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Systemic and Cerebral Haemodynamics During Craniotomy Under Mild Hypothermia in Patients with Acute Subarachnoid Haemorrhage

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Summary

Background. Mild hypothermia provides cerebral protection against ischaemic insults in various animal models. We compared systemic and cerebral oxygenation between mild hypothermic and normothermic management in 60 patients with acute subarachnoid haemorrhage who underwent clipping of cerebral aneurysms.

Method. The temperature in the pulmonary artery was maintained at 36 °C in 28 patients and was reduced to 34 °C in 32 patients. Parameters in the systemic and cerebral haemodynamics from pulmonary artery and internal jugular vein catheters were compared between the two groups immediately after the induction of anaesthesia (T1), and just before temporary occlusion or aneurysm clipping (T2).

Findings. Cardiac index, oxygen delivery index, oxygen consumption index, and oxygen saturation of the jugular bulb were significantly lower at T2 in hypothermic group (H) (2.9 ± 0.6 L/min/m^2, 400.8 ± 106.3 ml/min · m^2, 87.0 ± 14.8 ml/min · m^2, 55.2 ± 6.6%, respectively) than in normothermic group (N) (3.7 ± 0.6, 521.0 ± 105.5, 109.9 ± 21.7, 60.9 ± 6.6) ($p < 0.05$). The arterial lactate and arteriojugular difference in oxygen content were significantly higher in H (2.3 ± 1.3 mmol/L, 6.5 ± 1.5 ml/dl, respectively) than in N (1.7 ± 1.0, 5.6 ± 1.2) ($p < 0.05$). Arteriojugular differences in carbon dioxide tension and hydrogen ion content were significantly lower at T2 in H (-10.8 ± 2.1 mm Hg, -6.4 ± 1.3 nmol/L, respectively) than in N (-8.9 ± 2.8, -5.3 ± 1.0) ($p < 0.05$).

Interpretation. The balance between oxygen supply and demand systemically and in the brain may worsen during aneurysm surgery for patients with acute subarachnoid haemorrhage under mild hypothermia. Oxygenation of the brain and the whole body should be monitored closely during this surgery, and adequate circulatory assistance is recommended under mild hypothermia.

Keywords: Haemodynamics; cerebral circulation; mild hypothermia; subarachnoid haemorrhage.

Introduction

Hypothermia at 25 °C or less was used during cerebral aneurysm surgery in the 1950's for brain protection against ischaemia due to temporary occlusion [13, 31]. However, serious systemic complications, such as cardiac arrest, severe arrhythmias [25], circulatory inhibition, bleeding tendency, and high rates of infection led to poor outcomes [16, 50]. In contrast, recent experiments have shown that mild or moderate hypothermia, i.e., 30 to 34 °C, can provide cerebral protection through inhibition of excitatory amino acid release, prevention of brain edema by blood-brain barrier protection, delayed influx of intracellular Ca^{++}, inhibition of adenosine triphosphate decrease by stabilisation of cell membranes, inhibition of leukotriene production, and increased free radical scavenger functions [5, 6, 24, 36]. In addition, mild or moderate hypothermia causes fewer side effects [10, 32]. Therefore, mild hypothermia is now used during cerebral aneurysm surgery, as well as in the treatment of brain swelling and intracranial hypertension after severe head injury and cardiopulmonary resuscitation [32, 40]. Marion *et al.* [26] demonstrated that 62% in the treatment of moderate hypothermia and 38% in normothermia had good outcomes at 12 months in a randomised control trial. Shiozaki *et al.* [45–47] analysed the efficancy of inducing mild hypothermia (34 °C) in severe head injured patients, and concluded that mild hypothermia was effective for preventing intracranial pressure (ICP) elevation in patients without diffuse brain swelling whose ICP remained higher than 20 mm Hg but less than 40 mm Hg after conventional therapies, including fluid restriction, hyperventilation, and high dose barbiturate therapy. Holzer *et al.* [18] analysed the effects of mild therapeutic hypothermia after ventricular fibrillation cardiac arrest, and demonstrated the outcome measured at 6 months with cerebral performance category score was found to have a two-fold improvement compared to historic control.

In patients with the acute stage of subarachnoid haemorrhage (SAH), systemic haemodynamics are unstable due to catecholamine release [41]. Cerebral blood flow (CBF) reduction may occur due to a rapid increase in intracranial pressure, and cause primary brain damage. Hindman *et al.* [17] analysed patients with and without SAH undergoing aneurysmal clipping under mild hypothermia (33.5 °C) or normothermia (36.5 °C), and compared the neurological outcomes. They found that among patients with SAH, patients managed under mild hypothermia had a lower frequency of neurological deficit at 24, 72 hours after surgery and more returned home than under normothermia, although not statistically significant; but that among patients without SAH, there was no difference in neurological outcomes between those managed under mild hypothermia and normothermia. However, the effects of hypothermia, induced during the acute phase of SAH, on cerebral circulation and metabolism are still unclear.

This study investigated the changes in systemic circulatory dynamics and cerebral oxygen supply-demand balance during mild hypothermia in patients with acute SAH undergoing craniotomy.

Patients and Methods

Subjects

Sixty patients with SAH were involved in this study with approval of institutional ethics committee and informed consents. All of these cases were operated on within 72 hours after the onset of SAH, under mild hypothermic management in 32 patients (hypothermic group: H) and normothermic in 28 (normothermic group: N). Table 1 shows patient population including age, sex, Hunt and Hess grade, and location of the ruptured aneurysms.

Table 1. *Patient Population in the Normthermic and Hypothermic Group*

		Normothermic group	Hypothermic group
Case number		28	32
Age		35–81 (mean 55.9)	37–72 (mean 56.7)
Sex	(male/female)	12/16	12/20
H-H	(I/II/III/IV)	4/13/7/4	0/19/11/2
AN location	AcomA	12	12
	ICA	4	11
	MCA	7	5
	PICA	1	3
	ACA	3	0
	BA	1	1

H-H Hunt and Hess grade; *AN Location* location of the ruptured aneurysms; *AcomA* anterior communicating artery; *ICA* internal carotid artery; *MCA* middle cerebral artery; *PICA* posterior inferior cerebellar artery; *ACA* peripheral anterior cerebral artery; *BA* basilar artery.

Methods

Anaesthesia. Anaesthetic management was similar in both groups. Anaesthesia was induced with propofol, 1–2 mg/kg, and fentanyl, 3–7 μg/kg. Following administration of vecuronium, 0.1 mg/kg, patients were intubated and ventilated. The arterial carbon dioxide tension ($PaCO_2$) was maintained at 35 mm Hg using α-stat management. Anaesthesia was maintained by continuous infusion of propofol, 5–15 mg/kg/h, and intermittent administration of fentanyl. Muscle relaxation was monitored and vecuronium was administered intermittently when necessary. Haemoglobin (Hb) levels of less than 9 g/dl were corrected by infusing irradiated red cells. Central venous pressure (CVP) was maintained at 3 to 6 mm Hg by crystalloid with supplemental colloid infusion to maintain oncotic pressure. Neither inotropes nor vasodilators, except for administration of 0.01 μg/kg/min of prostaglandin E_1 (PGE_1) to all cases, was used during this study. Previous reports demonstrated that PGE_1 is beneficial to maintain liver and renal circulation [21, 35], and suitable for induced hypotension during cerebral aneurysm surgery because flow/metabolism coupling of brain and regional cerebral oxygenation are well maintained and cerebral vascular reactivity to carbon dioxide preserved during hypotensive anaesthesia [1, 23].

Monitoring and Measurement. Electrocardiography (ECG), percutaneous oxygen saturation, noninvasive arterial pressure, invasive arterial pressure, and end-tidal carbon dioxide concentration were monitored. Following induction of anaesthesia, a pulmonary artery catheter (Swan Ganz CCOmbo 744HF75; Baxter Healthcare Co., CA) and jugular bulb catheter (fiberoptic intravascular catheter, P540-H; Abbott Laboratories, IL) were inserted, and cardiac index (CI), pulmonary arterial pressure, CVP, mixed venous oxygen saturation (SvO_2), and jugular bulb oxygen saturation (SjO_2) were monitored continuously. Arterial, mixed venous, and jugular bulb blood were sampled and analysed immediately after induction of anaesthesia (T1) and just before temporary occlusion or aneurysm clipping (T2) (Rapidlab 860 with 800 CO oximeter module; Chiron Diagnostics Co., MA).

Arterial oxygen content (CaO_2), mixed venous oxygen content (CvO_2), jugular bulb venous oxygen content (CjO_2), systemic oxygen delivery index (DO_2I), systemic oxygen consumption index (VO_2I), systemic oxygen extraction ratio (O_2ER), systemic vascular resistance index (SVRI), arteriojugular difference in oxygen content ($AjDO_2$), arteriojugular difference in hydrogen ion content ($AjDH^+$), arteriojugular difference in carbon dioxide tension ($AjDPCO_2$), and lactate oxygen index (LOI) were calculated using the following equations.

$$CaO_2 = 1.34 \cdot Hb \cdot SaO_2/100 + 0.0031 \cdot PaO_2 \text{ (ml/dl)}$$
$$CvO_2 = 1.34 \cdot Hb \cdot SvO_2/100 + 0.0031 \cdot PvO_2 \text{ (ml/dl)}$$
$$CjO_2 = 1.34 \cdot Hb \cdot SjO_2/100 + 0.0031 \cdot PjO_2 \text{ (ml/dl)}$$
$$DO_2I = CI \cdot CaO_2 \cdot 10 \text{ (ml/min/m}^2\text{)}$$
$$VO_2I = CI \cdot (CaO_2 - CvO_2) \cdot 10 \text{ (ml/min/m}^2\text{)}$$
$$O_2ER = VO_2I/DO_2I$$
$$SVRI = (MAP - CVP)/CI \cdot 80 \text{ (dyn} \cdot \text{sec/cm}^5\text{/m}^2\text{)}$$
$$AjDO_2 = CaO_2 - CjO_2 \text{ (ml/dl)}$$
$$AjDH^+ = 10^{9-pHa} - 10^{9-pHj} \text{ (nmol/L)}$$
$$AjDPCO_2 = PaCO_2 - PjCO_2 \text{ (mm Hg)}$$
$$LOI = -(\text{arterial lactate} - \text{jugular vein lactate}) \text{ (mmol/L)}/AjDO_2 \text{ (ml/dl)} \cdot 2.24 \text{ [44]}$$

Where SaO_2 = arterial oxygen saturation, PaO_2 = arterial oxygen tension, PvO_2 = mixed venous oxygen tension, PjO_2 = jugular bulb oxygen tension, MAP = mean arterial pressure, pHa = pH of arte-

Table 2. *Haemodynamic and Oxygenation Values in the Normothermic and Hypothermic Groups at T1 and T2*

	Normothermic group		Hypothermic group	
	T1	T2	T1	T2
CI	3.7 ± 0.7	3.7 ± 0.6	4.0 ± 1.0	2.9 ± 0.6**++
SvO2	83 ± 3.1	79 ± 4.0**	83 ± 4.6	77 ± 5.5**
lactate	1.9 ± 1.3	1.7 ± 1.0	2.4 ± 1.3	2.3 ± 1.3+
DO2I	580 ± 120	521 ± 105	611 ± 168	400 ± 106**++
VO2I	98.5 ± 20.2	109.9 ± 21.7*	99.5 ± 16.0	87.0 ± 14.8*++
O2ER	0.17 ± 0.03	0.22 ± 0.05**	0.17 ± 0.05	0.22 ± 0.05**
MAP	78.5 ± 13.1	78.5 ± 6.2	77.2 ± 12.2	76.7 ± 8.7
SVRI	1634 ± 502	1605 ± 348	1514 ± 454	2102 ± 585**++
SjO2	60.0 ± 8.5	60.9 ± 6.6	55.5 ± 7.7	55.2 ± 6.6++
AjDO2	6.23 ± 1.4	5.62 ± 1.2	7.14 ± 1.6	6.48 ± 1.5+
AjDH+	−5.7 ± 1.6	−5.3 ± 1.0	−6.1 ± 1.8	−6.4 ± 1.3++
AjDPCO2	−11 ± 4.8	−8 ± 2.8	−9 ± 2.8	−11 ± 2.1*+
LOI	−0.011 ± 0.072	−0.013 ± 0.073	−0.056 ± 0.077	−0.045 ± 0.078

Values are expressed as mean ± standard deviation. *T1* immediately after the induction of anaesthesia; *T2* just before temporary occlusion or aneurysm clipping; *CI* cardiac index; *SvO2* oxygen saturation of mixed venous blood; lactate: arterial lactate; *DO2I* oxygen delivery index; *VO2I* oxygen consumption index; *O2ER* oxygen extraction ratio; *MAP* mean arterial pressure; *SVRI* systemic vascular resistance index; *SjO2* oxygen saturation of jugular bulb; *AjDO2* arteriojugular difference in oxygen content; *AjDPCO2* arteriojugular difference in carbon dioxide tension; *AjDH+* arteriojugular difference in hydrogen ion content; *LOI* lactate oxygen index. Comparison of values between the normothermic and hypothermic groups at T2, statistical significance: +($p < 0.05$), ++($p < 0.01$). Comparison of values between T1 and T2 in each group, statistical significance: *($p < 0.05$), **($p < 0.01$).

rial blood, pHj = pH of jugular venous blood, $PaCO_2$ = arterial carbon dioxide tension, and $PjCO_2$ = jugular bulb carbon dioxide tension.

Temperature Control. Body temperature was continuously measured at the tip of the pulmonary artery catheter, and adjusted to 34 °C (H) or 36 °C (N) using a water-circulating blanket (Blanket II; Cincinnati Sub-Zero Products Inc., OH), an air-cooling blanket (Warm Touch Model 5200; Mallinckcrodt Medical Inc., MO), and cold (7 °C) or warm (37 °C) transfusion after the completion of measurement at T1. Rewarming was started after clipping of the aneurysms using the blankets and warm transfusion. Induction of hypothermia, rewarming, and extubation were performed routinely in the operating room.

Statistics. Parameters within each group were analysed by the paired T test. Intergroup differences in parameters were compared by the unpaired two groups T test, differences in sex and location of the aneurysm were assessed by the chi-square test, and differences in Hunt and Hess grades were compared by the Mann-Whitney u test. Values were expressed as mean ± SD, and $p < 0.05$ were considered statistically significant.

Results

There were no statistical differences in age, sex, or Hunt and Hess grade between the two groups. Mean body temperature at T1 was 36.2 °C in H and 36.4 °C in N, being not statistically different. As intended, at T2, patients assigned to H were significantly colder than those assigned to N, with mean body temperatures of 34.1 °C versus 36.5 °C, respectively. Hypothermia was induced in 20 to 195 minutes (mean 65 minutes), and rewarming required in 45 to 250 minutes (mean 115 minutes). The mean duration of anaesthesia in H was 608.7 ± 164.8 minutes, significantly longer than in N, 507.2 ± 202.3 minutes ($p < 0.05$).

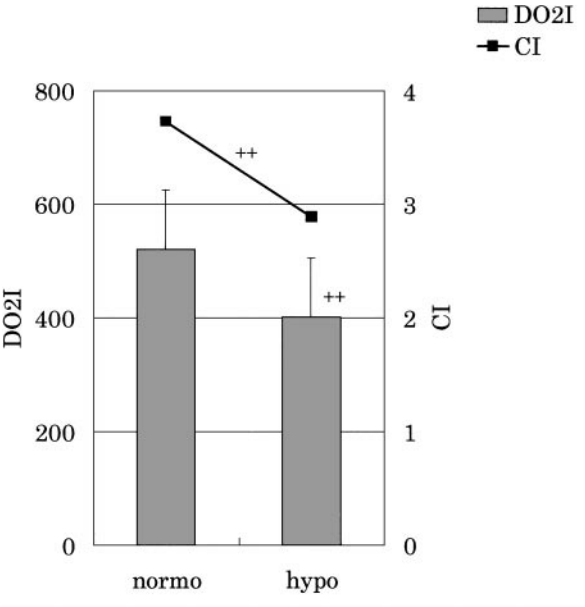

Fig. 1. Comparison of cardiac index (CI) and oxygen delivery index (DO2I) between the two groups just before temporary occlusion or aneurysm clipping (T2). CI and DO2I were significantly lower in the hypothermic group (hypo) than in the normothermic group (normo). Statistical significance: ++($p < 0.01$)

Table 2 shows values of the parameters, and Figs. 1 and 2 compare the parameters at T2 between the two groups. There were no significant differences in any

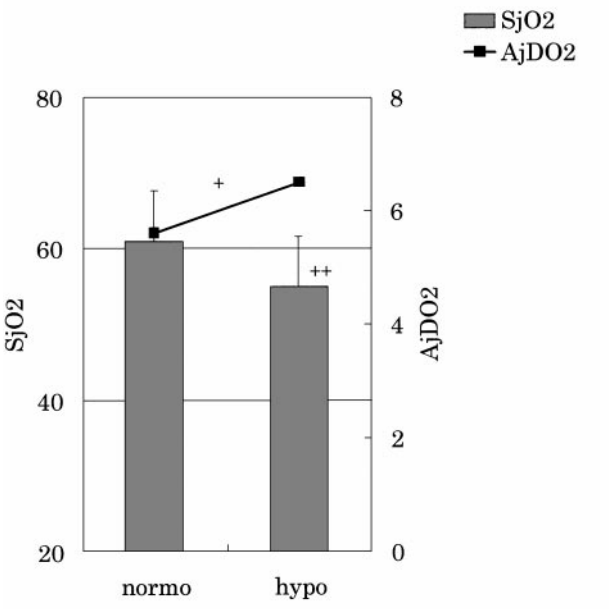

Fig. 2. Comparison of oxygen saturation of jugular bulb (SjO2) and arteriojugular difference in oxygen content (AjDO2) between the two groups just before temporary occlusion or aneurysm clipping (T2). SjO2 was significantly lower and AjDO2 was significantly higher in the hypothermic group (hypo) than in the normothermic group (normo). Statistical significance: $+(p < 0.05)$, $++(p < 0.01)$

parameters, including body temperature, between the two groups at T1. In contrast, differences were found at T2 as follows.

Systemic Circulatory Dynamics and Systemic Oxygen Supply-Demand Balance

CI and DO_2I were significantly lower in H at T2 ($p < 0.01$). VO_2I was also significantly lower in H ($p < 0.01$). There were no intergroup differences in O_2ER and SvO_2. Arterial lactate content and SVRI were significantly higher in H ($p < 0.05$ and $p < 0.01$, respectively). MAP was not significantly different between the two groups.

Cerebral Oxygen Supply-Demand Balance

SjO_2 was significantly lower in H at T2 ($p < 0.01$). $AjDO_2$ was significantly higher ($p < 0.05$) in H. $AjDPCO_2$ and $AjDH^+$ were significantly lower ($p < 0.05$, and $p < 0.01$, respectively) in H. The LOI was normal (<0.03) in all patients, and there was no significant difference in LOI between the two groups.

Discussion

Previous investigations of cardiac function during hypothermia have shown that profound hypothermia causes severe arrhythmias such as ventricular fibrillation more frequently than during mild or moderate hypothermia. However, assessments of cardiac output are varied. Bacher *et al.* [2] compared 13 patients managed with hypothermia at 32 °C and 6 patients with normothermia at 35.5 °C during scheduled neurosurgical operations, and found no significant intergroup differences in CI and DO_2I, but significant reduction in VO_2I, resulting in a significant reduction of O_2ER during hypothermia. Stable oxygen delivery and a decreased oxygen metabolic rate caused no change or reduction in O_2ER, and no reduction in the systemic oxygen supply-demand balance [3, 32]. However, 9 patients with severe head trauma, managed with hypothermia at 32.5 to 33 °C, showed decreased cardiac output within 24 hours after the induction of hypothermia [29]. Experimental animals showed decreased cardiac output more than 24 hours after the induction of hypothermia, suggesting a worsening oxygen supply-demand balance [30]. In contrast, our study showed a lower CI, DO_2I, and VO_2I in H at T2, but no significant intergroup difference in O_2ER. We believe that discrepancies between our results and previous findings are due to the differences in subjects and management of hypothermia. Management of hypothermia in Bacher's series was similar to ours, although the aneurysms in his subjects had not ruptured. Patients with acute SAH develop an unstable systemic circulation due to sympathetic hypertonia accompanied by reduced intravascular capacity and circulatory complications [12, 14, 34, 42, 52]. Approximately 50 to 75% of patients with SAH demonstrate ECG abnormalities [53]. Catecholamine toxicity causes band necrosis of the cardiac muscles leading to impairment of left ventricular performance in such patients, and delayed cerebral vasospasm occurs, especially in patients with decreased cardiac output [27]. Thus, cardiac function may be worsened in patients with acute SAH so that even mild hypothermia may impair cardiac function.

Previous studies have suggested that reduction of VO_2I following induction of hypothermia is caused by both reduced oxygen metabolism due to lower body temperature and a decreased cellular oxygen extraction ratio because the Hb oxygen dissociation curve is shifted to the left [4, 7, 43]. The present study also

shown no significant difference in SvO_2 between the two groups at T2, but found a significantly higher level of lactate, an indicator of anaerobic metabolism, in H. This finding might suggest that the systemic oxygen supply-demand balance was aggravated by inhibition of circulation and inhibition of oxygen transfer from Hb to the tissues under mild hypothermia.

SjO_2 is determined by SaO_2, Hb, CBF, and cerebral metabolic rate of oxygen ($CMRO_2$), and can be calculated by the following equation.

$$SjO_2 = SaO_2 - 100 \cdot CMRO_2/(1.34 \cdot Hb \cdot CBF/100) \quad [37]$$

Rapid changes in SaO_2 or Hb require careful interpretation [11], but changes in SjO_2 may reflect changes in CBF and $CMRO_2$. Reduction of SjO_2 to $\leq 54\%$ suggests cerebral hypoperfusion, and $\leq 40\%$ suggests diffuse cerebral ischaemia [33, 38]. In the present series, SjO_2 at T2 was significantly lower in H, $55.2 \pm 6.6\%$, than in N, $60.9 \pm 6.6\%$ ($p < 0.01$).

$AjDO_2$ is calculated by the Fick principle as $AjDO_2 = CMRO_2/CBF$. Robertson *et al.* [38] demonstrated that CBF could be predicted reliably from the $AjDO_2$, if LOI was less than 0.08, as $CMRO_2$ were held constant at a particular value. Roux *et al.* [39] investigated post-treatment changes in $AjDO_2$ in 32 patients with severe head injury and found that patients with increased $AjDO_2$ after treatment suffered from delayed cerebral infarction and had poor outcomes. In the present series, $AjDO_2$ at T2 was significantly greater in H ($p < 0.05$). Cerebral perfusion pressure (MAP – intracranial pressure) is stable during craniotomy, but cerebrovascular autoregulation is impaired during the acute stage of SAH [14, 52]. Therefore, circulatory inhibition under intra-operative mild hypothermia might have directly caused lower CBF rather than being coupled to $CMRO_2$ reduction induced by mild hypothermia, and resulted in significantly greater $AjDO_2$ in H at T2.

The normal value of $AjDPCO_2$ is -10 ± 2.4 mm Hg [15]. Greater differences in $AjDPCO_2$ may be caused by CO_2 retention due to slow capillary blood flow, the bicarbonate buffering effect on acid-base balance, and increased CO_2 production by anaerobic metabolism caused by the reduced oxygen supply-demand balance [22, 48]. In the present series, $AjDPCO_2$ at T2 was -10.8 ± 2.1 mm Hg in H, significantly lower than -8.9 ± 2.8 mm Hg in N ($p < 0.05$). The normal value of $AjDH^+$ is -4.92 ± 1.74 nmol/L [15]. $AjDH^+$ is an indicator of brain tissue hypoxaemia [51, 54]. In this series, $AjDH^+$ at T2 was -6.4 ± 1.3 nmol/L in H, significantly lower than -5.3 ± 1.0 nmol/L in N ($p < 0.01$). Chieregato *et al.* [9] reported that serial changes in $AjDPCO_2$ and $AjDH^+$ may be an effective indicator of cerebral oxygen supply-demand balance following CBF reduction in the acute stage of severe head injury. Our study showed that the cerebral oxygen supply-demand balance might worsen under mild hypothermia, compared with normothermia, in the acute stage of SAH.

The normal level of LOI is < 0.03; > 0.08 indicates global cerebral ischaemia [44]. Moss *et al.* [33] measured LOI during clipping of cerebral aneurysms in 26 patients and found that patients with LOI > 0.08 had worse neurological function than patients with LOI < 0.08. In all patients in this study, LOI was within normal limits at both T1 and T2, and LOI at T2 was not significantly different between the two groups. But changes in LOI from T1 to T2 showed a tendency to increase in H. However, standard deviations of LOI were too high to predict reliably the cerebral oxygen supply and demand balance from LOI only. Taking the results of the comparison between the two groups at T2 in $AjDO_2$, $AjDH^+$, and $AjDPCO_2$ into consideration, this finding might suggest that the cerebral oxygen supply-demand balance aggravated to such a degree that anaerobic matabolism did not increase. Robertson *et al.* [38] defined the state in which LOI was less than 0.08 and $AjDO_2$ was higher than normal "compensated hypoperfusion". The brain was compensating for the decreased blood flow by extracting oxygen more completely, and the low blood flow did not appear to have altered overall cerebral metabolism, because $CMRO_2$ did not change or decrease by mild hypothermia, and anaerobic metabolism was not increased. Moreover, The rate at which lactate moves from brain to venous blood has been controversial [29]. Lactate produced by anaerobic metabolism induced hypoxaemia releases not only into the blood but also into the cerebrospinal fluid [20]. Measuring the lactate level in the blood as well as in the cerebrospinal fluid [49] or the cerebral intercellular matrix using microdialysis [19] may provide more accurate assessment of the cerebral oxygen supply-demand balance.

In this study, the oximetry catheter was inserted into the internal jugular vein on the side with greater venous return confirmed by pre-operative cerebral angiography, in most cases on the right side. However, the values of the parameters may vary depending on the side of the catheterisation, i.e. ipsilateral or con-

tralateral to the craniotomy. Bilateral internal jugular venous catheterisation will provide a more accurate assessment, especially for investigating the whole-brain oxygen supply-demand balance during intra-operative mild hypothermia [8, 28].

In conclusion, intra-operative mild hypothermia for patients in the acute stage of SAH may cause circulatory inhibition and possible worsening of the systemic and cerebral oxygen supply-demand balances. Therefore, strict monitoring of the systemic and cerebral haemodynamics and oxygenation, and adequate circulatory assistance are recommended during mild hypothermia. More investigation of the effects of intra-operative mild hypothermia especially in the acute stage of SAH on the cerebral and systemic haemodynamics and oxygenation are warranted to clarify this conclusion.

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Comments

The effects of hypothermia on cardiac function, systemic haemodynamics and oxygenation have already been extensively studied in the anaesthesia of cardiac-surgery in the past four decades. During the last 10 years mild to moderate hypothermia have been repeatedly demonstrated to reduce neuronal injury in animal models of focal cerebral ischaemia and traumatic brain injury. However, the cerebral effects of mild cooling has not been investigated in clinical situations such as acute subarachnoid haemorrhage. Furthermore, neuroclinicians eagerly seek the answer to the question wether mild intraoperative hypothermia is a protective measure during aneurysm clipping.

This study – precisely and carefully carried out – adds some new information on the issue of mild hypothermia in subarachnoid haemorrhage.

T. Dóczi – J. Futó

The authors have compared parameters of systemic and cerebral oxygenation during intra-operative hypothermic and normothermic management of 60 patients with subarachnoid haemorrhage. Patients treated with intra-operative hypothermia (34 °C) had significantly higher arteriovenous oxygen difference, lower jugular venous oxygen saturation, lower cardiac index and lower oxygen delivery index compared to those treated with normothermia during surgery. From these findings, the authors emphasise that mild hypothermia during the acute stage of SAH may worsen the cerebral oxygen supply-demand balance.

Intra-operative hypothermia is increasingly used to provide a maximum of neuronal protection during temporary clipping or other potential causes of intra-operative ischaemia (e.g. intra-operative rupture). There are no hard data on outcome to recommend intra-operative hypothermia as a standard management in patients with SAH, however, there is convincing experimental evidence that hypothermia increases the neuronal ischaemic tolerance. An international multicenter study is now under way, which will hopefully provide more clinical data.

Many of the observed effects of intra-operative hypothermia may be explained by our current knowledge, except the very interesting finding of increased arteriovenous lactate difference. This somewhat

contrary to the beneficial effect that has been observed in many experimental studies and clinical case reports. Therefore, the clinical significance is unclear.

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