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Cerebral oxygen saturation measured by near-infrared spectroscopy and jugular oxygen saturation

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Keywords

Brain, haemodynamics, spectroscopy, transcranial doppler

Comments

This paper examines an important question of whether extrapolation of data from normal subjects to those with critical illness, is valid. The answer would seem to be that in this case, it is not. In addition to errors with equipment algorithms or technical failures, the dramatically altered physiology in acute closed head injury would appear to make SCO_2an invalid monitor of cerebral oxygenation.

Introduction

Several studies have shown that cerebral oxygen saturation (SCO_2), measured by near infrared spectroscopy (NIRS), responds rapidly to changes in cerebral perfusion in normal subjects. The method has not been studied in more complex situations such as in neuro ICU. In this setting, changes in cerebral blood flow and vascular tone may alter the arteriovenous (AV) distribution of blood in the scalp and extracerebral tissues, leading to inaccuracies.

Aims

To study the relationship between NIRS SCO_2 and jugular venous oxygen saturation ($SjvO_2$) during changes in arterial carbon dioxide tension and blood pressure, in adults with head trauma.

Methods

In total, nine patients with severe closed head injury and diffuse brain swelling or multifocal contusions were studied in the first 10 days following their injury. All patients were mechanically ventilated and sedated, those who were hypotension (MAP < 70mm Hg) prior to the start of the study

received a norepinephrine infusion. A jugular catheter was inserted into the side of the dominant jugular vein. SCO_2 was recorded using NIRS and middle cerebral artery blood velocity (MACv) was measured using pulsed Doppler. $PaCO_2$ was then altered to a level of moderate hypocapnia ($PaCO_2$ 30-35), intense hypercapnia ($PaCO_2$ 20-25 mm Hg) or moderate hypercapnia. Cerebral vascular resistance was calculated as cerebral perfusion pressure (CPP) divided by MCAv and was measured before and after the CO_2changes. A response to changes in MAP was then examined by either starting or stopping a norepinephrine infusion depending on whether the patient was at the upper or lower limit of the cerebral autoregulation curve.

Results

Data were plotted according to the method of Bland and Altmann. The plots showed decreasing differences between changes in SCO_2 and $SvjO_2$ when the mean of this difference increased, suggesting that the bias was not random. Regression analysis showed that both tests were different in terms of magnitude and also that the relationship between changes in SCO_2 and $SvjO_2$ during $CO_2$and pressure were opposite.

Discussion

SCO_2, assessed by NIRS, does not adequately reflect changes in $SvjO_2$ in patients with severe head injury. The changes in extracerebral tissues on SCO_2 is critical. The use of norepinephrine, in cases with impaired autoregulation, will lead to increased cerebral blood flow in proportion to MAP, and decreased blood flow to the extracerebral tissues. Conversely increased CO_2leads to global increases in blood flow in both regions. These findings may not be apparent in studies looking at decreases in oxygen saturation which would be the same in all compartments (eg hypoxic challenges). In addition the physiology of these patients is complex, with regional variations in cerebral blood flow and areas of ischaemia and hyperaemia.

References

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