Estimation of Cerebral Venous Saturation in Newborn Infants by Near Infrared Spectroscopy

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ABSTRACT. The purpose of the study was to evaluate the near infrared spectroscopy technique for determination of the cerebral venous oxygen saturation. By tilting the patient's head down 15°, changes in OD may be caused by changes in cerebral blood volume. On the assumption that increases in cerebral blood volume consist of venous blood only, cerebral venous oxygen saturation can be calculated as the measured change in oxygenated Hb divided by the change in total Hb. Two groups of mechanically ventilated, newborn infants were investigated: 10 asphyxiated, term infants and another 22 preterm infants with respiratory distress syndrome. All were monitored by near infrared spectroscopy during tilting, and cerebral blood flow was estimated by the ^{133}Xe clearance technique immediately before tilting. Cerebral venous oxygen saturation could not be calculated in 13 preterm infants, as the blood volume remained constant during tilting. In the remaining 19, cerebral venous oxygen saturation averaged 0.67 (SD = 0.09) in asphyxiated infants and 0.53 (SD = 0.15) in preterm infants ($p = 0.03$). The corresponding values of cerebral blood flow were closely and inversely related to oxygen extraction calculated from cerebral venous saturation as estimated by near infrared spectroscopy. (*Pediatr Res* 33: 52–55, 1993)

Abbreviations

CBF, cerebral blood flow (mL/100 g/min)
CBV, cerebral blood volume (mL/100 g)
CMRO₂, cerebral metabolic rate of O₂ (mL O₂/100 g/min)
MABP, mean arterial blood pressure
NIRS, near infrared spectroscopy
COD, cerebral oxygen delivery (mL/100 g/min)
OE, oxygen extraction (mL O₂/100 g/min)
SaO₂, arterial saturation
CSvO₂, cerebral mixed venous saturation
HbO₂, oxygenated hemoglobin
deoxy-Hb, deoxygenated hemoglobin
PaO₂, arterial oxygen tension
PaCO₂, arterial carbon dioxide tension

In at least two situations, it would be valuable to have information on CSvO₂ in the sick, newborn infant. Very low levels of CBF have been found among mechanically ventilated, preterm infants, and these infants may be at high risk of ischemic brain lesions (1). When CBF is low, however, the brain may escape from ischemia by enhancing the OE from the blood, resulting in low CSvO₂ values. Conversely, CBF is markedly elevated after

severe birth asphyxia (2). In this hyperperfusion state, a high CSvO₂ would represent "luxury perfusion," indicating serious brain damage (3), whereas a normal CSvO₂ might demonstrate an intact coupling between CBF and the metabolic needs.

It is not practical to sample cerebral venous blood, *e.g.* from the internal jugular vein, in view of the invasive nature of the technique (4). Recently, it has been suggested that the cerebral venous oxygen content can be estimated noninvasively by NIRS (5).

The purpose of this study was to evaluate whether the estimated CSvO₂ was related to the simultaneously measured CBF.

MATERIALS AND METHODS

Patients. Two groups of mechanically ventilated, newborn infants were investigated: 10 asphyxiated, term infants with umbilical cord pH less than 7.10 and/or a 5-min Apgar score less than 5 and another 22 preterm infants with respiratory distress syndrome (Table 1).

NIRS. NIRS depends on the relative transparency of the neonate's head to near infrared light. The light is attenuated by scattering and absorption in the tissues. Our NIRS instrument (Radiometer, Copenhagen, Denmark) uses four semiconductor laser diodes with wavelengths of 775, 805, 845, and 904 nm. The lasers are operated sequentially and pulsed for 200 ns with a repetition rate of 500 Hz. Optical fiber bundles are used to transmit and receive the light (6, 7). In the present study, the NIRS recordings were performed in transmission mode in low-birth-weight infants, whereas larger infants were investigated in reflection mode. In both modes, the relation between optical path length and interoptode spacing appears to be constant, as recently observed in time-of-flight studies (8). The calculation of CSvO₂ does not require any information on the optical path length, as CSvO₂ is expressed in fractions. Conversely, the optical path length must be known for the calculation of changes in CBV (Table 1). Significant changes in CBV were required for estimation of CSvO₂, and CBV was calculated as previously suggested (9). We assumed that the optical light path length is $4.4 \times$ the interoptode distance (10).

Estimation of CSvO₂. CSvO₂ was estimated by tilting the infant head-down 15°. The acute alterations in the cerebral concentration of HbO₂ and deoxy-Hb that result from tilting may be assumed to be caused by an increase in venous blood only. By comparing the incremental changes in HbO₂ and deoxy-Hb, CSvO₂ can be calculated from the formula:

$$\text{CSvO}_2 = \Delta(\text{HbO}_2) / \Delta[(\text{HbO}_2) + (\text{deoxy-Hb})]$$

where CSvO₂ is expressed as a fraction. CSvO₂ was derived from stable 90-s periods before and during tilting.

CBF. CBF was measured by the ^{133}Xe clearance technique, as previously described (11). Briefly, 20–40 MBq/kg ^{133}Xe were injected into a peripheral vein, and the clearance was recorded by scintillation detectors placed over one frontoparietal region and the thorax, respectively. A recording time of 15 min was

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Table 1. Comparison of physiologic data between preterm infants and asphyxiated term infants*

	Preterm		Term (n = 10)	p
	Nonreacting (n = 13)	Reacting (n = 9)		
Birth weight (g)	1184 (313) ^a	1436 (523) ^b	3012 (650) ^{a,b}	<0.0001
Gestational age (wk)	27.9 (1.7) ^b	29.4 (2.2) ^a	38.1 (1.1) ^{a,b}	<0.0001
Postnatal age (h)	22.8 (16.0)	32.0 (16.4) ^b	15.7 (8.2) ^b	0.06
FiO ₂	0.41 (0.10)	0.53 (0.25)	0.49 (0.23)	0.35
Ventilation (breaths/min)	41 (16) ^{a,b}	69 (31) ^b	32 (11) ^a	<0.001
MABP (mm Hg)	38.0 (7.8)	37.3 (7.5)	45.0 (10.5)	0.11
PaO ₂ (kPa)	8.9 (1.6)	10.3 (3.0)	9.6 (2.2)	0.41
PaCO ₂ (kPa)	4.9 (1.6)	4.1 (1.0)	4.0 (0.6)	0.15
SaO ₂	0.97 (0.04)	0.96 (0.05)	0.94 (7.0)	0.42
Hb (mM)	9.0 (1.5)	9.9 (1.4)	10.3 (1.9)	0.13
CBF (mL/100 g/min)	11.1 (3.9) ^a	12.6 (6.4) ^b	26.5 (17.9) ^{a,b}	0.005
CBV change (mL/100 g)		0.13 (0.08)	0.16 (0.07)	0.37
CMRO ₂		1.0 (0.4)	1.4 (0.8)	0.17
COD (mL/100 g/min)	2.5 (0.5) ^a	2.0 (0.7) ^b	5.7 (4.3) ^{a,b}	0.005
OE (mL O ₂ /100 g/min)		0.09 (0.03) ^b	0.06 (0.02) ^b	0.01
CSvO ₂ (%)		53.44 (15.36) ^b	67.30 (9.38) ^b	0.03
Interoptode distance (cm)	5.3 (0.5) ^a	5.4 (0.7) ^b	4.6 (0.9) ^{a,b}	0.02

* Values are mean ± SD. Preterm infants are grouped according to the response to tilting down. *p* values are calculated by analysis of variance. FiO₂, fraction of inspired oxygen. Superscript letters indicate significant differences between two groups.

used. CBF was calculated from the time when the activity of ¹³³Xe in the lung had decreased to 15% of the peak activity.

COD was calculated as the product of physically dissolved and Hb-bound O₂ and CBF:

$$\text{COD} = [(\text{PaO}_2 \times 0.227 + \text{SaO}_2 \times 21.0 \times \text{Hb}) / 1000] \times \text{CBF mL O}_2 / 100 \text{ g/min}$$

SaO₂ was measured by pulse oximetry (Datex, Helsinki, Finland). MABP was recorded from an indwelling umbilical catheter, and the transducer was kept at heart level.

Study design. CBF and CSvO₂ were measured after the infant had been undisturbed for at least 30 min. At the end of the CBF measurement, the infant was tilted head-down for a 3-min period and he or she was subsequently returned to horizontal level. Arterial blood was drawn for analysis of blood gases (ABL, Radiometer), Hb concentration, and oxygen saturation (OSM3, Radiometer). It was assumed that CBF and oxygen consumption remained constant during the tilting maneuver.

Statistics. Linear regression analysis and analysis of variance were used when appropriate. Multiple comparisons between groups were performed by the Student-Newman-Keuls procedure.

The study was approved by the Ethics Committee for Greater Copenhagen, and parental informed consent was obtained for each infant.

RESULTS

Response to tilting. A marked and significant increment in CBV in response to tilting was observed in 19 infants. However, in 13 of the 22 preterm infants, CBV did not respond significantly during the procedure. Apparently, these nonreacting preterm infants required lower ventilatory rates compared with the nine reacting preterm infants, whereas other parameters were identical (Table 1). The nonreacting infants were excluded from the following calculations.

In reacting infants, the response to tilting appeared within 15 to 20 s with an increase in HbO₂, and deoxy-Hb, and hence in CBV. These variables returned to baseline levels when the infant was returned to the horizontal position (Fig. 1). The increase in CBV was identical in asphyxiated, term infants and preterm infants (Table 1), and these changes were not statistically related to changes in MABP or to the PaCO₂ level.

CSvO₂ and CBF. In all infants, CSvO₂ ranged from 0.30 to

Fig. 1. Changes in cerebral concentration of CBV (■), oxyhemoglobin (+), and MABP (◇) in response to tilting in a preterm infant. CBF = 18 mL/100 g/min, CSvO₂ = 0.61.

0.80 (mean 0.61), whereas CBF averaged 19.9 mL/100 g/min (range 5.1–60.5). CSvO₂ as well as CBF were higher in the asphyxiated, term infants compared with preterm infants with respiratory distress (Table 1). CSvO₂ was not related to the level of PaCO₂ or PaO₂.

OE, COD, and CMRO₂. OE, calculated as the product of blood Hb concentration and difference between SaO₂ and CSvO₂, was significantly higher in preterm infants than in asphyxiated, term infants (Table 1). Despite interindividual variation, CSvO₂ was curvilinearly related to COD, with a high slope at low COD levels and a plateau at higher levels (Fig. 2). Similarly, an apparent linear relationship was observed between OE and 1/CBF (*P* < 0.001), where the slope (OE × CBF) represents CMRO₂ (Fig. 3). CMRO₂ was identical between the two groups (Table 1).

Case study. Low CBF, low OE, and hence a very low CMRO₂ were demonstrated in a term infant with presumed prenatal asphyxia. This infant had an isoelectric EEG, and he died on the 3rd d of life. Extensive cerebral cystic degeneration, which could be dated to 3–4 wk before delivery, was demonstrated at autopsy, thus explaining the discrepancy between a high CSvO₂ and a low CBF.

DISCUSSION

The increase in CBV during a head-down tilt was significant in the majority of infants allowing calculation of CSvO₂. We

Fig. 2. CSvO₂ vs COD of 19 infants. ▼, preterm infants; □, asphyxiated infants; *, term infant with presumed prenatal asphyxia.

Fig. 3. Cerebral OE vs 1/CBF of 19 infants. The slope represents CMRO₂ (CMRO₂ = OE × CBF). ▼, preterm infants; □, asphyxiated infants; *, term infant with presumed prenatal asphyxia.

have, however, no explanation for why CBV remained constant in 13 preterm infants although the ventilatory rate was lower than that of reacting infants. We did not measure the central venous pressure, and it is possible that high pressures influence changes in CBV during tilting. Another hypothetical explanation is the reactivity of CBV at different levels of PaCO₂ and PaO₂, but these variables were similar among reacting and nonreacting infants. Finally, the presence of a venous autoregulatory capacity in the more healthy infants may be relevant (12). Future studies are needed to evaluate whether the failing response is due to biologic or methodologic factors. Besides, the reproducibility of the method should be tested.

There are reasons to believe that CSvO₂ was estimated reliably in the reacting infants: 1) CBV increased rapidly during tilting, thereby indicating a vascular origin; 2) the magnitude of changes of 0.15 mL/100 g corresponds to 5–10% of total CBV; and 3) the estimated ranges of CSvO₂ and OE are physiologically acceptable.

We recognize that the ideal would have been to examine infants with normal brains in which a normal CMRO₂ can be taken for granted. For practical purposes (lack of reaction, excessive movement artifacts), this is not possible. We have reasons, however, to suppose that CMRO₂ in the ill infants we studied did not vary markedly: 1) In preterm infants with very low CBF, there are no signs of electrical failure, *i.e.* no sign of a major decrease in oxygen consumption (13). 2) In asphyxiated term infants with severe asphyxia and very high CBF, an isoelectrical EEG and/or subsequent brain damage indicate that CMRO₂ is not increased in parallel. CMRO₂ may be somewhat above or

even below normal depending on the sum of normal and abnormal metabolic processes occurring during the transformation of the initial insult into cell death and tissue damage. Hence, although the relation between CSvO₂ and CBF cannot be expected to be perfect, it should be sufficient for a preliminary validation to indicate that this method gives physiologically meaningful values. The accuracy of the method must await further studies.

Some possible confounding factors should be considered. An arterial contribution to the increase in CBV during the tilt-down may have occurred though the increases of CBV were not related to the concomitant changes in MABP. Accurate quantification of CSvO₂ requires that the optical path length remains constant during the tilt, and two sources of error are likely to occur in this regard: 1) The increase in CBV may induce a small increase in head circumference and hence an increase in optode distance, and 2) higher cerebral Hb concentration will increase the absorption. Higher absorption will tend to attenuate photons that have traveled a greater distance, thus shortening the optical path length. Such errors could lead to under- or overestimation of CSvO₂. The observed changes in OD at the four wave lengths could not entirely be explained by changes in Hb concentrations as the residuals, although small, after having accounted for Hb nevertheless were systematically different from zero.

Venous saturation has previously been measured in animal experiments using multiwavelength derivative spectroscopy (14), and CSvO₂ estimated by this method was in good agreement with the oxygen saturation of blood drawn from the superior sagittal sinus over the range of 0.015 to 0.70. In that study, the optical fibers were placed directly on the bone over the sagittal sinus with an interoptode distance of 1–2 cm and hence with a very small sampling volume. It is possible that this method may be adapted to preterm infants in whom the scalp is only a few millimeters thick.

The potential usefulness of measuring CBF and CMRO₂ in infants has already been discussed by Garfunkel *et al.* in 1954 (15). Three of the 33 infants studied by the nitrous oxide method after internal jugular venous catheterization were newborn (age 2–19 d), and they had very low CMRO₂ values (mean 1.5 mL O₂/100 g/min) that actually are comparable to those of the present study, indicating that the absolute values of CSvO₂ obtained by NIRS are of the right order of magnitude. This interpretation is supported by the observations of normal neuronal outcome in preterm infants with a very low CBF (16, 17).

Conversely, we found very high CBF and low OE in some asphyxiated infants, indicating cerebral luxury perfusion. If the cerebral hyperperfusion was coupled to a high CMRO₂, higher OE values should have been recorded. These results are in agreement with observations by Frewen *et al.* (18) using the Kety-Schmidt method. The high CSvO₂ in one infant with low CBF and pathologic evidence of past brain injury also supports the accuracy of the CSvO₂ measurements.

Conclusion. We suggest that CSvO₂ may be estimated in sick newborn infants by NIRS. The ranges of CSvO₂ and OE were physiologically acceptable and closely related to CBF as measured by an independent technique. Future studies are necessary to clarify why the NIRS technique fails in some preterm infants.

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