

# Preoperative Embolization of Intracranial Meningiomas

Claude Manelfe<sup>1</sup>  
 Pierre Lasjaunias<sup>2</sup>  
 Jordi Rusalleda<sup>3</sup>

The goal of preoperative embolization of intracranial meningiomas is to facilitate their surgical removal by reducing tumor vascularity and decreasing blood loss during surgery. This study is based on personal experience with about 100 embolized meningiomas and on the experience of others. Embolization is performed during the same session as diagnostic angiography. The appropriate embolic materials (absorbable or nonabsorbable) are chosen according to the location of the tumor, the size of the feeding arteries, the blood flow, and the presence of any potentially dangerous vessels (dangerous anastomoses between external carotid artery and internal carotid or vertebral arteries, arteries supplying the cranial nerves). Preoperative embolization appeared to be very useful in large tumors with pure or predominant external carotid artery supply (convexity meningiomas), in skull-base meningiomas, and in middle fossa and paracavernous meningiomas. It was also useful in falx and parasagittal meningiomas receiving blood supply from the opposite side and in posterior fossa meningiomas. CT low densities demonstrated after embolization did not always correlate with necrosis on microscopic examination, and large areas of infarction could be found despite normal CT. Embolic material was found on pathologic examination in 10%–30% of cases; fresh or recent ischemic and/or hemorrhagic necrosis consistent with technically successful embolization was demonstrated in 40%–60% of cases. With careful technique complications are rare.

Meningiomas are considered to be benign and potentially curable tumors. They account for 13%–18% of all intracranial neoplasms [1]. After total tumor removal, tumor recurrence varies from 9% to 11% if the dural attachment is resected and from 19% to 22% if the dural attachment site is cauterized but not excised. In cases of partial tumor removal, the risk of recurrence increases to 37%–40% [2, 3]. Parasagittal and falx meningiomas recur nearly twice as often as do the meningiomas of the convexity. Meningiomas of the sphenoid ridge and posterior fossa recur in more than 20% of cases [3]. Incomplete tumor removal is related to many factors, such as tumor hypervascularity, dural venous attachment, tumor size, and tumor location deep within brain or near vital neural structures. Of these factors the ones most susceptible to control are the degree and origin of the hemorrhage during surgery.

For all these reasons several authors have used transcatheter embolization in highly vascular tumors supplied by the external carotid artery, either to decrease preoperative bleeding and facilitate surgical removal or to palliate inoperable cases [4–10]. In 1973, we published our preliminary results with embolization of intracranial meningiomas [11]. Since then, other experiences have been reported [12–20], some with large series [21–24].

Since the development of computed tomography (CT), the indications for angiography in the diagnosis of intracranial meningiomas have changed markedly. Accuracy of CT diagnosis of meningiomas is in the range of 90%–95% [25]. Calcifications, hyperostosis, dural abutment, and increased tumor density are seen on plain CT scans; homogeneous enhancement is usually observed in postcontrast

Received March 13, 1986; accepted after revision May 28, 1986.

Presented at the annual meeting of the American Society of Neuroradiology, New Orleans, February 1985.

<sup>1</sup> Department of Neuroradiology, Hôpital Purpan, 31059 Toulouse Cédex, France. Address reprint requests to C. Manelfe.

<sup>2</sup> Department of Radiology, Hôpital Bicêtre, Université Paris XI-Orsay, 94270 Kremlin-Bicêtre, France.

<sup>3</sup> Department of Neuroradiology, Hospital de la Santa Creu i San Pau, 08025 Barcelona, Spain.

**AJNR 7:963–972, September/October 1986**

0195–6108/86/0705–0963

© American Society of Neuroradiology

CT. For these reasons some neurosurgical teams operate on most meningiomas on the basis of the CT findings and do not use angiography.

However, angiography remains very important for several reasons: (1) CT diagnosis may be in doubt because of atypical CT appearance including cyst, ring enhancement, or absence of enhancement. Hemorrhage, necrosis, lipomatous degeneration, scarring, and cystic change are frequently observed on pathologic examination of benign meningiomas [26]. (2) Only angiography can provide the vascular data necessary for planning surgery, specifically the origin, size, and course of the feeding arteries, demonstration of arterial encasement or occlusion at the base of the brain, and dural sinus patency [27]. (3) There is no obvious relation between CT findings and angiographic vascularity and there is no specific CT appearance that indicates purely external carotid artery supply to the tumor.

The goals of preoperative embolization are (1) to reduce vascularity and facilitate surgical removal by decreasing blood loss during the operation and (2) to decrease the likelihood of tumor recurrence by specifically producing necrosis at the site of dural attachment [11, 19]. We will review the techniques, indications, and results of preoperative embolization of intracranial meningiomas according to our own experience and to that of others.

### Technique

Because embolization is regarded as a tool to facilitate surgical removal of the tumor, embolization must be as safe, painless, and easy to perform as possible. For that reason embolization is usually performed by the Seldinger technique via the femoral route in the same session as diagnostic angiography. Early in our experience we used general anesthesia [11, 21] to avoid patient discomfort during catheterization and embolization of those external carotid artery branches that supply the meningioma. More recently, nonionic contrast media with reduced osmolality have been used to decrease the sensation of heat, local pain, and other unpleasant side effects, permitting us to employ neuroleptic analgesia instead. With neuroleptic analgesia the patient is drowsy but remains conscious and arousable, so he can be tested for neurologic function as needed. Digital subtraction angiography (DSA) is particularly well adapted to interventional procedures because it has superior contrast resolution and provides immediate subtraction images [28]. DSA also allows the angiographer to use smaller catheters and to reduce the total dose of contrast medium by about 50%.

We routinely use 4–5 French catheters (Ingenor, Paris) with simple curves for superselective angiography and embolization in the external carotid artery territory. When we need to reach distal and tortuous vessels, like branches of the internal maxillary artery, without producing arterial spasm, we use the polyurethane catheter of Berenstein and Kricheff [29, 30] (USCI International, Paris) that tapers from 7 to 5 French, with a distal 105° curve. Superselective catheterization is mandatory to avoid migration of emboli to normal vascular territories [31].

Selection of the appropriate embolic materials depends on the location of the tumor, size of the feeding arteries, blood flow, and presence of any potentially dangerous collateral pathways. Meningiomas are highly vascular tumors with hypertrophied meningeal pedicles; however, tumoral circulation time is often slow, and arteriovenous shunts are unusual [32]. Absorbable embolic agents are chosen whenever emboli could possibly migrate aberrantly during embolization of the tumor despite superselective catheterization. Thus, during embolization in the middle meningeal artery or the internal maxillary artery, the possibility of migration of emboli into the superficial temporal artery, sphenopalatine artery, or dental branches requires use of absorbable embolic material. Similarly one must use absorbable material whenever embolization is undertaken in vessels that have potential anastomoses between external carotid artery branches and internal carotid or vertebral arteries and whenever the arteries that supply the vasa nervorum of the lower cranial nerves are embolized. There are the “dangerous vessels,” and full knowledge of their origins and interconnections is mandatory for safety during embolization. For all these reasons, gelatin sponge (Gelfoam) is the most frequently used material in preoperative embolization of meningiomas [4, 11, 18, 21, 23, 24, 31, 33]. It is absorbable, easy to handle, and well tolerated [34]. Embolization with Gelfoam is performed either with small strips or particles ranging in size from 0.5 × 0.5 mm to 1 × 5 mm or with Gelfoam powder (particles around 60 μm). Of these two, Gelfoam powder penetrates farther into the tumor bed and produces distal vascular occlusion. A higher incidence of tumor necrosis is noted at pathologic examination after embolization with Gelfoam powder (18, 19, 35) than after embolization with particles of Gelfoam.

Nonabsorbable embolic material may be used when aberrant embolization of adjacent normal territories is unlikely to produce ischemia. Nonabsorbable emboli achieve more durable occlusion. Such nonabsorbable agents include blenderized (150–250 μm and 300–500 μm) or finely cut polyvinyl alcohol foam (PVA) particles [36] and lyophilized dura mater (Langemarckplatz, Erlangen, W. Germany) [17, 23, 24]. Horton et al. [37] demonstrated that embolization with a mixture of Gelfoam and PVA foam (PVA particles/Gelfoam powder or PVA microparticles/Gelfoam particles) retains the advantages and eliminates the disadvantages of each. Hieshima et al. [22] have used 1 mm barium-impregnated Silastic spheres and believe that they offer the best combination of precise placement and permanent vascular occlusion.

We have not been using this technique as it cannot satisfy our requirements for presurgical embolization in tumors (see below). We also have used 300 μm radiopaque calibrated polyethylene microspheres (Ingenor, Paris) to produce very distal occlusions when there are no dangerous vessels or anastomoses. Gelfoam, PVA, and dura mater are not radiopaque. They need to be injected under fluoroscopic control in a solution of nonheparinized saline mixed with contrast medium. Emboli must be injected slowly, using the normal blood flow to direct the particles to the tumor.

Embolization is stopped (1) when the tumor blush of feeding vessels close to the tumor can no longer be seen and (2)

when the blood flow toward the tumor decreases. The goal of preoperative embolization is to occlude vessels within the tumor, not just the large feeding vessels. Therefore, the size of the emboli is predetermined by the diameters of the feeding arteries, the blood flow, and the vascular territories supplied by each vessel. As a general rule one must avoid particles smaller than 100  $\mu\text{m}$  when the arterial trunk to be occluded gives rise to either (1) potentially dangerous collateral supply to the internal carotid or vertebral arteries or (2) supply to the vasa nervorum of cranial nerves (VII, IX, X, XI, and XII) [38]. Dangerous external carotid collateral routes are summarized in Figure 1.

In patients with large meningiomas of the skull base that invade the cavernous sinus or in patients with recurrent and/or malignant meningiomas, a more aggressive endovascular technique is needed. In these patients, the internal carotid artery can be occluded proximal to the origin of the ophthalmic artery by a detachable balloon, according to the technique of Debrun et al. [39, 40]. Accurate placement of the balloon in the cavernous portion of the internal carotid artery is sometimes difficult because of encasement of this vessel by the tumor (Fig. 2).

Balloon occlusion of the internal carotid artery is performed after functional testing of the circle of Willis. If temporary occlusion of the internal carotid artery is not clinically tolerated, a superficial temporal artery–middle cerebral artery an-

astomosis can be performed before balloon occlusion of the internal carotid artery. The internal carotid artery is occluded below the ophthalmic artery to preserve the patient's vision. However, with the balloon in this position, collateral, retrograde flow through the ophthalmic artery from branches of the external carotid artery may result in bleeding during surgery [40]. This bleeding must be accepted, since vision must be preserved if at all possible. When both internal carotid artery and middle meningeal artery embolization have to be performed, they should be carried out in the this order: middle meningeal artery followed by internal carotid artery. Internal carotid occlusion will open internal maxillary artery branches—ophthalmic anastomoses. If, however, the eye is already blind, then the balloon should be detached opposite to the origin of the ophthalmic artery to occlude this source of hemorrhage as well. Thereafter, any remnant feeders can be embolized with microspheres, microparticles of PVA, dura mater, or even isobutyl-2-cyanoacrylate.

### Indications

The indications and results of preoperative embolization are based on our own experience with about 100 meningiomas embolized since 1972 [9, 11, 21] and on the experience of others [19, 22–24]. The indications for preoperative embolization depend on the (1) specific neurosurgical team with which you work, (2) external carotid artery supply, (3) tumor location, and (4) tumor size. Some neurosurgeons operate on most meningiomas on the basis of CT findings alone and consider embolization useless. During surgery, they can easily clip the meningeal feeders and remove the tumor, avoiding excessive bleeding. However, some meningiomas, because of their location, size, and vascularity, are exceptionally difficult to remove. In such tumors, preoperative embolization can be very valuable. In special cases, embolization may even permit successful resection of a meningioma after the initial attempt proved unsuccessful because of uncontrollable hemorrhage [22].

The vascular supply of meningiomas has been well demonstrated by Salamon et al. [41]. Independent of the location of the tumor, intracranial meningiomas have two different arterial supplies: (1) feeders at the site of dural attachment that arise from meningeal arteries (pedicles d'insertion) and radiate deeply to the center of the tumor and (2) feeders of the tumor capsule that arise from pial and cortical arteries (pedicles de capsule), entering the capsule from the brain surface to supply the periphery of the tumor. This vascular pattern can be well demonstrated on surgical specimens after injection of the meningeal pedicle with barium sulfate (Fig. 3). In vivo, angiography readily differentiates "capsular" pedicles arising from cortical and pial branches (Fig. 4) from "dural" pedicles arising from the external carotid artery (middle meningeal artery, accessory middle meningeal artery, neuromeningeal branch of the ascending pharyngeal artery, and stylo-mastoid branch of the occipital artery), from the internal carotid artery (ethmoidal, cavernous, clival, and tentorial branches), and from the vertebral artery (posterior meningeal artery).

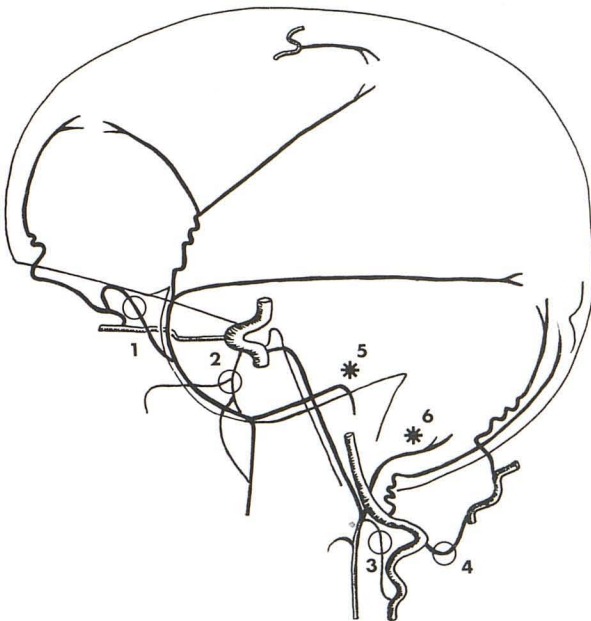


Fig. 1.—Schematic representation of dangerous anastomoses (circles) and dangerous vessels (asterisks) supplying cranial nerves: (1) anastomosis between ophthalmic artery system and middle meningeal artery; (2) anastomosis between intracavernous internal carotid artery and accessory meningeal and middle meningeal arteries; (3) anastomosis between vertebral artery and ascending pharyngeal artery through odontoid arterial arch; (4) anastomosis between vertebral artery and occipital artery (first and second cervical spaces); (5) petrous branch of middle meningeal artery supplying seventh cranial nerve; and (6) neuromeningeal branch of ascending pharyngeal artery supplying cranial nerves IX–XII.

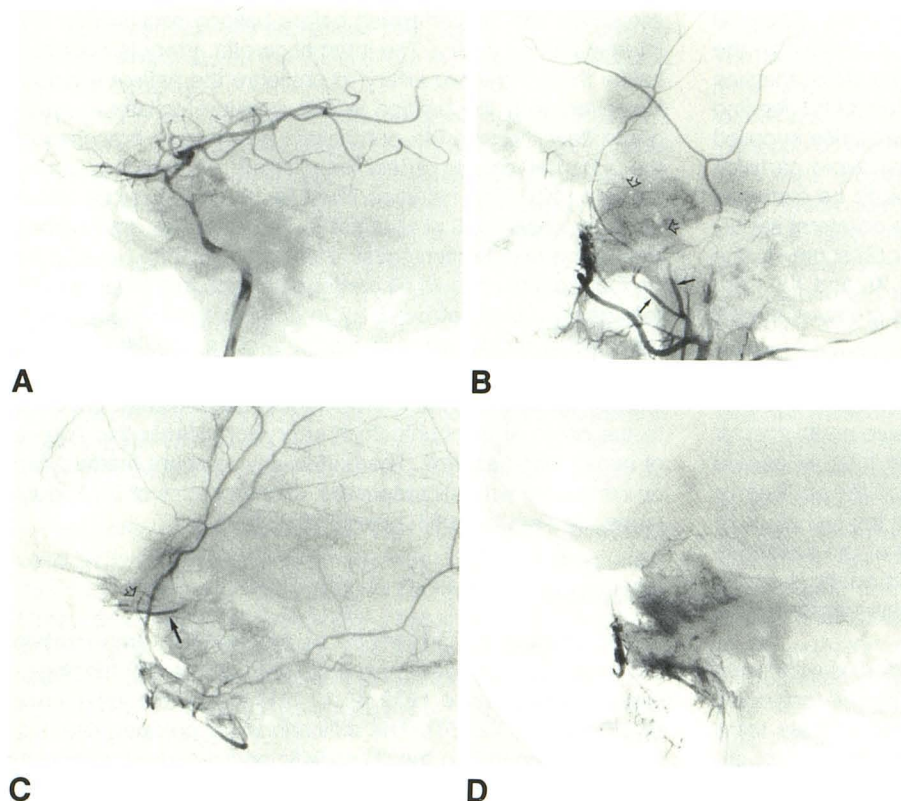


Fig. 2.—Right cavernous sinus meningioma. **A**, Right internal carotid angiogram. Marked irregular stenosis of cavernous portion of internal carotid artery, which is encased by tumor and supplied by multiple intracavernous branches. Two balloons were detached in internal carotid artery: the first at C3 (knee of internal carotid artery under anterior clinoid process) below ophthalmic artery; the second in ascending portion of C5 (ganglionic segment). **B**, Right external carotid artery. Meningioma is fed by middle meningeal and accessory meningeal arteries (solid arrows). Note clips (open arrows) of the two detached balloons in carotid siphon. **C**, Selective injection in right middle meningeal artery shows faint tumoral blush but opacifies ophthalmic artery (solid arrow) through meningioophthalmic anastomosis (open arrow). **D**, Superselective injection in accessory meningeal artery shows marked tumoral blush. This vessel was embolized with Ivalon particles of 500  $\mu$ m to protect ophthalmic artery; middle meningeal artery was occluded with large piece of Gelfoam.

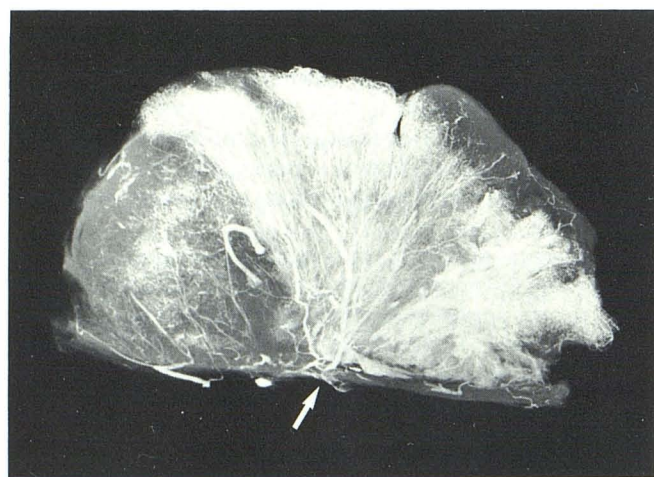


Fig. 3.—Microangiogram of surgical specimen of convexity meningioma after injection of main meningeal pedicle with barium sulfate. From dural attachment (arrow), meningeal branches radiate deeply to center of tumor. Nonopacified portion at periphery of tumor corresponds to territory of pial and cortical branches arising from internal carotid artery.

The vascular supply of meningiomas can be considered in four categories: (1) meningiomas supplied solely by the external carotid artery; (2) meningiomas with mixed external/internal carotid arterial supply but with dominant feeders from the external carotid artery territory; (3) meningiomas with

mixed external/internal carotid artery supply but with dominant feeders from the internal carotid artery territory; and (4) meningiomas with pure internal carotid artery supply such as sellar meningiomas (diaphragmatic, tuberculum) or proximal tentorial meningiomas. The "balance" between the dural and the cortical or pial vascular supply depends mainly on the location and, to a lesser degree, on the size of the tumor. Sometimes, anatomic variations, such as ophthalmic origin of the anterior branch of the middle meningeal artery, preclude the embolization of one territory.

Meningiomas can be divided arbitrarily into three different groups according to their size [3]: (1) giant meningiomas 7 cm in diameter or larger; (2) large tumors 4.5–7.0 cm in diameter; and (3) small tumors 4.5 cm or less in diameter. The best indications for preoperative embolization are large, highly vascular tumors supplied mainly by the external carotid artery (Fig. 5). Embolization is particularly indicated in meningiomas of the skull base and middle cranial fossa in which the arterial supply is otherwise difficult, if not impossible, to occlude before surgical removal of the bulk of the tumor. Falx and parasagittal tumors, which represent almost one-third of all meningiomas, frequently receive their main vascular supply from the opposite middle meningeal artery and may be easier to resect after embolization (Fig. 6).

The meningeal component of large posterior fossa meningiomas—mainly those of the clivus, cerebellopontine angle, and posterior aspect of the petrous bone—can be embolized through the meningeal branch of the ascending pharyngeal

Fig. 4.—Left parietal convexity meningioma. Selective internal (A) and external (B) carotid lateral subtraction angiograms. Pial and cortical branches of middle cerebral artery supplying periphery of tumor (arrows) and branches of middle meningeal artery at site of dural attachment (arrows).

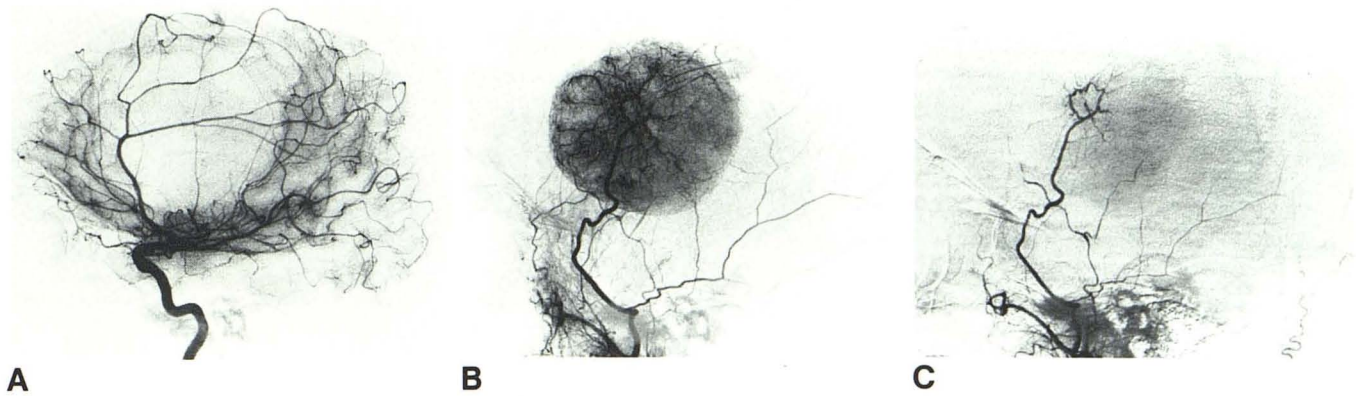
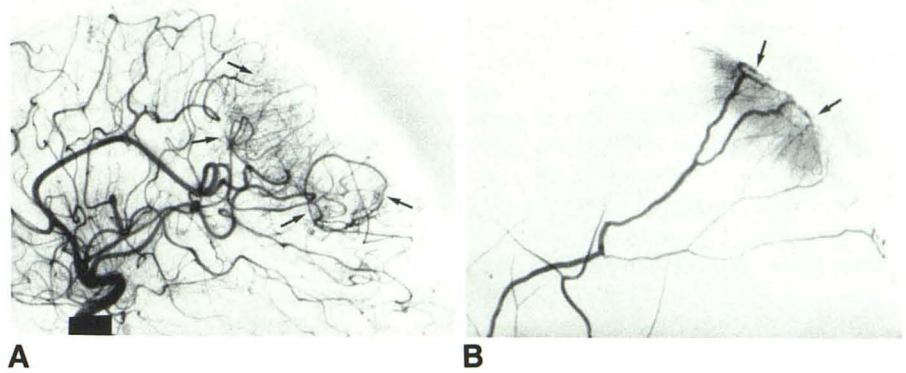
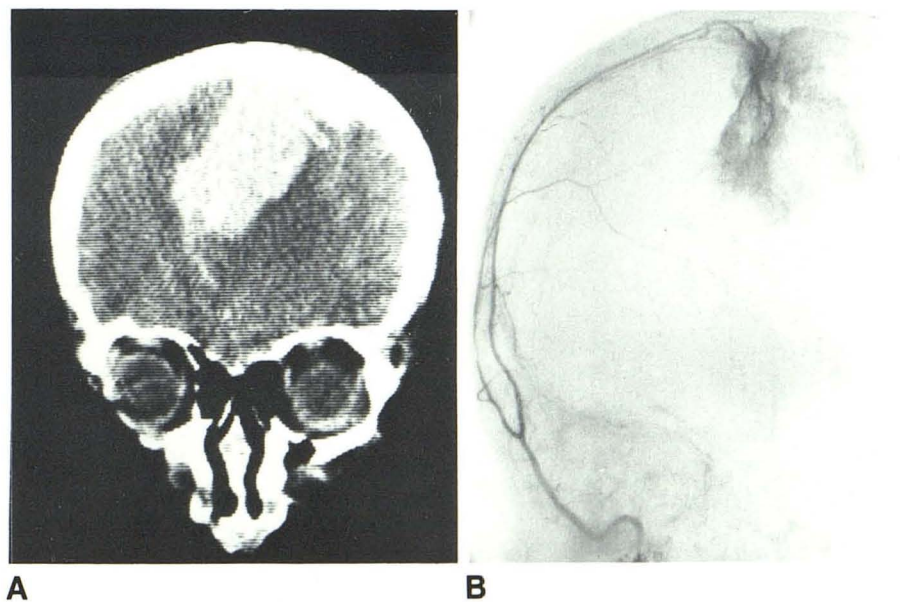


Fig. 5.—Large left frontorolandic convexity meningioma. Selective internal (A) and external (B) carotid lateral subtraction angiograms. Tumor is almost exclusively supplied by anterior branch of middle meningeal artery. C, After

selective embolization in middle meningeal artery with Ivalon particles of 300  $\mu$ m tumor blush disappears with preservation of main trunk of middle meningeal artery.

Fig. 6.—Left falx and parasagittal meningioma. A, Postcontrast CT scan, direct coronal section. Large homogeneous enhancing mass involves left side of falx and frontorolandic parasagittal region. B, Right external carotid subtraction angiogram, anteroposterior view. Dural attachment of meningioma on falx is supplied by right middle meningeal artery, which was embolized with dura matter particles. Left external carotid artery was normal.



artery [15] (Fig. 7). The less common orbital meningiomas may be embolized via the ophthalmic artery if the eye is already blind or is to be sacrificed [35].

Tumors with mixed internal and external carotid arterial supply have been reported to show increased flow from the internal carotid artery after occlusion of the external carotid feeders. If this is the situation, embolization may be of limited benefit or even contraindicated [4, 23, 24]. In our experience [9, 11, 21] as well as in that of others [23], however, embolization of the external carotid component of mixed vascular supply (cavernous and middle temporal fossa meningiomas) usually decreases the bleeding from the internal carotid artery branches. The reduced vascularity then makes surgery easier. Even in cases of meningiomas with mixed external/internal carotid artery supply, very distal embolization of the meningeal source may occlude small vessels within the tumor bed, causing softening and necrosis of the central portion of the tumor [18, 22, 35]. As a result, the surgeon may be able to retract the tumor more easily, establish a cleavage plane between tumor and brain, and reach the cortical (capsular) supply of the tumor to control hemorrhage before resecting

the bulk of the tumor. This leads to simpler and safer tumor removal (Fig. 8).

**Results**

The effects of embolization can be appreciated by different means: (1) preoperatively by angiography and CT; (2) intraoperatively by direct inspection and quantification of blood loss; (3) postoperatively by pathologic examination; and (4) by amount of recurrence rate.

*Postembolization Angiography*

In some cases, embolization achieves near-obliteration of the small vessels within the tumor. In other cases, it merely obstructs large vessels proximal to the tumor without truly occluding the tumor vessels themselves. Since the best embolizations and best surgical results are obtained with very small particles that enter the tumor nidus, postembolization angiography is performed to determine the extent of obliteration of the tumor bed and the feeding vessels. In our expe-

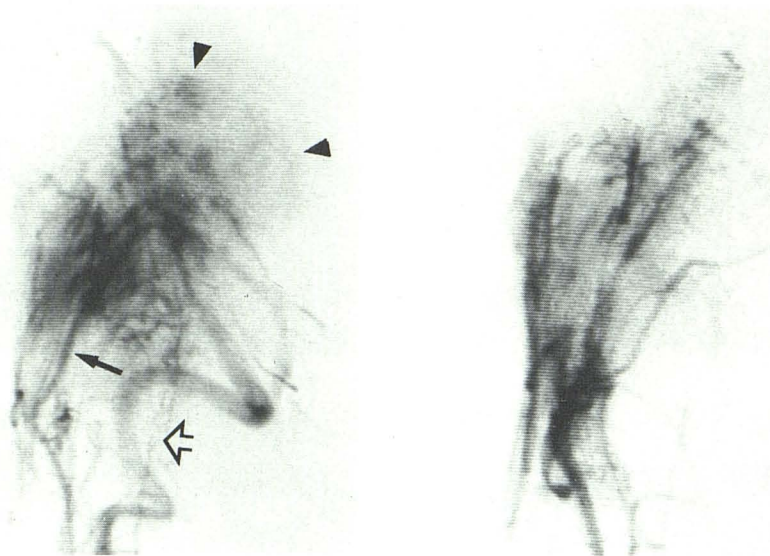


Fig. 7.—Right posterior fossa meningioma. A, Lateral digital subtraction angiogram of right ascending pharyngeal artery before embolization. Tumor involving posterior aspect of petrous bone (arrowheads) is supplied by branches from neuro-meningeal trunk (solid arrow) of ascending pharyngeal artery. Ipsilateral vertebral artery is faintly injected through anastomotic arch of odontoid (open arrow). B, After selective embolization in ascending pharyngeal artery with Ivalon particles of 500  $\mu$ m, tumor blush is not seen; anastomotic arch of odontoid is preserved.

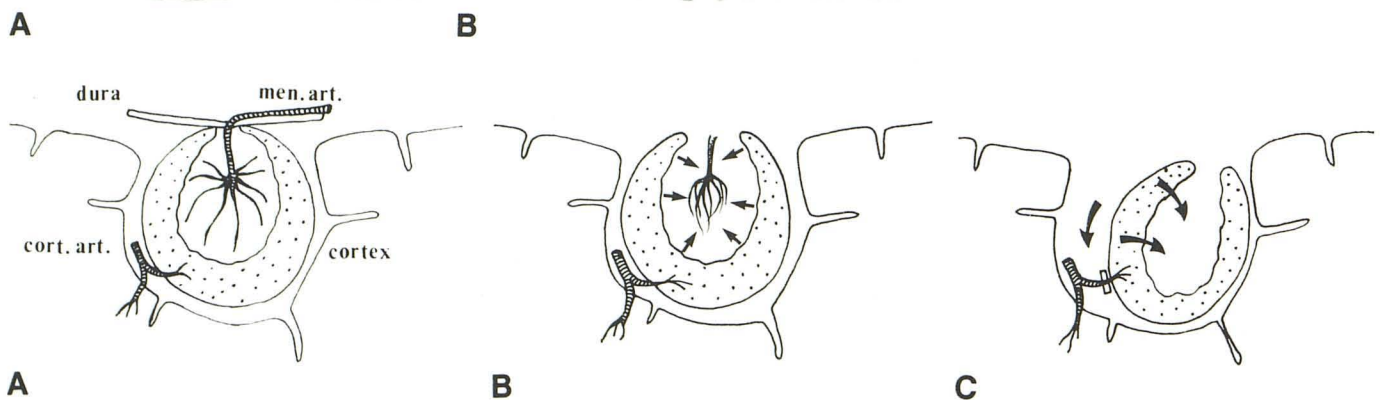


Fig. 8.—Diagram of vascular supply to meningioma. A, Dural or meningeal arteries supply central part of tumor, whereas pial and cortical arteries supply periphery. B, Embolization of meningeal pedicles produces central softening

allowing surgeon (C) to separate tumor from cortex more easily to reach and clip capsular arteries.

rience, it is preferable to perform postoperative embolization angiography of the external carotid artery only. When external carotid arteriography reveals disappearance of the tumor blush, embolization has been successful.

When external carotid arteriography reveals occlusion of some, even many, major arterial feeders but persistence of some tumor blush, greater blood loss may be expected at surgery. Early in our experience [11, 21] we also performed postembolization internal carotid angiography to evaluate the remnant or additional arterial supply from this territory and to be sure of the absence of inadvertent internal carotid emboli. However, we no longer perform internal carotid angiography after embolization of external carotid territory. Rather, we agree with Teasdale et al. [24] that postembolization internal carotid arteriography is unwise and may indeed be a source

of complications if microemboli remain within the catheter. Moreover, failure to visualize tumor vessels issuing from the internal carotid artery does not exclude an arterial supply from this territory [23].

#### Postembolization CT

Postembolization CT was performed in our institution mainly after 1977. Most patients were investigated within 3 days after embolization; one patient was scanned immediately after embolization and 24 hr later (Fig. 9). In about half of the contrast-enhanced CT scans obtained after embolization, low-density areas were demonstrated within previously homogeneous tumors and were interpreted as necrosis. When these tumors were examined pathologically, however, CT and

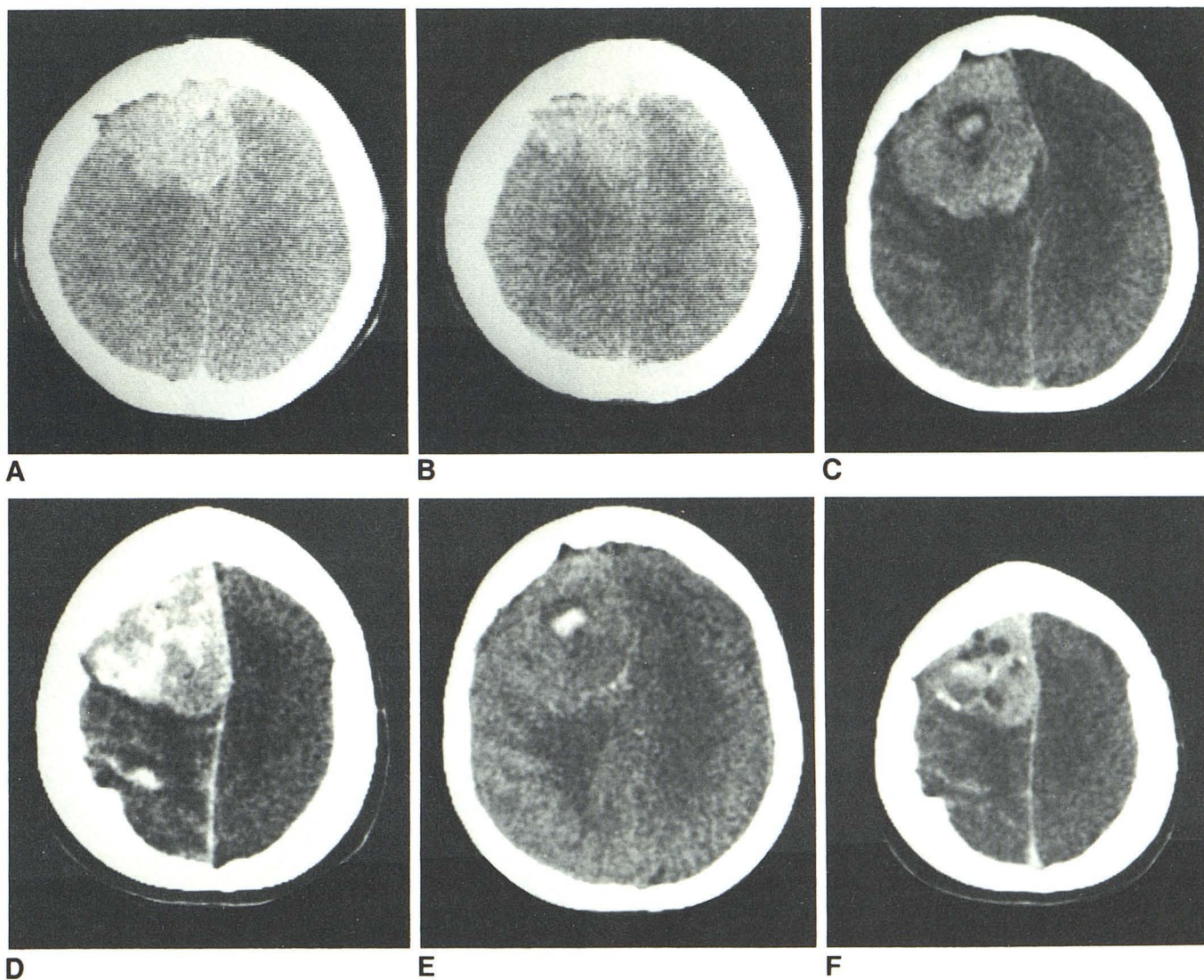


Fig. 9.—Right frontal meningioma. **A** and **B**, Postcontrast CT scans. Homogeneous enhancing mass in frontal parasagittal convexity region. **C** and **D**, After embolization with Gelfoam particles in right external carotid artery. Multiple, irregular patchy densities within tumor. Rounded increased density within

tumor in **C** was interpreted as small focus of hemorrhage. **E** and **F**, Noncontrast scans 24 hr after embolization. Low-density areas within tumor consistent with necrosis. No change in central increased density.

pathologic examination were in agreement in only one-third of cases. In two-thirds of the specimens, CT low densities did not correlate with necrosis on microscopic examination, and large areas of infarction were present despite normal CT. Teasdale et al. [24] found changes suggesting necrosis in eight of 17 patients examined by CT, of whom seven had only external carotid artery feeding vessels. In most patients without change on contrast-enhanced postembolization CT, there was mixed external/internal carotid or a dominant internal carotid artery supply. We have not found any increase in the size of the tumor or of the peritumoral edema after embolization. We have not found, as Hieshima et al. [22] noted, a decrease in tumor size after embolization on either CT or isotope brain scans.

#### Surgical Findings

Results are appreciated according to the facility of surgical removal and to the importance of blood loss. In our series [11, 21], preoperative embolization appeared very useful in large tumors with pure or predominant external carotid artery supply, such as convexity meningiomas (Fig. 4), in skull base meningiomas, and in middle fossa and paracavernous meningiomas (Fig. 2). Preoperative embolization appeared useful in falx and parasagittal meningiomas receiving contralateral blood supply (Fig. 6) and in posterior fossa meningiomas (Fig. 7).

Teasdale et al. [24] reported that embolization facilitated surgery in 13 of 26 patients. Of the 13 patients in whom embolization was considered beneficial, nine had purely external carotid feeders. Of those with persistent bleeding or no appreciable angiographic improvement, only one had an entirely external supply; in that case, despite the apparent lack of benefit, the tumor showed evidence of necrosis on post-embolization CT [24]. Marked hemorrhage during surgery occurred only in four patients, and all of them had significant internal carotid artery supply.

No obvious relations were observed between the subjective assessment of usefulness of embolization and either the histologic type of meningioma or the precise interval between embolization and surgery [21]. However, resection must be performed within 2–5 days after embolization.

#### Pathological Findings

Histologically, meningiomas may be classified as meningo-thelial, transitional, fibroblastic, angioblastic, and malignant. Objective assessment of the effects of embolization is difficult because tumor specimens given to the pathologist consist most often of multiple small fragments rather than an intact tumor. Therefore, a central zone of infarction is seldom demonstrated [22, 24]. It is noteworthy that many studies have failed to show massive necrosis of the tumor after embolization [11, 21, 23, 24]; however, multiple areas of necrosis and hemorrhagic infarctions have occurred fairly often after embolization with Silastic spheres [22].

Emboic material is found in arterial feeders in 10%–30% of examinations; Gelfoam particles may be seen in vessels of

100  $\mu$ m [11, 21] and frequently are associated with thrombosis (30%) (Fig. 10). Fresh or recent ischemic and/or hemorrhagic necrosis consistent with a technically successful embolization was seen in 40%–60% of cases (Figs. 11 and 12). However, small foci of necrosis are not uncommon in meningiomas, and histopathologic demonstration of iatrogenic infarction must be based on strict criteria such as a large circumscribed area of pallor associated with nuclear disintegration with or without an inflammatory response [24].

#### Complications

Preoperative embolization of intracranial meningiomas must be as safe as possible to avoid complications, which can be divided into minor and severe.

*Minor complications* are mainly represented by painful trismus and/or facial pain; they are related to ischemic phenomena when some normal internal maxillary artery branches (dental, buccal, masseteric) are occluded during the proce-

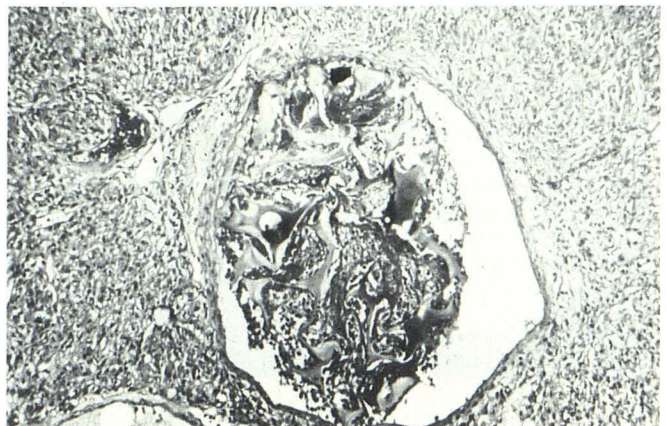


Fig. 10.—Histopathologic specimen of meningioma embolized with Gelfoam 3 days before. Gelfoam particles associated with early organized arterial thrombus within artery. No perivascular necrosis. (Masson's trichrome  $\times 10$ .)

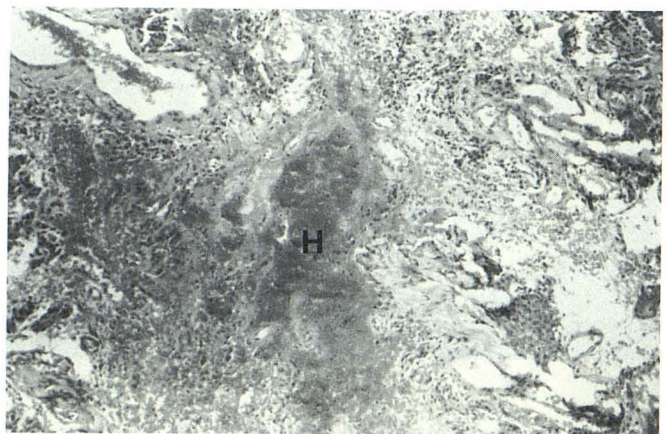


Fig. 11.—Focal area of hemorrhage (H) within embolized meningioma with Ivalon. (Hemalum-eosin  $\times 2.5$ .)



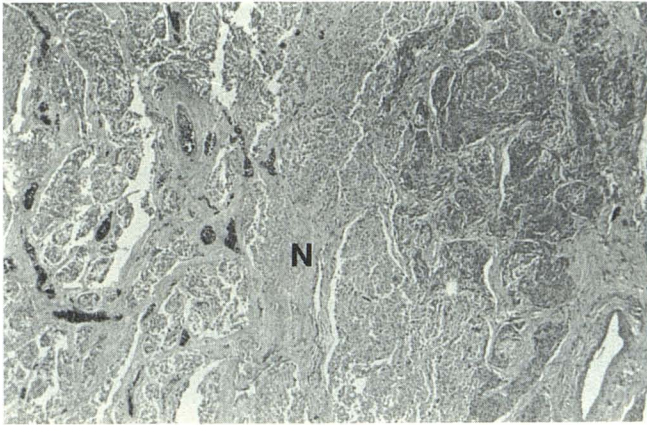


Fig. 12.—Area of pallor related to ischemic necrosis (N). (Hemalum-eosin  $\times 2.5$ .)

ture. They were present in about 20%–30% of our cases [21], but seem less common now probably because of the systematic use of corticosteroids in the days after embolization. When present, pain usually lasts 2–3 days and resolves without sequelae. Symptomatic treatment requires analgesics, but aspirin should be avoided due to its inhibitory effect on platelet aggregation.

Severe complications are represented by stroke and cranial nerve palsies. Stroke is exceptional [10] and usually is related to reflux of emboli in the internal carotid or vertebral artery. As we mentioned, careful technique and permanent fluoroscopic control are mandatory to avoid reflux of embolic material. Stroke can also result from embolization of territories vulnerable to dangerous anastomoses between the external carotid artery and internal carotid or vertebral artery (Fig. 1). Cranial nerve palsies (mainly VII and XII) represent a true therapeutic risk as the supply of most meningiomas is the same as the supply of the cranial nerves (Fig. 1). Currently, this risk represents the main limitation in the use of fluid agents. Recent technical advances (opened guidewire) allow catheterization of the tumoral feeders distal to the origin of the dangerous vessels. In most meningiomas preoperative embolization with particles can be carried out with minimal risks.

Because of the presence of estrogen and progesterone receptors in some tumors like meningiomas [42, 43], Suzuki and Komatsu [44] have used estrogen for embolization in three cases of middle cranial fossa and convexity meningiomas. The mechanism of vessel occlusion after estrogen administration is not completely clarified and probably results in injury of the vascular endothelium and increased vascular permeability. Even if the first results are promising, hormonal therapy should be considered as an alternative in patients too elderly or debilitated to undergo conventional surgical removal.

#### ACKNOWLEDGMENTS

We thank Janine Gallien and Annie Mele for secretarial assistance, Claude Vachon and Michel Gorda for iconography, Thomas Naidich

for reviewing the translation, and Maryvone Pradère for reviewing the pathologic material.

#### REFERENCES

- Russell DS, Rubinstein LJ. *Pathology of tumors of the nervous system*. Baltimore: Williams & Wilkins, 1977:65–100.
- Simpson D. The recurrence of intracranial meningiomas after surgical treatment. *J Neurol Neurosurg Psychiatry* 1957;20:22–39
- Chan RC, Thompson GB. Morbidity, mortality, and quality of life following surgery for intracranial meningiomas. A retrospective study in 257 cases. *J Neurosurg* 1984;60:52–60
- Djindjian R, Cophignon J, Theron J, Merland JJ, Houdart R. Embolization by super-selective arteriography from the femoral route in neuroradiology: review of 50 cases. III: Embolization in craniocerebral pathology. *Neuroradiology* 1973;6:143–152
- Manelfe C, Fardou H, David J, Combes PF. Embolisation thérapeutique par cathétérisme femoral pecutané. (A propos de vingt quatre cas.) *Ann Radiol (Paris)* 1974;17:571–592
- Manelfe C, Djindjian R, Picard L. Embolisation par cathétérisme fémoral des tumeurs irriguées par l'artère carotide externe. A propos de 40 cas. *Acta Radiol [Suppl]* (Stockh) 1975;347:175–185
- Hilal SK, Michelsen JW. Therapeutic percutaneous embolization for extra-axial vascular lesions of the head, neck, and spine. *J Neurosurg* 1975;43:257–287
- Kendall B, Moseley I. Therapeutic embolization of the external carotid arterial tree. *J Neurol Neurosurg Psychiatry* 1977;40:937–950
- Manelfe C, Picard L, Bonafe A, Roland J, Sancier A, L'Esperance G. Embolisations et occlusions par ballonnets dans les processus tumoraux. Sept années d'expérience. *Neuroradiology* 1978;16:395–398
- Brismar J, Cronqvist S. Therapeutic embolization in the external carotid artery region. *Acta Radiol [Diagn]* (Stockh) 1978;19:715–731
- Manelfe C, Guiraud B, David J, et al. Embolisation par cathétérisme des méningiomes intracrâniens. *Rev Neurol (Paris)* 1973;128:339–351
- Hekster RE, Matricali B, Luyendijk W. Presurgical transfemoral catheter embolization to reduce operative blood loss. *J Neurosurg* 1974;41:396–398
- Oberson R, Tribolet N, Campiche R, Probst A. Embolisation de l'artère méningée moyenne cathétérisée par voie fémorale pour l'hémostase pre-opératoire d'un méningiome de la convexité. *Schweiz Med Wochenschr* 1974;104:388–392
- Benati A, Maschio A, Perini S. Embolisation pré-opératoire par cathétérisme sélectif des méningiomes intracrâniens. *J Radiol* 1975;56:646–647
- Dilenge D, Calderon H. Cathétérisme super-sélectif et embolisation des pédicules artériels de deux méningiomes de la fosse postérieure. *Neurochirurgie* 1976;22:711–720
- Schumacher M, Gilsbach J, Seeger W, Mennel HD, Voigt K. Techniken und Ergebnisse bei Meningiome-Embolisationen. *Eur Arch Psychiatry Neurol Sci* 1979;227:241–260
- Fagioli L, Mavilla L, Nuzzo G, Calbucci F, Trevisan C. Preoperative embolization of brain meningiomas. *Acta Neurochir (Wien)* 1981;57:307
- Berenstein A, Russell E. Gelatin sponge in therapeutic neuroradiology: a subject review. *Radiology* 1981;141:105–112
- Berenstein A, Kricheff II. Microembolization technique of vascular occlusion: radiologic, pathologic and clinical correlation. *AJNR* 1981;2:261–267

20. Del Favero G. Preoperative embolization of intracranial meningiomas. *Radiol Med (Torino)* **1984**;70:113-117
21. Manelfe C. Transfemoral catheter embolization of intracranial meningiomas. In: Salamon G, ed. *Advances in cerebral angiography*, vol 1. Berlin: Springer-Verlag, **1975**:184-191
22. Hieshima GB, Everhart FR, Mehringer CM, et al. Preoperative embolization of meningiomas. *Surg Neurol* **1980**;14:119-127
23. Richter HP, Schachenmayr W. Preoperative embolization of intracranial meningiomas. *Neurosurgery* **1983**;13:261-268
24. Teasdale E, Patterson J, McLellan D, MacPherson P. Subselective preoperative embolization for meningiomas. A radiological and pathological assessment. *J Neurosurg* **1984**;60:506-511
25. New PFJ, Aronow S, Hesselink JR. National Cancer Institute study: evaluation of computed tomography in the diagnosis of intracranial neoplasms. IV. Meningiomas. *Radiology* **1980**;136:665-675
26. Russell EJ, George AE, Kricheff II, Budzilovich G. Atypical computed tomography features of intracranial meningioma. Radiologic-pathologic correlation in a series of 131 consecutive cases. *Radiology* **1980**;135:673-682
27. Handel SF. Computed tomography vs. angiography in the diagnosis of intracranial neoplasms. *Clin Neurosurg* **1981**;28:502-519
28. Brant-Zawadzki M, Gould R, Norman D, Newton TH, Lane B. Digital subtraction cerebral angiography by intraarterial injection: comparison with conventional angiography. *AJNR* **1982**;3:593-599
29. Berenstein A. Brachiocephalic vessel: selective and superselective catheterization. *Radiology* **1983**;148:437-441
30. Berenstein A, Kricheff II. Catheter and material selection for transarterial embolization: technical considerations. I. Catheters. *Radiology* **1979**;132:619-630
31. Djindjian R, Cophignon J, Theron J, Merland JJ, Houdart R. Embolization by superselective arteriography from the femoral route in Neuroangiography. Review of 60 cases. I. Technique, indications, complications. *Neuroangiography* **1973**;6:20-26
32. Wickbom I. Tumor circulation. In: Newton TH, Potts DG, eds. *Radiology of the skull and brain. Angiography*, vol 2, book 4. *Specific disease processes*. Saint Louis: Mosby, **1974**:2257-2285
33. Berenstein A, Kricheff II. Catheter and material selection for transarterial embolization: technical considerations. II. Materials. *Radiology* **1979**;132:631-639
34. Light RU, Prentice HR. Surgical investigation of new absorbable sponge derived from gelatin for use in hemostasis. *J Neurosurg* **1945**;2:435-455
35. Russell EJ, Berenstein A. Neurologic applications of interventional radiology. Neurologic symposium on neuroimaging. *Neurol Clin* **1984**;2:873-902
36. Tadavarthi SM, Moller JH, Amplatz K. Polyvinyl alcohol (Ivalon). A new embolic material. *AJR* **1975**;125:609-616
37. 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
38. Lasjaunias P. *Craniofacial and upper cervical arteries. Functional, clinical and angiographic aspects*. Baltimore: Williams & Wilkins **1981**
39. Debrun G, Lacour P, Caron JP, et al. Traitement de fistules artérioveineuses et d'anévrysmes par ballon gonflable et largable. *Nouv Presse Med* **1975**;4:2315-2318
40. Riche MC, Cophignon J, Thurel C, et al. Embolization of the cavernous and petrous segments of the internal carotid artery in severe basilar skull and petrous bone lesions. *J Neuroradiol* **1981**;8:301-315
41. Salamon G, Guerin G, Combalbert A, Faure JJ, Giudicelli G. Etude artériographique des méningiomes intracrâniens. Correlations radio-anatomiques. *Ann Radiol (Paris)* **1969**;12:661-679
42. Donnell MS, Meyer GA, Donegan WL. Estrogen-receptor protein in intracranial meningiomas. *J Neurosurg* **1979**;50:499-502
43. Poisson M, Magdalenat H, Foncin JF, et al. Récepteurs d'oestrogènes et de progestérone dans les méningiomes. *Rev Neurol (Paris)* **1980**;136:193-203
44. Suzuki J, Komatsu S. New embolization method using estrogen for dural arteriovenous malformation and meningioma. *Surg Neurol* **1981**;16:438-442