

Case Report

Differential interhemispheric cooling and ICP compartmentalization in a patient with left ICA occlusion

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Summary

We report a case of a 69-year-old white female who presented with a large left internal carotid artery occlusive stroke from a cardiogenic embolus. She was enrolled in an institutional study using a specially designed cooling helmet. Bilateral intracranial pressure (ICP) and temperature probes were placed to determine if there was any differential cooling and ICP compartmentalization between the two hemispheres. We demonstrated a significant temperature gradient between the infarcted and the non-infarcted hemisphere. A significant inter-hemispheric ICP gradient was also observed. We believe that this is the first demonstration of preferential cooling of the infarcted hemisphere over the non-infarcted hemisphere with regional surface hypothermia.

Keywords: Hypothermia; stroke; head injury; helmet; brain cooling.

Introduction

Hypothermia is one of the most potent neuronal protective agents in animal studies and probably has the greatest therapeutic potential [1, 5–8]. However, its timely and safe delivery remains to be a significant clinical challenge. Complications associated with systemic hypothermia or the inevitable delay in instituting hypothermia in the hospital setting may greatly compromise its potential therapeutic effects. Regional hypothermia may achieve sufficient brain cooling while minimizing systemic effects and has the potential of being used in the pre-hospital setting. A specially designed cooling helmet using NASA spin-off spacesuit technology may have a potential of meeting these goals [10]. We present a case using this helmet in a patient with a large acute

infarct. The patient was enrolled in a trial to determine if this helmet was effective in preferentially lowering the brain temperature compared to the core temperature. Bilateral intracranial pressure (ICP) and temperature probes were placed in this patient to determine if there was differential cooling and differential ICP measurements between the noninfarcted and infarcted hemisphere.

Case report

This 69-year-old female presented with a large left hemispheric stroke secondary to occlusion of the left ICA from a cardiogenic embolus. Her NIHSS was 26 on admission. The patient presented beyond the treatment window for intravenous or intra-arterial thrombolytic therapy. The patient deteriorated further on hospital day 2 despite maximal medical treatment and a repeat CT scan of the brain showed a 1.7 cm midline shift. Her family consented for the IRB approved COOL BRAIN-Stroke Trial (CBST) protocol with bilateral brain monitoring.

The patient was fully sedated and paralyzed. Bilateral Neurotrend[®] sensors (Codman & Shurtleff, Inc., Raynham, MA) were placed with the tips of the probes in both the infarcted area and contralateral normal brain tissue (Fig. 1). Brain temperatures were continuously monitored at a depth of 0.8 cm below the cortical surface (Through theoretical modeling and calculation, it has been suggested that average brain temperature should be measured at 7.5 mm below the cortical surface in adults [11].) as were the core temperatures and ICP (Codman MicroSensor, Johnson & Johnson Professional, Inc., Raynham, MA).

At baseline, the healthy hemisphere temperature (HHT) was 35.1 °C and the body temperature was 35 °C. The infarcted hemisphere temperature (IHT) was approximately 1.8 °C below the body temperature. Within one hour of helmet cooling, the HHT dropped approximately 5.6 °C while the IHT dropped approximately 8 °C (Fig. 2). An average temperature gradient (brain–body) of –14 °C in the infarcted hemisphere



Fig. 1. The tips of the Neurotrend® probes were in both the infarcted area and contralateral healthy brain tissue

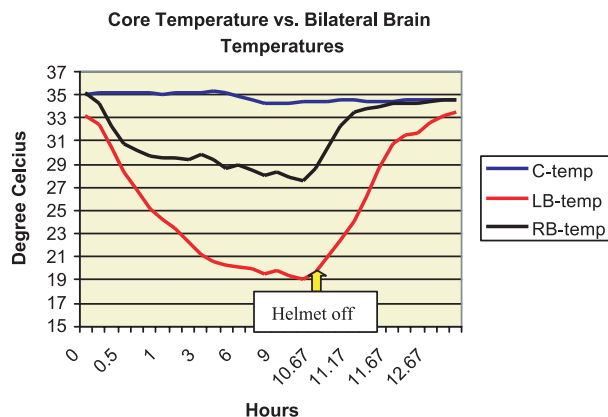


Fig. 2. At baseline, the HHT was 35.1 °C and the body temperature was 35 °C. The IHT was approximately 1.8 °C below the body temperature. Within one hour of helmet cooling, the HHT dropped approximately 5.6 °C while the IHT dropped approximately 8 °C. An average temperature gradient (brain–body) of –14 °C in the IH and –6 °C in the HH was maintained throughout the cooling period. Warming blankets were used to maintain body temperature at approximately 35 °C. Just before the patient expired, the HHT was approximately the same as the body temperature while the IHT was approximately 1 °C lower than the body temperature. (C-temp Core temperature; LB-temp Left brain temperature; RB-temp Right brain temperature)

(IH) and –6 °C in the healthy hemisphere (HH) was maintained throughout the cooling period (Fig. 2). Warming blankets were used to maintain the body temperature at about 35 °C. The patient showed no improvement with further treatment. At hour 10, the cooling was dis-

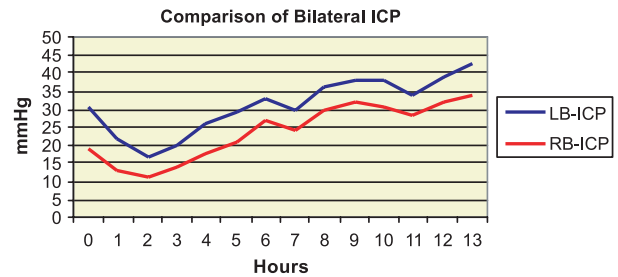


Fig. 3. A clinically significant inter-hemispheric ICP gradient (IH–HH) of 7 mmHg was observed. (LB-ICP Left brain intracranial pressure; RB-ICP Right brain intracranial pressure)

continued and the care was withdrawn per her family's request. The patient expired shortly afterwards. Just before the patient expired, the HHT was approximately the same as the body temperature while the IHT was approximately 1 °C lower than the body temperature. Of note, there was a clinically significant inter-hemispheric ICP gradient (IH–HH) of approximately 7 mm Hg throughout the period of recording (Fig. 3). The patient never developed unilateral or bilateral dilating pupils throughout the cooling period.

Discussion

Resuscitative hypothermic neuroprotection has been conclusively established in animal models of both ischemic and traumatic brain injuries [1, 5–8]. In patients with severe ischemic strokes, significant temperature gradients with a temporal profile between the infarcted and contralateral hemispheres have been documented [9]. The infarcted hemisphere appeared to have had higher temperatures than the contralateral hemisphere within the first 6 hours post-stroke but have had lower temperatures after 12 hours [9]. To the best of the authors' knowledge, our case is the first report (our randomized study was published [10] prior to the enrollment of this patient) that documents the effects of regional surface brain cooling with bilateral cerebral temperature monitoring in a patient with severe ischemic stroke. Our patient was between 48–72 hours post-stroke when bilateral temperature probes were placed. At baseline, the body temperature was 35 °C, the HHT was 0.1 °C higher and the IHT was approximately 1.8 °C lower than the body temperature. Our patient's data suggests that poorly perfused brain tissue may have a much higher susceptibility to regional surface cooling. Within one hour of helmet cooling, the HHT dropped approximately 5.6 °C while the IHT dropped approximately 8 °C (Fig. 2). Throughout the cooling period, an average temperature gradient (brain–body) of –14 °C was maintained in the IH in contrast to a gradient of –6 °C in the HH (Fig. 2). Poorly perfused brain tissue may receive minimal warming effects from cerebral blood flow. This

could account for the increased susceptibility to regional surface cooling observed in the IH in this patient. The implication is that possibly much more rapid and profound regional hypothermia may be accomplished in the ischemic region prior to the definitive thrombolytic therapy if EMS personnel can initiate local brain cooling in the pre-hospital setting. Also, the fact that there appears to be differential cooling between the infarcted and normal hemispheres has potential implications when monitoring patients for future and ongoing studies using hypothermia for the treatment of ischemic strokes.

Large hemispheric infarction is commonly associated with significant ICP elevation from cerebral edema. Although ICP probes are traditionally placed in the right frontal cortex to minimize potential injury to the eloquent tissue from procedural related complications, recent evidence has suggested that significant ICP gradient develops with large hemispheric injuries [2–4]. However, it remains controversial whether such gradients persist and whether monitoring ipsilateral to the infarcted hemisphere would provide more accurate therapeutic guidance. Our patient was between 48–72 hours post-stroke when bilateral ICP probes were placed. A clinically significant interhemispheric ICP gradient (IH–HH) of approximately 7 mmHg was observed throughout the recording period (Fig. 3). Ipsilateral monitoring may be indicated in large hemispheric infarctions to more accurately assess the regional cerebral perfusion pressure.

Conclusion

We believe that this is the first demonstration of preferential cooling of an infarcted hemisphere over a normal hemisphere in patient undergoing regional surface hypothermia. The reported data, being in an extreme case of completed infarction, may not reflect findings in evolving ischemia or less extensive infarction. However, this case may suggest that regional surface hypothermia could potentially allow a therapeutic depth of cooling in the ischemic hemisphere prior to patient's arrival in the emergency department. Also, this data may be important for investigators planning future hypothermia trials for ischemic stroke.

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Comment

The efficacy of hypothermia as a treatment in acute neurological pathology remains an opportunity for development. This case report from Wang *et al.* demonstrates clinically significant inter-hemispheric differences in both ICP and temperature. The cooling helmet that they have used provides temperature reduction and that have made measurements at single points within each hemisphere. Further work is required to establish the spatial patterns of temperature and pressure within abnormal pathology throughout the hemisphere and then to ultimately determine whether this methodology can result in improvements in outcome.

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