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Therapeutic Hypothermia for all Cardiac Arrest Patients

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Dr Michael Holzer presents the rationale for cooling all comatose patients after cardiac arrest.

About 17 million people worldwide die from cardiovascular diseases each year (World Health Organization 2002), and many of these deaths are due to sudden cardiac arrest. The incidence of out-of-hospital sudden cardiac arrest in industrial countries lies between 36 and 128 per 100,000 inhabitants per year. Unfortunately, full cerebral recovery after cardiac arrest is still a rare event and good neurological recovery of patients admitted to a hospital can only be achieved in 11% to 48% of the cases. The rest of the patients die during the hospital stay or remain in vegetative state (Vreede-Swagemakers et al. 1997).

Therapeutic hypothermia is a new therapy that acts in a multifactorial way on the mitigation of post-resuscitation disease. This includes the slowing of destructive enzymatic processes, the protection of lipid membranes fluidity and the reduction of oxygen needs without impairing microvasculatory blood flow in low-flow regions during reperfusion after ischemia. Additionally, therapeutic hypothermia also acts on lipid peroxidation, brain oedema, intracellular acidosis and apoptotic neuronal cell death (Chopp et al. 1989; Lei et al. 1994; Sterz et al. 1992; Zhu et al. 2004). Therapeutic hypothermia not only protects neurons, but also has beneficial effects on white matter injury and astroglial cell proliferation (Hachimi-Idrissi et al. 2004; Roelfsema et al. 2004).

Recently two randomized trials documented that reducing the body temperature to 32-34°C after successful restoration of spontaneous circulation could substantially improve neurological recovery (Bernard et al. 2002; The Hypothermia After Cardiac Arrest (HACA) study group 2002). Based on these trials, recent published guidelines of resuscitation recommend cooling comatose patients after cardiac arrest due to ventricular fibrillation. The guidelines state that this might also be beneficial in patients with a cardiac arrest due to a non shockable rhythm or in-hospital arrest (European Resuscitation Council 2005).

Experimental evidence shows that therapeutic hypothermia is also beneficial in other cerebral ischemic states (e.g. stroke). Studies analysing the protective mechanisms have shown that the effect of therapeutic hypothermia is largely independent of the underlying cause of ischemia. Further, a small randomized trial including only patients resuscitated after asystole or pulseless electrical activity found a non significant increase in survival and improvement of neurological recovery (Hachimi-Idrissi et al. 2001). A recent metaanalysis of all three randomized hypothermia trials showed a significant improvement of survival and shortterm neurologic recovery (Holzer et al. 2004). It could therefore be concluded that as long as the patient has restoration of spontaneous circulation, there is some brain tissue left which could be rescued by therapeutic hypothermia, independent of the underlying cause, location or rhythm of cardiac arrest.

There are also side effects of using therapeutic hypothermia and in each case whether the risk outweighs the benefit needs to be assessed. Negative experiences with lower temperatures (26-32°C, moderate hypothermia) at the beginning of the therapeutic hypothermia era raised concerns that the developing complications outweighe any favourable effects of hypothermia.

More recent experiences with mild therapeutic hypothermia (32-34 °C), however, show that the beneficial effect exceeds the complications by far. The following complications can occur and should be considered when treating patients with therapeutic hypothermia. A significantly higher rate of pneumonia has been reported, although only in one study (Yanagawa et al. 1998), in which it was also reported that none of the cases of pneumonia was a direct cause of death. A higher incidence of complications has been found in other studies with arrhythmias, haemodynamic instability, bleedings, thrombocytopenia, pneumonias, sepsis and convulsions (Bernard et al. 1997; Bernard et al. 2002; The Hypothermia After Cardiac Arrest (HACA) study group 2002). The total complication rate was not significantly higher in the cooled group in any of the reported studies. However, the number of patients studied was small. Treatment with therapeutic hypothermia requires defined protocols, to identify possible arising complications promptly.

Induced mild hypothermia after resuscitation from cardiac arrest improves neurological outcome. Extending further the existing resuscitation guidelines, all unconscious adult patients with spontaneous circulation after out-of-hospital cardiac arrest should be cooled to 32 to 34 °C for 24 hours as long as there is no contraindication (ongoing haemorrhage, pregnancy, terminal disease). Although the optimal target temperature, duration and mode of re-warming are still the subject of investigation, it is clear that therapeutic hypothermia should be started as soon as possible to yield the maximum benefit. A very effective method of inducing therapeutic hypothermia is the rapid infusion of 30 ml/kg cold (4°C) lactated Ringer's solution.

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