



European Resuscitation Council Guidelines for Resuscitation 2005

Section 2. Adult basic life support and use of automated external defibrillators

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4c. ALS treatment algorithm

Bicarbonate (p.48).

Administering sodium bicarbonate routinely during cardiac arrest and CPR (especially in out-of-hospital cardiac arrests) or after return of spontaneous circulation is not recommended. Give sodium bicarbonate (50 mmol) if cardiac arrest is associated with hyperkalaemia or tricyclic antidepressant overdose; repeat the dose according to the clinical condition and result of repeated blood gas analysis. Some experts give bicarbonate if the arterial pH is less than 7.1, but this is controversial. During cardiac arrest, arterial blood gas values do not reflect the acid-base state of the tissues (96); the tissue pH will be lower than that in arterial blood. Mixed venous blood values give a more accurate estimate of the pH in the tissues (96), but it is rare for a pulmonary artery catheter to be in situ at the time of cardiac arrest. If a central venous catheter is in situ, central venous blood gas analysis will provide a closer estimate of tissue acid/base state than that provided by arterial blood.

4e. Assisting the circulation

Drugs and fluids for cardiac arrest

Buffers (p.62-63)

Cardiac arrest results in combined respiratory and metabolic acidosis caused by cessation of pulmonary gas exchange and the development of anaerobic cellular metabolism, respectively. The best treatment of acidaemia in cardiac arrest is chest compression; some additional benefit is gained by ventilation. If the arterial blood pH is less than 7.1 (or base excess more negative than -10 mmol l⁻¹) during or following resuscitation from cardiac arrest, consider giving small doses of sodium bicarbonate (50 ml of an 8.4% solution). During cardiac arrest, arterial gas values may be misleading and bear little relationship to the tissue acid-base state (96); analysis of central venous blood may provide a better estimation of tissue pH (see Section 4c). Bicarbonate causes generation of carbon dioxide, which diffuses rapidly into cells. This has the following effects:

- It exacerbates intracellular acidosis.
- It produces a negative inotropic effect on ischaemic myocardium.
- It presents a large, osmotically active, sodium load to an already compromised circulation and brain.

- It produces a shift to the left in the oxygen dissociation curve, further inhibiting release of oxygen to the tissues.

Mild acidaemia causes vasodilation and can increase cerebral blood flow. Therefore, full correction of the arterial blood pH may theoretically reduce cerebral blood flow at a particularly critical time. As the bicarbonate ion is excreted as carbon dioxide via the lungs, ventilation needs to be increased. For all these reasons, metabolic acidosis must be severe to justify giving sodium bicarbonate.

Several animal and clinical studies have examined the use of buffers during cardiac arrest. Clinical studies using Tribonate® (215) or sodium bicarbonate as buffers have failed to demonstrate any advantage (216-220). Only one study has found clinical benefit, suggesting that EMS systems using sodium bicarbonate earlier and more frequently had significantly higher ROSC and hospital discharge rates and better long-term neurological outcome (221). Animal studies have generally been inconclusive, but some have shown benefit in giving sodium bicarbonate to treat cardiovascular toxicity (hypotension, cardiac arrhythmias) caused by tricyclic antidepressants and other fast sodium channel blockers (Section 7b) (222). Giving sodium bicarbonate routinely during cardiac arrest and CPR (especially in out-of-hospital cardiac arrests) or after return of spontaneous circulation is not recommended.

Consider sodium bicarbonate for life-threatening hyperkalaemia or cardiac arrest associated with hyperkalaemia, severe metabolic acidosis, or tricyclic overdose. Give 50 mmol (50 ml of an 8.4% solution) of sodium bicarbonate intravenously. Repeat the dose as necessary, but use acid/base analysis (either arterial or central venous) to guide therapy. Severe tissue damage may be caused by subcutaneous extravasation of concentrated sodium bicarbonate. The solution is incompatible with calcium salts as it causes the precipitation of calcium carbonate.

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Section 6. Paediatric life support

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6b Paediatric advanced life support Fluids and drugs

Sodium bicarbonate (p.109)

Giving sodium bicarbonate routinely during cardiopulmonary arrest and CPR or after ROSC is not recommended (167,168). After effective ventilation and chest compressions have been achieved and adrenaline given, sodium bicarbonate may be considered for the child who has had a prolonged cardiopulmonary arrest and severe metabolic acidosis. Sodium bicarbonate may also be considered in the case of haemodynamic instability and co-existing hyperkalaemia, or in the management of tricyclic overdose. Excessive quantities of sodium bicarbonate may impair tissue oxygen delivery, produce hypokalaemia, hypernatraemia and hyperosmolality and inactivate catecholamines.

6c Resuscitation of babies at birth

Bicarbonate (p.119)

If effective spontaneous cardiac output is not restored despite adequate ventilation and adequate chest compressions, reversing intracardiac acidosis may improve myocardial function and achieve a spontaneous circulation. Give 1-2 mmol kg⁻¹ IV.

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