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Generic CT and MRI Contrast Agents



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Editorial

Interventional Neuroradiology

Neuroradiology, as with any branch of medicine, is constantly evolving. Three recent, challenging developments are having profound effects on the scope of neuroradiology and its practice.

The first development, computed tomography, has had and will continue to have far-reaching diagnostic and socioeconomic consequences. The second development is the use of metrizamide, the new water-soluble contrast agent, for myelography, cisternography, and ventriculography. The use of this contrast agent is only now being explored, and its full potential has not yet been realized. The third area of progress, the growth of interventional therapy for many types of vascular lesions of the head, face, and spine, has seen many recent advances. Though less well heralded than the first two, interventional embolization has now become firmly established in neuroradiology.

While therapeutic endovascular occlusions date from as early as 1904 [1], it is in the last 20 years that major progress has occurred. The development of catheters that permit superselective catheterization and subsequent injection of embolic agents, such as tissue adhesives that polymerize on contact with blood and cause intravascular thrombosis, represents a remarkable technical improvement [2]. For example, compare the therapy of cerebral arteriovenous malformations devised by Luessenhop and Spence in 1960 [3], who performed an open arteriotomy of the carotid artery on a patient for introduction of four large (2.5, 3.0, 4.0, and 4.2 mm) methyl methacrylate emboli that were passively carried by the blood flow to the arteriovenous malformation, with the present day transfemoral approach of catheterization of branches of the middle cerebral artery and precise injection of tissue adhesives directly into the malformation [4].

The refinement of catheters has progressed from the conventional tapered and nontapered types to the single-lumen, double-lumen, flow-guided single-lumen, and detachable balloon catheters. The selection of the appropriate catheter depends on both the size of the feeding arteries

and the location of the lesion [5]. There has been a similar growth in the development of different embolization agents. Gelfoam, polyvinyl alcohol foam, silicone spheres, and tissue adhesives such as isobutyl-2-cyanoacrylate and silicone fluid mixtures have all been used. The decision as to the type of agent injected depends on the flow dynamics and the location of the lesion. Each catheter and each embolization agent has its advantages and disadvantages, its proponents and critics.

The technical advances have expanded the scope of embolization. Not only can it be used for the therapy of cerebral arteriovenous malformations, but it can also be used to treat facial angiomas, juvenile angiofibromas, and glomus jugulare tumors, and to control intractable epistaxis [6]. Carotidocavernous and vertebral arteriovenous fistulas have been treated with detachable balloons [7]. Embolization has been used as a preoperative aid to reduce the vascularity of meningiomas [6]. Spinal arteriovenous malformations have been successfully treated by embolization [8–10].

The aim of therapeutic embolization of an arteriovenous malformation is to occlude the nidus of the lesion, prevent the development of collateral channels, and effect a permanent cure. The aim of therapy of angiofibromas and glomus jugulare tumors is either to promote necrosis of the lesions or to reduce the vascularity as an adjunct to surgery or radiation therapy. Tissue adhesives such as isobutyl-2-cyanoacrylate, which polymerizes on contact with blood and the intima of vessels and causes a mild inflammatory reaction, appear to be ideal agents for intravascular occlusion [4]. Silicone fluid mixtures can also be used [6] but, since they do not adhere to tissue, it is necessary to have complete stasis in the vessel during injection. Gelfoam, polyvinyl alcohol, and silicone spheres generally do not enter the nidus of the lesions but lodge at precapillary sites; however, they are ideal agents if the aim of therapy is to reduce vascularity prior to surgery. In the treatment of carotidocavernous and vertebral arteriovenous fistulas, the aim is to

occlude the fistulas while preserving the patency of the carotid and vertebral arteries. Detachable balloons placed in the venous side of the fistulas appear to be most effective in these cases [7].

The risk of stroke is always present when embolizing intracranial arteriovenous malformations. On rare occasions, intracranial embolization with tissue adhesives may cause rupture of the malformation with catastrophic results. However, it is remarkable to what degree the brain can tolerate aberrant particulate emboli, such as Silastic beads, lodging in normal arteries [11]. Probably, the potential for immediate opening of collateral arteries accounts for the temporary nature of any resulting neurologic complications. It has been shown that sudden obliteration or excision of a large intracranial malformation may lead to a severe edema or hemorrhage in the brain surrounding the arteriovenous malformation. The cause for this complication has been called normal perfusion breakthrough by Spetzler et al. [12]. They considered that the hemisphere surrounding an arteriovenous malformation is chronically ischemic with impairment of normal autoregulation. Then, after resection of the arteriovenous malformation, normal perfusion is reestablished and, since the arteries in the chronically ischemic brain have lost their capacity to autoregulate and decrease their diameter under the sudden excess blood load, intracerebral edema or hemorrhage results.

With embolization of the external carotid artery, the risks are: (1) facial nerve palsies when the accessory meningeal artery supplies the geniculate ganglion [13]; (2) vertebrobasilar stroke when either the ascending pharyngeal artery-vertebral artery anastomosis or the occipitovertebral artery anastomosis is patent [14, 15]; (3) carotid artery stroke if the emboli reflux alongside the catheter and enter the internal carotid artery [16]; and (4) tissue necrosis when all potential collateral channels to one area are occluded [13]. To prevent these complications, it is necessary to carry out meticulous preembolization angiography to identify and exclude those patients in whom the anastomoses are present. Careful monitoring while injecting the emboli, to insure that a runoff is always present in the external arterial branch, should prevent retrograde reflux.

Paraplegia or quadriplegia due to occlusion of the major spinal arteries is always possible after embolizing spinal arteriovenous malformations. Therefore, it is mandatory to identify the artery of the cervical enlargement or the artery of Adamkiewicz to the dorsal cord prior to carrying out embolization in either of these two areas [9].

Because of the potential for complications after embolization of the arteries of the neuraxis, it is crucial that the indication for embolization be specifically defined. Periodically, we need to ask ourselves what is being accomplished by embolization. Not every cerebral arteriovenous malformation should be embolized; in many cases a cortical lesion, particularly if situated in the poles of the brain, can be safely excised with minimal morbidity. In such cases we should ask whether the benefits of embolization outweigh the risks. Since intracranial arteriovenous malformations are seldom completely obliterated by embolization, what are the risks of leaving part of the arteriovenous malformation intact? We

know that additional arterial feeders develop in time, but is the patient with a partly embolized arteriovenous malformation at greater or lesser risk for subsequent seizures or hemorrhages? In a study in 1975, Luessenhop and Presper [17] found that subsequent to embolization with Silastic emboli, the risk of hemorrhage in patients who had not had a prior cerebral hemorrhage was less than in nonembolized patients. However, embolization had no effect on the incidence of hemorrhage in patients who previously had hemorrhages. The frequency of seizures was decreased by embolization.

It is critical that the results of embolization be meticulously reported. There is no alternative to providing candid, precise, and complete information. It is too early in the development of embolization to advocate one method, one catheter system, or one type of embolic material exclusively. Variant data and divergent viewpoints must not be suppressed, and everyone performing embolotherapy must try to remove any unconscious prejudices against alternative techniques. Dogmatism or exaggeration can easily creep into reporting the data because of a fear of seeming irresolute. On the other hand when the circumstances are justified, the data must be expressed absolutely and with conviction. The proponents of embolization should concentrate on reporting the long-term effects of their various techniques. Admittedly, in a rapidly advancing field, this can be difficult when insufficient time has elapsed to assess these results. Nevertheless, only by rigorous follow-up studies can the efficacy of embolization be determined.

It is evident that the neuroradiologist advocating and carrying out embolization has a direct responsibility to the patient. Close cooperation with the referring physician is necessary throughout every phase of the procedure. The physician team must carefully choose the cases, discuss the vessels to be embolized, monitor the patient's condition throughout the continuously changing radiologic studies, and evaluate the effect of therapy [6].

In the same way that microneurosurgery with the operating microscope is a technique used only by qualified neurosurgeons, embolization should be carried out only by qualified neuroradiologists. Specialization within neuroradiology is healthy, desirable, and ultimately to the advantage of the patient. There is no substitute for repetitive trials of the different techniques on animals until one becomes completely familiar with the newer catheters and different embolic materials being used. Only when the neuroradiologist is thoroughly comfortable with the equipment should patient procedures be undertaken, and then under supervision. The development of competence is a slow process. Undoubtedly, with improvement in skills and with development of newer and safer catheters and embolic materials, the complication rate will decrease.

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REFERENCES

1. Dawborn RHM. The starvation operation for malignancy in the external carotid area. *JAMA* 1904;17:792-795
2. Djindjian R, Picard L, Manelfe C, Merland JJ, Theron J. Devel-

- opment de la neuroradiologie therapeutique. *Neuroradiology* **1978**;16:381-384
3. Luessenhop AJ, Spence WT. Artificial embolization of cerebral arteries: report of use in a case of arteriovenous malformation. *JAMA* **1960**;172:1153-1155
 4. Kerber CW. Intracranial cyanoacrylate. A new catheter therapy for arteriovenous malformation (letter). *Invest Radiol* **1975**;10:536-538
 5. Berenstein A, Kricheff II. Catheter and material selection for transarterial embolization: technical considerations. I. Catheters. II. Materials. *Radiology* **1979**;132:619-639
 6. Hilal SK, Michelsen JW. Therapeutic percutaneous embolization for extra-axial vascular lesions of the head, neck, and spine. *J Neurosurg* **1975**;43:275-287
 7. Debrun G, Lacour P, Caron JP, Hurth M, Comey J, Keravel Y. Detachable balloon and calibrated-leak balloon techniques in the treatment of cerebral vascular lesions. *J Neurosurg* **1978**;49:635-649
 8. Doppman J, DiChiro G, Ommaya A. Percutaneous embolization of spinal cord arteriovenous malformations. *J Neurosurg* **1971**;34:48-55
 9. Djindjian R, Houdart R, Cophignon J, Hurth M, Comoy J. Premiers essais d'embolisation par voie femorale dans un cas d'angiome medullaire. *Rev Neurol (Paris)* **1971**;125:119-130
 10. Djindjian R. Embolization of angiomas of the spinal cord. *Surg Neurol* **1975**;4:411-419
 11. Wolpert SM, Stein BM. Factors governing the course of emboli in the therapeutic embolization of cerebral arteriovenous malformations. *Radiology* **1979**;131:125-131
 12. Spetzler RF, Wilson CB, Weinstein P, Mehdorn M, Townsend J, Telles D. Normal perfusion pressure breakthrough theory. *Clin Neurosurg* **1978**;25:651-672
 13. Bentson J, Rand R, Calcaterra T, Lasjaunias P. Unexpected complications following therapeutic embolization. *Neuroradiology* **1978**;16:420-423
 14. Lasjaunias P, Moret J. The ascending pharyngeal artery. *Neuroradiology* **1976**;11:77-82
 15. Lasjaunias P, Moret J, Theron J. The so-called anterior meningeal artery of the cervical vertebral artery. Normal radioanatomy and anastomoses. *Neuroradiology* **1978**;17:51-55
 16. Djindjian R, Cophignon J, Merland JJ, Houdart R. Embolization by superselective arteriography from the femoral route in neuroradiology. Review of 60 cases. I. Technique, indications, complications. *Neuroradiology* **1973**;6:20-26
 17. Luessenhop AJ, Presper JH. Surgical embolization of cerebral arteriovenous malformations through internal carotid and vertebral arteries. Long-term results. *J Neurosurg* **1975**;42:443-451