Editorial

Is there a role for hyperoxia in the management of severe traumatic brain injury?

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The pathophysiological features of neurotrauma, including traumatic brain injury (TBI) and spinal cord injury, involve a primary mechanical injury that reflects the complex interplay of contusion, compression, and shear and rotational forces as well as a series of vascular, metabolic, molecular, and inflammatory processes that synergistically culminate in a process termed secondary injury.2,5,8

Although improved outcomes after TBI have occurred due to the use of restraint systems in automobiles, protocol-driven critical care management, and judicious use of decompressive surgery, no significant attenuation of secondary injury mechanisms has yet occurred in the clinical setting, despite promising data from animal models.

There are persuasive data implicating mitochondrial dysfunction and delayed apoptosis or programmed cell death in the pathobiological mechanisms of neuronal cell death after TBI. Based on some animal models and preliminary clinical data, it has been postulated that impairments in oxygen delivery and extraction could be involved in the mechanism of posttraumatic mitochondrial dysfunction in neurons.1,7

With this background implying that hyperoxic therapy could improve cerebral metabolism after TBI, Diringer and colleagues have examined the effect of 100% oxygen on cerebral metabolism and cerebral blood flow by using positron emission tomography scanning techniques. Five patients with TBI (median Glasgow Coma Scale Score 7; range 3–9) were studied within 24 hours of injury (mean time 17.9 hours posttrauma). Patients received 1 hour of 100% fraction of inspired O2, before and after which positron emission tomography scanning assessments of the effect of hyperoxic therapy on TBI using “gold standard” positron emission tomography scanning methodology. Although this study does not definitively exclude a potential role for hyperoxic therapy in which hyperbaric approaches are used at later time points, the absence of even a trend in physiological benefit from hyperoxic therapy does call into question this therapeutic modality. Further clinical research is required to establish whether hyperoxic therapy is of value in TBI.

References


RESPONSE: We agree completely with the editorial comments.

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