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Abstract

A number of approaches have been put forward to monitor spinal cord ischemia during thoracic and thoracoabdominal aortic occlusion. However, none of these can ultimately prevent devastating complications which result from ischemic spinal cord injury. A direct measurement of the oxygen content of the spinal cord may accurately indicate the perfusion state, but in practice it is impractical. We surmised that intrathecal and/ or epidural oxygen concentration(I-pO_2 and E-pO_2, respectively) accurately reflect oxygen content in the spinal cord. So, we examined whether or not I-pO_2 and/or E-pO_2 correlated with the spinal cord pO_2 (S-pO_2) in dogs. In nine mongrel dogs, a model of graded spinal cord ischemia was developed by stepwise alternation of the level of aortic occlusion with an intraaortic balloon catheter. I-pO_2, E-pO_2 and S-pO_2 were measured with a mass spectrometer. Our results show that, both I-pO_2 and E-pO_2 significantly correlated with S-pO_2. I-pO_2 correlated with S-pO_2 better than E-pO_2 did. Therefore, I-pO_2 can be used as a new indicator for spinal cord ischemia, and I-pO_2 monitoring would be useful to prevent paraplegia associated with thoracic aortic surgery.

KEYWORDS: spinal cord ischemia, thoracic and thoracoabdominal aortic surgery, intrathecal pO_2

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Intrathecal Oxygen Concentration as a New Indicator of Spinal Cord Ischemia

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A number of approaches have been put forward to monitor spinal cord ischemia during thoracic and thoracoabdominal aortic occlusion. However, none of these can ultimately prevent devastating complications which result from ischemic spinal cord injury. A direct measurement of the oxygen content of the spinal cord may accurately indicate the perfusion state, but in practice it is impractical. We surmised that intrathecal and/or epidural oxygen concentration(I-pO_2 and E-pO_2, respectively) accurately reflect oxygen content in the spinal cord. So, we examined whether or not I-pO_2 and/or E-pO_2 correlated with the spinal cord pO_2 (S-pO_2) in dogs. In nine mongrel dogs, a model of graded spinal cord ischemia was developed by stepwise alternation of the level of aortic occlusion with an intraaortic balloon catheter. I-pO_2, E-pO_2 and S-pO_2 were measured with a mass spectrometer. Our results show that, both I-pO_2 and E-pO_2 significantly correlated with S-pO_2. I-pO_2 correlated with S-pO_2 better than E-pO_2 did. Therefore, I-pO_2 can be used as a new indicator for spinal cord ischemia, and I-pO_2 monitoring would be useful to prevent paraplegia associated with thoracic aortic surgery.

Key words: spinal cord ischemia, thoracic and thoracoabdominal aortic surgery, intrathecal pO_2

The incidence of paraplegia after thoracic and/or thoracoabdominal aortic surgery has been reported to be 16 % or more (1, 2). The catastrophic results of this complication led to the development of a variety of new methods to prevent spinal cord ischemia, including distal aortic perfusion, hypothermia, and pharmacotherapy (3–5). In addition, neurophysiologic monitoring methods, such as spinal motor evoked potentials (MEP) (6, 7), somatosensory evoked potentials (SSEP) (8, 9), and evoked spinal potentials (ESP) (10) have been used to monitor spinal cord function. Unfortunately, none of these have proven effective enough to eradicate the incidence of paraplegia. The most important problem of these neurophysiologic monitoring methods is that changes in these evoked potentials are only visible after a significant ischemic injury of the spinal cord, *i.e.*, after the damage has been done. Therefore, we wanted to develop a new method to monitor the perfusion state of the spinal cord. The essential cause of spinal cord ischemia is the lack of the oxygen supply following a decrease in blood flow in the spinal cord. It is most reliable to directly measure the oxygen tension (pO_2) in the spinal cord (S-pO_2). However, it is clinically impossible to place a pO_2 probe in the parenchyma of the spinal cord itself. On the other hand, it is possible to measure pO_2 in the intrathecal and epidural spaces (I-pO_2 and E-pO_2, respectively). The purposes of this study are to reveal the changes in S-pO_2, I-pO_2 and E-pO_2 in conjunction with changes in ESP, and to examine whether or not I-pO_2 and/or E-pO_2 would correctly reflect S-pO_2.

Materials and Methods

Animal preparations. Nine mongrel dogs, weighing from 8 to 12 kg, were anesthetized initially by ketamine hydrochloride (10 mg/kg, intramuscularly (i.m.)) with atropine sulfate (0.25 mg, i.m.). A venous line was established to infuse lactate Ringer solution supplemented with 5 % sorbitol (10 to 15 ml/kg/h). After endotracheal intubation, animals were sufficiently anesthetized and mechanically ventilated (oxygen: 4 l/min, halothane: 0.5 vol %, a tidal volume: 20 ml/kg, respiratory rate: 16 times/min.). A Bauman balloon catheter (French size 7)

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was advanced into the abdominal aorta via the left common carotid artery for aortic occlusion (Fig. 1). To confirm the position of the occlusion balloon, a small laparotomy was made through the left lateral abdominal wall. The celiac and superior mesenteric arteries were ligated to eliminate collateral blood influx into the aorta.

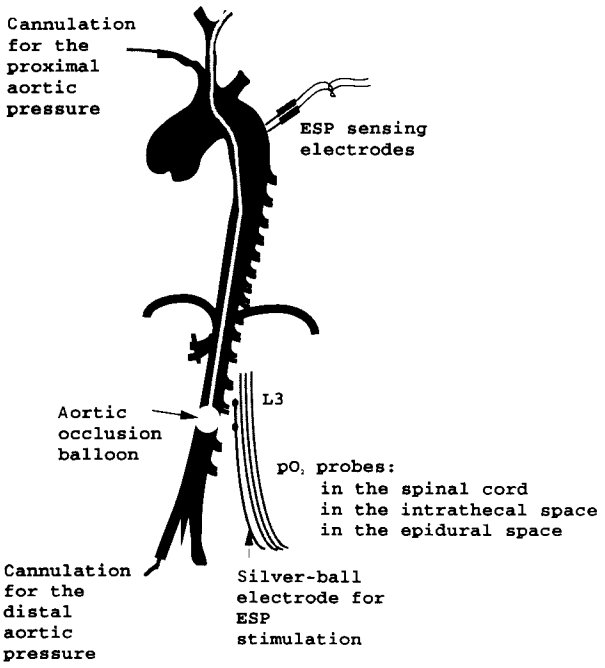

Fig. 1 Schematic diagram of the experimental preparations. Three oxygen tension (pO_2) probes were placed to measure spinal cord oxygen concentration (S-pO_2), intrathecal oxygen concentration (I-pO_2), and epidural oxygen concentration (E-pO_2) at the level of the 3rd lumbar vertebra (L3). A silver-ball electrode was placed in the epidural space for the electric stimulation for evoked spinal potentials (ESP), the sensor of which was placed in the epidural space between the 4th and 5th thoracic vertebrae. An aortic occlusion balloon was initially placed in the lower abdominal aorta.

The right brachial and femoral arteries were cannulated to represent the aortic pressures at the proximal and distal points of the occlusion balloon, respectively.

Measurement of S-pO_2, I-pO_2 and E-pO_2. With animals laying on their right sides, laminectomy was performed at the level of the 4th lumbar vertebra. After the dura mater was opened, a pO_2 probe (Physio-probe®) was placed in the center of the parenchyma of the spinal cord at the 3rd lumbar vertebra (L3) level to measure S-pO_2 (Figs. 1 and 2). The 2nd pO_2 probe was fixed in the intrathecal space at the L3 level, nearby to the anterior spinal artery to measure I-pO_2. The 3rd pO_2 probe was placed in the epidural space at the L3 level to measure E-pO_2 (Figs. 1 and 2). The outer diameter of the pO_2 probes used was 0.64 mm and the length of the effective diffusion membrane was 25.4 mm. PO_2 measurements were made with a medical mass spectrometer (Medspect II, Chemetron, MO, USA).

Measurement of evoked spinal cord potential (ESP). As an indicator of spinal cord function, ESP was measured with a Neuromatic 2000C (DISA, Denmark) as follows (Figs. 1 and 2): A bipolar, silver-ball electrode was inserted through the site of laminectomy and fixed at the L3 level for electrical stimulation. The ESP-sensing electrode was placed between the 4th and 5th thoracic vertebrae in the epidural space using the hanging-drop method. Electrical stimulation was given with an amplitude of 10 mA, a pulse-width of 0.1 ms, and a frequency of 7 Hz. The responses were evaluated by measuring amplitudes of the 1st negative (N1) and the 2nd negative (N2) components (16), averaged over 20 stimulations, and expressed as a percentage of each control amplitude determined before aortic occlusion.

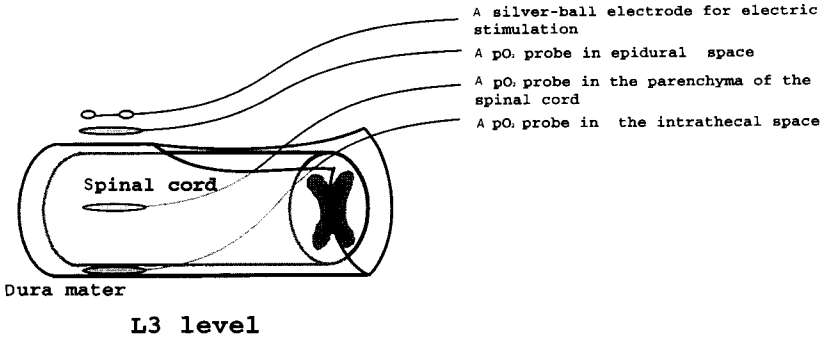

Fig. 2 Placement of the pO_2 probes and the electric stimulation electrode for ESP. After opening the dura mater, three pO_2 probes and an electric stimulation electrode for ESP were placed at the level of the 3rd lumbar vertebra (L3). ESP: See Fig. 1.

Induction of graded spinal cord ischemia. After determining the baseline value of all pO_2 and ESP, the aortic occlusion balloon was inflated. The level of ischemia of the spinal cord was elevated incrementally by drawing the occlusion balloon in the cephalad direction to the next proximal pair of lumbar or intercostal arteries. After equilibration for 6 min at each occlusion level, S-pO_2, I-pO_2, E-pO_2 and ESP were measured. The pO_2 values were corrected in accordance with the paraspinal temperature, which was measured in the paravertebral muscle with an electric needle thermometer, kept higher than 34 °C using an electric warming blanket.

Reperfusion. After the aortic occlusion balloon reached the level of the 8th thoracic vertebra (Th8), it was deflated, and the changes in all pO_2 and ESP were recorded.

Statistical analysis. Data are expressed as means ± SD.

Statistical analysis was done using a Student's *t*-test to compare the variables between groups and a simple linear regression analysis (Statview J 4. 0. 2, Abacus Concepts Inc., USA) to examine the correlation between two variables. The difference between correlation coefficients (r) was also calculated after z-transformation of r-values. A P value < 0.05 was considered statistically significant.

All animals received humane care in accordance with the Principles of Laboratory Animal Care formulated by the National Society of Medical Research and the Guide for the Care and Use of Laboratory Animals prepared by the National Academy of Sciences and published by the National Institute of Health (NIH Publication no. 80-23, revised in 1978).

Results

Changes in aortic blood pressure. The proximal and distal aortic pressures before aortic occlusion were 104.4 ± 23.3 and 97.6 ± 24.9 mmHg, respectively; there was no significant difference (Fig. 3). Immediately after occlusion of the aorta at the 5th lumbar vertebra (L5) level, the distal aortic pressure significantly decreased to 38.8 ± 23.4 mmHg ($P < 0.01$). As the occlusion balloon was pulled in the cephalad direction, distal pressure tended to decrease further and proximal pressure tended to gradually increase (Fig. 3). Throughout the time when the balloon was inflated, distal aortic pressure was always significantly lower than the proximal pressure ($P < 0.01$ at every level). Soon after deflation of the balloon, both pressures returned to each baseline, and the pressure difference disappeared.

Changes in S-pO_2, I-pO_2 and E-pO_2. Be-

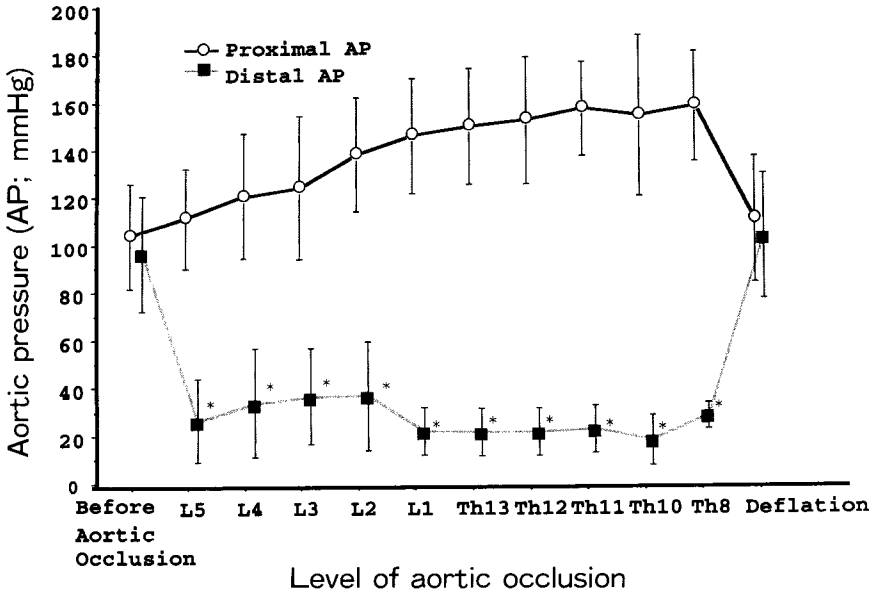

Fig. 3 Changes in proximal and distal aortic blood pressures. L5 and Th13 indicate the 5th lumbar vertebra and the 13th thoracic vertebra, respectively. Similar abbreviations indicate the vertebrae in the same way.
*$P < 0.01$ as compared to not only the baseline value but also the corresponding proximal aortic pressure.

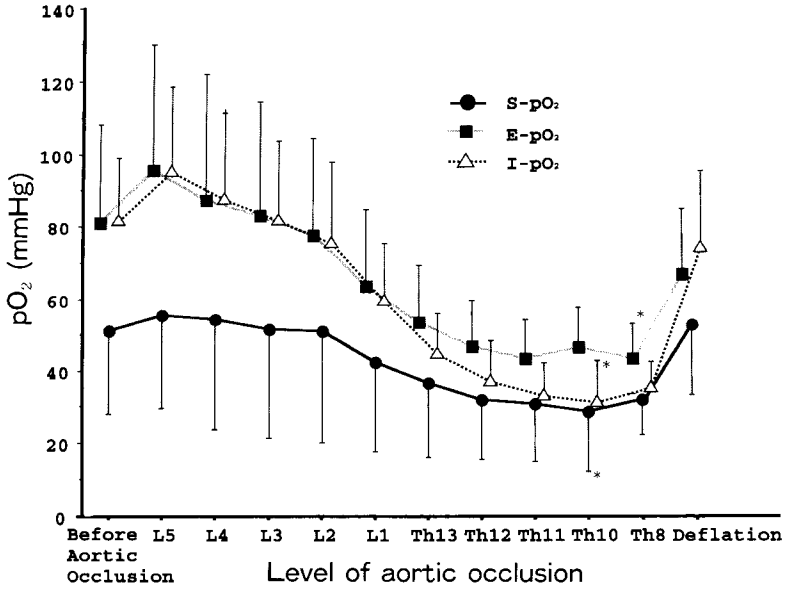

Fig. 4 Changes of pO_2 in relation to aortic occlusion level. There was a trend for S-pO_2 to always show the lowest value with little change. This was not statistically significant. Abbreviations are the same as in Fig. 1 and Fig. 3. *The lowest value of each pO_2; $P < 0.01$ as compared to each control value.

Table 1 Correlation of E-pO_2 and I-pO_2 to S-pO_2

Dog No.	E-pO_2		I-pO_2	
	r	P	r	P
1	0.875	< 0.01	0.947	< 0.01
2*	0.700	< 0.05	0.787	< 0.01
3	0.906	< 0.01	0.992**	< 0.01
4	0.969	< 0.01	0.984	< 0.01
5	0.768	< 0.05	0.920	< 0.01
6	0.937	< 0.01	0.932	< 0.01
7	0.837	< 0.01	0.900	< 0.01
8	0.794	< 0.01	0.949	< 0.01
9	0.837	< 0.01	0.930	< 0.01

*; Subarachnoid hemorrhage accidentally occurred.
**; $P < 0.05$ as compared to the r-value of E-pO_2.
E-pO_2, I-pO_2 and S-pO_2 indicate oxygen concentration (pO_2) in the epidural and intrathecal spaces and the spinal cord parenchyma, respectively.
r, correlation coefficient

fore aortic occlusion, S-pO_2, I-pO_2 and E-pO_2 were 50.9 ± 22.9, 81.3 ± 18.5 and 81.1 ± 26.9 mmHg, respectively (Fig. 4). It was observed that all pO_2 decreased as the occlusion catheter was drawn in the cephalad direction. The lowest values of S-pO_2, I-pO_2 and E-pO_2 were 28.3 ± 16.3 mmHg at the occlusion level of Th10, 30.8 ± 11.6 mmHg at Th10, and 42.8 ± 9.7 mmHg at Th8, respectively, representing a significant difference from each baseline value ($P < 0.01$ for each). After deflation of the balloon, all pO_2 returned to baseline.

Correlation of I-pO_2 and E-pO_2 to S-pO_2. To evaluate the correlation of I-pO_2 and E-pO_2 to S-pO_2, a linear regression analysis was done, and the r values of I-pO_2 and E-pO_2 to S-pO_2 in each dog are presented in tabular form (Table 1). In dog 2, which showed the lowest r-value not only for I-pO_2 but also for E-pO_2, a subarachnoid hemorrhage accidentally occurred. It was observed that not only I-pO_2 but also E-pO_2 significantly correlated to S-pO_2 in all dogs. The r-values of I-pO_2 were higher than those of E-pO_2 in 8 of 9 dogs, suggesting that I-pO_2 correlated to S-pO_2 better than E-pO_2 did. In fact, in dog 3, the correlation of I-pO_2 to S-pO_2 was statistically better ($P < 0.05$) than that of E-pO_2.

Changes in ESP. During aortic occlusion, a significant decrease of the N2 component (to less than 50 % of the baseline value) was observed in only four dogs (Dog. 1, 2, 8 and 9) (Table 2). Although the N2 component tended to decrease after the occlusion balloon reached the Th12 level, this change was not consistent, neither in individual dogs, nor from one dog to another. On the

Table 2 Percent changes of N1 and N2 amplitudes in ESP by aortic occlusion level

Dog.		Aortic occlusion levels										Def. (%)
		L5	L4	L3	L2	L1	Th13	Th12	Th11	Th10	Th8	
1	N1	105	105	100	105	95	105	114	123	123	132	105
	N2	104	108	104	108	96	104	76	56	56	48	92
2	N1	110	100	90	70	75	80	80	80	80	75	60
	N2	85	85	76	39	39	39	39	0	0	0	76
3	N1	100	86	97	107	103	97	103	100	114	124	103
	N2	100	100	120	140	80	100	100	80	140	140	140
4	N1	100	100	103	95	92	92	92	92	92	82	NR
	N2	100	100	120	90	80	90	80	120	120	90	NR
5	N1	120	120	128	124	136	128	136	132	104	124	NR
	N2	100	100	100	100	100	100	100	75	75	75	NR
6	N1	104	107	110	107	106	105	104	96	89	87	114
	N2	103	103	100	100	92	100	94	89	78	67	100
7	N1	87	85	89	89	83	85	85	91	91	91	85
	N2	85	100	85	85	92	92	54	69	69	69	77
8	N1	101	93	94	101	104	96	105	112	108	108	97
	N2	100	100	82	91	45	36	0	18	0	0	36
9	N1	98	98	93	99	102	102	104	109	109	109	98
	N2	25	0	81	50	50	75	0	0	0	0	0

Data are expressed as percentages of each control value determined before aortic occlusion. Underlines indicate a significant decrease (to less than 50% of control).

Def.: Deflation of the aortic occlusion balloon; ESP: Evoked spinal potentials; N1 and N2: The 1st negative and the 2nd negative components; NR, not recorded because of poor condition of the experimental dogs. Other abbreviations are the same as in Fig. 3.

other hand, the N1 component did not show a significant change.

Discussion

Paraplegia is one of the catastrophic complications of thoracic aortic surgery. Although a number of strategies have been put forward to prevent spinal cord ischemia in experimental models (3–5), none of these are satisfactory. It has been reported that neurophysiologic monitoring methods such as ESP and MEP proportionally indicate the occurrence of postoperative paraplegia (13, 17). However, by the time a change in ESP is observed, critical ischemia has caused irreversible damage (13). Thus, there are some problems with neurophisiologic indicators, such as ESP and MEP (13, 14), limiting their widespread use. Moreover, while MEP is thought to be a highly sensitive indicator of paralysis, it cannot be used in combination with muscle relaxants that are usually necessary in clinical cases.

Wadough *et al.* directly measured surface pO_2 of the spinal cord in pigs, and reported that the primary cause of spinal cord injury after aortic clamping was oxygen deficiency in the area distal to the aortic occlusion and supplied by the artery of Adamkievicz (15). We previously reported a good correlation between E-pO_2 and spinal blood pressure (16). Therefore, we believed that I-pO_2 and/or E-pO_2 would significantly reflect spinal cord ischemia, and wanted to know whether or not I-pO_2 and/or E-pO_2 correlated well with S-pO_2.

As a preliminary study, we closely examined the vascular structure of the spinal cord in dogs. This revealed that there are many radicular arteries that ran into the anterior spinal artery at almost every lumbar or thoracic vertebra level; a representative specimen is presented (Fig. 5). Therefore, we believed that it would be possible to develop a model of graded spinal cord ischemia by incrementally withdrawing an aortic occlusion balloon in the cephalad direction. The pia mater is well vascularized and receives blood supply from the anterior spinal artery. This was proven by injection of indigo carmine dye into the anterior spinal artery; the pia mater

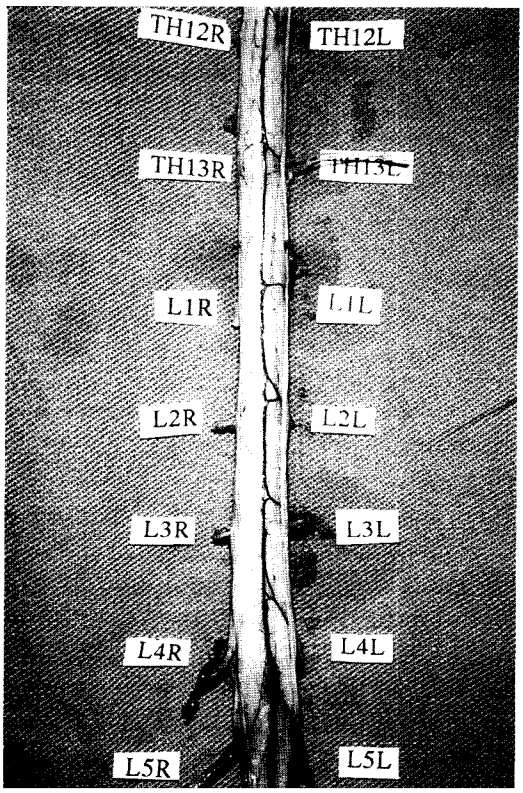

Fig. 5 A representative specimen of the vascular structure of the anterior spinal artery. The anterior spinal artery was clearly stained with indigo carmine solution. TH12R and TH12L indicate the right and left sides at the 12th thoracic vertebra level, respectively, and L1R and L1L similarly indicate the 1st lumbar vertebra level. Similar abreviations indicate the side and the thoracic and/or lumbar vertebra level.

was stained more densely than the spinal cord itself. This anatomical feature, namely, that the outer membrane of spinal cord is better vascularized than the inside of the spinal cord, can also explain the observations that I-pO_2 is almost always higher than S-pO_2 and that I-pO_2 shows more sensitive changes with greater fluctuations than S-pO_2. Therefore, I-pO_2 significantly correlated with S-pO_2.

E-pO_2 also correlated with S-pO_2, but the r-value was lower in round numbers, though not always significantly, than that of I-pO_2. This was chiefly attributed to difficulty in precise positioning of the pO_2 probe in the epidural space. Unless this problem is solved, it will be difficult to use E-pO_2 as an indicator accurately reflecting oxygenation of the spinal cord. On the contrary, I-pO_2 was not subject to this limitation. Therefore, instantaneous monitoring of spinal cord oxygenation can be achieved by measuring I-pO_2. That is, spinal cord ischemia can be detected while it is not yet severe enough to cause changes in ESP and while the ischemic damage is still reversible.

The reasons for a lack of consistent changes in ESP during aortic occlusion in this study were considered in three ways. First, ESP did not change until the occlusion balloon reached so critical a level as to induce marked spinal cord ischemia. Second, a longer tenure of ischemia may be necessary for a significant change in ESP to be induced. This means that both the ischemic time at each occlusion level and the cumulative ischemic time after reaching the critical level were insufficient in our experiment. Third, there was a problem with the completeness of the aortic occlusion by balloon. Since the balloon was incrementally withdrawn to produce graded ischemia, a leakage, small though it might have been, by the periphery of the balloon, must have occurred during movement of the balloon. Even a small leakage meant reperfusion of the spinal cord, resulting in improvement and/or delayed manifestation of ESP changes. Furthermore, since the diameter of the aorta is larger at the proximal end than at the distal end, the air content of the occlusion balloon should have been increased each time the balloon was pulled cephaladly.

Nevertheless, it is worthwhile to know that the spinal cord must suffer significant ischemia before remarkable changes in ESP occur. Svensson *et al.* have succeeded in identifying the key-arteries feeding the spinal cord. They used an intrathecal electrode to detect a hydrogen-induced current impulse (HICI) created by injecting hydrogen-saturated saline into selected lumbar or intercostal arteries (6). Similarly, our method also can be used to identify the feeding arteries by detecting the oxygen current impulse by injecting oxygen-saturated saline or blood into each group of arteries. Furthermore, it may be possible to perfuse these feeding arteries with a small pump so that the time of aortic cross-clamping can be prolonged with safety.

In conclusion, both I-pO_2 and E-pO_2 significantly correlated to S-pO_2, the former showing a superior correlation. Therefore, we propose I-pO_2 as a reliable indicator of spinal cord ischemia, and we recommend it as the expedient and practical indicator to be monitored for prevention of critical paraplegia after thoracic aortic surgery.

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