



# Every breath you take: Hyperventilation and intracranial pressure

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**H**yperventilation is one of the most effective methods available for the rapid reduction of intracranial pressure (ICP). The CO<sub>2</sub> reactivity of intracerebral vessels is one of the normal mechanisms involved in the regulation of cerebral blood flow (CBF). Experimental studies using a pial window technique clearly demonstrate that the action of CO<sub>2</sub> on cerebral vessels is exerted via changes in extracellular fluid pH.<sup>1</sup> Molecular CO<sub>2</sub> and bicarbonate ions do not have independent vasoactivity on these vessels. Although even a mild traumatic brain injury (TBI) can reduce the ability of cerebral vessels to react to changes in pCO<sub>2</sub>, most patients with moderate or severe TBI retain at least some global CO<sub>2</sub> reactivity. As a result, hyperventilation consistently lowers ICP.<sup>2</sup>

## ■ THE MAIN DRAWBACK: REDUCED CEREBRAL BLOOD FLOW

Despite the effectiveness of hyperventilation in lowering ICP, use of this treatment modality has fallen out of favor, primarily because of the simultaneous effect on CBF. Current guidelines for the management of TBI recommend avoiding hyperventilation during the first 24 hours after injury, when CBF is generally lowest, and recommend that moderate hyperventilation may be used subsequently, but only as a treatment for an elevated ICP.<sup>3</sup>

The effect of a reduction in pCO<sub>2</sub> on CBF in a normal subject is approximately 3% per mm Hg. Early studies in normal subjects using the Kety Schmidt technique for measuring global CBF demonstrated that reducing pCO<sub>2</sub> from 37 to 19

mm Hg resulted in a decrease in global CBF from 45 to 25 mL/100 g/min.<sup>4,5</sup> Cerebral oxygen extraction was increased, but cerebral oxygen consumption (CMRO<sub>2</sub>) remained unchanged. Only when pCO<sub>2</sub> was further reduced to an average value of 10 mmHg was CMRO<sub>2</sub> significantly reduced, suggesting that ischemia may have resulted from the reduction in CBF.

Studies in patients with TBI follow this same pattern, with hyperventilation resulting in a consistent decrease in global CBF and increasing global cerebral oxygen extraction, but no reduction in CMRO<sub>2</sub> until very extreme levels of pCO<sub>2</sub> are reached. However, patients with TBI often have areas of brain that are hypoperfused as a result of their brain injury, and these patients may be more vulnerable to regional effects of hyperventilation on CBF. Recently, studies using positron emission tomography have clearly shown that reduction in pCO<sub>2</sub> to levels of 25 to 30 mm Hg does reduce regional CBF, even in areas of the brain that are hypoperfused at baseline.<sup>6,7</sup> Furthermore, hyperventilation increases the volume of the brain that is marginally perfused, but no significant reduction in regional CMRO<sub>2</sub> has been observed at these levels of pCO<sub>2</sub>.<sup>6,7</sup> The conclusion of these recent studies seems to be that hyperventilation regularly reduces CBF and increases the proportion of the brain that is critically hypoperfused, but does not result in ischemia at the levels of pCO<sub>2</sub> that are commonly used in clinical practice.

The consequences of these hemodynamic effects of hyperventilation on outcome after severe TBI have been studied multiple times, and there is no consistent neuroprotective effect. One randomized clinical trial has shown an adverse effect of chronic hyperventilation in TBI patients.<sup>8</sup> Experimental studies using the cortical impact injury model demonstrate that hyperventilation for 5 hours after TBI increased CA3 hippocampal neuron loss.<sup>9</sup>

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### ■ EFFECTS ARE TRANSIENT

Another characteristic of hyperventilation that limits its usefulness as a treatment modality for intracranial hypertension is the transient nature of its effect. Because the extracellular space of the brain rapidly accommodates to the pH change induced by hyperventilation, the effects on CBF and on ICP are transient. In fact, after a patient has been hyperventilated for more than 6 hours, rapid normalization of arterial pCO<sub>2</sub> can cause a significant rebound increase in ICP.

### ■ SUMMARY

Hyperventilation can rapidly lower ICP, but because it induces a consistent reduction in CBF and because the effects on ICP are transient, the only role that hyperventilation plays in the management of intracranial hypertension is in the management of acute elevations in ICP. In these circumstances, hyperventilation can be life-saving and can temporize until more definitive treatment of the intracranial hypertension can be undertaken.

### ■ REFERENCES

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