Preventing hypoxemia in closed head injury

Topic: Preventing Hypoxemia in Closed Head Injury Brain injury results when there is a sudden, external, and physical assault to the brain. Brain injury occur in two ways, namely: (a) closed brain injury, and (b) penetrating brain injury. In closed brain injury, there is no break in the skull, and is caused by a rapid forward and backward movement as well as shaking of the brain resulting to bruising and tearing of the tissue and blood vessels of the brain. Closed brain injury usually results from car accidents, falls, and shaking of the baby (also known as shaken baby syndrome). Brain injuries may be mild, and others are more severe and may further result to permanent disability or death.

In closed head injury, brain is susceptible to further injury caused by cerebral edema since the brain is confined in a calvarium and therefore, cannot expand. Cerebral edema is the accumulation of excess water into the intra and extra cellular spaces of the brain (Cerebral Edema, 2003), and is most often encountered in patients with brain injury. Cerebral edema can cause a lethal effect, which includes cerebral ischemia due to compromised regional or global cerebral blood flow (CBF) and intracranial compartmental shifts due to intracranial pressure gradients that result in compression of vital brain structures (Ahmed, 2007). Thus, it is the primary goal in medicine to manage cerebral edema to maintain regional and cerebral blood flow in order to meet the metabolic requirements of the brain as well as prevent secondary neuronal injury brought about by cerebral ischemia.

Tegtmeyer (1998) noted that in order to minimize secondary injuries brought about by closed head injury, priorities of treatment must be geared towards airway, breathing, and circulation. Airway must be maintained to facilitate oxygen delivery to the brain to prevent cascading of secondary injuries.

During the injury, the cerebral perfusion pressure drops off rapidly as the blood goes down, and brain volume increases with swelling and edema. Cytotoxic edema which results from closed head injury must be prevented to further damage the brain. And in doing so, hypercarbia and hypoxemia must be corrected. As indicated in the University of Oxford database (2008), secondary brain injury may have been caused by inadequate brain perfusion, and therefore, avoiding and treating hypoxia, hypercarbia, and hypertension are of utmost importance. Increased morbidity and mortality are associated with hypoxemia, hypercarbia, and hypotension in patients with severe head trauma. In line with this, brain injury causing compromised cerebral oxygen delivery, further causes vasodilatation increasing cerebral blood flow and intracranial pressure, respectively.

Lastly, Ahmed and Bhardwaj (2007) noted that hypoxemia and hypercarbia must be avoided in patients with cerebral edema because of its effect as potent cerebral vasodilators thereby causing further cerebral edema.

References

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