

THE CCU CORNER

Advances in Therapeutic Hypothermia After Cardiac Arrest

BY GEORGE PHILIPPIDES, M.D., AND ERIC H. AWTRY, M.D. EDITED BY THOMAS J. RYAN, M.D.

In the first "CCU Corner" column, we provided an overview of the growing body of evidence supporting the use of mild hypothermia for the treatment of anoxic brain injury in survivors of cardiac arrest (CARDIOLOGY NEWS, March 2007, p. 14). The American Heart Association and the European Resuscitation Council recommended in 2005 that unconscious adult patients with spontaneous circulation after resuscitation from cardiac arrest be cooled to 32-34° C for a period of 12-24 hours, if the initial rhythm is ventricular tachycardia or ventricular fibrillation and that this therapy should be considered for other rhythms. Subsequently, several nonrandomized implementation studies using historical controls have provided additional support for therapeutic hypothermia.

Despite the published data and guidelines supporting the treatment, recent physician surveys in Germany, England, and the United States suggest that therapeutic hypothermia has yet to be broadly incorporated into clinical practice.

We anticipate that published guidelines for therapeutic hypothermia (TH) will gradually result in its increased use and spur the development of hypothermia protocols. The Boston Medical Center has created a multidisciplinary, multi-department TH protocol that attempts to meet the challenges of treating these critically ill patients as they pass through the prehospital setting, emergency department and often, the cardiac catheterization laboratory, before admission to the CCU. (See box.)

Therapeutic Hypothermia Protocol

The following TH protocol was implemented at BMC in May 2008 to hasten cooling in survivors of cardiac arrest resulting from any rhythm who demonstrate hemodynamic stability and signs of brain injury.

Prehospital Setting

- ▶ Place esophageal thermometer probe to establish baseline body temperature.
- ▶ Document vital signs and temperature at baseline and every 5 minutes throughout transport. (Target cooling body temperature is 32-34° C.)
- ▶ Place ice packs in armpits, neck, torso, and groin areas.
- ▶ Establish two peripheral intravenous lines to infuse chilled normal saline (2-4° C) at 500-cc increments to a maximum of 2,000 cc.
- ▶ Continue the cooling protocol (IVs) until arrival at the ED even if the patient becomes unstable.
- ▶ Administer lorazepam (2-4 mg) and vecuronium (0.1 mg/kg) to control shivering.
- ▶ Transport the patient to a hospital capable of continuing the cooling therapy for 12-24 hours.

Emergency Department

Upon arrival at the ED, the patient is reevaluated. If no contraindications exist, such as systolic BP of less than 90 mm Hg despite pressors or unstable cardiac rhythm, hypothermia is maintained with surface cooling vest/pads. The on-call CCU cardiologist is often consulted to help with this decision.

Cardiac Catheterization Laboratory

Cooling is maintained during coronary interventions. If femoral venous access is obtained during the procedure, an endovascular cooling device can be placed before the patient is transferred to the CCU.

Coronary Care Unit

Therapeutic hypothermia is continued for 24 hours whenever possible. The patient is then actively rewarmed at a rate of 0.5° C/hr. When normothermia is achieved, intravenous sedatives and neuromuscular blockade are weaned and the patient's neurologic condition is assessed, often in conjunction with the neurology service.

Education

In an effort to educate all members of the ED and critical care teams on the use of TH, all ICU nurses have been trained on the use of therapeutic hypothermia devices and all internal medicine and emergency department house officers receive a 1-hour lecture on the topic. The BMC electronic order set for hypothermia has been revised in order to standardize clinical care across the medical center, encourage the use of evidence-based therapies, and collect data on this patient population.

Future Challenges

To date, no consensus exists on the best way to initiate and maintain hypothermia, or how quickly patients should be rewarmed. Simple surface cooling techniques such as ice packs and cooling blankets have the advantage of being noninvasive, relatively inexpensive, and easy to use, but the achievable cooling rate with these modalities is relatively slow, around 0.8° C/hr. Newer, more sophisticated surface cooling devices comprising a vest and limb pads are now commercially available and can achieve cooling rates of 1.3-1.5° C/hr. Another system, called the Thermosuit, consists of an inflatable, contoured "body suit" that covers the limbs and entire torso and allows flowing ice water to come into contact with the skin of the patient. Cooling rate is extremely rapid—1° C every 7 minutes—allowing the attainment of target temperature of 32-34° C within 30 minutes. Both are more complicated to use and expensive. All surface cooling techniques have the disadvantage of limiting access to the patient during the cooling phase.

Endovascular cooling devices offer relatively rapid cooling rates (4° C/hr), less "overshoot" of target temperatures (which occurs in up to 80% of patients cooled with surface methods), and controlled rewarming rates, but are expensive and require femoral venous cannulation.

Infusion of cold saline is easy to administer, is inexpensive, does not hinder access to the patient, and is an attractive method for initiating hypothermia in the prehospital setting. A recent pilot study

randomized 125 patients to receive standard care with or without intravenous cooling with 500-2,000 cc of 4° C normal saline before hospital arrival. In-field cooling was associated with significantly lower temperatures upon arrival, no adverse events, and a trend toward better neurologic outcomes in VF patients.

Future clinical studies are needed to determine whether very early prehospital cooling, longer duration of cooling, and cooling of patients with asystole or longer/unknown downtimes will further improve outcomes and whether TH has a therapeutic role in the treatment for patients with traumatic brain injury, stroke, and myocardial infarction undergoing reperfusion therapy. Investigators at Ochsner Medical Center in New Orleans are planning a pilot clinical trial assessing rapid TH with the Thermosuit to improve reperfusion in acute MI patients. Rapid therapeutic cooling will be achieved in the emergency department before taking the patient to the catheterization laboratory within the mandated 90-minute door-to-balloon time period, Dr. Christopher White, director of the Ochsner Heart and Vascular Institute, told us.

In the meantime we recommend that all medical centers develop protocols for the use of TH, using any of the above methods, for patients with anoxic brain injury following cardiac arrest.



DR. AWTRY is assistant professor of medicine and director of education at Boston Medical Center. DR. PHILIPPIDES is assistant professor of medicine and director of the Coronary Care Unit at BMC. To respond to this column or suggest topics for consideration, write to our editorial offices or e-mail us at cardnews@elsevier.com.

Increasingly, COPD Raises Mortality Risk in MI Patients

BY MITCHEL L. ZOLER
Philadelphia Bureau

CHICAGO — Chronic obstructive pulmonary disease is a lethal comorbidity for myocardial infarction patients, and its deadly punch has grown over time, according to a community-based review of more than 3,000 patients.

In addition, chronic obstructive pulmonary disease (COPD) has become increasingly com-

mon among patients who have a myocardial infarction, affecting 16% of patients who had an a myocardial infarction during 2000-2005, compared with 8% during 1979-1985, Dr. Francesca Bursi and her associates reported in a poster at the annual meeting of the American College of Cardiology.

The deadly impact of coexisting COPD was so strong that it negated an overall temporal trend toward fewer patients dy-

ing following a myocardial infarction. The risk of death following an MI in patients with COPD, compared with those without COPD, rose from a 21% increased risk in 1979-1985 to a 2.6-fold increased risk during 2000-2005, reported Dr. Bursi, a cardiologist at the Mayo Clinic in Rochester, Minn.

Although the Mayo Clinic researchers who performed this analysis had no explanation for why the impact of COPD on

post-MI mortality has increased, they said that their findings underscored the need to enhance therapy and follow-up for patients who face this double whammy.

The Mayo team reviewed data collected on 3,259 residents of Olmsted County, Minn., who had a myocardial infarction during 1979-2005 and were monitored through the Mayo Clinic's county-based community surveillance program. During an average fol-

low-up of 4.8 years, 1,436 (44%) of these myocardial infarction patients died.

For the group overall, the confluence of MI and COPD boosted the risk of death by a statistically significant 38%, compared with patients without COPD, in an analysis that adjusted for several demographic and clinical differences including age, gender, smoking, hypertension, medications used, and revascularization treatment. ■