Section 8: Postresuscitation Care

Return of Spontaneous Circulation After a No-Flow Cardiac Arrest

Cardiovascular and hemodynamic derangements are common when spontaneous circulation returns (ROSC) after a cardiac arrest. These abnormalities include hypovolemic shock, cardiogenic shock, and the vasodilatory shock associated with the systemic inflammatory response syndrome (SIRS).

Multiple pathogenic factors contribute to the postresuscitation syndrome:

- Reperfusion failure
- Reperfusion injury
- Cerebral intoxication from ischemic metabolites
- Coagulopathy

After restoration of circulation, 4 phases of the postresuscitation syndrome occur, dependent on the degree and duration of organ ischemia.1

1. Almost one-half of postresuscitation syndrome deaths that occur take place within 24 hours of the event. In the hours after the ROSC, cardiovascular dysfunction is present to a variable degree, with a tendency to normalize over 12 to 24 hours. Microcirculatory dysfunction from the multifocal hypoxia leads to rapid release of toxic enzymes and free radicals into the cerebrospinal fluid and blood. Cerebral and microvascular abnormalities persist as metabolic disorders progress.

2. Over 1 to 3 days cardiac function and systemic function improve but intestinal permeability increases, predisposing to sepsis syndrome. Several organs have progressive dysfunction, particularly the liver, pancreas, and kidneys, leading to the multiple organ dysfunction syndrome (MODS).

3. Finally, days after the cardiac arrest serious infection occurs and the patient declines rapidly.

4. Death occurs.

The principal objective of the postresuscitation phase is the complete reestablishment of regional organ and tissue perfusion. Simple restoration of blood pressure alone and improvement in tissue gas exchange do not necessarily improve survival. Notably, these end points fail to indicate appropriate resuscitation of peripheral organ systems and their blood supply, particularly the splanchnic and renal circulation, which contribute importantly to MODS after hypoxic-ischemic arrest.3–5

In most cases the acidemia associated with cardiac arrest improves spontaneously when adequate ventilation and perfusion have been restored. Persistent unrecognized splanchnic hypoperfusion will be identified only with specific monitoring and requires targeted therapy.6 In addition to invasive hemodynamic monitoring with pulmonary artery catheters, which remains controversial,8–10 splanchnic resuscitation should be directed by quantitative gastric tonometric measurement of the systemic:gastric mucosal PCO₂ gradient. Targeted correction of systemic:gastric mucosal PCO₂ gradient may be an important adjunct to invasive hemodynamic monitoring in the ICU but is presently unproven and is not widely available.11,11a,12

The purpose is to maximize splanchnic perfusion in the early postresuscitation phase and avoid progression to MODS.

These 2000 ACLS guidelines incorporate our evolving understanding of the hemodynamic abnormalities encountered in patients who survive resuscitation. Recommendations are generally based on data derived from studies of posttraumatic and medical SIRS. Very few clinical randomized studies have been published dealing specifically with hemodynamic support after neurocerebral resuscitation for cardiac arrest.

The immediate goals of postresuscitation care are to

- Provide cardiorespiratory support to optimize tissue perfusion, especially to the brain
- Transport the prehospital cardiac arrest patient to the hospital Emergency Department and then to an appropriately equipped critical care unit
- Attempt to identify the precipitating causes of the arrest
- Institute measures such as antiarrhythmic therapy to prevent recurrence

Immediately after resuscitation, patients may exhibit a wide spectrum of physiological states. Patients may recover fully with normal hemodynamic and cerebral function. At the other end of the spectrum, patients remain comatose with cardiorespiratory abnormalities. All patients require careful, repeated assessments to establish the status of their cardiovascular, respiratory, and neurological systems. Clinicians should identify complications, such as rib fracture, hemothorax, pericardial tamponade, intra-abdominal trauma, and misplaced tracheal tube.

Optimal Response to Resuscitation

In the optimal situation after resuscitation, the patient is awake, responsive, and breathing spontaneously. Apply ECG monitor leads and provide supplemental oxygen. If not already done during resuscitation, start an IV infusion with normal saline. Glucose administration is reserved for patients with documented hypoglycemia. Change peripheral or central intravenous lines placed without proper sterile technique or those maintained inadequately. If the arrest rhythm was VF or VT and no antiarrhythmic treatment was given, consider use

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of a lidocaine bolus followed by maintenance infusion unless contraindicated (ie, in patients with ventricular escape rhythm) and continue the infusion for several hours while primary ventricular fibrillation secondary to an acute coronary syndrome is excluded and other correctable causes are assessed.

Clinicians should consider the precipitating cause of the cardiac arrest, particularly an AMI, electrolyte disturbances, or primary arrhythmias. If an antiarrhythmic agent was used successfully during the resuscitation, administer a continuous infusion of that agent. If hemodynamically significant bradycardia is present, initiate therapy as described in the guidelines section on bradycardia. Consider fibrinolytic therapy for patients who survive resuscitations of short duration and with minimal trauma with evidence of acute ST-segment elevation MI on their postresuscitation 12-lead ECG and who have no contraindications to fibrinolytic therapy. Patients with contraindications to fibrinolytic therapy should be considered for urgent coronary angiography and appropriate intervention. Consider the patient’s neurological status, but coma should not preclude indicated interventions. Acute coronary syndromes must be evaluated with serial ECG and cardiac markers in all patients. Assess the hemodynamic status, vital signs, and urine output.

Perform laboratory investigations, including a 12-lead ECG; portable chest x-ray; determination of arterial blood gases, electrolyte, glucose, serum creatinine, blood urea nitrogen, magnesium, and calcium levels; and other appropriate chemical analyses. Treat aberrations in potassium, magnesium, calcium, and sodium levels aggressively. In candidates for fibrinolytic therapy, perform arterial punctures only if less invasive assessments of oxygenation (pulse oximetry), ventilation (expired CO₂), and acid-base status (venous sample) are unavailable and clinically relevant information is unavailable by other noninvasive methods. Prearrest status must be reviewed carefully, particularly if the patient was receiving drug therapy. After completion of these steps, transfer the patient with oxygen and ECG monitoring to a special care unit for observation, continuous monitoring, and further therapeutic intervention. Resuscitation equipment and an adequate number of trained personnel must accompany the patient in transport.

**Temperature Regulation**

Regional cerebral metabolic rate determines the regional blood flow requirements of the brain. The cerebral metabolic rate increases approximately 8% per degree Celsius (2 degrees Fahrenheit) of body temperature elevation. After resuscitation, temperature elevation above normal can create a significant imbalance between oxygen supply and demand and impair brain recovery. Treat fever aggressively in the postischemic period.

**Hypothermia**

Hypothermia, in contrast, is an effective method to suppress cerebral metabolic activity. Although previously used widely during cardiovascular surgery, hypothermia has significant detrimental effects that might adversely affect the post–cardiac arrest patient, including increased blood viscosity, decreased cardiac output, and increased susceptibility to infection. Many reports indicate benefit after brain ischemia, although some document detrimental effects or lack of improvement. Recent evidence indicates that mild levels of hypothermia (eg, 34°C [93°F]) are effective in mitigating posts ischemic brain damage without detrimental side effects. In the normal brain, a 7% reduction in the cerebral metabolic rate occurs with every 1°C (2°F) reduction in brain temperature.

After cardiac arrest, hypermetabolism may cause fever and disrupt the balance between cerebral oxygen supply and demand. This suggests a possible clinical role for induced mild hypothermia.

Interest in hypothermia as a treatment modality for brain injury was rekindled in the late 1980s and early 1990s when experiments performed in carefully controlled rodent models of brain ischemia (by cerebral vascular occlusion techniques) and dog experiments of cardiac arrest showed that even mild intras ischemic hypothermia could be neuroprotective. The ability to improve neurological outcomes bycooling brain-injured humans quickly and safely was demonstrated by Marion et al in a randomized, controlled trial comparing the effects of moderate hypothermia (32°C to 33°C [89.5°F to 91.5°F] for 24 hours) with normothermia in 82 patients with severe closed-head injuries. As of early 2000 there was an active, randomized European multicenter trial of resuscitative hypothermia after cardiac arrest. The investigators anticipate an enrollment of 500 patients.

Side effects of hypothermia include coagulopathy, cardiac dysrhythmias, impaired cardiac function, and increased susceptibility to infection. The prevalence and severity of these side effects is proportional to the depth and duration of hypothermia. Investigations inducing mild to moderate hypothermia in humans after cardiac arrest (minimum temperature ≥32°C [89.5°F]) for 24 to 36 hours have reported hypothermia-related side effects.

In summary, hemodynamically stable patients who develop a mild degree of hypothermia (>33°C [91.5°F]) spontaneously after cardiac arrest should not be actively warmed. Mild hypothermia may be beneficial to neurological outcome and is likely to be well tolerated (Class IIb). However, hypothermia should not be induced actively after resuscitation from cardiac arrest (Class Indeterminate).

**Hyperthermia**

There are many studies in animal models of brain injury that show exacerbation of injury if body/brain temperature is increased during (intras ischemic) or after cardiac arrest. Moreover, several studies have documented worse neurological outcome in humans who have fever after ischemic brain injury. Closely monitor temperature after resuscitation from cardiac arrest, and treat fever aggressively (Class IIa).

**Single- or Multiple-Organ System Failure: Requires Total or Near-Total Support**

After ROSC, patients may remain comatose for a variable period of time. Spontaneous breathing may be absent, and a period of mechanical ventilation via tracheal tube may be required. The hemodynamic status may be unstable with...
abnormalities of cardiac rate, rhythm, systemic blood pressure, and organ perfusion. Hypoxemia and hypotension exacerbate brain injury and must be avoided. The patient may be in a coma or show decreased responsiveness. The baseline postarrest status of each organ system must be defined and monitored and appropriate interventions instituted. With adequate ventilation and reperfusion, the acediaemia of arrest will normally improve spontaneously in most cases without the need for buffer administration.

During transportation of the patient to a critical care unit, mechanical ventilation and oxygenation must be maintained along with ECG monitoring. Assessment of circulatory status in transport with physical palpation of carotid or femoral pulses, continuous intra-arterial pressure monitoring, or pulse oximetry will allow for immediate initiation of CPR should another arrest occur. Equipment and personnel to accomplish immediate defibrillation and drug therapy must accompany the patient in transport.

**Respiratory System**

After ROSC, patients may exhibit various degrees of respiratory dysfunction. Some patients will remain dependent on mechanical ventilation and will need supplementary oxygen. Perform a complete clinical examination and review the chest x-ray. Pay special attention to potential complications of resuscitation, such as pneumothorax and misplacement of the tracheal tube. The level of mechanical ventilatory support is determined by the blood gas values, respiratory rate, and perceived work of breathing. As spontaneous ventilation becomes more efficient, the level of respiratory support can be decreased until respiration is entirely spontaneous (decreasing intermittent mandatory ventilation rates). If high oxygen concentrations are needed, it is important to establish whether the cause is pulmonary or cardiac dysfunction. Positive end-expiratory pressure (PEEP) may be helpful in the patient with pulmonary dysfunction complicated by left ventricular failure if the patient is hemodynamically stable. If cardiac dysfunction is present, support of the failing myocardium is important. Adjust inspired oxygen concentration, PEEP, and minute ventilation based on sequential arterial blood gas analyses and/or noninvasive monitoring, such as pulse oximetry and capnography. To facilitate repeated arterial blood sampling, an arterial cannula may be necessary. The systemic blood pressure can also be accurately and continuously monitored from this arterial line.

**Ventilatory Parameters**

Recent evidence supports the theory that sustained hypocapnea (low Pco₂) may worsen cerebral ischemia. After cardiac arrest, restoration of blood flow results in an initial hyperemic blood flow response lasting 10 to 30 minutes, which is followed by a more prolonged period of low blood flow. During this period of delayed hyperperfusion a mismatch between blood flow (oxygen delivery) and oxygen metabolism may occur. If the patient is hyperventilated at this stage, the additional cerebral vasoconstriction resulting from a low Pco₂ may further decrease cerebral blood flow and worsen cerebral ischemia. There is no evidence that hyperventilation protects vital organs from further ischemic dam-

age after cardiac arrest. The potential risk for further brain ischemia is real, and hyperventilation after cardiac arrest should be avoided. Safar et al also showed indirectly that hyperventilation results in worse neurological outcome. After cardiac arrest, dogs treated with mild hypothermia enhanced by hypertension and ventilated to normocarbia had improved outcome with this clinical management.

Hyperventilation may generate airway pressures and auto-PEEP, leading to an increase in cerebral venous and intracranial pressures. The increase in cerebral vascular pressure results in a decreased cerebral blood flow and a further worsening of brain ischemia. This mechanism is independent of the effects of Pco₂ or pH on cerebral vessel reactivity.

In summary, after either cardiac arrest or head trauma, ventilate the comatose patient to achieve normocarbia (Class IIa). Routine hyperventilation may be detrimental and should be avoided (Class III). In specific situations hyperventilation to achieve hypocarbia may be beneficial. Treat cerebral herniation syndrome with hyperventilation (Class IIa). Hyperventilation may also have a role when pulmonary hypertension is the cause of arrest (Class IIa). With restoration of cardiac output, metabolic acidosis usually corrects over time, and hyperventilation should not be used as a primary treatment modality. The use of buffer therapy is also not indicated and should be used for specific indications only (see above).

**Cardiovascular System**

Evaluation must include a complete vascular examination and review of serial vital signs and urine output. Compare a 12-lead ECG with previous tracings if available. Assess the chest x-ray: serum electrolyte levels, including calcium and magnesium; and cardiac marker levels. Review current and previous drug therapy. Serum cardiac marker levels may be elevated because of resuscitative efforts alone as global ischemia occurs during arrested or low-flow states. If the patient’s condition is hemodynamically unstable, assess both circulating fluid volume and ventricular function. Avoid even mild hypotension because it can impair recovery of cerebral function. Noninvasive assessment of blood pressure may be inaccurate in patients with low cardiac output and peripheral vasoconstriction. Intra-arterial assessment of blood pressure is usually more accurate in these patients and allows better titration of potentially dangerous catecholamine infusions. In the presence of severe vasoconstriction, blood pressure measurement from the radial artery may be inaccurate, and a femoral artery catheter may be considered.

In the critically ill patient, invasive hemodynamic monitoring is often undertaken with a pulmonary artery catheter. The use of these devices is controversial. Obtain pressure measurements of the pulmonary circulation using a pulmonary artery flow–directed catheter. These catheters also permit cardiac output measurements using the thermodilution technique. If both cardiac output and pulmonary artery occlusive pressures are low, fluid challenge with reassessment of pressures and cardiac output is indicated. In the patient with an AMI, ventricular compliance may be reduced and filling pressures elevated. The precise level of pulmonary occlusive pressure needed to achieve optimal cardiac output...
will vary, but it is often 18 mm Hg, which is higher than normal and may vary depending on patient and pathological conditions. If hypotension or hypoperfusion persists after filling pressure is optimized, inotropic (dobutamine), vaso-pressor (dopamine or norepinephrine), or vasodilator (nitroprusside or nitroglycerin) therapy may be indicated. The use of these agents is outlined in the algorithm for acute pulmonary edema, hypotension, and shock (Part 7, Section 1) and is discussed in the related text.

Renal System
The bladder must be catheterized so that urine output can be measured hourly and an accurate volume can be estimated (output includes suctioned gastric secretions, diarrheal fluid, and vomitus as well as urine). In the oliguric patient, measurement of pulmonary artery occlusive pressures and cardiac output along with evaluation of the urine sediment, electrolyte values, and measurement of the fractional excretion of filtered sodium may be helpful in differentiating prerenal from renal failure. Furosemide may maintain urine output despite developing renal failure. Dopamine at low doses (1 to 3 μg/kg per minute) does not improve splanchic blood flow or provide specific renal protection and is no longer indicated in acute oliguric renal failure.31–36 Nephrotoxic drugs and drugs eliminated via the kidneys should be used with caution and monitored appropriately, and doses should be adjusted. Progressive renal failure is indicated by a steadily rising serum urea nitrogen and creatinine, usually permissible. Progressive and irreversible renal damage results in reduced renal perfusion pressure. The cerebral perfusion pressure is equal to mean arterial pressure minus intracranial pressure and cerebral blood flow becomes dependent on cerebral autoregulation of cerebral blood flow and glycogen stores of the brain are depleted, and after 4 to 5 minutes ATP is exhausted. Autoregulation of cerebral blood flow is lost after extended hypoxemia or hypercarbia, or both, and cerebral blood flow becomes dependent on cerebral perfusion pressure. The cerebral perfusion pressure is equal to mean arterial pressure minus intracranial pressure (CPP=MAP−ICP). Following ROSC, after a brief initial period of hyperemia, cerebral blood flow is reduced (the “no-reflow phenomenon”) as a result of microvascular dysfunction. This reduction occurs even when cerebral perfusion pressure is normal. Any elevation of intracranial pressure or reduction in systemic mean arterial pressure may reduce cerebral perfusion pressure and further compromise cerebral blood flow.

Therapy for the unresponsive patient should include measures to optimize cerebral perfusion pressure by maintaining a normal or slightly elevated mean arterial pressure and reducing intracranial pressure if it is increased. Because hyperthermia and seizures increase the oxygen requirements of the brain, normothermia should be maintained and seizure activity controlled with phenobarbital, phenytoin, or diazepam or barbiturate. The head should be elevated to approximately 30° and maintained in a midline position to increase cerebral venous drainage. Care should be observed during tracheal suctioning because of the increase in intracranial pressure during this procedure. Preoxygenation with 100% oxygen helps prevent hypoxemia during suctioning. Although there is exciting experimental data on preserving central nervous system function, no treatment is sufficiently established at present to warrant its routine use after resuscitation. Nonetheless, vigilant attention to the details of oxygenation and perfusion of the brain after resuscitation can significantly reduce the possibility of secondary neurological injury and maximize the chances of full neurological recovery.

Gastrointestinal System
A nasogastric tube should be inserted if bowel sounds are absent and in those patients with a reduced level of consciousness who are mechanically ventilated. Start enteric feeding as soon as possible. If enteric feeding is not tolerated, administer histamine H2-receptor blockers or sucralfate to reduce the risk of stress ulceration and gastrointestinal bleeding.

SIRS and Septic Shock
SIRS is a complex process that may be triggered by a variety of initial insults, such as trauma, burn, or infection.37–39 The inflammatory response results in tissue damage and initiates a self-perpetuating process that results in local tissue damage and MODS. Signs of a systemic inflammatory response (fever and leukocytosis) may also occur after prolonged CPR. When infection is the cause, the resulting clinical syndrome is by definition sepsis.40 Patients with septic shock have MODS usually combined with vasodilatory shock, resulting in a relative and absolute hypovolemia.

The goal of hemodynamic management is normal tissue oxygen uptake. Initial management consists of volume replacement. Following volume replacement, an inotrope or vasopressin is usually required.41–43 Dobutamine and norepinephrine may be useful in severe septic shock.44–47 Improved outcome, however, has not been shown with the use of volume expansion and inotropic support. When sepsis is suspected, empirical antibiotic therapy is indicated and should be directed at common and usual organisms.

The use of glucocorticoid therapy in septic shock has been the subject of unresolved debates in critical care for almost half a century. The predominant controversies focus on the normal adrenal responses to sepsis, “normal” cortisol levels in the stressed state, exacerbation of active infectious processes, and significant metabolic derangements.

Relative hypoadrenalism occurs in septic shock even in the presence of normal and high cortisol levels. Methylprednisolone was found to have no mortality benefit and, in fact, in patients taking methylprednisolone there was a slight increase in mortality. No significant differences were found in the prevention of shock, the reversal of shock, or overall mortality. Patients treated with methylprednisolone experienced more deaths from secondary infection than the control group.48 Studies using lower, “supraphysiological” corticosteroid doses have been published.49–51 These studies have concluded
that methylprednisolone shortens the pressor-dependent phase of shock and reduces the amount of organ system dysfunction. At present there is no evidence that corticosteroids improve survival rates. Supraphysiological doses of corticosteroids may be beneficial for patients with persistent vasopressor-resistant shock maximally treated with broad-spectrum or organism-specific antimicrobial therapy (Class IIb).

In summary, care of the patient after resuscitation from cardiac arrest involves a careful assessment of the many organs subjected to an anoxic-hypoxic insult. Information in the patient with the postresuscitation syndrome is continuing to evolve as pathophysiological mechanisms of the SIRS and MODS are elucidated. The splanchnic circulation and gut are focal for patients with persistent vasopressor-resistant shock dysfunction. At present there is no evidence that corticosteroids may be beneficial for patients with persistent vasopressor-resistant shock.

References


Part 6: Advanced Cardiovascular Life Support: Section 8: Postresuscitation Care

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