Managing the Post-resuscitation Patient in the Field

David E. Persse, MD, Brian S. Zachariah, MD, MBA, Jane G. Wigginton, MD

Abstract

The principal goal after successful resuscitation of a cardiac arrest patient is to maintain the patient's pulse and avoid a pulseless state. Of equal importance in the post-resuscitation patient are efforts to prevent myocardial dysfunction and increase the likelihood of a good neurologic outcome. To optimize cardiac and hemodynamic resuscitation, paramedics should obtain good background information, which could provide clues to factors contributing to the cardiac arrest, such as the use of certain drugs or being overdue for dialysis, and could aid in customizing therapy for rhythm disturbances and hemodynamic aberrations. Treatment of rhythm disturbances depends on the type of arrhythmia identified, the history of present illness, and the resuscitation efforts provided. Common post-resuscitation dysrhythmias are wide-complex tachycardia, narrow-complex tachycardia, and bradycardia. Optimizing neurologic resuscitation is difficult, but evidence suggests that hypertensive reperfusion, hemodilution, and mild hypothermia may be of benefit in improving neurologic outcome after resuscitation. Unfortunately, to date, no proven therapies are available to improve neurologic outcome after resuscitation from cardiac arrest. **Key words:** cardiac arrest; post-resuscitation syndrome; neurologic outcome; resuscitation.

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For the emergency medical services (EMS) provider, the immediate goal of post-resuscitation management is to maintain the patient's pulse, prevent recurrence of a pulseless state, and optimize the patient's hemodynamic condition. Once this has been achieved, efforts should be made to achieve the ultimate goal of post-resuscitation management—enhancing the likelihood that the patient will have a good neurologic outcome.

Historically, the focus of EMS and paramedic resuscitation training has been on reestablishing spontaneous blood flow. The art of stabilizing the critically ill survivor of out-of-hospital cardiac arrest has been given little emphasis, and EMS personnel have received almost no preparation on efforts to ameliorate neurologic damage and maximize a patient's chance of returning to a productive and enjoyable life. This oversight is due in part to the considerable efforts made in training emergency medical technicians and paramedics on how to reestablish pulses and in part to the relative lack of knowledge regarding methods that can be used in the early post-resuscitation period that might increase the likelihood of a good neurologic outcome.

The hemodynamic stability of a patient just resuscitated from cardiac arrest is difficult to predict and manage and depends on many factors, including rhythm disturbances, oxygenation, ventilation, acid–base balance, pulmonary capacity, diffusion capabilities, cardiac output, myocardial damage, vascular conditioning, and blood viscosity. For paramedics trying to manage multiple physiologic parameters in a relatively uncontrolled setting, the post-resuscitation patient is one of their greatest challenges. To facilitate success in this endeavor, paramedics should be provided guidelines for managing the post-resuscitation patient that are easy to understand and remember.

To produce useful guidelines for post-resuscitation care, we must identify the issues, review the current understanding of the pathophysiology unique to the resuscitated patient, discuss the current state of the art regarding treatment of resuscitated patients, and translate this information into a product that the average paramedic can incorporate into care at the scene. Among the areas that require specific attention in developing post-resuscitation care guidelines are post-resuscitation syndrome, management of post-resuscitation cardiac dysfunction, and management of post-resuscitation neurologic dysfunction.

Post-resuscitation Syndrome

Negovsky[1] first proposed the concept of a specific post-resuscitation disease in 1972. "Negovsky's disease," as it was known in early work, was subsequently reclassified into a post-resuscitation syndrome consisting of four features: 1) reperfusion failure; 2) reoxygenation injury; 3) cerebral intoxication from derangement of extracerebral organs; and 4) changes in blood cell activity and coagulation

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Address correspondence and reprint requests to: David E. Persse, MD, Physician Director of EMS, City of Houston, 601 Sawyer Street, Suite 500, Houston, TX 77007. e-mail: <david.persse@cityofhouston.net>.

abnormalities.[2] Briefly, the overall post-resuscitation syndrome is believed to progress through several stages, as outlined in Table 1. The severity of the damage resulting from the ischemic insult that initiates the post-resuscitation syndrome primarily depends on the duration of anoxia ("no flow") and resuscitative efforts.

Characteristics of Post-resuscitation Myocardial Dysfunction

Transient coronary artery occlusion results in temporary impairment of myocardial capabilities. This impairment will persist longer and be more severe depending on the duration of the ischemic insult.[3–7] The reversible ventricular dysfunction phenomenon described by Braunwald and Kloner[8] has been labeled the "stunned myocardium." Although the stunned myocardium dysfunction and recovery phenomenon was first described in terms of regional ischemia, it has also been observed in the setting of cardiac arrest and resuscitation.[9–11]

The exact mechanisms believed to be responsible for myocardial dysfunction after global ischemia remain unclear. However, the following theories are the two most generally accepted:

Depletion of high-energy phosphates theory. This theory is based on the observation that myocardial mitochondria depend heavily on oxygen and aerobic metabolism to produce adenosine triphosphate (ATP). In dogs, mitochondrial recovery from global ischemia can take as long as 72 hours despite adequate reperfusion.[12–15]

Reperfusion injury theory. This theory suggests that there is little damage to the myocyte during the no-flow period and that the damage actually occurs when circulation is restored. Aerobic oxidative phosphorylation is reduced and hypoxanthine accumulates as a consequence of degradation of ATP. After blood flow and therefore oxygen delivery are restored, hypoxanthine is oxidized with the aid of xanthine oxidase, which results in generation of oxygen-derived free radicals, specifically superoxide anions (O_2–), hydroxy radicals (–OH), and H_2O_2.[16] These active metabolites, collectively referred to as oxygen-free radicals, are believed to be responsible for most reperfusion injury. Oxygen-free radicals have been shown to damage the sarcolemma and mitochondria,[16,17] induce calcium overload in the cytoplasmic space,[18,19] and produce cellular swelling, lysosomal rupture, and myofibrillary contraction.[20,21]

Table 1. Stages of Post-resuscitation Syndrome

Stage I (insult to 6 to 9 hours) Rapid changes in cerebral and systemic hemodynamics, clotting disturbances, increased blood viscosity, multifocal tissue hypoxia.
Stage II (10 to 24 hours) Normalization of cardiovascular function, persistent cerebral dysfunction, severely impaired microcirculation. Fifty percent of deaths occur during this period as a result of recurrent cardiac arrest, bleeding, and brain and lung edema.
Stage III (1 to 3 days) Normalization of systemic indices, improvement of cerebral function, increased intestinal permeability leading to bacteremia and development of multiple organ dysfunction.
Stage IV (beyond 3 days) Localized or generalized infection.

Characteristics of Post-resuscitation Neurologic Injury

The sudden no-flow state created by cardiac arrest followed by the low-flow state and incomplete ischemia of cardiopulmonary resuscitation initiates the cerebral ischemic insult that often results in death or permanent disability. Sudden and complete loss of cerebral perfusion followed by normotensive reperfusion will produce the cerebral post-resuscitation syndrome if the no-flow state is of sufficient duration. This syndrome involves many complex interactions of pathophysiologic processes, including depletion of ATP; respiratory (anaerobic) acidosis; failure of ATP-dependent ion gradient maintenance efforts, resulting in depolarization; calcium influx into the cell, resulting in activation of calcium-dependent enzymes, such as phospholipases and proteases, that degrade cell membranes and certain proteins; and glutamate release, resulting in excitotoxicity of neurons.[22,23]

After reperfusion, brain ATP, ion pumps, and tissue pH quickly return to normal. However, perfusion failure, reoxygenation injury with chemical cascades leading to lipid peroxidation and cell necrosis, extracerebral organ derangements, and blood derangements due to stasis will lead to the structural incompetence of the neuron and cellular death. Because these processes are already under way while the patient is being cared for by the paramedic and before the patient arrives at the hospital, there may be little that can be done to mitigate the damage caused by the initial no-flow period. Efforts to ameliorate neurologic dysfunction in resuscitated patients using calcium channel blockers, anticonvulsants, free-radical scavengers, corticosteroids, and aminosteroids have produced disappointing results.[24–27]

A key characteristic of the post-resuscitation syndrome is the loss of cerebral autoregulation.[28] In the healthy brain, autoregulation maintains a global cerebral blood flow (gCBF) of about 50 mL per 100 g of brain tissue per minute within the cerebral perfusion pressure range of approximately 50 to 150 mm Hg. Cerebral perfusion pressure is the mean arterial pressure minus intracranial pressure. When cerebral perfusion pressure drops below 50 mm Hg, cerebral blood flow decreases. The low-flow states of cardiopulmonary resuscitation

threaten the viability of normal neurons, with cerebral perfusion pressures of less than 30 mm Hg allowing for cerebral blood flow of less than 15 mL per 100 g of brain tissue per minute. Inadequate oxygen delivery is evidenced by a cerebral venous oxygen tension of less than 20 torr.[28–30] The brain may tolerate low flow (gCBF of 10% of normal) better than no flow,[31] but "trickle flow" (gCBF < 10%) can sometimes be worse than no flow.[32]

Cerebral blood-flow problems (i.e., reperfusion failure) in the post-resuscitation patient progress through four stages:

1. *Multifocal no reflow* occurs immediately and seems to be readily overcome by normotensive or hypertensive reperfusion.[33,34] After pulses are restored, patients demonstrate a post-arrest no-reflow phenomenon that can be overcome when reperfusion pressure is high.[33,35]
2. *Transient global "reactive" hyperemia (vasoparalysis)* lasts 10 to 30 minutes.[33,36–38] The hyperemic flow patterns are believed to last longer in the brain stem and basal ganglia than in the neocortex.
3. *Delayed, prolonged, global and multifocal hypoperfusion* begins about two hours after circulation is restored. After the transient period of hyperemia, gCBF is decreased to as little as 50% of normal because of loss of autoregulation capabilities.[33,36,38–40] Despite the hyperperfusion continuing in some limited areas of the brain, gCBF is reduced to about 50% of baseline, while cerebral metabolic oxygen consumption returns to or above baseline values, creating a mismatch of oxygen delivery and uptake.[33,36–38,41]
4. *Late resolution* is marked by either normal gCBF and cerebral oxygen consumption or low gCBF and cerebral oxygen consumption, which is a clinical indicator of brain death.

Optimizing Cardiac and Hemodynamic Resuscitation

Immediately after restoring the patient's pulse, paramedics typically focus on the continued presence of a pulse and the patient's rhythm. Because the scene of a cardiac arrest resuscitation is often chaotic, it can be difficult for paramedics to obtain background information. Nonetheless, they should try to determine whether a resuscitated patient's rhythm disturbance is intrinsic to the cause of the cardiac arrest or a result of the resuscitative efforts. The contribution of tricyclic antidepressants, beta-blockers, calcium channel blockers, cocaine, amphetamines, pesticides, missed dialysis, history of accessory pathways, and other conditions may not be appreciated unless EMS providers specifically ask about these conditions. Often hints to these contributors are present in the prodromal symptoms the patient reported before collapse or the physical signs noticed either before collapse or during the early stages of resuscitation. Conversely, rescuers should remember that in many patients with classic sudden death syndrome, the heart has no structural or vascular abnormalities.

Resuscitation medications are a common extrinsic cause of rhythm disturbances in the post-resuscitation patient. Epinephrine is a potent cause of tachydysrhythmia. Atropine also can contribute to the development of tachycardias. Lidocaine, amiodarone, and other antiarrhythmics can contribute to arrhythmia problems as well. Epinephrine and lidocaine have short half-lives and will be quickly metabolized. Thus their effects, such as tachycardia and profound hypertension from epinephrine, will dissipate over several minutes and should not be treated. Other medications will have more prolonged effects on the heart, blood vessels, and other organs and may contribute substantially to the difficulties encountered trying to stabilize a patient.

Common post-resuscitation dysrhythmias can be categorized as wide-complex tachycardia, narrow-complex tachycardia, and bradycardia, which is often associated with varying degrees of atrioventricular block.

Wide-complex Tachycardia

In non-cardiac-arrest settings, differentiating ventricular tachycardia (VT) from a supraventricular rhythm with aberrant conduction may be difficult, but wide-complex tachycardia is most often VT. However, in the immediate post-resuscitation patient with a pulse, this is quite likely *not* the case. A heart that has been subjected to global ischemia and then develops VT is not likely to sustain a pulsing VT for long and can be expected to deteriorate into ventricular fibrillation quickly. Therefore, if a just-resuscitated patient with a pulse has a wide-complex tachycardia, the patient likely has a supraventricular rhythm with aberrant conduction. Treatment of this rhythm with synchronized countershock or antiarrhythmics may be unnecessary and quite possibly detrimental to the patient.

Narrow-complex Tachycardia

In patients with narrow-complex tachycardia after resuscitation, the indicated therapy is often conservative "watchful waiting," particularly if the patient has received intravenous epinephrine. The International Resuscitation Guidelines 2000 recommend countershock for narrow-complex tachycardia of more than 150 beats/min only when the patient has serious signs and symptoms related to the tachycardia (e.g., chest pain, short-

ness of breath, decreased level of consciousness, low blood pressure, shock, pulmonary congestion, congestive heart failure, or acute myocardial infarction).[42] In the post-arrest setting, the signs and symptoms of critical illness (e.g., unresponsiveness, hypotension) are likely to be related to the anoxic or hypoxic period, and the tachycardia is either protective or simply the effect of epinephrine. Therefore, the tachycardia should not necessarily be treated as if it were a presenting sign. Patients with rapidly progressive tachycardia deteriorating into ventricular fibrillation may be demonstrating the effects of administered epinephrine. As a result, a conservative approach to the post-resuscitation patient with tachycardia that allows the beta-adrenergic effects of epinephrine to subside may enhance the likelihood of the patient's developing a normal rate and pressure.

Bradycardia

Most patients resuscitated from cardiac arrest who are bradycardic are suffering from either serious conduction abnormalities or global myocardial ischemia. Treatment options are to pace the heart, administer atropine, or do nothing. In a hypotensive post-resuscitation patient with a bradycardic rhythm, atropine can generally be given quickly in most EMS systems. However, for those systems equipped with transthoracic pacing capabilities, pacing is the intervention of choice.

Optimizing Neurologic Resuscitation

Currently, beyond providing high-flow oxygen and striving to maintain a heart beat, EMS providers do little specifically to resuscitate the brain. Current research suggests there may be some things paramedics can do to improve the neurologic outcome of cardiac arrest survivors. An emerging theory regarding cerebral dysfunction after cardiopulmonary arrest is that pathophysiologic processes are initially set into motion during the hyperemic period after restoration of normotensive reperfusion. Increasing evidence indicates that continued neurologic damage occurs by further initiation of the same and other pathophysiologic processes during the nonhomogeneous flow patterns that develop shortly after return of spontaneous circulation.[22,23] Therefore, efforts to minimize or eliminate the nonhomogeneous flow patterns of the post-resuscitation patient should be helpful. Among them are hypertensive reperfusion, hemodilution, and mild hypothermia (Table 2).

Table 2. Methods of Optimizing Neurologic Resuscitation

Hypertensive reperfusion Aim for a systolic blood pressure that is higher than normal (i.e., 150 mm Hg).
Hemodilution Promote increased cerebral blood flow by providing hemodilution (normal saline) as part of the vasopressor support.
Mild hypothermia Allow for mild hypothermia to begin by stripping the patient to undergarments and avoiding anything that would increase the patient's body temperature.

Hypertensive Reperfusion

Soon after spontaneous circulation is restored, a fairly short-lived increase in cerebral blood flow occurs. This hyperemic period lasts from 10 to 30 minutes. The hyperemic flow patterns are believed to last longer in the brain stem and basal ganglia than in the neocortex. Following the hyperemic period is a period of variable perfusion to vulnerable regions of the brain. Multiple studies have demonstrated cellular morphologic changes in these brain areas that are believed to represent post-ischemic brain damage.[35,43–45] This damage is believed to result from mismatch of cerebral oxygen consumption and delivery.

As previously mentioned, a serious sequela of cerebral ischemia is the loss of autoregulation of cerebral blood flow.[28] Evidence suggests that induced hypertension or enhanced flow states might overcome problems resulting from loss of autoregulation.[33,35,46] During the period of decreased cerebral blood flow, vulnerable regions in the brain are usually more severely damaged. These areas are characteristically found in the border zones between cerebral arterial distribution zones.[22,23] In animal models, hypertensive reperfusion can overcome the immediate no-reflow phenomenon,[33,34,38] enhance circulation to the vulnerable brain areas, and improve electroencephalographic recovery.[47] In a dog model of cardiac arrest, a brief episode of induced hypertension (mean arterial pressure of 150 to 200 mm Hg for 1 to 5 minutes) followed by controlled normotension eliminated evidence of immediate cerebral no reflow and correlated with improved cerebral outcome.[35] After return of spontaneous circulation in humans, brief hypertension correlates with a good cerebral outcome and hypotension with a poor cerebral outcome.[48,49] However, prolonged severe hypertension after cardiac arrest might not be tolerated by the ischemic heart and can worsen vasogenic cerebral edema.[43,50]

Hemodilution

A contributing factor to the no-reflow phenomenon is sludging. Consequently, hemodilution has been shown to be effective in increasing cerebral blood flow.[33,35] Post-ischemic hypoperfusion can be prevented by hypertensive reperfusion plus normovolemic hemodilution.[33] However, hemodilution is not without its dangers. Overdilution to hematocrit values of less than 30% can decrease arterial oxygen transport capabilities even with increased cardiac output and cerebral blood flow. A hematocrit of 30% seems beneficial.[45]

Mild Hypothermia

Deep hypothermia (<25°C) causes ventricular fibrillation or asystole but is successfully used in the operative theater. Moderate hypothermia (30°C), induced before circulatory arrest, protects the brain during no flow of up to 20 minutes. Mild hypothermia (33–34°C) can improve neurologic outcomes in select patients with stroke or head trauma.[51–55] Mild hypothermia has also shown promise in multiple cardiac arrest studies in animals.[44,56–61] This benefit, however, seems highly time-dependent, with improved outcomes seen only when mild hypothermia is initiated and achieved early in the post-resuscitation period.[62,63] The use of mild hypothermia in human survivors of cardiac arrest has shown promise and is believed to be safe.[64–69]

A variety of animal studies looking at single interventions has produced disappointing results compared with some using combination therapy. Animal studies using a combination of techniques to improve cerebral blood flow combined with mild hypothermia have been remarkably successful.[70,71]

Hyperventilation

Overaggressive hyperventilation can impair cerebral blood flow and promote cerebral edema in post-resuscitation patients.[72] Historically, hyperventilation has been recommended to reverse the metabolic acidosis that has accumulated during the cardiac arrest. Problems with hyperventilation are now recognized, specifically in terms of reduced cerebral blood flow resulting from hypocarbic vasoconstriction. During the period of cerebral oxygen debt, a decrease in oxygen delivery resulting from reduced blood flow *must* be avoided. Capnography and capnometry devices are available to EMS personnel and can reliably guide paramedics as to the arterial carbon dioxide content. While the patient has evidence of adequate cardiac output (i.e., easily palpable pulses), the patient's ventilations should be titrated to maintain end-tidal carbon dioxide levels near 40 mm Hg or just below.

CONCLUSION

Resuscitating a patient who has suffered cardiac arrest is a considerable accomplishment, but to achieve the best possible patient outcome, post-resuscitation myocardial dysfunction and neurologic injury must be minimized. To do so, paramedics must recognize the various types of arrhythmias that can occur after resuscitation and take appropriate steps to end the abnormal rhythm. Arrhythmias in the resuscitated patient can be due to a variety of etiologies different from those in the non-arrested patient, and the patient must be treated accordingly. Neurologic injury resulting from decreased cerebral blood flow can be difficult to prevent or minimize, but use of hypertensive reperfusion, hemodilution, and mild hypothermia has shown promise in improving neurologic outcomes. Furthermore, maintenance of normal $PaCO_2$ levels measured by capnometry will decrease the likelihood of iatrogenic inhibition of cerebral blood flow.

Consensus Presentation

"I've got a pulse back; what do I do now?" This was the overriding question the consensus group sought to answer, as they addressed several aspects of post-resuscitation care of the adult cardiac arrest patient. The consensus participants agreed that after a patient is successfully resuscitated, the goal is to maintain hemodynamic stability through appropriate management of arrhythmias and blood pressure and to improve neurologic outcome.

Rhythm Disturbances

Post-resuscitation rhythm disturbances can be categorized as either tachycardias (wide complex or narrow complex) or bradycardia and as either intrinsically abnormal or extrinsically abnormal (i.e., resulting from the effects of the resuscitation attempt itself). Most tachycardias that occur after resuscitation are likely due to the catecholamine release that accompanies the arrest or the treatment of the arrest itself. Unless the patient is hypotensive, "watchful waiting" is the recommended approach for post-resuscitation tachycardias. For the hypotensive patient who becomes tachycardic after resuscitation, the first step is to determine whether the rhythm disturbance is sinus tachycardia or some other tachyarrhythmia. A 12-lead electrocardiogram is useful in making this distinction and in determining whether an acute myocardial infarction was the cause of the cardiac arrest. Patients who suffer arrest secondary to an acute myocardial infarction or other treatable etiology can potentially benefit from being transported to a facility equipped with a catheterization laboratory. The group considered whether all post-resuscitation patients should be transported to such a facility, but the participants agreed that not enough data were available to support such a recommendation.

The second step in managing the hypotensive tachycardic patient after resuscitation is to administer fluids, unless there is clear evidence of volume overload. The recommendation was to start with 250 to 500 mL of normal saline, to repeat this dose if the patient remained hypotensive, and to titrate the dose to effect or adverse effects. Although most textbooks recommend starting with fluids and then adding either an inotrope or a vasopressor, the consensus group did not endorse this approach, given the paucity of data in this area. The primary concern of the consensus participants was that it is difficult to determine in the field whether a patient has a significant left ventricular wall abnormality or impaired systemic vascular resistance. It is also often difficult to determine whether the patient is hypotensive secondary to a tachycardia or tachycardic secondary to hypotension. Accurately identifying the underlying problem based on history or physical examination might not be possible. Thus empirically adding either an inotrope or a vasopressor might not only be ineffective but could even be harmful to the patient. The best approach for these patients is to administer fluids, monitor for signs of pulmonary edema, and rapidly transport them to the hospital. However, during transport, paramedics should continue aggressive ventilatory management and constant reassessment.

Bradycardia in the post-resuscitation patient is primarily due to ventilatory problems, such as poor airway management or poor ventilation. The first task, then, is to look for potential causes of impaired ventilation. Pacing the bradycardic heart is generally considered first-line therapy. Although no strong evidence indicates that pacing is any more effective than atropine in a bradycardic patient, it can be done fairly quickly, particularly if combination pads have been used for the cardiac arrest, and its effects are quickly reversible, simply by shutting off the machine. Atropine, which can also be administered fairly quickly, cannot be "turned off" as can pacing if adverse effects develop. Little evidence supports the use of a vasopressor in post-resuscitation bradycardia.

The consensus group did agree that bradycardia in the post-resuscitation patient should be treated more aggressively than it currently is. Paramedics are traditionally taught that bradycardia in a patient who is not hypotensive is of little concern. This philosophy may be changing, as more data accumulate on the potential benefits of a higher-than-normal blood pressure in bradycardic post-resuscitation patients.

Blood Pressure

Patients who are hypertensive after resuscitation from cardiac arrest tend to have better outcomes than do those who are hypotensive. Whether hypertension is a marker for patients who will do better or whether the hypertension itself contributes to better outcomes is unclear. In any case, given the potential benefits of hypertension, post-resuscitation patients who are hypotensive should not be given high-dose dopamine as a single agent unless vasodilation is clearly the cause of the hypotension. The difficulty in differentiating primary tachycardia with secondary hypotension from primary hypotension with secondary tachycardia was previously mentioned. Acknowledging that concern, the consensus group noted that combining dopamine with norepineph-

rine may be of benefit in hypotensive post-arrest patients, because these patients often are relatively volume-depleted and vasodilated. However, most data supporting use of this combination were gathered from septic shock models rather than true post-resuscitation cardiac arrest models. Dobutamine might also be a good choice for post-resuscitation hypotension. However, until further data are available, the consensus group would not make any strong recommendation on the management of post-resuscitation hypotension beyond the administration of fluids.

For post-resuscitation patients who are hypertensive, the consensus participants were unanimous in their recommendation—do not treat hypertension after resuscitation.

NEUROLOGIC OUTCOME

The ultimate goal of post-resuscitation management is to optimize neurologic outcome. Certainly, appropriate management of arrhythmias and blood pressure contributes to improved neurologic outcomes. Whether mild hypothermia (33–34°C) is of value is not completely clear, nor is it clear how best to achieve a mildly hypothermic state. Use of fluorocarbons to rapidly cool patients to an appropriate temperature might be possible in the future. At present, the consensus group recommended passive hypothermia. This approach entails removing the patient's clothing to the limits of the environment. Patients can then cool passively but should not be allowed to become overly hypothermic (<30°C).

Hyperthermia and seizures increase the metabolic demands on the brain at a time when the brain may not have enough supply to meet those demands. For that reason, both should be avoided. If seizures do occur, they should be treated aggressively.

OTHER CONSIDERATIONS

The consensus participants agreed that gathering useful data in the field on post-resuscitation care is difficult. The group did recommend use of end-tidal carbon dioxide ($ETCO_2$) monitoring to confirm and monitor endotracheal tube placement, guide ventilatory support after resuscitation, determine return of spontaneous circulation, and provide a surrogate measurement for cardiac output. To optimize neurologic outcome, paramedics should avoid hyperventilating patients below an $ETCO_2$ of 30 mm Hg.

CONCLUSION

Few data are available on post-resuscitation care of the cardiac arrest patient. What data are available have come mostly from other models, such as those for septic shock. Nonetheless, paramedics can take steps to improve the outcome of post-resuscitation patients, including appropriate management of arrhythmias, blood pressure, and body temperature. In the future, improved modalities for cardiopulmonary resuscitation and minimally invasive thoracotomy procedures may lead not only to improved resuscitation but also to improved cerebral perfusion pressure, which is an outcome that would help minimize secondary brain injury.

Although the consensus participants had several recommendations for post-resuscitation care, they emphasized that rapid return of spontaneous circulation is the cornerstone of good post-resuscitation management. Emergency medical services providers should not become so focused on treating complications or drug adverse effects that they ignore the fact that the best means of avoiding neurologic injury is to achieve return of spontaneous circulation as quickly as possible.

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