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Preoperative Embolization of the Head and Neck: Indications, Patient Selection, Goals, and Precautions

Anton Valavanis[1]

Preoperative embolization was performed in 39 patients with 44 paragangliomas of the head and neck. Because of their complex vascular supply and their relation to vital structures such as the internal carotid artery and the lower cranial nerves, paragangliomas of the temporal bone represent challenging lesions to both the neuroradiologist and the otoneurosurgeon. Detailed classification by high-resolution CT and recognition of the multi- or monocompartmental vascular composition and of dangerous situations by selective angiography are essential prerequisites for safe and effective devascularization of paragangliomas of the temporal bone. Major complications that may occur if embolic material reaches intraaxial vessels through anastomoses between external carotid artery branches and the internal carotid and/or the vertebral artery can be avoided with the use of specific precautionary techniques. Palsies of the facial and lower cranial nerves can also be avoided if reabsorbable material is used for embolization of vessels supplying cranial nerves in asymptomatic patients. In selected cases with significant supply from the internal carotid artery, special interventional techniques, including embolization of the pericarotid tumor portion through the caroticotympanic artery and pre- or peroperative balloon occlusion of the petrous internal carotid artery, allow radical removal of extensive paragangliomas of the temporal bone. Techniques and selection of materials for embolization of carotid body, vagal body, and other paragangliomas of the head and neck mainly depend on the vascular composition of the tumor and on the specific vascular territory in which the tumor is located. In this series, preoperative embolization significantly improved surgical conditions of paragangliomas of any location in the head and neck and proved to represent an essential prerequisite for successful surgery of extensive paragangliomas of the temporal bone.

Paragangliomas of the head and neck arise from the symmetrically and segmentally distributed, multicentric extraadrenal paraganglion system [1]. Therefore, typical locations for paragangliomas are, in decreasing order of frequency: the temporal bone (glomus tympanicum and jugulare tumors), the carotid bifurcation (glomus caroticum tumors), the upper parapharyngeal space (glomus vagale tumors), and rarely other regions of the head and neck harboring paraganglionic tissue, such as the orbit, the larynx and the upper airway [1]. Independent of their location in the head and neck, paragangliomas are highly vascular and histologically benign, but locally infiltrating, potentially lethal, and difficult to treat. In up to 10% of the cases, paragangliomas may appear at multiple sites. Rarely the tumors may exhibit clinically evident catecholamine secretion [1].

The neuroradiologic approach to paragangliomas of the head and neck proceeds in sequential steps. High-resolution computed tomography (CT) is used to define the extent and topography of the tumor [2–6] and is usually followed by selective angiography, which provides essential information concerning the vascular supply and the angioarchitecture of the tumor [7–9]. Finally, embolization may be performed for preoperative devascularization of large, highly vascular paragangliomas [8].

Recent advances in interventional neuroradiology, including the availability of

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different embolic materials [10] and catheter systems [11] as well as a better understanding of the functional vascular anatomy [12] and hemodynamic conditions [13], permit safe and effective embolization of paragangliomas [8]. Because embolization has been shown to significantly improve the conditions under which surgery is performed, it is now an established and accepted modality for preoperative devascularization of paragangliomas in any location in the head and neck [8, 14].

Among all types of paragangliomas of the head and neck, those arising in the temporal bone represent the most challenging lesions to both the neuroradiologist and the otoneurosurgeon. It is our purpose to present our experience with preoperative embolization of 44 paragangliomas of the head and neck, with special emphasis on indications, techniques, dangers, precautions, and material selection for embolization of paragangliomas of the temporal bone.

Subjects and Methods

Thirty-nine patients with a total of 44 paragangliomas of the head and neck underwent preoperative embolization. Four patients had multiple tumors. Of them, one patient had bilateral paragangliomas of the temporal bone and a carotid body paraganglioma, two patients had ipsilateral paragangliomas of the temporal bone and carotid body, and one patient had ipsilateral paragangliomas of the temporal bone and the carotid and vagal bodies. Thirty-five of the paragangliomas were located in the temporal bone, five in the carotid body, three in the vagal body, and one in the infratemporal fossa. The 39 patients were 28–71 years old (mean, 41 years). There were 25 women and 14 men. All patients were initially examined with contrast-enhanced multiplanar high-resolution CT, according to a technique described previously [3, 15]. Selective angiography and embolization was performed in the same session and under general anesthesia in all patients. Selective angiography and embolization in the last eight patients in this series were exclusively performed with the digital subtraction technique.

Most of the angiographic studies and embolizations were performed with a simply curved, 4 French polyethylene catheter. Catheterization (two cases) and embolization (one case) of internal carotid artery (ICA) branches was performed with a calibrated-leak microballoon catheter system [11]. Temporary (28 cases) or permanent (three cases) balloon occlusion of the ICA was performed with Debrun's detachable balloon catheter system [10]. Depending on the vascular composition of the tumors and on the type of the feeding arteries, one or more of the following embolic materials were used for embolization of each paraganglioma: particles of Gelfoam, powder of Gelfoam, microparticles of polyvinyl alcohol foam (Ivalon), particles of lyophilized human dura mater (Lyo Dura), and isobutyl-2-cyanoacrylate (Bucrylate). The mode of preparation and instructions for the use of these materials have been described [10].

Paragangliomas of the Temporal Bone

Experience with preoperative embolization of paragangliomas of the temporal bone obtained in different centers during the past decade indicates that this method considerably improves surgical conditions [8, 16–25]. The surgical approach to paragangliomas of the temporal bone depends primarily on tumor size. A major advance in the management of large paragangliomas was the introduction of the infratemporal fossa approach by Fisch [26] in 1977. With the exception of lesions exhibiting significant intradural extension, this approach allows radical removal of most paragangliomas with minimal morbidity and almost no mortality [14, 27]. For accurate preoperative assessment of tumor size and extension, a classification system was introduced [14, 28]. In our consecutive series of 82 surgically removed tumors, of whom 48 were examined by high-resolution CT and 35 underwent preoperative embolization, this classification proved highly useful, since it helped to select appropriate cases for embolization, predicted the extent of embolization, and guided selection of the appropriate surgical approach [3, 14, 27].

Classification

On the basis of high-resolution CT findings, paragangliomas of the temporal bone are classified into one of four main types (A–D) and several subtypes (C_1–C_4, De_{1-3}, and Di_{1-3}). *Type A* paragangliomas are confined to the tympanic cavity and represent glomus tympanicum tumors. *Type B* paragangliomas originate in the hypotympanic bone plate and may extend into the hypotympanum or mastoid bone.

Type C paragangliomas represent glomus jugulare tumors. Depending on the degree of involvement of the carotid canal, type C tumors are further subdivided into types C_1–C_4. *Type* C_1 lesions cause only minimal erosion of the vertical segment of the carotid canal. *Type* C_2 lesions cause complete erosion of the vertical segment of the carotid canal. *Type* C_3 lesions cause additional erosion of the horizontal segment of the carotid canal. *Type* C_4 lesions cause involvement of the entire carotid canal including the foramen lacerum.

Type D paragangliomas represent glomus jugulare tumors with intracranial extension. Depending on the extra- or intradural position of the intracranial component, type D lesions are defined as type De or Di. Depending on the size of the extra- or intradural portions, tumor types De and Di are further subdivided into subtypes De_{1-3} and Di_{1-3}.

Indications for Embolization

Tumor types A and B are small and can be removed easily by conventional tympanoplastic techniques without prior embolization [27].

Tumor types C_1–C_4 and De_{1-3} require the infratemporal fossa approach for radical removal. For these tumor types, embolization is an essential prerequisite for successful surgery [14].

Tumor types Di_{1-3} require a combined two-stage otologic (i.e., infratemporal fossa) and neurosurgical (i.e., lateral suboccipital) approach. Although embolization usually does not act upon the intradural portion of the tumor, it significantly improves surgical conditions of type Di lesions by devascularizing the main intratemporal and extradural bulk of tumor [14].

Vascular Composition and Embolization

Selection of the appropriate embolic material and technique of embolization primarily depend on the vascular composition

of the tumor, as determined by selective angiographic investigation. Moret et al. [29, 30] were the first to recognize that paragangliomas may be composed either of multiple compartments or of a single vascular compartment. In our consecutive series of 35 embolized types C and D paragangliomas, 83% were found to be multicompartmental and 17% monocompartmental tumors.

Each vascular compartment of a multicompartmental paraganglioma is hemodynamically independent. Therefore, injection of one feeding artery in a multicompartmental tumor invariably results in opacification of only one tumor compartment (Fig. 1). Angiographic demonstration of the entire extent of a multicompartmental paraganglioma therefore requires injection of all possible feeding arteries (Fig. 1). On the other hand, injection of each of the feeding arteries in a monocompartmental tumor invariably results in opacification of the entire lesion (Fig. 2). Correlation of the angiographic findings in types C and D paragangliomas with the known vascular

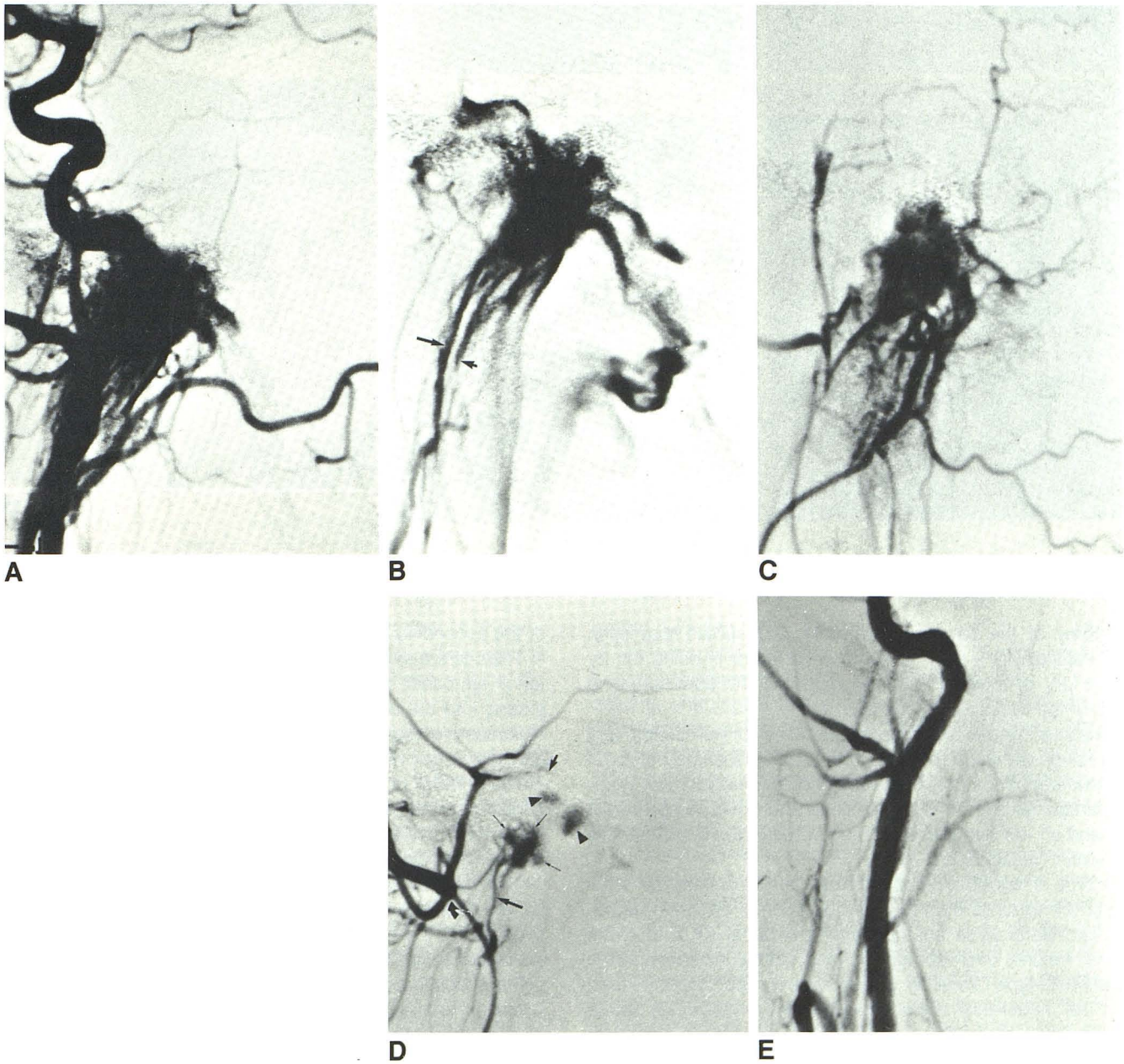


Fig. 1.—Type C_3 multicompartmental paraganglioma. **A**, Common carotid artery injection. Opacification of entire lesion. **B**, Injection of ascending pharyngeal artery. Opacification of inferomedial compartment via dilated inferior tympanic artery (*long arrow*) and neuromeningeal trunk (*short arrow*). **C**, Injection of stylomastoid artery. Opacification of posterolateral compartment. **D**, Injection of internal maxillary artery at level of branching of middle meningeal artery (*curved arrow*). Opacification of anterior compartment (*thin arrows*) via anterior tympanic artery (*long thick arrow*) and of superior compartment (*arrowheads*) via superior tympanic artery (*short thick arrow*). **E**, Injection of common carotid artery after embolization of each compartment. Complete devascularization of lesion with preservation of ECA branches.

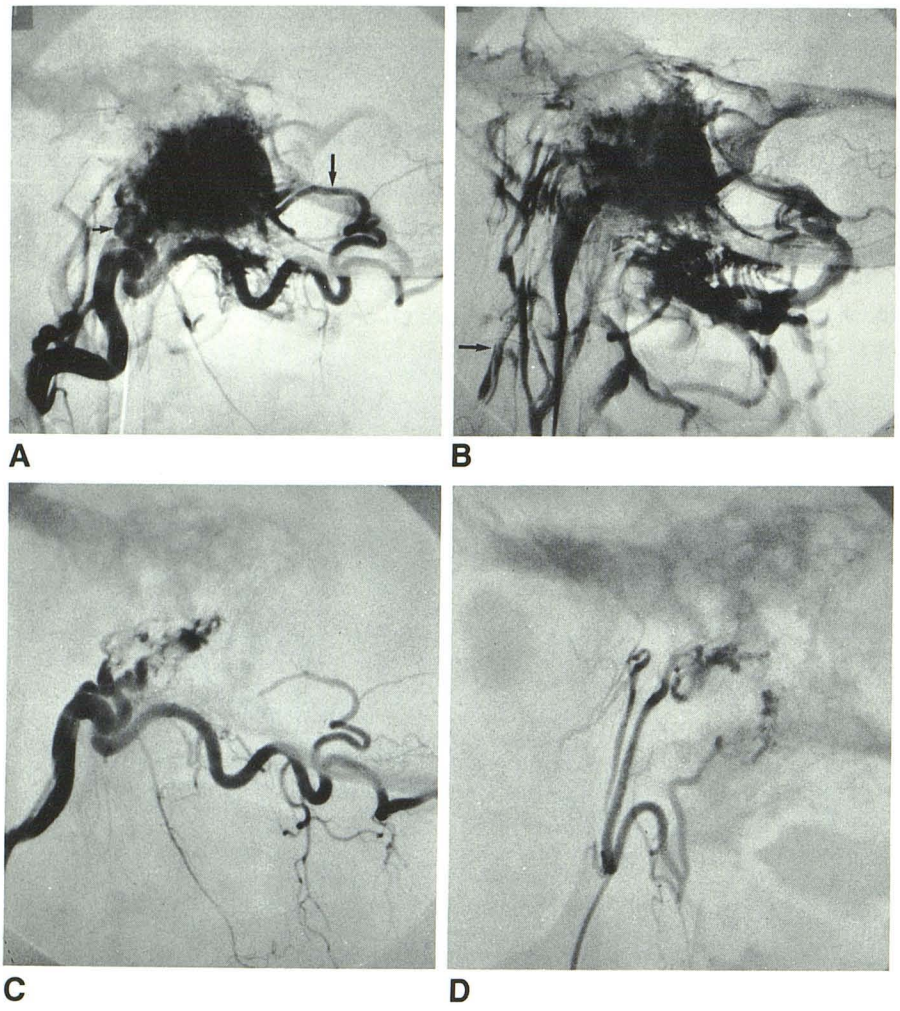


Fig. 2.—Type C_3 monocompartmental paraganglioma. **A**, Injection of occipital artery. Opacification of tumor via stylomastoid (*short arrow*) and mastoid (*long arrow*) arteries. **B**, Injection of ascending pharyngeal artery (*arrow*). Identical tumor opacification as with injection of occipital artery. **C**, Injection of occipital artery after embolization of tumor with Bucrylate through stylomastoid artery. No opacification of tumor bed. **D**, Injection of nonembolized ascending pharyngeal artery. No opacification of tumor bed confirms effective embolization of tumor. In both **C** and **D**, pooling is seen only in distal arterial segments.

anatomy of the temporal bone [31, 32] shows that each compartment in a multicompartmental tumor is being fed by the artery or arteries normally supplying the corresponding area of the temporal bone [30].

Analysis of the angiographic findings in our series and correlation with high-resolution CT findings showed that both the number of compartments composing a multicompartmental paraganglioma and the size of each individual compartment depend on the size and extension of the lesion and therefore on tumor classification.

A type C and D multicompartmental tumor may be composed of no more than four compartments: an inferomedial, a posterolateral, an anterior, and a superior [30]. In types C_1–C_3 lesions, each compartment is strictly intratemporal in location. In type D lesions, one or more compartments extend into the intracranial extradural (De) or intradural (Di) space and thereby annex the supply of the corresponding dural or parenchymal vascular systems.

The *inferomedial compartment* (Fig. 1B) occupies the jugular bulb area and hypotympanum. With further growth it also occupies the infralabyrinthine space. It is the first compartment to develop in the evolution of a paraganglioma. It is supplied by the inferior tympanic artery and the jugular branch of the ascending pharyngeal artery [29, 30].

The *posterolateral compartment* (Fig. 1C) occupies the posterior tympanic cavity and the mastoid bone. It may also extend into the external auditory canal. It is supplied by the stylomastoid artery, a branch of the occipital or posterior auricular artery [29, 30].

The *anterior compartment* (Fig. 1D) occupies the protympanum and the pericarotid area. It is supplied by the anterior tympanic branch of the internal maxillary artery and/or by the caroticotympanic branch of the ICA [30, 33].

The *superior compartment* (Fig. 1D) occupies the epitympanum and may extend into the supralabyrinthine space. It is supplied by the superior tympanic artery, which arises from the petrous branch of the middle meningeal artery [29, 30].

For effective devascularization of multicompartmental type C lesions, each compartment should be individually embolized through its feeding artery or arteries (Fig. 1E). If there is no anterior compartment present or if an anterior compartment is present but not supplied by the caroticotympanic artery, complete devascularization of the tumor will be achieved (Fig. 1E). If the anterior compartment is supplied by both the anterior tympanic and caroticotympanic arteries, fluid material embolization through the anterior tympanic artery may result

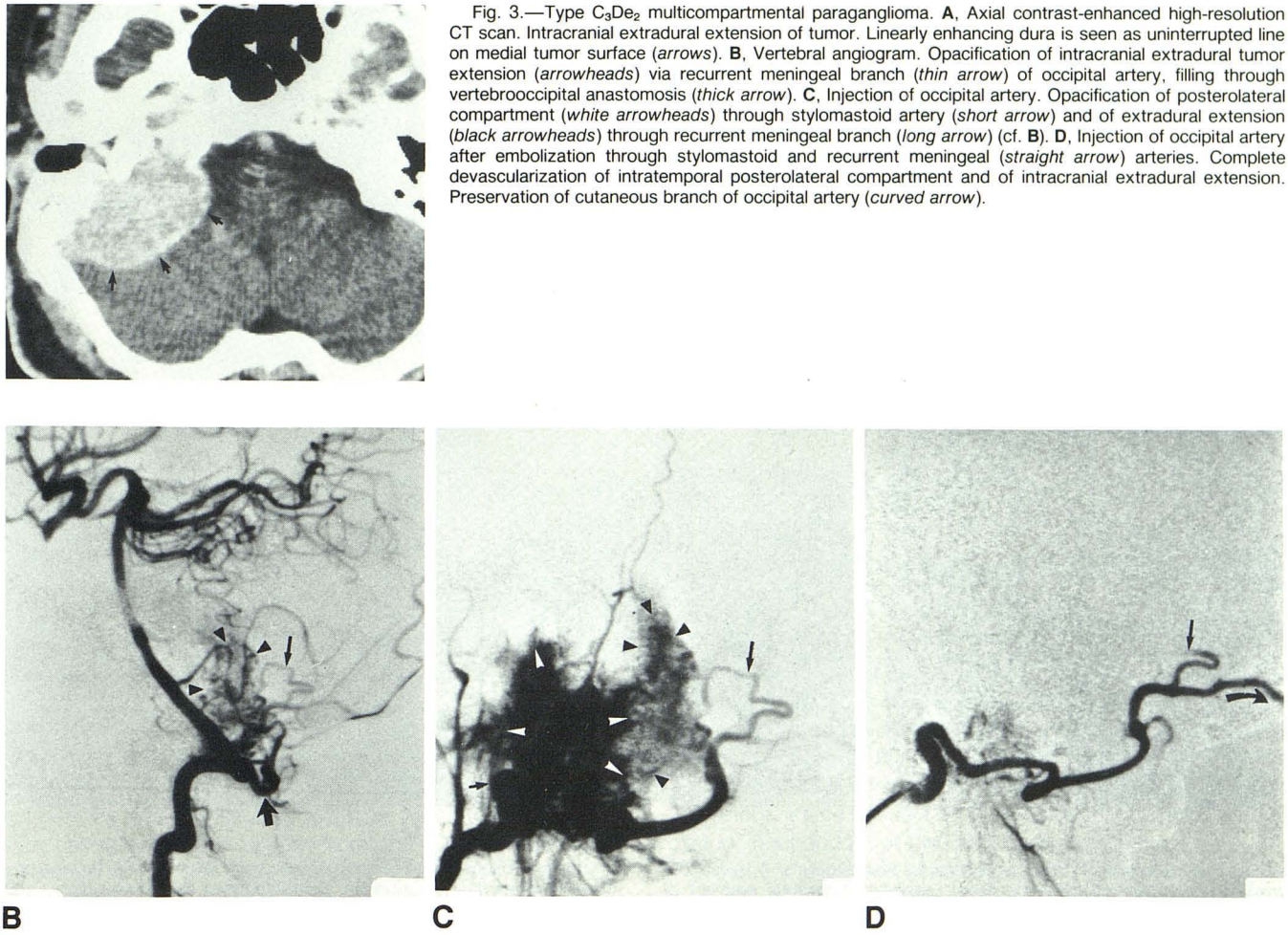


Fig. 3.—Type C_3De_2 multicompartmental paraganglioma. **A**, Axial contrast-enhanced high-resolution CT scan. Intracranial extradural extension of tumor. Linearly enhancing dura is seen as uninterrupted line on medial tumor surface (*arrows*). **B**, Vertebral angiogram. Opacification of intracranial extradural tumor extension (*arrowheads*) via recurrent meningeal branch (*thin arrow*) of occipital artery, filling through vertebrooccipital anastomosis (*thick arrow*). **C**, Injection of occipital artery. Opacification of posterolateral compartment (*white arrowheads*) through stylomastoid artery (*short arrow*) and of extradural extension (*black arrowheads*) through recurrent meningeal branch (*long arrow*) (cf. **B**). **D**, Injection of occipital artery after embolization through stylomastoid and recurrent meningeal (*straight arrow*) arteries. Complete devascularization of intratemporal posterolateral compartment and of intracranial extradural extension. Preservation of cutaneous branch of occipital artery (*curved arrow*).

in significant diminution of the ICA contribution. If there is significant supply from the caroticotympanic artery, special interventional techniques may be used. These will be discussed under management of the ICA.

In monocompartmental tumors, effective embolization of the entire lesion can be performed through one of the feeding arteries, provided that a fluid material is used. In these cases, postembolization angiography of the nonembolized arteries shows no opacification of the lesion and thus confirms successful devascularization of the tumor (Fig. 2). If this technique of "single-vessel" embolization of a monocompartmental paraganglioma is not possible, either because Bucrylate has to be injected through a dangerous vessel or the selected artery for embolization cannot be catheterized distally, embolization with microparticles through all identified feeders is a nearly equally effective alternative technique.

Type D paragangliomas are further divided into extradural types De and intradural types Di, based on contrast-enhanced high-resolution CT. In type De lesions, the enhancing dura is seen as an uninterrupted line on the medial surface of the tumor (Fig. 3A). In type Di lesions, contrast-enhanced high-resolution CT regularly shows focal interruption of the linearly enhancing dura and nodular tumor extension through interrupted dura (Fig. 4A).

The extradural extension of type De and Di paragangliomas is supplied centrally by the meningeal branches of the ascending pharyngeal artery and posteriorly by the recurrent meningeal (or mastoid) branch of the occipital artery [30] (Fig. 3). The central extradural portion represents the intracranial extension of the inferomedial compartment and the posterior extradural portion represents the intracranial extension of the posterolateral compartment.

If the extradural extension is large, the anterior intracranial portion of the tumor is supplied by the clival and even the cavernous branches of the ICA. The superior extradural extension is supplied by the petrous branch of the middle meningeal artery [30] (Fig. 4C). The intradural portion of type Di paragangliomas is constantly supplied by parenchymal branches of the vertebrobasilar system. Intradural extensions at the level of the jugular foramen receive their blood supply from the posterior inferior cerebellar artery (PICA) and intradural extensions into the cerebellopontine angle receive their blood supply from the anterior inferior cerebellar artery (AICA) [30] (Fig. 4B).

In our experience, extradural type De_1 and De_2 tumors and the extradural component of type Di_1 lesions usually do not

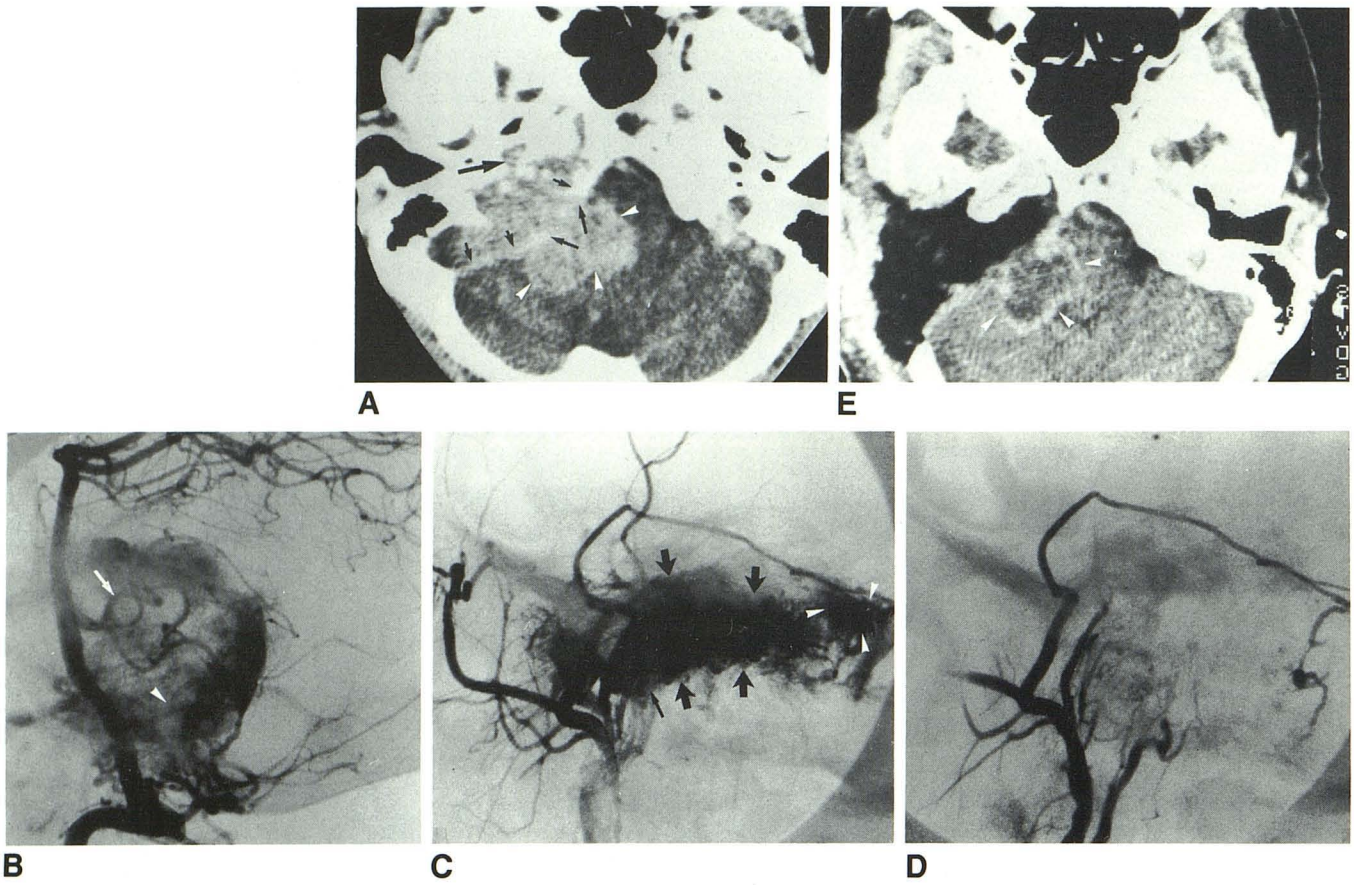


Fig. 4.—Type $C_3De_2Di_2$ multicompartmental paraganglioma. **A**, Axial contrast-enhanced high-resolution CT scan. Erosion of horizontal segment of carotid canal (*large arrow*), indicating type C_3; moderate extradural extension (*short arrows*), indicating type De_2 and moderate intradural extension (*white arrowheads*) through interrupted dura (*two long arrows*), indicating type Di_2. **B**, Vertebral angiogram. Opacification of intradural extensions via AICA (*arrow*) and PICA (*arrowhead*). **C**, Injection of internal maxillary artery. Opacification of anterior compartment (*thin arrow*), of superior compartment (*thick arrows*), and of intradural extension into cerebellopontine angle (*arrowheads*). **D**, Injection of maxillary artery after embolization. Complete tumor devascularization. Opacification just above bend of distal ECA represents staining of temporal muscle, a phenomenon observed frequently when, after repeated injections, contrast material accumulates within slow-flow vascular bed of that muscle. **E**, Axial contrast-enhanced high-resolution CT scan after embolization and surgical removal of intratemporal and extradural parts. Necrosis and size reduction of remaining intradural extension (*arrowheads*) (cf. **A**).

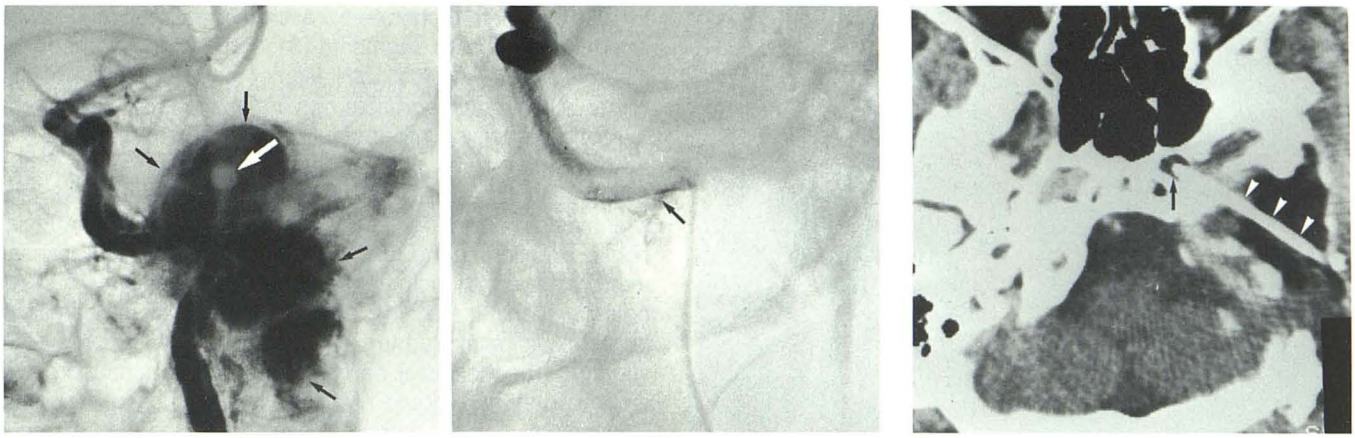
receive additional supply from the clival and cavernous branches of the ICA and, therefore, can be devascularized completely (Fig. 3). Tumor types De_3, Di_2, and Di_3 usually receive additional supply from the clival and cavernous branches of the ICA. If this supply is significant, special interventional techniques may be used.

The intradural component of type Di paragangliomas cannot be reached by embolization. However, embolization of the intratemporal and extradural intracranial portions of the tumor significantly improved surgical conditions in the type Di lesions in our series. In an exceptional case of a type Di_2 paraganglioma, the intradural extension exhibited angiographically a monocompartmental vascular composition, being simultaneously supplied by the AICA and the petrous branch of the middle meningeal artery (Figs. 4B and 4C). In this case, embolization with microparticles of Ivalon through the middle meningeal artery resulted in necrosis and size diminution of the intradural tumor portion (Fig. 4).

Management of the ICA

One of the main advantages of the infratemporal fossa surgical approach is that it provides access to the whole length of the intratemporal ICA [27]. Extensive paragangliomas of types C_2, C_3, De, and Di destroy the carotid canal, may infiltrate the adventitia of the ICA, and may take significant supply from the intratemporal and extradural ICA branches [34]. In these cases, mobilization of the anterior pericarotid part of the tumor pole from the petrous ICA represents one of the most difficult and critical steps during surgery [14, 34].

In selected cases of larger paragangliomas with significant supply from the caroticotympanic artery, embolization of the pericarotid tumor portion through this vessel may be performed. Essential prerequisites for this technique are superselective catheterization of the caroticotympanic artery with no evidence of reflux of contrast material into the ICA on



A B

Fig. 5.—Embolization of pericarotid tumor portion. **A**, ICA angiogram after placement of calibrated-leak microballoon (*white arrow*) through caroticotympanic artery in pericarotid tumor portion. Extensive supply (*black arrows*) from caroticotympanic artery. **B**, Selective angiogram of caroticotympanic artery (*arrow*) after embolization. Complete devascularization of tumor and reflux of contrast material into ICA.

Fig. 6.—Intraoperative balloon occlusion of petrous ICA. Axial contrast-enhanced high-resolution CT scan after radical removal of type C_3De_3 paraganglioma. Introducer catheter (*arrowheads*) in operative field with tip in distal horizontal segment of ICA (*arrow*). Through this catheter, detachable balloon was inserted in ICA during operation.

preembolization angiography. Manelfe et al. [35] were the first to report on catheterization and embolization of the caroticotympanic artery in a case of large paraganglioma of the temporal bone. In our series this technique was successfully used in one case of type C_3De_2 paraganglioma being supplied by a dilated caroticotympanic artery (Fig. 5).

In cases with both supply from the ICA *and* extensive infiltration of the ICA wall, permanent occlusion of the ICA with a detachable balloon may be performed [36]. Prerequisite for this technique is patient tolerance of temporary balloon occlusion of the ICA [36].

An alternative to these techniques is temporary or permanent intraoperative balloon occlusion of the ICA after puncture of the vessel through the operative field and insertion of a detachable balloon in the petrous ICA (Fig. 6). Prerequisite for this technique is patient tolerance for temporary balloon occlusion, tested preoperatively during angiography and embolization. The advantage of this technique is that the ICA is not being sacrificed a priori, but rather this decision is made during surgery and according to the problems encountered during mobilization of the pericarotid tumor portion.

Dangers, Complications, and Precautions

Minor and frequent complications of embolization of paragangliomas include pain in the ear and postembolization fever. Both are of short duration and are attributed to tumor ischemia [8]. In our series, pain and fever were present in about 80% of cases (28 of 35). In our experience, pain and fever together with cessation of pulsatile tinnitus are reliable clinical indicators of successful embolization of paragangliomas.

Another minor complication of embolization of paragangliomas is postoperative wound healing problems. This complication is likely to occur if the cutaneous branches of arteries supplying the tumor are occluded during embolization. The large retroauricular and temporal skin incision used in the infratemporal fossa approach for paragangliomas is located in the vascular territory of the cutaneous branch of the occipital, posterior auricular, and superficial temporal arteries [14]. Therefore, these vessels must be preserved during embolization, independent of the embolic material used.

Major complications of embolization of paragangliomas theoretically include stroke and palsies of the seventh and lower cranial nerves. Stroke is likely to occur if embolic material injected into the external carotid artery (ECA) branches supplying the tumor reaches the vertebrobasilar or ICA circulation through arterial anastomoses. The most important anastomoses that may be present in embolization of paragangliomas are those connecting the ascending pharyngeal and/or the occipital artery with the vertebral artery [12, 13, 37]. The pharyngovertebral anastomoses are (1) the so-called odontoid arterial arch system connecting the hypoglossal branch of the ascending pharyngeal artery with the vertebral artery at the C2–C3 level and (2) the musculospinal artery connecting the main stem of the ascending pharyngeal artery with the vertebral artery at the C3 level [12, 38]. The occipitovertebral anastomoses are the C1 and C2 collaterals connecting the occipital artery with the vertebral artery at the C1 and C2 levels, respectively [12, 39]. The presence of such an anastomosis represents one of the most dangerous situations in the embolization of paragangliomas. However, it does not represent a contraindication to embolization, provided that specific precautions are applied: (1) the use of particles larger in size than the anastomotic artery, (2) temporary occlusion of the anastomotic artery with a large, reabsorbable particle of Gelfoam, and (3) temporary occlusion of the vertebral artery at the entry site of the anastomotic artery with a balloon (Fig. 7). In our series, these types of arterial anastomoses were

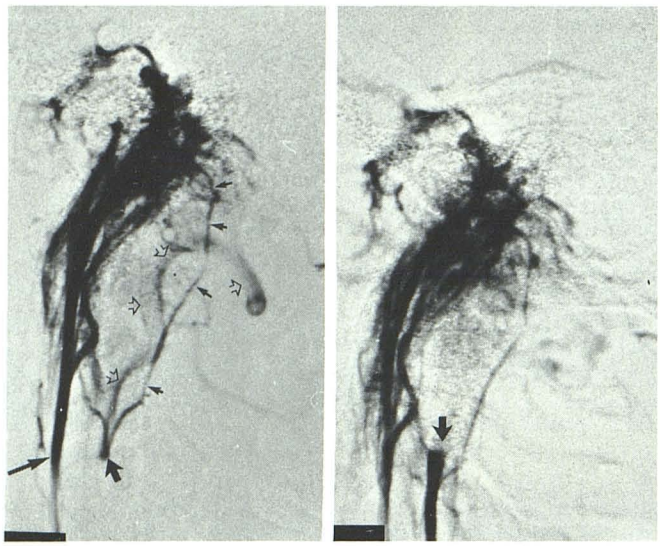


Fig. 7.—Temporary balloon occlusion of vertebral artery in patient with pharyngovertebral anastomosis. **A**, Injection of ascending pharyngeal artery (*large arrow*). Opacification of inferomedial compartment and filling of vertebral artery (*open arrows*) through anastomosis (*thick arrow*) of odontoid arch system (*small arrows*). **B**, Simultaneous injections of ascending pharyngeal artery and vertebral artery after inflation of balloon in vertebral artery at level of entry of anastomotic artery (*arrow*). Balloon protects vertebrobasilar system.

present in 12 (34%) of the 35 cases. With the use of the above-mentioned precautionary techniques, influx of embolic material into the vertebrobasilar system was prevented in all cases.

Cranial nerve palsies are likely to occur if embolic material occludes the distal portion of ECA branches known to supply cranial nerves [40, 42]. The dangerous vessels in embolization of paragangliomas are the neuromeningeal branch of the ascending pharyngeal artery, supplying cranial nerves IX–XII [42], as well as the stylomastoid and middle meningeal arteries, both supplying the intratemporal portion of the seventh nerve [43, 45]. A permanent palsy is likely to occur if nonabsorbable particulate or fluid materials are injected in one of these dangerous vessels. On the other hand, a transient palsy may occur if absorbable particles, for example, Gelfoam, are used for embolization through these dangerous vessels. In patients with clinically evident palsy of one or more of the caudal cranial nerves, the use of nonabsorbable materials is justified. However, in patients without clinically evident palsy, a resorptive material should be used. In this series, microparticles of Gelfoam or Gelfoam powder were used for embolization through the ascending pharyngeal artery in patients without palsy of the lower cranial nerves. With this technique one transient ninth nerve palsy occurred lasting for 5 days. There were no permanent palsies.

In 75% of cases, the intratemporal portion of the facial nerve is supplied nearly equally by two vessels: the tympanic segment is supplied by the petrous branch of the middle meningeal artery and the mastoid segment is supplied by the stylomastoid artery [12, 31, 43, 44]. In 10% of cases the stylomastoid artery supplies both the mastoid and tympanic segments [31, 43]. In these cases the petrous branch is short. In 15% of cases the petrous branch of the middle meningeal artery supplies both segments [31, 43]. When a long stylomastoid artery supplies both segments, embolization of the artery with a nonabsorbable material may cause permanent facial nerve palsy. Unfortunately, tumor blush obscures the stylomastoid artery, and, therefore, its supply to the facial nerve cannot be seen directly. To determine whether the stylomastoid artery does supply both segments of the facial nerve, injection of the middle meningeal artery and assessment of the length of its petrous branch is necessary. A short petrous branch, reaching only to the geniculate ganglion, indicates indirectly that the stylomastoid artery supplies both segments of the facial nerve. Therefore, a nonabsorbable embolic material should not be used in such a situation. Conversely, a long petrous branch reaching the mastoid segment of the facial canal indicates that the stylomastoid artery is short and supplies only the mastoid segment of the facial nerve. Therefore, a nonabsorbable material may be used safely in such a situation. In this series, two transient facial nerve palsies occurred after embolization of the posterolateral compartment of two paragangliomas through the stylomastoid artery with Gelfoam particles.

Other rare complications reported to occur after embolization of paragangliomas include aspiration pneumonia following occlusion of the ascending pharyngeal artery with ferrosilicone [46] and uncontrollable fluctuations of blood pressure following embolization of a secretorily active paraganglioma [47]. No such complication occurred in this series. In two patients with secretorily active paragangliomas causing severe hypertensive crises, embolization was followed by normalization and stabilization of blood pressure.

Selection of Embolic Materials

Selection of the appropriate embolic material for embolization of paragangliomas depends mainly on two factors: safety and effectiveness. Maximally safe is embolization with reabsorbable microparticles or Gelfoam powder. However, this technique is not always effective. For the embolization to be effective, the vascular composition of the tumor must be considered.

In monocompartmental tumors, effective embolization may be performed with a fluid material, such as bucrylate or silicone, injected through a safe artery that can be catheterized distally. An artery is considered safe if it is not involved in the supply of a cranial nerve and it does not anastomose with the vertebral artery or ICA. An artery is also considered safe if it supplies a cranial nerve in a patient with palsy of this nerve or if it anastomoses with the vertebral artery or ICA and the anastomosis can be temporarily blocked.

In multicompartmental tumors, different materials may be used for different compartments. Compartments supplied by safe arteries may be embolized with fluid materials (e.g., Bucrylate, silicone) or nonabsorbable microparticles (e.g., Ivalon). Compartments supplied by dangerous arteries should be embolized with resorptive microparticles (e.g., Gelfoam). An artery is considered dangerous if it supplies a cranial nerve in a patient without palsy of this nerve or if it anastomoses with the vertebral or ICA and this anastomosis cannot be temporarily blocked.

Other Paragangliomas of the Head and Neck

Paragangliomas of the carotid body appear on high-resolution CT as homogeneously and intensively enhancing, round to oval, sharply demarcated masses in the carotid space at hyoid level [6]. Since the carotid body is located on the inner surface of the carotid bifurcation, carotid body paragangliomas characteristically displace the bifurcation laterally [6]. In addition, these tumors grow between the main stem of the ECA and the proximal segment of the ICA and, therefore, tend to displace the former anteriorly and the latter posteriorly. Angiographically, carotid body paragangliomas may be composed of multiple compartments or of a single vascular compartment [8, 13]. In our experience, carotid body paragangliomas are more frequently multicompartmental tumors. The most frequently involved vessels in the supply of these tumors are the musculospinal branch of the ascending pharyngeal artery and the ascending cervical artery. The so-called artery of the carotid body is also supposed to supply these tumors, but this small vessel is rarely identified angiographically [13]. Depending on their growth direction, larger tumors may additionally derive their supply from the facial, lingual, thyroid, posterior auricular, occipital, deep cervical, and C4–ECA collateral arteries [13]. Preoperative embolization of carotid body paragangliomas follows the same principles and techniques described above for paragangliomas of the temporal bone. Multicompartmental tumors should be embolized through all identified feeding arteries. Monocompartmental tumors may be embolized with a fluid material through a safe feeding artery. In our experience, the musculospinal branch of the ascending pharyngeal artery is a safe and effective route for complete devascularization of monocompartmental paragangliomas of the carotid body (Fig. 8).

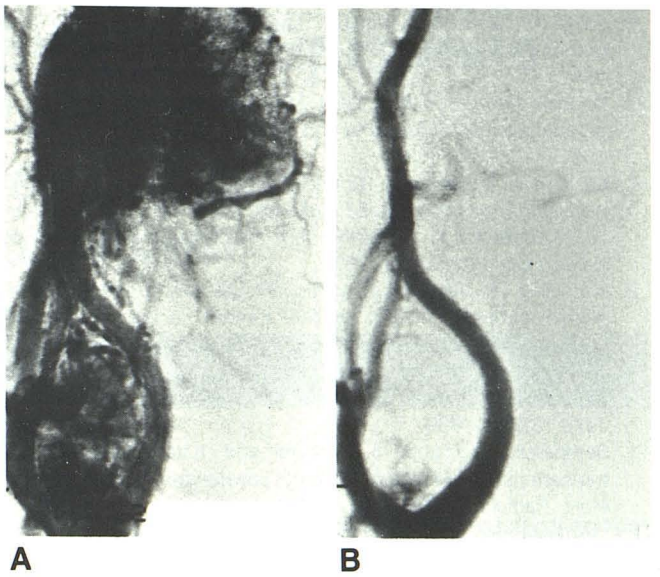


Fig. 8.—Monocompartmental carotid body paraganglioma. **A**, Injection of common carotid artery. Medium-sized carotid body and large temporal bone paraganglioma. **B**, Injection of common carotid artery embolization of monocompartmental carotid body tumor through musculospinal branch and multicompartmental temporal bone tumor through all identified feeding arteries. Complete devascularization of both tumors.

Paragangliomas of the vagal body are located in the upper cervical area below the exocranial aspect of the posterior skull base. Large lesions may exceptionally extend through the jugular foramen into the temporal bone [7]. Vagal body tumors characteristically displace both the ICA and ECA anteriorly and medially [7]. These tumors are located in the vascular territory of the pharyngooccipital system and derive their supply primarily from the musculospinal branch of the ascending pharyngeal artery as well as from proximal muscular branches of the occipital artery [13]. Like paragangliomas of other locations, vagal body tumors are more frequently composed of multiple compartments than of a single compartment. In this series, two of the three lesions had a multicompartmental and one a monocompartmental composition. The anterior extension of large vagal body tumors receives additional supply from the linguofacial system, the posterior extension supply from the deep cervical artery, the superior extension from the occipital artery, and the inferior extension from the ascending cervical artery [13]. Techniques and embolic materials for embolization of vagal body tumors are similar to those used for temporal bone paragangliomas and depend mainly on the vascular composition of the lesion as determined by selective angiographic exploration. Since the ICA usually does not supply vagal body paragangliomas, complete devascularization usually can be achieved. The main dangers of preoperative embolization of paragangliomas of the vagal body involve passage of emboli from the occipital and ascending pharyngeal artery into the vertebrobasilar system through pharyngo- and occipitovertebral anastomoses as well as distal occlusion of the neuromeningeal trunk of the ascending pharyngeal artery with the risk of palsy of cranial nerves IX–XII [13]. These complications can be avoided using the techniques described above for temporal bone paragangliomas. In these series no complication occurred with preoperative embolization in eight cases of carotid and vagal body paragangliomas.

Paragangliomas of other areas of the head and neck are extremely rare. Possible locations are the larynx, the upper airways, the orbit, and the parapharyngeal space [1, 8]. Techniques of preoperative embolization of such rare lesions depend on the specific vascular territory involved and on the vascular composition of the tumor.

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