Hypothermia after Cardiac Arrest
December 2013

Managing Common Issues:

Bradycardia:

Bradycardia alone (even to as low as 35 bpm) may occur during induced hypothermia and, except in rare cases, is NOT a reason to discontinue the protocol. If bradycardia is severe, associated with persistent hypotension, and is not responsive to fluid and vasopressor therapy, a decision in conjunction with Neurocritical Care may be made to discontinue the protocol and rewarm the patient to 36°C. In this case, the endovascular device should be left in and temperature should remain at 36°C for the duration of the "maintenance" phase. Rewarming can then proceed to 37°C per protocol.

Lactate:

Due to multiple factors (decreased cardiac output, shivering, lab measures, vasopressor administration), lactate levels will often be mildly elevated (usually $\leq$ 5 mmol/L) during hypothermia. Once patient has reached target temperature, lactate should remain stable. Thus, the lactate trend during the protocol should be noted. An increasing lactate during the maintenance phase may indicate underresuscitation, which should be treated aggressively.

Methods of Temperature Measurement:

The benefit of therapeutic hypothermia relies on the induction and maintenance of an appropriate core body temperature without overcooling or accidental rewarming, both of which may be harmful to the patient. For this reason, temperature must be monitored closely and the method used to do so must be accurate and continuous. Of the methods available, both esophageal and bladder measurements have been shown to most accurately reflect core body temperature in most circumstances. Due to the importance of continuous temperature measurement and the possibility of inaccuracies, it is safest to measure BOTH esophageal and Foley temperatures continuously throughout the protocol. This requires a temperature-sensitive Foley catheter to be placed immediately as the protocol begins, followed by an esophageal probe as soon as possible.

Rectal temperature probes, although often accurate when core body temperature is static, have been shown to lag behind changes in core body temperature. This "probe lag" may lead to the over-cooling and inappropriately rapid rewarming. Thus, rectal probes should not be used during the protocol unless both esophageal and Foley probes are unavailable or contraindicated.

Tympanic Membrane and axillary temperature measurements do not provide continuous temperature monitoring and should not be used to monitor temperature in patients undergoing therapeutic hypothermia.

Dysrhythmia:

Hypothermia-induced dysrhythmias generally do not occur unless temperatures fall $\leq$ 30°C, and hypothermia-related ventricular fibrillation is rare unless temperature is $\leq$ 28°C. If temperatures this low do occur, monitor closely for dysrhythmias. If hemodynamically unstable dysrhythmias do occur at the low temperatures noted above, rewarm the patient rapidly to 32°C.

Hypotension:

Hypotension often occurs during the hypothermia protocol. If the source is cardiogenic, it is often the post-arrest state (and not the hypothermia) that is the cause. During rewarming,

peripheral vasodilation may be the cause, and this is usually responsive to IV fluids. Except in rare cases, the hypothermia protocol should NOT be stopped due to hypotension. Continue to give hemodynamic support as above. If hypotension continues despite these measures, contact Neurocritical Care to discuss further options, which may include rewarming.

Diuresis:

Hypothermia induces diuresis in most patients. This so called "cold diuresis" may lead to an artificially high urine output. This may result in both a relative hypovolemia as well as an overestimation of the patient's volume status if conventional urine output goals are applied. Thus, a slightly higher goal urine output of 1 cc/kg/hr is reasonable in most cases to ensure continued adequacy of intravascular volume. Further, hypothermic patients are especially at high risk of hypotension during the rewarming phase, when 24 hours of diuresis has occurred along with peripheral vasodilation occurring with the increase in temperature. Along with higher urine output goals, it may be beneficial to increase intravascular fluid administration just prior to rewarming.

Bleeding:

The antiplatelet and antithrombotic effect of hypothermia is low (especially at a temperature above 34 °C). Further, most bleeding risk associated with hypothermia is limited to an impaired ability to generate new clots (not necessarily in disruption of clots already formed) and is increased in the setting of acidosis. Thus, for minor bleeding (i.e. around IV sites, sites of minor trauma, etc.) do NOT stop the hypothermia protocol. Instead, correct acidosis and minimize any further trauma to the patient. For cases of clinically significant bleeding (ex massive gastrointestinal bleeding, intracranial hemorrhage, etc.) early rewarming to 36^{o}C or higher may be initiated, but ONLY in consultation with the Neurocritical Care Fellow.

Electrolyte Shifts:

Decreases in temperature often lead to intracellular potassium shifts without depletion in whole body potassium stores. This corrects during rewarming, which, if aggressively replaced during low body temperatures, may lead to hyperkalemia as the patient is rewarmed. Use caution when replacing potassium in these patients. Further, replace magnesium (>2.0), free Cal ("high-normal"), and phosphorous (normal) aggressively as they may decrease during hypothermia.

Arterial Blood Gases:

It is imperative that the lab knows the patient's current temperature when analyzing blood gases. This should be communicated directly to the lab when sending the sample, and should be checked to confirm the temperature used for analysis is listed on the result and is consistent with the patient's actual body temperature. Lab analysis of PaCO2 and PaO2 at the incorrect temperature may lead to unrecognized respiratory alkalosis and/or hypoxia. In cases where it is impossible for the lab to measure at the patient's actual body temperature (rare) and a second sample cannot be sent, values may be adjusted as follows:

-<u>For each 1 °C below 37°C</u>:

-PO2 – 5 mmHg = corrected value

-PCO2 – 2 mmHg = corrected value

-pH + 0.012 = corrected value

Diagnosis of Infection:

Since the patient's hemodynamic, cellular, and thermoregulatory response to infection will be blunted during hypothermia, it is necessary to monitor closely for the development of infection using other methods. Daily chest x-rays as well as screening blood, urine, and sputum cultures (BAL or PAL) every 48 hours should be obtained while temperature is being regulated.

Aggressive treatment should be initiated when there is evidence of infection, and the patient, while hypothermic, should be treated as immunocompromised. In general, evidence of infection is NOT an indication for early rewarming, but should be discussed on a case-by-case basis with the Neurocritical Care fellow.

Medication Administration:

Do not administer any medications labeled **"Do Not Refrigerate"** through the Icy-Cath or Cool-Cath while being used for active cooling. These medicines may precipitate in the central line. These medications may be administered via a separate peripheral or central line – even while the patient is hypothermic. The cold central line causes the precipitation, not the hypothermic patient.

Shivering:

Shivering is an innate and effective response to a decrease in core body temperature. It is problematic for the post-cardiac arrest patient, as it increases cerebral oxygen consumption, systemic oxygen consumption, CO2 production, and increases cardiac. Further, it can severely impede achievement and maintenance of therapeutic goals. Thus, it should be monitored closely and aggressively treated by EITHER of the two methods above: Neuromuscular Paralysis OR the Anti-Shivering Protocol (not both). If the Anti-Shivering Protocol is applied, patients should be watched closely to ensure effectiveness of the sedative and nonmedical measures. If neuromuscular paralysis is employed, the need for this should be frequently reassessed, and paralysis weaned when indicated.

Surface Counterwarming is a nonmedical therapy that applies heat to the skin while maintaining the appropriate core body temperature. This results in a perceived increase in body temperature by the hypothalamus, which results a decrease in shivering. If the cooling catheter has not been placed, and ice packs and a cooling blanket have been applied, surface counterwarming may be achieved by placing warm blankets on the patient's exposed distal upper and lower extremities. Once the cooling catheter has been placed, a Bair Hugger may be used over the patient's skin to achieve this purpose. Surface Counterwarming should NOT be applied to patients undergoing neuromuscular paralysis.

Seizure Monitoring:

Patients who have undergone a cardiac arrest are at high risk for seizures. This is especially detrimental for these patients, as seizures can increase cerebral metabolic rate and contribute to reperfusion injury. Since these patients will be sedated and may have received neuromuscular paralysis, neurocritical care should initiate Continuous Video EEG monitoring as close to the initiation of hypothermia as possible. If seizures are detected by EEG monitoring, these should be treated aggressively.

Neuroprognostication:

Neurologic prognosis post cardiac arrest treated with hypothermia is controversial. The optimal timing for appropriate prognosis is unclear. The primary team should refrain from any attempt at prognosis until at least 72 hours post cardiac arrest. This is especially important if the patient remains neurologically altered or comatose upon rewarming. Recent literature has demonstrated good neurologic outcomes despite initially poor neurologic exams post-rewarming. Additional tests such as MRI or EEG may be employed to assist with prognosis. Neurology should be consulted during the hypothermia period to assist with the long-term prognosis once rewarming is complete.

Managing Fluctuations in Temperature:

Wide swings in temperature may be harmful in the post-arrest period and should be prevented. Patients should NOT be overcooled (< 32°C) or allowed to rewarm in an uncontrolled fashion after induction. To prevent this, a bladder OR an esophageal temperature probe should be placed as soon as possible after the patient's arrival. Temperature should be monitored closely and corrective measures should be taken immediately if the patient becomes overcooled or rewarming begins to occur. Further, placement of an endovascular device should occur as soon as possible after the patient's arrival, regardless of the target temperature.

Hyperthermia:

Fever can be damaging to the injured brain and may impede neurologic recovery after cardiac arrest. In all cases of post-arrest management, regardless of the initial target temperature, hyperthermia (> 37.5°C) should be avoided for AT LEAST 72 hours after return of spontaneous circulation, preferably longer.

Stopping Hypothermia:

Though it is rare, there are circumstances in which the patient must be rewarmed prior to the rewarming phase. This should only be done in conjunction with Neurocritical Care. If this is necessary, the patient should be rewarmed at a maximum rate of 0.5°C (vs 0.35°C)/hr to 36°C. In general, temperature should be maintained at 36°C until the rewarming phase, at which time the care should continue as per protocol. There may be very rare cases in which a higher temperature is needed (for example, in some cases of acute bleeding). In this case, though a temperature above 36°C is allowable, the patient should be still kept at a temperature < 37.5°C at all times.

Resources:

Troubleshooting the Thermogard:

Alsius Cooling System:

Operating Manual Available on CPQE

24 hour support line (877) 225-748