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Spinal epidural oxygen partial pressure and evoked spinal cord potential in relation to the severity of spinal ischemia during cross-clamping of the thoracic aorta.

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Abstract

Experiments were undertaken to determine the relationship between evoked spinal cord potential (ESP) and the partial pressure of oxygen in tissue in the epidural space (E-pO2) during aortic clamping. Eighteen adult mongrel dogs were studied as follows. In group I (n = 6), the descending thoracic aorta was clamped partially at the proximal site for 15 min to maintain the distal arterial pressure at 60, 40, and 20 mmHg consecutively at 15 min intervals. In group II (n = 6), the descending thoracic aorta was clamped proximally for 30 min. In group III (n = 6), the descending thoracic aorta was cross-clamped at proximal and distal sites for 30 min. Postoperative complete paraplegia was observed in 4 of 6 dogs in group III, but none in group II. The change in ESP with aorta cross-clamping was very mild in groups I and II. Transient increases and decreases in the ESP amplitude were observed in group III. The decrease of E-pO2 correlated well with the distal arterial pressure, and a rapid return to baseline of the E-pO2 was observed after declamping. The E-pO2 changed in response to spinal ischemia more rapidly than did ESP in all groups. The critical level of E-pO2 was 50 mmHg or a 40% decrease from baseline. Because the ESP reflects spinal function and the E-pO2 reflects spinal blood pressure, we propose that combined recording of ESP and E-pO2 would improve spinal monitoring during thoracic aortic surgery.

KEYWORDS: thoracic aortic surgery, spinal cord monitoring, paraplegia, evoked spinal cord potential, partial pressure of oxygen

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Spinal Epidural Oxygen Partial Pressure and Evoked Spinal Cord Potential in Relation to the Severity of Spinal Ischemia during Cross-Clamping of the Thoracic Aorta

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Experiments were undertaken to determine the relationship between evoked spinal cord potential (ESP) and the partial pressure of oxygen in tissue in the epidural space (E-pO_2) during aortic clamping. Eighteen adult mongrel dogs were studied as follows. In group I (n = 6), the descending thoracic aorta was clamped partially at the proximal site for 15 min to maintain the distal arterial pressure at 60, 40, and 20 mmHg consecutively at 15 min intervals. In group II (n = 6), the descending thoracic aorta was clamped proximally for 30 min. In group III (n = 6), the descending thoracic aorta was cross-clamped at proximal and distal sites for 30 min. Postoperative complete paraplegia was observed in 4 of 6 dogs in group III, but none in group II. The change in ESP with aorta cross-clamping was very mild in groups I and II. Transient increases and decreases in the ESP amplitude were observed in group III. The decrease of E-pO_2 correlated well with the distal arterial pressure, and a rapid return to baseline of the E-pO_2 was observed after declamping. The E-pO_2 changed in response to spinal ischemia more rapidly than did ESP in all groups. The critical level of E-pO_2 was 50 mmHg or a 40 % decrease from baseline. Because the ESP reflects spinal function and the E-pO_2 reflects spinal blood pressure, we propose that combined recording of ESP and E-pO_2 would improve spinal monitoring during thoracic aortic surgery.

Key words : **thoracic aortic surgery, spinal cord monitoring, paraplegia, evoked spinal cord potential, partial pressure of oxygen**

Postoperative paraplegia is a devastating sequela after cross-clamping of the descending thoracic aorta. Numerous interventions designed to prevent spinal cord ischemia have been attempted in animal models (1-3). Somatosensory cortical evoked potential (SSEP) monitoring has been regarded as a simple, non-invasive method by which spinal cord ischemia could be monitored during cross-clamping of the thoracic aorta (4, 5). Evoked spinal cord potential (ESP) monitoring has recently been used clinically because it is stable, highly reproducible and affected little by anesthesia (6). However, several limitations were described for SSEP and ESP monitoring (7, 8), and the need for a new monitoring system for spinal ischemia which is more sensitive than SSEP or ESP has been emphasized. We describe measurements of epidural pO_2 in dogs (E-pO_2) during aortic clamping from the ischemic stand point. This study had three purposes: (a) to determine if the E-pO_2 correlates with the spinal arterial pressure, (b) to determine the relationship between ESP and E-pO_2 during aortic clamping, and (c) if the E-pO_2 would be a more sensitive indicator of spinal ischemia during aortic cross-clamping.

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Subjects and Methods

Eighteen adult mongrel dogs, weighing 8 to 15 kg (mean 9.2 kg), were sedated with ketamine hydrochloride (10 mg/kg; i.m.) and atropine sulfate (0.25 mg; i.m.). After an intravenous line was established and Ringer's lactate solution supplemented with sorbitol

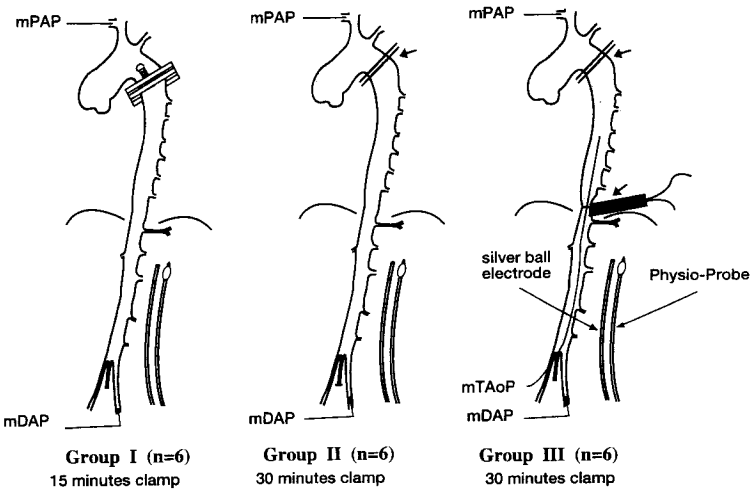

Fig. 1 Schematic diagram of the experimental method. In group I (n = 6), the descending thoracic aorta was partially clamped at the proximal site for 15 min to maintain the distal arterial pressure at 60, 40 and 20 mmHg. In group II (n = 6), the descending thoracic aorta was clamped at the proximal site for 30 min, and in group III (n = 6), the descending thoracic aorta was cross-clamped for 30 min at two sites.

was infused (10–15 mg/kg/h), tracheal intubation was performed. Anesthesia with oxygen and halothane was carried out and ventilation was controlled at a tidal volume of 20 ml/kg and a respiratory rate of 16/min. The muscle relaxant, pancuronium bromide, was injected intravenously at a total dose of 2–4 mg.

The thoracic cavity was opened through the fifth and eleventh intercostal spaces to expose the left subclavian artery and the descending thoracic aorta. After heparin sulfate (100 u/kg) was injected, the aorta was clamped as depicted in Fig. 1. In group I, the descending thoracic aorta was clamped partially with a screw clamp at a proximal site, *i.e.*, immediately distal to the left subclavian artery. First, the aorta was clamped partially for 15 min to maintain the distal arterial pressure at 60 mmHg. The clamp was released and 15 min later the clamp was applied again for 15 min to maintain the distal arterial pressure at 40 mmHg. After another interval of 15 min after the clamp was released, the aorta was clamped to maintain the distal arterial pressure at 20 mmHg for another 15 min. In group II, the descending thoracic aorta was cross-clamped at only one proximal site for 30 min. In group III, the descending thoracic aorta was clamped at two sites, immediately distal to the left subclavian artery and immediately proximal to the diaphragm. The left subclavian artery remained unclamped. The total clamped time was 30 min.

Evoked spinal cord potential (ESP) measurement. To monitor intraoperatively spinal cord function, ESPs were measured. First, the dog was placed in the prone position. A bipolar silver ball electrode was inserted into the lower lumbar epidural space by the hanging drop method. Following thoracotomy, a needle electrode was placed into the T5–T6 intervertebral disc space. Another needle electrode of the same type was placed into the subcutaneous space as the reference electrode. Stimuli were given by the silver ball electrode in the epidural space at an amplitude of 10 mA, pulse width of 0.2 ms and frequency of 7 Hz. The responses sensed by the chest needle electrode were accumulated 20 times. Stimuli were applied and recordings of ESP were made using the Neuromatic 2000 C (DANTEC Denmark).

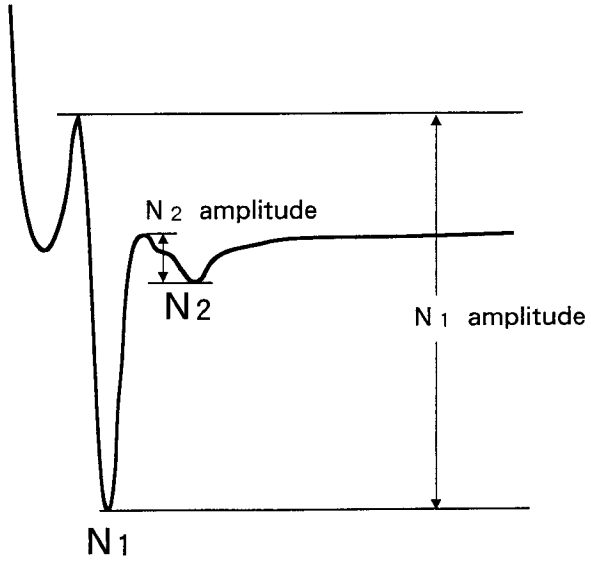

Fig. 2 ESP baseline; composed of two spike potentials. (N_1, N_2)

Before clamping the aorta, ESP were recorded to confirm reproducibility and to obtain control measurements. The ESP (recorded in each animal) was composed of two spike potentials, the first (N_1) and the second spike (N_2). As indicated in Fig. 2, N_1 and N_2 amplitudes were measured from the peak to the baseline. The ESP was recorded every 5 min after aortic clamping and again

after declamping. The changes of the ESP were evaluated as the percent change of the control wave amplitude for both the first negative (N_1) and the second negative components (N_2).

Measurement of the partial pressure of oxygen in the epidural space. The partial pressure of oxygen in the epidural space (E-pO_2) was measured with a mass spectrometer (Medspect: Chemetron, USA) (9, 10). A Physio-probe for tissue gas analysis was inserted through a lower lumbar intervertebral space into the epidural space (diaphragm level) in a manner similar to that used for the silver ball electrode. The probe for tissue gas analysis is designed to absorb a trace amount of gas dissolved in the tissue through the diffusion membrane. The partial pressure of oxygen was measured every 5 min after aortic clamping and every 5 min after declamping.

Body temperature was continuously measured with a needle thermometer placed into the muscle of the leg during the operation and was kept relatively constant between 34.5–37.0°C. Plastic catheters were placed in the right brachial and left femoral arteries to monitor the mean proximal arterial pressure (mPAP) and mean distal arterial pressure (mDAP). In addition, a catheter was inserted through the right femoral artery into the thoracic aorta to measure the mean blood pressure in the clamped thoracic aorta (mTAoP) in group III. Arterial blood gas analysis was performed before and after clamping and post-clamping metabolic acidosis was corrected with $NaHCO_3$.

After the clamp was released, each dog in groups II and III recovered from anesthesia and were followed postoperatively for 48 h to monitor the development of neurologic deficits. Dogs of group I did not recover from anesthesia. The paralysis was graded according to Fried's grading system (11): Grade 0, normal motor function; Grade 1, minimal motor disability, abnormal reflexes in which the hind legs could still be used in climbing; Grade 2, moderate paraparesis in which standing was accomplished with the support of the forelegs; Grade 3, marked paraparesis in which the hind legs could move against gravity but could not be used for support,; Grade 4, paraplegia in which there was minimum or no voluntary movement of the hind legs.

The measurements were expressed as the mean ± standard deviation. Significant differences were tested using a Student's t-test. And the postoperative paraplegis was evaluated by χ^2 test.

Results

Changes in mean blood pressure during clamping and after unclamping the thoracic aorta. In group I, partial clamping of the thoracic aorta maintained the mDAP 62.8 ± 8.5 mmHg, 39.0 ± 4.9 mmHg, or 20.0 ± 2.6 mmHg at 15 min after clamping (Fig. 3). The mDAP in groups II and III changed from aortic clamping to 30 min after declamping as shown in Figs. 4 and 5. The preclamping mDAP was 111.2 ± 17.8 mmHg in group II and 107.9 ± 19.7 mmHg in group III. After 15 min of aortic clamping, the mPAP increased to 136.5 ± 23.0 mmHg in

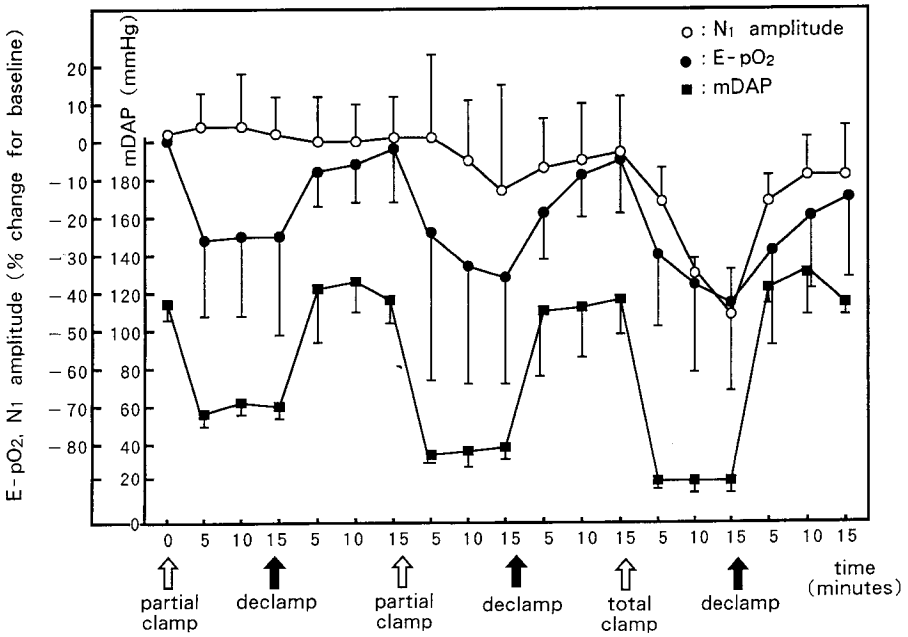

Fig. 3 Changes in the mDAP, ESP (N_1) amplitude and E-pO_2 during and after cross-clamping in group I (n = 6). The E-pO_2 decreased in parallel to the stepwise decrements of mDAP. Although there was little change in the ESP while the mDAP was 60 or 40 mmHg, the changes became severe when the mDAP was 20 mmHg.

group II and 139.7 ± 9.7 mmHg in group III. The mDAP decreased within 15 min after clamping to 23.0 ± 3.5 mmHg in group II and to 10.8 ± 3.6 mmHg in group III, which was significantly lower than that in group II ($p < 0.01$). At that time, the mTAoP was 83.0 ± 12.3 mmHg in group III. The mTAoP in group III was

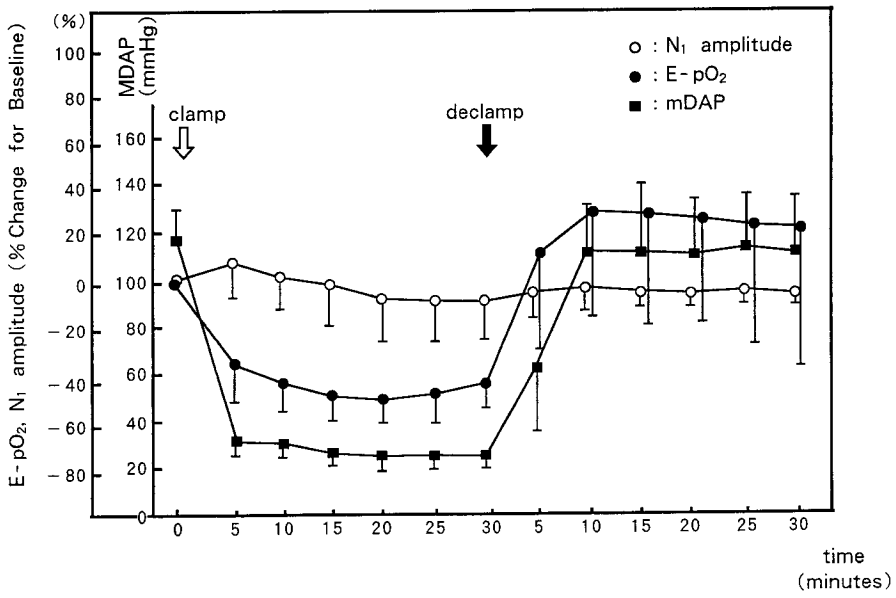

Fig. 4 Changes in the mDAP, ESP (N_1) amplitude and E-pO_2 during and after cross-clamping in group II (n = 6). The E-pO_2 decreased markedly after clamping and increased rapidly after clamp release. However, the ESP N_1 amplitude remained nearly unchanged.

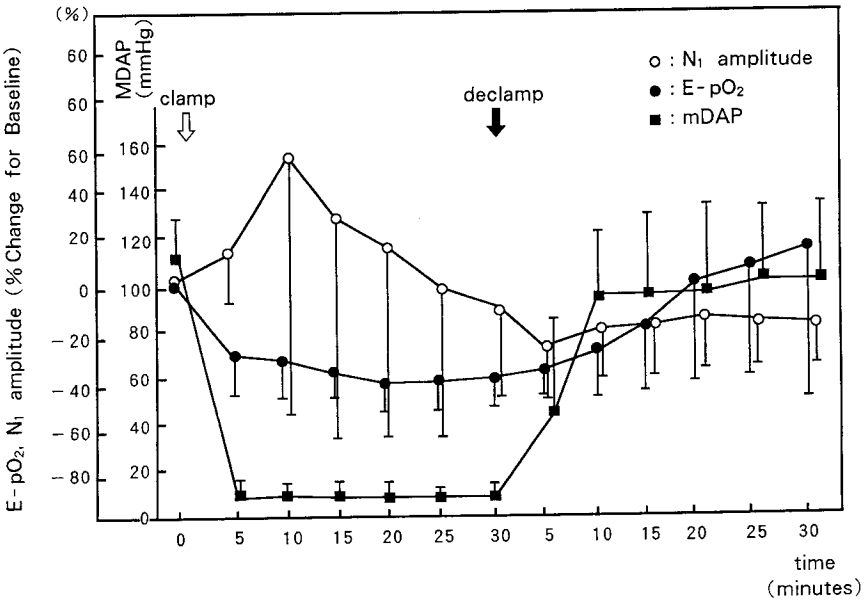

Fig. 5 Changes in the mDAP, ESP (N_1) amplitude and E-pO_2 during and after cross-clamping in group III (n = 6). The N_1 amplitude increased transiently in 4 of 6 cases. The E-pO_2 decreased markedly immediately after clamping and increased more slowly than in group II after clamp release.

approximately one half of the mPAP and there was no obvious correlation between the mTAoP and postoperative paraplegia. Fifteen min after the clamp was released, the mDAP returned to 111.3 ± 21.1 mmHg in group II and 97.8 mmHg ± 24.0 mmHg in group III (Table 1).

Changes in ESP (N_1) during and after cross-clamping of the thoracic aorta. Changes in ESP (N_1) amplitude were evident in each group relative to the pre-clamping value. In group I, although there was little change in the ESP while the mDAP was 60 mmHg (130.4 ± 57.6 μV to 132.8 ± 53.1 μV; 102.8 ± 10.5 % of the baseline at 15 min. after clamping) or 40 mmHg (to 108.1 ± 50.0 μV; 88.0 ± 27.3 %, the change became severe when the mDAP was 20 mmHg (to 63.0 ± 30.4 μV; 54.1 ± 13.1 %). In group II, the N_1 amplitude changed very little and remained almost constant, changing from 120.5 ± 46.7 μV at baseline to 112.1 ± 37.7 μV; 99.5 ± 20.8 % at 15 min after clamping. It remained at 106.9 ± 36.9 μV (92.3 ± 11.9 %) at 30 min after clamping and changed little within 30 min of clamp release 112.5 ± 45.2 μV; 92.7 ± 5.6 %. In group III, the N_1 amplitude was augmented at 5 min after clamping in 4 of 6 dogs (67 %). It was 109.4 ± 62.4 μV before clamping, which increased to 118.4 ± 61.3 μV; 109.3 ± 25.8 % at 5 min ($p < 0.05$), and 118.6 ± 49.9 μV; 153.9 ± 113.4 % at 10 min. It decreased in parallel to the stepwise decrease of the distal blood pressure to 60, 40 and 20 mmHg, respectively. When the mDAP was 60 mmHg, the E-pO_2 decreased to 65.7 ± 27.8 mmHg, 74.8 ± 26.7 % of baseline at 15 min after partial clamping. When mDAP was 40 mmHg, it decreased 54.5 ± 23.5 mmHg, 63.7 ± 27.0 %, and when mDAP was 20 mmHg, it decreased 46.3 ± 13.2 mmHg, 58.3 ± 24.5 %. The E-pO_2 was 82.1 ± 25.7 mmHg before clamping in group II, and it was 93.4 ± 23.5 mmHg in group III. It decreased markedly within 5 min after clamping to 67.7 ± 16.4 % in group II and 71.5 ± 19.6 % in group III. After that, the E-pO_2 decreased gradually in both groups to 56.2 ± 17.8 % in group II and 56.8 ± 16.3 % in group III at 30 min after clamping. After the clamp was released, the E-pO_2 recovered at a relatively rapid rate in group II and more gradually in group III (at 10 min after clamp release, 126.5 ± 47.7 % in group II and 70.0 ± 23.4 % in group III; $p < 0.05$). In 4 dogs in group II and 2 dogs in group III, the E-pO_2 increased above the baseline after clamp release (Table 1, Figs. 3–5).

Incidence of postoperative paraplegia. In group II, no dog had paraplegia at 48 h after the operation, although transient paresis (Grade 1) was observed in 2 dogs. Four dogs (67 %) in group III suffered from complete para-

Table 1 Changes in the mDAP, ESP(N_1), E-pO_2 during and after cross-clamping in groups I–III

			Pre-clamp	After clamp		After declamp	
				15 min	30 min	15 min	30 min
Group I							
(1st clamp)	mDAP	(mmHg)	123.3 ± 8.1	62.8 ± 8.5		119.2 ± 12.3	
	ESP(N_1)	(μV)	130.4 ± 57.6	132.8 ± 53.1		131.3 ± 53.7	
	E-pO_2	(mmHg)	87.0 ± 23.7	65.7 ± 27.8		91.0 ± 33.2	
(2nd clamp)	mDAP	(mmHg)	119.2 ± 12.3	39.0 ± 4.9		116.3 ± 17.1	
	ESP(N_1)	(μV)	131.3 ± 53.7	108.1 ± 50.0		124.6 ± 47.7	
	E-pO_2	(mmHg)	91.0 ± 33.2	54.5 ± 23.5		85.7 ± 30.5	
(3rd clamp)	mDAP	(mmHg)	116.3 ± 17.1	20.0 ± 2.6		118.3 ± 0.9	
	ESP(N_1)	(μV)	124.6 ± 47.7	63.0 ± 30.4		112.0 ± 46.1	
	E-pO_2	(mmHg)	85.7 ± 30.5	46.3 ± 13.2		77.3 ± 6.9	
Group II	mDAP	(mmHg)	111.2 ± 17.8	23.0 ± 3.5	24.1 ± 4.2	111.3 ± 21.1	120.1 ± 18.8
	ESP(N_1)	(μV)	120.5 ± 46.7	112.1 ± 37.7	106.9 ± 36.9	110.9 ± 43.3	112.5 ± 45.2
	E-pO_2	(mmHg)	82.1 ± 25.7	41.7 ± 10.9	41.5 ± 15.2	101.7 ± 41.0	100.3 ± 43.7
Group III	mDAP	(mmHg)	107.9 ± 19.7	10.8 ± 3.6*	11.1 ± 3.5	97.8 ± 24.0	110.2 ± 20.5
	ESP(N_2)	(μV)	109.4 ± 62.4	94.8 ± 60.0	87.5 ± 75.6	94.8 ± 65.2	97.0 ± 66.8
	E-pO_2	(mmHg)	93.4 ± 23.5	63.7 ± 44.7	57.9 ± 40.7	90.7 ± 51.2	108.7 ± 68.6

mDAP: mean distal arterial pressure
ESP: evoked spinal cord potential; E-pO_2: epidural pO_2
*$p < 0.01$ (to Group II)

Table 2 The times taken for N_2 component of ESP to disappear

Group	Condition	Required time (min) Dog number 1	2	3	4	5	6
I*	$T_{1/2}$	—	—	5	—	5	—
	T_0	—	—	5	—	5	—
II	$T_{1/2}$	—	—	25	—	Unknown	—
	T_0	—	—	30	—	Unknown	—
III	$T_{1/2}$	—	10	5	—	5	5
	T_0	—	20	5	—	30	25

The time taken for the N_2 amplitude to decrease to 50 % or less of the baseline ($T_{1/2}$) and to disappear completely (T_0). —; N_2 component neither diminished nor disappeared.
*N_2 disappeared when mean DAP was 20 mmHg.

Table 3 Incidence of postoperative paraplegia

Group	Postoperative time (h)	Paralysis grade[a] Dog number 1	2	3	4	5	6
II	24	1	0	1	0	0	0
	48	0	0	0	0	0	0
III	24	0	4	4	0	4	4
	48	0	4	4	0	4	4

a: Paralysis was graded according to Fried's grading system (described in text).

plegia (grade 4) and the other two showed no paralysis (Grade 0) (Talbe 3). There was a significant difference in the occurrence of postoperative paraplegia at 48 h after the operation between groups II and III according to the χ^2 test ($p < 0.05$).

Discussion

Spinal cord injury after cross-clamping of the thoracic aorta is due to decreasing the spinal oxygen supply. In our experiments, we proposed that monitoring of spinal oxygenation would be more useful during aortic surgery than the conventional monitoring, and studied relationship between the E-pO_2 and ESP.

For measuring the spinal oxygenation, Wadouh *et al.* (12) studied direct measurement of oxygen tension on the spinal cord surface of pigs. Placement of a needle electrode into the spinal cord itself is clinically impractical because of the risk of causing spinal cord damage by the needle. Thus, the partial pressure of oxygen in the spinal fluid or in the epidural space were selected as the new parameters which may vary with the spinal circulation pressure (13–14). It is a much safer technique to insert a probe into the epidural space, particularly in dogs. Therefore, the latter parameter was chosen for the study in the present series of experiments.

We attemped to make a mild spinal ischemic model by partial clamping of thoracic aorta in group I, a moderate spinal ischemic model in group II, and a severe ischemic model in group III, and studied the relationship between the ESP and E-pO_2 in each group. It was thought ideal to clamp the aorta for 30 min in group I. However, based on the study that 15 min of clamping did not cause spinal necrosis (15), we used group I for the acute study mainly to investigate the correlation between mDAP and the E-pO_2. A mass spectrometer, Medspect (9,10) was used in the present experiments to measure E-pO_2 continuously and is, in general, useful for monitoring the microcirculation dynamically.

In our experiments, the E-pO_2 changes correlated closely with the mDAP and was a more sensitive indicator of spinal ischemia than any changes in the ESP. If one assumes that the distal clamping site was immediately proximal to Adamkiewicz's arteries (16) (Fig. 1), spinal artery pressure around the diaphragm correlated well with the mDAP. Although critical spinal arterial pressure is generally believed to be 50 mmHg (17), the level of ischemia seen at 50 mmHg does not always cause ESP changes. When the mDAP was 40 mmHg, the E-pO_2 was 54.5 ± 23.5 mmHg, 63.7 ± 27.0 % of baseline. Therefore, in our study the critical level of E-pO_2 was approximately 50 mmHg or 60 % of the baseline.

ESP is considered to be an effective tool for monitoring spinal ischemia in the operating room, which commonly has various sources of electronic interference, because ESP is affected minimally by anesthesia, and is highly reproducible and stable. It was reported to correlate well with the presence or absence of postoperative paraplegia (8). A 50 % change of the initial N_1 amplitude has been suggested as criteria for irreversible spinal cord ischemia and correlates well with postoperative paraplegia (18). In our experience, a transient increase in N_1 amplitude occurred in 4 of the 6 dogs in group III. A transient increase in the ESP N_1 amplitude is believed to arise from a lowering of the threshold of neurons as ischemia increases the number of excited neurons. Thus, this

change is the earliest important abnormal finding to occur when the ischemic state is relatively mild, in that conductivity has not yet deteriorated in the posterior and lateral funiculi.

However, once the ESP change is seen, the spinal cord injury may sometimes be irreversible. In our experience, the ESP (N_1) changed very little when the mDAP was around 25 mmHg, and changed significantly when the mDAP reached below 20 mmHg. Wadouh (12) showed that an arterial pressure of 20 mmHg in the aorta does not provide a sufficient blood supply to the spinal cord, with or without spinal fluid drainage. Therefore, it may be too late to prevent the spinal injury when the ESP changes. If the E-pO_2 was monitored intraoperatively, it would be possible to identify the important intercostal arteries which should be reconstructed. When the sudden decrease in E-pO_2 is observed during aortic surgery, even if the ESP is stable, the important intercostal arteries may be clamped and spinal arterial pressure may be critical. At such time, perfusion of the clamped intercostal arteries could mitigate the risk of paraplegia. Furthermore, it is important to reattach the intercostal arteries to the new graft. Svensson *et al.* (19) reported 34 patients in whom intrathecal papaverine was used and intercostal and lumbal arteries were not reanastomosed to the new aortic graft. Although no immediate postoperative paraplegia occurred, 2 patients experienced delayed paraparesis.

After declamping the aorta, the ESP and E-pO_2 recovered gradually. The return of the E-pO_2 levels to baseline would indicate that reconstruction of spinal arteries is successful, *i.e.*, arterial blood supply to the spinal cord is restored. However, it may not predict motor function recovery. A rapid hyperperfusion of the spinal artery may cause a reperfusion injury or spinal cord edema. The metabolic products of the ischemic tissue, particulary superoxides, were reported to be associated with paraplegia after aortic cross-clamping. In our experiments, hyperperfusion after declamping was suspected in 6 of 12 dogs of groups II and III.

We conclude that combined monitoring of the ESP and the E-pO_2 would be preferred method to use during thoracic aortic surgery. Although the change of the ESP correlates closely post-operative paraplegia, it occurs too late to prevent paraplegia. The E-pO_2 does not correlate closely post-operative paraplegia, but reflects spinal blood pressure. We recommend that the E-pO_2 should be kept relatively constant above 60 mmHg, and that the ESP should be kept constant by using the adequate adjunct and reattachment of intercostal arteries.

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