ABC of Resuscitation

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POST RESUSCITATION CARE

Successful resuscitation

Arrest was witnessed

Underlying arrhythmia was
ventricular fibrillation

Successful cardioversion
achieved in 2-3 minutes and not longer
than 8 minutes

When defibrillation was delayed for longer
than 2-3 minutes cardiopulmonary
resuscitation was started and maintained

- Restore spontaneous cardiac output
- Control pH, blood gases, and electrolytes
- Electively ventilate

Full recovery from cardiac arrest is rarely immediate. The restoration of electrocardiographic complexes marks the start and not the end of a successful resuscitation. The true end point is a fully conscious, neurologically intact patient with a spontaneous stable cardiac rhythm and an adequate urine output.

The chances of achieving this are greatly enhanced if the conditions in the box are met.

Once spontaneous cardiac output has been restored ensure that a senior doctor is summoned and makes a decision about transferring the patient to an intensive care area. Elective ventilation is often necessary and such decisions require experience and authority. Clearly if the time from the onset of cardiac arrest to return to full consciousness is about two to three minutes elective ventilation may prove unnecessary. Unfortunately this is rare and is likely to occur only when the patient is already under intensive care. On a hospital ward, although the arrest is often witnessed, it may be many minutes before definitive treatment can be started. These patients will invariably need transfer to an intensive care unit for further assessment, monitoring, and treatment.

Accident and emergency departments receive patients who have had unwitnessed arrests. In these patients special attention must be given to cerebral resuscitation. All doctors who resuscitate patients must be aware of the consequences when resuscitation is incomplete and brain damage occurs. Nevertheless, more brains are damaged by inadequate than by inappropriate resuscitation. Experienced senior doctors must be involved early in the management of cardiac arrest, ideally from the start.

When the heart stops the brain may be damaged both by the initial ischaemia and by failure of adequate reperfusion. The latter is ultimately the most damaging. The brain may survive a period of hypoxia if cerebral flow is maintained. When cerebral blood flow ceases the electroencephalogram is flat within 10 seconds and cerebral glucose used up within one minute. Neuronal activity may, however, continue for up to an hour. Unfortunately progressive deterioration of the brain cannot be reversed or halted after about three minutes. A carotid pulse may be restored but the myriad of tiny cerebral vessels cannot be reperfused. Factors implicated are thought to include the following.

Vasospasm—Movement of calcium ions is implicated in vasospasm, and calcium antagonists have improved cerebral survival in animals. The administration of calcium during cardiac arrest must therefore be questioned and can be endorsed only for electromechanical dissociation.

Cerebral oedema—Hypoxia leads to cerebral oedema and this is augmented by the high blood levels of carbon dioxide associated with apnoea. Administration of sodium bicarbonate may release yet more carbon dioxide and compound the problem. Hyperventilation and elective lowering of the PCO_2 may reduce cerebral oedema and obviate the need for bicarbonate.

Microthrombus formation and sludging—When cerebral flow ceases the smaller vessels silt up. Once formed these blockages cannot be removed.

Cerebral damage after cardiac arrest can be minimised by the following manoeuvres.

Immediately after restoration of cardiac rhythm complete this checklist

- (1) With a laryngoscope ensure that the endotracheal tube is correctly placed in the trachea and not in the oesophagus.
- (2) Ensure that the patient is being adequately ventilated with 100% oxygen. Listen with a stethoscope and confirm adequate and equal air entry. If you suspect *pneumothorax* insert a chest drain.
- (3) Estimate arterial pH and gases, preferably from an arterial sample, but use a central venous sample if necessary.
- (4) Estimate serum potassium.
- (5) Obtain a chest radiograph. An anteroposterior supine view is adequate.
- (6) Measure the arterial blood pressure.
- (7) Insert a urinary catheter and measure the urinary output.
- (8) Insert a nasogastric tube and aspirate the contents of the stomach.
- (9) Measure the central venous pressure.
- (10) Obtain a 12 lead electrocardiograph.

Rapid return of spontaneous cardiac output—Cardiopulmonary resuscitation sometimes achieves no more than 5% of normal cerebral flow and minimal cerebral oxygenation. It does, however, prevent total cessation of cerebral blood flow.

Meticulous control of pH, blood gases, and electrolytes in the period immediately after the arrest. In most cases this can be achieved only by elective ventilation of a sedated paralysed patient in an intensive care unit.

It is also worth considering barbiturate coma, hypothermia, steroid treatment, and intracranial pressure monitoring.

Barbiturate coma may prevent the damaging effect of epileptic fits and protects against incomplete ischaemia, possibly by reducing cerebral activity. Barbiturates are mild calcium channel blockers and some are free radical scavengers. Their effectiveness is not proved or widely accepted.

Hypothermia reduces the activity of the brain but may itself induce cardiac arrest. It is technically difficult to achieve and is usually used when the patient is already hypothermic—for example, after near-drowning.

Steroids—There is some evidence that steroids may modify the action of free radicals and so protect an ischaemic brain. They are valuable in controlling cerebral oedema associated with slow growing tumours but may also have a place after an arrest.

Intracranial pressure monitoring may detect changes in intracranial pressure and help evaluate the efficacy of treatment.

Although spontaneous respiratory effort may return quite soon after a heart beat, it is rarely adequate and the gag reflex is usually impaired. A cuffed endotracheal tube will protect the airway and facilitate positive pressure ventilation. 100% oxygen should be used in the early stages to ensure adequate oxygenation of poorly perfused areas. If the patient resists the endotracheal tube an immediate decision must be made about *elective ventilation*. This will allow hyperventilation to be used. A reduction of the PCO_2 to about 4 kPa (30 mm Hg) will reduce cerebral oedema, and control of ventilation will allow some correction of blood gas abnormalities and scrupulous maintenance of the PO_2 .

A self expanding bag and valve are inadequate for more than a few minutes of resuscitation. They will allow carbon dioxide to build up, which can lead only to an increase in intracranial pressure and a deterioration in the acid base status. A mechanical ventilator must be used.

External chest compression may create a flail segment. This may be the result of rib fracture or, more commonly, of costochondral or sternal dislocation. Elective ventilation is mandatory for this condition. Do not wean a patient off a ventilator unless you know the rib cage is intact.

Acid base balance

When an arrest was witnessed there is no need to administer sodium bicarbonate immediately. It need be given only when other methods have failed to reverse the acidosis. Even when the arrest was unwitnessed it is wise to administer bicarbonate only when there is a measured acidosis. External chest compression produces poor cerebral blood flow but even less peripheral circulation. There is little effective venous return, so lactic acid builds up but is not returned to the circulation until the heart is restarted. It is in the phase immediately after the arrest that serious acid base problems may occur, and continuous monitoring of the blood gases every 15 minutes, possibly for several hours, is essential. Sodium bicarbonate provides a large hyperosmolar sodium load to an extremely compromised circulation. It can produce a precipitous fall in the serum potassium concentration. It neutralises acid by the release of carbon dioxide, and a rise in the PCO_2 will

Dopamine infusion

Add 800 mg of dopamine to 500 ml of 5% dextrose and mix well. Infuse very slowly through a burette and adjust rate according to response.

Lignocaine infusion

Add 2 g of lignocaine (10 ml of 20% solution) to 500 ml of 5% dextrose. Infuse at 5-15 drops per minute.

Caution

The pupillary response is of little value immediately after an arrest. Pupils dilate during cardiac arrest for several reasons. Concentrations of circulating endogenous catecholamines are very high immediately before and during an arrest and exogenous catecholamines are likely to have been given. Atropine may also have been administered. Ischaemia to the anterior chamber can dilate the pupils. Only when hypoxia has been corrected and the effects of drugs allowed to wear off can tests of brain stem function be employed. This may take several hours.

When feeling for carotid pulses, inserting a nasogastric tube, or replacing an endotracheal tube always watch the electrocardiographic monitor. Hypoxia augments many vagally mediated reflexes and stimulation of the carotid sinus or oropharynx under these circumstances may lead to marked reflex bradycardia and asystole.

lead to increased cerebral oedema. Carbon dioxide will move into the cerebrospinal fluid, making central monitoring of the pH by the brain extremely precarious once cardiac output has been restored. Blood gas and acid base abnormalities should initially be controlled by ventilation and the restoration of renal function. If these prove inadequate then small doses of bicarbonate can be given if blood gases are constantly monitored.

Hypokalaemia may precipitate cardiac arrest, particularly in elderly patients taking digoxin and diuretics. Bicarbonate administration may further lower the serum potassium value. If the serum potassium concentration is very high, usually as the result of renal failure, then it can be lowered with glucose and insulin.

Each resuscitation attempt must be accompanied by a chest radiograph. Check the position of the central venous line and ensure that the endotracheal tube is not down a main bronchus. Pneumothorax often occurs and is an important reversible cause of electromechanical dissociation. When electrical activity is recorded by the electrocardiogram but there is no cardiac output it is important to eliminate any other easily reversible condition. Cardiac tamponade may be diagnosed by an enlarged cardiac shadow and a raised central venous pressure. If in doubt obtain an echocardiogram.

Once a rhythm has been restored it is only of value if it produces an output. If you cannot feel a pulse continue external chest compression and treat the patient for electromechanical dissociation. If the patient is hypotensive a dopamine infusion may be required.

If a short period of hypertension ensues this is not harmful and may even be beneficial to the brain.

Consider a lignocaine infusion after ventricular fibrillation.

An adequate blood pressure will produce 40-50 ml of urine every hour. A urinary catheter may be necessary to monitor urine output.

Early attempts at mouth to mouth and bag and mask ventilation will have introduced air into the stomach. An initially misplaced endotracheal tube will do the same. Gastric distention provokes vomiting and is uncomfortable. It is usually necessary to insert a nasogastric tube.

The cause of the arrest and its effects on the heart may be shown by a 12 lead electrocardiogram. Any subsequent changes can be related to this early reading.

Conclusion

If patients are to be protected from the dangers inherent in our decision to resuscitate them we must accept that a commitment to treating cardiac arrest is a commitment to intensive postresuscitation care. The patient can be managed only in an intensive care unit and will need at least a short period of elective ventilation.

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A man aged 52 has had three attacks of erysipelas affecting the lower part of his right leg since a coronary artery bypass operation. The erysipelas appears to have occurred around the scar from the removal of the long saphenous vein. Is this a recognised complication of such surgery? If the attacks continue would long term penicillin prophylaxis be advisable?

The medial side of the lower leg is a common site for inflammation. Recurrent episodes may be phlebitis or, more usually, the lipodermatosclerosis of chronic venous insufficiency. Recurrent episodes of erysipeloid cellulitis may occur in relation to sites of previous injury or operation. It is not specifically related to the removal of vein for bypass grafting. The presence of foreign material such as non-absorbable ligature may keep the focus of infection active. Patients with chronic lymphoedema are particularly prone to infection. The other predisposing condition to exclude is any site of streptococcal ingress to the foot such as chronic or recurrent skin hack, bunion, corn, or paronychia. The commonest entry route of secondary infection in patients prone to recurrent cellulitis is a skin fissure between the toes in patients with athlete's foot. Attention should therefore be paid to hygiene and antifungal powder should be used regularly. Should the

cellulitis recur, and given that none of these factors is present, it might be reasonable to give a long term course of antibiotic, though this depends on the time scale. If the attacks have been coming at frequent intervals a three month course of oral penicillin could be tried. If the attacks have been many months apart it would seem more sensible to provide the patient with a supply of antibiotic to be started at the first sign of symptoms.—C RUCKLEY, consultant vascular surgeon, Edinburgh.

Correction

The Mandwa experiment, an alternative strategy

We regret that an error occurred in this paper by Mr N H Antia (3 May, p 1181-3). The introduction stated that the infant mortality rate in India was about 300 this should have read about 130. In addition, in the discussion section, "30 of every 1000 children" should have read "130 of every 1000 children." We apologise for these errors.