# INTRACRANIAL HEAD AND NECK TUMORS: ENDOVASCULAR CONSIDERATIONS, PRESENT AND FUTURE

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TO REVIEW THE literature on endovascular therapies available to clinicians to aid in the management of head, neck, and intracranial tumors. Hypervascular tumors of the head and neck region, as well as the intracranial region, are associated with large amounts of blood loss intraoperatively. Preoperative embolization of selected hypervascular tumors has been proposed in the literature as a method of reducing blood loss intraoperatively. This technique involves superselective catheterization of the feeding arteries to the tumor bed and then by infusion of embolic particles to saturate the tumor bed in the hopes of inducing necrosis. For less vascular tumors, selective infusion of chemotherapeutic agents has been reported as a method of reducing the systemic toxic effects of these medications. Endovascular therapies for hypervascular and less vascular tumors hold promise, although multicenter randomized controlled trials are required to help identify the patients that will benefit the most.

**KEY WORDS:** Angiography, Embolization, Endovascular therapy, Intra-arterial chemotherapy, Neoplasm

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There are roughly 39,000 new cases of central nervous system (CNS) tumors (46) and 37,000 new cases of head and neck cancer (64) diagnosed in the United States each year. Embolization of these tumors has become an important adjunct to the surgical treatment of these tumors. In addition to facilitating surgery, they can be used in isolation as palliative treatment for nonresectable tumors and for delivering chemotherapeutic agents. One of the earliest reported cases of a successful embolization was in 1974 by Hekster et al. (29). Since then, many reports or series have been published examining the potential benefits of this therapy, but no consensus has been reached.

Endovascular therapies are not limited to embolization procedures for head and neck tumors. Nonvascular CNS tumors, such as lymphomas, can be treated with blood–brain barrier (BBB) disruption, and squamous cell carcinomas (SCC) can be treated with intra-arterial chemotherapy infusions. In this review, we will discuss the endovascular management of CNS and head and neck tumors.

## INDICATIONS AND GENERAL PRINCIPLES

Embolization is generally used only in the management of vascular tumors. *Table 1* summarizes the indications for embolization of tumors. *Table 2* summarizes the common hypervascular tumors that are treated with embolization preoperatively.

Embolizations can be performed by either an endovascular approach or direct injection of embolic agents into the tumor. The aim of embolization is to devascularize the tumor bed by saturating these capillaries, with the hope of promoting tumor necrosis. Sacrificing proximal arterial feeders will do little to help with this endeavor. The smallest particles that are feasible should be used with embolization to penetrate the small capillary beds. The size of these capillary beds varies based on tumor pathology. The limiting factor in using smaller particles is recognition of dangerous anastamoses that can occur between the external carotid artery (ECA) and internal carotid artery (ICA) branches and arteriovenous shunting within the tumor bed. *Table 3* lists some of these anastomoses (71). In addition, there is

**TABLE 1. Indications for tumor embolization**

| |
|---|
| 1. Control surgically inaccessible arterial feeders |
| 2. Decrease surgical morbidity by reducing blood loss |
| 3. Shorten the operative procedure time |
| 4. Increase the chances of complete surgical resection |
| 5. Decrease the risk of damage to adjacent normal tissue |
| 6. Relieve intractable pain |
| 7. Decrease expected tumor recurrence |
| 8. Allow better visualization of the surgical field with decreased overall surgical complication |

blood supply to vital structures such as cranial nerves and nerve roots via ECA and vertebral artery branches as listed in *Table 4*. In general, particles smaller than 150 $\mu$m in diameter and liquid embolization should be avoided if these anastamoses are seen or if embolization is being performed in branches supplying cranial nerves or nerve roots (7).

Embolic material can be divided into three major categories: liquid, particulate, and coils. *Table 5* summarizes these various embolic agents and the advantages and disadvantages of each of them. Liquid and particulate agents are used primarily to aid in necrosing of the tumor capillary bed when the microcatheter can be placed in a safe position. Selection of each agent is based on location of the feeding artery (i.e., risk of injuring normal structures) and the desired effect in relation to the planned surgery. If surgery is planned within a few days of embolization, selection of temporary occlusive materials such as particles is reasonable. Fibered and nonfibered detachable coils are used to sacrifice the feeding pedicle to reduce the rate of recanalization, especially when using particulate embolization.

**TABLE 2. Common head and neck tumors that are treated with endovascular embolization**

| |
|---|
| 1. Hemangioblastomas |
| 2. Meningiomas |
| 3. Intracranial and extracranial metastases |
| 4. Hemangiopericytomas |
| 5. Neurogenic tumors (e.g., schwannomas) |
| 6. Paragangliomas |
| 7. Juvenile nasopharynegeal angiofibromas |
| 8. Hemangiomas |

## TECHNIQUES

A diagnostic angiogram is performed using the transfemoral technique with cannulation of the common carotid arteries and selective injection of the ICA and ECA. Selective catheterizations of the feeding vessels are performed to provide complete vascular mapping of these lesions. Embolization procedures are initiated with superselective catheterization of the distal most vessel. By entering the distal most vessel, the operator avoids the problem of proximal vasospasm precluding embolization of more distal vessels. On entering the distal arterial feeding vessel to the tumor bed, the feeding pedicles are tested for supply to cranial nerves. An injection of 3 ml of lidocaine is administered, and the patient is tested for cranial nerve and vision deficits before embolization proceeds. If the patient does not incur a deficit with testing, the tumor's capillaries are slowly saturated with embolic particles under constant fluoroscopic guidance. It is important to maintain a slow but steady injection rate while watching for reflux of particles, which would signal the operator to stop injecting. Once the tumor is saturated, the proximal feeding vessel is occluded with either Gelfoam (Upjohn Co., Kalamazoo, MI) or platinum coils before the process is repeated on the next most distal vessel and on through all of the feeding vessels.

For tumors with small arterial feeders coming off larger vessels supplying normal neural structures, we use a technique of liquid embolization using alcohol. By temporarily occluding the larger vessel with a balloon distal to the feeding artery site, we have found that alcohol can be safely injected into these small feeders to aid in devascularizing a tumor (32). Others have described similar techniques (80). However, this technique is still controversial because it can lead to complications. After the balloon is deflated, particles may still be floating in the ICA, and they might distally embolize and cause stroke.

Periprocedurally, it is important to ensure that the patient is well hydrated with intravenous fluids to help protect the kidneys from the iodinated contrast load. Postembolization, patients undergo surgical resection

**TABLE 3. Dangerous anastamoses from external carotid artery branches to consider before embolization procedures[a] (71)**

| Vessel | Anastamoses |
|---|---|
| Anterior deep temporal artery | Ophthalmic artery |
| Accessory meningeal | ICA |
| Middle meningeal | Ophthalmic, inferolateral trunk (cavernous ICA) |
| Ascending pharyngeal | VA (via hypoglossal art), ICA (via carotid branch) |
| Occipital artery | VA |
| Facial artery | Ophthalmic |
| Vidian artery | Remnant communication between ECA and petrous ICA |
| Artery of foramen rotundum | Internal maxillary artery to cavernous ICA connection |

[a] ICA, internal carotid artery; ECA, external carotid artery; VA, vertebral artery.

within 7 days. This is to avoid neovascularization and new collaterals that can form rapidly. Swelling after embolization is often a concern, and patients can be treated with steroids pre- and postprocedure to help reduce edema.

## MENINGIOMAS

Meningiomas account for roughly 15% of all intracranial tumors and typically occur in adults between the ages of 40 and 60 years (71). These tumors can be cured through surgical excision (1), although considerable debate exists in the literature as to the advantages of presurgical embolization.

Meningiomas are usually supplied by dural arteries. These arteries include the middle meningeal artery, accessory meningeal, ascending pharyngeal, or occipital transmastoid-perforating branches of the ECA. Dural arteries also include the tentorial and inferolateral trunk branches of the ICA, as well as the posterior meningeal branch of the vertebral artery. There is often additional supply derived from pial vessels (56). The vascular supply to meningiomas typically varies based on the location of the tumor (*Table 6*).

The aim of embolization is to help reduce blood loss intraoperatively and induce necrosis of the tumor (81). Studying the effectiveness of embolization has been challenging because of the varied sizes, location, and vascular supply to meningiomas. In addition, most studies have relied on subjective reports of blood loss during surgeries, although recent studies have considered gadolinium enhancement on magnetic resonance imaging as a marker of efficacy (25). *Figure 1* shows an example of pre- and postembolization treatment of a sphenoid wing meningioma with marked reduction in gadolinium enhancement. A reduced perioperative blood loss was highly correlated with a reduction in the degree of gadolinium enhancement (25). One of the larger randomized controlled trials has shown that preoperative embolization can reduce blood transfusions perioperatively and may also be more cost-effective in treating patients with meningiomas (14). Wakhloo et al. (81) showed that the blood loss was moreover related to the size of particles used during embolization. A 500- to 2600-ml perioperative blood loss was noted in 70% of cases in which polyvinyl alcohol (PVA) particles that were 150 to 300 $\mu$m in diameter were used. When PVA particles that were 50 to 150 $\mu$m in diameter were used, the blood loss was less than 500 ml in all but two patients of a total of 20 patients. In those two patients, the blood loss was limited to 800 ml. On histological examination, it was noted that 15 out of the 20 patients treated with particles 50 to 150 $\mu$m in diameter had evidence of particles in the tumor capillary bed (81).

Superselective angiography of the feeding vessels with mi-

**TABLE 4. Vascular supply to cranial nerves and nerve roots of commonly embolized vessels**

| Artery | Nerves |
|---|---|
| Middle meningeal artery | VII, Vm, V3, gasserian ganglion |
| Accessory meningeal artery | V3, Vm, V2, VII |
| Inferolateral trunk | III, IV, V1, V2, V3, Vm, gasserion ganglion, VI |
| Marginal tentorial artery | III, IV |
| Ascending pharyngeal artery | Gasserion ganglion, VI, IX, X, XI, XII, C3 and C4 roots, Jacobson nerve |
| Occipital artery | C1 and C2 roots |

**TABLE 5. Summary of the various embolic agents used during endovascular treatment of central nervous system and head and neck tumors[a]**

| Agent | Specific material | Advantages | Disadvantages |
|---|---|---|---|
| Liquid | Ethanol<br>NBCA<br>Onyx<br>Hydrogels | Can penetrate into the capillary bed of the tumor | Can cause angionecrosis<br>Injure normal structures (i.e., cranial nerves)<br>Cytotoxic edema<br>Requires changing of microcatheter for each pedicle |
| Particulate | PVA<br>Gelfoam<br>Microfibrillar collagen | Less likely to injure normal structures with increased particle size | Less likely to penetrate capillary bed<br>Not felt to be permanent<br>Necrosis/edema possible |
| Microspheres | Gelatin<br>Dextran<br>Poly (D, L lactide/ glycolide) copolymer | Precise control<br>Less likely to injure normal structures | Often resorbable<br>Temporary effects |
| Coils | Fibered<br>Detachable (i.e., GDC) | Used in conjunction with particles to reduce rate of recanalization | Temporarily reduces blood supply to tumor<br>No effect at capillary level |

[a] NBCA, *n*-butylcyanoacrylate; PVA, polyvinyl alcohol; GDC, Guglielmi detachable coils.

**TABLE 6. Typical blood supply to meningiomas based on location[a] (9)**

| Location meningioma | Typical blood supply |
|---|---|
| Parasagittal/falx | MMA, contralateral MMA, anterior ethmoidal |
| Olfactory groove | Anterior/posterior ehtmoidal |
| Sphenoid wing | Sphenoidal branches MMA |
| Parasellar | ICA branches, MMA, artery of the foramen rotundum |
| Tentorial | Marginal tentorial artery, basal tentorial artery |
| Posterior fossa | MMA, occipital artery, ascending pharyngeal artery |

[a] MMA, middle meningeal artery; ICA, internal carotid artery.

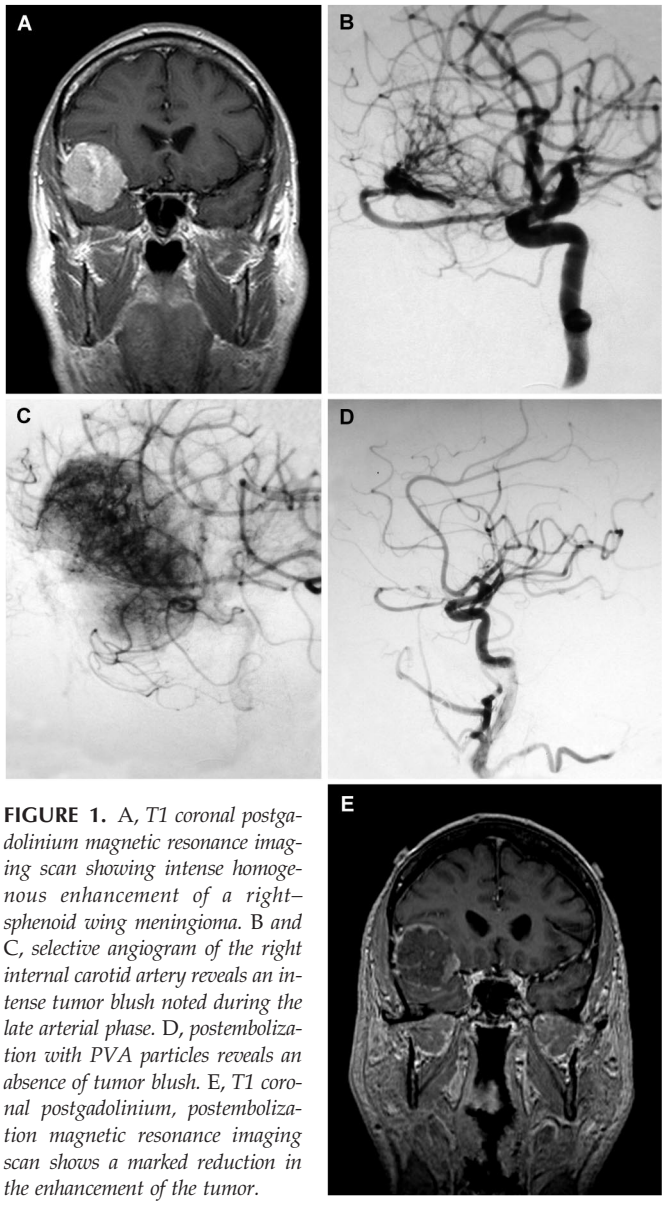


**FIGURE 1.** A, *T1 coronal postgadolinium magnetic resonance imaging scan showing intense homogenous enhancement of a right–sphenoid wing meningioma.* B *and* C, *selective angiogram of the right internal carotid artery reveals an intense tumor blush noted during the late arterial phase.* D, *postembolization with PVA particles reveals an absence of tumor blush.* E, *T1 coronal postgadolinium, postembolization magnetic resonance imaging scan shows a marked reduction in the enhancement of the tumor.*

crocatheters aids with planning the size and type of materials that will be used during the procedure. The purpose of this technique is to lay out the dangerous anastomoses that can occur, especially from the ECA circulation. Some of the complications reported in the literature occur from the opening of these anastomoses during embolization. If a dangerous anastomosis is identified, the catheter is repositioned or the anastomosis is occluded using a microcoil. Provocative testing is performed as described before with lidocaine to identify blood supply to cranial nerves. If neurological changes occur with lidocaine testing, the catheter is repositioned and the test is repeated. The other option would be to use particles larger in diameter than the vasa nervorum, which would be at least 300 $\mu$m.

Particulate material such as PVA and Trisacryl gelatin microspheres are commonly used for embolization (81). The permanence of liquid agents, such as alcohol and cyanoacrylate, is not needed because most of these lesions are resected after embolization. Moreover, liquid agents are riskier because they can cross the anastomotic channels and can cause damage of important neural structures. Other agents that have been used include fibrin glue, lyophilized dura Gelfoam particles, and *n*-butylcyanoacrylate (47, 62). Embolization is done slowly, and vigorous embolization is avoided to prevent reflux of embolic material into normal proximal branches. The pial supply is generally not embolized because of the higher risk of stroke.

This emphasizes the importance of using smaller particles, but care must be taken because this also increases the risk of the procedure. A risk of using particles less than 150 $\mu$m in diameter is injuring cranial nerves via the vasa nervorum (43). Embolization procedures should be performed distally to these branches if smaller particles are to be used. Larger particles can be used if the catheter is proximal to branches supplying normal structures.

Large centers report low complication rates with embolization of meningiomas. Berenstein et al. (7) reported that three patients out of 185 (1.6%) developed permanent neurological deficits as a result of embolization, whereas five (2.7%) had transient neurological events. Our experience at the University of Pittsburgh combined with the University of Texas Southwestern Medical Center has shown that four patients out of 111 (3.6%) developed cranial nerve injury or monocular blindness as a result of embolization. The two patients who developed blindness were embolized with 50- to 150-$\mu$m PVA particles despite passing provocative testing with lidocaine.

A technique using temporary balloon occlusion has been used for branches of the ICA that may feed the tumor. The authors inflated a balloon distal to the tumor's arterial feeders

and then injected PVA particles, thereby occluding the arterial feeders to the tumor (80). As noted earlier, this technique is controversial and requires further study.

Embolization of meningiomas is feasible and can be done safely at an experienced center, although the risk of this procedure may elevate with the use of smaller particles (81). Preliminary data from case series and single-center randomized trials seem to favor the use of this method for larger tumors, although larger clinical trials are required to delineate the precise role of embolization.

## PARAGANGLIOMAS

Paragangliomas are derived from neuroendocrine cells and are a rare entity in the head and neck region. The most common location in the head and neck region for these tumors is along the temporal bone involving the tympanic nerve or jugular fossa, followed by the carotid bifurcation and vagus nerve (4, 74). Angiographically, the typical appearance involves the "splaying" of the carotid bifurcation, with an intense blush denoting its hypervascularity (39). Clinical presentation for these tumors varies based on location. *Table 7* outlines the names given to paragangliomas based on location along with typical clinical presentations. These tumors can be multicentric, although this is more common in patients with a positive family history (21, 26, 61). Paragangliomas rarely secrete catecholamines, especially when they are located at the carotid bifurcation, although patients with labile hypertension should arouse clinical suspicion (63, 66).

It has generally been accepted that definitive treatment for these tumors involves surgical excision. A recent review of the case series presented between 1992 and 1995 of 178 patients undergoing surgical excision of carotid body tumors showed a 0% mortality, 2.2% rate of stroke, and 22% rate of cranial nerve palsies (54). This was in contrast to earlier reports from the 1940s, which had shown a 40% morbidity from excision (52). Preoperative arterial embolization has been used for select cases of head and neck paragangliomas. Because these tumors are rare, it is difficult to study the potential benefits of embolization. Currently, reports from experienced centers have helped in the identification of patients who may potentially benefit from embolization (60).

Preoperative angiography is considered vital by many surgeons because it can aid in determining the arterial supply to the tumor based on vessels that have been displaced (36). In addition, the circle of Willis can help determine how safe it is to temporarily occlude the carotid artery during surgery (60). Once arteriography has been performed, a decision must be made as to the benefits and risks of arterial embolization. These surgeries can often result in significant blood loss of up to 2 L (28). Tikkakoski et al. (78) compared blood loss between patients embolized before surgical excision with that of nonembolized patients and showed a significantly lower amount of blood loss in the embolization group (588 versus 1374 ml, $P < 0.04$) (78). Much of this decision rests on the experience of the interventionalist, along with location and size of the tumor.

It is generally felt that larger tumors will likely benefit from embolization (33, 40), although a cutoff value for size has never been established. Arterial supply to these tumors often involves branches from the ECA and ICA, but almost all patients have some supply from the ascending pharyngeal arteries (60). *Figure 2* shows an example of a typical carotid body tumor with supply from the ascending pharyngeal artery. Different techniques for embolization have been described in the literature. Many advocate catheter placement in the distal part of the feeding vessel with fluoroscopy-guided administration of embolic particles such as PVA, Gelfoam, or cyanoacrylate. Administration should occur with continuous fluoroscopy, and care should be taken to avoid reflux of particles into the normal circulation (33). Proximal embolization of feeding vessels often does not help with reduction of blood loss because ICA collaterals can develop around the tumor. In addition, access to further embolization procedures is lost (60). The capillary bed to these tumors is roughly 200 $\mu$m, and thus 150- to 250-$\mu$m particles are usually appropriate (7). Catheter-directed embolization can be performed safely, as shown in a series of 47 patients from New York University. Only one patient had a permanent sequela (facial nerve palsy after the procedure); three patients had cranial nerve palsies but also had tumor encapsulating those nerves, one patient had an asymptomatic dissection, and one patient had a transient hemiparesis (60). Surgical excision should occur within 1 week after embolization to ensure that the new collaterals do not form in the interim.

When a large number of branches come off of the ICA and feed the tumor, consideration can be given to a balloon test occlusion. At our institution, we use xenon computed tomography as a physiological test (77) to determine patient tolerance to a balloon test occlusion. If a patient tolerates the

**TABLE 7. Clinical symptoms associated with paragangliomas (17, 18)**

| Location | Tumor name | Clinical symptoms |
|---|---|---|
| Middle ear | Tympanic paraganglioma | Hearing loss, tinnitus, discharge, VIIth nerve palsy |
| Vagus nerve | Vagal paraganglioma | Neck mass, painful, involvement of Xth and XIIth cranial nerves |
| Carotid bifurcation | Carotid body tumor | Neck mass, painless, rare hoarseness or hemiatrophy of tongue (XIIth) |
| Jugular bulb | Glomus jugularae | Involvement of the XIth and XIIth cranial nerves |
| Adrenal gland | Pheochromocytoma | Labile hypertension |

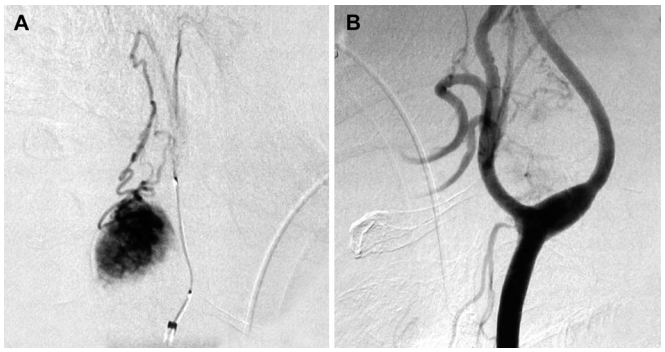


**FIGURE 2.** A, *selective microcatheter injection of the right ascending pharyngeal artery revealing a hypervacular blush to a carotid body tumor.* B, *postembolization angiography of the right common carotid artery showing splaying of the bifurcation, with an absence of hypervascularity. The patient underwent successful resection of the tumor.*

occlusion, consideration can be given to sacrificing the ICA, although some have recently reported the use of grafted stents to maintain ICA patency while excluding arterial feeders to the tumor (12). Others have reported success with direct percutaneous injection of cyanoacrylate or alcohol into these hypervascular tumors, with successful devascularization of the tumor, but without significant complications (9).

Paragangliomas prove to be challenging tumors to treat and require a team approach towards the goal of successful removal. Endovascular approaches can be used to help reduce blood loss through devascularization of the tumor bed.

## HEMANGIOPERICYTOMA

Hemangiopericytoma is a rare tumor of the CNS that accounts for roughly 1% of all intracranial tumors and 2 to 4% of meningiomas (24, 27). These tumors often present clinically and have radiographic features that are similar to those of meningiomas. On magnetic resonance imaging scans, hemangiopericytomas are less likely to have associated calcifications and may have a tendency to uptake contrast in a heterogenous pattern (13, 58). Based on a small number of case series in the literature, it is felt that these tumors have a higher frequency of recurrence in comparison with meningiomas (27). Currently, most authors recommend a radical surgical excision of these tumors (3).

Angiographically, these tumors are typically supplied by branches coming off the ICA and vertebrobasilar circulation and, occasionally, branches from the ECA. The classic feature to the tumor is an intense tumor blush followed by a long-lasting venous phase, along with corkscrew-type vessels within the tumor itself (2, 49).

These tumors are highly vascular and associated with large amounts of blood loss intraoperatively. Earlier reports note intraoperative mortality attributable to blood loss from surgical resection (34). Presurgical embolization has been performed in a limited number of cases in the literature. In addition, direct tumor embolization through percutaneous entry has also been described (23).

## JUVENILE NASOPHARYNGEAL ANGIOFIBROMA

Juvenile nasopharyngeal angiofibroma (JNA) is a rare tumor of adolescence that most commonly affects males (55), although there are rare reports involving females (30). The typical clinical presentation for patients presenting with a JNA is unilateral nasal obstruction with epistaxis. These masses are highly vascular and are associated with arteriovenous fistulae that generate increased feeding artery pressures (73) and may extend intracranially in 10 to 20% of cases (35). JNAs can be staged according to the Fisch classification system (18) (*Table 8*), and surgical excision has been advocated by many authors (59). Despite removal of these tumors, recurrence rates have been reported between 20 and 40% (17, 65). Recurrence rates are most likely related to initial staging of the tumor (45).

The role of preoperative embolization for these vascular tumors has been debated in the literature (51, 53). As with other vascular tumor surgeries, presurgical embolization seems to reduce blood loss during surgery (45, 48, 72). In addition, it may be useful in larger tumors and those extending intracranially because devascularization can help shrink the tumor and make surgical excision easier. The majority of the blood supply comes from the internal maxillary artery, sphenopalatine artery, ascending pharyngeal artery, and smaller branches off the carotid artery (41). Angiography of both carotid systems is necessary because supply can be bilateral in 30% of patients and from branches of the ICA in 30% of patients (59). *Figure 3* shows the typical radiographic features of a JNA tumor pre- and postembolization.

Complication rates from these procedures reported in the literature seem to be low at experienced centers (42, 48). Many of the complications reported in the literature are attributable to poor recognition of dangerous anastomoses, inappropriate embolic material, and size or reflux of particles (59). There has been concern that embolization may be a risk factor for recur-

**TABLE 8. Fisch classification for staging of juvenile nasopharyngeal angiofibromas (18)**

| Class | Location |
|---|---|
| I | Mass in nasopharynx and nasal cavity without bony disruption |
| II | Invasion of the maxillary, ethmoid, and sphenoid sinus |
| III | Invasion of the pterygo–palatine fossa, intratemporal fossa, orbit, and parasellar region |
| IV | Invasion of the cavernous sinus or optic chiasm or pituitary fossa |

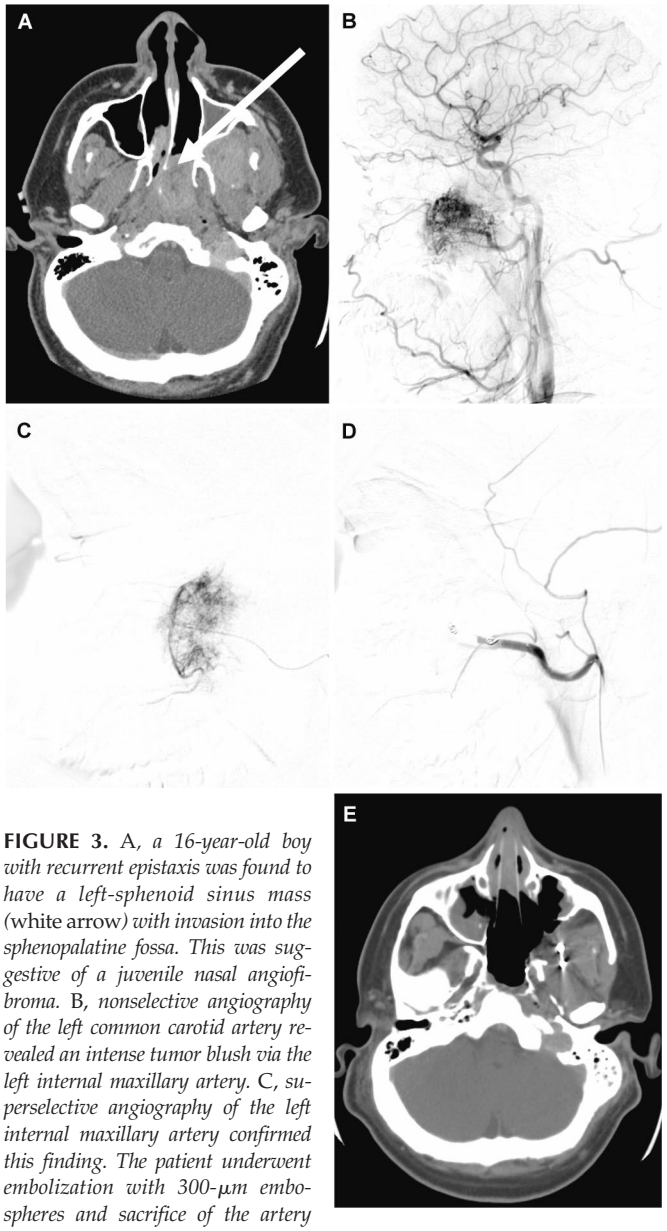


**FIGURE 3.** A, *a 16-year-old boy with recurrent epistaxis was found to have a left-sphenoid sinus mass* (white arrow) *with invasion into the sphenopalatine fossa. This was suggestive of a juvenile nasal angiofibroma.* B, *nonselective angiography of the left common carotid artery revealed an intense tumor blush via the left internal maxillary artery.* C, *superselective angiography of the left internal maxillary artery confirmed this finding. The patient underwent embolization with 300-μm embospheres and sacrifice of the artery with fibered coils.* D, *postembolization reveals an absence of tumor blush.* E, *computed tomographic scan after surgical removal of the tumor reveals resection of the mass and the nasal septum.*

rence of tumor, although this was from a small series of 33 patients (51).

As with other embolization procedures, if significant blood supply occurs from the ICA, some have used balloon inflation distally to the branches, but proximally to the ophthalmic artery. Particles are then released into the ICA under continuous fluoroscopy to watch for reduction of tumor blush via these ICA branches. Saline can be infused after ceasing embolization to dilute the particles before deflation of the balloon (20). In addition, direct puncture of the tumor can be performed with an 18-gauge needle followed by injection of cyanoacrylate. This can be used in patients with prior proximal ligations within the ECA, thereby restricting endovascular access to feeding arteries; it can also be used in patients with a large supply from ICA branches (79). There are few reports of this technique, and further study is required to assess safety and efficacy.

As with the other tumors discussed thus far, no multicenter large clinical trial data are available to help define the role of embolization of JNAs. These tumors can be safely embolized, although the single-center report of embolization procedures leading to an increased rate of recurrence (51) is concerning.

## SQUAMOUS CELL CARCINOMA

SCC is the most common tumor of the head and neck region and is most commonly treated with a combination of surgical resection and radiation therapy. Unfortunately, long-term survival is poor for patients with advanced disease (15). Surgery can often be disfiguring, with associated difficulties in swallowing and chewing secondary to resection of the oral mucosa (68). Cisplatin with or without 5-fluorouracil is used as the chemotherapy regimen for SCC, but it has toxic side effects with escalating doses (37). Intra-arterial administration of cisplatin has been used in limited centers as an alternative to systemic chemotherapy.

Intra-arterial administration of cisplatin into the feeding vessels of the tumor offers the advantage of reduced systemic toxicity along with the ability to give higher doses of the medication. A second problem with systemic doses of cisplatin is that resistance may develop after a few doses, thereby rendering this therapy ineffective (5). Giving higher doses may potentially offset resistance and help induce the tumorcidal effects of cisplatin (67).

These tumors are generally avascular; thus, embotherapy is difficult to perform. Infusion of chemotherapy via intra-arterial means has not gained widespread acceptance, although many reports have been published in the literature showing the potential benefits of this approach (7, 69). These tumors are fed by ECA branches and can be accessed via catheterization of the femoral artery. The catheter can be placed in the proximal ECA before determining which vessels supply the tumor. The catheter is then advanced into the feeding vessel, where infusions of chemotherapeutic agents can be given.

*Table 9* summarizes the long-term results from some of the recent studies performed with intra-arterial infusions. Historical comparison shows that patients with advanced head and neck cancer have poor long-term survival, ranging from 15 to 40% (50). Unfortunately, large-scale randomized controlled studies are lacking. The largest, a series of 385 patients, looked at complications associated with intra-arterial chemotherapy infusions and found 10.6% of patients had chemotoxic events, with the majority involving the mucosa, 5.7% groin hematomas, and 1.5% neurological events (22). The chemotoxic events

**TABLE 9. Summary of recent studies looking at long-term survival rates in patients with advanced head and neck cancer being treated with intra-arterial cisplatin**

| Series (ref. no.) | No. of patients | Adjuvant therapy | 3-yr survival | No. (%) of patients with stage T3/T4 |
|---|---|---|---|---|
| Kovacs, 2004 (38) | 52 | Surgery | 82% | 19 (37%) |
| Robbins et al. (68) | 25 | Surgery and radiation therapy | 54% (5 yr) | 20 (80%) |
| Balm et al., 2004 (6) | 79 | Radiation | 43% | 79 (100%) |
| Homma et al., 2005 (31) | 53 | Radiation | 54% | 53 (100%) |

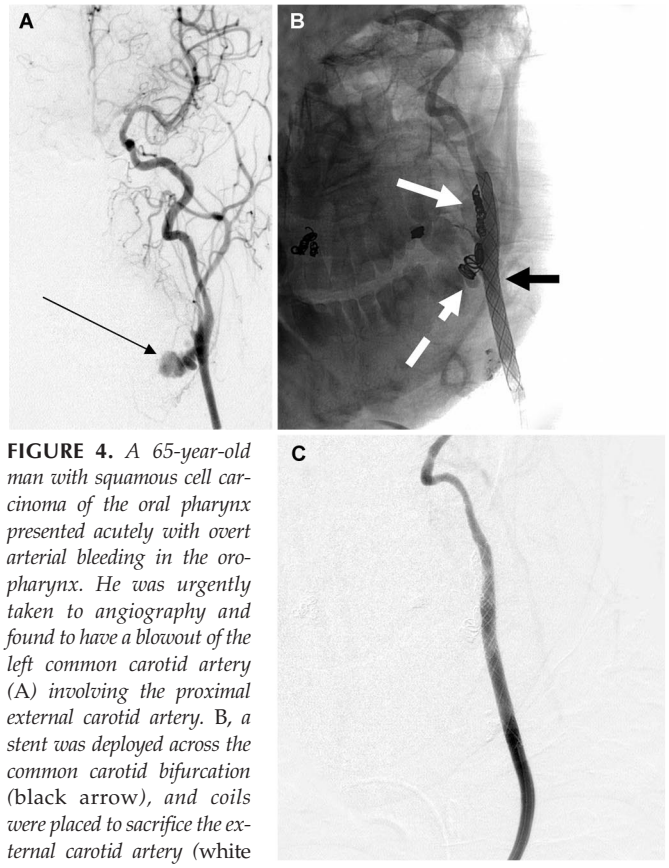


**FIGURE 4.** *A 65-year-old man with squamous cell carcinoma of the oral pharynx presented acutely with overt arterial bleeding in the oropharynx. He was urgently taken to angiography and found to have a blowout of the left common carotid artery (*A*) involving the proximal external carotid artery.* B, *a stent was deployed across the common carotid bifurcation (*black arrow*), and coils were placed to sacrifice the external carotid artery (*white arrow*) and into a pseudoaneurysmal pouch (*white dashed arrow*). A Wallgraft (Gortex-covered stent) was then placed across the common bifurcation to exclude the tumor-encased arterial segment from the arterial circulation.* C, *poststent and coil placement, the extravasation ceased.*

reported were lower then those noted with intravenous administration of cisplatin (8).

Intra-arterial infusions of chemotherapy seem to be safe and effective relative to historical controls. Results of these studies may help guide future study of intra-arterial therapy for intracranial tumors. Future randomized control studies will hopefully help to clarify the indications and dosages for this therapy.

## CAROTID BLOWOUT SYNDROME

Carotid blowouts typically occur in the extracranial segment of the carotid artery as a result of invasion of malignant head and neck carcinomas into the carotid artery. The mortality rate for blowouts is 40% because of extravasation (10). The typical presentation is copious, pulsating bleeding from the oropharynx.

This condition can be managed through endovascular methods. In patients with an intact circle of Willis who are able to tolerate a balloon test occlusion of the ipsilateral artery, complete sacrifice of the carotid artery is an option. This can be performed with detachable coils or detachable balloons with low morbidity (11). Patients deemed to be at a high risk of stroke based on a failed balloon test occlusion can potentially be treated with stents covering the injured segment of artery (*Fig. 4*). The placement of such stents is feasible and can stop extravasation (44), but this is not a long-term solution. In the longer term, the artery continues to deteriorate, and the stents can extrude or become occluded over time, thus causing neurological morbidity (76, 82). Additionally, patients are placed on antiplatelet therapy to maintain the patency of the stent, which may not be optimal in this group of patients.

## CNS LYMPHOMA/GLIOMAS

Current therapies for malignant gliomas and lymphomas involve combination therapy using radiation, chemotherapy, and palliative surgery. Survival rates have been dismal despite such aggressive therapies, and newer therapeutic interventions have been studied using BBB disruption in combination with intra-arterial chemotherapy infusions.

This technique involves placing patients under general anesthesia. Selective catheterization of the target vessel ipsilateral to the tumor (i.e., ICA or vertebral artery) is performed. After injections of contrast material are performed, a determination is made as to the rate of infusion of mannitol based on the lowest infusion rate to achieve slight retrograde arterial filling from the catheter. At this point, 25% mannitol is infused for 30 seconds into the catheter at the rate determined necessary to disrupt the BBB. After 15 to 20 minutes, infusion of the chemotherapeutic agent is initiated (57). Methotrexate is used for lymphomas, and carboplatin regimens are used for gliomas. Such protocols have been shown to be safe in Phase II studies enrolling more than 6000 patients and can be replicated across specialized centers (16).

A Phase III randomized trial comparing the infusion of intra-arterial carmustine versus intravenous delivery postresection of malignant gliomas showed no difference between

the two groups. Moreover, the intra-arterial group had significant side effects including white matter changes and encephalopathy (75). Currently, a Phase III study is underway to determine the effectiveness of combing BBB disruption to intra-arterial delivery of chemotherapeutic agents in the hopes of extending life expectancies for these malignant tumors (19).

## CONCLUSION

We have summarized some of the therapies used by neurointerventionalists in the treatment of intracranial and head and neck cancers. Small case series have shown that there may be a role for these therapies in the future. Large multicenter randomized clinical trials are required to answer vital questions as to the risks and benefits of these therapeutic interventions.

## REFERENCES


1. Adegbite AB, Kahn MI, Paine KW, Tan LK: The recurrence of intracranial meningiomas after surgical treatment. **J Neurosurg** 58:51–56, 1983.
2. Akiyama M, Sakai H, Onoue H, Miyazaki Y, Abe T: Imaging intracranial haemangiopericytomas: Study of seven cases. **Neuroradiology** 46:194–197, 2004.
3. Alen JF, Lobato RD, Gomez PA, Boto GR, Lagares A, Ramos A, Ricoy JR: Intracranial hemangiopericytoma: Study of 12 cases. **Acta Neurochir (Wien)** 143:575–586, 2001.
4. Alford BR, Guilford FR: A comprehensive study of tumors of the glomus jugulare. **Laryngoscope** 72:765–805, 1962.
5. Andrews PA, Jones JA, Varkl NM, Howell SB: Rapid emergence of acquired cis-diamminadichloroplatinum (II) resistance in an in vivo model or human ovarian carcinoma. **Cancer Commun** 2:93–100, 1990.
6. Balm AJ, Rasch CR, Schornagel JH, Hilgers FJ, Keus RB, Schultze-Kool L, Ackerstaff AH, Busschers W, Tan IB: High-dose superselective intra-arterial cisplatin and concomitant radiation (RADPLAT) for advanced head and neck cancer. **Head Neck** 26:485–493, 2004.
7. Berenstein A, Lasjaunias P, ter Brugge KG: *Surgical Neuroangiography.* Berlin, Springer-Verlag, 2004, ed 2.
8. Brizel DM, Albers ME, Fisher SR, Scher RL, Richtsmeier WJ, Hars V, George SL, Huang AT, Prosnitz LR: Hyperfractionated irradiation with or without concurrent chemotherapy for locally advance head and neck cancer. **N Engl J Med** 338:1798–1804, 1998.
9. Chaloupka JC, Mangla S, Huddle DC, Roth TC, Mitra S, Ross DA, Sasa CT: Evolving experience with direct puncture therapeutic embolization for adjunctive and palliative management of head and neck hypervascular neoplasms. **Laryngoscope** 109:1864–1872, 1999.
10. Chaloupka JC, Putman CM, Citardi MJ, Ross DA, Sasaki CT: Endovascular therapy of the carotid blowout syndrome in head and neck surgical patients: Diagnostic and managerial considerations. **AJNR Am J Neuroradiol** 17:843–852, 1996.
11. Citardi MJ, Chaloupka JC, Son YH, Ariyan S, Sasaki CT: Management of carotid artery rupture by monitored endovascular therapeutic occlusion (1988–1994). **Laryngoscope** 105:1086–1092, 1995.
12. Cohen JE, Ferrario A, Ceratto R, Miranda C, Lylyk P: Covered stent as an innovative tool for tumor devascularization and endovascular arterial reconstruction. **Neurol Res** 25:169–172, 2003.
13. Cosentino CM, Poulton TB, Esguerra JV, Sands SF: Giant cranial hemangiopericytoma: MR and angiographic findings. **AJNR Am J Neuroradiol** 14:253–256, 1991.
14. Dean BL, Flom RA, Wallace RC, Khayata MH, Obuchowski NA, Hodak JA, Zabramski JM, Spetzler RF: Efficacy of endovascular treatment of meningiomas: Evaluation with matched samples. **AJNR Am J Neuroradiol** 15:1675–1680, 1994.
15. Dimery IK, Hong WK: Overview of combined modality therapies for head and neck cancer. **J Natl Cancer Inst** 85:95–111, 1993.
16. Doolittle ND, Miner ME, Hall WA, Siegal T, Jerome E, Osztie E, McAllister LD, Bubalo JS, Kraemer DF, Fortin D, Nixon R, Muldoon LL, Neuwelt EA: Safety and efficacy of a multicenter study using intraarterial chemotherapy in conjunction with osmotic opening of the blood-brain barrier for the treatment of patients with malignant brain tumors. **Cancer** 88:637–647, 2000.
17. Fagan JJ, Snyderman CH, Carrau RL, Janecka IP: Nasopharyngeal angiofibromas: Selecting a surgical approach. **Head Neck** 19:391–399, 1997.
18. Fisch U: The infratemporal fossa approach for nasopharyngeal tumors. **Laryngoscope** 93:36–44, 1983.
19. Fortin D: The blood-brain barrier should not be underestimated in neuron-oncology. **Rev Neurol** 160:523–532, 2004.
20. Garcia-Cervigon E, Bien S, Rufenacht D, Thurel C, Reizine D, Huy PB, Merland JJ: Pre-operative embolization of naso-pharyngeal angiofibromas. Report of 58 cases. **Neuroradiology** 30:556–560, 1988.
21. Gardner P, Dalsing M, Weisberger E, Weisberger E, Sawchuk A, Miyamoto R: Carotid body tumors, inheritance, and a high incidence of associated cervical paragangliomas. **Am J Surg** 172:196–199, 1996.
22. Gemmete JJ: Complications associated with selective high-dose intraarterial cisplatin and concomitant radiation therapy for advanced head and neck cancer. **J Vasc Intervent Radiol** 14:743–748, 2003.
23. George G, Casasco A, Deffrennes D, Houdart E: Intratumoral embolization of intracranial and extracranial tumors: Technical note. **Neurosurgery** 35: 771–774, 1994.
24. Goellner JR, Laws ER Jr, Soule EH, Okazaki H: Hemangiopericytoma of the meninges: Mayo Clinic experience. **Am J Clin Pathol** 70:375–380, 1978.
25. Grand C, Bank WO, Baleriaux D, Matos C, Dewitte O, Brotchi J, Delcour C: Gadolinium-enhanced MR in the evaluation of preoperative meningioma embolization. **AJNR Am J Neuroradiol** 14:563–569, 1993.
26. Grufferman S, Gillman MW, Pasternak LR, Peterson CL, Young WG Jr: Familial carotid body tumors: Case report and epidemiologic review. **Cancer** 46:2116–2122, 1980.
27. Guthrie BL, Ebersold MJ, Scheitauer BW, Shaw EG: Meningeal hemangiopericytoma: Histopathological features, treatment, and long-term follow-up of 44 cases. **Neurosurgery** 25:514–522, 1989.
28. Hallet JW, Nora JD, Hollier LH, Cherry KJ, Pairolero PC: Trends in neurovascular complications of surgical management for carotid body and cervical paragangliomas: A fifty-year experience with 153 tumors. **J Vasc Surg** 7:284–291, 1988.
29. Hekster RE, Matricali B, Luyendijk W: Presurgical transfemoral catheter embolization to reduce operative blood loss. Technical note. **J Neurosurg** 41:396–398, 1974.
30. Hiranandani LH, Melgiri RD, Juveker RV: Angiofibroma of the ethmoidal sinus in a female. **J Laryngol Otol** 81:935–939, 1967.
31. Homma A, Furuta Y, Suzuki F, Oridate N, Hatakeyama H, Nagahashi T, Ushikoshi S, Asano T, Nishioka T, Shirato H, Fukuda S: Rapid superselective high-dose cisplatin infusion with concomitant radiotherapy for advanced head and neck cancer. **Head Neck** 27:65–71, 2005.
32. Horowitz M, Whisnant RE, Jungreis C, Snyderman C, Levy EI, Kassam A: Temporary balloon occlusion and ethanol injection for preoperative embolization of carotid-body tumor. **Ear Nose Throat J** 81:536–538, 2002.
33. Iafrati MD, O'Donnell TF Jr: Adjuvant techniques for the management of large carotid body tumors. A case report and review. **Cardiovasc Surg** 7:139–145, 1999.
34. Jaaskelainen JJ, Servo A, Wahlstrom T, Haltia M: Diagnosis of intracranial haemangioperictomas with angiography and CT scanning. **Neuroradiology** 27:38–43, 1985.
35. Jafek BW, Nahum AN, Butler RM: Surgical treatment of juvenile nasopharyngeal angiofibroma. **Laryngoscope** 83:707–720, 1973.
36. Kafie FE, Freischlag JA: Carotid body tumors: The role of preoperative embolization. **Ann Vasc Surg** 15:237–242, 2001.
37. Kish JA, Ensley JF, Jacobs JR, Binns P, al-Sarraf M: Evaluation of high-dose cisplatin and 5-FU infusion as initial therapy in advanced head and neck cancer. **Am J Clin Oncol** 11:553–557, 1988.
38. Kovacs AF: Intra-arterial induction high-dose chemotherapy with cisplatin for oral and oropharyngeal cancer: Long-term results. **Br J Cancer** 90:1323–1328, 2004.
39. Lack EE, Cubilla AL, Woodruff JM, Farr HW: Paragangliomas of the head and neck region: A clinical study of 69 patients. **Cancer** 39:397–409, 1977.

40. LaMuraglia GM, Fabian RL, Brewster DC, Pile-Spellman J, Darling RC, Cambria RP, Abbott WM: The current surgical management of carotid body paragangliomas. **J Vasc Surg** 15:1038–1044, 1992.
41. Lasjaunias P: Nasopharyngeal angiofibromas: Hazards of embolization. **Radiology** 136:119–123, 1980.
42. Lasjaunias P, Picard L, Manelfe C, Moret J, Doyon D: Angiofibroma of the nasopharynx. A review of 53 cases treated by embolization. The role of pretherapeutic angiography. Pathophysiological hypotheses. **J Neuroradiol** 7:73–95, 1980.
43. Latchaw RE: Preoperative intracranial meningioma embolization: Technical considerations affecting the risk-to-benefit ratio. **AJNR Am J Neuroradiol** 14:583–586, 1993.
44. Lesley WS, Chaloupka JC, Weigele JB, Mangla S, Dogar MA: Preliminary experience with endovascular reconstruction for the management of carotid blowout syndrome. **AJNR Am J Neuroradiol** 24:975–981, 2003.
45. Liu L, Wang R, Huang D, Han D, Ferguson EJ, Shi H, Yang W: Analysis of intra-operative bleeding and recurrence of juvenile nasopharyngeal angiofibromas. **Clin Otolaryngol** 27:536–540, 2002.
46. Maity A, Pruitt AA, Judy KD, Phillips PC: Cancer of the central nervous system, in Abeloff MD (ed): *Clinical Oncology.* Philadelphia, Churchill-Livingston, 2004, pp 1374–1432, ed 3.
47. Manelfe C, Lasjaunias P, Ruscalleda J: Preoperative embolization of intracranial meningiomas. **AJNR Am J Neuroradiol** 7:963–972, 1986.
48. Mann W, Jecker P, Amedee RG: Juvenile angiofibroma: Changing surgical concept over the last 20 years. **Laryngoscope** 114:291–293, 2004.
49. Marc JA, Takei Y, Schechter MM, Hoffman JC: Intracranial hemangiopericytomas. Angiography, pathology, and differential diagnosis. **Radiology** 125:823–832, 1975.
50. Marcial VA, Pajek TF: Radiation therapy alone or in combination with surgery in head and neck cancer. **Cancer** 55 [Suppl 9]:2259–2265, 1985.
51. McCombe A, Lund VJ, Howard DJ: Recurrence in juvenile angiofibroma. **Rhinology** 28:97–102, 1990.
52. Monro RS: The natural history of carotid body tumours and their diagnosis and treatment; with a report of five cases. **Br J Surg** 37:445–453, 1950.
53. Moulin G, Chagnaud C, Gras R, Gueguen E, Dessi P, Gaubert JY, Bartoli JM, Zanaret M, Botti G, Cannoni M: Juvenile nasopharyngeal angiofibroma: Comparison of blood loss during removal of in embolized versus non-embolized group. **Cardiovasc Intervent Radiol** 18:158–161, 1995.
54. Muhm M, Polterauer P, Gstottner W, Temmel A, Richling B, Undt G, Niederle B, Staudacher M, Ehringer H: Diagnostic and therapeutic approaches to carotid body tumors. Review of 24 patients. **Arch Surg** 132:279–284, 1997.
55. Neel HB 3rd, Whicker JH, Devine KD: Juvenile angiofibroma. Review of 120 cases. **Am J Surg** 126:547–560, 1973.
56. Nelson PK, Setton A, Choi IS, Ransohoff J, Berenstein A: Current status of interventional neuroradiology in the management of meningiomas. **Neurosurg Clin N Am** 5:235–259, 1994.
57. Neuwelt EA, Goldman DL, Dahlborg SA, Crossen J, Ramsey F, Roman-Goldstein S, Braziel R, Dana B: Primary central nervous system lymphoma treated with osmotic blood-brain barrier disruption: Prolonged survival and preservation of cognitive function. **J Clin Oncol** 9:1580–1590, 1991.
58. Osborne DR, Dubois P, Drayer B, Sage M, Burger P, Heinz ER: Primary intracranial meningeal and spinal hemangiopericytoma: Radiological manifestations. **AJNR Am J Neuroradiol** 2:69–74, 1981.
59. Paris J, Guelfucci B, Moulin G, Zanaret M, Triglia JM: Diagnosis and treatment of juvenile nasopharyngeal angiofibroma. **Eur Arch Otorhinolaryngol** 258:120–124, 2001.
60. Persky MS, Setton A, Niimi Y, Hartman J, Frank D, Berenstein A: Combined endovascular and surgical treatment of head and neck paragangliomas—A team approach. **Head Neck** 24:423–431, 2002.
61. Pratt LW: Familial carotid body tumors. **Arch Otol** 97:334–336, 1973.
62. Probst EN, Gryzyska U, Westphal M, Zeumer H: Preoperative embolization of intracranial meningiomas with a fibrin glue preparation. **AJNR Am J Neuroradiol** 20:1695–1702, 1999.
63. Pryse-Davies J, Dawson IMP, Wesbury G: Some morphologic, histochemical and chemical observations on chemodectomas and the normal carotid body, including a study of the chromaffin reaction and possible ganglion cell elements. **Cancer** 17:185–202, 1964.
64. Quon H, Hershock D, Feldman M, Sewell D, Weber RS: Cancer of the head and neck, in Abeloff MD (ed): *Clinical Oncology.* Philadelphia, Churchill-Livingston, 2004, pp 1497–1560, ed 3.
65. Radkowski D, McGill T, Healey GB, Ohlms L, Jones DT: Angiofibroma. Changes in staging and treatment. **Arch Otolaryngol Head Neck Surg** 122:122–129, 1996.
66. ReMine WH, Weiland LH, ReMine SG: Carotid body tumors: Chemodectomas. **Curr Prob Cancer** 2:3–26, 1978.
67. Robbins KT, Kumar P, Regine WF, Wong FH, Weir AB 3rd, Flick P, Kun LE, Palmer R, Murry T, Fontanesi J, Ferguson R, Thomas R, Hartsell W, Paig CU, Salazar G, Norfleet L, Hanchett CB, Harrington V, Niell HB: Efficacy of targeted supradose cisplatin and concomitant radiation therapy for advanced head and neck cancer: The Memphis experience. **Int J Radiat Oncol Phys** 38:263–271, 1997.
68. Robbins KT, Samant S, Vieira F, Kumar P: Presurgical cytoreduction of oral cancer using intra-arterial cisplatin and limited concomitant radiation therapy (Neo-RADPLAT). **Arch Otolaryngol Head Neck Surg** 130:28–32, 2004.
69. Robbins KT, Storniolo AM, Kerber CW, Seagren S, Berson A, Howell SB: Rapid superselective high dose cisplatin infusion for advanced head and neck malignancies. **Head Neck** 14:364–371, 1992.
70. Russell EJ: Functional angiography of the head and neck. **AJNR Am J Neuroradiol** 7:927–936, 1986.
71. Russell DS, Rubinstein LJ: *Pathology of Tumors of the Nervous System.* Baltimore, Williams & Wilkins, 1989, ed 5.
72. Scholtz AW, Appenroth E, Kammen-Jolly K, Scholtz LU, Thumfart WF: Juvenile nasopharyngeal angiofibroma: Management and therapy. **Laryngoscope** 111:681–687, 2001.
73. Schroth G, Haldemann AR, Mariani L, Remonda L, Raveh J: Preoperative embolization of paragangliomas and angiofibromas. Measurement of intratumoral arteriovenous shunts. **Arch Otolaryngol Head Neck Surg** 122:1320–1325, 1996.
74. Shamblin WR, ReMine WH, Sheps SG, Harrison EG Jr: Carotid body tumor (chemodectoma). Clinicopathologic analysis of ninety cases. **Am J Surg** 122:732–739, 1971.
75. Shapiro WR, Green SB, Burger PC, Selker RG, VanGilder JC, Robertson JT, Mealey J Jr, Ransohff J, Mahaley MS Jr: A randomized comparison of intra-arterial versus intravenous BCNU, with or without intravenous 5-fluorouracil for newly diagnosed patients with malignant glioma. **J Neurosurg** 76:772–781, 1992.
76. Simental A, Johnson JT, Horowitz M: Delayed complications of endovascular stenting for carotid blowout. **Am J Otolaryngol** 24:417–419, 2003.
77. Steed DL, Webster MS, DeVries EJ, Jungreis CA, Horton JA, Sehkar L, Yonas H: Clinical observations on the effect of carotid artery occlusion on the cerebral blood flow mapped by xenon computed tomography and its correlation with carotid artery back pressure. **J Vasc Surg** 11:38–43, 1990.
78. Tikkakoski T, Luotonen J, Leinonen S, Siniluoto T, Heikkila O, Paivansalo M, Hyrynkangas K: Preoperative embolization in the management of neck paragangliomas. **Laryngoscope** 107:821–826, 1997.
79. Tranbahuy P, Borsik M, Herman P, Wassef M, Casasco A: Direct intratumoral embolization of juvenile angiofibroma. **Am J Otolaryngol** 15:429–435, 1994.
80. Tymianski M, Willinsky RA, Tator CH, Mikulis D, TerBrugge KG, Markson L: Embolization with temporary balloon occlusion of the internal carotid artery and in vivo proton spectroscopy improves radical removal of petrous-tentorial meningioma. **Neurosurgery** 35:974–977, 1994.
81. Wakhloo AK, Juengling FD, Van Velthoven VV, Schumacher M, Hennig J, Schwechheimer K: Extended preoperative polyvinyl alcohol microembolization of intracranial meningiomas: Assessment of two embolization techniques. **AJNR Am J Neuroradiol** 14:571–582, 1993.
82. Warren FM, Cohen JI, Nesbit GM, Barnwell SL, Wax MK, Andersen PE: Management of carotid "blowout" with endovascular stent grafts. **Laryngoscope** 112:428–433, 2002.


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