2010 BLS and ACLS Guideline Changes: Post-Cardiac Arrest Syndrome and Therapeutic Hypothermia

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The International Liaison Committee on Resuscitation (ILCOR), along with the American Heart Association and the Heart & Stroke Foundation of Canada, released the 2010 resuscitation guidelines in October 2010. The guidelines are divided into basic life support (BLS) and advanced cardiac life support (ACLS). The significant changes for BLS are (1) ABC (airway, breathing, circulation) has been replaced by CAB (compression first, airway, breathing) to bring the importance of compressions to the forefront, and (2) lay persons are encouraged to provide continuous compressions only, omitting the need for mouth-to-mouth contact. The focus of this clinical column is on post-cardiac arrest syndrome and therapeutic hypothermia.

Post-Cardiac Arrest Syndrome

The addition of a new link in the chain of survival has brought post resuscitation care to the forefront of advanced life support. Post-cardiac arrest syndrome (formally known as post-resuscitation disease) is a unique and complex combination of processes, which include (1) post-cardiac arrest brain injury, (2) post-cardiac arrest myocardial dysfunction, and (3) systemic ischemia. This state is often complicated by a fourth component: the unresolved pathological process that caused the cardiac arrest (Neumar et al., 2008). The 2010 guidelines suggest that the individual components of post-cardiac arrest syndrome are potentially treatable. Treatment must focus on reversing the manifestations of the post-cardiac arrest syndrome through prioritization and timely execution from a multi-disciplinary team.

Past practice and treatment protocols must be revisited and goal-directed therapy made a priority when treating post-cardiac arrest syndrome. Clinicians are asked to focus on achieving early hemodynamic stability through invasive monitoring and timely optimization of preload, arterial oxygen content, afterload, and contractility. The recommended treatments include intravenous fluids, inotropic support, vasopressors, and blood transfusions where indicated. The simultaneous need to perfuse the post-ischemic brain adequately without putting unnecessary strain on the post-ischemic heart is unique to post-cardiac arrest syndrome (Neumar et al., 2008).

There are currently no recommendations for maintenance of mean arterial pressure (MAP) in post-cardiac arrest syndrome. However, the increase in intracranial pressure associated with the syndrome suggests that MAP plays a heightened role in maintaining adequate cerebral blood flow. Consensus suggests a MAP of 65 mm Hg to 100 mm Hg will offer adequate cerebral perfusion pressures.

The optimal central venous pressure (CVP) for post-cardiac arrest patients has not been defined by prospective clinical trials, but a range of 8 mm Hg to 12 mm Hg has been used in most published studies (Neumar et al., 2008). Post-cardiac arrest syndrome causes intravascular volume depletion soon after return of spontaneous circulation (ROSC) and volume expansion is usually required. The historical debate over choice of fluid remains a clinician preference. In the process of preload optimization and elevating the CVP, the clinician must be aware that signs and symptoms of cardiac tamponade and right ventricular infarction may be masked.

Although treatment practices can be facility specific, it is important to understand that research suggests ventilation with 100% oxygen for the first hour after ROSC resulted in worse neurological outcomes than immediate adjustment of the FiO_2 to produce an arterial oxygen saturation of 94% to 96% (Peberdy et al., 2010). Evidence indicates that hyperventilation should be avoided in the post-cardiac arrest patient. Ventilation should be adjusted to achieve normocarbia and should be monitored by regular measurement of arterial blood gas values. The 2010 guidelines suggest

that waveform capnography to measure end-tidal CO_2 should be employed as soon as possible after endotracheal intubation and is a significant tool to guide the effectiveness of chest compressions and circulation following ROSC (Peberdy et al., 2010).

Based on the evidence available, clinicians treating post-cardiac arrest syndrome should aim for a MAP of 65 mm Hg to 100 mm Hg, CVP of 8 mm Hg to 12 mm Hg, $ScvO_2$ >70% (if available), and urine output of 1 ml/kg/hr (Neumar et al., 2008; Peberdy et al., 2010).

Therapeutic Hypothermia

In 2005, ILCOR suggested that therapeutic hypothermia (TH) should be part of a standardized treatment strategy for comatose survivors of cardiac arrest. In 2010, ILCOR incorporated TH into the chain of survival under post-cardiac arrest care. Two randomized clinical trials and a meta-analysis (Bernard et al., 2002; Holzer, Bernard, & Hachimi-Idrissi, 2005; Hypothermia after Cardiac Arrest Study Group [HACA], 2002) identified improved outcomes in adults who remained comatose after initial resuscitation from out-of-hospital ventricular fibrillation (VF) cardiac arrest. These patients were cooled to 33 degrees Celsius (range 32 to 34 degrees) within minutes to hours after ROSC for a period of 12 to 24 hours (Neumar et al., 2008; Peberdy et al., 2010).

Although the evidence base is small, the HACA (2002) and Bernard (2005) trials suggest that TH is the only therapy shown to improve neurological outcome post-cardiac arrest. Evidence suggests that the number needed to treat to improve neurological outcome with the use of TH post arrest is between six and seven (HACA, 2002). Researchers have identified two distinct windows of opportunity for clinical use of hypothermia. In the early intra-ischemia period, hypothermia changes abnormal cellular free radical production, poor calcium management, and poor pH management. In the later post-reperfusion period, hypothermia changes the necrotic, apoptotic, and inflammatory processes that cause delayed cell death (Neumar et al., 2008). These changes translate into improved neurological outcomes. However, improved cooling and monitoring technologies are required to realize the full potential of this therapy.

The practical approach to therapeutic hypothermia can be divided into three phases: induction, maintenance, and rewarming. Induction can be initiated with 4-degree intravenous saline or simply external cooling with the application of ice packs to the head, axilla, and groin areas. Neuromuscular blockade is indicated to prevent shivering that leads to rewarming and an increase in PCO_2 (Neumar et al., 2008; Peberdy et al., 2010).

Maintenance can be achieved with the application of cooling blankets, vests, and helmets. Intravascular cooling catheters are internal cooling devices that are usually inserted into a femoral or subclavian vein. However, unfamiliarity and the invasive nature of the devices limit their use. Clinicians need to be aware that iced saline alone cannot maintain the cooled state (Neumar et al., 2008).

The rewarming phase can be regulated with the devices used for cooling or by other heating systems, but passive rewarming is recommended in order to prevent rebound hyperthermia. The optimal rate of rewarming is not known, but current consensus is to rewarm at approximately 0.25°C to 0.5°C per hour (Peberdy et al., 2010). Particular care should be taken during the cooling and rewarming phases because metabolic rate, electrolyte concentrations, and hemodynamic conditions can change rapidly.

Therapeutic hypothermia is associated with several complications. Shivering is common, particularly during the induction phase. Mild hypothermia increases systemic vascular resistance, which reduces cardiac output. Dysrhythmias may be induced by hypothermia with bradycardia being the most common. Hypothermia induces a diuresis and could lead to electrolyte imbalances. Hypothermia decreases insulin sensitivity and insulin secretion, which results in hyperglycemia. Alterations in platelet and clotting function can lead to impaired coagulation and increased bleeding. Hypothermia can impair the immune system and increase infection rates (Neumar et al., 2008). In the HACA (2002) study, pneumonia was more common in the cooled group, but this difference did not reach statistical significance. Of particular note, the clearance of sedative drugs and neuromuscular blockers is reduced by up to 30% at a temperature of 34°C (Neumar et al., 2008).

Magnesium sulfate reduces shivering thresholds and can be given to reduce shivering during cooling. Magnesium is also a vasodilator and, therefore, increases cooling rates. Magnesium sulfate (5 g) can be infused over five hours, which covers the period of hypothermia induction (Neumar et al., 2008). A review of seven provincial protocols within Canada did not reveal magnesium as a standard order.

In summary, evidence supports mild therapeutic hypothermia as an effective therapy for the post-cardiac arrest syndrome. Unconscious adult patients with ROSC after out-of-hospital VF cardiac arrest should be cooled to 32°C to 34°C for at least 12 to 24 hours. Most experts currently recommend cooling for at least 24 hours. Trials have used 32°C to 34°C; however, the optimal temperature has not been determined. In addition, therapeutic hypothermia might also benefit unconscious adult patients with spontaneous circulation after out-of-hospital cardiac arrest from a non-shockable rhythm

or in-hospital cardiac arrest (Peberdy et al., 2010). Prognostication has also been identified in the 2010 guidelines as an issue for clinicians to reconsider; more than the traditional 72 hours may be required if TH has been used. If TH is contraindicated then avoidance of elevated body temperatures should be employed, as there is a direct correlation between pyrexia and poor neurological outcomes in post-cardiac arrest patients (Neumar et al., 2008).

Conclusion

Much anecdotal evidence exists in support of this treatment modality. However, it is scientific evidence that drives practice. Nursing is in a pivitol role to embrace this treatment and develop scientific evidence surrounding the impact on quality of life post therapy. I challenge every nurse in Canada with a passion for improving the quality of life for sudden cardiac death patients to ask: "Is this patient a candidate for induced / therapeutic hypothermia?" ♥

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References

Bernard, S.A., Gray, T.W., Buist, M.D., Jones, B.M., Silvester, W., Gutteridge, G., & Smith, K. (2002). Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. *New England Journal of Medicine 346*, 557–563.

Holzer, M., Bernard, S.A., & Hachimi-Idrissi, S. (2005). Hypothermia for neuroprotection after cardiac arrest: Systematic review and individual patient data meta-analysis. *Critical Care Medicine 33*, 414–418.

Hypothermia after Cardiac Arrest Study Group. (2002). Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. *New England Journal of Medicine 346*, 549–556.

Neumar, R., Nolan, J., Adrie, C., Aibiki, M., Berg, R., Bottiger, B., …Vanden Hoek, T.L. (2008). ILCOR consensus statement: Post cardiac arrest syndrome. *Circulation 118*, 2452–2483.

Peberdy, M.A., Calloway, C., Neumar, R., Geocadin, R.G., Zimmermann, J., Donnini, M., …Kronick, S. (2010). Part 9: Post cardiac arrest care: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation, 122,* s768–s786.