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Clinical reports

Jugular venous oxygen saturation (S_jo_2) monitoring in a patient with fulminant hepatic failure

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Introduction

The mortality rate in patients with fulminant hepatic failure (FHF) is high, and the major cause of death is cerebral edema [1,2]. It has been suggested that, in these patients, autoregulation of cerebral blood flow is absent and that CO_2 reactivity is also compromised [3,4]. Therefore, both cerebral hyperperfusion and hypoperfusion can easily develop. Although some institutions favor the routine use of intracranial pressure (ICP) monitoring [5,6], the placement of an intracranial probe is associated with a risk of bleeding and infection. We had a patient with FHF whose S_jo_2 was continuously monitored during orthotopic liver transplantation (OLT).

Case report

A 26-year-old man (170 cm, 56 kg) developed FHF due to non-A, non-B hepatitis. Because his hepatic encephalopathy (HE) did not respond to conservative management for 10 days, OLT was planned. His HE was classified as stage 4, coma grade 3, according to Bismuth et al. [2]. His electroencephalogram showed low voltage and slow waves. A CT scan of the brain showed minimal brain edema with a severity score of 21 out of a possible 22 according to Wijdicks et al. [5]. His prothrombin time and serum total bilirubin were 25.1 s (control, 11.1 s) and 16.2 mg·dl^{-1}, respectively.

Anesthesia was induced with fentanyl 0.25 mg and isoflurane 0.5%, and was maintained with fentanyl (total 10.5 mg), isoflurane 0.8%–1.0%, propofol (total 1120 mg), and vecuronium (total 54 mg). During the anhepatic period, blood from the right branch of the portal vein was diverted to the left saphenous vein using a biopump with a flow rate of 1.8–2.0 l·min^{-1}. The anhepatic time and the operation time were 1 h 2 min and 22 h 55 min, respectively. Mean arterial blood pressure (MABP) was monitored in the radial artery. S_jo_2 was monitored continuously using a 5.5 F fiberoptic catheter (OptiCath, Oximetrix, Mountain View, CA, USA) connected to an Oximetrix-3 (Abbot, North Chicago, IL, USA). Regional cerebral oxygenation (rSo_2) was monitored with a near-infrared spectroscopy probe (INVOS 3100, Somanetics, Troy, MI, USA) applied to the right forehead.

We failed to obtain a true trace of continuously monitored S_jo_2 values during the early part of the pre-anhepatic stage (Fig. 1 and Table 1). This artificial reading was probably caused by the attachment of the catheter tip to the wall of the blood vessels, because markers indicating a high-quality signal were absent during this period. This problem was solved by withdrawing the catheter by about 1 cm.

Serial changes in S_jo_2, MABP, and $ETco_2$ during anesthesia are shown in Fig. 1. During the pre-anhepatic stage, S_jo_2 fluctuated within the lower ranges. The removal of the recipient's liver coincided with an increase in S_jo_2 followed by an immediate decline, which was confirmed by co-oximetry. This change in S_jo_2 during the anhepatic stage was associated with an increased gradient in pH ($pH_{(j\text{-}a)}$, from −0.075 to −0.352), Pco_2 ($P_{(j\text{-}a)}co_2$, from 7 to 25 mmHg), and blood lactate ($D_{(j\text{-}a)}$lactate, from −3 to 40 mg·dl^{-1}) between the jugular bulb venous blood and the arterial blood (Table 1). There was a transient fall in S_jo_2 following reperfusion.

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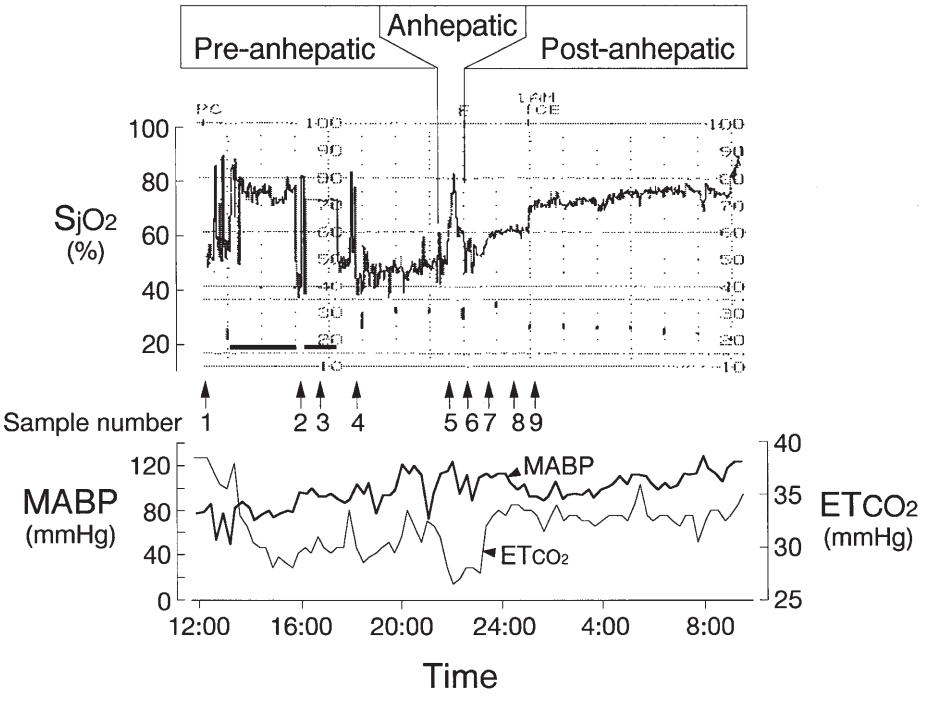

Fig. 1. Changes in continuously monitored jugular venous oxygen saturation (S_jO_2), mean arterial blood pressure (*MABP*), and end-tidal CO_2 ($ETCO_2$). Nine blood samples were taken during the course of the surgery. Sample 1 was taken just before surgery commenced. Samples 2 through 4 were taken during the pre-anhepatic stage. Sample 5 was taken 50 min into the anhepatic stage. Sample 6 was taken 15 min after reperfusion. Samples 7 through 9 were taken 60, 120, and 200 min into the post-anhepatic stage, respectively. The *horizontal bars* on the S_jO_2 trace indicate an artificial recording of fiberoptically determined S_jO_2, because markers indicating a high-quality signal are absent. The abrupt change in S_jO_2 at sample 9 was due to in vivo calibration

Table 1. Changes in co-oximetrically-obtained S_jO_2 and its related parameters

Parameter	Sample								
	1	2	3	4	5	6	7	8	9
Co-oximetric S_jO_2 (%)	57.9	44.4	53.6	47.0	72.2	64.2	64.7	73.5	71.1
Fiberoptic S_jO_2 (%)	50.4	44.6	72.4	41.4	59.7	54.2	58.6	60.5	60.4
rSO_2 (%)	n.d.	58	69	62	71	73	77	77	76
Temperature (°C)	36.0	35.6	35.7	35.8	35.4	35.2	35.4	35.4	35.4
Hb ($g \cdot dl^{-1}$)	8.0	8.8	9.7	11.0	10.8	11.4	12.6	12.9	10.1
MABP (mmHg)	78	80	95	87	123	112	109	99	107
P_aCO_2 (mmHg)	45.3	36.0	36.4	36.1	35.2	36.3	37.1	37.7	n.d.
$P_{(j-a)}CO_2$ (mmHg)	7.0	3.9	7.1	7.4	24.5	12.3	3.2	n.d.	n.d.
$pH_{(j-a)}$	−0.063	−0.095	−0.091	−0.075	−0.352	−0.167	−0.058	−0.059	n.d.
$D_{(j-a)}$lactate ($mg \cdot dl^{-1}$)	−13	11	1	−3	40	21	−1	−11	n.d.
$D_{(j-a)}$glucose ($mg \cdot dl^{-1}$)	12	31	33	−11	34	−16	−26	−52	n.d.

For the sample number, see Fig. 1 legend.

After this episode, it increased gradually for a few hours and then remained stable. Co-oximetrically determined S_jO_2 correlated well with MABP (correlation coefficient, 0.754; $P < 0.05$ by two-tailed Fisher's exact test) and rSO_2 (correlation coefficient, 0.894; $P < 0.005$). There was no correlation between S_jO_2 and P_aCO_2. The jugular-arterial glucose concentration difference ($D_{(j-a)}$glucose) decreased from 34 to $-52\,mg \cdot dl^{-1}$ during the post-anhepatic stage. The patient's neuropsychological faculties gradually recovered, and his disorientation disappeared on the eighth postoperative day. His recovery was satisfactory, and he was discharged a month later.

Discussion

In our patient, S_jO_2 was shown to be closely correlated with MABP but not with P_aCO2, which coincides with

previous reports that CBF in patients with FHF depends on cerebral perfusion pressure [4,7,8]. The values of S_jO_2 seemed to be greater and its fluctuations seemed to be smaller during the later part of the post-anhepatic stage than during the pre-anhepatic stage. The probable cause is that circulatory stability was established after reperfusion. Another explanation is that the reestablishment of CBF autoregulation occurred within several hours after the restoration of liver function. Strauss et al. have reported that the reestablishment of CBF autoregulation in patients with FHF was observed within 24–48 h after OLT [7].

We observed some interesting changes in S_jO_2. The first was a characteristic, transient increase in S_jO_2 during the anhepatic stage. Considering a report that ICP increases transiently after removal of the liver in patients with FHF [8], it seems likely that CBF increased first, followed by an increase in ICP. It might be that an increased intracranial arteriovenous shunting increased both CBF and S_jO_2: the underlying mechanism might be the absence of the liver or the introduction of a portocaval bypass. This vascular engorgement resulted in cerebral ischemia, which was manifested as increases in $pH_{(j\text{-}a)}$, $P_{(j\text{-}a)}CO_2$, and $D_{(j\text{-}a)}$lactate. An increase in MABP could be secondary to an increased ICP. On the other hand, it also seems possible that the anhepatic stage was associated with an enhanced cerebral hyperemic response against an increase in MABP. Whatever the cause(s), the cerebral ischemia ceased after the termination of the anhepatic stage (sample 6). We might not have been able to detect this possible cerebral ischemic episode without sampling jugular bulb blood. The anhepatic stage in patients with FHF might be most critical in terms of brain ischemia. The second was an abrupt decrease in S_jO_2 immediately after reperfusion (sample 6). Although this was probably due to a decrease in MABP, a reperfusion-induced increase in ICP and a decrease in CBF might be also responsible [9].

The brain in patients with FHF has been shown to exhibit depressed glucose consumption [10]. Our patient exhibited a progressive decrease in $D_{(j\text{-}a)}$glucose during the post-anhepatic stage. Although $D_{(j\text{-}a)}$glucose is influenced by CBF, the observed change might indicate an immediate restoration of the brain's glucose-utilizing capacity.

Monitoring of S_jO_2 in patients undergoing OLT has been recently introduced by Skak et al. [11]. They have observed that an initial S_jO_2 value of 73% did not change significantly during the anhepatic stage, increased after reperfusion, and then declined to basal values at the end of the surgery. They mentioned that there was no statistically significant difference in S_jO_2 among 22 patients with end-stage liver disease and 9 with FHF. However, distinctions between acute and chronic types of HE have been recognized [12]. Furthermore, it has been suggested that in patients with FHF, CBF decreased in patients with stage 1 to 3 coma, whereas it increased in those with cerebral edema [3]. Skak et al. did not refer to the preoperative stages of HE or the use of veno-venous bypass during the anhepatic stage in their patients. Aggarwal et al. observed a clear difference in the reperfusion-induced changes in CBF parameters between patients with chronic liver disease without HE and those with FHF in a grade 4 coma: the latter showed a decrease in CBF, but the former did not [9]. We had a 50-year-old woman who underwent living-related OLT for her FHF due to autoimmune hepatitis. In contrast to the case discussed in this report, she was without HE. Her pre-anhepatic and anhepatic rSO_2 values were 65%. rSO_2 increased up to 69% transiently after reperfusion and then returned to around 65%. Therefore, the changes in S_jO_2 in patients with FHF and associated HE might not be so simple as shown by Skak et al.

In summary, we reported a patient with FHF and HE undergoing OLT, during which his S_jO_2 was continuously monitored. Maintaining MABP seemed to be important to avoid a decrease in S_jO_2 or cerebral hypoperfusion. Because the impairment of the cerebral oxygen demand-supply balance could vary among patients with FHF and among stages of the disease, S_jO_2 monitoring might be useful to avoid both cerebral hypoperfusion and hyperperfusion in these patients. Although continuous fiberoptic monitoring of S_jO_2 is useful for early detection of the changes in cerebral oxygen demand-supply balance, determining S_jO_2 values co-oximetrically is also important for artificial recording of fiberoptically-determined S_jO_2. Furthermore, our patient clearly showed that a high value of S_jO_2 failed to guarantee an adequate cerebral oxygen demand-supply balance during the anhepatic stage. To evaluate the adequacy of the cerebral oxygen demand-supply balance, $pH_{(j\text{-}a)}$, $P_{(j\text{-}a)}CO_2$, and $D_{(j\text{-}a)}$lactate should also be measured in conjunction with S_jO_2.

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