Do Severe Traumatic Brain Injuries Affect Brain Blood Flow Differently than Healthy Children

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Introduction

The most common cause of death in children is traumatic brain injury. In order to prevent further brain damage, neuro intensive care for children with severe TBI aims to stop insults to the brain. Following a brain injury, it is critical to monitor Cerebral Blood Flow (CBF) and auto regulation. There hasn't been a lot of research on children to look into this, though. Recent research suggests that the age dependent rise in CBF only lasts for a short time. Low initial CBF after TBI has been linked to negative outcomes and might be more common than previously thought. A worse outcome is also associated with impaired cerebral pressure auto regulation, however it is unclear whether this association is causative. The limited studies that are now available are primarily centered on a small group of patients between the ages of 0 and 18. To collect information that helps optimise the therapeutic and clinical management of children who have had TBI, larger trials with more restricted age ranges and multimodality monitoring are needed. This happens as people age because of changes in CBF and cerebral pressure auto regulation.

Description

Worldwide, traumatic brain injury is a leading cause of death in children and affects young people. Many children who have severe TBIs require neuro intensive care and neuro monitoring even though the majority of TBIs are modest. There are still no evidence based suggestions on how to treat TBI in young individuals, despite the enormous number of cases.

Acute TBI includes both primary and secondary injuries. The initial impact related harm done directly to the brain parenchyma is referred to as primary brain injury. When taking into account the effects of inflicted TBI, the initial damage in children is much more varied and complex than in adults. A common outcome of numerous initial injuries is the activation of secondary processes that cause secondary injury, such as rupture of the blood brain barrier, harm to neurons, disturbed cerebral pressure auto regulation, ischemia, and brain herniation. The primary goal of TBI treatment during the acute period is to achieve enough CBF in order to halt further brain injury and insults. This is mostly achieved by monitoring ICP and CPP to maximise CBF, which is necessary for supplying substrates to the injured brain. The average brain requires 15% of cardiac output even though it only makes up roughly 2% of body weight. It consumes 60% of the available glucose and 20% of the oxygen when the organism is at rest, indicating a high energy need. At age 5, the brain starts consuming twice as much glucose per day as an adult's brain. Although this gap diminishes with age, boys utilise more brain glucose than girls do. Blood flow and oxygen supply must be closely regulated for neurons to survive, which is even more important in the injured brain.

This regulation is governed by a number of homeostatic processes. The most important ones are cerebral pressure auto regulation, brain metabolism, $PaCO_2$ and PaO_2.

The cerebral pressure auto regulation mechanism maintains a constant cerebral blood supply despite variations in blood pressure by changing the diameter of brain arteries. Vasodilation results from drops in MAP/CPP while constriction results from increases. This prevents bleeding, edema, and cerebral ischemia. $PaCO_2$, the most potent vasodilator, increases CBF by 2%/mmHg-4%/mmHg. The therapeutic use of hyperventilation to lower ICP relies on the fact that the CBF response to PaO_2 changes occurs quickly.

PaO_2 is less useful clinically and has a far lesser effect on cerebral blood flow than $PaCO_2$. CBF rises when PaO_2 is less than 50 mmHg, however changes take longer than 6 minutes to take place. PaO_2 changes exceeding 50 mmHg are negligible.

In a healthy brain, the interactions between ICP, MAP, CPP, and CBF can be straightforward, but in an injured brain, particularly one that is still developing, they can be more challenging. Numerous physiological variables, such as cerebral pressure auto regulation, CO_2 reactivity, and O_2 reactivity, might be out of balance and jeopardise the need for a sufficient supply of substrates. Hypo perfusion and poor cerebral pressure auto regulation have been seen in children with TBI, and these conditions have been associated with worse outcomes. Under 4 years olds are more vulnerable to poor cerebral pressure auto regulation and experience worse outcomes. Even though much effort has been done to track and understand the cerebral hemodynamic changes following TBI, little is known about children who have TBI. Optimizing treatment and care approaches for children with severe TBI requires a full understanding of the normal physiology and cerebral hemodynamic changes caused by TBI. In this review, we have made an effort to summarise the most recent studies on CBF monitoring in children with severe brain lesions and both healthy controls.

Conclusion

It is obvious that the pathophysiology of brain injury changes between birth and the age of 18 years. As children grow and their brains develop, major changes occur in their physiology, architecture, skull size, suture elasticity, vascular maturation, tortuosity, and the ratio of grey to white matter. The possibility that the brain's response to the injury and the biomechanics of brain injury would vary is increased by this. The variations in CBF seen in young, healthy children at various ages and reported in this study suggest that the tolerance and vulnerability of the brain fluctuate greatly with age and may exist even at very modest age differences.

It has been established as a result that the prognosis following TBI changes with age, with children under the age of 4-5 years having a worse prognosis. More study is needed on specific cerebral hemodynamic abnormalities following TBI in different age groups of children in order to improve the management of kids with severe TBI.

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