Comparison of the Effects of Total Intravenous Anesthesia and Intravenous Inhalational Anesthesia on Brain Metabolism during Surgery

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ABSTRACT

To compare the effects on the brain metabolism of propofol-remifentanil total intravenous anesthesia and desflurane-remifentanil intravenous inhalational anesthesia in neurosurgery. Methods: Thirty-four neurosurgical patients with intracranial space-occupying surgery were randomly divided into two groups: Group A was conducted with the propofol-remifentanil total intravenous anesthesia and Group B with desflurane-remifentanil intravenous inhalational anesthesia. The patients were monitored for the brain metabolism prior to anesthesia, 1 hour after surgery, 2 hours after surgery, and half an hour after surgery. Results: The cerebral venous oxygen saturation, jugular bulb oxygen partial pressure, jugular vein oxygen content, and arterial oxygen content of the patients in group B were significantly higher than that of group A ($P<0.001$); arterial-jugular oxygen content difference and brain oxygen uptake rate of group B were lower than that in group A. Conclusions: (1) Both anesthesia methods can reduce the cerebral oxygen consumption and brain oxygen uptake, and thus reduce the cerebral oxygen metabolism.

Key Words: Total Intravenous Anesthesia, Intravenous Inhalational Anesthesia, Surgery, Cerebral Metabolism

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Introduction

Neurosurgical anesthesia is mainly conducted through general anesthesia. Anesthetics affect brain metabolism by changing nerve cell activity. Many drugs can achieve satisfactory neuro-protection performance when they are used on animals while their effects are difficult to observe when used in clinical research (Li *et al.*, 2012). In addition, the method of conducting anesthesia may also affect brain metabolism. With the increasingly delicate operation of neurosurgery, continuous improvement of anesthesiology, and development of monitoring equipment, brain metabolism monitoring is considered to be an important method for early detection of cerebral ischemia and hypoxia, thus its clinical value is gradually attracting attention. The brain metabolic monitoring during operation is guidance for selecting ideal anesthetic drugs and anesthetic methods, and in the brain protection during the surgery (Lin *et al.*, 2014). SJVO2 as the most widely used measurement method among the various modern cerebral oxygen monitoring techniques, detects the reflux venous blood oxygen saturation in brain tissue through retrograde placement of the catheter in the jugular vein into the jugular bulb (Sørensen *et al.*, 2013). Since the reflux venous blood of the brain tissue collect in the jugular bulbs with rare external venous blood, the sample blood from the

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retrograde placement of catheter in jugular vein can more accurately reflect the condition of cerebral oxygen supply and demand balance and brain metabolism. The blood gas index of the jugular bulb can reflect cerebral oxygen metabolism (Holzer *et al.*, 2013).

Most neurosurgeries are of high risk, which itself will cause the body's strong stress, thus resulting in sympathetic nerve activation. Such effects will sometimes impact the surgical operation, and may lead to brain complications and other postoperative complications in some severe cases and even be fatal (Helmy *et al.*, 2007). Therefore, anesthesia for neurosurgery is highly significant. One of the important tasks of anesthesiologists during the entire operation is cerebral protection. Cerebral protection can minimize the intraoperative brain damage and reduce postoperative complications by decreasing the brain oxygen metabolism, increasing brain resistance to ischemia and hypoxia, removing the oxygen free radicals and protecting cell membranes, so as to improve the patient's life quality. The major neurosurgical anesthesia is general anesthesia, in which multiple drugs are combined to can achieve the synergistic and complementary function, and therefore reduce the drug use, and improve the effects of anesthesia. Anesthetic drugs affect brain metabolism by altering neural cell activity in a manner consistent with the effect of general anesthetic drugs on neuronal function, without affecting the energy metabolism process that maintains cell integrity. Many drugs that show satisfactory neuro-protective effects in ex vivo or in vivo animal don't present observable protective effects in clinical studies. In addition, the method of anesthesia may also affect the cerebral oxygen metabolism. The conventional maintenance drug for total intravenous anesthesia is propofol. When it is used for neurosurgical anesthesia, depending on the dose, propofol can contract cerebral blood vessels, reduce cerebral blood flow, brain metabolic rate, and intracranial pressure, maintains cerebral blood flow related to carbon dioxide index, protects integrity of ion channels and cells and maintains the function of the blood-brain barrier. The representative drugs for intravenous inhalational anesthesia are the inhaled anesthetic agents, which can expand cerebral blood vessels, increase cerebral blood flow, reduce brain metabolism, and thus protects the brain during neurosurgical anesthesia (Bossers *et al.*, 2013). What is the effect of desflurane-remifentanil intravenous inhalational anesthesia on brain metabolism during surgery? What is the difference when it is compared with promofol-remifentanil total intravenous anesthesia? Does the monitoring and analysis of the cerebral oxygen metabolism in the jugular bulb blood and arterial blood gas have any significance for the method and operation of neurosurgery anesthesia? Are they relevant with each other? There is still no report yet.

In this paper, the above two drugs are used along with remifentanil in the total intravenous anesthesia and intravenous inhalational anesthesia for neurosurgery, for the comparison of the effects of the two methods of anesthesia.

Methods

General information

Thirty-six patients undergoing elective surgery for neurosurgery intracranial space occupation from December 2014 to November 2015 were randomly assigned to group A and group B, with 18 cases in each group. Among the patients in group A, 2 patients were excluded due to the long operation time, large bleeding, and blood transfusion during operation. There was no statistical significance in the difference between the age, gender, height, weight and other general information between the two groups of patients.

Anesthetic method

Thirty-four cases were routinely monitored after entering the room. After B ultrasound-guided local anesthesia, retrograde internal jugular vein catheterization and arterial puncture catheterization (Figure. 1, Figure. 2) were performed with atropine 0.01 mg/kg, propofol 2 mg/kg, fentanyl 3 μg/kg, rocuronium 0.8 mg/kg. Anesthesia-induced endotracheal intubation are followed by mechanical ventilation by a ventilator, with tidal volume 8 to 10 m L/kg, respiratory rate 12 times/min, maintaining Sp O_2 98% to 100% and Pet-CO2 35 to 45 mm Hg (1 mm Hg=0.133 kPa). Then, TCI anesthetic maintenance was performed with micro-pumps. Remifentanil TCI plasma concentration was 2.0 ng/mL, and the target-controlled plasma concentration was 2.6-3.5 ng/m L after the initiation of the surgery; the patients in group B inhaled desflurane, with MAC maintained at 1.0; and patients in group A had the propofol TCI plasma concentration of 2.0 μg/m.

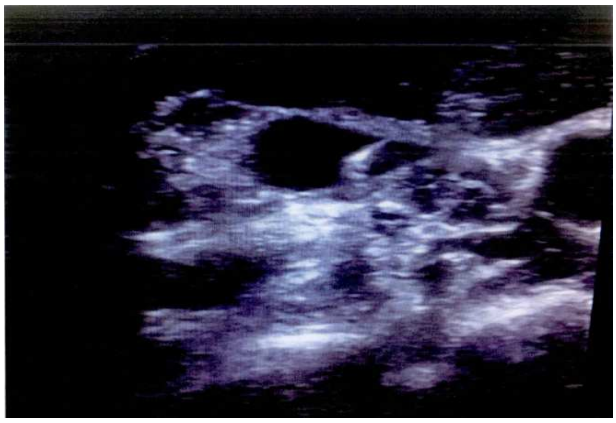

Figure 1. Ultrasound guided internal jugular vein catheterization retrograde catheterization pressure

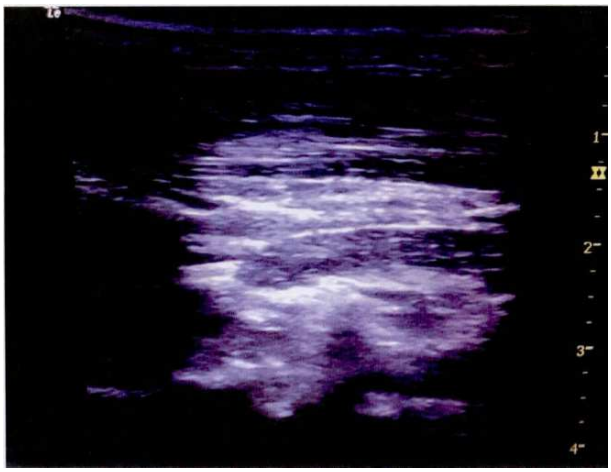

Figure 2. Internal jugular vein retrograde placement ultrasound puncture tube pressure

Monitoring Index

Monitor and record the Sjv O2, Pjv O2, Hbv, Ljv, Gv, SaO2, PaO2, Hba, La, and Ga of the T1, T2, T3, and T4 of the two groups. According to Fick's formula, CaO2, CjvO2, AVDO2, CEO2, CLP, CGU are calculated:

CaO2=Hb×1.36×SaO2+0.0031×PaO2;
CjvO2=Hb×1.36×SjvO2+0.0031×PjvO2;
AVDO2=CaO2-CjvO2; CEO2=(CaO2-Cj-vO22)/CaO2; CLP=(Ljv-La)/La; CGU=(Gv-Ga)/Ga (Purins *et al.*, 2014) .

Statistical analysis

SPSS statistical software is applied to analyze the data. The results are presented with mean ± standard deviation (x±s). The inter-group comparison used repeated measurement data variance analysis, while the intra-group comparison used paired t test. P<0.05 is the difference that is statistically significant.

Results and discussion

For the inter-group comparison, the Sjv O2, Pjv O2, CjvO2, and CaO2 of group B was significantly higher than that of group A (P<0.001); AVDO2 and CEOO2 of group B were lower than that of group A (P<0.05). The Gv and Ga of group B were higher than that of the group A (P<0.05); the CGU, Ljv, La between the two groups had no difference (P>0.05). The CLP of group B was lower than that of group A (P<0.05). For the intra-group comparison, PaO2, SaO2, PjvO2, and SjvO2 of both groups increased with time (P<0.05), and CaO2, CjvO2, AVDO2, and CEO2 of the two groups decreased with time (P<0.05). Refer to Table 1 and Figures 3, 4, and 5 for the results.

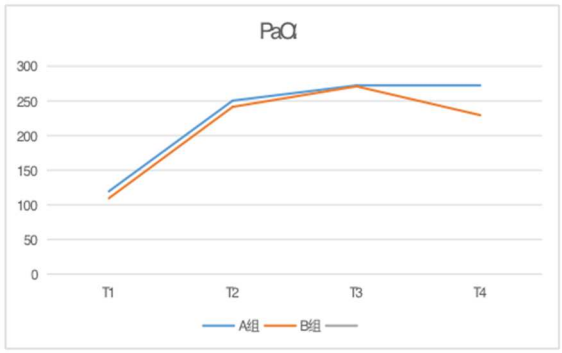

Figure 3. PaO_2 Trend of change

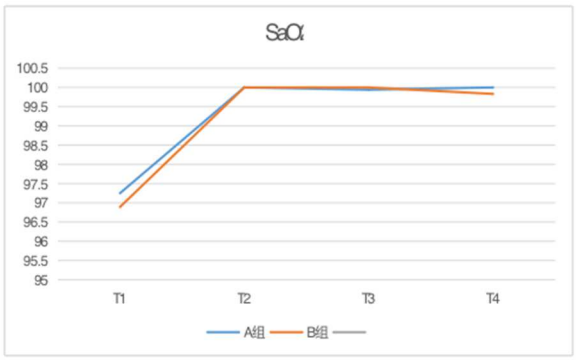

Figure 4. SaO_2Trend of change

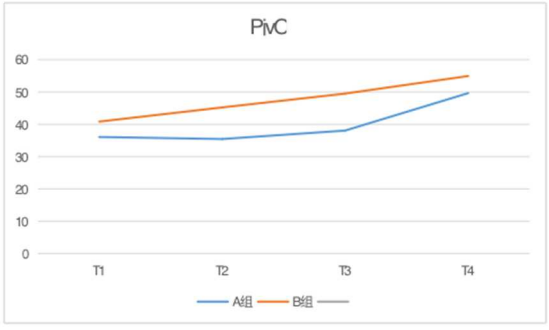

Figure 5. $PjvO_2$Trend of change

Table 1. comparison of brain metabolic indices at each time point (x±s)

Observationindicators	Groups	T1	T2	T3	T4
PaO_2 (mmH) g	A	119.44±51.30	250.44±78.85•	272.31±72.34•	271.56±65.46•
	B	109.50±34.09	241.39±51.53•	271.06±50.82•	229.44±49.31•
SaO_2 (%)	A	97.25±1.95	100•	99.94±0.25•	100•
	B	96.89±1.78	100•	100•	99.83±0.71•
Pjv O_2 (mm H) g	A	36.06±2.84	35.44±8.43	38.06±7.35•	49.63±15.92•
	B	40.83±6.97	45.22±7.50▲•	49.50±14.26▲•	54.94±7.00▲•
$SjvO_2$ (%)	A	64.38±5.43	62.31±11.18	67.25±9.96•	76.25±11.09•
	B	71.44±9.81	75.06±6.87▲•	79.06±10.36▲•	83.39±7.03▲
CaO_2 (m L/L)	A	181.86±20.69	159.12±15.42•	153.06±24.22•	138.97±21.44•
	B	184.33±20.00	174.00±17.79▲•	164.19±20.19▲•	439.77±84.27▲•
$CjvO_2$ (mL/L)	A	120.61±19.65	101.02±16.54•	101.75±15.55•	108.05±26.07•
	B	135.30±22.14	130.52±18.51▲•	127.77±20.50▲•	124.27±23.11▲•
$AVDO_2$ (mL/L)	A	61.25±15.17	58.11±20.18	51.31±24.87•	30.92±16.69•
	B	49.03±24.61	43.48±11.20▲	36.42±12.92▲•	315.50±72.07▲•
CEO_2 (%)	A	33.69±7.68	36.14±11.19•	32.53±11.93•	22.47±12.00•
	B	26.05±13.24	25.10±6.33▲•	22.20±7.98▲•	71.35±4.50▲•
Ga (mmol/L)	A	4.44±0.79	4.43±0.80	4.58±0.82	5.38±1.04
	B	4.77±1.27	5.26±1.39▲	5.44±1.48▲	5.94±1.16▲
Gv (mmol/L)	A	3.88±0.92	3.94±0.91	4.19±0.86	5.04±1.06
	B	4.28±1.10	4.84±1.11▲	5.26±1.10▲	5.79±0.93▲
CGU (%)	A	-13.00±9.25	-11.08±9.56	-8.64±7.02	-6.30±7.18
	B	-5.03±38.48	-0.26±50.45	-3.67±41.78	-6.15±26.37
La (mmol/L)	A	3.11±0.82	2.94±1.02	2.38±0.75	2.37±1.15
	B	2.49±0.80	2.34±0.91	2.17±0.58	2.96±0.98
Ljv (mmol/L)	A	2.76±0.92	2.34±0.73	2.03±0.74	2.06±1.12
	B	2.26±0.74	2.33±0.68	2.17±0.78	2.71±1.08
CLP (%)	A	11.67±15.78	18.75±15.43	13.69±19.04	12.81±18.06
	B	7.06±17.42	9.36±46.87▲	0.28±27.39▲	8.60±14.19▲

Compared with group A, ▲P<0.05; compared with T1, P<0.05.

Conclusion and outlook

Sj O2 monitoring is considered to be the golden standard for assessing cerebral oxygen metabolism, which reflects the balance between oxygen supply and oxygen metabolic rate in brain tissue. The normal value of Sj O2 is 54% to 75%. Sj O2<50% indicates the risk of ischemic damage in the cerebral hemisphere; Sj O2> 75% indicates an increasing cerebral oxygen supply or cerebral blood flow, suggesting a possibility of cerebral congestion or reduced brain metabolism. It may also be caused by low catheter position and mixed surface venous blood (Saidman, 1991). The comparison between the two groups showed that the Sj-v O2, PjvO2, CjvO2, and CaO2 of group B at T2, T3 and T4 were higher than that of group A, and AVDO2 and CEO2 were lower. Such results indicate that intravenous inhalational anesthesia reduces brain metabolism, resulting a cerebral blood flow higher than metabolic requirements, therefore the oxygen ingested by brain tissue from the blood stream decreases, leading to increased oxygen levels in the brain's venous blood, and subsequent increased tolerance of brain tissue to hypoxia.

The comparison of CGU between the 2 groups had no statistical significance. The Gv, Ga of group B was higher than that of group A. Ljv and La of the two groups showed no difference, and CLP of group A was significantly higher than that of group B. Such changes can't be explained with single factor due to the brain tissue's ability of release and uptake of lactic acid. It only shows that total intravenous anesthesia lead to a more obvious anaerobic metabolism, thus the anesthesiologists should give more attention to brain metabolism. For the intra-group comparison, PaO2, SaO2, PjvO2, and SjvO2 increased over time (P<0.05), and CaO2, CjvO2, AVDO2, and CEO2 decreased over time (P<0.05), indicating that both methods of anesthesia can reduce brain oxygen consumption, decrease the cerebral oxygen uptake and cerebral oxygen metabolism, and therefore achieve the brain protection function. In summary, both of total intravenous anesthesia and intravenous inhalational anesthesia can reduce the cerebral oxygen metabolism, with intravenous inhalational anesthesia generate lower level of lactic acid than total intravenous anesthesia and thus lead to lower anaerobic metabolism; it seems that

intravenous inhalational anesthesia has a stronger brain protection effects than the total intravenous anesthesia.

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