AJNR

Radiographic management of juvenile angiofibromas.

R N Bryan, R B Sessions and B L Horowitz

This information is current as of April 19, 2024.

Radiographic Management of Juvenile Angiofibromas

R. Nick Bryan[1]
Roy B. Sessions[2]
Barry L. Horowitz[1]

Juvenile angiofibromas are benign, vascular, locally aggressive neoplasms that are preferably treated by surgical resection, or irradiation if surgery is not possible. Adequate surgery in the past has been limited by incomplete knowledge of the anatomy of the tumor and technical difficulties related to the vascularity. To better define the tumor, 12 patients with juvenile angiofibroma have been studied by axial and coronal high resolution computed tomography (CT). The extent of the neoplasm was better demonstrated by CT than by other techniques. Based on the CT findings, we propose an anatomic classification that is helpful in determining treatment methods. Nine patients were considered operable, and eight of these underwent preoperative embolization with Silastic spheres and Gelfoam. The preoperative embolization significantly reduced operative difficulty and the necessity for blood transfusions. Based on these cases, we believe the current radiographic management of juvenile angiofibromas should consist of plain films, CT, angiography and, in surgical cases, preoperative embolization.

Juvenile angiofibromas are intriguing lesions that are highly vascular, noninfiltrating, benign neoplasms, occurring in the nasopharyngeal region of pubescent males [1–3]. Continuing interest in this lesion is enhanced by its unusual patient population, presumed hormonal influences [4], and clinical challenge. Despite the benign histology and limited growth potential, these tumors have proven recalcitrant to any single therapy method. This is primarily due to their anatomically complex and critical location, extreme vascularity, and the problems of high dose radiation for benign lesions in young patients.

Improved diagnostic definition of angiofibroma by pluridirectional tomography [5], angiography [6], and most recently computed tomography (CT) [7–9] has greatly improved the understanding of the gross anatomy of these lesions and offered some insight into the dynamic growth patterns of the tumors. This knowledge, combined with preoperative embolization, has greatly improved therapy [10, 11]. However, there is still no integrated system for using all of this information plus prior pathologic and surgical knowledge to logically classify each lesion and plan treatment. Based on our experience with 12 patients with angiofibromas over a 2½ year period, we propose a classification based on anatomic location as determined by CT. Appropriate treatment regimens based on this classification system are suggested.

Materials and Methods

We evaluated 12 patients with juvenile angiofibromas either surgically proven (nine) or diagnosed on typical clinical presentation, appropriate angioarchitecture, and appropriate response to radiation therapy (three). All patients had plain radiography, angiography including selective internal and external carotid injections, and CT using a third or fourth generation scanner. Scans were performed in the axial and coronal projections before and after intravenous contrast injection. The patients, 10–17-year-old boys, all had diagnostic

TABLE 1: **Juvenile Angiofibromas: Case Summaries**

Case No.	Symptoms and Signs	Location*	Blood Supply	Surgery			Follow-up	Classification†
				Approach	OR Time	EBL (ml)		
1	Nasal obstruction, epistaxis, L posterior nasal mass	L choanal NP	L maxillary III (E), PBFA	Tr pal, tr ant (unnecessary)	5'25"	350	NET 2 yr, neg angio	Ipsi IA
2	Nasal obstruction, epistaxis, small L choanal mass	L choanal, sphenoid sinus	L maxillary III (E)	Tr pal	3'15"	100	NET 1 yr, neg CT	Ipsi IB
3	Obstruction, epistaxis, rhinorrhea, large R nasal and NP mass crossing midline	R choanal NP, bowing septum across midline	R maxillary III (E), PBFA (E)	Tr pal	3'15"	200	NET 1 yr, neg CT	Ipsi IA
4	Obstruction, epistaxis, rhinolalia, R posterior nasal and NP mass	R choanal NP and PPF	R maxillary II and III (E), ascending pharyngeal (E)	Tr pal	2'30"	600	NET 10 mo	Ipsi IIB, 1A
5	Nasal obstruction, epistaxis, large R nasal mass obliterating NP	R choanal, R and L NP, R maxillary and ethmoid sinus, PPF and ITF	R maxillary II and III (E), R ascending pharyngeal (E), L maxillary III (E)	Tr pal, Tr nas, Tr ant	4'40"	1,100	Residual tumor 4 mo, reop, NET 6 mo	Bilateral IIC, IB
6	Nasal obstruction epistaxis, proptosis, R nasal and NP mass with septal deviation	R choanal NP, PPF, maxillary, ethmoid and sphenoid sinus, and orbit	R maxillary II and III (E), L maxillary III	Tr pal, Tr eth, Tr ant, sublabial	7'25"	1,700	NET 3 yr, neg angio	Bilateral IIB, IB
7	Nasal obstruction, rhinorrhea, rhinolalia, anosmia, R NP mass	R NP, PPF, sphenoid sinus, cavernous sinus, medial wall middle fossa	R maxillary III, R ascending pharyngeal, R internal carotid	...	...	...	Asymptomatic 2 yr, pos CT	Ipsi III, IIB, IB
8	Nasal obstruction, epistaxis	R NP, PPF, infraorbital fissure, posterior orbit, medial wall middle fossa	R maxillary III	...	...	...	1 mo	Ipsi III, IIB, IB
9	HA, nasal obstruction, facial swelling, L proptosis, L NP and maxillary mass	L NP, PPF-PMF, infratemporal fossa, maxillary, ethmoid, and sphenoid sinus, floor anterior fossa, medial wall middle fossa, cavernous sinus	L maxillary I, II, III (E), L ascending pharyngeal (E), L internal carotid, R maxillary III (E), R ascending pharyngeal (E)	Tr pal, Tr nas, Tr ant, Sublabial	11'55"	3,500	Partial resection followed by XRT; asymptomatic 1 yr, pos CT	Bilateral III, IIC, IB
10	1973: Epistaxis, neg PE	R NP, PPF-PMF, sphenoid and cavernous sinus (pre-CT)	R maxillary III, R internal carotid	Tr eth, Tr ant	...	...	Postop residual	?Ipsi III, IIC, IB
	6/79: Asymptomatic, neg PE	PPF	...		...	...	Asymptomatic	IIB
	7/79: Epistaxis, neg PE	R PPF, NP, sphenoid sinus	R maxillary III (E), R internal carotid	Tr eth, Tr Ant	5'40"	2,500	NET 6 mo, neg CT	IIB, IB

TABLE 1 Continued

Case No.	Symptoms and Signs	Location*	Blood Supply	Surgery			Follow-up	Classifica-tion†
				Approach	OR Time	EBL (ml)		
11	1975: Nasal obstruction, R NP mass	R NP, PPF-PMF, sphenoid sinus, cavernous sinus (pre-CT)	?	Tr Pal, Tr Ant	...	6,000	Evaluated and operated else-where; referred for XRT; asympto-matic 4 yr	?Ipsi III, IIB, IB
	1980: Epistaxis	PPF	R internal maxillary III		...	...	Resect if more epistaxis	IIB
12	1975: Obstruction, rhinorrhea, rhinolalia, L NP, choanal mass with septal deviation	L choanal, bilateral, NP, L PPF, sphenoid sinus, cavernous sinus (pre-CT)	L maxillary II and III, L internal carotid	...	...	...	...	?Ipsi III, IIB, IB
	1979: Asymptomatic, small NP mass	PPF, sphenoid sinus	...	...	...	...	Decreased tumor size	III, IIB

Note.—(E) = vessel embolized preoperatively; EBL = estimated blood loss; HA = headache; Ipsi = ipsilateral; ITF = infratemporal fossa; NET = no clinical evidence of tumor; NP = nasopharyngeal; OR = operating room; PBFA = palatine branch facial artery; PE = physical examination; PMF = pterygomaxillary fissure; PPF = pteryogopalatine fossa; Tr ant = transantral; Tr eth = transethmoid; Tr nas = transnasal; Tr pal = transpalatine; and XRT = x-ray therapy. Maxillary I, II, and III refer to branches off segments of the maxillary artery as defined by Allen et al. [12]. Irradiation was administered in the following cases: case 7, 5,600 rad (56 Gy); case 8, 5,500 rad (55 Gy); case 9, 4,600 rad (46 Gy); case 10, 5,600 rad (56 Gy); case 11, 4,400 rad (44 Gy); and case 12, 5,600 rad (56 Gy).
* Location was determined by CT unless otherwise noted.
† Classification criteria are defined in the text.

angioarchitecture on angiography. Nine were operated, eight after catheter embolization. Three were deemed inoperable and treated with irradiation. Several patients had multiple radiologic studies, surgeries, and x-ray treatments. They are summarized in table 1.

Discussion

Review of our patients and earlier reports leads to the obvious conclusion that for treatment purposes juvenile angiofibroma cannot be considered a single entity. Rather, it is a single histologic lesion that occurs in different ana-tomic compartments; it is the anatomic location that deter-mines the treatment method.

There are three main anatomic compartments that these tumors may occupy, each requiring its own treatment method. We offer the following classification (fig. 1):

Type I. Lesions in the median aspect of the skull base without intracranial extension. These tumors basically lie between the pterygoid processes. This type is divided into two subcategories: type IA is restricted to the nasal cavity and nasopharynx and type IB includes extension into the maxillary, ethmoid, and/or sphenoid sinuses.

Type II. Lesions extending laterally from the sphenopala-tine foramen. If only a small part of tumor occupies the pterygopalatine fossa, it is a IIA lesion; if it extends across the pterygomaxillary fissure, it is IIB; if it extends more laterally into the infratemporal fossa, it is type IIC.

Type III. Lesions extending intracranially. Usually they involve the medial wall of the middle fossa, including the cavernous sinus.

Using this system, we classified our cases in the final column of table 1. This classification is based on simple anatomic units and indicates appropriate treatment regi-mens. Type I lesions are surgically resectable by median or paramedian approaches. Type IA may be treated by the transpalatal approach (fig. 2). A transnasal or transethmoid approach may be added to the transpalatal incision for type IB lesions (fig. 3). Type II lesions usually require a more lateral approach, mainly transantral (Caldwell-Luc). Small type IIA tumors might be reached transpalatally. The more lateral type IIC lesions may require a lateral sublabial exten-sion of the transantral approach. Tumors with both type I and type II components will require medial and lateral ap-proaches. Type III lesions are not completely resectable and require irradiation [2, 13, 14].

One of the problems in evaluating previous treatment regimens has been the lack of a classification system. This was mainly due to the lack of a diagnostic tool to accurately show the full extent of the tumor. Clinical evaluation alone is not adequate.

Pluridirectional tomography provides information as to tumor location by the indirect evidence of bone erosion and replacement of air-filled structures. However, it does not differentiate between tumorous or nontumorous soft tissues. This limitation is particularly important in evaluating lateral extension and sinus opacification. Enlargement of the pter-ygomaxillary fissure (Holman sign) (fig. 4) implies lateral growth, but only CT clearly shows how far the tumor has extended. A sinus may be opaque either from tumor or ostial
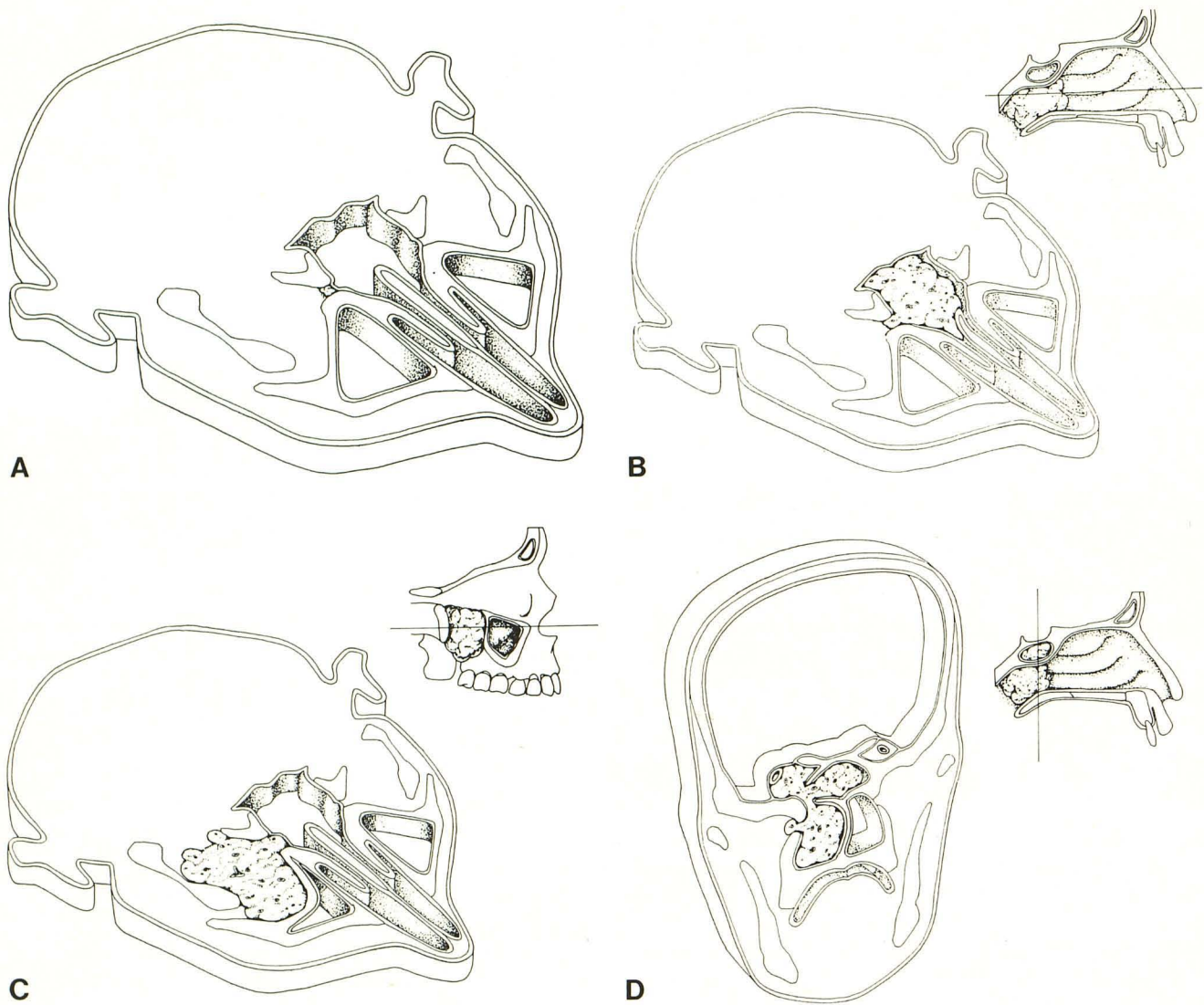
A

B

C

D

Fig. 1.—Anatomic classification of juvenile angiofibromas. **A,** They arise in area of sphenopalatine foramen and pterygopalatine fossa. **B,** Type I lesions extend medially into nasopharynx. **C,** Type II lesions extend laterally through pterygomaxillary fissure into infratemporal fossa. **D,** Type III lesions extend superiorly into middle fossa and cavernous sinus region via foramina at skull base.
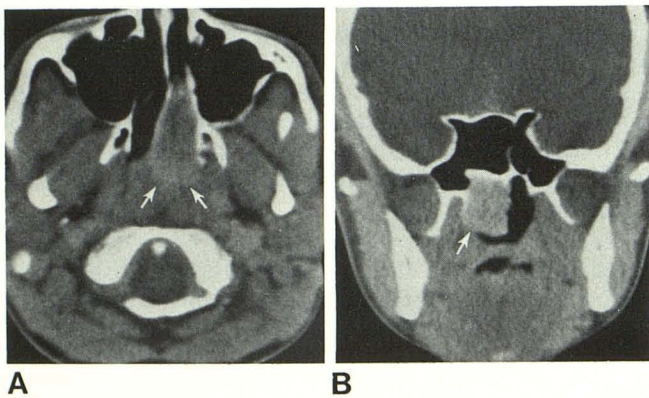

A   **B**

Fig. 2.—Case 1, type IA. Axial (**A**) and coronal (**B**) scans after contrast injection. Enhancing lesion within left posterior nasal cavity and nasopharynx (*arrows*).

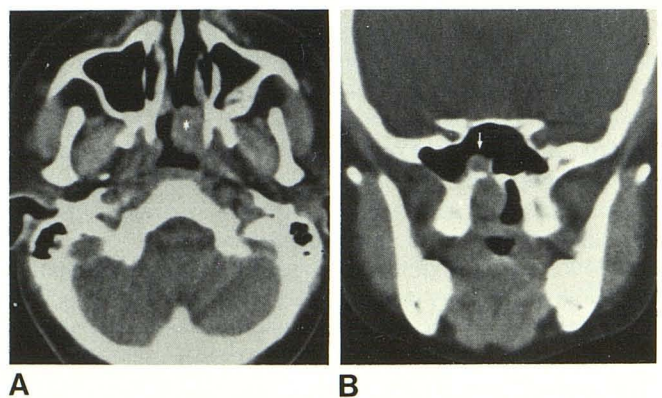**A**   **B**

Fig. 3.—Case 2, type IB. Axial (**A**) and coronal (**B**) scans after contrast injection. Small left posterior choanal lesion (*) extends slightly into sphenoid sinus (*arrow*).
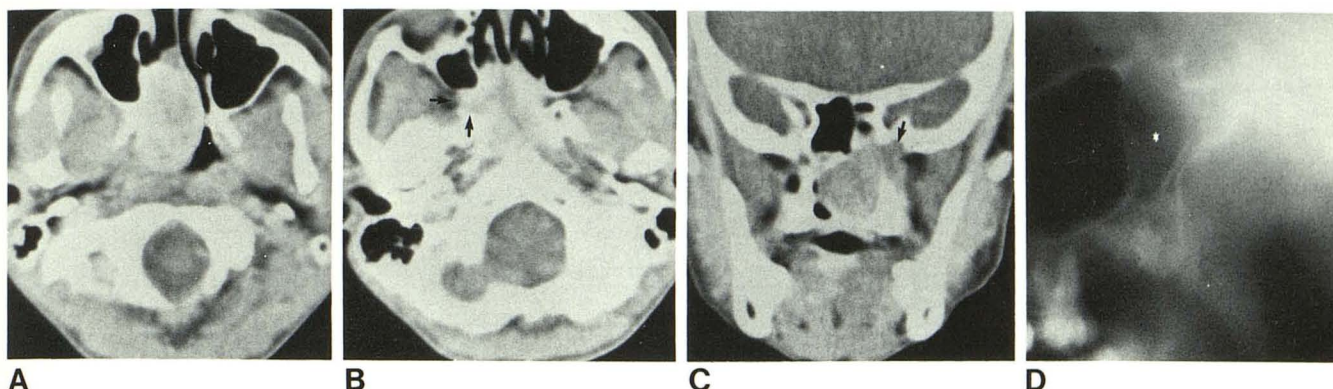
Fig. 4.—Case 4, combined types IIB and IA. **A,** Postcontrast axial scan. Obvious enhancement of large right posterior nasal and nasopharyngeal mass. Higher axial (**B**) and coronal (**C**) scans reveal critical lateral extension of tumor into pterygopalatine fossa and pterygomaxillary fissure (*arrows*). **D,** Lateral polytomogram. Widening of pterygomaxillary fissure (*) indicates mass in this area, and might suggest more lateral extension than seen on CT.
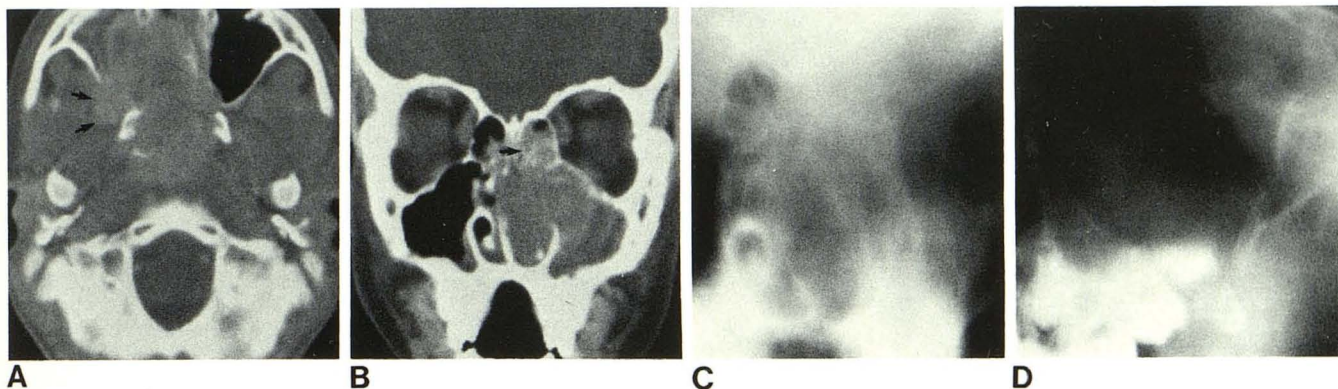

Fig. 5.—Case 5, combined types IIC and IB. **A,** Postcontrast axial scan. Large, lobulated enhanced mass fills right posterior nasal cavity and nasopharynx with extension to opposite nasopharyngeal wall. Lesion has extended anteromedially into obstructed maxillary sinus and grown laterally through pterygopalatine fossa and pterygomaxillary fissure into medial infra-temporal fossa (*arrows*). **B,** Coronal scan. Superior extension into middle ethmoid sinus (*arrow*). Anteroposterior (**C**) and lateral (**D**) polytomograms of lesion show bone erosion and sinus opacification, but differentiation between tumor and fluid-filled sinus is seen only on CT.

obstruction. Contrast enhanced CT makes this differentiation (fig. 5).

Angiography may accurately demonstrate the tumor by its vascularity and confirm the histologic diagnosis. However, in most cases, the overwhelming mass of vessels defines the periphery of the tumor, but obscures the details of gross anatomic extension. This is particularly important because these tumors tend to grow, not as a sphere, but in a lobulated fashion into and through various fissures, foramina, and sinus compartments. While it is generally true that parts of tumors are supplied by appropriate vessels, this may be deceiving, particularly in postsurgical or irradiated patients. For instance, intracranial extension is said to always be associated with feeding vessels from the cavernous carotid artery [13]. In figure 6, CT clearly shows intracranial extension to the cavernous sinus region, but selective internal carotid angiography reveals no cavernous tumor supply.

We believe CT reveals the gross anatomy of these lesions far better than other imaging techniques. The geography of the lesions is usually obvious even without contrast injection. However, contrast injection is important to better define the limits of the tumor and suggest the correct histologic diag-

nosis. Other nasopharyngeal lesions occur in young males and may mimic angiofibromas. We scanned boys with antral choanal polyps, lymphoma, and squamous carcinoma. All of these lesions had different anatomic distributions and enhanced no more than adjacent muscle tissue. All the angiofibromas enhanced more than adjacent muscle. It is important that the scans be performed shortly after or during contrast injection. Because of the rapid equilibration of intra- and extravascular contrast concentrations, the initially high iodine levels within the lesion may not be obvious if scanning is delayed.

We believe a classification system will allow more effective comparison of treatment methods. It is clear from our cases that surgical treatment is simpler (as indicated by surgery time, blood loss, and personal experience) in type I and IIA lesions. These lesions should not be compared with type IIB and III tumors.

Many of the tumors cross the midline and the classification type should be prefixed with either "ipsilateral" or "bilateral." However, this is not as important as it might seem. These tumors are not (?ever) "bilateral" tumors in the sense of symmetry. They arise from one side and expand across
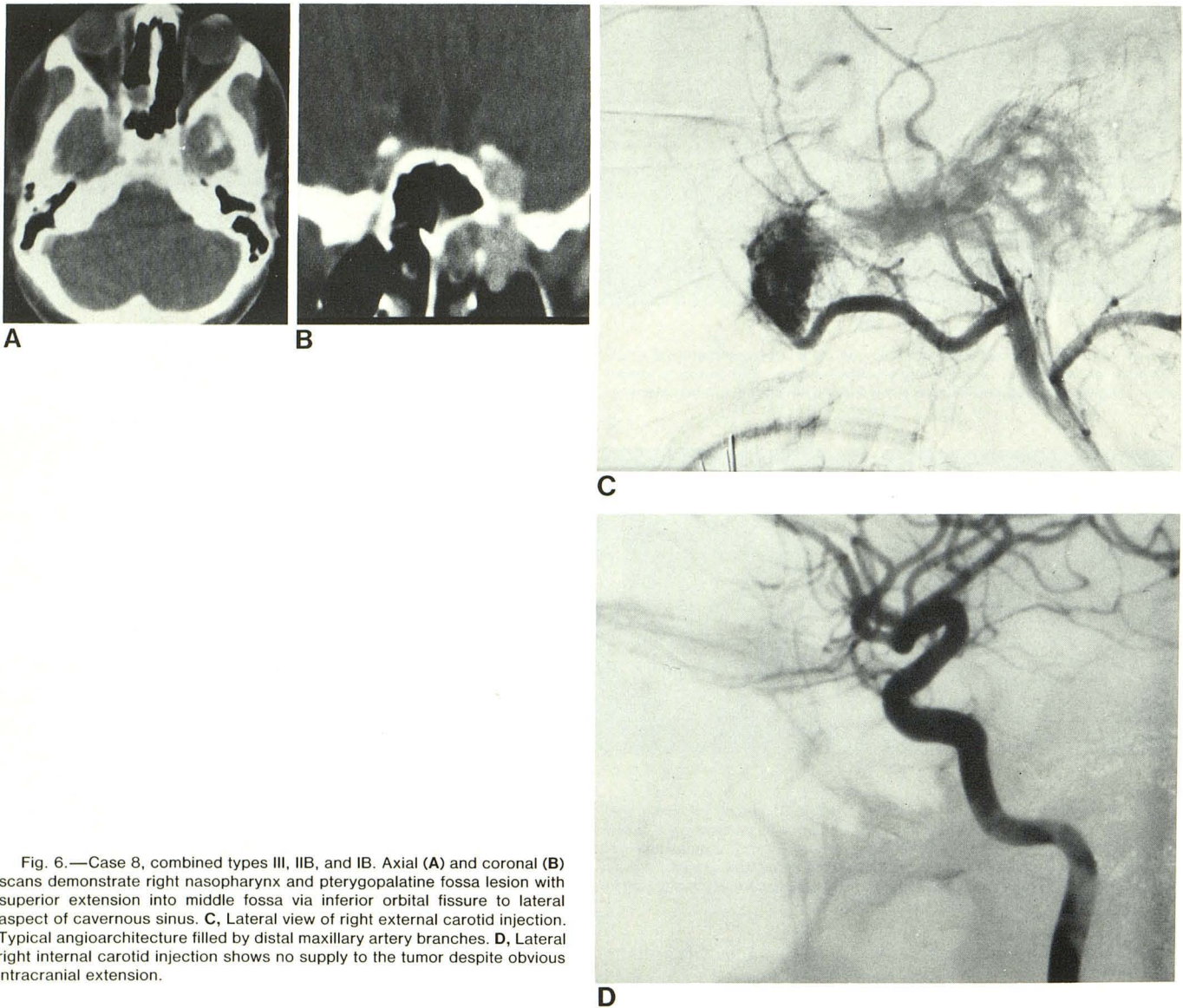
Fig. 6.—Case 8, combined types III, IIB, and IB. Axial (**A**) and coronal (**B**) scans demonstrate right nasopharynx and pterygopalatine fossa lesion with superior extension into middle fossa via inferior orbital fissure to lateral aspect of cavernous sinus. **C,** Lateral view of right external carotid injection. Typical angioarchitecture filled by distal maxillary artery branches. **D,** Lateral right internal carotid injection shows no supply to the tumor despite obvious intracranial extension.

the midline. They seldom extend beyond the nasopharyngeal wall of the opposite side. The transpalatal approach usually allows resection of any extension across the midline. Gross anatomic extension across the midline is easily determined by CT, but laterality of blood supply is better determined by angiography. This determination is important in planning embolization or other hemostatic procedures.

Complete surgical resection of types I and II tumors is possible, and we believe it is the treatment of choice. This may (and probably should) be preceded by preoperative embolization. Eight of our nine surgical cases had preoperative embolization. Our embolization technique includes first angiography to identify the major supplying vessels, selective catheterization of the appropriate feeding vessels, and initial embolization with small (0.5 and 1.0 mm Silastic spheres). We prefer to start with these solid emboli because we believe they are flow directed better than pledgets of Gelfoam and less likely to obstruct very small vessels, such as vasa nervorum, which might lead to neurologic deficit

[15, 16]. After flow has been significantly reduced by the Silastic spheres, the selected vessel is then carefully and completely occluded by small pledgets of Gelfoam. We believe this combination also allows a longer lasting occlusive effect, while still allowing subsequent recanalization of the major feeding vessels. This is particularly important to us because, for logistical purposes, we perform the embolization the afternoon before surgery and Gelfoam alone does not provide adequate occlusion over the intervening 12-hr period (fig. 7). We have had no complications from embolization other than the expected local pain, transient fever, and groin tenderness.

We intended to compare our embolized cases with previous nonembolized cases, however it quickly became apparent that we had no reliable means of selecting comparable cases. We believe this is probably true of nearly all previous reports comparing various treatment methods for these tumors. This includes reports comparing surgical approaches, hormone therapy, irradiation, and embolization.
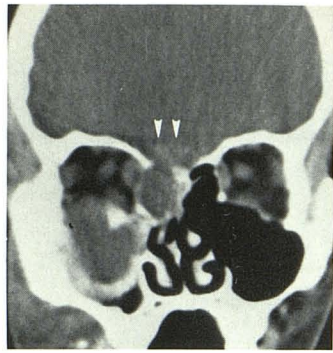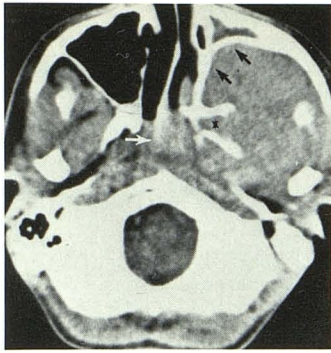
Fig. 7.—Case 9, combined types III, IIC, and IB. **A,** Axial scan. Massive tumor with only small nasopharyngeal extension (*white arrow*) from pterygopalatine fossa–pterygomaxillary fissure (*), but large component in infratemporal fossa bowing back of maxillary sinus (*black arrows*). **B,** Coronal scan. Superior extension into ethmoid sinus and through cribriform plate into anterior fossa (*arrowheads*). **C,** Initial lateral left external carotid angiogram. Large tumor with typical angioarchitecture fed by branches of all parts of internal maxillary artery (*long arrow*), ascending pharyngeal artery (*arrowhead*), and branches off middle meningeal artery including orbital branches (*short arrows*). **D,** Postembolization film. Occlusion of most feeders. Ascending pharyngeal and first and second parts of internal maxillary artery were embolized with Silastic spheres (note balls in second part of maxillary artery [*arrow*]). Middle meningeal and origin of maxillary artery were embolized with pledgets of Gelfoam. **E,** Internal maxillary artery angiogram 12 hr later. Partial recanalization of infratemporal (*arrow*) and posterior pterygoid branch (*arrowhead*) of proximal internal maxillary artery. **F,** Additional Gelfoam embolization further diminished blood supply. Tumor was partially resected to decrease necessary radiation volume.
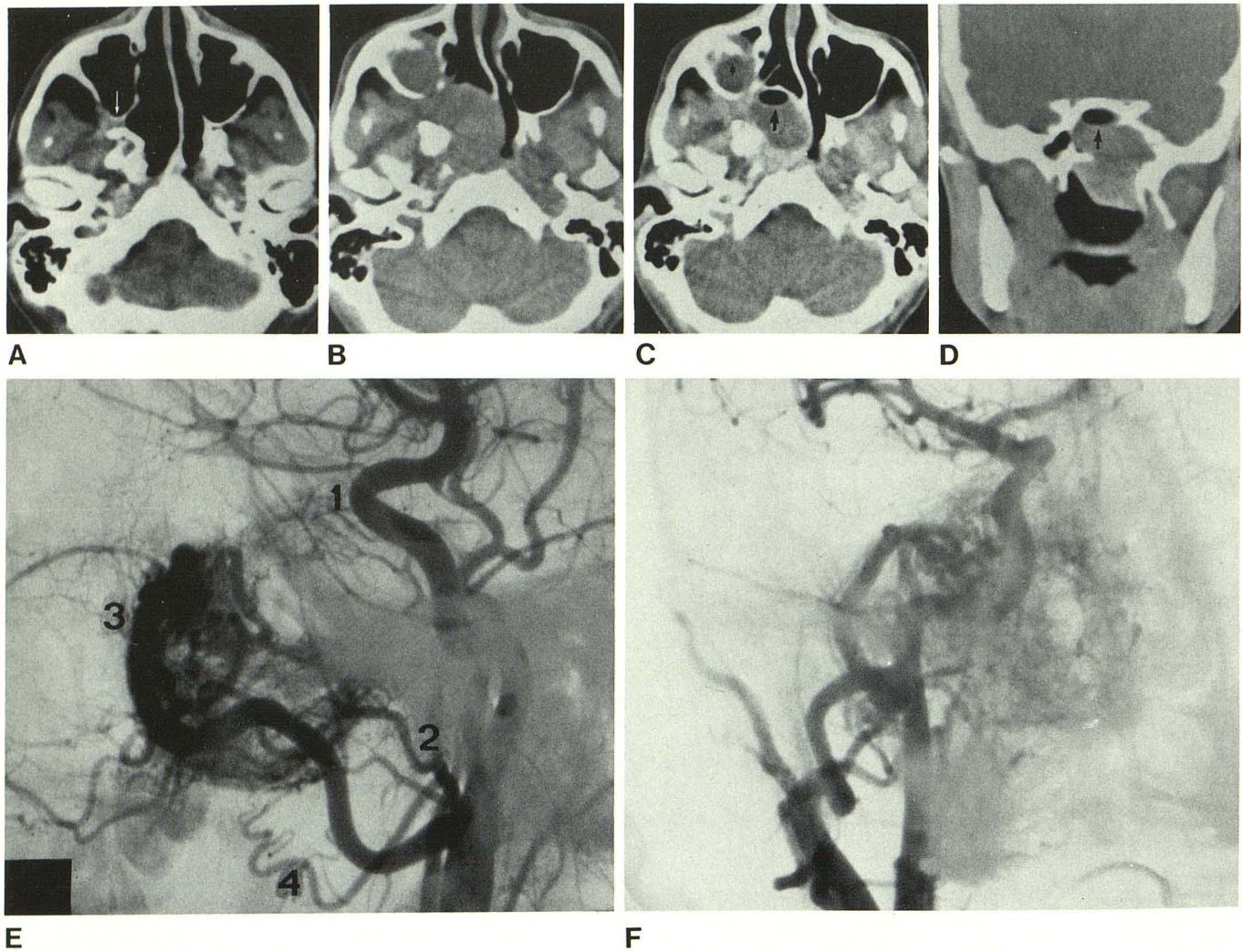
A

B
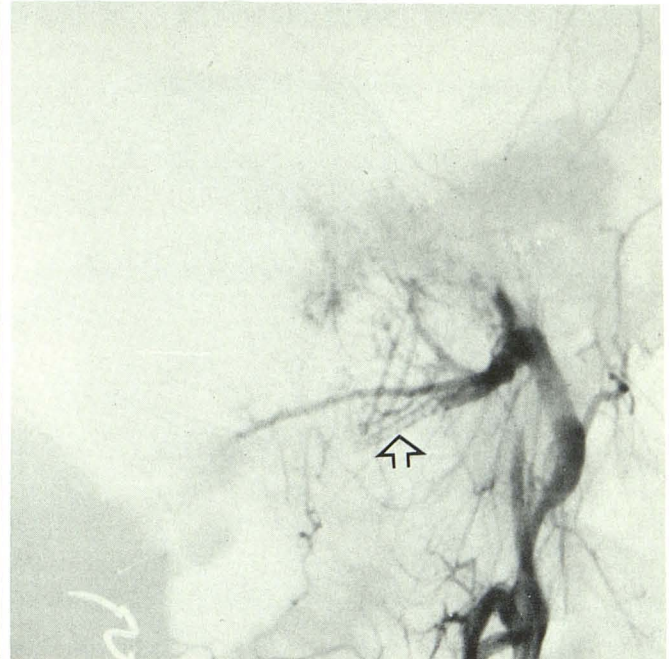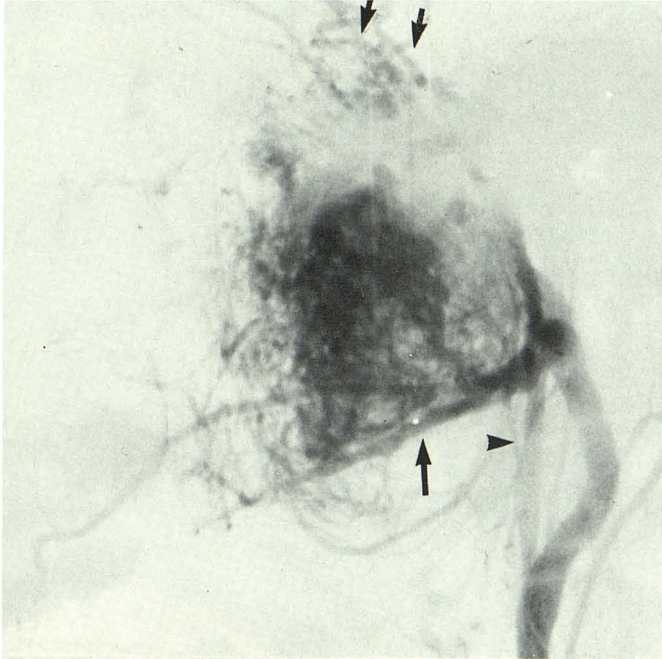
C

D
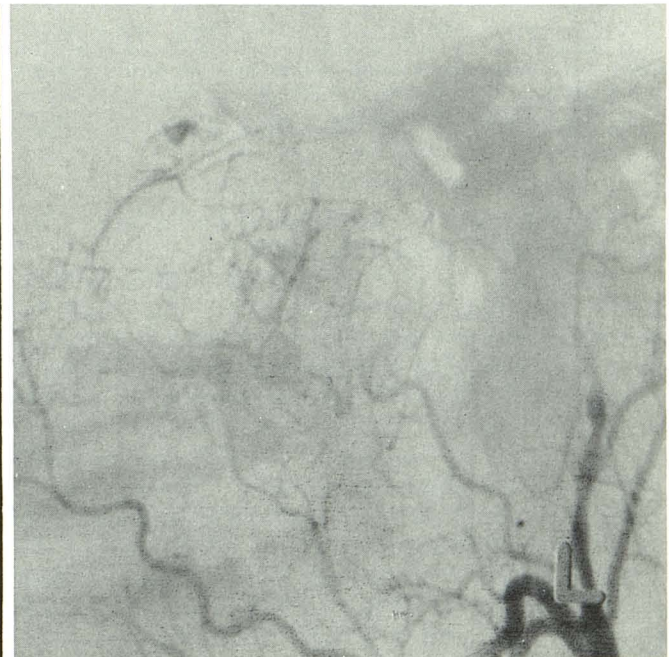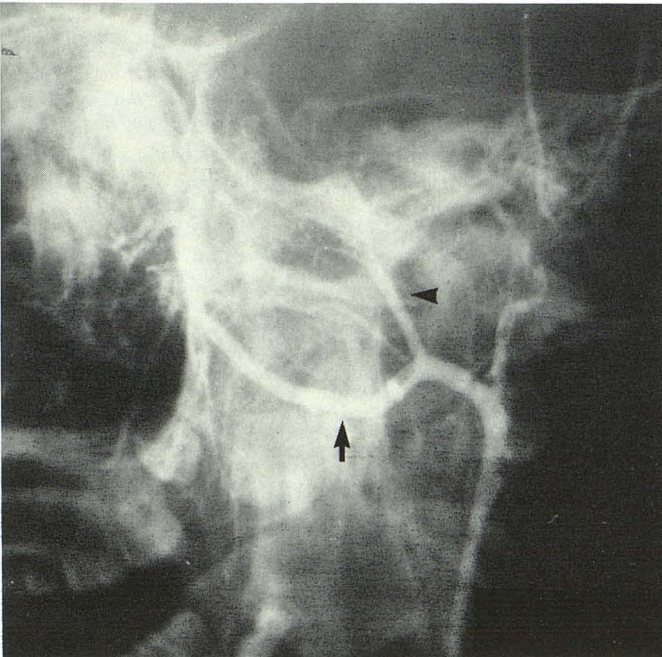
E

F

Fig. 8.—Case 10, types IIB becoming IIB-IB. **A,** Postcontrast axial scan. Only small residual tumor (*arrow*) in pterygopalatine fossa–pterygomaxillary fissure 6 years postirradiation. Precontrast (**B**) and postcontrast (**C**) axial and coronal (**D**) scans 7 weeks later. Regrowth of tumor with peripherally enhancing, cystic component in nasopharynx and sphenoid sinus. Air within cyst (*arrow*) and nonenhancing obstructed maxillary sinus (*). Lateral (**E**) and anteroposterior (**F**) angiograms at same time. Typical angioarchitecture with supply from internal carotid (1), ascending pharyngeal (2), III portion maxillary (3), and palatine (4) arteries. True cystic nature of tumor is difficult to appreciate. All except internal carotid branches were embolized. Lesion was easily resected including cystic part which collapsed after aspiration and dropped out of sphenoid sinus.

We hope that use of a classification system as we propose will alleviate this problem in the future.

In our experience, irradiation has been limited to those patients with intracranial extension. Five cases have received irradiation and been followed with CT for intervals up to 5 years. While there was clinical evidence of regression of tumor in all cases, there was also radiographic evidence of residual neoplasm in all cases. It is hoped that these residual lesions will continue to regress and completely resolve when the patients reach young adulthood.

We have found that clinical evaluation in these patients is not accurate in determining residual tumor. Four of five irradiated patients were clinically without evidence of neoplasm, although they clearly had tumor on CT and/or angiography. This limitation must be considered when evaluating earlier reports [17]. Furthermore, diminution of tumor

size after irradiation does not mean that its potential for growth is lost. In case 10 (fig. 8), the tumor had resolved to a small nubbin of tissue in the pterygopalatine fossa before it dramatically grew to a medium-sized tumor in the nasopharynx and sphenoid sinus. We believed this pattern of incomplete regression is the usual response to irradiation (figs. 8 and 9).

Further review of these cases leads us to the following postulates on the origin and growth of these tumors (fig. 1). All lesions have a component in the sphenopalatine foramen. We suggest that the tumors arise in this area, but do not know why. This origin is perhaps related to residual vascular erectile tissue, as postulated by Shiff [18] in 1959. We believe this location is a more likely origin for these tumors than other postulated regions at the base of the skull, including the area of the spheno-occipital synchondrosis
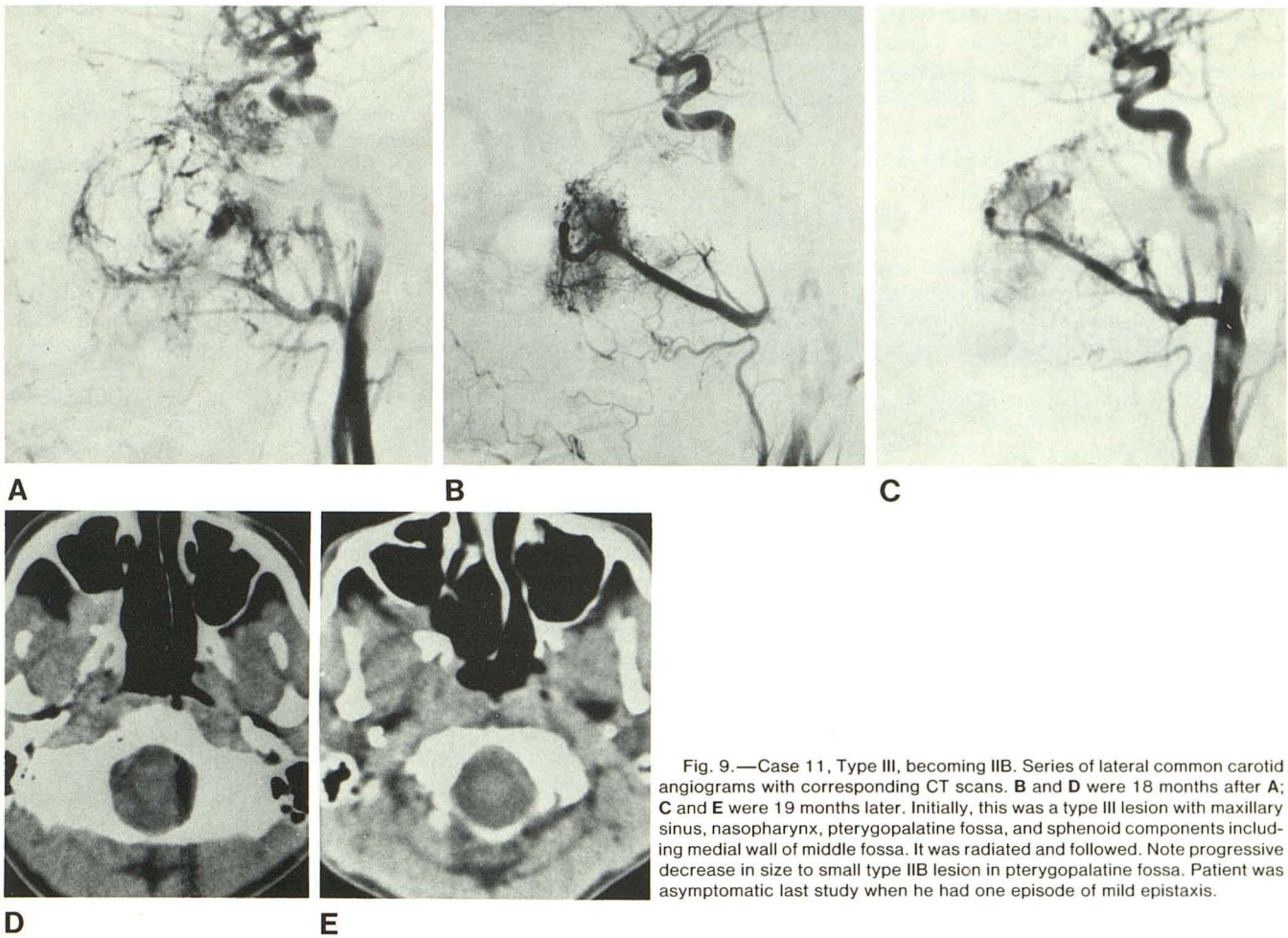
A　　　　　　　　B　　　　　　　　C

Fig. 9.—Case 11, Type III, becoming IIB. Series of lateral common carotid angiograms with corresponding CT scans. **B** and **D** were 18 months after **A**; **C** and **E** were 19 months later. Initially, this was a type III lesion with maxillary sinus, nasopharynx, pterygopalatine fossa, and sphenoid components including medial wall of middle fossa. It was radiated and followed. Note progressive decrease in size to small type IIB lesion in pterygopalatine fossa. Patient was asymptomatic last study when he had one episode of mild epistaxis.

D　　　　　　　　E

[19]. The tumors may then grow in the natural bony channels about this foramen. Medial growth produces tumor in the posterior choanal opening and nasopharynx. These medial tumors (type I) may then grow superiorly, and secondarily involve the ethmoid and sphenoid sinuses. Lateral growth from the sphenopalatine foramen results in tumor within the pterygopalatine fossa and still more laterally into the pterygomaxillary fissure and finally the infratemporal space. These are type II tumors. Superior growth is possible via the pterygopalatine fossa through the foramina at the base of the skull, particularly foramen rotundum and inferior orbital fissure to the area of the cavernous sinus and medial margins of the middle fossa. Superior growth such as this produces a type III tumor. Significant inferior growth is seldom seen, probably due to the minimal soft tissue space in the inferior aspect of the pterygopalatine fossa. Anterior extension into the maxillary sinus is nearly always secondary to invasion from medial nasal components of the tumor or from lateral components in the pterygopalatine fossa.

Probably the most critical anatomic area in these cases is the pterygopalatine fossa. Essentially all tumors will have a component contiguous to or within this small space. Even tumors that initially appear to be completely within the nasopharynx and nasal cavity will almost always have a small nubbin of tissue in the medial aspect of the pterygopalatine fossa. One must look closely for this component as it is the part of tumor most likely to be left after a paramedian approach to a type I or IIA tumor. If there is significant lateral extension into the full width of the pterygopalatine fossa (to the plane of the lateral plate of the pterygoid process) or if there is any question in the surgeon's mind during surgery as to residual neoplasm in the fossa, then a transantral approach to the pterygopalatine fossa must be made.

From our experience, we believe the optimum radiologic evaluation of patients with possible juvenile angiofibroma includes plain films followed by CT in the axial and coronal projections, with and without contrast injection. Based on the CT scans, the presumptive diagnosis is made and the lesion anatomically classified according to our criteria. Based on the tumor location, appropriate therapy is planned. However, any therapeutic program is preceded by angiography to confirm the diagnosis by demonstrating appropriate angioarchitecture. If the lesion is operable, transcatheter embolization is performed in conjunction with the angiography. This approach allows not only confident diagnosis, but also appropriate therapy planning. Such planning will

allow complete surgical resection with reasonable morbidity in all cases except those with intracranial extension. We also believe the classification system will aid in comparison of treatment modalities and results from different institutions.

REFERENCES

1. Harrison DFN. Juvenile postnasal angiofibroma—an evaluation. *Clin Otolaryngol* **1976**;1:187–197
2. Jereb B, Anggard A, Baryd I. Juvenile nasopharyngeal angiofibroma. A clinical study of 69 cases. *Acta Radiol [Diagn]* (Stockh) **1970**;9:302–310
3. Martin JS. Nasopharyngeal fibroma and its treatment. *J Laryngol Otol* **1954**;68:39–54
4. Johns ME, MacLeod RM, Cantrell RW. Estrogen receptors in nasopharyngeal angiofibromas. *Laryngoscope* **1980**;90:630–634
5. Holman CB, Miller WE. Juvenile nasopharyngeal fibroma—roentgenologic characteristics. *AJR* **1965**;94:292–298
6. Wilson GH, Hanafee WN. Angiographic findings in 16 patients with juvenile nasopharyngeal angiofibroma. *Radiology* **1969**;92:279–284
7. Duckert LG, Carley RB, Hilger JA. Computerized axial tomography in the preoperative evaluation of an angiofibroma. *Laryngoscope* **1978**;88:613–618
8. Gonsalves CG, Briant TDR. Radiographic findings in nasopharyngeal angiofibromas. *J Can Assoc Radiol* **1978**;29:209–215
9. Weinstein MA, Levin H, Duchesneau PM, Tucker HM. Diagnosis of juvenile angiofibroma by computed tomography. *Radiology* **1978**;126:703–705
10. Pletcher JD, Newton TH, Dedo HH, Norman D. Preoperative embolization of juvenile angiofibromas of the nasopharynx. *Ann Otol Rhinol Laryngol* **1975**;84:740–746
11. Roberson GH, Price AC, Davis JM, Gulati A. Therapeutic embolization of juvenile angiofibroma. *AJR* **1979**;133:657–663
12. Allen WE, Kier EL, Rothman SLG. The maxillary artery: normal arteriographic anatomy. *AJR* **1973**;118:517–527
13. Ward PH, Thompson R, Calcaterra T, Kadin MR. Juvenile angiofibroma: a more rational therapeutic approach based upon clinical and experimental evidence. *Laryngoscope* **1974**;84:2181–2194
14. Sessions RB, Wills PI, Alford BR, Harrell JE, Evans RA. Juvenile nasopharyngeal angiofibroma: radiographic aspects. *Laryngoscope* **1976**;86:2–18
15. Hilal SK, Michelsen JW. Therapeutic percutaneous embolization for extra-axial vascular lesions of the head, neck and spine. *J Neurosurg* **1975**;43:275–287
16. Lasjaunias P. Nasopharyngeal angiofibromas: hazards of embolization. *Radiology* **1980**;136:119–123
17. Briant TDR, Fitzpatrick PJ, Berman J. Nasopharyngeal angiofibroma: a twenty year study. *Laryngoscope* **1978**;88:1247–1251
18. Schiff M. Juvenile nasopharyngeal angiofibroma. A theory of pathogenesis. *Laryngoscope* **1959**;69:981–1016
19. Osborn DA. The so-called juvenile angiofibroma of the nasopharynx. *J Laryngol Otol* **1959**;73:295–316