MANAGEMENT OF VESTIBULAR SCHWANNOMAS THAT ENLARGE AFTER STEREOTACTIC RADIOSURGERY: TREATMENT RECOMMENDATIONS BASED ON A 15 YEAR EXPERIENCE

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OBJECTIVE: Stereotactic radiosurgery is an effective alternative to surgical resection for the majority of patients with vestibular schwannomas (VS). However, after radiosurgery, the imaging characteristics of VSs are variable, and correct interpretation is critical to prevent unnecessary surgery for these patients.

METHODS: A retrospective study of 208 consecutive patients with unilateral VS having radiosurgery between March 1990 and December 2001. Thirty (14%) patients had tumors that enlarged at least 2 mm after radiosurgery. The median follow-up after radiosurgery was 56 months (range 24–132 mo).

RESULTS: The median time to tumor enlargement was 9 months (5–60 mo). The median volume increase was 75%. A loss of central enhancement was noted in 28 (93%) patients. Six (20%) patients had new symptoms noted at the time of tumor enlargement including hemifacial spasm (n = 2), ataxia (n = 2), trigeminal neuralgia (n = 1), and facial numbness (n = 1). Additional treatment was performed at the time of initial enlargement in 3 patients (resection, n = 2; ventriculoperitoneal shunt, n = 1). In the 28 patients who did not undergo resection at the time of initial enlargement, three patterns were identified on later imaging. Sixteen (57%) patients showed eventual tumor regression (type 1), and eight (29%) patients had tumors that increased and remained larger but did not show progressive enlargement (type 2). Four (14%) patients showed progressive enlargement on serial imaging (type 3) and underwent additional treatment (resection, n = 3; stereotactic radiation therapy, n = 1).

CONCLUSION: Tumor expansion after VS radiosurgery rarely denotes a failed procedure, and the majority of patients only require further imaging. Approximately one third of tumors that enlarge will remain increased in size compared with the time of radiosurgery but will not show sequential growth. Additional tumor treatment should be reserved only for patients who demonstrate progressive tumor enlargement on serial imaging (2% in this series).

KEY WORDS: Acoustic neuroma, Stereotactic radiosurgery, Vestibular schwannoma

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Stereotactic radiosurgery is an accepted alternative to surgical resection for patients with small- to moderate-sized vestibular schwannomas (VS). Although performed for more than 3 decades (15, 16), changes implemented over the past 15 years have vastly improved outcomes for VS patients having radiosurgery (3). Current expectations of this technique include a risk of facial weakness or numbness of less than 3% and hearing preservation in more than 50% of treated patients (3, 4, 6, 12, 15, 18, 21, 23). Despite more than 25,000 patients having VS radiosurgery (10), misinterpretation of the follow-up imaging is not uncommon, and physicians may prematurely conclude that radiosurgery has failed. Such errors can lead to patients having unnecessary and inappropriate surgery. In this report, management recommendations are presented on the basis of the imaging characteristics and clinical course of 30 VS patients whose tumors enlarged after radiosurgery over a 15 year interval.

MATERIALS AND METHODS

Between March 1990 and December 2001, 216 patients with unilateral VS underwent radiosurgery using the Leksell

Gamma Knife (Elekta Inc., Norcross, GA) at the Mayo Clinic, Rochester, MN. Twenty patients with neurofibromatosis type II having VS radiosurgery over this interval were excluded from the study population. Preoperative, dosimetric, and postoperative information was placed into a prospectively maintained database. Patients were requested and typically had follow-up evaluation and magnetic resonance imaging performed at 6, 12, 24, and 48 months then bi-yearly after radiosurgery. Tumors were measured in the x, y, and z planes and compared with a study performed on the day of radiosurgery. Tumor size was classified as unchanged, decreased, or increased. Tumors more than 2 mm larger in average diameter were coded as enlarged. Eight (4%) patients did not have any follow-up information available. Thirty of the remaining 208 (14%) patients were noted at some point after radiosurgery to have tumor enlargement and comprise the sample for this review. Comparison of the patients with tumor enlargement with those without tumor enlargement noted after radiosurgery showed no difference in mean age (58.0 versus 58.1 yr, $P = 0.99$), tumor volume (3.0 versus 3.2 cm^3, $P = 0.78$), tumor margin dose (13.9 versus 14.5 Gy, $P = 0.21$), or maximum radiation dose (28.1 versus 29.6 Gy, $P = 0.11$).

The clinical and dosimetric characteristics of the 30 patients are outlined in *Table 1*. The median patient age was 57 years (range 32–79 yr). No patient had received prior radiation for the tumor. The median initial tumor volume was 1.5 cm^3; the median tumor margin dose was 13.5 Gy. All the available imaging for these patients was reviewed using the method of Linskey et al. (11) by the author to determine tumor volumes. As noted by Linskey et al. (11), the change in tumor volume necessary for tumors to be objectively changed compared with the time of radiosurgery must account for potential measurement errors and the initial tumor size. Volume changes greater than 100%, 50%, and 25% were required to classify tumors with an average diameter of less than 1 cm, 1 to 2 cm, or more than 2 cm, respectively, as having changed in size compared with radiosurgery. The median follow-up after radiosurgery was 56 months (range 24–132 mo).

TABLE 1. Patient and dosimetric information

Factor	No. of patients (%)
Male/female	13/17
Prior resection	7 (23)
Tumor volume (cm^3)	
<1	13 (43)
1–4	10 (33)
>4	7 (23)
Tumor margin dose (Gy)	
≤12	12 (40)
13–14	10 (33)
>14	8 (27)
Maximum tumor dose (Gy)	
≤24	8 (27)
26–28	12 (40)
>28	10 (33)

RESULTS

The median time to tumor enlargement was 9 months (range 5–60 mo). The median volume increase was 75% (range 26–280%). A loss of central enhancement was noted in 28 (93%) patients at the time of tumor expansion. The two patients without a loss of enhancement did not demonstrate progressive growth. One patient underwent tumor resection at the time tumor enlargement was noted; the second patient showed eventual tumor shrinkage. Six (20%) patients had symptoms noted at the time of tumor enlargement including hemifacial spasm (HFS) (n = 2), ataxia (n = 2), trigeminal neuralgia (n = 1), and facial numbness (n = 1). Two patients underwent resection at the time of initial enlargement. One patient was a 79-year-old woman with a residual tumor (mean diameter 28 mm) who had increased ataxia 12 months after radiosurgery. The tumor had a loss of central enhancement and was 40% larger (3 mm). The patient improved after a course of corticosteroids and ventriculoperitoneal shunt placement, but the steroids were stopped after 2 weeks, and her symptoms recurred. She underwent a gross total resection 18 months after radiosurgery, and the facial nerve was severed during the operation. The second patient was a 66-year-old man who developed HFS 24 months after radiosurgery of a 17 mm tumor. Imaging showed the tumor to be 187% larger (4 mm) without a loss of central enhancement. He underwent a near gross total resection (24 mo after radiosurgery) with preservation of the facial nerve. At last follow-up, he had a mild facial weakness (House-Brackmann grade II) (5). One patient had increased ataxia after a large tumor (mean diameter 32 mm) increased 28% (3 mm) 5 months after radiosurgery. His symptoms resolved after placement of a ventriculoperitoneal shunt and a course of corticosteroids. The tumor returned to its original size on later imaging. The remaining three patients were managed medically for facial numbness (corticosteroids), trigeminal neuralgia (carbamazepine), and HFS (observation). The onset of symptoms was 6 months, 14 months, and 17 months, respectively. The patient with facial numbness has persistent facial numbness despite the tumor decreasing 50% in volume. Medical therapy controlled the trigeminal neuralgia; the patient with HFS slowly improved but continues to have some minor facial twitching more than 6 years after radiosurgery. Neither patient has required any further tumor treatments. Of note, the patient with trigeminal neuralgia also developed HFS 26 months after radiosurgery despite no further evidence of tumor enlargement.

Four patients underwent additional treatment after confirmed progressive tumor enlargement on sequential imaging between 23 and 60 months after radiosurgery. The final tumor volumes of these four patients were 35%, 110%, 203%, and 252% larger than at the time of radiosurgery, respectively. All these patients remained clinically stable despite clear tumor growth. Three patients underwent tumor resection, whereas

one patient was treated with stereotactic radiotherapy (SRT). One patient had a gross total tumor resection and postoperatively had a complete facial palsy. Two patients had a near total resection. Both patients recovered to near-normal facial movement after surgery (House-Brackmann grade II) (5). The patient having SRT received 30 Gy in 10 fractions. After SRT, the tumor has shown no further growth, and she continues to have normal facial movement.

Three patterns of tumor imaging were noted in the 28 patients who did not undergo resection at the time of initial enlargement and were classified as type 1, 2, or 3, respectively (*Fig. 1*). Type 1 patients (n = 16, 57%) showed eventual tumor regression to either the same (n = 12) or reduced (n = 4) volume at a median of 62 months (range 25–132 mo) (*Fig. 2*). Type 2 patients (n = 8, 29%) had tumors that increased and remained larger but did not show progressive enlargement (*Fig. 3*). The median follow-up after radiosurgery for the eight patients with a type 2 pattern was 56 months (range 23–87 mo). Type 3 patients (n = 4, 14%) showed progressive enlargement on serial imaging at a median follow-up of 31 months (range 23–60 mo) (*Fig. 4*). Patients having a type 3 pattern received a higher maximum radiation dose and a shorter follow-up interval compared with the patients demonstrating type 1 and 2 imaging patterns (*Table 2*).

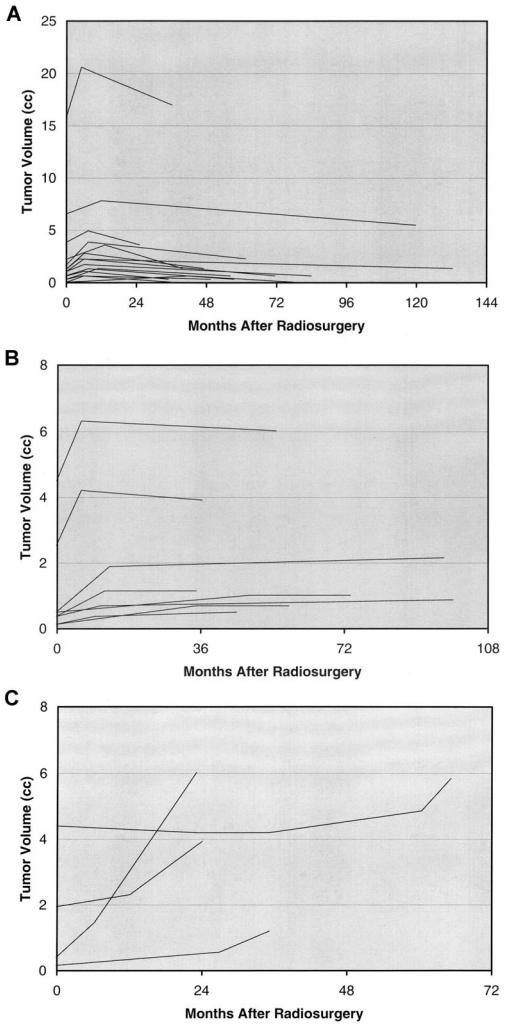

FIGURE 1. *Imaging of enlarged vestibular schwannomas after radiosurgery.* A, *type 1 pattern (n = 16) characterized by initial volume increase followed by later tumor reduction to either the same or reduced size compared to radiosurgery.* B, *type 2 pattern (n = 8) characterized by tumor volume increase that persists, but tumor does not enlarge on later imaging.* C, *type 3 pattern (n = 4) characterized by progressive tumor volume increase on serial imaging.*

DISCUSSION

There remains some controversy regarding the definition of failure after VS radiosurgery despite more than 30 years of clinical use. To correctly answer this question, some critical points regarding the imaging features of VSs after radiosurgery must be understood. First, it has been shown that as many as 80% of schwannomas will lose their central enhancement and may temporarily enlarge by several millimeters after radiosurgery (11, 14, 17, 23). Norén (15) noted that approximately 5% of tumors will exhibit such "swelling" and that at least 2 years should pass before determining a lack of response from radiosurgery. Kondziolka et al. (8) found that 5 of 162 (3%) patients had tumor expansion secondary to central necrosis but showed no later evidence of true neoplastic growth. As a result, surgical resection should be delayed until serial imaging over 2 or more years confirms that the tumor

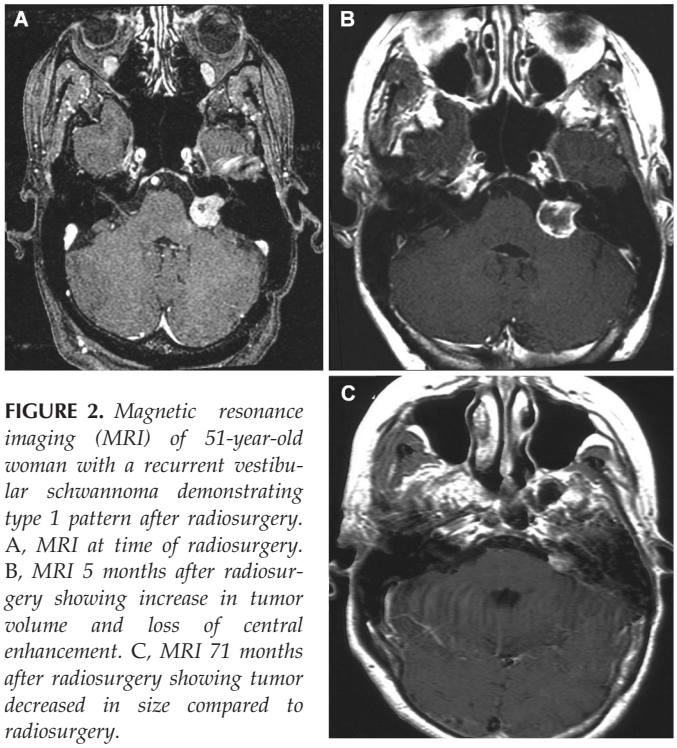

FIGURE 2. *Magnetic resonance imaging (MRI) of 51-year-old woman with a recurrent vestibular schwannoma demonstrating type 1 pattern after radiosurgery.* A, *MRI at time of radiosurgery.* B, *MRI 5 months after radiosurgery showing increase in tumor volume and loss of central enhancement.* C, *MRI 71 months after radiosurgery showing tumor decreased in size compared to radiosurgery.*

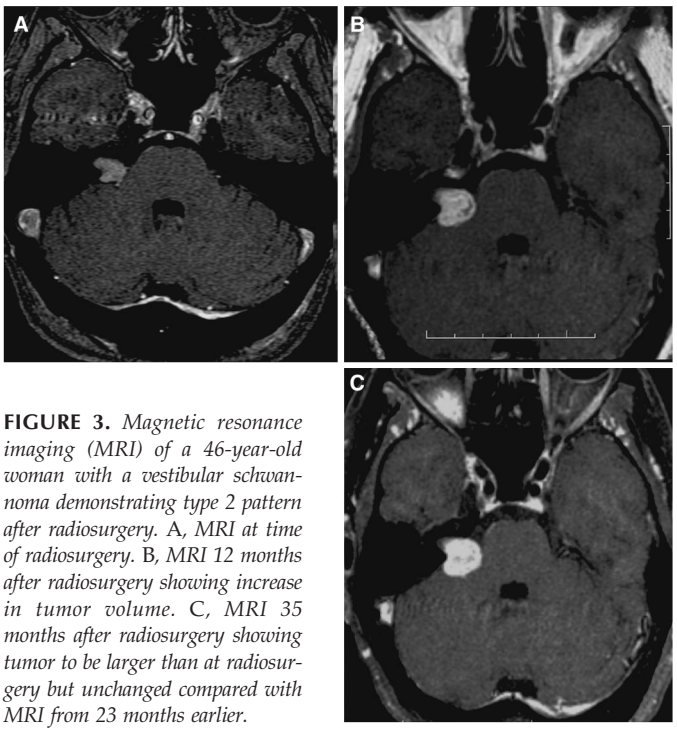

FIGURE 3. *Magnetic resonance imaging (MRI) of a 46-year-old woman with a vestibular schwannoma demonstrating type 2 pattern after radiosurgery.* A, *MRI at time of radiosurgery.* B, *MRI 12 months after radiosurgery showing increase in tumor volume.* C, *MRI 35 months after radiosurgery showing tumor to be larger than at radiosurgery but unchanged compared with MRI from 23 months earlier.*

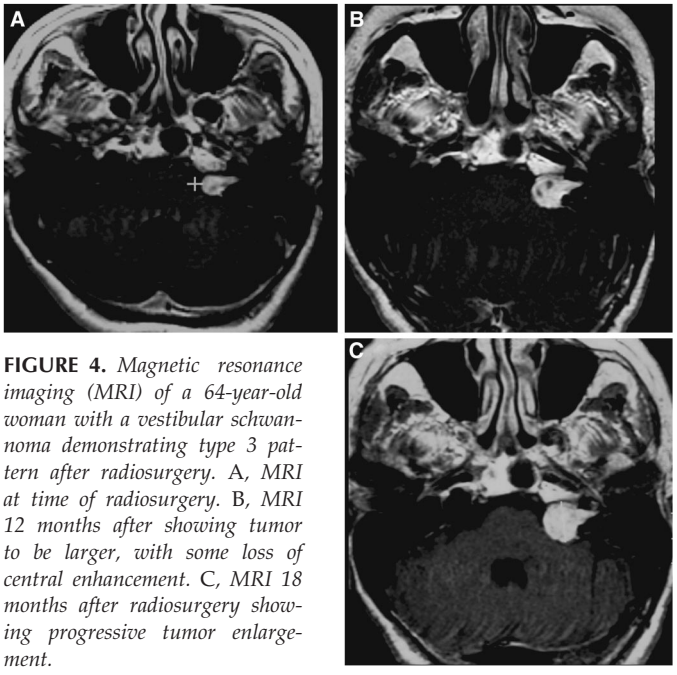

FIGURE 4. *Magnetic resonance imaging (MRI) of a 64-year-old woman with a vestibular schwannoma demonstrating type 3 pattern after radiosurgery.* A, *MRI at time of radiosurgery.* B, *MRI 12 months after showing tumor to be larger, with some loss of central enhancement.* C, *MRI 18 months after radiosurgery showing progressive tumor enlargement.*

continues to enlarge. Second, the number of patients showing tumor enlargement can vary significantly depending on the method used to measure the tumor. In this study, we used five linear measurements as described by Linskey et al. (11) to determine tumor volumes. We noted that 14% of our patients demonstrated tumor enlargement of more than 2 mm at some point after radiosurgery. Using similar methodology, Delsanti et al. (2) found that 15% of patients showed a significant increase in tumor size. However, this method may miss subtle changes in tumor volume and is prone to human error. A more accurate method than relying on two-dimensional measurements is to assess the tumor volume by digitally segmenting postradiosurgical magnetic resonance imaging to determine tumor volumes. In this manner, Yu et al. (28) found that 63% of patients had a transient increase in tumor volume that peaked at 6 months after the procedure. Five of 91 (6%) patients showed a persistent volume increase. Okunaga et al. (17) noted a temporary volume increase in 19 of 42 (45%) patients followed for more than 1 year after linear accelerator-based VS radiosurgery. In this series, eight (19%) patients had persistent tumor enlargement, but only three (7%) patients demonstrated continuous enlargement on serial magnetic resonance imaging. Third, to conclude that radiosurgery has failed on the basis of the onset of a new onset facial numbness or weakness is incorrect. Experience over the past 3 decades has demonstrated that many cranial neuropathies are temporary and will resolve without treatment. Moreover, surgical resection performed at the time of a deficit likely correlates with poor cranial nerve outcomes because the nerves are already dysfunctional and may be permanently injured by surgical manipulation. Fortunately, inappropriate surgery based on cranial nerve deficits rarely occurs anymore because the incidence of facial or trigeminal neuropathies after VS radiosurgery has dropped to less than 3%. By the definition of sequential tumor enlargement, the failure rate after VS radiosurgery at our center from 1990 through 2001 was 1.9% (4 of 208 patients).

This analysis of our center's 15 year experience with VS radiosurgery revealed two details that have not been fully addressed in previous papers but whose recognition may prevent some patients from having unnecessary surgery. One, some schwannomas may initially enlarge or "swell" after radiosurgery but never return to their original volume. In our series, 8 of 28 (29%) patients had tumors that initially enlarged and remained larger than at radiosurgery. This imaging sequence was defined as Type 2 to differentiate it from the more typical and well-described pattern of tumor expansion followed by volume reduction (type 1). By this method, patients with progressive growth on serial imaging were defined as Type 3. In our experience, no patient with a Type 2 pattern has shown later tumor progression at follow-up intervals up to 8.2 years (median 56 mo) after radiosurgery. The median tumor volume increase in these eight patients was 105%. Okunaga et al. (17) also observed early tumor enlargement followed by a growth plateau in their paper on volumetric tumor changes after linear accelerator-based VS radiosurgery. In that paper, 5 of 27 (19%) patients demonstrated a Type 2 pattern by our definition. Still, additional follow-up is needed to conclude that these tumors are incapable of growing, and we are not simply observing the natural history of a benign tumor that may have spontaneous periods of growth arrest.

TABLE 2. Characteristics of patients with tumor enlargement[a]

Enlargement pattern	Age (yr)	Volume (cm^3)	Margin Dose (Gy)	Maximum Dose (Gy)	Follow-up (mo)
Type 1 (n = 16)	54	1.2	12.5	27.4	62
Type 2 (n = 8)	59	1.2	14.0	28.0	56
Type 3 (n = 4)	58	2.2	15.5	33.0[b]	31[b]

[a] All numbers shown are median values.
[b] Patients with a type 3 enlargement pattern received a higher maximum radiation dose compared with patients with type 1 and 2 enlargement patterns ($P = 0.02$, Student's t test) and had a shorter follow-up interval ($P < 0.01$, Student's t test).

The second observation was that a small percentage of patients having VS radiosurgery develop HFS. Overall, six patients (three in this study) developed HFS of our total series of 337 (2%) VS patients having radiosurgery before 2005. The time to developing HFS after radiosurgery ranged from 13 to 26 months (median 20 mo), and in each case, the tumor was larger by several millimeters when compared with radiosurgery. One patient's tumor was removed shortly after the onset of symptoms, whereas the other five patients have undergone additional imaging. To date, none of the five tumors has shown further enlargement at a range from 24 to 78 months after radiosurgery. In four patients, significant improvement in the HFS was noted with either observation alone or a small dose of carbamazepine. Norén (15) noted HFS in 2% of the patients having VS radiosurgery at the Karolinska Institute from 1969 to 1994. In that experience, the HFS usually resolved after several months and was not associated with facial weakness. The one exception reported by Norén was a patient who developed HFS 2 years after radiosurgery that continued to worsen despite no evidence of tumor progression. Eleven years after radiosurgery, the patient underwent tumor removal and had a complete facial palsy postoperatively. This patient is Case 1 in the paper by Slattery and Brackmann (25). Régis et al. (23) noted HFS in 3% of their VS patients after radiosurgery. Information regarding the time to developing HFS or tumor size for these patients was not detailed.

One of the primary criticisms of VS radiosurgery is that if the tumor does not respond and continues to enlarge, then subsequent microsurgery is more difficult and surgical morbidity is increased (1, 9, 25, 27). Slattery and Brackmann (25) reported the results of VS resection in five patients who had undergone previous radiosurgery or radiation therapy 5 months to 12 years (median 12 mo) earlier. In each case, the authors commented that the tumor was scarred to the adjacent facial and vestibulocochlear nerves. The two patients with facial movement before surgery both had facial palsies postoperatively. They concluded that surgical morbidity after prior irradiation was increased. Battista and Weit (1) reported the results of a survey sent to members of the American Neurotology Society on the topic of surgical difficulties encountered in treating acoustic neuroma patients who had undergone stereotactic radiosurgery. The overall response rate of the survey was 36%; details on 12 patients having tumor resection after radiosurgery were provided. The mean time to surgery after radiosurgery was 35 months (range 3–72 mo). Of note, 7 of the 12 tumors were larger than 3 cm, and 4 of the 12 had recurrent tumors after prior surgical resection. Nine of the 12 operating surgeons commented that there was no clear plane between the tumor and the facial nerve. They concluded that microsurgical resection was technically difficult regardless of the time after radiosurgery and that cranial nerve results are uniformly poor. Lee et al. (9) presented the microsurgical results of four VS patients who had undergone either radiosurgery (n = 2) or SRT (n = 2) 1 to 2 years earlier. Three patients had developed HFS, whereas one patient developed facial numbness. Compared with the time of radiosurgery, the tumors had increased between 2 and 5 mm in greatest dimension. Fibrosis and scarring were noted in all four cases, making tumor dissection from the facial and caudal cranial nerves more difficult. A complete tumor resection was possible for each patient, and three patients retained House-Brackmann grade I or II function. On the basis of such reports and the perceived risk of malignant transformation, most neurotologists have concluded that surgical removal is clearly the treatment of choice for the majority of VS patients (22).

Conversely, advocates of radiosurgery quote five retrospective studies comparing the results of surgical resection and radiosurgery for VS patients (7, 13, 19, 23, 26). Each study found radiosurgery had improved facial nerve outcomes, hearing preservation rates, and lower overall morbidity. Patients returned to work faster after radiosurgery, and the costs associated with radiosurgical management were less than open surgery. The fact that surgical resection after radiosurgery is associated with greater morbidity has also been questioned. An earlier review of 13 patients from our center in conjunction with the University of Pittsburgh found no relationship between the use of radiosurgery and the subsequent ease or difficulty of later VS resection (20). Roche et al. (24) reported 20 patients having surgical resection of unilateral VS after radiosurgery. The mean time between radiosurgery and tumor removal was 36 months; the median tumor volume increase was 160%. In this series, the operating surgeon noted unusual surgical difficulty in nine (45%) cases because of adhesions to adjacent neurovascular structures. Tumor removal was graded as total in 14 cases, near-total in four cases, and subtotal in two cases. At last follow-up, 10 (50%) patients retained either normal or near normal facial movement (House-Brackmann grade I–II) (5). In our series, the two patients having gross total resection after failed radiosurgery had complete facial palsies postoperatively, whereas the three patients with near-total tumor resections preserved good fa-

cial movement. Therefore, if a tumor does show progression on serial imaging after VS radiosurgery, it seems that the best functional outcomes occur when a subtotal or near-total resection is performed rather than a gross total removal. Moreover, repeat radiosurgery or SRT should be considered if preservation of facial function remains a goal after failed radiosurgery. More information is needed to assess the results of such salvage radiation-based procedures.

It is notable that three of the four patients in the series of Lee et al. (9) on acoustic neuroma surgery after stereotactic radiosurgery had HFS. In their experience, patients developed HFS 1 to 2 years after radiation; the median time to onset of HFS in our patients was 21 months. Similar to our patients, the linear enlargement of the tumors was modest, ranging between 2 and 5 mm in greatest dimension compared with the time of radiosurgery (estimated volume change 39–240%). The primary difference in these two reports is that after our first case, we directed our treatment at the HFS, whereas they concluded that radiosurgery had failed and performed tumor removal. With observation and medical therapy, our last four patients have shown no further evidence of tumor progression, and their HFS is either absent or markedly reduced. These observations suggest that HFS after radiosurgery is distinct from cranial neuropathies that have been well described. The onset of HFS after VS radiosurgery seems to be delayed when compared with facial weakness. Whereas most facial neuropathies appear by 6 months after radiosurgery (11, 12, 15), HFS generally does not start until a year or more after radiosurgery. The fact that the HFS is usually temporary, is not associated with facial weakness, and often improves with the use of anticonvulsant medications suggests that it is caused by an irritation of the facial nerve. Tumor expansion as noted in this series and by Lee et al. (9) could very well stretch the facial nerve or compress it against the temporal bone. It can be postulated that because the tumor does not continue to enlarge, the facial nerve adapts over time, and the HFS resolves. Another possible explanation could be a delayed vascular insult to the facial nerve. Of interest, none of the four patients with serial progression on imaging developed HFS or any other new symptom. Consequently, tumor resection based on the development of HFS after VS radiosurgery should be avoided unless the tumor demonstrates clear enlargement on scans performed over a year or more.

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COMMENTS

The biological effect of radiosurgery on a schwannoma is a combination of direct effects on tumor cells (beginning with apoptosis and necrosis) and indirect effects related to delayed vascular occlusion

(1). The acute effect of radiosurgery is manifested via inflammation; tissue repair later proceeds through chronic inflammation and, later, granulation tissue formation. The subacute inflammation phase can lead to a tumor that appears slightly swollen and enlarged on imaging. Such tumors have often lost central contrast enhancement. Often the enlarged tumor is seen in patients with a "quick tumor death," subsequently followed by tumor regression. Overall, about 70% of tumors do regress after radiosurgery, as identified in systematic long-term analyses for 15 years. Dr. Pollock has provided a thoughtful analysis of the different responses observed on imaging.

When a tumor later appears to be a little bigger, it may be owing to tumor growth, but is more likely caused by the formation of excess granulation tissue. These fibroblasts and capillaries can lead to tumor "scar," but also to a picture of tumor re-enhancement. Thus, a tumor that enlarges 1 or 2 mm should be observed, but, often, the process stops. If a tumor continues to enlarge on serial imaging studies, it likely indicates tumor growth and this may need to be addressed with resection or repeat radiosurgery. Fortunately, this outcome is uncommon, with different gamma knife series reporting an incidence of 2%. The observations noted in this report need to be understood both by surgeons and radiologists, but also by our patients in advance of receiving radiosurgery.

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1. Witham T, Okada H, Fellows W, Hamilton R, Flickinger J, Chambers W, Pollack I, Watkins S, Kondziolka D: The characterization of tumor apoptosis after experimental radiosurgery. **Stereotact Funct Neurosurg** 83:17–24, 2005.

We really appreciate the original contribution from Pollock about the volumetric behavior of vestibular schwannomas that were carefully followed after gamma knife radiosurgery. Of particular interest is the information about a small subgroup of patients (Type 2) who should not be misunderstood as actual failures, but who do not display what we can expect as successful treatment. We also share the same experience of this subgroup and, like the author, think that this particular behavior does not justify a microsurgical removal, but rather requires long-term serial imaging (3). However, it is notable that several groups are observing a higher rate (approximately 30%) of tumor enlargement after radiosurgery, possibly owing to a difference in follow-up methods.

This study deserves several comments from a methodological perspective. Concerning the inclusion of patients in the follow-up group, the author only excluded eight patients from the analysis who did not have any follow-up information available. Thereby, he implicitly recognized that he took into consideration incomplete data from a number of patients. We can thus postulate that the sequential prospective follow-up was not achieved for all patients. Because this study is mainly based on radiological data, the author did not provide information about who assessed the magnetic resonance images. Did he assess them himself or did independent observers? We are not even sure that all patients underwent magnetic resonance imaging instead of computed tomographic scans at the time of treatment and during the follow-up period. As the measurement methodology, the author selected the Linskey (2) criteria to assess changes in tumor volume. We agree that this was an acceptable method at the start of this study, but we now consider it inaccurate. The generalized use of Gammaplan and fused computed tomographic and magnetic resonance imaging protocol at the time of treatment allow the calculation of tumor volume using the method recommended by Yu et al. (4). In the same way, is it appropriate to consider that volume changes of less than 100% for tumors with an average diameter of less than 1 cm should be classified as unchanged tumors? However, three-dimensional methodologies are raising technical problems, specially for older cases. Using a linear method of measurement (1), we found that 15% of the tumors enlarged compared with 14% in Pollock's series.

The author makes comments about surgery after microsurgery, and particularly about technical considerations regarding the microsurgical difficulties. Theses considerations are not broadly based on the results from his own study, but from literature that was not extensively analyzed. However, there is no doubt that facial nerve preservation is the main issue during the surgical management of vestibular schwannomas. We agree with the author that it is better for the patient to keep a small piece of tumor against the facial nerve instead of the radical removal and radical facial nerve deficit.

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1. Delsanti C, Tamura M, Galanaud D, Régis J: Changing radiological results, pitfalls and criteria of failure. **Neurochirurgie** 50:312–319, 2004.
2. Linskey ME, Lunsford LD, Flickinger JC: Neuroimaging of acoustic nerve sheath tumors after stereotaxic radiosurgery. **AJNR Am J Neuroradiol** 12: 1165–1175, 1991.
3. Roche PH, Régis J, Devèze A, Delsanti C, Thomassin JM, Pellet W: Surgical removal of unilateral vestibular schwannomas after failed gamma knife surgery. **Neurochirurgie** 50: 383–393, 2004.
4. Yu CP, Cheung JY, Leung S, Ho R: Sequential volume mapping for confirmation of negative growth in vestibular schwannomas treated by gamma knife radiosurgery. **J Neurosurg** 93[suppl 3]:82–89, 2000.

The authors present an interesting study on 30 (14%) patients out of 208 treated patients with unilateral vestibular schwannomas that enlarge after gamma knife treatment. In two patients, microsurgical resection was performed, one complete and one subtotally, and one patient received a shunt. Four (1.9%) patients showed progressive enlargement and needed further therapy, whereas the other 24 remained stable or eventually regressed.

New symptoms in the patient group with enlarging tumors were hemifacial spasm (2 out of 30), trigeminal neuralgia (1 out of 30), ataxia, and facial numbness. Interestingly, no symptoms such as hearing impairment, tinnitus, nausea, or headache were noted.

While the rate of occurrence of trigeminal neuralgia was reported to be 4%, and permanent in 1.6%, of patients (2), the occurrence of hemifacial spasm in 6% of this selected group of patients is cause for concern. Together with the cited data of Lee et al. (1) with three out of four patients having recurrent vestibular schwannoma after radiosurgery developing hemifacial spasm, this points to a higher occurrence of hemifacial spasm in patients with failed radiosurgery, than the presented 2% of all treated vestibular schwannomas.

Regarding the four patients with further growth after radiosurgery, three were treated with microsurgical resection, two subtotally and one with gross total tumor removal. One patient received additional radiosurgery. The patients with complete tumor removal developed complete facial palsy and thus the authors recommend subtotal tumor removal and the consideration of additional radiosurgery instead of gross tumor removal. It seems ethically questionable to recommend an already failed therapy in diseases that can be cured by other means.

In view of the benign nature of vestibular schwannomas, we would recommend microsurgical tumor removal as the first consideration for treatment. It offers the chance of a cure with a recurrence rate of 0.7 % in non-neurofibromatosis Type 2 tumors (3). With an anatomical facial

nerve preservation rate of 97% and hearing preservation rates of 43 to 54% in T1–T3 tumors that are amenable for both treatment options, the results are comparable to the radiosurgery results (4). In contrast, trigeminal neuropathy or facial spasm did not occur after surgery, but improved if it was a presenting symptom. Microsurgical tumor removal after failed radiosurgery still carries increased risks to the cranial nerve function, as described by the authors. Therefore, we would recommend radiosurgery only to patients, who are, for other reasons, not able to undergo a microsurgical tumor removal.

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1. Lee DJ, Westra WH, Staecker H, Long D, Niparko JK: Clinical and histopathologic features of recurrent vestibular schwannomas (acoustic neuroma) after stereotactic radiosurgery. **Otol Neurotol** 24:650–660, 2003.
2. Prasad D, Steiner M, Steiner L: Gamma surgery for vestibular schwannoma. **J Neurosurg** 92:745–759, 2000.
3. Samii M, Matthies C: Management of 1000 vestibular schwannomas (acoustic neuromas): Surgical management and results with an emphasis on complications and how to avoid them. **Neurosurgery** 40:11–21, 1997.
4. Samii M, Matthies C: Gamma surgery for vestibular schwannoma. **J Neurosurg** 92:892–894, 2000 (comment).

In this study, the authors bring up some very important points regarding the treatment of vestibular schwannomas with radiosurgery. The most important is that an increase in the size of the tumor does not necessarily mean failure. In fact, increase in size was a sign of lack of response in only four out of 28 vestibular schwannomas in the present study. In the other 24 tumors, it was a sign of a temporary internal reaction, likely indicating an unusually brisk response to the radiation and, as such, was probably a good prognostic sign.

One can only guess how many vestibular schwannomas have been unnecessarily resected over the past two or three decades because of misinterpreted temporary swelling. I remember very well the first such case I witnessed: a woman with a 2-cm tumor, who underwent radiosurgery in 1976 in Stockholm. Seven months later computed tomographic scans demonstrated reduced enhancement in the central area and increased size of the tumor. It was resected with the belief that the treatment was a failure. The patient did well postoperatively, but developed a complete facial paralysis. Pathology demonstrated widespread radiation-induced necrosis throughout most of the tumor. We then realized that increased tumor size was, perhaps, not the same as treatment failure.

Another source of misinterpretation is related to cranial neuropathies. Decreased or increased excitability of the Vth, VIIth or VIIIth cranial nerves can never be used as an indicator for a change of the size of the vestibular schwannoma, whether a decrease or an increase. Only imaging can be used for this purpose.

Regarding the time of onset of hemifacial spasm versus facial weakness, we did not find the difference reported by the author. Except for the patient quoted in the present article, where the spasm began 2 years after the treatment, the range was 2 to 11 months for the 2% (11 of 530) of patients who developed hemifacial spasm and 1 to 15 months for those with facial weakness (1). Maybe the difference in response of the nerve is mostly a matter of radiation dose (low dose → spasm, high dose → weakness), and that the basic pathophysiological mechanism is the same, or is at least similar.

The author's take-home message cannot be emphasized enough. Tumor expansion after vestibular schwannoma radiosurgery rarely denotes a failed procedure. Additional treatment should only be offered when the tumor demonstrates progressive enlargement on serial imaging. Cranial nerve function cannot be used as an indicator of what is going on with the tumor.

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1. Norén G: Long-term complications following gamma knife radiosurgery of vestibular schwannomas. **Stereotact Funct Neurosurg** 70[suppl 1]: 65–73, 1998.

This report examines a 15-year single institution experience treating vestibular schwannomas with stereotactic radiosurgery. The primary focus is on patients whose tumors enlarge after radiosurgical treatment. The study tries to determine when it is reasonable to reoperate for presumed treatment failure and when to patiently opt for conservative management. Patients are divided into three categories based on postoperative radiographic responses to radiosurgery. The key groups are Type 1 (tumors increased and then regressed) and Type 2 patients (tumors increased and remained larger but did not show progressive enlargement) who should be managed conservatively without surgery. Type 3 patients (tumors increased and progressed) ultimately required additional treatment, but this group is a small minority accounting for only 14% of all patients with tumor enlargement.

This study is derived from an initially large patient population and is only modestly compromised by its retrospective nature, the heterogeneous treatment group and radiosurgery protocol changes evolving over the 15 years. These experimental impurities notwithstanding, the study's conclusions are convincing and the finding of radiographic enlargement after radiosurgery should not initially be considered a treatment failure. This is a useful study that validates patience and conservative management in patients after radiosurgery. Dr. Pollock has provided physicians with a logical system for understanding postradiosurgery imaging changes in patients with vestibular schwannomas through this practical contribution to the literature.

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