Embolization of Intramedullary Spinal Arteriovenous Malformation Fed by the Anterior Spinal Artery with Monitoring of the Corticospinal Motor Evoked Potential

—Case Report—

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Abstract

Intramedullary spinal AVMs fed by the anterior spinal artery cannot be embolized without risking unacceptable motor deficits, since the feeding arteries may supply the corticospinal tract (CST). An 8-year-old boy underwent successful embolization of such an AVM under general anesthesia using intermittent infusion of embolic material with monitoring of the CST integrity with the corticospinal motor evoked potential (MEP). This case illustrates the value of corticospinal MEP monitoring during therapeutic procedures under general anesthesia which risk interrupting the blood supply to the CST.

Key words: arteriovenous malformation, embolization, pyramidal tract, evoked potential, spinal cord

Introduction

Intramedullary spinal arteriovenous malformation (AVM) may be fed by the anterior spinal artery.1–4,6,7,15–17 The medullary arteries supplying such AVMs may also supply the normal cord tissue, including the corticospinal tract (CST).15,16 Almost all the CST blood supply comes from the end perfusion bed of the anterior spinal artery. Such AVMs cannot be excised or embolized without risking unacceptable motor deficits.15,16 Patients with intramedullary spinal AVMs are usually less than 30 years old. General anesthesia has to be employed for embolization in younger children. This further magnifies the problem, since the development of motor deficits cannot be detected during embolization procedures or test infusions of short-acting barbiturates.12,13 Although the somatosensory evoked potential (SEP)1–4,12,21 has been used to monitor the cord function during interventional neuroradiological procedures, it is not suitable in this case because a major component of the SEP is mediated by the dorsal column6,11 which is supplied by the posterior spinal artery.

We present here a successful embolization of an intramedullary AVM fed by the anterior spinal artery with CST monitoring using the motor evoked potential (MEP) in an 8-year-old boy under general anesthesia. Muscle responses to motor cortex stimulation are unstable under general anesthesia. We therefore adopted monitoring of the corticospinal MEP2,5,8,13,17 which directly reflects impulses mediated by the CST.8,10 This case illustrates the value of monitoring the corticospinal MEP during therapeutic procedures under general anesthesia which risk interrupting the blood supply to the CST.

Case Report

An 8-year-old boy suddenly suffered paraparesis on October 16, 1988. A spinal tap revealed no evidence of subarachnoid hemorrhage. Intra-arterial digital subtraction angiography demonstrated an intramedullary AVM fed by the right vertebral artery through an enlarged anterior spinal artery at the
upper thoracic level on December 7, 1988. He underwent the first intravascular embolization on February 1, 1989. He was fully capable of walking, running, and riding a bicycle.

On December 15, 1989, he again suddenly suffered paraparesis. No evidence for hemorrhage was noted. Bilateral increased tendon reflexes and Babinski reflex were noted. Slight urinary retention was also observed. Touch and proprioceptive sensations were not disturbed. Thermal and pain sensations were slightly decreased in the lower extremities bilaterally. Intra-arterial digital subtraction angiography revealed that rapid flow through the AVM was re-established (Fig. 1 left). He recovered gradually from the above neurological deficits and underwent a second AVM embolization on February 7, 1990.

### Procedures for MEP Monitoring

The radiographic and neurological findings strongly suggested that the diversion of blood flow from the ventral spinal cord to the AVM caused the neurological deficits in this patient. This implied that the same medullary arteries fed both normal cord tissue and the AVM. Therefore, surgical excision or embolization of the AVM might cause devastating motor deficits. We therefore monitored the CST integrity. As indicated above, we used the corticospinal MEP because the procedure required general anesthesia. The parents gave informed consent for all the procedures described below.

Under fluoroscopic control, a pair of flexible platinum electrodes (Medtronic M8483, Medtronic Co., Minneapolis, Minn., U.S.A.) were inserted into the epidural space and advanced rostrally to the middle thoracic level (Th7). A detailed description of the technique employed has been given elsewhere. The monopolar corticospinal MEP response to motor cortex stimulation was recorded at the epidural electrodes with a reference electrode over the paravertebral muscles. Signals from the electrodes were fed into an amplifier with a bandpass range of 5 Hz to 5 kHz and 16-32 sweeps averaged using a signal processor.

We initially attempted to record the corticospinal MEP using transcranial stimulation of the motor cortex. Transcranial stimulation with higher currents causes large artifacts which obscure the corticospinal MEPs, but the corticospinal MEP can be recorded with relatively low currents in children because of the relatively thin cranial vault. However, the corticospinal MEP traces recorded in the present case were insufficiently stable to distinguish small pathological changes. We therefore decided to stimulate the motor cortex epidurally through small burr holes in the bilateral frontal skull 20 mm

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off the midline. Platinum-plate electrodes with a 5 mm diameter (Medtronic M3586) were inserted into the epidural space and advanced posteriorly to contact the motor cortex bilaterally. The bilateral motor cortices were stimulated bipolarly using these two electrodes. An anodal current was delivered to the electrodes alternately to stimulate subcortical CST fibers from the trunk and leg areas of the motor cortex bilaterally. Contraction of various leg muscles was obtained bilaterally, in the absence of muscle relaxants. The stimuli were monophasic square wave pulses of 0.3 msec duration at 4 Hz and triggered by electrocardiograms. The stimulus intensity was less than maximum (5 mA).

**Embolization and Changes in MEP**

A normal corticospinal MEP was clearly recorded before starting the embolization. An initial biphasic positive-negative response followed by a small additional biphasic positive-negative response were observed (Fig. 2). These responses, termed the direct wave (D wave) and indirect wave, respectively, were followed by a weak muscle response detected by the reference electrode on the paravertebral muscle. The D wave followed stimulation at frequencies as high as 600 Hz, which showed the response was mediated by axons with no intervening synapses. Only the CST is known to mediate such a response to cortex stimulation recorded at the spinal cord. The indirect wave and muscle response were unstable under general anesthesia. The physiological characteristics of these waves recorded by the same technique have been described elsewhere.

A catheter (Tracker #18) was inserted into the anterior spinal artery feeding the AVM. The D wave amplitude began to decrease gradually several minutes after insertion and returned to the original level when the catheter was withdrawn. The catheter was again inserted into the anterior spinal artery and embolic material (Ivaron®) infused intermittently (Fig. 1 center). After each infusion, serial corticospinal MEPs were recorded as fast as every 15-30 seconds. The catheter was withdrawn to confirm D wave recovery to the original level. After repeated procedures, most of the AVM eventually became invisible radiologically (Fig. 1 right). The anterior spinal artery distal to the AVM became visible at the same time. Soon after the subsequent infusion of embolic material, a definite decrease in D wave amplitude (−35%) suddenly occurred accompanied by a slight delay in D wave latency (+0.2 msec) and the indirect wave and muscle response became obscure (Fig. 2). This combination of changes strongly suggested that the CST was dysfunctional. No further embolic material was therefore infused. The D wave amplitude and latency then gradually recovered to the original level approximately 20 minutes after termination of the embolization procedures (Fig. 2). The indirect wave and muscle response reappeared at the same time. The patient suffered only minor deterioration of motor function and could walk 1 month postoperatively.

**Discussion**

Corticomyographic MEP monitoring of CST integrity during interventional neuroradiological procedures has been described previously. However, there were three major reasons why we preferred corticospinal MEPs for this purpose. First, corticospinal MEPs directly reflect the conductivity of the CST. In contrast, corticomyographic MEPs are the response relayed by spinal motor neurons. As the excitability of spinal motor neurons is influenced by several neural pathways in addition to the CST, changes in corticomyographic MEPs may not necessarily be specific to CST dysfunction. Second, corticospinal MEPs can be evoked by lower intensity stimulation. General anesthesia considerably reduces
the excitability of spinal motor neurons, so large currents are usually required to evoke corticomyographic MEPs. Third, the level of anesthesia greatly influences the corticomyographic MEPs but not the corticospinal MEPs, again due to the influence of general anesthesia on spinal motor neurons. We therefore recommend corticospinal MEP, rather than corticomyographic MEP, for monitoring CST function during therapeutic procedures, especially under general anesthesia. The present case does show that disappearance of the indirect waves and muscle response provides good confirmation of CST dysfunction indicated by a depressed D wave.

Rapid decreases in SEP amplitude are consistently observed during neuroradiological procedures where the anterior spinal artery is occluded or filled with contrast medium. The present case also demonstrated decreased corticospinal MEP amplitude when the catheter was inserted in the anterior spinal artery. This change was totally reversed by catheter withdrawal. Since a major component of the SEP is mediated by the dorsal column which has a different blood supply, any correlation between SEP changes and postoperative neurological deficits is debatable.

Persistent changes in the D wave of the corticospinal MEP followed obliteration of most of the AVM. The blood flow of the medullary arteries was probably diverted from the normal tissue to the AVM before embolization. This implies that infused embolization material is predominantly delivered to the AVM. As embolization proceeds, however, the blood flow through the AVM decreases and the risk of embolization of the normal tissue may increase. Embolization material should therefore be infused carefully when the blood flow through the AVM is greatly diminished. The present case suggests that this critical point may be identified from serial corticospinal MEP recordings.

The corticospinal MEP recorded with epidural stimulation was demonstrably much clearer than that recorded with transcranial stimulation. As corticospinal MEP has great clinical value, epidural stimulation should be used without hesitation, even though invasive, if clearer recording is essential.

References


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