. Vasopressors

Experimentally adrenaline/epinephrine improves myocardial and cerebral blood flow and resuscitation rates in animals, and higher doses are more effective than the 'standard' dose of 1 mg [54,55]. There is no clinical evidence that adrenaline/epinephrine improves survival or neurological recovery in humans irrespective of whether standard or high dose is used [56,57]. Some clinical trials have reported slightly increased rates of spontaneous circulation with high dose adrenaline/epinephrine but without improvement in overall survival rate [58–62]. The reasons for the difference between experimental and clinical results are likely to reflect differences in underlying pathology and the relatively long periods of arrest before the ALS team is able to give adrenaline/epinephrine in the out-of-hospital clinical setting. It is also possible that higher doses of adrenaline/epinephrine may be detrimental in the post-resuscitation period [63,64]. Pending definitive placebo-

controlled trials the indications, dosage, and time interval between doses for adrenaline/epinephrine are unchanged. In practical terms for non VF/VT rhythms each loop of the algorithm lasts 3 min and therefore adrenaline/epinephrine is given with every loop. For VF/VT rhythms, the process of rhythm assessment, three defibrillatory shocks followed by 1 min of CPR will take 2–3 min. Thus, adrenaline/epinephrine could generally be given with each loop if precise timing of administration is impracticable.

Considerable caution should be employed before routinely administering adrenaline/epinephrine in patients whose arrest is associated with solvent abuse, cocaine, and other sympathomimetic drugs [65–68].

The evidence with regard to other adrenergic and non-adrenergic vasopressors is limited. Experimentally, vasopressin leads to significantly higher coronary perfusion pressures and preliminary data in relation to ROSC rates may be encouraging [69,70], but at present, no pressor agent other than adrenaline/epinephrine can be recommended.

3.4.2. Anti-arrhythmic agents

There is incomplete evidence to make firm recommendations on the use of *any* anti-arrhythmic agent, although our knowledge of lidocaine is greater than for the others. Early studies suggested that lidocaine increased the ventricular defibrillation threshold in animals [71–74], but this may have been influenced by experimental techniques [75]. In humans, the administration of lidocaine prior to defibrillation may not increase the energy requirements for defibrillation [76,77]. In one randomised placebo-controlled trial there was a beneficial effect on the threshold in the special circumstance of patients undergoing myocardial reperfusion after coronary artery bypass grafting [78].

For these reasons and pending the results of trials, it is recommended that no change is made in the previous recommendations with regard to lidocaine, bretylium and other anti-arrhythmic agents [79].

Atropine has a well established role in the treatment of haemodynamically compromising bradyarrhythmias and some forms of heart block [10]. It was advocated for asystole in the 1992 guidelines on the basis that increased vagal tone could contribute to the development or unresponsiveness of this arrhythmia. Evidence of value in this condition is equivocal and limited to small series and case reports [80–83]. Since any adverse effect is unlikely in this situation its use can still be considered in a single dose of 3 mg IV. This dose is known to be sufficient to block vagal activity effectively in fit adults with a cardiac output [84].

3.4.3. Buffer agents

In previously healthy individuals, arterial blood gas analysis does not show a rapid or severe development of acidosis during cardiorespiratory arrest provided effective BLS is carried out [85–87]. Simply measuring arterial (or even mixed venous blood) gas tensions may, however, be misleading and bear little relationship to the internal milieu of myocardial or cerebral intracellular values [88–92].

With this background, the role of buffers in CPR is still uncertain. Much of the evidence against the routine use of bicarbonate is based on animal studies, and may have limited applicability to the human situation as the doses of bicarbonate used have often been high [93–95]. One prospective randomised controlled trial has been reported on the use of buffers in patients with out-of-hospital cardiac arrest [96]. The buffer used was Tribonat (a mixture of sodium bicarbonate, trometamol, disodium phosphate and acetate). There was no improvement in hospital admission or discharge rates. In this study the dispatch-response time interval was short, and the confidence interval with the odds ratio was wide [97].

Pending further studies, it is suggested that the judicious use of buffers is limited to severe acidosis as defined in the previous guidelines (arterial pH < 7.1 and base excess < -10) and to certain special situations, such as cardiac arrest associated with hyperkalaemia or following tricyclic anti-depressant overdose. For sodium bicarbonate, a dose of 50 mmol (50 ml of an 8.4% solution) is appropriate, with further administration dependant upon the clinical situation and repeat arterial blood gas analysis.

4. Using the universal algorithm

Each step that follows in the ALS algorithm (Fig. 1) assumes that the preceding one has been unsuccessful.

A precordial thump may, in certain situations such as a witnessed event, precede (albeit by a few seconds only) the attachment of a monitor/defibrillator [98,99].

ECG monitoring then provides the link between BLS and ALS procedures. Electrocardiographic rhythm assessment must be always interpreted within the clinical context as movement artefact, lead disconnection, and electrical interference can mimic rhythms associated with cardiac arrest.

Following this assessment, the algorithm splits into two pathways—VF/VT and other rhythms.

4.1. VF/VT rhythms

The first defibrillating shock must be given without any delay. If unsuccessful it is repeated once and if necessary, twice. This initial group of three shocks should occur with successive energies of 200 J, 200 J, and 360 J. If VF/VT persists, further shocks are given with 360 J energies or the biphasic equivalent. A pulse

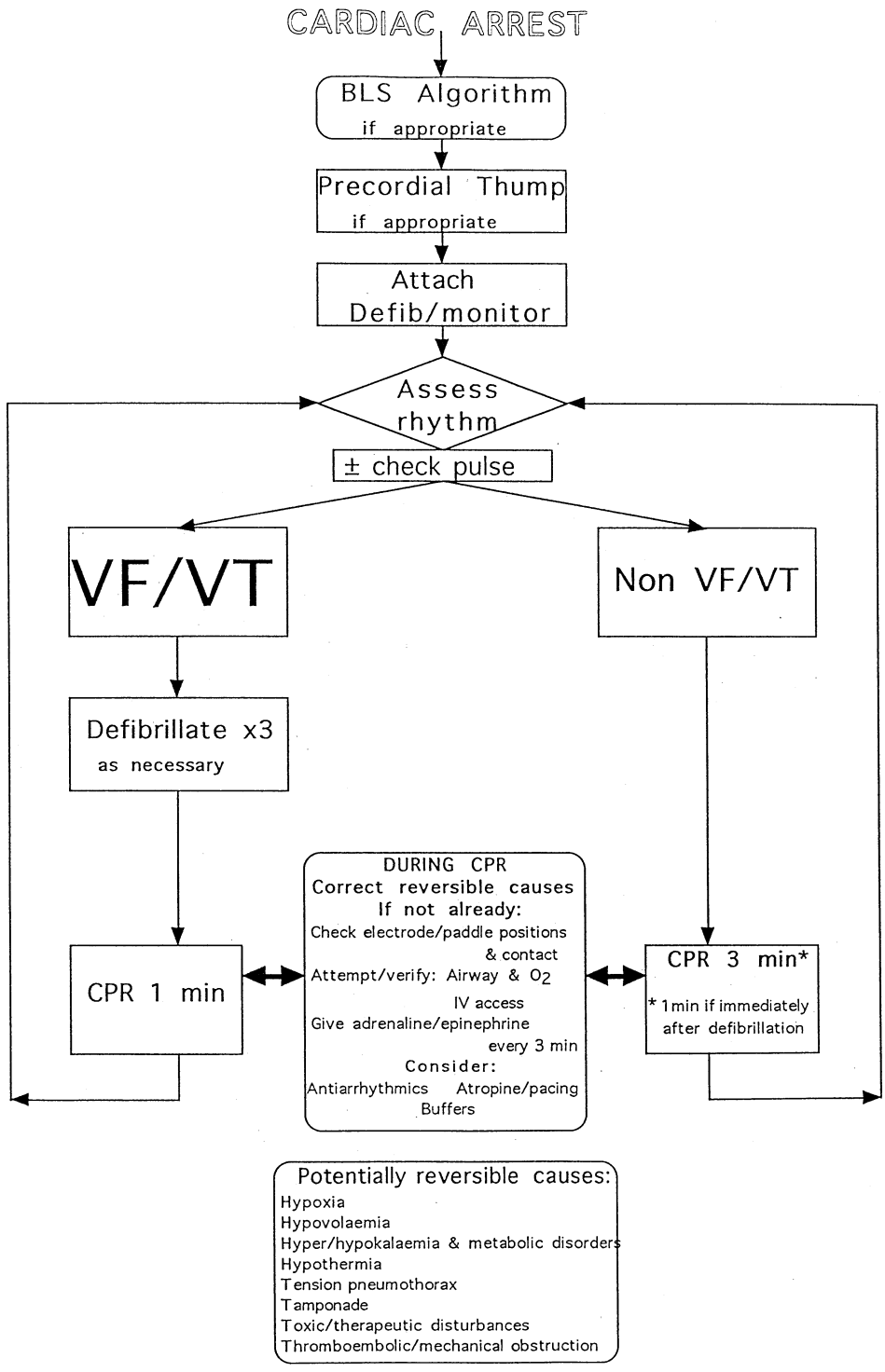

Fig. 1. ALS algorithm.

check is carried out, and should be prompted by an AED if, following a defibrillating shock a change in waveform is produced which is compatible with output. If the monitor/defibrillator indicates that VF/VT persists, then further DC shocks are administered without a further pulse check.

It is important to note that after a shock the ECG monitor screen will often show an isoelectric line for several seconds. This is commonly due to a transient period of electrical and/or myocardial 'stunning', and does not necessarily mean that the rhythm has converted to asystole, as a co-ordinated rhythm or return of VF/VT may supervene subsequently. If the monitor screen of a manual defibrillator shows a 'straight' line for more than one sweep immediately after a shock, 1 min of CPR should be given without a new dose of

adrenaline/epinephrine, and the patient reassessed. Only if the result of this reassessment is a non VF/VT rhythm without a pulse should a new dose of adrenaline/epinephrine be administered and CPR given for a further 2 min before the patient is assessed again. Algorithms of AED's should also take account of this phenomenon.

Emphasis must be placed on the correct performance of defibrillation including the use of couplants. The safety of the resuscitation team is paramount. During defibrillation, no-one must be in contact with the patient. Liquids, wet clothing or the spreading of excess electrode gel can cause problems. Transdermal patches should be removed to prevent the possibility of electrical arcing [100]. Paddle/pads should be kept 12–15 cm away from implanted pacemakers. During manual defibrillation, the operator must give a command, e.g. 'Stand clear!' and check that this is obeyed before the shock is given. With automated systems an audio command is given, and all team members must comply with this command.

Over 80% of individuals who will be successfully defibrillated have this achieved by one of the first three shocks [17,19,20,101]. Subsequently, the best prospects for restoring a perfusing rhythm still remain with defibrillation, but at this stage the search for and correction of potentially reversible causes or aggravating factors is indicated, together with an opportunity to maintain myocardial and cerebral viability with chest compressions and ventilation. During CPR attempts can be made to institute advanced airway management and ventilation, venous access, and to administer drugs if appropriate to do so.

The time interval between the third and fourth shocks should not exceed two minutes. Although the interventions which can be carried out during this period may improve the prospects for successful defibrillation, this is unproven, while it is well-established that with the passage of time the chances of success for defibrillating shocks lessen.

4.2. 'Looping' the algorithm

For the patient with persistent VF/VT, potential causes or aggravating factors may include electrolyte imbalance, hypothermia, and drugs and toxic agents for which specific treatment may be indicated. Where it is appropriate to continue resuscitation, successive loops of the algorithm are followed, allowing further sequences of shocks, basic life support, and the ability to carry out and secure advanced airway and ventilation techniques, oxygenation and drug delivery. Antiarrhythmic drugs may be considered after the first two sets of three shocks, though maintaining the previous policy of deferring this treatment until four sets would be acceptable.

4.3. Non VF/VT rhythms

If VF/VT can be positively excluded, defibrillation is not indicated as a primary intervention, (although it may be required later if ventricular fibrillation develops), and the right-sided path of the algorithm is followed.

For patients in cardiac arrest with non VF/VT rhythms, the prognosis is in general much less favourable. The overall survival rate with these rhythms is approximately 10–15% of the survival rate with VF/VT rhythms, but the possibility of survival should not be disregarded. In some series, approximately 20% of eventual survivors present with a non VF/VT rhythm [102–105].

With the passage of time, all electrical rhythms associated with cardiac arrest deteriorate with the eventual production of asystole. The abysmal prognosis of this degenerated rhythm is well justified. There are, nevertheless, some situations where a non VF/VT rhythm may be caused or aggravated by remediable conditions, especially if this was the *primary* rhythm. As a consequence the detection and treatment of reversible causes become relatively more important.

During the search for and correction of these causes, CPR together with advanced airway management, oxygenation and ventilation, and any necessary attempts to secure venous access should occur, with adrenaline/epinephrine administered every 3 min.

The use of atropine for asystole has been discussed above. Atropine 3 mg IV is given once, along with adrenaline/epinephrine 1 mg for asystole on the first loop. Pacing may play a valuable role in patients with extreme bradyarrhythmias, but its value in asystole is questionable, except in cases of trifascicular block where P waves are seen. In patients where pacing is to be carried out, but a delay occurs before it can be achieved, external cardiac percussion (also known as 'fist' or 'thump' pacing) may generate QRS complexes with an effective cardiac output, particularly in cases where myocardial contractility is not critically compromised [106–108]. External cardiac percussion is performed with blows at a rate of 100 min^{-1}, given with less force than a precordial thump and delivered over the heart, not the sternum. Conventional CPR should be substituted immediately if QRS complexes with a discernable output are not being achieved.

After 3 min of CPR, the patient's electrical rhythm is reassessed. If VF/VT has supervened, the left-sided path of the algorithm is followed, otherwise loops of the right-sided path of the algorithm will continue for as long as it is considered appropriate for resuscitation to continue. Resuscitation should generally continue for

at least 20–30 min from the time of collapse unless there are overwhelming reasons to believe that resuscitation is likely to be futile.

4.4. Post-resuscitation care

There are no changes in the recommendations for post resuscitation care. The most vulnerable organ for the ischaemic/hypoxic damage occurring in association with cardiac arrest is the central nervous system (CNS). Approximately 1/3 of the patients who have return of spontaneous circulation die a neurologic death, with 1/3 of long-term survivors having recognisable motor or cognitive deficits [109–111]. Fortunately only 1–2% of these individuals do not achieve an independant existence [112].

Intensive research efforts are rapidly increasing our knowledge about the pathophysiology of CNS ischaemia/hypoxia, but there are no new clinically validated treatment strategies for the cerebral damage sustained with cardiac arrest. Efforts should be directed to the avoidance and/or correction of hypotension, hypoxia, hypercarbia, electrolyte imbalance, and hypo- or hyperglycaemia [113–116].

Many victims of cardiac arrest have features indicating that the event was precipitated by acute myocardial infarction [117]. In these patients, there is an urgent need for appropriate management including such aspects as thrombolysis or other methods for obtaining coronary reperfusion and maintaining electrical stability to reduce the chances of further episodes of cardiac arrest and to improve the overall prognosis. These aspects are covered by the publications on the Management of Acute Myocardial Infarction of the European Society of Cardiology and the ESC/ERC task force on the pre-hospital management of myocardial infarction [118–120].

References

[1] Guidelines for advanced life support. A statement by the advanced life support working party of the European Resuscitation Council, 1992. Resuscitation 1992;24:111–21.

[2] Standards and guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). J Am Med Assoc 1986;255:2905–89.

[3] The ALS working group of the International advisory Committee on Resuscitation. The Universal Algorithm. Resuscitation 1997;34:109–11.

[4] Chamberlain DA, Cummins RO, Eisenberg M, et al. Resuscitation. Recommended guidelines for uniform reporting of data from out-of-hospital cardiac arrest: the Utstein style. Resuscitation 1991;22:1–26.

[5] Cummins RO, Chamberlain DA, et al. Recommended guidelines for uniform reporting of data from in-hospital resuscitation: the in-hospital "Utstein style". Resuscitation 1997;34:151–83.

[6] Spain D, Bradess V, Mohr C. Coronary atherosclerosis as a cause of unexpected and unexplained death. J Am Med Assoc 1960;174:384–8.

[7] Schatzkin A, Cupples LA, Fisher R, Heeren T, Morelock S, Mucatel M, Kannel WB. The epidemiology of sudden unexpected death: risk factors for men and women in the Framingham heart study. Am Heart J 1984;107:1300–6.

[8] Holmes DR, Davis K, Gersh BJ, Mock MB, Pettinger MB. Risk factor profiles of patients with sudden cardiac death and death from other cardiac causes: a report from the Coronary Artery Surgery Study (CASS). J Am Coll Cardiol 1989;13:524–30.

[9] Myerburg RJ, Kessler KM, Bassett AL, Castellanos A. A biological approach to sudden cardiac death: structure, function and cause. Am J Cardiol 1989;63:1512–6.

[10] Chamberlain D. Periarrest arrhythias. Br J Anaesth 1997;79:198–202.

[11] Surawicz B. Ventricular fibrillation. J Am Coll Cardiol 1985;5:438–548.

[12] Sedgewick ML, Dalziel K, Watson J, Carrington DJ, Cobbe SM. The causative rhythm in out-of-hospital cardiac arrests witnessed by the emergency medical services in the Heartstart Scotland project. Resuscitation 1994;27:55–9.

[13] Deluna AB, Courrel P, Ledercq JF. Ambulatory sudden cardiac death: mechanisms of production of fatal arrhythmia on the basis of data from 157 cases. Am Heart J 1989;117:151–9.

[14] Adgey AAJ, Devlin JE, Webb SW, Mulholland H. Initiation of ventricular fibrillation outside hospital in patients with acute ischaemic heart disease. Br Heart J 1982;47:55–61.

[15] Roth R, Stewart RD, Rogers K, Cannon GM. Out-of-hospital cardiac arrest: factors associated with survival. Ann Emerg Med 1984;13:237–43.

[16] Ekstrom L, Herlitz J, Wennerblom B, Axelsson A, Bang A, Holmberg S. Survival after cardiac arrest outside hospital over a 12-year period in Gothenburg. Resuscitation 1994;27:181–8.

[17] Tunstall-Pedoe H, Bailey L, Chamberlain D, Marsden A, Ward M, Zideman D. Survey of 3765 cardiopulmonary resuscitations in British hospitals (the BRESUS Study). Br Med J 1992;304:1347–51.

[18] Tortolani AJ, Risucci DA, Rosati RJ, Dixon R. In-hospital cardiopulmonary resuscitation: patient, arrest and resuscitation factors associated with survival. Resuscitation 1990;20:115–28.

[19] Hargarten KM, Steuven HA, Waite EM, et al. Prehospital experience with defibrillation of coarse ventricular fibrillation: a ten-year review. Ann Emerg Med 1990;19:157–62.

[20] Cobbe SM, Redmond MJ, Watson JM, Hollingsworth J, Carrington DJ. Heartstart Scotland—initial experience of a national scheme for out of hospital defibrillation. Br Med J 1991;302:1517–20.

[21] Neumar RW, Brown CG, Robitaille PL, et al. Myocardial high energy phosphate metabolism during ventricular fibrillation with total circulatory arrest. Resuscitation 1990;19:199–226.

[22] Mapin DR, Brown CG, Dzuonczyk R. Frequency analysis of the human and swine electrocardiogram during ventricular fibrillation. Resuscitation 1991;22:85–91.

[23] Larsen MP, Eisenberg MS, Cummins RO, Hallstrom AP. Predicting survival from out-of-hospital cardiac arrest: a graphic model. Ann Emerg Med 1993;22:1652–8.

[24] Greene HL, Dimarco JP, Kudenchuk PJ, et al. Comparison of monoplasic and biphasic defibrillating pulse waveforms for transthoracic cardioversion. Am J Cardiol 1995;75:1135–9.

[25] Bardy GH, Gliner BE, Kudenchuk PJ, et al. Truncated biphasic pulses for transthoracic defibrillation. Circulation 1995;91:1768–74.

[26] Kerber RE, Spencer KT, Kallock M, et al. Overlapping sequential pulses: a new waveform for transthoracic defibrillation. Circulation 1994;89:2369–79.

[27] Kerber RE, Kouba C, Martins JB, et al. Advance prediction of transthoracic impedence in human defibrillation and cardioversion: importance of impedence in determining the success of low energy shocks. Circulation 1984;70:303–8.

[28] Kerber RE, Martins JB, Kienzle MG, et al. Energy, current and success in defibrillation and cardioversion: Clinical studies using an automated, impedence-based energy adjustment method. Circulation 1988;77:1038–46.
[29] Bossaert L, Koster R. Defibrillation: methods and strategies. A statement for the Advanced Life Support Working Party of the European Resuscitation Council. Resuscitation 1992;24:211–25.
[30] Lermann BB, Deale OC. Relation between transcardiac and tranthoracic current during defibrillation in humans. Circ Res 1990;67:1420–6.
[31] Adgey AAJ, Dalzell GNW. Paddle/pad placement for defibrillation in adults. Br J Int Care 1994;Suppl:14–6.
[32] Sirna SJ, Ferguson DW, Charbonnier F, Kerber RE. Electrical cardioversion in humans: factors affecting transthoracic impedence. Am J Cardiol 1988;62:1048–52.
[33] Kerber RE. Electrical treatment of cardiac arrhythmias: defibrillation and cardioversion. Ann Emerg Med 1993;27:296–301.
[34] Alyward PE, Keiso R, Hite P, Charbonnier F, Kerber RE. Defibrillator electrode-chest wall coupling agents: Influence on transthoracic impedence and shock success. J Am Coll Cardiol 1985;6:682–6.
[35] Pagan-Carlo LA, Spencer KT, Robertson CE, et al. Transthoracic defibrillation: Importance of avoiding electrode placement directly on the female breast. J Am Coll Cardiol 1996;27:449–52.
[36] Kerber RE, Grayzel J, Kennedy J, Jensen SR. Elective cardioversion: influence of paddle electrode location and size on success rates and energy requirements. New Engl J Med 1981;305:658–62.
[37] Kerber RE, Martins JB, Kelly J, et al. Self-adhesive pre-applied electrode pads for defibrillation and cardioversion: experimental and clinical studies. J Am Coll Cardiol 1984;3:815–20.
[38] Bardy GH, Ivey TD, Allen MD, Johnson G, Greene HL. Evaluation of electrode polarity on defibrillation efficacy. Am J Cardiol 1989;63:433–7.
[39] Strickberger SD, Hummel JD, Horwood LE, et al. Effect of shock polarity on ventricular defibrillation threshold using a transvenous system. J Am Coll Cardiol 1994;24:1069–72.
[40] Weaver WD, Martin JS, Wirkus MJ, et al. Influence of external defibrillator electrode polarity on cardiac resuscitation. Pace 1993;16:285–90.
[41] Baskett PJF, Bossaert L, Carli P, et al. Guidelines for the advanced management of the airway and ventilation during resuscitation A statement by the Airway and Ventilation Management Group of the ERC. Resuscitation 1996;31:201–30.
[42] Gabbott DA, Baskett PJF. Management of the airway and ventilation during resuscitation. Br J Anaesth 1997;79:159–71.
[43] Nolan JP, Parr MJA. Aspects of resuscitation in trauma. Br J Anaesth 1997;79:226–40.
[44] Brimacombe JR, Berry A. The incidence of aspiration associated with the Laryngeal mask Airway: a meta-analysis of published literature. J Clin Anaesth 1995;7:297–305.
[45] Owens TM, Robertson P, Twomey C, et al. The incidence of gastro oesophageal reflux with the laryngeal mask. Anesth Analg 1995;80:980–4.
[46] Davis K, Johannigman JA, Johnson RC, Branson RD. Lung compliance following cardiac arrest. Acad Emerg Med 1995;2:874–8.
[47] Ornato JP, Bryson BL, Donovan PJ, et al. Measurement of ventilation during CPR. Crit Care Med 1983;11:79–82.
[48] Baskett PJF, Nolan J, Parr MJA. Tidal volumes which are perceived to be adequate for resuscitation. Resuscitation 1996;31:231–4.
[49] Cohen TJ, Tucker KJ, Lurie KG, et al. Active compression-decompression. A new method of cardiopulmonary resuscitation. J Am Med Assoc 1992;267:2916–23.
[50] Schwab TM, Callaham ML, Madsen CD, Utecht TA. A randomised clinical trial of active compression-decompression CPR vs. standard CPR in out of hospital cardiac arrest in two cities. J Am Med Assoc 1995;273:1261–8.
[51] Steill IG, Hebert PC, Wells GA, et al. The Ontario trial of active compression-decompression cardiopulmonary resuscitation for in-hospital and prehospital cardiac arrest. J Am Med Assoc 1996;275:1417–23.
[52] Plaisance P, Adnui F, Viuuni E, et al. Benefit of active compression decompression CPR as a prehospital advanced cardiac life support. Circulation 1997;95:955–61.
[53] Hapnes S, Robertson C. CPR-drug delivery routes and systems. Resuscitation 1992;24:137–42.
[54] Redding JS, Pearson JW. Evaluation of drugs for cardiac resuscitation. Anaesthesiology 1963;24:203–7.
[55] Redding JS, Pearson JW. Adrenaline in cardiac resuscitation. Am Heart J 1963;66:210–4.
[56] Woodhouse SP, Cox S, Boyd P, Case C, Weber M. High dose and standard dose adrenaline do not alter survival compared with placebo, in cardiac arrest. Resuscitation 1995;30:243–9.
[57] Herlitz J, Ekstrom L, Wennerblom B, Axelsson A, Bang A, Holmberg S. Adrenaline in out of hospital ventricular fibrillation. Does it make any difference? Resuscitation 1995;29:195–201.
[58] Callaham ML, Modsen CD, Barton CW, Saunders CE, Pointer J. A randomised clinical trial of high-dose epinephrine and noradrenaline vs. standard dose epinephrine in prehospital cardiac arrest. J Am Med Assoc 1992;268:2667–72.
[59] Abramson NS, Safar P, Sutton-Tyrrel K. A randomised clinical trial of escalating doses of high dose epinephrine during cardiac resuscitation [Abstract]. Crit Care Med 1995;23:178.
[60] Lindner KH, Ahnefeld FW, Prengel AW. Comparison of standard and high-dose adrenaline in the resuscitation of asystole and electromechanical dissociation. Acta Anaesth Scand 1991;35:253–6.
[61] Steill IG, Hebert PC, Weitzman BW, et al. High dose epinephrine in adult cardiac arrest. New Engl J Med 1992;327:1045–50.
[62] Brown CG, Martin DR, Pepe PE, et al. A comparison of standard-dose and high-dose epinephrine in cardiac arrest outside the hospital The Multicenter High-dose Epinephrine Study Group. New Engl J Med 1992;327:1051–5.
[63] Rivers EP, Wortsnan J, Rady MY, et al. The effect of total cumulative epinephrine dose adminstered during human CPR on haemodynamic, oxygen transport and utilisation variables in the post-resuscitation period. Chest 1994;5:1315–6.
[64] Tang W, Weil MH, Sun S, Noc M, Yang L, Gasmuri RJ. Epinephrine increases the severity of post-resuscitation myocardial dysfunction. Circulation 1995;92:3089–93.
[65] Lathers CM, Tyau LSY, Spino MM, et al. Cocaine-induced seizures, arrhythmias and sudden death. J Clin Pharmacol 1988;28:584–93.
[66] Lange RA, Cigarroa RG, Yancy CW, et al. Cocaine-induced coronary artery vasoconstriction. New Engl J Med 1989;321:1557–62.
[67] Sheperd RT. Mechanism of sudden death associated with volatile substance abuse. Hum Toxicol 1989;8:287–92.
[68] Boon NA. Solvent abuse and the heart (Editorial). Br Med J 1987;294:722.
[69] Lindner KH, Prengel AW, Pfenninger EG, et al. Vasopressin improves vital organ blood flow during closed-chest cardiopulmonary resuscitation in pigs. Circulation 1995;91:215–21.
[70] Lindner KH, Prengel AW, Brinkmann A, et al. Vasopressin administration in refractory cardiac arrest. Ann Intern Med 1996;124:1061–4.
[71] Babbs CF, Yim GKW, Whistler SJ, Tacker WA, Geddes LA. Elevation of ventricular defibrillation threshold in dogs by antiarrhythmic drugs. Am Heart J 1979;98:345–50.

[72] Chow MSS, Kluger J, Lawrence R, Fieldman A. The effect of lidocaine and bretylium on the defibrillation threshold during cardiac arrest and cardiopulmonary resuscitation. Proc Soc Exp Biol Med 1986;182:63–7.
[73] Dorian P, Fain ES, Davy JM, Winkle RA. Lidocaine causes a reversible, concentration-dependent increase in defibrillation energy requirements. J Am Coll Cardiol 1986;8:327–32.
[74] Echt DS, Black JN, Barbey JT, Coxe DR, Cato E. Evaluation of antiarrhythmic drugs on defibrillation energy requirements in dogs. Sodium channel block and action potential prolongation. Circulation 1989;79:1106–17.
[75] Natale A, Jones DL, Kim Y-H, Klein GJ. Effects of lidocaine on defibrillation threshold in the pig: Evidence of Anesthesia Related Increase. Pace 1991;14:1239–44.
[76] Kerber RE, Pandian NG, Jensen SR, et al. Effect of lidocaine and bretylium on energy requirements for transthoracic defibrillation; Experiemental studies. J Am Coll Cardiol 1986;7:397–405.
[77] Kerber RE, Jensen SR, Gascho JA, Grayzel J, Hoyt R, Kennedy J. Determinants of defibrillation: prospective analysis of 183 patients. Am J Cardiol 1983;52:739–45.
[78] Lake CL, Kron IL, Mentzer RM, Crampton RS. Lidocaine enhances intraoperative ventricular defibrillation. Anesth Analg 1986;65:337–40.
[79] von Planta M, Chamberlain D. Drug treatment of arrhythmias during cardiopulmonary resuscitation. Resuscitation 1992;24:227–32.
[80] Brown DC, Lewis AJ, Criley JM. Asystole and its treatment: The possible role of the parasympathetic nervous system in cardiac arrest. J Am Cardiol Exp Pharmacol 1979;8:448–52.
[81] Iseri LT, Humphrey SB, Sirer EJ. Prehospital bradyasystolic cardiac arrest. Ann Intern Med 1978;88:741–5.
[82] Coon GC, Clinton JE, Ruiz E. Use of atropine for bradyasystolic prehospital cardiac arrest. Ann Emerg Med 1981;10:462–7.
[83] Steuven HA, Tonsfeldt DJ, Thomson BM. Atropine in asystole: Human studies. Ann Emerg Med 1984;13:815–7.
[84] Chamberlain DA, Turner P, Sneddon JM. Effects of atropine on heart-rate in healthy man. Lancet 1967;ii:12–5.
[85] Steedman DJ, Robertson CE. Acid base changes in arterial and central venous blood during cardiopulmonary resuscitation. Arch Emerg Med 1992;9:169–76.
[86] Henreman PL, Ember JE, Marx JA. Development of acidosis in human beings during closed chest and open chest CPR. Ann Emerg Med 1988;17:672–5.
[87] Gilston A. Clinical and biochemical aspects of cardiac resuscitation. Lancet 1965;2:1039–43.
[88] von Planta J, Weil MH, Gazmuri RJ, Bisera J, Rackow EC. Myocardial acidosis associated with CO_2 production during cardiac arrest and resuscitation. Circulation 1989;80:684–92.
[89] Capparelli EV, Chow MSS, Kluger J, Fieldman A. Difference in systemic and myocardial blood acid–base status during cardiopulmonary resuscitation. Crit Care Med 1989;17:442–6.
[90] Gudipati CV, Neil MH, Gazmuri RJ, et al. Increases in coronary vein CO_2 during cardiac resuscitation. J Appl Physiol 1990;68:1405–8.
[91] Kette F, Weil MH, Gazmuri RJ, Bisera J, Rackow EC. Intramyocardial hypercarbic acidosis during cardiac arrest and resuscitation. Crit Care Med 1993;21:901–6.
[92] Javaheri S, Clendending A, Papadakos N, et al. pH changes in the surface of brain and in cisternal fluid in dogs in cardiac arrest. Stroke 1984;15:553–8.
[93] Kette F, Weil MH, Gazmuri RJ. Buffer solutions may compromise cardiac resuscitation by reducing coronary perfusion pressure. J Am Med Assoc 1991;266:2121–6.
[94] Gazmuri RJ, von Planta M, Weil MH, Rackow EC. Cardiac effects of carbon dioxide-consuming and carbon dioxide-generating buffers during cardiopulmonary resuscitation. J Am Coll Cardiol 1990;15:482–549.
[95] Bleske BE, Rice TL, Warren EW, et al. The effect of sodium bicarbonate administration on the vasopressor effect of high-dose epinephrine during cardiopulmonary resuscitation in Swine. Am J Emerg Med 1993;11:439–43.
[96] Dybvik T, Strand T, Steen PA. Buffer therapy during out-of-hospital cardiopulmonary resuscitation. Resuscitation 1995;29:89–95.
[97] Koster RW. Correction of acidosis during cardio-pulmonary resuscitation. Resuscitation 1995;29:87–8.
[98] Caldwell G, Millar G, Quinn E, Vincent R, Chamberlain DA. Simple mechanical methods for cardioversion: defence of the precordial thump. Br Med J 1985;291:627–30.
[99] Robertson C. The precordial thump and cough techniques in advanced life support. Resuscitation 1992;24:133–5.
[100] Paracek EA, Munger MA, Rutherford WF, Gardner SF. Report of nitropatch explosions complicating defibrillation. Am J Emerg Med 1992;10:128–9.
[101] Adgey AAJ. The Belfast experience with resuscitation ambulances. Am J Emerg Med 1984;2:193–209.
[102] Sedgwick ML, Dalziel K, Watson J, Carrington DJ, Cobbe SM. Performance of an established system of first responder out of hospital defibrillation. Resuscitation 1993;26:75–88.
[103] Myerburg RJ, Conde CA, Sing RJ, et al. Clinical, electrophysiological and haemodynamic profile of patients resuscitated from prehospital cardiac arrest. Am J Med 1980;68:568–76.
[104] Isen LT, Humphrey SB, Sirer EJ. Prehospital bradyasystolic cardiac arrest. Ann Emerg Med 1978;88:741–5.
[105] Herlitz J, Ekstrom L, Wennerblom B, Axelsson A, Bang A, Holmberg S. Survival among patients with out-of-hospital cardiac arrest found in electromechanical dissociation. Resuscitation 1995;29:97–106.
[106] Sandoe E. Ventricular standstill and percussion (Editorial). Resuscitation 1996;32:3–4.
[107] Dowdle JR. Ventricular standstill and cardiac percussion. Resuscitation 1996;32:31–2.
[108] Scherf D, Bornemann C. Thumping of the precordium in ventricular standstill. Am J Cardiol 1960;5:30–40.
[109] Edgren E, Hedstrand N, Kelsey S, Sutton-Tyrrell K, Safar P, BRCT 1. Study group assessment of neurological prognosis in comatose survivors of cardiac arrest. Lancet 1994;343:1055–9.
[110] Roine RO, Kaste M, Kinnunen A, Nikki P, Sarna S, Kajaste S. Nimodipine after resuscitation from out-of-hospital ventricular fibrillation: A placebo controlled, double blind, randomized trial. J Am Med Assoc 1990;264:3171–7.
[111] Brain Resuscitation Clinical Trial II Study Group A randomized study of calcium entry blocker (lidoflazine) administration in the treatment of comatose survivors of cardiac arrest. New Engl J Med 1991;324:1225–31.
[112] Graves JR, Herlitz J, Axelsson A, Ekstrom L, Holmberg M, Lidquist J, Sunnerhagen K, Holmberg S. Survivors of out of hospital cardiac arrest: their prognosis, longevity and functional status. Resuscitation 1997;35:117–22.
[113] Cantu RC, Ames A, Digancinto G, et al. Hypotension: a major factor limiting recovery from cerebral ischaemia. J Surg Res 1969;9:525–9.
[114] Sieber FE, Traystman RJ. Special issues, glucose and the brain. Crit Care Med 1991;20:104–14.
[115] Longstreth WT, Inin TS. High blood glucose level on hospital admission and poor neurological recovery after cardiac arrest. Ann Neurol 1984;15:59–63.
[116] Buylaert WA, Calle PA, Honbreckts HN. The cerebral resuscitation study group Serum electrolyte disturbances in the post resuscitation period. Resuscitation 1989;17:S189–96.
[117] Cobb LA, Werner J, Trobough G. Sudden cardiac death: a decade's experience with out of hospital resuscitation. Mod Concepts Cardiovasc Dis 1980;49:31–9.

[118] The task force on the management of acute myocardial infarction of the European Society of Cardiology Acute Myocardial Infarction: prehospital and in-hospital management. Eur Heart J 1996;17:43–63.

[119] Guidelines for the early management of patients with acute myocardial infarction. A report of the ACC/AHA task force on assessment of diagnostic and therapeutic cardiovascular procedures. J Am Coll Cardiol 1996;16:249–92.

[120] Arntz HR, Bossaert L, Carli P, Chamberlain D, Davies M, Dellborg M et al. The Prehospital management of Acute Heart attacks: Report of a task force of ESC/ERC Resuscitation 1998 (in press).