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Embolization of a Spinal Arteriovenous Malformation: Correlation between Motor Evoked Potentials and Angiographic Findings: Technical Case Report

Author(s): Sala, Francesco M.D.; Niimi, Yasunari M.D.; Krzan, Matevz J. M.D.; Berenstein, Alex M.D.; Deletis, Vedran M.D., Ph.D.

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Institution(s): Division of Intraoperative Neurophysiology (FS, MJK, VD) and Center for Endovascular Surgery (YN, AB), Institute for Neurology and Neurosurgery, Beth Israel Medical Center, Singer Division, New York, New York

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Reprint requests: Francesco Sala, M.D., Division of Intraoperative Neurophysiology, Institute for Neurology and Neurosurgery, Beth Israel Medical Center, Singer Division, 170 East End Avenue, New York, NY 10128.

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OBJECTIVE AND IMPORTANCE: Endovascular procedures for the treatment of spinal arteriovenous malformations place the spinal cord at risk of ischemia. This report illustrates the usefulness of motor evoked potentials (MEPs) in detecting functional changes within the spinal cord motor pathways during embolization of a spinal arteriovenous malformation under general anesthesia.

CLINICAL PRESENTATION: A 28-year-old man presented with a history of progressive lower extremity numbness and weakness followed by bladder dysfunction. Magnetic resonance imaging and angiography disclosed a T11-T12 spinal arteriovenous malformation.

INTERVENTION: During the endovascular procedure, before injection of particles, the disappearance of MEPs from the tibialis anterior muscle led to prompt angiographic reevaluation, which disclosed the arrest of spinal blood flow secondary to radiculomedullary artery occlusion by the catheter. Embolization and catheter withdrawal were followed by temporary recovery of spinal blood flow and MEPs. A second arrest of spinal cord blood flow, caused by severe vasospasm of the feeding radiculomedullary artery, was documented by a control angiogram, and its functional relevance was revealed by a second disappearance of MEPs. The therapeutic effect of papaverine infusion and induced moderate hypertension was confirmed angiographically by complete reopacification of the anterior spinal artery and confirmed neurophysiologically by the complete recovery of MEPs. At the end of the procedure, no additional neurological deficits were noted.

CONCLUSION: During spinal cord embolization, MEPs may play a critical role in early detection of spinal cord dysfunction by aiding in the prevention of damage to the spinal cord as well as by predicting the clinical outcome.

Therapeutic options for the treatment of spinal arteriovenous malformations (AVMs) include endovascular embolization, surgery, or both (9, 16, 17).

Intravascular procedures usually consist of preliminary meticulous and complete spinal angiography, followed by embolization

(2). Because this procedure can require several hours, general anesthesia is usually preferred by the endovascular surgeon and the patient. Under general anesthesia, however, spinal cord ischemia secondary to arterial vasospasm or unrecognized occlusion of an arterial feeder to the cord may not be detected. When unrecognized, both events may result in spinal cord infarction with deleterious consequences for the patient. Our group has used intraoperative neurophysiology for the last 15 years to assist us with continuous feedback on the functional status of the sensory and motor pathways at risk for ischemia (3, 18). Over the past 15 years, somatosensory evoked potentials (SEPs) have been documented as unable to predict postoperative motor deficits (6, 14). Motor evoked potentials (MEPs) elicited with a short train of transcranial electrical stimuli and recorded from muscles (i.e., muscle MEPs) have been proposed recently as a noninvasive, sensitive, and specific tool to monitor the functional integrity of motor pathways during surgical manipulation of the spinal cord (5, 12).

The purpose of this report is to illustrate the usefulness of intraoperative MEPs in detecting functional changes within the spinal cord motor pathways during the embolization of a spinal AVM. In addition, we present correlations between neurophysiological, angiographic, and clinical findings.

CASE REPORT

History

A 28-year-old man first presented at the age of 16 years with transitory weakness involving both lower extremities, primarily in the right leg, and gait disturbances. He was treated with corticosteroids and recovered completely within 2 months.

After 12 years without symptoms, he developed progressive numbness and weakness in his left leg followed by bladder dysfunction, described as urinary retention and urgency. Magnetic resonance imaging revealed intrinsic spinal cord abnormalities on T2-weighted images. Spinal angiography revealed a T11-T12 vascular malformation. Surgical exploration was performed at another hospital, but no resection of the AVM was attempted because of significant intraoperative changes in both SEPs and MEPs (MEPs were lost, and SEPs decreased more than 50% in amplitude). The surgical exploration ruled out the presence of hematoma and disclosed two large draining veins but no feeding arteries. After surgical exploration, the patient was paraplegic with a sensory level at T11, but over a 10-day period postoperatively, he partially recovered to the level of deep paraparesis. Three weeks after surgery, the patient was referred to our hospital for angiography and possible embolization.

Examination

At admission, the patient was alert. His cranial nerves were intact. Sensory and motor findings in the upper extremities were normal. He was unable to walk; motor strength in the lower extremities was 2 to 3/5 in proximal muscles and 0 to 1/5 in distal muscles. He was spastic in both legs, the left more than the right. There was hyperreflexia and ankle clonus in the left leg. The Babinski sign was present bilaterally. The sensory level was at T11 bilaterally. He was continent but complained of urinary retention and urgency.

Angiography

After informed consent was obtained from the patient, spinal cord angiography was performed under general anesthesia. A selective angiogram of the left T11 intercostal artery demonstrated the anterior spinal artery (ASA) supply to the lesion (Fig. 1 B). The small nidus of the malformation was observed overlying the T11 vertebral body. The direction of venous drainage was primarily upward, but there was some downward drainage as well. Vascular congestion of the lower spinal cord was present. The spinal cord angiogram revealed no other supply to the malformation or other contribution to the ASA. Selective catheterization of the left and right T10 intercostal artery revealed a posterior spinal artery on each side, but no supply to the malformation.

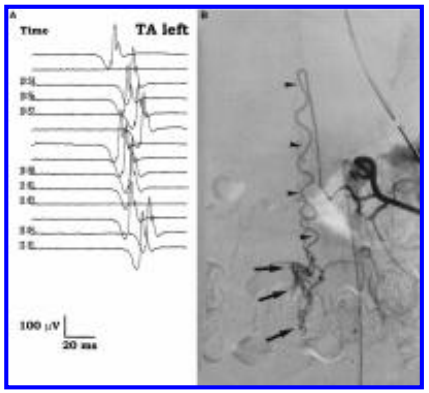

FIGURE 1. *A*, MEP recorded from the left TA muscle. *B,* selective angiogram of the left T11 intercostal artery with opacification of the AVM (*arrows*) supplied by the anterior spinal artery (*arrowheads*).

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Interventional neurophysiological monitoring

Recording of the cortical and subcortical SEPs was attempted according to a routine procedure described elsewhere (4). However, despite a clear response after stimulation of the median nerve at the wrist, which was used as a control recording, no reproducible responses were obtainable after stimulation of the posterior tibial nerves at the ankles, and the somatosensory pathways from both lower extremities were therefore considered unmonitorable.

Muscle MEPs were elicited with transcranial electrical stimulation of the motor cortex using corkscrew-like electrodes (CS electrode; Neuromedical, Inc., Herndon, VA). Short trains of up to seven square-wave stimuli of 500 μs duration and interstimulus intervals of 4 ms were applied at a repetition rate of 1 Hz through electrodes placed at C1 and C2 scalp sites, according to the International 10/20 Electroencephalogram System. The stimulation intensity did not exceed 145 mA. Muscle responses were recorded via needle electrodes inserted into the tibialis anterior (TA) muscles bilaterally. Recordings from thenar muscles served as controls (12).

The Sentinel 4 evoked potential system (Axon Systems, Inc., Hauppauge, NY) with modified software was used for both stimulation and recording. At baseline (time, 10:54), before any interventional procedure, muscle MEP responses were obtainable only from the left TA muscle (Fig. 1A), and there was no response from the right TA muscle. Recordings from right thenar muscles were present.

Endovascular treatment

After the angiographic study was performed, the decision to perform endovascular embolization was made in view of the single supply to the malformation from the left T11 intercostal artery, which was also the primary supply to the spinal cord. By a coaxial technique, a Prowler 10 microcatheter (Cordis, Miami Lakes, FL) was advanced into the radiculomedullary artery arising from the left T11 intercostal artery and beyond the hairpin loop. Shortly thereafter (time, 13:10), disappearance of the MEP from the left TA muscle was noted (Fig. 2 A). At this time, angiography revealed complete arrest of the flow in the ASA distal to the tip of the microcatheter (Fig. 2B). This was thought to be a result of the small size of the ASA and stretching of the sharp hairpin loop by the microcatheter (Fig. 2C). The patient was fully heparinized immediately by an intravenous bolus injection of 7000 U heparin followed by continuous intravenous infusion of heparinized saline. Because of the muscle

MEP disappearance, the decision was made to expedite embolization. The AVM was embolized with small polyvinyl alcohol particles (45-150 µm) using a push technique (2). The microcatheter was then withdrawn, and a control angiogram of the left T11 intercostal artery was performed. This study documented reestablished blood flow in the ASA, which showed multiple vasospastic changes (Fig. 3 B) (time, 13:16), and was followed by the reappearance of left TA muscle MEP (Fig. 3A) (time, 13:20). Approximately 9 minutes later, however, the vasospasm was so severe that a repeat angiogram of the left T11 intercostal artery revealed a complete disappearance of the radiculomedullary artery from this pedicle (Fig. 4 B). Shortly thereafter, a second disappearance of left TA muscle MEP was noted (Fig. 4A) (time, 13:32). Repeated angiography of other pedicles failed to demonstrate any other contributions to the ASA at this level.

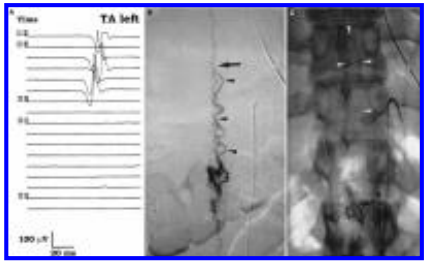

FIGURE 2. *A,* disappearance of muscle MEP from left TA muscle after catheterization of the anterior spinal artery through the left T11 intercostal artery (time, 13:10). *B,* complete arrest of blood flow in the ASA (*arrowheads*) distal to the tip of the microcatheter (*arrow*). *C,* nonsubtracted image demonstrating the opened hairpin loop of the radiculomedullary artery by the microcatheter (*arrowheads*). Compare with Figure 1B.

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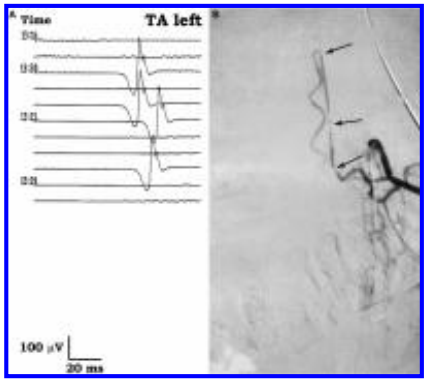

FIGURE 3. *A,* reappearance of muscle MEP from left TA muscle (time, 13:20). *B,* reappearance of slow blood flow in the anterior spinal artery, which shows multiple spastic changes (*arrows*) (time 13:16).

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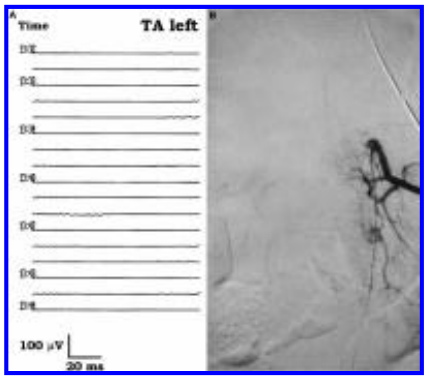

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FIGURE 4. *A,* muscle MEPs from left TA muscle are absent during severe left T11 radiculomedullary artery vasospasm (time, 13:32). *B*, angiographic evaluation of the left T11 intercostal artery demonstrates complete disappearance of the radiculomedullary artery from this pedicle, a result of severe vasospasm (time, 13:25).

The microcatheter was placed at the orifice of the radiculomedullary artery leading to the ASA, and the patient was then treated with intra-arterial papaverine infusion (a total of 20 mg) and mild systemic hypertension (systolic blood pressure, 170 mm Hg). The control angiogram demonstrated almost complete resolution of the vasospasm of the radiculomedullary artery and less opacification of the malformation nidus (Fig. 5 B) (time, 14:02). A few minutes later, approximately 35 minutes after the second disappearance of the muscle MEP, the left TA muscle MEP recovered again (Fig. 5A) (time, 14:08). The blood pressure was then gradually restored to the patient's preangiogram values (115/80 mm Hg). The muscle MEPs were continuously monitored, and they remained stable to the end of the procedure (Fig. 6 A). At this time, the vasospasm was completely resolved (Fig. 6B).

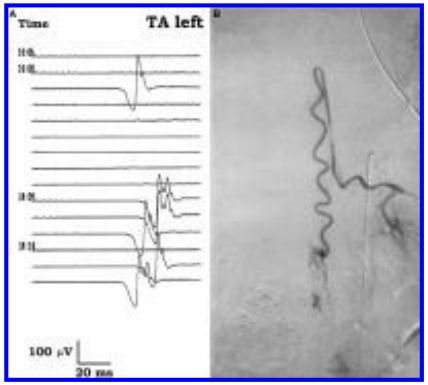

FIGURE 5. *A,* reappearance of the muscle MEP response from left TA muscle (time, 14:08). *B,* repeat angiogram of the left intercostal artery after 15 mg of papaverine infusion demonstrates reopacification of the radiculomedullary artery and almost complete resolution of the vasospasm (see Fig. 4B) (time, 14:02).

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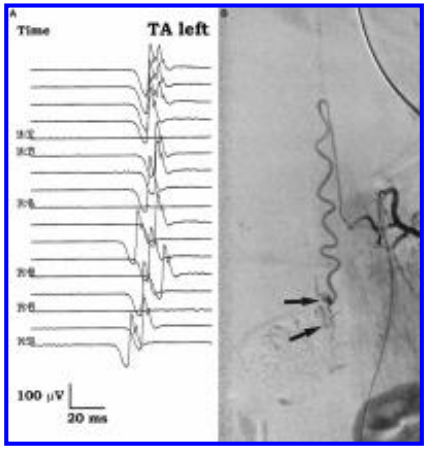

FIGURE 6. *A,* muscle MEP response from left TA muscle at the end of the procedure. *B,* complete resolution of the vasospasm; the nidus of the AVM is less opacified as a result of embolization (*arrows*).

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When the patient awoke, his neurological status was unchanged compared with the preoperative evaluation. The patient was transferred to a rehabilitation center for physical therapy 4 days after the procedure. After 3 months of follow-up, the patient is able to walk with the use of a walker, although there have not been significant changes in bladder function.

DISCUSSION

Intravascular procedures for the treatment of spinal AVMs carry the potential risk of vascular damage to the spinal cord. Morbidity secondary to spinal angiography and embolization of spinal AVMs has been described (2, 8, 11, 15). When general anesthesia is required and therefore neurological testing is not feasible, intraoperative neurophysiology is essential to perform the procedure safely. In our experience, implementation of neurophysiological monitoring using a provocative test with superselective injection of amobarbital and xylocaine significantly adds to the safety of the embolization (3).

The role of SEPs in monitoring sensory pathways during interventional neuroradiology has been documented repeatedly since 1983 (3, 7, 18). Dorsal columns, however, are supplied mainly from posterior spinal arteries, and ischemic derangement involving the anterior spinal cord may not affect SEPs. Thus, although SEPs may provide indirect information about the motor pathways, the occurrence of postoperative motor deficits despite intraoperatively unchanged SEPs has discouraged the use of these potentials for the assessment of corticospinal tracts during neurosurgical procedures (6, 14). Furthermore, SEPs may be absent from the beginning, as in the present case. Therefore, MEPs are the optimal modality of neurophysiological monitoring when the motor pathways are at risk.

Muscle MEPs are easily elicited using a short train of electrical stimuli applied transcranially. This technique is highly sensitive and specific in predicting early postoperative motor status. Data from more than 200 intraoperative muscle MEP recordings during surgery for spinal cord tumors suggest that the disappearance of muscle MEPs from TA muscles, using a yes/no criterion, is consistent with postoperative monoplegia or paraplegia (5, 12).

The role of MEPs in detecting functional changes in the motor pathways during embolization of spinal AVMs and in predicting outcomes has been documented previously (1, 10, 13). Katayama et al. (10) observed a 35% decrease in the amplitude of the epidural MEP (i.e., the D-wave) associated with disappearance of muscle MEPs from paravertebral muscles during embolization of an intramedullary AVM in the upper thoracic spinal cord. In this patient, the embolization procedure was terminated and the D-wave amplitude gradually recovered to the original amplitude, although the patient experienced only a minor and transitory deterioration of motor function. In a recent report by our group (13), the appearance of muscle MEPs from the lower extremities after endovascular occlusion of a thoracic spinal dural arteriovenous fistula correlated with the improvement in strength of both legs immediately after the patient awakened from anesthesia.

This report correlates angiographic and neurophysiological changes during intravascular embolization of a spinal AVM, suggesting the pathophysiological sequence connecting functional with morphological findings. We emphasize the importance of this correlation on the basis of three phenomena that occurred during our treatment of this patient. First, pre-embolization changes in muscle MEPs were demonstrated before radiological changes and prompted a rapid angiographic reevaluation that revealed complete arrest of blood flow in the ASA. Embolization and rapid withdrawal of the microcatheter allowed for recovery of both spinal blood flow and MEPs. Second, the disappearance of muscle MEPs after embolization revealed the neurophysiological relevance of severe vasospasm associated with arrest of spinal blood flow, which was detected by angiography shortly before muscle MEP changes. Finally, the therapeutic effect of papaverine infusion and induced moderate hypertension was confirmed by complete reopacification of the ASA and recovery of MEPs.

CONCLUSION

This case report illustrates that MEPs may play a critical role in the early detection of spinal cord dysfunction, that they can prevent damage to the spinal cord, and that they can predict clinical outcome during spinal AVM embolization. Although larger studies are needed to confirm these data, we suggest the use of MEP monitoring during any interventional procedure that places the spinal cord at risk.

ACKNOWLEDGMENT

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COMMENTS

Sala and et al. report their experience using motor evoked potentials (MEPs) during embolization of a spinal cord arteriovenous malformation. This report is from a group with extensive experience with neurophysiological monitoring during endovascular procedures. They have worked this problem out in the endovascular suite and were early to report their experience with somatosensory evoked potentials during embolization of spinal lesions as well. MEP monitoring is very attractive when the anterior spinal circulation is going to be manipulated and is potentially at risk. Their case is extremely illustrative, and they describe exactly how they perform this procedure, which should allow others to duplicate their efforts. This is a technique that should be performed routinely when embolizing these lesions.

Robert H. Rosenwasser

Philadelphia, Pennsylvania

The authors report their experience using MEP monitoring during an embolization procedure on a 28-year-old man with a spinal arteriovenous malformation. The authors briefly describe their technique, its application, and its benefits for their patient. They also demonstrate its potential use as a monitoring modality during spinal embolization procedures, particularly

when motor deficits are anticipated.

This form of spinal monitoring certainly has advantages over traditional somatosensory evoked potential monitoring. From an endovascular standpoint, intraprocedural monitoring of MEPs is more useful in detecting functional changes within the spinal cord motor pathways during interventions along the anterior spinal axis.

The authors mention the “push technique” of embolization with polyvinyl alcohol particles. This is a term that is usually reserved for liquid adhesive embolization, not polyvinyl alcohol particle embolization. In addition, this article would be more useful if it went into greater detail describing the technique of MEP monitoring, its applications, and technical considerations, so that other centers can consider using this technology. Nevertheless, this is a significant contribution to the literature by a group with significant experience in intraoperative neurophysiology monitoring.

Donald W. Larsen

Interventional Neuroradiologist Los Angeles, California

Embolization; Intraoperative monitoring; Motor evoked potential; Spinal arteriovenous malformation; Transcranial electrical stimulation

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