Intraoperative Monitoring of the Somatosensory Evoked Potentials and Cerebral Blood Flow During Aneurysm Surgery

—Safety Evaluation for Temporary Vascular Occlusion—

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Abstract

Somatosensory evoked potentials (SEPs) and cerebral blood flow (CBF) were monitored during temporary vascular occlusion in 67 aneurysm operations to evaluate the usefulness of SEP as an indicator for cerebral ischemia. The SEP N₂₀ component completely disappeared during temporary vascular occlusion in 24 cases, 23 of which demonstrated complete recovery following recirculation and had no postoperative neurological sequelae. The only one with postoperative sequelae demonstrated rapid, within 1 minute, loss of N₂₀ followed by no recovery. Another eight cases showed prolongation of central conduction time during vascular occlusion and had no postoperative sequelae. The SEP N₂₀ attenuation reflected the CBF reduction in the middle cerebral artery (MCA) territory during MCA occlusion. However, CBF changes in the internal carotid artery (ICA) territory during ICA occlusion greatly varied. No detectable changes in SEP were found during anterior cerebral artery occlusion for anterior communicating artery aneurysms. This study indicates that intraoperative SEP monitoring is useful to detect ischemia in the MCA territory and that rapid disappearance of the N₂₀ component is a danger signal.

Key words: cerebral aneurysm, temporary occlusion, intraoperative monitoring, somatosensory evoked potential, cerebral blood flow

Introduction

Intraoperative aneurysmal rupture can be prevented by first temporarily occluding the major cerebral vessels and then treating the aneurysmal neck.²⁶⁻²⁹⁾ This technique has increasingly been used recently because of its effectiveness.^{2,6,9,14)} The principal problem with temporary vascular occlusion is the possibility of induced ischemic brain damage. Various techniques are available to protect the brain, including use of the Sendai cocktail containing mannitol, vitamin E, and phenytoin.

Our experimental^{1,25)} and clinical investigations²⁷⁻²⁹⁾ have shown that this agent, given intravenously, allows occlusion of any combination of cerebral blood vessels at any location for up to 40 minutes without resultant brain damage, if the occlu-

sion is completed within 100 minutes after the administration. However, experimental work has also shown that lower tolerance to temporary vascular occlusion results in severer ischemia.^{11,16)} In addition, decreases in cerebral blood flow (CBF) caused by occlusion differ with the sites of occlusion and the individuals, so the allowable duration of temporary vascular occlusion will vary. In our early study,²⁸⁾ postoperative neurological sequelae occurred in only four of 384 cases (1%) receiving mannitol administration to protect the brain, and all four underwent relatively short vascular occlusion (5-20 min).

Cerebral function monitoring during neurosurgical procedures is used more frequently today. Electroencephalographic (EEG) monitoring during temporary occlusion in carotid endarterectomy has been performed.^{3,5,20,22-24)} Since the somatosensory evoked potentials (SEPs) accurately reflect cerebral ischemia and can be evaluated quantitatively more

Received June 20, 1988; Accepted March 29, 1989

easily than EEG, SEP monitoring has also been used during temporary occlusion in both carotid endarterectomy and aneurysm surgery.^{4,7,10,12,15,17,18,21} We therefore studied SEP and CBF changes during aneurysm surgery, particularly during temporary vascular occlusion, and the indications for subsequent ischemic brain damage.

Clinical Materials and Methods

Intraoperative SEP monitoring was possible in 61 of the 86 cases undergoing direct aneurysm surgery between July, 1986 and June, 1987. Five cases had multiple aneurysms, giving a total of 67 aneurysms in this study. Twenty aneurysms were on the middle cerebral artery (MCA), 24 on the internal carotid artery (ICA), 20 on the anterior communicating artery (AcomA), and three on the vertebrobasilar arterial system (VBA). Preoperatively, the Hunt and Kosnik⁸⁾ grades of the patients were 0 in one, 1 in five, 2 in 38, 3 in 13, and 4 in four. Most operations were performed in the acute stage, with 21 cases operated on within 24 hours of ictus. Temporary vascular occlusion was performed in all cases. A summary of the sites and durations is given in Table 1.

The aneurysm site controlled whether the feeding or both the feeding and draining arteries were occluded. However, the present study only examined the occlusion of the feeding arteries. There were no significant differences in the occlusion times with aneurysm site, and the mean occlusion time was 20 ± 10 minutes.

SEPs were monitored with a Neuropak 8 system (Nihon Kohden Corp., Tokyo). In most cases, the

Table 1 Site and duration of temporary vascular occlusion for each aneurysm site

Aneurysm site	No. of aneurysms	Occlusion site*	Occlusion time (min)	
			Range	Mean $\pm$ SD
MCA	20	M ₁ (M ₂)	4-48	20.1 $\pm$ 10.5
ICA	24	ICA (PcomA)	2-52	18.2 $\pm$ 10.9
AcomA	20	both A ₁ (A ₂)	5-38	21.1 $\pm$ 9.1
VBA	3	BA or VA	21-39	28.5 $\pm$ 7.7
Total	67		2-52	20.1 $\pm$ 10.4

*() indicates with or without. Thus M₁ (M₂) indicates M₁ or M₁ + M₂.

SEPs were evoked by stimulating the median nerve at the wrist, but for AcomA aneurysm cases, the posterior tibial nerve was also stimulated. The stimulation parameters for the median nerve were 10-12 mA, 0.2-msec duration, and 4-Hz square waves. A saddle-shaped bipolar stimulator was used to stimulate the bilateral median nerves alternately, and the bilateral SEPs were recorded. The recording electrodes were located at C3' and C4' (international 10-20 system) and the second cervical vertebra (Cv2) and Erb's point. A frontal reference (Fz) was used. The band-pass filters were set to 20-500 Hz for recording cortical components (N₂₀) and to 100-1500 Hz for analyzing short-latency components (N₁₄). The analysis time was 50 msec, and 500 responses were averaged. Following Symon *et al.*,^{30,32)} we have focused on the central conduction time (CCT) and amplitude of the N₂₀ peaks (Fig. 1A). The stimulus parameters for the posterior tibial nerve were 12-15 mA, 0.2-msec duration, and 5-Hz square

Fig. 1 Intraoperative median nerve (A) and posterior tibial nerve (B) SEP recordings. CCT is defined as the interpeak latency between N₂₀ and N₁₄. Line 1: P₃₀ generated from dorsal column nucleus, line 2: N₃₄ generated from medial lemniscal pathway, lines 3 and 4: P₃₈ generated from primary sensory cortex. C3': left sensory cortex, Cv2: second cervical spinous process, Cv7: 7th cervical spinous process, Cz': 2 cm posterior to the Cz, Erb: Erb's point, Fz: frontal vertex.

Fig. 2 Intraoperative CBF monitoring using a thermal diffusion flow probe. In this case, CBF was reduced from 30 to 10 ml/100 gm/min during temporary MCA occlusion (arrows).

waves. The recording and reference electrodes were placed following Urasaki *et al.*³¹⁾ The band-pass filter was set to 100–1000 or 20–500 Hz. The analysis took 70 msec, averaging 500–1000 responses (Fig. 1B). SEP monitoring was started after the induction of anesthesia to minimize preoperative stress and continuously recorded before and after temporary vascular occlusion. All results were stored on floppy disks.

CBF measurements used a thermal diffusion flow probe (BTG-III, Biomedical Inc., Kanazawa).^{13,33)} The CBF probe was placed directly on the cortex in the area of interest after opening the dura mater (Fig. 2).

Results

I. Classification of the SEP changes caused by temporary vascular occlusion

Changes in the median nerve SEPs following temporary occlusion were classified into four grades as shown in Table 2 and Fig. 3A–D. Posterior tibial nerve SEP monitoring only for AcomA aneurysm cases showed no changes, making grading unnecessary (Fig. 3E).

II. Relationship between SEP changes and aneurysm site

The SEP changes during temporary vascular occlusion with different aneurysm sites are summarized in Table 3.

Fifteen (75%) of the 20 MCA aneurysm cases demonstrated Grade 3 SEP changes. The occlusion time in these ranged from 4 to 42 minutes, with a mean of 19.5 minutes. SEP attenuation began 3 to 16 minutes (mean 8.8 min) after the start of vascular occlusion. SEP recovery had already occurred by the first postrecirculation measurement in 11 cases. The other four demonstrated signs of recovery within 8 minutes. All the 15 cases eventually showed complete recovery of N₂₀ amplitude and return to the preocclusion level of CCT and had no postoperative neurological sequelae caused by the temporary vascular occlusion. The duration of N₂₀ absence ranged from 2 to 38 minutes (mean 13.3 min) (Fig. 4A). The other five MCA aneurysm cases demonstrated lower grade changes and subsequent recovery of the SEPs and had favorable postoperative outcomes.

Eight (33%) of the 24 ICA aneurysm cases showed Grade 3 changes. The mean occlusion time was 22.8 minutes. The SEP disappeared at 1–30 minutes (mean 9.5 min) from the start of occlusion. After an average of 4.9 minutes of recirculation, seven of the eight cases showed considerable recovery of the SEPs, which were eventually similar to the preocclu-

Table 2 Classification of SEP changes during temporary vascular occlusion

Grade	Criterion
0	no change
1	<1.0 msec prolongation of CCT, or <50% decrease in N ₂₀ amplitude
2	≥1.0 msec prolongation of CCT, or ≥50% decrease in N ₂₀ amplitude
3	loss of N ₂₀

Table 3 Summary of SEP changes for each aneurysm site

Aneurysm site	Grade of SEP changes			
	0	1	2	3
MCA	2 (24)	1 (6)	2 (27.7)	15 (19.5 ± 10.9)
ICA	11 (11.8 ± 6.8)	5 (24.7 ± 14.0)	0	8 (22.8 ± 8.2)
AcomA	20 (21.1 ± 9.1)	0	0	0
VBA	2 (23.5)	0	0	1 (39)
Total	35 (18.5 ± 9.2)	6 (21.6 ± 14.6)	2 (27.7)	24 (21.4 ± 10.6)

Mean occlusion time ± SD given in parentheses (min).

Fig. 3 A: Median nerve SEP changes, Grade 0. In this case, SEP N_{20} was preserved with no changes in latency and amplitude after 20 minutes of MCA occlusion. B: Median nerve SEP changes, Grade 1. In this case, CCT prolonged from 7.6 to 8.4 msec after temporary occlusion of ICA and returned to the preocclusion level after reperfusion. C: Median nerve SEP changes, Grade 2. This case showed a prolongation of CCT from 6.0 to 7.0 msec and a reduction in N_{20} amplitude of more than 50% of the preocclusion level after temporary MCA occlusion. D: Median nerve SEP changes, Grade 3. In this case, N_{20} disappeared 10 minutes after MCA occlusion and recovered rapidly following recirculation. E: Posterior tibial nerve SEP changes, Grade 0. All AcomA aneurysm cases examined showed no changes during bilateral ACA occlusion.

sion level. The duration of SEP attenuation was between 8 and 37 minutes (mean 17.7 min). The other one case did not show SEP recovery following recirculation. This was the only case in the series to have permanent postoperative sequelae due to the temporary vascular occlusion (Fig. 4B). Vascular occlusion was relatively short (17 min), but the SEP was absent immediately following occlusion. The other 16 ICA aneurysm cases showed Grade 1 or 0 changes

and had favorable postoperative courses.

There were no detectable SEP changes among the 20 AcomA aneurysm cases. The SEPs were elicited by median nerve stimulation in 13 cases and by posterior tibial nerve stimulation in seven. None had postoperative sequelae attributable to the temporary vascular occlusion.

There were two cases with vertebral-posterior inferior cerebellar artery aneurysms and only one with

Fig. 4 Occlusion time, time to N_{20} loss from the start of temporary occlusion, time to N_{20} recovery from the start of recirculation, and time of absent N_{20} (occlusion time - time to N_{20} loss + time to N_{20} recovery) in cases with Grade 3 SEP changes during MCA occlusion (A, $n = 15$) or ICA occlusion (B, $n = 8$). Each closed circle and bar represent mean $\pm$ SD. ■: case with permanent deficit.

a basilar tip aneurysm. The former two cases showed no changes in the SEP due to the temporary occlusion of the vertebral artery (VA). In the latter one, the basilar artery (BA) and the bilateral posterior cerebral arteries (PCAs) were occluded for 39 minutes. The SEP became attenuated after 7 minutes but recovered immediately following recirculation. No sequelae due to the temporary vascular occlusion were evident postoperatively.

III. CBF changes during temporary vascular occlusion

Reliable data were obtained in a total of 17 cases, 10 with MCA aneurysms and seven with ICA aneurysms. In the MCA aneurysm cases, vascular oc-

Fig. 5 Relationship between changes in N_{20} amplitude and CBF induced by temporary vascular occlusion in cases with MCA aneurysm ($\square$, $n = 10$) or ICA aneurysm ($\blacktriangle$, $n = 7$).

Table 4 Summary of CBF changes during temporary vascular occlusion

Occlusion site	No. of cases	CBF (ml/100 gm/min)	
		Preocclusion	During occlusion*
MCA	10	31.8 $\pm$ 16.0	10.5 $\pm$ 5.4 (34.2%)
ICA	7	45.7 $\pm$ 11.8	25.0 $\pm$ 10.7 (55.5%)

*Percent of control given in parentheses.

clusion reduced the CBF to 34.2% of the preocclusion level, but in the ICA aneurysm cases, the CBF decreased to 55.5% (Table 4). The variation in individuals was much greater in the ICA than MCA

aneurysm cases (Fig. 5).

SEP attenuation occurred in nine of the 10 MCA aneurysm cases, during which CBF fell to 18–55% of the preocclusion level. The case without SEP attenuation demonstrated a CBF reduction to 33% during vascular occlusion for 28 minutes. There were fewer ICA aneurysm cases, but some correlation between CBF and SEP changes was evident. The N₂₀ potential tended to disappear as soon as the CBF decreased to 40% (Fig. 5).

Discussion

The N₂₀ component of the median nerve SEP is thought to originate in the cerebral cortex (somatosensory cortex for the upper extremities) or the thalamocortical radiations. Although the origin of the evoked potentials is uncertain, both regions are perfused by the MCA. We found that the N₂₀ component was frequently suppressed during the MCA occlusion and completely disappeared in 75% of the MCA aneurysm cases. In contrast, only 33% of the ICA aneurysm cases receiving the ICA occlusion showed N₂₀ suppression. The blood flow to the MCA territory is thought to be influenced by the degree of collateral circulation through the AcomA and posterior communicating artery (PcomA). The CBF monitoring of the present study supports this view. Following MCA occlusion, the CBF uniformly decreased to about 1/3 of the preocclusion level, but following ICA occlusion, the decrease in CBF was between 0 and 70%, with large individual variations. It should be noted, however, that CBF was measured at one location only. Multiple probes will assess more accurately the effects of occlusion on CBF.

Temporary occlusion of the BA and bilateral PCAs demonstrated N₂₀ attenuation. Since this occlusion reduced the blood flow in the thalamoperforating arteries, *etc.*, the N₂₀ attenuation is thought to be due to disturbances in the thalamocortical neurotransmission. The absence of SEP changes in the few VA cases is probably due to small changes in CBF in the sensory pathways following unilateral VA occlusion.

It is noteworthy that monitoring the N₂₀ component of the median nerve SEP has little significance in AcomA aneurysm cases requiring anterior cerebral artery (ACA) occlusion. We used stimulation of the upper extremities in a few early cases, but no changes in N₂₀ were observed following ACA occlusion. Stimulation of the lower extremities was tried in seven cases, but again no SEP changes occurred. The SEP from the somatosensory area of the

lower extremities was named P₃₈ by Urasaki *et al.*³¹⁾ This cortical region is clearly fed by ACA branches. In our study, however, no changes in the P₃₈ amplitude or latency were demonstrated following ACA occlusion. The reasons for this are thought to be: i) the somatosensory area of the lower extremities is the distal ACA territory, and collateral circulation from the MCA or PCA may be involved when the ACA is occluded; and ii) P₃₈ may reflect activity in subcortical pathways rather than in the primary sensory cortex of the lower extremities. Whatever the cause is, we found that the SEP was inadequate for monitoring cerebral ischemia in the ACA territory. A more suitable technique for monitoring cerebral functions in this region is still required.

The prediction of possible postoperative neurological sequelae is the most important objective of the intraoperative monitoring of cerebral functions. Kidooka *et al.*¹²⁾ found that eight of 13 cases with CCT prolongation by more than 1.2 msec or N₂₀ attenuation developed postoperative symptoms. They considered CCT prolongation by more than 1.2 msec to be critical in temporary vascular occlusion. Momma *et al.*¹⁷⁾ also considered CCT prolongation and N₂₀ attenuation as danger signals during temporary vascular occlusion. In particular, special caution is required if N₂₀ disappears within 3–4 minutes of vascular occlusion. In our series, N₂₀ disappeared 1–30 minutes after the start of vascular occlusion in 24 cases. With one exception, in 23 cases, N₂₀ peaks reappeared following recirculation by the first measurement (within 1 minute), and returned to the baseline recordings 1–8 minutes (mean 2.6 min) in MCA aneurysm cases and 1–20 minutes (mean 4.9 min) in ICA aneurysm cases. None of these 23 cases showed postoperative sequelae. The mean length of disappearance of N₂₀ during temporary occlusion (occlusion time – time to N₂₀ loss, Fig. 4) was 10.7 minutes in MCA aneurysm cases and 13.3 minutes in ICA aneurysm cases. Therefore, it is considered that the postoperative outcome is expected to be favorable if the temporary clip is released within about 10 minutes after attenuation of N₂₀. From our results, the permissible temporary occlusion time was thought to be relatively longer than those of the previous reports.^{11,12)} It might be due to the differences of the methods of brain protection. As before mentioned, we routinely use the Sendai cocktail to protect the brain from ischemic damages during temporary occlusion.^{1,25,26,28,29)} However, the most rapid N₂₀ attenuation, within 1 minute of vascular occlusion, occurred in the case in which SEPs did not recover leaving postoperative sequelae (Fig. 4). This case had a broad-based aneurysm at the

origin of the PcomA from the ICA and received vascular occlusion at four sites: the cervical ICA (17 min), A₁ segment of the ACA (13 min), M₁ segment of the MCA (14 min), and PcomA (12 min). Although the CBF monitoring was not done in this case, it is considered that either severe ischemia in the perforating artery territory or thrombus formation during the temporary occlusion has caused the sequelae.

SEP only monitors the sensory pathways. A more direct method for monitoring motor functions is necessary to prevent postoperative motor deficit.¹⁹⁾ Such techniques are still being developed and are presently more invasive than SEP monitoring. Further technical developments, however, should achieve an effective method for intraoperative monitoring of motor functions.

The present study has found that the N₂₀ component of the median nerve SEP accurately reflects the ischemic state of the MCA territory. This should therefore be useful for intraoperative monitoring of MCA or ICA aneurysm surgery. It is also useful for monitoring the development of stenosis or occlusion of perforating arteries or major vessels secondary to inadequate operative procedures.

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