

**NEURORADIOLOGY OF POSTERIOR
CRANIAL BASE MASSES: DECISION MAKING**

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In the evaluation of posterior skull base masses, many decisions are predicated upon neuroradiologic procedures. When evaluating a skull base mass the following questions must be addressed:

- 1) Will computed tomography (CT) or Magnetic Resonance (MR) be more helpful in evaluating the lesion? Will both studies be needed?
- 2) Is surgery indicated based on clinical and radiologic findings?
- 3) What surgical approach will be optimal?
- 4) Is pre-operative test occlusion of the carotid artery indicated?
- 5) Is pre-operative embolization indicated?
- 6) How successful was the resection based on post-operative clinical and imaging findings?

We will touch briefly on each of these questions in the following discussion.

CHOOSING IMAGING TECHNIQUES

Cross sectional imaging is essential in all phases of skull base surgery; diagnosis, surgical planning, assessment of complications, and long term follow up. It's role is so universal that we often forget that it has been widely available only for about a decade. Computed tomography (CT) with bone algorithms replaced pluridirectional tomography in the early 1980's and magnetic resonance (MR) imaging has been widely available only since the mid-80's. MR has continued to improve, first with the introduction of MR contrast agents and more recently with the development of MR angiography and tissue specific pulsing sequences, such as fat suppression imaging.

The first neuroradiologic decision in approaching a patient with a skull base mass is which imaging technology to utilize. The advantages of MR are superior soft tissue contrast, the ease of multi-planar imaging, the lack of bone artifact, and the excellent demonstration of the relationships of skull base tumors to the carotid artery and the jugular vein. CT does retain several advantages such as superior demonstration of cortical bone and the ability to differentiate bone from the air filled spaces of the temporal bone. This differentiation of bone from air is critical in the evaluation of the carotid canal, the jugular fossa, the bony labyrinth, sinuses, middle ear, and the facial nerve canal.

Angiography is no longer an important tool in differential diagnosis. It remains useful in surgical planning. The display of the entire arterial or venous system on a single image is appealing. Improvements in MR angiography have reached the stage that they will probably

replace angiography in the demonstration of the great vessels. Many predict that even the detailed small vessel anatomy of vascular skull base lesions such as paragangliomas will be adequately demonstrated by MR angiography in the near future. The role of conventional angiography is restricted to preoperative testing and embolization.

INDICATIONS FOR SURGERY: POTENTIAL PITFALLS

The specific diagnosis of skull base lesions is usually made by biopsy. The role of imaging in most cases is to precisely define the extent of the lesion so that complete surgical removal can be achieved. However sometimes imaging will show a lesion for which surgery is not indicated. Examples include vascular abnormalities of the middle ear such as aberrant carotid artery, dehiscent jugular bulb, and aneurysm of the petrous carotid artery. In the evaluation of potentially vascular masses of the middle ear CT is the dominate modality. Aberrant carotid artery is excluded by seeing the thin plate of bone which separates the petrous carotid artery from the middle ear cavity. On MR this plate of bone may be invisible because it has the same signal as the flowing blood in the carotid artery itself and of the air in the middle ear cavity. For similar reasons CT is the best way to detect dehiscence of the jugular bulb.

Petrous carotid aneurysms are rare but can mimic skull base neoplasms particularly if they are partially thrombosed. Inadvertent or inappropriate surgical intervention can be disastrous. CT is essential in these lesions to show the remodeling of the carotid canal. It can also reveal calcification in the walls of aneurysms. MR may be helpful by showing the luminal flow void or blood products within mural thrombus.

Another pitfall to avoid in MR imaging is confusion between jugular thrombosis (not a surgical lesion) and a small glomus jugulare tumor. We've encountered MR scans showing enhancing thrombus within the jugular fossa which looks like a glomus jugulare tumor except that it lacks "salt and pepper" appearance created by the flow voids within this very vascular lesion. However these flow voids may be absent in small paragangliomas. CT is able to make the distinction in ambiguous cases because it will show erosion of the lateral wall of the jugular fossa in glomus tumors. Such erosion will be absent in jugular thrombosis.

Fibrous dysplasia is usually a self limited disease for which surgery is not indicated. On MR scans fibrous dysplasia can mimic an aggressive skull base neoplasm. Areas of fibrous dysplasia are usually intermediate in signal on T1 weighted images and low signal

on T2 weighted images. The metabolically active bone within the lesion enhances following intravenous contrast. The enhancement is striking on MR because of the lack of signal from the bone. To the unwary, the MR scans of fibrous dysplasia may look like a chordoma or chondrosarcoma. One clue is that almost all chordomas and chondrosarcomas are high signal on the T2 weighted images. CT is more definitive than MR in the diagnosis of fibrous dysplasia since it shows the classic "ground glass" appearance of this lesion. Rarely surgery is indicated in fibrous dysplasia when there is severe vascular or neural compression caused by the dysplastic bone.

PLANNING THE SURGICAL APPROACH

Generally the operative approach to a posterior skull base mass depends on the location and extent of the lesion as determined by imaging and the skill training of the operating surgeon. Occasionally imaging findings will alter surgical approach by allowing a specific diagnosis to be made preoperatively. One such situation involves cholesterol granulomas of the petrous apex. On CT, there may be confusion between an epidermoid tumor of the petrous apex and a cholesterol granuloma. Epidermoid tumors must be completely resected whereas cholesterol granulomas may be treated with a simpler drainage operation. The two may have similar appearances on CT although cholesterol granuloma is usually more expansile, like a mucocele. The definitive test in this case is MR. A cholesterol granuloma will be high signal on both T1 and T2 weighted images whereas most epidermoids are low signal on T1 weighted images.

Imaging is also helpful in differentiating eighth nerve schwannomas from meningiomas of the cerebellopontine angle cistern. The latter are usually more broad based along the petrous bone, rarely undergo cavitation, and less often extend into the internal auditory canal. Meningiomas are usually removed by a retrosigmoid approach whereas eighth nerve schwannomas may be removed from either a translabyrinthine or a retrosigmoid approach.

PRE-OPERATIVE TESTING OF THE GREAT VESSELS

Posterior cranial base masses frequently involve the high cervical or petrous segment of the carotid artery. If the carotid is to be resected the consequences of such a carotid sacrifice must be assessed. Pre-operative balloon test occlusion of the carotid artery has become popular in many centers. Although 80% of individuals can tolerate sacrifice of the carotid artery 15 to 20% of individuals will have a stroke. The cause of the stroke may be insufficient collateral circulation,

embolization, thrombosis due to low flow, or a combination of these. Balloon test occlusion of the carotid artery is able to identify those individuals with insufficient collateral supply.

In our experience patients who have no neurologic deficit from temporary occlusion of the carotid and who show no significant asymmetric drop in measured cerebral blood flow during balloon test occlusion are at low risk from sacrifice of the carotid artery. Test occlusion of the vertebral artery is more rarely performed. We have performed so few test occlusions of the vertebral artery that we cannot comment on its predictive value.

Test occlusion of the jugular vein has also only rarely been performed. Many skull base lesions which involve the jugular vein occlude it gradually as they enlarge and before the patient comes to surgical attention. A more difficult situation arises when the vein is still patent but occlusion is a necessary or desired result of surgical intervention.

Frequently the decision that the patient can tolerate jugular sacrifice is based on "eyeballing" the venous sinuses and subjectively deciding if the contralateral jugular vein will be able to handle the added flow. We have attempted test occlusion of the jugular vein with limited success. One patient who tolerated temporary occlusion with a single balloon developed a hemorrhagic stroke following permanent occlusion with two balloons. This may have occurred because we isolated a segment of venous sinus and its tributaries with the two permanent balloons. The single balloon, if not inflated directly over the entry point of tributary veins would permit proximal or distal communication with the rest of the venous system.

EMBOLIZATION

The decision whether or not to embolize a lesion rests with both the neuroradiologist and the neurosurgeon. The benefit of decreased bleeding at surgery should outweigh the relatively small but definite risk of embolization. Rarely embolization is used as a primary therapeutic modality for vascular tumors in patients who are unable to tolerate surgical resection.

POST OPERATIVE IMAGING

Imaging in the immediate post operative period is performed to assess complications of skull base surgery. CT is the preferred modality since it is easy to perform on critically ill patients, is widely available, and is excellent in showing most of the complications of surgery such as infarction, hematoma, vascular thrombosis, and hydrocephalus.

The more difficult problem in the post operative period is deciding how successful the surgical resection has been. Searching for small areas of residual or recurrent tumor within the complex tissues of the operative bed is a diagnostic challenge. The density and enhancement characteristics of post operative scar, packing, or flaps may closely approximate those of the original tumor. The same is true of MR signal characteristics. We have been disappointed in our ability to differentiate scar, packing, and flaps from residual tumor. Often we are unable to detect tumor regrowth until it grossly distorts the tissue planes within the operative bed. For this reason serial scans are quite important. We obtain a baseline post operative study three months following resection.

**NON-SURGICAL TREATMENT OF TUMORS OF THE POSTERIOR SKULL BASE:
EFFECTIVENESS AND SAFETY**

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Radiation therapy, brachytherapy, or radiosurgery may be used effectively to treat selected patients with tumors of the posterior skull base in an adjuvant setting or as the primary modality, either initially or at recurrence. Unsuccessful outcomes following these local radiation procedures are caused by inadequate dose within the treatment volume, by tumor extension beyond the treatment volume, or by unacceptably high dose to normal tissue within the irradiated volume. Successful treatment depends on close cooperation between the radiation therapist, neurosurgeon, and neuroradiologist, and on careful treatment planning with modern imaging techniques and sophisticated three-dimensional treatment planning software. This presentation will focus on nonsurgical management of several tumors of the posterior skull base: meningioma, chordoma and chondrosarcoma, glomus tumor, acoustic neuroma, and metastases. Much of the available data is from an era when 3-D imaging-based planning was unavailable. It is expected that control and complication rates will improve, in some cases substantially, with modern techniques and possibly with the use of hyperfractionation.

Side Effects and Complications of Radiation Treatment

During the course of treatment some patients may suffer an acute increase in preexisting neurological deficits or demonstrate a clinical syndrome indicative of increased intracranial pressure. Usually such reactions are mild and of little significance. They are commonly thought to be due to radiation-induced edema, although objective evidence to prove this hypothesis is lacking. CT scans obtained during treatment usually do not demonstrate increased peritumoral edema (11). Modern clinical experience indicates that constant daily radiation doses of 22 cGy are well tolerated acutely. Uncommonly, new neurological deficits may be seen a few weeks to a few months following irradiation (5,41). Such reactions are usually transient and associated with an uneventful recovery. CT scans may reveal changes consistent with demyelination during the inhibition of myelin synthesis (17).

The latency and recovery times of such reactions correspond to the turnover time of myelin (15,22). Reactions manifesting several months to years following radiotherapy are usually progressive and irreversible, and sometimes fatal. Radiologically, such injury may present as focal necrosis or as diffuse white matter injury. On CT, focal necrosis produces a low-density region with surrounding edema and variable mass effect, with an irregular margin of contrast enhancement. MRI demonstrates edema with increased signal on T2-weighted images. Neither CT nor MRI produces images specific for tumor versus focal necrosis. Occasionally the distinction can be made by examining the blood-brain barrier and local glucose metabolism with positron emission tomography (14). Diffuse

white matter injury may occur at a somewhat lower dose and after a longer latent period than focal necrosis (36); it is more frequently appreciated on MRI than CT. The incidence of such injury increases with volume of brain irradiated, radiation dose, interval between irradiation and imaging, and patient age. Severe radiologic changes correlate with clinical neurological findings, whereas the significance of mild and moderate radiological abnormalities is unknown (9). Infrequently, damage to intracranial arteries may occur, possibly resulting in brain necrosis at a site not within or immediately adjacent to the radiation field; arteriography may demonstrate occluded or stenosed arteries within the radiation field (6,35). In 1980 Scheline et al (46) reviewed all reported cases of radiation-induced brain necrosis and estimated the incidence of brain necrosis following 5200 cGy/26 fractions at 200 cGy/day to be less than 0.4%. This was subsequently corroborated by Marks et al, (32,34) who demonstrated that the risk of radionecrosis is extremely low for patients treated to 5400 cGy in 30 fraction over 42 days, but increases rapidly as dose rises above 6000 cGy. Radiation damage to the brain stem is thought to be infrequent for a dose of 5400 cGy in fractions of 180-200 cGy. Recent experience indicates that the brain stem dose can be increased substantially, to 7200-7800 cGy with twice daily fractions of 100 cGy. It is thought that dose to other structures, such as cranial nerves, can be increased with BID fractionation schemes, although this is not well documented at present.

The risk of cranial nerve damage is correlated with daily fraction size and total dose. At approximately 165-190 cGy/fraction, the risk of optic nerve injury, for example, is close to 0% if 4500 cGy is delivered, and about 10% if 6000-7300 cGy is delivered (40). In this latter dose range, daily fractions of 195 cGy or greater increase the risk to about 40%. Occasional reports of optic nerve damage following a dose of 5000 cGy or less in standard fractions are seen in patients who present with optic nerve deficits secondary to mass effect. Other cranial nerves, more relevant in the treatment of tumors of the posterior skull base, may also be damaged following radiotherapy but are not as well studied as the optic nerves. It is usually assumed that the risk for cranial nerve damage is similar to or possibly lower than that associated with the CNS. For example, approximately half of the patients treated with high dose radiation therapy for nasopharyngeal cancer who have pretreatment and posttreatment audiometric evaluation are noted to have developed sensorineural hearing loss (38). Little is known concerning effects of conventional radiotherapy on vestibular function, although some patients who complain of vertigo in the postradiation period demonstrate abnormal results of ice water caloric tests (28). Occasional histologic

studies have shown absence of the organ of Corti, atrophy of the spiral ganglia and cochlear nerve, loss of hair cells, etc. (28,45).

With modern supervoltage therapy the skin is usually not a dose-limiting tissue when treating skull base tumors. However, when the beam enters the body tangentially or when bolus is placed on the skin the skin-sparing effects are lost. Skin reactions vary with the size of the area irradiated, dose, overall treatment time, number of fractions, energy of radiation, and patient-related variables. Subcutaneous fibrosis in unoperated tissue may appear 6-12 months following delivery of greater than 6800 cGy in fractions of 180-200 cGy, measured at 5mm depth. Such fibrosis is rarely painful and is more frequently seen in patients with large amounts of subcutaneous adipose tissue. Alopecia can be permanent with skin doses above 5000-6000 cGy.

Experienced radiotherapists realize that the dose to skin and subcutaneous tissue may be higher than that delivered to the tumor if parallel opposed fields are used with cobalt-60 equipment or with 4 Me V linear accelerators rather than with higher energy machines. Xerostomia may result if entrance or exit beams deposit significant dose in the parotid gland. Only a small percentage of patients receiving 4000-6000 cGy have measurable parotid flow following salivary stimulation (33). Salivary function may recover six months following 3000-3500 cGy. Fibrosis of the muscles of mastication and ankylosis secondary to irradiation of the temporomandibular joint with resulting trismus may follow high dose irradiation of these areas. With modern treatment planning techniques it should be possible to avoid this complication in most patients with posterior fossa skull base tumors. The risk of temporal bone necrosis is related to total dose and fraction size, and is very uncommon for temporal bone doses less than 7600 cGy at 200 cGy/fraction with megavoltage equipment (52). Likewise, cartilage usually tolerates conventionally fractionated high-dose irradiation well (12,39).

Radiation-induced tumors including meningioma, glioma, and sarcoma are occasionally reported (29,43). The latency period is between 6 and 31 years. Fortunately, radiation-induced brain tumors are rare and the possibility of their induction should not by itself be considered a contraindication to radiotherapy. However, with improved survival in childhood malignancies the incidence of radiation-induced brain tumors may increase.

Most groups practicing radiosurgery report serious new neurologic deficits in approximately 5% or fewer of patients treated. The risk of such side effects is thought to be closely related to extent of radiosurgery target volume and dose, site in the brain, and possibly other factors such as patient age, concomitant medical problems, prior treatment history, etc. In most cases a dose in the range 1500-2000 cGy at the periphery of

the target volume is tolerated well, although this dose may result in temporary cranial neuropathies in patients whose target volume encompasses a portion of a cranial nerve.

Occasional patients with posterior fossa skull base tumors may be treated with brachytherapy techniques, usually involving permanent placement of radioactive seeds. In such cases radiation is delivered at a relatively low dose rate over many months. The cumulative dose may be in the range of 10,000 cGy or greater. Side effects directly attributable to this radiation dose are infrequent.

The risk of all types of radiation injury is increased in patients with underlying vascular disease secondary to diabetes, hypertension, Cushing's disease, or acromegaly, in those with underlying infection, and in those who have received chemotherapy or are under the age of 3 years (in whom myelination is incomplete) (1,4,7,8,46,47,53).

Metastases

Carcinomas of the breast or prostate are the most common malignancies to metastasize to the osseous base of skull (19,24,42). Such patients are often treated with 3000 cGy administered in 10 fractions to the skull base via lateral opposed fields. Pain and cranial nerve involvement are common symptoms of base of skull involvement and symptomatic response to radiotherapy is seen in 80-90%; objective response is about 50% (2/3 partial, 1/3 incomplete) (19). Response is increased if duration of symptoms is less than 1 month and for increased dose. Since at least 2/3 of patients will have metastatic involvement at other sites, radiotherapy to symptomatic osseous metastases is usually employed although chemotherapy and/or hormonal manipulation may also be used. Since most patients with osseous metastases have limited life expectancy, the standard treatments to administer larger daily fractions over a relatively short period, usually 3-weeks. However, in patients with a solitary metastasis, especially for those with breast cancer, survival duration may be many years. In such cases long-term side effects may be reduced if lower daily fractions to a total dose of 4500-2000 cGy in five weeks are delivered.

Vikram and Chu (50) have shown that few patients with base of brain involvement will subsequently require whole brain irradiation. DeAngelis et al (10) have demonstrated that severe dementia may occur in 10-20% of patients who receive whole brain radiation therapy and survive at least one year. Such dementia can be avoided with protracted fractionation such as 4500 cGy in five weeks rather than 3000 cGy in three weeks to the whole brain. Therefore, unless patients have demonstrated metastatic disease at other intracranial sites, it would seem that whole

brain irradiation delivered in large fractions is usually contraindicated. Occasional patients may present with posterior fossa brain metastases adjacent to the skull base. Such patients have an elevated risk for intradural spinal metastases (as high as 25% actuarial risk at one-year (37)).

Such patients could be treated with external beam radiation therapy, with considerations similar to those mentioned for patients with osseous posterior skull base metastases. In general several factors are known to be associated with prolonged survival in patients with intracranial metastases: KPS 70-100, age less than 60, primary tumor absent or controlled, no metastases outside the brain, small number of brain metastases (13,49). Recently Loeffler (30) has summarized several results of radiosurgery for brain metastases. Of 214 patients treated with a minimum tumor dose of 1650-2500 cGy, lack of tumor progression was seen in 85-95%, with follow-up of 3-7.5 months. Patients treated with radiosurgery frequently show clinical improvement and have decreased steroid requirements despite having received prior brain irradiation. One must caution, however, that new cranial neuropathies can be a complication of treatment.

In summary, patients with short expected survival can be palliated effectively with 3000 cGy delivered to the whole brain in 2 weeks. For patients with long expected survival and a solitary osseous skull base metastasis or a solitary brain metastasis, focal radiation therapy to 5000 cGy over 5 weeks is recommended. Radiosurgery may be considered for patients with a solitary brain metastasis.

Chordoma and Chondrosarcoma

Conclusions regarding the efficacy of x-rays for chordoma or chondrosarcoma must be considered tenuous. First, most series report small numbers of patients. Second, median follow-ups are often too short for adequate evaluation. Finally, and most importantly, series with length follow-up are from an era when CR of MRI scans were not available for treatment planning purposes, and treatment plans that would be considered unacceptable by today's standards were often used. Therefore, it is not surprising that there is disagreement between radiotherapy series in terms of presence of absence of a clear dose response curve, or in terms of correlation of degree of post-radiotherapy local control with extent of resection. Fuller and Bloom (18) reported 13 patients with base of skull chordomas treated with linear accelerator x-rays but without the benefit of CT treatment planning. The delivered dose was 1500-6500 cGy. Pain relief was seen in all patients but cranial nerve deficits usually did not improve. No complications were seen. Local control (defined as freedom from radiologic or clinical progression) was 23% at 5-years and 16% at 10-years and showed a positive correlation with increasing dose and with increasing degree of surgical resection, although not all authors have reported these correlations.

Patients with posterior fossa lesions had a lower control rate than those with middle fossa lesions, likely because patients with posterior fossa lesions were treated to a lower dose to avoid damage to the brain stem. Based on histology the 5 year local control rates were: chondrosarcoma (77%), chordoma (55%), chondroid chordoma (36%). All patients were planned with modern imaging and treatment planning techniques, and were treated to higher doses than in most reported x-ray series. Nevertheless approximately 1/3 of patients developed cranial nerve complications or damage to the brain stem or pons. Austin-Seymour et al (2) reported 68 patients with base of skull chordomas or chondrosarcomas treated with protons and x-rays. Again, modern imaging and planning techniques were used and high doses were employed (median 6900 cGy). The five year local control rate in 68 patients was 82% at five years. No differences were seen based on histology.

Scattered reports in literature indicated that it is possible to use brachytherapy techniques to deliver 4000-40,000 cGy with radioactive seed implant. (20,26) Reported complication rates are low and control rates with very limited follow-up appear to be good. Much more experience is needed before definitive statements can be made regarding this modality.

In summary, local control is good in patients treated with particles and planned with modern techniques; it is poor in patient treated with x-rays and planned with outmoded techniques. It is not known whether BID x-rays and modern planning will also yield high control rates. Several additional issues have not been resolved. First, it is not known whether radiation therapy should be used in patients following total resection or whether is preferable to wait until recurrence. Second, the benefits and risks of re-irradiation for patients who recur following previous high dose radiation therapy have not been defined. (Although preoperative external beam therapy may allow tumor shrinkage and reduction of the surgical field, such shrinkage may take six months or longer and may lead to decreased surgical healing and increased wound infection.)

Improved results may be seen with the use of neutron radiotherapy (27) although follow-up is still limited. Yet more promising results are seen with the use of heavy ions (plus or minus x-rays) or protons (plus x-rays) to a high dose. It is not known whether the improved results are due to the use of heavy ions or protons per se or to the use of modern imaging and treatment planning techniques or to the use of higher dose. Berson et al (3) reported 27 patients with chordomas or chondrosarcomas of the base of skull and reported a 74% control rate at 5-years. Patients with less than 20 cc of gross tumor had a significantly higher local control rate than those with greater than 35 cc of tumor (80% at five-years versus 33%; p - 0.05). Patient received up to 8000 cGy.

Glomus Tumors

Series with long follow-up after radiation therapy treatment are from an era prior to advanced imaging and planning techniques. Nevertheless, local control (defined as lack of radiologic or clinical evidence of progression) is excellent and complication rates are low. Springate and Weichselbaum (48) summarized all radiotherapy series with treatment dates in the range 1932-1983. For patients receiving preoperative or planned postoperative therapy local control was 146/169 (86%). In this group there was a 7% complication rate, including bone or brain necrosis, auditory canal stenosis, brain abscess, TMJ fibrosis, or panhypopituitarism. Over the same treatment period 13 series reported treatment with radiotherapy alone or subsequent to surgical failure, with a local control rate of 183/195 (94%). In this group the complication rate was 4%, including sarcoma, brain abscess, and bone or brain necrosis. Every reported complication, either in the preoperative/postoperative radiotherapy group or in the radiotherapy alone group was a result of the use of orthovoltage x-rays (no longer used for glomus tumors) or the use of electrons alone (no longer recommended). Patients in the two groups received 1500-1700 cGy; most analyses show that a dose of 5000 cGy is adequate to ensure a high degree of local control and a low rate of complication. With modern imaging and planning techniques and with modern linear accelerator equipment, bone or brain necrosis of TMJ fibrosis should be exceedingly rare.

The regression rate following radiotherapy is usually slow. Some patients eventually demonstrate complete gross disappearance of tumor although many have a persistent stable lesion for many years. With current techniques the patient may be treated with a wedged pair arrangement of beams and many patients experience small areas of temporary hair loss after 3 or 4 weeks of treatment. In most cases hair will regrow over a period of several months.

Acoustic Neurinoma

The efficacy of fractionated radiation therapy for acoustic neurinomas has been discussed by Wallner et al, (51) who reviewed the records of 124 patients treated at UCSF from 1945 through 1983. Follow-up was 2.6-40.7 years. 38 patients received irradiation based on recommendations of the surgeon and consulting radiotherapist. One of 17 patients (6%) who underwent subtotal resection followed by at least 4500 cGy of external beam radiation therapy developed a recurrence, compared to 6 of 13 (46%) who did not receive external beam radiation. This difference is statistically significant. Three additional patients had biopsy only and were treated with external beam radiation therapy; none recurred. Wallner et al suggest that tumors that are subtotally resected should receive external beam radiotherapy to a dose of

5000-5500 cGy. Unfortunately, this paper did not address changes in cranial nerve function following radiotherapy. However, cranial nerve deficits to this dose with a standard fraction of 180 cGy per day are expected to be infrequent.

Recent data demonstrates the effectiveness of radiosurgery for management of acoustic neurinomas. The group in Pittsburgh (16) has reported results in 85 patients, with follow-up of 1-29 months. The group from Stockholm (21) has reported 126 patients, with follow-up of 8-154 months. In both groups patients had an initial tumor size less than 30mm. Maximum tumor doses were in the range 2200-5000 cGy and minimum tumor doses were in the range 1400-2500 cGy. Results at Pittsburgh (Stockholm) are: decreased size 41% (44%), stable lesion 56% (42%), increased size 3% (14%), central necrosis (63%). Following treatment, useful hearing was preserved in Pittsburgh (Stockholm) in 46% (26%) of patients, new trigeminal neuropathy 37% (18%), new facial neuropathies 33% (15%). In most cases facial neuropathies and trigeminal neuropathies were considered transient. The Pittsburgh group was unable to find a relationship between radiation dose and decreased hearing or trigeminal or facial nerve deficit. This group did note that no patient with a tumor less than 10 mm in diameter suffered loss of useful hearing.

More data are needed to better define the management of large acoustic neurinomas. Recently Lownie and Drake (31) published results of radical intracapsular removal performed in 12 patients. Tumor recurrence developed in two patients (18%) and facial function was preserved in 9 (82%). An alternative approach is that of Ikeda et al (23) who described 3 patients who were treated preoperatively with 2340-3000 cGy over 3-3 1/2 weeks, with radical operations performed 6-8 weeks following external beam radiation. It was concluded that radical surgery, which may have been impossible because of uncontrolled massive bleeding, was successful without significant intraoperative bleeding after radiotherapy.

Meningioma

It is generally agreed that the risk for local recurrence following surgery for meningioma is inversely related to the extent of surgery performed, and that the extent of surgery performed is dependent on tumor location. In some studies the percentage of patients undergoing total resection for a posterior fossa benign meningioma is about half that of patients with convexity meningiomas. Radiation therapy should be considered for patients who do not have a total resection. Unfortunately, most studies which evaluate the risks and benefits of radiation therapy in

patients who have had less than total excision are not randomized and thus do not allow a direct comparison of surgery alone versus surgery plus radiotherapy. Some studies suggest no benefit from radiation therapy whereas others report improved local control. A recent meta-analysis of 13 studies involving 1898 patients indicates improved local control with the addition of external beam radiation therapy (44). The percentage of patients having a recurrence within 5 years is: 40% (subtotal excision alone), 18% (subtotal excision plus radiation therapy), 76% (subtotal excision alone following recurrence), 43% (subtotal excision plus radiation therapy following recurrence). Such an analysis is strongly suggestive that radiation therapy is of benefit in improving local control. It is not clear, however, if radiation therapy simply delays the time to recurrence or whether actual cures are produced. Some studies involving postoperative radiation therapy show a delay in actuarial local control or survival cures without a break to indicate cure; others do show such a break. Several series report that recurrence has an adverse affect on survival, that the time interval between successive recurrences decreases, and the successive recurrences are associated with malignant transformation. Therefore, radiation should probably be used following the first subtotal resection rather than later. A similar meta-analysis involving 73 patients with malignant meningiomas shows a 50% local recurrence rate in patients undergoing total excision and a 90% recurrence rate in patients undergoing subtotal excision. The addition of radiation therapy markedly reduces these recurrence rates, although the number of patients in various categories is small.

Following are guidelines for the use of external beam fractionated radiotherapy in the management of meningiomas: (1) Incompletely resected benign meningiomas should be treated with external beam radiation therapy to 5000-5500 cGy at 180-200 cGy/fraction. It is possible that bulky residual tumors require 6000 cGy, although this has not been demonstrated definitively. (2) Patients who have not received radiation therapy but recur should receive external beam radiation therapy. (3) Patients with aggressive histology, even those with total resection, require external beam radiation therapy to 6000 cGy in standard daily fractions, to 3 cm beyond the MRI presurgical tumor volume.

Recently radiosurgery has been used as the only radiation modality in the treatment of patients with meningiomas with average diameter no greater than 35 mm (25). In most of the reported patients no prior radiation was used; in some patients no surgery was performed. Patients received a mean radiation dose of approximately 1700 cGy in a single fraction. The actuarial tumor control rate at 2 years is 96%. Longer follow-up is required, of course, but this approach appears quite promising.

An additional approach which may prove promising involves implantation of brachytherapy sources directly within the tumor bed (20,26). Such an approach has the advantage of delivering a very high dose, with low complication rate, over a period of weeks or months, depending on the isotopes selected.

References

1. Aristizabel SA, Boone ML, Laguna J. Endocrine factors influencing radiation injury to central nervous tissue. *International Journal of Radiation Oncology, Biology, Physics* 5:349-353, 1975.
2. Austin-Seymour M, Munzenrider J, Goitein M, Verhey L, et al, Fractionated proton radiation therapy of chordoma and low-grade chondrosarcoma of the base of skull. *Neurosurgery* 70:13-17, 1989.
3. Berson AM, Castro JR, Petti P, et al. Charged particle irradiation of chordoma and chondrosarcoma of the base of skull and cervical spine: The Lawrence Berkeley Laboratory experience. *Radiation Oncology, Biology, Physics* 15:559-565, 1988.
4. Bloom B, Kramer S. Conventional radiation therapy in the management of acromegaly. In: Black PM, Zervas NT, Ridgeway EC, Martin JB, ed. *Secretory Tumors of the Pituitary Gland*. New York: Raven Press, 1984:179-190.
5. Boldrey E, Sheline GE. Delayed transitory clinical manifestations after radiation treatment of intracranial tumors. *Acta Radiologica* 5:5-10, 1967.
6. Brant-Zawadski M, Anderson M, DeArmond SJ. Radiation-induced large intracranial vessel occlusive vasculopathy. *American Journal of Reontgenology* 134:51-55, 1980.
7. Burger PC, Mahaely MS Jr., Dudka L, Vogel FS. The morphologic effects of radiation administered therapeutically for intracranial gliomas: a postmortem study of 25 cases. *Cancer* 44:1256-1272, 1979.
8. Cumberlin RL, Luk KH, Wara WM. Medulloblastoma: Treatment results and effect on normal tissues. *Cancer* 43:1014-1020, 1979.
9. Curran WJ, Hecht-Leavitt C, Schut L. Magnetic resonance imaging of cranial radiation lesions. *International Journal of Radiation Oncology, Biology, Physics* 13:1093-1098, 1987.

10. DeAngelis LM, Delattre J-Y, Posner JB. Radiation-induced dementia in patients cured of brain metastases. *Neurology* 39:789-796, 1989.
11. Deck MDF. Imaging techniques in the Diagnosis of Radiation Damage to the Central Nervous System. In: Gilbert HA, Kagan AR, ed. *Radiation Damage to the Nervous System*. New York: Raven Press, 1980: 107-127.
12. Del Regato JA, Dental lesions observed after roentgen therapy in cancer of the buccal cavity, pharynx, and larynx. *American Journal of Roentgenol Radium Therapy* 42:404-410, 1939.
13. Diener-West M, Dobbins TW, Phillips TL, Nelson DF. Identification of an optimal subgroup for treatment evaluation of patients with brain metastases using RTOG study 7916. *International Journal of Radiation Oncology, Biology, Physics* 16:669-673, 1989.
14. Doyle WK, Budinger TF, Valk PE. Differentiation of cerebral radiation necrosis from tumor recurrence by (¹⁸F) FDG and ⁸²Rb positron emission tomography. *Journal of Computer Assisted Tomography* 11:563-570, 1987.
15. Edward MSB, Wilson CB. Treatment of radiation necrosis. In: Gilbert HA, Kagan AR, ed. *Radiation Damage to the Nervous System*. New York: Raven Press, 1980:129-143.
16. Flickinger JC, Lunsford LD, Coffey RJ, et al. Radiosurgery of acoustic neurinomas. *Cancer* 67:345-352, 1990.
17. Freeman JE, Johnston PGG, Voke JM. Somnolence syndrome after prophylactic cranial irradiation in children with acute lymphoblastic leukemia. *British Medical Journal* 4:523-525, 1973.
18. Fuller DB, Bloom JG. Radiotherapy for chordoma. *International Journal of Radiation Oncology, Biology, Physics* 15:331-339, 1988.
19. Greenberg HS, Dek MDF, Vikram B, et al. Metastasis to the base of the skull: Clinical findings in 43 patients. *Neurology* 31:530-537, 1981.
20. Gutin PH, Leibel SA, Hosobuchi Y, Crumley RL, Edwards MSB, et al. Brachytherapy of recurrent tumors of the skull base and spine with iodine -125 sources. *Neurosurgery* 20:938-945, 1987.
21. Hirsch A, Noren G, Audiological findings after stereotactic radiosurgery in acoustic neurinomas. *Acta-Otolaryngologica* 106: 224-251, 1988.
22. Hoffman WF, Levin VA, Wilson CB. Evaluation of malignant glioma patients during the postirradiation period. *Journal of Neurosurgery* 50:624-628, 1979.
23. Ikeda J, Ito H, Kashihara K, Fujisawa H, Yaamoto S. Effective preoperative irradiation of highly vascular cerebellopontine angle neurinomas. *Neurosurgery* 22:566-573, 1988.
24. Kistler J, Pribram HW, Metastatic disease of the sella turcica. *American Journal of Roentgenol.* 123: 13-21, 1975.
25. Kondziolka D, Lunsford LD, Coffey RJ, Flickinger JC. Stereotactic radiosurgery of meningiomas. *Neurosurgery* 74:552-559, 1991.
26. Kumar PR, Good RR, Leibrock LG, Patil AA, et al. Tissue tolerance and tumor response following high activity iodine-125 endocurietherapy for skull base tumors. *Endocurie, Hypertherm, Oncology* 6:223-230, 1990.
27. Laramore GE, Griffith JT, Boespflug M, et al. Fast neutron radiotherapy for sarcomas of soft tissue, bone, and cartilage. *American Journal of Clinical Oncology* 12:320-326, 1988.
28. Leach W. Irradiation of the ear. *Laryngology, Otology* 79:870-880, 1965.
29. Liwnicz BH, Berger TS., Liwnicz RG, Aron BS. Radiation-associated gliomas: a report of four cases and analysis of postradiation tumors of the central nervous system. *Neurosurgery* 17:435-436, 1985.
30. Loeffler JS, Alexander I E., Kooy HM, Wen PY, Fine HA, Black PM. Radiosurgery for brain metastases. *Principles & Practice of Oncology* 5:1-12, 1991.
31. Lownie SP, Drake CG. Radical intracapsular removal of acoustic neurinomas: long-term follow up review of 11 patients. *Neurosurgery* 74:422-425, 1991.
32. Marks JR, Wong J. The risk of cerebral radionecrosis in relation to dose, time and fractionation. *Progress in Experimental Tumor Research* 29:210-218, 1985.
33. Marks JE, Davis CC, Gottsman VL, Purdy JE, Lee F. The effects of radiation on parotid salivary function. *Radiation Oncology, Biology, Physics* 7:1013-1019, 1981.

34. Marks JE, Baglan RJ, Prasad SC, Blank WF. Cerebral radionecrosis: incidence and risk in relation to dose, time fractionation and volume. *International Journal of Radiation Oncology, Biology, Physics* 7:243-252, 1981.
35. Marks MP, Delapaz FL, Fabrikant JI, et al. Intracranial vascular malformations: imaging of charged-particle radiosurgery: Part II. Complications. *Radiology* 168:457-462, 1988.
36. Mikhael MA. Radiation damage to the central nervous system: a delayed therapeutic hazard. In: Gilbert HA, Kagen AR, ed. *Radiation Damage to the Nervous System*. New York: Raven Press, 1980:59-91.
37. Mirimanoff RO, Choi NC. Intradural spinal metastases in patients with posterior fossa brain metastases from various primary cancers. *Oncology* 44:222-236, 1987.
38. Moretti JA. Sensorineural hearing loss following radiotherapy to the nasopharynx. *Laryngoscope* 85:598-602, 1976.
39. Parker RG, Wildermuth O. Radiation therapy of lesions overlying cartilage: I. Carcinoma of the pinna. *Cancer* 15:57-65, 1962.
40. Parsons JT, Fitzgerald CR, Hood CI, et al. The effects of irradiation of the eye and optic nerve. *International Journal of Radiation Oncology Biology and Physics* 9:609-622, 1983.
41. Rider WD. Radiation damage to the brain - a new syndrome. *Journal of the Canadian Association of Radiologists* 14:67-69, 1963.
42. Roessman, U, Kaufman B, Friede RL. Metastatic lesions in the sella turcica and pituitary gland. *Cancer* 25:478-480, 1970.
43. Ron E, Modan B, Boice JD. Tumors of the brain and nervous system after radiotherapy. *New England Journal of Medicine* 319:1033-1039, 1988.
44. Salazar OM, Ensuring local control in meningiomas. *International Journal of Radiation Oncology, Biology, Physics* 15:501-504, 1988.
45. Samaan NA, Vieto R, Schultz PN, et al. Hypothalamic, pituitary, and thyroid dysfunction after radiotherapy to the head and neck. *Radiation Oncology, Biology, Physics* 8:1857-1867, 1982.
46. Sheline GE, Wara WM, Smith V. Therapeutic irradiation and brain injury. *Journal of Radiation Oncology, Biology, Physics* 6:1215-1228, 1980.
47. Smith BM, McGinnis W, Cook J, Latourette H. Central nervous system changes complicating the use of radiotherapy for the treatment of a nasopharyngeal neoplasm in a diabetic patient. *Cancer* 43:2239-2242, 1979.
48. Springate SC, Weichselbaum RR. Radiation or surgery for chemodectoma of the temporal bone: a review of local control and complications. *Head and Neck* 12:303-307, 1990.
49. Swift PS, Phillips TL, Martz K, et al. CT Characteristics of an optimal subgroup for treatment evaluation of patients with brain metastases using RTOG study 7916. *Radiation Oncology, Biology Physics (Suppl.)* 19:206, 1990.
50. Vikram B, Chu FCH. Radiation therapy for metastases to the base of the skull. *Radiology* 130:465-468, 1979.
51. Wallner KE, Sheline GE, Pitts LH, Wara WM, Davis RL, Boldrey EB. Efficacy of irradiation for incompletely excised acoustic neurilemmomas. *Neurosurgery* 67:858-863, 1987.
52. Wang CC, Doppke L. Osteoradionecrosis of the temporal bone: Consideration of nominal standard dose. *International Journal of Radiation Oncology, Biology, Physics* 1:881-883, 1976.
53. Weiss HD, Walker MD, Wiernik PH. Neurotoxicity of commonly used antineoplastic agents. *New England Journal of Medicine* 291:127-133, 1974.

EXTRADURAL TUMORS OF THE POSTERIOR CRANIAL BASE

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INTRODUCTION

The posterior cranial base is a common site of origin of benign and malignant tumors, which may be conveniently divided into intra- and extradural tumors. Unlike other regions of the cranial base, posterior cranial base tumors continue to be the most challenging surgical lesions due to several factors:

1. The intimate relationship of such tumors to critical structures such as the brain stem, cranial nerves, and major cranial blood vessels;
2. Difficulty in obtaining adequate surgical access to this area;
3. For malignant tumors, radical tumor resection with tumor free margin is necessary to ensure total tumor resection. This frequently implies resection of tumor involved cranial nerves and vessels with reconstruction, which have a relatively high associated risks.

Extradural posterior cranial base neoplasms can be considered under three major categories: benign, low-grade malignancies (i.e., slowly growing neoplasms), and high-grade malignancies (i.e., rapidly growing neoplasms). The list of the various types of neoplasms occurring on this region are outlined in Table I. Common benign tumors in this area include paragangliomas, neurilemmomas of cranial nerves VII, IX-XI, cholesterol granulomas and extradural meningiomas. Low-grade malignancies predominantly consist of chordoma, chondrosarcoma and, less frequently, adenoid cystic carcinoma. High-grade malignancies include a variety of lesions such as squamous cell carcinoma, adenocarcinoma, basal cell carcinoma, and osteogenic sarcoma.

SURGICAL MANAGEMENT

Preoperative Evaluation

All patients undergo CT scans, using 3mm cuts through areas of interest using bone and soft tissue algorithms. MRI scans are performed with and without gadolinium contrast agent. If intraoperative neurophysiological monitoring with evoked potentials or electroencephalography is contemplated, a preoperative baseline study is obtained.

Preoperative cerebral angiography is performed in all patients to evaluate cerebral circulation and tumor blood supply. Preoperative tumor embolization is performed in cases where an accessible blood vessel is available. If the internal carotid artery (ICA) is encased by tumor, or if manipulation of the ICA is anticipated during the exposure, a balloon test occlusion (BTO) with continuous neurological monitoring, followed by cerebral blood flow (xenon/CT-CBF) evaluation is performed, if the patient passes the clinical phase of the test (17).

ANESTHESIA AND NEUROPHYSIOLOGIC MONITORING

After induction of anesthesia, if intraoperative monitoring of cranial nerve function is desired, an inhalational anesthetic technique, such as isoflurane with avoidance of muscle relaxants, is used. For extradural operations, a lumbar subarachnoid drain can aid with brain relaxation. Intraoperative monitoring of evoked potential and electroencephalogram is used liberally to reduce or eliminate postoperative morbidity.

Monitoring of function of cranial nerves III, IV, VII, X, and XII is quite useful to locate these nerves when the anatomy is altered and to reduce injury to them during the removal of tumor (9,13).

OPERATIVE APPROACHES

The selection of a surgical approach to an extradural posterior cranial base tumor depends on the origin of the tumor and the extent of local invasion. The surgical approaches commonly utilized for surgical resection of extradural posterior cranial base neoplasms are outlined in Table 2.

In general, tumors involving the petroclival region are best approached through one of the anterior or lateral approaches. For larger tumors which extend laterally into the petrous bone and sphenoid sinus, the combined anterior and lateral approaches works best (11,15). Petrous bone tumors are best managed by resection of the tumor-infiltrated bone with the object of leaving a tumor-free bony margin through an approach based on the petrous temporal bone (e.g., total petrosectomy approach) (10,12). Tumors occurring in and around the foramen magnum, occipital condyles, and jugular foramen are best approached through the extreme lateral transcondylar +/- transcondylar approach (18). For tumors involving the jugular foramen, a postauricular transtemporal approach (infralabyrinthine approach) is used (5,6).

CRITERIA OF SURGICAL APPROACH SELECTION

Division of the clivus into three anatomical regions (i.e., upper, mid, and lower clivus) is helpful in surgical approach selection (Figure 1). The upper clivus is the area above the petrous apices and above the crossing points of the trigeminal and the abducens nerves from the posterior to the middle cranial fossa. It includes the dorsum sellae and the posterior clinoid processes. It is bonded laterally, by the intracavernous carotid arteries and the cavernous sinuses with contained structures, the structures in the tentorial notch area, and the temporal lobes; posteriorly, by the basilar

artery and its branches, and the mid-brain; and anteriorly by the sella turcica and the sphenoid sinus. The mid-clivus extends from the sixth cranial nerve down to the exit foramina (pars nervosa of the jugular foramen) of the ninth, tenth and eleventh cranial nerves. The mid-clivus is related posteriorly to the basilar artery and branches, the vertebrobasilar junction, and the pons; laterally, by the inferior petrosal sinuses, the petrous apices and the seventh and eighth cranial nerves; and anteriorly, by the nasopharynx and retropharyngeal tissues. The lower clivus is the area below the ninth, tenth and eleventh cranial nerves and includes the occipital condyles, the foramen magnum, and the hypoglossal canals. Posteriorly, the lower clivus is related to the vertebral arteries, the pontomedullary junction, the medulla, and the spinomedullary junction; laterally, to the hypoglossal nerves, the sigmoid sinus, and the jugular bulb; and anteriorly, to the nasopharynx and retropharyngeal tissues. This type of division of the clivus into upper, middle and lower areas is helpful to the surgeon to plan the surgical approaches based on the anatomical location of the tumor.

For lesions involving the upper clivus, we prefer the subtemporal, transcavernous, and transpetrous apex approach (9,17). For neoplasms of the midclivus, the surgical approach selection depends on the tumor location with respect to the midline. For centrolateral tumors where one-half of the clivus and the petrous apex regions are involved by tumor, the subtemporal and infratemporal approach is optimal (11,14). In the more extensive lesions involving the entire midclivus and petrous apex and sphenoid area, either the subtemporal infratemporal approach with division of V3, or the subtemporal infratemporal approach in combination with the basal frontal approach (combined anterolateral exposure) is utilized (11,15).

When the neoplasm is confined to the midline only and involves the whole clivus, it can be approached by one of the anterior approaches (1-4,7,8,11,15). The anterior approaches offer the advantage of providing the most direct route to the clivical structures and are most applicable for extradural tumors. Despite this, they have a number of disadvantages, including the following: the approaches involve traversing contaminated spaces (sinus and nasal cavity); the operative field is at great depths and is restricted laterally by the optic nerves, carotid arteries and the hypoglossal foramina; and the ability to reconstruct the anterior cranial base is limited. We prefer to use the basal frontal approach (11,15) (a modification of transbasal approach of Derome) for these lesions since it does not have some of the disadvantages previously mentioned. The transbasal approach of Derome (4), the basal subfrontal approach and the maxillotomy approach of Cocke and Robertson (1) are the only approaches that allow the possibility of immediate cranial base reconstruction.

For tumors of the lower clival and foramen magnum area, the extreme lateral transcondylar, transcondylar approach is used if the lesions extend to either side of the midline. If the tumor is strictly in the midline and extradural, a transoral or transmaxillary approach may be appropriate (2,7,8). Although intradural midline tumors have been successfully operated on by the transoral approach (3), we feel the risks of complication are too high to recommend as a routine approach by the inexperienced surgeon.

The surgical treatment of benign or malignant tumors involving the temporal bone continues to be one of the most difficult of all skull base procedures. The surgical difficulties arise from having to deal with the carotid artery, venous channels, and multiple cranial nerves that surround or traverse the temporal bone and are frequently involved by temporal bone tumors. For malignant tumors of the middle ear and mastoid, the traditional recommended treatment involves en bloc radical temporal bone resection. In the past, this in fact meant a subtotal resection since the petrous apex was excluded from the resection. We have been using a more radical "total petrosectomy" approach which involves excision of the external ear canal, middle ear and its contents, the mastoid, and the inner ear, along with the petrous apex (10,12). The approach involves skeletonization of the petrous carotid and retracting it anteriorly, followed by retraction of the skeletonized nerve posteroinferiorly. The petrous bone is then removed in a piecemeal fashion. If the carotid artery is involved by tumor, it can be resected and grafted with a saphenous vein graft (16). Cranial nerves involved by tumor are resected and grafted.

Auxiliary Operations

Extradural posterior cranial base tumors frequently present with lower cranial nerve deficits. This situation is further compounded by the frequent occurrence of temporary or permanent lower cranial nerve palsies following surgical intervention. For cases of facial nerve palsies, early tarsorrhaphy is recommended to prevent exposure keratitis. Patients with lower cranial nerve palsies require early tracheostomy and feeding gastrostomy to prevent aspiration and to maintain adequate nutrition. Vocal cord injections with Teflon or Gelfoam is performed later as needed. Speech therapy is helpful in aiding the patient in the relearning process of swallowing.

EXPERIENCE WITH EXTRADURAL POSTERIOR CRANIAL BASE TUMORS

Between 1983 to 1990, the senior author (LNS) operated on 91 patients with extradural tumors of the posterior cranial base in collaboration with neurosurgical, otolaryngological, and plastic surgical colleagues at the University of Pittsburgh Hospitals. The results of these operations are summarized in Tables 3 and 4. The tumors are considered under three major categories: benign, low-grade malignancy, and high-grade malignancy (Tables 3 and 4). Benign lesions included a wide spectrum of tumors such as meningiomas, neurilemmomas of cranial nerves IX and X as well as glomus jugulare tumors with a major component of the tumor involving the extradural petroclival and petrous temporal bone region. Low-grade malignancies consisted predominantly of chordomas and chondrosarcomas but also included some invasive pituitary adenomas with adenoid cystic carcinomas. High-grade malignancies were also quite varied, with squamous cell carcinoma and osteogenic sarcoma being the most frequently encountered lesions. Many of these tumors were noted to be very extensive, and a large number of them had been previously operated, some more than once. Some patients had received radiation therapy following partial resection at other institutions.

Our philosophy in the surgical management of these tumors has been to attempt wide radical tumor excision immediately from the time of diagnosis. In keeping with this philosophy the more extensive lesions frequently required staged operations in order to achieve complete tumor resection. Frequently, for the extensive lesion, more than one approach was necessary in order to obtain optimal tumor exposure.

Tumor-involved dura, tentorium, and brain were resected en bloc with the tumor. Involvement of the petrous internal carotid artery by malignant tumor was managed by resection and reconstruction of that segment of the artery with saphenous vein graft. Infiltration of the lower cranial nerves with malignant tumors was managed by resection with sural nerve grafts. Dural sinus involvement was treated with inclusion of the sinus in the tumor resection. Reconstruction of the operative defect particularly in the petrous bone malignancy was performed by utilizing rotation flaps locally or free muscle flaps (11 or the 20 cases).

Tumor resection was evaluated by a standard protocol of CT/MRI scans performed 2-3 months postoperatively with and without intravenous contrast agents. With increasing experience, our ability to achieve complete tumor resection has improved steadily, with continued reduction in the postoperative morbidity. Follow-up has ranged from a few months to 7 years. As can be seen from Table 5, when total tumor resection was achieved particularly in the benign and low grade malignancies, local recurrence was very uncommon. However, when overall patient status was considered, patients with highly

malignant lesions failed, due to the development of metastatic disease (Tables 3 and 4). It is apparent from these results that for highly malignant tumors radical surgical resection alone is not going to be enough, but that improved methods of adjuvant therapy will need to be developed to improve the prognosis.

Complications

The postoperative complications of extensive extradural posterior cranial base neoplasms are summarized in Table 6. These complications will be discussed further below.

One patient with an extensive squamous cell carcinoma and bilateral ICA encasement was operated with the idea of reducing tumor size prior to radiation therapy. The ipsilateral upper cervical ICA was injured intraoperatively, but was successfully repaired. The artery remained exposed to the nasopharynx since the temporalis muscle flap was used in reconstruction was avascular. One week postoperatively the patient sustained a massive hemorrhage from rupture of ICA. The ruptured ICA was repaired and a rectus abdominis free flap was used to reconstruct the cranial base. A few days later the patient sustained rupture of the contralateral ICA which was occluded. The patient recovered from this, but eventually died of an intractable local infection. The case highlights the risk of leaving arteries exposed at the end of an operation to the contaminated field of the paranasal sinus. The separation of these vessels from the exposed sinus by the utilization of a well-vascularized flap could have averted this complication.

Three patients suffered cerebral infarction. A two-year-old child developed a delayed occlusion of the ICA opposite to the side of the operation, presumably related to excessive rotation of the head during the operation, kinking of the ICA by the styloid process, and perioperative hypercoagulability. In two additional cases, brain stem perforator injuries occurred in patients who had large previously operated tumors with extensive involvement of the basilar artery from tumor or scar tissue.

Three patients with petrous bone malignancies had temporary dysphasia and confusion (one following resection of tumor invaded vein of Labbe' on the dominant hemisphere) which resolved within several weeks. This was felt to be related to cerebral edema of the dominant temporal lobe.

Cerebrospinal fluid (CSF) leaks occurred in patients in whom the tumor was both intra and extradural, or where the dura was excised along with the tumor. This also occurred in two patients who underwent a total petrosectomy for squamous cell carcinoma of the petrous bone. This complication rate was surprisingly low considering water-tight dural closure is never achieved following petrous

bone resection. All CSF leaks were successfully repaired. Cranial nerve palsies were for the most part temporary. In the petrous bone tumor group, temporary lower cranial nerve palsies occurred frequently, since in many cases total tumor resection could only be achieved at the expense of sacrificing the lower cranial nerves. While 17 facial nerves were resected along with petrous bone tumors, 12 were reconstructed using cable graft or hypoglossal to facial nerve anastomosis. In addition, in four of the eight cases involving cranial nerves IX-XI, reconstruction was achieved using cable graft. The remaining cases of facial palsy followed mobilization of the nerve in its infratemporal segment. When cranial nerve VII was not mobilized from the temporal bone, temporary cranial nerve VII palsy was followed by an early recovery to a House Grade I or II function. When cranial nerve VII was displaced from the temporal bone or grafted, the recovery was usually to a House Grade III. Unilateral deficits of cranial nerves IX and X, were reasonably well tolerated with the aid of vocal cord injections. Four wound breakdowns occurred in the petrous bone malignancy group which required secondary wound repair. Two of these patients had undergone surgery with incomplete tumor resection, and radiation therapy prior to our operations. Another patient developed a large hematoma under his rectus abdominis free flap which required evacuation.

The other major complications in the petrous bone neoplasm groups included five cases of pneumonia in patients with lower cranial nerve palsies, and one case of a large pulmonary embolism which developed two days postoperatively. All patients made a good recovery.

Overall, an increased complication rate was noted in the following patients: those who had been operated on previously; those with giant sized tumors; those who had had previous radiation therapy; and those with intra and extradural tumors.

The overall outcome of the complications was quite good, with the majority of patient being able to return to their prior occupation or independent living; except when more permanently disabled.

CONCLUSION

The radical resection of both large benign and malignant extradural tumors of the posterior cranial base in the era of modern skull base surgery can be performed safely with an acceptable low morbidity. This approach appears to offer a definite advantage to the outcome of benign tumors. As far as malignant tumors are concerned, surgery will have to be completed by improvement in adjuvant therapies to improve the outlook for these tumors.

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TABLE 1

Extradural Tumors of the Posterior Cranial Base

PETROCLIVAL REGION

<u>Benign</u>	Meningiomas Paragangliomas Cholesterol Granuloma Epidermoid Cyst Neurilemmomas of CNs IX-XI
<u>Low-Grade Malignancies</u>	Chordoma Chondrosarcoma Adenoid Cystic Carcinoma Pituitary Adenoma (invasive)
<u>High-Grade Malignancies</u>	Squamous Cell Carcinoma Nasopharyngeal Carcinoma Osteogenic Sarcoma Metastasis

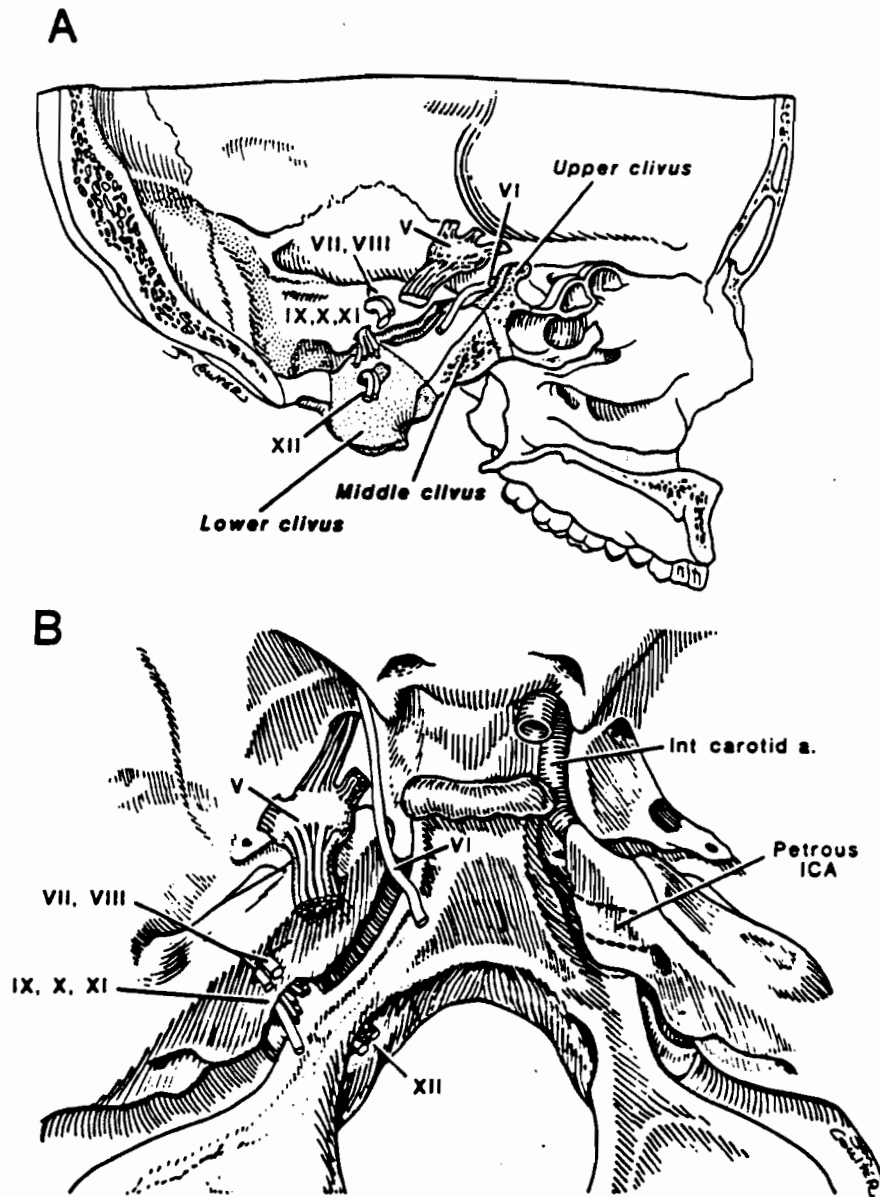
PETROUS BONE

<u>Benign</u>	Schwannoma of CN VII, IX-XI Meningiomas Glomus Tumor
<u>Low-Grade Malignancies</u>	Adenoid Cystic Carcinoma Adenocarcinoma
<u>High-Grade Malignancies</u>	Squamous Cell Carcinoma Osteosarcoma Rhabdomyosarcoma Basal Cell Carcinoma Metastasis

FIGURE 1

Anatomical division of the clivus into upper, mid, and lower regions, and the adjacent important cranial base structures are shown.

- A. Lateral View
- B. Superior View



SURGICAL APPROACHES TO EXTRADURAL TUMORS OF THE POSTERIOR CRANIAL BASE

Approach	Region Exposed	Advantages	Disadvantages
I) Anterior Extradural			
Transoral/Transpalatine	mid and lower clivus down to C ₂	-most direct approach for midline lesion -technically fast and easy	-limited exposure -working at great depth -contaminated operative field (risk of meningitis)
Transsphenoidal	sphenoid sinus and upper clivus	-technically easy	-exposure limited to midline
Maxillotomy (LeFort I osteotomy)	upper and mid clivus sphenoid sinus upper nasopharynx	-rapid and technically easy	-working through -contaminated operative field
Maxillectomy	upper clivus to C ₅	-good exposure	-facial incision scar -oral cavity prosthesis required
Transbasal (Derome)/ Basal Subfrontal (modified transbasal)	base of dorsum sella to C ₁ sphenoid and ethmoid. sinus	-aerodigestive tract not violated -allows cranial base reconstruction with vascularized flap	-bilateral olfactory denervation -risk of injury to frontal lobes and CNs II and VI
II) Lateral Extradural			
Subtemporal Infratemporal	mid and lower clivus petrous apex + adjacent 1/2 of clivus	-give excellent anterolateral view of brain stem without retraction	-loss of Eustachian tube + temporomandibular joint -risk of ICA injury

TABLE 2 (continued)

Approach	Region Exposed	Advantages	Disadvantages
Subtemporal Infratemporal with V ₃ division	petrous apex entire mid + lower clivus sphenoid sinus	-improves exposure of mid clivus and sphenoid sinus	-above + loss of V ₃ function (return of V ₃ if reanastomosed)
Subtotal/Total Petrosectomy	partial upper + mid clivus, petrous bone region	-wide exposure without brain retraction	-loss of hearing -risk of facial nerve injury and ICA injury
Postauricular Transtemporal (Infralabyrinthine/ Infratemporal (Fisch))	infratemporal fossa jugular foramen	-good exposure	-loss of conductive hearing (Fisch) -temporary facial paralysis
Extreme Lateral, Trans- condylar, Transjugular	lower clivus, occipital condyle, C ₁ jugular foramen	-working anterior to craniocervical junction through sterile field -wide exposure	-need for craniocervical fusion if condyle removed -lengthy procedure
III) Anterior and Lateral			
Basal Subfrontal and Subtemporal Infratemporal	clivus to C ₁ , petrous bone, sphenoid sinus and ipsilateral ICA	-very wide exposure with minimal brain retraction -able to reconstruct floor or cranial base	-time-consuming -upper clivus not exposed -loss of olfaction and Eustachian tube
IV) Lateral Intradural			
Subtemporal, Transcavernous and Transpetrous Apex	upper clivus and petrous apex (ipsilateral) -cavernous sinus	-upper clivus approach through middle fossa	-risk of CN III, IV, V, VI and ICA injury -only useful when tumor invades cavernous sinus