Over time, the management options for arteriovenous malformations (AVMs) have expanded and evolved. As our knowledge base improves, individualized multimodal treatment has become a more recent management philosophy for AVMs. However, although more than 71,000 Gamma Knife surgery (GKS) AVM procedures have been performed at centers worldwide (1991–2011), an algorithm to appropriately manage all scenarios remains elusive. The goal of all management strategies has never waivered, because only complete obliteration of the AVM nidus dramatically reduces the risk of subsequent hemorrhage. The rates of AVM occlusion after GKS generally range from 70% to 80% over a 5-year observation interval.

The prevalence of an AVM is approximately 18 per 100,000 people, but only 5%–19% are found within or adjacent to the ventricular system. Such AVMs are located in a distinct anatomical environment that may affect both the clinical presentation and the response to stereotactic radiosurgery. We further hypothesized that periventricular and intraventricular AVMs may have an increased rate of pre- and postprocedure hemorrhage. Our review of the most recent 20 years of medical literature found limited information relating to AVMs in the ventricular region. This finding prompted us to review the outcomes and hemorrhage risks associated with intraventricular and periventricular AVMs managed by GKS at our center.

Object. The outcomes of stereotactic radiosurgery for arteriovenous malformations (AVMs) within or adjacent to the ventricular system are largely unknown. This study assessed the long-term outcomes and hemorrhage risks for patients with AVMs within this region who underwent Gamma Knife surgery (GKS) at the University of Pittsburgh.

Methods. The authors retrospectively identified 188 patients with ventricular-region AVMs who underwent a single-stage GKS procedure during a 22-year interval. The median patient age was 32 years (range 3–80 years), the median target volume was 4.6 cm³ (range 0.1–22 cm³), and the median marginal dose was 20 Gy (range 13–27 Gy).

Results. Arteriovenous malformation obliteration was confirmed by MRI or angiography in 89 patients during a median follow-up of 65 months (range 2–265 months). The actuarial rates of total obliteration were 32% at 3 years, 55% at 4 years, 60% at 5 years, and 64% at 10 years. Higher rates of AVM obliteration were obtained in the 26 patients with intraventricular AVMs. Twenty-five patients (13%) sustained a hemorrhage during the initial latency interval after GKS, indicating an annual hemorrhage rate of 3.4% prior to AVM obliteration. No patient experienced a hemorrhage after AVM obliteration was confirmed by imaging. Permanent neurological deficits due to adverse radiation effects developed in 7 patients (4%).

Conclusions. Although patients in this study demonstrated an elevated hemorrhage risk that remained until complete obliteration, GKS still proved to be a generally safe and effective treatment for patients with these high-risk intraventricular and periventricular AVMs.
Radiosurgery for ventricular AVMs

Methods

Patient Population

This single-institution retrospective analysis was approved by the University of Pittsburgh institutional review board. We selected patients with an AVM nidus that was in direct contact with a ventricular surface. This selection also included AVMs with a nidus that was completely contained within the ventricular system. These two groups have been subdivided where appropriate and will be referred to as periventricular and intraventricular, respectively. We identified 188 patients who underwent GKS during a 22-year interval between 1987 and 2009. The outcome data were collected through medical record review and analyzed by neurosurgeons who did not participate in the initial patient management. The median patient age was 32 years (range 3–80 years) and included 98 male and 90 female patients (Table 1). The AVM was diagnosed after brain imaging was performed because of an initial intracranial hemorrhage in 118 patients (63%), a seizure in 26 patients (14%), headache development in 25 patients (13%), or unexplained neurological deficit in 10 patients (5%). In 9 patients (5%), the AVM was diagnosed incidentally after brain imaging was performed for other reasons. The AVM location was periventricular in 162 patients (86%) and intraventricular in 26 patients (14%; Table 1). Arteriovenous malformation involvement of the lateral ventricles occurred in 133 patients, the third ventricle in 46 patients, and fourth ventricle in 9 patients. A hemorrhagic presentation was more common in the 21 patients with intraventricular AVMs (81% with initial bleed presentation) compared with the 98 patients who had a periventricular AVM (60% presented with a bleeding event).

Neurological deficits prior to GKS were observed in 72 patients (38%). Hemorrhage was the cause of the deficit in 58 patients, stroke after embolization in 8 patients, and stroke related to endocarditis or prior brain surgery (1 patient each). Hemiparesis was the most common finding (53 patients), followed by visual field and ocular motor deficits (21 patients), speech-related symptoms (7 patients), memory difficulty (4 patients), and other cranial nerve problems (3 patients). Fourteen patients had more than 1 deficit.

A coexisting aneurysm was identified in 23 patients (12%) and a venous outflow varix in 28 patients (15%). Endovascular embolization was conducted one or more times in 34 patients (18%) prior to GKS (Table 1). Adverse effects from embolization occurred in 8 patients. Surgical intervention was required in 25 patients (13%) prior to GKS. Nine patients underwent a craniotomy for clot evacuation, 7 patients had a ventriculostomy, 5 patients had partial AVM resection, and 4 patients underwent clipping of a coexisting aneurysm. Surgery prior to GKS was undertaken more often in female patients (n = 15) than male patients (n = 10; p = 0.037). This difference was also noted for embolization, in which 22 female patients were treated compared with 12 male patients (p = 0.030).

The Spetzler-Martin grading was determined by 2 experienced neurosurgeons.31 A Grade I AVM was diagnosed in 4 patients (2%), Grade II in 27 patients (14%), Grade III in 115 patients (61%), Grade IV in 27 patients (14%), and Grade VI in 15 patients (8%; Table 1). The Pollock-Flickinger score was calculated as Grade I (<1) for 48 patients (26%), Grade II (1–1.50) for 60 patients (32%), Grade III (1.51–2.0) for 55 patients (29%), and Grade IV (>2) for 25 patients (13%).

Table 1: Summary of demographics and AVM characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>no. of patients</td>
<td>188</td>
</tr>
<tr>
<td>age (yrs)</td>
<td>median 32</td>
</tr>
<tr>
<td></td>
<td>range 3–80</td>
</tr>
<tr>
<td>sex</td>
<td>male 98 (52)</td>
</tr>
<tr>
<td></td>
<td>female 90 (48)</td>
</tr>
<tr>
<td>location</td>
<td>periventricular 162 (86)</td>
</tr>
<tr>
<td></td>
<td>intraventricular 26 (14)</td>
</tr>
<tr>
<td>presentation</td>
<td>bleed 121 (64)</td>
</tr>
<tr>
<td></td>
<td>headache 25 (13)</td>
</tr>
<tr>
<td></td>
<td>seizure 26 (14)</td>
</tr>
<tr>
<td></td>
<td>incidental 11 (6)</td>
</tr>
<tr>
<td></td>
<td>neurological deficit 5 (3)</td>
</tr>
<tr>
<td>prior embolization</td>
<td>34 (18)</td>
</tr>
<tr>
<td>prior surgery</td>
<td>25 (13)</td>
</tr>
<tr>
<td>varix present</td>
<td>28 (15)</td>
</tr>
<tr>
<td>coexisting aneurysm</td>
<td>23 (12)</td>
</tr>
<tr>
<td>Spetzler-Martin Grade</td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>4 (2)</td>
</tr>
<tr>
<td>II</td>
<td>27 (14)</td>
</tr>
<tr>
<td>III</td>
<td>115 (61)</td>
</tr>
<tr>
<td>IV</td>
<td>27 (14)</td>
</tr>
<tr>
<td>VI</td>
<td>15 (8)</td>
</tr>
<tr>
<td>Pollock-Flickinger score</td>
<td></td>
</tr>
<tr>
<td>1 = &lt;1</td>
<td>48 (26)</td>
</tr>
<tr>
<td>2 = 1–1.50</td>
<td>60 (32)</td>
</tr>
<tr>
<td>3 = 1.51–2</td>
<td>55 (29)</td>
</tr>
<tr>
<td>4 = &gt;2</td>
<td>25 (13)</td>
</tr>
</tbody>
</table>

Radiosurgical Technique

On the day of treatment, patients were administered intravenous sedation or, in the case of children, general anesthetic and endotracheal intubation. The Leksell stereotactic frame was applied with local anesthetic administered to the pin sites. High-resolution axial imaging (MRI after 1991) was then conducted, followed by biplanar stereotactic angiography. Gamma Knife surgery was calculated to deliver, in a single procedure, an AVM edge margin dose that corresponded to the 3D nidus volume. This study spans the use of Leksell Gamma Knife (Elekta AB) models U, B, C, 4C, and Perfexion. At the conclusion of treatment all patients received 20–40 mg of in-
travenous methylprednisolone. Patients were discharged within 2–24 hours after the procedure. Expanded technical elements of this technique have been detailed in our previous publications.19,27

The median target volume was 4.6 cm³ (range 0.1–22 cm³), which was associated with a median maximum AVM nidus diameter of 2.6 cm (range 0.6–5.2 cm). The median marginal dose was 20 Gy (range 13–27 Gy) and median maximum dose was 36 Gy (range 18–50 Gy). The median number of isocenters used was 4 (range 1–15; Table 2).

**Patient Follow-Up**

Clinical and imaging follow-up was requested at 6, 12, 24, and 36 months after GKS. If any changes in neurological symptomatology occurred, the patient was investigated promptly with imaging or angiography to assess for hemorrhage or adverse radiation effects. After 3 years, if MRI or CT demonstrated total AVM obliteration (no flow voids identified), then angiography was requested. Complete AVM obliteration via angiography was defined as an elimination of the AVM nidus and the absence of early draining veins.10,11 If a residual nidus was evident on imaging, then repeat GKS was considered to obtain complete AVM obliteration. Any patient receiving a second GKS treatment was once again followed with a similar protocol.

**Statistical Analysis**

Kaplan-Meier survival analysis was used to demonstrate correlations and for graphic interpretations of obliteration rates based on significant factors. The timing of documented obliteration was determined to be the date that CT, MRI, or angiography demonstrated complete occlusion of the AVM. Basing obliteration results solely on the timing of angiography biases outcome data. For example, 26 patients who demonstrated complete obliteration by MRI failed to have a confirmatory angiogram, then 63 patients had total AVM obliteration. As documented in previous studies, angiography results are falsely lowered by patients who fail to undergo angiography after obliteration is determined on MRI.8,10 Several variables were significant predictors for AVM obliteration based on statistical analyses. A smaller AVM nidus volume led to an increased rate of confirmed obliteration (p = 0.013), especially in patients whose AVM was \( \leq 4 \) cm³ (Fig. 2). The GKS margin dose was higher in patients who attained total AVM obliteration (p = 0.004; Fig. 3). Patients with a prior hemorrhage had a higher rate of complete AVM obliteration (p = 0.046). The probability of AVM obliteration correlated with a lower Pollock-Flickinger score (p = 0.025).

**Total AVM Obliteration**

An AVM obliteration was attained in 89 patients as documented by MRI or angiography. The actuarial rates of total AVM obliteration were 32% at 3 years, 55% at 4 years, 60% at 5 years, and 64% at 10 years (Fig. 1). The median time until AVM obliteration was documented as 43 months (95% CI 37–48 months). If angiography was used alone to confirm obliteration (26 patients with MRI evidence of obliteration failed to have a confirmatory angiogram), then 63 patients had total AVM obliteration. As documented in previous studies, angiography results are falsely lowered by patients who fail to undergo angiography after obliteration is determined on MRI.8,10

Several variables were significant predictors for AVM obliteration based on statistical analyses. A smaller AVM nidus volume led to an increased rate of confirmed obliteration (p = 0.013), especially in patients whose AVM was \( \leq 4 \) cm³ (Fig. 2). The GKS margin dose was higher in patients who attained total AVM obliteration (p = 0.004; Fig. 3). Patients with a prior hemorrhage had a higher rate of complete AVM obliteration (p = 0.046). The probability of AVM obliteration correlated with a lower Pollock-Flickinger score (p = 0.025).

**Results**

The median imaging follow-up after GKS was 65 months (range 2–265 months). At the time of review 17 patients had died. Thirteen deaths were directly attributable to AVM hemorrhage, 2 were unrelated (leukemia and myocardial infarction), and 2 were undetermined.

**TABLE 2: Summary of GKS treatments**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Median (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>target volume (cm³)</td>
<td>4.6 (0.1–22)</td>
</tr>
<tr>
<td>max diameter (cm)</td>
<td>2.6 (0.6–5.2)</td>
</tr>
<tr>
<td>margin dose (Gy)</td>
<td>20 (13–27)</td>
</tr>
<tr>
<td>max dose (Gy)</td>
<td>36 (18–50)</td>
</tr>
<tr>
<td>no. of isocenters</td>
<td>4 (1–15)</td>
</tr>
</tbody>
</table>

**Fig. 1.** Kaplan-Meier curve for total AVM obliteration based on MRI and angiography combined. Patients remaining in the analysis are indicated at 3 and 5 years.
Radiosurgery for ventricular AVMs

Periventricular AVMs had an actuarial rate of obliteration of 29% at 3 years, 51% at 4 years, 57% at 5 years, and 61% at 10 years. In comparison, intraventricular AVM obliteration rates were 50% at 3 years, and 76% at 4, 5, and 10 years after GKS (Fig. 4). This difference in rates was statistically correlated with a higher obliteration rate of intraventricular AVMs (p = 0.029). It is noteworthy that the treatment volume of intraventricular AVMs was significantly smaller than in the periventricular group (p = 0.019).

Hemorrhage Risks

A hemorrhage prior to GKS was more frequent in younger patients (p = 0.008) and patients with a smaller AVM volume (p < 0.001). Intraventricular AVMs were more prone to a presentation from hemorrhage (p = 0.041) than periventricular AVMs. Patients with a prior bleeding event were also more likely to have deep venous drainage (p = 0.049). However, a venous outflow varix was associated with fewer hemorrhages (p = 0.0003).

Twenty-five patients (13%) experienced a hemorrhage during the latency period at a median of 24 months (range 2–118 months). Thirteen of those patients died as a result of the hemorrhage at a median of 16 months after GKS. One patient had a second hemorrhage 3 months after the initial event. The cumulative rate of AVM hemorrhage after GKS was 6.5% at 1 year, 12% at 3 years, 13.6% at 5 years, and 16.4% at 10 years (Fig. 5). This correlated with 756 patient-years of estimated hemorrhage risk, for an overall annual rate of 3.4% during the latency interval (time from treatment until obliteration or last follow-up with a patent AVM). The likelihood of having a bleeding event during the latency period was significantly greater in patients who had deep venous drainage (p = 0.01), but paradoxically...
lower if a venous outflow varix was present \((p = 0.05)\). In addition, a larger AVM volume \((p = 0.01)\) and a lower GKS margin dose \((p = 0.004)\) were risk factors for a bleeding event after GKS. A postprocedure bleed occurred in 13 patients \((11\%)\) with a hemorrhage prior to GKS and in 12 patients \((17\%)\) without a prior hemorrhage. Twenty-four of the hemorrhages after GKS occurred in the periventricular group and only 1 in the intraventricular group. No patients suffered a hemorrhage after confirmed AVM obliteration by MRI and angiography.

As the result of post-GKS hemorrhages, 5 patients required intervention beyond radiosurgery. Three patients required operative management for clot evacuation, during which resection of the AVM nidus was conducted in 2 of these patients. The timing of these procedures was 4, 6, and 35 months after their respective GKS procedures. One patient required a shunt due to an intraventricular hemorrhage at 42 months after GKS. Finally, a single patient underwent embolization 4 months after GKS in response to a hemorrhage.

**Clinical Outcomes**

There were 72 patients with neurological deficits prior to GKS; at the last recorded follow-up evaluation, 16 patients had shown improvement (Table 3). Improvement was reported at a median of 27 months \((\text{range } 4–44 \text{ months})\) after GKS. However, only 4 patients experienced a complete return to baseline function. Recovery from a previous hemorrhage appeared to be a factor in 11 patients, benefit from GKS obliteration of the AVM in 4 patients, and recovery from an ischemic event after embolization event in 1 patient. Sixteen patients had permanent worsening of their preexisting neurological deficits or developed a new deficit after GKS. New deficits resulted from latency interval hemorrhage in 8 patients \((4\%)\). Thirteen patients \((7\%)\) developed a symptomatic adverse radiation effect, defined as new T2 MRI signal changes adjacent to the AVM target associated with a new neurological deficit not caused by hemorrhage. The median onset of adverse radiation effect symptoms or signs was 14 months \((\text{range } 1–48 \text{ months})\) after GKS. Transient deficits accounted for 5 \((3\%)\) of these patients. These latter patients improved after a short course of oral corticosteroids. Associated T2 signal adjacent to the AVM target resolved over a median of 6 months \((\text{range } 2–31 \text{ months})\). Neurological deficits after GKS were found less frequently in patients with intraventricular AVMs \((p = 0.033)\). The risk of a new neurological deficit increased as the AVM volume treated increased \((p = 0.043)\). Delayed asymptomatic cyst formation was identified after GKS in 3 patients at a median of 11 months \((\text{range } 9–44 \text{ months})\). These patients did not require neurosurgical intervention and no other side effects were identified in this group.

**Repeat Radiosurgery**

Twenty-five patients with residual AVMs at the 3-year follow-up underwent a second GKS procedure at a median of 42 months \((\text{range } 36–125 \text{ months})\). Thirteen of the 25 patients had MRI-confirmed AVM obliteration at a median interval of 39 months \((\text{range } 15–60 \text{ months})\) after the second procedure. A single patient reported headache and disequilibrium 52 months after the second GKS procedure; these symptoms resolved within 7 months.

**Discussion**

**Factors Related to AVM Obliteration**

Individualized multimodal management has gained increasing traction for patients with newly diagnosed AVMs. The selected intervention strategies must provide a balance between safety and effectiveness, but ultimately lead to AVM obliteration. The rate of AVM obliteration in this study after a single GKS procedure was 60% at
Radiosurgery for ventricular AVMs

5 years. Multiple variables were associated with successful obliteration. An AVM volume ≤ 4 cm³ led to a higher rate of AVM obliteration. This volume effect has been well-defined in prior studies. The AVM margin dose is closely related to the success of obliteration, with higher doses usually delivered to smaller-volume AVMs. The present study again verified the relationship between obliteration and dose delivered to the margin. Arteriovenous malformations below 4 cm³ received a median of 22 Gy compared with 17 Gy in the > 4 cm³ group. There was a wide AVM volume distribution in this study, with a few AVMs exceeding 20 cm³. All AVMs in this study were treated with a single GKS procedure. However, as documented by Kano et al., these large AVMs may have benefited from a staged procedure. In that report, the average AVM nidus volume per procedure was 9.5–11.5 cm³. The decision to perform upfront staged stereotactic radiosurgery is based on analysis of the AVM location, prior bleeding history, estimated safe and effective dose, and estimated risk of adverse radiation effect.

Patients who had a prior hemorrhage also experienced a higher rate of AVM obliteration. We found that many of the patients who presented with an AVM bleed had smaller AVM volumes and were eligible to receive a higher dose to increase the chance of obliteration. The overall rate of obliteration in this study is comparable to other stereotactic radiosurgery outcome studies for patients with AVMs of the brainstem, thalamus, and basal ganglia. In such studies the initial single-procedure obliteration rates range from 52% to 73% in the literature.

We also noted that periventricular AVMs had an obliteration rate of 57% at 5 years compared with the 77% obliteration rate noted for patients with intraventricular AVMs. This finding is related to the decision to deliver higher doses to smaller AVMs located in the ventricle as well as the greater safety of delivering higher doses because of the rapid fall off of dose within a CSF structure rather than brain tissue.

Factors in AVM Management Decisions

The expected outcomes of AVM management options have been improved by the use of prospective grading systems and studies of AVM angioarchitecture. However, no single algorithm has emerged to provide definitive guidelines for all patients. For eligible patients, complete surgical removal of the AVM provides the benefit of early hemorrhage protection, but at the potential expense of new neurological morbidity. Two publications related to surgical removal of AVMs predominantly involving the lateral ventricle found that a new postoperative morbidity developed in 12%–14% of patients. Endovascular embolization of periventricular AVMs resulted in a 50% occlusion rate and an 8% risk of new permanent neurological disability. Although only 4% of patients in the present study developed a new or worsened permanent neurological deficit, 25 patients suffered a hemorrhage and 13 patients died during the 3-year latency interval after GKS. Patients selected for stereotactic radiosurgery usually have deep-seated ventricular or periventricular AVMs, considered high risk for surgical removal. In the present experience, 8% of the patients had Spetzler-Martin Grade VI AVMs.

Our analysis also indicates that such deep-seated AVMs in or adjacent to the ventricle have a higher risk of hemorrhage in comparison with more superficial supratentorial AVMs. The percentage of patients presenting with hemorrhage in the periventricular group was 60%, and was even higher in the intraventricular group (81%). This hemorrhage rate also exceeds the averaged intracranial prior hemorrhage rate of 52% in meta-analyses. Prior publications indicate an intraventricular AVM hemorrhagic presentation rate of 86%–99%, which is similar to what our study revealed. Meta-analysis data have also noted significantly increased bleeding risks in patients with prior hemorrhage, aneurysms, and deep venous drainage. In the current study, 64% of patients presented with a hemorrhage and 86% of these patients had deep venous drainage.

Our study provides further support to the observation that a larger AVM volume and a lower GKS margin dose are associated with a higher rate of hemorrhage during the latency period. Nagy et al. also demonstrated an elevated post–stereotactic radiosurgery bleed rate greater in unruptured large thalamic and basal ganglia AVMs; inevitably, due to their ventricular proximity, these regions overlap the regions investigated in this study, which could make the results from the 2 studies comparable. There was no statistical difference in the risk for bleeding after radiosurgery between patients who had already sustained a hemorrhage and patients who had never had a bleeding event. The overall annual risk of hemorrhage during the latency interval was 3.4%. Interestingly, the annual risk for latency interval rebleeding in intraventricular AVMs, which often present with an initial bleed, was < 1%. No patient had a bleeding event once the AVM was confirmed as obliterated.

Repeat GKS was undertaken when incomplete AVM obliteration was noted on MRI at 3 or more years after initial GKS, to improve the chances of AVM obliteration. We confirmed that 13 of 25 additional patients then achieved total obliteration. There was only 1 transient neurological symptom in the retreatment group.

Study Limitations

The authors acknowledge that this report would benefit from a longer duration of patient follow-up. Although all patients were prospectively entered into a database at the time of GKS, this report is a retrospective review of outcomes. The small number of patients with purely intraventricular AVMs and the great variation in AVM volumes prevented our ability to use a case-matching methodology to compare outcomes in the 2 subgroups. In the future, a calculation of the AVM surface area in contact with the ventricular system might define whether the adjacent brain parenchyma provides any tamponade effect that would reduce the risk of AVM hemorrhage during the latency interval.

Conclusions

Factors that improved the rate of total AVM oblit-
eration included an intraventricular location, a volume ≤ 4 cm³, a GKS margin dose ≥ 20 Gy, and a hemorrhagic presentation prior to GKS. Despite the unfavorable location of AVMs associated with the ventricular system, a permanent treatment-related neurological deficit developed in only 4% of patients. Although intraventricular and periventricular AVMs have been increasingly treated by stereotactic radiosurgery, it is important to understand that this subgroup may have an elevated hemorrhage risk. The annual hemorrhage rate during the latency interval period after GKS was 3.4%, resulting in 13 deaths from AV hemorrhage. After obliteration was confirmed, no patient suffered a hemorrhage. The 7% mortality rate of these AVMs within 3 years after GKS and the 0% mortality rate after obliteration provide evidence of the high-risk nature of these AVMs, as well as the potential achievable benefit if GKS is performed.

Acknowledgment

The authors thank Professor Douglas Kondziolka, M.D. (New York University Langone Medical Center), for significant contribution to patient management.

Disclosure

Dr. Lunsford is a consultant and stockholder for AB Elekta. The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Kano, Bowden, Lunsford. Acquisition of data: Kano, Bowden, Yang. Analysis and interpretation of data: Bowden. Drafting the article: Kano, Bowden, Lunsford. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Kano. Statistical analysis: Bowden. Study supervision: Kano, Lunsford.

References

Radiosurgery for ventricular AVMs

40. Zipfel GJ, Bradshaw P, Bova FJ, Friedman WA: Do the morphological characteristics of arteriovenous malformations affect the results of radiosurgery? *J Neurosurg* 101:393–401, 2004

---

Manuscript submitted September 5, 2013.
Accepted April 21, 2014.

Please include this information when citing this paper: published online May 30, 2014; DOI: 10.3171/2014.4.JNS131943.

Address correspondence to: Hideyuki Kano, M.D., Ph.D., Department of Neurological Surgery, University of Pittsburgh, Ste. B-400, UPMC Presbyterian, 200 Lothrop St., Pittsburgh, PA 15213. email: kanoh@upmc.edu.