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Correlation Between Jugular Bulb Oxygen Saturation and Partial Pressure of Brain Tissue Oxygen During CO_2 and O_2 Reactivity Tests in Severely Head-Injured Patients

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Summary

Purpose. To correlate the jugular bulb oxygen saturation ($SjvO_2$) and brain tissue oxygen pressure ($PbtO_2$) during carbon dioxide (CO_2) and oxygen (O_2) reactivity tests in severely head-injured patients.

Methods and Results. In nine patients (7 men, 2 women, age: 26 ± 6.5 years, GCS of 6.5 ± 2.9), a polarographic microcatheter (Clark-type) was inserted into nonlesioned white matter (frontal lobe). $PbtO_2$ and $SjvO_2$ were monitored simultaneously and cerebral vasoreactivity to CO_2 and O_2 was tested on days three, five and seven after injury. Simultaneous measurements of vasoreactivity by transcranial Doppler (TCD) were undertaken. A total of twenty-one CO_2 and O_2 reactivity tests were performed. Critical values of $PbtO_2$ (<15 mm Hg) during induced hyperventilation could be observed four times in two patients. High $PbtO_2$ values up to 80 mm Hg were observed during hyperoxygenation (FiO_2 100%). CO_2 vasoreactivity by means of $PbtO_2$ was absent in four tests in which measurements by TCD showed intact responses. A stronger correlation between $SjvO_2$ and $PbtO_2$ during the O_2 reactivity tests was observed ($r = 0.6$, $p < 0.001$), in comparison to values obtained during the CO_2 reactivity tests ($r = 0.33$, $p < 0.001$). In addition, there was no statistically significant correlation ($r = 0.22$, $p = 0.26$) between CO_2 reactivity values measured by TCD ($4.5 \pm 5.7\%$) and $PbtO_2$ ($3 \pm 2.8\%$).

Conclusions. Correlation between $SjvO_2$ and $PbtO_2$ during CO_2 reactivity test is low, even if significant differences between normo- and hyperventilation values are present. In comparison to $SjvO_2$, monitoring of $PbtO_2$ might more accurately detect possible focal ischaemic events during rapidly induced hyperventilation in severely head-injured patients. The CO_2 vasoreactivity by means of changes in Vm MCA seems to be higher in comparison to changes of $PbtO_2$. These observations lead to the hypothesis that vasoreactivity measured by TCD overestimates the cerebrovascular response to CO_2.

Keywords: Head injury; brain tissue oxygen pressure; jugular bulb oxygen saturation; CO_2 and O_2 reactivity.

Introduction

In normal cerebral circulation, acute alterations in arterial CO_2 lead to changes in Cerebral Blood Flow (CBF) of 3 to 5% per mm Hg change in $PaCO_2$ [13, 14, 25, 37]. Carbon dioxide (CO_2) stimulation has been used to test the cerebrovascular reactivity in order to assess the adequacy of brain perfusion in patients with carotid artery disease [30, 34], subarachnoid haemorrhage (SAH) [39], intracerebral haemorrhage (ICH) [21], and brain injury [6, 10, 12, 17, 34]. The effects of hyperventilation on cerebral autoregulation have been investigated by transcranial Doppler sonography (TCD) [2, 25, 34]; nevertheless, this technique allows the determination of cerebral haemodynamics, but not be assessment of focal cerebral oxygenation patterns. In severely head-injured patients dynamic autoregulation may be impaired despite normal CO_2; however, it has been demonstrated that moderate transient hyperventilation can temporarily improve the efficiency of the autoregulation response [35]. The mechanism of this "improvement" during induced hyperventilation remains unclear and has been evaluated only in terms of flow velocities in the middle cerebral artery. Changes of focal cerebral oxygenation patterns during transient hyperventilation are unknown.

In the last decade, the introduction of continuous jugular bulb oxygen saturation monitoring ($SjvO_2$) and measurements of the arteriovenous difference of oxygen content ($AVDO_2$) and lactate (AVDL) have allowed new therapeutic approaches in severely head-injured patients [8, 9, 15, 38, 41]. More recently, brain tissue oxygen pressure monitoring ($PbtO_2$) has been introduced into the clinical practice [11, 20, 23, 31, 46, 47]. The brain $PbtO_2$ monitoring is based on the polarographic principle: at the sensitive site of a polarographic pO_2 electrode, oxygen, dissolved in aqueous

electrolyte solution, is converted to OH- in minimal quantity ($O_2 + 2H_2O + 4e- \rightarrow 4OH-$) [3, 23, 31]. Measurement of $PbtO_2$ constitutes another attempt to detect ischaemia and prevent secondary brain damage. Preliminary clinical studies have suggested the association between episodes of low $PbtO_2$ (< 15 mm Hg) and poor outcome or death [24, 44, 45].

The main purpose of this study was the assessment of focal cerebral oxygenation patterns during CO_2 and O_2 vasoreactivity tests by means of $PbtO_2$. Correlation between $SjvO_2$ and $PbtO_2$ values, and between the cerebrovascular response to CO_2 measured by TCD and $PbtO_2$ were evaluated.

Patients and Methods

Patient Population

Nine severely head-injured patients (seven men and two women) admitted to our Trauma Intensive Care Unit during a six-month period were included in this study. Demographic characteristics are shown in Table 1. The mean age was 26 ± 6.5 years (range: 17–37). Seven out of eight patients had an initial GCS ≤ 8 (6.5 ± 2.9). All patients underwent radiological evaluation with skull projections and computerized tomography (CT) immediately after admission to the hospital. CT classifications were made according to the National Institutes of Health Traumatic Coma Data Bank (TCDB) [30]. Eight patients had *diffuse lesions* and one patient had a *nonevacuated mass lesion* (Patient 8). Two patients presented with postraumatic subarachnoid haemorrhage (Patient 3 and 4). None of the patients required craniotomy for the removal of a haematoma. In one patient a large decompressive craniotomy was performed on day 3 after trauma and the $PbtO_2$ was replaced and reinserted in nonlesioned tissue. During ICU management, patients underwent CT follow-up within 48 hours after admission, before wake-up procedure, if ICP increased or became intractable with critical CPP, or if unexplained deterioration of cerebral oxygenation parameters appeared. The mean Injury Severity Score (ISS) [4] in this series was 26 ± 12.4. Six out of the nine patients had an ISS greater than 25. The Glasgow Outcome Scale score (GOS) was determined six months after injury by personnel who were unaware of the cerebral oxygenation data obtained during the intensive care management [16]. The patients were routinely controlled at the neurological department in a special outpatient unit for brain-injured patients. A standard battery of memory tests, social integration assessment and neurological examination were included in the evaluation.

Table 1. *Demographic Characteristics*

Patient	Age/sex	GCS	ISS	CT findings (TCDB category)	GOS
1	24/M	6	40	Diffuse Injury IV (shift)	GR
2	24/M	6	25	Diffuse Injury II	GR
3	33/F	7	41	Diffuse Injury II, tSAH	MD
4	22/M	3	26	Diffuse Injury II (bilateral), tSAH	MD
5	26/M	6	57	Diffuse Injury II	MD
6	32/F	7	17	Diffuse Injury II	GR
7	37 M	8	24	Diffuse Injury II (bilateral)	D
8	17/M	13	25	Nonevacuated mass lesion	MD
9	20/M	3	26	Diffuse Injury II	SD

M Male; *F* female; *ISS* injury severity score [4]; *GCS* Glasgow Coma Scale score; *tSAH* postraumatic subarachnoid haemorrhage; *GOS* Glasgow Outcome Scale score [16] after 3 months (*GR* good recovery; *MD* moderate disability; *SD* severe disability; *D* death); *TCDB* National Institutes of Health Traumatic Coma Data Bank [31].

Management of Intracranial Hypertension

The management protocol of brain-injured patients in our unit has been described elsewhere [42, 43]. The main goals of treatment included (1) immediate life support, especially achievement and maintenance of circulatory stability (mild hypervolaemia) and adequate gas exchange; (2) maintenance of CPP ≥ 70 mm Hg; (3) surgical treatment of extracranial lesions under ICP monitoring. Monitoring and therapeutic tools included haemodynamic monitoring with pulmonary artery catheter (Swan-Ganz), invasive arterial mean arterial blood pressure monitoring (MAP); cerebrospinal fluid (CSF) drainage, hyperventilation under $SvjO_2$ monitoring, osmotherapy with mannitol and mild hypothermia. In this series, four patients (44%) underwent barbiturate coma because of refractory high ICP values. The thiopenthal dose was adjusted according to bedside EEG registration (6 bursts/min). Neither corticosteroids nor anti-epileptic prophylaxis were included in the management protocol. Methods for ICP measurement included ventriculostomy in eight patients and additional infrared devices[1] in two patients. Wake-up procedure was indicated if no ICP increase above 15 mm Hg within 24 hours happened under normothermia and normoventilation, if the amount of CSF drainage was below 80 ml/24 hours, if $SvjO_2$ and the arteriovenous difference of lactate (AVDL) values were normal, and if no signs of intracranial hypertension were evident in the CT scan. ICP monitoring was discontinued as soon as neurological assessment was possible and no therapeutic interventions were necessary for 24 hours.

Continuous Cerebral Oxygenation Monitoring ($SjvO_2$ and $PbtO_2$)

To monitor $SvjO_2$, a No. 5.5 French fiberoptic oxygen double-lumen catheter[2] was inserted percutaneously into the internal jugular vein through a NO. 6 French introducer sheath according to the technique described in detail elsewhere [1, 15]. The position of the catheter was controlled radiologically with anteroposterior and lateral skull projections. The catheter for measurement $SjvO_2$ was placed on the right side unless a unique unilateral lesion in the left hemisphere was present, the left jugular circulation was demonstrated to be dominant, or if thrombosis or thrombi in the right jugular vein were documented in the colour-coded duplex sonography. On-line printouts of continuous $SjvO_2$ measurements were available. Every effort was made to start the monitoring as early after admission as possible. Pre-insertion and in vivo calibration were performed after insertion and every six hours thereafter. If $SjvO_2$ changed abruptly or if technical measurement problems were sus-

[1] Fiberoptic digital pressure monitor, Model 420, manufactured by Camino® Laboratories, San Diego, CA.

[2] Oximetrix® fiberoptic catheter (Model P575), Abbott Laboratories, North Chicago, IL.

pected, in vivo calibration was immediately repeated and documented. SaO_2 and haemoglobin concentration was kept constant throughout the ICU management. Normal $SjvO_2$ values were considered between 60% and 75%. "Desaturation episode" was defined as $SjvO_2 < 60\%$. $SjvO_2$ under 55% was considered a consequence of hypoperfusion after causes of artifactual measurements were ruled out. Simultaneous measurements of $SjvO_2$ and AVDL were done in order to identify episodes of luxury perfusion and ischaemia. AVDL values of 0.2 µmol/L or greater were interpreted as increased cerebral lactate production due to ischaemia/infarction episode. Bedside monitoring parameters included intracranial pressure (ICP), mean arterial pressure (MAP), cerebral perfusion pressure (CPP), and arterial (invasive) continuous monitoring of SaO_2, PaO_2, $PaCO_2$ and PH[3] Arterial blood was sampled for blood gas analyses[4] and lactate measurements in our laboratory[5].

$PbtO_2$ was monitored with a polarographic microcatheter (Clark-type) with a diameter of 0.5 mm and a 5-mm-long partial pressure oxygen-sensitive area[6]. The PO_2 sensitive area of the probe is about 7.9 sq. mm. Technical principles of this method were described previously [23]. The catheter was inserted into nonlesioned white matter (frontal lobe) using a special intracranial bolt with one, two, or three entries (for the temperature sensor and eventually the ICP monitoring infrared device). Before insertion of the $PbtO_2$ catheter, catheter-specific calibration values specified by the manufacturer were registered on the $PbtO_2$ device. Prior to insertion, the catheter was calibrated in a N_2-saturated fluid ($PO_2 = 0$) and in open air ($PO_2 = 154$ if room air pressure is 760 mm Hg). Mean zero drift was 0.8 ± 0.6. A temperature sensor was inserted in six out of nine patients (the temperature coefficient of the sensor sensitivity is 2.4–2.5%/°C). Since the temperature probe is integrated into the sensor, the $PbtO_2$ values were automatically corrected when the tissue temperature changed. In three patients, temperature values were given manually according to the blood temperature in the jugular bulb (measured in the tip of the catheter). Before starting a measurement, a 2-point calibration (one calibration at an oxygen pressure of 0 mm Hg and one at normal air oxygen pressure) was performed. $PbtO_2$ monitoring was started within the first 48 hours after brain injury (mean 18.5 hours) and was prolonged during a mean period of 198 hours. $PbtO_2$ values were stored at 30-second intervals. In three patients, the probe was changed because of technical problems, indication of craniotomy, or prolonged monitoring time (more than five days). $PbtO_2$ values of 15 mm Hg or greater were interpreted as "adequate" according to reported studies [23, 31, 46]. Values of 10 mm Hg or lower were considered a consequence of an ischaemic event. No infection, haematoma, or CSF-leakage could be noted as complications during $PbtO_2$ monitoring.

CO_2 and O_2 Reactivity Tests

After brain injury, the so-called $PbtO_2$–CO_2 and O_2 reactivity tests were performed on days 3, 5, and 7 according to the protocol described elsewhere (Fig. 1) [11, 45]. For testing CO_2, hyperventilation was induced by increasing the inspiratory minute volume (IMV) by 20% and subsequently decreasing the IMV to initial values.

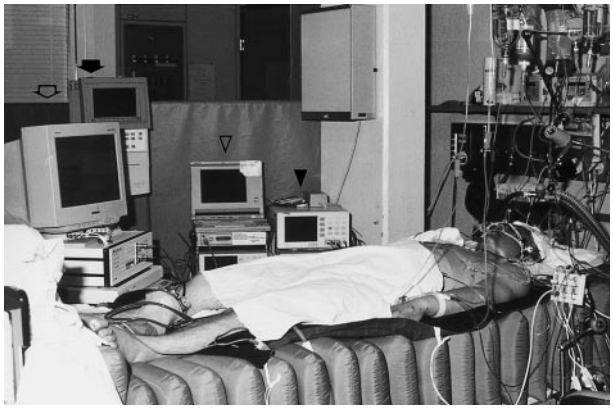

Fig. 1. Multimodal bedside monitoring during CO_2 and O_2 reactivity tests including on-line invasive blood gas analysis (black arrow), transcranial Doppler sonography (white arrow), $PbtO_2$ and $SjvO_2$ (white arrow head), and continuous haemodynamic monitoring (black arrowhead)

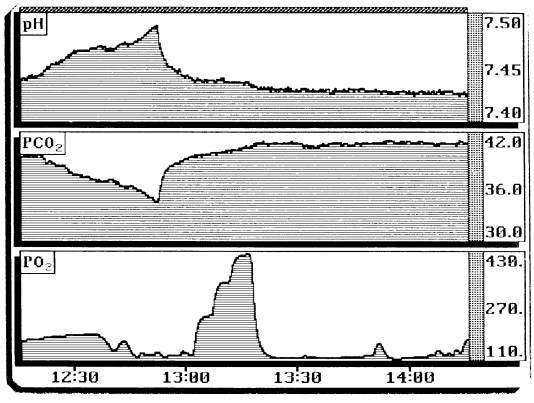

Fig. 2. On-line invasive monitoring of arterial pH, $PaCO_2$ and PaO_2 during CO_2 and O_2 reactivity tests. During the induced hyperventilation a $PaCO_2$ of 34 mm Hg with a simultaneous increase of pH value up to 7.5 was achieved. The CO_2 reactivity test was followed by the O_2 reactivity test (FiO_2 100%), causing an increase of PaO_2 up to 430 mm Hg

After the ventilator was returned to its original setting and baseline $PaCO_2$, $SvjO_2$, and $PbtO_2$ values had been reached, the $PbtO_2$–O_2 reactivity was tested. The fractional inspired oxygen (FiO_2) was increased in steps of 20% up to 100%, inducing an increase of PaO_2. Arterial and jugular-venous blood was sampled after each change of the ventilator setting. In addition, continuous invasive monitoring of PaO_2, $PaCO_2$ and pH was performed throughout the tests (Fig. 2). Flow velocity in both middle cerebral arteries (Vm MCA) was measured by TCD[7] using a 2 MHz probe fixed to the head during the performance of the tests. Vm MCA was measured from a depth of 54 to 57 mm ipsilateral to the hemisphere where $PbtO_2$ was monitored. $PbtO_2$–CO_2 reactivity was calculated as the percentage change in $PbtO_2$ divided by the actual change in $PaCO_2$. $PbtO_2$–O_2 reactivity was calculated as the percentage change in $PbtO_2$ divided by the actual change in PaO_2. The CO_2 reactivity test (induced hyperventilation) was prematurely ended if ICP increased to > 25 mm Hg, or $SvjO_2$ values became $\leq 60\%$. The so-called TCD–CO_2 vasoreactivity was calculated by dividing the percentage of Vm MCA

[3] Paratrend 7® Multiparameter Intravascular Sensor, Biomedical Sensors, Malvern, PA.

[4] ABL System 625®, Radiometer Medical A/S, Copenhagen, Denmark.

[5] TDx FLx®, Lactate Analyzer, Abbott Laboratories, Abbott Park, IL.

[6] Licox®, GMS, Kiel-Milkendorf, Germany.

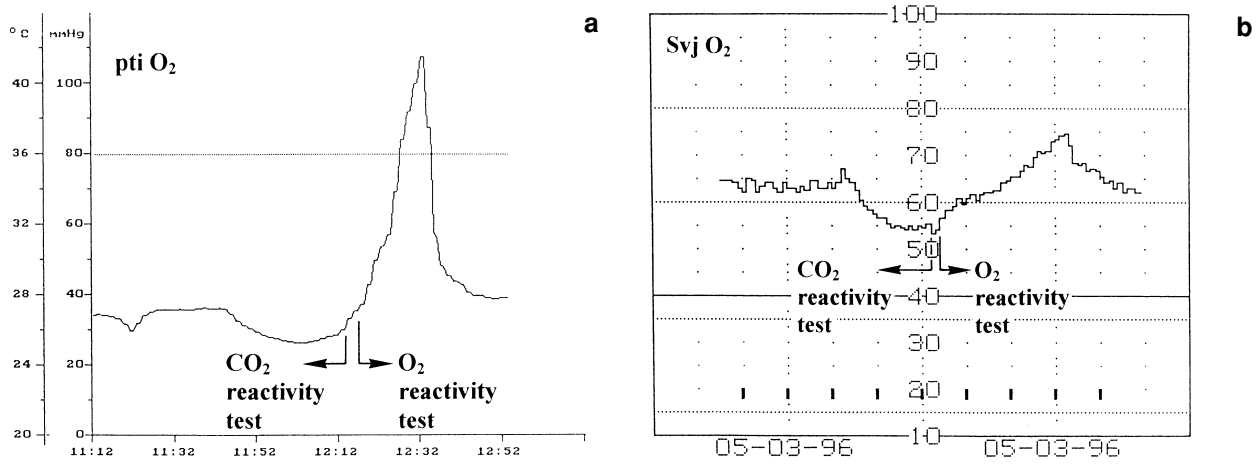

Fig. 3. Graph presentation of CO_2 reactivity test followed by O_2 reactivity test (FiO_2 increased to 100%) in Patient 5. During the first phase the patient was hyperventilated from $PaCO_2$ values of 39.7 mm Hg to 29.2 mm Hg showing a decrease in $PbtO_2$ from 36 mm Hg to 26 mm Hg (a). A simultaneous drop of the $SjvO_2$ until 55% is demonstrated (b). $PbtO_2$ value of 100 mm Hg was rapidly reached during O_2 reactivity test (FiO_2 100%). During the test the brain temperature was maintained stable at 36 °C (dotted line)

decrease by the absolute change of $PaCO_2$ [6, 10, 12, 25, 32, 37]. Patients with CO_2 vasoreactivity greater than 1% were considered to have an intact response to CO_2.

Statistical Methods

Statistical analysis was performed using STATISTICA® for Window 5.1 (StatSoft Inc., Tulsa, OK). Summary data are reported as the mean ± standard deviation. Student's t-test was used to compare group means. The significant level used to determine statistical significance was 0.05.

Illustrative Case

Case 5

This 26-year-old man suffered a severe head injury after falling from a scaffold. The patient was intubated and sedated at the scene of the accident then transferred by helicopter to the hospital. The patient had an initial GCS of 3 and multiple fractures in the lower extremities. No hypoxic episode was documented. On admission, he presented with a closed head injury and normal pupillary size and response. The initial CT revealed multiple contusions in the right temporal lobe and a thin right subdural haematoma causing no midline shift or hemisphere compression (*diffuse injury II*). No presence of postraumatic subarachnoid haemorrhage was observed. A right ventriculostomy was performed and a $PbtO_2$ probe was implanted in the right frontal lobe approximately two hours after trauma. The initial ICP and $PbtO_2$ values were 17 mm Hg and 24 mm Hg respectively. In the ICU, $SjvO_2$ and haemodynamic monitoring using a pulmonary artery catheter was initiated. The $PbtO_2$ monitoring showed initial low values during the first 24 hours after trauma (up to 10 mm Hg), with $SjvO_2$ between 65% and 70% and without signs of lactic acidosis (AVDL = < 0.2 µmol/l). The follow-up CT showed better limitation of the temporal lobe contusion, appearance of perifocal oedema and no volume expansion of the subdural haematoma. ICP values remained controllable (< 20 mm Hg) with adequate CPP throughout the hospitalization. Vasoreactivity tests were performed on days 3, 5 and 7 after injury. Figure 3 shows the CO_2 and O_2 performed on day 5. At this time the $PbtO_2$ ranged between 30 and 35 mm Hg under normocapnia and CPP values were over 70 mm Hg. During induced hyperventilation ($PaCO_2$ up to 29.2 mm Hg), a simultaneous drop of $PbtO_2$ and $SjvO_2$ was demonstrated (up to 26 mm Hg and 55% respectively). The $PbtO_2$–CO_2 reactivity was 3.77% and the TCD–CO_2 vasoreactivity 2.17%. During the O_2 reactivity test, after increasing FiO_2 to 100%, $PbtO_2$ values over 100 mm Hg were rapidly reached (Fig. 3). The patient could be discharged after 18 days and was moderately disabled according to the outcome evaluation three months after trauma.

Results

Correlation Between $PbtO_2$ and $SvjO_2$ During CO_2 Vasoreactivity Test

A total of 186 blood samples were obtained during twenty-one CO_2 reactivity tests in order to confirm continuous on-line monitoring values. Cerebral perfusion and oxygenation parameters during CO_2 vasoreactivity tests are shown in Table 2. Initial $PaCO_2$ values from 36 ± 3.7 mm Hg decreased significantly 14.6 ± 6 mm Hg ($p < 0.001$). The ICP and CPP did not change significantly. The mean baseline ICP value before starting hyperventilation was 18.9 ± 8.5 mm Hg (range, 5 to 31) and decreased to 17.8 ± 10.3 mm Hg (12.3 ± 23.8%, range, −22 to 62). ICP decreased in 18 tests and remained unchanged in 4 tests. The mean CPP at the beginning of hyperventilation was 81.1 ± 13.7 mm Hg (range, 70 to 102)

[7] Multi Dop X4®, DWL, Slipplingen, Germany.

Table 2. *Physiological Parameters During CO_2 Reactivity Tests*

	ICP (mm Hg)	CPP (mm Hg)	$PaCO_2$ (mm Hg)	Vm MCA (cm/s)	$SjvO_2$ (%)	$PbtO_2$ (mm Hg)
Normoventilation	18.9 ± 8.5	81.1 ± 13.7	36.0 ± 3.7	73 ± 25	74 ± 7	28.4 ± 6.9
Hyperventilation	17.8 ± 10.3	87.0 ± 13.8	31.54 ± 3.46	58 ± 20	54 ± 3.4	24 ± 8.2
Percentage of change	12.3 ± 23.8	7.2 ± 13.7	14.6 ± 6.0	18.5 ± 10.6	8.9 ± 7.3	16.4 ± 17.3
p-value*	NS	NS	< 0.001	< 0.001	< 0.001	< 0.001

ICP Baseline intracranial pressure; *CPP* cerebral perfusion pressure; *$PaCO_2$* arterial carbon dioxide; *Vm MCA* mean flow velocity in the middle cerebral artery ipsilateral to $PbtO_2$ monitoring (cm/s); *$SjvO_2$* jugular vein oxygen saturation; *$PbtO_2$* brain tissue oxygen pressure. *Statistical significance between normo- and hyperventilation values.

Table 3. *Cerebrovascular Response to CO_2 by Measuring Relative Changes of $PbtO_2$ ($PbtO_2$–CO_2 Reacitivity) and Flow Velocity in the Middle Cerebral Artery (TCD–CO_2 Reactivity) in Nine Patients* ($r = 0.22$, $p = 0.26$)

Test # (day)	$PbtO_2$–CO_2 reactivity (%/mm Hg)	TCD–CO_2 reactivity test (%/mm Hg)
1 (3)	−0.53	1.59
2 (5)	4.25	5.7
3 (7)	4.22	1.57
4 (3)	1.75	1.98
5 (5)	4.02	4.2
6 (7)	3.08	3.9
7 (3)	1.22	3.48
8 (5)	1.38	3.78
9 (3)	2.90	1.61
10 (5)	4.95	8.6
11 (7)	5.00	28.1
12 (3)	0.35	4.43
13 (5)	3.77	2.17
14 (3)	−2.40	3.73
15 (5)	0.12	3.38
16 (7)	1.60	2.34
17 (3)	1.11	1.68
18 (5)	8.67	3.08
19 (3)	2.99	0.88
20 (5)	8.20	3.93
21 (7)	7.20	4.01
mean ± SD	3 ± 2.8	4.5 ± 5.7

$PbtO_2$ Brain tissue oxygen pressure; *TCD* transcranial Doppler (flow velocities of the middle cerebral artery ipsilateral to $PbtO_2$ monitoring).

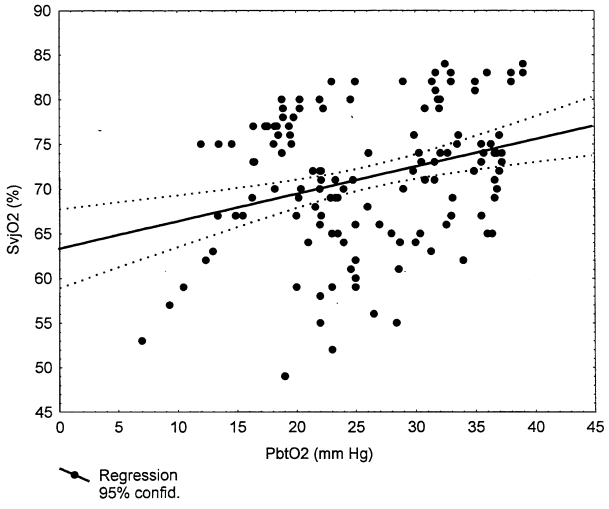

Fig. 4. Linear regression $SjvO_2$ against $PbtO_2$ during CO_2 reactivity test ($r = 0.33$, $p < 0.001$)

and increased to 87.0 ± 13.8 mm Hg (7.2 ± 13.7%). During induced hyperventilation, a significant decrease of PaO_2 (≥ 2.25 mm Hg) was observed in only three tests. Vm MCA, $SjvO_2$ and $PbtO_2$ decreased significantly during hyperventilation ($p < 0.001$). $SjvO_2$ values decreased from 74.3 ± 7.1% mm Hg to 31.54 ± 3.4 mm Hg and (8.9 ± 7.3%). Initial $PbtO_2$ values of 28 ± 6.9 mm Hg decreased to 24.05 ± 8.23 (16.4 ± 17.3%). Flow velocities of the MCA decreased from 73 ± 25.7 cm/s to 58.6 ± 20 cm/s (18.5 ± 10.6%).

The mean CO_2 vasoreactivity, measured by changes in $PbtO_2$, was 3 ± 2.8% (range, −2.4 to 8.6) (Table 3). Impaired or abolished vasoreactivity to CO_2 was observed in three patients on day 3 (Tests # 1, 12 and 14) and day 5 after trauma (Test # 15). One CO_2 reactivity test was prematurely ended because of critical $SjvO_2$ values (< 60%); nevertheless, $PbtO_2$ remained normal in this situation. During induced hyperventilation, critical values of $PbtO_2$ (< 15 mm Hg) were observed in four CO_2 reactivity tests. In two of these episodes, $PbtO_2$ normalized after restoration of normoventilation. A rapid and simultaneous decrease of $PbtO_2$ and $SjvO_2$ was observed in twelve out of twenty-one CO_2 reactivity tests (57%) (Illustrative Case); nevertheless, in nine tests (43%), a slow and non-simultaneous drop of both values was demonstrated. The correlation between and $PbtO_2$ and $SjvO_2$ during the CO_2 reactivity test was weak, and also varied from patient to patient ($r = 0.33$, $p < 0.001$). The mean CO_2 reactivity in day 5 was higher (4.42 ± 2.95) than in Day 3 (0.92 ± 1.79) and Day 7 (4.22 ± 2.1). Figure 4 shows the linear regression between $SjvO_2$ and $PbtO_2$ during CO_2 reactivity tests. Finally, no

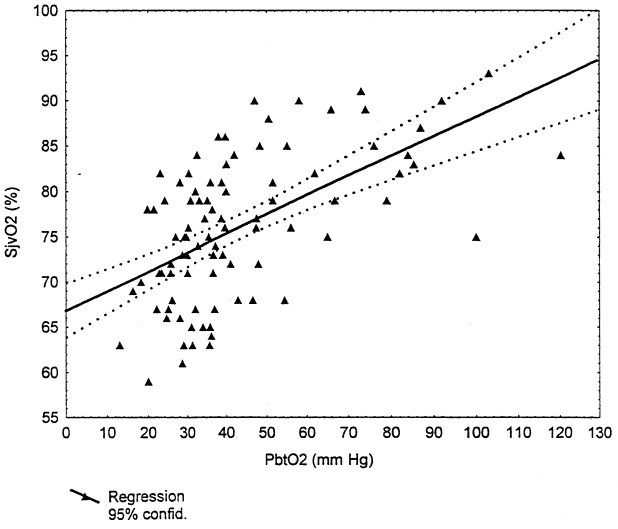

Fig. 5. Linear regression $SjvO_2$ against $PbtO_2$ during O_2 reactivity test ($r = 0.6$, $p < 0.0001$)

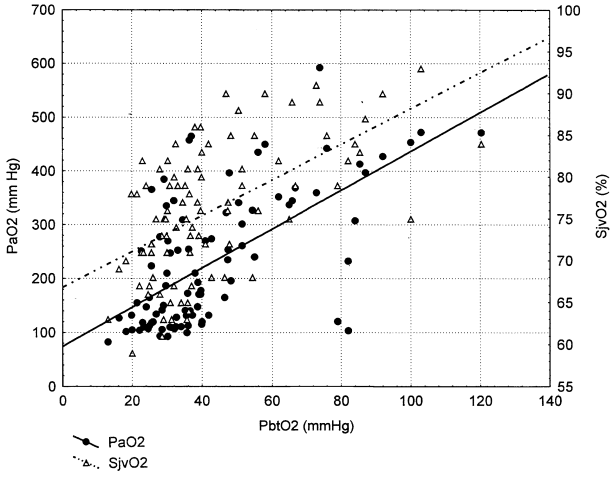

Fig. 6. Double Y scatterplot showing the correlation between PaO_2, $SjvO_2$ and $PbtO_2$ during O_2 reactivity test during stepwise FiO_2 increases up to 100% (correlation PaO_2–$PbtO_2$: $r = 0.63$, $p < 0.0001$; correlation $SjvO_2$–$PbtO_2$: $r = 0.6$, $p < 0.0001$)

statistically significant association between CO_2 vasoreactivity and outcome was shown.

Correlation Between $PbtO_2$ and $SvjO_2$ During O_2 Vasoreactivity Test

The mean O_2 reactivity was $0.6 \pm 0.5\%$ (range, 0.04 to 1.86). After increasing FiO_2 to 100%, PaO_2 increased from 220.88 ± 108.7 mm Hg to 483.93 ± 210.57 mm Hg. Simultaneously, the initial $PbtO_2$ values increased from 29.5 ± 14.5 mm Hg to 64.5 ± 28.13 mm Hg. The percentage increase of PaO_2 and $PbtO_2$ during hyperoxygenation was $249.31\% \pm 159$ and $316.23\% \pm 85.33$ respectively. In comparison with the CO_2 reactivity test, the correlation between $PbtO_2$ and $SjvO_2$ was better in the O_2 reactivity test ($r = 0.6$, $p < 0.001$). $PbtO_2$ values over 80 mm Hg were demonstrated in eight O_2 reactivity tests after increasing FiO_2 up to 100%. As shown in Fig. 5, the correlation analysis between $PbtO_2$ and $SvjO_2$ during O_2 reactivity tests revealed a correlation coefficient of 0.6 ($p < 0.001$). Changes of PaO_2 during hyperoxygenation correlated significantly with changes of $PbtO_2$ ($r = 0.63$, $p < 0.0001$). Figure 6 shows in a double-Y scatterplot graph the simultaneous increase of $SjvO_2$ and PaO_2 in relation to $PbtO_2$ during the O_2 reactivity tests. Other parameters like CPP, ICP and $PaCO_2$ did not statistically correlate with $PbtO_2$ changes. In our series, we did not observe a statistically significant association between O_2 reactivity and outcome after three months.

$PbtO_2$ and Flow Velocity Responses to CO_2

As shown in Table 2, the baseline Vm MCA ipsilateral to the $PbtO_2$ probe was 73 ± 7.1 cm/sec (range, 36 to 120). During hyperventilation, this value decreased by $8.9 \pm 7.3\%$. Three patients presented on three different days Vm MCA >100 cm/sec (Test # 8, 11 and 21); nevertheless, the responses to CO_2 changes in terms of $PbtO_2$ and Vm MCA changes remained adequate ($PbtO_2$–CO_2 reactivity = 1.38%, 5% and 7.2% respectively; and TCD–CO_2 reactivity = 3.78,28,0 and 4.0% respectively). Table 3 shows the vasoreactivity values by means of $PbtO_2$ and Vm MCA changes. The mean vasoreactivity measured by means of changes in Vm MCA was higher ($4.5 \pm 5.7\%$/mm Hg) than that obtained measuring the $PbtO_2$ changes ($3 \pm 2.8\%$ mm Hg). A low correlation between Vm MCA and $PbtO_2$–CO_2 reactivity was demonstrated ($r = 0.22$, $p = 0.26$).

Discussion

Disturbed cerebral autoregulation may result in an abnormal balance between CBF, blood volume, and the metabolic requirement of the cerebral tissues. Impaired or persistent loss of autoregulation on the first two days after injury seems to be correlated with an unfavourable outcome [10, 22]. Lam *et al.* studied 26 brain-injured patients with laser Doppler flowmetry (LDF) and analysed the relationship between the change in CPP and LDF as an index of cerebral au-

toregulation [22]. The authors found three patterns of autoregulation after brain injury: intact, transient loss, and persistent loss. In 11 patients who had persistent loss of autoregulation, nine died and two were severely disabled. Czosnyka *et al.* studied autoregulatory mechanisms in 82 patients, correlating flow velocities of the MCA (mean and during cardiac systole) and CPP [10]. They demonstrated that a positive correlation and impaired autoregulation between Vm MCA and CPP on the first two days after injury was seen in patients with unfavourable outcomes. On average, patients with favourable and unfavourable outcomes had intact autoregulation during days 3 to 5 after injury. Thereafter, patients with unfavourable outcomes showed a significant loss of autoregulation during days 6 to 8. The findings of these and other studies [5, 26, 36], related with different pathological mechanisms which may result in postraumatic cerebral ischaemia, can be explained by the different cerebral haemodynamic phases following severe head injury that were nicely described recently by Martin *et al.* [29]. The authors studied 125 patients with severe head injuries and concluded that three phases with different perfusion, oxygenation, and CBF patterns can be chronologically characterized. The Phase I, or *hypoperfusion phase*, occurs on the day of injury (Day 0) and is defined by a low CBF, normal Vm MCA, and normal $AVDO_2$. During Phase II, or *hyperaemia phase*, (Days 1–3) CBF increases, $AVDO_2$ falls, and Vm MCA rises. In Phase III or *vasospasm phase* (Days 4–15) there is a fall in CBF and further increases in Vm MCA. These phases of cerebral haemodynamic disturbance coincide with the chronological development of impaired or lost autoregulation described elesewhere [10, 17, 22]. Postraumatic vasospasm occurs in 25–40% of brain-injured patients, and may contribute to the pathogenesis of cerebral autoregulation disturbance [7, 27, 28, 29, 34]. The time course of this complication resembles that of vasospasm associated with aneurysmal SAH, with onset occurring two or more days after injury. Although some patients may develop mild-to-moderate vasospasm during *Phase II* after injury, almost all of the severe cases are seen during *Phase III*. Martin *et al.* reported that CBF is low in almost 50% of patients who have TCD findings which suggest severe vasospasm (15%) [29]. In these cases, the cerebral metabolic rate of oxygen ($CMRO_2$) remains normal, suggesting a coupling of CBF and cerebral metabolic requirements. In the remaining 50% of patients in whom CBF does not remain coupled to the metabolic requirement, a disturbance of autoregulation can also be expected. This situation can be associated with postraumatic SAH [28, 34], but also occurs in the presence of normal CT findings. Jünger at al. determined the cerebral autoregulation following minor head injury and demonstrated poorly functioning or absent cerebral autoregulation in 28% of the patients [17]. In our series, two patients (Patient 3 and 4) who presented with postraumatic SAH developed TCD-vasospasm signs (Vm MCA >110 cm/s) on days 5 and 7 (*Phase III*). Only in patient 3 was autoregulation impaired. Concerning the cerebrovascular response to CO_2 in the same two cases, an intact vasoreactivity measured by TCD was found; nevertheless, low vasoreactivity values during hyperventilation were demonstrated by means of $PbtO_2$ measurements ($PbtO_2$–CO_2 reactivity $= 1.22$ and 1.38). In this case, a large discrepancy between the two methods was observed (Table 3).

Since the introduction of TCD to clinical practice in the mid 1980's, this method has been accepted as the gold standard technique for measuring the cerebrovascular autoregulation and vasoreactivity to CO_2 [25]. From different published studies, the mean percentage change of CBF per mm Hg $PaCO_2$ in normal, awake, hyperventilated volunteers ranged from 1.8 to 2.3 [18, 19]. The lower percentile of cerebrovascular response to CO_2 reported by McHenry *et al.* [24] was 0.8 and 1.2 according to Kety *et al.* [19]. Newell *et al.* reported a Vm MCA response to hyperventilation varying from 0 to 4.6% change/mm Hg [35]. In severe head injury, the clinical impact of hypocapnic CO_2 reactivity seems to be considerable, since the loss of cerebral CO_2 vasoreactivity is associated with poor outcome [40]. In this study, the mean CO_2 reactivity measured by TCD was higher than vasoreactivity by means of $PbtO_2$ changes. In four patients, vasoreactivity by means of focal oxygenation parameters like $PbtO_2$ was lower or even abolished in four patients in comparison with values measured by TCD. These findings indicate that "real" responses to CO_2 in terms of cerebral oxygenation parameters are much lower than those measured by TCD, and correlate weakly to each other. Since autoregulation can be influenced by hyperventilation [35], vasospasm [28], or just change throughout the different cerebral haemodynamic phases following severe head injury [29], the main goal during intensive care is the monitoring of cerebral oxygenation patterns in order to prevent secondary brain injury. In the last decade, the introduction of $SjvO_2$ has

contributed to the understanding of cerebral oxygenation during intensive management of head-injured patients [8, 9, 38, 40]. $SjvO_2$, in combination with $AVDO_2$ and AVDL measurements, enable the identification of global luxury perfusion, normal coupling of global cerebral flow with global cerebral metabolism, global cerebral hypoperfusion and global cerebral ischaemia [38]. Technical limitations of $SjvO_2$ and the impossibility of detecting focal ischaemia encouraged other alternatives for evaluating focal cerebral oxygenation patterns. $PbtO_2$ monitoring is an alternative method for evaluating changes in cerebral oxygenation caused by changes in oxygen offer, as well as by changes in cerebral blood flow, and oxygen demand of cerebral tissue [23]. Measurement of $PbtO_2$ seems to be a safe, reliable, and stable method, with less technical shortcomings than $SjvO_2$, and more suitable for long-term monitoring. Recently, Kiening *et al.* reported a good correlation between $SjvO_2$ and $PbtO_2$ [20]. The authors found a hypoxic threshold of 8.5 mm Hg $PbtO_2$ (range 3–12 mm Hg), correlating with a $SjvO_2$ of 50%. These findings were based on a monitoring duration of nine days (range, 5–12) without hyperventilation manoeuvers. Rapid changes of CO_2 during intensive care management were not described in the study. These interesting results encouraged us to correlate both methods during abrupt changes of CO_2 in terms of vasoreactivity. We observed that $SjvO_2$ and $PbtO_2$ correlate weakly during hyperventilation in spite of the fact that significant differences in $PaCO_2$ were demonstrated. The reason for that was likely to be a rapid and simultaneous decrease of $PbtO_2$ and $SjvO_2$, as shown in the illustrative case (Figure 3), and was observed in only 57% of the CO_2 reactivity tests. According to our results, brain $PbtO_2$ measurements during rapid induced hyperventilation dropped more quickly constantly than $SjvO_2$ did. These findings suggest that so-called desaturation episodes during hyperventilation could be detected earlier and more reliably with $PbtO_2$ monitoring. In addition, the relationship of $PbtO_2$ to the outcome after severe head injury has been recently reported by Valadka *et al.* [44]. The authors suggest that the likelihood of death increases with increasing duration of time at or below a $PbtO_2$ of 15 torr, or with the occurrence of any $PbtO_2$ values ≤ 6 torr. With respect to the effects of hyperoxygenation, we found a better correlation between $SjvO_2$ and $PbtO_2$ during the so-called O_2 reactivity test than during induced hyperventilation. As reported in other studies, we found that changes in PaO_2 seem to correlate significantly with changes in brain $PbtO_2$ [45, 46]. In our study we observed that after increasing PaO_2 from 220.88 ± 108.7 mm Hg to 483.93 ± 210.57 mm Hg, there was a statistically significant increase in $PbtO_2$ values from 29.5 ± 14.5 mm Hg to 64.5 ± 28.13 mm Hg ($p < 0.001$). These observations reinforce the need to specify whether to increase CPP or FiO_2 is the best method for improving $PbtO_2$, taking into account the already reported consequences of this parameter in the outcome [44]. $PbtO_2$ seems to increase rapidly to values over 80 mm Hg during hyperoxygenation (FiO_2 100%). According to our observations, $PbtO_2$ and $SjvO_2$ increase simultaneously with the increase of PaO_2 during hyperoxygenation as shown in Figure 6. The O_2 reactivity values by means of $PbtO_2$ observed in our study seem to be roughly the same as those observed by other authors [45]; nevertheless, we did not find a statistically significant correlation with the outcome.

Finally, we think that the monitoring of $PbtO_2$ is a reliable method for identifying in an easy and rapid way patients who will tolerate rapid hyperventilation during episodes of intracranial hypertension. This method seems to be more accurate than TCD or detection of $SjvO_2$ desaturation episodes during evaluation of cerebrovascular response to CO_2; nevertheless, for long-term monitoring of hyperventilated patients, this technique has to be combined with global measure of cerebral metabolism ($AVDO_2$ and AVDL).

Conclusions

Correlation between $SjvO_2$ and $PbtO_2$ during CO_2 reactivity test is low, despite the fact that significant differences between normo- and hyperventilation values were observed. In comparison to $SjvO_2$, monitoring of $PbtO_2$ might more accurately detect focal ischaemic events during rapidly induced hyperventilation in severely head-injured patients.

The CO_2 vasoreactivity by means of changes in Vm MCA seems to be higher in comparison to changes in brain $PbtO_2$. These observations lead to the hypothesis that the vasoreactivity measured by transcranial Doppler tends to overestimate the cerebrovascular response to CO_2.

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Comments

This is an extremely interesting clinical study, as monitoring of $PbtO_2$ recently introduced into clinical practice has become a promising modality for improving the management of severely head-injured patients.

Since the introduction of TCD into clinical practice it has been widely employed as the the most valuable technique for monitoring cerebrovascular autoregulation and vasoreactivity to CO_2 to be able to achieve the main goal of intensive care: the prevention of secondary complications by the maintenaince of appropriate tissue oxygenation. However, this study indicated that the real response to CO_2 in terms of cerebral oxygenation parameters are much lower than those measured by TCD and correlate weakly to each other. It is suggested that vasoreactivity as measured by TCD may overestimate the cerebrovascular response to CO_2.

$SjvO_2$ in combination with $AVDO_2$ and AVDL measurements enable the identification of luxury perfusion, normal coupling of flow and metabolism, hypoperfusion and ischaemia on a global level. $PbtO_2$ monitoring is a new alternative method for the evaluation of oxygen offer and demand, and level of CBF on a regional basis. This study found that $SjvO_2$ and $PbtO_2$ correlate weakly during rapid changes of CO_2 in spite of the fact that significant differences in $PaCO_2$ were demonstrated. These findings suggest that $PbtO_2$ measurements are more reliable than $SjvO_2$ studies. The study has also helped to answer the question wether increase of CPP or that of FiO_2 is the best method for improving cerebral oxygenation.

Although the number of patients is relatively small, I think, it is a very valuable and important paper. The working hypothesis is sound, and the results are moderately interpreted. The conclusions transmit important message for those involved in the management of severely head-injured patients. I strongly encourage the authors to continue with the study.

T. Dóczi

The authors examine the correlation between jugular bulb oxygen saturation, partial pressure of brain tissue oxygen and reactivity tests by transcranial Doppler in severely head injured patients. They found a low correlation between jugular bulb oxygen saturation and partial pressure of brain O_2. Further, the CO_2 vasoreactivity measured by transcranial Doppler in the MCA was higher in comparison to changes of brain tissue oxygen pressure. The study is methodologically very well done and the conclusions drawn from the results of the present investigations are correct. The only weak point is that partial pressure of brain tissue oxygen measurements represents a focal parameter of brain metabolism, whereas transcranial Doppler measurements of the MCA is a regional parameter and jugular bulb oxygen saturation is a global parameter of CBF and cerebral metabolism. There may be regional differences in disturbed autoregulation after severe head injury. Therefore, the three parameters are not strictly comparable.

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