Hyperventilation

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Recommendations

Level I

There are insufficient data to support a Level I recommendation for this topic.

Level II

Prophylactic hyperventilation should be avoided.

Level III

Hyperventilation is recommended as a temporising measure for the reduction of ICP.

Hyperventilation should be avoided during the first 24 h after injury when CBF often is critically reduced. If hyperventilation is still used, it is recommended to monitor brain oxygen delivery by SjO_2 or $PtiO_2$ measurements or other means of continuous brain oxygen monitoring.

60.1 Overview

Aggressive hyperventilation (arterial $PaCO_2 \leq$ 3.3 kPa) has been a cornerstone in the management of severe traumatic brain injury (TBI) for more than 20 years because it can cause a rapid reduction in ICP. Brain swelling and elevated ICP develop in 40% of patients with severe TBI (Miller et al. 1977), and high or uncontrolled ICP is one of the most common causes of death and neurologic disability after TBI (Becker et al. 1977). Therefore, the assumption has been made that hyperventilation benefits all patients with severe TBI. In a 1995 survey, Ghajar et al. found that hyperventilation was being used by 83% of US trauma centres (Ghajar et al. 1995). This number has declined over the years, but as recent as 2008, the BrainIT group made a survey in European centres showing that early prophylactic hyperventilation was used in more than 50% of cases (Neumann et al. 2008). However, hyperventilation reduces ICP by causing cerebral vasoconstriction and a subsequent reduction in CBF and cerebral blood volume and not by reducing brain oedema (Raichle and Plum 1972). Research conducted over the past 20 years clearly demonstrate that CBF during the first day after injury is less than half that of normal individuals (Bouma et al. 1992; Muizelaar et al. 1989; Robertson et al. 1988) and that there is a risk of causing or worsening cerebral ischaemia with aggressive hyperventilation, especially in the already ischaemic penumbra zone. Histologic evidence of cerebral ischaemia has been found in most victims of severe TBI who died (Graham and Adams 1971; Ross et al. 1993). A randomised study found significantly poorer outcomes at 3 and

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6 months when prophylactic hyperventilation was used, as compared to when it was not (Muizelaar et al. 1991). Thus, limiting the use of hyperventilation following severe TBI may help to improve neurologic recovery following injury or at least avoid iatrogenic cerebral ischaemia.

Tips, Tricks and Pitfalls

- If the ICP is stable and below 20 mmHg, keep the $PaCO_2$ above 4.5 kPa.
- If the patient is monitored with $ETCO_2$ and the pulmonary condition is stable, compare $ETCO_2$ with $PaCO_2$ on a regular basis (once or twice a day). This will diminish the need for arterial blood gas monitoring since the difference between $PaCO_2$ and $ETCO_2$ will, for all good measures, be approximately the same if the pulmonary condition is stable.
- If the ventilator is on pressure control and you want to decrease $PaCO_2$, increase the frequency of ventilations rather than the tidal volume because this will not alter the difference between $ETCO_2$ and $PaCO_2$.
- If the ventilator is on volume control and you increase the minute volume, keep an eye on the tidal volume to decrease the risk of overextension of the lungs.
- It is easier to keep adequate ventilation volume if the ventilator setting is on volume control.

60.2 Background

60.2.1 CBF Following TBI

Three studies provide Class III evidence that CBF can be dangerously low soon after severe TBI (Bouma et al. 1992; Marion et al. 1991; Sioutos et al. 1995). Two CBF measurements were performed with xenon-CT/CBF method during the first 5 days following severe TBI in a total of 67 patients. CBF measurements were obtained during the first 24 h after injury in one study, and they were less than 18 mL/100 g/min in 31.4% of patients (Bouma et al. 1992). In the second study, the mean CBF during the first few hours after injury was 27 mL/100 g/min (Marion et al. 1991). The third study measured CBF with a thermodiffusion blood flow probe, again during the first 5 days post-injury, in 37 severe TBI patients (Sioutos et al. 1995). Twelve patients had a global CBF less than 18 mL/100 g/min up to 48 h post-injury.

60.2.2 $PaCO_2$/CBF Reactivity and Cerebral Oxygen Utilisation

Three Class III studies provide the evidence base for this topic (Imberti et al. 2002; Oertel et al. 2002 and Sheinberg et al. 1992). Results associating hyperventilation with SjO_2 and $PtiO_2$ values in a total of 102 patients are equivocal. One study showed no consistent positive or negative change in SjO_2 or $PtiO_2$ values (Imberti et al. 2002). A second study associated hyperventilation with a reduction of $PaCO_2$ and subsequent decrease in SjO_2 from 73% to 67%, but the SjO_2 values never dropped below 55% (Oertel et al. 2002). The third study reported hyperventilation to be the second most common identifiable cause of jugular venous oxygen desaturation in a sample of 33 patients (Sheinberg et al. 1992). Studies on regional CBF showed significant variation in reduction in CBF following TBI. Two studies indicated lowest flows in brain tissue surrounding contusions or underlying subdural haematomas and in patients with severe diffuse injuries (Marion et al. 1991; Salvant and Muizelaar 1993). Similarly, a third study found that CO_2 vasoreactivity was most abnormal in contusions and subdural haematomas (McLaughlin and Marion 1996). Considering that CO_2 vasoreactivity could range from almost absent to three times normal in these patients, there could be a dangerous reduction in CBF during hyperventilation,

especially in areas surrounding contusions or underlying subdural clots. Only one of these three studies (Marion et al. 1991) had adequate design and sample to be included as evidence. Two studies associated hyperventilation-induced reduction in CBF with a significant increase in oxygen extraction fraction, but they did not find a significant relationship between hyperventilation and change in the $CMRO_2$ (Diringer et al. 2002; Hutchinson et al. 2002).

60.2.3 Effect of Hyperventilation on Outcome

One Class III study involving 890 patients intubated prehospitally showed adverse outcome for both hypo- and hypercapnic patients; intubated patients arriving at the trauma centre with an $PaCO_2$ between 4.0 and 6.5 kPa had a better survival than those who had been hypo- or hyperventilated, after adjustment for confounding factors (adjusted OR 2.17) (Davis et al. 2006).

One Class II RCT of 113 patients used a stratified, randomised design to compare outcomes of severe TBI patients provided normal ventilation ($PaCO_2$ 4.66 ± 0.25 kPa; $n = 41$; control group), hyperventilation ($PaCO_2$ 3.33 + 0.25 kPa; $n = 36$) or hyperventilation with tromethamine ($n = 36$) (Muizelaar et al. 1991). One benefit of hyperventilation is the minimisation of cerebrospinal fluid (CSF) acidosis. However, the effect on CSF pH may not be sustained due to a loss of HCO^{-3} buffer. Tromethamine (THAM) buffer treatment was introduced to test the hypothesis that it could reverse the effects of the loss of buffer. Patients were stratified based on the motor component of the Glasgow Coma Scale (GCS) score (1–3 vs 4–5). The Glasgow Outcome Scale (GOS) score was used to assess patient outcomes at 3, 6 and 12 months. For patients with a motor GCS of 4–5, the 3- and 6-month GOS scores were significantly lower in the hyperventilated patients than in the control or THAM groups. However, the effect was not sustained at 12 months. The recommendation today is that hyperventilation should be avoided.

60.3 Specific Paediatric Concerns

There are no specific paediatric concerns; children should be treated in accordance with the adult guidelines.

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