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Impact of the Mean Cochlear Biologically Effective Dose on Hearing Preservation After Stereotactic Radiosurgery for Vestibular Schwannoma: A Retrospective Longitudinal Analysis

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BACKGROUND AND OBJECTIVES: Stereotactic radiosurgery (SRS) is a useful alternative for small- to medium-sized vestibular schwannoma. To evaluate whether biologically effective dose ($BED_{Gy2.47}$), calculated for mean ($BED_{Gy2.47}$ mean) and maximal ($BED_{Gy2.47}$ max) cochlear dose, is relevant for hearing preservation.

METHODS: This is a retrospective longitudinal single-center study. Were analyzed 213 patients with useful baseline hearing. Risk of hearing decline was assessed for Gardner–Robertson classes and pure tone average (PTA) loss. The mean follow-up period was 39 months (median 36, 6-84).

RESULTS: Hearing decline (Gardner–Robertson class) 3 years after SRS was associated with higher cochlear $BED_{Gy2.47}$ mean (odds ratio [OR] 1.39, $P = .009$). Moreover, $BED_{Gy2.47}$ mean was more relevant as compared with $BED_{Gy2.47}$ max (OR 1.13, $P = .04$). Risk of PTA loss (continuous outcome, follow-up minus baseline) was significantly corelated with $BED_{Gy2.47}$ mean at 24 (beta coefficient 1.55, $P = .002$) and 36 (beta coefficient 2.01, $P = .004$) months after SRS. Risk of PTA loss (>20 dB vs ≤) was associated with higher $BED_{Gy2.47}$ mean at 6 (OR 1.36, $P = .002$), 12 (OR 1.36, $P = .007$), and 36 (OR 1.37, $P = .02$) months. Risk of hearing decline at 36 months for the $BED_{Gy2.47}$ mean of 7–8, 10, and 12 $Gy_{2.47}$ was 28%, 57%, and 85%, respectively.

CONCLUSION: Cochlear $BED_{Gy2.47}$ mean is relevant for hearing decline after SRS and more relevant as compared with $BED_{Gy2.47}$ max. Three years after SRS, this was sustained for all hearing decline evaluation modalities. Our data suggest the $BED_{Gy2.47}$ mean cut-off of ≤8 $Gy_{2.47}$ for better hearing preservation rates.

KEY WORDS: Biologically effective dose, Cochlea, Hearing preservation, Vestibular schwannoma

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Vestibular schwannomas (VSs) are the most common neoplasm of the cerebellopontine angle in adults.[1] They are generally slow-growing, extra-axial benign tumors, with a diagnosis that is usually made in adults with a mean age from 46 to 58 years.[2] During the past 4 decades, with the common accessibility of MRI, the incidence of newly diagnosed VSs has continuously

ABBREVIATIONS: BED, biologically effective dose; **CISS,** constructed interference in steady-state; **GK,** Gamma Knife; **GR,** Gardner–Robertson; **IAM,** internal auditory meatus; **PTA,** pure tone average; **SRS,** stereotactic radiosurgery; **VS,** vestibular schwannoma.

increased to currently around 19 tumors per million per years.[3] Management options include observation, radiosurgery, and microsurgical resection.[4] Large VSs with a symptomatic mass effect benefit from microsurgical resection.[5-7] One of the key factors in deciding the appropriate therapeutic option is hearing preservation.[8,9] Recent data for small-volume VSs suggested, based on both volume and hearing preservation, that when resection is not considered, early stereotactic radiosurgery (SRS) in contrast to observation results in improved long-term control and hearing preservation rates.

Stereotactic radiosurgery has become increasingly used for small- to medium-sized VSs.[8,10-13] Key factors for hearing preservation after SRS for VSs have been considered the mean dose received by the cochlea,[14-16] the maximal dose received by the ventral cochlear nucleus,[17] or, more recently, the biologically effective dose (BED) received by the tumor.[8,18]

It is thus considered that cochlear dose might be one of the many variables associated with hearing preservation after SRS for VSs.[19] A key question is whether the BED received by the cochlea would be more relevant as compared with the physical dose, considering that BED incorporates not only the factor time but also the delivered dose. Here, we aimed at studying whether the BED to the cochlea is more pertinent regarding hearing deterioration as compared with the mean and maximal dose delivered to the same structure.

METHODS

Study Design

This is a retrospective, single-center, longitudinal study. The Ethical Committee was requested by our Ear, Nose, and Throat group (2020-01989) as part of a larger VS clinical research analysis. Patients provided written informed consent for the procedure.

Patient Population

We followed and included 213 consecutive patients with pre-radiosurgery serviceable hearing (Gardner–Robertson [GR] class I and II[20]). They were treated in the Gamma Knife Center, Lausanne University Hospital, Switzerland, between June 2010 (opening of our radiosurgery activity) and December 2019. Exclusion criteria were as follows: inability to give written informed consent, previous irradiation, patients previously operated, or the presence of type II neurofibromatosis or intracochlear tumors.

Pre- and Postradiosurgery Evaluation

All cases benefited from a standard clinical (including audiological) and neuroimaging assessment. Hearing was evaluated using the GR class[20] (both using discrimination score— speech discrimination score—and pure tone average, PTA). Serviceable hearing included patients with speech discrimination score higher than 50% and PTA less than 30 dB. Facial nerve function was evaluated using the House and Brackmann[21] grading. For tumor classification, we considered the Koos grading.

Postradiosurgery evaluation was performed at 6, 12, 24, 36, 60, and 84 months using the same outcome measurements.

Follow-up Period

The mean follow-up period was 39 months (median 36 months, range 6-84). A minimum follow-up of 2, 3. and 5 years was available for 165, 127, and 82 patients. The basic demographic data are presented in Table 1. The mean age was 52.2 years (median 54, 21.7-86.1). The baseline hearing level corresponded to GR class I in 154 (71.6%) and II in 59 (28.4%) patients.

Radiosurgical Technique

In our center, the used radio-neurosurgery technique is the Leksell Gamma Knife Perfection (June 2010-June 2016, Elekta Instruments, AB) and ICON (from June 2016, onward). We always apply the Leksell stereotactic G frame, followed by a 3-dimensional (3D) stereotactic

TABLE 1. Basic Demographic Data

Variable	n, % or mean, SD (range)
Left:right	109 (50.7%):106 (49.3%)
Age	Mean 52.2, median 54 (21.7-86.1)
Sex	
Male:female	104 (48.4%):111 (51.6%)
Symptom at discovery	
Hearing loss	124 (57.7%)
Vertigo	42 (19.5%)
Tinnitus	25 (11.6%)
Incidental	24 (11.2%)
Koos grade at baseline	
I	65 (30.2%)
II	75 (34.9%)
III	72 (33.5%)
IV	3 (1.4%)
Baseline hearing (GR class)	
1	154 (71.6%)
2	59 (28.4%)
PTA	
Baseline	32.9 ± 14.4 (3.7-66.2)
6 mo after GK	38.7 ± 18.7 (2.5-110)
12 mo after GK	42.2 ± 18.6 (2.5-130)
36 mo after GK	44.4 ± 19.2 (2.5-97.5)
60 mo after GK	47.1 ± 18.5 (5-95)

GK, Gamma Knife; GR, Gardner–Robertson; PTA, pure tone average.
Adapted from the study by Tuleasca et al,[18] by permission from the Congress of Neurological Surgeons.

volumetric acquisition, including computed tomography and MRI (T1- and T2-weighted constructed interference in steady-state (CISS)/fast imaging using steady-state acquisition (Fiesta) sequences, without and with contrast enhancement).

We commonly prescribe a physical dose of 12 Gy, after the dose de-escalation study by Kondziolka et al.[22] In the current series, 208 patients received 12 Gy (97.7%) and 5 patients (2.3%) received 11 Gy.

The mean dose received by the cochlea was 2.9 ± 0.8 (0.6-6.6) Gy. The maximal dose received by the cochlea was 4.2 ±1.4 (1.5-10.4) Gy. Dosimetric data are presented in Table 2.

TABLE 2. Dosimetric Data

Variable	Mean, SD (range)
Target volume (mL)	0.9 ± 1.3 (0.005-7.8)
Prescription isodose volume (mL)	1.1 ± 1.4 (0.015-8.5)
Physical dose (marginal dose, Gy)	12 Gy in 210 (97.7%) cases; 11 Gy in 5 (2.3%) cases
Coverage (%)	98.5 ± 1.3 (93.8-100)
Paddick index	0.74 ± 0.1 (0.28-1.42)
Gradient index (units)	3.1 ± 0.7 (2.2-7.9)
RDR (Gy/min)	2.8 ± 0.6 (1.7-3.8)
No. of isocenters (units)	
Corresponding to the tumor	8.9 ± 7.1 (1-32)
Corresponding to the internal acoustic meatus	Mean 2.5, median 2 (1-9)
Time (min)	
Beam-on time	36.3 ± 18.1 (7.3-101.8)
Treatment time	38.8 ± 18.5 (9-106)
Treatment time minus couch-in and couch-out	37.9 ± 18 (8.9-102.8)
Couch-in and couch-out (together)	0.9 ± 0.7 (0.1-3.2)
Beam-on time corresponding to isocenters in the IAM	16.9 ± 8.9 (2.44-56.2)
Mean dose to the cochlea	2.9 ± 0.8 (0.6-6.6)
BED corresponding to the mean dose to the cochlea ($Gy_{2.47}$)	5.8 ± 2.5 (0.71-21.27)
Maximal dose to the cochlea	4.2 ± 1.4 (1.5-10.4)
BED corresponding to the maximal dose to the cochlea ($Gy_{2.47}$)	10.6 ± 6 (2.2-46.9)
Integral dose	
VS (tumor)	14.7 ± 21 (0.1-116.7)
IAM (all volume)	2 ± 1 (0.6-6.3)

BED, biologically effective dose; IAM, internal auditory meatus; RDR, radiation dose rate; VS, vestibular schwannoma.

Primary Aim

The primary aim was to correlate changes in hearing outcome from serviceable to nonserviceable hearing with BED (both mean and maximal) received by the cochlea.

Hearing deterioration outcomes were assessed as follows:

1. Decline in GR class: I and II (coded 0) vs III, IV, and V (coded 1);
2. Changes in PTA (Δ = follow-up point—baseline) as continuous values;
3. Decline in PTA as binary: Δ ≤ 19 dB (coded 0) vs Δ > 20 dB (coded 1).

Biologically Effective Dose to the Cochlea

The biologically effective dose to the cochlea was calculated for an alpha/beta ratio of 2.47 ($BED_{Gy2.47}$), using an approach initially developed by Fowler[23,24] and further discussed by Barendsen[25] and Hopewell.[26] Couch-in and couch-out, corresponding to complete closure of Cobalt sources, were excluded from total time calculation. We initially considered the beam-on time, from which treatment time was generated using the following formula: [n * t + (n − 1) * 0.1 minutes], with n being the numbers of isocenters and t the isocenter treatment duration.

The mean $BED_{Gy2.47}$ corresponding to the mean dose received by the cochlea was 5.8 ± 2.5 (0.71-21.27) $Gy_{2.47}$. The maximal $BED_{Gy2.47}$ corresponding to the maximal dose received by the cochlea was 10.6 ± 6 (2.2-46.9) $Gy_{2.47}$.

Statistical Analysis

Statistical analysis was performed using Stata 16.1 (StataCorp. 2019, Stata Statistical Software: Release 16: StataCorp LLC). Descriptive statistics were related as proportion/frequency for categorical data and mean, median, and range for continuous variables. The association between the BED (both mean and maximal) and the two binary outcomes, decline in GR class and decline in PTA, was assessed using the logistic regression model. The strength of the association was reported using the odds ratio (OR) and its calculated *P*-value. For the change in PTA (continuous outcome), the association with the BED (both mean and maximal) was assessed using the linear regression model. The strength of the association was reported using the β Coefficient and its calculated *P*-value. All analyses were performed without and with adjustment for baseline hearing level and age.

RESULTS

A detailed overview of the results is presented in Table 3.

Risk of Hearing Decline from GR Classes I and II to III, IV, or V (Binary Outcome)

At 3 years after SRS, the risk of hearing decline as per GR class was associated with higher $BED_{Gy2.47}$ mean received by the cochlea (OR 1.39, $P = .009$; Table 3; Figure 1); after adjustment for baseline GR class and age, the result remained statistically significant (OR 1.52, $P = .006$; Table 3). Moreover, $BED_{Gy2.47}$ mean was more relevant as compared with $BED_{Gy2.47}$ max before (OR 1.13, $P = .04$) and after adjustment (OR 1.19, $P = .01$).

The mean ($P = .91$) and maximal ($P = .66$) doses received by the cochlea were not statistically significant.

Risk of Hearing Decline Regarding Increase in PTA (Continuous Outcome, Follow-up Minus Baseline)

The risk of PTA loss (continuous outcome, follow-up minus baseline) was significantly corelated with $BED_{Gy2.47}$ mean at 24 (beta coefficient 1.55, $P = .002$) and 36 (beta coefficient 2.01, $P = .004$) months after SRS; after adjustment for age and baseline PTA, this result was even more relevant at both time points, 24 (beta coefficient 1.61, $P = .001$) and 36 (beta coefficient 2.34, $P = .001$) months (Table 3, Figure 2).

The mean ($P = .27$) and maximal ($P = .87$) doses received by the cochlea were not statistically significant. Moreover, the intracanalicular volume was not statistically significant for hearing deterioration at 6 ($P = .87$), 12 ($P = .72$), 24 ($P = .4$), 36 ($P = .18$), and 60 months ($P = .58$).

Risk of Hearing Decline Regarding Changes in PTA [Continuous Outcome, as Binary Δ ≤ 19 dB (Coded 0) vs Δ > 20 dB (Coded 1)]

The risk of PTA loss [as binary Δ ≤ 19 dB (coded 0) vs Δ > 20 dB (coded 1)] was associated with higher $BED_{Gy2.47}$ mean at 6 (OR 1.36, $P = .002$), 12 (OR 1.36, $P = .007$) and 36 (OR 1.37, $P = .02$) months; after adjustment for age and baseline PTA, this result was even more relevant at 6 (OR 1.36, $P = .003$), 12 (OR 1.38, $P = .009$) and 36 (OR 1.49, $P = .007$) months (Table 3, Figure 3).

The mean dose received by the cochlea was statistically significant only at 24 months. Moreover, the intracanalicular volume was not statistically significant for hearing deterioration at 6 ($P = .92$), 12 ($P = .52$), 24 ($P = .31$), 36 ($P = .22$), and 60 months ($P = .28$).

Risk of Hearing Decline at 36 Months after SRS

Risk of hearing decline at 36 months for the $BED_{Gy2.47}$ mean of 7–8, 10, and 12 $Gy_{2.47}$ was 28%, 57%, and 85%, respectively (Figure 4).

The risk of hearing decline at 36 months was for the $BED_{Gy2.47}$ max of 8, 9, 10, 12, 14, and 15 $Gy_{2.47}$ was 21.3%, 26.6%, 30%, 38.6%, 45.1%, and 46.8%, respectively.

DISCUSSION

In this study, we evaluated whether biologically effective dose ($BED_{Gy2.47}$), calculated for mean ($BED_{Gy2.47}$ mean) and maximal ($BED_{Gy2.47}$ max) cochlear dose and for an alpha/beta ratio of 2.47, is relevant for hearing preservation after SRS for VSs. Our data suggest that cochlear $BED_{Gy2.47}$ mean is relevant for hearing decline after SRS and more relevant as compared with cochlear $BED_{Gy2.47}$ max. At 3 years after SRS, such a result was sustained for all hearing decline evaluation modalities, whether we evaluated a change in hearing GR class or a PTA loss (both continuous values and cutoff of 20 dB). Risk of hearing decline at 36 months for the $BED_{Gy2.47}$ mean of 7–8, 10, and 12 $Gy_{2.47}$ was 28%, 57%, and 85%, respectively. Based on our present findings, we propose a cutoff for $BED_{Gy2.47}$ mean received by the cochlea of equal to or less than 8 $Gy_{2.47}$ for better hearing preservation rates.

One of the key factors for hearing preservation after SRS for VSs has been classically considered the mean dose received by the cochlea.[14-16] However, it might be more relevant to evaluate the BED received by this structure because it considers both the time

TABLE 3. Summary of the Main Results of the Statistical Analysis

Time (mo)		GR class 0: I + II vs 1: III + IV + V OR (*P*-value)		PTA loss (follow-up point minus baseline) B Coefficient (*P*-value)		PTA loss 0: ≤20 units vs v1: >20 units OR (*P*-value)	
6	BED mean	1.01 (0.88)	1.01 (0.92)[b]	0.83 (0.04)	0.75 (0.07)[a]	1.36 (0.002)	1.36 (0.003)[a]
	BED max	0.99 (0.72)	0.99 (0.79)[b]	0.32 (0.06)	0.28 (0.11)[a]	1.10 (0.006)	1.10 (0.009)[a]
12	BED mean	1.11 (0.20)	1.11 (0.23)[b]	0.77 (0.09)	0.75 (0.10)[a]	1.36 (0.007)	1.38 (0.009)[a]
	BED max	1.02 (0.50)	1.03 (0.39)[b]	0.30 (0.12)	0.28 (0.15)[a]	1.11 (0.007)	1.10 (0.02)[a]
24	BED mean	1.11 (0.21)	1.14 (0.14)[b]	1.55 (0.002)	1.61 (0.001)[a]	1.19 (0.07)	1.22 (0.06)[a]
	BED max	1.02 (0.64)	1.02 (0.64)[b]	0.48 (0.03)	0.49 (0.03)[a]	1.07 (0.07)	1.07 (0.09)[a]
36	BED mean	1.39 (0.009)	1.52 (0.006)[b]	2.01 (0.004)	2.34 (0.001)[a]	1.37 (0.02)	1.49 (0.007)[a]
	BED max	1.13 (0.04)	1.19 (0.01)[b]	0.41 (0.23)	0.53 (0.12)[a]	1.12 (0.06)	1.14 (0.03)[a]
60	BED mean	0.98 (0.88)	0.95 (076)[b]	1.57 (0.11)	1.76 (0.08)[a]	1.27 (0.11)	1.29 (0.12)[a]
	BED max	1.01 (0.91)	1.00 (0.96)[b]	0.23 (0.61)	0.20 (0.66)[a]	1.09 (0.17)	1.08 (0.26)[a]

BED, biologically effective dose; GR, Gardner–Robertson; OR, odds ratio; PTA, pure tone average.
[a]Adjusted for the PTA baseline and age.
[b]Adjusted for the audition baseline (GR class) and age.

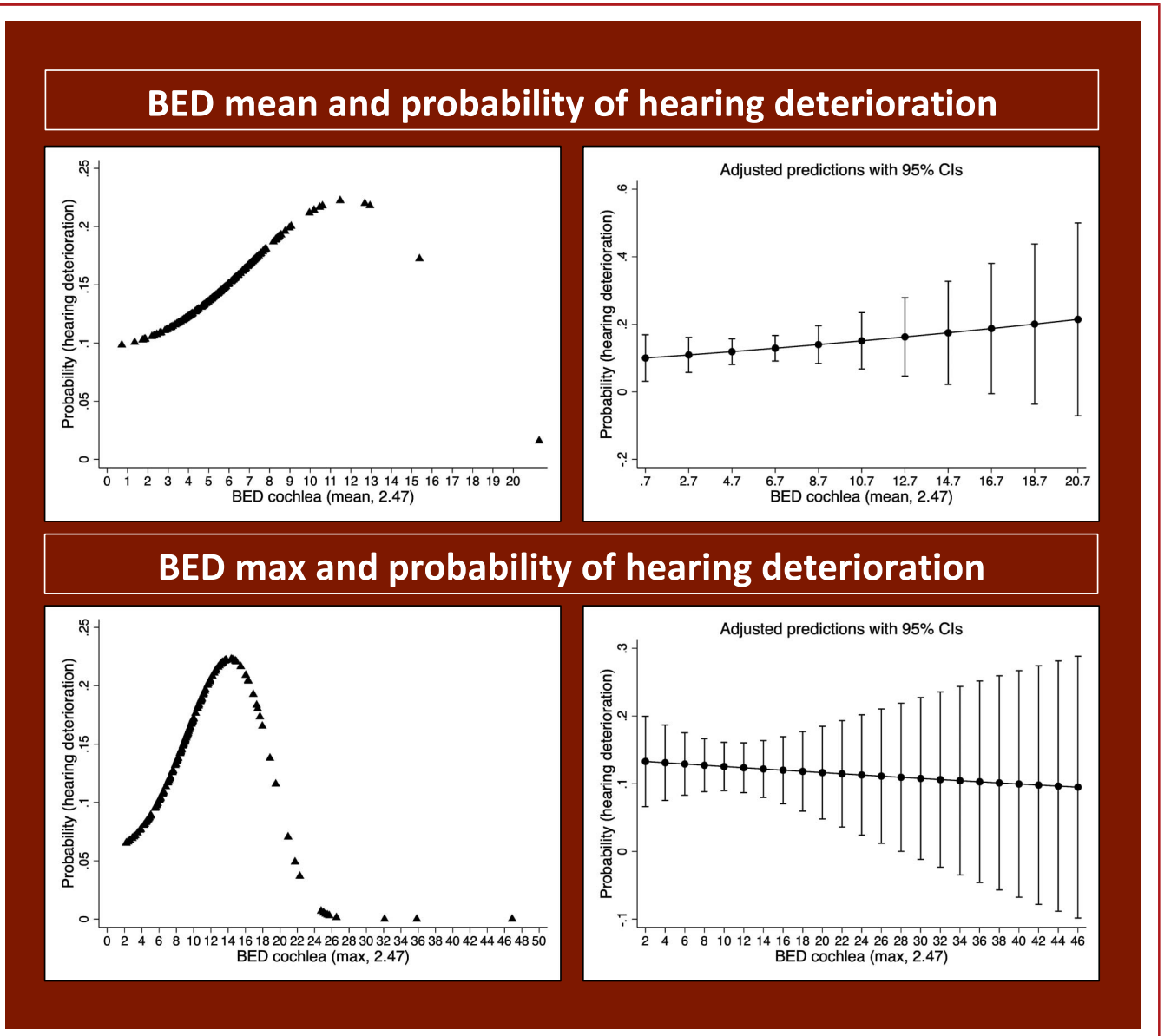

FIGURE 1. *Correlation between the $BED_{Gy2.47}$ (mean, upper part, and maximal, lower part) received by the cochlea and the probability of hearing decline (by Gardner–Robertson class); left, fractional polynomial, right, regression analysis. BED, biologically effective dose.*

factor and the delivered physical dose, as previously suggested in the recent literrature.[8,24,27-34]

The mechanisms of radiation-induced hearing loss are complex and poorly understood. After radiotherapy, such mechanisms include direct or indirect damage, with potential creation of free radicals that might damage double-stranded DNA, inflammatory cell recruitment, or activation of multiple signaling pathways.[35] Moreover, immediate deterioration is rare and considered as produced by neural edema, demyelination, or inflammation at the lesion's site.[35] After SRS, suggested mechanisms are loss of microvessels, thrombosis of the internal auditory artery, or direct and immune-mediated injury to the vestibulocochlear nerve or cochlea hair cells.[36] Indeed, components of the cochlea are sensitive to radiation and might be damaged after radiation-based treatments.[37] Previous studies support avoiding a cochlear dose of 3–5 Gy during SRS.[19,38,39] A recent systematic review proposed a mean cochlear dose of less than 4–6 Gy, without compromising tumor dose.[40] Brown et al[41] suggested that the cochlea volume irradiated above a certain level, rather than a specific point dose, predicted hearing loss. Moreover, hearing

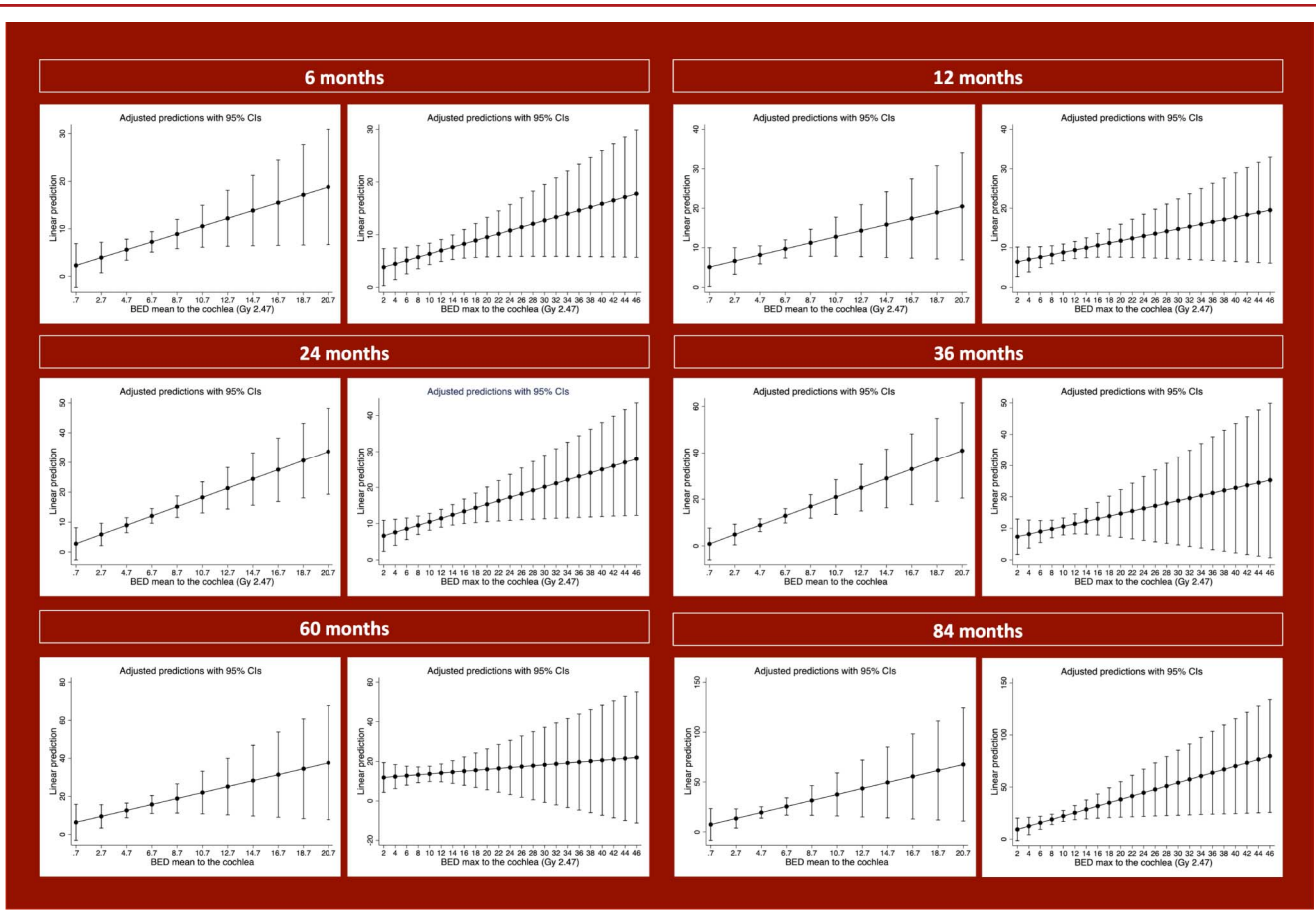

FIGURE 2. *Correlation between the $BED_{Gy2.47}$ (mean, left part, and maximal, right part, at each time point) received by the cochlea and the pure tone average loss (follow-up minus baseline). BED, biologically effective dose.*

pathways were suggested as pertinent, including structures such as the cochlear nucleus, which is suggested to receive less than 10 Gy to limit toxicity.[42] In this study, the mean doses received by the cochlea were rather low (2.9 Gy for the mean dose and 4.2 for the maximal dose, respectively). Since the beginning of our radio-neurosurgery program, we have been actively performing cochlear sparing during dosimetry planning, including using beam channel blocking (whenever necessary). We have thus tried, whenever feasible, to keep the dose delivered to the cochlea below the ranges reported as at risk for hearing loss after SRS in the literature. We thus hypothesize that our findings regarding the absence of statistical significance between the present cochlear doses (both mean and maximal) and the risk of hearing deterioration might be, in part, related to such a treatment policy.

Hypothetical pathological results also comprise tumor cell ischemia and hypoxemic cell death with subsequent cell loss.[42] The clinical consequence is the observed loss of central tumor contrast enhancement and delayed tumor shrinkage once ischemic cell predominates over swelling. Such observations are relevant 3–24 months after SRS, which might account for the specific time window of initial hearing deterioration. In addition to previous, the well-acknowledged transient tumor expansion might further engender cochlear nerve compression and further imply hearing decline.[43,44]

Protein sensitivity has been recently evaluated in 3-D CISS by Prabhu,[45] who suggested that the cochlear signal before treatment was not associated with the pretreatment hearing level. The authors determined that pre-SRS hearing loss is probably multifactorial and affected by other independent parameters of 3D-CISS signal or labyrinthine protein concentration, including neuronal or vascular compression or age-related hearing deterioration. Moreover, it has been suggested that there is a predictive value of the cochlear hypointense signal for hearing outcomes after surgery or SRS.

Dosimetric parameters involved in hearing toxicity, other than the physical dose received by the cochlea, include higher integral dose received by the intracanalicular part of the VSs treated by SRS,[15] higher radiation dose rate,[46] or, more recently, the BED received by the tumor.[8] In this respect, the cochlear physical dose and

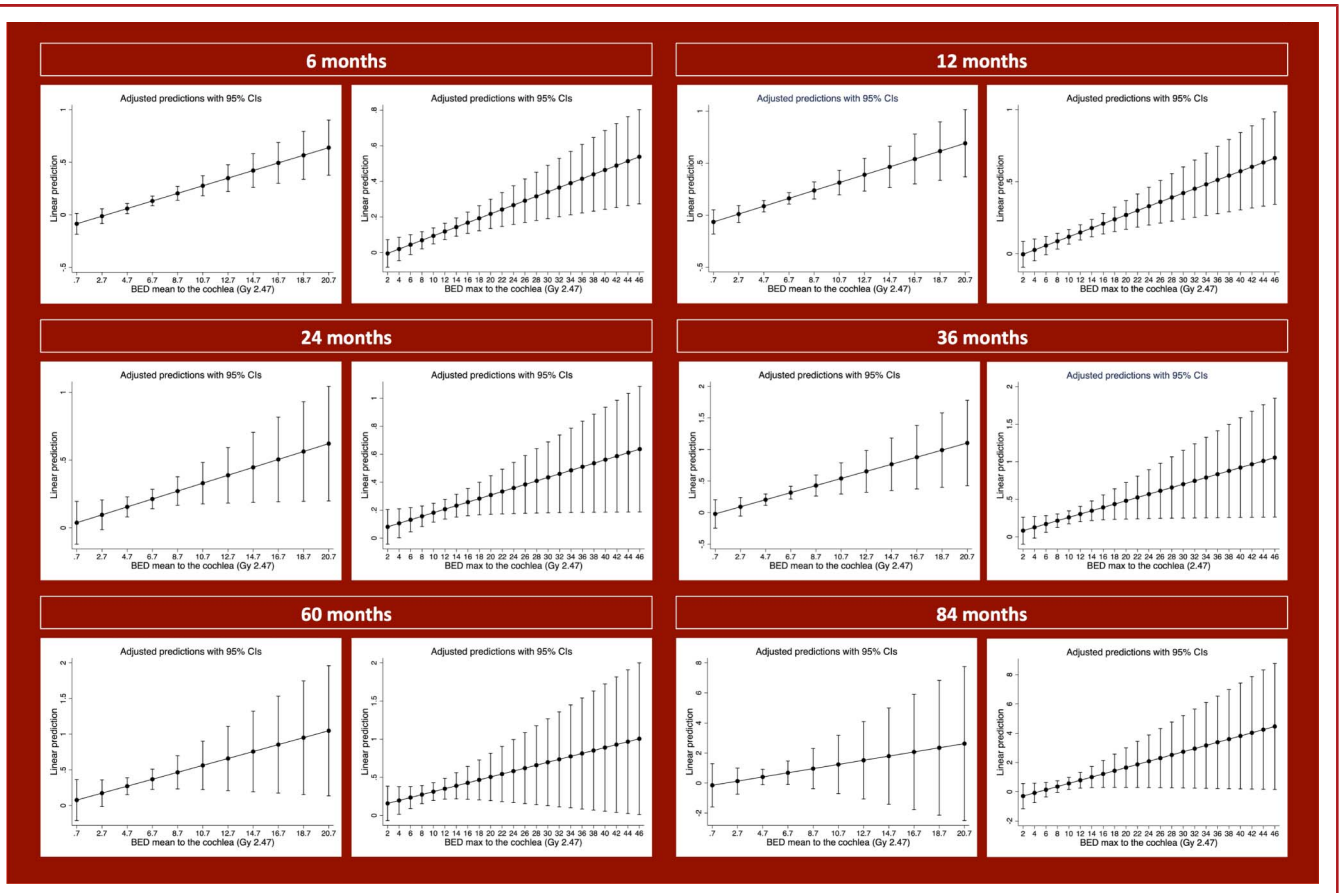

FIGURE 3. *Correlation between the $BED_{Gy2.47}$ (mean, left part, and maximal, right part, at each time point) received by the cochlea and the pure tone average loss (follow-up minus baseline, by loss of >20 dB vs less than or equal to 20). BED, biologically effective dose.*

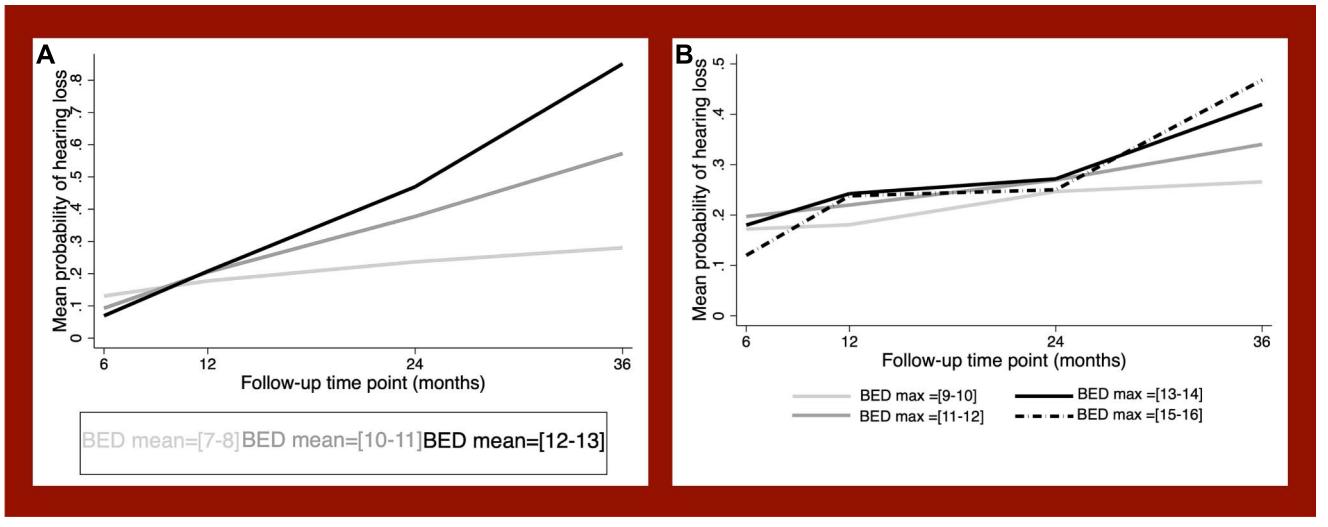

FIGURE 4. *Correlation between the $BED_{Gy2.47}$ (**A**, mean and **B**, maximal) and the overall probability of hearing loss. BED, biologically effective dose.*

corresponding BED are one of the multiple parameters predicting the outcomes.[45,47] We do not standardly prescribe corticosteroid treatment after SRS. In the case of acute or subacute hearing loss, we do prescribe a unique 1-week corticosteroid course, as previously described in the literature, which yields, in our experience, positive results with subsequent hearing recovery.[48,49]

A key question is whether the BED to the cochlea is an independent predictor of hearing preservation after SRS for VS. The results of this study suggest a key role of BED in hearing preservation after SRS for VSs. In the literature, one of the limiting factors in making a step forward is the small volume of the cochlea, thus precluding a straightforward dose-volume histogram analysis.

Limitations

Our study has several inherent limitations. The first is its retrospective nature, with all bias that such implies. The second is directly related to the BED formulae. Here, we used a biexponential fit; however, multiple approaches exist, assuming constant or nonconstant dose rate, bi- or monoexponential formula, and the other parameters, which might vary. The third is related to the limited number of patients. Indeed, such findings should be further validated in larger cohorts.

CONCLUSION

The $BED_{Gy2.47}$ mean delivered to the cochlea was statistically significant for predicting hearing loss after SRS for VSs, particularly at 3 years after SRS and independent of the evaluation modality (change in GR class or PTA). The risk of hearing decline at 36 months for the $BED_{Gy2.47}$ mean of 7, 10, and 12 $Gy_{2.47}$ was 28%, 57%, and 85%, respectively. Thus, our data suggest a cutoff for the $BED_{Gy2.47}$ mean of less than or equal to 8 $Gy_{2.47}$ for better hearing preservation rates.

Future prospective studies are crucial to further inform about BED received by the cochlea and the tumor and their further impact on hearing loss.

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COMMENTS

During stereotactic radiosurgery (SRS), there is variability in treatment gaps and overall treatment time even for tumors of a constant location like vestibular schwannoma. This affects the kinetics of repair of sublethal damage even with the same total dose; hence, the radiosurgery dose rate and the biologically effective dose (BED) calculation is very relevant. BED is not a new concept, and its role in the local control of various benign pathologies has been evaluated in previous studies. Earlier studies have shown that in vestibular schwannomas, BED received by the tumor correlates with hearing decline.[1a,2a] This study evaluates the relevance of BED to the cochlea in hearing preservation.

The authors have retrospectively analyzed their single-institution data on hearing preservation after SRS for vestibular schwannoma. The research question was whether the BED calculated for mean and maximal cochlear dose is an independent predictor of cochlear preservation over and above the total cochlear dose. 213 patients who received SRS as primary treatment and had a preradiosurgery serviceable hearing with a mean follow-up of 39 months were included in the study. Hearing outcomes were assessed as a decline in Gardner–Robertson (GR) class and pure tone average (PTA) loss. The cochlear BED mean correlated well with hearing outcomes; mean and maximal cochlear dose were not very relevant when cochlear sparing during dosimetry planning is performed. $BED_{Gy2.47}$ mean to the cochlea was more relevant than $BED_{Gy2.47}$ max in determining hearing outcomes. The authors suggest a cutoff for the cochlear $BED_{Gy2.47}$ mean of less than 8 Gy. Interestingly, intracanalicular tumor volume did' not correlate significantly with hearing decline post-SRS. Larger well-designed prospective studies are needed to validate these results.

Management options for small vestibular schwannomas with serviceable hearing include observation, microsurgical resection, and SRS. Hearing preservation should be a management priority for these patients. While SRS techniques are being developed to improve hearing preservation rates, the option of microsurgical resection should be definitely considered for all these tumors because it offers the best chances of tumor cure and long-term hearing preservation in safe hands.[3a-6a]

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