

Intraoperative monitoring of blood flow insufficiency in the anterior choroidal artery during aneurysm surgery

KYOUICHI SUZUKI, M.D., NAMIO KODAMA, M.D., TATSUYA SASAKI, M.D., MASATO MATSUMOTO, M.D., YUTAKA KONNO, M.D., JUN SAKUMA, M.D., MASAHIRO OINUMA, M.D., AND MASAHIRO MURAKAWA, M.D.

Departments of Neurosurgery and Anesthesiology, Fukushima Medical University, Fukushima, Japan

Object. The lack of a specified intraoperative method for monitoring anterior choroidal artery (AChA) blood flow insufficiency (BFI) led the authors to devise a method for checking the BFI in this artery during aneurysm surgery. To this end, the authors relied on the intraoperative motor evoked potentials (MEPs) elicited by electrical stimulation of the hand motor cortex.

Methods. The study population consisted of 108 patients with internal carotid artery (ICA) aneurysms who underwent surgery via a standard frontotemporal craniotomy. After the dura mater had been opened, a grid electrode strip with 16 small electrodes was inserted subdurally into the hand motor cortex from the edge of the craniotomy. To check BFI in the AChA, the hand motor cortex was stimulated at an intensity level between 10 and 18 mA. The MEPs were successfully recorded from the contralateral thenar muscles in all 108 patients. There was no postoperative motor paresis in 88 patients in whom the MEPs remained unchanged during the performance of various surgical maneuvers. Among the other 20 patients, 19 manifested transient MEP changes, but 15 of those patients experienced no postoperative motor paresis. In four patients who exhibited transient MEP changes, either after aneurysm clipping or during temporary occlusion of the ICA and/or AChA, hemiparesis occurred postoperatively but disappeared within 24 hours. In one patient with an ICA–posterior communicating artery aneurysm, the MEP disappeared and did not reappear by the time of dural closure. Severe hemiplegia developed in this patient and a computerized tomography scan obtained postoperatively revealed a new low-density area in the internal capsule.

Conclusions. The findings of this study suggest that the monitoring method that is introduced here is safe and reliable for detecting intraoperative BFI in the AChA.

KEY WORDS • intraoperative monitoring • motor evoked potential • aneurysm • anterior choroidal artery

It is very difficult to ascertain the patency of the AChA after an aneurysm of the ICA has been surgically clipped.⁹ Some patients have manifested AChA syndrome,^{1,8,9,21,27} even though blood flow in the AChA was apparently confirmed using Doppler ultrasonography with the aid of an operating microscope. At present, there is no reliable intraoperative method for detecting BFI in the AChA.

Our study is based on the concept that the AChA controls the blood flow to the region of the internal capsule. When flow to this region decreases or ceases, the function of the pyramidal tract may be compromised. We posited that if we checked pyramidal tract function by recording MEPs, it would be possible to detect BFI in the AChA intraoperatively.

Using a modified grid electrode, we successfully recorded MEPs at the contralateral thenar muscles on direct elec-

trical stimulation of the hand motor cortex. Our novel electrode can be easily inserted beneath the dura mater on the cortical surface, permitting adequate stimulation of the hand motor cortex without requiring extension of the craniotomy. As there is currently no reliable method for monitoring BFI in the AChA intraoperatively, we tested our hypothesis that changes in MEPs can be used to detect BFI during aneurysm surgery.

Clinical Material and Methods

We monitored MEPs intraoperatively in 108 patients with ICA aneurysms. The trial protocol was reviewed and approved by the local independent ethics committee, and informed consent was obtained from all patients or their legal representatives before enrollment in the study. Anesthesia was induced with a bolus injection of propofol (1.5–2 mg/kg) and fentanyl (2 µg/kg), and maintained by the continuous infusion of propofol (6–10 mg/kg/hr) and an additional injection of fentanyl (2 µg/kg) every 60 minutes. Before tracheal intubation, all patients received a bolus injection of vecuronium bromide (0.1 mg/kg) followed by drip infusion of the same drug (0.07–0.15 mg/kg/hr) to maintain a stable level of neuromuscular blockade. To mon-

Abbreviations used in this paper: ACA = anterior cerebral artery; AChA = anterior choroidal artery; BFI = blood flow insufficiency; CT = computerized tomography; ICA = internal carotid artery; MCA = middle cerebral artery; MEP = motor evoked potential; PCoA = posterior communicating artery; SSEP = somatosensory evoked potential; 3D = three-dimensional.

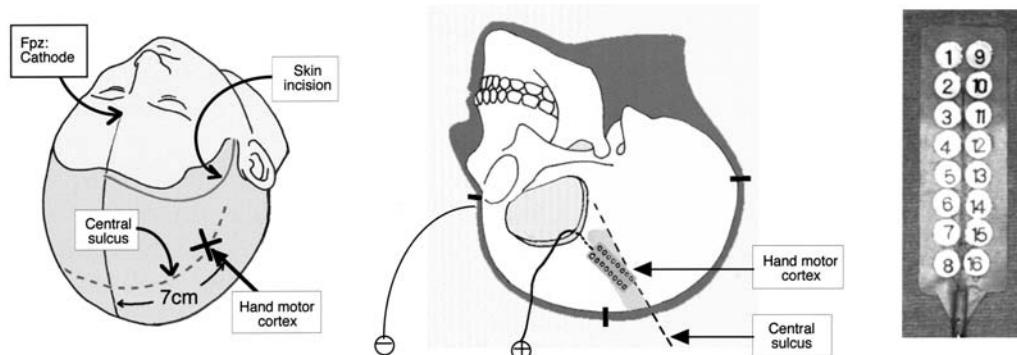


Fig. 1. *Left:* Schematic drawing showing the setup for monitoring MEPs. The central sulcus line is mapped and drawn on the scalp (dotted line). The point 70 mm lateral from the midline on the central sulcus line is marked (X) as the hand motor cortex. *Center:* After a standard frontotemporal craniotomy has been performed, the grid electrode is inserted into the subdural space at the site of the marked hand motor cortex. The cathode needle electrode is inserted at the frontal pole zero electrode location (Fpz). *Right:* Photograph showing the grid electrode used for electrical stimulation of the motor cortex. The strip contains 16 electrodes, each of which measures 3 mm in diameter. The distance between the electrodes is 2 mm.

itor muscle relaxation, we observed the compound muscle action potentials (M wave¹¹) of the abductor pollicis muscles or acceleration of thumb movement after electrostimulation of the ulnar nerve.¹⁰ The amplitude of the M wave was measured before vecuronium bromide administration and maintained at a stable 15 to 25% of the control level during the operation. Alternatively, we used a 2-Hz train-of-four electrical stimulation of the ulnar nerve and measured the acceleration of thumb movement with an acceleration transducer. The train-of-four ratio (acceleration by the fourth stimulation/acceleration by the first stimulation \times 100) was maintained at 1 to 15%. Before a standard frontotemporal craniotomy was performed, the hand motor cortex, which lies 70 mm lateral from the midline on the central sulcus line, was mapped^{2,4,14} and marked, and a grid electrode strip with 16 electrodes, which was especially designed for this study (Unique Medical, Tokyo, Japan), was inserted into the subdural space to facilitate electrical stimulation of the hand motor cortex (Fig. 1). Less than 1 minute was required to insert the subdural electrode, and the procedure did not require extension of the craniotomy. The electrode providing the largest MEP amplitude was chosen for stimulation. To determine the threshold level, the stimulation intensity was increased in a step-wise manner, beginning at 5 mA. Intraoperatively, the motor cortex was stimulated at 2 mA above the threshold level. The cathode was placed at the frontal pole zero electrode location. A monopolar anodal electrical stimulus with five pulses was applied. The frequency of the train pulse was 500 Hz and the duration of each single pulse was 200 μ sec.²³ An electronic stimulator (model 3F46; NEC Medical Systems, Tokyo, Japan) was used. Compound muscle action potentials were recorded from the contralateral thenar muscles by using a pair of subcutaneous stainless-steel needle electrodes (No. 45244; NEC Medical Systems). The filters were at 20 Hz (low band pass) and 3 kHz (high band pass). A signal processor (Syntax 1100; NEC Medical Systems) was used to record the MEPs.

Results

We successfully recorded MEPs in a series of 108 con-

secutive patients harboring ICA aneurysms who underwent surgery at our university between January 1997 and February 2002. The threshold stimulation intensity ranged from 8 to 16 mA. Usually, the responses were recorded as a group of positive and negative deflections. The amplitude and onset latencies of the MEPs were $405.2 \pm 287.3 \mu$ V and 22.2 ± 1.6 msec (means \pm SDs), respectively. Because responses produced different amplitudes and waveforms, the operator received a warning when an MEP disappeared and/or decreased in amplitude to less than 50% of the control level in the course of three or more consecutive recordings.

Of the 108 patients studied, 88 manifested no intraoperative change in MEPs and no postoperative motor paresis. Postoperative CT scans revealed no newly developed low-density areas. In the remaining 20 patients, a reduction or disappearance of MEP amplitude was noted (Table 1). In one of the 20 patients (Case 1), the MEPs disappeared intraoperatively and did not reappear by the time the dura was closed. Postoperatively, this patient experienced permanent hemiplegia. In the other 19 cases, the MEP was reduced or disappeared in response to temporary clipping of the ICA (eight cases), clipping of the aneurysm (six cases), or aneurysm rupture (one case). In four cases this occurred in response to short-term occlusion (< 40 seconds) of the AChA by forceps, a procedure that we used to identify the true AChA when a few similar thin arteries diverted from the ICA^{19,27} close to the aneurysm. When a decrease in amplitude or disappearance of the MEP occurred, the surgical maneuvers inducing the MEP changes were corrected as soon as possible. In 19 patients, this led to the recovery of the MEP and maintenance of its amplitude at the normal range by the time of dural closure.

No postoperative neurological symptoms developed in 15 of these 19 patients (Cases 6–20, Table 1). Four patients (Cases 2–5) manifested transient and mild unilateral hemipareses that disappeared within 24 hours postoperatively. The duration of the decrease in amplitude or disappearance of the MEPs was 8 to 16 minutes (mean 11.5 minutes) in patients with transient postoperative motor paresis and 30 seconds to 12 minutes (mean 4 minutes, 50 seconds) in those without this deficit. In patients with transient postop-

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TABLE 1
*Profiles of 20 patients in whom the MEPs disappeared or decreased in amplitude**

Case No.	Age (yrs), Sex	Location of An	Intraop MEP Changes	Time Until MEP Recovery	Cause of MEP Changes	Postop Motor Paresis (MMT score)	Newly Developed LDA on Postop CT
1	81, F	lt ICA-PCoA	disappeared	permanent	temporary clip on ICA (17 mins)	permanent (2/5)	LDA in internal capsule none
2	75, F	lt ICA-PCoA	disappeared	transient (10 mins)	An clipping	transient (4/5)	
3	25, M	lt ICA-PCoA	disappeared	transient (12 mins)	clipping	transient (4/5)	none
4	62, F	lt ICA-PCoA	disappeared	transient (16 mins)	temporary clip on ICA (3 mins)	transient (4/5)	none
5	55, F	rt ICA (AW)	disappeared	transient (8 mins)	temporary clip on ICA (4 mins)	transient (4/5)	none
6	73, F	rt ICA-AChA	disappeared	transient (8 min)	An clipping	none	none
7	78, F	lt ICA-PCoA	disappeared	transient (12 mins)	An rupture	none	none
8	62, F	rt ICA-AChA	disappeared	transient (1 min)	temporary occlusion of AChA (15 secs) by forceps	none	none
9	83, F	lt ICA-PCoA	disappeared	transient (8 mins)	temporary clip on ICA (3 mins)	none	none
10	77, F	rt ICA-PCoA	disappeared	transient (10 mins)	temporary clip on ICA (2 mins)	none	small LDA in thalamus
11	57, F	rt ICA-AChA	90% decreased	transient (1 min)	temporary occlusion of AChA (30 secs) by forceps	none	none
12	50, F	lt ICA-AChA	80% decreased	transient (3 mins)	temporary occlusion of ICA (60 secs) by forceps	none	none
13	61, M	lt ICA-PCoA	80% decreased	transient (6 mins)	An clipping	none	none
14	64, M	lt ICA (AW)	70% decreased	transient (11 mins)	temporary clip on ICA (2 mins)	none	none
15	49, F	lt ICA-PCoA	60% decreased	transient (3 mins)	An clipping	none	none
16	54, F	lt ICA-AChA	55% decreased	transient (80 secs)	temporary occlusion of AChA (10 secs) by forceps	none	none
17	69, F	rt ICA-bifur	55% decreased	transient (30 secs)	temporary occlusion of AChA (15 secs) by forceps	none	none
18	54, F	lt ICA-PCoA	50% decreased	transient (2 mins)	temporary occlusion of ICA (40 secs) by forceps	none	none
19	74, M	rt ICA (PW)	50% decreased	transient (3 mins)	An clipping	none	none
20	57, F	rt ICA-AChA	50% decreased	transient (3 mins)	temporary occlusion of ICA (60 secs) by forceps	none	none

* An = aneurysm; AW = anterior wall; bifur = bifurcation; LDA = low-density area; MMT = manual muscle test; PW = posterior or wall.

erative hemiparesis, the duration of MEP changes tended to be long. In Case 5, however, in which the patient experienced transient postoperative hemiparesis, MEP deterioration lasted only 8 minutes, and in Case 7 in which the patient did not experience hemiparesis the change in MEP lasted 12 minutes. On the basis of our current data we could not identify a duration of MEP changes that is critical for the development of postoperative hemiparesis.

We encountered system failure (false-positive findings) in only one patient (Case 9), an 83-year-old woman. Her MEP disappeared after cerebrospinal fluid was suctioned through the Liliequist membrane. We posit that the grid electrode became detached from the brain surface due to slack. After we inserted a cottonoid between the dura mater and the electrode for reattachment, the amplitude of this patient's MEP recovered to the control level and she manifested no postoperative neurological symptoms. In two of 20 patients with intraoperative changes in MEP, postoperative CT scans revealed new infarctions. In Case 1, the pa-

tient's MEP disappeared and permanent hemiparesis developed. In Case 10 we observed thalamic infarction that was probably due to BFI in the perforating arteries arising from the PCoA. That patient's MEP disappeared for 10 minutes and postoperatively, transient hemiparesis developed. All 88 patients in whom there was no evidence of MEP changes were free from new infarction on postoperative CT scans. We encountered no complications such as convolution or subdural hematoma in any of the 108 patients.

Illustrative Cases

Case 1

In this 81-year-old woman who had suffered a subarachnoid hemorrhage, 3D CT angiography demonstrated an aneurysm at the junction of the left ICA-PCoA (Fig. 2). A standard frontotemporal craniotomy was performed and, intraoperatively, the aneurysm ruptured. After temporary oc-

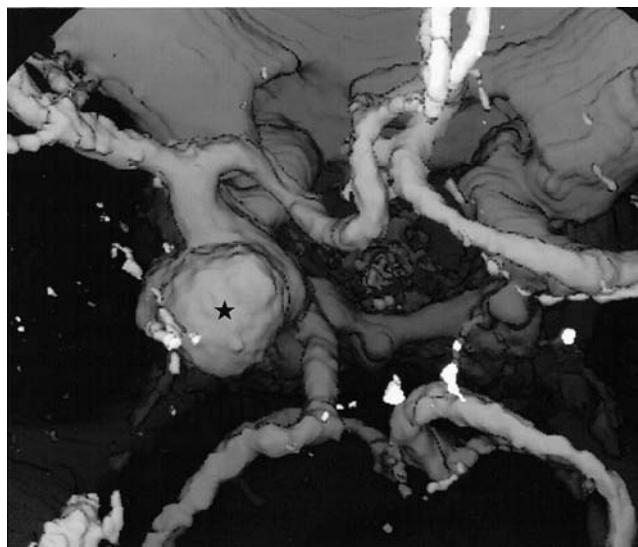


FIG. 2. Case 1. Preoperative 3D CT angiogram (superior view) revealing a left ICA-PCoA aneurysm (star).

clusion (17 minutes) of a proximal portion of the ICA, the aneurysm was clipped. The patient's MEP disappeared after this temporary occlusion and did not reappear by the time of dural closure (Fig. 3). According to the findings on Doppler ultrasonography, the AChA was intact and normal. Postoperatively, right hemiplegia developed in this patient and a CT scan revealed a low-density area in the internal capsule (Fig. 4).

Case 3

This 25-year-old man harbored an unruptured left ICA-PCoA aneurysm (Fig. 5) that was so large that multiple clip applications were necessary for its occlusion. The proximal portion of the AChA was involved in the aneurysm wall. The patient's MEP remained unchanged until the third clipping procedure and disappeared after the fourth clipping procedure. At that point, the AChA appeared white under the surgical microscope, suggesting that blood flow had ceased as a result of some pulling effect on the aneurysm wall due to the clips. After removal of all clips, the MEP reappeared and its amplitude recovered to the control level within 10 minutes. We observed that the blood recirculated in the AChA. Then, we temporarily occluded the left ICA, the A₁ segment of the left ACA, and the M₁ segment of the left MCA. The MEP disappeared again following these procedures, but reappeared immediately after the temporary occlusion was released. The aneurysm was occluded using six fenestrated clips and the amplitude of the MEP remained at the control level until dural closure (Fig. 6). Postoperatively, this patient experienced right hemiparesis that lasted 1 day. He was discharged without any neurological dysfunction. A postoperative CT scan revealed no newly developed low-density area, and patency of the AChA was confirmed angiographically (Fig. 7).

Case 19

In this 74-year-old man with subarachnoid hemorrhage, 3D CT angiography demonstrated a saccular aneurysm that

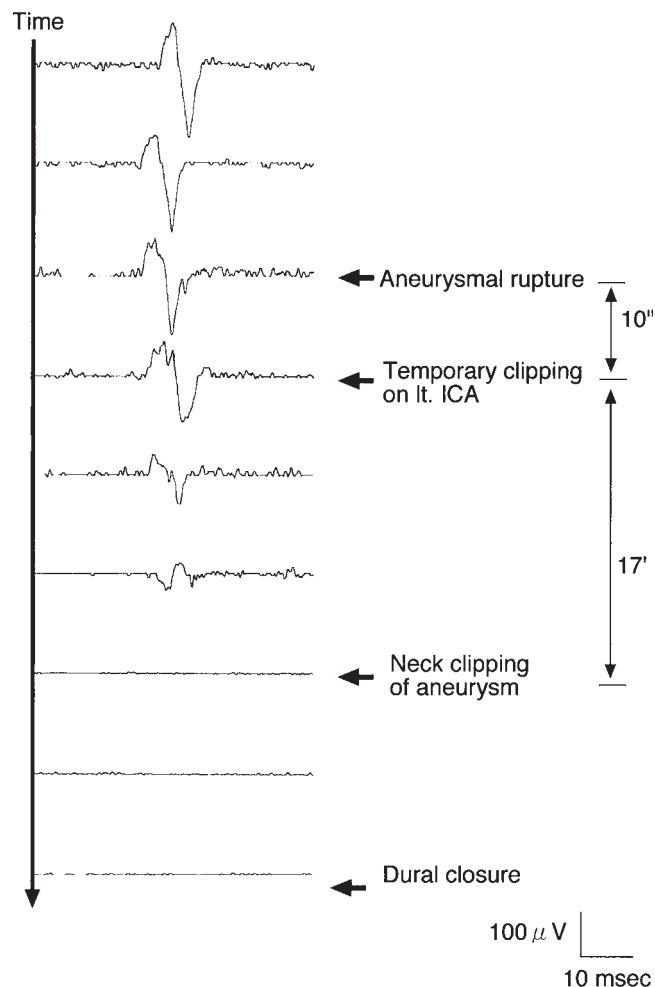


FIG. 3. Case 1. Intraoperative MEP findings. After a premature rupture of the aneurysm and a temporary occlusion of the left ICA, which lasted 17 minutes, the aneurysm was clipped. The MEP disappeared immediately after aneurysm rupture and did not reappear by the time of dural closure.

projected posteromedially from the posterior wall of the dilated right ICA (Fig. 8 *upper left*). A standard frontotemporal craniotomy was performed. Because we were unable to visualize the AChA directly, we used a micromirror (Fig. 8 *upper right*). Two fenestrated clips were used to treat the aneurysm. The MEP amplitude decreased to 50% of the control level approximately 20 seconds after the second clip had been placed and remained there for 2 minutes. Because the AChA was stenotic (Fig. 8 *lower left*), we repositioned the second clip and the patient's MEP recovered to the control level within 40 seconds. The AChA was confirmed to be intact (Fig. 8 *lower right*). On reappearance of the MEP, its amplitude remained at the control level until closure of the dura (Fig. 9). A postoperative CT scan did not reveal any abnormal findings.

Discussion

The effects of occlusion of the AChA were described in

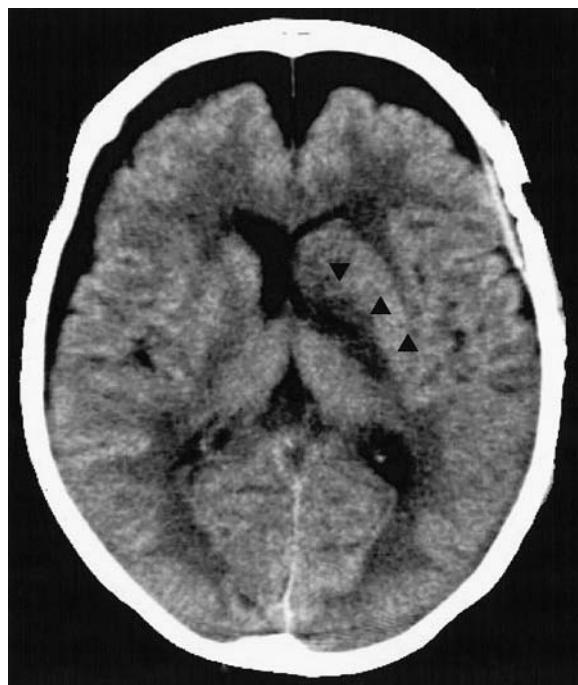


FIG. 4. Case 1. Postoperative CT scan revealing a low-density area in the internal capsule, which is supplied by the AChA (arrowheads).

1925 by Foix, et al.,⁸ as contralateral hemiplegia, hemianesthesia, and hemianopsia. Abbie¹ later attributed the contralateral hemiplegia to infarction in the posterior two thirds of the posterior limb of the internal capsule and in the middle one third of the cerebral peduncle. In 1954, Cooper,⁷ who applied clips to the AChA in 34 patients with Parkinson disease, reported that hemiplegia developed in only two of those patients. On the other hand, there are later reports of AChA syndrome following aneurysm surgery.^{9,21,27} This discrepancy is perplexing and two explanations come to mind. There have been reports of the AChA arising as a duplicate or double artery from the ICA.^{19,27} Under these conditions, sacrifice of one AChA may not induce hemiplegia because the other AChA continues to supply blood to the internal capsule. Alternatively, as suggested by Yaşargil, et al.,²⁷ and Friedman and colleagues⁹ the full utilization of potential collateral routes of blood flow is often hampered by the presence of vascular spasm, diminished hemispherical blood flow, and altered autoregulation after aneurysmal subarachnoid hemorrhage. We contend that the frequency of hemiparesis following AChA occlusion during aneurysm surgery is actually much higher than was suggested by the report of Cooper.

Methods such as electroencephalography,¹⁵ SSEP monitoring,^{15,17} microvascular Doppler ultrasonography,²⁰ and intraoperative angiography^{3,6,22} have been used for the identification of BFI; however, no reliable technique for monitoring BFI in the AChA has been established to date. Electroencephalography is useful for detecting insufficiency in the major arteries, for example, during carotid endarterectomy.¹⁵ Monitoring of SSEPs is not an effective indicator of ischemia, unless the ischemic zone includes the sensory pathways. Mizoi and Yoshimoto¹⁷ reported on three pa-

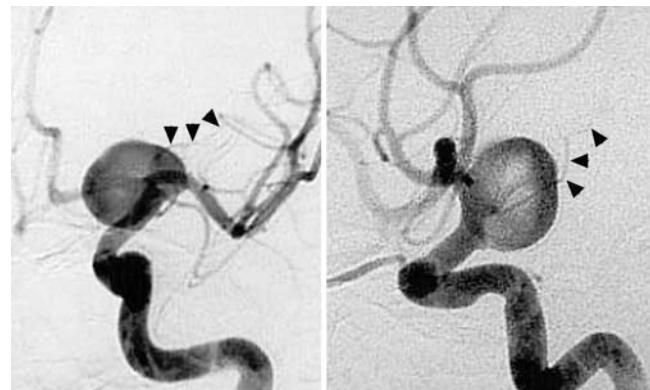


FIG. 5. Case 3. Preoperative angiograms demonstrating a large left ICA–PCoA aneurysm and the AChA (arrowheads).

tients who exhibited postoperative sensorimotor deficits, although their SSEPs had remained unchanged throughout surgery. The AChA has been reported to arise as a duplicate or double artery from the ICA.^{19,27} In these cases, confirmation of AChA blood flow by microvascular Doppler ultrasonography and/or intraoperative angiography does not guarantee blood flow in the internal capsule and cerebral peduncle. We considered three causes of the new infarction of the internal capsule in Case 1, despite Doppler ultrasonographic confirmation of AChA patency. 1) Although Doppler ultrasonography can detect blood flow per se, based on the information it provides it is impossible to determine whether the flow is sufficient to avoid infarction. 2) There are often two branches of the AChA.^{19,27} It is likely that the first branch, whose blood flow was confirmed by Doppler ultrasonography, did not supply the internal capsule. Another branch supplying the internal capsule, which happened to be unchecked, was occluded by the applied aneurysm clip. 3) Infarction of the internal capsule might have been caused by the 17-minute temporary occlusion of the ICA.

We suggest that monitoring of the pyramidal tract by recording the MEP intraoperatively is an excellent method for guarding against postoperative motor paresis due to BFI in the AChA. As the motor cortex is not exposed by a standard frontotemporal craniotomy, its direct stimulation is difficult. The distance between the edge of the craniotomy and the hand motor cortex is approximately 4 cm. Therefore, we designed a grid electrode strip with 16 electrodes measuring 4 × 2 cm. This can be inserted from the edge of the craniotomy into the subdural space, thus facilitating direct stimulation of the motor cortex. Before surgery, we marked the scalp to identify the site of the hand motor cortex and then introduced the grid electrode subdurally under the marked area. This technique facilitated the correct placement of the electrode and direct stimulation of the cortex.

Transcranial electrostimulation^{18,25} and magnetic stimulation^{24,25} have been used to stimulate the brain; however, these methods are not suitable for aneurysm surgery. The area activated by transcranial electrostimulation remains uncertain. Yamamoto, et al.,²⁶ reported that the lower portion of the medulla oblongata is the stimulation point. If the stimulation reaches the medulla oblongata, this method cannot be used to detect deficits in either the motor cortex

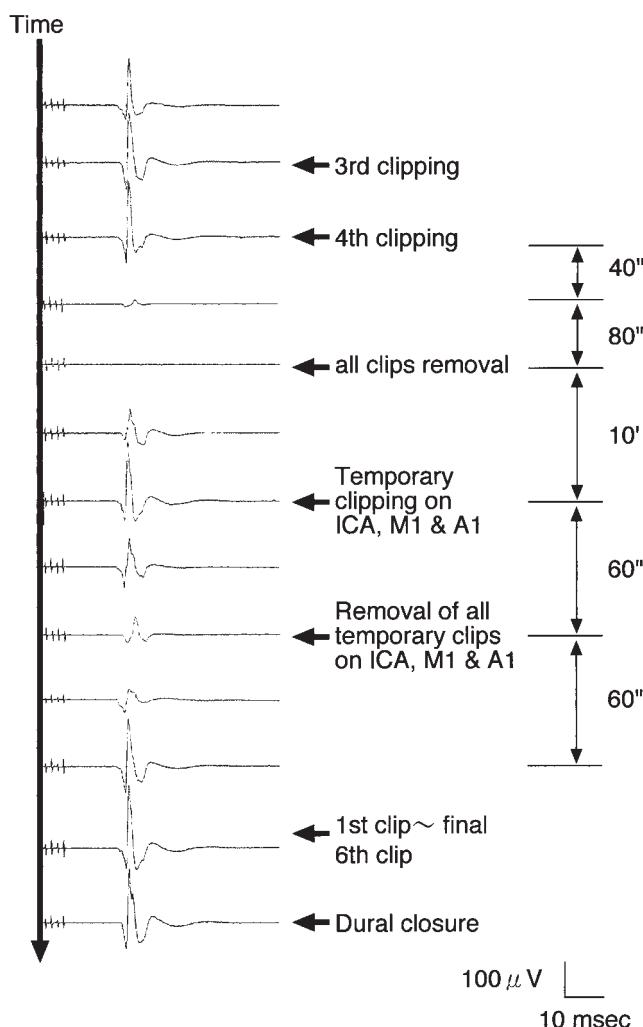


FIG. 6. Case 3. Intraoperative MEP findings. The MEP remained unchanged until placement of the third clip, disappeared after placement of the fourth clip, and reappeared after all clips had been removed. The MEP decreased in amplitude after temporary clipping of the left ICA, the A₁ segment of the left ACA, and the M₁ segment of the left MCA. It recovered after release of the temporary clips. Finally, the aneurysm was occluded using six fenestrated clips and the amplitude of the MEP remained at the control level until dural closure.

or the internal capsule. Transcranial magnetic stimulation is also unsuitable, because it is difficult to achieve constant stimulation of the motor cortex²⁴ and because the stimulated area remains uncertain. In addition, the effect of metal clips on magnetic stimulation cannot be ignored. Induced by transcranial magnetic stimulation, the MEPs were abolished in 43 to 86% of patients during induction of anesthesia with propofol, etomidate, methohexitol, or thiopental.²⁴ Monitoring of MEPs in patients in a state of anesthesia is hampered by the conduction block of lower motor neurons by anesthetic agents. Anesthetic gases, such as nitrous oxide, halothane, and isoflurane, block conduction at the synapse in the anterior horn of the spinal cord.^{16,28} To avoid an inhalant-induced conduction block, MEPs at cervical levels

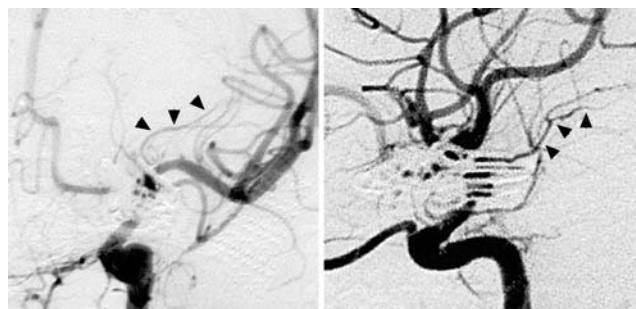


FIG. 7. Case 3. Postoperative angiograms clearly demonstrating the left AChA (arrowheads).

are generally recorded via epidural electrodes.^{12,26} The insertion of catheter electrodes into the cervical epidural space is invasive, however, and it cannot be performed safely, especially in patients undergoing aneurysm surgery during the acute stage.

Two major breakthroughs improved this situation. One is the use of propofol,¹⁰ which produces little synaptic conduction block activity,¹³ and the other is the modification of stimulating methods. Taniguchi and associates²³ elicited MEPs from target muscles in the upper limb with high-frequency repetitive electrical stimulation by applying an anodal rectangular pulse directly to the motor cortex; this brought the α -motor neuron to the firing threshold by the temporal summation of excitatory postsynaptic potentials.

The safety of electrical stimulation of the human brain has been investigated. Taniguchi, et al., concluded that high-frequency repetitive stimulation is relatively safe. Cedzich, et al.,⁵ who restricted the stimulation intensity to within 20 mA, encountered no complications in 99 patients in whom tumors were located near the central sulcus. In our series, the threshold of evoked MEPs was less than 16 mA and we observed no complications attributable to cortical stimulation. Cedzich, et al., found that patients in whom intraoperative MEPs disappeared transiently experienced transient and/or mild postoperative hemiparesis, whereas patients in whom MEPs disappeared without reappearing suffered permanent hemiparesis. In our series of 108 patients, 88 patients in whom the MEPs remained unchanged experienced no postoperative motor paresis. In 19 of the remaining 20 patients, the MEPs disappeared and/or decreased transiently after aneurysm clipping, aneurysm rupture, and temporary occlusion of the ICA and/or AChA. After clip repositioning and/or release of the temporary occlusion, the MEPs reappeared. Although four of these 19 patients experienced transient postoperative motor paresis, the other 15 did not. It is possible that without MEP monitoring, all 19 patients could have suffered permanent postoperative motor paresis.

Based on the results of our study, we suggest that the cause of intraoperative changes in MEPs should be determined immediately and removed as soon as possible. In patients in whom we detected transient changes in MEPs and recovery of MEP amplitudes to the control level by the time of dural closure, postoperative hemiparesis was transient, if it occurred at all. In our study of 108 patients there were no false-negative findings. Therefore, we propose that this method is reliable, easy, minimally invasive, and highly

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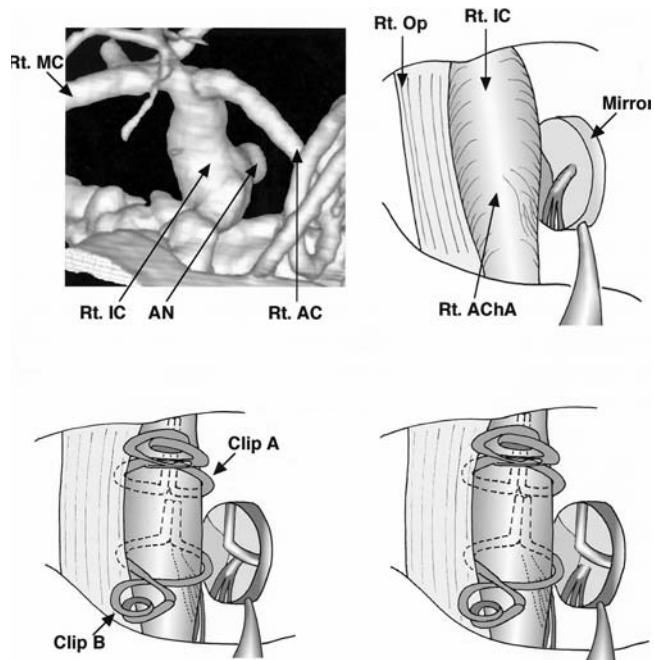


FIG. 8. Case 19. *Upper Left:* A 3D CT angiogram (anteroposterior view) revealing a right ICA aneurysm. *Upper Right:* Drawing showing how the AChA was visualized using a micromirror. *Lower Left:* Drawing demonstrating how the aneurysm was clipped using two fenestrated clips. The proximal portion of the AChA was stenotic. *Lower Right:* Drawing showing repositioning of the second clip, which released the stenosis of the AChA. The artery was confirmed to be intact. AC = ACA; IC = ICA; MC = MCA; Op = optic nerve.

useful. It can be used to detect BFI not only in the AChA but also in the ICA and MCA. In fact, using this method, we were able to detect BFI in these major arteries in many patients undergoing surgery (unpublished data). In this report, we chose to focus on the AChA because there has been no reliable method to detect BFI in this vessel intraoperatively. This is the first reported method for detecting ischemia in the AChA territory. It is of predictive value in assessing treatment outcomes in patients who undergo surgery for aneurysms. Basic studies are necessary to illuminate the mechanism(s) underlying the observed changes in MEPs.

Conclusions

We evaluated intraoperative changes in MEPs in 108 patients with ICA aneurysms. Our findings indicate that MEP changes can be used to detect BFI in the AChA. Our method is simple and minimally invasive; it is therefore ideally suited for detecting BFI in the AChA during aneurysm surgery. It can also be used to monitor blood flow in patients undergoing surgery for arteriovenous malformations and tumors in the frontal base.

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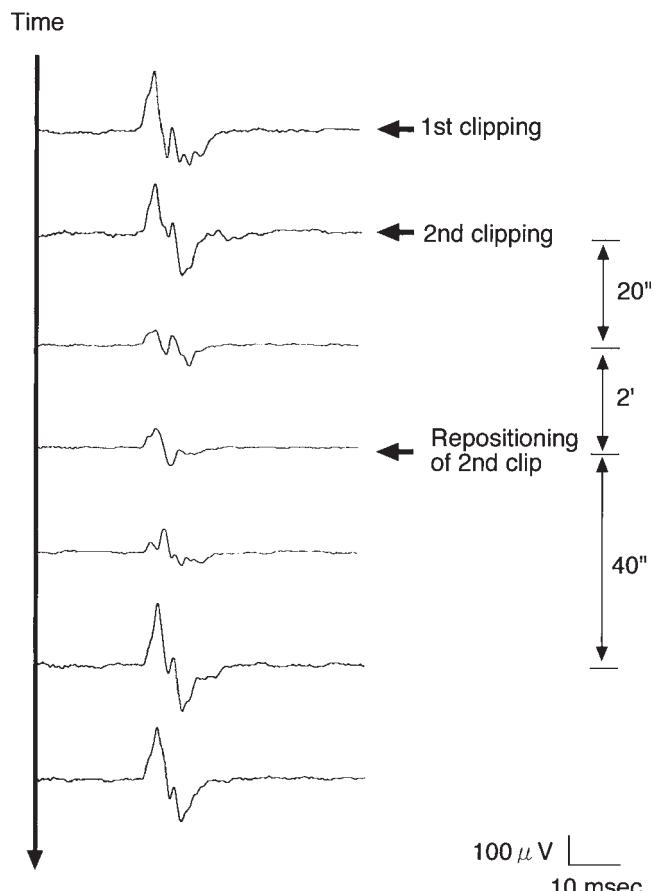


FIG. 9. Case 19. Intraoperative MEP findings. The aneurysm was clipped using two fenestrated clips. The MEP amplitude decreased approximately 20 seconds after the second clip was placed and this decrease persisted for 2 minutes. Repositioning of the clip released the stenosis of the AChA. The amplitude of the MEP recovered to the control level within 40 seconds after repositioning of the clip and remained at the control level until dural closure.

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Address reprint requests to: Kyouichi Suzuki, M.D., Department of Neurosurgery, Fukushima Medical University, 1 Hikarigaoka, Fukushima 960-1295, Japan. email: kyon@fmu.ac.jp.