Therapeutic Hypothermia in Post-Cardiac Arrest Patients

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Abstract

Cardiac arrest, or when the heart stops beating, is associated with high mortality rates and those that do survive tend to end up with severe neurological impairments. Without the heart beating, no blood is being pumped which causes ischemia, or lack of oxygen supply, to tissues. The brain is often affected leaving patients cognitively impaired and physically disabled. Hypothermia is when the core body temperature is reduced to 35˚C or lower. It has been researched as an intervention when treating post-cardiac arrest patients and it has been proven to be beneficial in reducing ischemic tissue damage and improving neurological outcomes. Hypothermia treatments usually consist of twelve to twenty-four hour periods of the body being cooled down to about 32˚C - 34˚C through either surface or intravenous cooling mechanisms. It has shown to decrease patient mortality rates and has presented no serious or harmful side effects when kept within a safe temperature range.

*Keywords*: Cardiac arrest, therapeutic hypothermia, ischemia, neurological impairments

Therapeutic Hypothermia in Post-Cardiac Arrest Patients

Hypothermia is usually thought of as something to avoid. However, research has shown it to be beneficial in several health related conditions, especially post-cardiac arrest. The small percentage of survivors are often left with widespread cerebral ischemia and edema (Fenwick, 2010). This causes severe neurological dysfunction, such as cognitive and memory impairments. By lowering the body’s metabolic demand, therapeutic hypothermia has been shown to inhibit tissue injury and improve the neurological status in post-cardiac arrest patients.

Cardiac arrest results from an arrhythmia, or an abnormal heartbeat, that causes the heart to stop beating. Ventricular fibrillation and ventricular tachycardia are the two most common arrhythmias associated with cardiac arrest (Collins & Samworth, 2008). Without the heart beating, blood is not being pumped out to the body and oxygen is not being delivered to vital tissues or organs, like the brain. This lack of oxygen, or ischemia, can eventually lead to cell death and tissue necrosis. In addition, when cerebral perfusion is regained and blood flow is reestablished, reperfusion injury can result. “The increased release by presynaptic terminals of the excitatory neurotransmitter glutamate causes calcium to shift from the extracellular fluid to the intracellular fluid which leads to an accumulation of free radicals and activates enzymes that have degrading actions” (Fenwick, 2010, p. 33).

Hypothermia is thought to suppress many of the chemical reactions associated with reperfusion injury and improve neurological status and mortality rates in post-cardiac arrest patients. Decreasing the core temperature reduces the body’s respiratory rate, heart rate, and most importantly metabolic rate (Collins & Samworth, 2008). Both oxygen consumption and carbon dioxide production are also decreased. “Hypothermia reduces the metabolic rate by 6% to 7% for every decrease of 1˚C in temperature” (Fenwick, 2010, p. 33). Since cerebral metabolic rate for oxygen is the primary determinant of cerebral blood flow, a lower metabolic rate can improve oxygen supply and reduce oxygen consumption in the brain, thus prolonging cell life and preventing injury to the tissues.

Typically, mild hypothermia is chosen for treatment because it keeps the patient’s core body temperature in a safe range, between 32˚C-34˚C, reducing the risk for adverse effects. There are three phases or steps to this process including induction, maintenance, and re-warming. Cooling the body can be done by several different mechanisms. Ice packs can be placed on different parts of the body, including the head, neck, armpits, and groin and cooling vests and leg wraps can also be used. In order to decrease the body temperature at an appropriate rate, 70% of the body surface area must be covered or wrapped with these devices (Presciutti, Bader, & Hepburn, 2012). Although this technique is cost efficient and doesn’t require specialized personnel, the equipment can limit the provider’s access to the patient when assessing and doing procedures. In addition, this method is more likely to encounter accidental over-cooling, which occurs when the body temperature drops below 32˚C, therefore putting the patient at greater risk for complications (Fenwick, 2010).

Intravenous infusions of cooled normal saline or lactated ringers solution may be implemented to achieve a lower body temperature as well. Several research results suggest “that after an infusion of [two liters] of 4˚C normal saline, a decrease in temperature between 1.7˚C and 2˚C can be expected” (Fenwick, 2010, p. 35). There is evidence showing that the rapid infusion of cooled fluids produces no harmful effects on left ventricular function, left atrial filling pressures, pulmonary artery pressures, or central venous pressures (Fenwick, 2010). This method is much faster and doesn’t limit patient access; however it is more expensive, poses increased risk for infection, and requires trained personnel for insertion. Re-warming methods are similar and can consist of warming blankets or warmed IV fluids. The methods chosen to cool and re-warm post-cardiac arrest patients are individualized to each person and based off of physician preference and facility protocol.

Cardiac arrests can occur both inside the hospital and out, both with relatively poor prognoses. It is estimated that “3% to 27% of patients who have had an in-hospital cardiac arrest survive until discharge” and the survival rate for pre-hospital cardiac arrest is much worse with “only 7% surviving until discharge” (Collins & Samworth, 2008, p. 145). As stated before, the patients who do manage to live often have detrimental neurological deficits that leave them disabled. There have been several studies examining hypothermia’s effectiveness and potential complications when using it as a treatment therapy for post-cardiac arrest patients.

In 1996, the Hypothermia After Cardiac Arrest (HACA) Study Group conducted a four year study throughout five European countries: Austria, Belgium, Finland, and Germany (Zeitzer, 2005). This randomized control study was done to examine the role of therapeutic hypothermia in patients who underwent out-of-hospital cardiac arrest. Out of 275 participants, 138 people were chosen for the normothermic group while the other 137 patients were randomly assigned to the hypothermic group. The participants’ body temperatures in the hypothermic group were cooled down 32˚C to 34˚C via cooling blankets and ice packs. They remained in this state for twenty-four hours, where their temperatures were continuously monitored, first by a tympanic thermometer and then by a bladder catheter, before finally progressing to a pulmonary artery catheter. These patients were then gradually re-warmed for the following eight hours (Zeitzer, 2005).

After six months, each patient was assessed using the Glasgow-Pittsburgh cerebral performance category tool (CPC) which is a well known neurological assessment tool that was developed to be able to assess patient outcomes from cerebral injury. It is a commonly used instrument and works by rating patients’ severity of brain damage on a scale from 1 to 5; one indicting good recovery; two indicating moderate disability; three meaning severe disability; four implying vegetative state; and five signifying death (Collins & Samworth, 2008). Findings revealed there was a 55% chance of favorable neurological outcomes for the hypothermic group compared with the 39% chance for the normothermic group. In addition, six months after their cardiac arrest, “only 41% died in the hypothermic group compared with 55% in the normothermic group” (Collins & Samworth, 2008, p. 148). The study went on to say how there was no significant difference in terms of complications during the treatment period.

 Several other papers, including the ones written by Mocom (2003) and Zeitzer (2005), review and discuss another study that took place in Australia in 2002. Seventy-seven adult patients who experienced out-of-hospital cardiac arrest were included in this study. Forty-three people were given hypothermia treatment via ice packs while thirty-four patients were given normothermia treatment. In addition, these participants were started on hypothermia treatment in the ambulance on the way to the hospital. The hypothermia group received therapy for twenty-four hour sessions “on the basis of odd versus even days, with patients only getting treatment on odd-numbered days” (Morcom, 2003, p. 25). Temperatures were measured using a pulmonary artery catheter and maintained at 33˚C and re-warming was done over a six hour period using heated oxygen and surface heat (Zeitzer, 2005).

In comparison to the European study that evaluated patient outcomes at six months using the Glasgow-Pittsburgh cerebral performance category tool, this study’s results were obtained when the patients were discharged. The researcher determined patients’ neurological outcomes based on where each patient would go after being discharged (Zeitzer, 2005). “[Forty-nine percent] of those treated with hypothermia were discharged home or to rehabilitation units, indicating a good prognosis, compared with the 26% of those treated without hypothermia” (Morcom, 2003, p.26). Bernard et al. (2002) also found out later on that 51% of the hypothermic group died compared with 68% of the normothermic group. In addition, there were no clinically significant adverse effects when using this treatment.

 Though neither study experienced any significant negative effects, there are potential complications that come with therapeutic hypothermia. “Shivering is a response that is activated as a defense mechanism in response to an altered temperature and can cause metabolic stress for patients” (Presciutti et al., 2012, p. 33). It raises the metabolic rate two to five times greater than the normal rate and increases oxygen consumption by up to 400%. Shivering consumes a lot of energy, increases carbon dioxide production, and can increase intracranial pressure (Presciutti et al., 2012). In general it poses a risk for hemodynamic instability, which is why shivering must be treated as soon as it is detected. Sedatives and neuromuscular blocking agents are typically given to relax the patient’s muscles and prevent shivering (Presciutti et al., 2012).

In addition to shivering, bradycardia, or a slowing of the heart rate can also be expected during hypothermia treatments. Arrhythmias, such as atrial or ventricular fibrillation may be seen as well. Increased systemic vascular resistance and reduced cardiac output are other common effects of hypothermia. In fact, cardiac output is decreased by 7% for each temperature drop of 1˚C (Presciutti et al., 2012). Coagulopathy, mild thrombocytopenia, and rebound hyperthermia, are also potential complications that may be evident (Fenwick, 2010). As the body temperature is lowered there is decreased renal and hepatic perfusion, which can impair their function. If kidney and liver function slows or stops completely, metabolic disturbances, like hyperkalemia and hyperglycemia, can occur. Furthermore, drugs that rely on these organs for metabolism or excretion will stay in the body longer, posing risk for drug toxicity. During the re-warming process, vasodilatation occurs and fluid requirements increase, sometimes requiring inotropic or vasoconstricting drugs to be administered (Fenwick, 2010). Although these potential complications must be acknowledged, they do not occur often if the body temperature is kept above 30˚C - 32˚C. Furthermore, if they do occur, most of them are easily treated in any intensive care unit (ICU) “or can be reduced by slowly increasing the temperature by 1˚C - 2˚C” (Fenwick, 2010, p. 34).

 Because of the low risk for adverse effects, therapeutic hypothermia is recommended to treat post-cardiac arrest patients for at least twelve to twenty-four hours. Lowering the metabolic demand has proven beneficial in limiting the amount of oxygen consumption in the brain and therefore reducing tissue injury due to ischemia. Patients who have undergone hypothermia treatments have had better neurological outcomes and better mortality rates than those who underwent normothermia. With the minimal complications and the significant benefits of therapeutic hypothermia, it will most likely continue to expand as a therapeutic treatment. Perhaps people will stop thinking of hypothermia as a negative thing and instead start viewing it as a medical intervention.

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