Practical Aspects Of Postcardiac Arrest Therapeutic Hypothermia

Abstract

Neurologically intact survival following cardiac arrest remains a challenge of modern medicine. Internationally, the rate for survival of out-of-hospital cardiac arrest has hovered at a disappointingly low 6%. Over the past decade, the early postcardiac arrest period has emerged as a critical window for therapeutic intervention to minimize cerebral injury and improve recovery. Therapeutic hypothermia is a cornerstone of this early postresuscitation care. Despite accumulating evidence and widespread endorsement, adoption of therapeutic hypothermia is incomplete. This review focuses on overcoming barriers to enable practical application of this life-saving treatment.

Note From The Editor-In-Chief

Readers of EM Critical Care will recall our 2012 issue by Rittenberger et al that emphasized the guiding principles of cardiocerebral resuscitation in the emergency department after cardiac arrest. In this issue, we will expand on the discussion of the use of therapeutic hypothermia after cardiac arrest and will address a number of the challenges and logistical considerations for implementing hypothermia. More than a decade after the landmark trials by Bernard et al and the Hypothermia After Cardiac Arrest (HACA) Study Group, there remain challenges with uptake, implementation, and inclusion/exclusion criteria for therapeutic hypothermia. This issue by Pearson and Heffner succinctly addresses the spectrum of questions and complexities that have evolved since therapeutic hypothermia’s powerful benefit was demonstrated. Enjoy! - Rob Arntfield

CME Objectives

Upon completion of this article, you should be able to:
1. Describe the indications and contraindications of therapeutic hypothermia in postcardiac arrest.
2. Describe the most effective strategies to implement therapeutic hypothermia in your institution.
3. Troubleshoot or prevent common complications of therapeutic cooling.

Prior to beginning this activity, see the back page for faculty disclosures and CME accreditation information.
Case Presentations

A 63-year-old male experienced a witnessed, out-of-hospital collapse. The patient's wife called 911 and initiated CPR. Ventricular fibrillation was recognized upon paramedic arrival, but it was resistant to initial biphasic countershock. ROSC was ultimately achieved 21 minutes following cardiac arrest. Hypotension (BP of 92/42 mm Hg, MAP of 59 mm Hg) and persistent coma (GCS score = 3) are noted upon arrival to the ED. The nurse asks you if this patient is a candidate for the therapeutic hypothermia protocol, despite his prolonged downtime.

A short time later, EMS brings in a 39-year-old woman with a history of asthma who called 911 for shortness of breath. When EMS arrived, the patient was in severe respiratory distress. During transport, her respiratory status worsened, and she became unresponsive. The monitor revealed a bradycardic idioventricular rhythm, and the patient was noted to be pulseless. ACLS was started by EMS, and ROSC was achieved following 10 minutes of resuscitation. The patient remains comatose upon arrival to the ED. The exam reveals symmetric diffuse expiratory wheezes, and her chest x-ray is negative for pneumothorax. You continue to aggressively treat the status asthmaticus and initiate consultation with the critical care team. You consider postcardiac arrest therapeutic hypothermia, but you wonder whether you should cool this patient following PEA arrest?

Introduction

Over 300,000 patients per year in the United States experience sudden cardiac arrest.\(^\text{1}\) Despite initial resuscitation, neurological injury sustained during cardiac arrest is the leading cause of death and contributes to the historic survival rate of only 6% to 8%.\(^\text{1,2}\) Additionally, 30% of survivors have permanent neurological injury.

Therapeutic hypothermia has emerged as the only effective intervention to ameliorate anoxic brain injury and improve neurological outcome.\(^\text{3}\) Despite strong evidence and endorsement by the American Heart Association, therapeutic hypothermia remains an underutilized modality, with implementation rates as low as 30% to 40%.\(^\text{4-8}\)

Perceived barriers to implementation of therapeutic hypothermia include lack of treatment awareness, premature negative prognostication, and perceived high workload demands.\(^\text{9,10}\) Regardless of the reason, failure to implement this evidence-based therapy deems its ineffectiveness absolute.

This review focuses on the practical aspects of implementing therapeutic hypothermia for postcardiac arrest patients, regardless of practice locale, and highlights the key resuscitation adjuncts in the immediate and early postresuscitation phase of illness that provide the greatest opportunity to achieve the goal of neurologically intact survival of cardiac arrest victims.

Critical Appraisal Of The Literature

The PubMed database was searched for articles published from 1950 to August 2012. The terms used in the search included English publications containing 1 or more of the following key words: therapeutic hypothermia, induced hypothermia, cardiopulmonary resuscitation, and cardiac arrest. This search revealed over 440 publications. The results were reviewed, and relevant citations from each study were searched manually. An Internet search for practice guidelines and clinical policies identified the following:

- American Heart Association: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations
- Canadian Association of Emergency Physicians, Critical Care Committee: The Use of Hypothermia After Cardiac Arrest
- Scandinavian Society of Anesthesiology and Intensive Care Medicine: Task Force on Scandinavian Therapeutic Hypothermia Guidelines, Clinical Practice Committee
- The Hong Kong Society of Critical Care Medicine Position Statement: The Use of Hypothermia After Cardiac Arrest

Efficacy Of Therapeutic Cooling

Evidence For Cooling Patients With Ventricular Tachycardia And Ventricular Fibrillation

Therapeutic hypothermia is the only neuroprotective therapy that demonstrates neurological and survival benefit following cardiac arrest.\(^\text{11-13}\) A decade of supportive investigation culminated in completion of 2 multicenter randomized trials of therapeutic hypothermia in 2002. Both trials enrolled comatose survivors of out-of-hospital cardiac arrest with a first recognized rhythm of ventricular tachycardia and ventricular fibrillation (VT/VF).\(^\text{11,12}\) Nonshockable rhythms were excluded to avoid experiment confounding. Both trials showed improved survival with good neurological outcome. (See Table 1.) The Hypothermia After Cardiac Arrest (HACA) study defined a Cerebral Performance Category (CPC) score of 1 (patient is alert with normal cerebral function) or 2 (patient is alert and has sufficient cerebral function to live independently and work part time) as a good neurological outcome. The Bernard study defined a “good” outcome as normal or minimal disability (able to care for self; discharged directly to home). A powerful treatment impact was demonstrated with 1 survivor, with good neurological outcome achieved for every 6 patients treated (ie, the number needed
Evidence For Cooling Patients With An Initial Rhythm Other Than Ventricular Tachycardia And Ventricular Fibrillation

One challenge to improving outcomes after cardiac arrest is the epidemiologic trend of patients presenting with nonshockable rhythms, which is independently associated with worse prognosis. Whether or not patients with nonshockable primary arrest rhythms (pulseless electrical activity [PEA] and asystole) are eligible for therapeutic hypothermia remains controversial. To date, no randomized trials have focused on therapeutic hypothermia for these patients. Champions for this group highlight strong biologic plausibility and irrelevance (to the brain) of the nonperfusing rhythm that initiated the injury. Observational evidence regarding therapeutic hypothermia in this patient group is conflicting.

We believe that defining cardiac arrest patients by primary arrest rhythm is an overly simplistic approach that does little to discriminate individual patient acuity and physiology. Regardless of the rhythm, once ROSC is achieved, neurological outcome—not the illness precipitating cardiac arrest—is the primary limitation for patient recovery and survival. In contrast to the strict criteria we would expect of clinical trials, real-world implementation should aim to exclude fewer potential candidates for cooling, given the low risk and low cost of this treatment and the absence of an alternative postarrest treatment that is proven to improve survival. Multiple guidelines support application of cooling for all comatose cardiac arrest victims regardless of the initial rhythm or arrest precipitant. These recommendations were based primarily on observational trials showing benefit of cooling following out-of-hospital cardiac arrest regardless of initial rhythm. Detractors of such a strategy point to the absence of a randomized trial supporting use in this group. However, the heterogeneity of patient and arrest factors resulting in a first-identified rhythm of PEA and asystole make this a challenging subset of patients to study, and future trials risk the ethical dilemma of randomizing patients to normothermia in light of our current evidence.

Many institutions with therapeutic hypothermia protocols cool patients regardless of arrest rhythm. This affords the postarrest patient the best chance at neurological recovery with minimal risk. Therapeutic hypothermia represents a cost-effective resource that should be deemed part of comprehensive postresuscitative cardiac arrest care; thus, we encourage cooling regardless of initial arrest rhythm.

Evidence For Cooling Patients With Nonprimary Cardiac Events

Death due to brain injury represents the final common pathway of patients experiencing anoxic or hypoxic injury, regardless of the inciting event. Thus, the best available evidence supports extrapolation of therapeutic hypothermia to patients experiencing nondysrhythmogenic arrest, including patients with asphyxia or traumatic nonexsanguinating arrest. (See Table 2.) Neuroprotective cooling should not be withheld due to the lack of an “ideal” candidate, as the potential neurological benefit of cooling outweighs the potential risk of hypothermia in these patients. Neurological recovery is the primary limiting factor to a positive outcome in most of these circumstances.

Inclusion And Exclusion Criteria For Therapeutic Cooling

Institutions have slight variations of criteria for inclusion and exclusion to their postcardiac arrest care bundle. Our institution’s criteria are described in Tables 3 and 4 (page 4). In this protocol, cardiac arrest patients are cooled regardless of initial rhythm or arrest location (in-hospital or out-of-hospital). Patients with severe or terminal illness and those un-

Table 1. Survival After Cardiac Arrest With Good Neurological Outcome In Landmark Trials

<table>
<thead>
<tr>
<th></th>
<th>Hypothermia</th>
<th>Control</th>
<th>P</th>
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<tbody>
<tr>
<td>HACA</td>
<td>75/136 (55%)</td>
<td>54/137  (39%)</td>
<td>.009</td>
</tr>
<tr>
<td>Bernard et al</td>
<td>21/43 (49%)</td>
<td>9/34 (28%)</td>
<td>.046</td>
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Abbreviation: HACA, Hypothermia After Cardiac Arrest trial.
likely to survive the intensive care unit (ICU) based on comorbid disease (irrespective of cardiac arrest) are poor candidates for cooling.

The relative contraindications of therapeutic hypothermia (listed in Table 4) are discussed below.

Age: The sentinel trials focused on adults, but therapeutic hypothermia also improves cerebral outcomes following perinatal hypoxia. Although a gap in the evidence for pediatric patients remains,\textsuperscript{29,30} selected use in children is endorsed and widely adopted.\textsuperscript{4}

Pregnancy: Successful therapeutic hypothermia of pregnant patients, although a rare circumstance, emphasizes the favorable risk-reward ratio of therapy in this situation.\textsuperscript{31-32}

Hemodynamic instability: Hemodynamic instability and postcardiac arrest shock are common. Postcardiac arrest shock complicates management of half of all comatose patients and is directly associated with the length of cardiac arrest. Although patients in shock were excluded from early trials of therapeutic hypothermia, accumulating evidence supports the safety and efficacy of therapeutic hypothermia among patients with serious hemodynamic instability, including those requiring catecholamine support and intra-aortic balloon pump for severe cardiogenic shock.\textsuperscript{33} As such, shock is no longer considered a contraindication to therapeutic hypothermia induction. New evidence also shows that therapeutic hypothermia may be associated with a reduced need for vasopressor and inotropic support in patients with cardiogenic shock.\textsuperscript{34,35}

<table>
<thead>
<tr>
<th>Table 3. Inclusion Criteria For Postcardiac Arrest Therapeutic Hypothermia</th>
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<tbody>
<tr>
<td>• ROSC &lt; 60 min after cardiac arrest</td>
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<tr>
<td>• Inhospital or out-of-hospital cardiac arrest</td>
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<tr>
<td>• Time at induction &lt; 6 h from ROSC</td>
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<tr>
<td>• Coma (GCS score ≤ 8)</td>
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<td>• Patient intubated and mechanically ventilated</td>
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Abbreviations: GCS, Glasgow Coma Scale; ROSC, return of spontaneous circulation.

<table>
<thead>
<tr>
<th>Table 4. Exclusion Criteria For Postcardiac Arrest Therapeutic Hypothermia</th>
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Absolute Contraindications
• Severe terminal illness or Do Not Resuscitate (DNR) / Do Not Intubate (DNI) order

Relative Contraindications
• Age < 18 y
• Pregnancy
• Hemodynamic instability
• Traumatic arrest
• Severe systemic infection
• Clinical bleeding

| Traumatic arrest: Traumatic arrest is often listed as a contraindication to cooling due to the concern for hypothermia-induced coagulopathy in the setting of life-threatening hemorrhage. However, cardiac arrest is a frequent precipitant of trauma. A fall or motor vehicle collision precipitated by cardiac arrest is a common scenario, so ascertaining historical events accurately is imperative. Patients who have nonhemorrhage-related traumatic hypoxia or arrest should also be considered for therapeutic hypothermia. Some centers are utilizing therapeutic hypothermia after gaining full control of the bleeding source in patients experiencing traumatic arrest from hemorrhagic shock; however, little is known about the application of therapeutic hypothermia in this setting.\textsuperscript{36}

Systemic Infection: Hypothermia-associated immune suppression warrants weighing the perceived risk and benefit of cooling patients who have cardiac arrest due to sepsis.\textsuperscript{37} Acute aspiration pneumonia should not be considered a contraindication to hypothermia.

Bleeding: Since hypothermia induces coagulopathy, active bleeding is a relative contraindication. Therapeutic hypothermia induces platelet dysfunction at temperatures ≤ 35°C and inhibition of the coagulation cascade at temperatures ≤ 33°C.\textsuperscript{38} Thus, some experts advocate for cooling at a slightly higher temperature range of 34.5°C to 35°C in patients with suspected or active bleeding.\textsuperscript{39} Additionally, cooling is under investigation to help treat traumatic and vascular cerebral hemorrhage. Patients ineligible for therapeutic cooling warrant active measures to avoid post-ROSC hyperthermia due to its potential to exacerbate brain injury.\textsuperscript{40} Moderate-to-severe accidental or spontaneous hypothermia associated with cardiac arrest warrants rewarming to the goal mild hypothermia temperature range (32°C-34°C).

Timing Of Therapeutic Cooling

In addition to the primary ischemic insult during no-flow and low-flow cardiac arrest, a second insult of cerebral reperfusion injury evolves over hours to days.\textsuperscript{31,42} This window affords an opportunity for initiating targeted neuroprotective therapies to mitigate the reperfusion insult and improve neurological outcome.

Prehospital Cooling

Animal studies demonstrate that cooling that is initiated early results in improved survival and neurological outcome, while delays negate the beneficial effect.\textsuperscript{43-47} The potential benefit of early therapy has led to prehospital adoption of cooling. This provides the earliest possible initiation for most patients, and it is most commonly achieved with ice packs or cold intravenous (IV) fluids. A prospective study
of 142 patients documented a 20% increased risk of death for every hour of delay in cooling initiation.\textsuperscript{48} Additionally, a retrospective study of 172 patients revealed increased odds of poor neurological outcome with each 5-minute delay in the initiation of cooling.\textsuperscript{49} Garrett et al evaluated 551 patients after cardiac arrest and showed that prehospital intra-arrest cooling via 4°C normal saline was associated with improved ROSC (36.5%) when compared to normothermia resuscitation (26.9%).\textsuperscript{50}

In contrast, 3 prospective randomized trials showed no difference in neurological outcome between the prehospital and in-hospital cooling groups, despite the prehospital group reaching the target temperature more rapidly.\textsuperscript{51-53}

The optimal timing of the initiation of cooling to maximize benefit remains unclear. However, the general consensus is that early cooling should be prioritized as a part of resuscitation.

**Cooling In The Emergency Department**

It is important to note that prehospital cooling does not mandate continued therapy, since many prehospital protocols are designed to ensure consistent and wide implementation. Upon arrival to the emergency department (ED), patients should be individually evaluated for candidacy for therapeutic hypothermia based on institutional guidelines and the potential risk and benefit of continued treatment. If a patient is deemed inappropriate for cooling, gradual rewarming is advised due to the potential for deleterious electrolyte shifts, hemodynamic instability, and cerebral injury associated with rapid rewarming.\textsuperscript{54,55}

The landmark trials for therapeutic hypothermia reached the goal temperature in a mean of 2 to 8 hours.\textsuperscript{11,12} One approach for treating cardiac arrest patients with therapeutic cooling would be to achieve a target temperature of 32°C-34°C in 6 hours, with the goal to cool as rapidly as possible. However, cooling may offer benefit up to 24 hours after cardiac arrest (and possibly beyond), so delayed initiation of cooling or delays in reaching target temperature does not necessarily diminish the potential benefit. Given the potential neurological and cardiac benefit of early initiation of cooling, we advocate for the earliest possible induction time. Additionally, cooling should be considered in all cardiac arrest patients, even in the setting of delayed induction.

**Avoiding Delays**

For in-hospital arrests (ED or hospital inpatients), supplies to allow rapid initiation cooling (ie, prechilled 4°C IV fluids) should be readily available; we recommend maintaining bags of normal saline in refrigerators that are easily accessible to the ED or ICU.

Extensive delays in the initiation of cooling due to concerns over patient candidacy should be avoided. Since many commercially available devices have nonreusable and costly equipment (ie, catheters or surface pads costing thousands of dollars), cold IV fluids and ice packs are effective initial measures while exploring candidacy of individual patients.

**Transfer**

Postcardiac arrest interventions are time sensitive, so early initiation of treatment is essential. Facilities that do not have percutaneous coronary intervention (PCI) and therapeutic hypothermia capabilities are encouraged to initiate therapeutic cooling prior to transfer. Induction of cooling can be achieved with cold IV fluid infusion and surface ice packs applied to the neck, groin, and axillae. Proactive development of clinical pathway criteria in conjunction with the development of protocols for referral to a cardiac arrest center will help ensure smooth transitions of care.

**Techniques For Therapeutic Cooling**

Therapeutic cooling is divided into 4 phases. These include: (1) induction; (2) maintenance; (3) rewarming; and (4) controlled normothermia. In the ED, the induction phase of therapeutic cooling will be the primary focus. This may represent initiation of the cooling process or continuing the cooling efforts started by prehospital providers. The maintenance, rewarming, and controlled normothermia phases will primarily be done in the ICU, but it is important to understand the clinical caveats of these phases, as it may be necessary to maintain or rewarm the patient in the ED at times.

**Cooling Induction**

Induction of therapeutic hypothermia with cold (4°C) isotonic fluid is safe, inexpensive, and effective.\textsuperscript{56,57} Application of surface ice packs augments induction and may be required for patients who are intolerant of volume loading. Two benefits of cold fluids and ice packs are their low cost and their ease of use (including in the prehospital setting). The concomitant use of cold IV fluids, ice packs, and servo-controlled automated cooling devices for rapid cooling is helpful. Overshooting the goal temperature range (< 32°C) to a point that it poses significant risk is rare and should not make the provider hesitate to be aggressive with cooling measures during the induction phase.

Reliable core temperature measurement is an important element for monitoring, and it provides essential feedback for the servo-controlled automated cooling systems. Core temperature measurement should be initiated upon arrival to the ED. Bladder or esophageal temperature sensors are most commonly used for core temperature monitoring. Newer temperature-sensing Foley catheters do not require urinary flow; instead, they sense bladder outlet temperature. However, esophageal temperature...
monitoring is preferred because bladder and rectal temperature measurements more slowly reflect core temperature during rapid cooling. Oral or axillary temperature measurements are not recommended, as they may not reflect core temperature adequately, especially when used in patients experiencing rapid temperature changes.\(^{38,59}\)

Sedation and analgesia should be employed for all comatose postarrest patients in order to attenuate adrenergic response, hypermetabolism, and shivering during cooling.\(^{60}\) Adjunctive measures to control shivering are often employed; these measures are discussed in the Troubleshooting Therapeutic Cooling section of this article.

Commercially available cooling systems utilize either external (noninvasive) or internal (endovascular) devices to complete the induction phase and transition to the maintenance phase.\(^{61}\) (See Table 5.) There are important logistical issues to consider regarding setup and use for both invasive and noninvasive cooling devices, and when compared to each other, neither is superior with regard to survival or neurological outcomes.\(^{62-64}\) New devices continue to emerge for rapid induction. The RhinoChill\(^{®}\) (Benechill, Inc., San Diego, CA) delivers a coolant to the nasopharyngeal passages, whereas the Thermosuit\(^{®}\) (Life Recovery Systems HD, LLC, Kinnelon, NJ) uses cold-water immersion to induce rapid hypothermia.\(^{33,65}\)

**Cooling Maintenance**

Therapeutic cooling for 12 to 24 hours at a goal temperature of 32°C to 34°C is recommended based on the protocols of the landmark trials. The optimal depth and period of cooling is currently undergoing investigation. Surface cooling with commercially available water-circulating blankets or gel pads and intravascular cooling appear to be more effective than conventional cooling (eg, ice packs and cold fluids) and air-circulating blankets.\(^{66}\) These new-generation devices provide automated control to prevent temperature fluctuations and to facilitate slow, controlled rewarming (< 0.5°C/h). There is no clear advantage between invasive and noninvasive units. Cost is comparable among the commercially available surface devices and intravascular cooling devices. Selection of a cooling strategy should involve the multidisciplinary team caring for these patients.\(^{52,64}\)

**Rewarming**

Early rewarming prior to completing the hypothermia treatment time period is rare. In such cases, slow controlled rewarming (0.3°C/h to 0.5°C/h) to a safe or normal core body temperature is recommended.\(^{39}\)

The induction and rewarming phases tend to be higher-risk periods for the complications of therapeutic hypothermia, so they require judicious monitoring for dysrhythmia, potassium shifts, and hemodynamic instability. Warming-associated vasodilation can lead to hypotension, requiring fluid and/or vasopressor support. Rapid rewarming causes potassium to move extracellularly, which risks hyperkalemia.\(^{38}\) However, we caution against aggressive correction of potassium during rewarming. Low-dose repletion for potassium < 3.5 mEq/L, with frequent electrolyte reanalysis, is recommended. Equally important, cerebral injury has been associated with rapid rewarming and is the basis for slow, controlled rewarming rates.\(^{54,55}\)

**Controlled Normothermia**

The brain remains vulnerable to heat stress for several days following initial injury. Hyperthermia after warming is common and can lead to neurological deterioration. If feasible, keep the cooling device in place to maintain normothermia after completing cooling. This fourth phase of controlled normothermia is often continued for 2 to 3 days after rewarming.

**Additional Considerations For Therapeutic Cooling**

**Troubleshooting Therapeutic Cooling**

The most common challenge during the induction phase of therapeutic hypothermia is failure to reach goal temperature in a timely fashion. Unrecognized shivering plays a significant role. Shivering is often difficult to discern on examination. Failure to reach or maintain goal temperature may be an impor-

**Table 5. Cooling Methods And Devices**

<table>
<thead>
<tr>
<th>Noninvasive Cooling Devices</th>
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<tbody>
<tr>
<td>Arctic Sun(^{®}) 2000 and 5000 (by CR Bard, formerly Medivance Inc., Louisville, CO)</td>
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<tr>
<td>Stryker (formerly Gaymar) Medi-Therm(^{®}) III (Stryker, Orchard Park, NY)</td>
</tr>
<tr>
<td>Phillips InnerCool STX Surface Pad System (Phillips Healthcare, Andover, MA)</td>
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<tr>
<td>Blanketrol(^{®}) III Body Temperature Regulation System (Cincinnati Sub-Zero Products, Inc, Cincinnati, OH)</td>
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<th>Invasive Cooling Devices</th>
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<tr>
<td>Philips InnerCool RTx Endovascular System, Accutrol(^{®}) catheter (Phillips Healthcare, Andover, MA)</td>
</tr>
<tr>
<td>ZOLL (formerly Alsius) Intravascular Temperature Management (IVTM(^{™})) system CoolGard 3000(^{®}) device &amp; Cool Line(^{®}), ICY(^{®}), and Quattro(^{®}) catheters (ZOLL Medical Corporation, Chelmsford, MA)</td>
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<tr>
<th>Cooling Adjuncts</th>
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<tbody>
<tr>
<td>Ice packs and cold saline (4°C normal saline is most commonly used)</td>
</tr>
<tr>
<td>EMCOOLS, refrigerated surface pads (EMCOOLS, Vienna, Austria)</td>
</tr>
<tr>
<td>Thermosuit(^{®}) (Life Recovery Systems HD, LLC, Kinnelon, NJ)</td>
</tr>
<tr>
<td>RhinoChill(^{®}) intranasal cooling system (Benechill, Inc., San Diego, CA) (At the time of publication, this device was not yet available for sale in the United States.)</td>
</tr>
</tbody>
</table>
Complications Of Therapeutic Cooling

The unique physiological changes of therapeutic cooling can occasionally lead to complications. (See Table 6.) In this section, the hypothermic response on the cardiovascular, immune, hematologic, metabolic, and endocrine systems will be reviewed.

Sinus bradycardia, typically in the range of 40 to 50 beats/min, is expected, and it indicates that the patient undergoing therapeutic hypothermia is being properly managed. If sinus bradycardia is not seen, inadequate sedation or incomplete hemodynamic resuscitation may be the cause. Rarely, severe bradycardia resulting in hypoperfusion during cooling will warrant mild elevation of the target temperature. Concurrent use of negative chronotropes such as beta blockers and amiodarone (Cordarone®), Pacerone®) should be avoided unless strongly indicated.

Hypothermia suppresses immune system function; however, increased infection rates and sepsis in postarrest patients undergoing therapeutic hypothermia have not been observed. Regardless, postarrest patients have a notable risk of pulmonary infection due to a high incidence of aspiration. Temperature control obscures fever as a marker of infection, which has been linked to delay in antibiotic administration. Thus, early empiric antibiotics are frequently employed for patients with evidence of aspiration (ie, pulmonary secretions, radiographic infiltrates). Changes in the water temperature of the cooling bath within the commercial cooling device can act as a surrogate marker for fever and should be routinely monitored.

Coagulopathy is a known complication of therapeutic cooling, but it rarely results in spontaneous bleeding. Conventional anticoagulant medications should be used when indicated. Additionally, thrombolytic therapy for pulmonary embolism and STEMI should be used when necessary, despite the anticipated need for cooling. Significant temperature-related coagulopathy is unusual above 33.5°C. Patients with severe coagulopathy or suspected bleeding may be preferentially maintained above this threshold. Additionally, subcutaneous desmopressin (DDAVP®) reverses hypothermia-induced platelet dysfunction, and it should be considered as a therapeutic adjunct for patients with active bleeding.

Life-threatening bleeding not amenable to surgical hemostasis warrants rewarming.

Hyperglycemia is common following cardiac arrest. Decreased insulin secretion and sensitivity occur with hypothermia and may exacerbate hyperglycemia. IV insulin infusion is recommended to maintain moderate glucose control (< 180 mg/dL). Complications may also occur due to the cooling devices used. The use of invasive cooling devices may result in an increased risk of bleeding, infection, vascular puncture, and venous thrombosis. In contrast, surface cooling devices, especially if placed incorrectly, may lead to skin breakdown. Evidence is limited with regard to device-specific complications.

Therapeutic cooling may impact lactate production and diminish clearance. Serum lactate is increasingly used as a prognostic marker and resuscitation endpoint in acute critical illness. The expected kinetics of lactate in patients undergoing therapeutic cooling following cardiac arrest remains to be clarified.

Early Postarrest Prognostication

Prognostication of neurological outcome in the ED remains a challenge. No physician desires a futile resuscitation; however, premature negative prognostication remains an obstacle to optimal care. There is no evidence to support accurate early neurological prognostication. Pre-arrest and intra-arrest parameters such as duration of arrest, bystander CPR, or presenting rhythm are unreliable prognosticators alone or in combination. For reference, the median

Table 6. Potential Adverse Physiologic Responses And Complications Associated With Cooling

| • Coagulopathy and thrombocytopenia |
| • Cold diuresis |
| • Dysrhythmias (sinus bradycardia during cooling is most common) |
| • Electrolyte abnormalities (K, Mg, Phos) |
| • Hypotension (including vasodilation with rewarming) |
| • Infection (and masked signs of infection) |
| • Insulin resistance and hyperglycemia |
| • Pancreatitis |
| • Shivering |
## Clinical Pathway: Adult Patients After Cardiac Arrest With Return Of Spontaneous Circulation

**Induction:**
- Infuse cold IV fluids.
- Administer NS 30 cc/kg IV bolus as tolerated.
- Apply ice packs to groin, axilla, and neck.
- Maintain early sedation and paralysis until goal temperature is achieved.
- Initiate cooling device for 33°C.

**Goals:**
- **Cooling goals:**
  - Achieve 32°C-34°C (Class I) in 60 min.
- **Hemodynamic and ventilatory goals:**
  - Maintain MAP ≥ 70 mm Hg (the preferred vasopressor is norepinephrine).
  - Minimize FiO₂, while maintaining oxygen saturation > 95%.
  - Maintain PaCO₂ 38-42 mm Hg.

**Critical care considerations:**
- Central venous catheter to assess CVP & ScvO₂ (avoid left subclavian catheter, as this may be the preferred site for a pacemaker/defibrillator).
- Echocardiogram to assess myocardial function.
- Arterial monitoring line.
- Glucose control.
- Seizure control.
- Evaluate and address etiology of arrest.

**Maintenance:**
- Goal temperature of 32°C-34°C.
- Continue cooling for 24 h.
- Assess for shivering: short-acting analgesia or sedation (paralytic agents if shivering uncontrolled with these measures).
- Assess electrolytes.
- Monitor fluid status: input & output.

**Rewarming:**
- Rewarm approximately 0.5°C/h. (Class I)
- Assess electrolytes closely.
- Assess neurological status once at normothermia.

**Controlled normothermia:**
- Keep the cooling device in place for 2 to 3 days after rewarming to maintain normothermia (37°C).

**Abbreviations:**
- CVP, central venous pressure; FiO₂, fraction of inspired oxygen; IV, intravenous; MAP, mean arterial pressure; NS, normal saline; PaCO₂, partial pressure of carbon dioxide; PEA, pulseless electrical activity; PCI, percutaneous coronary intervention; ScvO₂, central venous oxygen saturation; VT/VF, ventricular tachycardia/ventricular fibrillation

### Class Of Evidence Definitions

Each action in the clinical pathways section of *EM Critical Care* receives a score based on the following definitions.

**Class I**
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

**Level of Evidence:**
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

**Class II**
- Safe, acceptable
- Probably useful

**Level of Evidence:**
- Generally higher levels of evidence
- Nonrandomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

**Class III**
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

**Level of Evidence:**
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

**Indeterminate**
- Continuing area of research
- No recommendations until further research

**Level of Evidence:**
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

downtime between collapse and ROSC for many of these patients is > 20 minutes. In a recent observational study of therapeutic hypothermia, 36% of patients with an arrest interval > 30 minutes experienced a good neurological outcome. Although advanced age generally worsens prognosis, 30% of patients > 75 years of age had good neurological outcome in the same series. The immediate postarrest neurological examination (including low Glasgow Coma Scale [GCS] score, poor motor examination, and absent brainstem reflexes) does not predict neurological outcome. Prediction of prognosis based on neurological examination becomes more reliable after at least 72 hours following resuscitation. Given these limitations, early neuroprotective measures should be strongly considered for all potentially eligible patients.

The Postcardiac Arrest Syndrome
A discussion involving the practical implementation of therapeutic hypothermia necessitates the contextual background of the postcardiac arrest syndrome. One cannot simply expect to cool a patient as an isolated therapy without addressing the multitude of other resuscitation therapies that define contemporary postcardiac care, including acute coronary interventions and goal-directed critical care. (See the Clinical Pathway.)

The postcardiac arrest syndrome is a unique multiorgan condition. The complex immunological cascade that ensues from reperfusion after a transient period of total body ischemia results in a systemic inflammatory response that mimics severe sepsis. Clinically, this is reflected in hemodynamic instability and early organ dysfunction. For ease of targeting the unique clinical aspects of the illness, the International Liaison Committee on Resuscitation categorizes the syndrome in the following way:

- Postcardiac arrest brain injury
- Postcardiac arrest myocardial dysfunction
- Systemic ischemia-reperfusion response
- Persistent precipitating pathology

By targeting these clinical components of the postarrest state with a comprehensive resuscitative protocol, the emergency physician can have the greatest impact on improving outcomes.

Therapeutic cooling is only one aspect of an effective, multipronged resuscitative protocol. The early postarrest period is an opportunity to address additional priorities for effective cardiocerebral resuscitation. Priorities of the postarrest period include stabilization of organ perfusion and oxygenation, identification and treatment of reversible causes of cardiac arrest, and initiation of neuroprotective therapy. As such, contemporary emergency care now emphasizes intensive support during this vulnerable and modifiable phase of illness. (See Tables 8 and 9 on page 10.) For a detailed review on the postcardiac arrest syndrome, see the October 2012 issue of EM Critical Care titled, “Postarrest Cardiocerebral Resuscitation: An Evidence-Based Review.”

Reperfusion Therapy For Postcardiac Arrest Patients
Acute coronary syndromes (ACS) are the most common cause of sudden cardiac arrest in adults. The rate of acute coronary occlusion in patients with ACS is estimated to be 30% to 50%. Unfortunately, clinical history is often limited, and the electrocardiogram after ROSC can be nondiagnostic in discriminating acute coronary occlusion requiring emergent PCI. Revascularization is independently associated with survival and should be considered for all patients with strongly suspected acute coronary disease or electrocardiogram evidence of ST-segment elevation myocardial infarction (STEMI). PCI is the preferred method of revascularization and was safely performed concurrent with therapeutic cooling in 2 prospective observational studies. With appropriate coordination of cooling and PCI priorities, induction of hypothermia does not delay cardiac catheterization. PCI should not be delayed due to an uncertain neurological prognosis.

Thrombolysis is an acceptable reperfusion strategy for STEMI if PCI is not immediately available. Thrombolysis is associated with improved survival and neurological outcome, while the hemorrhagic risk is comparable to patients not thrombolysed. Coadministration with therapeutic cooling does not appear to increase the risk of significant bleeding.

Table 7. Postcardiac Arrest Syndrome Pathophysiology

<table>
<thead>
<tr>
<th>Acute Myocardial Dysfunction</th>
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<tbody>
<tr>
<td>Acute coronary syndromes</td>
</tr>
<tr>
<td>Myocardial stunning</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Brain Injury</th>
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<tbody>
<tr>
<td>Anoxic brain insult</td>
</tr>
<tr>
<td>Impaired autoregulation</td>
</tr>
<tr>
<td>Ischemic reperfusion injury</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Persistent Precipitating Pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decompensated cardiomyopathy</td>
</tr>
<tr>
<td>Myocardial ischemia</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Systemic Ischemia-Reperfusion Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dysregulated coagulation</td>
</tr>
<tr>
<td>Early organ dysfunction</td>
</tr>
<tr>
<td>Impaired microvascular function</td>
</tr>
<tr>
<td>Inappropriate vasodilation</td>
</tr>
<tr>
<td>Systemic inflammatory response</td>
</tr>
</tbody>
</table>
Adjunctive aspirin and heparin are recommended for suspected or confirmed ACS. Withholding acute beta-blocker and angiotensin-converting enzyme (ACE)-inhibitor therapy should be considered due to the potential for hemodynamic deterioration and shock in postarrest patients.

Based on a similar mechanism as neuroprotective benefit, cardioprotective effects of hypothermia are under exploration. Animal models show benefit with early cooling. Early trials in noncardiac-arrest patients with STEMI who received hypothermia prior to PCI showed decreased infarct size, without significant treatment delay.

**Summary Of Controversies In Therapeutic Cooling**

While therapeutic hypothermia has recognized value, there remain controversies in regard to timing and techniques; these issues are summarized below. Additional controversy surrounds the effectiveness of prehospital cooling and the role of therapeutic hypothermia in patients experiencing PEA of asystolic arrests; well-designed prospective studies with adequate power are needed in order to resolve these debates.

**Devices for cooling**: There continues to be debate over the best way to cool (invasive or non-invasive). Studies demonstrate improved temperature control with invasive devices compared to older generation noninvasive cooling pads. Comparison of newer hydrocolloid gel-pad-based surface cooling devices and endovascular cooling devices is limited. What is key to remember is that the execution of a cooling strategy to ensure that

- Provide adequate oxygenation and ventilation.
  - Avoid hyperventilation (goal PaCO$_2$ of 38-42 mm Hg).* 
  - Avoid hyperoxia (titrate FiO$_2$ to SpO$_2$ > 95% [or PaO$_2$ > 70 mm Hg]).* 
- Reverse shock and stabilize hemodynamics.
- Identify and treat reversible cause of cardiac arrest.
- Apply neuroprotective therapies (ie, therapeutic hypothermia).
- Correct metabolic disturbances.

*The solubility of gases (ie, PaCO$_2$ and PaO$_2$) changes as a function of temperature. At goal temperature, a patient's PaCO$_2$ may be up to 8 mm Hg lower than reported on the blood gas warmed to normal temperature. Unfortunately, the clinical impact of temperature correction via pH-stat methodology is unclear. In the absence of clear data, targeting high-normal PaCO$_2$ may avoid unintended cerebral vasconstrictive stemming from arterial hypcapnea.

Abbreviations: FiO$_2$, fraction of inspired oxygen; PaCO$_2$, partial pressure of carbon dioxide; PaO$_2$, partial pressure of oxygen; SpO$_2$, oxygen saturation by pulse oximeter.

patients receive this neuroprotective therapy is most important.

**Rate of cooling**: Another challenge involves identifying the ideal rate of cooling, with several studies showing that patients who experienced a faster rate of cooling had a less favorable outcome. One hypothesis to explain this is that more severely brain-injured patients may lose their ability to thermoregulate, and, thus, they cool more quickly. However, Mooney et al found no association between outcomes and time to goal temperature among 140 postcardiac arrest patients. Nonetheless, given that animal studies show improved outcomes with faster cooling, rapid cooling should remain the goal until contradictory evidence is unveiled.

**Initiation of cooling**: Additionally, the ideal time to initiate cooling remains controversial. Three prospective randomized trials showed no difference between cooling initiated prehospital or in hospital, despite reaching target temperature more rapidly. Conversely, Mooney et al found that every hour of delay in the initiation of cooling led to an increase in mortality of 20% per hour. Another study revealed increased odds of poor neurological outcome with each 5-minute delay in initiation of cooling.

**Table 9. Early Hemodynamic Resuscitation Goals Following Cardiac Arrest**

<table>
<thead>
<tr>
<th>Resuscitation Priority</th>
<th>Monitor and Goal</th>
<th>Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Preload optimization</td>
<td>Response to fluid challenge: fluid therapy is guided by:</td>
<td>Fluids</td>
</tr>
<tr>
<td></td>
<td>• CVP: 8-12 mm Hg</td>
<td>Norepinephrine*</td>
</tr>
<tr>
<td></td>
<td>• Echocardiogram: cardiac function and IVC variation</td>
<td>Epinephrine</td>
</tr>
<tr>
<td></td>
<td>• Markers of SVV</td>
<td>Vasopressin</td>
</tr>
<tr>
<td>2. Perfusion pressure</td>
<td>MAP 65-100 mm Hg</td>
<td>Dobutamine</td>
</tr>
<tr>
<td>3. Perfusion optimization</td>
<td>Global perfusion markers:</td>
<td>Milrinone</td>
</tr>
<tr>
<td></td>
<td>• ScvO$_2$ &gt; 70%</td>
<td>IABP</td>
</tr>
<tr>
<td></td>
<td>• Lactate clearance Clinical perfusion markers:</td>
<td>PRBC transfusion</td>
</tr>
<tr>
<td></td>
<td>• Urine output &gt; 0.5 cc/kg/h</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Peripheral skin perfusion</td>
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</tbody>
</table>

*First-choice vasopressor

Abbreviations: CVP, central venous pressure; IABP, intra-aortic balloon pump; IVC, inferior vena cava; MAP, mean arterial pressure; ScvO$_2$, central venous oxygenation saturation; IABP; intra-aortic balloon pump; PRBC, packed red blood cells; SVV, stroke volume variation.
**Duration of cooling**: Finally, the optimal duration of cooling has yet to be determined. In the landmark cooling trials, the period of cooling once at goal temperature was 12 to 24 hours. Based on these recommendations, current American Heart Association guidelines recommend cooling between 12 to 24 hours; however, it is unclear whether extended periods of cooling offer additional neuroprotective benefit.

**Disposition**

The disposition of postcardiac arrest patients depends on whether the facility has comprehensive postcardiac arrest care capabilities. If your hospital already has therapeutic hypothermia capability but does not have a protocol in place, your goal is aggressive resuscitation that focuses on initiation of therapeutic hypothermia and hemodynamic optimization while considering and addressing the possible causes of the arrest. If your facility has limited resources or infrequently cares for postcardiac arrest patients, a transfer protocol that includes cooling initiation is recommended.

**Regionalization Of Postarrest Care**

Many obstacles make the comprehensive management of a successfully resuscitated cardiac arrest patient challenging. An important consideration prior to establishing a hypothermia protocol is to assess your hospital resources and volume of cardiac arrest patients. The relative infrequency of applying the intervention, equipment costs, emergency nursing resource allocation, and the multidisciplinary specialized team required all make a postcardiac arrest care bundle difficult to implement effectively. Effective management of the unique aspects of the postcardiac arrest syndrome, beyond therapeutic hypothermia, must be considered. There exists a well-defined relationship between increased patient volume or procedures and improved outcomes for other complex, time-sensitive disorders. Higher-volume centers treating patients after cardiac arrest are associated with improved survival. Regional systems of care have been successfully established for STEMI, trauma, burns, and stroke, and the rationale for regional systems of care for postcardiac arrest patients is analogous to the need for these centers.

The American Heart Association recommends transporting postarrest patients to a comprehensive postcardiac arrest treatment system that is capable of acute coronary interventions, goal-directed critical care, and therapeutic hypothermia. No external certification for designation of a cardiac arrest center currently exists. These regional centers should not be restricted to academic medical centers. Academic and community-receiving hospitals with adequate patient volume, multidisciplinary care, and resources for postarrest patients will need to share in this endeavor. Additionally, essential systems of care include a supportive community, protocol-driven emergency medical services, and transferring hospitals.

At present, several prehospital systems (eg, Charlotte, New York City) prioritize out-of-hospital cardiac arrest patients to designated cardiac arrest centers despite the availability of more proximate facilities. The challenge lies when one considers catchment size and regions with longer transport times, resource allocation for prehospital systems with limited ambulances and personnel, and the fact that not all cardiac arrest patients are good candidates for aggressive care. In our opinion, the best means to address these challenges is via predetermined treatment protocols that take the unique aspects of each prehospital system and regional resources into consideration. Although exploration of the ideal regionalized system of care is just beginning, lengthy critical care transport is safe and feasible.

Some regions have a transfer-destination hospital model in order to provide a larger catchment area with access to comprehensive postarrest care. The Minneapolis Heart Institute receives patients from a network of 33 hospitals from a catchment area of 150 miles. Another example is our cardiac resuscitation center, which receives patients from many regional hospitals within a 100-mile radius via a postcardiac arrest care pathway known as CODE COOL. Transferring facilities have a transfer protocol whereby cooling is initiated with cold fluids and the patient is subsequently transferred to the cardiac resuscitation center for resuscitative care, including therapeutic hypothermia. Table 10 (page 12) shows the guidelines from the CODE COOL TM poster that is distributed to all outlying facilities to serve as a guide to ease the resuscitative and transfer process.

**Summary**

Therapeutic hypothermia is an effective therapy to improve chances of neurological recovery after cardiac arrest. Ensuring broader utilization of therapeutic cooling is essential to improving outcomes. Successful execution of therapeutic cooling must consider the hospital’s capabilities as well as the complexities of the postcardiac arrest syndrome. Design and implementation of a postcardiac arrest resuscitation protocol emphasizing hemodynamic resuscitation, neuroprotective cooling, and critical care support will enable centers to optimize cerebral recovery following cardiac arrest.
Risk Management Pitfalls For Therapeutic Hypothermia In The Emergency Department

1. Delaying initiation of cooling due to excessive imaging. Computed tomography head and chest imaging is frequently performed to assess for precipitants of arrest and early brain injury. Unfortunately, these tests provide uncertain benefit and frequently delay attention to therapeutic cooling. Focused attention to the details of the patient’s decompensation and arrest event, along with simple bedside tests (electrocardiogram, echocardiogram, blood analysis), are frequently sufficient to establish a short list of differential diagnoses. When cooling is indicated, imaging should not delay initiation of cooling.

2. Leaving the patient on 100% FiO₂. Too little oxygen is bad for the brain; however, too much oxygen may also cause harm. Hyperoxia is associated with increased mortality in postcardiac arrest patients. Rapidly titrate oxygen to 30% FiO₂ (as tolerated) to maintain oxygen saturation > 95%.

3. Assuming a poor prognosis. Arrest intervals > 20 minutes, postarrest GCS score of 3 to 4, and age > 70 do not equate to a dismal prognosis. Recognizing the major limitations to early prognostication is the first step in avoiding the therapeutic nihilism that occurs when patients are excluded from the very treatment that may improve their outcome.

4. The patient not reaching goal temperature. Occult shivering is common and frequently thwarts effective induction to goal temperature. Sedation and paralysis should be routine to assist in reaching goal temperature—at which point, most patients can be maintained on sedation alone. Additionally, supplementing ice packs and additional boluses of cold IV fluids may be necessary to ensure rapid induction.

5. Not establishing a postcardiac arrest care plan and protocol. If your hospital does not have a predetermined plan for managing postarrest patients, now is the time to create one. Assess your current ED and hospital resources and determine whether your patients can be comprehensively managed or whether they require transfer to a regional PCI-capable and hypothermia-capable cardiac resuscitation center for optimal care. If transfer is preferred, create a “transfer protocol” such that cold fluids or ice packs are readily available for use and cooling is initiated early. Coordination with an established destination ensures a smooth transfer of care.

Case Conclusions

With the prehospital cold fluids continuing to infuse in the first patient, you note that he fits the demographics (VF/VT of presumed cardiac origin) and arrest intervals (downtime of 21 min) of the largest prospective therapeutic hypothermia trial; therefore, cooling this patient has Level 1 evidence. Given that your facility does not have PCI or cooling

Table 10. Postcardiac Arrest Resuscitation: Carolinas Medical Center CODE COOL™ Transferring Guideline

Inclusion Criteria

- Adults (age ≥ 18 yrs)
- ROSC within 60 min of arrest
- Persistent coma: inability to follow commands and/or GCS score < 9

Exclusion Criteria

- Absolute: severe or terminal illness with anticipated nonaggressive care
- Relative: active hemorrhage; systemic infection/sepsis; severe refractory shock

Resuscitation Priorities

- Airway
  - Intubation
- Breathing
  - Avoid hyperventilation (goal PaCO₂, of 38-42 mm Hg).
  - Avoid hyperoxia (rapidly decrease FiO₂ to maintain SpO₂ > 95%).
- Circulation
  - Goal MAP is > 70.
  - Anticipate and avoid hypotension.
  - Norepinephrine is the preferred vasopressor.
  - Screen for STEMI using ECG.

Cooling Induction

- Initiate cooling as soon as possible after ROSC.
- Administer refrigerated (4°C) NS 30 cc/kg IV bolus as tolerated.
- Ice packs to groin, axilla, and neck.
- Control shivering with propofol 10 mcg/kg/min.
- Paralyze patient with vecuronium 0.1 mg/kg q1h.

Do

- Initiate transfer early.
- Use paralytics during the induction phase of cooling.
- Document the time of arrest, time of ROSC, and neurological examination.

Do Not

- Delay cooling for CT scanning or extensive testing before transfer, unless clinically indicated.

Abbreviations: ECG, electrocardiogram; FiO₂, fraction of inspired oxygen; GCS, Glasgow Coma Scale; IV, intravenous; MAP, mean arterial pressure; NS, normal saline; PaCO₂, partial pressure of carbon dioxide; ROSC, return of spontaneous circulation; SpO₂, oxygen saturation by pulse oximeter; STEMI, ST-segment elevation myocardial infarction.

Used with permission from Carolinas Medical Center.
capability, you initiate the transfer protocol and place ice packs on the patient. In the meantime, you continue aggressive resuscitation of the patient. After a careful neurological exam, you confirm nonreactive pupils and a GCS score of 3, and you proceed to sedation with propofol and paralysis with vecuronium to avoid shivering during the induction phase. With the blood pressure at 92/42 mm Hg (MAP of 59 mm Hg), you continue fluid resuscitation concurrently with initiation and titration of norepinephrine to reach a MAP > 70 mm Hg to ensure appropriate brain perfusion. Next, you titrate the FiO2 for goal SpO2 of 95% to avoid hyperoxia-related injury. You ask for an arterial blood gas to ensure that the PaCO2 is in the target range of 38 to 42 mm Hg. Shortly thereafter, the patient is transported to the cardiac arrest center.

For your patient with PEA due to a respiratory arrest secondary to status asthmaticus, you acknowledge the Level II evidence that exists for therapeutic hypothermia. You activate your postarrest resuscitative bundle and begin cooling the patient with 30 cc/kg of 4°C normal saline. The patient has cooling pads placed, ice packs applied, and sedation administered. An hour into cooling, her temperature remains > 35°C. You realize that unrecognized shivering may be the cause, so you paralyze her with vecuronium. An hour later, she is at goal temperature. The patient is subsequently transferred to the ICU of the nearest cardiac arrest receiving hospital and completes 24 hours of therapeutic hypothermia at 33°C via protocol. She is then rewarmed slowly over 10 hours. On day 3 postarrest, her sedatives are weaned and she ultimately awakens. Her CPC score is 1, which equates to a good neurological outcome. Several days later, she is ready for discharge. She is discharged directly to home with appropriate asthma management.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study, will be included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, will be noted by an asterisk (*) next to the number of the reference.

234 patients)


84.* Garot P, Lefevre T, Eltchaninoff H, et al. Six-month outcome of emergency percutaneous coronary intervention in resuscitated patients after cardiac arrest compli-


---

**CME Questions**

1. Which of the following statements is currently true about therapeutic hypothermia for VT/VF arrest?
   a. The practice is performed often but has no known benefit.
   b. It is endorsed by consensus guidelines, but implementation remains a challenge at some centers.
   c. The practice has a prohibitively large number needed to treat.
   d. It is not as effective as cooling a PEA/asystolic arrest.

2. Regarding patients that are candidates for therapeutic hypothermia:
   a. PEA arrest is an absolute exclusion criteria.
   b. Asystolic arrest is an absolute exclusion criteria.
   c. Severe terminal illness is an exclusion criteria.
   d. ROSC < 5 minutes after arrest is an exclusion criteria.

3. Why is traumatic active bleeding a contraindication for therapeutic hypothermia for VT/VF arrest?
   a. Patients that are cooled cannot get transfusions.
   b. Patients that are hypotensive should not be cooled.
   c. Patients with a sympathetic surge are at greater risk for shivering.
   d. There is a concern that hypothermia will induce coagulopathy.
4. The authors suggest cooling for ____ at a goal temperature of ____.
   a. 6 to 12 hours, 34°C to 38°C
   b. 12 to 24 hours, 34°C to 36°C
   c. 12 to 24 hours, 32°C to 34°C
   d. 24 to 48 hours, 34°C to 38°C
   e. 24 to 48 hours, 32°C to 34°C

5. The rewarming phase increases the patient’s risk for which of the following?
   a. Hypertension and hypokalemia
   b. Hypertension and hyperkalemia
   c. Hypotension and hypokalemia
   d. Hypotension and hyperkalemia

6. Which of the following statements is true regarding patients with cardiac arrest and signs of a STEMI?
   a. Therapeutic hypothermia is not advised.
   b. Therapeutic hypothermia should be undertaken after PCI.
   c. Priority should be given to hypothermia; PCI is not indicated.
   d. Therapeutic hypothermia has been safely performed along with PCI in 2 studies.
   e. A patient who receives thrombolytic therapy should not be cooled because they have a significantly higher risk of bleeding.

7. What is the most common dysrhythmia encountered during cooling?
   a. Third-degree block
   b. Atrial fibrillation
   c.VF
   d. Sinus tachycardia
   e. Sinus bradycardia

8. Strategies that can be used to avoid postcardiac arrest syndromes include:
   a. Avoiding hyperventilation with a goal PaCO₂ of 45 to 50 mm Hg
   b. Avoiding hyperoxia by not allowing SpO₂ to exceed 90%
   c. Allowing hypotension at a MAP between 40 to 50 mm Hg, as this is cardioprotective
   d. Correcting metabolic disturbances

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Coming Soon In EMCC

Ventilator Management In The Intubated Emergency Department Patient

AUTHORS:

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Emergent airway management is one of the defining skills of the practice of emergency medicine. Emergency physicians must be comfortable with the initial intubation and stabilization of these patients as well as with ongoing ventilator management during the patient’s stay in the emergency department. A retrospective review of a large national data set found that patients who require mechanical ventilation represent only 0.23% of emergency department visits, but they have an inhospital mortality rate of 24%. The same study found that 75% of mechanically ventilated patients spent >2 hours in the emergency department, and 25% were there for >5 hours. Retrospective studies have found that an emergency department boarding time of >2 hours before transfer to the ICU is associated with increased ventilator days and hospital length of stay, and boarding time of >6 hours is associated with increased mortality. Close attention to the optimal management of mechanically ventilated patients in the emergency department may help improve outcomes.

Upon completion of this article, you should be able to:

1. Summarize the data behind low-tidal-volume ventilation, and describe which patients should and should not receive such therapy.
2. Describe treatment approaches for a crashing intubated patient, a patient with urgent ventilation or oxygenation difficulty, and a patient with ventilator dyssynchrony.
3. Cite situations in which patients may be extubated in the ED.
# The EM Critical Care Archives

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<tr>
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<th>Publication Date</th>
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Erratum

In the “Emergency Ultrasound In Patients With Respiratory Distress” article in EM Critical Care Vol. 2, No. 1, an incorrect version of Figure 18D, Subxiphoid was published. The “LA” appearing on the bottom right-hand side of liver should have been labeled “LV.” The correct version of this image appears below and on our website at www.ebmedicine.net/EMCC. We regret any confusion this may have caused.

CME Information

Date of Original Release: June 1, 2013. Date of most recent review: May 1, 2013. Termination date: June 1, 2016.

Accreditation: EB Medicine is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. This activity has been planned and implemented in accordance with the Essential Areas and Policies of the ACCME.

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Needs Assessment: The need for this educational activity was determined by a survey of medical staff, including the editorial board of this publication; review of morbidity and mortality data from the CDC, AHA, NCHS, and ACEP; and evaluation of prior activities for emergency physicians.

Target Audience: This enduring material is designed for emergency medicine physicians, physician assistants, nurse practitioners, and residents as well as intensivists and hospitalists.

Goals: Upon completion of this article, you should be able to: (1) demonstrate medical decision-making based on the strongest clinical evidence; (2) cost-effectively diagnose and treat the most critical ED presentations; and (3) describe the most common medicolegal pitfalls for each topic covered.

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