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Therapeutic Hypothermia to Improve Postresuscitation Outcomes From Sudden Cardiac Arrest

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The survival rate from sudden cardiac arrest is dishearteningly dismal. Even when there is initial return of spontaneous circulation, most victims of cardiac arrest do not leave the hospital alive due to severe ischemic brain injury.¹

Mild hypothermia (cooling the brain to about 90°F to 93°F) has been shown to offer protective effects against various complex pathological processes. Applying this therapy to mitigate the effects of postresuscitation brain injury following cardiac arrest, however, has only recently gained acceptance. In 2003 the Advanced Life Support Task Force of the International Liaison Committee on Resuscitation (ILCOR) issued an advisory statement in support of the use of thera-

peutic hypothermia for selected victims of sudden VF cardiac arrest following return of spontaneous circulation.² The 2005 *AHA Guidelines for CPR and ECC* ratified the earlier ILCOR statement.^{3,4}

Evidence providing the basis for these recommendations comes mainly from 2 key randomized trials.⁵⁻⁸ Interested readers can find more information about the scientific rationale in the 2005 *AHA Guidelines for CPR and ECC*.⁴

Therapeutic Hypothermia Treatment Plans

Successfully instituting therapeutic hypothermia for the out-of-hospital cardiac arrest victim requires a collaborative effort of prehospital EMS, emergency department, respiratory therapy, cardiology, intensive care, and neurology personnel. All parties must understand the basic principles and goals and adhere to a clear management plan. The hospital should create a treatment protocol with clear inclusion and exclusion criteria as well as an easily understood method for delivering hypothermia therapy.

Key medical personnel developing the protocol must decide who will be cooled, within what time frame, by what method, and for how long. Many examples of protocols currently in use in various areas of the country are available at the University of Chicago's Post Resuscitation Resource Pages <http://hypothermia.uchicago.edu/>.

Key Features of a Treatment Protocol

Method

Adequate brain cooling can be achieved using external or internal cooling. External cooling methods include surface cooling with ice packs, cooling blankets, mattresses, or vest systems. Internal cooling methods include the use of cooling catheters inserted into the femoral vein or the peripheral administration of ice-cold IV fluids. Most hospitals select 1 or 2 methods and use them exclusively so that nursing and medical staff gain experience and competency in those techniques.

Timing

Little evidence exists about the window of opportunity for success of this therapy, but most experts recommend that cooling begin as soon as possible, ideally within 4 hours of ROSC in eligible patients. Cool the patient rapidly so that the target temperature of 32°C to 34°C is reached as quickly as possible but avoid overshooting the target. Once the target temperature is attained, keep the patient cool for 12 to 24 hours, or until 24 hours have elapsed from the beginning of induction.

Rewarm the patient slowly to lessen reperfusion injury and avoid rebound hyperthermia. Controlled rewarming at a rate of 0.35°C to 0.65°C per hour to a target temperature

2005 AHA Guidelines for CPR and ECC Mild Therapeutic Hypothermia

- Unconscious adult patients with return of spontaneous circulation (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).
- Hemodynamically stable patients with spontaneous mild hypothermia (>33°C [91.5°F]) after resuscitation from cardiac arrest should not be actively rewarmed.

of 36.5°C (97.7°F) is reasonable and takes between 4 and 12 hours. Once the patient is rewarmed, many experts keep the patient at the target temperature for an additional 12 hours or so to ensure that no fever develops.

Temperature Monitoring

Continuous electronic core temperature monitoring is required to ensure that patients are quickly cooled and maintained in the range 32°C to 34°C without excessive cooling. Cooling below 32°C appears to increase the risk of complications without adding benefit. Good monitoring sites include the bladder (accurate as long as the patient has urine output), the esophagus, or a pulmonary artery catheter if available. Rectal temperature lags behind the core temperature and is not as quick to equilibrate.

The body's natural response to cold is shivering, which generates heat and slows cooling. Shivering also increases oxygen consumption and metabolic demands during a critical healing time. Thus, shivering is undesirable and should be prevented by neuromuscular blockade using continuous administration of an agent such as vecuronium.

Since neuromuscular blockade prevents clinical assessment of the patient's level of consciousness, patients must receive continuous intravenous administration of sedatives, such as propofol or midazolam, to ensure that they do not wake up while they are cold and pharmacologically paralyzed. Use of the train of four (TOF) to measure the depth of paralysis is common in critical-care units, but this method of assessment may not be reliable during hypothermia. Maintaining adequate levels of paralysis and forgoing heated ventilation circuits and hot overhead lights will help to make the induction phase more rapid and direct.

Monitoring for Potential Complications

Potential complications of cooling include arrhythmias, electrolyte abnormalities, diuresis, infection, and coagulopathies. Bradycardia is common, with heart rates frequently slowing to the high 30s. Osborne waves (J waves) are often observed at the

end of the QRS complex during hypothermia. Bradycardia need not be treated unless there is also evidence of poor perfusion, such as hypotension or diminished urinary output.

A mean arterial pressure goal of at least 90 mm Hg is desired to promote cerebral perfusion. The blood pressure may remain elevated during hypothermia because the patient develops peripheral vasoconstriction. Hypotension may occur during rewarming; if present it should be treated aggressively with fluids and vasoconstrictor medications.

Monitor potassium levels closely because hypokalemia is common during cooling and may be exacerbated by insulin administration. Most experts suggest checking electrolyte levels often, particularly during induction of cooling. Supplemental potassium infusions may be needed to achieve a K⁺ of about 4 mmol/L. Stop potassium infusions at the onset of rewarming because potassium will then leave the cells and may cause hyperkalemia.

Significant arrhythmia, bleeding, or hemodynamic instability requires discontinuation of the therapy and active rewarming.

Practical Considerations

Whatever method is used, hypothermia therapy must be considered as a dose-response agent. It is necessary to achieve and maintain the therapeutic dose until rewarming. Wild swings in temperature may counteract the protective benefits of the therapy and may be detrimental. It is possible to continue cooling therapy while the patient is in the cath lab, and interruptions in cooling for other tests should be minimized.

The neuromuscular blockade required for hypothermia therapy can mask seizure activity. Seizure activity in patients with neuromuscular block can be detected using continuous EEG monitoring, but the appropriate role for continuous EEG monitoring or intermittent EEG is still to be determined.

Conclusion

As with any new treatment, the first few patients undergoing mild therapeutic hypothermia treatment will challenge the nursing

and medical staff to learn the nuances of the treatment. The treatment is not difficult to manage and has clear benefits in saving lives. You can be a positive force for change in your institution and help implement this treatment to improve neurologically intact survival from cardiac arrest.

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