Cardiopulmonary-cerebral resuscitation

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Introduction

Cardiopulmonary resuscitation (CPR) is necessary whenever the oxygen supply to the brain is insufficient to maintain cerebral function. Oxygen supply to the brain is dependent on:

1. Cardiac output

2. Haemoglobin concentration
    $CaO_2 = $ Hb g/dl x SaO_2 x 1,34ml O_2/dl + (PaO_2 x 0,003)

3. Oxygen-haemoglobin saturation, which is mainly dependent on respiratory function.

CPR is thus essential in any condition in which cardiac output is critically low.

Cerebral hypoxia

The brain is the organ most sensitive to hypoxia. The brain cannot store oxygen and has a very limited capacity for anaerobic metabolism. Permanent brain damage will thus result after three to four minutes of total hypoxia at normal temperatures. Should cerebral blood flow be restored after such an incident, the patient may survive, but usually not without some form of permanent neurological damage.

The factors most often associated with poor outcomes after cardiac arrest are:
•   Prolonged arrest time before CPR;
•   Prolonged ventricular fibrillation without definitive treatment;
•   Inadequate coronary and cerebral perfusion during cardiac massage.

The only modalities which are accepted universally as effective during CPR are:
•   Rapid application of closed chest compressions; and
•   Early defibrillation.

The most common cause of brain damage after cardiac arrest is delay in commencing compressions. When the diagnosis of cardiac arrest is made, CPR should be initiated immediately.

Diagnosis of cardiac arrest

1. **An unconscious patient and absence of breathing**. These are the only diagnostic signs of cardiac arrest. Absent central pulses (e.g. carotid, femoral) can confirm the diagnosis. During anaesthesia, it may be difficult to distinguish between severe hypotension and cardiac arrest. If central pulses are not palpable, external cardiac massage must be initiated immediately.

    It is also of extreme importance to commence CPR if the cardiac output has decreased to such an extent that tissue perfusion and oxygenation might be inadequate. In this situation, a slow or faint pulse may still be palpable. Clinically it may be observed as dramatically decreasing oxygen saturation on the pulse oximeter.

2. **Dilated pupils**, usually within one minute after cardiac arrest. This clinical sign is not valid during general anaesthesia. Certain drugs also affect the pupil size (e.g. adrenaline and atropine).

3. **Cyanosis** of central origin.

4. **No bleeding** from the surgical wound.

5. **Appropriate ECG trace**.

    a. **Ventricular fibrillation (VF)** in 80% of cases. Fine fibrillation can be mistaken for asystole. This can be excluded by simply moving one electrode.

        Recent research indicates the incidence of VF to be somewhat lower than initially

thought, in the region of 60 - 70%. There is also evidence of VF spontaneously converting to asystole after a short duration, and this is much more difficult to resuscitate.

b. **Asystole** in 5 - 15% of cases, possibly as high as 25 - 30%.

c. **Pulseless Electrical Activity (PEA)** in less than 5% of cases. This implies a normal ECG trace with no cardiac output (no central pulses). The causes of PEA include ruptured abdominal aorta aneurysm resulting in no preload, severe or bilateral tension pneumothorax, and acute cardiac tamponade.

An easy way of remembering the causes of PEA:

Hypovolaemia	Tablets and toxins
Hypoxia	Tamponade cardiac
Hypothermia	Tension pneumothorax
Hydrogen ions (acidosis)	Thrombosis (myocardial
Hyper- and hypokalaemia	infarction)
Hypoglycaemia	Thrombosis (pulmonary
	embolism)
	Trauma (hypovolaemia)

The phases of CPR

Phase I

Basic life support which attempts to keep the patient alive, i.e. cardiac compressions, ventilation, intubation, infusion, etc.

Phase II

Restoring cardiac function by means of defibrillation and adrenaline.

Phase III

Increasing and maintaining the cardiac output with drugs after the heart has started beating again.

Physiology of the circulation during closed chest compressions

During closed cardiac compressions, two mechanisms come into action.

1. The **cardiac pump mechanism**, in which the heart is compressed between the sternum and the spine, resulting in ejection of blood into the aorta. The venous valves prevent retrograde flow.

2. The **thoracic mechanism**, by which chest compressions result in an increase in the intra-

thoracic pressure, forcing the blood out of the thorax, while the venous valves and the dynamic venous compression prevent backward flow. The heart acts as a passive conduit.

Fluctuations in intrathoracic pressure play a major role in blood flow during CPR, with the cardiac pump contributing to a lesser degree under some circumstances. Which mechanism predominates varies from patient to patient, and even during CPR in the same patient.

Cardiac output is severely depressed during CPR, and ranges from 10 - 33% of pre-arrest values in experimental animals. The majority of cardiac output is directed to organs above the diaphragm. Brain blood flow is 50 - 90% and cardiac flow 20 - 50% of normal, while lower extremity and abdominal visceral flow are reduced to only 5% of normal. Total blood flow tends to decrease as time elapses, but the relative distribution of flow does not change. Flow to the brain and heart is improved by the administration of vasopressors, whilst flow to the rest of the body below the diaphragm is unchanged or even reduced further.

Assessing the adequacy of circulation during CPR

If successful resuscitation is achieved, myocardial blood flow will be 15 - 30 ml/min/100 g body mass. To achieve such flows, closed chest compressions must generate adequate cardiac output and coronary perfusion pressures.

Coronary perfusion occurs primarily during the diastolic phase of chest compression. Myocardial blood flow is optimum during CPR when aortic diastolic pressure exceeds 40 mmHg and myocardial perfusion pressure exceeds 20 – 25 mmHg. (Aortic end-diastolic pressure minus right atrial diastolic pressure). Vascular pressures below the critical levels are associated with poor results; however, pressures above the desired levels do not ensure success.

Exhaled CO_2 is an excellent non-invasive indicator of effective resuscitation effort. After intubation, CO_2 levels are primarily dependent on blood flow rather than ventilation. Decreased pulmonary blood flow during CPR causes lack of perfusion to many alveoli. The alveolar content of these units has no CO_2 in the mixture, thus mixed-alveolar end-tidal CO_2 will be very low and correlate poorly with arterial CO_2. However, if flow during CPR starts to increase, more alveoli are perfused and less alveolar dead space occurs, and end-tidal CO_2 measurements rise. If CPR is successful, CO_2 values of 20 mmHg indicate successful CPR.

When spontaneous circulation resumes, an increase in end-tidal CO_2 values of up to 40 mmHg indicates effective circulation. Thus, within a wide range of cardiac output, end-tidal CO_2 corresponds with coronary perfusion pressure, cardiac output and survival. End-tidal CO_2 measured in humans during CPR can be used to predict outcome. No patient with an end-tidal CO_2 value of less than 10 mmHg can be successfully resuscitated.

Be aware that the administration of $NaHCO_3$ results in the release of CO_2 in venous blood and a temporary rise in end-tidal CO_2. Therefore, end-tidal CO_2 monitoring will not be useful for judging the efficacy of chest compressions for a period of 3 – 5 minutes following the administration of $NaHCO_3$.

It has now been proven that cardiac arrest due to ventricular fibrillation can be divided into three phases:

1. The **electrical phase** occurs during the first 4 – 5 minutes of arrest. Early defibrillation is critical, during this period, for achieving success. This is why the benefit of the automatic external defibrillator (AED) has been shown in a wide variety of settings.[1]

2. The **haemodynamic phase** (or circulatory phase) follows for the next 10 – 15 minutes, when perfusion of the myocardium with oxygenated blood is essential. (Some authors maintain this period lasts only 5 minutes.) Compressions are the mainstay of this phase.[2] During this time, generation of adequate cerebral and coronary perfusion pressure is critical to neurologically normal survival. However, if an AED is the first intervention applied during this phase, the subject is much less likely to survive. As time passes without perfusion, ATP stores become depleted. Accompanying the decrease in ATP, myocardial ischaemia produces CO_2 and H^+, creating an acidic environment. Cardioversion during this timeframe may convert a potentially salvageable rhythm to a terminal asystole or pulseless electric activity. With an optimal chest compression strategy providing improved coronary perfusion, ATP may be increased, thus creating a more ideal environment for successful electric cardioversion

3. The **metabolic phase** is characterised by ischaemic injury to the heart. This injury can be so destructive that there is uncertainty as to what interventions will be successful. This is the phase for which innovative new concepts are needed, the most promising of which seems to be the application of hypothermia.[3]

It is obvious that early intervention is of the essence. Bystander intervention should take place as soon as possible. However, this may not happen for three reasons:

1. Lack of training;
2. Complexity of the task;
3. Fear of harm.

Many concerns focus on mouth-to-mouth ventilation. However, experts now stress the importance of continuous closed chest compressions at the expense of ventilation. Research shows that, in a witnessed arrest with cardiac origin, closed chest compressions without ventilation are as efficacious as full CPR. The possibility of intubation should be available in a relative short period of time thereafter.

Importantly, compressions should not be interrupted, as is often the case for short periods of time for repeated evaluations, ventilations, intubation, and central line placements. These interruptions are detrimental to the ultimate success of the resuscitation attempt.

The management of cardiac arrest

When cardiac arrest has been witnessed, the following procedures should be carried out immediately.

1. **Precordial thump.** Administered with the side of a clenched fist. This may defibrillate the heart in cases of ventricular fibrillation. This manoeuvre is done only in a "witnessed arrest", and only as a single thump. If the arrest occurred a few minutes earlier, no precordial thump should be executed.

2. **Early defibrillation**. This is the major determinant of survival in cardiac arrest due to ventricular fibrillation. In the case of a witnessed arrest where a defibrillator is **immediately available**, an **immediate defibrillation** is done. Apply a shock of 360 J with older defibrillators, and 150 J with the newer biphasic defibrillation machines. A precordial thump is not done under these circumstances. Should the arrest have taken place some minutes earlier, the normal CPR techniques should be applied.

Reports mention that delays in recharging the defibrillator may be detrimental to the whole effort of CPR. Thus, immediately after giving a single shock, and without reassessing the rhythm or feeling the pulse, resume CPR (30 compressions with 2 ventilations) for 2 minutes before delivering another shock. Even after successful defibrillation, it is very difficult to feel a pulse after defibrillation. The delay in trying to feel pulses will further compromise the myocardium

if a strong rhythm has not been restored. The single shock strategy is applicable to both monophasic and biphasic defibrillators.

All cases of witnessed cardiac arrest must be immediately defibrillated. This should be carried out even in cases with an asystolic trace on the screen, as fine fibrillation and displacement of the electrical axis of the heart can simulate asystole. Defibrillation is not the usual treatment for proven cases of asystole.

The principle of early defibrillation is supported by the following:
- The majority of cases of cardiac arrest are initially VF;
- The most effective treatment of VF is defibrillation;
- The probability of defibrillation being successful decreases over time;
- VF tends to convert to asystole after a short period of time.

A survival rate of 90% has been suggested if defibrillation occurs within 1 minute after arrest. This survival rate decreases dramatically to 50% after 5 minutes, 30% after 7 minutes, 10% after 9 - 11 minutes, and 2 - 5% if defibrillation is delayed for more than 12 minutes.

Although previous guidelines have recommended immediate defibrillation for all witnessed arrests, recent evidence suggests that a period of immediate CPR before defibrillation may be beneficial after prolonged collapse.

Defibrillation

The placement of the paddles is to the right of the sternum below the right clavicle, and lateral to the left nipple in the mid-axillary line. Electrode paste or normal saline should be placed between the electrodes and the skin. Stand clear of the bed when defibrillating.

New guidelines: Deliver a single shock at 150 J (biphasic). Immediately continue with cardiac compression at a tempo of 100 per minute.
- Children: 2 J/kg initially; 4 J thereafter.
- Open chest defibrillation: 50 J (with internal paddles).

The purpose of resuscitation is to restore oxygen delivery to vital organs, such as the brain, heart and kidneys, as rapidly as possible. The circulation should be maintained by external cardiac massage (ECM) and oxygenation by artificial ventilation until spontaneous action of the heart has been restored. After the initial defibrillation in a witnessed arrest, and only after CPR has been commenced, may one proceed to the administration of adrenaline with sequential cycles of defibrillation.

With a fixed arrest (longer than 3 minutes), all methods to ensure successful CPR should be undertaken, e.g. endotracheal intubation, intra-venous infusion, diagnosis. This is followed by, firstly, the administration of adrenaline, and then defibrillation of one shock at 360 J (150 J with the new biphasic machine). This action should be repeated three times during a period of 10 - 12 minutes, during which time only adrenaline and defibrillation is administered.

Adrenaline administration should be continued during asystole until the patient starts fibrillating, after which he or she may be defibrillated.

CPR must be continued during defibrillation efforts at all times.

External cardiac massage

ECM is the only way in which blood can be circulated when cardiac arrest has taken place and, therefore, it is imperative that ECM should be commenced immediately. The exception to this rule is when cardiac arrest has taken place due to hypoxia. Ventilation will then have first priority. This is particularly important in children.

In adult cardiac arrest, compressions have now taken preference over ventilation. The reasons for this are as follows:
- The lungs usually contain enough oxygen to prevent serious desaturation of the blood for up to 3 minutes or longer. This oxygen is stored in the functional residual capacity (FRC) of the lungs. Therefore, the two early rescue breaths are actually ignored in cases of witnessed arrest.
- The brain is more resistant to hypoxia than to ischaemia.
- It is usually easier to commence ECM than artificial ventilation. Compressions should be done at a rate of 100 per minute.
- If ECM is initiated before the myocardium becomes hypoxic, the ECM per se may cause spontaneous defibrillation.

Haemodynamics of ECM

It is accepted that the reason for forward blood flow during ECM is primarily due to the increase in intrathoracic pressure. Of less importance is the compression of the heart between the sternum and spinal column.

Together with the above are heart valves, valves in the venous system and compression of the thin walled

veins due to the raised intrathoracic pressure, which ensure that blood flows forward, i.e. in the direction of the aorta, arteries and vital organs. Arteries like the aorta are not compressed and blood flows in the direction of least resistance.

Positive pressure ventilation also contributes to the raised intrathoracic pressure, leading to improved cardiac output during CPR. It is therefore imperative that the patient is intubated and mechanically ventilated as soon as possible.

When using the Active Compression-Decompression (ACD) apparatus, during the passive and active decompression phase of the thorax cavity, air and blood is entrained into the thorax due to a negative pressure being created. Negative pressure is not created during standard CPR, but is of much more importance during the active decompression phase and is also maintained much longer.

Factors that determine this negative pressure
• Patency of the airways
• Degree of bronchial and thoracic compliance and collapse
• Degree of elasticity
• Thoracic wall compliance
• Efficiency of suction

Contributing mechanisms:
• Circulating blood volume
• Myocardial diameter and compliance (stiffness)
• Right atrial compliance
• Power of thorax compression

Venous return to the heart is dependent on:
• The difference between the pressure in the peripheral venous system and that in the right atrium.
• Diastolic pressure difference between the large veins and the thorax.
• Blood flow in the large veins.

Cardiac output is reflected by the gradient between left ventricle and peripheral arterial pressure. Numerous studies have shown the advantages of attaining increased thoracic pressures by increased sternal displacement, ensuring an improved cardiac output.

Very little was done to improve right atrial filling until active compression-decompression was introduced.

Cardiac output and blood flow are determined by:
• The volume of blood in the left ventricle
• The velocity of blood flow through the mitral valve
• The forward flow of blood to the thoracic cavity and heart

• The flow of blood between the heart chambers
• End compression volume
• Stroke volume

Ventilation and end-tidal CO_2

End-tidal CO_2 (EtCO_2) gives an indication of the cardiac output but is dependent on circulation, adequate ventilation and CO_2 production. To determine the efficiency of CPR, CO_2 levels should ideally be measured in the pulmonary artery and, therefore, a pulmonary artery catheter (Swan-Ganz catheter) should be inserted. This is not always feasible.

Ventilation by means of endotracheal intubation should be commenced immediately in order to maintain acceptable CO_2 levels.

Recommended techniques for ECM

If cardiac arrest occurs during anaesthesia the following should be done:
• Inform the surgeon.
• Place the table in the Trendellenburg position.
• Cease the administration of all anaesthetic agents.
• Ventilate the patient with 100% oxygen.
• Initiate ECM, even if the abdomen is open.

Cardiac compressions and ventilation should take place simultaneously in the intubated patient. The patient should be resuscitated on a resuscitation board or a table with a firm surface.

The correct compression technique should be applied. The rescue worker stands on the right side of the patient, places one hand over the middle part of the sternum (the other hand on top of the first) and commences compression with the heel of the lower hand. The elbows are locked, arms are straightened and thrusts are performed in a straight down direction at a tempo of 100 per minute. The sternum must be depressed by 5 cm in the normal-sized adult.

ECM techniques

The European Resuscitation Council Guidelines for Resuscitation (2005) contain several changes in comparison to the guidelines that were published in 2000.

A. If resuscitation is being done by one person alone, the method of 30 compressions followed by 2 ventilations should be applied at a tempo of 100 per minute.

B. If two people are available, different techniques can be applied.

i.   Traditional method

In the unintubated patient, alternate cardiac compressions and mouth-to-mouth ventilations at a ratio of 30:2, with a pause for ventilation of 1,5 - 2 seconds, are applied.

In the intubated patient, the currently accepted method is ECM at a rate of 100/min with ventilations at 10 breaths/min. The actions occur independently of each other. If cardiac compressions and ventilations happen to occur simultaneously, the intrathoracic pressure is increased further, improving cardiac output for that specific moment.

ii.   Alternating abdominal and cardiac compressions (six-handed technique).

Endotracheal intubation is mandatory, as regurgitation and aspiration is a real danger when this technique is used. The abdominal compressions are applied mainly in the right hypochondrium, in an upward direction towards the thorax. The pressure is approximately 25% of that applied to the chest during ECM. The pressure on the liver and large veins increases venous return to the right side of the heart, with a consequent increase in cardiac output. Ventilation is carried out at the usual rate (10 breaths/min), with 100% oxygen.

This technique requires three people who have been adequately trained and is not recommended for the casual resuscitator. Incorrect application of this technique will cause more harm than good to the patient. The patient should also be intubated to prevent aspiration.

iii. Active compression-decompression method

This method entails active compression followed by active decompression of the thoracic cavity. An apparatus equipped with a large suction cup is firmly applied to the chest and, with vigorous up-and-down movements, blood is actively drawn into the chest during the negative pressure phase. This increases venous return and cardiac output. These movements are executed at a tempo of 100 times per minute. Although a fair amount of air is entrained into the lungs during the decompression phase, it is recommended that the patient be intubated for optimal manual ventilation. Research shows that short-term haemodynamics are improved by between 11 and 43% compared to traditional hand compressions and mouth-to-mouth ventilations. Studies confirming long-term advantages are not available.

iv.   Simultaneous compression and ventilation

This technique necessitates endotracheal intubation. The rate of ventilation is much more rapid than in the traditional method (i), a simultaneous ventilation being given for each compression, but the consequent increase in intrathoracic pressure results in vastly improved cardiac output. The goal of the rapid ventilatory rate is to counteract the initial respiratory acidosis resulting from washout of CO_2. This is not at all recommended

Open cardiac massage

It has been claimed that open cardiac massage is more effective than ECM but, due to complications, this technique is not routinely applied any more. Open cardiac massage is only performed under the following circumstances:
• Tension pneumothorax
• Serious chronic obstructive airway disease e.g. emphysema
• Cardiac tamponade
• Where the chest is already open

Artificial ventilation

Rescue ventilation

If mouth-to-mouth ventilation is not carried out correctly, regurgitation and aspiration can be a danger during CPR. Low ventilatory pressures are applied in an attempt to avoid forcing open the cardio-oesophageal sphincter. A pause of 1,5 – 2,0 seconds during ECM is necessary to attain the recommended 500 ml tidal volume in adults.

Concern has been expressed about the possible risk of transmitting diseases during CPR. It has never been proven that HIV can be transmitted in this way. However, diseases like tuberculosis and severe acute respiratory syndrome (SARS) could possibly. To avoid direct contact with the patient's mouth, different barrier devices are available. Many of these are not very effective. The application of a handkerchief to the patient's mouth, or the use of an airway device which protrudes above the patient's face (Resusciade) can be considered.

All cases of cardiac arrest should be intubated as soon as possible to facilitate artificial ventilation.

Endotracheal intubation

Endotracheal intubation allows easier and more effective ventilation. Intubation should be performed within 30 seconds.

The reasons for endotracheal intubation include:
- Administration of 100% O_2.
- Increasing intrathoracic pressure to improve cardiac output.
- Protecting the airway against aspiration after regurgitation and vomiting.
- Preventing shunting in the lungs by applying high positive ventilation pressure, thereby forcing open alveoli and preventing atelectasis.
- Administration of certain drugs by this route (e.g. adrenaline, atropine, vasopressin and lignocaine) in the absence of an open line.

New American guidelines

It is estimated that, annually, between 250 000 and 450 000 patients die in the United States after sudden cardiac arrest. The majority of these deaths occur before the patient reaches the hospital. There are no statistics available for South Africa.

Cardiocerebral resuscitation (CCR) is a new approach to the resuscitation of patients with cardiac arrest. It is composed of 3 components:[3]

1. Continuous chest compressions for bystander resuscitation at 200 compressions (100 per minute);
2. A new emergency medical services (EMS) algorithm; and
3. Aggressive post-resuscitation care.

The CCR method has been shown to dramatically improve survival in those patients most likely to survive i.e. witnessed arrest and shockable rhythm on arrival of EMS. The CCR method advocates continuous chest compressions without mouth-to-mouth ventilations for witnessed cardiac arrest.[3] (See below.)

However, EMS personnel most often arrive after the electrical phase, in the circulatory phase of VF arrest. By this time, the fibrillating myocardium has consumed most or all of its energy stores, and chest compressions that perfuse the heart are mandatory prior to and immediately after a defibrillator shock. Endotracheal intubation is delayed, excessive venti- lations are avoided, and early administration of adrenaline is advocated.

Major predictors of survival of patients with out-of- hospital primary cardiac arrest are witnessed collapse and a shockable rhythm. Since early bystander- initiated resuscitation efforts prolong the electrical or shockable phase of untreated ventricular fibrillation, areas with higher rates of early onset bystander resuscitation efforts and shorter EMS response times have higher survival rates.

The first problem contributing to the poor survival rates of out-of-hospital cardiac arrest is the lack of bystander-initiated basic CPR. Although the majority of out-of-hospital cardiac arrests are witnessed arrests, only one patient in five receives bystander- initiated CPR. In the absence of early defibrillation, bystander-initiated chest compression is essential for improved survival for patients with out-of-hospital cardiac arrest.[4]

If it wasn't a witnessed arrest, one should assume that the patient is in the haemodynamic phase of cardiac arrest. It is then recommended to give 200 chest compressions, deliver one shock, and perform another 200 chest compressions before rhythm analysis. As noted above, this sequence is followed 3 times before an attempt is made to intubate. Before intubation, the patient is ventilated via bag-valve mask.

Excessive ventilation is a major problem in CPR, reducing the chances of survival. With simultaneous chest compressions and ventilations, there is a dramatic increase in intrathoracic pressure, decreasing venous return and, thus, perfusion pressures. Aufderheide et al found that 12 to 15 ventilations per minute are preferable to the near 30 ventilations per minute that are often delivered. [5]

The major EMS modifications include the following:
- Eliminating the emphasis on early intubation and positive pressure ventilations.
- Eliminating the recommendation for immediate defibrillation, and the use of stacked shocks.
- Eliminating all unnecessary interruption of chest compressions.
- Advocating chest compressions before and immediately after a single defibrillation shock.
- Early administration of epinephrine.
- Use of passive insufflation of O_2

It was found that the survival rate after out-of-hospital cardiac arrest was superior with passive insufflation of oxygen when compared with active assisted ventilation.

A change in defibrillation application was also advocated. A single shock instead of stacked shocks, followed immediately by 200 compressions, improved survival rate dramatically.

In a study by Alex Garza et al the investigators concluded that the typical patient in cardiac arrest has a relatively prolonged downtime (usually beyond the 5 minute electric phase) with minimal resuscitative efforts during the first minutes. Therefore, the majority of cardiac arrest patients have not benefited from the addition of AEDs to EMS vehicles. Although

immediate defibrillation is lifesaving and beneficial to patients in the electric phase of cardiac arrest, it may be hazardous to patients in the circulatory phase.[6]

The myocardium is highly metabolic. Cardiac arrest is the ultimate insult to the myocardium, resulting in a substantial and severe energy deficit. This is particularly so during ventricular fibrillation arrest when the fibrillating heart still requires energy substrates, even more so than the normally beating heart. Substantial data show the gradual depletion of cellular energy substrates during cardiac arrest, and degradation of the fibrillation activity of the myocardium. Immediate countershock during the times of low-frequency fibrillation and loss of energy substrates have been shown to result in conversion to a nonsurvivable rhythm.[7]

Even more important was the finding that chest compressions before defibrillation lessen mortality for any patient, regardless of response time interval. It was apparent that responder arrival at the patient's side usually occurred during the transition from the electric to circulatory phase, or in the circulatory phase proper. Therefore, chest compressions must begin before attempted defibrillation in all cases except rescuer-witnessed sudden cardiac arrest. It is well documented, however, that increased compressions improve coronary perfusion pressure, which directly affects survival in both animal and human studies.

Studies have shown that persons receiving continuous chest compressions from bystanders had higher survival rates than those subjected to standard CPR with a pause for ventilation.

Although oxygen delivery to the myocardium is an important determinant of survival in cardiac arrest, it is believed that aggressive chest compressions with passive oxygen delivery will likely ensure adequate oxygenation for the patient. Using continuous insufflation of oxygen at 15 l/min through an endotracheal tube, it was possible to achieve equivalent oxygenation compared with mechanical ventilation in cardiac arrest patients.

Hyperventilation has been shown to be detrimental in both out-of-hospital and in-hospital resuscitation. Studies demonstrated that rescuers almost always significantly hyperventilate patients, whether they are intubated, or not. Furthermore, hyperventilation leads to decreased survival because of the resultant increase in intrathoracic pressure, which decreases coronary perfusion pressure in cardiac arrest patients.

Rescuers placed an oral airway immediately to correct or prevent any upper airway obstruction and provided continuous flow of oxygen via a nonrebreather mask. Two "gentle" ventilations were delivered over 2 seconds. The 2 ventilations were thought to bridge the time between the switching of the compression providers and to offer some ventilatory support. It was thought that this small number of ventilations would not produce increased intrathoracic pressures and would pose a minimal risk to the patient. Endotracheal intubation attempts were made after the first three series of 200 compressions were performed.

Ewy and Kern, both leaders in the field of cardiac resuscitation, reviewed CCR and described ideal postresuscitation care. They described the three pillars of CCR.[8]

1. Compression-only CPR by anyone who has witnessed the event.

Figure 1.
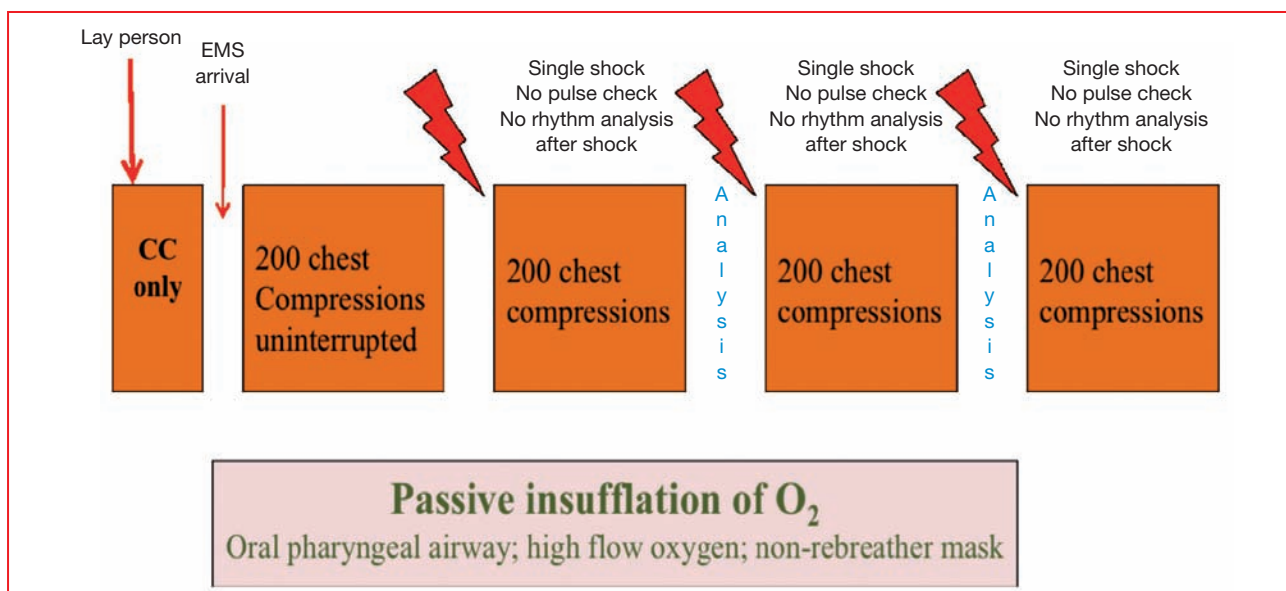
2. CCR by emergency medical service personnel, assumed to be arriving more than 5 minutes after the arrest:
   a. Administer 200 chest compressions (at 100/minute), and delay intubation; second person to apply defibrillation pads and initiate passive oxygen insufflation (i.e. 100% oxygen via facemask).
   b. Administer a single shock, if indicated, immediately followed by 200 more chest compressions (no pulse check after shock).
   c. Check for pulse and rhythm; note that this pulse check occurs 4 minutes after the CCR has begun.
   d. Administer adrenaline, intravenously or intra-osseously, as soon as possible, to improve central circulation, coronary circulation, and diastolic blood pressure.
   e. Repeat (b) and (c) 3 times; intubate if no return of spontaneous circulation after 3 cycles; note that neither bag-valve-mask ventilation nor intubation occurs until 12 minutes after the CCR has begun.
   f. Continue resuscitation efforts, with minimal interruption of chest compressions, until resuscitation is successful or the person is pronounced dead.

3. Postresuscitation care to include mild hypothermia (32 – 34 °C) for patients in coma postarrest. Urgent cardiac catheterisation and percutaneous coronary intervention, unless contra-indicated.[9]

Some facts and figures about CPR

- 4 minutes: time it takes for brain damage to set in, without adequate blood flow.
- 7 minutes: time it takes for irreversible brain damage to set in.
- 7 - 10%: reduction in the chance of survival for every minute of delay until defibrillation takes place.
- 95%: proportion of cardiac arrest victims who die before reaching the hospital.
- 49 - 75%: survival rate in adult patients when CPR plus defibrillation is performed within 3 to 5 minutes of collapse.
- 294 851: annual number of EMS-treated out-of-hospital cardiac arrests in the US.
- 100: number of compressions per minute to be delivered during CPR.
- 80%: proportion of all out-of-hospital cardiac arrests that occur in private homes.
- 2% - 10%: survival rate in paediatric patients who develop out-of-hospital cardiac arrest.
- 14 - 38%: proportion of CPR attempts by bystanders in out-of-home cardiac arrests.
- 89%: proportion of respondents willing to do something to help if witness to a medical emergency.
- 21%: proportion of respondents who were confident that they could perform CPR.
- 15%: proportion of respondents who believed they could use an AED in an emergency.

When asked why they did not perform CPR in an emergency situation, the most common reasons provided by resondents were:
- Lack of confidence;
- Fear of doing more harm than good; and, not surpisingly,
- Concern about legal consequences.

Drugs used in advanced cardiac life support

Oxygen

Though not really a drug, per se, adequate oxygen supply to the brain is at the core of CPR. Expired air delivers 16 - 17% of oxygen to the patient. This will produce an average alveolar oxygen tension of 80 mmHg. But there are factors which may result in inadequate oxygenation during mouth-to-mouth respiration. These include low cardiac output associated with ECM, intrapulmonary shunts, and ventilation/perfusion abnormalities.

This will result in hypoxaemia, due to alveolar-arterial O_2 tension differences, which necessitates the use of 100% oxygen under positive pressure ventilation as soon as possible to optimise haemoglobin saturation and oxygen delivery to the tissues. These patients must be intubated.

Intravenous fluids

Expansion of the circulating blood volume is critical in patients with acute blood loss. Until blood is available, this can be achieved by the rapid administration of crystalloid (preferably 0,9% saline or Ringer's lactate) and colloid solutions (starches e.g. Voluven, or gelatines e.g. Gelofusine). Should there be no loss of intravascular volume, either absolute or relative (such as in cardiac and anaphylactic shock), care should be taken not to overload the patient with fluid. Dextrose and water solutions should never be used.

Adrenaline hydrochloride (epinephrine)

Adrenaline is the top priority drug in cases of cardiac arrest, and should be administered immediately.

This drug is of benefit for the patient in cardiac arrest, mainly due to its α-adrenergic receptor stimulating properties. α-effects cause constriction of the skin and splanchnic vessels, while β_2-stimulation

causes dilatation of the vessels to the muscles. **The net effect is an increased peripheral vascular resistance**. This raises the total pressure against which the heart must contract (afterload). The aorta also undergoes vasoconstriction, and this raises the current end-diastolic pressure. The combined effect is that of improved myocardial and cerebral blood flow during mechanical ventilation and chest compression. In other words, it increases coronary and cerebral perfusion during ECM in diastole. During CPR, coronary perfusion occurs primarily during the diastolic phase. Critical myocardial blood flow is reached when aorta diastolic pressure exceeds 40 mmHg or coronary perfusion pressure exceeds 20 – 25 mmHg.

Myocardial blood supply increases, because adrenaline causes the aorta end-diastolic pressure to increase. The right atrial pressure stays constant, and an even greater pressure gradient is created with improved myocardial perfusion. We now have a resultant selective increase in the vascular tone of the non-cerebral and non-coronary vessels. An example of this is the increased cerebral flow due to vasoconstriction of the extracerebral peripheral arteries to the tongue and facial muscles, with subsequent redistribution of blood to the essential intracerebral area.

An additional beneficial effect is that the capacitance vessels also undergo slight vasoconstriction, which leads to central pooling of circulating blood and an increased preload as a result. This theory is, however, controversial.

The value and safety of the ß–effects of adrenaline also remain controversial. One view is that these are disadvantageous because of the resultant increase in myocardial work, viz. increased inotropy and coarsening of VF (positive inotropic effect), and decrease in subendocardial perfusion. A larger oxygen demand, therefore, arises. There is a difference in opinion regarding its positive inotropic effects and the tendency to convert an asystole or fine fibrillation to a coarse ventricular fibrillation, which can be seen as highly beneficial. One fact remains, however. If the heart is arresting due to asystole or fibrillation, ß-effects will have a negative influence on the myocardium. Nevertheless, the positive effects of α-stimulation outweigh the potentially negative effects of the ß-stimulation.

Adrenaline is the drug of choice for the treatment of asystole. The original opinion was that, by utilising the ß-effects, the heart muscle fibres will be made more excitable and the non-active fibres will be made to fibrillate, in either a coarse or a fine manner. In case of a fine fibrillation, the dose of adrenaline should be repeated until a coarse fibrillation pattern is established, after which this can be defibrillated.

Lately, it has been postulated that it is actually the α–effects of the drug that cause the heart to start contracting after a period of asystole. This is due to the resultant improved myocardial perfusion and increasing oxygen supply that restores the excitability of the myocardial cells. In other words, it is the availability of oxygen that activates the fibres again.

Dosage

The dose-response curve of adrenaline was investigated during the 1980s, and it was found that the optimal effects of adrenaline are obtained at dosages of 0,045 - 0,2 mg/kg. From these studies, it also became evident that higher doses of adrenaline are required to bring about improved haemodynamics and successful resuscitation. This is the origin of the principle of **high-dose adrenaline**.

Several studies during the 1990s failed to replicate these results, and thus prove that high-dose adrenaline is superior to the traditional 1,0 mg dose. Simultaneously, there was no proof that the higher dose is inferior to the traditional dose. However, subsequent studies have found that high cumulative doses of adrenaline have been associated with a poorer post-resuscitation haemodynamic and neurological outcome, although a causal relationship has not been established. High doses may result in improved circulation and coronary perfusion initially, but may also lead to a severe toxic hyperadrenergic state in the post-resuscitation phase.

The latest recommendations are that adrenaline 1,0 mg be administered every 3 to 5 minutes. The first dose must be administered as 10 ml of a 1:10 000 solution, purely as a method to allow mixing of drug with blood, or a 20 ml fluid flush must be administered after each dose to ensure that the drug reaches the central compartment.

Adrenaline is absorbed sufficiently by the tracheobronchial tree if given via the endotracheal tube. This is of value if intravenous access has not yet been established. In this case, give 8 mg, diluted in 20 ml of sterile water. Larger volumes allow better spread over the alveolar surfaces together with positive pressure ventilation. The latest literature questions the validity of this possible effect.

Caution should be taken with intracardiac administration, because of the possible dangers of coronary vasospasm, laceration, cardiac tamponade and tension pneumothorax.

Adrenaline is also associated with:
• Increased myocardial oxygen consumption,
• Ventricular dysrhythmias, and

- Myocardial dysfunction during the immediate post-resuscitation period.

The administration of adrenaline is followed by sequential cycles of 1 defibrillation 360 J (150 J biphasic). Once 3 cycles of adrenaline and defibrillation have been administered, one can proceed to the use of other medications.

Vasopressin

After the latest work done in Europe, arginine vasopressin (ADH) is strongly recommended as the primary pharmacological agent in the treatment of cardiac arrest.

The reasons for this are:
- Absence of the negative effects of the ß-adrenergic stimulation of adrenaline, which worsens fibrillation.
- Increased blood flow to vital organs.
- Increased cerebral O_2 delivery.
- Greater recovery of spontaneous circulation.
- Higher 24 hour survival rate.

Interestingly, after administration of equal doses of either intravenous or endobronchial vasopressin, equal coronary perfusion pressures were obtained. However, with endobronchial administration of adrenaline, a 10-fold higher dose was required to achieve an equivalent coronary perfusion pressure. Endobronchial vasopressin is absorbed rapidly and considerably increases the potential for a successful resuscitation.

The addition of nitroglycerine to vasopressin during cardiac resuscitation significantly improves endocardial blood flow. The endocardial:epicardial blood flow ratio also shows dramatic improvement, compared with the administration of vasopressin alone.

Vasopressin is a powerful vasoconstrictor, but does not have much direct effect on the blood pressure under normal physiological conditions. Its chief function is increased water absorption from the kidneys, with sodium retention and potassium excretion. The half-life of vasopressin in animal models with intact circulation is 10 – 20 minutes, which is longer than that of adrenaline during CPR.

Endogenous vasopressin levels in patients who have undergone CPR are considerably higher in those who survive compared with those who have no recovery of spontaneous circulation. This may indicate that exogenous vasopressin may have a greatly advantageous effect during cardiac arrest.

After a short period of VF, vasopressin is found to have the following effects:

- Increased coronary perfusion pressure;
- Increased vital organ blood flow;
- Increased ventricular fibrillation median frequency;
- Increased cerebral O_2 delivery.

The same effects are found in cases of extended cardiac arrest and PEA. Importantly, vasopressin does not cause bradycardia after recovery of spontaneous circulation.

The effects of vasopressin on smooth muscle are due to its action on V_1-receptors. During CPR, it causes profound vasoconstriction of skin, skeletal muscle, the gut and fatty tissue, with relatively little vasoconstriction of renal and coronary vessels and vasodilatation of cerebral vessels. No increase in O_2 demand occurs, because vasopressin has no ß-effects.

Laboratory studies have shown that vasopressin may be administered via the intra-osseus route at the same dose as the intravenous route.

An animal model to determine the dose of vasopressin found that, to attain an equipotent effect with adrenaline, 0,8 U/kg vasopressin was required.

In summary it can be said that, at the present, vasopressin is an effective alternative drug to adrenaline for ventricular fibrillation. It may also be effective in asystole and PEA. Vasopressin may be used effectively in cases where adrenaline has no effect during CPR. There is, at present, insufficient data to recommend the use of vasopressin as first drug of choice in CPR because the long-term outcome in patients leaving hospital does not differ significantly from that of adrenaline.

From the New England Journal of Medicine (2004)

"The effects of vasopressin were similar to those of adrenaline in the management of VF and PEA, but vasopressin was superior to adrenaline in patients with asystole. Vasopressin followed by adrenaline may be more effective than adrenaline alone in the treatment of refractory cardiac arrest."

From the ASA meeting (Atlanta, 2005)

"Latest recommendation is that it should only be used as an alternative to the first dose of adrenaline during ventricular fibrillation in a dose of 40 units IV. For additional vasopressor therapy and for asystole or PEA, adrenaline is recommended.

It should not be used in the awake or conscious patient with coronary artery disease, because it may provoke an attack of angina due to the increased peripheral vascular resistance.

Further studies have found that the haemodynamic effects of vasopressin, compared to adrenaline, are especially impressive during long cardiac arrests. The use of vasopressin therefore may be of great benefit in prolonged resuscitation efforts."

Drugs used in asystole

In cases of asystole, it is now recommended that atropine (not glycopyrrolate) be given, because many cases of asystole may be of vagal origin. It is therefore necessary to administer atropine 3 mg (as a single dose) immediately, when the diagnosis of asystole or PEA (if the cause is vagal) is made. The American guidelines suggest a dose of 1 mg every 10 minutes up to a maximum of 3 mg.

Drugs used for control of heart rate and rhythm

Lignocaine

Lignocaine is still the drug mostly used for the management of ventricular fibrillation resistant to defibrillation, ventricular tachycardia and ectopy. Lignocaine stabilises cellular membranes and suppresses electrical activity by blocking sodium channels. Lignocaine per se will not reverse fibrillation to sinus rhythm. It merely renders the cells less excitable. After lignocaine has been administered, defibrillation should again be commenced in order to restore the normal rhythm.

A bolus dose of 1,5 mg/kg should be administered, followed every 8 - 10 minutes by a dose of 0,5 mg/kg, up to a maximum of 3 mg/kg. This regimen can be followed by a constant infusion of 2 - 4 mg/min, once normal rhythm has been established.

As lignocaine is a negative inotropic drug, care should be taken in the following cases:
• low cardiac output states;
• congestive cardiac failure;
• shock;
• patients suffering from liver disease; and
• patients older than 70 years.

Commence with half the bolus dose and be on the lookout for signs of toxicity. The therapeutic-to-toxic levels are delicate. There is serious disapproval of the administration of lignocaine in myocardial infarction.

Although lignocaine is still used as an antidysrhythmic drug in the treatment of VF and pulseless VT, the true effectiveness in these cases is questioned. Lignocaine is thus no longer approved for cases of ventricular dysrhythmia in acute myocardial infarction.

The negative inotropic effects of lignocaine may be considerably more prevalent than its positive effects,

and care must be taken to monitor myocardial depression.

Amiodarone

Amiodarone is a pharmacologically complex drug with sodium, potassium, calcium, and α- and ß-adrenergic blocking properties that are useful for the treatment of atrial and ventricular dysrhythmias. Amiodarone can cause bradycardia and a severe reduction in blood pressure when administered too rapidly. This can be treated by slowing the rate of administration, and also by the administration of efficient amounts of fluids, vasopressors and chronotropic drugs.

Amiodarone is becoming popular as the drug of choice for the control of resistant dysrhythmias during CPR.

Amiodarone is a class IIB antidysrhythmic drug, and it is recommended that it should be used before lignocaine in the initial treatment of haemodynamically stable broad-complex tachycardias. It can also be used effectively in haemodynamically unstable VT, and in VF.

Amiodarone has vasodilatory and negative inotropic effects, which may worsen an already haemodynamically unstable state. These effects are dose-dependent and are also influenced by rate of administration.

Amiodarone is recommended in cases of shock-refractory cardiac arrest due to VF or pulseless VT. This means that the drug may be administered in cases where sustained VT occurs after defibrillation has been performed and adrenaline has already been given.

It is alleged that amiodarone has greater effectiveness and fewer prodysrhythmic effects than other antidysrhythmic drugs under similar conditions.

Dosage

150 mg administered over 10 minutes, dissolved in 20 ml dextrose water, followed by 1 mg/min infusion for 6 hours, followed by 0,5 mg/min.

Supplementary doses of 150 mg can be repeated as required for recurrent or resistant dysrhythmias, to a maximum recommended dose of 2 g per 24 hours.

The dose during cardiac arrest, due to VF or pulseless VT, is 300 mg in 30 ml normal saline or 5% dextrose water, given intravenously, over a period of 10 minutes.

The initial dose may be followed by the supplementary dose already mentioned.

Although these effects must still be confirmed, amiodarone seems to produce better outcomes than any of the other antidysrhythmic drugs.

The disadvantages of amiodarone include:
• hypotension;
• bradycardia;
• high cost;
• difficult method of administration: presently available as a 6 ml ampoule, the drug must be diluted to 20 ml with 5% dextrose water before administration.

Procainamide

Procainamide is a class 1A anti-arrhythmic drug, and it increases the threshold to electric stimulation in both atria and ventricles. Conduction in the atria and ventricles is diminished, while the refractory period of the atria is prolonged.

In cases of severely resistant VF, it may be used to stabilise the situation. It is also recommended, instead of lignocaine, in cases of haemodynamically stable broad complex tachycardias.

Dosage

The normal dose is an infusion of 20 mg/min, until:
• The dysrhythmia is suppressed;
• Hypotension occurs;
• The QRS-complex is lengthened to 50% of the original complex; or
• A total dose of 17 mg/kg has been administered.

In serious situations, up to 50 mg/min can be administered, up to the maximum dose of 17 mg/kg. The maintenance dose of procainamide is 1 – 4 mg/min, and must be reduced in cases of renal failure.

Magnesium sulphate

Magnesium sulphate is presently being used as an antidysrhythmic drug during cardiac arrest. The reason is that Mg^{2+} counteracts the release of Ca^{2+}, which originates during ischaemia of the brain.

It is also recommended in hypomagnesaemic states and in "torsades de pointes". The recommended dose is 1 – 2 g in 100 ml 5% dextrose water, given slowly intravenously.

Beta-adrenergic blockers

Beta-blockers can be used to treat repeated episodes of supraventricular tachycardia that can compromise cardiac output. The dose of propanolol that must be administered is 1 - 3 mg every 5 minutes intravenously, up to a maximum of 0,1 mg/kg. Propranolol should not be administered initially.

Atropine sulphate

Atropine is indicated in the treatment of sinus bradycardia not due to hypoxia, and also for AV block at the level of the AV node. Atropine can cause a nodal rhythm.

Atropine is now also administered in cases of asystole. Very often, asystole may be the result of increased parasympathetic activity.

It is also indicated in cases of PEA with a pulse rate of < 60 beats per minute on the ECG monitor.

Verapamil

Verapamil, a calcium channel blocker, can be used to treat bouts of supraventricular tachycardia. An intravenous bolus of 5 mg is administered, followed by 10 mg over the following 15 - 30 minutes, should the tachycardia persist and no adverse effects are noted after the first dose. Severe bradycardia, hypotension and congestive heart failure can occur after treatment with verapamil. The hypotension can sometimes be reversed with $CaCl_2$ 0,5 – 1 mg.

Morphine sulphate

Morphine is not an antidysrhythmic agent and is only mentioned here because of its essential role in the management of patients with myocardial infarction which may result in cardiac arrest.

Morphine is the agent of choice in the treatment of acute myocardial infarction and pulmonary oedema. It increases the venous capacitance, decreasing venous return and inducing mild arterial vasodilatation. These effects relieve pulmonary congestion and reduce left ventricular wall stress, thereby decreasing myocardial oxygen requirements.

By reducing the sensitivity of the J-receptors, the stretch reflex of the lungs is depressed and the patient is able to breathe with greater ease.

2 - 5 mg must be administered intravenously, as needed. The patient must be monitored for respiratory depression and hypotension. If necessary, a diuretic such as furosemide can be administered in the treatment of pulmonary oedema.

Drugs used to improve cardiac output and blood pressure

Adrenaline

This drug has been discussed already.

Once a beating heart has been established, a constant infusion of adrenaline (8 - 10mg in 200 ml 5% dextrose water) is administered in the adult at a rate of 1 - 4 µg/min, or is titrated according to the effect desired.

Dopamine hydrochloride

This is the chemical precursor of noradrenaline and has dopaminergic and ß- and α-adrenergic effects. Dopaminergic dosages range from 0,5 – 2,5 µg/kg/min, and the ß-effects fall within 2,5– 5,0 µg/kg/min, possibly as high as 7,5 µg/kg/min.

Doses of more than 7,5 µg/kg/min are in the α-adrenergic range, resulting in peripheral vaso-constriction.

Dobutamine hydrochloride

Dobutamine has predominant ß-effects, which improve cardiac contractility and increase heart rate. It can cause initial peripheral vasodilatation, with subsequent hypotension and decrease in peripheral vascular resistance. The usual dose range is 2,5 - 10 µg/kg/min. An increase of more than 10% in the heart rate should be avoided, as this may induce myocardial ischaemia.

Dobutamine should not be administered for the management of acute cardiac arrest because of its possible deleterious effects.

Amrinone

Amrinone is a non-adrenergic, positive inotropic agent, of which the mechanism of action is unknown.

An initial dose of 0,75 mg/kg is administered initially over 2 - 3 minutes, followed by an infusion at a rate of 5 - 10 µg/kg/min.

There is seldom an indication for use of amrinone during the acute episode, but it can be commenced thereafter. Recently, research has shown very good results with amrinone in the acute phase of a cardiac arrest, but more studies are needed.

Calcium

Although it plays a major role in myocardial contractility, several studies have failed to show any benefit from the use of calcium in the cardiac arrest setting. It may be deleterious for brain resuscitation and should only be used in the following situations:
• hyperkalaemia
• hypocalcaemia
• calcium channel blocker toxicity

During cardiac arrest, the myocardium and brain become flooded with circulating Ca^{2+} ions. During the stage of anoxia/ischaemia, ATP stores become depleted, causing a disruption in Ca^{2+} homeostasis. There is an uncontrolled influx of Ca^{2+} from the extravascular compartment into the intracellular space. Due to the malfunction of the ATP-dependent ion pumps, Ca^{2+} becomes sequestrated within the intracellular compartment. Ca^{2+} is also being effluxed from its intracellular stores, i.e. the endoplasmic reticulum. This Ca^{2+} overload causes mitochondrial damage, platelet aggregation, vaso-spasm, membrane lipid peroxidase release and the accumulation of arachidonic acid metabolites and oxygen free radicals. Increased Ca^{2+} concentrations in the cerebral circulation also cause increased cerebral vasoconstriction. Brain oedema is often a complication of the abovementioned processes, and usually materialises within the 24 - 48 hours after a successful resuscitation.

Dose: $CaCl_2$ in doses of 2 ml 10% solution (2 – 4 mg/kg). If necessary, this dose may be repeated.

Diuretics

With pulmonary oedema, furosemide 0,5 mg/kg is administered slowly intravenously.

Potassium chloride

Potassium chloride is administered when the potassium level is below 4 mmol/l, or as a last resort after numerous unsuccessful defibrillations. It might contribute to the successful reversal of asystole to VF, or finer fibrillation waves to coarse fibrillation. 5 - 10 ml of a 15% solution is normally sufficient, and is given by continuous slow infusion.

Magnesium sulphate

This drug has effectively been used as an anti-dysrhythmic, and is administered as a dosage of 2 g, slowly intravenously.

Sodium bicarbonate (NaHCO$_3$)

The initial acidosis of cardiac arrest is a respiratory acidosis. Adequate alveolar ventilation is the most important aspect in the initial maintenance of the acid-base balance during cardiac arrest. Metabolic acidosis is inevitable during prolonged

cardiopulmonary arrest. Alveolar hypoventilation is the most important contributing factor. This causes increased levels of CO_2 which diffuse easily over cellular membranes, resulting in intracellular acidosis. The respiratory acidosis is the most important causative factor of myocardial dysfunction during the later phases of cardiac arrest. Hyperventilation tends to restore the initial respiratory acidosis by extracting the CO_2 which diffused freely over membranes. This also leads to delayed metabolic acidosis.

There is little research that proves that buffers improve outcomes in CPR. On the contrary, there are data proving that $NaHCO_3$:
• Does not improve the ability to defibrillate in experimental animals.
• Shifts the oxyhaemoglobin dissociation curve to the left, thereby inhibiting the release of oxygen to the tissues.
• Induces hypernatraemia, hyperosmolality and metabolic alkalosis, and the resultant side effects.
• Produces paradoxical acidosis due to production of carbon dioxide. This situation can only be improved by hyperventilation, which is probably already taking place. Hypercarbia may depress myocardial function and increase cerebral blood volume.
• Worsens central venous acidosis and its effects. If bicarbonate is administered without effective ventilation, an increase in CO_2 is caused with worsening of the acidosis. The aim of bicarbonate administration is only to clear the circulating H^+ ions during acidosis.
• May inactivate simultaneously administered cate-cholamines

Bicarbonate should only be administered after interventions such as CPR, ventilation with an endotracheal tube, defibrillation and pharmacological therapy have been unsuccessfully utilised for 12 - 15 minutes. It is used at the discretion of the treating physician.

Empirical dose: 1 mmol/kg initially, and no more than half this dose every 10 minutes thereafter.

If laboratory tests reveal metabolic acidosis or hyperkalaemia before the cardiac arrest, bicarbonate may be used earlier.

In the postresuscitation phase, the administration of bicarbonate should be guided by arterial blood gases. The formula is as follows:

weight (kg) x base excess x 0,3 = meq $NaHCO_3$

(Always rectify base excess to a value of –5)

Administer only half of the amount during the first 10 - 15 minutes and repeat the blood gases before giving the rest. In cases where volume overload is a problem, use the 8,4% solution.

Cerebral oedema

The two most important complications after cardiac arrest, irrespective of the damage that it causes to the heart, are kidney failure and brain oedema.

Cerebral oedema usually develops within the first 24 - 48 hours after a successful resuscitation, and can cause problems if the physician is not aware of this possibility. It usually manifests as a patient who is moderately confused and who becomes more and more so, gradually evolving into complete unconsciousness.

Treatment

The patient must be sedated and, if necessary, intubated and mechanically ventilated after been given a muscle relaxant. Ventilate in order to maintain an adequate CO_2 level (between 28 – 30 mmHg is acceptable). Hypercapnoea causes cerebral vasodilatation and enhances the deleterious effects of the brain oedema. The CO_2 must not be "blown off" too aggressively, as this would compromise cerebral blood flow due to severe vasoconstriction, thereby worsening the ischaemic damage to the brain.

The patient must be nursed in the "head up" position (± 15°) to decrease cerebral venous congestion and worsening of the oedema.

Diuretics

Mannitol is considered the best option due to its osmotic effect. It withdraws fluid from the extravascular space towards the intravascular compartment, and is excreted in its unchanged form by the kidneys. In cases of true renal failure, mannitol should be administered with great caution. Furosemide could also be given to reduce the brain oedema.

If available, the Ca^{2+} channel blocker nimodipine could also be administered. During cardiac arrest, there is an abundant efflux of Ca^{2+} ions into the circulation that causes further vasospasm and therefore enhances the cerebral oedema. This can be prevented by nimodipine. This is also the reason why calcium, in essence an excellent positive inotropic drug, is not administered during cardiac arrest.

Steroid administration still remains controversial but may be effective.

Conclusion

Successful resuscitation is determined by:

1. Basic life support
2. Early cardiac compressions
3. Immediate defibrillation
4. Endotracheal intubation and ventilation
5. α-adrenergic agonist support

α-agonism improves the two most important haemodynamic variables that determine survival:

1. Aortic diastolic pressure
2. Associated myocardial perfusion gradient

All other drugs are of no worth. Myocardial and cerebral protection is compromised if defibrillation is delayed and CPR is not commenced immediately.

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