Facial nerve injury in acoustic neuroma (vestibular schwannoma) surgery: etiology and prevention

PRAKASH SAMPATH, M.D., MICHAEL J. HOLLIDAY, M.D., HENRY BREM, M.D.,
JOHN K. NIPARKO, M.D., AND DONLIN M. LONG, M.D., PH.D.

Departments of Neurological Surgery and Otolaryngology–Head and Neck Surgery, The Johns Hopkins Hospital, Baltimore, Maryland

✔ Facial nerve injury associated with acoustic neuroma surgery has declined in incidence but remains a clinical concern. A retrospective analysis of 611 patients surgically treated for acoustic neuroma between 1973 and 1994 was undertaken to understand patterns of facial nerve injury more clearly and to identify factors that influence facial nerve outcome.

Anatomical preservation of the facial nerve was achieved in 596 patients (97.5%). In the immediate postoperative period, 62.1% of patients displayed normal or near-normal facial nerve function (House–Brackmann Grade 1 or 2). This number rose to 85.3% of patients at 6 months after surgery and by 1 year, 89.7% of patients who had undergone acoustic neuroma surgery demonstrated normal or near-normal facial nerve function.

The surgical approach appeared to have no effect on the incidence of facial nerve injury. Poor facial nerve outcome (House–Brackmann Grade 5 or 6) was seen in 1.58% of patients treated via the suboccipital approach and in 2.6% of patients treated via the translabyrinthine approach. When facial nerve outcome was examined with respect to tumor size, there clearly was an increased incidence of facial nerve palsy seen in the immediate postoperative period in cases of larger tumors: 60.8% of patients with tumors smaller than 2.5 cm had normal facial nerve function, whereas only 37.5% of patients with tumors larger than 4 cm had normal function. This difference was less pronounced, however, 6 months after surgery, when 92.1% of patients with tumors smaller than 2.5 cm had normal or near normal facial function, versus 75% of patients with tumors larger than 4 cm.

The etiology of facial nerve injury is discussed with emphasis on the pathophysiology of facial nerve palsy. In addition, on the basis of the authors' experience with these complex tumors, techniques of preventing facial nerve injury are discussed.

KEY WORDS • facial nerve • acoustic nerve tumor • vestibular schwannoma • injury • outcome

ACOUSTIC neuromas are considered by many surgeons to be one of the most difficult brain tumors to remove without producing dysfunction;[16,18,19] successful removal has long been considered a neurosurgical tour de force. Ever since Sir Charles Ballance first successfully resected an acoustic neuroma in 1894,[7] surgical techniques have been continuously refined to reduce patient morbidity and mortality rates. Over the last quarter century acoustic neuroma surgery has undergone immense changes[6] and the focus of surgery has shifted from prolongation of life to the preservation of cranial nerve function.[5,41,42,44,45]

In recent years, with advances in intraoperative cranial nerve monitoring,[11,26,30,32] considerable effort has been placed on preservation of both facial and cochlear nerve function.[36,44,45,49] Loss of facial nerve function is a debilitating and psychologically devastating condition. According to the Acoustic Neuroma Association, facial nerve dysfunction remains the number one concern among patients undergoing cerebellopontine angle surgery.[35,40,47] Given the functional and psychological effects of facial nerve dysfunction, it is important to identify perioperative factors that influence facial nerve outcome.

In this paper, we review our experience with 611 patients who underwent acoustic neuroma surgery performed by a single surgical team. Our goals were to determine the rate of facial nerve transection, the incidence of postoperative palsy, and to correlate facial nerve outcome with tumor size, surgical approach, and intraoperative facial nerve injury. We focus on the pathophysiology and prevention of facial nerve injury before, during, and after surgery. We conclude that with appropriate perioperative and intraoperative management, facial nerve injury from acoustic neuroma surgery can be minimized, with a very low incidence of poor facial nerve outcome (1.3% currently). We believe, given the preservation of facial nerve function, the concomitant low morbidity rate, and the low incidence of recurrence, surgery should remain the first line of treatment for most people with acoustic neuromas.

Clinical Material and Methods

This study focuses on 611 patients surgically treated for acoustic neuroma at The Johns Hopkins Hospital between September 1973 and June 1994. Patient charts, operative notes, follow-up clinic notes, and radiographic studies and

TABLE 1
House–Brackmann facial nerve grading scale

Grade	Description	Characteristics at Rest	Characteristics in Motion
1: normal	normal facial function in all muscles	normal	normal
2: mild dysfunction	slight weakness noticeable only on close inspection	normal symmetry & tone	no synkinesis, contracture, or hemi-facial spasm
3: moderate dysfunction	obvious, but not disfiguring weakness; no functional impairment	normal symmetry & tone	noticeable, but not severe synkinesis or contracture, hemifacial spasm, or both
4: moderately severe dysfunction	obvious weakness, disfiguring asymmetry; positive functional impairment	normal symmetry & tone	obvious synkinesis or mass action, hemifacial spasm
5: severe dysfunction	only barely perceptible motion	asymmetry w/ droop at corner of mouth & decreased nasal labial fold	synkinesis, contracture, & hemifacial spasm usually absent
6: total paralysis	no motion	no tone, asymmetry; no synkinesis, contracture, or hemifacial spasm	

reports were carefully reviewed. All patients who had pre-operative facial nerve dysfunction, schwannoma of other cranial nerves, previous acoustic neuroma surgery, other pathology, or indefinite follow-up findings were excluded from the study.

Each patient's facial nerve function was assessed in the immediate postoperative period (at the time of discharge from the hospital) and at 6-month and 1-year follow-up intervals using the House–Brackmann facial nerve out-come scale[20] (Table 1). For patients who did not have a direct 1-year follow-up examination, facial nerve outcome was assessed at the 6-month follow-up visit.

All patients underwent surgery performed by a combined neurosurgical/neurootological team. The operative approach was described in detail in every case and was dictated by the size of the tumor and the patient's preoperative hearing status. In patients with absent hearing and small-to-moderate-sized tumors, a translabyrinthine approach was used.[23] In patients with preserved hearing or a moderate to large tumor size, a retrosigmoid or suboccipital approach was used.[12,37,43] In selected patients with small intracanalicular tumors and intact hearing, a middle fossa approach was used.[14,25] Facial nerve outcome as of the 6-month follow-up examination was examined with respect to surgical approach.

After 1990, 75 patients underwent quantifying preoperative magnetic resonance (MR) imaging performed with a 1.5-tesla system (Siemens, Iselin, NJ) at The Johns Hopkins Hospital. The maximum extent of the tumor was noted either by direct measurement from the MR image or from the radiographic report. Tumors were classified as small (< 2.5 cm), medium (2.5–4 cm), or large (> 4 cm). The immediate and 6-month facial nerve outcomes were examined with respect to tumor size.

Patients in whom intraoperative transection of the facial nerve occurred were identified. The patients who underwent immediate facial nerve repair (neurorrhaphy) were also noted and the facial nerve outcomes of these patients were compiled at the 1-year follow-up examination.

Results

Table 2 provides an overview of 611 patients who underwent acoustic neuroma surgery at The Johns Hopkins Hospital between September 1973 and June 1994.

There was similar gender distribution, with 317 females (51.9%) and 294 males (48.1%) in the series. The mean age of the patients was 51.4 years and the median age was 48.5 years (range 14–83 years). There was one perioperative death (0.16%) early in the series. Six patients experienced life-threatening morbidity (0.98%): four patients had cerebellar hemorrhages that required evacuation, one had a capsular hemorrhage of unknown cause, and one patient had a postoperative cerebrovascular accident. Five of these patients made a good recovery; one (0.16%) remained hemiparetic.

Postoperative cerebrospinal fluid (CSF) leaks, which usually manifested as rhinorrhea, were seen in 154 patients (25.2%), but only six (0.98%) required reoperation for CSF closure. Ten patients (1.64%) developed meningitis and were treated successfully with antibiotic agents alone. The CSF leaks resolved in the overwhelming majority of patients (95.6%) with observation, serial lumbar punctures, or lumbar drainage.

All patients underwent gross-total resection of their tumors, except one elderly patient who had planned subtotal resection. Follow-up imaging (either MR or computerized tomography) was performed annually for 5 years in every patient. Tumor recurrence was observed in five patients (0.82%) during the course of the study.

In 596 patients (97.6%) the facial nerve was anatomically preserved. Fifteen patients (2.45%) were noted to have facial nerve transection after the tumor was removed; eight (1.3%) of these underwent immediate end-to-end repair. Of the eight patients in whom facial nerve repair was demonstrated 1 year after surgery, five patients had House–Brackmann Grade 3 or 4, whereas three patients continued to have a poor facial functional outcome (House–Brackmann Grade 5 or 6).

In the series, 72.7% of the patients underwent acoustic neuroma resection via a suboccipital approach, 25.5% via a translabyrinthine approach, and 1.8% via the middle fossa approach. When facial nerve outcome was examined with respect to surgical approach, the incidence of poor facial nerve function (House–Brackmann Grade 5 or 6) at least 6 months after surgery was 1.58% (seven patients) via the suboccipital approach, and 2.5% (four patients) via the translabyrinthine approach.

As graphically demonstrated in Fig. 1, facial nerve outcomes change over time. Immediately after surgery,

TABLE 2

Characteristics of 611 patients who underwent acoustic neuroma surgery at The Johns Hopkins Hospital, 1973–1994

Characteristic	No. of Patients (%)
total no. of patients	611 (100)
sex	
female	317 (51.9)
male	294 (48.1)
age (yrs)	
mean	51.4
range	14–83
median	48.5
tumor recurrence*	5 (0.82)
death	1 (0.16)
complications	
serious surgical morbidity†	6 (0.98)
transient CSF leak (rhinorrhea)	154 (25.2)
operative CSF leak repair	6 (0.98)
meningitis	10 (1.64)
facial nerve preservation	
anatomically preserved	596 (97.54)
transected	15 (2.45)
immediate repair	8 (1.3)
operative approach	
translabyrinthine	156 (25.5)
suboccipital	444 (72.7)
middle fossa	11 (1.8)

  * Recurrent tumor found on imaging study. Follow-up imaging: mean 8.7 years; range 1–21 years.
  † Includes one cerebrovascular accident and four cerebellar and one supratentorial capsular hemorrhages.

52.4% of patients had normal facial nerve function, 9.7% had House–Brackmann Grade 2, 20.1% had Grade 3 or 4, and 17.8% had Grade 5 or 6 (Fig. 1 *upper*). Seventy-five patients were evaluated at 6 months. At that time 85.3% of the 75 patients had House–Brackmann Grade 1 or 2, 9.3% Grade 3 or 4, and 5.3% Grade 5 or 6 (Fig. 1 *center*).

The remaining 536 patients were evaluated at 1 year postsurgery; 89.7% of them had House–Brackmann Grade 1 or 2, 8.9% Grade 3 or 4, and 1.3% Grade 5 or 6 (Fig. 1 *lower*).

Figure 2 demonstrates how facial nerve outcome correlated with tumor size immediately after surgery (Fig. 2 *upper*) and 6 months later (Fig. 2 *lower*) in 75 patients. Immediately postsurgery, of the 51 patients who had tumors less than 2.5 cm in maximum diameter, 60.8% had normal facial nerve function, 3.9% had House–Brackmann Grade 2, 21.6% Grade 3 or 4, and 13.7% Grade 5 or 6. Of the 16 patients with tumors sized between 2.5 cm and 4 cm, 25% had normal facial nerve function, 6.25% had House–Brackmann Grade 2, 25% Grade 3 or 4, and 43.75% Grade 5 or 6. Of the eight patients with tumors larger than 4 cm in diameter, 37.5% had normal facial nerve function and 62.5% had House–Brackmann Grade 5 or 6.

As of the 6-month follow-up examination, 72.5% of the patients with tumors smaller than 2.5 cm had normal facial nerve function, 19.6% had House–Brackmann Grade 2, 5.9% Grade 3 or 4, and 2% Grade 5 or 6. In the patients with tumors sized between 2.5 cm and 4 cm, 31.25% had normal facial nerve function, 37.5% had House–Brackmann Grade 2, 18.75% Grade 3 or 4, and
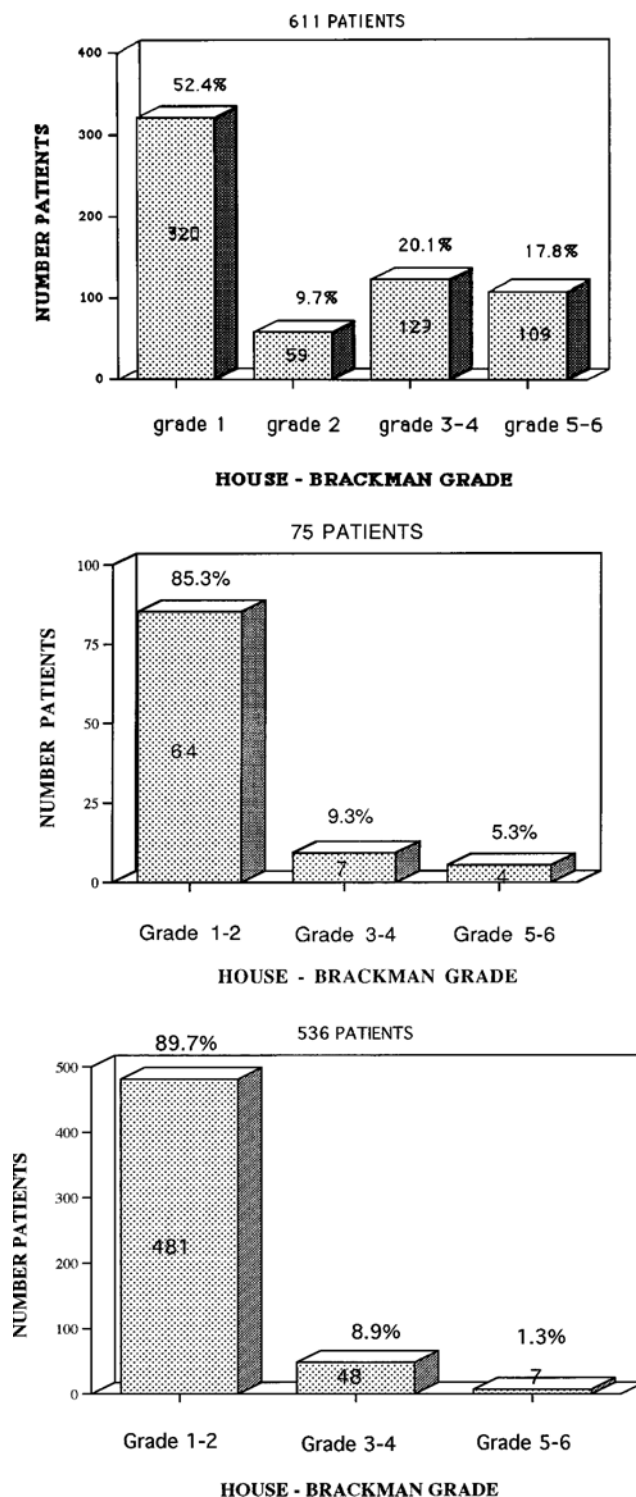

FIG. 1. Bar graphs displaying facial nerve outcome after acoustic neuroma surgery immediately postoperatively *(upper)* and at 6-month *(center)* and 1-year *(lower)* follow-up reviews.

12.5% Grade 5 or 6. In the patients with tumors larger than 4 cm, 50% had normal facial nerve function, 25% had House–Brackmann Grade 2, 12.5% Grade 3 or 4, and 12.5% Grade 5 or 6.
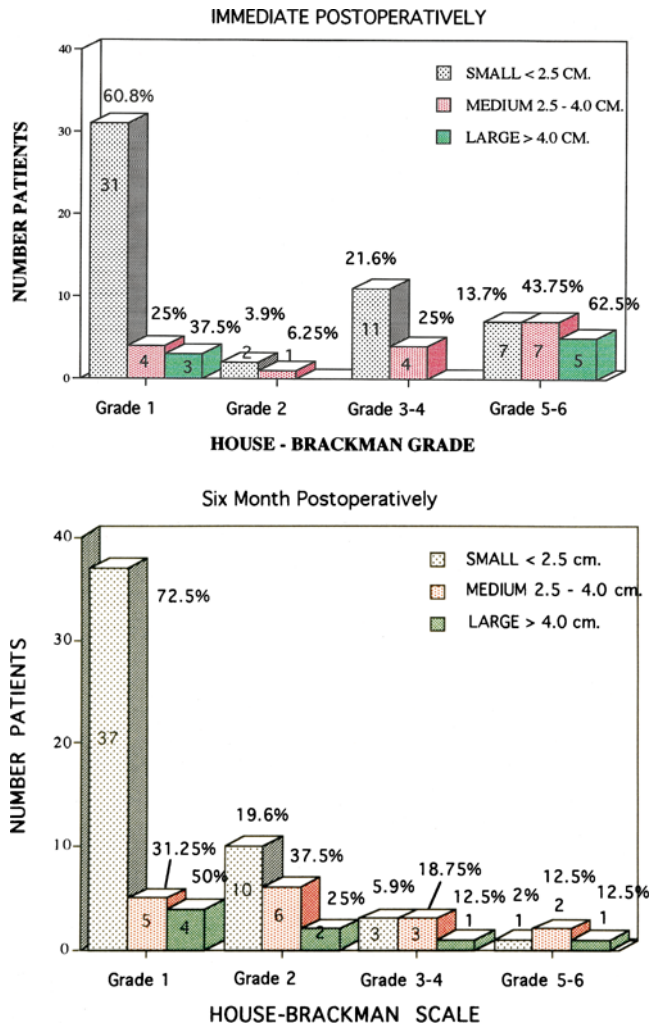
FIG. 2. Bar graphs depicting the correlation between facial nerve palsy and tumor size after acoustic neuroma surgery immediately postoperatively *(upper)* and at 6-month *(lower)* follow-up review.

Discussion

Early History of Acoustic Neuroma Surgery

In the early part of this century most patients with acoustic neuromas presented with very large tumors and significant brainstem compression. The aim of surgery was to decompress the posterior fossa and, not surprisingly, surgical mortality was over 80%. In 1917 Cushing[7] advocated subtotal resection of acoustic tumors and with improved surgical technique and hemostasis was able to lower perioperative mortality to 20%.[22]

Dandy[8] was the first American surgeon to perform total resection of acoustic tumors successfully and the first to describe the unilateral approach to the cerebellopontine angle.[9] Because many patients at this time presented with facial palsy, postoperative facial nerve paralysis was a uniform complication. Indeed, Dandy[10] himself wrote that "paralysis of the facial nerve must usually be accepted as a necessary sequel of the operation." This attitude pre-

vailed until the latter half of this century; it is only in recent decades that emphasis has been placed on preserving facial nerve function.

In 1931 Cairns[4] was the first surgeon to document preserved facial nerve function. It was not until 1940, however, when Olivecrona[39] employed an operating room nurse to observe facial twitching during tumor resection, that any systematic attempt was made to preserve the anatomy of the facial nerve.

Introduction of the Operating Microscope

In 1961, House[21] pioneered a new era in acoustic neuroma surgery with the introduction of the operating microscope. Translabyrinthine and middle fossa approaches to the internal auditory canal, which had been described years before, were refined and greatly enhanced the surgeon's ability to dissect tumor while preserving facial nerve function.[24] Between 1961 and 1968, House and Leutje[24] reported a 72% rate of preserved facial nerve function in 141 consecutively treated patients. Rand and Kurze[41] were among the first neurosurgeons to use the operating microscope for the suboccipital approach to these tumors. They demonstrated, along with Atkinson,[2] the importance of preserving the anterior inferior cerebellar artery during surgery. Yaşargil[48] later systematized microdissection of acoustic tumors.

Intraoperative Facial Nerve Monitoring

Following the widespread application of the operating microscope to acoustic neuroma surgery, the incidence of postoperative facial nerve palsy decreased dramatically. However, even the best results still revealed a 40 to 50% rate of poor postoperative facial nerve function.[6,23,29,44] The most recent advance in preserving facial nerve function was the introduction of intraoperative facial nerve monitoring by Delgado, et al., in 1979.[11] Since that time intraoperative monitoring techniques have rapidly evolved and now enable the surgeon to obtain instantaneous feedback on facial nerve firing during tumor dissection.[28,33]

Today, with advances in microsurgical techniques and intraoperative monitoring, the incidence of facial nerve paralysis has declined dramatically. Since the era of facial nerve monitoring began, many reports have included descriptions of facial nerve outcome.[1,3,12,18,24,33,36,37,44,46] Although almost all authors describe 90 to 95% anatomical preservation of the nerve, most report approximately 80% functional preservation (Table 3). This is a vast improvement from the early days of acoustic neuroma surgery, but still leaves many patients with disfiguring facial motor deficits.

The Johns Hopkins Experience

In the present series, 90% of patients displayed normal or near-normal facial nerve function (House–Brackmann Grade 1 or 2) 1 year after surgery (Fig. 1 *lower*) and all but 1.3% of patients had at least moderate function. However, immediately postoperatively, only 62.1% of patients demonstrated normal or near-normal function (Fig. 1 *upper*). Clearly a subset of patients develop transient facial nerve palsy in the immediate postoperative period only to recover function slowly over the next several months. The

TABLE 3
*Review of outcomes in acoustic neuroma surgery**

Authors & Year	No. of Patients	Anatomical Preservation (%)	Excellent Facial Outcome (%)†	Total Tumor Removal (%)
House, 1979	500	96.6	48	93.4
Glasscock, et al., 1986	616	82	NA	99
Benecke, et al., 1987	40	92	83	94
Sterkers & Bowdler, 1988	800	94	50	NA
Tos & Thomsen, 1988	400	95	67	NA
Ebersold, et al., 1992	255	92.6	64	NA
Arriaga, et al., 1993	515	NA	87	98.6
Ojemann, 1993	410	NA	56–95‡	80
Lalwani, et al., 1994	129	99.2	90	77
Matula, et al., 1995	364	NA	93	NA
Sampath, et al., 1997 (present study)	611	97.5	89.7	99.5

* NA = not addressed in article.
† House–Brackmann Grade 1 or 2.
‡ Reported as a function of tumor size.

challenge remains to identify those patients who eventually will have poor facial nerve recovery, so that early reconstructive intervention can be initiated.

The choice of operative approach did not result in a significant difference in the incidence of facial nerve paralysis in our series. Several previous studies have indicated that the translabyrinthine approach to acoustic tumors allows earlier exposure of the facial nerve and, consequently, is associated with a lower incidence of facial nerve injury.[23,31,34] The results of our study, however, indicate that equivalent outcomes can be achieved with each approach and therefore, the operative approach should only be dictated by the size and location of the tumor, the patient's preoperative hearing function, the nerve of origin, and the experience of the surgeon.

The exception to this rule occurs when a middle fossa approach is being considered for treating inferior vestibular nerve tumors. Because these tumors can be readily identified preoperatively using electronystagmography,[29] our experience suggests that they are best approached by a retrosigmoid approach, which both preserves hearing and avoids unnecessary retraction of the facial nerve. In general, we believe the middle fossa approach is best suited for intracanalicular superior vestibular lesions.

When postoperative facial nerve outcome is examined with respect to tumor size, it is not surprising that the incidence of temporary facial nerve palsy is higher with very large tumors. This difference was particularly pronounced in the immediate postoperative period during which 62.5% of patients with tumors larger than 4 cm had a House–Brackmann Grade 3 or greater, compared with only 35.3% of patients with tumors less than 2.5 cm (Fig. 2 *upper*). However, the effect of tumor size on facial nerve outcome becomes much less prominent 6 months after surgery when 75% of patients with tumors larger than 4 cm have normal or near-normal facial nerve function and 87.5% have acceptable function (Fig. 2 *lower*). This emphasizes that, as long as the facial nerve is preserved in continuity and precautions are taken to minimize facial nerve injury perioperatively, excellent facial nerve outcomes can be achieved even with very large tumors.

Of the 611 patients who underwent acoustic neuroma resection, 15 (2.5%) had intraoperative facial nerve transection. Eight (1.3%) of these patients underwent facial nerve repair by immediate primary epineural neurorrhaphy.[13] None of these patients recovered normal facial nerve function; however, five patients did improve function to House–Brackmann Grade 3 or 4. Immediate neurorrhaphy should be attempted in all patients in whom facial nerve transection occurs. Although these patients do not regain normal function (in our series, all were House–Brackmann Grade 3 or more), regained function minimizes sequelae related to globe protection (for instance, keratoconjunctivitis) and facilitates future nerve reanimation, albeit with varying degrees of synkinesis.

Pathogenesis and Prevention

Facial nerve weakness that is evident immediately after acoustic neuroma surgery is caused by a number of possible mechanisms. A thorough understanding of these can help minimize intraoperative facial nerve injury.[27]

The most common cause of postoperative facial nerve palsy is direct trauma or nerve stretching during surgery. Theoretically, both neuropraxia and axonotmesis are reversible phenomena and facial nerve function should fully return. Not surprisingly, very large tumors place the nerve under greater tension, which increases the likelihood of stretch injury and may explain the high rate of facial palsy seen in patients with tumors larger than 4 cm. Alternatively, nerve dysfunction may result from devascularization of nerve segments that are effaced by large tumors.

Measures can be taken to minimize trauma to the facial nerve during surgery. First, the ability to compress and retract the tumor capsule rather than the nerve by debulking the tumor prior to nerve dissection is of major importance, particularly in cases of large tumors. Second, excessive pressure on the facial nerve should be avoided. Cotton and microsuction devices should be used at all times. Sharp dissection should be used until a clear dissection plane is established to avoid unnecessary stretch injury. Third, it is important to avoid excessive cerebellar retraction to minimize the tension placed on the facial nerve. Finally, it is essential that dissection proceed from known to unknown structures. If hearing preservation is not a consideration, early identification of the facial nerve near the lamina spiralis allows better appreciation of its relationship with the tumor.

Another common mechanism of facial nerve injury is compromise of the vascular supply to the facial nerve. The facial nerve is supplied by three separate vascular systems: the labyrinthine artery off the anterior inferior cerebellar artery, the greater superficial petrosal branch off the middle meningeal artery, and the stylomastoid artery off the external carotid system. Maintaining the blood supply to the facial nerve is critical if postoperative palsy is to be avoided. The surgeon must be careful to avoid inadvertent vascular injury. Bipolar cautery should be used cautiously; when possible, blunt dissection should be used near all vascular structures. Topical administration of papaverine after tumor resection can also aid in preventing vasospasm. Because most of the microvascular blood supply to the facial nerve is in the subarachnoid space, it is essential that dissection proceed in the correct plane between the

tumor capsule and the underlying arachnoid. Overly aggressive dissection of the tumor capsule from the facial nerve may strip the facial nerve of its vital microvascular supply and lead to postoperative nerve dysfunction.

Thermal injury can also cause temporary facial nerve palsy or paralysis. Overly cold irrigation may "stun" the nerve and is avoidable with use of warmed saline solutions. This phenomenon is usually transient, but occasionally it may lead to local vasoconstriction and cause secondary ischemic injury to the nerve. Thermal injury can be more permanent if the laser is used for tumor extirpation. Both potassium-titanyl-phosphate and CO_2 lasers have become increasingly popular in recent years, especially for use in treating vascular tumors.[15,17] Laser vaporization can be quite helpful in devascularizing tumor remnants to prevent recurrence, but it should be used with great caution near the facial nerve. Cool intermittent irrigation and continuous suction of the laser plume can help minimize thermal injury.

We use several preventive measures in all our patients. First, we use a perioperative course of steroid medications to minimize both cerebellar and perineurial swelling. We also advocate postoperative head elevation, appropriate intravenous hydration, and maintenance of suitable electrolytes to reduce the likelihood of cerebral and neural swelling. Meticulous attention should also be given to bone-waxing all mastoid air cells and ensuring a tight dural closure to avoid postoperative CSF leak. If a CSF leak does occur we routinely use serial lumbar puncture or lumbar drainage aggressively.

If the facial nerve is inadvertently transected during surgery, restoration of facial nerve function becomes much more problematic. Our experience suggests that facial nerve disruption generally leads to poor outcome even with apparently satisfactory immediate repair. In our series the patients were only able to recover to House–Brackmann Grade 3 at best. We encourage close follow-up review of these patients and consider facial nerve reanimation (either 12th–seventh or 11th–seventh cranial nerve anastomosis) if there is no evidence of recovery by 12 months postoperatively. After 1 year, facial nerve function with reanimation surgery is much less predictable.

Conclusions

Facial nerve weakness is the complication of most concern for patients undergoing acoustic neuroma surgery. Advances in anesthesia, surgical technique, and postoperative care have made morbidity and mortality from cerebellopontine angle surgery extremely low, allowing surgeons to focus on preservation of cranial nerves. With appropriate perioperative management and a more thorough understanding of mechanisms of facial nerve injury, facial nerve dysfunction after acoustic neuroma surgery can be minimized.

Although immediate postoperative palsies are not uncommon, ultimately up to 90% of patients will regain normal or near-normal facial nerve function and nearly 98% should have functionally acceptable results. The surgical approach appears to have no bearing on ultimate facial nerve paralysis. Even patients with large tumors (> 4 cm) can expect to demonstrate acceptable facial nerve out-

comes 1 year after surgery. Only patients who have intraoperative facial nerve transection can expect to have poor function and should consider early facial reanimation strategies. This occurred in 2.45% of our patients.

This large cohort study is of importance to neurosurgeons, particularly in light of emerging treatment options such as gamma knife surgery. Despite recent advances in radiosurgery, given the overall facial nerve outcome, the exceedingly low mortality and morbidity rates, and the low incidence of tumor recurrence, we believe that surgery should remain the first choice of treatment for most patients with acoustic tumors. The experience of the surgeon, appropriate perioperative care, anesthesia, and electrophysiological monitoring all contribute to achieving optimum patient outcomes. The challenges for the future are to minimize the incidence of postoperative facial nerve palsy and to identify patients who may have poor long-term facial nerve recovery after surgery, so that reanimation can be considered in a timely fashion.

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