ELSEVIER
SAUNDERS

Neurosurg Clin N Am 19 (2008) 379–392

NEUROSURGERY
CLINICS
OF NORTH AMERICA

Guiding Patients Through the Choices for Treating Vestibular Schwannomas: Balancing Options and Ensuring Informed Consent

Douglas D. Backous, MD, FACS[a,*], Huong T. Pham, MD[b]

[a]*Otology, Neurotology and Skull Base Surgery, Section of Otolaryngology-Head and Neck Surgery, Virginia Mason Medical Center, 1100 Ninth Avenue, X10-0N, Seattle, WA 98111-0900, USA*
[b]*Section of Radiation Oncology, Virginia Mason Medical Center, P O. Box 900, Seattle, WA 98111-0900, USA*

Counseling patients who are diagnosed with vestibular schwannomas (VS), formerly known as acoustic neuromas, can be challenging. These benign neoplasms, which originate from either the superior or inferior vestibular nerves, have an average growth rate of 1 mm^3 per year. Larger tumors can cause brainstem compression with few noticeable symptoms, whereas smaller VS can cause vertigo, tinnitus, and hearing loss.

Patients are confronted by the difficult task of choosing a treatment method based on the advice of caregivers, currently available literature, and Internet-based information sources. Patients often visit the Internet either before or during their decision period, which can be helpful or even more confusing for them as they weigh their options. The health care provider has the responsibility to explain, in understandable language, to the patient or legal representative governing the patient's care the proposed treatment options, risks and complications associated with each form of treatment, and alternatives to treatment, including no therapy. The medical record must contain evidence of the patient's informed consent, with the exception of emergency situations in which a delay in intervention could compromise outcomes in a life or limb-threatening situation. Aside from cases with brainstem compression and hydrocephalus, patients should be encouraged to gather information before making a treatment decision. For the physicians managing these patients, information should be delivered in a balanced way to ensure patient understanding of their options leading to adequate informed consent.

Options for treatment include radiation therapy, surgical excision, and observation with serial MRI. Discerning treatment advantages from a particular modality is made difficult because of nonstandardized definitions of tumor control and hearing preservation and varied posttreatment intervals presented in the medical literature. Surgical techniques and radiotherapy dosage paradigms have evolved considerably over the past two decades. Currently, no randomized, prospective clinical trial has compared the three treatment options and there are no clearly accepted, evidence-based, best practices for managing VS.

The treatment of VS requires a multidisciplinary team not only to deliver the chosen therapy but also to assist in the decision-making process. At our center, a nurse familiar with VS treatment coordinates appointments with a neurotologist, a neurosurgeon, and a radiotherapist. Patients are encouraged to take the amount of time necessary to make a decision with full understanding of potential risks and benefits. The amount of time necessary to come to a decision depends on the needs of a particular patient.

This article evaluates the English language literature, dating back to 1994, to present long-term results for tumor control and complication rates for the treatment of VS, excluding cases of

A version of this article originally appeared in Otolaryngologic Clinics of NA, volume 40, issue 3.

* Corresponding author.
E-mail address: otoddb@vmmc.org (D.D. Backous).

1042-3680/08/$ - see front matter
doi:10.1016/j.nec.2008.02.004

neurofibromatosis type II. The goal is to provide a guide to otolaryngologists who provide initial counseling for patients with newly diagnosed VS.

Stereotactic radiation therapy

Radiation therapy was initially used as an adjunct to surgery in patients with incompletely resected VS. In a study reported from University of California San Francisco, Wallner and colleagues [1] demonstrated that conventional fractionated radiation therapy to more than 45 Gy significantly reduced regrowth from 46% to 6% in incompletely resected VS. In this series, 31 patients treated between 1945 and 1983 were followed from 2.6 to 40.7 years. The authors concluded that postoperative radiation therapy reduced the "local recurrence rate" of VSs that were incompletely excised or only biopsied and demonstrated the effectiveness of radiation therapy in the treatment of acoustic neuromas.

Stereotactic radiosurgery, first developed in 1951 by Leksell [2], is a method of delivering a highly conformal single dose of ionizing radiation with submillimeter accuracy to an intracranial target. The goal of a single high-dose delivery was to cause tumor necrosis and control of growth as an alternative to surgery for patients who were suboptimal candidates for excision. The first stereotactic radiosurgery treatment for VS was performed in 1969 with the gamma knife (GK), also developed by Leksell [3]. The GK is a highly specialized radiation delivery system that uses 201 radioactive cobalt 60 sources to deliver high-dose radiation accurately to tumor masses. A stereotactic frame is fixed to the skull and attached to the treatment table to provide rigid immobilization of the patient's head and ensure accurate localization of the radiation dose.

Thousands of patients have been treated with the GK. Initially, doses were high, and although tumor control was excellent, toxicity was significant [4–6]. Several refinements, including dose reduction, improved target definition, and treatment accuracy, have provided excellent tumor control while minimizing toxicities of treatment [7,8]. Lower radiation doses were shown to be effective in controlling tumor growth, whereas tumor shrinkage could take several years to document radiographically.

The linear accelerator (LINAC) also can be adapted to perform stereotactic radiosurgery [9]. Multiple beam positions or arcs are used to create a conformal dose distribution around the target. Although the tumor margin doses are similar to the GK, LINAC radiosurgery typically uses fewer isocenters and the dose to the tumor is more homogeneous. Similar to the GK, a non-relocatable invasive head frame is required for patient immobilization during treatment.

Fractionated stereotactic radiation therapy is the most recently developed technique for delivering high-dose and localized treatment. Unlike with GK or LINAC, a noninvasive relocatable head frame or thermoplastic mask is used for fractionated stereotactic radiation therapy. This head frame increases patient comfort but may result in less dose conformality when compared with other radiosurgery techniques. Subscribers of this technique believe that fractionation takes advantage of radiobiologic principles to reduce late toxicity while maintaining tumor control [10,11]. The fractionated treatment regimens range from doses given over several days to standard fractionation given over 4 to 5 weeks, similar to the scheme originally used by Wallner.

Data regarding the outcomes GK, LINAC, and FSRT are reviewed in the next section. Because few centers in the world offer proton beam radiation therapy to treat VS, studies that used proton-based treatments were excluded. All pertinent papers published from 1994 to the present that assess local control rates with a median follow-up of at least 2 years were reported. Outcomes included tumor control, hearing preservation, facial neuropathy, and trigeminal neuralgia. In most patients, tumors were sporadic and had a maximum diameter of $\leq$3 cm.

The goal of radiation therapy is to arrest tumor growth. Local control rate in radiotherapy studies can be defined as the percentage of tumors that do not increase in size on follow-up imaging. Many researchers define the local control rate as the percentage of tumors that do not require salvage therapy. This determination could overestimate the control rate because some tumors may have progressed but are not symptomatic enough to require further treatment. All three techniques seem to achieve excellent local control with a range of 87% to 100% (Tables 1–3) [8,10,12–45].

Up to 50% of radiated tumors developed central necrosis that results in transient increase in tumor volume (23% of cases) [12]. This phenomenon was observed up to 4 years after treatment and took from 6 months to 5 years to disappear. If tumor progression was defined as tumor growth, then some patients may have

Table 1
Gamma knife radiosurgery outcomes (median marginal dose 12–14 Gy)

Author (year)	Number of patients	Local control rate	Hearing preservation rate	Facial neuropathy rate	Trigeminal neuropathy rate	Other complications
Flickinger, 2004 [14]	313	98.6% @ 6 y	78%@ 6 y	0% @ 6 y	4.4% @ 6 y	
Andrews, 2001 [10]	63	98%	33%	2%	5%	hc 2.9% vertigo 1.4% ataxia 1.4%
Massager, 2006 [15]	82	98%	65%			
Ottaviani, 2002 [16]	30	87%	73%	3% temp	16% mild symptoms	
Wackym, 2004 [17]	29	94%		0%	3% temp	
Prasad, 2000 [12]	153	92%	75% for tumors <1 cc, 57% for tumors >1 cc	2.3%	1.7% perm, 2.5% temp	
Chung, 2005 [18]	195	95% @ 7 y	60%	1.4% temp	1.1%	hc 3.5%
Wowra, 2005 [19]	111	95% @ 6 y		2.7%	11.5%	
Lundsford, 2005 [8]	829	97% @10 y	78.6%	0%	4.4%	ventriculoperitoneal shunt 0.8%, tinnitus 0.2%, peritumoral cyst 3.6%, no radiation-induced malignancy
Petit, 2001 [20]	45	96%	88%	4% temp	0%	
Hasegawa, 2005 [13]	317	92% @10 y	68%	1% temp	2%	hc 4.1%, malignant transformation 0.3%
Pollock, 2006 [21]	46	100%	63%	4%	2.1%	hc 4%
Mysreth, 2005 [22]	103	89.2%		5.2%		hc 3.9%
Paek, 2005 [23]	25	100%	46%	0%	5%	
Litvack, 2003 [24]	134	97.7%	61.7%	2.2% temp	5.8% temp	hc 3.0%
Hirato, 1996 [25]	29	93%	59%	10.3% temp	3% temp	hc 6.8%
Muacevic, 2004 [26]	219	97%	49%	0.5%	5% temp	
Iwai, 2003 [27]	51	96%	56%	0%	4%	hc 8%
Kwon, 1998 [28]	88	95%	67%	4% temp, 4% perm	3.4% temp	hc 3.5%

Hearing preservation rate is the percentage of patients who maintained or gained useful hearing (Gardner-Robertson class 1-2) after treatment.
Abbreviations: perm, permanent; hc, hydrocephalus; temp, temporary.

Table 2
LINAC stereotactic radiosurgery outcomes (median marginal dose 12–14 Gy)

Author (year)	Number of patients	Local control rate	Hearing preservation rate	Facial neuropathy rate	Trigeminal neuropathy rate	Other complications
Friedman, 2006 [7]	390	90% @ 5 y, only 1% required surgery		4.4% (0.7% w/dose ≤12.5 Gy)	3.7% (0.7% w/dose ≤12.5 Gy)	
Chung, 2004 [29]	45	100%	all patients deaf before tx	4.4% perm	7.5% perm	hc 4.4%
Combs, 2006 [30]	26	91% @ 10 y	55%	5% perm, 15% temp	8%	1 patient with radionecrosis of cerebellum
Meijer, 2003 [31]	49	100%	75%	7%	8%	hc 2%
Speigelman, 2001 [32]	44	98%	71%	2.4% temp, 8% perm	18%	
Foote, 2001 [33]	149	95.4%		11.8% temp	9.5% temp	
Lee, 2001 [34]	42	100%	82%	2.5% perm	2.5% perm	hc 7.5%

Hearing preservation rate is the percentage of patients who maintained or gained useful hearing (Gardner-Robertson class 1-2) after treatment.
Abbreviations: hc, hydrocephalus; perm, permanent; temp, temporary.

undergone surgery for salvage treatment unnecessarily [13].

Lundsford and colleagues [8] and Hasegawa and colleagues [13] reported 10-year local control rates of more than 90% in separate series of patients treated with GK. The median follow-up period in Hasegawa's study was 7.8 years. Partial or complete radiographic response to treatment occurred in 62% of radiated tumors, and tumors <15 cm^3 had a better progression-free survival than tumors >15 cm^3 (96% versus 57%, $P < .001$). Tumors not compressing the brainstem or obstructing the fourth ventricle had a better progression-free survival (97% versus 74%, $P < .008$). Tumor progression occurred within 3 years from the time of treatment in most cases. Forster and colleagues [46] documented local control rates for tumors >3 cm, 2 to 3 cm, and <2 cm at 33%, 86%, and 89%, respectively. Similar findings were reported by Kondziolka and colleagues [4].

Friedman and colleagues [7] reviewed the outcomes of 390 patients treated with LINAC radiosurgery for VS. With a median follow-up of 40 months and a median dose of 12.5 Gy, the 5- and 10-year local control rates were 90% with only 1% of patients requiring surgery for treatment failure during the follow-up period. With a median follow-up period of 48.5 months, Combs and colleagues [30] also reported a local control rate of 91% at 10 years.

The longest follow-up periods for the FSRT studies are from Combs and colleagues [35], Sawamura and colleagues [40], and Chan and colleagues [42]. The median follow-up periods in these studies averaged 48 months, and the 5-year local control rates in all three studies are more than 90% (see Table 3).

Unlike with use of GK, tumor volume or size has not been shown to be of prognostic value in predicting response to treatment with LINAC radiosurgery or fractionated stereotactic radiotherapy for acoustic neuromas. Different fractionation regimens were used depending on tumor size [11,37].

Hearing preservation has not been documented according to a consistent standard (Table 4), and no randomized studies regarding tumor control and hearing preservation have been reported to date. The use of pure tone audiometry and discrimination testing before and at a standardized interval after treatment would be optimal. In most of the radiotherapy papers reviewed, the statistic that is most often reported is the percentage of patients who maintained or gained

Table 3
Fractionated stereotactic radiation therapy outcomes

Author (year)	Number of patients	Dose	Local control rate	Hearing preservation rate	Facial neuropathy rate	Trigeminal neuropathy rate	Other complications
Andrews, 2001 [10]	46	50 Gy/25 fx	97%	81%	2%	7%	hc 3.6%, vertigo 1.7%, ataxia 3.6%
Combs, 2005 [35]	106	57.6 Gy/32 fx	93%	98%	2.3%	3.4%	
Chung, 2004 [29]	27	45 Gy/25 fx	100%	57%	4% temp	7% temp	hc 4%
Shirato, 1999 [36]	33	36–44 Gy/20–22 fx	98%	53%	5% temp	12% temp	
Williams/Shokek, 2004 [11,37]	375	25 Gy/5 fx (<3 cm diam), 30 Gy/10 fx (>3 cm diam), 40 Gy/20 fx	97%	59%	1.5% temp 0.5% perm	1.2% temp	
Meijer, 2003 [31]	80	20–25 Gy/5 fx	94% @ 5 y	61%	3%	2%	
Fuss, 2000 [38]	42	57.6 Gy/32 fx	97.5% @ 5 y	85%	0%	4.8%	
Selch, 2004 [39]	48	54 Gy/30 fx	100%	91.4%	2.8%	3.8%	Tinnitus 5%, hc 0%, ataxia 2%
Sawamura, 2003 [40]	101	40–50 Gy/20–25 fx	97%	71	4% temp	14%	hc 11% disequilibrium 16.8%
Poen, 1999 [41]	31	21 Gy/3 fx/24 h	97%	77%	3%	16%	
Chan, 2005 [42]	70	54 Gy/30 fx	92%	84%	1%	4%	
Chang, 2005 [43]	61	21 Gy/3 fx/24 h	98%	74%	0% perm	0%	
Lederman, 1997 [44]	38	20 Gy/4 fx or 20 Gy/5 fx	100%	93.5% (does not distinguish useful from no useful hearing)	2.6% temp	0%	
Ishihara, 2004 [45]	38	15–20.5 Gy/1–3 fx	94%	93%	2.6% temp	2.6%	

Hearing preservation rate is the percentage of patients who maintained or gained useful hearing (Gardner-Robertson class 1-2) after treatment.
Abbreviations: fx, fraction; hc, hydrocephalus; perm, permanent; temp, temporary.

Table 4
Hearing classification scales

AAO-HNS classification		
Class	Pure tone average (0.5, 1, 2, 3 kHz measured in dB HL)	Speech discrimination score (%)
A	0–30	70–100
B	31–50	50–100
C	>50	50–100
D	Any	<50
Gardner-Robertson Classification		
Class	Pure Tone/Speech Reception Threshold (dB HL)	Speech Discrimination Score (%)
1	0–30	70–100
2	31–50	50–69
3	51–90	5–49
4	>90	1–4
Word Recognition Scores		
Class	Word Recognition Score (%)	
I	70–100	
II	50–69	
III	1–50	
IV	0%	

Data from Meyer TA, Canty PA, Wilkinson EP, et al. Small acoustic neuromas: surgical outcomes versus observation or radiation. Otol Neurotol 2006;27(3):380–92.

useful hearing (Gardner-Robertson classes 1 and 2). Intervals for testing hearing after treatment are not clear, and in some studies residual hearing was defined by whether patients could talk on the telephone on the side of the affected ear. Hearing loss is rare, tends to occur over 6 to 24 months, and can continue to decline for years after treatment. Prasad [12] reported a useful hearing preservation rate of 58% with a median follow-up of 4.2 years. In most patients, hearing decline occurred after 2 years and continued up to 8 years after treatment, with a hearing preservation rate of 75% for tumors $<1\ cm^3$ compared with 57% for tumors $>1\ cm^3$. Massager [15] demonstrated better useful hearing preservation rates in tumors with intracanalicular volumes $<100\ mm^3$ compared with larger tumors (82.6% versus 44.8%, $P = .045$).

Improved hearing outcome has been demonstrated at doses $\leq$13 Gy. In an early paper from the University of Pittsburg, Kondziolka and colleagues [4] reported a "useful" hearing preservation rate of 46% at an average marginal dose of 16 Gy. Later studies documented hearing preservation in up to 68% to 78% of cases treated in the range of 12 to 13 Gy [13,14]. Because there were no differences in the local control rates with the lower doses, it is common practice to prescribe 12 to 13 Gy to the tumor margin when using stereotactic radiosurgery to control VS growth and maximize hearing preservation.

Others attempt to minimize toxicity and improve hearing preservation rates by fractionating the dose. This practice is based on a radiobiologic principle that there is a direct relationship between late normal tissue damage and dose per treatment delivered to the tissue. Andrews and colleagues [10] performed a prospective, nonrandomized study to compare the outcomes of treatment for acoustic neuromas with GK radiation to FSRT. The GK dose was 12 Gy and the FSRT dose was 50 Gy in 25 fractions given daily. They found no difference in local control rates, but the hearing preservation rate was 33% for GK treatment compared with 81% for fractionated stereotactic radiotherapy. These results should be viewed with caution because they had relatively short mean follow-up of less than 3 years. Although most of the FSRT studies reported excellent hearing preservation rates, they have shorter follow-up than other radiosurgery series. The different fractionation regimens make it difficult to make any conclusions regarding dose and hearing preservation.

Facial neuropathy is a potential complication with all three treatment options for VS. Kondziolka and colleagues [4] performed a multivariate analysis of 162 patients treated with GK and found that tumor volume and dose of radiation

to the tumor margin were associated with the risk of neuropathy ($P < .001$). With an average marginal dose of 16 Gy, the overall rate of facial neuropathy was 15%. In subsequent reports, when 12 to 13 Gy were prescribed to the margin, the facial neuropathy rate dropped to 0%. Friedman and colleagues [7] presented a similar finding when they lowered their LINAC radiosurgery dose to 12.5 Gy (4.4% versus 0.7%). As seen in Tables 1 and 2, the risk of facial neuropathy with radiosurgery is minimal and often temporary with a dose of 12 to 13 Gy. Similar rates of facial neuropathy are reported with FSRT despite variable doses and fractionation regimens (see Table 3). Unfortunately, no consistent facial nerve function scale was used for reporting.

After performing a multivariate analysis, Kondziolka and colleagues [4] documented that tumor volume and radiation dosage to the tumor margin were associated with the risk of trigeminal neuropathy ($P < .001$). With average marginal doses of 16 Gy, the overall rate of trigeminal neuropathy was 16% compared with 4.4% at 12 to 13 Gy. There was no evidence of trigeminal nerve damage for intracanalicular tumors. Friedman and colleagues [7] noted a similar finding when they lowered their LINAC radiosurgery dose to 12.5 Gy (3.7% versus 0.7%).

Meijer and colleagues [31] reported the outcomes of treatment with LINAC compared with FSRT for their patients with VS. They found a statistically significant increased incidence of trigeminal neuropathy in patients treated with FSRT (8%) versus LINAC radiosurgery (2%) ($P = .048$).

Other potential side effects from radiation therapy include vertigo, tinnitus, ataxia, headache, hydrocephalus, cyst formation, radiation-induced edema or necrosis, intratumoral bleeding, and malignant transformation (see Tables 1–3). The reporting of these toxicities has not been consistent. The rate of hydrocephalus ranges from 0 to 11% [10,29,39]. Sawamura and colleagues [40] reported the highest incidence of hydrocephalus, with 11% of 101 patients treated with FSRT 40 to 50 Gy in 20 to 25 daily fractions manifesting communicating hydrocephalus that requires ventriculoperitoneal shunting. The hydrocephalus resolved in all patients with shunt placement and was assumed to be caused by cerebrospinal fluid (CSF) malabsorption associated with VS.

Delayed malignant transformation or radiation-induced malignancy may occur with radiation therapy for acoustic neuromas. Malignant transformation occurred in one patient 51 months after radiotherapy treatment. The malignant transformation rate in this study was 0.3% among patients who were followed longer than 5 years after GK [13]. Two other case reports of malignant transformation have been reported with GK [47,48]. One case was reported of a patient who developed a glioblastoma multiforme adjacent to an acoustic neuroma that was treated with GK 7.5 years earlier [49]. Another patient treated with FSRT developed a low-grade malignant nerve sheath tumor 216 months after initial treatment [50]. Delayed malignant transformation or radiation-induced malignancies are rare. Long-term, yearly follow-up of these patients provides a more accurate assessment of incidence.

Recent papers compared the outcomes of microsurgery to stereotactic radiosurgery to attempt to determine which treatment is better for sporadic VS ≤ 3 cm. Myrseth and colleagues [22] reported a retrospective study of 189 consecutive patients—86 treated by microsurgery and 103 by GK. The mean follow-up period was 5.9 years. In addition to local control and cranial nerve preservation, they also evaluated quality of life through standardized questionnaires. The overall local control rates for microsurgery and GK were 89.2% versus 94.2%, which was not statistically significant. Facial nerve function (House-Brackmann grade 1-2) was preserved in 79.8% of the microsurgery group and in 94.8% of the GK group ($P = .0026$). The middle fossa approach was not used in these patient cohorts. Overall, the quality-of-life scores were significantly lower in the microsurgery group compared with the GK group. The authors concluded that these results favored GK as the treatment of choice for this group of patients.

Pollock and colleagues [21] presented a prospective cohort study of 82 patients with unilateral, unoperated VS <3 cm in greatest dimension undergoing surgical resection ($n = 36$) or GK ($n = 46$). Other than age (patients undergoing microsurgery were younger), all other pretreatment characteristics were matched in the two treatment groups. The mean follow-up period was 42 months. There was no difference in the local control rate between microsurgery and GK (100% versus 96%, $P = .50$). They found that facial nerve preservation (96% versus 75%, $P < .01$) and serviceable hearing rates (63% versus 5%, $P < .001$) were better in the GK group than microsurgery group. With regard to quality of life, patients who underwent microsurgery

had a decline in physical functioning and bodily pain scores, whereas patients who had GK had lower Dizziness Handicap Inventory scores. All of these differences in quality of life were statistically significant. Although the authors concluded that radiosurgery should be considered the best treatment for this group of patients, they commented on the necessity of longer follow-up.

Microsurgery

Three principal approaches are used to surgically remove VS. The translabyrinthine (TL) route is used for tumors with either no hearing or in cases with little chance of preserving hearing. TL typically would be chosen in patients with less than 50% speech discrimination and 50 dB speech reception threshold, in intracanalicular tumors extending to the lateral margin of the internal auditory canal, or in tumors >3 cm in largest dimension. Two approaches, the middle cranial fossa (MCF) and retrosigmoid (RS), are used in cases with potential hearing preservation. MCF is ideal for intracanalicular tumors with minimal extension into the cerebellopontine angle. The RS approaches the cerebellopontine angle with better visualization of the cranial nerves, the pons, and brainstem. Anatomic limitations may foster recidivism with the hearing preservation approaches. The falciform crest obscures the inferior portion of the lateral fundus when using the MCF. Inferior vestibular nerve tumors may extend into this region to an inaccessible space of 1.82 to 2.33 mm [51,52]. The RS approach leaves up to the lateral 3 mm of the internal auditory canal underexposed. The addition of operative endoscopy has proved useful in accessing the lateral 30% of the internal auditory canal [53]. Larger tumors with significant brainstem compression may be accessed via an RS route in the setting of poor hearing because of the better visibility of brainstem structures. Prognostic factors that favor hearing preservation include a small tumor, greater than 70% speech discrimination and a 30dB speech reception threshold, auditory brainstem response with a normal wave V amplitude and latency, and no medical contraindications for surgery [54]. The choice of approach also depends on the preferences of the surgical team.

The most commonly reported major complications from microsurgery include hearing loss, facial nerve dysfunction, balance abnormalities, cerebrospinal fluid leakage, headache, meningitis, and stroke. Harsha and Backous [55] performed a Medline search for all articles pertaining to VS surgery published from 1994 to 2004. They refined the search to include only English-language articles that reported hearing outcome using either the AAO-HNS hearing or Gardner-Robinson hearing outcome scales, facial nerve outcomes using the AAO-HNS (House-Brackmann) facial nerve grading scale for facial nerve outcome, and complication rates based on approach used. Of 1132 articles evaluated, 31 met the criteria for inclusion and only 14 compared one or more outcome measure for more than one approach from the same institution. Two additional articles, which specifically addressed hearing preservation after middle fossa surgery, have been published subsequent to this article (Table 5) [56–69].

Overall, hearing outcome was serviceable (AAO-HNS class A or B/Gardner-Robinson class 1 or 2) in 618 of 2034 (30%) of patients treated with a hearing preservation approach. Results were considerably better with MCF (523/1017 [51%]) than with RS (95/304 [31%]). In six papers, MCF was compared with RS in the same institution. Forty-eight percent preserved serviceable hearing with MCF compared with 31% with RS [55]. The two most recent articles that addressed hearing preservation and the MCF approach reported high levels of maintained hearing in the serviceable range. Arts and colleagues [67] reviewed 73 consecutive patients excised via MCF. Of the 27 with class A hearing preoperatively, 62% remained class A, 18% deteriorated to class B, and 20% fell to class C. Overall, of the 62 patients presenting with class A or B hearing before surgery, 73% remained in class A or B. No patients improved their hearing class with surgery [66].

Meyer and colleagues [68] reported on 162 consecutive VS resected by the MCF approach. Class A or B/Class 1 or 2 hearing was preserved in 66 of 162 (41%) patients overall. Eight patients improved to class 1 hearing with surgical removal of their VS. Of the 113 patients with word recognition scores (WRS) more than 70% preoperatively, 56 (50%) maintained WRS of more than 70% after excision. Tumor size ranged between 0.2 and 2.5 cm in largest diameter. Smaller tumors (0.2–1.0 cm) had 59% maintain WRS more than 70% and 9% improved to more than 70% when falling below preoperatively. For tumors that measured 1.1 to 1.4 cm, 39% maintained at least 70% WRS and 3 additional patients (9%) improved to more than 70% postoperatively. Only

Table 5
Hearing outcomes using AAO-HNS/Gardner-Robinson classifications

Postoperative hearing classification [Number (%)]				
Author (ref)	A/1	B/2	C/3	D/4
Retrosigmoid approach				
Colletti, 2003 [56]	2 (8%)	3 (32%)	4 (16%)	11 (44%)
Staecker, 2000 [57]	6 (40%)	1 (7%)	1 (7%)	7 (46%)
Hecht, 1997 [58]	4 (10%)	5 (12%)	4 (10%)	29 (68%)
Arriaga, 1997 [59]	8 (31%)	6 (23%)	1 (4%)	11 (42%)
Sanna, 2004 [60]	8 (18%)	7 (16%)	4 (9%)	29 (66%)
Magnan, 2002 [61]	17 (15%)	18 (16%)	23 (19%)	61 (51%)
Lassaletta, 2003 [62]	0 (0%)	5 (17%)	1 (3%)	23 (79%)
Total	45 (15%)	50 (16%)	38 (13%)	171 (56%)
Middle Cranial Fossa Approach				
Colletti, 2003 [56]	3 (12%)	10 (40%)	3 (12%)	9 (36%)
Staecker, 2000 [57]	5 (33%)	3 (20%)	2 (13%)	5 (33%)
Hecht, 1997 [58]	4 (22%)	3 (17%)	1 (5%)	10 (56%)
Arriaga, 1997 [59]	15 (44%)	8 (24%)	2 (6%)	9 (26%)
Sanna, 2004 [60]	4 (7%)	15 (26%)	10 (17%)	30 (50%)
Slattery, 1997 [63]	35 (25%)	39 (27%)	5 (4%)	64 (45%)
Brackmann, 2000 [64]	109 (33%)	87 (26%)	16 (5%)	121 (36%)
Weber, 1996 [65]	5 (10%)	13 (27%)	3 (6%)	28 (57%)
Satar, 2002 [66]	30 (22%)	42 (31%)	18 (13%)	64 (34%)
Arts, 2006 [67]	21 (29%)	24 (33%)	6 (8%)	22 (30%)
Meyer, 2006 [68]	37 (23%)	29 (18%)	4 (2.5%)	83 (51%)
Total	268 (26%)	255 (25%)	64 (6%)	430 (42%)

33% of patients with tumors between 1.5 and 2.5 cm maintained WRS more than 70%. In the hands of the same surgical team, statistically significant improvement was noted in cases in which near field eighth nerve compound action potential monitoring was used. With tumors <1.4 cm, MCF done in experienced hands provided significant hearing preservation not previously reported. This article suggested changing the hearing classification to report only WRS because it more accurately reflects rehabilitation potential for "residual" hearing.

Hearing preservation rates reported in Table 5 address the number and percent of patients with serviceable hearing when compared with all patients treated by approach. This rate does not take into account the number of patients who maintained or improved their hearing status after operative tumor removal. In the two studies that specifically examined MCF for intracanalicular tumors, the results are much more favorable. Long-term (>5 years) hearing preservation in this group of patients should be compared with patients who received radiotherapy at matched intervals.

The AAO-HNS (House-Brackmann) facial nerve grading scale score, determined at the 6- to 12-month visit, is the gold standard for reporting facial nerve outcomes after VS treatment. In the review by Harsha and Backous [55], overall grade I/II function was 82%, with 92% of RS, 89% of MCF, and 73% of TL having a good outcome. Only one center reported results of all three approaches within the same institution. Grade I/II function was maintained as follows: RS (91%), MCF (88%), and TL (77%). Centers with more than 100 cases with a single approach were analyzed separately and good function (AAO-HNS I/II) was maintained as follows: RS (97%), MCF (93%), and TL (78%). These results were not analyzed separately for tumor size, which may have contributed to the choice of surgical approach. Meyer and colleagues [68] reported grade I/II function in 97% of 162 patients (86% grade I) operated via the MCF, whereas Arts and colleagues [67] maintained 96% grade I/II function (85% grade I).

No specific data were presented with standardized methodology to assess postoperative vestibular and other balance dysfunction.

CSF leakage is the most common complication after VS resection. Typically, CSF leaks occur 2 to 3 days postoperatively (early) and at 10 to 14 days after surgery (late). Pooling the data from

19 studies revealed an incidence of 8% (360/4297) for CSF leakage. Leakage rate stratified by approach was: 6% MCF (67/1038), 11% RS (42/380), and 8% TL (253/2881) [55]. Slattery and colleagues [70,71] reported 1697 patients from the House Ear Clinic and found a higher incidence of CSF leakage in the RS group (15%) compared with TL (11%) and MCF (6%). Selesnick [72] performed a meta-analysis and found the lowest CSF leakage rate in the TL group (9.5%) when compared with the RSA and MCF groups (both 10.6%). Overall, the CSF leak rate is low for each of the three approaches and may vary according to the preferences and experiences of individual surgeons and skull base teams.

Headache is common in the first few days to weeks after VS resection. Prolonged postoperative headache—beyond 3 months—occurs in roughly 10% of cases. Bone dust in contact with the CSF and meninges causing secondary aseptic meningitis, entrapment of the occipital nerve in neck musculature and scar tissue, scarring of the neck muscles to the dura, and migraines are considered the four mechanisms for prolonged headaches [54]. No standardized reporting scheme has been developed for postoperative headache, but pooled data from six articles revealed clinically significant headache in 21% of RS, 8% MCF, and 3% TL [55]. Staecker [57] found more than double the headache rate in RS (47%) when compared with MCF (20%).

The rate of bacterial meningitis was 2% to 3% for all three approaches [55]. Meningitis peaked at 3 to 5 days postoperatively and was most commonly caused by *Staphylococcus aureus*. Aseptic meningitis can occur in up to 20% of patients and responds to intravenous or oral steroids. Seizure, hydrocephalus, and stroke rates caused by surgery are rare and occur in less than 2% of cases.

Observation with serial neuroimaging

An often undervalued treatment modality for VS is observation with serial neuroimaging using MRI scanning at regular intervals. A report from Saudi Arabia retrospectively reviewed 205 patients with intracanalicular tumors for an average of 40 months (range 12–180 months). No growth was found in 66.3% of patients, 23.9% showed slow growth, and 4% had rapid growth. Six patients (3%) had tumor regression [73]. Charabi and colleagues [74] reported on 127 tumors in 123 patients followed from 1973 to 1993 in Denmark. At a mean follow-up of 3.8 years, tumor growth was found in 82% of tumors, no growth in 12%, and regression in 8%. Growth patterns were variable and unpredictable. Leeuwen and colleagues [75] found no correlation among tumor size, symptoms, and patient age in 164 patients treated and over a period of 13 years in Holland. Yoshimoto [76] reviewed 1340 patients from a Medline search of 26 studies. The overall growth of VS during a mean follow-up period of 38 months was 46%. Growth averaged 1.2 mm/y. Raut and colleagues [77] reviewed conservative management of 72 patients in the United Kingdom. Growth was seen in 38.9%, no growth in 41.7%, and regression in 19.4% after a mean follow-up of 80 months. Pure tone and speech discrimination scores deteriorated regardless of tumor growth. In Aarhus County, Denmark, 162 VSs were diagnosed and 64 patients opted for conservative management. Twenty-three percent of tumors grew more than 1 mm/y, 55% did not grow, and 22% regressed. The observation period extended to 15 years (180 months) [78]. Unfortunately, no common predictors or ''red flags'' could be extracted from these studies to predict growth patterns for VS.

Battaglia and colleagues [79] reviewed 164 patients treated with radiation therapy from 1986 to 2004 at the Southern California Permanente Group and compared their long-term growth rates with the results of a meta-analysis of five studies of cases managed conservatively. They reported an average growth rate of 0.7 mm/y. They determined that small intracanalicular tumors grew more slowly, if at all, when compared with tumors in the cerebellopontine angle. They concluded that to determine whether radiotherapy alters tumor growth rate, a direct comparison of small, medium, and large tumors is necessary.

The option of observation with serial MRI scanning is a valid form of managing VS. Severity of symptoms does not correlate with lesion size, and determination of efficacy of radiotherapy in small tumors VS the natural history of intracanalicular lesions is unclear. If no brainstem compression or hydrocephalus is present, patients should be offered an observation course with an understanding that no clear risk factors for growth have been defined. Our center considers observation and interval symptom review, physical examination, and neuroimaging a form of treatment and document it as such.

Summary

Patients diagnosed with VS are faced with a difficult decision-making process based on

medical literature weakened by inconsistent reporting of results. Radiotherapy protocols and surgical techniques have evolved in the past 30 years. Most reporting is retrospective and does not adhere to consistent and validated standards for hearing preservation, facial nerve function, or balance disturbance.

Confusion surrounding recidivism versus recurrence of VS and no clear and validated definition of tumor control further clouds the interpretation of results. The lack of randomized, blinded clinical trials has retarded the development of evidence-based clinical best practices for recommending optimal treatment for patients dictated by such factors as tumor size, location, and hearing level. Patients often review data on the Internet, which is not peer reviewed and can complicate preoperative counseling.

The mandate for establishing and documenting informed consent in a format that is timely and understandable to individual patients remains. A trend is developing in which neurosurgeons and neurotologists are acquiring certification in GK and other forms of radiosurgery. These surgeons provide a balanced informational process because they are involved in either option selected by a patient. In other centers, a multidisciplinary team of surgeons and radiotherapists individually consults with patients within their own area of expertise to present options to patients and foster appropriate informed consent. Whichever the approach taken to explain options to patients, one physician should remain the gatekeeper of the patient's care. Answering additional questions and ensuring the opportunity for interval clinical, audiometric, and radiographic follow-up remains the responsibility of the care team. The choice to follow surveillance is a patient choice.

Standardized quantification of tumor size (volumetric or greatest dimension), consistent adherence to the AAO-HNS facial nerve reporting scale, consistent acceptance of one reporting standard for hearing and agreed-upon intervals for testing, and follow-up for longer intervals improve data and foster outcomes-based best practices for reporting results and advising patients for treatment.

Currently, in patients without significant brainstem compression, hydrocephalus, or ataxia, a conservative approach with annual MRI scans and physical examinations remains an acceptable option. Although the literature is not clear, larger tumors with extension into the cerebellopontine angle seem to grow more quickly and intracanalicular VSs grow more slowly. The interval for imaging may vary depending on tumor characteristics in individual patients.

Stereotactic radiosurgery is a valid option for tumors <3 cm in largest dimension. Tumors >3 cm are poorer candidates for radiotherapy and should be considered for surgical removal unless the patient is medically unstable. An intracanalicular VS that is <1.4 cm in greatest dimension with CSF between the lateral margin of the tumor and the labyrinth on T2-weighted MRI imaging, has a normal wave V on auditory brainstem response, has more than 70% WRS, and is in a patient with good health can be excised via MCF with hearing preservation approaching 70% and good facial nerve function in more than 96% of cases.

Longer term follow-up determines the longevity of hearing preservation in surgical and radiotherapy patients and catalogs late complications possible after both. Most surgical complications occur within the first weeks after resection, whereas radiotherapy complications may occur many years after treatment is complete. The difficult cases are found in patients with VS <3 cm. All three options are potentially useful, and unbiased provision of information to help them decide on a path of treatment is mandatory for proper informed consent.

At our institution, we use a multidisciplinary team that includes a neurotologist, neurosurgeon, and radiotherapist to explain the risks and benefits of each option for treatment. We give the patient a packet with selected articles, and a nurse-coordinator follows up to be sure all questions are relayed to the appropriate physician and answered. The process is designed to allow the patient a balanced presentation of information to foster a timely decision that ensures adequate informed consent.

Acknowledgments

This work was supported by the Listen for Life Foundation Fund at the Virginia Mason Medical Center.

References

[1] Wallner KE, Sheline GE, Pitts LH, et al. Efficacy of irradiation for incompletely excised acoustic neurilemomas. J Neurosurg 1987;67:858–63.

[2] Leksell L. The stereotactic method and radiosurgery of the brain. Acta Chir Scand 1951;102:316–9.

[3] Leksell L. A note on the treatment of acoustic tumors. Acta Chir Scand 1971;137:763–5.
[4] Kondziolka D, Lunsford D, McLaughlin, et al. Long-term outcomes after radiosurgery for acoustic neuromas. N Engl J Med 1998;339:1426–33.
[5] Ito K, Kurita H, Sugasawa K, et al. Neuro-otological findings after radiosurgery for acoustic neurinomas. Arch Otolaryngol Head Neck Surg 1996;122: 1229–33.
[6] Noren G. Long-term complications following gamma knife radiosurgery of vestibular schwannomas. Sterotact Funct Neurosurg 1998;70(Suppl 1):65–73.
[7] Friedman WA, Bradshaw P, Myers A, et al. Linear accelerator radiosurgery for vestibular schwannomas. J Neurosurg 2006;105:657–61.
[8] Lunsford LD, Niranjan A, Flickinger JC, et al. Radiosurgery of vestibular schwannomas: summary of experience in 829 cases. J Neurosurg 2005; 102(Suppl):195–9.
[9] Foote KD, Friedman WA, Buatti JM, et al. Linear accelerator radiosurgery in brain tumor management. Neurosurg Clin N Am 1999;10:203–43.
[10] Andrews DW, Suarez O, Goldman W, et al. Stereotactic radiosurgery and fractionated stereotactic radiotherapy for the treatment of acoustic schwannomas: comparative observations of 125 patients treated at one institution. Int J Radiat Oncol Biol Phys 2001; 50(5):1265–78.
[11] Williams JA. Fractionated stereotactic radiotherapy for acoustic neuromas. Int J Radiat Oncol Biol Phys 2002;54(2):500–41.
[12] Prasad D, Steiner M, Steiner L. Gamma surgery for vestibular schwannoma. J Neurosurg 2000;92: 745–59.
[13] Hasegawa T, Fujutani S, Katsumata S, et al. Stereotactic radiosurgery for vestibular schwannomas: analysis of 317 patients followed more than 5 years. Neurosurgery 2005;57:257–65.
[14] Flickinger JC, Kondziolka D, Niranjan A, et al. Acoustic neuroma radiosurgery with marginal tumor doses of 12 to 13 Gy. Int J Radiat Oncol Biol Phys 2004;60(1):225–30.
[15] Massager N, Nissim O, Delbrouck C, et al. Role of intracanalicular volumetric and dosimetric parameters on hearing preservation after vestibular schwannoma radiosurgery. Int J Radiat Oncol Biol Phys 2006;64(5):1331–40.
[16] Ottaviani F, Neglia CB, Ventrella L, et al. Hearing loss and changes in transient evoked otoacoustic emissions after gamma knife radiosurgery for acoustic neuromas. Arch Otolaryngol Head Neck Surg 2002;128:1308–12.
[17] Wackym PA, Rung-Samuelson CL, Poetker DM, et al. Gamma knife radiosurgery for acoustic neuromas performed by a neurotologist: early experiences and outcomes. Otolo Neurotol 2004;25: 752–61.
[18] Chung WY, Liu KD, Shiau CY, et al. Gamma knife surgery for vestibular schwannoma: 10-year experience of 195 cases. J Neurosurg 2005; 102(Suppl):87–96.
[19] Wowra B, Muacevic A, Jess-Hempen A, et al. Outpatient gamma knife surgery for vestibular schwannoma: definition of the therapeutic profile based on a 10-year experience. J Neurosurg 2005; 102(Suppl):114–8.
[20] Petit JH, Hudes RS, Chen TT, et al. Reduced-dose radiosurgery for vestibular schwannomas. Neurosurgery 2001;49(6):1299–307.
[21] Pollock BE, Discoll CLW, Foote RL, et al. Patient outcomes after vestibular schwannoma management: a prospective comparison of microsurgical resection and stereotactic radiosurgery. Neurosurgery 2006;59(1):77–85.
[22] Myrseth E, Moller P, Pederson PH, et al. Vestibular schwannomas: clinical results of quality of life after microsurgery or gamma knife radiosurgery. Neurosurgery 2005;56(5):927–35.
[23] Paek SH, Chung HT, Jeong SS, et al. Hearing preservation after gamma knife stereotactic radiosurgery of vestibular schwannoma. Cancer 2005;104:580–90.
[24] Litvack ZN, Noren G, Chougle PB, et al. Preservation of functional hearing after gamma knife surgery for vestibular schwannoma. Neurosurg Focus 2003; 14(5):1–5.
[25] Hirato M, Inoue H, Zama A, et al. Gamma knife radiosurgery for acoustic schwannomas: effects of low radiation dose and functional prognosis. Stereotact Funct Neurosurg 1996;66(Suppl 1):134–41.
[26] Muacevic A, Jess-Hempen A, Tonn JC, et al. Results of outpatient gamma knife radiosurgery for primary therapy of acoustic neuromas. Acta Neurochir Suppl 2004;91:75–8.
[27] Iwai Y, Yamanaka K, Shiotani M, et al. Radiosurgery for acoustic neuromas: results of low-dose treatment. Neurosurgery 2003;53:282–8.
[28] Kwon Y, Kim JH, Lee DJ, et al. Gamma knife treatment of acoustic neuroma. Stereotact Funct Neurosurg 1998;70(Suppl 1):57–64.
[29] Chung HT, Ma R, Toyota B, et al. Audiologic and treatment outcomes after linear accelerator-based stereotactic irradiation for acoustic neuroma. Int J Radiat Oncol Biol Phys 2004;59(4):1116–21.
[30] Combs SE, Thilmann C, Debus J, et al. Long-term outcome of stereotactic radiosurgery (SRS) in patients with acoustic neuromas. Int J Radiat Oncol Biol Phys 2006;64(5):1341–7.
[31] Meijer OW, Vandertop WP, Baayen JC, et al. Single-fraction vs. fractionated linac-based stereotactic radiosurgery for vestibular schwannoma: a single-institution study. Int J Radiat Oncol Biol Phys 2003;56(5):1390–6.
[32] Spiegelmann R, Lidar Z, Gofman J, et al. Linear accelerator radiosurgery for vestibular schwannoma. J Neurosurg 2001;94:7–13.
[33] Foote KD, Friedman WA, Buatti JM, et al. Analysis of risk factors associated with radiosurgery for vestibular schwannomas. J Neurosurg 2001;95:440–9.

[34] Lee GY, Roos DE, Brophy BP. Radiosurgery for vestibular schwannomas: preliminary results of the Adelaide experience. Stereotact Funct Neurosurg 2001;76:209–12.
[35] Combs SE, Volk S, Schultz-Ertner D, et al. Management of acoustic neuromas with fractionated stereotactic radiotherapy (FSRT): long-term results in 106 patients treated in a single institution. Int J Radiat Oncol Biol Phys 2005;63(1):75–81.
[36] Shirato H, Sakamoto T, Sawamura Y, et al. Comparison between observation policy and fractionated stereotactic radiotherapy (SRT) as an initial management for vestibular schwannoma. Int J Radiat Oncol Biol Phys 1999;44(3):545–50.
[37] Shokek O, Terezakis S, Hughes MA, et al. Hypofractionated stereotactic radiotherapy as primary treatment of acoustic neuroma: interim results of the Johns Hopkins experience. Int J Radiat Oncol Biol Phys 2004;60(Suppl 1):s314.
[38] Fuss M, Debus J, Lohr F, et al. Conventionally fractionated stereotactic radiotherapy (FSRT) for acoustic neuromas. Int J Radiat Oncol Biol Phys 2000;48(5):1381–7.
[39] Selch MT, Pedroso A, Lee SP, et al. Stereotactic radiotherapy for the treatment of acoustic neuromas. J Neurosurg 2004;101(Suppl 3):362–72.
[40] Sawamura Y, Shirato H, Sakamoto T, et al. Management of vestibular schwannoma by fractionated stereotactic radiotherapy and associated cerebrospinal fluid malabsorption. J Neurosurg 2003;99: 685–92.
[41] Poen J, Golby AJ, Forster KM, et al. Fractionated stereotactic radiosurgery and preservation of hearing in patients with vestibular schwannoma: a preliminary report. Neurosurgery 1999;45(6):1299–307.
[42] Chan AW, Black PM, Ojemann RG, et al. Stereotactic radiotherapy for vestibular schwannomas: favorable outcome with minimal toxicity. Neurosurgery 2005;57:60–70.
[43] Chang SD, Gibbs IC, Sakamoto GT, et al. Staged stereotactic irradiation of acoustic neuroma. Neurosurgery 2005;56:1254–63.
[44] Lederman G, Lowry J, Werthein S, et al. Acoustic neuroma: potential benefits of fractionated stereotactic radiosurgery. Stereotact Funct Neurosurg 1997;69:175–82.
[45] Ishihara H, Sato K, Nishizaki T, et al. CyberKnife radiosurgery for vestibular schwannoma. Minim Invasive Neurosurg 2004;47:290–3.
[46] Forster D, Kemeny A, Pathak A, et al. Radiosurgery: a minimally interventional alternative to microsurgery in the management of acoustic neuroma. Br J Neurosurg 1996;10:169–74.
[47] Hanabusa K, Morikawa A, Murata T, et al. Acoustic neuroma with malignant transformation: case report. J Neurosurg 2001;95:518–21.
[48] Shin M, Ueki K, Kurita H, et al. Malignant transformation of a vestibular schwannoma after gamma knife radiosurgery. Lancet 2002;360:309–10.
[49] Shamisa A, Bance M, Nag S, et al. Glioblastoma multiforme occurring in a patient treated with gamma knife surgery. J Neurosurg 2001;94: 816–21.
[50] Maire JP, Huchet A, Milbeo Y, et al. Twenty years' experience in the treatment of acoustic neuromas with fractionated radiotherapy: a review of 45 cases. Int J Radiat Oncol Biol Phys 2006;66:170–8.
[51] Haberkamp TJ, Meyer GA, Fox M. Surgical exposure of the fundus of the internal auditory canal: anatomic limits of the middle fossa versus the retrosigmoid approach. Laryngoscope 1998; 108:1190–4.
[52] Driscoll CL, Jackler RK, Pitts LH, et al. Is the entire fundus of the internal auditory canal visible during the middle fossa approach for acoustic neuroma? Am J Otolaryngol 2000;21(3):382–8.
[53] Blevins NH, Jackler RK. Exposure of the lateral extremity of the internal auditory canal through the retrosigmoid approach: a radioanatomic study. Otolaryngol Head and Neck Surg 1994;111:81–90.
[54] Wackym PA. Stereotactic radiosurgery, microsurgery, and expectant management of acoustic neuroma: basis for informed consent. Otolaryngol Clin North Am 2005;38(4):653–70.
[55] Harsha WJ, Backous DD. Counseling patients on surgical options for treating acoustic neuroma. Otolaryngol Clin North Am 2005;38(4):643–52.
[56] Colletti V, Fiorino F. Middle fossa versus retrosigmoid-transmeatal approach in vestibular schwannoma surgery: a prospective study. Otol Neurotol 2003;24:927–34.
[57] Staecker H, Nadol J, Ojeman R, et al. Hearing preservation in acoustic neuroma surgery: middle fossa versus retrosigmoid approach. Am J Otolaryngol 2000;21(3):399–404.
[58] Hecht C, Honrubia V, Wiet R, et al. Hearing preservation after acoustic neuroma resection with tumor size as a clinical prognosticator. Laryngoscope 1997;107:1122–6.
[59] Arriaga M, Chen D, Fukushima T. Individualized hearing preservation in acoustic neuroma surgery. Laryngoscope 1997;107:1043–7.
[60] Sanna M, Khrais T, Picarilo E, et al. Hearing preservation surgery in vestibular schwannoma: the hidden truth. Ann Otol Rhinol Laryngol 2004;113: 156–63.
[61] Magnan J, Barbieri M, Mora R, et al. Retrosigmoid approach for small and medium sized acoustic neuromas. Otol Neurotol 2002;23:141–5.
[62] Lassaletta L, Fontes L, Mecon E, et al. Hearing preservation with the retrosigmoid approach for vestibular schwannoma: myth or reality? Otolaryngol Head and Neck Surg 2003;129:397–401.
[63] Slattery W, Brackmann D, Hitselberger W. Middle fossa approach for hearing preservation with acoustic neuromas. Am J Otolaryngol 1997;18:596–601.
[64] Brackmann D, Owens R, Friedman R, et al. Prognostic factors for hearing preservation in vestibular

schwannoma surgery. Am J Otolaryngol 2000;21(3): 417–24.
[65] Weber P, Gantz B. Results and complications from acoustic neuromas excision via middle cranial fossa approach. Am J Otolaryngol 1996;17:669–75.
[66] Satar B, Jackler R, Oghalai J, et al. Risk-benefit analysis of using the middle fossa approach for acoustic neuromas with >10 mm cerebello-pontine angle component. Laryngoscope 2002;112 1500–6.
[67] Arts HA, Telian SA, l-Kashlan H, et al. Hearing preservation and facial nerve outcomes in vestibular schwannoma surgery: results using the middle cranial fossa approach. Otol Neurotol 2006;27(2): 234–41.
[68] Meyer TA, Canty PA, Wilkinson EP, et al. Small acoustic neuromas: surgical outcomes versus observation or radiation. Otol Neurotol 2006;27(3):380–92.
[69] Arriaga M, Chen D. Facial function in hearing preservation acoustic neuroma surgery. Arch Otolaryngol Head Neck Surg 2001;127:543–6.
[70] Slattery W, Sabina F, House K. Perioperative morbidity of acoustic neuroma surgery. Otol Neurotol 2001;22(6):895–902.
[71] Korinek AM. Risk factors for neurosurgical site infections after craniotomy: a prospective multicenter study of 2944 patients. Neurosurgery 1997;41: 1073–9.
[72] Selesnick SA, Liu JC, Jen A, et al. The incidence of cerebrospinal fluid leak after vestibular schwannoma surgery. Otol Neurotol 2004;25(3):387–93.
[73] Al Sanosi A, Fagan PA, Biggs ND. Conservative management of acoustic neuroma. Skull Base 2006;16(2):95–100.
[74] Charabi S, Thomsen J, Tos M, et al. Acoustic neuroma/vestibular schwannoma growth: past, present and future. Acta Otolaryngol 1998;118(3):327–32.
[75] van Leeuwen JP, Cremers CW, Thewissen NP, et al. Acoustic neuroma: correlation among tumor size, symptoms, and patient age. Laryngoscope 1995; 105(7Pt1):701–7.
[76] Yoshimoto Y. Systematic review of the natural history of vestibular schwannoma. J Neurosurg 2005; 103(1):59–63.
[77] Raut VV, Walsh RM, Bath AP, et al. Conservative management of vestibular schwannomas: second review of a prospective longitudinal study. Clin Otolaryngol Allied Sci 2004;29(5):505–14.
[78] Mirz F, Pedersen CB, Fiirgaard B, et al. Incidence and growth pattern of vestibular schwannomas in a Danish county, 1977–98. Acta Otolaryngol Suppl 2000;543:30–3.
[79] Battaglia A, Mastrodimos B, Cueva R. Comparison of growth patterns of acoustic neuromas with and without radiosurgery. Otol Neurotol 2006;27(5):705–12.