Somatosensory and brainstem auditory evoked potentials in cardiac arrest patients treated with hypothermia*

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Objective: To evaluate the prognostic value of short-latency median nerve somatosensory evoked potentials and brainstem auditory evoked potentials in outcome prediction for comatose cardiac arrest patients treated with hypothermia.

Design: Prospective, randomized, controlled trial of mild hypothermia after out-of-hospital cardiac arrest; a substudy of the European Hypothermia After Cardiac Arrest study.

Setting: Intensive care unit of a tertiary referral hospital (Helsinki University Central Hospital).

Patients: Sixty consecutive patients (aged 18–75 yrs) resuscitated from out-of-hospital ventricular fibrillation and comatose at 24 hrs after cardiac arrest; all patients were randomly assigned either to therapeutic hypothermia of 33°C or normothermia.

Interventions: All patients received standard intensive care for at least 2 days. Patients randomized to hypothermia were cooled with an external cooling device for 24 hrs and then allowed to rewarm slowly for 12 hrs. In the normothermia group, the core temperature was kept below 38°C with antipyretics and by phys-

ical means. The clinical outcome was assessed 6 months after cardiac arrest.

Measurements and Main Results: Somatosensory evoked potentials and brainstem auditory evoked potentials were recorded 24–28 hrs after cardiac arrest. All wave latencies were significantly prolonged in the hypothermia group. Bilaterally absent N20 waves predicted permanent coma with a specificity of 100% in both treatment groups. Brainstem auditory evoked potential recordings did not correlate with the outcome in either treatment group.

Conclusions: The prognostic ability of median nerve short-latency somatosensory evoked potentials does not seem to be affected by therapeutic hypothermia. Brainstem auditory evoked potentials had no additional value in outcome prediction. (Crit Care Med 2005; 33:1736–1740)

KEY WORDS: cardiac arrest; hypothermia; somatosensory evoked potentials; brainstem auditory evoked potentials; hypoxic-ischemic encephalopathy; prognosis

Advances in emergency medicine have increased the number of cardiac arrest patients in the emergency department. The duration and severity of global cerebral ischemia and secondary mechanisms of ischemia related to reperfusion contribute to the extent of brain damage. Mild hypothermia of 33°C for 12 or 24 hrs has been shown to improve neurologic outcome after cardiac arrest (1, 2) and also to increase the chance of survival (1).

Early prediction of neurologic outcome for patients resuscitated from cardiac arrest is a challenging task. The median nerve somatosensory evoked potentials (SEPs) have been reported to accurately predict poor outcome after cardiac arrest (3, 4). Hypothermia is known to prolong all neuronal conduction velocities, including the SEP and brainstem auditory evoked potential (BAEP) latencies, and temperature has also been reported to affect the amplitudes of SEP responses (5–8). Mild to moderate hypothermia does not abolish cortical responses to sensory evoked potentials (9). The cortically generated component N20 has been shown to disappear only at mean nasopharyngeal temperature of 20°C under normal hemodynamic conditions (6, 10). The measurement of BAEPs has been used to monitor neuronal function at the level of the brainstem. Hypothermia increases the BAEP interwave latencies (5, 11, 12). BAEP components have also been reported to be abolished at temperatures below 20°C (12).

The aim of this study was to evaluate the prognostic value of the short-latency median nerve SEPs and BAEPs in out-

come prediction for comatose cardiac arrest patients treated with hypothermia.

SUBJECTS AND METHODS

The protocol and consent procedure of this study was approved by the ethics committee of Helsinki University Central Hospital in accordance with institutional guidelines. A deferred consent was used for all patients. The patient's family was informed about the trial and could withdraw the patient at any time from the study. Each patient was informed about the trial both orally and in writing, when he or she was able to receive this information.

Patients randomized into the Hypothermia After Cardiac Arrest trial were included (1). All adult patients admitted to the emergency department of Helsinki University Central Hospital after resuscitation from out-of-hospital cardiac arrest were screened for the trial. The criteria for inclusion were an age of 18–75 yrs, a witnessed cardiac arrest, ventricular fibrillation or nonperfusing tachycardia as the initial cardiac rhythm, presumed cardiac origin of the arrest, an estimated interval of 5–15 mins from collapse to the first attempt at resuscitation by emergency medical personnel, and an interval of <60 mins from collapse to restoration of spontaneous circulation (ROSC). If

*See also p. 1868.

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cardiac arrest occurred after the arrival of the first responding unit or the patient responded to verbal command after ROSC, the patient was excluded. Additional exclusion criteria were cardiac arrest due to intoxication or trauma; tympanic temperature $<30^{\circ}\text{C}$ at admission; evidence of hypotension (mean arterial pressure <60 mm Hg) for >30 mins after ROSC and before randomization; evidence of hypoxia (arterial oxygen saturation $<85\%$) for >15 mins after ROSC and before randomization; a preceding terminal illness; known pre-existing coagulopathy; pregnancy; factors that made participation in follow-up unlikely; and enrollment in another study (1).

All cardiac arrest data were collected in the Utstein style (13). Cardiac arrest was defined as the absence of both palpable pulse and spontaneous respiration. ROSC was defined as return of palpable arterial pulse. Within the Helsinki metropolitan area, basic life support and advanced cardiac life support were provided by the three-tiered Helsinki emergency medical services. Outside the city of Helsinki, basic life support was provided by the staff of regional fire brigade-based emergency medical services and advanced cardiac life support by the emergency medical helicopter. All emergency medical service units are equipped with semiautomatic defibrillators, and the staffs are trained to intubate, insert intravenous catheters, and start medication.

Patients were transferred to the intensive care unit (ICU) after assessment of respiratory and hemodynamic function and initial neurologic evaluation in the emergency department. All patients underwent standard intensive care management and monitoring, including mechanical ventilation and placement of arterial catheters, central venous catheters, and Foley catheters, with a temperature sensor and a pulmonary artery catheter used as necessary.

Sedation and analgesia were accomplished with administration of midazolam (0.125 mg/kg per hr) and fentanyl (0.002 mg/kg hr intravenously), titrated for 32 hrs. To avoid shivering, pancuronium (0.05 mg/kg per hr) was initially used. The lowest dose that would permit complete muscle relaxation was used for a total of 32 hrs. Mean arterial pressure was targeted at 80 mm Hg. Decreases in systemic blood pressure were treated primarily with crystalloid fluids or hydroxyl ethyl starch. Inotropic agents were used if sufficient blood pressure control could not be achieved by fluid therapy alone. No glucose-containing solutions were infused. Intravenous insulin infusion was administered if blood glucose exceeded 10 mmol/L, and normoglycemia was targeted. Optimal head-up position (30 degrees) was maintained.

Patients randomized to normothermia were allowed to rewarm passively to normo-

thermia (core temperature, $<38^{\circ}\text{C}$) and were then kept normothermic. Those randomized to hypothermia treatment were actively cooled externally to a core target temperature of 33°C (range, $32\text{--}34^{\circ}\text{C}$) with a cooling device (Thera-kool, Kinetic Concepts, Wareham, UK). The cooling device consists of a mattress and cover that delivers cold air over the entire body. Ice packs were applied on the patient's axilla and groin to enhance the cooling. Hypothermia ($33^{\circ}\text{C} \pm 1^{\circ}\text{C}$) was maintained for 24 hrs from start of cooling, and patients were then allowed to rewarm for 12 hrs. Life support was maintained for at least 3 days for all patients and at least 7 days for patients responding to pain in any manner. The decision to withdraw treatment was based on clinical status. Withdrawal of care meant withdrawal of inotropic intravenous medications but maintenance of airway and ventilator treatment. The SEP and BAEP results were not available during the acute phase and thus did not affect the decision to withdraw treatment. Family members were informed of the decision to withdraw treatment but did not participate in the decision-making process.

Assessment of Outcome. Neurologic examination was performed daily during treatment in the ICU; on days 3, 7, and 14; at discharge from hospital; at 3 months; and at 6 months after cardiac arrest. The primary end point was the recovery of consciousness, defined as ability to obey verbal command, and the secondary outcome was favorable outcome at 6 months after cardiac arrest, as assessed by the Pittsburgh Outcome Scale (14, 15). This is a five-category scale of cerebral performance categories (CPCs). These categories are defined as follows: CPC 1, conscious and alert with normal cerebral function; CPC 2, conscious and alert with moderate cerebral function; CPC 3, conscious with severe cerebral disability; CPC 4, comatose or in persistent vegetative state; CPC 5, dead. For statistical analyses, the neurologic outcome was dichotomized as good (CPC 1 and 2) or poor (CPC 3, 4, and 5).

SEPs and BAEPs. The SEPs and BAEPs were recorded 24–28 hrs after cardiac arrest with a four-channel electromyography/evoked potentials device (Medelec Sapphire, Medelec Limited, Surrey, UK). For SEPs, square-wave pulses with duration of 0.2 msec at a repetition rate of 3 pulses per sec were used as stimuli to the median nerve at wrist. Stimulus intensity was 25–30 mA. Ag/AgCl surface electrodes were placed according to the International 10–20 System as follows: for N9 peak on ipsilateral Erb's point (referenced to contralateral Erb's point), for N13 peak on cervical spinous process C7 (referenced to sternum), for P15 and N18 peaks on Fpz (referenced to ipsilateral mastoid), and for N20 peak on contralateral parietal (referenced to ipsilateral parietal). A sweep time of 100 msec, an amplifier sensitivity of 20–50 $\mu\text{V}/\text{div}$, a display sen-

sitivity of 1.0–2.5 $\mu\text{V}/\text{div}$, and a bandwidth of 20 Hz to 1 kHz were used. Electrode impedances were maintained below 5 kOhm. Trials with excessive artefacts were rejected automatically. A mean 610 trials (range, 350–1050) were averaged. Recordings were performed twice to both right and left median nerve stimulation. The distance between stimulation site and Erb's point was measured to calculate the nerve conduction velocity.

BAEPs were recorded with clicks of 105-dB intensity presented monaurally at a rate of 9/sec through earphones. A mask of 20-dB intensity was used in the contralateral ear. A sweep time of 10 msec, an amplifier sensitivity of 5–10 $\mu\text{V}/\text{div}$, a display sensitivity of 0.05–0.1 $\mu\text{V}/\text{div}$, and a bandwidth of 100 Hz to 2 kHz were used. The Ag/AgCl surface electrodes were placed on ipsilateral mastoid and referenced to Fz. Responses of 2048 stimuli were averaged for an individual test, and the procedure was performed twice to both right and left ear stimulation. The SEP and BAEP recordings were all analyzed retrospectively as a block. The physician analyzing SEPs and BAEPs was blinded to the treatment and outcome for the patient.

Statistical Analysis. Categorical variables are given as counts and percentages. Data are given as median and interquartile range. Means of left-sided and right-sided evoked potential stimulations were used for calculations. Amplitudes and latencies were calculated from cases with present responses. Continuous data were compared with the Mann-Whitney U-test. Outcome data are binary, and the chi-square test was used to compare proportions between the hypothermia and normothermia groups. $p < .05$ were considered statistically significant. We used the Statistica data analysis software system (StatSoft, Tulsa, OK) to analyze the data.

RESULTS

Seventy consecutive unconscious (Glasgow Coma Scale <9) patients met the inclusion criteria and were randomly assigned to hypothermia ($n = 36$) or normothermia ($n = 34$) treatment. In both groups one patient died before 24 hrs. SEPs to median nerve stimulation and BAEPs were recorded for 60 consecutive patients (subjects 9–70, with the exception of two patients who died before 24 hrs) at 24–28 hrs after cardiac arrest. SEP and BAEP recordings were not available for the first eight randomized patients for technical reasons, after which all consecutive patients surviving for at least 24 hrs were studied. Thirty of the 60 patients were randomized to the hypothermia treatment and 30 to the normothermia treatment. All patients were sedated, relaxed, and artificially ventilated during the evoked potentials recording

session. The median core temperature during recording was 33.0°C in the hypothermia group and 37.5°C in the normothermia group.

Clinical and demographic data of patients (n = 60) are presented in Table 1. No statistically significant differences were observed in any baseline characteristics between the two treatment groups. Forty-five patients recovered consciousness (26 in the hypothermia group and 19 in the normothermia group; $p = .04$). At 6 months after cardiac arrest, the outcome was favorable for 77% (n = 23) of the hypothermia-treated patients and 50% (n = 15) of the normothermia-treated patients ($p = .03$).

SEP. Data from 57 patients (30 in the hypothermia group and 27 in the normothermia group) could be analyzed. For two normothermic subjects, reliable analysis of SEP recordings was prevented by excess muscle artefact due to insufficient relaxation, and one normothermic

patient had severe edema of the head and upper trunk, which made the analysis of SEP recordings impossible. The nerve conduction velocities, latencies to N13 responses, latencies to cortical N20 responses, and amplitudes of N20 responses and cervicocortical conduction times (times from cervicomedullary N13 to cortical N20) are presented in the Table 2. Nerve conduction velocity was statistically significantly lower in the hypothermia group ($p < .001$), as expected. The latencies to N13 response and to early cortical N20 response were significantly longer in the hypothermia group ($p < .001$). The amplitudes of N20 responses were comparable in the two treatment groups and did not correlate with outcome. Cervical N13 peak was detected in all patients. The cortical N20 responses were bilaterally absent in 11 patients, of which three were in the hypothermia group and eight in the normothermia group. None of these patients regained

consciousness, and they died after a median of 10 days (range, 2–354 days). Bilaterally absent cortical N20 responses predicted permanent coma with a specificity of 100% (95% confidence interval, 92–100%). Three patients (one in the hypothermia group and two in the normothermia group) had bilateral N20 responses but did not awaken. Thus, the sensitivity was 75% (95% confidence interval, 30–95%) in the hypothermia group and 80% (95% confidence interval, 49–94%) in the normothermia group. Table 3 summarizes the relation between outcome and presence of N20 responses.

BAEP. Data from all 60 patients (30 in the hypothermia group and 30 in the normothermia group) could be analyzed. The latencies of wave I, III, and V responses and intervals of I–III, III–V, and I–V waves are presented in Table 4. All these latencies were statistically significantly longer in hypothermia-treated patients ($p < .02$ –.001). Within the treatment groups the latencies did not correlate with age, awakening, or achieving favorable outcome. At least one of the waveforms I, III, or V were constantly missing in two hypothermia-treated and four normothermia-treated patients. In two patients these responses were missing only unilaterally; both patients were known to have a hearing loss and awakened later. In four patients these responses were missing bilaterally, and three of them did not regain consciousness. Of them, one patient had a hearing loss, which probably explained the findings in BAEP, since he had cortical N20 responses in SEPs. One patient also had bilaterally absent N20 responses on SEPs. One patient had severe edema of the head and upper trunk, and analysis of the SEP recording was not possible. The one patient who awakened despite bilaterally missing waveforms had a hearing deficit and used a hearing aid. Thus, in this study the addition

Table 1. Clinical and demographic characteristics of patients treated with hypothermia or normothermia

Characteristic	Median Value (Range)	
	Hypothermia (n = 30)	Normothermia (n = 30)
Age, yrs	60 (23–75)	58 (18–75)
Male/female, no. (%)	26/4 (87/13)	22/8 (73/27)
Bystander-initiated CPR, no. (%)	16 (53)	16 (53)
BLS, min	7 (4–14)	7 (4–11)
ACLS, min	14.5 (7–59)	13 (5–39)
ROSC, min	17 (9–34)	18.5 (8–45)
Defibrillations before ROSC	3 (1–12)	2 (1–30)
Total dose of epinephrine before ROSC, mg	2.0 (0–9.0)	2.0 (0–6.5)
Glasgow Coma Scale score on admission	5 (3–7)	5 (3–8)
Blood glucose on admission, mmol/L	10.0 (6.1–18.7)	9.7 (4.9–25.9)
Tympanic temperature on admission, °C	35.2 (33.4–36.9)	35.5 (33.6–36.9)

CPR, cardiopulmonary resuscitation; BLS, basic life support; ACLS, advanced cardiac life support; ROSC, restoration of spontaneous circulation.

Table 2. Characteristics of somatosensory evoked potential (SEP) recordings

Characteristic	Median Value (Interquartile Range)		
	Hypothermia Group (n = 30)	Normothermia Group (n = 27)	p Value
Temperature during SEP recording, °C	33.0 (32.7–33.4)	37.5 (36.6–37.8)	
Nerve conduction velocity, m/sec	46.2 (42.9–48.5)	53.5 (49.2–56.9)	<.001
Latency to N13 response, msec, RS stimulation	18.1 (16.8–19.0)	15.0 (14.0–16.0)	<.001
Latency to N13 response, msec, LS stimulation	17.5 (16.5–19.0)	14.8 (14.0–16.0)	<.001
Latency to cortical N20 response, msec, RS stimulation	25.0 (24.0–26.8)	21.0 (20.0–22.0)	<.001
Latency to cortical N20 response, msec, LS stimulation	24.7 (23.3–26.0)	20.5 (20.0–21.2)	<.001
CCT, msec, RS stimulation	7.2 (6.5–7.8)	6.0 (5.5–6.5)	<.001
CCT, msec, LS stimulation	7.0 (6.5–7.5)	6.0 (5.0–6.3)	<.001
Amplitude of N20 response, µV, RS stimulation	1.3 (1.0–1.7)	1.4 (1.2–1.9)	NS
Amplitude of N20 response, µV, LS stimulation	1.1 (1.0–1.5)	1.4 (0.9–1.8)	NS

RS, right-sided; LS, left-sided; CCT, cervicocortical conduction time; NS, not significant.

Table 3. Relation between outcome and the presence of N20 responses in somatosensory evoked potential (SEP) recordings

Variable	No. of Patients	
	Hypothermia Group (n = 30)	Normothermia Group (n = 30)
No. of patients with analyzable SEP recordings	30	27
Awakened, N20 responses present	26	17
Outcome at 6 mos		
CPC 1	20	9
CPC 2	3	4
CPC 3	2	3
CPC 4	0	0
CPC 5	1	1
Awakened, N20 responses absent	0	0
Remained in coma, N20 responses present	1	2
Outcome at 6 months		
CPC 5	1	2
Remained in coma, N20 responses absent	3	8
Outcome at 6 months		
CPC 4	0	1
CPC 5	3	7

CPC, cerebral performance category.

Table 4. Characteristics of brain stem auditory evoked potential (BAEP) recordings

Characteristic	Median (Interquartile Range)		p Value
	Hypothermia Group (n = 30)	Normothermia Group (n = 30)	
Temperature during BAEP recording, °C	33.0 (32.7–33.4)	37.5 (37.2–37.8)	
Latency of wave I, msec, RS stimulation	1.85 (1.77–1.99)	1.70 (1.60–1.94)	.02
Latency of wave I, msec, LS stimulation	1.85 (1.69–1.97)	1.64 (1.55–1.80)	.005
Latency of wave III, msec, RS stimulation	4.59 (4.33–4.73)	4.08 (3.89–4.23)	<.001
Latency of wave III, msec, LS stimulation	4.54 (4.29–4.91)	4.06 (3.84–4.13)	<.001
Latency of wave V, msec, RS stimulation	6.88 (6.61–7.10)	6.08 (5.78–6.44)	<.001
Latency of wave V, msec, LS stimulation	6.90 (6.71–7.19)	5.88 (5.64–6.51)	<.001
Interpeak latency I–III, msec, RS stimulation	2.70 (2.53–2.87)	2.32 (2.09–2.43)	<.001
Interpeak latency I–III, msec, LS stimulation	2.73 (2.54–3.04)	2.33 (2.19–2.48)	<.001
Interpeak latency III–V, msec, RS stimulation	2.28 (2.14–2.48)	1.99 (1.85–2.14)	<.001
Interpeak latency III–V, msec, LS stimulation	2.35 (2.07–2.50)	1.94 (1.75–2.19)	<.001
Interpeak latency I–V, msec, RS stimulation	5.01 (4.76–5.25)	4.33 (4.08–4.57)	<.001
Interpeak latency I–V, msec, LS stimulation	5.13 (4.86–5.35)	4.22 (3.89–4.45)	<.001

RS, right-sided; LS, left-sided.

of BAEP recording to SEP recording did not increase sensitivity for identifying patients with poor prognosis, as compared with SEP recording alone.

DISCUSSION

Our results suggest the possibility that the prognostic ability of median nerve SEP recordings is preserved in cardiac arrest patients treated with hypothermia of 33°C. The bilateral absence of early N20 cortical responses also was a reliable predictor of permanent coma after cardiac arrest in these patients. The high sensitivity is most likely attributed to the small number of patients.

Therapeutic hypothermia significantly reduces the nerve conduction velocities

and increases the cervicocortical conduction time. This reflects slowing of neural conduction along the axons and an increase in synaptic delay (16). In peripheral nerves, sensory nerve conduction velocity has been reported to decrease at a rate of about 2.0 m/sec per each degree Celsius (17). In our study the difference in nerve conduction velocity between the two treatment groups was about 7 m/sec, and the difference in core temperature was 4.5°C. The amplitudes of N20 responses did not differ between the two treatment groups. In the study of Porkkala et al. (8) examining median nerve SEPs under hypothermic cardiopulmonary bypass, the N20 amplitudes at temperatures of 36.4°C and 31.8°C were

similar but decreased significantly at a temperature of 27.8°C.

SEP and BAEP recordings are noninvasive, reproducible techniques that can be easily performed at the ICU bedside. Induced hypothermia requires muscle relaxation, which prevents the adjustment of SEP stimuli intensity on the basis of thumb twitch. The intensity of the stimuli has to be sufficient to evoke an ipsilateral supraclavicular response in patients with clinical muscle relaxation. Significant improvements in SEP have been reported within 24 hrs after ROSC (18), and allowing a period of at least 24 hrs after cardiac arrest is recommended for obtaining a reliable prognosis based on SEP. Short-latency SEPs and BAEPs are quite resistant to anesthetic agents. In the study of McPherson et al. (19), thiopental increased the latency of N20 and an intravenous bolus of fentanyl 25 µg/kg increased the latency of N20 and decreased the amplitude of N20. In our study the dosage of fentanyl was only 2 µg/kg intravenously every hour. For BAEP, fentanyl with doses up to 50 µg/kg intravenously had no significant effect on absolute or interpeak latencies of wave I-, III-, or V-evoked potentials (20).

Theoretically, abnormal BAEP would not be expected to be a sensitive marker of neuronal damage after global ischemia, since cortical neurons are more sensitive to hypoxic-ischemic damage than neurons in the brainstem. According to our results, BAEP recordings are not useful in identifying a poor prognosis for cardiac arrest patients either when used alone or when used in combination with short-latency SEPs. Missing waveforms in BAEP can indicate a brainstem lesion, but this finding may also be attributed to hearing loss, which is a common confounding factor, especially in elderly patients.

There are some limitations to this study. Because of the strict inclusion and exclusion criteria, these patients represent only a minor proportion of all resuscitated cardiac arrest patients, and thus the results cannot be applied to all resuscitated patients treated with hypothermia. Still, this is the patient group in which studies have shown therapeutic hypothermia to be effective in ameliorating neurologic outcome after cardiac arrest (1, 2). The number of patients was relatively small and the proportion of patients whose outcome was favorable was exceptionally high. Although the target population was not identical with that of

The prognostic ability of median nerve short-latency somatosensory evoked potentials does not seem to be affected by therapeutic hypothermia, and brainstem auditory evoked potentials had no additional value in outcome prediction.

the Hypothermia After Cardiac Arrest trial, there was no additional bias in patient selection. One limitation of this study is the relatively small proportion of patients not recovering consciousness. Also, the number of patients whose outcome was poor and who had detectable N20 SEP peaks was small. The power of this study is not sufficient enough to detect a possible correlation between outcome and N20 peak latencies or N20 amplitudes. In normothermic cardiac arrest survivors, hypoxic-ischemic brain damage with poor outcome has been shown to be associated with prolonged latencies and decreased amplitudes of SEP N20 peaks (21). Short-latency SEPs lack the capacity to identify patients whose outcomes will be favorable after cardiac arrest. Long-latency SEPs have been found useful even for this purpose by Austrian investigators (22). Madl et al. have previously shown that in normothermic cardiac arrest patients, a latency >130 msec to cortical evoked potential N70 peak or absent N70 peaks predict poor outcome with a specificity of 97% and sensitivity of 94%. They also found that the N70 long-latency sensory evoked potential was more accurate in predicting individual outcome than was a panel of three experienced emergency physicians reviewing clinical data 24 hrs after cardiac arrest (23). Because of the slowing of neural transmission with hypothermia, the N70 cut-off values determined for normothermic patients cannot be applied to hypothermic patients.

To avoid falsely pessimistic prognosis, tests used in critical care are required to have high specificity for poor outcome,

with less emphasis on sensitivity. Induced hypothermia requires muscle relaxation, sedation, and mechanical ventilation, which may complicate the clinical assessment. Reliable tests predicting unfavorable neurologic outcome for these patients are needed. In a previous study, we found that the use of therapeutic hypothermia reduces the prognostic value of both serum neuron-specific enolase and S-100B protein in predicting poor outcome after cardiac arrest (24). This small study suggests that the prognostic ability of median nerve short-latency SEPs may not be affected by therapeutic hypothermia. Recording of BAEPs did not provide additional benefit in outcome prediction. Larger trials are needed to confirm these findings.

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