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Spinal function monitoring by evoked spinal cord potentials in aortic aneurysm surgery

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Abstract: Evoked spinal cord potentials (ESCPs) were monitored in 12 patients who underwent repair of thoracoabdominal aortic aneurysm with a high risk of spinal ischemia. A pair of bipolar catheter electrodes were introduced into the epidural space, one at the level of the C5–T2 vertebrae and the other at the level of T11–L2. Conductive mixed ESCP in seven patients, conductive sensory ESCP in one patient, and segmental descending ESCP in three patients were observed by applying a rectangular electric current to one of each pair of epidural electrodes and recording through the other. Segmental ESCP in response to posterior tibial nerve stimulation was observed in one patient. Following aortic cross-clamping, the I wave of conductive mixed ESCPs gradually decreased in amplitude with latency prolongation in five of the seven patients and disappeared in one of these five; transient augmentation of amplitude was observed before eventual decline in four of these five patients. The N wave of segmental descending ESCP subsequently flattened in two of the three patients and the N_1 wave of segmental ESCP in the one patient. Three of the four patients in whom the ESCPs disappeared during aorta clamping recovered the ESCPs after declamping and showed no neurological disorders postoperatively. Intraoperative ESCP monitoring appears to be useful to detect spinal cord ischemia in the early stage and to alert surgeons and anesthesiologists so that timely resuscitative steps can be taken.

Key words: Spinal evoked potential, Aortic aneurysm, Aorta clamp, Spinal cord ischemia, Spinal cord damage

Introduction

Spinal cord damage caused by ischemia during surgery for aortic aneurysm is a most serious complication, the prevalence of which still remains between 3% and 40% [1,2]. As neurological signs and symptoms of ischemic lesions by aortic cross-clamping (AXC) are masked under anesthesia, an alternative measure should be undertaken for monitoring spinal cord function. The ideal neuromonitoring procedure should provide early information about critical changes whereby the therapeutic procedures in progress can be rapidly modified or adapted.

Somatosensory evoked potentials (SEP) from the scalp are currently used in the evaluation of spinal cord function during surgical procedures on the aorta [3,4]. However, SEP monitoring has yielded low specificity and sensitivity to postoperative neurological deficits of the spinal cord, as the potentials represent integrated functions from the stimulated peripheral nerves to the cerebral cortex via the posterior columns of the spinal cord. The area of the spinal cord most sensitive to ischemia is thought to be the anterior horn of the lumbar enlargement, as shown by the distribution of postoperative spinal cord injury [1,2,4]. Cunningham et al. [3], by monitoring SEP during aortic surgery, reported 6 of 30 patients with false-positive responses (20%), and Crawford et al. [4] noted 33 of 99 patients with false-positive (33%) and 7 of 99 patients with false-negative responses (7%). Gugino et al. [5] adovocated monitoring of motor evoked potentials elicited by trascranial magnetic stimulation during aortic surgery, but the potentials also involve the motor cortex, spinal motor tract, peripheral motor nerves, and myoneural junctions other than the anterior horn cells in the traveling pathway.

Shimoji et al. [6] have developed a technique for the epidural recording of evoked spinal cord potentials (ESCP) using a thin stainless wire electrode, introduced safely through a catheter for continuous epidural anesthesia. At present, a bipolar flexible catheter electrode, with a diameter of 0.8 mm, is commercially avilable (UKG-100-2PM, Unique Medical, Tokyo, Japan). With this method we have conducted intraoperative ESCP

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monitoring, along with cerebrospinal fluid (CSF) pressure monitoring and mild hypothermia, for the past 3 years in patients undergoing surgical repair of aortic aneurysm with high risk of spinal ischemia.

Subjects and methods

After approval from the Institutional Ethics Committee on Human Research, 12 patients (7 men and 5 women) undergoing resection and grafting of thoracoabdominal aortic aneurysm with high risk of spinal ischemia were studied (Table 1). Informed consent for intraoperative ESCP monitoring was obtained from all of the subjects. The average age was 57.5 years (range 30–72) and the average weight 57.9 kg (range 37–76). Preoperative aortoradiography in these patients revealed that a large radicular artery (suspicious of Adamkiewicz's artery) arose from the extending region of the aortic aneurysm. Replacement of the aneurysm using an artificial vessel was planned with clamping of the aorta at two sites immediately proximal and distal to the aneurysm under initiation of femoro-femoral venoarterial partial bypass (F-F bypass). Mild hypothermia was also intended to protect the spinal cord during AXC using a heat exchanger assembled in the F-F bypass circuit.

The patients were premedicated with intramuscular scopolamine 0.25–0.5 mg and morphine 5–10 mg. Electrodes for ESCP monitoring were introduced on the day prior to surgery for the first 3 patients. As they complained of discomfort or sleeplessness at night due to the electrodes, electrodes were introduced before induction of anesthesia and at least 1 h before heparinization on the day of surgery for the remaining 9 patients. After placement of an intravenous catheter, a blood pressure cuff, an Spo_2 probe, and electrodes for electrocardiogram, patients were placed in the lateral decubitus position. A pair of flexible bipolar catheter electrodes, with a diameter of 0.8 mm, (UKG-100-2PM, Unique Medical, Tokyo, Japan) were aseptically introduced into the posterior spinal epidural space through an 18-gauge Tuohy needle. One was placed near the cervical enlargement of the spinal cord at the level of the C5–T2 vertebrae and the other near the lumbosacral enlargement at T11–L2. The longitudinal level of epidural electrodes was confirmed by antero-posterior X-rays prior to anesthesia on the day of surgery. In addition to the electrodes for ESCP monitoring, a polyethylene tube with a diameter of 0.8 mm was introduced into the subarachnoid space through the Tuohy needle at the L2–3 or L3–4 interspace, which was used for pressure monitoring and drainage of the CSF.

The spinal cord was stimulated by applying a rectangular electric current (0.1–0.2 ms pulse width, 3–10 Hz frequency and 2–8 mA intensity) to one of the two epidural electrodes and ESCP was recorded through the other. The electric current was applied to the posterior tibial nerve at the popliteal fossa in the recording of segmental (seg.) ESCP from the lumbosacral enlargement. The band-pass filter was set at 100–1500 Hz. Additive averaging was performed 50 times.

Anesthesia was induced with intravenous diazepam 5–10 mg and fentanyl 0.8–2.2 mg, and the trachea was intubated after intravenous vecuronium or pancuronium 6–8 mg. Subsequent surgical preparations and measurements of ESCP were carried out with the administration of incremental doses of diazepam, fentanyl, and vecuronium or pancuronium, if needed. Less than 0.8% isoflurane in oxygen was inhaled during thoracotomy or laparotomy for blood pressure control in six of the patients. Respiration was mechanically controlled to maintain $Paco_2$ at around 40 mmHg. The right internal jugular vein was cannulated for pressure monitoring of the superior vena cava and as a route of drug administration. The right radial artery at the wrist and the dorsal artery of the foot, contralateral to the following femoral artery cannulation for F-F bypass, were cannulated for continuous monitoring of arterial blood pressue and blood sampling. A balloon catheter was inserted into the bladder and an esophageal probe introduced for temperature monitoring.

Prior to thoracotomy, a 24–28 Fr venous cannula was introduced via the femoral vein to the inferior vena cava and an 18 Fr arterial cannula via the femoral artery to the abdominal aorta for the institution of F-F bypass. The F-F bypass circuit including a membrane oxygenator was primed basically with lactated Ringer's solution (1000 ml), 20% mannitol (40 g), 25% albumin (25 g), 8.4% sodium bicarbonate (80 mEq), urinastatin (250 000 U), poloxamer-188 (42 ml), and heparin (30 mg). Activated coagulation time was adjusted to 400–600 s during F-F bypass. Surgical preparation of the aneurysms was carried out with F-F bypass ready to start.

Results

The operation time was 8.9 ± 4.0 h (mean ± SD) and anesthesia time was 11.9 ± 4.0 h. The amounts of fentanyl and diazepam administered throughout surgery were 3.4 ± 1.5 mg and 14 ± 4.9 mg, respectively. Duration of AXC, flow of F-F bypass, range in the distal arterial pressure during AXC, and peak CSF pressures pre-, during, and post-AXC are shown in Table 1. Minimum and maximum spinal cord perfusion pressure (distal perfusion pressure—CSF pressure) during AXC period was 26 mmHg (range 3–63) and 50 mmHg (range 18–99), respectively. Five to 30 ml of CSF was withdrawn during AXC to raise spinal cord perfusion

Table 1. Subject profiles, location of aneurysm, duration of aorta cross-clamp (X-clamp), femoro-femoral venoarterial partial bypass (F-F bypass) flow, pressure range in the dorsal artery of the foot during aorta X-clamp, peak cerebrospinal fluid (CSF) pressures pre-, during, and post-aorta clamp (pre-dur-post), and drained volume of CSF

No.	Age (years)/ Sex	Weight (kg)	Aneurysm	Duration of aorta X-clamp (min)	F-F bypass flow (1/min)	Arter. Pres. in Foot (mmHg)	CSF Pres. (mmHg) Pre-dur-post	CSF drainage (ml)
1	47/M	61	dTAA	72	1.6	41–57	23–31–15	30
2	47/F	56	dTAA	85	1.8	45–55	10–17–9	—
3	71/F	71	dTAA	57	1.6	28–41	8–11–6	—
4	55/M	67	dTAA	76	1.2	8–10–12	—	
5	44/F	44	dTAA	125	2.2	35–56	8	
6	59/F	49	dTAA	187	2.0	60–83	17–24–20	5
7	62/M	54	TAAA	63	1.6	48–76	6–8–9	10
8	63/M	48	dTAA	69 + 55	1.7	46–71	5–1–1	10
9	71/M	56	AAA[a]	44				
10	30/M	76	TAAA	85	1.6	20–35	12–17–13	—
11	72/F	37	TAAA	50	1.6	75–111	10–12–13	—
12	69/M	76	TAAA	184 + 97	1.2, (0.6_b)	10–60		
Mean	57.5	57.9		89	1.6	41–65	11–15–11	
SD	12.7	12.1		44	0.3	18–21	5–8–5	

dTAA, descending thoracic aortic aneurysm; TAAA, thoracoabdominal aortic aneurysm; AAA, abdominal aortic aneurysm; Arter. Pres., arterial pressure; CSF Pres., cerebrospinal fluid pressure; SD, standard deviation.
[a]F-F bypass was ready but not performed.
[b]Selective perfusions to the superior mesenteric artery and the right renal artery.

Table 2. Vertebral levels of electrodes, evoked spinal cord potentials (ESCPs) recorded, changes in latency and amplitude of ESCPs during aorta X-clamping, and the lowest esophageal temperature during aorta X-clamp (esophageal temperature immediately before aorta X-clamping)

No.	Stim. electro.	Rec. electro.	ESCP	ESCP changes during aorta X-clamp: Latency	ESCP changes during aorta X-clamp: Amplitude	Esophag. temp. (°C)
1	T11	C7	cond. mix.	n.c.	n.c	36.1 (35.8)
2	T12	C7–T1	cond. mix.	↓ (0.24 ms)	n.c.	34.4(33.6)
3	T1	T12–L1	cond. mix.	↑ (0.22 ms)	↓(76%)	32.8 (33.9)
4	T11–12	C7–T1	cond. mix.	↑ (0.44 ms)	↑ (107%) ~ ↓ (44%)	35.0 (36.5)
5	C5–6	T11–12	cond. mix.	↑ (0.16 ms)	↑ (25%) ~ ↓ (16%)	35.1(36.1)
6[a]	T1–2	T12	cond. mox.	↑ (0.20 ms)	↑ (20%) ~ ↓ (76%)	33.9(35.8)
7	C5–6	T12–L1	cond. mix.	↑	↑ (198%) ~ ↓ ~ 0	34.0(34.1)
8	L1–2	T1–2	cond. sens.	n.c.	n.c.	36.0(36.0)
9	PTN	T12–L1	seg.	↑	↓ ~ 0	34.8(34.8)
10	C7	T12-L1	seg. desc.	↑	↓ ~ 0	30.8(32.5)
11	T1–2	T12-L1	seg. desc.	↓ (1.10 ms)	↓(23%)	35.5(34.1)
12[b]	T1–2	T12-L1	seg. desc.	↑	↓ ~ 0	32.6(32.7)

Stim. electro., stimulation electrode; Rec. electro., recording electrode; Esophag. temp., esophageal temperature; PTN, posterior tibial nerve; cond. mix., conductive mixed; cond. sens. conductive sensory; seg., segmental; seg. desc., segmental descending; n.c., no change; ↓, decreased; ↑, increased; ↑ ~ ↓, decreased following temporary augmentation; ↓ ~ 0, decreased and disappeared.
[a]Developed ataxia due to cerebellar infarction.
[b]Died due to massive bleeding within 24 h postoperatively.

pressure in 5 patients. Esophageal temperature was 34.7° ± 1.3°C immediately before AXC and the lowest value during AXC was 34.3° ± 1.5°C (Table 2).

ESCPs recorded from the epidural space were divided into four patterns: conductive (cond.) mixed ESCP in seven patients (patients 1–7), cond. sensory ESCP in 1 patient (patient 8), seg. ESCP in 1 patient (patient 9), and seg. decending ESCP in three patients (patients 10–12) (Table 2). Cond. mixed ESCP was recorded through epidural electrodes over the caudal (T11–12, T12, T12–L1) or rostral (C7, C7–T1) spinal cord by applying stimulation over the rostral (C5-6, T1, T1–2) or caudal (T11, T12, T11–12) spinal cord, respectively. In general, cond. mixed ESCP consisted of two

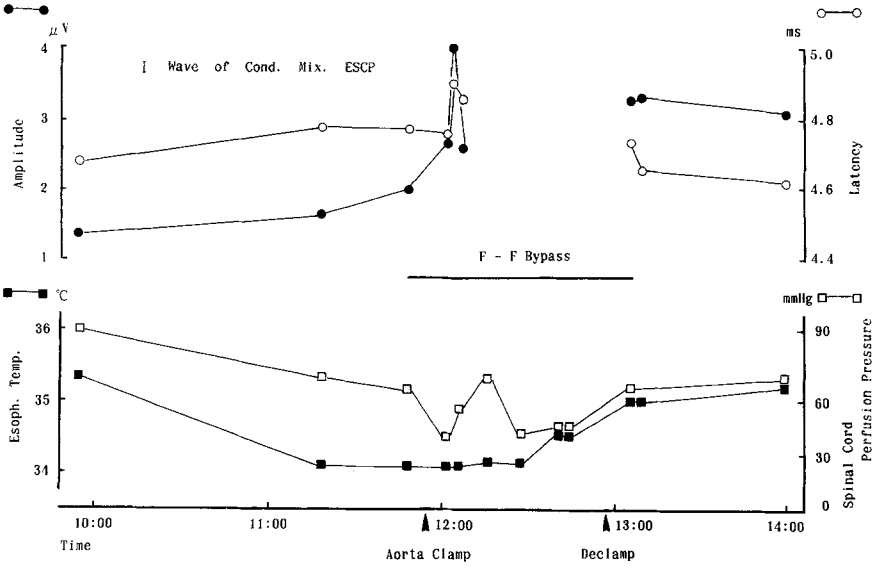

Fig. 1. Changes in I wave amplitude and latency of conductive mixed evoked spinal cord potentials (ESCPs) (*upper lines*), esophageal temperature, and spinal cord perfusion pressure (*lower lines*) in the course of surgery in patient 7. ESCP was recorded epidurally at T12–L1 with stimulation at C5–6. The I wave of conductive mixed ESCP, which disappeared following temporary amplitude augmentation after aortic cross-clamping, reappeared immediately after declamp with no neurological deficits. F-F bypass, femoro-femoral venoarterial partial bypass; Esoph. temp., esophageal temperature; Spinal cord perfusion pressure, mean dorsalis pedis artery pressure—mean cerebrospinal fluid (CSF) pressure

fast components: an I wave with low threshold and high amplitude followed by a small II wave, considered to originate in both the ascending and descending tracts in the posterolateral spinal cord [7]. Cond. sensory ESCP was obtained through epidural electrodes over the rostral (T1–2) spinal cord by applying stimulation over the cauda equina (L1–2), consisting of early triphasic spikes (C1, C2, C3) followed by a slow negative wave [8]. These cond. ESCPs reflect the function of white matter of the spinal cord [7,8].

On the othr hand, the following seg. ESCPs mainly reflect the function of gray matter in the spinal cord [6,9]. Seg. ESCP, recorded epidurally over the lumbosacral enlargement in response to posterior tibial nerve stimulation, consisted of an initial positive spike (P_1), reflecting the afferent volley along the roots [6], followed by a slow negative wave (N_1), believed to be synchronized activity of dorsal horn neurons [6], and a slow positive wave, assumed to reflect primary afferent depolarization [6,8,9]. Seg. descending ESCP, recorded through epidural electrodes over the lumbosacral enlargement (T12–L1) by applying stimulation over the rostral (C7, T1–2) spinal cord, also consisted of early spike waves followed by late negative and positive slow waves (N and P) [9]. The wave forms and time courses of the descending N and P waves were very similar to those of the N_1 and P_2 waves, respectively, produced by segmental volleys. Both the P_2 wave of seg. ESCP and the P wave of seg. descending ESCP were depressed prior to AXC under deep fentanyl anesthesia.

During AXC, the I wave of cond. mixed ESCPs progressively decreased in amplitude with prolonging latency in five of the seven patients (Table 2) and subsequently disappeared in one (patient 7) of these five, as demonstrated in Fig. 1. The N_1 wave of seg. ESCP was abolished in one patient (patient 9) (Fig. 2) and the N wave of seg. descending ESCP in two (patients 10, and 12) of the three patients. Figure 3 shows changes of the ESCP in patient 10. The four patients (patients 7, 9, 10, and 12) with ESCP disappearance during AXC received the distal aortic clamp below the celiac artery, as the aortic aneurysm extended to the abdominal aorta. In addition, spinal cord perfusion pressure did not exceed 20 mmHg at least for 65 min during AXC in patients 10 and 12, although it remained constantly above 40 mmHg in patient 7. In patient 9, F-F bypass was ready to start but was not initiated because the aneu-

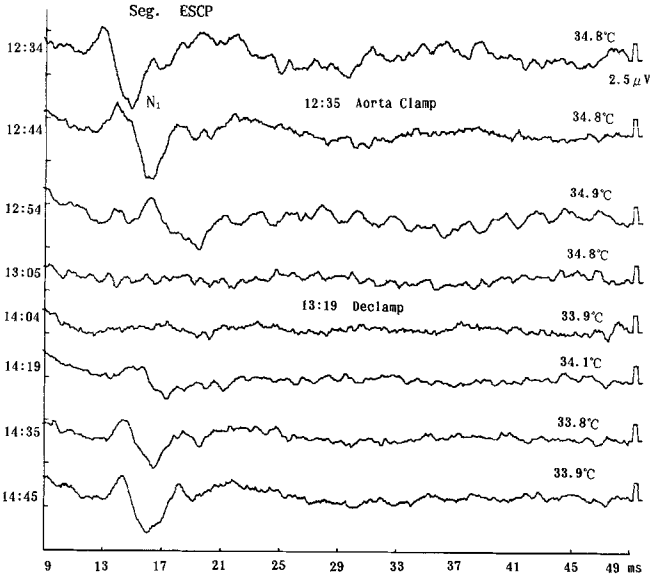

Fig. 2. Sequential changes of segmental ESCP recorded epidurally at T12–L1 in response to posterior tibial nerve stimulation in patient 9. The N_1 wave of segmental ESCP disappeared 30 min after aortic cross-clamping and reappeared about 53 min after declamp with no neurological deficits

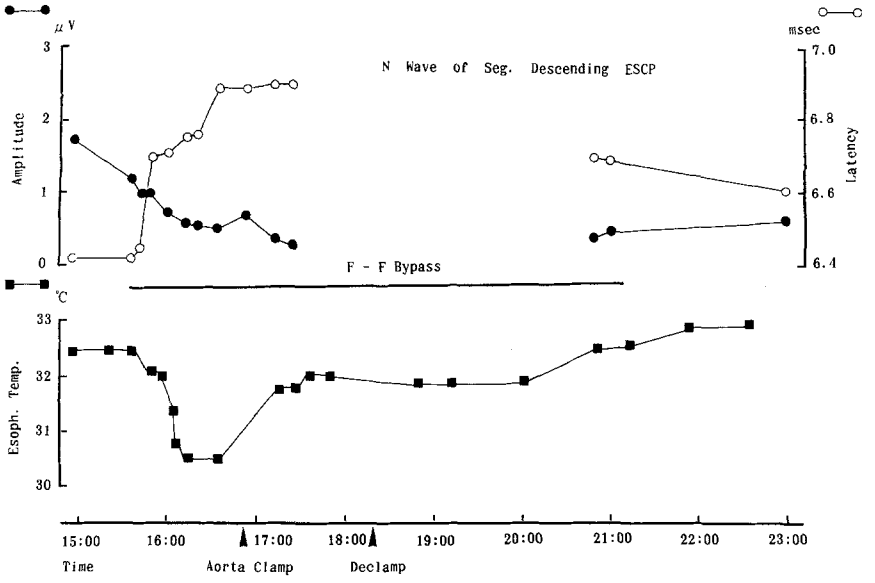

Fig. 3. Changes in the N-wave amplitude and latency of segmental descending ESCP (*upper lines*) and esophageal temperature (*lower line*) in the course of surgery in patient 10. ESCP was recorded epidurally at the level of the T12–L1 vertebrae with stimulation at the C7 level. The N wave of segmental descending ESCP, which gradually decreased in amplitude and increased in latency with cooling under initiation of F-F bypass and disappeared after aortic cross-clamping, reappeared about 190 min after declamp with no neurological deficits. Spinal cord perfusion pressure (mean dorsalis pedis artery pressure—mean CSF pressure) never rose above 20 mmHg during the 85 min aorta cross-clamping period

rysm extended down to the iliac arteries. Patients 7, 9, and 10, whose ESCPs were restored with a tendency to return to the pre-clamp amplitudes and latencies after declamping, showed no spainal cord dysfunction postoperatively. The disappearing period of ESCPs and the esophageal temperature at that period were as follows: 53 min and 34.0°–35.1°C in patient 7, 70 min and 34.1°–34.8°C in patient 9, and 188 min and 30.8°–32.4°C in patient 10. Patient 12, without reappearance of the ESCP, died due to massive intraoperative bleeding during the first 24 h postoperatively. Initial spikes of cond. sensory ESCP did not change, while the follwing slow negative wave became positive and subsequently flattened with time during AXC.

Temporary augmentation of amplitude was observed before eventual decline in the I wave of cond. mixed ESCPs of four of the seven patients (57%). A typical case is shown in Fig. 4. ESCP latency decreased during AXC in 2 patients, in whom body temperature during AXC was maintained higher than the baseline before AXC (Table 2). Patient 6 developed ataxia postoperatively, which computerized tomography revealed to be due to cerebellar infarction. Neurological disorders suspicious of epidural or subdural hematoma were not seen in the postoperative course even in the patients who received epidural placement of the catheter electrodes on the day of surgery.

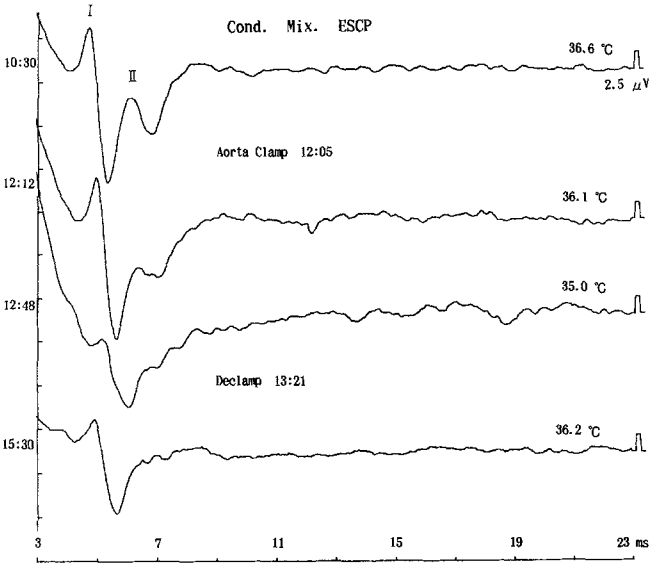

Fig. 4. Sequential changes of conductive mixed ESCP recorded epidurally at the level of the T12 vertebrae with stimulation at the T1–2 level in patient 4. The I wave of conductive mixed ESCP increased in amplitude prolonging latency temporarily after aortic cross-clamping before eventual decline

Discussion

In the present study, ESCPs disappeared during AXC in 4 of the 12 subjects. Three of these four patients were free from spinal cord damages with ESCPs reappearing after release of AXC. Presumably, spinal ischemia would tend to occur toward the end of AXC in these patients. When the ESCPs disappeared, we informed the surgeons and anesthesiologists about the impending ischemia. Anesthesiologists made efforts to increase the spinal perfusion pressure and to advance the cooling in cooperation with a perfusionist. Surgeons prepared selective perfusion for the arteries leaving the aorta between the proximal and distal AXCs in patient 12, who eventually died due to massive bleeding. Even cond. mixed ESCP, which was thought to be more resistant to ischemia as compared with seg. ESCPs, was abolished for 53 min in patient 7, although the spinal cord perfusion pressure was maintained at more than

40 mmHg and the esophageal temperature above 34°C throughout AXC. This suggests that the main feeding artery of the spinal cord left the aorta between the proximal and distal AXC, and also that the spinal function can be recovered even after such a long disappearance of the nonsynaptic ESCP. In addition to the peculiar blood supply system to the spinal cord, mild hypothermia, and hemodilution and heparinization accompanied with the initiation of F-F bypass, might have contributed to the good revovery. The good recoverability of spinal function means that the spinal cord is more resistant to ischemia than the brain. Grabitz et al. [10] stated that AXC up to 60 min in dogs does not necessarily induce irreversible ischemic damage, although it demonstrates loss of physiological function.

Lee et al. [11] reported that cond. ESCPs gradually decreased in amplitude and increased in latency after AXC in all 8 patients, particularly when the distal blood pressure dropped or the distal clamp was lower than T12. One of the eight patients developed paraplegia with sensory dissociation and the amplitude did not return after declamping the aorta. Takano et al. [12] recognized changes in cond. ESCPs in 10 of 23 patients during AXC and noted the possibility that hypothermia may also change the evoked potential. In their report, ESCPs decreased in amplitude and did not return to the control level in 2 of the 10 patients. One patient showed paraplegia and the other died during surgery due to massive intraoperative bleeding. Recently, Stuhmeier K-D et al. [2] reported that cond. ESCPs disappeared within 15 min after AXC in 40 of 100 patients undergoing repair of thoracoabdominal aortic aneurysm and that postoperative neurological deficits were seen in 12 (30%) of the 40 patients. Based on the observation, they recommended waiting for 15 min after AXC before permanently dividing the aortic aneurysm.

In general, synaptic components such as the N_1 and P_2 waves of seg. ESCP and the N and P waves of seg. descending ESCP are more easily depressed by anesthesia [13,14], hypothermia [15], and ischemia [12,16], compared with nonsynaptic components, and this might explain why seg. ESCPs are seldom used as a clinical indicator of spinal ischemia under anesthesia combined with induced hypothermia. However, this study demonstrated that the N_1 wave of seg. ESCP or the N wave of seg. descending ESCP is a useful and sensitive indicator of spinal cord ischemia under deep fentanyl anesthesia and mild (33°–34°C) hypothermia. Subsequent P_2 or P waves, which are more susceptible to anesthetic agents [13, 14] and hypothermia [15], had almost flattened prior to AXC and were not available as an indicator of ischemia. The high-cut setting of the band-pass filter in the ESCP recording might be also attributed to the suppression of slow P_2 or P waves. The N wave of seg. descending ESCP in patient 10 was restored about 190 min after declamping the aorta. In this patient, not only ischemia but long-lasting hypothermia (30°–32°C) was considered to be responsible for the delayed reappearance of the N wave. Our previous study using dogs anesthetized with N_2O (60%)-O_2-isoflurane (1.15%) demonstrated that the I wave of cond. ESCP could be available for the monitoring of spinal ischemia under hypothermia down to 23°–24°C and the N wave of seg. descending ESCP down to 30°C [15].

Seg. descending ESCP was first recorded by Shimizu et al. [9] from the human epidural space at the level of the lumbosacral enlargement in response to electrical stimulation of the cervical spinal cord. They observed the occlusion phenomenon between the seg. descending ESCP elicited by descending volleys (cervical or upper thoracic epidural stimulation) and the seg. ESCP produced by segmental volleys (posterior tibial nerve stimulation). Shimizu et al. [9] suggested use of a unipolar electrobe with reference electrode on the interspinosus ligament for the recording of seg. descending ESCP. Among the T12–L1 epidural recordings of the present study, seg. descending ESCP was obtained in some patients and cond. mixed ESCPs in others. Our previous animal experiment [15] revealed that a bipolar recording electrode was situated not along the spinal cord but across it when seg. descending ESCP was recorded from the canine epidural space over the lumbosacral enlargement. In the present clinical study, we confirmed the longitudinal levels of the bipolar recording electrodes by X-ray photographs, but could not confirm their exact locations in the spinal epidural space.

We have demonstrated that autoregulation of the spinal cord blood flow is impaired below the mean arterial blood pressure of 60 mmHg in the dog [17]. Oka and Miyamoto [18] recommended maintaining spinal cord perfusion pressure above 40 mmHg by CSF drainage in a canine ESCP study under partial AXC. Murray et al. [19] withdrew an average of 47 ml of CSF to maintain a CSF pressure below 10–15 mmHg in 29 patients under AXC without F-F bypass. In our present study, spinal cord perfusion pressure was not always kept above 40 mmHg. However, the low spinal cord perfusion pressure was attributable mainly to low distal arterial pressure rather than to high CSF pressure in this study. We measured the distal arterial pressure at the dorsal artery of the foot contralateral to the femoral cannulation for sending oxygenated blood toward the abdominal aorta. It is conceivable that aortic pressure distal to AXC might be higher than the peripheral arterial pressure. The initiation of F-F bypass seems to have suppressed an increase in CSF pressure secondary to AXC. Only two patients exhibited CSF pressure higher than 20 mmHg during AXC.

In our study there was temporary augmentation of the amplitude in 57% of the cond. mixed ESCP after AXC. Takano et al. [12] also observed temporary amplitude augmentation of cond. ESCPs in four of five patients whose body temperature decreased during AXC. Amplitude augmentation of the cond. mixed ESCPs has already been reported at the initial phase of spinal ischemia [11,12,20], compression [21], or cooling [15] before eventual decline, although the mechanism is still not clear. One possible explanation for amplitude augmentation during AXC is additional excitation of the subliminal nerve fibers [20]. The threshold of a nerve action potential is known to decrease temporarily, early following induction of ischemia [22] or hypothermia [23,24].

One of 12 patients in our study developed ataxia due to cerebellar infarction. Lee et al. [11] reported that one of eight patients died due to cerebral infarction postoperatively. It seems inevitable in AXC surgery that clots or atheromas tear off from the thickened arterial intima leading to emboli in the arteries branching from the aorta. Searches for safe AXC zones using an echogram may be useful for prevention of the embolism. Maruyama et al. [25] recommended intraoperative monitoring of multispatial evoked potentials during aortic surgery to detect early central nervous dysfunction and also to define the sites of lesions.

Investigators may refrain from recording the ESCPs from the epidural space in heparinized condition. Maruyama et al. [25] used a specially designed catheter electrode which had two side holes near its tip and carried out an aspiration test frequently through the catheter electrode during and after the operation. Epidural electrodes should be introduced on the day before surgery or at least 1 h prior to heparinization on the day of surgery and should be removed after inactivating heparin with protamine to avoid epidural or subdural hematoma. Contamination of myogenic components in ESCPs obtained will lead to false-positive responses. Complete muscle paralysis should be confirmed by monitoring muscle twitch responses to peripheral nerve stimulation during ESCP measurements.

In summary, as the subjects in the present study had been suspectd to be at high risk of spinal ischemia during AXC, F-F bypass was instituted to establish distal perfusion during the AXC period in all the patients except one. Nevertheless, ESCPs during AXC decreased in amplitude with prolonging latency in 9 (75%) of 12 patients and eventually disappeared in 4 patients (33%). Although the size of our population was too small to make any firm conclusion, the incidence of spinal ischemia during AXC, which may lead to postoperative neurological deficits, appears to be relatively high. ESCP monitoring is useful for early detection of impending spinal ischemia. Severe suppression or disappearance of ESCPs will alert surgeons and anesthesiologists so as to make timely resuscitative interventions.

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