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Clinical Reviews

Current Approaches to Cardiopulmonary Resuscitation

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Key Points

- New guidelines emphasize the importance of basic life support and defibrillation over advanced life support.
- Prompt initiation of CPR has an impact in survival following cardiac arrest.
- CPR should be performed with a compression-ventilation ratio of 30:2, with minimal interruptions.
- Defibrillation should be performed immediately when appropriate.
- Chest compressions should be resumed immediately after each shock, and any drugs should be administered during CPR.
- Importantly, the new changes identified the gaps in evidence in resuscitation knowledge.
- Further research in these areas is vital for further improvement in CPR management.

Introduction

Cardiac arrest occurs when there is abrupt cessation of effective pumping activity of the heart. Among the likely causes are ventricular asystole (electrical or mechanical), pulseless ventricular tachycardia or ventricular fibrillation. Clinically this may present as 'sudden cardiac death'.

Sudden cardiac death is an unexpected natural death, from a cardiac cause, within one hour of onset of symptoms, in a person without known cardiac disease. It is a major clinical problem worldwide accounting for 3 to 400,000 deaths per annum.[1]

The likelihood of return of spontaneous circulation (ROSC) varies with the presenting rhythm, with a reported survival rate of 0.9% for pulseless electrical activity and asystole, which increases to between 9.5% and 40% for ventricular tachycardia or ventricular fibrillation.[2,3]

Historically, the first successful cardiac massage was performed via a thoracotomy by Kristian Igelsrud following an anaesthetic induced cardiac arrest in 1901.[4]

CS Beck, a thoracic surgeon in the University Hospitals, Cleveland reported successful electrical defibrillation in 1947.[5]

Almost 60 years later it was discovered, accidentally, that external chest compression was associated with an increase in arterial blood pressure and this technique was subsequently introduced in patient care by Kouwenhoven in 1960.[6]

Also, in 1960, the American Heart Association started a program to acquaint physicians with close-chest cardiac resuscitation, the forerunner of CPR training for the general public.

In-spite of years of public training in CPR, the survival rates among patients with out-of-hospital cardiac arrest remains depressingly low (5-18%).

This low survival rate makes it difficult to perform clinical trials with sufficient power to demonstrate improved long-term outcomes (i.e., neurologically intact survival to hospital discharge). Indeed, high-level evidence is insufficient to support many recommendations in cardiopulmonary resuscitation and emergency cardiovascular care.

The 2005 American Heart Association Guidelines for CPR are, however, based on a comprehensive review of the available evidence as reviewed by the 2005 Consensus Conference.[7] These guidelines, and the theory / evidence which underpin them, are summarised in this article.

'Non Cardiac' Cardiac Arrest

The majority of this article will deal with the major changes in Advanced Life Support (ALS) guidelines. However, it is also important to remember that, particularly within the hospital environment, cardio-respiratory arrest may be a 'terminal' manifestation of a myriad of non cardiac disorders. Death may be inevitable in many of these cases and as a consequence resuscitation may be both inappropriate and futile. However, many studies site unexpected cardio-respiratory arrest as a surrogate marker of suboptimal 'medical' care.

The widespread introduction of Critical Care Outreach services, use of Early Warning Scores and moves towards locating higher risk patients in more appropriate environments are attempts to tackle this problem. The long term success of these interventions is yet to be assessed.

Cardiopulmonary Resuscitation

1. Compression-Ventilation ratio

The guidelines recommend a compression-ventilation ratio of 30:2 for lone rescuers in all cases of cardiac arrest. When 2 rescuers are involved (chiefly aimed at healthcare professionals), a compression-ventilation ratio of 15:2 is recommended for infants and children but 30:2 ratio for all other victims.

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It is emphasised that rescuers should push "hard and fast" during chest compressions and maintain the recommended rate (100/minute).

The aim of the cardiac compressions is to maintain adequate blood flow to the cerebral and coronary circulation.

The change to this aspect is based on the observations that long interruptions in cardiac compressions occur during delivery of rescue breaths (14-16 seconds), with a fall in effective compression rate to 39-40/minute,[8,9] compromising vital organ blood flow and reducing the probability of ROSC.[10]

This observation applies equally to lay, paramedical and medical resuscitators.[11,12]

Studies of animal models have shown an improved matching of ventilation and pulmonary blood flow with compression-ventilation ratio more than 15:2.[13,14]

The variation for infants and children addresses a concern in this group about the adequacy in ventilation with ratios higher than 15:2, and this could be equally applied to cases of asphyxial arrest.

Incidentally, better outcomes have also been demonstrated with continuous cardiac compressions (minimally invasive cardiac resuscitation), compared to traditional ventilation-compression.[15]

2. Number of shocks for defibrillation

The new guidelines advise treatment of ventricular fibrillation or pulseless ventricular tachycardia with a single shock, followed by immediate recommencement of CPR. Rhythm check is to be done only after 2 minutes of CPR, followed by further shocks if required.

This is an important change from the previous guidelines of a stack of 3 consecutive shocks, with rhythm check following each shock.

The previous recommendation of a stacked sequence of 3 shocks was based on the low efficacy of monophasic shock, and in an attempt to decrease the transthoracic impedance (normally about 70-80 ohms) with subsequent shocks. However, it was argued that modern biphasic defibrillators terminate ventricular fibrillation in up to 90% of patients.[16,17]

Failure to cardiovert VF immediately may indicate low amplitude VF; hence a period of CPR before attempting cardioversion again, is beneficial.

Moreover, even after successful defibrillation, restoration of cardiac output is often not immediate while rhythm analysis has a time lag of 29-37 seconds. Thus CPR should be reinstituted after cardioversion until self-sustained cardiac function is evident.[18,19]

3. Monophasic versus Biphasic waveforms – Energy Content

The new guidelines recommend an initial energy level of 150J for biphasic defibrillators, with escalating energy for subsequent shocks, and 360J for monophasic defibrillators.

Monophasic defibrillators essentially direct energy through the myocardium in one direction (between the 'paddles') In biphasic defibrillators, current travels in one direction initially, and then changes polarity to travel in the opposite direction. The full cycle takes about 10 milliseconds. The higher efficacy of the biphasic defibrillators is not fully understood, but may be due to the conditioning prepulse, which reduces the excitation threshold for the second phase.[20] Hence biphasic defibrillators use less electrical energy (the potential difference required is 1600 to 1750 volts) than monophasic defibrillators (potential difference of 5200 volts). These reduced energy levels produce less impairment of ventricular contractility following successful resuscitation.

Given this, the new recommendations needed to redefine the optimal energy for the initial shock. First shock efficacy for long duration VF/VT is greater with biphasic (86-98%), than monophasic shock, and hence use of the former is recommended, whenever available.[17, 21-23]

Many places still use monophasic defibrillators and here the initial and subsequent energy level should be 360J.

Of the 2 types of biphasic defibrillators, the shock should be no lower than 120 J for a Rectilinear Biphasic (RLB) waveform, and 150 J for Biphasic Truncated Exponential (BTE) waveform.[24] This is because the former waveform controls for the impedance of the chest (which can vary between 25-180 ohms) directly by a change in the resistor of the machine, and can maintain a relatively constant current. In the latter, there is a gradual decline in current.

To avoid confusion, however, an initial biphasic shock is recommended of 150 J is recommended. If the first shock is unsuccessful, then either a fixed or escalating energy level may be used.

4. CPR before defibrillation

The ALS guideline of 2000 recommended immediate defibrillation for all shockable rhythms. However, recent evidence suggests that, in the case of prolonged cardiac arrest (more than 5 minutes), a period of cardiac compression before defibrillation may improve survival.[25]

As it is often difficult to quantify the duration of collapse, out of hospital, the new guidelines recommend a 2 minute period of CPR before attempted defibrillation in this group. This does not apply to an arrest witnessed by healthcare personnel, or for in-hospital cardiac arrest, where defibrillation is still recommended as soon as possible.

5. Role of Automated External Defibrillators (AEDs)

Early defibrillation is the single most important intervention which improves survival following cardiac arrest,[26] both out of hospital, when used by the general public[27] and in-hospital scenarios.[28]

Hence the new guidelines recommend the use of AEDs as soon as it is available. However, CPR is to be continued till the AED is available and while the AED pads are being positioned, as this often takes a few minutes.

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Current Approaches to Cardiopulmonary Resuscitation

Pharmacological therapies

Available evidence suggests that basic life support and defibrillation are the most important factors for ROSC, especially in out-of-hospital arrest.[29]

However the adjuvant use of drugs in this emergency setting may be beneficial. The new recommendations take into account the current best practice regarding pharmacotherapy.

1. Adrenaline (epinephrine)

Adrenaline (1 in 10,000) is recommended as a 1 mg dose (10ml) in all forms of adult cardiac arrest. The first dose should be given immediately after confirmation of rhythm before delivery of shock, or at the start of chest compressions in cases of non-shockable rhythm. Subsequently doses should be administered every 3-5 minutes.

Adrenaline was first used for resuscitation in the early 1900s.[30]

In vitro evidence has shown that adrenaline helps in the ROSC via its actions on alpha-adrenergic receptors; essentially acting as a vasoconstrictor. The most important factor in restoring spontaneous circulation appears to be development of adequate coronary blood flow by increasing coronary perfusion pressure (aortic diastolic minus right atrial pressure). Adrenaline also helps in the redistribution of blood, by decreasing splachnic circulation, and increasing blood flow to the heart and brain.

However, as adrenaline is a nonselective adrenergic agonist, it also acts on the beta-adrenergic receptors, and probably increases the oxygen consumption of the fibrillating myocardium, a potentially deleterious effect.[31,32]

Despite the widespread use of adrenaline during resuscitation, there is a distinct lack of 'in vivo' evidence demonstrating that any vasopressor given routinely at any stage increases the rate of survival. As such a proposal to remove all recommendations was considered. However, both due to an absence of good, placebo controlled, data and due to in-vitro evidence of the beneficial effects of adrenaline on haemodynamics during cardiac arrest, this proposal was not approved.[33]

Dose of adrenaline

There has been some controversy regarding the appropriate dose of adrenaline during cardiac support, with some animal data favouring high dose regimens.

In a large multicentre prospective study, 3327 patients with out-of-hospital cardiac arrest, were randomly assigned to 2 groups, one receiving 1 mg of adrenaline, while the other received 5 mg. Up to 15 doses were given at an interval of 5 minutes.

There was no significant difference in the rates of discharge from hospital or neurological recovery at discharge, when higher doses of adrenaline were used.[34]

2. Vasopressin vs. Adrenaline

The new guidelines do not incorporate the use of vasopressin in resuscitation.

Vasopressin is an endogenous peptide, which, at high concentrations, causes vasoconstriction via V1 receptors. Its vasoconstrictor properties have aroused interest in the context of cardiac arrest. Its longer half-life (about 20 minutes), and its efficacy in the acidic milieu makes it an attractive alternative to adrenaline. A small study published in 1997 suggested better return of ROSC and higher 24 hour survival in out-of-hospital cardiac arrest with 40 IU of vasopressin, compared to adrenaline.[35]

However, subsequent larger studies have failed to show any advantage over adrenaline.[36,37]

A recent meta-analysis concluded that there is yet no clear advantage of vasopressin over adrenaline in the management of cardiac arrest.[38]

3. Atropine

3 mg of atropine is recommended as a single dose after the first dose of adrenaline in cardiac arrest associated with asystole, and slow PEA (HR <60/minute). The dose is based on the maximum vagolytic effect of atropine.

Atropine is an anticholinergic drug with a half-life of 2 hours. Though it is used in the management of bradycardic arrest and pulseless electrical activity, there is very little evidence to show that it is effective.

4. Antiarrythmics

The new guidelines recommend amiodarone 300 mg as a bolus injection, if VF/VT persists after three shocks, during the brief rhythm analysis before delivery of the fourth shock. A further dose of 150 mg may be given for recurrent or refractory VF/VT, which is to be followed by an infusion of 900 mg over 24 h.

There is no evidence that routine administration of any antiarrhythmic drug increases survival during cardiac arrest.

Amiodarone is used in the management of shock-refractory ventricular fibrillation and tachycardia. The use of this drug is based on the findings of two clinical trials in out-of-hospital-cardiac arrest.[39,40]

However, inspite of improved short term outcome (survival to hospital admission), there is no evidence to suggest that amiodarone improves survival to hospital discharge.

Given the lack of evidence of pharmacologic therapies improving long-term outcome after cardiac arrest, the new guideline deemphasizes the role of drugs, and emphasizes the importance of basic life support.

Post Resuscitation care

This is now receiving greater attention in the management of cardiac arrest. The principal objective is to restore effective perfusion of different organ systems, and to treat the consequences of hypoxaemia/ischaemia suffered during the cardiac arrest.

The measures include haemodynamic support (including the management of myocardial stunning, and cardiac arrhythmias), prevention of hyperthermia (which can result following hypoxic injury to the brain, and has been shown to cause exacerbation of brain injury), and

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avoidance of hyperglycaemia. However, there is a lack of standardized care pathways.

Therapeutic Hypothermia

The International Liaison Committee on Resuscitation (ILCOR) has recommended that unconscious adult patients with spontaneous circulation after out-of-hospital cardiac arrest should be cooled to between 32°C and 34°C for 12 to 24 hours when the initial rhythm is VF

For favourable neurologic outcome following cardiac arrest, it is imperative to address any CNS ischaemia, caused by the circulatory failure, and also to reduce the severity of post-resuscitation syndrome. This is characterised by increased inflammatory response, production and activation of cytotoxic free radicals, which result in multisystem dysfunction, and increased morbidity and mortality.[41,42]

Hypothermia has been shown in experimental studies to have a multifactorial neuroprotective effect.[43-49] The exact mechanism of the protective effect is not clear, and may be related to reduction in cerebral oxygen consumption, reduction of free radical formation, slowing down of deleterious enzymatic reactions, and other multifactorial physic-chemical mechanisms. Two clinical trials demonstrated the improvement in survival and neurological outcome in patients with out-of-hospital cardiac arrest (OHCA). These trials involved active cooling to 32-34 degree C for 12 -24 hours.[50,51]

A recent metanalysis of the clinical studies involving post cardiac arrest patients suggests that mild therapeutic hypothermia (<35 degree C), initiated within 6 hours of arrival to the hospital improves short term survival and neurologic outcome. This was independent of the method of inducing hypothermia.[52] Such cooling may also be beneficial for other rhythms or in-hospital cardiac arrest.[53]

Therapeutic hypothermia is not recommended for patients with cardiogenic shock or persistent life threatening arrhythmias, pregnancy and known coagulopathy.

Further research is needed to identify optimal methods of cooling, timing and duration, before there is universal acceptance.

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