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## Intravascular Embolization of a Cerebellar Arteriovenous Malformation for Treatment of Hemifacial Spasm

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Hemifacial spasm consists of involuntary contractions of the facial muscles, usually caused by vascular compression of cranial nerve VII, near its exit zone from the brainstem [1–7]. Arteriovenous malformations (AVMs) are a rare cause of this condition [2, 3]. We report a case of hemifacial spasm caused by an extensive cerebellar AVM, which was successfully treated by intravascular embolization.

### Case Report

A 58-year-old man presented with a chief complaint of right hemifacial spasm occurring 15–20 times a day for 2 months. He also complained of a right-sided bruit present for 1 year, and intermittent dizziness 10–15 times a day for 3–4 months. Physical examination was remarkable only for a bruit, which was heard over the right mastoid region.

An MR scan revealed a large posterior fossa, pial AVM and enlarged vascular structures in the right cerebellopontine angle (Fig. 1). Vertebral angiography demonstrated predominate blood supply from enlarged right posterior inferior cerebellar artery and (PICA) anterior inferior cerebellar artery (AICA) branches (Fig. 2). Correlation of the angiogram and MR findings disclosed an enlarged AICA–PICA loop in proximity to the exit zone of the seventh cranial nerve.

A nondetachable balloon was temporarily placed into the right vertebral artery, just distal to the PICA origin. This was followed by intravascular embolization of the AVM from a catheter in the cervical portion of the right vertebral artery by using approximately 12 pieces of hand-cut polyvinyl alcohol (PVA) particles measuring 1–2 mm in diameter. Postembolization angiograms revealed marked reduction in blood flow through the AVM (Fig. 3). The distal right PICA was occluded.

After embolization, the patient's hemifacial spasm, bruit, and episodes of dizziness subsided completely. The patient continues to be asymptomatic 18 months after the procedure.

### Discussion

Hemifacial spasms are usually unilateral and consist of intermittent contractions of the facial muscles, frequently

starting with the orbicularis oculi muscle and continuing downward to involve all the muscles of the face [1–5]. It is caused by hyperactive dysfunction of the facial nerve and can have a variety of causes. The most common cause is vascular compression by a small artery or vein at the exit zone for the seventh nerve [1–7]. This portion of the nerve appears to be particularly susceptible to compression because of an anatomically discernible junction where the thinner central myelin is replaced by thicker, peripheral myelin [4, 5]. Other causes for hemifacial spasm include aneurysms, AVMs, and tumors of the cerebellopontine angle [2, 8, 9]. Decompression of the nerve will usually relieve the symptoms.

Larger posterior fossa AVMs are difficult to treat surgically [10–12]. Results obtained by using intravascular techniques have been poor when trying to obliterate the entire malformation [10, 13]. However, interventional techniques can be used in selected cases to augment surgery or to partially treat an AVM. The latter is usually considered when the primary goal is simply a reduction of neurologic symptoms. Our patient had evidence of vascular compression of the facial nerve by enlarged vascular structures on the radiologic studies. By reducing the blood flow to the AVM, it was thought that a decrease in the size of the supplying arteries and draining veins might lessen the amount of nerve compression and cranial nerve dysfunction. The postembolization angiogram demonstrated a significant reduction in blood flow to the AVM with occlusion of the distal right PICA. Immediate relief of hemifacial spasm and the bruit was noted.

A review of the literature disclosed only five previously reported cases of hemifacial spasm caused by an AVM [2, 3]. All were surgically treated and had postsurgical relief of symptoms. However, two patients reported by Gardner and Sava [2] had surgical complications; one with mild facial weakness and the other with facial paralysis that completely resolved after 3 months. Ours is the first report of successful treatment of this condition by means of intravascular embolization. Partial embolization has not been shown to decrease the risk of hemorrhage in AVMs. However, in selected cases

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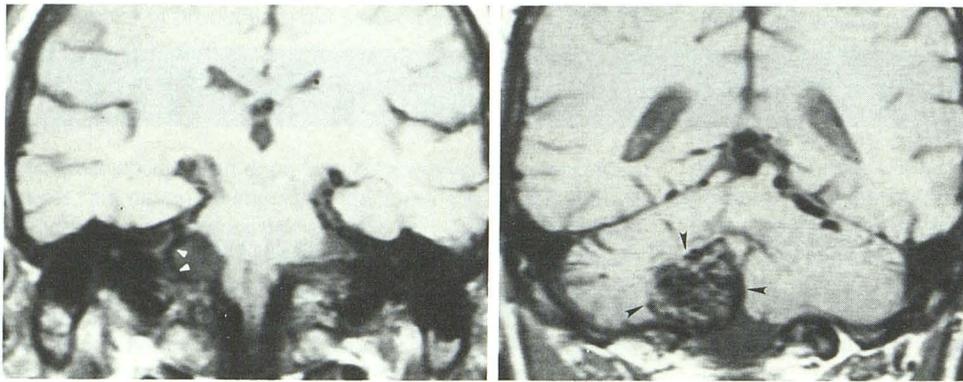
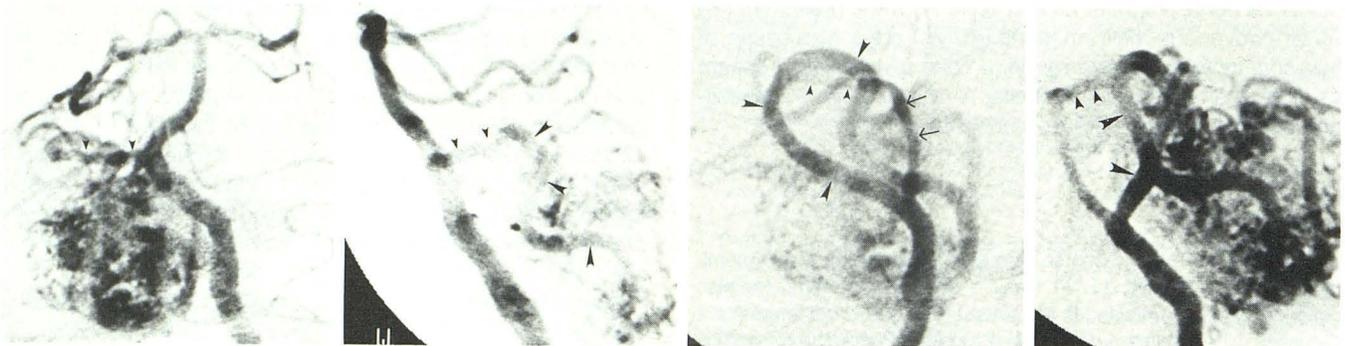


Fig. 1.—A, Coronal T1-weighted MR image through cerebellopontine angles. Note large vascular loop (*arrowheads*) on right side.  
 B, Coronal T1-weighted MR image, more posterior than A, reveals a large AVM involving the medial, right cerebellar hemisphere (*arrowheads*).

A

B



A

B

C

D

Fig. 2.—A, Left vertebral artery injection, anteroposterior view, shows large right AICA (*arrowheads*) with supply to AVM.  
 B, Left vertebral artery injection, lateral view, reveals connection between large right AICA (*small arrowheads*) and enlarged distal right PICA (*large arrowheads*) supplying AVM.  
 C, Right vertebral artery injection, anteroposterior view, shows enlarged PICA (*large arrowheads*) feeding AVM. Also, flash-fill of basilar artery (*arrows*) and right AICA (*small arrowheads*) are noted.  
 D, Right vertebral artery injection, lateral view, shows vascular loop of PICA (*large arrowheads*) and AICA (*small arrowheads*).



A

B

C

Fig. 3.—A, Right vertebral artery injection, lateral view, postembolization film reveals markedly decreased flow through AVM with distal occlusion (*arrowhead*) of PICA. Note increased opacification of basilar artery and normal cerebellar branches.  
 B, Left vertebral artery injection, Towne view, postembolization study shows decreased flow to AVM. Right AICA cannot be identified clearly.  
 C, Left vertebral artery injection, lateral view, postembolization injection shows diminished opacification of right PICA (*arrowheads*) and AVM as compared with similar preembolization study (Fig. 2B).

these techniques can be used to achieve a significant reduction in AVM blood flow, resulting in temporary or permanent relief of neurologic symptomatology.

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