Shivering Avoidance in the Neuronally Injured Patient: Impact on Temperature Management Technology Decisions

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Introduction

Temperature reduction therapies have been proven to provide substantial protection against ischemic brain injury [1] and to slow or prevent secondary brain injury. [2] Recent studies have demonstrated that induced moderate hypothermia improves neurological outcome in survivors of cardiac arrest.[3,4] Temperature management protocols include therapeutic hypothermia, which purposely lowers body temperature below normal (to 32-34°C / 89.6°F-93.2°F), or fever control, maintaining body temperature within normal range. Traumatic Brain Injury (TBI) guidelines cite maintenance of normothermia as the minimum standard of care and recommend mild to moderate hypothermia for neuro protection and improved patient outcomes.[5] Shivering, one of the common side effects seen with therapeutic cooling, remains a serious limitation to this therapeutic modality and must be controlled in order to avoid serious physiologic consequences.[6]

The American Heart Association (AHA) strongly recommends the avoidance of shivering during hypothermia induction, normothermia or rewarming periods.[16]

Physiology of Shivering

Shivering is a normal physiological response of an individual's *sensed temperature* and the thermostatic-like limitation of their *threshold zone*. The body's ability to homeoregulate involves regulating and defending its set point or temperature levels within the *threshold zone*. Skin thermoreceptors lie near the surface, detecting temperature changes of as little as a few thousandths of a degree Celsius (C).[7]

Incoming signals of "cold" from the periphery (i.e skin) provide the input information to the central control mechanisms (hypothalamus) and initiate thermoregulatory responses. When hypothermia develops (either accidentally or intentionally induced), the body will immediately try to counteract this disturbance to decrease heat loss by vasomotor mechanism control (vasoconstriction) and piloerection ("gooseflesh") followed by emergency thermoregulatory mechanisms (shivering).

Shivering can occur at normothermic, or even hyperthermic, stages due to elevation in the hypothalamic thermoregulatory set-point (fever) and raises the *threshold zone*.[8] During fever, the set-point is elevated due to the influence of inflammatory chemicals called pyrogens. Because body temperature is lower than the new elevated *threshold zone*, warming responses (e.g. vasoconstriction, piloerection, shivering) are stimulated. Febrile shivering is the "shaking chill" experienced during fever. The observed increase in skeletal muscle activity results in increased heat production until the body temperature reaches the new thermostatic set-point.

The Impacts of Shivering

Shivering can be divided into different stages and can be visible or invisible (subclinical shivering). The first indications of shivering may be labored breathing and a fall in mixed venous oxygen saturation followed by heightened muscular tone. Visible shivering may vary from involuntary facial and neck muscle contractions to shaking so severe that patients rattle the bedrails, often referred to as "rigors". Following profound shivering, patients often complain of muscle soreness and exhaustion.[9] A **Bedside Shivering Assessment Scale** (**BSAS**) was modified from an earlier observation tool, and measures the severity and duration of shivering in daily clinical practice (Table 1).[10]

Table 1 Bedside Shivering Assessment Scale (BSAS)

0	No shivering	Neither visually nor with palpation of thorax
1	Mild	Localized to the neck and /or thorax only; may manifest on EKG rhythm
2	Moderate	Shivering involves gross movement of the upper extremities (in addition to neck and thorax)
3	Severe	Shivering involves gross movements of the trunk and upper and lower extremities

A growing body of evidence shows that vigorous shivering can increase metabolic heat production up to 600% above basal level,[11] even in febrile patients. Shivering is not only remarkably uncomfortable, it will also increase intracranial pressure,[12] a particularly undesirable effect in patients with primary neurological and/or posthypoxic brain injury.[9] In one clinical study, shivering patients also showed a decrease from baseline Glasgow Coma Scale (GCS) at 24 hours that may be associated with adverse effects on level of consciousness.[13] Shivering can double or even triple oxygen consumption,[15] causing hypoxemia, myocardial ischemia, and myocardial infarction in high-risk patients because of increased myocardial demands. This has a particularly negative impact on a post cardiac-arrest patient whose heart has just been resuscitated. Therefore, American Heart Association (AHA) strongly recommends the avoidance of shivering during hypothermia induction, normothermia or rewarming periods.[16]

Interventions to Control Shivering

Shivering is most likely to occur when the core temperature is 34-36°C,[17] diminishing with core temperatures below 34°C. Therefore, rapidly cooling patients below 34°C is considered ideal for optimal outcomes.[18]

The ideal goal in shivering management is prevention. Every effort should be made to avoid shivering stimuli and to stop shivering activity when it starts.[19] The strongest evidence in the literature related to the cause and prevention of shivering supports protection of cold-sensitive cutaneous receptors from direct cold contact.[20] Avoiding skin exposure and contact with cold surfaces should be the first step to minimize the risk of shivering in patients undergoing therapeutic hypothermia. Other non-pharmacological treatment of shivering is the application of warm packs to the face and arms.[21] Because only a small portion of the total skin surface is affected, isolated hand or face warming only trivially reduces the shivering threshold, and is often counterproductive during induction and maintenance of therapeutic cooling[22] and fever control.

In current clinical practice, several sedatives, anesthetics, and opiate drugs such as meperidine, buspirone, dexmedetomidine, and/or continuous infusions of magnesium or propofol are utilized to suppress shivering activities. Many of these agents can significantly compromise airway defenses and respiration. Meperidine has traditionally been the drug of choice to treat both postoperative shivering and shaking chills.[23,24] Buspirone has antishivering properties that appear to be mediated centrally by serotonin (5-HT) 1_A receptors. As a single agent, high-dose buspirone has limited use in controlling shivering. However, it has been reported that the combination of meperidine and buspirone synergistically impairs shivering threshold without producing significant sedative effects.[25, 26]

Occasionally, aggressive pharmacologic control of shivering is needed, combining high doses of sedatives and narcotics along with neuromuscular blocking agents (NMBAs). These medications, (e.g. cisatracurium, pancuronium, vecuronium) cause paralysis and thus prevent shivering.[27] They can only be administered to patients who have been intubated and receiving mechanical ventilation. NMBAs are considered the drugs of choice to prevent shivering during therapeutic hypothermia after sudden cardiac arrest,

however their use is not without clinical drawbacks. Large doses of NMBAs not only prohibit neurological examination, an important step in assessing brain-injured patients,[28] but they prolong ventilation which has been associated with Acute Respiratory Distress Syndrome (ARDS) and Ventilator Associated Pneumonia (VAP).[29,30]

Incidence of Shivering with Different Cooling Interventions

Surface cooling with water-filled cooling blankets, ice packs or other attempts to lower skin temperature have shown increased peripheral vasoconstriction and shivering as the body attempts to thermoregulate and conserve body heat.[31] Attempts to directly cool the skin have limited impact on core cooling as mean skin temperature must be reduced considerably before core temperature decreases. Because heat only flows down a temperature gradient, peripheral tissue temperatures must be well below 32°C in order to achieve a core temperature of that value.[32]

When cooling is applied directly to the skin, shivering occurs because of the skin's role as a thermoreceptor. In one clinical study using a skin surface cooling device (Arctic Sun®, Medivance, Louisville, CO), gel pads were applied directly to the skin of critically ill patients and cold water was circulated through the pads, simulating water immersion. Shivering occurred in 86% of febrile, mechanically ventilated, and sedated patients, all of whom were receiving propofol.[33] In a similar study, patients showed a statistically significant higher rate of shivering when cooled with the Arctic Sun skin surface cooling device compared to a traditional surface water circulating cooling blanket (Blanketrol®, Cincinnati Sub-Zero, Cincinnati, OH) which had limited skin contact (39% vs 8%, p= 0.013).[34]

Intravascular temperature management (IVTM™) is effective in transferring or removing heat directly within the core thermal compartment via a central venous catheter. Of note, this procedure has shown significantly less shivering and less thermal gradient between the peripheral and core compartment. Additionally, use of IVTM has shown greater accuracy and control of temperature with little or no overshoot of the target temperature compared with clinical situations where external cooling is used.[35] More recently, a large prospective, randomized, controlled trial demonstrated that the Cool Line® Catheter (Alsius, Irvine, CA) was able to reduce the fever burden in a large number of NICU patients compared to patients who received the standard of care (defined as a combination of antipyretics and external cooling with water-circulating blankets, ice packs and gastric lavage). Only 3.7% of patients who received intravascular cooling catheter experienced shiver.[36] Shivering must be avoided or controlled for post cardiac arrest patients during therapeutic hypothermia. Less sedation and NMBAs were used with IVTM than surface cooling during therapeutic hypothermia in post cardiac arrest patients.[37]

Nursing Experience with Neuroscience Patients using IVTM™ at Tampa General Hospital

Neuroscience nurses often encounter a multitude of challenges managing fevers in their patient population. The efficiency of the cooling modality is critically important to both the patient and the nurse since the therapeutic window to implement neuroprotection is narrow, and "time is brain." The neurological ICU nurse's bedside practice focuses on the ease of initiating cooling therapy, the speed of fever reduction, and maintaining tight temperature control.

Traditional cooling blankets and even the newer skin surface cooling methods are labor intensive and cumbersome for the bedside nurse. They require constant temperature monitoring to prevent over/undershoot of goal temperature, and are often coupled with around the clock anti-pyretic medication administration in an attempt to achieve fever control. At Tampa General Hospital, we experienced many drawbacks to surface cooling including the need for multiple nurses in order to turn the patient for pad placement, increased shivering requiring high doses of sedative and/or paralytic medications, frequent and time consuming skin checks for breakdown, thermal injury, decreased peripheral circulation, and inconsistent temperature control to include overshoot of target temperature. The nurse must also couple surface cooling with adjunctive cold water baths, removal of heated circuit of ventilator, and bedside fans for convective cooling in an attempt to maintain the target temperature. Condensation from the surface cooling onto the patient creates an unsafe environment for the nurses if defibrillation is necessary. Lastly, surface cooling is almost impossible to use in the multi-trauma patient with limited available skin surface area.

Our experience with intravascular temperature management for the last four years in more than 300 patients at Tampa General Hospital has been very positive. Use of the Cool Line® and ICY® Catheters (Alsius, Irvine, CA) provided immediate and consistent temperature control, with minimal peripheral vasoconstriction and shivering. Because these are multi-lumen catheters, other infusion lines are available for the administration of fluids, medications, blood products, and for monitoring central venous pressure. If a patient does begin to shiver, this can often be abetted with a light blanket and placement of warm packs to their hands and/or feet. Intravascular cooling relieves the workload of the bedside nurse by allowing for rapid reduction of temperature without overshoot, and allows for tight temperature control with little to no nursing intervention necessary. This method of cooling also allows for easy controlled re-warming and prevention of rebound intracranial pressure problems. Our neurosurgery and stroke neurology teams have aggressively addressed fever management in our ICU by prophylactically using the intravascular cooling technology. Additionally, we use the Alsius CoolGard 3000®/Thermogard XP™, device to induce hypothermia for the management of elevated intracranial pressure in high grade hepatic encephalopathy and to maintain/warm burn patients during burn debridement surgery.

Conclusion

When considering methods to induce therapeutic hypothermia and fever control to optimize neurological outcomes, shivering should be anticipated as a normal thermoregulatory response which must be prevented and controlled. Intravascular cooling technology has been shown to be more effective and accurate in terms of cooling technologies and shown to be superior in reduction of visible and sub-clinical shivering compared to several methods of skin surface cooling.

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