

Clinically Induced Hypothermia

Why Chill Your Patient?

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ABSTRACT

Clinically induced hypothermia is an evidence-based intervention strategy that can improve the neurological outcome of unconscious patients after sudden cardiac arrest. Until recently, clinically induced hypothermia has been primarily used during surgery as a mechanism of preserving cardiovascular and neurologic stability of patients. Current evidence suggests that early use of mild hypothermia therapy in select populations of patients improves survival and neurologic outcome postdischarge. While clinically induced hypothermia is beneficial as a treat-

ment to preserve neurologic function, it is not without complications. The purpose of this article is to review current literature and evidence-based nursing practice implications for managing the induction of a hypothermic state in adult patients who remain comatose after initial resuscitation from sudden cardiac arrest. Physiologic benefits of hypothermia, complications, and nursing care considerations will be presented.

Keywords: clinically induced hypothermia, neurological outcome, postresuscitation care, shivering

Clinically induced hypothermia as an intervention strategy for improving neurological outcome of patients after sudden (ventricular fibrillation) cardiac arrest has been advocated since the 1950s.^{1,2} Early studies found that reducing the body's core temperature below normal could improve patient survival and neurological outcomes. Challenges associated with inducing hypothermia, in addition to complications from the therapy, have made the treatment difficult to use in many clinical situations. Until recently, clinically induced hypothermia has been primarily used during surgery as a mechanism of preserving cardiovascular and neurologic stability of patients. Current evidence suggests that induced hypothermia outside the operating suite for patients after cardiac arrest may improve patient neurological outcomes as well as survival after hospital discharge.³⁻⁹ This article reviews the current literature and evidence-based practice implications for managing hypothermia treatment in adult patients

who remain comatose after a sudden cardiac arrest. Physiologic benefits of hypothermia, complications, and nursing care considerations will be presented.

Why Cool Your Patient?

In the United States, the prevalence of coronary heart disease and myocardial infarction are estimated to affect 13 000 000 and 7 100 000 individuals respectively,¹⁰ and sudden cardiac arrest is responsible for more than 250 000 deaths in the United States each year.^{8,11} As our population continues to age and the prevalence of obesity and type II diabetes mellitus continue to increase,¹⁰ the prevalence and incidence of cardiovascular

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diseases that lead to sudden cardiac arrest will unfortunately also increase.

After initial resuscitation, goals for postresuscitation care are to maximize blood pressure and effective perfusion to organs and tissues, control temperature (prevent hyperthermia), control glucose concentration, and avoid hyperventilation.^{8,9} Ideally, patients that are successfully resuscitated after a sudden cardiac arrest will also be neurologically intact after hospital discharge. Current resuscitation research continues to identify interventions that improve neurological function of patients who survive sudden cardiac arrest.⁹ One treatment found to enhance neurological function of patients that suffer a sudden cardiac arrest is inducing hypothermia for a short period of time (12 to 24 hours postresuscitation) as soon as possible after the cardiac event. Current studies are suggesting that early, controlled induced hypothermia in the postresuscitation phase of patients remaining comatose after a sudden cardiac arrest have improved survival with better neurologic outcomes postarrest.^{3-5,8-13}

Not all patients that experience a cardiac arrest are candidates for clinically induced hypothermia therapy. The American Heart Association and the International Liaison Committee on Resuscitation have recommend criteria for which patients should be treated with hypothermia therapy. The current criteria state that unconscious adult patients with spontaneous circulation after out-of-hospital sudden cardiac arrest should be cooled (32°C to 34°C) for 12 to 24 hours when the initial rhythm was pulseless ventricular tachycardia or ventricular fibrillation.^{4,8-10} The science of using hypothermia to treat other patient populations and diseases (such as victims of traumatic brain injury, stroke, subarachnoid hemorrhage, hepatic encephalopathy, children, and lethal cardiac dysrhythmias or in-hospital cardiac arrests) remains an active area of research, but discussion of these injuries and populations are beyond the scope of this article.

Hypothermia is defined by core (blood) body temperature and is broken down into ranges of mild (33°C to 35°C), moderate (28°C to 32°C) and severe (<28°C) hypothermia.¹⁴ Mild to moderate states of hypothermia has been found to have a neurologic protective mechanism within the brain that can improve a patient's outcome after a sudden cardiac arrest. Patients experiencing sudden cardiac arrest should be cooled as soon as possible and

maintained in a state of mild hypothermia for 12 to 24 hours and then be gradually rewarmed. The actual time delay that can be tolerated before initiating hypothermia treatment remains unclear; however,¹³ Polderman suggests that in postcardiopulmonary resuscitation, a delay of up to 8 hours may be acceptable and still optimize neurologic outcome of patients from the protective effects of cooling.

Hypothermia provides several brain preserving effects; however, it also has associated complications that require attentive nursing care and interventions to prevent complications from the therapy. It is important that the nurse understand both the benefits and adverse effects of therapeutic hypothermia while providing postresuscitation care to unconscious survivors of sudden cardiac arrest.

Benefits and Adverse Effects of Hypothermia

During cardiac arrest, neurologic deficit can result from decreased cerebral oxygen delivery because of hypotension or lack of perfusion. Hypoxic states in the brain cause cerebral edema, and consequences of reperfusion can exacerbate the cerebral edema compromising neurological function after successful resuscitation from a cardiac event. Cooling the patient has been found to limit the negative effects from hypoxic and reperfusion states. Inducing mild hypothermia slows cerebral metabolism, decreasing oxygen consumption by 5% to 7% for each degree centigrade in reduction in body temperature.^{13,14}

During ischemic and reperfusion states cells in the brain release enzymes and experience intracellular ion changes that damage cellular mitochondria, causing a programmed cell death that is called apoptosis. Anaerobic metabolism in the brain disrupts ATP dependent cellular pumps and excessive calcium and the excitatory neurotransmitter glutamate are found to be at abnormally high levels in the cells. The presence of high levels of calcium and glutamate create an excitotoxic cascade¹²⁻¹⁴ of hyperexcitability in the brain cells, exacerbating the hypoxic state furthering mitochondrial and cellular death. As cells in the brain die, cerebral ischemia persists in perpetuating the negative cascade of events that leads to cerebral edema, compounding the adverse sequence of events within the brain. The blood brain barrier is also disrupted during low perfusion states, allowing an influx of fluid

worsening cerebral edema. Hypothermia slows the neuroexcitatory processes stabilizing the influx of calcium and glutamate limiting cellular death and reduces disruptions in the blood brain barrier lessening cerebral edema. Apoptosis is believed to last for up to 48 hours after initial insult, which may explain why clinically induced hypothermia therapy can be neuroprotective.¹³ Following ischemic injury and during reperfusion states, the inflammatory and immune systems respond by releasing proinflammatory mediators: neutrophils and macrophages. Phagocytic actions of these cells and release of large amounts of oxygen radicals overwhelm injured cells transitioning cell injury to cell death,^{5,13,15} worsening cerebral edema and cerebral perfusion.

Induction of a hypothermic state suppresses the ischemia-induced inflammatory reactions that occur after cardiac arrest. Neutrophil and macrophage functions are decreased at temperatures $<35^{\circ}\text{C}$.¹⁵ Although this suppression assists in minimizing cerebral edema, it also poses a risk of infections for the patient because of changes in white blood cell function. Mild hypothermia also induces insulin resistance and hyperglycemia, which further increases the risk of infection. Pulmonary and wound infections are the most common infectious complications experienced during induced cooling.^{16–18} Prevention of infection by rigorous surveillance and prophylactic nursing interventions can minimize these risks for the patient. Table 1 provides suggested nursing interventions to prevent infection and monitoring parameters during postresuscitation care of patients with clinically induced hypothermia treatment.

Hypothermia causes mild bleeding from platelet dysfunction. Laboratory analysis of coagulation studies may not accurately report alterations in function because tests are usually performed at 37°C ;^{12,19} thus, the nurse recognizes alterations in coagulation occur during hypothermia and observes for signs and symptoms of delayed clotting. Current reviews of hypothermia therapy studies have not found significant risks of bleeding and, therefore, decreased coagulation during hypothermia, if it is a clinical concern, should be managed with the administration of platelets and/or fresh frozen plasma.^{6,15,17,19,20}

Fluids and electrolyte shifts occur during induction of hypothermia. Mild diuresis can occur with initiation of cooling, and interventions to replace fluid and electrolytes may be

indicated. Hypothermia lowers cardiac output by up to 25%,¹⁹ causing a mild acidosis and rise in serum lactate levels. Electrolytes shift because of changes in cellular membranes with ischemia and states of cellular acidosis. Electrolyte disorders commonly seen during hypothermia therapy are low levels of magnesium, potassium, and calcium. Electrolytes are replaced if cardiovascular signs and symptoms result from low levels of these serum electrolytes. However, magnesium has been found to have neuroprotective properties and high normal levels of magnesium are recommended during cooling.¹⁹

Shivering is the body's natural response to cold. Shivering increases metabolic activity and oxygen consumption in an effort to generate heat. It is also uncomfortable for the patient. The goal of cooling is to decrease oxygen consumption; thus, prevention of shivering during the cooling processes is essential. Medications, primarily sedating agents and neuromuscular blocking agents (NMBA), such as cisatracurium or vecuronium, can be used to attenuate adverse effects of shivering.

Mild hypothermia therapy has neuroprotective benefits by reducing apoptosis and cerebral edema. The primary benefit of cooling is enhanced neurologic recovery after a cardiac arrest. However, hypothermia can also have negative consequences for the patient. The cooler a patient becomes, the more significant the adverse effects of hypothermia. Thus, great care needs to be taken when inducing hypothermia so that target goals for therapy are not below mild hypothermic temperature ranges and continuous, accurate monitoring of body temperature must be maintained throughout the therapy.

Methods to Induce Hypothermia

There are several methods that can be used to induce mild hypothermia. The methods can be categorized as being invasive and non-invasive. The invasive methods involve the use of endovascular catheters ranging in size from 9 French (Fr) to 14.7 Fr and are typically placed in the femoral vein. Blood comes in direct contact with a closed loop catheter that circulates cold water within the tip. Radiant (Radiant Medical, Redwood City, CA), Innercool (Innercool Therapies, San Diego, CA), and Alsius (Alsius Corporation, Irvine, CA) are commercial devices available for invasive hypothermia therapy.

Table 1: Nursing Care Considerations in the Management of Patients With Clinically Induced Hypothermia Postresuscitation From Sudden Cardiac Arrest³¹⁻³³**Aspiration precautions**

- Head of bed >30°.
- Maintain airway and check cuff pressures.
- Provide frequent oral care.

Hyper-/Hypoglycemia management

- Implement intensive insulin therapy.
- Maintain blood glucose 80 to 110 mg/dL.

Temperature regulation

- Follow hospital hypothermia protocol.
- Administer neuromuscular block agents (NMBA) and sedation agents to prevent shivering.
- Discontinue NMBA when the patient is in the rewarming phase.
- Verify temperature using 2 sources; if using bladder temperature, make sure that the patient has adequate output.
- Document temperatures every hour and PRN; ensure patient's temperature does not go below 33°C.

Fluid management

- Monitor electrocardiogram rhythm.
- Check electrolytes and replace as needed.
- Monitor arterial blood gases and base deficit.
- Strict input and output.

Infection protection

- Turn every 2 hours.
- Provide oral hygiene.
- Maintain aseptic technique with invasive devices.
- Monitor for any signs and symptoms of infection.

Pain management

- Assess patient's pain level using an appropriate pain scale.
- Administer pain medication in addition to sedation and NMBA.
- Note that patient may require less pain medication when cooling and more during the rewarming due to vasoconstriction and vasodilation.

Skin surveillance

- Inspect skin for areas of breakdown prior to applying cooling device.
- Continue to inspect skin during the cooling treatment for any signs of maseration, mottling, or breakdown.
- Patient's skin may be very red from hypothermic vasoconstriction.
- Turn the patient every 2 hours and elevate heels off the bed.
- Document any skin or mucous membrane changes.
- Document skin assessment (eg, Braden Score) per hospital guidelines.
- Consider pressure relief/reduction mattress therapy.

Preliminary studies show the invasive devices to be effective for rapid cooling and temperature control.^{19,21} However, the invasive devices have inherent risks, such as bleeding, infection, deep vein thrombosis, and vascular puncture.

Noninvasive methods utilize surface cooling techniques. Cooling with ice and water immersion has been reported to be the most effective way of surface cooling.²² Traditional methods of surface cooling include the use of ice packs, alcohol baths, and fans. These techniques have practical limitations in that they are labor intensive, messy, and present electrical hazards to the staff and patient. Temperature control may be imprecise and inefficient, resulting in either prolonged time to reach patient target temperature or significant over cooling in patient temperature (eg, $<33^{\circ}\text{C}$).^{19,23}

The noninvasive or surface methods can be further separated into 2 categories: the traditional methods (which require manual control of water or air temperature) and the newer systems that provide automatic control. Manual systems typically employ single-use woven cloth or reusable vinyl water blankets. The patient either lies on one blanket or has one blanket below and one blanket on top. Air trapped between the blanket and the patient acts as an insulator, resulting in slow thermal transfer from the patient. Some of the newer automated systems have expanded on this concept by evolving the water blankets into wraps that cover approximately 70% to 80% of the patient's body surface area. Although these latter methods are an improvement in the original water blanket design, the degree of surface area coverage can impede patient care.^{18,22,24} Other complications that may occur with water blankets include accidental puncture of the blanket causing leaks and skin erythema, mottling, and breakdown from hypothermic skin vasoconstriction.²⁵

One of the more recent advances in temperature control employs an automated temperature control system and adhesive, hydrogel energy transfer pads (eg, Arctic Sun, Medavance, Louisville, Colo). Temperature controlled water circulates through the pads in response to patient temperature and a preset target temperature. With the adherence of the pads to the skin, conductive heat transfer occurs for rapid, precise control for inducing hypothermia and rewarming. The surface area required for this system is only 40% of the body, covering the back, abdomen, and thighs. The design

circulates water under negative pressure thereby eliminating the chance of leaks that can be seen with other devices.^{11,18,22,24} This design also allows for better access to the patient to provide care, such as turning the patient to prevent skin breakdown and pneumonia, without compromising cooling therapy. A study by Mayer et al²³ found the arctic sun temperature management system to be superior to conventional cooling blanket therapy in the controlled induction of hypothermia and rewarming of patients. Additional studies comparing other external cooling devices are needed.

Other nursing considerations during initiating of hypothermia therapy are that the effectiveness of cooling techniques are dependent upon normal physiology. Older patients have a decreased capacity to control body temperature, lower rate of metabolism, lower body mass index, and less effective vascular response (eg, less vasoconstriction), thus older patients will cool faster than younger patients.¹³ Obese patients may take longer to cool and reach target hypothermic temperature because of the insulating properties of fat tissue.

Whatever method is selected to induce mild hypothermia, it is critical that all members of the team are adequately trained on the cooling protocol or device. If a commercial cooling device is used, nursing staff needs to be familiar with the use and potential complications associated with the device to ensure mild hypothermia is achieved quickly and rewarming is gradual.

Monitoring Temperature

Once a patient is identified as a candidate for mild hypothermia therapy, goals are to rapidly cool the patient to a temperature in the range of 33°C to 35°C . When aggressively cooling the patient, care needs to be taken to prevent excessive hypothermia (eg, 28°C to 32°C). If the patient is cooled to a temperature of $<33^{\circ}\text{C}$, the patient becomes more at risk for complications associated with hypothermia.

Two means of monitoring patient temperature are recommended to ensure you have accurate measurement because peripheral and core temperature may not always correlate. The goal is to have the 2 measurements as similar as possible. The assumption that core and peripheral temperature are equal is false.²² A core temperature is obtained from a pulmonary artery catheter and peripheral

temperatures sites include bladder, rectal, oral, esophageal, and axillary. Tympanic temperature measurements can be considered to be reflective of core temperature because the infrared signal senses blood temperature from the tympanic membrane. However, the tympanic measurement is not a reliable source as it can become dislodged and accuracy is based on clinician technique.^{26,27}

Bladder temperature can be used to trend cooling; however, a reasonable urinary flow is needed to obtain accurate measurement. Bladder temperatures have been found to correlate well with fluctuations in core temperature,²⁸ making it a convenient means of continuously measuring body temperature. Esophageal and rectal temperature measurement methods have also been used during cooling. Esophageal temperature monitoring requires a thermister be inserted into the patient's esophagus and temperature readings are displayed on an external monitoring device. Dislodgement of the esophageal probe is unlikely unless other oropharyngeal tubes are placed or removed during therapy. Rectal temperature measurements are easy to obtain; however, disadvantages of the rectal temperature measurement are inability to maintain accurate assessment of correct placement in the patient's rectum and stool may lead to false readings and dislodgement during defecation.^{26,27}

Some cooling devices have a capacity to electronically regulate the cooling of the water that is circulating through the device. For these devices to accurately regulate and automatically set water temperature based on feedback to the machine from the temperature measurement probe, the accuracy of the source of temperature reading (eg, bladder probe) must be frequently reassessed. Failure to ensure accurate temperature feedback to the cooling device can result in under- or overcooling the patient. Nursing interventions during monitoring of the hypothermic patient requires frequent reassessment and trouble shooting the measurement techniques during hypothermic therapy.

Nursing Care Considerations of the Hypothermic Patient

Caring for the hypothermic patient challenges the nurse's clinical skills. The nurse needs to provide vigilant surveillance over the patient's changing physical condition. The nurse's knowledge of the pathophysiology associated

with hypothermia will require nursing assessment of the following: prevention of rewarming, electrolyte imbalance, arrhythmia detection, prevention of infection, skin care, and pain. The nursing interventions will vary according to each individual patient and how they are responding to the cooling and the other medical conditions that may be occurring concurrently. Understanding the side effects and physiological changes associated with cooling will prevent detrimental side effects that will overshadow the positive benefits of cooling. Prior to initiating hypothermia therapy, the nurse should perform a complete assessment to include surveillance of the skin, patient's response to pain, and baseline neurologic function. Neurologic assessment is needed to allow for comparison after therapies, as well as the fact that some patients have been known to wake up before cooling is initiated, thus clinically induced hypothermia would not be a treatment option in a conscious patient as part of postresuscitation care.

Once the target temperature is reached, the temperature needs to be maintained within the target range. This can be challenging if you are using cooling blankets and ice. The nurse needs to constantly monitor the temperature and ensure that cool air is not lost when performing nursing functions that require lifting the blankets. Likewise, the nurse needs to know when to remove the ice bags so that temperature does not go below 33°C. Excessive cooling or overshooting the target temperature (eg, temperature below 33°C) puts the patient at risk for moderate hypothermia with significant complications. Typically, patients are not cooled for longer than 24 hours. After this time period, the rewarming phase occurs and usually takes 8 hours to achieve normothermia. Patients should be rewarmed at 0.5°C to 1°C every hour to prevent adverse effects during the rewarming process.^{12,18,29} This is easily accomplished with devices that can be programmed to monitor a patient's temperature and automatically cool or warm the circulating water. If such a device is not available, rewarming can be accomplished using warm blankets and monitoring the patient's temperature every 15 minutes.

Effective airway management is maintained through endotracheal intubation and mechanical ventilation.^{5,19,29} Current evidence supports the use of sedation protocols that monitor effective analgesia and sedation without oversedating

patients and prolonging intensive care stays.³ Following sedation protocols during cooling maximizes patient comfort and hopefully will minimize prolonged mechanical ventilation at the close of hypothermia therapy. Benzodiazepines have been found to be effective sedating agents and are believed to inhibit neurotransmitters in the brain lowering cerebral oxygen consumption.¹⁴ Propofol may also be used as a sedating agent. In addition to sedation, pharmacologic agents that attenuate pain need to be administered. Fentanyl and morphine are preferred agents because meperidine has an active metabolite that is epileptogenic and could worsen the brain injury.^{19,29}

An important part of nursing care during induction of hypothermia is to prevent shivering. The natural response to cooling is for a patient to shiver to warm the body. Shivering can lead to increased in oxygen consumption between 40% and 100%,¹³ which is an undesirable response. Shivering may be difficult to assess in its early stages especially when the patient is not able to inform the nursing staff.³⁰ Early signs of shivering include a drop in mixed venous oxygen saturation, increased in respiratory rate, facial tensing, “noise” or static tracing on the continuous electrocardiogram tracing, and palpation of muscle fasciculations of the muscles of face or chest.^{23,30} Sedating agents and/or use of NMBA may be needed to prevent shivering. The nurse should establish a train of four (TOF) before initiating NMBA therapy; however, peripheral vasoconstriction during hypothermia may present with difficulties in on going monitoring of the TOF while the patient is at target temperature during cooling. Hypothermia changes drug metabolism and pharmacokinetics effecting desired action of medications. Dosing of sedation, pain, and NMBA agents may need to be decreased because of changes in metabolism during hypothermia requiring less drug to be administered.¹⁷ Neuromuscular blocking agents need to be discontinued during the start of rewarming because the patient no longer needs these agents to prevent complication of shivering and increase oxygen consumption.

During mild hypothermia therapy, it is anticipated that the patient will experience some cardiac complications. As the body temperature decreases, cardiac output and stroke volume decrease and systemic vascular resistance and blood pressure increase in a response to vasoconstriction. Sinus bradycardia is a com-

mon finding with cooling related to hypothermic myocardial depression.¹³ Continuous cardiac monitoring is needed to detect changes in cardiac rhythm because the risk of arrhythmias increases as the patient reaches levels of moderate hypothermia (eg, <32°C) Moderate hypothermia can cause negative chronotropic effects on pacemaker tissues that may potentiate AV blocks and make the patient more susceptible to atrial and ventricular fibrillation. Antihypertensive therapy may be needed to lower blood pressure in some patients optimizing cardiac output.²²

Another side effect of hypothermia is hyperglycemia caused by insulin resistance. Hyperglycemia is associated with increased infection rates, higher incidence of renal failure, and critical illness neuropathy.³ The amount of insulin required to maintain glucose levels within the normal range (eg, 80 to 110 mg/dL) is usually higher than expected because of this insulin resistance. Electrolyte imbalances are also seen during cooling because of intracellular ion shifts. During hypothermia fluxuations in potassium, calcium, and magnesium occur. Magnesium levels are treated to maintain normal to high normal levels of this electrolyte because of its role mitigating neurological injuries.^{13,20} Potassium levels will drop during cooling and rise as the patient is rewarmed. Caution should be used in treating potassium levels relative to the patient’s body temperature and associated signs and symptoms of potassium imbalances.

Patient’s immune response will be suppressed by hypothermia. The white blood cell count will be decreased, and cooling masks the normal response of a fever to indicate infection. Aggressive pulmonary prevention interventions are needed to pneumonia and nursing care should also include prevention and management of aspiration that can occur with sudden cardiac death. Mild hypothermia depresses platelet function; thus, assessment for signs and symptoms of prolonged bleeding are needed. Skin hygiene to prevent and monitor for signs of tissue breakdown and/or infection are an essential nursing function. If water leaks onto the skin, interventions to prevent skin maceration will also warrant implementation.

Conclusion

Hypothermia is evidence-based practice proven to improve neurological outcomes of some

unconscious cardiac arrest patients.^{1-6, 13,15-18} Society has benefited from a significant emphasis and education to the public and healthcare providers for early initiation of cardiopulmonary resuscitation and use of automated external defibrillators. However, poor neurological outcomes remain an unfortunate consequence of cardiac arrest. Early intervention with hypothermic therapy offers a realistic approach to preventing severe neurological deficits postcardiac arrest. Every patient who has a cardiac arrest deserves the opportunity for a full neurological recovery. Hypothermia treatment offers this patient population the best possible chance of a full neurologic recovery. Critical care nurses that are knowledgeable in the management of patients during clinically induced hypothermic therapy can positively impact patient care and prevent adverse neurologic outcomes from sudden cardiac arrest, as well as prevent complications associated with this therapy.

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