8 ■ VESTIBULAR SCHWANNOMA

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QUICK HIT ■ Vestibular schwannoma, previously called "acoustic neuroma," is a slow-growing, benign tumor of the cerebellopontine angle that typically presents with unilateral hearing loss. Treatment options include observation, microsurgical resection, and RT (SRS or fractionated). SRS is generally prescribed up to 13 Gy and conventional fractionation to 45 to 54 Gy. Tumor control outcomes appear equivalent between those of surgery and RT, but RT may minimize impact on QOL.

EPIDEMIOLOGY: Incidence is approximately 0.6 to 1.9/100,000, making up 8% of intracranial tumors. Incidence is increasing with increased utilization of diagnostic imaging.[1,2] Median age at diagnosis is 50 to 55, and incidence increases with age.[1,3]

RISK FACTORS: Increasing age, NF2 (96% of patients with NF2, often bilateral), NF1 (5% of patients with NF1, unilateral), childhood exposure to RT (RR 1.14/Gy).[4]

ANATOMY: VS typically arises from the vestibular portion of CN VIII and is unilateral in 90% of cases. CN VIII arises from the junction of the pons and medulla, enters the internal auditory foramen along with the facial nerve (CN VII), and then divides into the vestibular and cochlear nerves. The cochlear nerve runs to the spiral ganglion and innervates the spiral organ of Corti and the cochlea. The vestibular nerve runs to the vestibular ganglion and splits into three branches. The superior branch innervates the utricle and the superior and lateral semicircular ducts. The inferior branch innervates the saccule and the posterior branch innervates the posterior semicircular duct. VS arises with equal frequency in the superior and inferior branches and rarely in the cochlear nerve. It tends to occur in the vestibular region of the foramen, where the nerve acquires a Schwann cell sheath, although it can sometimes arise from or grow into the CPA.

PATHOLOGY: VS is composed of atypical proliferations of Schwann cells, which are found lining peripheral nerves. Histopathologically, they are similar to other peripheral schwannomas and are composed of alternating zones of dense and sparse cellularity, termed "Antoni A" and "Antoni B," respectively. IHC demonstrates S100 positivity.[5] Malignant degeneration is extremely rare.

GENETICS: Biallelic inactivation of NF2 on chromosome 22, which produces the tumor suppressor merlin, is common in sporadic VS and is the cause of bilateral VS in NF2.[6]

CLINICAL PRESENTATION: Hearing loss (95%; only two thirds are aware of it; average duration ~4 years although 16% develop sudden hearing loss), tinnitus (63%; average duration ~3 years), vestibular symptoms (61%; often mild to moderate, nonspecific, and fluctuating; average duration ~2 years), headache (12%; most often occipital), trigeminal symptoms (9%; typically facial numbness/hyperesthesia/pain; average duration ~1 year), facial nerve symptoms (6%; typically facial weakness, less commonly taste disturbance; average duration ~2 years), and other symptoms from brainstem compression (ataxia, hydrocephalus, dysarthria, dysphagia, hoarseness) are uncommon.[7] The House–Brackmann and Gardner–Robertson scales are common metrics of facial paralysis and hearing loss, respectively (Tables 8.1 and 8.2).

WORKUP: H&P including Weber and Rinne tests to evoke asymmetric sensorineural hearing loss and CN exam, audiometry; consider BAER testing (BAER/ABR; 60% to 90% sensitive with lower sensitivity for small tumors; 60%–90% specific).[8] Vestibular testing is uncommon.

Imaging: MRI of the brain with contrast is the gold standard for diagnosis. High-resolution CT with IV contrast if unable to obtain MRI. MRI shows isointense or slightly hypointense signal to brain on T1, typically with homogeneous contrast enhancement although occasional cystic degeneration can be seen.[9] Classic finding is "ice-cream cone" shape with widening of the porus acusticus.[10] Differential

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includes VS, meningioma, glomus tumor, ependymoma, facial or trigeminal schwannoma, epidermoid cyst, metastasis.

Table 8.1: House–Brackmann Facial Paralysis Scale[11]	
Grade I	Normal
Grade II	Mild dysfunction (slight weakness, normal symmetry at rest)
Grade III	Moderate dysfunction (obvious but not disfiguring weakness, synkinesis) with normal symmetry at rest Complete eye closure with maximal effort Good forehead movement
Grade IV	Moderately severe dysfunction (obvious and disfiguring asymmetry, significant synkinesis) Incomplete eye closure Moderate forehead movement
Grade V	Severe dysfunction (barely perceptive motion)
Grade VI	Total paralysis

Table 8.2: Gardner–Robertson Hearing Loss Scale[12]	
Grade I	Good–excellent (70%–100% speech discrimination)
Grade II	Serviceable (50%–69%)
Grade III	Nonserviceable (5%–49%)
Grade IV	Poor (1%–4%)
Grade V	None

PROGNOSTIC FACTORS: Baseline level of hearing loss, growth rate >2.5 mm/yr, and delay in diagnosis.[13–16] Initial tumor size is not prognostic.[15] Patients with growth rate >2.5 mm/yr have decreased rates of hearing preservation (32% vs. 75%, $p < .0001$) and decreased median time to total hearing loss (7.0 vs. 14.8 years, $p < .0001$).[15,16]

STAGING: VSs are not staged but can be graded on the Koos grading scale (Table 8.3).[17]

Table 8.3: Koos Grading Scale for VS[17]	
Grade I	Intracanalicular
Grade II	Tumor extending into the posterior fossa, with or without an intracanalicular component, without touching the brainstem
Grade III	Tumor extending into the posterior fossa, compressing the brainstem, but not shifting it from the midline
Grade IV	Tumor extending into the posterior fossa, compressing the brainstem, and shifting it from the midline

TREATMENT PARADIGM

Table 8.4: General Probability of Hearing Preservation in Favorably Selected Patients[18]			
	2 Yrs	**5 Yrs**	**10 Yrs**
Observation	>75% to 100%	>50% to 75%	Insufficient data
SRS	>75% to 100%	>50% to 75%	>25% to 50%
Surgery	>25% to 50%	>25% to 50%	>25% to 50%

Favorably selected patients include small- to medium-sized sporadic VS, good–excellent speech discrimination (Gardner–Robinson Grade I).

Observation: Consider observation with MRI every 6 to 12 months in patients without baseline hearing loss and stability or slow rate of growth. Observation is especially favored in older patients with significant comorbidities. Indications for treatment vary but can include >2.5 mm growth/year and new onset or worsening of symptoms. Patients undergoing observation should be counseled that they have a risk of hearing loss without treatment (see Table 8.4). Current consensus guidelines suggest annual imaging at least up to 5 years with possible longer duration of follow-up up to 10 years.[18,19]

Surgery: In general, surgery has excellent results for resection of the entire tumor but can have poor outcomes with hearing preservation. Hearing preservation is most likely when the tumor is <1.5 to 2 cm in size.[20] Other major morbidities include CSF leaks, tinnitus, headaches, and facial paralysis.[21] Surgery is still the most common treatment for VS and is especially considered for younger patients, larger tumors, tumors causing mass effect or dizziness, cystic tumors, and small anatomically favorable tumors with good hearing.[22] There are three main surgical approaches for resection (see Table 8.5).[21–23] The goal of resection is to maximize tumor removal while minimizing morbidity.

Table 8.5 Surgical Techniques for VS

Approach	Pros	Cons
Retrosigmoid/ suboccipital	Possible hearing conservation and facial nerve sparing	Associated with increased risk of CSF leaks and HA
Translabyrinthine	Possible preservation of facial function	No hearing preservation, a fat graft is required, and the sigmoid sinus is more prone to injury
Middle fossa	Possible hearing conservation for small tumors (≤1.5 cm)	Facial nerve more vulnerable to injury, dural lacerations likely in older patients, may cause trismus from temporalis muscle injury

Chemotherapy: There is generally no role for systemic therapy, although bevacizumab has shown response in rare progressive situations associated with NF2.[24]

Radiation: Several options for treatment with RT exist. SRS (with GKRS or LINAC-based radiosurgery), FSRT, and proton beam RT have been used. RT is appropriate when the tumor is <3 to 4 cm in size or when surgery is not an option or refused.[25]

SRS: Doses above 12.5 to 13 Gy are associated with increased morbidity with regard to facial paralysis, trigeminal neuralgia, and hearing loss.[26,27] Long-term results show >95% control with minimal morbidity or impact on QOL. Impact on hearing preservation and relative differences between treatment modalities appears to vary with time.[28,29] In a series of 440 patients with long-term follow-up, one patient (0.3%) developed malignant transformation.[30]

FSRT: Treatments can range from 20 Gy/4 fx to 57.6 Gy/32 fx. Typical hypofractionated dose is 25 Gy/5 fx and 45 to 54 Gy/25 to 30 fx for conventional fractionation. Controversy exists whether FSRT is superior to SRS, but it is recommended with larger tumors (>3–4 cm) in an effort to spare adjacent normal structures such as the brainstem and cochlea.

Procedure: See *Handbook of Treatment Planning in Radiation Oncology*, Chapter 3.[31]

■ EVIDENCE-BASED Q&A

■ What are the outcomes of treatment with surgical resection for patients with VS?

Surgical resection is generally technically achievable with high rates of control.[20] The risk of significant complications is low. There may be a lower rate of complications with maximal safe resection, allowing for residual tumor rather than attempted GTR in all patients.[21] However, patients who undergo STR are at a higher risk of recurrence than patients who undergo GTR or NTR.[32]

Samii, Germany (*Neurosurgery* 1997, PMID 8971819): RR of 1,000 consecutive patients with VS resected by suboccipital approach between 1978 and 1993; 98% of tumors were completely removed.

Anatomic preservation of the facial nerve and the cochlear nerve was achieved in 93% and 68%, respectively. Major neurologic complications included tetraparesis in one patient, hemiparesis in 1%, lower cranial nerve palsies in 5.5%, and cerebrospinal fluid fistulas in 9.2%. There were 11 deaths (1.1%) occurring at 2 to 69 days postoperatively.

Carlson, Mayo Clinic (*Laryngoscope* 2012, PMID 22252688): RR of 203 patients treated at a single institution. Patients were classified by GTR, NTR, or STR; 144 patients underwent GTR, 32 NTR, and 27 STR; 12 patients (6%) had a recurrence at a mean of 3.0 years after surgery; 5-year RFS was estimated at 91%. Patients who received STR were 9 times more likely to fail than patients undergoing NTR or GTR. No significant difference between patients with NTR and GTR was noted. Patients with nodular enhancement on initial post-op MRI had a 16-times higher risk of recurrence compared to patients with linear patterns.

How does SRS compare with observation?

SRS appears to have limited impact on QOL compared to observation.[33]

Breivik, Norway (*Neurosurgery* 2013, PMID 23615094): Prospective cohort study of patients who underwent GKRS (113) or observation (124). Patients underwent GKRS with small tumors (<20 mm) after growth was observed by referring physician (n = 31), by patient choice (n = 26), or with tumors >20 mm who refused surgery. GKRS dose was 12 Gy to the tumor periphery. Serviceable hearing was lost in 76% of patients on observation and 64% with GKRS (NS). Patients treated with GKRS had significantly less need for future treatment. Symptoms and QOL did not differ between groups. **Conclusion: GKRS appears to prevent the need for further treatment and appears not to significantly impact rates of hearing loss, symptoms, or QOL compared to observation.**

How does RT compare with microsurgical resection?

Generally, studies have shown equivalent tumor control with SRS compared to microsurgical resection with generally better functional outcomes and less impact on QOL with SRS.[28,34–36] *However, there is no consensus on the optimal management. The ideal population of patients for each modality overlaps (small tumors with preserved hearing) but surgery may be preferred in larger tumors, especially in patients with mass effect.*

Pollock, Mayo Clinic (*Neurosurgery* 2006, PMID 16823303): Prospective cohort study of 82 patients with unilateral <3 cm VS undergoing surgical resection (n = 36) or GKRS (n = 46). GKRS mean dose was 12.2 Gy to tumor margin; mean maximum dose, 26.4 Gy. No difference in tumor control (100% vs. 96%, p = .50). GKRS patients had better facial nerve preservation at 3 months (100% vs. 69%, p < .001), 1 year (100% vs. 69%, p < .001), and last f/u (100% vs. 75%, p < .01). GKRS patients had better hearing preservation at 3 months (77% vs. 5%, p < .001), 1 year (63% vs. 5%, p < .001), and last f/u (63% vs. 5%, p < .001). GKRS patients had better physical functioning, energy, and pain at 3 months, 1 year, and last f/u. **Conclusion: Similar tumor control with GKRS or surgery but less morbidity with GKRS.**

Maniakas, Montreal (*Otol Neurotol* 2012, PMID 22996165): Meta-analysis of 16 studies comparing microsurgical resection and SRS. Overall, SRS showed significantly better long-term hearing preservation rates than microsurgery (70% vs. 50%, respectively, p < .001). Crude rates of long-term tumor progression were not significantly different between SRS and microsurgery (3.8% and 1.3%, respectively).

What are the long-term results of SRS?

Long-term results for SRS show excellent LC. However, with longer term follow-up, it appears that rates of hearing preservation may continue to decline.[29,30,37]

Hasegawa, Japan (*J Neurosurg* 2013, PMID 23140152): RR of 440 patients treated with GKRS between 1991 and 2000. MFU 12.5 years. Actuarial 5- and 10-year PFS was 93% and 92%, respectively. No patient failed >10 years after treatment. On MVA, significant brainstem compression, marginal dose ≤13 Gy, prior treatment, and female sex correlated with decreased PFS. Patients treated with ≤13 Gy had an increased rate of facial nerve preservation (100% vs. 97%); 10 patients (2.3%) developed delayed cyst formation. One patient (0.03%) developed malignant transformation.

Carlson, Mayo Clinic (*J Neurosurg* 2013, PMID 23101446): RR of 44 patients with long-term audiometric follow-up after SRS. SRS was given with 12 to 13 Gy to the periphery of the tumor. MFU 9.3

years; 36 patients developed nonserviceable hearing at mean of 4.2 years after SRS. Kaplan–Meier estimated rates of serviceable hearing at 1, 3, 5, 7, and 10 years following SRS were 80%, 55%, 48%, 38%, and 23%, respectively. MVA revealed that pretreatment ipsilateral pure tone average ($p < .001$) and tumor size ($p = .009$) were statistically significantly associated with time to nonserviceable hearing.

Can SRS be used for larger tumors (>3 cm)?

Yang, Pittsburgh (*J Neurosurg* 2011, PMID 20799863): RR of 65 patients with VS between 3 and 4 cm in one extracanalicular maximum diameter (median tumor volume 9 mL) who underwent GKRS; 17 patients (26%) had previously undergone resection; 2 years later, 7 tumors (11%) had grown; 18 (82%) of 22 patients with serviceable hearing before SRS still had serviceable hearing after SRS more than 2 years later; 3 patients (5%) developed symptomatic hydrocephalus and underwent placement of a VP shunt. In 4 patients (6%), trigeminal sensory dysfunction developed, and in 1 patient (2%) mild facial weakness (House–Brackmann Grade II) developed after SRS. In univariate analyses, patients who had a previous resection ($p = .010$), those with tumor volume >10 mL ($p = .05$), and those with Koos grade 4 tumors ($p = .02$) had less likelihood of tumor control after SRS.

How does fractionated RT compare WITH SRS?

Fractionation offers a theoretical radiobiologic advantage compared to single-fraction treatment, which should allow for improved sparing of normal structures. However, evidence for differences in outcome between SRS and five-fraction or longer treatment courses is limited to retrospective data and may only improve hearing preservation.[27,38,39]

Coombs, Heidelberg (*IJROBP* 2010, PMID 19604653): Prospective cohort study of 200 patients with 202 VS treated with either LINAC-based SRS ($n = 30$) or FSRT ($n = 172$). SRS dose was 13 Gy to 80% isodose line, and FSRT median dose was 57.6 Gy/32 fx. MFU 75 months. No difference in 5-year LC (96% overall). FSRT and SRS showed equivalent hearing preservation (76% at 5 years) for SRS dose ≤13 Gy. For SRS dose >13 Gy ($n = 11$), hearing preservation was significantly worse than FSRT. Both patients who developed trigeminal neuralgia in the SRS group were treated with >13 Gy. Rate of facial nerve weakness was 17% in the SRS group and 2% in the FSRT group. Only 1 patient treated with SRS to ≤13 Gy developed facial weakness. **Conclusion: SRS with doses ≤13 Gy is a safe and effective alternative to FSRT. FSRT should be reserved for larger lesions.**

Meijer, Netherlands (*IJROBP* 2003, PMID 12873685): RR of 129 consecutive patients treated with either single-fraction or five-fraction RT using LINAC-based SRS techniques. Patients were prospectively selected for single fraction if edentate and five fractions if dentate due to the immobilization device used. Single-fraction arm treated with 10 to 12.5 Gy and five-fraction arm treated with 20 to 25 Gy. Patients in the single-fraction arm were older (mean age 63 years vs. 49 years), but there were no other significant differences between groups. No significant differences in 5-year LC (100% vs. 94%), facial nerve preservation (93% vs. 97%), and hearing preservation (75% vs. 61%); 5-year trigeminal nerve preservation was significantly different (92% vs. 98%, $p = .048$) favoring the fractionated group.

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