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SHORT REPORT

Extracerebral absorption of near infrared light influences the detection of increased cerebral oxygenation monitored by near infrared spectroscopy

Timothy J Germon, Amber E R Young, Alexander R Manara, Richard J Nelson

Abstract

The detection of increased cerebral oxygenation secondary to cerebral hyperaemia, induced by hypercapnia has been studied in anaesthetised patients using a near infrared, reflectance mode, cerebral oxygenation monitor (Invos 3100 Somanetics, Troy, Michigan, USA). Two studies were performed, with and without a pneumatic scalp tourniquet, to distinguish between extracranial and intracranial changes in tissue oxygenation. In the control study a mean increase in end tidal CO_2 of 23·1 mm Hg was accompanied by a mean increase in middle cerebral artery flow velocity of 116%. Regional cerebral oxygen saturation (rSo_2) measured transcutaneously in the frontal distribution of the middle cerebral artery increased significantly from 70·5% to 74·6% (p = 0·001). During the second study with a scalp tourniquet inflated to maintain the extracranial tissues in a state of stable ischaemia a mean increase in end tidal CO_2 of 22·3 mm Hg was accompanied by a mean increase in middle cerebral artery flow velocity of 121%. The change in rSo_2 from 62·6% to 64·5% was not significant (p = 0·085). There was no correlation between the change in middle cerebral artery flow velocity and rSo_2 in the control or scalp ischaemia group.

This study shows that the Invos 3100 monitor is sensitive to tissue oxygenation but does not reliably detect changes in cerebral oxygenation as a result of profound cerebral hyperaemia. The contribution of extracerebral tissue to the attenuation of near infrared light and the lack of spatial resolution remain major problems to be overcome before this or other near infrared spectroscopy instruments can be introduced into clinical practice.

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Keywords: near infrared spectroscopy; increased cerebral oxygenation

Department of Neurosurgery
T J Germon
R J Nelson

Department of Anaesthetics, Frenchay Hospital, Frenchay, Bristol BS16 1LE, UK
A E R Young
A R Manara

Correspondence to: Mr T J Germon, Department of Neurosurgery, Frenchay Hospital, Frenchay, Bristol BS16 1LE, UK.

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Near infrared spectroscopy, a non-invasive technique for monitoring cerebral oxygenation and blood flow, semiquantitatively estimates the relative concentrations of oxyhaemoglobin and deoxyhaemoglobin within an unspecified volume of tissue illuminated by near infrared light of varying wavelengths. Absorption of the near infrared light takes place in the arterial, capillary, and venous circulations of both the extracranial and intracranial tissues. The technique possesses no intrinsic compartmental or spatial resolution and thus to be clinically useful the effects of changes in extracranial blood flow and oxygenation must be either quantified or made negligible by the design of the optical sensor and its software.

We have examined the capacity of the Invos 3100 cerebral oximeter to detect profound increases in cerebral blood flow and oxygenation induced by hypercapnia before and after the application of a pneumatic scalp tourniquet which results in stable scalp ischaemia.

Methods

Eight patients (mean age 44·5, range 30–66 years) undergoing cervical or lumbar discectomy with no predisposition to cerebrovascular disease gave informed verbal consent to the study, which was approved by the local ethics committee. A uniform anaesthetic technique was employed. The electrocardiogram (Hewlett Packard 78353B, GMBH, Germany), arterial blood pressure (Dinamap 845 XT, Critikon, Tampa, FL, USA), regional cerebral O_2 saturation (rSo_2), inspired O_2 and CO_2 concentrations, and end tidal CO_2 ($ETco_2$) concentrations (Nellcor N-1000, Hayward, CA, USA) were continuously monitored. The oximeter sensor was placed on the forehead, avoiding the midline and the temporalis muscle. Ipsilateral middle cerebral artery flow velocity was measured in the proximal middle cerebral artery at a depth of 5·0 to 6·0 cm with transcranial Doppler ultrasonography (PCDop 842, Scimed, Bristol, UK).

When $ETco_2$ was stable in the range 22–45 mm Hg five recordings were made. The inspired CO_2 concentration was increased to 5% and when $ETco_2$ had reached a stable level for three minutes five further recordings were made. The study was then repeated with a pneumatic tourniquet

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Mean values (SD) for the variables measured during normocapnia and hypercapnia in control and scalp ischaemia studies

	Control study			Scalp ischaemia		
	Normocapnia	Hypercapnia	P value	Normocapnia	Hypercapnia	P value
Heart rate (6 pm)	66 (10)	63 (9)	0·05	64 (13)	63 (12)	0·44
MAP (mm Hg)	77 (7)	83 (8)	0·05	79 (7)	82 (10)	0·03
$ETCO_2$ (mm Hg)	28·6 (6)	51·7 (5)	< 0·01	28·9 (6)	51·2 (6)	< 0·01
vMCA (cm/s)	37 (13)	80 (23)	< 0·01	38 (17)	84 (24)	< 0·01
rSO_2 (%)	70·5 (4)	74·6 (4)	0·001	62·6 (8)	64·5 (10)	0·085

inflated to a pressure of 200 mm Hg at the level of the supraorbital ridge for three minutes. Care was taken to ensure that the tourniquet did not impinge on the sensor.

A paired Student's *t* test was used to examine for significant differences between the study variables under normocapnic and hypercapnic conditions. The relation between the percentage change in middle cerebral artery velocity and the change in rSO_2 was studied with Pearson's rank correlation.

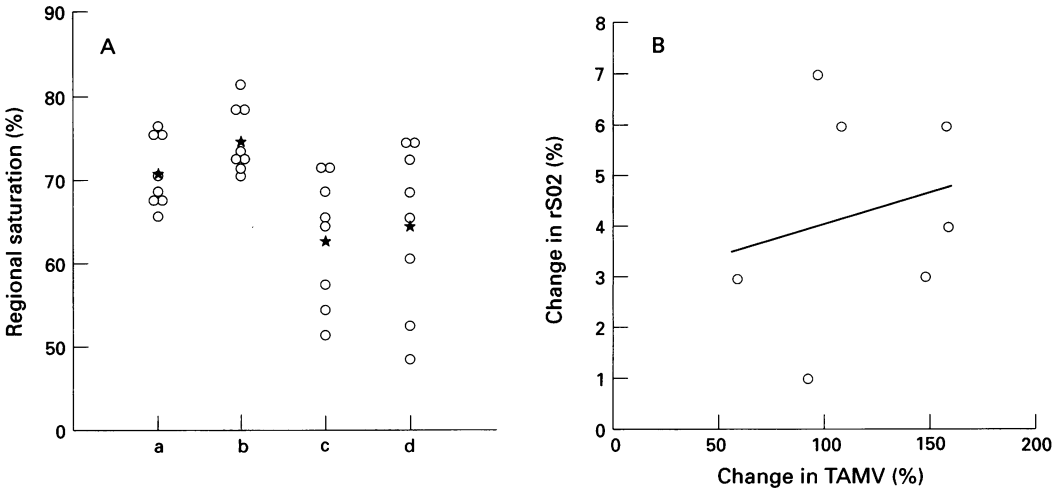
Results

The table summarises the results. The mean increase in $ETCO_2$ was 23·1 (SD 4) mm Hg in the control study and 22·3 (3) mm Hg when the scalp tourniquet was inflated. (In one patient without a satisfactory acoustic window for transcranial Doppler, $ETCO_2$ rose by 20 mm Hg without the tourniquet and by 23 mm Hg with the tourniquet, and therefore, his rSO_2 results are included.) There was no significant difference between the mean increase in middle cerebral artery velocity in the two studies (p = 0·21). There was a significant increase in mean rSO_2 from 70·5% to 74·6% in the control study (p = 0·001, figure). Under conditions of stable extracranial ischaemia, however, no significant change in rSO_2 was detected in response to hypercapnia. (Mean rSO_2 increased from 62·6% to 64·5%, p = 0·085, figure). There was no correlation between the changes in middle cerebral artery velocity and rSO_2 in the control (r = 0·229, p = 0·621) or scalp ischaemia (r = 0·183, p = 0·398) groups.

Discussion

Carbon dioxide is a potent cerebral vasodilator. The increase in cerebral blood flow mediated by cortical arteriolar dilatation[1] in response to increased CO_2 does not increase cerebral metabolic requirements for oxygen.[2] This results in an increased cerebral blood volume[3] of oxygenated arteriolar blood and an increase in the saturation of cerebral venous blood secondary to increased blood flow with constant O_2 extraction. It follows that rSO_2 should increase significantly in response to hypercapnia and there should be a close relation between increased flow velocity detected by transcranial Doppler and increased cerebral oxygenation detected by near infrared spectroscopy.

Assuming that 70% of cerebral blood volume is venous and 30% arterial and that normal jugular venous oxygen (SjO_2) saturation lies in the range 54% to 75% then the normal range for rSO_2 should lie between 67% and 85%. Mean rSO_2 in our control study was 70·5%. The increase in middle cerebral artery velocity in the two studies is compatible with an increase in global cerebral blood flow of about 120%. Assuming no appreciable change in the distribution of cerebral blood volumes or the oxygen dissociation curve for haemoglobin and a mean SjO_2 in the midrange of normal (65%) during normocapnia, we estimate that a 120% increase in cerebral blood flow would increase rSO_2 from 76% to 88%. With the same rationale, the mean normocapnic value for rSO_2 of 70·5% should have increased to 86%. Although the increase in rSO_2 from 70·5% to 74·6% in the



(A) Individual (circles) and mean (stars) values of rSO_2 for each of the four study conditions: a = control, normocapnia; b = control, hypercapnia; c = scalp ischaemia, normocapnia; d = scalp ischaemia, hypercapnia. (B) Scatter plot of change in rSO_2 against % change in time averaged mean flow velocity (TAMV) in the middle cerebral artery in response to hypercapnia under control conditions (no scalp ischaemia, r = 0·229, p = 0·621).

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control study was significant it clearly does not reflect the magnitude of change predicted and further, there was no correlation between changes in middle cerebral artery flow velocity and rSO_2 in either study.

In an attempt to overcome the problems of extracerebral attenuation of near infrared light and to increase the degree of spatial resolution the cerebral oximeter used in this study has two receiving optodes placed 3 and 4 cm from the emitting optode. Subtraction of the absorption characteristics detected at the proximal optode from those at the distal optode should reflect changes taking place in cerebral tissue alone.[4] With this optode arrangement we have previously shown that the induction of scalp ischaemia causes a significant reduction in rSO_2 as measured by the Invos 3100.[5] This may mean the mathematical algorithm that computes rSO_2 may not compensate for the changes in scalp oxygenation. It seems unlikely that the baseline mean rSO_2 of 62·6% in the second study reflects true regional O_2 saturation in the stable anaesthetised patient. Nevertheless, the principle of the proximal and distal path lengths should apply to the new stable conditions with the sensor capable of detecting the cerebral hyperaemia unaffected by extracranial change. The small and statistically insignificant increase in rSO_2 to 64·5% in the presence of scalp ischaemia strongly suggests that the increase in rSO_2 seen in response to hypercapnia in the absence of the tourniquet is largely due to changes in scalp oxygenation.

With this optode arrangement, the Invos 3100 monitor will detect tissue hypoxia deep to the scalp under conditions identical with those described.[5] We speculated whether the monitor was detecting changes in haemoglobin oxygenation in the diploic frontal bone and underlying dura, in the cerebral cortex, or in both. The failure to detect changes in oxygenation in response to hypercapnia with scalp ischaemia suggests that very little of the near infrared light signal change is taking place in the cerebral cortex and that hypercapnia does not increase blood flow to the skull. In similar work Harris and Bailey failed to detect any significant response to hypercapnia using a sensor configuration with a smaller separation between the light source and the two receivers.[6] It is possible that this sensor geometry was successful in excluding hyperaemic change in the scalp while remaining insensitive to change in the cerebral substance.

The problem of extracerebral attenuation of near infrared light in near infrared spectroscopy is not simply a theoretical consideration. Those patients most likely to benefit from this monitoring are also those most likely to have local change in the extracranial tissues—for example, after head injury or craniotomy—and systemic changes in cardiac output, peripheral resistance, and cutaneous vasodilatation, which also affect the extracranial tissues. Until the problems of quantifying and interpreting changes in rSO_2 under physiological and pathological conditions have been overcome near infrared spectroscopy monitors of this sort cannot be recommended for routine clinical monitoring. Other instruments with similar technology should be tested equally rigorously.

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