Acta Neurochir (Wien) (1996) 138: 1409–1415

Acta Neurochirurgica

Printed in Austria

Analysis of Abnormal Jugular Bulb Oxygen Saturation Data in Patients with Severe Head Injury

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Summary

Jugular bulb oximetry provides the first bedside, continuously available information on cerebral perfusion adequacy. An extensive analysis was made of all jugular bulb oxygen saturation (SjO_2) data obtained in 50 patients suffering from severe head injury. A total of 176 periods (more than 30 minutes) with reliable, abnormal SjO_2-values was observed, with 62 desaturation periods (SjO_2 < 55%) and 114 high SjO_2-periods (SjO_2 > 80%). Jugular desaturation periods were predominantly observed in the first 2 days of monitoring and seemed the most closely correlated to lowered cerebral perfusion pressure and lowered arterial carbon dioxide tension. The high SjO_2-values were more equally distributed over the first 5 days of monitoring and seemed mostly correlated to increased arterial carbon dioxide tension. Highlights of the general management of severely head injured patients is discussed, focussing attention on the importance of cerebral perfusion pressure and normoventilation.

Keywords: Jugular bulb venous oxygen saturation; severe head injury; cerebral perfusion pressure; hyperventilation.

Introduction

Jugular bulb oximetry provides the first bedside, continuously available information on cerebral perfusion adequacy. Most of the homeostatic mechanisms regulating cerebral perfusion are largely disturbed in patients suffering from severe head injury. This explains the demand for bedside cerebral monitoring modalities, estimating the adequacy of cerebral perfusion [3, 9, 17]. Moreover, guaranteeing an adequate cerebral perfusion is extremely important in the management of severe head injury to prevent the occurrence of secondary brain ischaemia [3, 17]. The nowadays available parameters, intracranial pressure (ICP) and cerebral perfusion pressure (CPP), are unfortunately only "indirect" parameters of cerebral perfusion adequacy.

Jugular bulb oximetry consists of the invasive fiberoptic measurement of the venous oxygen saturation in the jugular bulb [6, 7, 26]. Normal values of jugular bulb oxygen saturation (SjO_2) fluctuate between 55 and 75%. Increased SjO_2-values (>75%) reveal that oxygen supply to the brain (CBF) is exceeding oxygen demand ($CMRO_2$) of the brain (SjO_2 >75% reflects that CBF > $CMRO_2$). In contrast, decreased SjO_2-values (< 55%) indicate that cerebral blood flow is insufficient for the metabolic demands of the brain (SjO_2 < 55% reflects that CBF < $CMRO_2$).

For this report, we retrospectively analysed all obtained SjO_2-data in a homogenous subgroup of severely brain injured patients. Our objectives were to analyse the incidence of abnormal (lower than 55% or higher than 80%) SjO_2-values and to analyse the correlation of abnormal SjO_2-data with all other monitored parameters.

Patients and Method

Over a 24 months period (August 1992 to July 1994), 50 patients suffering from severe head injury were selected. Severe head injury was defined as a Glasgow Coma Score less than 8 on admission to our hospital. In all patients jugular bulb oximetry was started within the first 24 hours after trauma. All patients were older than 18 and younger than 70 years. All patients had an intracranial pressure monitoring inserted by means of an intraventricular catheter.

Primary neurotrauma care consisted of sedation (opioids and benzodiazepines), 30° head elevation, normoventilation ($PaCO_2$ between 32 and 40 mmHg), maintenance of cerebral perfusion pressure above 70 mmHg (with fluid loading and norepinephrine if necessary) and neurosurgical intervention whenever judged necessary. Intracranial hypertension was defined as an ICP exceeding 20 mmHg for more than 5 minutes. Intracranial hypertension was

first treated with cerebrospinal fluid drainage. If drainage alone seemed insufficient, mannitol was administered and eventually hyperventilation was considered. If under treatment refractory intracranial hypertension persisted barbiturates were administered as a last resort.

Jugular bulb oximetry was started on admission to the ICU. After puncture of the internal jugular vein, a retrograde Seldinger catheterization of the jugular bulb was performed, inserting a 5Fr introducer sheath. In this sheath, a 4Fr fiberoptic, double-lumen catheter (Opticath-Oximetrix, Abbott Laboratories) was inserted for about the estimated length (about 15–17 cm) to reach the jugular bulb. Correct positioning of the catheter tip was confirmed by x-ray (lateral view of the cervical spine) and it was accepted that a correctly located jugular bulb catheter should have its tip projecting above the second cervical vertebra [6]. Once the oximetry catheter was inserted, jugular bulb oxygen saturation values were displayed on the monitor screen.

All SjO_2-data were analysed for periods with abnormal values. A period with abnormal SjO_2-values was defined as more than 30 minutes with SjO_2-values below 55% or above 80%. Only data guaranteeing accurate positioning of the catheter tip, revealed by a perfect light intensity signal, and guaranteeing an acceptable reliability (less than 5% oxygen saturation difference between oximetry and co-oximetry control) were analysed. Routine co-oximetry control was performed every 6 hours and whenever a difference of more than 5% oxygen saturation between oximetry and co-oximetry was noted, in vivo recalibration of the fiberoptic monitoring system was routinely performed.

Whenever an abnormal SjO_2-value was observed, a search for the possible causes (analysing all cerebral and systemic parameters) was initiated and treatment was instituted in order to restore the jugular bulb oxygen saturation into normal range again.

In order to detect already established cerebral ischaemia, jugular as well as arterial lactate levels were intermittently (every 6 hours) determined [21]. In about 3% of all determinations, jugular-arterial differences in lactate were considered abnormal (Lactate Oxygen Index of 0.08 or more) [21], suggesting the presence of cerebral ischaemia. Abnormal jugular saturations occurring during these periods were excluded for further analysis, as abnormal jugular saturations seem of very little value in the presence of cerebral ischaemia [5].

Results

In these 50 patients, jugular bulb oximetry was performed for a mean duration of 7,5 days/patient (representing a total of 9096 hours for the whole study population). A total of 176 periods with reliable, abnormal SjO_2-values was observed, representing 1180 hours. This means that 13% of all monitored time revealed reliable, abnormal SjO_2-information.

A total of 62 jugular desaturation periods ($SjO_2 <$ 55%) was noted. These periods were observed in 31 patients (=62% of total study population revealed at least 1 jugular desaturation period). These 62 periods presented a mean duration of 2.4 hours/period. Most (60%) of the desaturation periods occurred in the first 2 days of monitoring or within the first 72 hours after trauma. Only very few desaturation periods (17%) occurred after 5 days monitoring. The distribution in time of all these periods is given in Table 1.

Table 1. *Time Distribution of All Periods wiht too Low Jugular Bulb Oxygen Saturation Values*

Day	Periods: total 62	%
Day 1	21 periods	34%
Day 2	16 periods	26%
Day 3	4 periods	6.5%
Day 4	7 periods	11%
Day 5	3 periods	5%
Day 6	3 periods	5%
Day 7	4 periods	6.5%
Day 8	–	–
Day 9	1 period	1.5%
Day 10	–	–
>Day 10	Day 11: 2 periods	3%
	Day 12: 1 period	1.5%

Jugular desaturation periods were further divided in 3 grades. Minor desaturations were defined as SjO_2-values between 50 and 54%, moderate desaturations as SjO_2-values between 45 and 49%, while severe desaturations were defined as SjO_2-values below 45%. We observed 30 minor, 20 moderate and 12 severe desaturation periods.

In 32 of these 62 desaturation periods, CPP was below 70 mmHg and was considered a possible cause of jugular desaturation. In 16 of these 32 periods, there was the combination of a low CPP together with an abnormal low $PaCO_2$-level (less than 32 mmHg). In 30 periods an abnormal low $PaCO_2$-level was found. In 16 of these periods there was the already above mentioned combination of a low CPP together with a low $PaCO_2$, whereas in 14 periods a low $PaCO_2$ was the only abnormality. In 4 of the 62 periods intracranial pressure was the only abnormality, accompanied by a normal CPP and a normal $PaCO_2$ level. And finally, in 12 periods no abnormality was observed neither in CPP, ICP or in $PaCO_2$. The possible causes found in these 12 periods were: too abrupt weaning of sedation in 6 periods, severe hypovolaemia in 4 periods, epileptic activity in 1 period and finally in 1 period no apparent cause could be found and jugular saturation returned to normal after 1 hour desaturation. Further information on the respective cerebral and systemic causes for the 3 grades of desaturation are given in Table 2.

For all 61 periods with an apparent cerebral or sys-

Table 2. *Cerebral and Systemic Factors for the Periods with too Low Jugular Bulb Saturation Values*

SjO_2-value	50–54%	45–49%	<45%
CPP < 70 mmHg	8 periods	5 periods	3 periods
nl $PaCO_2$ (32–40 mmHg)			
ICP < 20 mmHg	5 periods	3 periods	–
ICP > 20 mmHg	3 periods	2 periods	3 periods
$PaCO_2$ < 32 mmHg	7 periods	5 periods	2 periods
nl CPP (70–90 mmHg)			
ICP < 20 mmHg	5 periods	4 periods	2 periods
ICP > 20 mmHg	2 periods	1 period	–
CPP < 70 mmHg	7 periods	6 periods	3 periods
$PaCO_2$ < 32 mmHg			
ICP < 20 mmHg	2 periods	3 periods	2 periods
ICP > 20 mmHg	5 periods	3 periods	1 period
ICP > 20 mmHg	1 period	1 period	2 periods
nl CPP (70–90 mmHg)			
nl CPP (70–90 mmHg)	7 periods	3 periods	2 periods
nl $PaCO_2$ (32–40 mmHg)			
nl ICP (< 20 mmHg)			
Weaning off sedation	4 periods	2 periods	–
Hypovolaemia	3 periods	1 period	–
Epileptic attack	–	–	1 period
No apparent cause			1 period

temic causal factor, normal jugular saturation could be restored by correction of the causal factor.

A total of 114 high SjO_2-periods (SjO_2 >80%) was noted. These 114 periods were observed in 49 patients, implying that only one patient showed no high SjO_2-period. If we exclude the periods of high SjO_2-values with confirmed brain death (=8 periods for a total of 240 hours) abnormal high SjO_2-values which could be returned to normal lasted a mean of 4.8 hours/period. In contrast to the jugular desaturation periods, only a minority of the high SjO_2-values occurred in the first 2 days of monitoring (only 30% vs 60% of the jugular desaturation periods occurring within 72 hours after trauma) (see Table 3).

Table 3. *Time Distribution of All Periods with too High Jugular Bulb Oxygen Saturation Values*

Day	Periods: total 114	%
Day 1	13 periods	11.5%
Day 2	21 periods	18.5%
Day 3	15 periods	13%
Day 4	16 periods	14%
Day 5	14 periods	12%
Day 6	8 periods	7%
Day 7	8 periods	7%
Day 8	8 periods	7%
Day 9	3 periods	2.5%
Day 10	5 periods	4.5%
>Day 10	Day 12: 1 period	1%
	Day 14: 1 period	1%
	Day 16: 1 period	1%

In 55 of the 114 periods (=48.5%) with SjO_2-values greater than 80%, arterial carbon dioxide tension was above 40 mmHg (see Table 4). In 51 periods, a high $PaCO_2$ was the main cause of high SjO_2-values and in 4 periods there was the combination of a high $PaCO_2$-value together with a high CPP (above 90 mmHg). In 12 periods (=10.5%) high SjO_2-values were primarily associated with an increased cerebral perfusion pressure (above 90 mmHg). In 18 periods (=16%), there was a primary increase in intracranial pressure (without increase in $PaCO_2$ or in CPP). All these too high SjO_2-values could be returned to nor-

Table 4. *Cerebral and Systemic Factors for the Periods with too High Jugular Bulb Saturation Values*

SjO_2-value	>80%
$PaCO_2 > 40$ mmHg	51 periods
nl CPP (70–90 mmHg)	(45%)
ICP < 20 mmHg	25 periods
ICP > 20 mmHg	26 periods
CPP > 90 mmHg	12 periods
nl $PaCO_2$ (32–40 mmHg)	(10.5%)
ICP < 20 mmHg	9 periods
ICP > 20 mmHg	3 periods
CPP > 70 mmHg	4 periods
$PaCO_2 > 32$ mmHg	(3.5%)
ICP < 20 mmHg	3 periods
ICP > 20 mmHg	1 period
ICP > 20 mmHg	18 periods
nl CPP (70–90 mmHg)	(16%)
Brain death	8 periods
nl CPP (70–90 mmHg)	21 periods
nl $PaCO_2$ (32–40 mmHg)	(18%)
nl ICP (< 20 mmHg)	

mal by correction of the related cerebral or systemic causal factor.

Finally, in 29 periods (=25%) all other cerebral data were within normal range and no apparent cause could be detected, except for the 8 periods with the confirmed brain death diagnosis.

Discussion

Jugular bulb oximetry has been promoted as a new bedside cerebral monitoring technique estimating the adequacy of cerebral perfusion. Although the usefulness of its information has been largely underscored [12, 26], there are several major shortcomings that might limit its use [10]. Jugular bulb oximetry provides a unilateral and global information, referring to the balance of oxygen supply to demand of the brain. Next to these methodological shortcomings, there are technical drawbacks related to the use of a fiberoptic catheter retrogradely inserted in a low flow venous system. These technical problems might cause a reduced reliability and necessitate an increased vigilance as to the maintenance of normal fiberoptic signals with a routine cross check of jugular bulb saturation by co-oximetry control. For this analysis, we only analysed abnormal SjO_2-values with a perfect reliability, confirmed as well by normal light intensity signals as by routine co-oximetry control, in order to reduce possible technical artifacts. Sheinberg *et al.* [26] already reported that most of the erroneous saturation readings induced by vessel wall artifacts could be detected by light intensity alarm messages on the monitoring screen.

A major shortcoming of this report is that we did not dispose of a continuous data acquisitioning system, and that we had to rely on the medical charts with only 30 minutes controls. Therefore, we could only analyse periods of abnormal saturation lasting more than 30 minutes, excluding thereby all short lasting periods of abnormal jugular bulb saturation that might have been detected by a continuous data acquisitioning system [15]. Such a continuous monitoring system might also permit a much better multimodality analysis, revealing the exact relationship in time between several parameters, such as e.g. SjO_2, CPP and ICP.

Low Jugular Bulb Oxygen Saturation Data

We defined 55% as the lower margin of normal SjO_2-values, but divided all desaturation data in 3 grades, as already proposed by Dearden and others [7]. In the literature, true jugular desaturations are mostly defined as SjO_2-values below 50%. However, no clear cut threshold has been set for this lower margin and some data revealed the presence of cerebral hypoperfusion with SjO_2-values below 54% and withheld the lower margin at 55% [7, 8]. Because of these uncertainties and as SjO_2-values below 50% are independently associated with a worse neurological outcome, we wanted to enlarge our safety margins for detecting inadequate cerebral perfusion by maintaining the lower margin at 55%.

All our observed jugular desaturations were early events, with 60% of all desaturations occurring within 72 hours of trauma and even 30% occurring on ICU admission at a mean of 4.8 hours after trauma. Lewis *et al.* [15] reporting on cerebral venous desaturation ($SjO_2 < 55\%$) after severe head injury found the same predominance of jugular desaturations early in the posttraumatic course. Bouma *et al.* [1], performing very early invasive CBF measurements confirmed a surprisingly high incidence of very early cerebral hypoperfusion, underscoring the hypothesis that posttraumatic cerebral hypoperfusion is usually an early event. The high incidence of very early jugular bulb

desaturations might have important implications for the emergency management of severely head injured patients. Inadequate cerebral perfusion occurring in the first hours after trauma might be seen as a primary post-traumatic event, possibly secondarily potentiated by systemic factors known to reduce cerebral perfusion (too low a mean arterial blood pressure, too low a CO_2-tension). It might be extremely important to prevent and/or to treat these systemic influences.

The high incidence of early jugular desaturations illustrates moreover the importance of rapid and adequate haemodynamic and respiratory monitoring and management, even in patients suffering from isolated brain injury. Early invasive arterial pressure monitoring with continuous display of the mean arterial pressure seems mandatory, as is early respiratory monitoring with arterial blood gas analysis ($PaCO_2$-monitoring) and supplementary end-tidal CO_2-monitoring.

As in previous reports, jugular bulb desaturations were most closely correlated to a reduced CPP (due to a reduced MAP or increased ICP) or to a decreased $PaCO_2$, due to hyperventilation, or to a combination of both parameters. Schneider *et al.* [27] found 62% of their desaturations (SjO_2 < 50%) correlated to hypocapnia ($PaCO_2$ < 32 mmHg) and 38% to a CPP < 60 mmHg. Lewis *et al.* [15] found 45% of their desaturations (SjO_2 < 55%) correlated to hypocapnia (end-tital CO_2 < 30 mmHg), 31% due to a CPP <60 mmHg and 24% to a combination of both. Moreover, when comparing our 3 different desaturation grades, we found exactly the same distribution of both causal factors among the 3 different grades (see Table 2).

The need and the importance of maintaining an adequate cerebral perfusion pressure, with a threshold that should approximate 70 mmHg has been well established [2, 3, 23–25]. Jugular oxygen saturation monitoring in this study confirms once again the importance of CPP. In 17 of the 32 periods with SjO_2 < 55% due to CPP insufficiency, increases in ICP primarily caused the CPP insufficiency, whereas in the other 15 periods, ICP was within the normal range and a too low mean arterial pressure (MAP) was the primary cause. This is an important observation for the therapeutic implications as treatment should indeed be aimed both at reducing ICP and/or increasing MAP and reduction of the ICP should not interfer with systemic blood pressure.

The second most important cause of jugular desaturation was arterial hypocapnia, as in 49% of the desaturation periods a too low $PaCO_2$ (< 32 mmHg) was observed. As for the desaturations caused by CPP insufficiency, the major part of these desaturation periods (12 periods = 40%) was noticed on admission to the ICU. This can be explained by the use of so-called "blind hyperventilation", a practice which is still employed during transfer of severely head injured patients. Blind hyperventilation means application of hyperventilation without any knowledge of ICP, CPP (or SjO_2-value) and may be started at the scene of the accident to prevent or treat presumed intracranial hypertension. From our observations, we can only discourage the use of this "blind" hyperventilation. Hyperventilation should not be instituted as a prophylactic measure [18], but only once intracranial hypertension is well documented by ICP-monitoring and once information on cerebral perfusion is available (by e.g., SjO_2-monitoring). Only in cases of rapidly progressing intracranial catastrophes such as enlarging pupils, anisocoria or bilateral mydriasis could one still consider the use of blind hyperventilation with a consequent urgent need for neurosurgical intervention and cerebral monitoring (ICP- and SjO_2-monitoring).

Next to these accidental hypocapnic periods, we observed 5 periods of jugular desaturation due to therapeutic hypocapnia, where hyperventilation had been begun to reduce intracranial pressure. But hyperventilation resulted in an obvious insufficiency of the cerebral perfusion and normoventilation had to be gradually restored.

High Jugular Bulb Oxygen Saturation Data

High jugular saturation values were noted equally in the first 5 days of monitoring. Only 8% of the abnormal high SjO_2-values occurred on ICU admission, in contrast to the 30% incidence of abnormal low SjO_2-values noted on admission. This difference could be explained by the fact that, firstly, cerebral hyper-aemia after traumatic brain injury is not reported as an early event [16] and secondly the possible causes of high SjO_2-values detected in our study occurred at a later stage of the ICU management ($PaCO_2$-increases caused by pulmonary infection or by ventilation-perfusion mismatching). After 5 days of monitoring (and till day 16) still 42% of all high SjO_2-values were observed, while only very few desaturations were observed after 5 days monitoring (only 17% of all desaturations occurred after this 5 day period).

An increased arterial carbon dioxide tension

($PaCO_2 > 40$ mmHg) seemed to be the most reported cause of high SjO_2-values. The incidence of hypercapnia ($PaCO_2 > 40$ mmHg) is surprisingly high as indeed rigourous normoventilation was part of our primary neurotrauma care. Apparent causes for these hypercapnic periods were: in 4 periods (= 7%) hypercapnia was observed on admission to the ICU, in 3 periods (= 6%) it was noted after transport of the patient (e.g., to CT scan), in 14 periods (= 27%) during weaning off the ventilation, and finally in 34 periods (= 60%) it occurred suddenly, most probably due to concomitant pulmonary problems with sudden increases in ventilatory dead space, resulting in an increased $PaCO_2$. These 34 sudden increases of $PaCO_2$ were moreover associated with a high incidence of intracranial hypertension. These observations call for extreme vigilance concerning the possible sudden increases of $PaCO_2$ due to pulmonary complications. Perhaps, intermittent bloodgas analysis should be combined with continuous end-tidal carbon dioxide analysis for rapid detection and correction of $PaCO_2$ rises, avoiding possible deleterious intracranial pressure increases.

We found a minority of periods (12 periods) where high SjO_2-values were primarily associated with an increased CPP (above 90 mmHg). These observations may suggest an impairment of cerebral autoregulation, as may occur after severe head injury. Jugular bulb oximetry might indeed reveal an impairment of cerebral autoregulation, as changes in CBF will be reflected by respective alterations in SjO_2, as long as cerebral metabolism remains unchanged. Murr and others [19] recently reported that evaluation of SjO_2 : CPP relationships offers a new and easily accessible information about the status of cerebral autoregulation.

In 8 patients high SjO_2-values were found in association with brain death. As already reported that these high SjO_2-values (approaching the arterial oxygen saturation) strongly suggest the complete arrest of cerebral metabolism [13]. Finally, it should be emphasized that in not less than 21 periods (= 18%) no abnormality in either one of the other cerebral or systemic parameters could be found. The finding of such a high percentage raises a lot of questions as to the clinical value of too high SjO_2-values. Part of these high SjO_2-values could have been "false" high values attributed to increased extracerebral contamination [14]. There are some arguments that especially in situations of already reduced cerebral blood flow, extracerebral contamination of jugular bulb blood could become more important [22].

The question remains if too high SjO_2-values should be returned to normal by any therapeutic intervention. In contrast to jugular desaturations where it was clearly demonstrated that these are associated with a worse neurological outcome [12], very few data exist on the clinical value of too high SjO_2-values. Only in the presence of intracranial hypertension might they be used as a guide to, e.g., the use of therapeutic hyperventilation. However, as there is still a lot of debate around the exact meaning of high SjO_2-values (especially in the presence of cerebral ischaemia which might not always have been detected by abnormal cerebral lactate production in the jugular bulb blood) it seems mandatory to confirm the presence of cerebral hyperaemia by any other cerebral monitoring modality (e.g., transcranial doppler recordings) before installing therapeutic hyperventilation.

In conclusion, it seems that available information at the bedside concerning the adequacy of cerebral perfusion may provide new insights for the general management of severely head injured patients, focussing attention on the huge importance of an adequate cerebral perfusion pressure (perhaps even above 80 mmHg) and on the evenso extreme importance of normoventilation or maintaining $PaCO_2$ within a rather narrow range (above 32 mmHg and below 40 mmHg). These 2 systemic parameters illustrate well the importance of the overall systemic management of severely brain injured patients and they should ban definitively the idea that in severe traumatic brain injury the only involved and to be monitored organ is the brain. The recognition of these systemic influences on the final outcome point to a need for more appropriate systemic monitoring and management in the intensive treatment of severe traumatic brain injury. But the final question remains if by avoiding all situations that might have caused all jugular desaturations found in our material, a better outcome after severe head injury could be obtained.

References

1. Bouma GJ, Muizelaar JP, Stringer WA *et al* (1992) Ultra-early evaluation of regional cerebral blood flow in severely head injured patients with xenon-enhanced computerized tomography. J Neurosurg 77: 360–368
2. Chan KH, Dearden NM, Miller JD *et al* (1992) The effect of changes in cerebral perfusion pressure upon middle cerebral artery blood flow velocity and jugular bulb oxygen saturation after severe head injury. J Neurosurg 77: 55–61

3. Chesnut RM, Marshall LF, Piek J *et al* (1993) Early and late systemic hypotension as a frequent and fundamental source of cerebral ischemia following severe brain injury in the Traumatic Coma Data Bank. Acta Neurochir (Wien) [Suppl] 59: 121–125
4. Cold GE, Jensen FT, Malmros R (1977) The effects of $PaCO_2$ reduction on regional cerebral blood flow in the acute phase of brain injury. Acta Anaesthesiol Scand 21: 359–367
5. Cruz J, Jaggi J, Hoffstad OJ (1993) Cerebral blood flow and oxygen consumption in acute brain injury with acute anemia: an alternative for the cerebral metabolic rate of oxygen consumption. Crit Care Med 21: 1218–1224
6. Dearden NM (1991) Jugular bulb venous oxygen saturations in the management of severe head injury. Current Opin Anesthesiol 4: 279–286
7. Dearden NM (1992) SjO_2 and critical perfusion pressure after severe brain injury. Br J Int Care [Suppl 1]: 7–11
8. Moss E, Dearden NM, Berridge JC (1995) Effects of changes in mean arterial pressure on SjO_2 during cerebral aneurysm surgery. Br J Anesth 75: 527–530
9. De Deyne C, Decruyenaere J, Colardyn F (1994) New insights in the management of acute neurologic crises using jugular bulb oximetry. In: Vincent JL (ed) Update in Int Care and Emerg Medicine. Springer, Berlin Heidelberg New York Tokyo, pp 638–645
10. De Deyne C, Decruyenaere J, Colardyn F (1996) How to interpret jugular bulb oximetry? In: Vincent JL (ed) Update in Int Care and Emerg Medicine, Springer, Berlin Heidelberg New York Tokyo, pp 731–741
11. Enevoldsen EM, Jensen FT (1978) Autoregulation and CO_2-responses of cerebral blood flow in patients with acute severe head injury. J Neurosurg 48: 689–703
12. Gopinath SP, Robertson CS, Contant CF *et al* (1994) Jugular venous desaturation and outcome after head injury. J Neurol Neurosurg Psychiatry 57: 717–723
13. Hantson P, Mahieu P (1992) Usefulness of cerebral venous monitoring through jugular bulb catheterization for the diagnosis of brain death. Int Care Med 18 (1): 59 (Letter)
14. Jakobsen M, Enevoldsen E (1989) Retrograde catheterization of the right internal-jugular vein for serial measurements of cerebral venous oxygen content. J Cereb Blood Flow Metab 9: 717–720
15. Lewis SB, Myburgh JA, Reilly PL (1995) Detection of cerebral venous desaturation by continuous jugular bulb oximetry following acute neurotrauma. Anaesth Intens Care 23: 307–314
16. Marion DW, Darby JM, Yonas H *et al* (1991) Acute regional cerebral blood flow changes caused by severe head injury. J Neurosurg 74: 407–414
17. Miller JD (1985) Head injury and brain ischaemia: implications for therapy. Br J Anesthesiol 57: 102–129
18. Muizelaar JP, Marmarou A, Ward JD *et al* (1991) Adverse effects of prolonged hyperventilation in patients with severe head injury. A randomized trial. J Neurosurg 75: 731–739
19. Murr R, Schurer L (1995) Correlation of jugular venous oxygen saturation to spontaneous fluctuations of cerebral perfusion pressure in patients with severe head injury. Neurol Res 17: 329–333
20. Obrist WD, Langiftt TW, Jaggi JL *et al* (1984) Cerebral blood flow and metabolism in comatose patients with acute head injury. Relationship to intracranial hypertension. J Neurosurg 61: 241–253
21. Robertson CS, Grossman RG, Goodman JC *et al* (1987) The predictive value of cerebral anaerobic metabolism with cerebral infarction after head injury. J Neurosurg 67: 361–368
22. Robertson CS, Narayan RK, Gokoslan *et al* (1989) Cerebral arteriovenous oxygen difference as an estimate of cerebral blood flow in comatose patients. J Neurosurg 70: 222–230
23. Rosner MJ (1986) Cerebral perfusion pressure, intracranial pressure and head elevation. J Neurosurg 65: 636–641
24. Rosner MJ (1990) Cerebral perfusion pressure management in head injury. J Trauma 30: 933–940
25. Rosner MJ, Rosner SD, Johnson AH (1995) Cerebral perfusion pressure: management protocol and clinical results. J Neurosurg 83: 949–962
26. Sheinberg M, Kanter M, Robertson CS *et al* (1992) Continuous monitoring of jugular venous oxygen saturation in head-injured patients. J Neurosurg 76: 212–217
27. Schneider GH, v Helden A, Lanksch WR, Unterberg A (1995) Continuous monitoring of jugular bulb oxygen saturation in comatose patients – therapeutic implications. Acta Neurochir (Wien) 134: 71–75

Comments

The authors present a temporal analysis of jugular bulb oximetry recordings in 50 patients with severe head injury within an intensive care unit setting. They monitored patients for a mean of 7.5 days, allowing for a temporal analysis of their data for a longer time period than has been usually presented in the literature.

Of special interest was their identification of factors associated with high SjO_2, chiefly hypercarbia and systemic hypertension probably in the presence of impaired autoregulation. The prognostic significance of these episodes is not clear.

In their discussion the authors admit to the limitations of their intermittent SjO_2 monitoring routine. As they point out end-tidal CO_2 monitoring could well give earlier warning of impending cerebral perfusion changes. The authors caveat against *blind* hyperventilation is well taken.

Z. H. Rappaport

The paper deals with the analysis of low SjO_2-values, called desaturation periods and high SjO_2-periods in severely injured patients.

Desaturation episodes have already been described in the literature extensively.

The manuscript, however, gives new information about the occurrence of high SjO_2-values. The limit of 80% for "abnormally high" SjO_2-values seems quite appropriate since in earlier reports, normal SjO_2-values in healthy humans are reported even within 70 to 75%. To date, it is unclear whether truely abnormally high SjO_2-values should be treated or corrected, since it is not clear whether these episodes are harmful. It is very interesting that a high percentage of these periods could be corrected by changing the ventilation or decreasing CPP.

A. Unterberg

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