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Therapeutic Hypothermia After Cardiac Arrest

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In this article, Dr. Nolan discusses the benefits of induced hypothermia following cardiac arrest. He further explains the key factors that must be considered when implementing this therapy to optimize patient outcome.

Of the patients who sustain an out-of-hospital cardiac arrest caused by cardiac disease, just 7%survive to hospital discharge (Pell 2003). Survival to discharge after in-hospital cardiac arrest is 15-20% (Sandroni et al. 2006). Many of those who regain a spontaneous circulation die subsequently from neurological injury (Laver et al. 2004), which is sustained during the anoxic, no-flow period of cardiac arrest or as a result of reperfusion injury that occurs in the early post-resuscitation phase (Safar et al. 2002). Mild hypothermia in the early postresuscitation period may prevent or reduce this reperfusion injury (Sterz et al. 2001).

Two randomized controlled trials have demonstrated that mild therapeutic hypothermia improves outcome in unconscious patients with spontaneous circulation after initial resuscitation from cardiac arrest (Bernard et al. 2002; Hypothermia After Cardiac Arrest Study Group 2002). These studies enrolled only those patients with an initial rhythm of ventricular fibrillation (VF), and the vast majority of these were out-of-hospital cardiac arrests. The larger of the two studies, which was undertaken in several European centers, randomized 273 patients to be treated with either therapeutic hypothermia (32-34° for 24 hours) or normothermia (Hypothermia After Cardiac Arrest Study Group 2002). Fifty-five percent of the patients in the hypothermia group were discharged alive with a good neurological outcome, versus 39 percent in the group who were treated conventionally (p=0.009). The other study was undertaken in Australia and reported similar results, albeit with fewer patients (Bernard et al. 2002).

On the basis of these studies, the International Liaison Committee on Resuscitation (ILCOR) published an advisory statement recommending that “unconscious adult patients with spontaneous circulation after out of hospital cardiac arrest should be cooled to 32-34°C for 12-24 hours when the initial rhythm was ventricular fibrillation” (Nolan et al. 2003). The main recommendation was restricted to this specific group of cardiac arrest patients to reflect the highly selected group of subjects enrolled in the two clinical studies. A secondary recommendation made by ILCOR was that “such cooling may also be beneficial for other rhythms or in-hospital cardiac arrest.” Clinicians at many centers cool patients who remain comatose after cardiac arrest from non-shockable rhythms (Holzer et al. 2006), despite the lack of high-level outcome data for this group.

Generally accepted contraindications to inducing hypothermia are: severe systemic infection, severe cardiogenic shock, established multiple organ failure and pre-existing medical coagulopathy (patients given thrombolytic therapy can be cooled). There are several well-documented complications associated with the use of mild hypothermia, which need to be anticipated and managed appropriately if the post-cardiac arrest patient is to benefit from this therapy (Polderman 2004). Complications include infection (e.g. pneumonia), coagulopathy, vasoconstriction, arrhythmias (particularly bradycardia), hyperglycemia, electrolyte disorders and pancreatitis.

Shivering can occur at any time, but is particularly common during the cooling and rewarming phases. Sedation with propofol and an opioid will attenuate the problem, and buspirone may also be effective (Mokhtarani et al. 2001). An infusion of magnesium may reduce the shivering threshold and, as a vasodilator, increase the rate of cooling. If shivering persists, it may be necessary to give a bolus of a neuromuscular blocking drug. Many clinicians elect to use infusions of neuromuscular blocking drugs, but this is generally unnecessary and may mask convulsions.

Cooling should be initiated as soon as possible after return of spontaneous circulation, but appears successful even if it is delayed (e.g. 4-6 hours). Most centers will cool patients for 24 hours and then re-warmslowly at 0.25°C h-1.A significant systemic inflammatory response is very common in post-cardiac arrest patients, and considerable care is required to prevent hyperthermia, which would increase neurological injury.

Several cooling techniques exist, but none of these combines ease of use with high efficacy. External cooling methods are easy to apply, but are slow in reducing core temperature. The external techniques include cooling tents; cooling blankets (using circulating water or air); ice packs to the groins, axillae and neck; wet towels and fanning; and a variety of cooling helmets. One of the fastest and simplest methods to initiate cooling is to infuse 30 ml kg-1 of normal saline or Hartmann’s solution at 4°C. This reduces core temperature by 1.7°C and does not cause pulmonary

edema (Bernard et al. 2003). An endovascular cooling device, inserted into the femoral vein, enables precise temperature control (Holzer et al. 2006).

Further research needs to be undertaken to determine the optimal duration of the hypothermia, the optimum target temperature, the rate of cooling and re-warming and the optimal cooling technique (external or internal).

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