Letters to the Editor
NEUROSURGICAL FORUM

Save the nerve

TO THE EDITOR: We read with keen interest the article by Yamakami and associates[27] regarding the resection of small acoustic neuromas through the retrosigmoid approach (Yamakami I, Ito S, Higuchi Y: Retrosigmoid removal of small acoustic neuroma: curative tumor removal with preservation of function. *J Neurosurg 121:*554–563, September 2014). We commend the authors for the excellent outcome they achieved with their technique and believe that their article is a decisive one with regard to the preference of surgical removal of small vestibular schwannomas, particularly in younger patients, over other options. Although their emphasis was on preserving serviceable hearing, the success in preserving any hearing, regardless of how poor it might be, indicates the viability of the cochlear nerve. Preserving the hearing anatomy (the inner ear and cochlear nerve) in all patients for preservation of hearing or potential functional restoration has been our avid interest and has incited us to rethink the management of all acoustic tumors, regardless of the size or the patient's hearing status.

The remarkable success of cochlear implants in restoring hearing in patients with postlingual deafness is, in our opinion, a "game-changer" in the management of all vestibular schwannoma. In postlingual deaf patients, cochlear implants offer 70%–100% word recognition and 65%–80% sentence recognition.[17,20,21] Modern cochlear implants have the far more ambitious goal of restoring speech perception for patients. In favorable hearing conditions and with auditory training, implantation of these devices can lead to an appreciation of more complex sound, including aspects of music perception.[14] Impressive results have been obtained with cochlear implantation in patients with neurofibromatosis Type 2, as long as the nerve is kept intact.[4,6,21] Thus, the prospect of restoring functional hearing must necessarily influence today's management and approach to patients with unilateral acoustic neuromas.

The translabyrinthine approach inescapably sacrifices hearing, even though some pathological studies have suggested that some spinal ganglion cells might still be alive after labyrinthectomy.[7] The middle fossa approach puts the facial nerve at higher risk.[12,15,19,23] Although the middle fossa approach might be equal to the posterior fossa approach in preserving serviceable hearing,[23] it might risk the cochlea and the part of the nerve near the fundus. In particular, this approach might involve dissection distally in the fundus, and reports show that a distal extension into the meatus is a significant adverse factor in preserving hearing.[16] In addition, it was thought that a posterior fossa approach is more advantageous as far as complications are concerned.[23,27] In the quest to preserve the cochlear nerve and function, we believe that the posterior fossa approach should be the approach of choice to all acoustic tumors. To alleviate the inherent risk of cerebellar retraction, which is associated with the retrosigmoid approach, we use the transmastoid approach and finesse it with endoscopic techniques.[1,2]

Decreasing the prescribed radiosurgery dose to 12–13 Gy has been reported, with a rate of 61%–78% early hearing preservation of patients with small tumors.[10,18,26] Undeniably, however, there have been definitive, progressive, and permanent declines of hearing over the years after radiosurgery, to a very low level of hearing.[5,8,11,22] The damage appears on the cochlear nerve, at the cochlear level, and the salient, identifying factor is a cochlear dose higher than 3 Gy.[11,26] In one study, the only patients who maintained hearing were those who received a cochlear dose of less than 2 Gy.[3] Exceeding such a dose is expected in all radiosurgery patients, with rare exceptions. Linskey and colleagues[13] calculated the doses that different parts of the cochlea receive during radiosurgery for acoustic tumors, and found a range of 5–8 Gy. These facts suggest that radiation-induced injury would compromise the potential for hearing restoration through a cochlear implant, and that radiosurgery is disadvantageous for long-term preservation or potential restoration of hearing.

Preserving hearing, the cochlea, and the cochlear nerve is highly achievable in patients with tumors of all sizes, including giant ones, through the retrosigmoid approach.[9,24] The cochlear nerve was preserved in 84% of a recent series of 200 patients reported by Samii et al.[25] Most patients would be candidates for hearing restoration even if useful hearing was not preserved. Admittedly, the value and experience in applying a cochlear implant in the presence of another good hearing ear has yet to be determined, but the potential is vast.

Hence, as Yamakami and his colleagues recommended for the small tumors, we advocate curative tumor removal with the preservation of the cochlear nerve for potential hearing restoration in all surgically fit patients, regardless of the tumor size or the hearing status.

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Response

We greatly appreciate Dr. Al-Mefty and colleagues' interest in our article concerning the retrosigmoid curative removal of small acoustic neuroma with functional preservation.

Dr. Al-Mefty advocates the curative removal of tumor with hearing and cochlear nerve preservation in all patients with acoustic neuroma, regardless of the tumor's size or the patient's hearing status. We agree that, even in the large acoustic neuroma, the optimum goal is curative tumor removal with preservation of the facial nerve and hearing, and that surgeons must make every effort to accomplish this goal. Actually, during the same study period of 1998–2012 as our article, we accomplished curative tumor removal with facial nerve and hearing preservation in 8 patients with large acoustic neuroma (30- to 50-mm tumor diameter; our unpublished data). We have the possibility to accomplish both curative tumor removal and hearing preservation, even in large acoustic neuromas. However, the possibility is much smaller than 84% in small acoustic neuromas with preoperative hearing, which our article reported.

Expressing the possibility of hearing restoration by the future development of cochlear implants, Dr. Al-Mefty points out the importance of anatomical preservation of the cochlear nerve. Using continuous monitoring of cochlear nerve compound action potential (CNAP) during removal of small acoustic neuromas with hearing preser-

vation, we found out that mechanical injury of the cochlear nerve by surgical manipulation was the most common cause of postoperative hearing loss, and that the hearing did not recover in spite of the anatomical preservation of cochlear nerve.[1] Once the cochlear nerve loses the function of nerve conduction intraoperatively, the nerve preserved anatomically may not restore the nerve conduction and hearing function years after tumor removal. We are not so optimistic for future hearing restoration by the cochlear implant in acoustic neuroma patients postoperatively.

We agree with Dr. Al-Mefty and associates that the retrosigmoid approach is the surgical approach of choice to all acoustic neuromas in the quest for hearing preservation. Our published article showed that, compared with the middle fossa approach, the retrosigmoid approach accomplished better facial function and the same hearing preservation. However, we emphasize that even in the retrosigmoid approach, the surgical manipulations near the fundus of the internal auditory canal (IAC) are most critical for preserving the nerve function. Continuous CNAP monitoring during acoustic neuroma removal showed that patients with tumor dissection near the fundus of the IAC frequently developed a stepwise decrease of CNAP amplitude and latency elongation.[1]

We believe that the most critical point for preservation of hearing and facial function is sharp dissection performed using microscissors and microknives, and therefore we very frequently use microscissors during tumor debulking as well as tumor dissection. The frequent usage of microscissors is practiced without difficulty under the direct surgical field by using the microscope. Although the endoscopic microsurgical instrumentation is being progressively refined, blunt dissection is the predominant surgical technique under the endoscope at present.

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The transbasal approach: historical observation

TO THE EDITOR: I read with great interest and appreciation the article by Effendi et al.[3] (Effendi ST, Rao VY, Momin EN, et al: The 1-piece transbasal approach: operative technique and anatomical study. *J Neurosurg 121:*1446–1452, December 2014). This is an excellent anatomical study and will be useful in helping neurosurgeons to choose the right approach for extradural tumors of the anterior cranial base. I would just like to add that those who first applied this technique as a surgical standard were the French neurosurgeons Dr. Patrick J. Derome and his chairman, Dr. Gerard Guiot. In 1980 I had the privilege of spending my last year of residency in Dr. Guiot's department at the Hôpital Foch, Suresnes, Paris. There, I saw patients coming from around the world to be operated on, in particular, by Dr. Derome, who pioneered this technique along with a careful reconstruction of the bony cranial base, using split frontal bone, cancellous bone taken from the iliac crest, or both. He also used the carefully preserved bifrontal pericranial flap, which was to be sutured to the visible posterior border of the dura, along the sphenoid ridge on the contralateral side, applying stitches to the dura of the tuberculum sellae.[1] The flap covered the reconstructed bony cranial base and was lying over a partially preserved sphenoid ridge and nasal mucosa or a second free pericranial flap taken posteriorly. I think that his chapter on the transbasal approach published in the first edition of Schmidek and Sweet's *Operative Neurosurgical Techniques: Indications, Methods, and Results*[1] should always be quoted, as should another paper on cranial base surgery, "Bone problems in meningiomas invading the base of the skull," which appeared in 1977.[2]

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Response

Thank you, Dr. Pompili, for your response to our article. While the technique was first described by Dandy in 1941, Drs. Derome and Guiot were instrumental in popularizing the modern transbasal approach.[1–3] We thank you for your historical perspective regarding this approach.

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Optic radiations and anterior commissure

TO THE EDITOR: We read with interest the article by Goga and Türe[2] that discussed the anatomy of Meyer's loop (Goga C, Türe U: The anatomy of Meyer's loop revisited: changing the anatomical paradigm of the temporal loop based on evidence from fiber microdissection. *J Neurosurg* 122:1253–1262, June 2015). The authors demonstrated through fiber dissection the anatomy of the optic radiations. They also discussed the anatomy of the anterior commissure, whose posterior extent forms part of the sagittal stratum. It is unfortunate that the authors did not refer to our work on this subject.[4,5]

During our dissection of the limbic system in general and the Papez circuit in particular, we have demonstrated the anatomy of the anterior commissure.[4,5] The anterior commissure resembles a horizontally placed bow and runs anterior to the columns of the fornix. The anterior commissure runs in a canal of gray matter (canal of Gratiolet) parallel and deep to the uncinate fasciculus and occipitofrontal fasciculus. Some fibers of the anterior commissure run anteriorly, while most of the compact bulk of this white fiber tract runs posteriorly. The anterior fibers connect the anterior olfactory nucleus of one side to the contralateral side. The posterior portion of the anterior commissure runs posterolaterally deep to the lentiform nucleus and middle temporal gyrus and ultimately forms a component of the sagittal stratum. The uncinate fasciculus, which in the anterior temporal region is indistinguishable from the inferior occipitofrontal fasciculus, turns anteriorly to end in the anterior portion of the temporal lobe. The occipitofrontal fasciculus courses posterolaterally and forms another component of the sagittal stratum (Fig. 1). Thus, the sagittal stratum is composed of the fibers of the anterior commissure, the occipitofrontal fasciculus, and the optic radiations. Though these 3 fiber systems are difficult to distinguish after they merge with the optic radiations, we have found that the fibers of the anterior commissure and the occipitofrontal fasciculus lie laterally and the optic radiations lie medially. The point at which the 3 fiber systems converge was clearly identified in our fiber dissection and is shown in the figure (Figs. 2 and 3). At the point of intersection the fibers of the occipitofrontal fasciculus lie most laterally. Medial to these fibers lie the fibers of the anterior commissure, and more medially the temporal loop of the optic radiations can be visualized.

The optic radiations or the geniculocalcarine tract commences at the lateral geniculate body and ends in the calcarine cortex on the medial aspect of the occipital lobe.
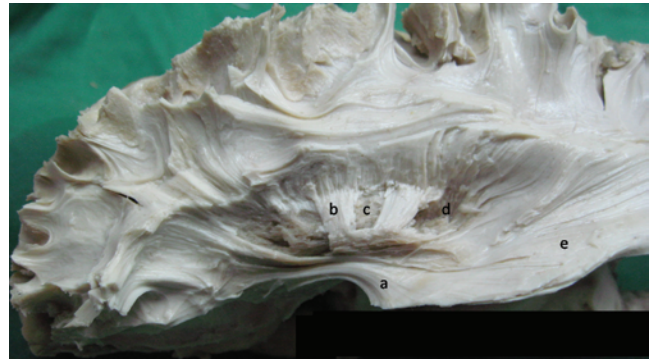

FIG. 1. Lateral surface of the hemisphere showing the region of the insula after removal of the insular gyri and the extreme capsule and claustrum. The superior longitudinal fasciculus has also been dissected away. The fibers of the occipitofrontal fasciculus are seen joining the sagittal stratum. **a:** Inferior occipitofrontal fasciculus. **b:** External capsule. **c:** Lentiform nucleus. **d:** Internal capsule. **e:** Sagittal stratum. Figure is available in color online only.

The optic radiations are composed of 3 bundles: anterior, central, and posterior. Though by the general direction of the fiber bundles 3 main bundles can be distinguished, it is not usually possible to clearly delineate the 3 bundles. The anterior bundle, also known as Meyer's loop, travels anteriorly from the lateral geniculate body, forward to the roof of the temporal horn, and then turns posteriorly in the lateral wall of the temporal horn. It continues posteriorly to terminate at the inferior bank of the calcarine sulcus. The central bundle courses laterally over the roof of the temporal horn and then courses posteriorly in the lateral wall of the trigone and occipital horn. It terminates at the occipital pole. The posterior bundle travels directly posteriorly over the trigone to end in the superior bank of the calcarine sulcus. The anterior bundle runs in the sublenticular portion of the internal capsule and the central and posterior bundles form the retrolenticular portion of the internal capsule (Figs. 2 and 3).

The internal capsule has been traditionally divided into an anterior limb, genu, posterior limb, a retrolenticular portion, and a sublenticular portion (Fig. 3). The anterior limb of the internal capsule is made up of the frontopontine fibers and the anterior thalamic radiations. The genu of the internal capsule is composed of the corticobulbar fibers and the superior thalamic peduncle. The posterior limb of the internal capsule is made up of the corticospinal fibers, the corticopontine fibers, the corticotegmental fibers, and the superior thalamic peduncle. The retrolenticular portion of the internal capsule comprises the central and posterior portions of the optic radiations and the posterior thalamic peduncle. The sublenticular portion of the internal capsule is made up of Meyer's loop (anterior bundle of the optic radiations) and the auditory radiations.

Goga and Türe with their dissection techniques noted that all the projection fibers of the sublenticular portion of the internal capsule make an initial anterior detour in the temporal region before turning posteriorly to join the sagittal stratum. They describe this whole bundle of fibers as the temporal loop and found it difficult to distinguish the optic radiations separately. It was also difficult to delineate the fine fibers that participate in the temporal loop from
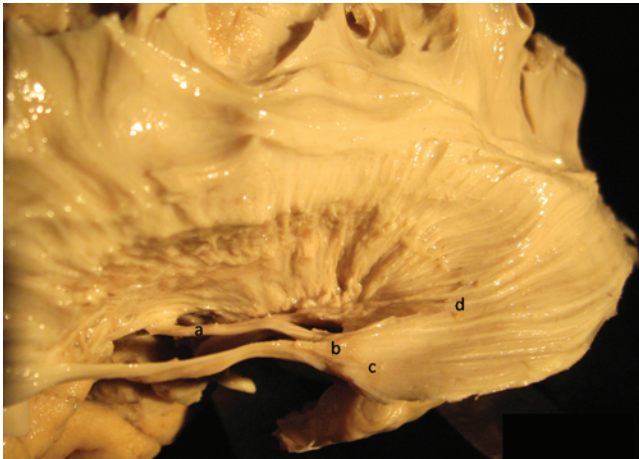
FIG. 2. Deeper dissection in the region. The bulk of fibers of the occipitofrontal fasciculus have been peeled away in the region of the tip of the temporal loop and even further posteriorly in the region of the sagittal stratum. Fibers of the anterior commissure are seen to enter the sagittal stratum. More posteriorly the 3 fiber systems cannot be distinguished. **a:** Anterior commissure. **b:** Tip of temporal loop where the fibers of the inferior occipitofrontal fasciculus, anterior commissure, and optic radiations meet. **c:** Meyer's loop. **d:** Central and posterior bundles of optic radiations in the retrolenticular portion of the internal capsule. Figure is available in color online only.

the intercrossing fibers of the anterior commissure and the occipitofrontal fasciculus. They were also unable to identify the tip of the temporal loop accurately.

In our dissections, we could identify the point of intersection of the anterior commissural fibers with the sagittal stratum as shown in Fig. 2. It was not necessary to sever the anterior commissure to identify the sublenticular portion of the internal capsule. The tip of the temporal loop or Meyer's loop has also been clearly identified by our dissection techniques. We also found that only a few fibers originating from the lateral geniculate body detoured anteriorly before turning posteriorly. This was identified as Meyer's loop (Fig. 4). We clearly identified these fibers originating from the lateral geniculate body. Some fibers passed directly backward without looping anteriorly. This has been noted in our dissection as well as fiber dissections by various other authors.[1,3] The average total length of the anterior commissure before its amalgamation with
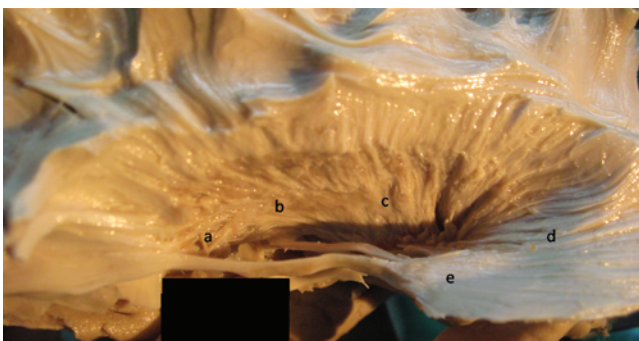

FIG. 3. Dissection showing the various components of the internal capsule. **a:** Anterior limb. **b:** Genu. **c:** Posterior limb. **d:** Retrolenticular portion. **e:** Sublenticular portion. Figure is available in color online only.
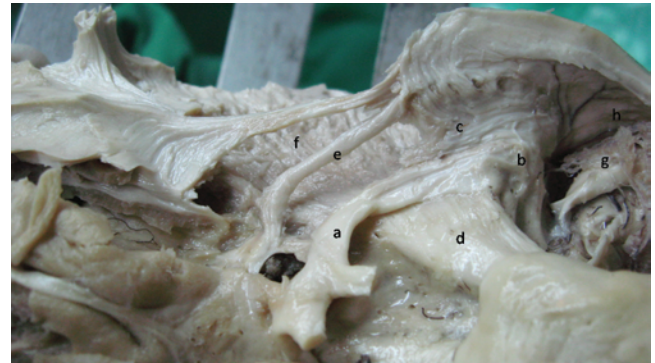

FIG. 4. Inferior view of the cerebral hemisphere. The inferior surface of the frontal and temporal lobes has been dissected. The hippocampus and the choroid plexus in the temporal horn have been reflected inferiorly. The fibers constituting Meyer's loop are seen to be arising from the lateral geniculate body, looping anteriorly and then posteriorly. **a:** Optic tract. **b:** Lateral geniculate body. **c:** Meyer's loop. **d:** Cerebral peduncle. **e:** Anterior commissure. **f:** Internal capsule. **g:** Choroid plexus in temporal horn reflected inferiorly. **h:** Temporal horn. Figure is available in color online only.

the sagittal stratum was 78 mm (range 74–80 mm). The distance of the anterior commissure form the temporal pole was a mean of 18 mm (range 16–20 mm). The tip of the temporal loop was an average of 25 mm (range 22–30 mm) from the temporal pole.

In the anterior portion of the sagittal stratum, fiber components of the inferior occipitofrontal fasciculus and the anterior commissure were recognizable, but further posteriorly it was difficult to delineate the 3 fiber systems, as also shown by the authors. We believe that the temporal loop is composed mainly of the anterior portion of the optic radiations (Meyer's loop) and these fibers can be seen to originate in the lateral geniculate body.

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Response

No response was received from the authors of the original article.

Gamma Knife radiosurgery for vestibular schwannoma

TO THE EDITOR: We carefully read the article by Boari et al.[1] (Boari N, Bailo M, Gagliardi F, et al: Gamma Knife radiosurgery for vestibular schwannoma: clinical results at long-term follow-up in a series of 379 patients. *J Neurosurg* 121 (Suppl 2):123–142, December 2014). The importance and the complexity of vestibular schwannoma (VS) management is indirectly confirmed by the constant appearance in the literature of new articles dealing with that topic. The article mentioned above, in which the results of radiosurgery in VS are described, may represent a very relevant contribution, adding data to the existing literature and confirming the validity of radiosurgery as an option. We appreciated very much the huge efforts of the authors, although some considerations should be mentioned.

First, the tumor growth definitely represents the most important end point for comparison of the main treatment options. The authors declare a very high rate of tumor control (97.1%), and in Fig. 3B a Kaplan-Meier curve shows this. Unfortunately, a very important parameter is lacking: the censoring. As is well known, Kaplan-Meier estimators can take into account if a patient withdraws from a study, and this is called censoring, which applies when patients drop out before the final outcome is observed. Censoring is indicated by small vertical tick marks interspersed on the plot. Those lines are present in the Kaplan-Meier curve of Fig. 5, which shows hearing results, but are lacking in the most important one; i.e., the tumor growth curve (i.e., Fig. 3B). Moreover, every Kaplan-Meier curve should report the sample size below the x axis at every time point: this is not a controversial issue, but can have important consequences during summarization of the data.

For example, the authors obtained a 97.1% rate of tumor control in their study group, with a mean follow-up of 68.3 months and for a maximum period of observation of 156 months. But how many patients dropped out? When did they actually drop out? And most importantly, do the authors consider them to have a tumor indefinitely under control? Unfortunately, this discrepancy frequently manifests in follow-up studies, and the reality is that in the majority of cases patients who dropped out are not censored, so they are considered "survivors" or "tumor controlled." This could also have strongly biased and overestimated the

final rate of tumor control in the present study. A possible confirmation that the above-mentioned bias occurred can be indirectly obtained by the shapes of the Kaplan-Meier curves in Fig. 5. Although as has already been underlined, sample modifications are not specified under the x axis, the high steps made by the curves going down and moving toward the right part of the plot are strongly suggestive of a small sample size at the long follow-up, so the 97.1% rate of tumor control reported could be referring to only the few patients who were left, and most were lost to follow-up and not censored.

Second, oddly, whereas other parameters are fairly summarized by mean and median (e.g., time between symptom onset and diagnosis, time from diagnosis to treatment, and so on), the length of follow-up is expressed only by the mean value of 68.3 months (maximum 156 months): the mean is not the most appropriate parameter to describe a follow-up, because it can mask a positive (right tailored) skew of a median distribution. For example, a very few patients followed for decades could strongly influence the mean, but not the median values, so the latter is therefore the most appropriate value to summarize a follow-up.

Third, the *Discussion* subheading *GKRS Versus Wait-and-See Strategy* is not very balanced, because the authors report only results from the literature that support their treatment modality, and conclude that the wait-and-see strategy would not be indicated for VS. Results of the wait-and-see policy presented in the literature are variable, and most are much better than those mentioned by the authors.[2] Anyway, if a single best treatment for every VS will ever exist, confirmation will only come from prospective randomized clinical trials, which are lacking at present.

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2. Patnaik U, Prasad SC, Tutar H, Giannuzzi AL, Russo A, Sanna M: The long-term outcomes of wait-and-scan and the role of radiotherapy in the management of vestibular schwannomas. **Otol Neurotol 36:**638–646, 2015

Response

We hereby provide our response to the letter by Alicandri-Ciufelli et al. about our recent paper on Gamma Knife radiosurgery for VS.

First, the observation that censoring was not applied to our retreatment-free Kaplan-Meier curve (our Fig. 3B) is unfounded. The censoring of patients who dropped out of

the study was considered, and it is reported in the curve as very small vertical tick-marks interspersed on the plot. Their density and small dimension may require some magnifying by attentive readers.

Had we actually done no censoring, we would have applied logistic regression instead of survival analysis, to benefit from some well-known possible advantages of it. Otherwise, considering dropout patients as "survivors" would be a trivial error. With respect to this issue, Alicandri-Ciufelli et al. offer an explanation of the shape of our Kaplan-Meier curve (Fig. 3B) that stems from the alleged occurrence of such a severe bias. Because this bias did not occur, this interpretation of the curve shape is itself biased. Actually, the Kaplan-Meier curve shows the failure timing, and particularly pinpoints that failures were clustered in the first 48 months after Gamma Knife radiosurgery treatment.

The statement that we obtained a 97.1% rate of tumor control with a mean follow-up of 68.3 months and for a maximum period of observation of 156 months does not correspond to any data reported in our manuscript. The value of 97.1% of tumor control was obtained over a series of 379 patients with a mean follow-up of 75.7 months, not 68.3 months. The 68.3 months value was reported for the 219 patients in the subgroup in which complete radiological volumetric MRI follow-up was obtained using the 'Gamma-plan' software.

Second, Dr. Alicandri-Ciufelli et al. stated that we reported the length of our follow-up only as the mean value of 68.3 months. We would like to remark that the reported mean value of the clinical follow-up was 75.7 months; furthermore, in the *Follow-Up* paragraph of the *Methods* section it is possible to disclose that the median values are reported, and they were 69.5 months for the clinical follow-up and 63 months for the quantitative radiological follow-up.

Dr. Alicandri-Ciufelli and colleagues claim that in the *GKRS Versus Wait-and-See Strategy* paragraph of the *Discussion* section, our arguments and the literature citations in favor of a proactive treatment are not very balanced. The very interesting manuscript by Patnaik et al., which is cited in the *References* of the Letter to the Editor, was published "ahead of print" in November 2014, whereas our manuscript was submitted for publication in the *Journal of Neurosurgery* in June 2014; it was therefore impossible for us to consider and cite it in the *Discussion*.

Discussing the very controversial issue of the best strategy to use when dealing with patients harboring VSs, we cited the manuscript published by Stangerup et al. in 2006 (reference 57 in our paper), which we consider a milestone in understanding the natural history of these tumors. Discussing the pros and cons of the "wait-and-see" strategy, we have cited other very interesting manuscripts written by the same group in 2008 and 2010 (references 56 and 58). We have carefully read the aforementioned recently published manuscript by Patnaik et al., and we have observed that these authors report a smaller series than that reported by Stangerup and colleagues, and that the mean follow-up is shorter; furthermore, audiological follow-up is not considered in the study.

It is important to keep in mind that many patients with a stable tumor at a radiological follow-up present with a worsening of hearing; in our opinion the hearing function has to be considered, as much as the tumor growth, a main end point in a study investigating the natural history of VSs. Nevertheless, we agree with the authors that other studies on this topic are needed in large series of patients with longer follow-up; prospective randomized clinical trials are advisable but they are extremely hard to run. Probably the best management strategy can vary in different subgroups of patients; according to the results reported in our manuscript, young patients in Gardner-Robertson Class I represent a subset of patients in whom a proactive radiosurgical treatment should be strongly recommended because they seem to have a higher probability of retaining functional hearing at long-term follow-up.

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