NURSING INTERVENTIONS IN THE CARE OF PATIENTS UNDERGOING INDUCED HYPOTHERMIA

by

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ABSTRACT

Use of induced hypothermia for the purpose of lowering intracranial pressure and preserving neuronal function has increased as research data reveals a trend of positive outcomes in patients treated with this therapy. Recently induced hypothermia following cardiac arrest due to ventricular fibrillation has been deemed successful. Current research has expanded to evaluate the effectiveness of induced hypothermia as a treatment modality for severe stroke and head trauma. In spite of its efficacy, complications exist with this treatment modality. The purpose of this literature review is to examine potential complications secondary to induced hypothermia and highlight the nurse's role in managing patient care. At the present, patient protocols for induced hypothermia are lacking. The success of treatment is largely dependent on the skill of the healthcare team to prevent further harm and enhance therapeutic outcomes by providing astute assessment and management of complications in patients undergoing induced hypothermia. The desired outcome of this review is to promote integration of research in the development of evidence-based protocols for induced hypothermia.

Key Words

Induced hypothermia, ischemia, cardiac arrest, traumatic head injury, adverse effects, metabolism, metabolic rate, complications, and nursing interventions.

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CHAPTER ONE: Introduction

Background

Cardiac arrest, stroke, and head trauma may lead to compromised cerebral perfusion. Ischemia in the brain triggers an inflammatory response resulting in cerebral damage. Compensatory mechanisms rapidly exhaust oxygen reserves, and the brain transitions into a state of anaerobic metabolism for survival (McKean, 2009). Anaerobic metabolism in response to ischemia precipitates a state of acidosis, which leads to disintegration of cell membranes and cell death (Finkelstein & Alam, 2010). Initiating a state of hypothermia in ischemic situations is believed to decrease the metabolic rate and lower cerebral oxygen demands. Animal studies have shown that a reduction in body temperature decreases the activity of sodium-potassium adenosine triphosphate synthase (ATPase) resulting in a conservation of adenosine triphosphate (ATP) (Finkelstein & Alam, 2010).

In the 1950s, hypothermia was first studied as an approach to preventing neurological dysfunction during cardiac surgery. Animal studies were followed by human trials which largely replicated the evidence that induced hypothermia (IH) lowers intracranial pressure, contributes to improved neurological outcomes, and reduces mortality in populations at risk for neurological dysfunction from cerebral ischemia (Oddo, Schaller, Feihl, Ribordy, & Liaudet, 2006; Zeitzer, 2005; Zhi, Zhang, & Lin, 2003). In 2002, two landmark studies presented supporting evidence that IH was the intervention of choice to preserve neurologic function for patients in post-cardiac arrest (Safer & Kochanek, 2002; The Hypothermia after Cardiac Arrest Study Group, 2002). In addition to cardiac arrest, research has expanded to explore application of IH in cases of

traumatic brain injury, stroke, spinal cord injury, bacterial meningitis, and neonatal hypoxic-ischemic encephalopathy (Finkelstein & Alam, 2010; Bernard & Buist, 2002).

Problem

Research study results point favorably toward ongoing use of IH. This treatment modality, however, encompasses a variety of complex variables that have the potential to adversely affect patient outcomes. The preemptive problem in guiding the care of these patients is the lack of evidence based protocols. Potential for rapid changes in patient condition and serious adverse effects require close monitoring by the nurse and the foreknowledge to anticipate needs and provide appropriate interventions.

Adverse effects of IH include shivering, which increases metabolic demands and oxygen consumption, increased risk for infection, and decreased platelet count and function (McKean, 2009; Finkelstein & Alam, 2010). Electrolyte imbalances, specifically hypokalemia, can develop rapidly during hypothermia and adversely affect heart rate and rhythm among other physiological functions (Finkelstein & Alam, 2010). Cardiovascular alterations, including bradycardia, hypotension, and dysrhythmias can further complicate the patient's status. Risks during rewarming include rebound hyperthermia and increased intracranial pressure (ICP) (McKean, 2009).

The complications associated with IH emphasize the importance of skilled nursing care and management for patients undergoing induced hypothermia. Development of evidence based protocols would promote patient safety by increasing awareness of complications and enhancing anticipation and management of patient needs.

Purpose

The purpose of this thesis was to provide a review of research findings on the physiological complications of IH and implications for nursing practice. Use of IH is increasing, yet patient care protocols are lacking. By increasing nursing awareness, complications can be managed in a way that promotes optimal patient functioning following treatment. This literature review is designed to integrate research findings that may promote development of evidence based patient care protocols.

Method

A synthesis of current research on IH was conducted for this thesis. The databases selected for the literature review included CINAHL, Medline, and the Cochrane Database of Systematic Reviews. Induced hypothermia in adult populations with witnessed cardiac arrest and traumatic head injury were the target populations. However, other forms of neuronal insult were explored as well. Search terms used were induced hypothermia, therapeutic hypothermia, complications, effects, fluid and electrolyte imbalance, arrhythmias, dysrhythmias, and review. Dates of research studies focused on studies published between the years 2000-2010 and included peer-reviewed articles and those written in the English language. Studies conducted in the United States and internationally were used in the literature review. Neonates receiving treatment for encephalopathy and pediatric populations were excluded from this thesis to direct focus toward complication management in adult populations.

Pathophysiology

The control center for thermoregulation is the hypothalamus. Normal body temperature is maintained within a narrow range of 36.6° ± 0.38°C to promote optimal metabolic function. Thermoreceptors located in the skin, hypothalamus, spinal cord, and abdominal organs provide feedback to the hypothalamus on the status of core and peripheral body temperature. Body heat, generated through metabolism and chemical thermogenesis, is distributed to all body tissues via the circulatory system. Limiting or increasing blood flow via vasoconstriction or vasodilation allows the body to regulate temperature, thereby maintaining a state of homeostasis (Polderman, 2004). When the hypothalamus senses a drop in temperature, peripheral vasoconstriction occurs and blood is shunted to the core where heat can be retained. Cooler body temperatures result in increased muscle tone in the form of shivering and contribute to a rise in temperature by increasing metabolic rate (Huether & McCance, 2008). In contrast, vasodilation occurs when body temperature rises, thereby increasing the volume of blood that reaches peripheral circulation for cooling (Polderman, 2004). Hyperthermia accelerates the rate of cell damage. When body temperature reaches 41°C, nerve damage occurs resulting in convulsions and eventual death (Huether & McCance, 2008).

Hypothermia decreases overall metabolic rate and specifically influences cerebral metabolic rate (Polderman, 2004). Desirable effects of hypothermia when ischemia and inflammation are present in the heart or brain are reduction in cellular oxygen demands and slowed rate of cellular death. In fact, donor organs being preserved for transplantation are kept on ice until transplant for this very reason. The neuroprotective quality of hypothermia has

created interest in inducing hypothermia as a therapeutic option for protecting the heart and brain when ischemia is present (Polderman, 2004; Zhi, Zhang, & Lin, 2003).

History of Induced Hypothermia

The first documented use of IH took place in 1937 on a cancer patient in an attempt to decrease the rate of metastatic cellular division. Results in this patient showed no change in the rate of cellular division with IH. Over the following decade IH continued to be used as a treatment for cancer. Patients reportedly tolerated the treatment well, but the therapy failed to slow the rate of cancer progression (Bernard & Buist, 2003). In the early 1940s, IH was first used for the purpose of improving neurological function in patients with head injury. Inducing a mild state of hypothermia during cardiac surgery was first introduced in 1950 and remains the recommended standard of practice for many cardiac surgical procedures because of its neuroprotective quality (Bernard & Buist, 2003). Animal trials began in the 1950s to examine the benefits of hypothermia as a therapeutic treatment and continued into the 1990s. Beginning in the 1990s, IH was specifically examined for its role in increasing central venous oxygen levels and decreasing arterial lactate concentrations in cases of head injury and witnessed cardiac arrest (McKean, 2009). Clinical trials validated the laboratory findings that IH reduced cerebral metabolism and improved neurological outcomes. The release of two landmark studies in 2002 presented IH as the best practice to preserve neurological function in patients following cardiac arrest (Safer & Kochanek, 2002; The Hypothermia after Cardiac Arrest Study Group, 2002). The use of IH to counteract secondary brain injury as a result of trauma and stroke are currently being implemented in some of the larger hospitals in the United States. Induction of IH is also now being considered for use by paramedics prior to hospital admission in patients with witnessed

cardiac arrest. The benefits of pre-hospital hypothermia are currently under investigation. Initial results appear promising, but research in this field is limited. In spite of research in support of this therapy, guidelines for implementing IH are lacking.

CHAPTER TWO: Research Findings

Indications for Treatment

Hypothermia continues to grow in use in contemporary medicine, encompassing a widening spectrum of medical conditions where ischemia is the common denominator. Considered a recommended therapy following witnessed cardiac arrest, IH is now being implemented in some cases of stroke, spinal cord injury, traumatic brain injury, hepatic encephalopathy, and bacterial meningitis with initial research initiatives showing favorable outcomes (Finkelstein & Alam, 2010; Bernard & Buist, 2002).

Cardiac Arrest and Anoxic Brain Injury

Patients who are unresponsive after witnessed cardiac arrest are recommended for IH. Patients admitted to the hospital in cardiac arrest often sustain severe neurologic injury due to the loss of cerebral perfusion (Bernard & Buist, 2003). The American Heart Association (AHA) recommends IH for patients of cardiac arrest whose underlying rhythm is ventricular fibrillation, although IH may also be beneficial in cases of non-ventricular fibrillatory arrest (McKean, 2009). Identification of appropriate cases for IH includes consideration of initiation and duration of resuscitation efforts.

In one study, acceptable criteria for IH included cardiac arrest patients on whom resuscitation efforts were initiated within 15 minutes of arrest (Bernard & Buist, 2003). Oddo et al. (2006) conducted a retrospective study on the impact of IH on 109 patients who were survivors of out-of-hospital cardiac arrest. Heart rhythms for these patients were ventricular fibrillation, asystole, or pulseless electrical activity (PEA). Patients were cooled to 33°C using

external cooling methods. Hypothermia was maintained for 24 hours followed by passive rewarming. During hypothermia, patients were mechanically ventilated and received sedation, analgesia, and paralysis. Study results compared patient outcomes between those treated with IH and patients treated with standard resuscitation methods. Standard resuscitation methods included maintaining mean arterial blood pressure (MAP) between 75-80 mm Hg and maintaining $PaCO_2$ between 36-40 mm Hg. Outcomes were measured using the Glasgow-Pittsburg Cerebral Performance Categories (CPC), which measures patient functionality. Of the patients in the experimental group whose presenting rhythm was ventricular fibrillation (VF), 55.8% had good outcomes (CPC 1-2) in contrast to 25.6% from the control group ($p=0.004$). Poor outcomes (CPC 3-5) were associated with patients from both groups whose presenting rhythm was either PEA or asystole. Results showed direct correlation between IH and improved functionality following cardiac arrest (Oddo, Schaller, Feihl, Ribordy, & Liaudet, 2006).

Traumatic Head Injury

IH was considered an acceptable treatment for traumatic head injury in the 1950s but was abandoned with the development of intensive care units. Treatment for traumatic head injury is directed at minimizing secondary cerebral injury by promoting cerebral oxygenation. IH is now being reconsidered and reintroduced by some hospitals as a treatment modality to manage complications of traumatic head injury (Bernard & Buist, 2003). The premise for IH in traumatic head injury is the decrease in oxygen demands and reduction of ICP afforded by this therapy. Existing research shows IH to be non-therapeutic in traumatic head injury and is largely controversial, although further investigation is warranted (Bernard & Buist, 2003).

A randomized study conducted by Clifton et al. (2001) examined the use of IH on 392 patients admitted to the hospital with severe head injury. Patients in the hypothermia group (n=199) were cooled to 33°C for 48 hours. Although patients in the hypothermia group (n=199) experienced a reduction in intracranial pressure greater than their counterparts in the control group (n=193), there was no difference in functional outcomes between the two groups 6 months following treatment (Clifton et al., 2001). Limitations in methodology may have impacted patient outcomes in this study. Patients with hypoxia or hypotension following initial resuscitation efforts were excluded from this study and may have benefited from reduced oxygen demands and consumption afforded by IH. Furthermore, the mean time for initiating IH was 8 hours following traumatic head injury. Lastly, rewarming was initiated after 48 hours of treatment regardless of intracranial pressure measurements, allowing patients with intracranial hypertension who may have benefitted from additional IH to be rewarmed (Clifton et al., 2004).

Stroke

Although first line treatment for ischemic stroke is aimed at early thrombolytic therapy, treatment for stroke patients is largely supportive in nature. When a stroke occurs, a core of irreversibly dead tissue exists at the site of infarction. Surrounding the infarction is a region of hypoperfused tissue known as the ischemic penumbra. The focus of stroke management is on restoring perfusion to the area of ischemia, thereby minimizing the extent of neurological damage (Bernard & Buist, 2003). IH is an intervention that may be beneficial in this patient population. Increased body temperature is an adverse effect of stroke and is associated with adverse outcomes and increased mortality rates. Preclinical trials have shown improved outcomes in the use of IH in patients with ischemic stroke (Bernard & Buist, 2003).

A prospective study of 390 stroke patients was conducted to see if laboratory findings that cerebral ischemia improved with hypothermia could be validated in humans following ischemic stroke. The study was conducted on patients who were admitted to the hospital within 6 hours of stroke symptom onset and examined the correlation between body temperature on admission and the infarct size, mortality rates, and survivor outcomes. Patients presenting with hyperthermia had increased rates of mortality and poorer outcomes at discharge. Functional outcomes were measured using the Scandinavian Stroke Scale (SSS). Findings affirmed that body temperature was directly related to stroke severity ($p<0.009$), infarct size ($p<0.0001$), mortality ($p<0.02$), and survivor outcomes ($p<0.002$) (Reith et al., 1996). Although this study was conducted prior to clinical application of IH in stroke patients, it validated the neuroprotective nature of IH in this specific population.

A preliminary clinical trial conducted on 50 patients with ischemic stroke used IH to lower ICP and minimize secondary infarction. Hypothermia-induced complications were as follows: 70% experienced thrombocytopenia, 62% had bradycardia, and 48% developed pneumonia. Complications during rewarming occurred in 30% of those studied. In spite of these complications, outcomes found IH to effectively control ICP and reduce functional and neurological deficits. Patient outcomes were evaluated at 4 weeks and 3 months using the National Institutes of Health Stroke Scale (average score of 29 at 4 weeks) and the Rankin Scale (average score of 2.9 at 3 months) (Schwab et al., 2001).

Hepatic Encephalopathy

IH for treatment of hepatic encephalopathy focuses on reducing increased ICP associated with acute liver failure. Animal studies have shown that IH reduced the levels of ammonia-

induced cerebral edema (Bernard & Buist, 2003). A study conducted by Rose et al. (2000) examined the impact of hypothermia on rats with ammonia-related cerebral edema secondary to liver failure. Cooled to 35°C, the experimental group showed decreased ammonia levels in cerebrospinal fluid and lower levels of extracellular glutamate than the control group, providing positive support for the theory that IH is effective in reducing ammonia-induced cerebral edema (Rose et al., 2000).

Two research studies conducted by Jalan et al. (1999; 2001) evaluated the effects of IH in patients with hepatic encephalopathy. In the first study, patients were cooled to 32°-33°C for a mean time of 32 hours. In the second, patients were cooled to the same temperature range 24 hours prior to surgery for liver transplantation and maintained between 32-33°C until the postoperative period. IH was effective in managing increased ICP for patients in acute liver failure awaiting liver transplants who did not respond to standard treatment with mannitol and ultrafiltration. Positive outcomes were a reduction in ICP by an average of 54 mm Hg, decreased arterial ammonia levels, and decreased cerebral uptake of ammonia. Neither study documented any long-term adverse effects of hypothermia (Jalan, Damink, Deutz, Lee, & Hayes, 1999; Jalan & Damink, 2001).

Bacterial Meningitis

IH has the potential to lower ICP, blunt any hyperemic response in cerebral blood flow, and decrease leukocyte count in cerebral spinal fluid (CSF) (Bernard & Buist, 2003). Animal studies have been conducted to explore the possibility of using IH in the treatment of bacterial meningitis. Pneumococcal meningitis was introduced to rats that were then treated with IH to assess the impact of IH on cerebral blood flow and ICP. The study found that IH lowered

cerebral blood flow and ICP in addition to decreasing tumor necrosis factor and cerebrospinal fluid leukocyte count (Angstwurm et al., 2000). Clinical trials have not been started, but application of IH in this patient population may be considered (Bernard & Buist, 2003).

Spinal Cord Injury

A limited number of animal studies analyzed the possibility of using IH to improve outcomes with spinal cord injury. While direct damage to the spinal cord is likely irreversible, IH may be useful in preventing secondary injury related to altered perfusion, ischemia, and inflammation (Finkelstein & Alam, 2010). Animal trials in the 1960s and 1970s looked for correlations between IH and improved outcomes with spinal cord injury. Results were inconclusive with only limited studies showing benefit of IH therapy. Animal studies on the use of IH for spinal cord injury resumed a decade ago as IH was becoming more widely used to treat cerebral inflammation and ischemia. Study results showed that IH provided a measure of cerebral protection, but improvement of functional outcomes was lacking (Inamasu, Nakamura, & Ichikizaki, 2003). Additional preclinical studies need to be completed before therapeutic benefits can be determined (Finkelstein & Alam, 2010).

Exclusion Criteria

In a literature review, McKean (2009) concluded that IH is not recommended for patients with life-threatening arrhythmias or those who are pregnant due to the lack of available research among these patient populations (McKean, 2009). Primary coagulopathy was also a contraindication for this therapy because of the potential for IH to further impede coagulation (McKean, 2009). Immunosuppression and sepsis were also considered contraindications for IH

due to increased risk for infection. IH was not recommended for persons with cardiac arrest secondary to respiratory compromise, or for patients with a systolic blood pressure of less than 90 mm Hg (McKean, 2009). Hypotension and decreased cardiac output have the potential to adversely affect cerebral perfusion pressure (CPP), which may be difficult to manage in patients presenting with hypotension (Huether & McCance, 2008). Similar exclusion criteria were listed in literature presented by Hoag Memorial Hospital Presbyterian. Exclusion criteria were subdivided into two groups: absolute and relative. Absolute exclusion criteria were listed as pregnancy, coma resulting from drug overdose, status epilepticus, and terminal illness. Relative exclusion criteria included preexisting coagulopathy, thrombocytopenia, preexisting hypotension, and bradycardia. Hoag Memorial established its exclusion criteria based on a review of functional outcomes of patients treated with hypothermia following cardiac arrest (Pyle, Pierson, Lepman, & Hewett, 2007).

Methods for Inducing Hypothermia

Noninvasive Cooling Measures

Cooling by conduction utilizes a cooling blanket that circulates chilled water. Cooling by convection uses fans to circulate cool air over the patient. These cooling methods generally produce slower reduction in core body temperature than invasive techniques. One study showed the use of cooling blankets to reduce body temperature by 1°C in a mean time of 178 minutes. Fans reduced core body temperature by a single degree in 142 minutes (Bernard & Buist, 2003). Additional methods of noninvasive cooling are alcohol baths, ice packs placed on the torso, groin and axillae, and immersion in ice water. Ice packs cool body temperature at an average rate of

0.9°C per hour. Immersion in ice water is effective in lowering body temperature but is difficult to implement in any hospital or pre-hospital setting (Bernard & Buist, 2003).

The above noninvasive methods of inducing hypothermia tend to be labor intensive for healthcare workers implementing the treatment and require close monitoring of the patient's temperature which can be erratic and unpredictable. Nursing judgment plays a significant role in the care of patients receiving noninvasive cooling since treatment requires continuous adjustment based on the patient's response to the intervention. Noninvasive cooling methods are associated with greater risk for accidental overcooling and excessively rapid rewarming. An additional concern is the prolonged time it may take to achieve target body temperature by surface cooling methods (McKean, 2009).

An exception to the aforementioned noninvasive cooling methods are external cooling devices such as the Arctic Sun. Advances in technology have led to the development of noninvasive cooling methods that regulate the rate at which body temperature is lowered and raised and provide continuous feedback on core body temperature. Arctic Sun uses adherent gel pads attached to the patient's torso and lower extremities to circulate chilled fluids. Arctic Sun contains a temperature feedback-control mechanism that improves thermoregulation by allowing temperature to be raised or lowered on demand. An additional benefit is the ability to reach target core body temperature more rapidly than with other external cooling devices (Heard et al., 2009).

A randomized control trial completed in 2009 compared the use of Arctic Sun to ice and cooling blankets for inducing hypothermia following cardiac arrest (Heard et al., 2009). The percentage of participants in the Arctic Sun group who reached target core body temperature

within four hours of induction was 71% in contrast with 50% for the control group ($p = 0.12$). Neurological function and survival rates were slightly higher in the Arctic Sun group at 46% compared with 38% in the control group ($p = 0.6$) (Heard et al., 2009). While cooling blankets and ice do effectively cool patients, improved technology, such as Arctic Sun, provide greater efficiency in cooling with the added benefit of precise temperature control (Heard et al., 2009).

Invasive Cooling Measures

Rapid infusion of chilled IV fluids and intravascular cooling catheters are invasive methods of inducing hypothermia. Intravascular catheters inserted via a central vein into the vena cava circulate chilled fluid from an external refrigerated pump through a closed catheter. Preliminary research shows intravascular catheters to be an effective technique for cooling core body temperature. Risks associated with this cooling method include bleeding, deep vein thrombosis, vascular puncture, and infection, and must be weighed against therapeutic benefits (Bernard & Buist, 2003).

A pilot study by De Georgia et al. (2004) assessed the use of intravascular cooling catheters in patients following ischemic stroke. Eighteen participants were included in the study. Intravascular cooling was chosen because it facilitated quick access and rapid cooling. Target core body temperature of 33°C was attained within 2 hours of catheter placement for 14 of the 18 participants, in contrast with the standard 4-6 hours required by other cooling methods. Hypothermia was successfully maintained for 24 hours. Occurrence of complications was comparable to those in the control group and patients remained hemodynamically stable for the duration of treatment. Participants who experienced complications had similar variables including advanced age and greater stroke severity. One exception was a patient who

experienced retroperitoneal hemorrhage. This patient was a poor candidate for intravascular cooling due to administration of antithrombolytics and anticoagulants prior to catheter insertion. Results of the study showed that hypothermia reduced the size of infarct growth, but functional outcomes were the same in both the experimental and control groups (De Georgia et al., 2004).

Regional Cooling Measures

Partial body hypothermia uses a regional cooling device such as a cooling helmet to provide localized brain cooling. Theoretically, providing therapeutic benefits of cooling without subjecting the body to the systemic effects of whole body hypothermia could optimize treatment outcomes by minimizing the risk for complications. Research, however, is conflicting and use of cooling helmets needs to be further researched. Some studies have shown that cooling helmets are useful in providing regional hypothermia; however, after 24 hours of use, they begin to reduce core body temperature (Bernard & Buist, 2003; Finkelstein & Alam, 2010). An article by Kuffler (2010) supported the use of head cooling in combination with mild total body hypothermia to minimize the complications associated with systemic, moderate IH.

Potential Adverse Effects of Induced Hypothermia

Shivering

Core body temperature is tightly regulated by the hypothalamus between 36.5°-37.5°C. Changes outside normothermic range initiate thermoregulatory responses within the hypothalamus. Hypothermia triggers the body's compensatory mechanisms to increase heat production. One mechanism is increased muscle tone in the form of shivering, which causes the body to generate heat two to five times the normal rate (Mahmood & Zweifler, 2007). In the

induced hypothermic state, shivering potentiates an increase in metabolic demands and cellular oxygen consumption. This is most likely to occur during the induction and rewarming phases of IH (McKean, 2009). Increased metabolic rate and oxygen consumption are counterproductive during IH, and must be overcome to increase the efficacy of treatment (Mahmood & Zweifler, 2007).

Pharmacologic agents are used to suppress shivering and include paralytics and nonvolatile anesthetics. Propofol (Diprivan) is a benzodiazepine that impairs vasoconstriction and is useful as a sedative and anxiolytic for mechanically ventilated patients (Sole, Klein, & Moseley, 2009). Propofol is the medication of choice to prevent shivering in IH patients (Mahmood & Zweifler, 2007). Meperidine (Demerol) is an opioid used as an anesthesia adjunct and has also been shown to suppress shivering during IH (Deglin & Vallerand, 2009; Mahmood & Zweifler, 2007). Other medications used for patient sedation and comfort are fentanyl (Sublimaze) and midazolam (Versed) (Deglin & Vallerand, 2009). Neuromuscular blocking agents provide therapeutic paralysis and include atracurium (Tracrium) and succinylcholine. Prior to administering paralytics and sedatives, the patient must be intubated to maintain airway patency and placed on a ventilator until the use of sedation is suspended (Sole, Klein, & Moseley, 2009). Current research validates the suppression of shivering from the onset of IH through the duration of treatment until the body is rewarmed and returned to a normothermic state (McKean, 2009).

Nursing assessment should include monitoring for signs of shivering such as increased respiratory rate and muscle tensing of chest or facial muscles. Other signs of shivering may include a reduction in venous oxygen saturation or a static tracing on the electrocardiogram

(ECG). Although it may be difficult to obtain due to peripheral vasoconstriction, a train of 4 should be established on the patient and monitored throughout the sedation period (McKean, 2009). Nursing assessment may be enhanced by the use of bispectral index monitoring to provide optimal sedation and reduce the risk of oversedation or excessive paralysis.

Cardiovascular Changes

The body's normal response to hypothermia is decreased heart rate, increased cardiac output, and peripheral vasoconstriction in an attempt to shunt blood to the vital organs and body core in an effort to conserve heat. Cardiovascular changes occur in two phases as body temperature lowers to a hypothermic state. During the induction phase heart rate is increased, thereby increasing cardiac output. Blood pressure also rises due to increased systemic vascular resistance. This compensatory mechanism allows greater volumes of warm blood to be circulated throughout the body to warm the tissues. As hypothermia persists and core body temperature lowers, bradycardia develops, afterload decreases, and cardiac oxygen demands are reduced (Polderman, 2004). Hypotension and dysrhythmias are two adverse cardiovascular effects seen during this phase of IH. The use of sedatives or paralytic agents compounds the body's natural response causing bradycardia and increased systemic vascular resistance (Bernard & Buist, 2003).

To compensate for increased arterial pressure, the kidneys respond with diuresis in an effort to lower blood pressure. Within hours, the body may lose large amounts of water (Guyton, 1991). Ensuing hypotension and decreased cardiac output have the potential to adversely affect CPP. If pressure in the vasculature of the brain is inadequate, oxygenation of cerebral cells will be insufficient (Huether & McCance, 2008). An injured brain is highly sensitive to changes in

blood volume and pressure and cannot survive long without adequate oxygenation (Finkelstein & Alam, 2010). Inadequate CPP leads to cerebral ischemia within three minutes and cerebral infarction in five minutes (Sole, Klein, & Moseley, 2009).

Hemodynamic monitoring is critical in these patients to assess fluid volume status and maintain adequate cerebral perfusion. Invasive hemodynamic monitoring devices include arterial lines, central venous catheters, and pulmonary artery catheters. Complications associated with these devices are infection, bleeding, pneumothorax or hemothorax, thrombosis, cardiac dysrhythmias, and pericardial tamponade. Proper management of arterial catheters includes zeroing the transducer at the start of every shift, when the patient is repositioned or moved, and if there is a significant change in patient status (Sole, Klein, & Moseley, 2009).

Fluid replacement is important to ensure adequate fluid volume and cerebral perfusion. The fluid of choice during hypothermia is 0.9% normal saline. Normal saline helps maintain optimal CPP and mean arterial pressure (MAP) (Sole, Klein, & Moseley, 2009).

Monitoring vital signs provides the nurse with critical information regarding the patient's response to hypothermia. Continuous blood pressure monitoring is essential in IH patients. Arterial catheters provide accurate measurement of systemic blood pressure and are the modality of choice for patients undergoing IH (Sole, Klein, & Moseley, 2009). Cerebral oxygenation monitoring is another valuable tool that allows the healthcare team to assess cerebral perfusion by the placement of a jugular venous bulb via the internal jugular vein. The monitor measures the level of oxygen saturation of the blood as it leaves the brain (Sole, Klein, & Moseley, 2009). Research on appropriate CPP levels is conflicting. During induced hypothermia one study suggested maintaining a CPP of 60 mm Hg (Finkelstein & Alam, 2010). According to Huether

and McCance (2008), a CPP of more than 70 mm Hg is recommended. Normal adult CPP is between 60-100 mm Hg. Decreased MAP or increased ICP causes CPP to be lowered. During hypothermia, a lower CPP may be acceptable because of decreased metabolic rate (Sole, Klein, & Moseley, 2009).

Hypothermic patients are also at risk for developing dysrhythmias. These can occur in response to decreased coronary perfusion and increased myocardial irritability. Without oxygen, the myocardium becomes fatigued and escape mechanisms replace normal conduction pathways. Although the heart can withstand episodes of ischemia for longer periods than the brain before infarct occurs, adequate perfusion is essential for proper function (Sole, Klein, & Moseley, 2009).

Hypokalemia is a common occurrence during IH due to cellular shifts of fluids and electrolytes. Potassium is essential for cellular metabolism and plays an essential role in neuromuscular function. Hypokalemia impacts resting membrane potential and specifically impacts the electrical conductivity of the myocardium by reducing cellular excitability. ECG changes associated with hypokalemia include flattened T waves, emergence of U waves, and peaked P waves (Lewis, Heitkemper, Dirksen, O'Brien, & Bucher, 2007). Dysrhythmias may develop at any point during IH, but are most likely to occur during the induction and rewarming phases (McKean, 2009).

Research findings showed the risk for dysrhythmias increased when body temperature fell below 30°C (McKean, 2009; Polderman, 2004). The most common ECG changes seen in patients with temperatures below 32°C were first-degree heart block and prolonged QT segment (Finkelstein & Alam, 2010). Tachydysrhythmias, most commonly atrial fibrillation, were

associated with temperatures between 30°-33°C. In deep, hypothermic states (less than 28°C), ventricular dysrhythmias were problematic (Finkelstein & Alam, 2010).

Nurses managing the care of IH patients should be aware of the risk for hypothermia-induced dysrhythmias and monitor for changes in heart rate and rhythm. Early recognition of ECG changes followed by early intervention reduces strain on the heart and helps reduce risk for more serious dysrhythmias (Sole, Klein, & Moseley, 2009). Treatment of dysrhythmias involves standard medical interventions, although dysrhythmias may be more difficult to treat due to the impact cold temperatures have on the myocardium. Awareness of alterations in drug metabolism is an important factor in treating dysrhythmias (Finkelstein & Alam, 2010; McKean, 2009). Hypothermic patients may require lower or less frequent doses of antiarrhythmic medications due to slower drug metabolism (McKean, 2009).

Coagulopathy

Hypothermia depletes clotting factors and clotting proteins and activates both intrinsic and extrinsic pathways causing the release of procoagulants. Consumption of clotting factors in the microcirculation increases systemic risk for bleeding. Uncontrolled clot formation throughout the microvasculature also increases the risk of organ ischemia and damage (Sole, Klein, & Moseley, 2009). Methods for assessing coagulation include platelet count, fibrinogen level, fibrin degradation products (FDPs), international normalized ratio (INR), prothrombin time (PT), and activated partial thromboplastin time (aPTT) (Sole, Klein, & Moseley, 2009).

The studies under review used PT and aPTT as the methods of choice for assessing risk for bleeding in hypothermic patients. Preclinical trials showed varying impact of IH on bleeding tendencies. A meta-analysis revealed only slight increase in PT between the hypothermic and

normothermic groups by 0.02 seconds (p=0.7) (Harris, Colford, Good, & Matz, 2002). One of four research studies conducted by a traumatic brain injury group showed an increased PT during IH (Finkelstein & Alam, 2010). According to Polderman (2004), IH was not associated with increased intracranial bleeding in clinical trials for patients with stroke, subarachnoid hemorrhage, or traumatic brain injury. Two human trials showed no increased risk for bleeding during mild to moderate hypothermia (28°-36°C) (McKean, 2009, Clifton et al., 2001). The risk for bleeding increased when body temperature was decreased to below 28°C (Finkelstein & Alam, 2010).

Assessment for signs and symptoms of coagulopathy should include monitoring for changes such as cyanosis of the fingers, toes, and nose, and delayed capillary refill. Oliguria, a possible sign of coagulopathy, may be difficult to monitor in patients who are vasoconstricted due to hypothermia. Patients should also be closely monitored for signs of bleeding such as gingival bleeding, petechiae, echymosis, oozing at venipuncture sites, and stool positive for occult blood (Sole, Klein, & Moseley, 2009).

Infection

Hypothermia inhibits proinflammatory responses by decreasing the number of circulating leukocytes, suppressing phagocytosis, and decreasing the synthesis of proinflammatory cytokines (Polderman, 2004). Hypothermia places patients at increased risk for developing infection and sepsis. Meningitis, pneumonia, and wound infections or prolonged wound healing are the most common infections associated with induced hypothermia (Finkelstein & Alam, 2010). Immunosuppression, in addition to intubation and mechanical ventilation, place ventilated patients at greater risk for developing ventilator-associated pneumonia (VAP) (Sole, Klein, &

Moseley, 2009). Hypothermia impairs ciliary function of the upper airways, thus inhibiting one of the body's natural defense mechanisms (McKean, 2009). Wound infections associated with IH are attributed to diminished leukocyte function and hypoperfusion resulting from hypothermia-induced vasoconstriction (Polderman, 2004).

Infection, specifically pneumonia, occurred in higher numbers when the hypothermic period lasted greater than 48 hours (Polderman, 2004). Incidence of hypothermia-related pneumonia was one of the documented complications found in 11 trials (n=559). Patients in hypothermic groups had a moderately higher risk for acquiring pneumonia than the control groups, but in these studies the increase was not shown to be significant (confidence interval 0.95-1.91) (Sydenham, Roberts, & Alderson, 2009). Infections that did occur responded well to treatment once the patients were rewarmed (Finkelstein & Alam, 2010).

Strategies to reduce the risk for infection during IH include maintaining the head of the patient's bed between 30°- 45°C if not contraindicated by patient condition. Raising the head of the bed helps reduce risk for aspiration and prevent gastric reflux. Suctioning of mechanically ventilated patients and providing oral hygiene are important aspects of nursing care to reduce the risk for pneumonia (Sole, Klein, & Moseley, 2009). Laboratory indicators for infection are white blood cell (WBC) count with differential and blood cultures and should be routinely monitored throughout the hypothermic period (Greer, 2005). Skin care and frequent turning help maintain skin integrity (McKean, 2009). Additional protective measures include antibiotic prophylaxis and decontamination of the gastrointestinal tract (Polderman, 2004). IH masks elevation in body temperature associated with infection, placing greater responsibility on nurses to use proactive

measures to prevent infection and astute assessment to detect the earliest signs of infection (McKean, 2009).

Fluid and Electrolyte Imbalances

Induction of hypothermia causes a significant shift of electrolytes from the extracellular fluid (ECF) to the intracellular fluid (ICF) causing serum levels of potassium to drop (Huether & McCance, 2008). In addition to the intracellular shift, decreased renal reabsorption of solutes leads to increased secretion of electrolytes (Bernard & Buist, 2003). A reduction in calcium, potassium, sodium, magnesium, and phosphate are all possible findings due to shifts in electrolytes and urinary diuresis (Finkelstein & Alam, 2010). One of the most concerning changes is a shift of potassium into the cells resulting in extracellular hypokalemia. Potassium plays a vital role in electrical impulse conductivity, and has specific impact on the myocardium. Cardiac dysrhythmias, including bradycardia and ECG changes such as a depressed ST segment, flattened T wave, and prolonged QT segment, are findings associated with hypokalemia (Sole, Klein, & Moseley, 2009). Hypomagnesemia is a finding associated with poor patient outcomes when experienced in conjunction with neurological injury and may be seen with hypothermia (Finkelstein & Alam, 2010).

Electrolyte monitoring is essential in patients undergoing IH. ECG readings should be closely monitored for changes consistent with hypokalemia. Replacement of potassium levels requires diligent and frequent monitoring as levels can drop dramatically and rapidly throughout the hypothermic phase. There are two options for replacing potassium during hypothermia. The first is to maintain normal serum potassium levels. The second is to replace measured losses of potassium. In one research study, patients whose potassium was maintained at a normal serum

level during hypothermia, developed hyperkalemia during the rewarming phase and more frequently experienced dysrhythmias. Patients whose potassium was replaced according to measured losses stayed within normal levels during rewarming and did not experience dysrhythmias (Koht, Cane, & Cerullo, 1983). While it is commonly thought that electrolyte levels should be maintained in the high range of normal, it may be wise to replace only measured losses in the case of potassium (Finkelstein & Alam, 2010).

Magnesium plays an important role in neuromuscular excitability. Effects of hypomagnesemia include hyperreflexia, muscle cramps, convulsions, tachycardia, and hypotension (Huether & McCance, 2008). Magnesium replacement during IH may benefit the patient by preventing vasospasm and aid in regulating the body's transition during reperfusion. According to Finkelstein and Alam (2010), hypomagnesemia during induced hypothermia was associated with increased mortality and poor outcomes in patients with neurological injury. In cases of traumatic brain injury, supplementing magnesium resulted in improved patient outcomes (Finkelstein & Alam, 2010).

In addition to replacing electrolytes, slow infusions of hypertonic saline may be administered if electrolyte levels are critically low and electrolyte levels are not improving sufficiently with replacement therapy (Sole, Klein, & Moseley, 2009).

Adverse Metabolic Effects

During periods of hypothermia, the body functions in a hypometabolic state. Metabolic rate decreases 5%-7% for each degree Celsius that body temperature is lowered (Polderman, 2004). Decreased metabolism impacts cellular uptake of insulin by increasing insulin resistance and decreasing pancreatic secretion of insulin. Both of these factors place the body at risk for

developing hyperglycemia (Finkelstein & Alam, 2010). Hyperglycemia increases susceptibility to infection in vulnerable patients whose immune function is suppressed (Polderman, 2004).

There are no clear guidelines for the management of hyperglycemia during IH. Two studies of patients with traumatic brain injury linked tight glucose control to improved patient outcomes. Higher amounts of insulin were required during the induction phase of hypothermia to maintain blood glucose within therapeutic range (Polderman, 2004). A clinical trial and meta-analysis, however, showed that tight glucose control involving frequent insulin administration appeared to adversely impact patients and possibly contributed to increased mortality (Finkelstein & Alam, 2010). A concern with aggressive administration of insulin during the hypothermic phase is rebound hypoglycemia during rewarming (Polderman & Ingeborg, 2009). A further consideration for glycemic control is the impact of hyperglycemia on blood viscosity. Hyperglycemia increases blood viscosity, which decreases perfusion (Sole, Klein, & Mosely, 2009). Critical care nurses need to diligently assess blood glucose levels in these patients and communicate with physicians to provide safe management of this potential adverse effect.

Research studies showed hyperglycemia to be the single greatest metabolic abnormality associated with IH (Polderman, 2004; Finkelstein & Alam, 2010). Additional metabolic risks do exist, however, including pancreatitis and impaired liver function that result in elevated liver enzymes and amylase levels (Finkelstein & Alam, 2010). Hypothermia can lead to acidosis due to increased levels of fatty acids, ketones, and lactate. This places the bowel at increased risk for ulcer formation. Bowel ischemia due to hypoperfusion reduces gastrointestinal (GI) motility and increases the risk of ileus and ulcer formation (Polderman, 2004). ICU protocols for GI antibiotic

prophylaxis should be closely adhered to in IH patients (Finkelstein & Alam, 2010; Oddo et al., 2006).

Reduced Drug Metabolism

The effects of hypothermia significantly impact pharmacokinetics and pharmacodynamics. Enzymes necessary in the metabolism of most medications are highly sensitive to changes in temperature. Drug clearance during hypothermia is decreased causing plasma drug levels to be higher for prolonged periods of time, requiring lower doses of medication to achieve the desired effects (Finkelstein & Alam, 2010; Polderman, 2004). Consequently, standard dosing schedules may lead to plasma drug accumulation and toxicity. Critical care nurses need to be cognizant of this fact and monitor for adverse effects of drug toxicity such as respiratory depression and changes in heart rate or blood pressure. Communication with the healthcare team, specifically physicians and pharmacists, is imperative as drug doses and frequency of medication administration may need to be adjusted to prevent toxicity (Lewis et al., 2007). Medications of specific concern for hypothermic patients are opiates, sedatives, vasoactive drugs, nitrates, beta blockers, and neuromuscular blocking agents (Polderman & Ingeborg, 2009).

Risks During Rewarming

Reperfusion injuries are exacerbated during the rewarming phase of IH. The brain's shift to anaerobic metabolism during initial loss of perfusion causes toxins to accumulate in the brain (Zeitzer, 2005). Free radicals, cytokines, catecholamines, and nitric oxide are released when reperfusion occurs (McKean, 2009). The presence of these chemicals furthers the injury when

the body transitions from a hypothermic to normothermic state. Electrolyte imbalances, specifically hyperkalemia, may occur during the rewarming phase as electrolytes shift from intracellular spaces to the extracellular. Cellular uptake of insulin also increases as the body returns to normothermia placing the patient at risk for hypoglycemia (Polderman & Ingeborg, 2009). Signs of medication toxicity should be closely assessed during the rewarming phase when the body's metabolism increases, thereby enhancing the uptake of pharmacotherapies (Finkelstein & Alam, 2010). Other concerns include rebound hyperthermia and hypotension. Rebound hyperthermia may increase cerebral edema and potentiates the risk for mitochondrial damage and cell death, thereby reversing the therapeutic benefits of IH (McKean, 2009). The rate at which body temperature is returned to a normothermic state must be monitored closely to reduce the risk of recurring cerebral edema and increased intracranial pressure (McKean, 2009).

To prevent adverse effects of rewarming, studies suggest gradually increasing body temperature by 0.5°-1.0°C per hour (McKean, 2009). Patient monitoring during the rewarming phase should include assessment for electrolyte imbalances and dysrhythmias. Blood glucose levels need to be monitored for hypoglycemia. Blood gasses should be assessed and oxygen levels titrated to prevent oxidative stress from occurring as excessive oxygen is detrimental in cases of post-ischemic neurological dysfunction. Vital signs should be continuously monitored for changes in blood pressure, heart rate, and temperature. Shivering should continue to be suppressed until normothermia is reestablished (McKean, 2009). Nurses should watch for ECG changes such as bradycardia, first-degree heart block, and prolonged QT segment (Finkelstein & Alam, 2010).

Induced Hypothermia in the Prehospital Setting

Early implementation of IH has been shown to be effective in improving neurological outcomes and minimizing secondary injury from ischemia. This finding has led to initiation of this therapy by emergency medical service (EMS) personnel during prehospital transport (Cabanas, Brice, Maio, Myers, & Hinchey, 2011). Numerous variables exist in the field, each with the potential to impact effectiveness of IH delivery and management. Transport time may range from less than ten minutes in a well-serviced city to more than an hour in rural settings. EMS personnel possess a broad range of certification and experience. Some operate as volunteers, others work full-time. Certification includes basic EMTs, paramedics, and in some states, intermediate level EMTs. Equipment and protocols vary between providers and areas of service. In order to analyze the benefits of IH, the risks of initiating this therapy in the prehospital setting must be considered.

As outlined in this literature review, there are a number of possible adverse effects associated with IH. Effectiveness of therapy is dependent on proper management of therapy-induced complications. In the field, paramedics do not have access to resources and equipment necessary for monitoring these complications. A question to consider is could potential harm be done by initiating this therapy in the field where there are limitations to what can be monitored? One variable that comes into play is the duration of patient transport. During a short transport time (<15-30 min.) it would be unlikely for body temperature to be lowered to the point where complications would present a problem. On the other hand, a longer transport time (>30 min.) may place the patient at risk for developing complications that could not be monitored until hospital arrival. This raises a further question as to how EMS personnel should best prioritize

their time. Performing lifesaving interventions, such as securing an airway, stopping hemorrhage, immobilizing the C-spine or injured extremities, placing one or more IVs, obtaining vital signs, and possibly ventilating a patient or performing CPR require full involvement by paramedics and EMTs making it difficult to initiate other interventions such as IH.

The underlying question that needs to be determined is do the benefits of IH outweigh the risks? Does initiation of IH in the field contribute to positive neurological outcomes, or would the patient be better served by providing a speedy transport with preparatory communication to emergency department staff to enable early intervention in the hospital where the patient can be adequately monitored and assessed for complications? In order to answer these questions, further research needs to be conducted on the use of IH in the prehospital setting (Cabanas, Brice, Maio, Myers, & Hinchey, 2011).

CHAPTER THREE: Conclusion

Implementation of Induced Hypothermia

Plan of Care

IH occurs in three phases: induction, hypothermic, and rewarming. Before the induction phase is initiated, however, several factors should be determined. Appropriate patients should be identified based on guiding criteria. IH may be beneficial for patients following cardiac arrest, traumatic head injury, stroke, hepatic encephalopathy, bacterial meningitis, spinal cord injury, and neonatal encephalopathy. IH is not recommended in patients with life-threatening arrhythmias or those who are pregnant. Preexisting coagulopathy, immunosuppression, or sepsis are also contraindications for IH. Use of IH with a systolic blood pressure of less than 90 mm Hg or a history of recent surgery may place the patient at increased risk for complications and should be avoided. (McKean, 2009).

A second consideration is unit staffing and resources. Physiological status may change dramatically and rapidly during IH. A one-to-one nurse to patient ratio may be required to ensure patient safety and should be considered when making patient assignments.

Better patient outcomes are associated with early initiation of IH. Studies concur that induction of hypothermia should be implemented within 2-5 hours of anoxic brain injury or cardiac arrest (Oddo et al., 2006). Studies currently suggest a target body temperature of 32°-34°C for a period of 24-48 hours (McKean, 2009). If IH is rapidly initiated, a period of 12 hours may be sufficient to produce therapeutic results. The rate at which body temperature is lowered

varies by cooling method. The time it should take to reach target temperature is recommended between 6-8 hours (Greer, 2005).

Vigilant nursing assessment is critical since patient status can change rapidly during the induction phase. Nurses should monitor for signs of shivering such as increased respiratory rate and muscle tensing of chest or facial muscles. Other signs of shivering may include a reduction in venous oxygen saturation and a static tracing on the ECG (Mahmood & Zweifler, 2007). Endotracheal intubation and mechanical ventilation may need to be established, requiring administration of opiates, paralytics, and neuromuscular blockers for providing patient comfort and enhancing therapeutic benefits (Mahmood & Zweifler, 2007). Bispectral index monitoring helps ensure optimal sedation and reduces the risk of oversedation.

During the induction phase vital signs should be closely monitored. Arterial lines facilitate continuous blood pressure monitoring. A systolic blood pressure greater that 90 mm Hg and mean arterial pressure (MAP) of greater than 80 mm Hg is desirable during IH (Greer, 2005). Temperature monitoring may be obtained via pulmonary artery temperature probes or bladder temperature probes. ECG monitoring with attention toward bradycardia should be performed. A secondary temperature monitoring device may be advised in patients with bladder probes, since the reading of core body temperature is dependent on adequate urine output (Greer, 2005).

Measures should be taken early to reduce patients' risk for infection. If tolerated, the head of the patient's bed should be elevated between 30°-45°. Suctioning secretions and providing oral hygiene are important aspects of nursing care to reduce the risk for pneumonia (Sole, Klein, & Moseley, 2009). White blood cell (WBC) count should be monitored. Skin care and frequent

turning help maintain skin integrity (McKean, 2009). If surface cooling methods are used, skin should be assessed every 2-6 hours for thermal injury (Bessman, Talavera, Setnik, Halamka, & Alder, 2010). Additional protective measures include antibiotic prophylaxis and antibiotic decontamination of the gastrointestinal tract (Polderman, 2004).

Fluid and electrolyte shifts begin during the induction phase. Hemodynamic monitoring and fluid replacement should be established at the beginning of treatment. Chemistry panels, CBC, and blood glucose levels should be obtained at 0, 12, and 24 hours. Blood cultures and ABG measurements are also recommended at 12 hour intervals (Greer, 2005).

Ongoing assessment should continue during the hypothermic period. During the hypothermic period each of the above functions requires close assessment. In addition to the assessments performed during the induction phase, timely monitoring and replacement of electrolytes are essential to prevent further complications. Potassium levels are of specific concern and need to be frequently assessed (Finkelstein & Alam, 2010). Medication administration for hypothermic patients may require lower than normal doses due to hypometabolism. Standard dosing schedules may lead to plasma drug accumulation and toxicity. Critical care nurses need to be cognizant of this potential effect and monitor for adverse effects of drug toxicity such as respirator depression and changes in heart rate or blood pressure (Lewis et al., 2007).

The rewarming phase may be passive or controlled. Passive rewarming involves the removal of cooling devices such as blankets or ice packs. Controlled rewarming is achieved by programming a cooling device such as the Arctic Sun or Cool Guard to the desired warming rate. For either method, the recommended rate of warming is 0.3°-1.0°C every hour until core body

temperature returns to 36°C. In general, normothermia should be achieved within 6-8 hours (Bessman et al., 2010).

During rewarming, the risk increases for rebound hyperkalemia, hyperthermia, hyperglycemia, and hypotension (McKean, 2009; Polderman & Ingeborg, 2009). Rewarming may be the most critical phase of IH as therapeutic effects may be reversed if complications and patient condition are poorly managed (Greer, 2005). The use of sedation and paralytics should continue until the patient achieves target, normothermic core body temperature. Medication doses may need to be adjusted as metabolism increases (Polderman & Ingeborg, 2009).

Nursing Education and Research

Efficacy of treatment is largely dependent on early implementation of IH in patients meeting criteria for treatment. Nurses should be trained in identification of patients who may benefit from IH to promote early intervention. The variety of complications associated with IH requires proficiency in nursing care. Continuing education on methods for inducing hypothermia and the associated interventions that may be required to maintain patient stability can play a vital role in preparing nurses to manage patient care.

Additional research needs to be conducted to assess therapeutic benefits in populations where clinical trials are lacking, including head trauma, stroke, and spinal cord injury. Specific focus on management of complications should be addressed to promote development of recommendations for best practice. Rather than focusing on complications in general, it may be beneficial to conduct research studies addressing individual complications.

Summary

Hypothermia is documented to improve outcomes following anoxic brain injury, cardiac arrest, and stroke. As exploration in this field advances, IH is being considered to treat specific cases of traumatic head injury, bacterial meningitis, spinal cord injury, and hepatic encephalopathy. Animal studies are well-documented and show promising results. Human clinical trials, however, are limited and small in number with variable quality. Results of clinical trials show varying support for IH, and further research needs to be conducted to validate these findings. Studies to date show correlations between IH and physiological complications. As future studies are performed, medical personnel need to be informed of these adverse effects, so care is properly managed and therapeutic benefits are maximized.

APPENDIX: Table of Evidence

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Author / Year / Title	Study / Purpose	Population Studied	Complications Evaluated	Results / Findings	Conclusion / Implications
Bernard & Buist, 2003 Induced hypothermia in critical care medicine: A review	Review IH as a treatment option for patients with neurologic disorders	Anoxic brain injury following cardiac arrest, traumatic head injury, stroke, hepatic enceph-alopathy, encephalitis, bacterial meningitis, ARDS, HF, and postoperative tachycardia	CNS, acid-base balance, electrolytes, and cardio-vascular, respiratory, renal, GI, and hematologic systems	Improved outcomes in two trials in patients with anoxic brain injury following cardiac arrest Variable results in patients with severe head injury Promising results in preliminary clinical trials of the use of IH with stroke, neurologic infection, and hepatic encephalopathy	ICU patients meeting specific criteria should be considered for IH. Critical care physicians should be knowled-geable in terms of physiologic effects of IH, methods of inducing IH, and potential complications.
Clifton et al., 2001 Lack of effect of induction of hypothothermia after acute brain injury	Compare outcomes in patients treated with IH and those who received standard treatment	Comatose following closed head injury	Fluid and electrolyte balance, lab value alterations, and hypotension	Comparable outcomes and mortality rates between to those receiving IH and those treated with standard interventions	Results were inconclusive. IH is not effective in improving outcomes in patients with severe brain injury.
Finkelstein & Alam, 2010 Induced hypothermia for trauma	Discuss potential benefits and complications of IH in the trauma population, including penetrating trauma with	Trauma with or without cardiac arrest, traumatic brain injury, spinal cord injury, and blunt trauma	Increased infections, coagulopathy, hemodynamic effects, dysrhythmias, alterations in drug metabolism, and fluid,	Uniform efficacy seen in preclinical trials with penetrating trauma with cardiac arrest, delaying cardiac arrest in trauma patients who are hypotensive, and	Further study needed in SCI and blunt trauma; IH beneficial with other types of trauma, specifically penetrating trauma with

Author / Year / Title	Study / Purpose	Population Studied	Complications Evaluated	Results / Findings	Conclusion / Implications
	cardiac arrest; evaluate the role of IH in delaying cardiac arrest in hypotensive patients with penetrating trauma, and traumatic brain injury		electrolyte, and metabolic disturbances	traumatic brain injury Conflicting results between preclinical and clinical trials in traumatic brain injury patients	cardiac arrest
Kuffler, 2010 Combinatorial techniques for enhancing neuroprotection	Neuro-protection provided by hypothermia Combining IH with other therapies and treatments	Variety of neuronal insults	Treatment of complications with neuro-trophins, anti-inflammatory agents, immune-therapy, and alkalinization	Survival rates and neurologic outcomes are significantly improved by whole body hypothermia	Effective neuro-protection possible when multiple treatments are implemented simul-taneously.
McKean, 2009 Induced moderate hypothermia after cardiac arrest	Overview of the patho-physiology of cerebral ischemia and physiologic changes associated with IH. Overview of the history and research of IH following cardiac arrest. Nursing management considerations for patients with IH and consideration of IH protocol development.	Inclusion criteria: Comatose following cardiac arrest Exclusion criteria: Patients with life-threatenting arrhythmias, primary coagulopathy, pregnancy, and sepsis	Prevention of shivering, VS monitoring, skin care issues, fluid and electrolyte imbalances, lab value monitoring, prevention of infection, and rewarming	55% of patients with IH had favorable outcomes	IH is beneficial following cardiac arrest, but implement-tation has been slow. Additional protocols including detailed instructions need to be developed to encourage consistent and safe implement-tation of IH.

Author / Year / Title	Study / Purpose	Population Studied	Complications Evaluated	Results / Findings	Conclusion / Implications
Oddo, Schaller, Feihl, Ribordy, & Liaudet, 2006 From evidence to clinical practice: Effective implementation of therapeutic hypothermia to improve patient outcome after cardiac arrest	Evaluate the use of IH in the ICU and whether IH improves outcomes of all comatose patients with cardiac arrest	Comatose following out-of-hospital cardiac arrest		IH had a positive impact on the outcome of patients in ventricular fibrillation and shock. Poor outcomes in patients with nonventricular dysrhythmias	IH can be successfully implemented in the ICU and is beneficial to patients in cardiac arrest and shock.
Polderman, 2004 Application of therapeutic hypothermia in the intensive care unit	Evaluate the benefits and risks of IH	Variety of neurological injuries	Cardiovascular and hemodynamic effects, coagulo-pathies, infection, hypovolemia, fluid and electrolyte imbalances, other metabolic effects, shivering, drug metabolism	IH useful in preventing or limiting damage to an injured brain or spinal cord	Successful application of IH requires vigilant care and team effort.
Sydenham, Roberts & Alderson, 2009 Hypothermia for traumatic head injury	Determine if the use of IH is beneficial in reducing the risk of death or severe disability in head trauma patients. Determine if IH places patients at risk for	Closed traumatic head injury	Pneumonia, unfavorable outcomes including vegetative state and severe disability	Results were not statistically significant.	No evidence that IH is beneficial in cases of head injury. IH may help reduce the occurrence of death, but significant benefit was only found in low quality trials.

Author / Year / Title	Study / Purpose	Population Studied	Complications Evaluated	Results / Findings	Conclusion / Implications
	pneumonia. Estimate the effects of IH on mortality and long-term functional outcomes in patients with traumatic head injury Determine if IH reduces the number of patients at the follow-up period who experienced unfavorable outcomes or death				High quality trials found no decreased risk of death with IH
Zeitzer, 2005 Inducing hypothermia to decrease neurological deficit: literature review	Examine the effectiveness of IH to decrease neurological deficit	Out-of-hospital, witnessed cardiac arrest	Sepsis, coagulo-pathies, neutropenia, thrombocy-topenia, arrhythmias, and electrolyte abnormalities	Six studies showed improved outcomes Four showed decreased mortality Minimal complications exist	Advanced practice nurses should promote the use of IH. Further research should be conducted on specific treatment protocols.
Zhi, Zhang & Lin, 2003 Study on therapeutic mechanism and clinical effect of mild hypothermia in patients with severe head	Patients with acute head injury within 24 hrs of trauma with major organ damage or functional failure without hypotension	Severe head injury with a GCS of $\leq$8 upon admission to the ED	Effects on ICP, blood glucose and lactic acid, vital sign changes, namely blood pressure and heart rate, blood gases, and electrolytes	IH is safe and effective for preventing brain damage, reducing mortality, and improving the prognosis in patients with severe head injury.	IH reduced mortality and improved outcomes in patients whose treatment included IH. IH decreased ICP, lactic acid levels and

Author / Year / Title	Study / Purpose	Population Studied	Complications Evaluated	Results / Findings	Conclusion / Implications
injury					hyper-glycemia and improved cerebral blood flow. No significant changes in vital signs, blood gas values, and electrolytes occurred in the IH group.

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