

Superselective Provocative Test with Propofol Using Motor-Evoked Potential Monitoring for Managing Cerebral Arteriovenous Malformations Fed by the Anterior Choroidal Artery

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When feeder artery obliteration is performed via an endovascular procedure to treat cerebral arteriovenous malformation (AVM), it is important to prevent the ischemic complications that are associated with feeder occlusion. A provocative test may be beneficial in some cases to protect against ischemic complications. We report the case of a 57-year-old man who developed an intracerebral hematoma in the left internal temporal lobe and who had an AVM with a varix in the ambient cistern, which was primarily fed by a branch of the left anterior choroidal artery (AChA) and the posterior lateral choroidal artery, and drained into the basal vein. Therefore, we planned endovascular obliteration of the AChA, followed by gamma knife radiosurgery for the residual posterior component. The patient underwent a superselective provocative test with a 3 mg of propofol under general anesthesia using motor-evoked potential monitoring. The feeder embolization was performed as planned after the provocative test, and the patient exhibited no neurological deficits, such as hemiparesis, during the procedure. **Key Words:** Motor-evoked potentials—cerebral arteriovenous malformations—superselective provocative test—embolization.

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Treatment of cerebral arteriovenous malformation (AVM) typically involves surgery or endovascular techniques, although the obliteration of a feeding artery or branch that supplies the corticospinal tract can lead to post-therapy motor deficits.^{1,2} The complication rate for this tech-

nique has been reported to be 10%-50%, and cerebral ischemia is a frequent major complication that is associated with feeder occlusion.³ Therefore, the provocative test may be beneficial in some cases to protect against the ischemic complications that are caused by arterial embolization.^{4,6} However, these pre-embolization tests have only been performed under local anesthesia or conscious sedation. We present a case in which we used the superselective provocative test with motor-evoked potential (MEP) monitoring under general anesthesia.

Case Report

A 57-year-old man who was taking medication for hypertension suddenly developed a headache and was admitted to a local hospital. Computed tomography and

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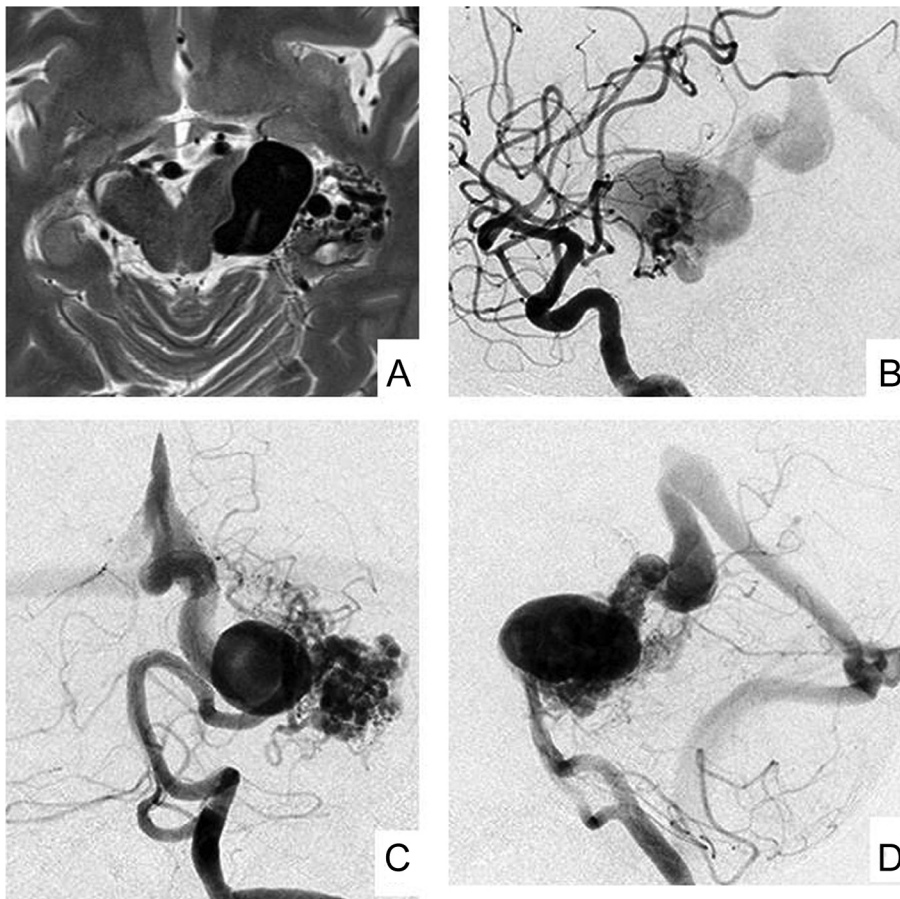


Figure 1. Preoperative magnetic resonance imaging reveals a cerebral AVM that is located in the left hippocampus and a large varix in the ambient cistern (A). The main feeders are the left anterior choroidal artery, as revealed on left internal carotid angiography (B), and the posterior/lateral choroidal artery, as revealed on left vertebral angiography (C and D). Because the AVM had deep venous drainage and the size of the nidus was approximately 3 cm, we graded it as Spetzler–Martin grade III. Abbreviation: AVM, arteriovenous malformation.

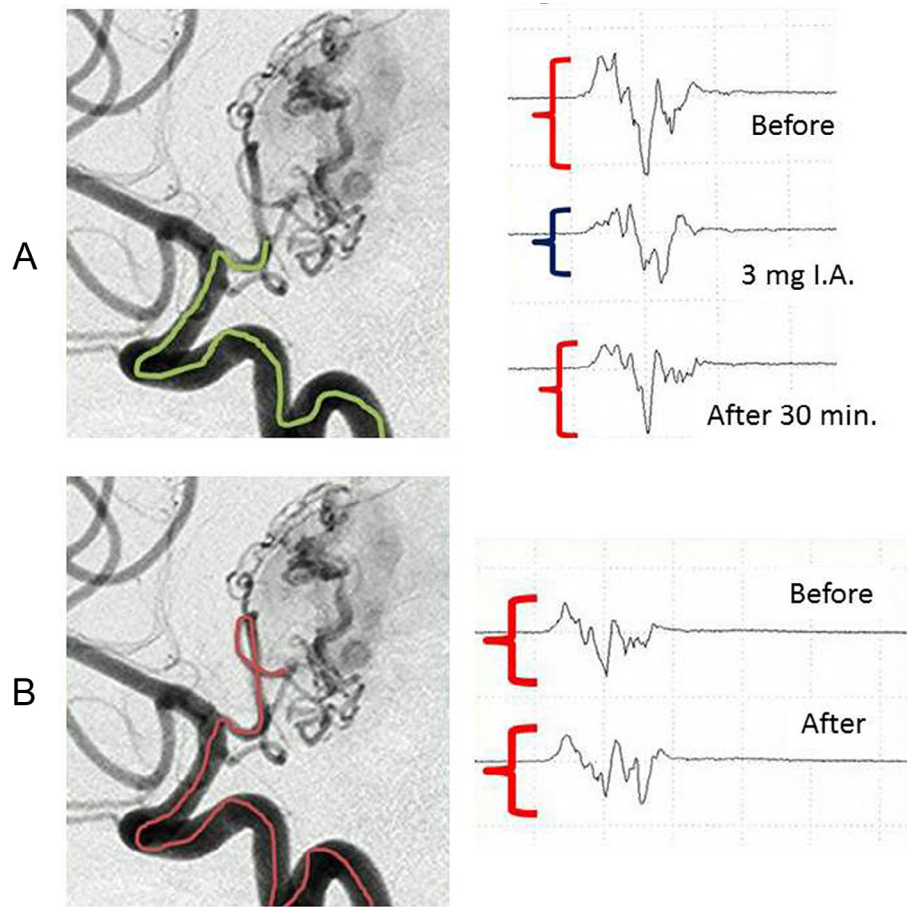
magnetic resonance imaging revealed an intracerebral hematoma in the left internal temporal lobe and abnormal flow void intensity in the left hippocampus. The patient was referred to our institution for treatment and underwent digital subtraction angiography, which revealed an AVM with a varix in the ambient cistern, which was fed by a branch of the left anterior choroidal artery (AChA), the posterior temporal artery (PTA), and the lateral posterior choroidal artery (LPChA) that drained into the basal vein (Fig 1). Although the size of the nidus was approximately 3 cm, it was also slightly diffused, with the anterior component fed by the AChA and the posterior component fed by the PTA and LPChA. This AVM was located in the eloquent brain area, had deep venous drainage, and was graded as Spetzler–Martin grade III. Therefore, we planned endovascular obliteration of the anterior component, followed by gamma knife radiosurgery for the residual posterior component.

Procedures for MEP Monitoring and Embolization

The Neuropack system (Nihon Kohden, Tokyo, Japan) was used for the MEP monitoring. The screw-shaped electrodes for stimulation were attached to the skin of the head on the bilateral motor area under general anesthesia. The plate electrodes for detecting the electromyogram

were attached to the bilateral thenar muscles and tibialis anterior muscles. The stimuli were 5 train pulses at 500 Hz, and a high voltage above 400 V was used. The patient underwent embolization via a transfemoral approach under general anesthesia and was administered heparin IA to achieve a periprocedural activated clotting time of approximately 250 seconds. The left internal carotid artery was then engaged using a 6-Fr Chaperon (Terumo Corporation, Tokyo, Japan) guiding catheter, and a Marathon (Covidien Ltd., Dublin, Ireland) flow-directed microcatheter was advanced to the main trunk of the AChA. A pre-embolization neurological assessment was performed using propofol (1 mg/mL intra-AChA) at 3-mg doses under corticomypogenic MEP monitoring, and we observed that the MEP amplitude fell to less than 50%, compared to the control (Fig 2). Next, a microcatheter was advanced into the pedicle near the AVM nidus, although the MEP amplitude did not change when the same assessment was performed (Fig 3). Therefore, a .07 mL mixture of n-BCA (20%) and ethiodized oil (80%), which contained ethyl esters of iodized fatty acids from poppy seed oil, was injected for occlusion of the feeding artery and part of the AVM nidus. The embolization proceeded as planned, and the patient exhibited no neurological deficits, such as hemiparesis. After the embolization, the patient underwent gamma knife surgery

Figure 2. Propofol (1 mg/mL) was injected at 3-mg doses into the AChA under corticomyogetic MEP monitoring. The MEP amplitude decreased by less than 50%, compared to the control (A). A microcatheter was advanced into the pedicle near the AVM nidus. The MEP amplitude did not change when the same assessment was performed. The AChA that was feeding the AVM nidus was occluded using n-butyl cyanoacrylate (B). Abbreviations: AChA, anterior choroidal artery; AVM, arteriovenous malformation; MEP, motor-evoked potential.



for the remaining components of the AVM and has been followed up at our hospital for 2 years after gamma knife surgery. The patient had no complication.

Discussion

Endovascular therapy for AVM is helpful in the preoperative or preradiosurgical setting for patients with AVM. In the present case, we could concentrate on the therapeutic procedures because the patient could not move during embolization due to the general anesthesia and his vital signs (such as blood pressure, heart rate, body temperature, and respiratory rate) being constantly monitored by an anesthesiologist.

Therefore, embolization under general anesthesia may be a safer or more useful procedure, compared to that performed under local anesthesia or conscious sedation, especially for embolization of narrow arteries such as the AChA.

As to the drug used in the provocation test, short-acting barbiturates such as amobarbital or pentobarbital were used frequently in the past.^{6,7} Lidocaine was mainly used in testing the peripheral nerve function,⁸ although there is a report of its intracranial concomitant use with amobarbital to reduce false-negative results.⁹

Propofol became one of the frequent anesthetic agents used in intra-arterial infusion for intracranial testing recently, as its efficacy and safety in inducing controlled transitory losses of neurological function in the perfused areas including those of small arteries were reported.^{4,5,10} Doses as low as 10 mg have been used for intracarotid provocative testing,^{5,10} and Feliciano et al⁴ reported that a 7 mg dose would be sufficient to induce a deficit in a smaller volume of distribution such as that of the distal cortical arteries. They reported that a 7 mg dose would be sufficient to induce a deficit in a smaller volume of distribution, such as that of the distal cortical arteries.⁴

In the present case, we used corticomyogetic MEP monitoring to facilitate the objective evaluation of the pre-embolization provocative test under general anesthesia for a patient with AVM that was mainly fed by the AChA. We observed a decreased corticomyogetic MEP amplitude of less than 50%, which indicated motor disturbance, when 3 mg doses of propofol were injected into the AChA from the microcatheter. A 3 mg dose was thought to be sufficient in this case because the AChA, the target artery in this procedure, has one of the smallest volumes of distribution (similar to the perforators). The optimal concentration and dose of propofol should be evaluated in future studies with a larger sample size.

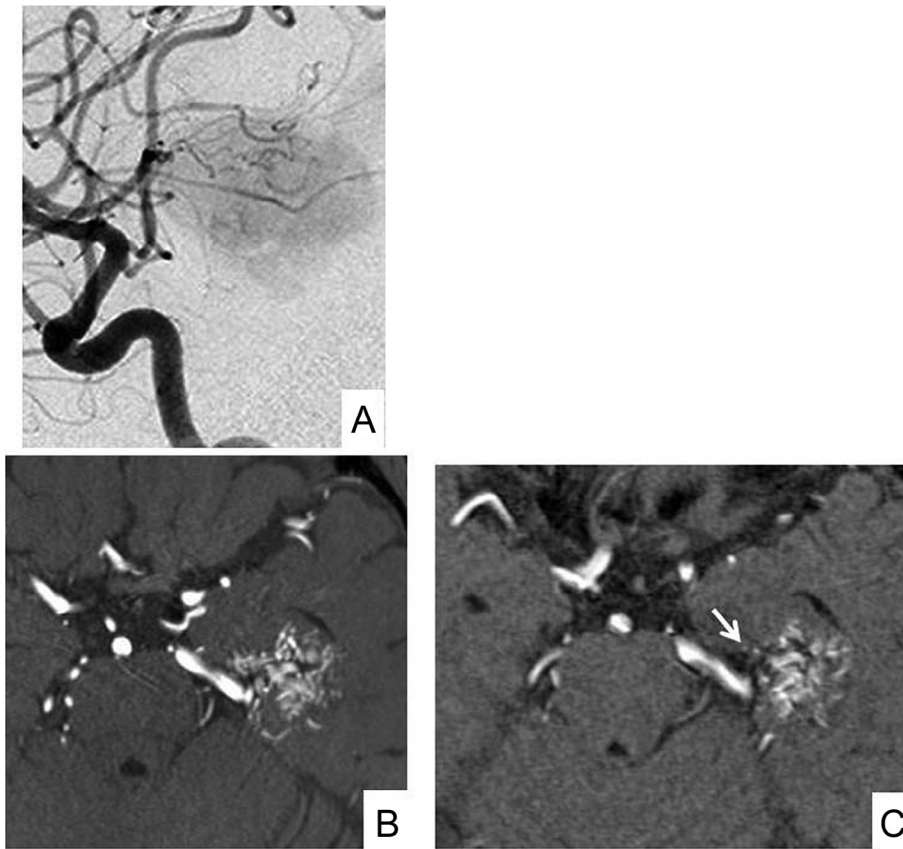


Figure 3. Postprocedural left internal carotid angiography reveals that the nidus that was fed by the anterior choroidal artery had disappeared (A). Magnetic resonance imaging after embolization (C) revealed that the volume of the arteriovenous malformation nidus had decreased (white arrow), compared to the pre-embolization state (B). The patient exhibited no neurological deficits during these procedures.

Previous studies have reported the usefulness of corticospinal MEP monitoring in the management of spinal AVM.¹¹ In this context, corticospinal MEP reflects the conductivity of the corticospinal tract more directly than corticomylogenic MEP, although this method is also more invasive, because it requires the insertion of plate electrodes into the epidural space. In the present case, corticomylogenic MEP monitoring may have been as useful as corticospinal MEP monitoring because the setup was simpler, and a recent study has reported high accuracy for predicting ischemic complications.¹²

In the cisternal segment of the AChA, some perforators feed the eloquent area such as the midbrain or the basal ganglia.¹³ Generally, if the tip of the microcatheter is in a terminal vessel to the AVM beyond the choroidal point, no complications may be expected unless upstream reflux occurs accidentally. However, in embolization therapy of the AChA, safety may not be ensured by anatomical knowledge only.

In this case, superselective administration of propofol from the orifice of AChA led to the decrease in MEP amplitude, indicating that propofol worked pharmacologically in this vascular territory. On the contrary, decrease in MEP amplitude did not occur when propofol was injected within the feeding artery, which was downstream the AChA. Thus, embolization of the feeder was performed. Care should be taken that the possibility of false-negative results cannot

be eliminated as this is a result from a single case, and this superselective provocative test with MEP under general anesthesia should be interpreted with other complementary information such as vascular anatomy until sufficient accumulation of experiences.

In summary, superselective provocative test with propofol under general anesthesia by using MEP monitoring may be useful as a supporting diagnostic method to determine the anatomy before embolization of a cerebral AVM, which was especially valid for the present case.

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