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ORIGINAL ARTICLE

Changes of motor evoked potentials during descending thoracic and thoracoabdominal aortic surgery with deep hypothermic circulatory arrest

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Abstract
Background Paraplegia is a serious complication of descending and thoracoabdominal aortic aneurysms (dTAAs and TAAAs) surgery. Motor evoked potentials (MEPs) enable monitoring the functional integrity of motor pathways during dTAA and TAAA surgery. Although MEPs are sensitive to temperature changes, there are few human data on changes of MEPs during mild and deep hypothermia. Therefore, we investigated changes of MEPs in deep hypothermic circulatory arrest (DHCA) in dTAA and TAAA surgery.
Methods Fifteen consecutive patients undergoing dTAA and TAAA surgery using DHCA were enrolled. MEPs were elicited and recorded during each degree Celsius change in nasopharyngeal temperature during both the cooling and rewarming phases. Hand and leg skin temperature were also recorded simultaneously.
Results In the cooling phase MEP amplitude decreased lineally in both the hand and leg. The MEP disappeared at ~16°C in both the hand and leg in 10 of 15 patients, but was still elicited in 5 patients. In the rewarming phase MEP in the hand recovered before the temperature reached 20°C for eight patients and 25°C for the other seven patients. In contrast, MEP in the leg recovered below 20°C for two patients and 30°C for three patients. For the other eight patients MEP waves did not recover during the rewarming phase.
Conclusion In the cooling phase of DHCA, MEP disappeared at ~16°C in some patients but was still elicited in others. MEP recovered below 25°C in the hand. Recovery of MEP in the leg was, however, extremely variable.

Keywords Aortic aneurysm, thoracic · Circulatory arrest, deep hypothermia-induced · Evoked potentials, motor

Introduction

Paraplegia is a serious potential complication of the surgical repair of descending thoracic aneurysm (dTAA) and thoracoabdominal aortic aneurysms (TAAAs) and still occurs in a high percentage of patients. The incidence of this complication varies between 2.6 and 13%, depending on the extent of the aneurysm, dissection, rupture, and cross clamp time [1–5].

Intraoperative monitoring of transcranial motor evoked potentials (MEPs) enables monitoring the functional integrity of motor pathways. Monitoring also enables evaluation of surgical strategies by enabling prompt response to spinal cord ischemia during dTAA and TAAA surgical repair [6–9]. However, the clinical use of these techniques has shown that elicited responses are very sensitive to suppression by anesthetics, muscle relaxants, and temperature [10–13]. Although previous studies have reported the effects of anesthetics and muscle relaxants on MEPs, data on the effect of temperature is limited [14, 15].

It has been suggested that hypothermia is an effective strategy for protecting the central nervous system against ischemia and is commonly used in the surgical repair of dTAAs and TAAAs. MEPs are sensitive to temperature,

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because values are obtained by electrophysiological monitoring. In mild hypothermia (32–34°C), MEPs are commonly used, because their efficacy for predicting postoperative neurological deficits has been established. Although human and animal data are available regarding the effect of hypothermia on MEP amplitude [10, 16], there are very few human data on deep hypothermic circulatory arrest.

Temperature changes from 36 to 18°C in deep hypothermic circulatory arrest enable the evaluation of MEP changes. With this information, the objective of our study was to investigate the change of MEPs in the surgical repair of dTAA or TAAAs using deep hypothermic circulatory arrest.

Materials and methods

This study was approved by the institutional review board for human research at the National Cerebral and Cardiovascular Center, and informed consent was obtained preoperatively from all study patients. Fifteen consecutive patients were enrolled who underwent the elective repair of dTAA and TAAA with the use of deep hypothermic circulatory arrest between July 2007 and March 2008. The patients included were free from neuromuscular disorders and untreated epilepsy.

Anesthesia technique

The anesthesia technique was standardized. No sedatives or other centrally acting drugs were given before the induction of anesthesia. Anesthesia was induced intravenously with fentanyl 5–10 μg/kg and midazolam 0.05–0.1 mg/kg. Tracheal intubation was performed with a left-sided double-lumen tube, the tube position being confirmed by fiberoptic bronchoscopy. Vecuronium bromide 0.1 mg/kg was administered to facilitate intubation. Anesthesia was maintained by continuous infusion of propofol 6 mg/kg/h and remifentanil 0.2–0.7 μg/kg/min without muscle relaxants and other drugs which interfere with MEPs, for example sevoflurane, midazolam, and nitrous oxide. The dose of propofol was reduced to 4 mg/kg/h during the DHCA period. Controlled ventilation was adjusted to maintain normocapnia (end-tidal carbon dioxide partial pressure 35–40 mmHg 4.0–4.5 kPa by the alpha stat method) and to administer oxygen 40–100% in air. In the rewarming phase, nitroglycerin and dopamine were used to come off cardiopulmonary bypass (CPB).

Two arterial lines were inserted into the right radial and the dorsalis pedis arteries. A 7.5-Fr. pulmonary artery catheter (OptiQ; Hospira, Lake Forest, IL, USA) was inserted in the right internal jugular vein. After placement of a transesophageal echocardiography (TEE) probe, the patient was positioned in the right lateral position and an intra-thecal catheter was placed in the L4–L5 interspace for cerebrospinal fluid (CSF) drainage and pressure monitoring. CSF drainage was not routinely used perioperatively, and was included as a method to potentially prevent spinal cord ischemia during the postoperative period. Nasopharyngeal (NPT) and bladder (BT) temperatures were monitored by use of the InteliVeu system (Philips Health Care, Boblingen, Germany). The skin temperature overlying the palmar right thenar muscle (PAT) and the plantar abductor hallucis muscle (PLT) on the opposite side of femoral arterial cannulation were measured by use of a Coretemp® thermometer (Terumo, Tokyo, Japan). The tip of the NPT probe was positioned just above the soft plate.

Technique for monitoring MEPs

A transcranial electrical stimulator D-185 (Digitimer, Welwyn Garden City, UK) was used to elicit MEPs. Stimuli were applied to the scalp by use of silver disc electrodes. The anode was placed at the C3 site, and the cathode at the C4 site (International 10–20 system for the placement of electroencephalogram electrodes). The electrical stimulation consisted of a train of five pulses with a 2.0 ms inter-stimulus interval. The stimulus intensity was set supra-maximally to 10% above the level that produced the maximum MEP amplitudes, typically 500 V. The pulse duration was set at 50 μs, resulting in an current of 800–900 mA. The resulting compound muscle action potentials were recorded from the skin overlying the right thenar muscle, the bilateral flexor hallucis brevis muscles, and the anterior tibial muscle on the side opposite femoral artery cannulation, by use of adhesive gel Ag/AgCl electrodes. The signals were recorded on a time base of 100 ms, passing through a bandpass filter of 50–1000 Hz. Data acquisition, processing, and analysis were performed on personal computer systems (Neuropak MEB-2200; Nihon Koden, Tokyo, Japan).

Surgical technique

The patient was placed in a modified right lateral decubitus position with the shoulders placed at 60° and the hips rotated 30° from the horizontal plane. Aneurysms limited to the descending thoracic aorta were approached through a full posterolateral thoracotomy in the fourth or fifth intercostal space. The thoracoabdominal incision varied in length and level, depending on the expected extent of aortic replacement.

The entire aorta was exposed with dissection of the retroperitoneal space and division of the costal cartilage and diaphragm. The left or right common femoral artery and vein were exposed for cannulation, and after anticoagulation with heparin (300 U/kg), a long venous cannula

was positioned in the right atrium. Proper positioning, which is essential to good flow in the CPB system, was checked by TEE. After establishment of a femorofemoral bypass circuit, a venous drainage cannula was inserted into the main pulmonary artery and an arterial line was inserted into the left axillary artery.

Systemic hypothermia was induced by CPB and the target temperature for hypothermia was set at 18°C. During the period of cooling, the abdominal portion of the incision was completed, if indicated. The left lung was collapsed and was gently retracted to minimize manipulation and injury. When the heart fibrillated, the left ventricle was vented through the apex if necessary for decompression. Cooling was continued until the NPT reached 18°C. After the patient had been placed in the head-down position and 20–40 mEq KCl had been administered into the venous reservoir, circulatory arrest of the upper body was established. The venting catheter was occluded to prevent suction of air into the heart. The aneurysmal portion was cross-clamped at the mid-thoracic level (T6 level) distal to the expected proximal anastomosis. The aorta was then opened, and the proximal anastomosis was made in an open fashion. During this anastomosis, ante-grade cerebral perfusion of the vertebral artery through an axillary arterial line (100–200 ml/min) was performed to prevent suction of air into the brain, and the distal aorta was perfused at a flow rate of 500 ml/min. ("distal aortic perfusion"). After the proximal aorta had been de-aired through the open distal end of the graft with the patient in the Trendelenburg position, CPB flow was reinstituted with a side branch of the graft. A cross-clamp was then placed just distal to the side branch cannulation site and flow (target cardiac index was 2.5) to the upper half of the body was reinstated.

The relevant intercostal arteries (particularly around T9 or T10) connecting to the Adamkiewicz artery (AKA), as demonstrated by MRA, were preserved or re-implanted during the deep hypothermic interval. When the aneurysm involved a long segment (for example TAAA), the clamps were placed sequentially, if possible ("segmental clamp technique"). The rewarming phase was then initiated to an NPT of 25–28°C. The distal anastomosis was also constructed in an open manner under moderate hypothermic circulatory arrest of the lower half of the body. After completion of the anastomosis, full CPB was re-established, resulting in active rewarming of both the upper and lower halves of the body. The heart was defibrillated when the perfuse temperature reached 32°C and the rewarming phase was continued until the NPT was 37°C.

Study protocol

We recorded MEP signals at an NPT of 35°C as a control value on CPB. MEP signals were recorded at every 1°C change to 18°C. In the rewarming phase, MEP signals were also recorded at every 1°C from 18 to 37°C, the terminating of CPB, and the termination of the operation. Amplitude (peak to peak) and latency (from the stimulation to the beginning of waves) of MEPs were measured offline. Amplitude over 10 μV, which could be distinguished from electrical noise, was deemed to be positive.

Results

Table 1 shows patient characteristics. Thirteen of 15 patients had a type B aortic dissection. There were no cases of postoperative paraparesis in any patients. Table 2 depicts details of CPB and surgical procedure. Fourteen of 15 patients used open proximal anastomosis and 13 of 15 patients open distal anastomosis. In the cooling phase, the temperature at MEP loss and the time that elapsed before loss of MEP is shown in Table 3; in the rewarming phase the temperature at MEP recovery and the time that elapsed before recovery of MEP is shown in Table 4.

Table 1 Patient characteristics

Age	52 ± 11 (range 31–66)
Sex	M/F 10/5
Preoperative variables	
HT	9
HLP	5
COPD	2
CAD	1
DM	3
Renal impairment	1
Aneurysm etiology	
Type B dissection	13
Marfan's syndrome	4
Atherosclerosis	1
Infection	1
Aneurysm extent	
dTAA	3
TAAA: Crowford	
Type I	5
Type II	7
Type III	0
Type IV	0

Data are expressed as mean ± SD

HT hypertension, *HLP* hyperlipidemia, *COPD* chronic obstructive pulmonary disease, *CAD* coronary artery disease, *DM* diabetes mellitus, *dTAA* descending thoracic aortic aneurysm, *TAAA* thoracic abdominal aortic aneurysm

Cooling phase

Figure 1 depicts the relationship between MEP amplitudes of the upper and lower extremities and the NPT. In the cooling phase, the increase in MEP amplitude in the hand at 25°C was small compared with that at 26°C. Table 3 depicts individual body temperature data for MEP loss and the time that elapsed before loss of MEP. In the hand, MEP waves disappeared by 16°C in 12 patients, but were still elicited in three patients at temperatures below 16°C and disappeared after circulatory arrest. MEP waves disappeared within 35 min in eight patients. PAT was higher than nasopharyngeal temperature at MEP loss in the hand for all patients. In the leg MEP waves disappeared by 16°C in 10 patients, but were still elicited at temperatures below 16°C in five patients and finally disappeared after circulatory arrest. PLT was higher than nasopharyngeal temperature at MEP loss in the leg for all patients. MEP waves in the leg disappeared within 35 min in eight patients.

Figure 2 reveals the time course of changes of core and local temperature in the cooling phase. NPT decreased rapidly and reached a plateau at ~17°C within 40 min. BT and PAT had an almost parallel trend in the cooling phase and reached a plateau at ~20°C. PLT also had a similar trend and reached a plateau at ~23°C. Overall, each core and peripheral temperature reached a plateau at a different temperature.

Figure 3a shows tat MEP latency increased linearly from 21.5 to 46.4 ms in the hand at 21°C, when MEP amplitude was ~25% of the control value. In Fig. 3b, MEP latency in the leg also increased from 42.3 to 77.0 ms linearly at 22°C, when MEP amplitude was ~25% of the control value.

Table 2 Details of cardiopulmonary bypass

Patient number	Ope time (min)	CPB time (min)	Lower extremity circulatory arrest (min)	Surgical procedure	
				Open proximal	Open distal
1	605	265	54	Yes	Yes
2	355	149	78	Yes	Yes
3	765	410	0	Yes	No
4	570	251	34	Yes	Yes
5	295	116	21	Yes	No
6	435	213	11	Yes	Yes
7	470	185	13	Yes	Yes
8	530	131	37	Yes	Yes
9	545	144	47	Yes	Yes
10	265	128	24	Yes	Yes
11	751	403	14	Yes	Yes
12	364	168	40	Yes	Yes
13	445	224	80	No	Yes
14	370	152	28	Yes	Yes
15	795	326	170	Yes	Yes

Ope operation, *CPB* cardiopulmonary bypass, *CA* circulatory arrest, *Open proximal* open proximal anastomosis with circulatory arrest, *Open distal* open distal anastomosis with circulatory arrest

Table 3 Body temperature at MEP loss and time for MEP loss

Patient number	MEP loss in the hand			MEP loss in the leg		
	Nasopharyngeal (°C)	Palm (°C)	Minutes to loss	Nasopharyngeal (°C)	Palm (°C)	Minutes to loss
1	18	No data	30	19	No data	28
2	20	23.9	17	20	27.2	17
3	After CA	18.1	65	After CA	23.2	65
4	18	21.0	43	After CA	24.3	43
5	18	23.1	22	24	29.6	16
6	19	25.7	29	18	28	30
7	17	22.5	45	16	23.5	47
8	After CA	24.2	34	After CA	23.8	34
9	17	23	32	17	20.8	32
10	17	26.6	32	19	20.3	22
11	18	23	35	After CA	31.4	38
12	After CA	19.3	33	After CA	26.1	33
13	18	20.1	52	18	24.8	61
14	22	28.4	83	22	27.9	83
15	16	17.1	61	16	21.6	61
Median	18	23	34	18.5	24.6	34

MEP motor evoked potential, *CA* circulatory arrest

Table 4 Body temperature at MEP reappearance and time for MEP recovery

Patient number	MEP recovery in the hand			MEP recovery in the leg		
	Nasopharyngeal (°C)	Palm (°C)	Minutes to recover	Nasopharyngeal (°C)	Palm (°C)	Minutes to recover
1	22	20.1	11	28		64
2	18	17.3	15	34	28.8	44
3	19	19.8	26	19	22.8	26
4	19	19.4	5	20	20.8	11
5	19	21.1	8	34.2	33.3	111
6	22	23.4	8	37	35.3	111
7	23	22.8	28	23	20.9	16
8	18	22.5	9	30	22.1	48
9	22	20.9	29	32	25	68
10	22	23	4	30	20.6	27
11	18	19	38	35.6	30.6	176
12	23	22.8	9	37	23.9	77
13	19	21.8	6	19	24.8	5
14	25	24.2	21	28	25.9	35
15	20	23.6	5	34	20.8	80
Median	20	21.8	9	30	24.4	48

MEP recovery time means the time from the end of circulatory arrest to the reappearance of MEP waves (<10 μV)

MEP motor evoked potentials

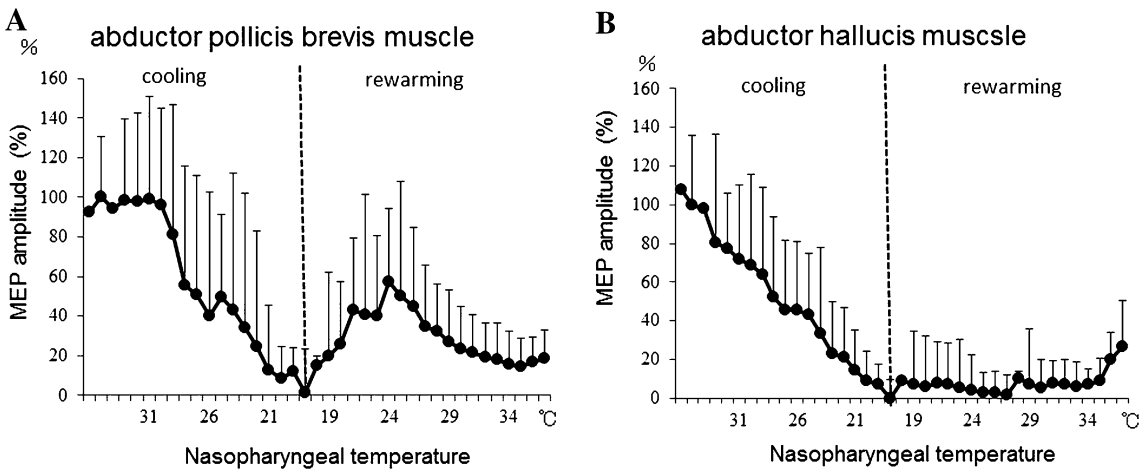

Fig. 1 The effects of temperature change on MEP amplitudes in the cooling and rewarming phases. **a** MEP amplitudes for the upper extremity. **b** MEP amplitudes for the lower extremity

Rewarming phase

MEP amplitude in the hand recovered to approximately 50% of the control value at 25°C once, but gradually decreased to 15.6% at 36°C (Fig. 1). In the leg, the temperature at which MEP recovered was extremely variable (19–36°C). Individually, MEP in the hand recovered below an NPT of 20°C in eight patients and below an NPT of 25°C in the other seven patients. In contrast, MEP in the leg recovered below an NPT of 20°C in two patients and from an NPT of 21 to 30°C in five other patients. In the other eight patients MEP waves recovered before 37°C. MEP in the hand recovered within 20 min of the end of circulatory arrest in 10 of 15 patients and within 30 min in 5 patients only. MEP in the leg recovered after more than 60 min in 6 patients (Table 4).

As is apparent from Fig. 2, the NPT and PAT began to increase to 34.1°C within 90 min in the rewarming phase. BT and PLT began to increase ~40 min after the start of the rewarming phase. BT caught up with NPT and PAT 135 min after the start of rewarming but PLT remained at approximately 31.5°C. In the cooling phase, temperatures

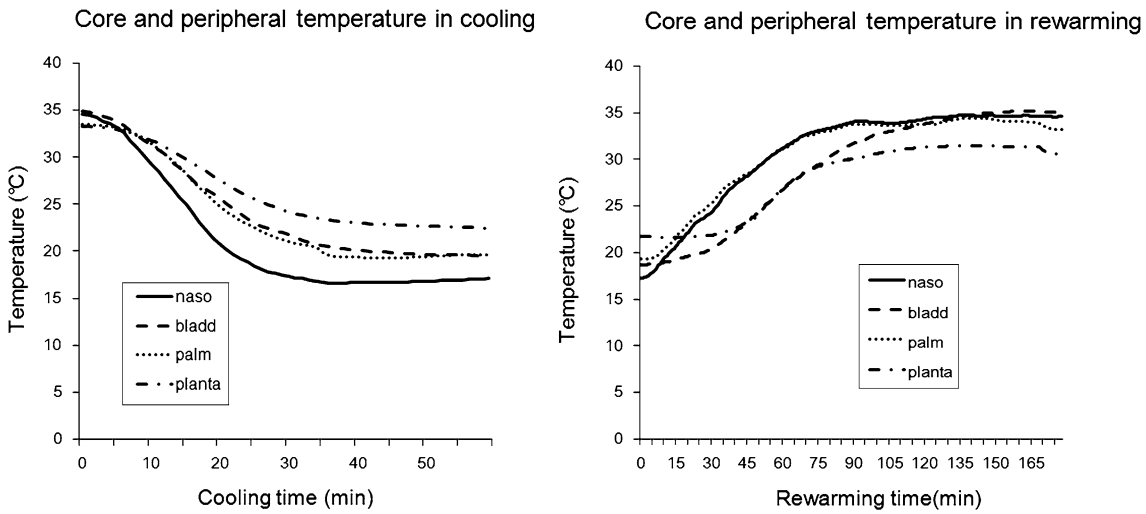

Fig. 2 Changes in core temperature (nasopharyngeal and bladder temperatures) and peripheral temperature (palm and plantar temperatures) over time in the cooling and rewarming phases

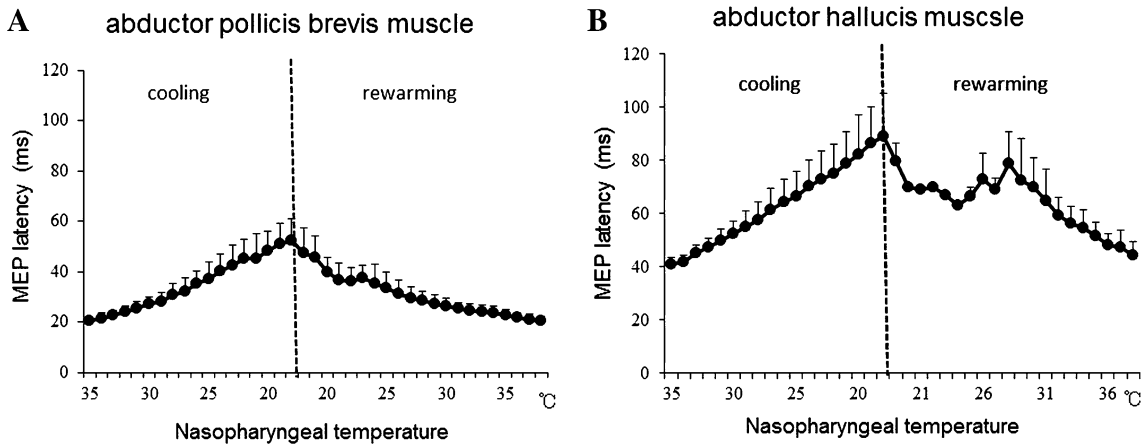

Fig. 3 Latency of MEP waves in the cooling and rewarming phases in the hand, mapped by change in temperature (**a**) and in the leg (**b**)

reached plateau within 30 min. In contrast, in the rewarming phase all temperatures reached a plateau in 120 min.

MEP latency decreased linearly to approximately control values (20.6 ± 1.6 ms) in the hand. In the leg, MEP latency once reduced to ~60 ms, but increased to 77.8 ms at an NPT of 28°C. MEP latency then began to decrease to 44 ms (Fig. 3).

Discussion

This study reveals the changes of MEP amplitude during systemic cooling and rewarming of CPB in humans. In the cooling phase MEP amplitude decreased as NPT decreased. In the rewarming phase MEP change in the hand had a peak at 24°C, but began to decrease after the peak, despite an increase of body temperature. Recovery of MEP in the leg required more time than in the hand. MEP latency increased linearly with a reduction of NPT and decreased linearly with increasing NPT. Also, a large discrepancy between core and peripheral temperatures exists both in the cooling and rewarming phases.

In the cooling phase, a small increase of MEP in the hand was observed at 25°C, but the results did not reach significance (Fig. 1). However, animal studies have demonstrated an increase in MEP amplitudes until 28°C of core temperature in the cooling phase [14, 16]. Browning et al. reported a linear increase of MEP amplitude as body temperature decreased to 28°C of rectal temperature in the cat model. Meylaerts et al. indicated that MEP amplitude was highest at 29.6°C for CSF in regional spinal cord hypothermia in the cooling phase in the pig model. The reason for the discrepancy between this study and previous studies is unknown. However, rapid cooling (1°C every 1 min) in this study might contribute to linear decrease of

MEP amplitude compared with the previous study (1°C every 10 min) [14].

MEP waves in the hand were still elicited even below 18°C in three patients and MEP in the leg in five patients. Meylaerts et al. [17] reported that MEP waves disappeared at 19.5 or 22°C in DHCA in TAAA surgery. The reason MEP was elicited below an NPT of 18°C was unknown. In humans, the low limit of MEP elicitation may be lower than in animals.

In the rewarming phase, MEP amplitudes in the hand recovered to 58.5% of the controls at 25°C, but decreased to 15.6% of the control values at an NPT of 36°C, as a whole. Some animal studies support this result. Browning et al. [14] demonstrated that MEP amplitudes decreased during rectal temperature increase up to 42°C in the cat model. Also, Oro et al. [15] investigated the effect of hyperthermia on MEP amplitudes in the rat model. MEP amplitudes were suppressed over 42°C. In our study, the peak of MEP amplitude in the hand was at an NPT of 25.0°C. The reason for these differences in the peak temperature compared with animal study results is unknown. However, decrease in MEP amplitude in the rewarming phase over 25°C may be because of the effect of the increase in body temperature during the hyperthermic phase in above cited animal studies.

Previous studies cited above suggest that MEP amplitudes begin to decrease above a certain temperature without postoperative motor deficits, similar to our results. Decreased MEP amplitudes in the rewarming phase of CPB should be taken into consideration because MEP amplitudes less than 25% of the control values have been connected to ischemic conditions [17]. In our study, there were no postoperative motor deficits. However, MEP amplitudes decreased to less than 25% of the control values in accordance with changes of body temperature, suggesting reduced MEP amplitudes may lead to the misunderstanding that ischemic conditions occurred in the rewarming phase.

MEP in the hand recovered when NPT became 25°C, and within 40 min for all patients. However, recovery of MPE in the leg required higher NPT and more time than in the hand. As revealed in Fig. 2, the peripheral temperature of the upper extremity increased in parallel with the NPT, but the peripheral temperature of the lower extremity began to increase 45 min after the start of rewarming. The PLT lingered at approximately 30°C when the NPT reached 34°C. When the muscle temperature decreased, the twitch response was reduced [18]. The lower PLT compared with the NPT may be one reason why MEPs in the leg did not recover in the rewarming phase, unlike in the hand.

Further in the leg, the effect of the ischemic condition during circulatory arrest for the open distal anastomosis should be taken into account. However, 20 min after the end of circulatory arrest in the leg, MEP recovered in 11 of 15 patients (Table 2). Further, nasopharyngeal temperature was still approximately 28°C during open distal anastomosis, meaning little effect of ischemia in the leg on MEP may be noted in the rewarming phase. In patient 5 MEP recovery needed 103 min without use of circulatory arrest by clamping the descending aorta, meaning that recovery of MEP after deep hypothermia may take much time without ischemic condition by circulatory arrest. However, unfortunately we did not measure the blood flow in the leg muscles. There still might be the possibility that changes in blood flow contributed to a variety of MEP change.

Figure 2 reveals a discrepancy between the core and peripheral temperatures. The plantar temperature was not as sensitive for the cooling or rewarming phase as the nasopharyngeal temperature. The reason the plantar temperature did not decrease in the same way as the other measured temperatures was not clear in the cooling phase, but after the start of rewarming circulatory arrest was still performed in the leg for the anastomosis of the distal side of aorta, resulting in delayed rewarming in the leg. We need to keep in mind that the temperature of lower extremity was still low at the end of CPB, suggesting that the MEP waves did not recover completely.

Our study had several limitations, including small sample size. Despite the limited sample size, typical MEP patterns were observed both in the cooling and rewarming phases in this human research. An additional issue was that we did not use a bispectral index monitor, therefore we could not evaluate anesthetic depth during CPB. Propofol concentration might increase because of clamping of the descending aorta [19], suggesting that MEP amplitude might decrease. However, the metabolic rate increases during the rewarming phase. Overall, the effect of hypothermia or CPB may be small during the rewarming phase and this could not be determined in this study. Further, it might be better to use the major spinal cord temperature to evaluate the effect of spinal cord temperature on MEP amplitude. However, it was invasive to measure spinal cord temperature because of the need to insert a large probe intrathecally. Therefore, we did not measure spinal cord temperature.

In summary, MEP amplitude decreased linearly in the cooling phase, but was still elicited at an NPT of approximately 16°C in some patients. In the rewarming phase, MEP in the hand recovered when NPT reached 25°C, but recovery of MEP in the leg required higher NPT and more time than in the hand, probably because of the lower temperature in the leg than in the hand and the ischemic condition in the leg. MEP amplitudes in the hand recovered to 58.5% of the control values at 25°C but decreased to 15.6% of the control values at an NPT of 36°C. We must keep in mind that MEP amplitude might decrease in the rewarming phase. The usefulness of MEP monitoring in

patients undergoing TAA and TAAA repair with DHCA is questionable because of variety of MEP results, irrespective of the absence of postoperative. Many factors could have an effect of MEP amplitude in addition to body temperature in DHCA.

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