It is possible of course that a method may some day be evolved whereby a Gasserian neurinoma or meningioma, even after it has crossed the ridge, may be safely approached and removed. Should this come to pass, it will be another conquest for neurosurgery.—Harvey Cushing

The treatment of trigeminal schwannomas is fast approaching the fulfillment of Dr. Cushing’s prediction. With the advent of microsurgical techniques and skull base approaches, a remarkable improvement has been seen in outcomes in patients undergoing surgical treatment for trigeminal schwannomas, a trend that parallels that of patients with vestibular schwannomas. Consequently, the emphasis of treatment has shifted to the patient’s quality of life, with the aim to recover or preserve cranial nerve function. In this report we focus on cranial nerve function and examine the eventual outcome of cranial nerve deficits, whether they are present preoperatively or develop postoperatively.

Object. As in patients with vestibular schwannomas, advances in surgical procedures have markedly improved outcomes in patients with trigeminal schwannomas. In this article the authors address the function of cranial nerves in a series of patients with trigeminal schwannomas that were treated with gross-total surgical removal. The authors emphasize a technique they use to remove a dumbbell-shaped tumor through the expanded Meckel cave, and discuss the advantage of the extradural zygomatic middle fossa approach for total removal of tumor and preservation or improvement of cranial nerve function.

Methods. Within an 11-year period (1989–2000), 25 patients (14 female and 11 male patients with a mean age of 44.4 years) with benign trigeminal schwannomas were surgically treated by the senior author (O.A.) with the aim of total removal of the tumor. Three patients had undergone previous surgery elsewhere. Trigeminal nerve dysfunction was present in all but two patients. Abducent nerve paresis was present in 40%. The approach in each patient was selected according to the location and size of the lesion. Nineteen tumors were dumbbell shaped and extended into both middle and posterior fossae. All 25 tumors involved the cavernous sinus. The zygomatic middle fossa approach was particularly useful and was used in 14 patients. The mean follow-up period was 33.12 months. In patients who had not undergone previous surgery, the preoperative trigeminal sensory deficit improved in 44%, facial pain decreased in 73%, and trigeminal motor deficit improved in 80%. Among patients with preoperative abducent nerve paresis, recovery was attained in 63%. Three patients (12%) experienced a persistent new or worse cranial nerve function postoperatively. Fifth nerve sensory deficit persisted in one of these patients, sensory and motor dysfunction in another, and motor trigeminal weakness in the third patient. In all patients a good surgical outcome was achieved. One patient died 2 years after treatment from an unrelated cause. In three patients the tumors recurred after an average of 22.3 months.

Conclusions. Preservation or improvement of cranial nerve function can be achieved through total removal of a trigeminal schwanna, and skull base approaches are better suited to achieving this goal. The zygomatic middle fossa approach is particularly helpful and safe. It allows extradural tumor removal from the cavernous sinus, the infratemporal fossa, and the posterior fossa through the expanded Meckel cave.

KEY WORDS • cranial nerve • cavernous sinus • Meckel cave • trigeminal schwannoma • cystic tumor • brain neoplasm • skull base surgery

Abbreviations used in this paper: CT = computerized tomography; MR = magnetic resonance; SPECT = single-photon emission CT.
Clinical Material and Methods

Within an 11-year period (1989–2000), a series of 25 patients with histologically verified benign trigeminal nerve schwannomas underwent surgery performed by the senior author (O.A.). Data from some of these patients were included in a previous publication on the overall treatment of tumors of the cavernous sinus.14 Three patients were referred to our department because they harbored a growing residual tumor after they had undergone a previous subtotal resection elsewhere. All patients underwent detailed general, ophthalmological, and neurological examinations. Audiograms were obtained in 12 patients.

Neuroimaging evaluation of these patients included preoperative MR imaging, CT scanning, and, early in the series, cerebral angiography with cross-compression studies or a balloon-occlusion test with SPECT scanning to evaluate cerebral collateral circulation. Magnetic resonance angiography replaced angiography later in the series.

All patients underwent surgical excision aimed at total removal of the tumor. Intraoperative monitoring included recording of brainstem auditory evoked responses and somatosensory evoked potentials with median nerve stimulation. Electromyographic recordings from the appropriate muscle were obtained for monitoring the third, fifth, sixth, and seventh cranial nerves. When necessary, the lower cranial nerves were monitored using electromyography.

The postoperative evaluation and follow-up review of patients involved general and neurological examinations. A CT scan was obtained early after surgery. Within 1 week after surgery MR imaging was usually performed to evaluate the extent of tumor excision. Additional MR images were obtained approximately 6 months after surgery and, later, annually to detect any recurrence. The pathological examination included intraoperative frozen section and formalin-fixed specimens to which routine and special stains were applied.

Results

This series consists of 14 female and 11 male patients. The mean age of the patients was 44.4 years (range 11–80 years), and the peak incidence of tumor discovery occurred during the patient’s fourth decade. Table 1 lists the clinical findings in the 25 patients. The mean duration of symptoms before treatment (known for 22 patients) was 18.5 months (range 2–96 months).

Signs and Symptoms

At the time of surgery, all but two patients manifested trigeminal nerve dysfunction (sensory, motor deficit, or pain). Fourteen patients experienced facial pain, four of whom displayed the typical tic douloureux. The other 10 patients

<table>
<thead>
<tr>
<th>Case No.</th>
<th>DOS (mos)</th>
<th>Facial Pain</th>
<th>Sensory Deficit</th>
<th>TM Deficit</th>
<th>Corneal Reflex Deficit</th>
<th>Other CN</th>
<th>Cerebellar Deficit</th>
<th>Brainstem Deficit</th>
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<td>1</td>
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<td>+</td>
<td>–</td>
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<td>+</td>
<td>–</td>
<td>–</td>
</tr>
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<td>+</td>
<td>–</td>
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<td>VI</td>
<td>–</td>
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<td>+</td>
<td>+</td>
<td>–</td>
<td>–</td>
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<tr>
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<td>? –</td>
<td>V2</td>
<td>–</td>
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<td>–</td>
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</tr>
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<tr>
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<td>–</td>
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<td>+</td>
<td>VI</td>
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<td>–</td>
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<td>+</td>
<td>+</td>
<td>IV, VI</td>
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<tr>
<td>19†</td>
<td>19</td>
<td>+</td>
<td>+</td>
<td>diplopia, headache, blurred vision, ringing</td>
<td>V1–3</td>
<td>N</td>
<td>N</td>
<td>–</td>
</tr>
<tr>
<td>20†</td>
<td>? +</td>
<td>+</td>
<td>diplopia, headache, blurred vision, ringing</td>
<td>V2, V3</td>
<td>N</td>
<td>N</td>
<td>IV, VI</td>
<td>+</td>
</tr>
<tr>
<td>21†</td>
<td>2</td>
<td>–</td>
<td>–</td>
<td>ear fullness, ringing</td>
<td>N</td>
<td>N</td>
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<td>–</td>
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<td>22†</td>
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<td>retroorbital</td>
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<td>48</td>
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<td>–</td>
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<td>dizziness</td>
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<td>VIII</td>
</tr>
<tr>
<td>25†</td>
<td>5</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>–</td>
</tr>
</tbody>
</table>

* CN = cranial nerve; DOS = duration of symptoms; N = normal; TM = trigeminal motor; + = present; – = absent; ? = unknown.
† Cystic lesion.
‡ Value represents the mean duration of symptoms.

Clinical findings in 25 patients with trigeminal schwannoma*
described their pain as periorbital or retroorbital (three patients), or as facial pain with undetermined characteristics (seven patients). Nineteen patients reported having numbness along one or more divisions of the trigeminal nerve. During the physical examination, 21 (84%) of the 25 patients displayed trigeminal dysfunction. Eighteen experienced sensory disturbances, namely, hypesthesia, dysesthesia, or hypalgesia, along one or more divisions of the trigeminal nerve. One patient exhibited a reduced corneal reflex only, without reduced sensation in any of the three dermatomes, and another patient only had only fifth nerve motor weakness. Nine patients (36%) displayed a decreased or absent corneal reflex. Decreased motor function, appearing as weakness or atrophy of the temporal or masseter muscles, was seen in six patients (24%).

Thirteen patients (52%) complained of diplopia, which was a presenting symptom in three patients. Four patients (16%) exhibited a deficit of the fourth cranial nerve, and 10 patients (40%) a deficit of the sixth nerve. Two patients each (8%) displayed deficits of the ninth and 10th cranial nerves (one due to tumor and the other as a result of previous surgery), and one patient (4%) displayed a deficit of the 12th nerve (due to a previous surgery). One patient experienced episodes of obscured vision associated with reduced visual acuity and restricted visual fields, and two patients experienced visual symptoms due to an unrelated pathological condition. Six patients displayed cerebellar signs. Of the 12 patients who underwent audiography, five had normal hearing, one had a positive decay reflex on the affected side, and five had high-frequency hearing loss.

Neuroimaging Evaluation

Preoperative CT scans were obtained in 18 patients and revealed bone erosion in six (33%). Contrast enhancement demonstrated a homogeneously enhanced lesion in 10 patients. In one patient, the tumor was not apparent on CT scans and was only revealed by MR imaging studies.

Magnetic resonance images were obtained routinely in all patients. The lesion was isointense or slightly hypointense on T₁-weighted images, hyperintense on T₂-weighted images, and homogeneously enhanced after administration of contrast agent. In 10 tumors a cystic component was identified. Magnetic resonance arteriography, which was performed in 15 patients, revealed narrowing of the internal carotid artery in only one patient. Magnetic resonance venography, which was performed in 10 patients, visualized the vein of Labbé and the exact venous sinus anatomy in all 10 patients.

The results of the CT and MR imaging studies and the surgical findings helped us accurately diagnose the trigeminal schwannomas and clearly identify the tumors’ locations, extensions, and origins. The tumor was located in the middle fossa in six patients (24%) and in both middle and posterior fossae in 19 patients (76%). The lesion extended to the orbit in one patient, and the maxillary sinus and infratemporal fossa were each involved in one patient.

Early in the series, cerebral angiography was performed in seven patients and a balloon occlusion test in six. One patient could not tolerate the occlusion, and tumor blush was detected in another. In two patients angiography demonstrated displacement of the nearby vessels. In three of the patients who had undergone angiography and tolerated the balloon occlusion, SPECT was performed. Normal perfusion was seen in all three cases. In one patient CT angiography was performed to distinguish the tumor from a thrombosed aneurysm.

Surgical Procedure

The surgical approach was selected after a thorough study of each patient’s preoperative radiological findings and was tailored to each case.

Because of the large size of the tumor in most patients, its extension into both middle and posterior fossae, and the involvement of the cavernous sinus, a skull-base approach was deemed advantageous for most patients. The cranioorbital zygomatic approach was used in three patients, the petrosal or extended petrosal in six patients, and the pterional in one patient. One extensive tumor required a combination of the cranioorbital zygomatic and the petrosal approaches (Fig. 1). We used the zygomatic middle fossa approach in 14 patients and found it particularly helpful and safe. It allowed extradural removal of tumor from the cavernous sinus and removal of tumor extension in the infratemporal fossa. We removed extensions into the posterior fossa through the expanded Meckel cave without sectioning the tentorium or drilling the petrous apex. Only large caudal extensions could not be reached in this way. Thus, the anatomical basis of the procedure and the zygomatic middle fossa approach deserve a description here.

Microsurgical Anatomy of the Meckel Cave. Johann Friedrich Meckel the Younger (1781–1833) described the dural cleft that was named after him in the following manner:

. . . at the upper edge of the petrous portion of the temporal bone it (the trigeminal nerve) enters a rounded and oblong sheath of the dura matter, which generally is entirely separated from the cavernous sinus. This sheath is at first loose, but afterwards is placed strongly on its surface. It thus goes from before downward and from behind forward on the anterior face of the petrous portion of the temporal bone.32

The Meckel cave is a dural recess extending from the posterior fossa into the posteromedial portion of the middle cranial fossa, providing a natural pathway that allows access from the middle fossa to the posterior fossa. Trigeminal schwannomas expand the Meckel cave, allowing entry to the posterior fossa (Fig. 2).

The oval mouth (porus trigeminus) of the Meckel cave is situated just below the superior petrosal sinus. The cave is elongated forward, like a three-fingered glove, covering the roots, ganglion, and divisions of the fifth nerve until they reach their respective foramina. The wrist of this glove is located where the roots are transmitted. The cave extends anteriorly to a level abreast of the anterior wall of the pituitary fossa, laterally to the foramen ovale, and upward to half the height of the cavernous sinus. The trigeminal nerve carries its arachnoid sheath into the middle cranial fossa. A probe inserted into the cave along the lateral wall may reach these levels without much resistance.32 Due to this invagination of the dura of the posterior fossa under that of the middle cranial fossa, a plane of cleavage is formed laterally between the two fused layers. This plane serves as the anatomical basis for the extradural approach to the Meckel cave.32,23

Surgical Procedure via the Zygomatic Middle Fossa Approach. The initial steps for the zygomatic middle fossa
approach are similar to those of previously described procedures. The dura mater of the middle fossa is elevated from the floor of the temporal fossa to expose the middle meningeal artery, which is coagulated and sectioned. The foramen ovale and rotundum are identified. Posterior to the foramen ovale, the lesser and greater petrosal nerves are identified, and traction on the greater petrosal nerve is avoided. Proximal control of the cavernous carotid artery is obtained in the petrous carotid artery canal, as previously described.

The sheaths of the second and third divisions of the trigeminal nerve are elevated to expose the lower cavernous sinus. The outer layer of the lateral wall of the cavernous sinus is peeled medially and posteriorly to expose the tumor, which usually displaces the gasserian ganglion, expands the Meckel cave, and splays the divisions of the trigeminal nerves (Fig. 3). The tumor capsule is entered between the divisions of the trigeminal nerve or behind the ganglion. The lesion is debulked using suction and followed posteriorly into the Meckel cave. It is then carefully dissected from the roots, ganglion, and divisions of the nerve to spare fascicles that are not the origin of the tumor in a manner similar to that used for other schwannomas. After the tumor is extirpated from the middle fossa and cavernous sinus extradurally, it is followed into the cerebellopontine angle through the mouth of the Meckel cave without drilling the petrous apex or sectioning the tentorium. The mouth can be further enlarged by an upward small incision toward the superior petrosal sinus, which might require coagulation and sectioning of the most anterior portion of the superior petrosal sinus. The tumor is carefully dissected from the fifth nerve rootlets and from the brainstem and the basilar artery and its branches by maintaining intraarachnoidal dissection (Figs. 4 and 5).

Tumors that extend into the infratemporal fossa or the pterygopalatine fossa are extirpated after removal of the floor of the middle fossa between the foramen ovale and rotundum. Tumors that extend along the superior orbital fissure are excised after removal of the floor of the middle fossa between the rotundum and the opening of the superior orbital fissure. For patients harboring large tumors, judicious placement of abdominal fat in the cavernous sinus can prevent venous bleeding and a cerebrospinal fluid leak, and will facilitate follow-up MR imaging examinations. The temporal muscle is split and the partially vascularized flap is laid on the floor of the middle fossa to support the closure with vascularized tissue.

Surgical Complications and Outcome

Twenty-seven surgical procedures were performed in 25 patients over a 10-year period. All patients had a preoperative diagnosis of trigeminal schwannoma. Twenty-five patients were followed up for a mean of 3.6 years, with a range of 1 to 10 years. The surgical approach was modified in 23 patients to improve exposure and surgical outcome. The surgical outcome was assessed using the modified Rankin Scale,

Fig. 1. Preoperative (A and B) and postoperative (C and D) MR images demonstrating a giant trigeminal schwannoma, the treatment of which necessitated a combination of the cranioorbital zygomatic and petrosal approaches. Note the extensive cystic degeneration. E and F: Photographs showing that the patient has recovered from a preexisting sixth cranial nerve dysfunction and a postoperative third cranial nerve dysfunction, but still has facial hypesthesia.
patients. Three patients who had permanent cranial nerve deficits after undergoing previous surgery elsewhere were excluded, and their corresponding nerve functions were not included in the analysis of recovery. Table 2 lists postoperative improvements as they relate to preoperative findings. Preoperative deficits of all other involved cranial nerves resolved by 4 to 6 months after surgery, except for two of the eight cases of abducotent nerve deficit. All cerebellar and brainstem manifestations resolved postoperatively.

The surgically induced cranial nerve dysfunction described here includes new deficits or worsening of presenting deficits in these nerves. It occurred in a total of 11 patients (44%) in different combinations and was alleviated in all but three patients (12%). No intraoperative complications occurred. In seven patients (Cases 1, 2, 7, 8, 9, 14, and 21) trigeminal nerve function (sensory, motor, or corneal reflex) worsened after surgery. Five patients (Cases 2, 7, 8, 9, and 21) experienced numbness of the face, although two of them had not noted any numbness before surgery. In one patient (Case 7) facial pain developed after surgery. Two patients (Cases 1 and 8) displayed fifth cranial nerve motor weakness and four patients (Cases 1, 2, 14, and 21) exhibited a decreased corneal reflex.

Sensory function returned to nearly normal in three of five patients, and the corneal reflex returned to normal in all but one patient. In two patients motor function of the fifth cranial nerve, which was affected by surgery, recovered in one and persisted mildly in the other.

Other cranial nerves were affected in four patients (Cases 7, 9, 11, and 20). These included trochlear nerve deficits in two patients (8%) that fully recovered 4 months postoperatively. Transient third nerve palsy in one patient and facial palsy in another patient observed 4 days after surgery recovered completely after 1 year. Function of the abducotent nerve was not worsened by surgery. Cerebrospinal fluid rhinorrhea occurred in one patient (4%) and resolved after lumbar drainage. One patient experienced Dilantin intoxication, but recovered completely. Frontalis muscle weakness from the peripheral frontal branches of the facial nerve over the temporal muscle occurred in four patients, but resolved during the follow-up period. Table 3 lists postoperative complications and follow-up clinical findings in all 25 patients.

Three patients (13%) experienced tumor recurrence after a mean period of 22.3 months (range 13–36 months). In the first patient regrowth was identified 18 months after total tumor removal. This recurrence was detected on MR images and the patient underwent a repeated operation 4 years and 10 months after the first surgery. She was free from recurrence for 5 years after the second operation, but the tumor again recurred. The second patient who experienced tumor recurrence exhibited ataxia and diplopia 3 years after total resection of tumor. The recurrent tumor was extirpated radically, and he was still free from recurrence 1 year after surgery. In the third patient tumor recurrence was revealed by MR imaging 13 months after surgery. There were no clinical manifestations of the disease and, therefore, the patient was followed up for 2 years until he died of an unrelated condition at the age of 82 years. In four patients, postoperative MR images demonstrated a small area of increased enhancement that did not change on subsequent MR images. These areas are believed to be postoperative changes, but we continue to follow them closely by performing MR imaging examinations.

Discussion

First reported by Dixon in 1846, schwannomas that arise from the intracranial portion of the trigeminal nerve are rare, accounting for 0.07 to 0.33% of intracranial tumors and 0.8 to 8% of intracranial schwannomas.10,26,38,52 In 1952, Cuneo and Rand50 published an excellent detailed history on the treatment of these lesions and stated that, in 1927, Peet had found 63 cases in the literature. Subsequent reviews have revealed approximately 402 cases to date.5,26,52

Because of the different origins and extensions of these tumors along the trigeminal nerve, several classification systems have been suggested that have implications on the clinical findings, surgical approach, and outcomes of patients with these tumors. Jefferson36 classified these tumors into three types: Type A, tumors located mainly in the middle fossa that arise from the gasserian ganglion; Type B, tu-
mors located predominately in the posterior fossa that arise from the root of the trigeminal nerve; and Type C, tumors with significant components in both middle and posterior fossae, which he described as dumbbell-shaped or hourglass tumors. Several modifications of this classification have been described.26,38,52

**Goal of Treatment**

Trigeminal schwannomas are overwhelmingly benign lesions; only a few malignant cases have been reported.6,10,12,17,28 Even when large, they displace surrounding neurovascular structures rather than engulf them. Therefore, total removal is believed to offer the best chance of cure,13,42 and subtotal removal is reported to be associated with higher rates of recurrence.35 Subtotal removal is also reported to be a source of postoperative bleeding from the tumor bed with associated instances of morbidity and mortality.5,28 In a review of the literature, Sindou and Pelissou40 found that total removal of the tumor had been achieved in only 50% of cases because of the close relationship of the lesion to the cerebellopontine angle, petrous apex, cavernous sinus, and important cranial nerves. Despite the emphasis on total removal in later series,31,45 this has not always been possible because of risks to vital structures.5,31,35 In their review of the literature, Samii and colleagues38 found that total or near-total removal was achieved in approximately 70% of cases during the past 15 years. Our aim in treating patients with these tumors has been to achieve total removal, even if the tumor is large and involves the cerebellopontine angle, petrous apex, or cavernous sinus (Table 4).

Early attempts to remove these tumors were associated with high rates of mortality.5 Hence, the emphasis on early detection of tumor is essential in preventing instances of morbidity and mortality.27,44 With the advent of microsurgical procedures, there has been a remarkable improve-
ment in the ability to remove trigeminal schwannomas to-
tally with low risks of mortality and morbidity.\textsuperscript{8,12,13,31,35,42,45,52}

Fifth cranial nerve deficits that developed postoperatively
have been almost universal.\textsuperscript{26} The absence of mortality or
major morbidity in our series corresponds to other excellent
results reported in recent series.\textsuperscript{12,13,42,52}

Stereotactic radiosurgery has been used recently to treat
patients with trigeminal schwannomas.\textsuperscript{19} Tumor shrinkage
was achieved in 56% of treated patients and tumor growth
arrest in 44%. No neurological deficit was observed after a
mean follow-up period of 44 months. Although the authors
recommended that stereotactic radiosurgery be used as the
primary treatment in patients with small and moderate-
sized trigeminal schwannomas, we reserve the use of radio-
surgery as adjuvant therapy for residual or recurrent tumors
that cannot be removed surgically, for patients unable to
undergo surgery and for patients who opt not to have sur-
gery. We agree with others that the best treatment for tri-
geminal neurinomas is complete microsurgical removal of
the lesion.\textsuperscript{13}

Cranial Nerve Function

Injury or permanent damage to the trigeminal branches
has been inevitable in many cases.\textsuperscript{49} Good health with tri-
geminal nerve symptoms and other neurological deficits is
considered to be a good outcome, even though injuries to
the sixth and seventh cranial nerves can be a complication
of surgery. Good health with only trigeminal nerve symp-
toms is considered to be an excellent outcome.\textsuperscript{49}

Currently, the emphasis of treatment has shifted to the
patient’s quality of life through the preservation or im-
provement of cranial nerve function, including that of the

\begin{table}[h]
\centering
\caption{Relationship between preoperative findings and
postoperative improvement*}
\begin{tabular}{|l|l|}
\hline
Preop Clinical Findings & No. of Cases W/ Postop Improvement (%) \\
\hline
facial pain & 8 of 11 (73) \\
facial numbness & 6 of 16 (38) \\
trigeminal motor deficit & 4 of 5 (80) \\
cerebellar signs & 4 of 4 (100) \\
brainstem signs & 1 of 1 (100) \\
CN deficits & \\
II & 1 of 1 (100) \\
IV & 3 of 3 (100) \\
VI & 6 of 8 (75) \\
VIII & 3 of 4 (75) \\
IX & 1 of 1 (100) \\
X & 1 of 1 (100) \\
\hline
\end{tabular}
\end{table}

* Three patients with prior surgically induced cranial nerve deficits were
excluded.
trigeminal nerve. Most patients have only a slight deficit in trigeminal function, which should remain the same or even improve. Our findings indicate that nontraumatic microsurgical dissection of the remaining fibers of the trigeminal roots, ganglion, and divisions effectively preserves or even improves trigeminal nerve function. The clinical sequence of symptoms and cranial nerve deficits is well described in the literature. The tumors in our patients fit the usual pattern of presentation. After surgery, preoperative facial numbness improved in 44% of patients, facial pain was alleviated in 73%, and trigeminal motor deficit improved in 80% of patients. The preoperative deficits of all other involved cranial nerves resolved by 4 to 6 months after surgery, except for two of the eight cases of abducent nerve deficit. All cerebellar and brainstem manifestations resolved postoperatively. Table 5 shows the effects of surgery on initial cranial nerve symptoms in major reported series. Cranial nerve deficits as a result of surgery and their clinical progress are listed in Table 6.

Our experience in this small series confirmed the reported value of neurophysiological monitoring in skull-base

---

**TABLE 3**

**Surgical intervention and follow-up results***

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Surgical Approach</th>
<th>Postop Complication</th>
<th>Clinical Findings</th>
<th>Duration (mos)</th>
<th>Tumor Recurrence (mos)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>ZMF</td>
<td>weak masseter muscle, mild frontalis muscle weakness, &amp; decreased corneal reflex</td>
<td>preop V2 &amp; V3 numbness remained, trigeminal pain improved; postop corneal reflex &amp; frontalis &amp; masseter muscles recovered</td>
<td>60</td>
<td>none</td>
</tr>
<tr>
<td>2</td>
<td>pet‡</td>
<td>weak frontalis muscle, &amp; decreased corneal reflex</td>
<td>preop facial pain persisted; postop numbness, corneal deficit, &amp; frontalis muscle recovered</td>
<td>29</td>
<td>none</td>
</tr>
<tr>
<td>3</td>
<td>ZMF</td>
<td>none</td>
<td>preop V2 &amp; V3 numbness &amp; corneal deficit persisted, pain &amp; masseter weakness improved</td>
<td>75</td>
<td>none</td>
</tr>
<tr>
<td>4</td>
<td>COZ</td>
<td>phenytoin toxicity</td>
<td>preop CN VI palsy &amp; pain improved</td>
<td>62</td>
<td>stable on MR image</td>
</tr>
<tr>
<td>5</td>
<td>ZMF (2×)</td>
<td>none</td>
<td>preop facial pain persisted</td>
<td>134</td>
<td>18, reop at 5 yrs, 2nd recurrence 5 yrs later</td>
</tr>
<tr>
<td>6§</td>
<td>pet (2×)</td>
<td>none</td>
<td>preop facial pain recovered</td>
<td>48</td>
<td>36, reop; 1 yr free from recurrence</td>
</tr>
<tr>
<td>7</td>
<td>pet</td>
<td>V1 &amp; V2 numbness, facial pain, weak CN IV</td>
<td>preop V1 &amp; V2 numbness improved; postop mild to moderate pain persisted, CN IV recovered completely</td>
<td>42</td>
<td>none</td>
</tr>
<tr>
<td>8†</td>
<td>ZMF</td>
<td>V2 &amp; V3 numbness</td>
<td>preop pain &amp; masseter muscle weakness persisted; postop V2 &amp; V3 numbness persisted</td>
<td>10</td>
<td>none</td>
</tr>
<tr>
<td>9</td>
<td>ZMF</td>
<td>V2 &amp; V3 numbness, facial palsy, headache</td>
<td>preop pain recovered; postop headache &amp; facial palsy recovered, numbness largely recovered</td>
<td>4</td>
<td>none</td>
</tr>
<tr>
<td>10§</td>
<td>COZ‡</td>
<td>none</td>
<td>preop V2 &amp; V3 numbness, decreased corneal reflex, &amp; masseter muscle weakness persisted; CN VI improved; lower CNs improved</td>
<td>60</td>
<td>none</td>
</tr>
<tr>
<td>11</td>
<td>pter</td>
<td>CN IV dysfunction</td>
<td>preop V1–3 numbness &amp; CN VI weakness persisted; postop CN IV dysfunction resolved</td>
<td>24</td>
<td>13, died 2 yrs later of other disease</td>
</tr>
<tr>
<td>12</td>
<td>COZ</td>
<td>none</td>
<td>preop V2 &amp; V3 numbness persisted, pain &amp; masseter muscle improved, CN VI palsy improved after 8 yrs of paralysis preop</td>
<td>60</td>
<td>none</td>
</tr>
<tr>
<td>13</td>
<td>pet</td>
<td>none</td>
<td>preop numbness &amp; pain improved, but retroorbital pain persisted</td>
<td>15</td>
<td>none</td>
</tr>
<tr>
<td>14§</td>
<td>pet</td>
<td>dysphasia, decreased corneal reflex</td>
<td>postop dysphasia &amp; corneal reflex fully recovered</td>
<td>20</td>
<td>none</td>
</tr>
<tr>
<td>15</td>
<td>pet</td>
<td>CSF rhinorrhea</td>
<td>preop V2 &amp; V3 numbness improved, pain &amp; CN VI dysfunction resolved, CSF leak stopped</td>
<td>58</td>
<td>none</td>
</tr>
<tr>
<td>16‡</td>
<td>ZMF</td>
<td>none</td>
<td>preop CN V &amp; VI improved</td>
<td>24</td>
<td>none</td>
</tr>
<tr>
<td>17</td>
<td>ZMF</td>
<td>mild frontalis muscle weakness†</td>
<td>preop numbness &amp; corneal reflex improved; postop frontalis branches improved</td>
<td>37</td>
<td>none</td>
</tr>
<tr>
<td>18</td>
<td>ZMF</td>
<td>none</td>
<td>preop CN V &amp; VI weakness &amp; corneal reflex recovered</td>
<td>12</td>
<td>none</td>
</tr>
<tr>
<td>19§</td>
<td>ZMF</td>
<td>none</td>
<td>preop pain improved</td>
<td>5</td>
<td>none</td>
</tr>
<tr>
<td>20§</td>
<td>COZ &amp; pet†</td>
<td>transient CN III paresis</td>
<td>preop ophthalmoplegia recovered, but CN V deficit persisted</td>
<td>24</td>
<td>none</td>
</tr>
<tr>
<td>21§</td>
<td>ZMF</td>
<td>weak frontalis muscle, V1 numbness, decreased corneal reflex</td>
<td>preop CN V numbness increased; postop frontalis branches improved</td>
<td>5</td>
<td>none</td>
</tr>
<tr>
<td>22§</td>
<td>ZMF</td>
<td>none</td>
<td>preop CN IV improved, persistent CN VI pain improved</td>
<td>7</td>
<td>none</td>
</tr>
<tr>
<td>23</td>
<td>ZMF</td>
<td>none</td>
<td>preop CN V &amp; cerebellar function improved</td>
<td>6</td>
<td>none</td>
</tr>
<tr>
<td>24</td>
<td>ZMF</td>
<td>none</td>
<td>normal</td>
<td>3</td>
<td>none</td>
</tr>
<tr>
<td>25§</td>
<td>ZMF</td>
<td>none</td>
<td>preop V1 &amp; V2 numbness; postop V2 numbness persisted</td>
<td>4</td>
<td>none</td>
</tr>
</tbody>
</table>

*Mean duration of follow up was 33.12 months during which three (15%) of 25 patients experienced tumor recurrence. The outcome in each case was good. Abbreviations: COZ = cranoorbitzygomatic; CSF = cerebrospinal fluid; pet = petrosal; pter = pterional; ZMF = zygomatic middle fossa.
† From skin flap.
‡ Underwent previous surgery elsewhere.
§ Cystic lesion.
surgery. It assists in locating cranial nerves displaced by tumor before they are inadvertently damaged and continually confirms their function during surgery. Electric stimulation is also a useful predictor of postoperative function.

Role of Skull Base Approaches

Recently, authors have used skull base approaches to treat patients with trigeminal schwannomas. In a comparison of conventional and skull base approaches, Taha and colleagues found that skull base approaches allowed better exposure of these tumors, multiple working angles with minimal brain retraction, and more complete removal without increased morbidity. They found residual or recurrent tumors in 65% of patients who had undergone conventional approaches compared with 10% of those who had undergone skull base approaches. In addition, cranial nerve dysfunction was seen in 70% of patients after conventional approaches. Yoshiida and Kawase also found that rates of total tumor removal and clinical outcomes were significantly better in patients treated via a skull base approach than in those treated via conventional approaches.

Skull base approaches were used in all but one of our patients, and gross-total resection was achieved in all patients. There was no incidence of mortality or major morbidity. Dumbbell-shaped tumors that involved both middle and posterior fossae (Type C in Jefferson’s classification and Type 4 in the classification of Lesoin and colleagues) are distinct in their clinical and surgical considerations. Some investigators have suggested that total removal of a dumbbell-shaped tumor is more difficult when the retrosigmoid and subtemporal approach is used. In most reported cases, however, the ability to remove these tumors totally was not significantly reduced (Table 4).

Dumbbell-shaped tumors were dominant in our series (19 cases), constituting 76% of all tumors. Tumors in all patients involved the cavernous sinus. Intraorbital, intramaxillary, intrasphenoidal, and infratemporal fossa extensions appeared in our series and did not preclude total removal of the tumor.

As the literature shows, patients who underwent extradural surgery did well, even as early as the 1950s. In the 1980s, surgeons mainly used an intradural approach to these tumors. Lesoin and colleagues advocated extradural approaches only for small tumors. More recently, the extradural approach has been used for extracranial tumors with intracranial extension, peripheral and gasserian lesions, and small posterior fossa tumors when an anterior transpetrosal approach is added.

This extradural tumor removal is readily achieved through the zygomatic middle fossa approach from the cavernous sinus and the infratemporal fossa. We add a zygomatic osteotomy to minimize retraction of the temporal lobe and allow better access to the anterior temporal fossa. A posterior fossa extension is removed through the expanded Meckel cave. Only large caudal extensions cannot be

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>No. of Cases</th>
<th>Pain</th>
<th>Sensory</th>
<th>Motor</th>
<th>Overall</th>
<th>CN III</th>
<th>CN IV</th>
<th>CN VI</th>
<th>Overall Diplopia</th>
<th>CN VII</th>
<th>CN VIII</th>
<th>Lower CNs</th>
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<tr>
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<td>14</td>
<td>8</td>
<td>7</td>
<td>11</td>
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<td>4</td>
<td>0</td>
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<tr>
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<td>6</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>5</td>
<td>0</td>
<td>—</td>
<td>—</td>
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<tr>
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<td>8</td>
<td>3</td>
<td>?</td>
<td>7</td>
<td>0</td>
<td>1</td>
<td>?</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
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<tr>
<td>Dolenc, 1994</td>
<td>44</td>
<td>3</td>
<td>3</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>23</td>
<td>11</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Taha, et al., 1995</td>
<td>15</td>
<td>3</td>
<td>100%</td>
<td>14</td>
<td>19%</td>
<td>5</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>2</td>
<td>2</td>
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</tr>
<tr>
<td>Day &amp; Fukushima, 1998</td>
<td>38</td>
<td>13</td>
<td>31</td>
<td>11</td>
<td>32</td>
<td>2</td>
<td>—</td>
<td>—</td>
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<td>—</td>
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</tr>
<tr>
<td>Present study</td>
<td>22†</td>
<td>11</td>
<td>8</td>
<td>16</td>
<td>7</td>
<td>5</td>
<td>4</td>
<td>—</td>
<td>—</td>
<td>0</td>
<td>0</td>
<td>3</td>
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</tbody>
</table>

* I = number of patients with improvement; N = total number of patients; — = data not available.
† Three patients with prior surgically induced cranial nerve deficits were excluded.
reached in this way. In these cases, the presigmoid petrosal approach allows access to large, dumbbell-shaped tumors and their posterior fossa components, which extend below the level of the seventh and eighth cranial nerves.

**Cystic Schwannomas**

Magnetic resonance imaging is indispensable in the diagnosis of trigeminal schwannomas and in surgical planning for patients harboring these tumors. It is particularly accurate in demonstrating cystic schwannomas, which occurred in 10 (40%) of our patients. In contrast to what has been reported for acoustic neuromas, there was no correlation between the appearance of cystic lesions on MR images and the totality of tumor removal or neurological outcomes in our series. In patients with acoustic schwannomas, a cystic component has been associated with a higher risk of facial nerve injury and postoperative hemorrhage. Cystic acoustic schwannomas have been found to be associated with larger size, a short clinical history, atypical initial symptoms, and histologically higher nuclear atypia. This was not the case in our trigeminal schwannoma series.

**Acknowledgments**

The authors are grateful to Mr. Ron Tribell for the artwork and to Mrs. Julie Yamamoto and Mrs. Amy Keeland for their editorial assistance.

**References**


**TABLE 6**

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>No. of Patients</th>
<th>CN V W/ Complications</th>
<th>Sensory N P</th>
<th>Motor N P</th>
<th>Total N P</th>
<th>CN III N P</th>
<th>CN IV N P</th>
<th>CN VI N P</th>
<th>Diplopia N P</th>
<th>CN VII N P</th>
<th>CN VIII N P</th>
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<td>6</td>
<td>3</td>
<td>—</td>
<td>—</td>
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<td>1</td>
<td>1</td>
<td>—</td>
<td>—</td>
<td>2</td>
<td>1</td>
</tr>
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<td>McCormick, et al., 1988</td>
<td>14</td>
<td>12</td>
<td>—</td>
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<td>9</td>
<td>9</td>
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<td>7</td>
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<td>4</td>
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<td>1</td>
<td>1</td>
<td>1</td>
<td>4</td>
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</tr>
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<td>44</td>
<td>44</td>
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<td>—</td>
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<td>10</td>
<td>10</td>
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<td>—</td>
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<td>8</td>
<td>—</td>
<td>—</td>
<td>63%</td>
<td>7%</td>
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<tr>
<td>Taha, et al., 1995</td>
<td>15</td>
<td>15</td>
<td>70%</td>
<td>70%</td>
<td>56%</td>
<td>56%</td>
<td>—</td>
<td>—</td>
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<td>63%</td>
<td>7%</td>
</tr>
<tr>
<td>Day &amp; Fukushima, 1998</td>
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<td>38</td>
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<tr>
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<td>27</td>
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<tr>
<td>Present study</td>
<td>25</td>
<td>11</td>
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<td>4</td>
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<td>1</td>
<td>7</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>

* N = total number of patients with postoperative cranial nerve dysfunction; P = number of patients with permanent deficits.
Trigeminal schwannomas


