



Providing Choice & Value

Generic CT and MRI Contrast Agents



**FRESENIUS
KABI**

CONTACT REP

AJNR

Lidocaine injection into external carotid branches: provocative test to preserve cranial nerve function in therapeutic embolization.

J A Horton and C W Kerber

AJNR Am J Neuroradiol 1986, 7 (1) 105-108

<http://www.ajnr.org/content/7/1/105>

This information is current as of July 9, 2025.

Lidocaine Injection into External Carotid Branches: Provocative Test to Preserve Cranial Nerve Function in Therapeutic Embolization

Joseph A. Horton¹
Charles W. Kerber²

Endovascular obliteration of hypervascular lesions of the head and neck has become clinically accepted, but it may cause stroke and peripheral cranial nerve palsy. By using a flow-controlled technique to deliver the materials and by knowing the vascular anatomy of the cranial nerves, these problems are less likely to occur. Occasionally, though, vascular anatomy is distorted by the lesion or is anomalous in its distribution. A provocative test of lidocaine injected into the appropriate artery seems to offer a functional test of whether the capillary bed will tolerate small-particle or liquid plastic occlusion. Twenty-six patients had various branches of their external carotid arteries challenged with lidocaine. Three developed transient palsies, and their treatments were modified. None of the 26 patients developed a complication of embolization.

Endovascular obliteration (therapeutic embolization) of hypervascular lesions of the head and neck has become an accepted clinical treatment. Still, it is not undertaken lightly because of two major possible complications: The first of these is the loss of an embolus into the intracranial circulation with subsequent potential cerebral infarction. The other is the development of a palsy of a peripheral cranial nerve by distal occlusion of its blood supply. We avoid the former by using a flow-control technique (the controlled deposition of materials in flowing blood while demonstrating the occlusion process) [1, 2]. We have observed that it may be possible to avoid the latter complication by a provocative test. It is often difficult in the face of abnormal and hypertrophied feeding vessels to be certain of the identification of each artery feeding a lesion. Thus, the potential blood supply to each cranial nerve can rarely be determined with certainty by the anatomic data given by an angiogram. When in doubt, we have tested vessels that may potentially supply cranial nerves by selectively injecting lidocaine into them under fluoroscopic control. The description and utility of this test is the purpose of this preliminary report.

Materials and Methods

In the past 3 years, we have challenged branches of the external carotid artery in 26 patients undergoing therapeutic embolization of facial and dural arteriovenous malformations (AVMs) and hypervascular tumors, usually glomus tumors.

Baseline testing of cranial nerves II–XII was carried out. After gentle catheterization of the artery in question, 2% lidocaine (Xylocaine 2%, Astra Pharmaceuticals, Worcester, MA) mixed in equal volumes with Conray 60 (Mallinckrodt, St. Louis) was injected using fluoroscopic monitoring to ensure that no reflux into other arteries occurred. Between 30 and 70 mg of lidocaine was injected at the vessel acceptance rate. Then, cranial nerves III–XII were tested again, and the patient's subjective feelings were recorded. Continuous cardiac monitoring was performed throughout the procedure. No patient was examined who had heart block. There was no appreciable change in the electrocardiographic pattern or rate.

If the provocative test showed no changes in cranial nerve function, we proceeded with a flow-controlled perfusion of polyvinyl alcohol (PVA) foam particles (Pacific Medical Industries,

Received November 28, 1984; accepted after revision June 21, 1985.

¹ Department of Radiology, Presbyterian-University Hospital, Pittsburgh, PA 15213. Address reprint requests to J. A. Horton.

² Alvarado X-Ray Medical Group, San Diego, CA 92120.

AJNR 7:105–108, January/February 1986
0195–6108/86/0701–0105

© American Society of Neuroradiology

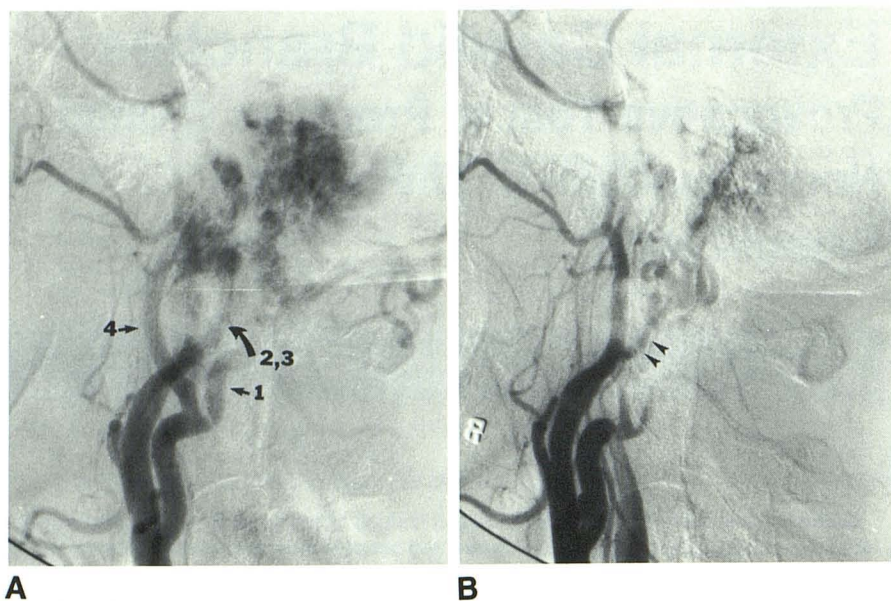


Fig. 1.—Case 1. **A**, Preembolization arteriogram, lateral view. Four external carotid artery branches supply tumor; the names of the branches are uncertain. Vessels 2 and 3 overlap. Lidocaine injection into vessels 1, 3, and 4 produced no deficit, but injection into vessel 2 produced a dense temporary seventh nerve palsy. Therefore, this is probably the posterior auricular artery. **B**, Vessel 2 (arrowheads) remains open after embolization of other three feeding arteries. The patient remained neurologically intact after embolization.

San Diego, CA) mixed with Gelfoam particles (Upjohn, Kalamazoo, MI) [3].

Follow-up angiography was then performed if feasible. If the test was positive, that is, if a cranial nerve palsy developed, we removed the catheter from the artery and waited for the palsy to resolve before other testing was carried out. Additional diagnostic runs were sometimes made during this waiting period.

Case Reports

Case 1

A 62-year-old woman was referred for angiography and preoperative embolization of her known glomus tumor. She had decreased ipsilateral eighth nerve function but no other cranial nerve palsies. Angiography demonstrated a total of four external carotid arterial branches supplying the tumor (fig. 1A). The internal carotid and the vertebral arteries were normal.

Lidocaine was injected into each of the four external branches, but only the second branch, probably the posterior auricular artery, seemed to supply the seventh nerve (fig. 1B). Forty mg of lidocaine injected into that artery promptly produced a dense seventh nerve palsy that cleared after 13 min. The other three vessels were embolized, and as we were unsure of the significance of our observation, we repeated the lidocaine test and reproduced the palsy. Thus, we elected not to occlude this vessel, and the postembolization angiogram shows it spared. No cranial nerve palsy developed after embolization, but a complete peripheral seventh nerve loss resulted after the subsequent operation.

Case 2

A 72-year-old woman had a pulsatile sound in the left side of her head that began after a chiropractic neck manipulation. Auscultation confirmed her bruit. The physical examination was otherwise normal. Angiography showed the external carotid artery supplying a dural AVM with contributions from the internal carotid artery via meningo-hypophyseal vessels (fig. 2A) and from the vertebral artery via a C2 posterior branch artery.

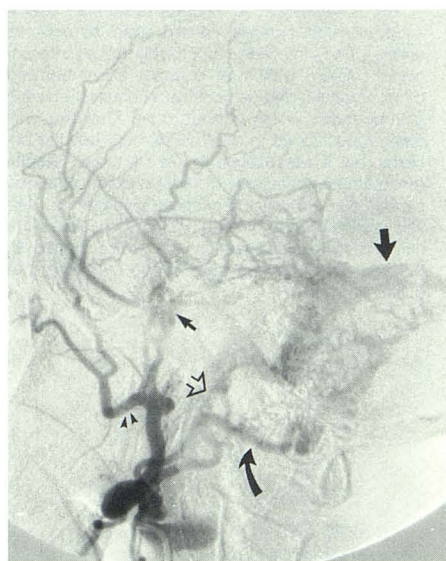
Early in the procedure we did not selectively catheterize the vessels fearing we might develop spasm. The middle meningeal artery appeared to be a principal feeder, but its origin was beyond two prominent loops located proximally in the external carotid artery. A lidocaine infusion low in the external carotid (fig. 2B) proximal to the origin of the middle meningeal artery (as seen by a fluoroscopically monitored test injection) produced a partial seventh nerve palsy. We then placed the catheter selectively into the middle meningeal artery (fig. 2C) and repeated the test. No neuropathy was produced; we embolized that vessel without further incident. Remembering that the patient's chief complaint was noise, and concerned that we might produce seventh nerve palsy by smaller-particle embolization, we occluded the external carotid artery by placing a coil proximally (fig. 2D). No deficit resulted. The bruit subjectively and objectively disappeared.

Case 3

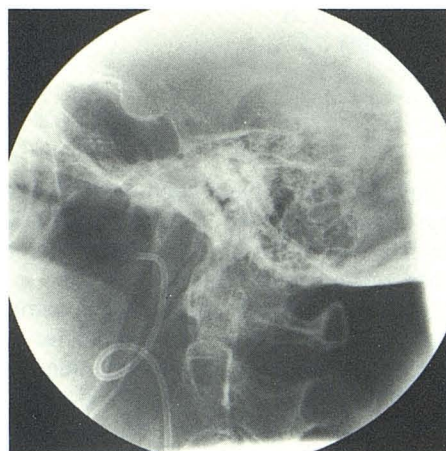
A 67-year-old woman had a large glomus tumor. Angiography demonstrated supply by several small arterial branches of the right external carotid artery (fig. 3A). Selective catheterization of the first vessel was accomplished, but it was difficult to keep the catheter out of the occipital artery. Lidocaine infusion produced no cranial neuropathy, but 30 sec later, the patient experienced severe vertigo, horizontal nystagmus, nausea, and vomiting. These symptoms resolved within 30 min. No embolization was performed in that vessel.

Discussion

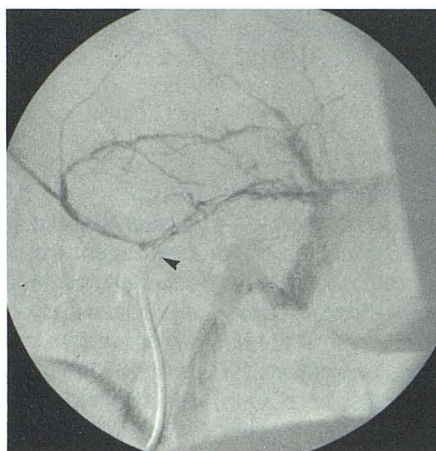
Tests that may predict functional loss are a desired part of the therapist's armamentarium. We have been using the superselective injection of Amytal into branch arteries before intracerebral embolization for nearly 10 years. This procedure, a refinement of the Wada and Rasmussen test [4] is done by injecting Amytal selectively into an intracerebral vessel. The test has been a reasonable but not infallible indicator of whether we will cause functional loss secondary to emboli-



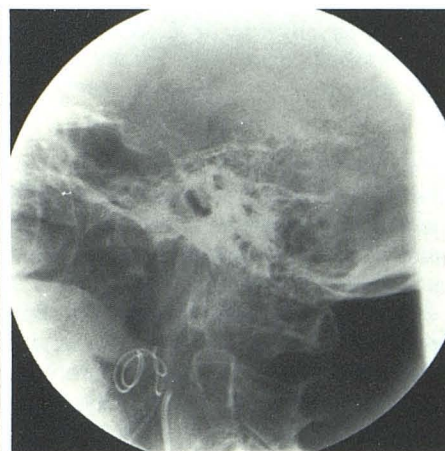
A



B



C



D

Fig. 2.—Case 2. **A**, Preembolization arteriogram. Dural AVM supplied by numerous branches of external carotid artery, including occipital (*curved arrow*), middle meningeal (*small solid arrow*), superficial temporal, and posterior auricular (*open arrow*). Tortuosity just distal to catheter tip made more distal catheterization difficult. Lidocaine injection with catheter in this position produced temporary facial paralysis. Sigmoid sinus (*large solid arrow*). **B**, Catheter repositioned more distally but still proximal to middle meningeal artery origin (as seen in fluoroscopically monitored injection), into internal maxillary artery (**A**, *arrowheads*), and lidocaine was again infused. Temporary facial palsy occurred immediately. **C**, Catheter was directed into middle meningeal artery (*arrowhead*). No paralysis resulted from lidocaine challenge. Infusion of PVA foam and Gelfoam particles was carried out. **D**, As patient's bruit had not been obliterated by this last embolization, and knowing that lidocaine had caused the seventh nerve palsy when injected proximally, a GAW coil was placed into proximal part of external carotid artery. Curves in coil are identical to those in artery in **A**. Nearly immediate cessation of the bruit occurred, and the patient remained asymptomatic without seventh nerve palsy.

zation. We now may have a predictor of potential functional loss during external carotid artery embolization. We expect, but have not yet found, false-negative results when a high-grade steal exists, as we have seen with certain intracranial AVMs.

It has been the unfortunate experience of early workers (personal communications, various authors) that smaller particles and the liquid embolic agents are likely to cause cranial nerve palsies when injected into the appropriate feeding vessel because they block capillary flow and preclude collateral pathway openings. One should theoretically then be able to avoid cranial nerve palsies simply by using larger particles. Large particles, however, though sometimes necessary, do permit frequent recurrence of these lesions. Also, having blocked proximal vessels, subsequent treatments are technically more difficult and less likely to succeed. To prevent this recurrence, some authors are using smaller particles, and again are using the liquid agents, most notably, cyanoacrylate; thus the need for a provocative test.

We believe that the technique of the provocation is important. Ahn et al. [5] showed extra- to intracranial shunts in normal patients during selective high-pressure injection of contrast agent. Therefore, we recommend the flow-control technique for both particle infusion and the test, lest either lidocaine or treatment agent pass unwanted into the central nervous system. Lidocaine was once injected into internal carotid arteries during carotid angiography in hopes of preventing the spasm secondary to subarachnoid hemorrhage. Today, however, its use in the central nervous system is not believed efficacious and, as suggested by our third patient, is most detrimental in the posterior circulation.

Case 1 illustrates a common problem of feeding artery identification. When hypervascular tumors and AVMs have recruited vessels, it is often difficult to find agreement among trained neuroradiologists as to the names of the particular hypertrophied vessels that are present, despite descriptions of the arterial anatomy of the craniofacial region [6]. Thus, identification of the anatomically known vascular supply to

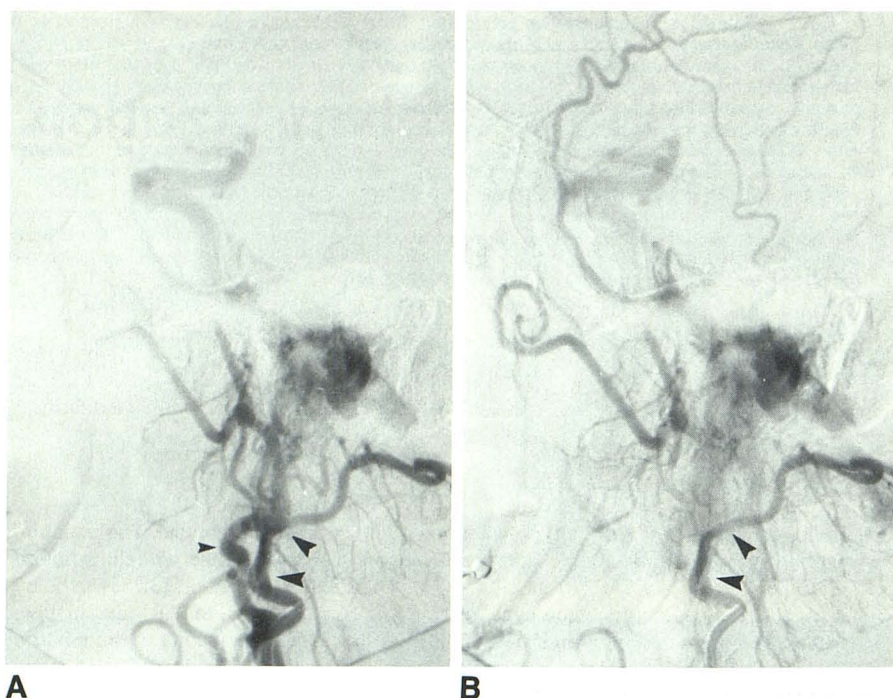


Fig. 3.—Case 3. **A**, Though positioned fluoroscopically into external carotid distal to occipital artery, during injection catheter tip moved into occipital branch. Reflux of contrast agent showed other branches and demonstrated densely staining glomus tumor within petrous bone. Catheter was again repositioned in external carotid, beyond occipital, but it was difficult to keep it there. Despite tenuousness of placement, infusion of lidocaine was carried out. Patient experienced nystagmus, vertigo, nausea, and vomiting, which took almost 30 min to resolve. Embolization was not performed. **B**, 0.5 sec after showing catheter tip migrating into occipital branch.

the cranial nerves is tenuous. Our first patient (case 1) did demonstrate a profound and repeatable response, and did show that the palsy produced would clear within a reasonable amount of time.

Case 2 was more complicated. Though certainly other treatment algorithms come to mind, the one followed did cure her main complaint. The procedure was difficult because of the tortuosity and the multiplicity of feeders. We are even now uncertain about the arterial supply to her seventh nerve and speculate that the physiologic/functional data given by the test may have been more important than the exact anatomic descriptions.

In case 3, we believe we inadvertently introduced lidocaine into the posterior circulation causing a severe and profound—but temporary—alteration in neurologic function. The supratentorial result of that experience was disturbing both to the patient and to us. It is likely that the lidocaine passed through the occipital artery and into the vertebral circulation.

Of the 26 patients in whom we have used this technique, three had profound clinical responses, and in each case we used this response to modify our therapeutic approach. Thus, we avoided the production of embolic seventh nerve palsies in two and probably a posterior fossa stroke in the last patient. Subsequent surgery did cause palsy in the first patient, but the surgeons advised her and her family in advance of the likelihood of this outcome. The patient's acceptance of this loss was much better, and, though the surgical results were not as good as desired, the psychologic trauma was lessened. Since we have begun using this test, no patient has sustained an embolization-related cranial neuropathy, even though small

particles and liquid embolic agents have been used. These observations lack the ultimate scientific verification: the embolic agent has not been delivered into an artery after a lidocaine test there has caused a neuropathy.

Lidocaine challenge of selective branches of the external carotid artery in our small group of patients appeared to enhance the safety of small-particle or liquid embolization of the external carotid artery. The test does add time to the procedure, but it can be performed while films are being processed. A wider clinical experience will, we hope, be forthcoming and will allow us to learn more about the drawbacks and pitfalls of the technique and the test itself.

REFERENCES

1. Kerber CW. Flow-controlled therapeutic embolization. A physiologic and safe technique. *AJR* 1980;134:557-561.
2. Kerber CW. Catheter therapy: fluoroscopic monitoring during deliberate embolic occlusion. *Radiology* 1977;125:538-540.
3. Horton JA, Marano GD, Kerber CW, Jenkins JJ, Davis S. Polyvinyl alcohol foam-gelfoam for therapeutic embolization: a synergistic mixture. *AJNR* 1983;4:143-147.
4. Wada J, Rasmussen T. Intracarotid injection of sodium amytal for the lateralization of cerebral speech dominance. *J Neurosurg* 1960;17:266-282.
5. Ahn HS, Kerber CW, Deeb Z. Recognition and role of extra- to intracranial anastomoses in therapeutic embolization. *AJNR* 1980;1:71-75.
6. Lasjaunias PL. *Craniofacial and upper cervical arteries: collateral circulation and angiographic protocol*. Baltimore: Williams & Wilkins, 1983.