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TO: ALL LUCAS COUNTY PARAMEDICS

DATE: April 22, 2008

FROM: Brent Parquette, NREMT-P
Continuing Education Program Administrator

RE: Continuing Education – May 2008

Emergency medical services have gone through dramatic changes in the last 25 years. Cutting edge drugs and diagnostic equipment give patients the best possible chance of survival, especially when it comes to out-of-hospital cardiac arrests. Despite these advances, however, the statistical chance of a patient making full neurological recovery following out-of-hospital cardiac arrest is still very poor. We need to consider how to help these patients not only survive, but have quality of life. Therapeutic hypothermia, a relatively new treatment that may increase the odds of neurological recovery, may be the answer.

Paramedics play an important role in beginning this potentially lifesaving treatment. EMS is a vital link in the therapeutic hypothermia process, which usually must continue a minimum of 12 hours following cardiac arrest. Since hypothermia benefits decrease drastically after a delay of even a few minutes following successful cardiac arrest resuscitation, EMS may be in the best position to begin immediate treatment. Current research has shown, almost universally, that therapeutic hypothermia indeed reduces brain damage following cardiac arrest.

These are exciting and innovative times for LCEMS. With the recent addition of the ResQGARD for trial, and now induced hypothermia, we now have a greater ability to affect positive outcomes with our patients. This month we will delve into the world of "induced hypothermia." We will be one of only 4 EMS agencies nationally providing this advanced therapy in the field.

Attached with this letter please find:

- May class agenda
- "I.C.E." Protocol (Induced Cooling by EMS)
- New Equipment/ Pharmacology Protocols
  - Braun Thermoscan Pro 4000 Tympanic Thermometer
  - Fentanyl
  - Etomidate
  - Vecuronium Bromide (Norcuron)
- Training Scenarios

Please take time to review all of the included material to help better prepare you before class attendance in the month of May. The scenarios provided will be the "actual" scenarios presented in each of the skill stations.

As always, if you have any questions or comments, please feel free to contact me at 419-213-6508. I look forward to seeing all of you in the coming month.

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Part 7.5: Postresuscitation Support

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Part 7.5: Postresuscitation Support

Few randomized controlled clinical trials deal specifically with supportive care following cardio-pulmonary-cerebral resuscitation (CPCR) from cardiac arrest. Nevertheless, postresuscitation care has significant potential to improve early mortality caused by hemodynamic instability and multi-organ failure and later mortality/morbidity resulting from brain injury.[1] This section summarizes our evolving understanding of the hemodynamic, neurologic, and metabolic abnormalities encountered in patients who are resuscitated from cardiac arrest.

Initial objectives of postresuscitation care are to

- Optimize cardiopulmonary function and systemic perfusion, especially perfusion to the brain
- Transport the victim of out-of-hospital cardiac arrest to the hospital emergency department (ED) and continue care in an appropriately equipped critical care unit
- Try to identify the precipitating causes of the arrest
- Institute measures to prevent recurrence
- Institute measures that may improve long-term, neurologically intact survival

Improving Postresuscitation Outcomes

Postresuscitation care is a critical component of advanced life support. Patient mortality remains high after return of spontaneous circulation (ROSC) and initial stabilization. Ultimate prognosis in the first 72 hours may be difficult to determine,[2] yet survivors of cardiac arrest have the potential to lead normal lives.[3–5] During postresuscitation care providers should (1) optimize hemodynamic, respiratory, and neurologic support; (2) identify and treat reversible causes of arrest; and (3) monitor temperature and consider treatment for disturbances of temperature regulation and metabolism. The first sections below discuss initial stabilization and temperature/metabolic factors that may be relevant to improving postresuscitation outcome, particularly in the critically ill survivor. Subsequent sections highlight organ-specific evaluation and support.

Return of Spontaneous Circulation

The principal objective of postresuscitation care is the reestablishment of effective perfusion of organs and tissue. After ROSC in the out-of-hospital or in-hospital setting, the provider must consider and treat the cause of the arrest and the consequences of any hypoxemic/ischemic/reperfusion injury. In most cases the acidemia associated with cardiac arrest improves spontaneously when adequate ventilation and perfusion are restored. But restoration of blood pressure and improvement in gas exchange do not ensure survival and functional recovery. Significant myocardial stunning and hemodynamic instability can develop, requiring vasopressor support. Most postresuscitation deaths occur during the first 24 hours.[6,7]

Ideally the patient will be awake, responsive, and breathing spontaneously. Alternatively the patient may initially be comatose but have the potential for full recovery after postresuscitation care.[3] Indeed, up to 20% of initially comatose survivors of cardiac arrest have been reported to have good 1-year neurologic outcome.[8] The pathway to the best hospital postresuscitation care of all initial survivors is not completely known, but there is increasing interest in identifying and optimizing practices that can improve outcome.[9] Regardless of the patient's initial status, the provider should support adequate airway and breathing, administer supplementary oxygen, monitor the patient's vital signs, establish or verify existing intravenous access, and verify the function of any catheters in place.

The clinician should assess the patient frequently and treat abnormalities of vital signs or cardiac arrhythmias and request studies that will further aid in the evaluation of the patient. It is important to identify and treat any cardiac, electrolyte, toxicologic, pulmonary, and neurologic precipitants of arrest. The clinician may find it helpful to review the H's and T's mnemonic to recall factors that may contribute to cardiac arrest or complicate resuscitation or postresuscitation care: hypovolemia, hypoxia, hydrogen ion (acidosis), hyper-/hypokalemia, hypoglycemia, hypothermia; toxins, tamponade (cardiac), tension pneumothorax, thrombosis of the coronary or pulmonary vasculature, and trauma. For further information see Part 10: "Special Resuscitation Situations."

After initial assessment and stabilization of airway, ventilation, and circulation, transfer the patient to a special care unit for observation, continuous monitoring, and further therapy. Personnel with appropriate training and resuscitation equipment must accompany the patient during transport to the special care unit.

Temperature Regulation

Induced Hypothermia

Both permissive hypothermia (allowing a mild degree of hypothermia >33°C [91.5°F] that often develops spontaneously after arrest) and active induction of hypothermia may play a role in postresuscitation care. In 2 randomized clinical trials (LOE 1[3]; LOE 2[4]) induced hypothermia (cooling within minutes to hours after ROSC) resulted in improved outcome in adults who remained comatose after initial resuscitation from out-of-hospital ventricular fibrillation (VF) cardiac arrest. Patients in the study were cooled to 33°C (91.5°F)[3] or to the range of 32°C to 34°C (89.6°F to 93.2°F)[4] for 12 to 24 hours. The Hypothermia After Cardiac Arrest (HACA) study[3] included a small subset of patients with in-hospital cardiac arrest.

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A third study (LOE 2)[10] documented improvement in metabolic end points (lactate and O_2 extraction) when comatose adult patients were cooled after ROSC from out-of-hospital cardiac arrest in which the initial rhythm was pulseless electrical activity (PEA)/asystole.

In the HACA[3] and Bernard[4] studies, only about 8% of patients with cardiac arrest were selected for induced hypothermia (ie, patients were hemodynamically stable but comatose after a witnessed arrest of presumed cardiac etiology). This highlights the importance of identifying the subset of patients who may most benefit. Although the number of patients who may benefit from hypothermia induction is limited at present, it is possible that with more rapid and controlled cooling and better insights into optimal target temperature, timing, duration, and mechanism of action, such cooling may prove more widely beneficial in the future.[11] A recent multicenter study in asphyxiated neonates showed that hypothermia can be beneficial in another select population.[12]

Complications associated with cooling can include coagulopathy and arrhythmias, particularly with an unintentional drop below target temperature. Although not significantly higher, cases of pneumonia and sepsis increased in the hypothermia-induction group.[3,4] Cooling may also increase hyperglycemia.[4]

Most clinical studies of cooling have used external cooling techniques (eg, cooling blankets and frequent applications of ice bags) that may require a number of hours to attain target temperature. More recent studies[13] suggest that internal cooling techniques (eg, cold saline, endovascular cooling catheter) can also be used to induce hypothermia. Providers should continuously monitor the patient's temperature during cooling.[3,4]

In summary, providers should not actively rewarm hemodynamically stable patients who spontaneously develop a mild degree of hypothermia (>33°C [91.5°F]) after resuscitation from cardiac arrest. Mild hypothermia may be beneficial to neurologic outcome and is likely to be well tolerated without significant risk of complications. In a select subset of patients who were initially comatose but hemodynamically stable after a witnessed VF arrest of presumed cardiac etiology, active induction of hypothermia was beneficial.[3,4,13] Thus, unconscious adult patients with ROSC after out-of-hospital cardiac arrest should be cooled to 32°C to 34°C (89.6°F to 93.2°F) for 12 to 24 hours when the initial rhythm was VF (Class IIa). Similar therapy may be beneficial for patients with non-VF arrest out of hospital or for in-hospital arrest (Class IIb).

Hyperthermia

After resuscitation, temperature elevation above normal can create a significant imbalance between oxygen supply and demand that can impair brain recovery. Few studies (with either frequent use of antipyretics or "controlled normothermia" with cooling techniques) have directly examined the effect of temperature control immediately after resuscitation. Because fever may be a symptom of brain injury, it may be difficult to control it with conventional antipyretics. Many studies of brain injury in animal models, however, show exacerbation of injury if body/brain temperature is increased during or after resuscitation from cardiac arrest.[14–17] Moreover, several studies have documented worse neurologic outcome in humans with fever after cardiac arrest (LOE 3)[18] and ischemic brain injury (LOE 7 extrapolated from stroke victims[18]). Thus, the provider should monitor the patient's temperature after resuscitation and avoid hyperthermia.

Glucose Control

The postresuscitation patient is likely to develop electrolyte abnormalities that may be detrimental to recovery. Although many studies have documented a strong association between high blood glucose after resuscitation from cardiac arrest and poor neurologic outcomes (LOE 4[21,22]; LOE 5[9,22–26]; LOE 6[27]), they did not show that control of serum glucose level alters outcome.

A prospective randomized study by van den Berghe (LOE 1)[28] did show that tight control of blood glucose using insulin reduced hospital mortality rates in critically ill patients who required mechanical ventilation. The study did not specifically focus on postresuscitation patients, but the effect of blood glucose control on outcome is compelling. The study documented not only improved survival but decreased mortality from infectious complications, a common problem in the postresuscitation setting.

In comatose patients, signs of hypoglycemia are less apparent, so clinicians must monitor serum glucose closely to avoid hypoglycemia when treating hyperglycemia. On the basis of findings of improved outcomes in critically ill patients when glucose levels are maintained in the normal range, it is reasonable for providers to maintain strict glucose control during the postresuscitation period. Additional study is needed, however, to identify the precise blood glucose concentration that requires insulin therapy, the target range of blood glucose concentration, and the effect of tight glucose control on outcomes of patients after cardiac arrest.

Organ-Specific Evaluation and Support

After ROSC patients may remain comatose or have decreased responsiveness for a variable period of time. If spontaneous breathing is absent or inadequate, mechanical ventilation via an endotracheal tube or other advanced airway device may be required. Hemodynamic status may be unstable with abnormalities of cardiac rate, rhythm, systemic blood pressure, and organ perfusion.

Clinicians must prevent, detect, and treat hypoxemia and hypotension because these conditions can exacerbate brain injury. Clinicians should determine the baseline postarrest status of each organ system and support organ function as needed.

The remainder of this chapter focuses on organ-specific measures that should be provided in the immediate postresuscitation period.

Respiratory System

After ROSC patients may exhibit respiratory dysfunction. Some patients will remain dependent on mechanical ventilation and will need an increased inspired concentration of oxygen. Providers should perform a full physical examination and evaluate the chest radiograph to verify appropriate

endotracheal tube depth of insertion and identify cardiopulmonary complications of resuscitation. Providers should adjust mechanical ventilatory support based on the patient's blood gas values, respiratory rate, and work of breathing. As the patient's spontaneous ventilation becomes more efficient, the level of respiratory support may be decreased until spontaneous respiration returns. If the patient continues to require high inspired oxygen concentrations, providers should determine if the cause is pulmonary or cardiac and direct care accordingly.

Debate exists as to the length of time patients who require ventilatory support should remain sedated. To date there is little evidence to guide therapy. One observational study (LOE 3)[29] found an association between use of sedation and development of pneumonia in intubated patients during the first 48 hours of therapy. The study, however, was not designed to investigate sedation as a risk factor for either pneumonia or death in patients with cardiac arrest. At this time there is inadequate data to recommend for or against the use of a defined period of sedation or neuromuscular blockade after cardiac arrest (Class Indeterminate). Use of neuromuscular blocking agents should be kept to a minimum because these agents preclude thorough neurologic assessments during the first 12 to 72 hours after ROSC.[2]

Sedation may be necessary to control shivering during hypothermia. If shivering continues despite optimal sedation, neuromuscular blockade may be required in addition to deep sedation.

Ventilatory Parameters

Sustained hypocapnea (low P_{CO_2}) may reduce cerebral blood flow.[30-31] After cardiac arrest, restoration of blood flow results in an initial hyperemic blood flow response that lasts 10 to 30 minutes, followed by a more prolonged period of low blood flow.[32,33] During this latter period of late hypoperfusion, a mismatch between blood flow (oxygen delivery) and oxygen requirement may occur. If the patient is hyperventilated at this stage, cerebral vasoconstriction may further decrease cerebral blood flow and increase cerebral ischemia and ischemic injury.

There is no evidence that hyperventilation protects the brain or other vital organs from further ischemic damage after cardiac arrest. In fact, Safar et al[34] provided evidence that hyperventilation may worsen neurologic outcome. Hyperventilation may also generate increased airway pressures and augment intrinsic positive end-expiratory pressure (so-called "auto PEEP"), leading to an increase in cerebral venous and intracranial pressures.[35,36] Increases in cerebral venous pressure can decrease cerebral blood flow and increase brain ischemia.

In summary, no data supports targeting a specific arterial P_{aCO_2} level after resuscitation from cardiac arrest. But data extrapolated from patients with brain injury supports ventilation to normocarbic levels as appropriate. Routine hyperventilation is detrimental (Class III).

Cardiovascular System

Both the ischemia/reperfusion of cardiac arrest and electrical defibrillation can cause transient myocardial stunning and dysfunction[37] that can last many hours but may improve with vasopressors.[38] Cardiac biomarker levels may be increased in association with global ischemia caused by absent or decreased coronary blood flow during cardiac arrest and CPR. Increased cardiac biomarkers may also indicate acute myocardial infarction as the cause of cardiac arrest.

Hemodynamic instability is common after cardiac arrest, and early death due to multi-organ failure is associated with a persistently low cardiac index during the first 24 hours after resuscitation (LOE 5).[6,39] Thus, after resuscitation clinicians should evaluate the patient's electrocardiogram, radiographs, and laboratory analyses of serum electrolytes and cardiac biomarkers. Echocardiographic evaluation within the first 24 hours after arrest is useful to guide ongoing management.[5,40]

One large case series (LOE 5)[6] of patients resuscitated following out-of-hospital cardiac arrest documented significant early but reversible myocardial dysfunction and low cardiac output, followed by later vasodilation. The hemodynamic instability responded to fluid administration and vasoactive support.[6] Invasive monitoring may be necessary to measure blood pressure accurately and to determine the most appropriate combination of medications to optimize blood flow and distribution. The provider should titrate volume administration and vasoactive (eg, norepinephrine), inotropic (eg, dobutamine), and inodilator (eg, milrinone) drugs as needed to support blood pressure, cardiac index, and systemic perfusion. The ideal target blood pressure or hemodynamic parameters associated with optimal survival have not been established.

Both cardiac arrest and sepsis are thought to involve multi-organ ischemic injury and microcirculatory dysfunction. Goal-directed therapy with volume and vasoactive drug administration has been effective in improving survival from sepsis.[41] The greatest survival benefit is due to a decreased incidence of acute hemodynamic collapse, a challenge also seen in the postresuscitation setting. Data extrapolated from a study of goal-directed therapy for sepsis (LOE 1[41] for sepsis; LOE 7 [extrapolated] for cardiac arrest) suggests that providers should try to normalize oxygen content and oxygen transport.

Relative adrenal insufficiency may develop following the stress of cardiac arrest, but the use of early corticosteroid supplementation in such patients to improve either hemodynamics or outcome is unproven and requires further evaluation.[42]

Although sudden cardiac arrest may be precipitated by cardiac arrhythmia, it is unclear if antiarrhythmics are beneficial or detrimental in the postresuscitation period. Thus, there is insufficient evidence to recommend for or against prophylactic administration of antiarrhythmic drugs to patients who have survived cardiac arrest from any cause. It may be reasonable, however, to continue an infusion of an antiarrhythmic drug that was associated with ROSC (Class Indeterminate). Also, given the cardioprotective effects of β-blockers in the context of ischemic heart disease, the use of β-blockers in the postresuscitation setting seems prudent if there are no contraindications.[9]

Central Nervous System

A healthy brain and a functional patient are the primary goals of cardio-pulmonary-cerebral resuscitation. 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.[43,44]

Neurologic support 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 elevated. Because hyperthermia and seizures increase the oxygen requirements of the brain, providers should treat hyperthermia and consider therapeutic hypothermia. Witnessed seizures should be promptly controlled and maintenance anticonvulsant therapy initiated (Class IIa). Because of a paucity of data, routine seizure prophylaxis is a Class Indeterminate recommendation at present.

Prognostic Factors

The period after resuscitation is often stressful to medical staff and family members as questions arise about the patient's ultimate prognosis. Ideally a clinical assessment, laboratory test, or biochemical marker would reliably predict outcome during or immediately after cardiac arrest. Unfortunately no such predictors are available. Determination of prognosis based on initial physical examination findings can be difficult, and coma scores may be less predictive than individual motor and brainstem reflexes found in the first 12 to 72 hours after arrest.[2]

In a meta-analysis (LOE 1)[44] bilateral absence of cortical response to median nerve somatosensory-evoked potentials predicted poor outcome in normothermic patients who were comatose for at least 72 hours after hypoxic-ischemic insult. A case report[46] also documents the usefulness of this evaluation. Therefore, median nerve somatosensory-evoked potentials measured 72 hours after cardiac arrest can be used to predict neurologic outcome in patients with hypoxic-anoxic coma.

A recent meta-analysis (LOE 1) of 11 studies involving 1914 patients[2] documented 5 clinical signs that were found to strongly predict death or poor neurologic outcome, with 4 of the 5 predictors detectable at 24 hours after resuscitation:

- Absent corneal reflex at 24 hours
- Absent pupillary response at 24 hours
- Absent withdrawal response to pain at 24 hours
- No motor response at 24 hours
- No motor response at 72 hours

An electroencephalogram performed >24 to 48 hours after resuscitation has also been shown to provide useful predictive information (LOE 5[47–50]) and can help define prognosis.

Other Complications

Sepsis is a potentially fatal postresuscitation complication.[51] Patients with sepsis will benefit from goal-directed therapy. Renal failure[52] and pancreatitis, while often transient, should be diagnosed and evaluated.[3,53]

Summary

The postresuscitation period is often marked by hemodynamic instability as well as laboratory abnormalities. This is also a period for which promising technological interventions such as controlled therapeutic hypothermia are being evaluated. Every organ system is at risk during this time, and patients may ultimately develop multi-organ dysfunction. A complete discussion of this topic is beyond the scope of this chapter. The goal of the postresuscitation period is to manage the patient's vital signs and laboratory abnormalities and support organ system function to increase the likelihood of intact neurologic survival.

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Therapeutic hypothermia after cardiac arrest. An advisory statement by the Advanced Life Support Task Force of the International Liaison Committee on Resuscitation☆

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1. ILCOR recommendations

On the basis of the published evidence to date, the Advanced Life Support (ALS) Task Force of the International Liaison Committee on Resuscitation (ILCOR) made the following recommendations in October 2002:

- Unconscious adult patients with spontaneous circulation after out-of-hospital cardiac arrest should be cooled to 32–34 °C for 12–24 h when the initial rhythm was ventricular fibrillation (VF).
- Such cooling may also be beneficial for other rhythms or in-hospital cardiac arrest.

2. Introduction

Induction of moderate hypothermia (28–32 °C) before cardiac arrest has been used successfully since the 1950s to protect the brain against the global ischaemia that occurs during some open-heart surgery. Successful use of therapeutic hypothermia after cardiac arrest in humans was also described in the late 1950s [1–3] but was subsequently abandoned because of uncertain benefit and difficulties with its use [4]. Since then, induction of hypothermia after return of spontaneous circulation (ROSC) has been associated with improved functional recovery and reduced cerebral histological deficits in various animal models of cardiac arrest [5–8]. Additional promising preliminary human studies have been completed [9–16]. At the time of publication of the *Guidelines 2000 for Cardiopulmonary Resuscitation and*

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Emergency Cardiovascular Care, the evidence was insufficient to recommend use of therapeutic hypothermia after resuscitation from cardiac arrest [17].

3. Clinical studies

In 2002 the results of two prospective randomised trials were published that compared mild hypothermia with normothermia in comatose survivors of out-of-hospital cardiac arrest [18,19]. One study was undertaken in nine centers in five European countries [19]; the other was conducted in four hospitals in Melbourne, Australia [18].

The criteria for entry into these trials were similar: ROSC, patients remaining intubated and ventilated, with persistent coma after out-of-hospital cardiac arrest due to VF. In the European study, the median Glasgow Coma Scale score on hospital admission in both groups was 3 with an interquartile range of 3–5 in the group with normothermia and 3–4 in the group with hypothermia [20]. Ten patients in the European study were resuscitated after in-hospital cardiac arrest (M. Holzer, written communication, October 2002). Additional criteria for entry into the European study were witnessed cardiac arrest, an estimated interval of 5–15 min from patient collapse to first resuscitation attempt by emergency medical services personnel, and an interval of ≤ 60 min from collapse to ROSC. Both studies excluded arrests that were probably of noncardiac aetiology and patients with severe cardiogenic shock.

In the European study, patients randomly assigned to the hypothermia group underwent cooling to a target temperature of 32–34 °C by use of a mattress made specifically for this purpose (with a cover that delivered cold air) and ice packs if necessary. The aim was to reach the target temperature within 4 h of ROSC, maintain it for 24 h, and follow with passive rewarming. In the Australian study, patients were pseudo-randomised (odd vs. even days) to treatment groups that allowed cooling, by application of cold packs to the head and torso, to begin in the field before admission to the hospital. The target temperature of the hypothermia group was 33 versus 37 °C in the control group. Hypothermia was maintained for 12 h after admission to the hospital; active rewarming started at 18 h.

In the European study, 75 of the 136 patients (55%) in the hypothermia group for whom data were available had a favorable neurological outcome (able to live independently and work at least part-time) at 6 months compared with 54 of 137 (39%) in the normothermia group (relative risk [RR] 1.40, 95% CI 1.08–1.81, number needed to treat [NNT] = 6] [19]. At 6 months there were 56 deaths in the 137 participants (41%) in the hypothermia group versus 76 of 138 (55%) in the normothermia group (RR 0.74, 95% CI 0.58–0.95, NNT = 7). The target temperature could not be achieved in 19 patients in the hypothermia group.

In the Australian study, 21 of 43 patients (49%) treated with hypothermia had good neurological function at discharge (to home or a rehabilitation facility) compared with 9 of 34 (26%) in the normothermia group (RR 1.85, 95% CI 0.97–3.49, NNT = 4) [18]. Mortality at discharge was 22 of 43 (51%) in the hypothermia group and 23 of 34 (68%) in the normothermia group (RR 0.76, 95% CI 0.52–1.10, NNT = 6).

Both of these studies involved a highly selected group of patients, excluding up to 92% of patients with out-of-hospital cardiac arrest initially assessed for eligibility [19]. Those excluded had persistent hypotension (systolic blood pressure < 90 mmHg despite use of inotropes) and causes of coma other than cardiac arrest (e.g. head injury, drug overdose, cerebrovascular accident). Other study limitations were that caregivers could not be blinded to treatment with hypothermia and that following ROSC, the normothermia group had an increase in core temperature of up to > 38 °C, as is often seen after cardiac arrest.

Some adverse events occurred more frequently in the hypothermia groups. The Australian hypothermia group had a lower cardiac index, higher systemic vascular resistance, and more hyperglycaemia than patients in the control group [18]. Although not statistically significant, in the European hypothermia group there were 22% more complications; in particular, there were more cases of pneumonia [number needed to harm (NNH) = 12], bleeding (NNH = 14), and sepsis (NNH = 16) [19].

4. Mechanisms of action

There are several possible mechanisms by which mild hypothermia might improve neurological outcome when used after reperfusion. In the normal brain, hypothermia reduces the cerebral metabolic rate for oxygen ($CMRO_2$) by 6% for every 1 °C reduction in brain temperature > 28 °C [21]. Some of this effect is due to reduced normal electrical activity [21], however, and after cardiac arrest in dogs, $CMRO_2$ is not significantly reduced by mild hypothermia [22]. Mild hypothermia is thought to suppress many of the chemical reactions associated with reperfusion injury. These reactions include free radical production, excitatory amino acid release, and calcium shifts, which can in turn lead to mitochondrial damage and apoptosis (programmed cell death) [23–25]. Despite these potential advantages, hypothermia can also produce adverse effects, including arrhythmias, infection, and coagulopathy.

5. Discussion

5.1. Selection of patients

There seems to be good evidence (Level 1 [see Appendix A]) to recommend the use of induced mild hypothermia in comatose survivors of out-of-hospital cardiac arrest caused by VF. Selection criteria for treatment were narrowly defined in the best evidence used and thus should be considered carefully when deciding to treat.

Several specific questions remain unanswered despite the results of these recently published controlled trials, previous clinical studies, and supporting experiments in animals. One controversial issue is whether findings from animal experiments and published clinical studies are enough to extend the use of therapeutic mild hypothermia to patients who remain comatose after cardiac arrest from any rhythm, after in-hospital cardiac arrest, and after cardiac arrest in children.

Any potentially beneficial effects of hypothermia on neuronal recovery must be counterbalanced by the known adverse effects of hypothermia. Although survivors of VF cardiac arrest have the most to gain from therapeutic hypothermia, some level 4 evidence suggests that survivors from out-of-hospital cardiac arrest of other causes may also benefit [9]. Further study is required. Many in-hospital cardiac arrests have non-cardiac causes, and because the use of therapeutic hypothermia has not been studied to a significant extent in this population, its relative risks and benefits are unknown. It is possible, however, that patients who remain comatose after an in-hospital arrest of cardiac aetiology will also benefit from therapeutic hypothermia.

Until further data are available, therapeutic hypothermia should not be used for patients with severe cardiogenic shock or life-threatening arrhythmias, pregnant patients, or patients with primary coagulopathy. Thrombolytic therapy does not preclude the use of hypothermia; patients who received thrombolytic therapy were included in both the European and Australian studies.

5.2. Timing of cooling

Cooling should probably be initiated as soon as possible after ROSC but appears to be successful even if delayed (e.g. 4–6 h). In the European study, the interval between ROSC and attainment of a core temperature of 32–34 °C had an interquartile range of 4–16 h [19].

Further research is needed to determine optimal duration of therapeutic hypothermia, optimum target temperature, and rates of cooling and rewarming. Animal data suggest that the sooner cooling is initiated after reperfusion from cardiac arrest, the better the outcome, although an impressive therapeutic benefit was seen in clinical studies when cooling was delayed for several hours. The therapeutic benefit may become much greater as better physical and pharmacological techniques to cool patients more rapidly become available. Although supporting data are limited, many critical care clinicians routinely sedate and ventilate the lungs of comatose survivors of cardiac arrest for at least 12–24 h; thus, application of therapeutic hypothermia over this period would be simple. Normothermia should be restored only slowly as rebound hyperthermia is common and should be avoided [14].

5.3. Cooling techniques and monitoring

A variety of cooling techniques have been described, but at this stage, none combines ease of use with high efficacy. External cooling methods are simple to use but slow in reducing core temperature. These techniques include the use of cooling blankets; application of ice packs to the groin, axillae, and neck; use of wet towels and fanning; and use of a cooling helmet [15]. In a recent study, intravenous infusion of 30 ml kg^{-1} of crystalloid at 4 °C over 30 min reduced core temperature significantly and did not cause pulmonary oedema [26]. Cooling by peritoneal and pleural lavage is possible but not generally used [27]. Extracorporeal cooling methods are efficient [12] but too invasive for use in the prehospital environment or most emergency departments. An intravascular heat exchange device, which enables rapid cooling and precise temperature control, has recently become available.

Shivering during cooling leads to warming and will increase overall oxygen consumption. Shivering should be prevented by use of a neuromuscular blocker and sedation (as done in the two definitive trials). Careful monitoring of temperature is important during use of therapeutic hypothermia. The incidence of complications such as arrhythmias, infection, and coagulopathy is likely to increase if the core temperature falls considerably below 32 °C. Continuous monitoring of temperature can be accomplished by use of a bladder temperature probe or a pulmonary artery catheter if one is in situ. Other temperature-monitoring techniques, including intermittent tympanic temperature measurements, are less reliable.

5.4. Use of therapeutic hypothermia in children

There is currently insufficient evidence to make a recommendation on the use of therapeutic hypothermia in children resuscitated from

cardiac arrest. The European and Australian clinical trials excluded children and cardiac arrests of noncardiac aetiology (e.g. respiratory failure or shock), which are typical of those in children [18,19]. In the 1970s therapeutic hypothermia was used to reduce secondary brain injury in children with severe anoxic/ischaemic insults. The practice was abandoned in the 1980s after a retrospective study of near-drowning victims reported that children treated with hypothermia were at an increased risk for death, neutropenia, and sepsis compared with children treated without hypothermia [28,29]. Important limitations of this study were the limited sample size, use of historic controls, and use of a lower target temperature for a longer duration than recommended in contemporary protocols (30–33 °C for ≥36 h vs. 32–34 °C for 12–24 h).

Until additional paediatric data become available, clinicians should tailor therapy for individual patients based on their assessment of the risks and benefits of hypothermia. Risk-benefit assessment should take into account relevant data from laboratory models of asphyxial arrest [8,30–32], results from trials of adult cardiac arrest [18,19], and reports on the use of hypothermia in treatment of neonatal asphyxia [33–38]. The results of therapeutic hypothermia are generally favorable in laboratory models of hypoxic ischaemic injury to immature brains of various species. Preliminary data from clinical trials of perinatal asphyxia indicate that induced hypothermia is feasible and safe, but data on long-term neurological morbidity are not yet available [33–38]. It is difficult to extrapolate from these disparate sources of information, and thus there is no consensus yet for use of therapeutic hypothermia among clinicians who care for these critically ill children.

6. Summary: ILCOR recommendations

On the basis of the published evidence to date, the ILCOR ALS Task Force has made the following recommendations:

- Unconscious adult patients with spontaneous circulation after out-of-hospital cardiac arrest should be cooled to 32–34 °C for 12–24 h when the initial rhythm was VF.
- Such cooling may also be beneficial for other rhythms or in-hospital cardiac arrest.

Appendix A: 1998 AHA ECC levels of evidence summary

Level of evidence	Definitions
Level 1	One or more RCTs in which the lower limit of CI for treatment effect exceeds the minimal clinically important benefit
Level 2	One or more RCTs in which the lower limit of the CI for treatment effect overlaps the minimal clinically important benefit
Level 3	Prospective cohort of patients not randomized to an intervention; control or comparison group available
Level 4	Historic, nonrandomized cohort or case-control studies
Level 5	Case series: patients compiled in serial fashion; a control group is lacking
Level 6	An animal or mechanical model study; a level 6A study is well designed, shows a homogeneous pattern of results; a level 6B study has a less powerful design and demonstrates an equivocal or heterogeneous pattern of results
Level 7	Reasonable extrapolations from existing data; quasiexperimental designs; pathophysiological and nonquantitative reasoning
Level 8	Rational conjecture (common sense); common practices accepted before evidence-based guidelines

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May 18, 2007

To Treat the Dead; The new science of resuscitation is changing the way doctors think about heart attacks–and death itself." Newsweek (May 7, 2007): 56

Byline: Jerry Adler

Consider someone who has just died of a heart attack. His organs are intact, he hasn't lost blood. All that's happened is his heart has stopped beating–the definition of "clinical death"–and his brain has shut down to conserve oxygen. But what has actually died?

As recently as 1993, when Dr. Sherwin Nuland wrote the best seller "How We Die," the conventional answer was that it was his cells that had died. The patient couldn't be revived because the tissues of his brain and heart had suffered irreversible damage from lack of oxygen. This process was understood to begin after just four or five minutes. If the patient doesn't receive cardiopulmonary resuscitation within that time, and if his heart can't be restarted soon thereafter, he is unlikely to recover. That dogma went unquestioned until researchers actually looked at oxygen-starved heart cells under a microscope. What they saw amazed them, according to Dr. Lance Becker, an authority on emergency medicine at the University of Pennsylvania. "After one hour," he says, "we couldn't see evidence the cells had died. We thought we'd done something wrong." In fact, cells cut off from their blood supply died only hours later.

But if the cells are still alive, why can't doctors revive someone who has been dead for an hour? Because once the cells have been without oxygen for more than five minutes, they die when their oxygen supply is resumed . It was that "astounding" discovery, Becker says, that led him to his post as the director of Penn's Center for Resuscitation Science, a newly created research institute operating on one of medicine's newest frontiers: treating the dead.

Biologists are still grappling with the implications of this new view of cell death–not passive extinguishment, like a candle flickering out when you cover it with a glass, but an active biochemical event triggered by "reperfusion," the resumption of oxygen supply. The research takes them deep into the machinery of the cell, to the tiny membrane-enclosed structures known as mitochondria where cellular fuel is oxidized to provide energy. Mitochondria control the process known as apoptosis, the programmed death of abnormal cells that is the body's primary defense against cancer. "It looks to us," says Becker, "as if the cellular surveillance mechanism cannot tell the difference between a cancer cell and a cell being reperfused with oxygen. Something throws the switch that makes the cell die."

With this realization came another: that standard emergency-room procedure has it

exactly backward. When someone collapses on the street of cardiac arrest, if he's lucky he will receive immediate CPR, maintaining circulation until he can be revived in the hospital. But the rest will have gone 10 or 15 minutes or more without a heartbeat by the time they reach the emergency department. And then what happens? "We give them oxygen," Becker says. "We jolt the heart with the paddles, we pump in epinephrine to force it to beat, so it's taking up more oxygen." Blood-starved heart muscle is suddenly flooded with oxygen, precisely the situation that leads to cell death. Instead, Becker says, we should aim to reduce oxygen uptake, slow metabolism and adjust the blood chemistry for gradual and safe reperfusion.

Researchers are still working out how best to do this. A study at four hospitals, published last year by the University of California, showed a remarkable rate of success in treating sudden cardiac arrest with an approach that involved, among other things, a "cardioplegic" blood infusion to keep the heart in a state of suspended animation. Patients were put on a heart-lung bypass machine to maintain circulation to the brain until the heart could be safely restarted. The study involved just 34 patients, but 80 percent of them were discharged from the hospital alive. In one study of traditional methods, the figure was about 15 percent.

Becker also endorses hypothermia–lowering body temperature from 37 to 33 degrees Celsius–which appears to slow the chemical reactions touched off by reperfusion. He has developed an injectable slurry of salt and ice to cool the blood quickly that he hopes to make part of the standard emergency-response kit. "In an emergency department, you work like mad for half an hour on someone whose heart stopped, and finally someone says, 'I don't think we're going to get this guy back,' and then you just stop," Becker says. The body on the cart is dead, but its trillions of cells are all still alive. Becker wants to resolve that paradox in favor of life.

Filed under ICE - Induced Cooling by EMS by admin

Lucas County EMS
Continuing Education Program
May 2008

Agenda

1 hour 20 minutes	Introduction to "Induced Hypothermia" • "I.C.E." Protocol • Pharmacology o Fentanyl o Etomidate o Vecuronium (Norcuron) • "I.C.E." Bypass Criteria
10 minutes	BREAK
2 hours 30 minutes	Skill Stations Stations 1-4: Cardiac Arrest Scenarios Station 5: ePCR Entry

Note: Each skill station will be 30 minutes in duration

Lucas County EMS

I.C.E – Hypothermia Protocol (Induced Cooling by EMS)

Current research has shown, almost universally, that therapeutic hypothermia reduces brain damage following cardiac arrest. It is well known that the brain responds poorly to hypoxic events. In most situations, the 4-6 minute "point of no return" still applies. Part of the problem (perhaps not realized by many responders) is that brain damage will continue for several hours following resuscitation; it doesn't simply stop because the patient's heart starts beating again. Therapeutic hypothermia can help increase the odds of these patients recovering completely.

Paramedics play an important role in beginning the therapeutic hypothermia process, which usually must continue for a minimum of 12 hours following cardiac arrest. Since therapeutic hypothermia benefits decrease drastically after a delay of even a few minutes following successful cardiac arrest resuscitation, EMS may be in the best position to begin immediate treatment.

Criteria for Induced Hypothermia:

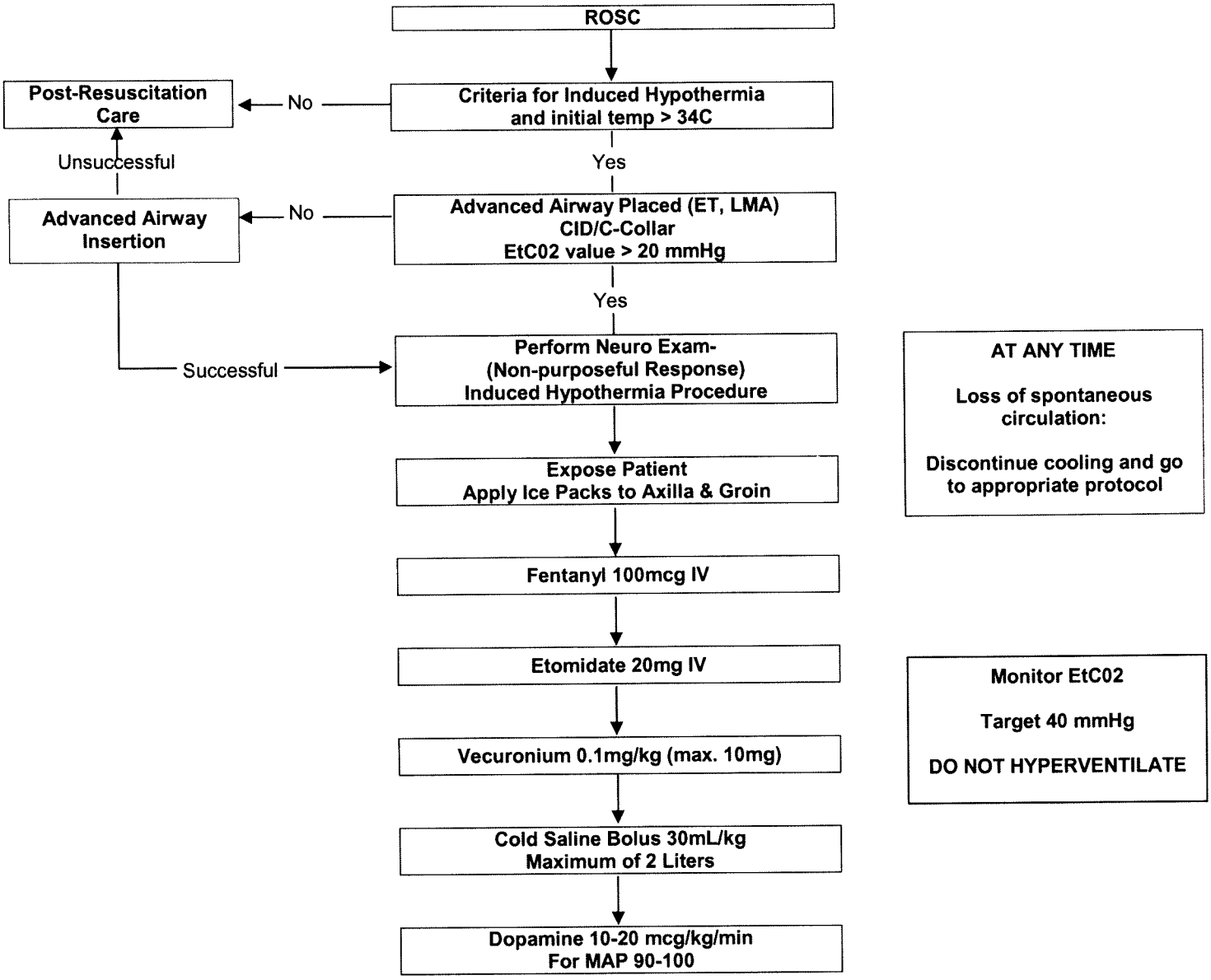
1. ROSC after cardiac arrest not related to trauma or hemorrhage
2. Age $\geq$ 16
3. Initial temperature > 34C
4. Patient has advanced airway in place (e.g., ETT, LMA) and remains comatose (no purposeful response to pain)
    a. If unable to secure an advanced airway in place, **DO NOT** initiate induced hypothermia
5. Not obviously pregnant

Assessment Notes:

1. When exposing patient for purpose of cooling, undergarments may remain in place. Be mindful of your environment and take steps to preserve the patient's modesty.
2. **DO NOT** delay transport for the purpose of cooling.
3. Reassess airway frequently and with every patient movement.
4. Patients develop metabolic alkalosis with cooling. **DO NOT** hyperventilate.
5. If there is a loss of ROSC at any time, discontinue cooling and go to appropriate protocol for treatment.
6. Continue to address specific differentials associated with original dysrhythmia or cause of arrest (H's and T's).
7. Patients with ROSC and/or induced hypothermia should be triaged to the closest "Hypothermia" center for continuation of the cooling process. A hypothermia "Alert" should be declared through LCEMS Dispatch.

I.C.E – Hypothermia Protocol (Induced Cooling by EMS)

History:	Signs/Symptoms:	Differential:
• Non-Traumatic Cardiac Arrest	• Return of Pulse (ROSC)	• Continue to address specific differentials associated with the original dysrhythmia



Assessment Notes:

- In the event that your patient's MAP increases to > 120, re-dose Etomidate at 20mg IV (at least 5 minutes after initial dose).
- With signs of patient movement (i.e., gasping, eye fluttering, seizure activity, shivering, movement), re-dose Vecuronium at 1/10 the original dose (.01mg/kg IV) and repeat Etomidate at 20mg IV.

Screening for Utilization

- ☐ Return of Pulse (ROSC)
- ☐ Age ≥ 16
- ☐ Temperature > 34C (Tympanic measurement)
- ☐ No purposeful pain response
- ☐ Intubated (or LMA) with EtC02 > 20 mm Hg
- ☐ Not obviously pregnant

Preparation for Induction - Hypothermia

- ☐ Conduct NEURO assessment:
  a. Pupils (size, reactivity, equality)
  b. Motor Response to Pain
- ☐ Remove clothing, protect modesty
- ☐ Apply cold packs to axilla and groin
- ☐ Goal EtC02 = 40; NO Hyperventilation
- ☐ Attempt second IV/IO (if not in place)

1

Induction of Paralysis

1. Administer Fentanyl 100mcg IV

2. Administer Etomidate 20mg IV

3. Administer Vecuronium 0.1mg/kg IV (max. 10 mg)

Weight (lbs)	Weight (kg)	Dose (mg)	Volume (cc)
88	40	4	4
110	50	5	5
132	60	6	6
154	70	7	7
176	80	8	8
198	90	9	9
220	100	10	10

Vecuronium 1mg/mL Only

2

Saline Infusion and Maintenance of Mean Arterial Pressure

1. Initiate cold saline bolus through up to two (2) IV or IO access points
2. Infuse cold saline at 30mL/kg to maximum of 2 Liters

Weight (lbs)	Weight (kg)	Volume Target (mL)
88	40	1200
110	50	1500
132	60	1800
> or =143	> or =65	2000

3. **Target Mean Arterial Pressure (MAP): 90-100**
4. Check MAP on the LP12, but manually monitor

Systolic	Diastolic	MAP
110	80	90
120	75-90	90-100
130	70-85	90-100
140	65-80	90-100

MAP = Diastolic Value + 1/3 Pulse Pressure

- **80 ≤ Target Diastolic ≤ 90**

5. If chilled saline does not maintain MAP go to 4

3

Maintenance of MAP with Pressors

1. Support BP with Dopamine as required to **maintain MAP of 90-100**

The values in this chart are drips per minute on a 60 drop/minute drip set:

Dopamine 400mg/250mL D5W

Weight (lbs)	Weight (kg)	5mcg/kg/min	10mcg/kg/min	20mcg/kg/min
88	40	8	15	30
110	50	9	19	38
132	60	11	23	45
154	70	13	26	53
176	80	15	30	60
198	90	17	34	68
220	100	19	38	75
242	110	21	41	83

NOTE: Discontinue Dopamine drip when diastolic pressure is ≥ 90 or MAP ≥ 100.

2. Cold saline is a strong vasoconstrictor. Watch MAP closely!

4

II.
Braun ThermoScan PRO 4000 Thermometer

The Braun ThermoScan thermometer has been developed for accurate, safe and fast human body temperature measurements in the ear. The shape of the thermometer probe prevents it from being inserted too far into the ear canal which could perforate the tympanic membrane.

Braun ThermoScan measures the infrared heat generated by the eardrum and surrounding tissues. To help ensure accurate temperature measurements, the sensor itself is warmed to a temperature close to that of the human body. When the Braun ThermoScan is placed in the ear, it continuously monitors the infrared energy until a temperature equilibrium has been reached and an accurate measurement can be taken.

A. Procedure for use

1. To achieve accurate measurements, make sure a new, clean probe cover is in place before each measurement.

2. When the probe cover is in place, the thermometer turns on automatically. Wait for the ready signal beep.

3. Fit the probe snuggly into the ear canal, then push and release the Start button.

4. If the probe has been inserted into the ear canal during the complete measuring process, a long beep will signal the end of the measuring process. The thermometer detects that an accurate temperature measurement has been taken. The result is shown on the display screen.
    a. When you take a temperature measurement, the <ExacTemp> light can be of help. It flashes during the measuring process when the probe is correctly positioned, and lights up continuously when the thermometer detects that an accurate measurement has been taken.

5. For the next measurement, eject the used probe cover (push ejector) and put on a new, clean probe cover. The thermometer will turn on automatically. Wait for the ready signal. Fit the probe snuggly into the ear canal, then push and release the Start button.

The Braun ThermoScan ear thermometer turns off automatically after 60 seconds of inactivity. It can also be turned off by pressing the <I/O> button for at least three seconds. The display will shortly flash <OFF> and steadily display the word <OFF> after releasing the button.

B. Temperature taking hints

1. A temperature measurement taken in the right ear may differ from a measurement taken in the left ear. Therefore, always take the temperature in the same ear.

2. The ear must be free from obstructions or excess cerumen (wax) build-up in order to take an accurate measurement.

3. External factors may influence ear temperatures, particularly when an individual has:
   - been lying on one ear or the other
   - had their ears covered
   - been exposed to very hot or very cold temperatures, or
   - been recently swimming or bathing.

4. For persons wearing hearing aids or ear plugs, remove the device and wait 20 minutes prior to taking a temperature.

5. Use the untreated ear if ear drops or other ear medications have been placed in the ear canal.

Body Temperature

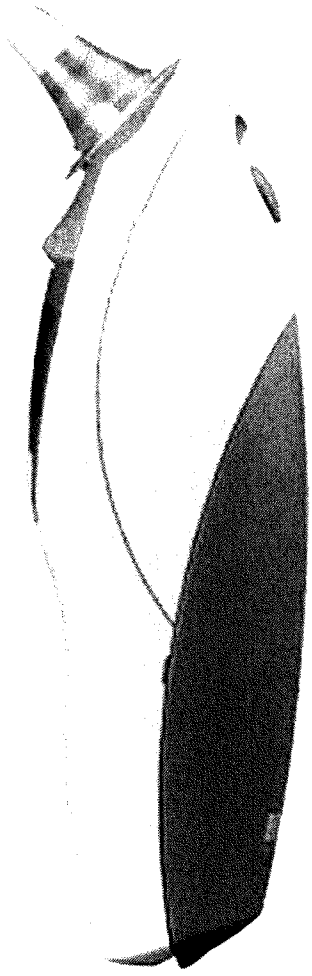
Normal body temperature is a range. The following table shows that ranges of normal also vary by site. Therefore, readings from different sites, even if taken at the same time, should not be directly compared.

Normal Ranges by site:

Axillary	94.5 – 99.1 F	34.7 – 37.3 C
Oral	95.9 – 99.5 F	35.5 – 37.5 C
Rectal	97.9 – 100.4 F	36.6 – 38.0 C
ThermoScan	96.4 – 100.4 F	35.8 – 38.0 C

A person's normal temperature range tends to decrease with age. The following table shows normal ThermoScan ranges by age:

0 – 2 years	97.5 – 100.4 F	36.4 – 38.0 C
3 – 10 years	97.0 – 100.0 F	36.1 – 37.8 C
11 – 65 years	96.6 – 99.7 F	35.9 – 37.6 C
> 65 years	96.4 – 99.5 F	35.8 – 37.5 C




98.6

Fentanyl (Sublimaze)

Onset 7 minutes
Duration 1-2 hours

Indications

A. Management of pain
B. Sedation for induced hypothermia in the setting of post-arrest ROSC.

Contraindications

A. Labor
B. Pediatrics less than 2 years old
C. In the presence of other CNS depressants

Adverse Reactions

A. Somnolence
B. Respiratory depression
C. Muscle Rigidity
D. Bradycardia
E. Seizures
F. Diaphoresis

Special Considerations:

A. Caution in children and the elderly (½ dose)

Reference in Protocol

Tab 500
Medical Procedures/Equip.
Q-3 FF-1

Tab 800
Cardiac Protocols
I.C.E. Induced Hypothermia
G-2; G-3; H-1; H-3; I-2
I-3; J-2; J-4; K-2; K-3;
L-2; L-4; M-2; M-3; O-1;
O-2

Tab 900
Medical Emergencies
P-1

Supplied
100mcg/2mL Carpujet

Etomidate

Onset Rapid, within 1 minute
Duration Dose dependent (usually 3-5 minutes)

Indications

A. Patient sedation in preparation for induced hypothermia subsequent to post-arrest ROSC.

Contraindications

A. None in the setting of post-arrest ROSC and no purposeful pain response

Adverse Reactions

A. Nausea
B. Vomiting
C. Involuntary muscle movements

Special Considerations:

A. Etomidate has anesthetic and amnestic, but no analgesic properties.

Reference in Protocol

Tab 800
Cardiac Protocols
I.C.E – Induced Hypothermia

Supplied
40mg/20mL Vial (2mg/mL)

Vecuronium Bromide (Norcuron)

Onset 1-5 minutes
Duration 25-40 minutes

Indications

A. Vecuronium is a muscle relaxant in the category of neuromuscular blocking agents of intermediate duration. It is used as an adjunct to sedation to facilitate induced hypothermia and to promote skeletal muscle relaxation during post-resuscitation care. Although vecuronium bromide is often thought of as a muscle relaxant, it may be more accurate to classify it as a paralyzing agent.

Contraindications

A. Known hypersensitivity

Adverse Reactions

A. Extension of the drug's pharmacological action beyond the time period needed

Special Considerations:

A. Vecuronium, if re-dosed, is administered at 1/10 the original dose.

Reference in Protocol

Tab 800
Cardiac Protocols
I.C.E – Induced Hypothermia

Supplied
10mg/10mL (1mg/mL)
Must be re-constituted

Scenario #1

Rhythm Sequence	Algorithm
1. Idioventricular Rhythm (No pulses)	Pulseless Electrical Activity (PEA)
2. Ventricular fibrillation	Ventricular fibrillation
3. Sinus Tachycardia	

A 55-YEAR-OLD, 80KG MAN WALKS INTO THE STATION COMPLAINING OF SEVERE CHEST PAIN. HE IS PLACED ON A STRETCHER AS YOU BEGIN TO GATHER INFORMATION. JUST AS YOUR PARTNER STARTS TO ATTACH THE MONITOR LEADS, HE FALLS BACK UNCONSCIOUS ON THE STRETHCER.

EKG:	"PULSELESS ELECTRICAL ACITIVITY	Correct Diagnosis?	Yes	No
Scen. 1A	Safe Scene Universal Precautions			
	Check for responsiveness			
	Primary and Secondary ABCD performed			
	Check vitals signs			
	Patient not breathing and pulseless			
	Starts CPR			
	Utilizes BVM with airway adjunct. ***Attaches ResQPOD to facemask (maintains adequate face seal)***			
	Applies Quick-Combo patches/Hard wire – (PEA)			
	Has team member start IV (1000mL NS)			
	Verbalizes need for large bore catheter (16ga if possible) ***NOTE: If no peripheral access found – insert IO***			
	Has team member intubate			
	• ***Confirmation with PE, breath sounds, TubeChek, capnography*** • ***Attaches ResQPOD to ET – engages timing light for ventilation*** • ***Consider ATV (10-12bpm) at appropriate TV for patient weigh*** • ***ET, LMA – (CID/C-Collar for continued airway control)***			
	Vasopressin 40 Units IV (single dose)			
	Circulate with 2 minutes of CPR			
	Epinepherine 1mg 1:10,000 IV			
	Circulate with 2 minutes of CPR			
	Atropine 1mg IV			
	Circulate with 2 minutes of CPR			

EKG:	"VENTRICULAR FIBRILLATION"	Correct Diagnosis?	Yes	No
Scen. 1B	Notes change in rhythm – Ventricular Fibrillation			
	Checks vital signs			
	Patient remains unresponsive, pulseless and apneic			
	Resumes CPR (if delay in immediate defibrillation)			
	Checks monitor			
	Patient in V-Fib ***Considers pre-cordial thump***			
	Clears area before defibrillating			
	Defibrillation (200J – Biphasic; 360J – Monophasic)			
	Has team members resume CPR			
	Evaluates effectiveness of CPR			
	Secondary ABCD survey performed			
	Epinepherine 1mg 1:10,000 IV			
	Circulate with 2 minutes of CPR			
	Clears area before defibrillating			
	Defibrillation (200J – Biphasic; 360J – Monophasic)			
	Amiodarone 300mg IV			
	Circulate with 2 minutes of CPR			
	Clears area before defibrillating			
	Defibrillation (200J – Biphasic; 360J – Monophasic)			
	Notes Rhythm Change (Organized Electrical Activity)			
	Assesses patient for change in condition			
	Notes change in rhythm – Sinus Tachycardia			

EKG:	"SINUS TACHYCARDIA"	Correct Diagnosis?	Yes	No
	Check for responsiveness and vital signs.			
	Patient appropriately responsive to painful stimulus ***P-120 (ROSC)*** ***BP – 132/80; Minimal gasping respiratory efforts*** ***EtC02 = 65mmHg*** ***Removal of ResQPOD from respiratory circuit***			
	Continue supportive care for patient			
	Ventilation efforts addressed to achieve EtCO2 = 40mm Hg ***Recognizes patient does not qualify for "Induced hypothermia" protocol***			
	12-Lead ECG Acquisition			
	12-Lead ECG non-diagnostic for ischemic or injury patterns			
	Transport Destination (Bypass Criteria)			
	Verbalizes need to transfer patient to closest PCI facility due to ROSC			

Critical Areas for Emphasis:

- Enhanced CPR performance with ResQPOD (facemask and ET attachment)
- 2 minute CPR intervals between interventions
- Proper sequencing of therapies for presenting rhythms (pharmacologic/electrical)
- Differential diagnosis for cause of arrest (H's and T's)
- Importance of EtC02 values during arrest and upon achieving ROSC
- Continuation of supportive care post-arrest (ROSC)
    - Serial vital signs
    - 12-Lead acquisition/interpretation
    - Continued cardiac monitoring
- Recognition of parameters for "hypothermia" induction
- Hospital bypass criteria for patient with ROSC

Scenario #2

Rhythm Sequence	Algorithm
1. Ventricular Tachycardia	Ventricular Tachycardia/Pulse Present/Unstable
2. Ventricular Fibrillation	Ventricular Fibrillation/Witnessed
3. Sinus Rhythm	Induced Hypothermia Protocol

YOU ARE EXAMINING A 60-YEAR-OLD 60KG (132LB) WOMAN WHO IS COMPLAINING OF PALPITATIONS, DIFFICULTY BREATHING AND SEVERE PRESSURE ON HER CHEST. WHILE BEGINNING TO EXAMINE HER SHE TELLS YOU SHE IS BEGINNING TO FEEL VERY WEAK AND THINKS SHE IS GOING TO PASS OUT. HER PULSE IS ABOUT 180 AND HER BP IS 80 SYSTOLIC.

EKG:	"VENTRICULAR TACHYCARDIA" (UNSTABLE) Correct Diagnosis?	Yes	No
Scen. 2A	Safe Scene Universal Precautions		
	Oxygen by appropriate method (Non-Rebreather Mask)		
	Vital signs (as noted above)		
	Has team member start IV, 1000mL NS		
	Verbalizes need for large bore catheter (16ga if possible)		
	Applies Quick-Combo patches/Hard wire		
	Identifies rhythm as Ventricular Tachycardia ***Prepares for synchronized cardioversion for unstable presentation***		
	Pre-medicates (if possible)		
	50mcg Fentanyl slow IV (no patient medication allergies)		
	Clears area before synchronized cardioversion		
	Immediate synchronized cardioversion at 100 Joules		
	******The patient loses consciousness and goes into V-Fib******		
	(Go to page 2)		

EKG:	"VENTRICULAR FIBRILLATION"	Correct Diagnosis?	Yes	No
	Notes change in rhythm – Ventricular Fibrillation (Witnessed)			
	Checks for responsiveness			
	Patient is unresponsive, pulseless and apneic			
	Begins CPR (If delay in Defibrillation)			
	Checks monitor			
	Patient in V-Fib, no pulse ***Considers pre-cordial thump (witnessed V-Fib)***			
	Clears area before defibrillating			
	Defibrillation (200J – Biphasic; 360J – Monophasic)			
	Has team members start/resume CPR			
	Evaluates effectiveness of CPR ***ResQPOD attached to facemask if delay in advanced airway placement***			
	Has team member Intubate			
	Confirmation with PE, breath sounds, TubeChek, capnography ***Attaches ResQPOD to ET – engages timing light for ventilation*** ***Consider ATV (10-12bpm) at appropriate TV for patient weight*** ***ET, LMA – (CID/C-Collar for continued airway control)***			
	Vasopressin 40 Units IV (single dose)			
	Circulate with 2 minutes of CPR			
	Clears area before defibrillating			
	Defibrillation (200J – Biphasic; 360J – Monophasic)			
	Amiodarone 300mg IV			
	Circulate with 2 minutes of CPR			
	Clears area before defibrillating			
	Defibrillation (200J – Biphasic; 360J – Monophasic)			
	********Notes change in rhythm********			
	(Go to Page 3)			

EKG:	"NORMAL SINUS RHYTHM"	Induction of Hypothermia	Yes	No
	Check for responsiveness and vital signs.			
	Patient remains unresponsive with no appropriate response to pain ***P – 98, reg. (ROSC); BP - 90/60*** ***NO spontaneous respiratory effort*** ***EtC02 = 55mmHg*** ***Removal of ResQPOD from respiratory circuit***			
	Continue supportive care for patient			
	Ventilation efforts addressed to achieve EtCO2 = 40mm Hg ***Recognizes patient qualifies for "Induced hypothermia" protocol***			
	12-Lead ECG (when possible during post-resuscitation care)			
	12-Lead ECG diagnostic for Antero-Septal Injury Pattern			
	Obtains patient temperature (Tympanic measurement)			
	Assures patient temperature > 34C (93.2 F)			
	Re-evaluates effectiveness of ventilation with advanced airway			
	ET, LMA - (CID/C-Collar for continued airway control) ***Assures EtC02 value > 20mm Hg***			
	Patient Neuro Examination			
	Assures No or Non-Purposeful response to painful stimuli			
	Patient exposure/Ice Pack Application			
	Ice Packs applied to axilla, groin ***NOTE: Application of ice packs to neck if absent C-Collar***			
	Attempt 2nd IV/IO line (when possible)			
	Fentanyl 50mcg IV			
	Initial 50mcg given prior to cardioversion ***If > 15 minutes from initial administration – 100mcg IV***			
	Amidate 20mg IV			
	Vecuronium 0.1mg/kg IV (maximum 10mg)			
	Initiate cold saline bolus at 30mL/kg (maximum 2 Liters)			
	Initiate cold saline bolus through up to two (2) IV or IO access points			
	(Go to Page 4)			

Cont.	INDUCTION OF HYPOTHERMIA (CONT)	Yes	No
	Re-Assess Patient		
	Patient remains unresponsive and apneic; ***P – 98; BP – 90/60;*** ***EtC02 = 40mm Hg ECG – NSR with no ectopy***		
	Prepares for administration of Dopamine Drip		
	400mg/250cc D5W with 60gtt administration set ***Verbalizes target for MAP at 90-100 (Check MAP on LP12)***		
	Initiates Dopamine Drip (10mcg/kg/min)		
	Patient BP is at 90/60 = MAP of 70 ***(Pt body weight 60kg = 600mcg/min = 23gtts/min)*** ***NOTE: Titrate Dopamine up to 20mcg/kg/min to achieve MAP of 90-100*** ***[80 ≤ Target Diastolic ≤ 90]*** ***Remember:*** ***Cold Saline is a strong vasoconstrictor. Watch MAP closely. In the event your target MAP is met (90-100), stop the Dopamine infusion.*** ***[Discontinue Dopamine when diastolic pressure is ≥ 90 or MAP ≥ 100]*** ***Re-start the Dopamine drip in the event the MAP falls below 90-100.***		
	**** Instructor verbalizes that approximately 1.5 Liters of chilled saline has been administered to the patient over the last 10 minutes ****		

Scen. 2B	Re-Assesses Patient		
	Patient remains unresponsive and apneic; ***P – 100 reg; BP – 120/90*** ***EtC02 = 40mm Hg;*** ***ECG – NSR with no ectopy***		
	Verbalizes cessation of Dopamine Drip		
	Target MAP achieved		
	Declares "I.C.E" Protocol with LCEMS Dispatch		
	Early Notification when possible ***Patient also fits STEMI criteria for bypass*** ***Bypass criteria for "Hypothermia" Centers (Toledo, SVMMC, UTMC, St Lukes)***		
	Hospital Arrival		
	Re-obtains patient temperature (tympanic measurement) ***Transfer to ED staff for continued cooling measures***		

Critical Areas for Emphasis:

- Enhanced CPR performance with ResQPOD (facemask and ET attachment)
- 2 minute CPR intervals between interventions
- Proper sequencing of therapies for presenting rhythms (pharmacologic/electrical)
- Importance of EtC02 values during arrest and upon achieving ROSC
- Continuation of supportive care post-arrest (ROSC)
    - Serial vital signs
    - 12-Lead acquisition/interpretation
    - Continued cardiac monitoring
- Recognition of parameters for "hypothermia" induction
    - Patient neuro assessment
    - Baseline tympanic ear temperature measurement
    - Patient sedation for induced hypothermia
    - Procedure for chilled saline administration
    - Maintenance of MAP throughout post-resuscitative care
- Hospital bypass criteria for patient with ROSC and Induced hypothermia protocol

Scenario #3

Rhythm Sequence	Algorithm
1. Ventricular Fibrillation	Ventricular Fibrillation
2. Idioventricular Rhythm (No pulse)	Pulseless Electrical Activity (PEA)
3. Sinus Tachycardia	Induced Hypothermia Protocol

YOUR PATIENT IS A 52-YEAR-OLD 70KG (154LB) WOMAN FOUND ON THE LIVING ROOM FLOOR. UPON ARRIVAL YOU FIND THE PATIENT INTUBATED AND AN IV STARTED BY THE FIRST RESPONDERS. THE PATIENT HAS BEEN DEFIBRILLATED ONCE AT 360 JOULES (MONOPHASIC LP12) AND 40 UNITS OF VASOPRESSIN HAS BEEN ADMINISTERED. ADEQUATE CPR IS BEING PERFORMED, AND THERE IS A RESQPOD ATTACHED TO THE ET TUBE.

EKG:	"VENTRICULAR FIBRILLATION"	Correct Diagnosis?	Yes	No
Scen. 3A	Checks for responsiveness			
	Patient is unresponsive, apneic and pulseless			
	Check of monitor – Patient still in V-Fib			
	Primary and Secondary ABCD performed			
	Checks ET tube placement, evaluates effectiveness of CPR			
	Attaches EtC02 filter line (if not already accomplished)			
	Consider ATV (10-12bpm) at appropriate TV for patient weight			
	Immobilization of head with CID/C-collar for airway control			
	Clears area before defibrillating			
	Assures 2 minutes of CPR before defibrillation			
	Defibrillate (200J – Biphasic; 360 J – Monophasic)			
	Has team members resume CPR			
	Evaluates effectiveness of CPR			
	Amiodarone 300mg IV			
	Circulate with 2 minutes of CPR			
	Clears area before defibrillating			
	Defibrillate (200J – Biphasic; 360 J – Monophasic)			
	********Organized rhythm achieved on monitor*********			
	(Go to page 2)			

EKG:	"IDIOVENTRICULAR RHYTHM" – (PEA)	Correct Diagnosis?	Yes	No
	Notes change in rhythm – Idioventricular Rhythm (NO Pulses)			
	Idioventricular rhythm @ 40bpm			
	Checks for responsiveness			
	Patient remains unresponsive, pulseless and apneic			
	Resumes CPR/ Hardwire patient (if not already Accomplished)			
	Epinepherine 1mg 1:10,000 IV			
	Circulate with 2 minutes of CPR			
	Checks monitor/rhythm			
	Continued Idioventricular rhythm (PEA)			
	Atropine 1mg IV			
	Circulate with 2 minutes of CPR			
	********Change in Rhythm noted on Monitor*******			

EKG:	"SINUS TACHYCARDIA"	Correct Diagnosis?	Yes	No
Scen. 3B	Check for responsiveness and vital signs.			
	Patient remains unresponsive with no appropriate response to pain ***P – 110, reg. (ROSC); BP - 110/80*** ***NO spontaneous respiratory effort*** ***EtC02 @ROSC = 75 mmHg*** ***Removal of ResQPOD from respiratory circuit***			
	Continue supportive care for patient			
	Ventilation efforts addressed to achieve EtCO2 = 40mm Hg ***Recognizes patient qualifies for "Induced hypothermia" protocol***			
	Obtains patient temperature (Tympanic measurement)			
	Assures patient temperature > 34C (93.2 F)			
	Re-evaluates effectiveness of ventilation with advanced airway			
	ET, LMA - (CID/C-Collar for continued airway control) ***Assures EtC02 value > 20mm Hg***			
	(Go to Page 3)			

Cont.	INDUCTION OF HYPOTHERMIA (CONT)	Yes	No
	Patient Neuro Examination		
	Assures NO or Non-Purposeful response to painful stimuli		
	Patient exposure/Ice Pack Application		
	Ice Packs applied to axilla, groin ***NOTE: Application of ice packs to neck if absent C-Collar***		
	Attempt 2nd IV/IO line (when possible)		
	Fentanyl 100mcg IV		
	Amidate 20mg IV		
	Vecuronium 0.1mg/kg IV (maximum 10mg)		
	Initiate cold saline bolus at 30mL/kg (maximum 2 Liters)		
	Initiate cold saline bolus through up to two (2) IV or IO access points		
	******** After cold saline is initiated, rhythm change is noted on monitor*******		

EKG:	"VENTRICULAR FIBRILLATION"	Yes	No
Scen. 3C	Re-Assesses Patient		
	Monitored/witnessed Ventricular Fibrillation ***Loss of spontaneous pulses***		
	Begins CPR (if delay in defibrillation)		
	Considers pre-cordial thump (Witnessed V-Fib)		
	Clears area before defibrillating		
	Defibrillate (200J – Biphasic; 360 J – Monophasic)		
	Has team members resume CPR		
	Evaluates effectiveness of CPR ***Re-attaches ResQPOD to ET tube (engages timing light for ventilation)*** ***Consider re-application of ATV at appropriate TV for patient weight***		
	(Go to Page 4)		

Cont.	"VENTRICULAR FIBRILLATION"	Yes	No
	Discontinues induction of cooling with chilled saline		
	Ambient temp. saline bag attached to IV line(s) OR chilled saline to KVO rate		
	Removal of applied ice packs		
	Epinepherine 1mg 1:10,000 IV		
	Circulate with 2 minutes of CPR		
	Clears area before defibrillating		
	Defibrillate (200J – Biphasic; 360 J – Monophasic)		
	********Notes Change in Rhythm********		

EKG:	"SINUS TACHYCARDIA"	Re-Induction of Hypothermia	Yes	No
	Check for responsiveness and vital signs.			
	Patient remains unresponsive with no appropriate response to pain			
	P – 120, reg. (ROSC); BP - 120/70			
	NO spontaneous respiratory effort			
	EtC02 @ROSC = 50 mmHg			
	Removal of ResQPOD from respiratory circuit			
	Continue supportive care for patient			
	Ventilation efforts addressed to achieve EtCO2 = 40mm Hg			
	Re-introduction of "hypothermia protocol"			
	Obtains patient temperature (Tympanic measurement)			
	Assures patient temperature > 34C (93.2 F)			
	Re-evaluates effectiveness of ventilation with advanced airway			
	ET, LMA - (CID/C-Collar for continued airway control)			
	Assures EtC02 value > 20mm Hg			
	Patient Neuro Examination			
	Assures NO or Non-purposeful response to painful stimuli			
	Patient exposure/Ice Pack Application			
	Re-introduce ice packs to axilla, groin			
	NOTE: Application of ice packs to neck if absent C-Collar			
	Attempt 2nd IV/IO line (if not previously accomplished)			
	If time permits			
	(Go to Page 5)			

Cont.	"SINUS TACHYCARDIA"	Re-Induction of Hypothermia	Yes	No
	Re-Dose Amidate 20mg IV			
	Re-dose Amidate 20mg IV if 5 minutes after initial dose			
	Re-initiates cold saline bolus 30mL/kg (maximum 2 Liters)			
	Cold saline bolus through up to two (2) IV or IO access points			
	******After 5 minutes of chilled saline infusion******			
Scen. 3D	Re-Assess Patient			
	Patient remains unresponsive and apneic ***P – 100, reg.*** ***BP – 130/70; MAP = 90*** ***EtC02 = 45mm Hg;*** ***ECG – Sinus Tachycardia***			
	Target MAP achieved			
	Watches MAP closely to maintain 90-100 ***NOTE: If MAP increases >120: Re-dose Amidate 20mg IV if 5 minutes after last administration*** ***Amidate 20mg can be re-dosed at 5 minute intervals for MAP > 120 to total of 4 doses.***			
	12-Lead ECG (when possible during post-resuscitation care)			
	12-Lead ECG diagnostic for antero-lateral "ischemia"			
	Declares "I.C.E" Protocol with LCEMS Dispatch			
	Early notification when possible ***Bypass criteria for "Hypothermia" Centers (Toledo, SVMC, UTMC, St. Lukes)***			
	Hospital Arrival			
	Re-obtains patient temperature (tympanic measurement) ***Transfer to ED staff for continued cooling measures***			

Critical Areas for Emphasis:

- Enhanced CPR performance with ResQPOD (facemask and ET attachment)
- 2 minute CPR intervals between interventions
- Proper sequencing of therapies for presenting rhythms (pharmacologic/electrical)
- Importance of EtC02 values during arrest and upon achieving ROSC
- Continuation of supportive care post-arrest (ROSC)
    - Serial vital signs
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    - Continued cardiac monitoring
- Recognition of parameters for "hypothermia" induction
    - Patient neuro assessment
    - Baseline tympanic ear temperature measurement
    - Patient sedation for induced hypothermia
    - Procedure for chilled saline administration
    - Maintenance of MAP throughout post-resuscitative care
- Recognition of parameters for re-introducing hypothermia protocol with intervening non-pulsatile states
- Parameters for re-dosing of Amidate in setting of increased MAP
- Hospital bypass criteria for patient with ROSC and Induced hypothermia protocol

Scenario #4

Rhythm Sequence	Algorithm
1. Asystole	Asystole
2. Ventricular Fibrillation	Ventricular Fibrillation (Witnessed)
3. Sinus Tachycardia/ Sinus Rhythm	Induced Hypothermia Protocol

A 44-YEAR-OLD 90KG (198 LB) MALE COLLAPSED WHILE CUTTING THE GRASS. ON ARRIVAL TO SCENE, BYSTANDER CPR BEING PERFORMED. FAMILY STATES THE PATIENT HAS NO MEDICAL PROBLEMS, BUT HAD BEEN FEELING "RUN DOWN" LATELY. YOU ESTIMATE HIS DOWN TIME TO BE APPROXIMATELY 5-7 MINUTES.

EKG:	"ASYSTOLE"	Correct Diagnosis?	Yes	No
Scen. 5A	Safe Scene/ Universal Precautions			
	Checks for responsiveness			
	Evaluates and notes effectiveness of bystander CPR			
	Assesses patient – unresponsive, apneic with no pulse			
	Starts CPR			
	Utilizes BVM with airway adjunct			
	Attaches ResQPOD to facemask (maintains adequate face seal)			
	Applies Quick-Combo patches/ Hard Wire - Asystole			
	Has team member start IV (1000mL NS)			
	Verbalizes need for large bore catheter (16ga if possible)			
	NOTE: If no peripheral access found – Insert IO			
	Has team member intubate			
	Confirmation with PE, breath sounds, TubeChek, capnography			
	Attaches ResQPOD to ET tube – engages timing light for ventilation			
	Consider ATV (10-12bpm) at appropriate TV for patient weight			
	ET, LMA – (CID/C-Collar for continued airway control)			
	Vasopressin 40 Units IV			
	Circulate with 2 minutes of CPR			
	Epinepherine 1mg 1:10,000 IV			
	Circulate with 2 minutes of CPR			
	(Go to Page 2)			

Cont.	"ASYSTOLE"		Yes	No
	Atropine 1mg IV			
	Circulate with 2 minutes of CPR			
	Re-Assess Patient			
	Patient remains unresponsive, pulseless and apneic ***Asystole continues*** ***Emphasis on Differential Diagnosis (H's and T's)***			
	Epinepherine 1mg 1:10,000 IV			
	Circulate with 2 minutes of CPR			
	******Change in Rhythm noted on Monitor*****			

EKG:	"VENTRICULAR FIBRILLATION"	Correct Diagnosis?	Yes	No
Scen. 5B	Notes change in rhythm – Ventricular Fibrillation			
	Checks Patient			
	Patient remains unresponsive, pulseless and apneic			
	Resumes CPR (If delay in immediate defibrillation)			
	Checks Monitor			
	Patient in V-Fib, no pulse ***Considers pre-cordial thump (witnessed V-Fib)***			
	Clears area before defibrillating			
	Defibrillation (200J – Biphasic; 360J – Monophasic)			
	Has team members resume CPR			
	Evaluates effectiveness of CPR			
	Amiodarone 300mg IV			
	Circulate with 2 minutes of CPR			
	Clears area before defibrillating			
	Defibrillation (200J – Biphasic; 360J – Monophasic)			
	(Go to Page 3)			

Cont.	"VENTRICULAR FIBRILLATION"		Yes	No
	Has team members resume CPR			
	Evaluates effectiveness of CPR			
	Epinepherine 1mg 1:10,000 IV			
	Circulate with 2 minutes of CPR			
	Clears area before defibrillating			
	Defibrillation (200J – Biphasic; 360J – Monophasic)			
	******Notes Rhythm Change (Organized Electrical Activity)******			

EKG:	"SINUS TACHYCARDIA" - ROSC	Correct Diagnosis?	Yes	No
	Check for responsiveness and vital signs			
	Patient remains unresponsive with no appropriate response to pain ***P – 130, reg. (ROSC); BP – 130/80*** ***NO spontaneous respiratory effort*** ***EtC02 = 72mm Hg*** ***Removal of ResQPOD from respiratory circuit***			
	Continue supportive care for patient			
	Ventilation efforts addressed to achieve EtC02 – 40mm Hg ***Recognizes that patient qualifies for "Induced hypothermia" protocol***			
	12-Lead ECG (when possible during post-resuscitation care)			
	12-Lead reveals Sinus Tachycardia with widened QRS complex ***Injury/Ischemic pattern not distinguishable***			
	Obtains patient temperature (Tympanic measurement)			
	Assures patient temperature > 34C (93.2 F)			
	Re-evaluates effectiveness of ventilation with advanced airway			
	ET, LMA – (CID/C-Collar for continued airway control) ***Assures EtC02 value >20mm Hg***			
	(Go to Page 4)			

Cont.	"INDUCTION OF HYPOTHERMIA"	Yes	No
	Patient Neuro Examination		
	Assures NO or non-purposeful response to painful stimuli		
	Attempt 2nd IV/IO line (when possible)		
	Fentanyl 100mcg IV		
	Amidate 20mg IV		
	Vecuronium 0.1mg/kg IV (maximum 10mg)		
	Initiate cold saline bolus at 30mL/kg (maximum 2 Liters)		
	Initiate cold saline bolus through up to two (2) IV or IO access points		
Scen. 5C	******After 500mL chilled saline infused – ECG rate change noted with subsequent drop in BP******		
	Re-Assess Patient		
	Patient remains unresponsive with no appropriate response to pain ***P – 80; BP – 100/50 (MAP = 66)*** ***NO spontaneous respiratory effort*** ***EtC02 = 35mm Hg*** ***ECG – NSR with wide QRS complex***		
	Prepares for administration of Dopamine Drip		
	400mg/250cc D5W with 60gtt administration set ***Verbalizes target for MAP at 90-100 (Check MAP on LP12)***		
	Initiates Dopamine Drip (10mcg/kg/min)		
	Patient BP is at 100/50 = MAP of 66) ***(Pt body weight 90Kg = 900mcg/min = 34gtts/min)*** ***NOTE: Titrate Dopamine up to 20mcg/kg/min to achieve MAP of 90-100*** ***80 ≤ Target Diastolic ≤ 90*** ***Remember:*** ***Cold saline is a strong vasoconstrictor. Watch MAP closely.*** ***In the event your target MAP is met (90-100), stop Dopamine infusion*** ***[Discontinue Dopamine when diastolic pressure is ≥ 90 or MAP ≥ 100*** ***Re-Start Dopamine drip in the event the MAP falls below 90-100***		
	(Go to Page 5)		

Cont.	"NSR" – INDUCTION OF HYPOTHERMIA (CONT.)	Yes	No
Scen. 5D	***Induced cooling continues – Approximately 5 minutes after start of Dopamine Drip – Change in Patient Noted***		
	Re-Assess Patient		
	Patient remains unresponsive; *Eye and facial twitching noted* *P – 96, reg.* *BP – 140/90; (MAP = 106)* *NO spontaneous respiratory effort* *EtC02 = 35mm Hg*		
	Cessation of Dopamine Drip		
	Target MAP achieved		
	Re-Dose Amidate 20mg IV		
	Re-Dose Vecuronium (1/10th original dose)		
	Patient weight 90Kg (initial dose of Vecuronium should have been 9mg *Re-Dose at 0.9mg*		
	Re-Assess Patient		
Scen. 5E	*Change in vital signs noted* *Cessation of eye/facial twitching* *Patient remains unresponsive* *P – 88, reg.* *BP – 130/80* *NO respiratory effort*		
	Declares "I.C.E" Protocol with LCEMS Dispatch		
	Early notification when possible during resuscitative care *Bypass criteria for "Hypothermia" Centers (Toledo, SVMC, UTMC, St Lukes*		
	Hospital Arrival		
	Re-obtains patient temperature (tympanic measurement) *Transfer to ED staff for continued cooling measures*		

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    - Procedure for chilled saline administration
    - Maintenance of MAP throughout post-resuscitative care
- Parameters for re-dosing of Amidate in setting of increased MAP
- Parameters for re-dosing Vecuronium in the setting of patient movement
- Hospital bypass criteria for patient with ROSC and Induced hypothermia protocol