ORIGINAL ARTICLE

Hearing Loss and Changes in Transient Evoked Otoacoustic Emissions After Gamma Knife Radiosurgery for Acoustic Neurinomas

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Objective: To evaluate the neuro-otological effects of gamma knife radiosurgery in patients with acoustic neurinoma.

Design: Prospective study.

Setting: University hospital in Milan, Italy.

Patients: Thirty consecutive patients with acoustic neurinoma who underwent gamma knife radiosurgery.

Intervention: Gamma knife radiosurgery.

Main Outcome Measures: Results of neuro-otological tests, including pure-tone audiometry, auditory brainstem responses, and transient evoked otoacoustic emissions, during a 2-year follow-up.

Results: Three patients showed slight tumor growth, 1 complained of a transient facial disturbance, and 5 complained of mild trigeminal disturbances. Seven of the 26 patients with a measurable threshold before radiosurgery experienced a 2-year decrease of more than 20 dB in at least 1 hearing level, and 2 of these became deaf in the affected ear. The analysis of auditory brainstem responses showed no significant increase in mean wave V latency after radiosurgery, but intensity of transient evoked otoacoustic emissions worsened in 9 of the 12 patients who had them before treatment. A statistically significant correlation was found between the 2-year decrease in low-tone average, pure-tone average, and high-tone average hearing levels and the 2-year decrease in transient evoked oacoustic emissions ($P<.001$, $P=.008$, and $P<.001$, respectively), and between the 2-year decrease in high-tone average hearing and the maximal cochlear dose ($P=.03$).

Conclusions: Although most patients had only a slight fluctuation of their hearing threshold after gamma knife radiosurgery, several experienced a remarkable hearing worsening. Hearing impairment was found to be mainly due to cochlear irradiation and maximal cochlear dose, which was correlated to hearing loss.

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DURING THE PAST 30 years, gamma knife stereotactic radiosurgery has become a valid alternative to the surgical treatment of acoustic neurinomas.[1-10] In the case of small tumors, it provides a good local control rate and a low incidence of complications that are comparable to or better than those observed in microsurgical series.[11-13] However, few studies are available concerning the neuro-otological effects of gamma knife radiosurgery in the treatment of acoustic neurinomas,[14-18] and to our knowledge, no previous study has simultaneously investigated the effects of radiosurgery on hearing threshold and changes in the neurophysiological measures of nervous transmission and cochlear function.

Traditional neurophysiological investigations using auditory brainstem responses (ABRs) make it possible to study nervous transmission through the acoustic nerve and the auditory pathway, but the the relatively recent clinical use of transient evoked otoacoustic emissions (TEOAEs) has provided an objective and noninvasive means of investigating cochlear function. It is believed that TEOAEs are generated by the electromotor activity of the outer hairy cells of the organ of Corti and that they are lost in the case of cochlear hearing loss.[19]

Previous studies have shown that TEOAEs are lost on the lesioned side in a variable number of patients with acoustic neurinoma, probably because of cochlear vascular damage.[20,21] Ferbert-Viart et al[22] found that the presence of TEOAEs on the lesioned side in 63 patients with acoustic neurinoma who were surgically treated with the aim of saving hearing function was a favorable prognostic factor for hearing

preservation, probably as a result of better cochlear vascularization.

The aim of this study was to evaluate changes in hearing thresholds and in ABR and TEOAE neurophysiological findings in a group of patients with acoustic neurinoma who underwent gamma knife radiosurgery. We also investigated the possible links between the audiological and neurophysiological changes and between the audiological findings and the main radiological and radiosurgical variables.

METHODS

Thirty consecutive patients with unilateral acoustic neurinoma (13 men and 17 women; mean±SD age, 54.6±13.3 years) underwent gamma knife stereotactic radiosurgery at the Department of Neurosurgery of the University of Milan, Milan, Italy, and were followed up by means of audiological and neurophysiological examinations for 2 years. Magnetic resonance imaging was used in all cases. At the time of radiosurgery, the tumor volumes ranged from 0.2 to 4.9 cm^3 (mean±SD, 1.6±1.6 cm^3).

All patients underwent a complete neuro-otological evaluation on the day before radiosurgery, including an otolaryngological examination, trigeminal and facial nerve function testing (using the House-Brackman grading system[23]), pure-tone audiometry, ABR examination, and TEOAE recordings.

Pure-tone audiometry was performed using an Amplaid 309 system (Amplifon, Milan) in a sound-treated room. The thresholds for each frequency from 250 to 8000 Hz were detected, and then the low- (LTA), pure- (PTA), and high-tone averages (HTA) were calculated.

The ABRs were recorded by an MK 15 Amplaid system (Amplifon) in a silent room while the subjects rested with their eyes closed, with the scalp electrodes placed to keep the impedance below 5000 Ω. A TDH 39 earphone (Telephonics Corp, Farmingdale, NY) delivered the acoustic stimuli, which consisted of 0.1 millisecond of unfiltered alternate polarity clicks at 120 dB of sound pressure level (SPL); the interstimulus interval was 75 milliseconds. Two thousand sequences were averaged for each trial, and at least 2 trials were performed on each side to obtain reproducible recordings. The analysis time was 10 milliseconds, and the digital off-line bandpass filters were set at 50 to 3000 Hz.

The TEOAEs were recorded using an ILO 88 (version 4.2 B) system (Otodynamics Ltd, Hatfield, England) while the subjects rested in a silent room. A probe fitted to the test ear delivered the acoustic stimuli and recorded the responses. The stimuli were nonlinear groups of clicks, ie, 3 positive clicks followed by 1 negative click, whose amplitude was 3 times larger to exclude the components of the response depending on the passive mechanical characteristics of the ear. The stimulus intensity in the ear canal was automatically kept within a range of 75 to 90 dB SPL. Two sets of 256 responses were averaged for each ear. The analysis time was 20.5 milliseconds. The first part of the response (2.5 milliseconds) was removed to avoid the artifacts related to click frequency and intensity, and the responses were digitally filtered to reduce noise from the environment and the passive structures within the ear. The band pass filters were set at 600 to 6000 Hz. The intensity of the responses below 600 Hz and above 6000 Hz was measured in decibels of SPL, and the reproducibility of the 2 sets of sequences for each ear was considered to distinguish further TEOAEs and noise.

A 201-source cobalt 60 gamma unit was used for the radiosurgical treatment at peripheral tumor doses of 1200 to 1400 rad (mean±SD, 1340±80 rad), and maximal tumor doses of 1750 to 2800 rad (mean±SD, 2500±260 rad). Cochlear volumes and the volume of the intracanalicular portion of the neurinoma were outlined using Gamma Plan software (Elekta AB, Stockholm, Sweden). The tumor-canalicular volume ratio was calculated with the peripheral and maximal cochlear doses.

All 30 patients underwent follow-up at 6 months and 1 and 2 years after radiosurgery. Follow-up visits consisted of a repeat complete neuro-otological evaluation. The radiological follow-up consisted of magnetic resonance imaging at 6, 12, and 24 months after radiosurgery.

The data are presented as mean±SD, unless otherwise indicated. The paired *t* test or Mann-Whitney statistic were used to compare the findings before and after radiosurgery as appropriate. Pearson correlation coefficients were calculated to evaluate the correlations between the results obtained using the different neuro-otological techniques and between the audiological and radiological or radiosurgical variables.

RESULTS

None of the patients experienced any significant acute complications after the treatment, and all were discharged on day 1. At the radiological follow-up, the tumor had decreased in size in 19 patients (63%) and remained stable in 7 (23%). Four patients (13%) showed slight growth of the tumor and underwent new radiosurgical treatment.

None of the patients experienced a significant exacerbation of facial palsy after the treatment. Only 1 patient complained of a facial spasm 8 months after treatment, which stopped within 1 year. Some trigeminal disturbances, paresthesias, or sensitive defects appeared or worsened in 5 patients, but none of them experienced trigeminal pain or severe trigeminal impairment.

The raw audiological and neuro-otolological findings of all the patients before and after radiosurgery and the main radiosurgical variables are shown in **Table 1**.

Table 2 shows the mean LTA, PTA, and HTA hearing levels at the time of radiosurgery and 6 months and 1 and 2 years later (the 4 patients whose affected ear was already deaf before radiosurgery were excluded from the analysis). At the time of radiosurgery, the mean LTA, PTA, and HTA hearing levels of the other patients were 35.6±13.3, 44.8±19.6, and 62.5±17.8 dB, respectively, and all were significantly different after 6 months ($P<.05$) and 1 and 2 years ($P<.01$). The mean 2-year decreases were 7.6±18.6, 11.2±15.2, and 10.9±12.3 dB, respectively.

Seven (27%) of the 26 patients with a measurable threshold before radiosurgery experienced a 2-year decrease of more than 20 dB for at least 1 hearing level, with mean decreases in LTA, PTA, and HTA hearing levels of 20.2±31.3, 31.9±12.8, and 26.5±11.3 dB, respectively. Two of these patients were considered deaf in the affected ear 1 and 2 years after radiosurgery, with an LTA, PTA, and HTA hearing level of 100.0 dB. The other 19 patients (73%) had hearing fluctuations of less than 20 dB, with mean decreases in LTA, PTA, and HTA hearing levels of 3.6±6.2, 5.2±6.1, and 3.0±4.5 dB, respectively.

All of the patients with a deaf ear before surgery had no measurable ABRs or TEOAEs in the affected ear. All

Table 1. Main Radiosurgical and Neuro-otological Data From the Series*

Patient No./ Age, y	Tumor Volume, cm^3	Tumor Dose, rad		Maximal Cochlear Dose, rad	Time of Radiosurgery				
					Hearing Loss, dB			ABR Wave V Latency, ms	TEOAE Intensity, dB SPL
		Peripheral, rad	Maximal, rad		LTA	PTA	HTA		
1/71	1.4	1400	2800	710	NM	NM	NM	Absent	0.0
2/74	2.3	1300	2600	1170	NM	NM	NM	Absent	0.0
3/61	4.9	1200	2400	1200	NM	NM	NM	Absent	0.0
4/55	0.6	1400	2800	1470	NM	NM	NM	Absent	0.0
5/37	3.3	1200	2400	1010	43.3	81.7	100.0	Absent	3.0
6/30	0.1	1400	1750	1500	83.3	78.3	68.3	8.11	0.0
7/47	3.3	1300	2600	1330	75.0	73.3	61.6	5.97	4.9
8/34	2.3	1300	2600	1320	68.3	65.0	91.6	Absent	0.0
9/66	3.2	1200	2400	640	73.3	65.0	76.6	Absent	0.0
10/67	1.4	1300	2360	530	35.0	63.3	75.0	6.30	0.0
11/53	0.2	1400	2330	1380	56.7	60.0	58.3	Absent	7.0
12/54	3.8	1300	2360	1050	38.3	51.7	70.0	Absent	0.0
13/61	0.9	1300	2600	540	50.0	50.0	61.6	7.20	0.0
14/65	3.4	1200	2400	590	26.6	50.0	70.0	5.86	4.2
15/57	0.5	1400	2800	860	41.6	48.3	78.3	Absent	6.4
16/64	0.6	1400	2800	620	25.0	45.0	66.6	6.34	0.0
17/63	0.8	1400	2330	460	15.0	43.3	68.3	6.14	3.7
18/58	1.0	1400	2150	640	20.0	43.3	65.0	Absent	5.5
19/39	0.5	1400	2800	1400	30.0	43.3	61.6	7.96	11.4
20/36	0.9	1400	1750	790	40.0	41.7	60.0	6.77	0.0
21/48	0.6	1400	2550	670	20.0	36.7	58.3	6.24	0.0
22/52	0.7	1300	2600	1070	20.0	35.0	70.0	7.72	14.4
23/68	0.3	1400	2550	790	25.0	35.0	76.7	Absent	0.0
24/63	6.7	1200	2400	720	20.0	31.7	65.0	6.30	0.0
25/36	1.9	1400	2800	520	21.6	28.3	38.3	5.97	0.0
26/77	1.8	1300	2600	660	15.0	42.0	50.0	7.20	0.0
27/58	2.6	1400	2550	710	51.6	18.3	36.6	6.04	0.0
28/30	0.3	1300	2000	1300	10.0	13.3	16.6	5.86	6.5
29/63	0.3	1400	2800	600	10.0	10.0	36.6	5.86	13.1
30/52	0.5	1400	2800	820	10.0	10.0	43.3	6.58	13.2

*LTA indicates low-tone average; PTA, pure-tone average; HTA, high-tone average; ABR, auditory brainstem response; TEOAE, transient evoked otoacoustic emission; SPL, sound pressure level; and NM, not measurable.

Table 2. Changes in Hearing Levels and ABR Wave V Latency*

	Pretreatment	6 mo After Treatment	1 y After Treatment	2 y After Treatment
Hearing level, dB (n = 26)				
LTA	35.6 ± 21.6	39.9 ± 23.5†	41.4 ± 23.4‡	43.2 ± 26.0‡
PTA	44.8 ± 19.6	50.1 ± 20.8†	52.4 ± 21.3‡	55.6 ± 24.0‡
HTA	62.5 ± 17.8	67.1 ± 19.7†	69.6 ± 17.5‡	73.4 ± 19.5‡
Wave V latency, ms (n = 18)	6.58 ± 0.75	6.54 ± 0.57	6.60 ± 0.64	6.53 ± 0.60

*Data are given as mean ± SD. LTA indicates low-tone average; PTA, pure-tone average; and HTA, high-tone average.
†$P<.05$ vs pretreatment.
‡$P<.01$ vs pretreatment.

of the other patients had some abnormal ABRs before radiosurgery, ie, prolonged wave V or wave III latencies, prolonged wave I through III or I through V intervals, or the absence of a measurable response. The ABRs were measurable in 18 patients, in whom wave V absolute latency was the only variable that was always detectable; the mean wave V latency was 6.58±0.75 milliseconds.

There was a slight 2-year increase in wave V latency in 8 (44%) of the 18 patients with measurable latency, and a slight decrease in the other cases (56%). No statistically significant difference was found in mean wave V latency before and 2 years after radiosurgery (Table 2).

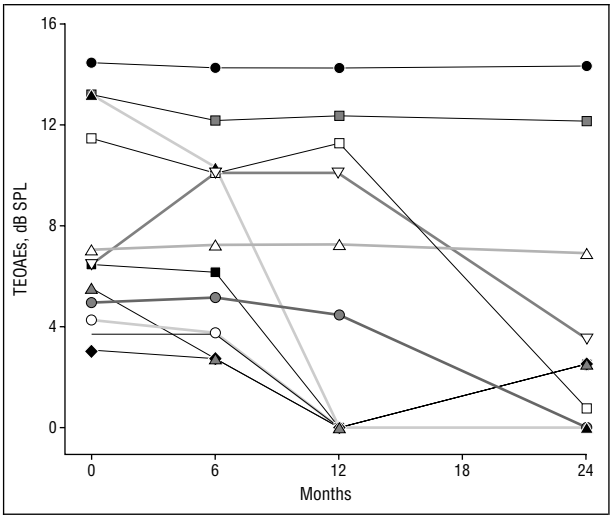
The TEOAE intensity worsened in 9 (75%) of the 12 patients with TEOAEs at the time of radiosurgery and was absent in 7 (58%) (**Figure**).

Four of the 7 patients experiencing a decrease in at least 1 hearing level of more than 20 dB had measurable TEOAEs before radiosurgery that were lost on the affected side in all cases within 2 years. These included the 2 patients who became deaf in the affected ear. On the contrary, among the 7 patients who experienced a de-

6 mo After Radiosurgery					1 y After Radiosurgery					2 y After Radiosurgery				
Hearing Loss, dB			ABR Wave V Latency, ms	TEOAE Intensity, dB SPL	Hearing Loss, dB			ABR Wave V Latency, ms	TEOAE Intensity, dB SPL	Hearing Loss, dB			ABR Wave V Latency, ms	TEOAE Intensity, dB SPL
LTA	PTA	HTA			LTA	PTA	HTA			LTA	PTA	HTA		
NM	NM	NM	Absent	0.0	NM	NM	NM	Absent	0.0	NM	NM	NM	Absent	0.0
NM	NM	NM	Absent	0.0	NM	NM	NM	Absent	0.0	NM	NM	NM	Absent	0.0
NM	NM	NM	Absent	0.0	NM	NM	NM	Absent	0.0	NM	NM	NM	Absent	0.0
NM	NM	NM	Absent	0.0	NM	NM	NM	Absent	0.0	NM	NM	NM	Absent	0.0
40.0	80.0	100.0	Absent	2.7	36.6	80.0	100.0	Absent	0.0	58.3	85.0	100.0	Absent	0.0
91.6	86.7	83.3	7.25	0.0	86.6	86.7	100.0	7.53	0.0	81.6	98.3	100.0	7.08	0.0
66.6	70.0	61.6	5.66	5.1	71.6	73.3	68.3	5.55	4.4	100.0	100.0	100.0	5.90	0.0
55.0	60.0	90.0	Absent	0.0	50.0	60.0	93.3	Absent	0.0	66.6	78.3	100.0	Absent	0.0
73.3	66.7	68.3	Absent	0.0	56.6	55.0	65.0	Absent	0.0	58.0	58.3	70.0	Absent	0.0
41.6	68.3	83.3	6.34	0.0	40.0	65.0	74.3	6.62	0.0	38.3	61.7	78.3	6.84	0.0
55.0	60.0	60.0	Absent	7.2	55.0	60.0	55.0	Absent	7.1	50.0	60.0	55.0	Absent	6.9
51.6	58.3	68.3	Absent	0.0	48.3	60.0	68.3	Absent	0.0	51.6	56.7	70.0	Absent	0.0
45.0	46.7	63.3	6.84	0.0	50.0	55.0	70.0	6.90	0.0	45.0	46.7	66.6	6.90	0.0
26.0	50.0	70.0	7.65	3.8	33.3	48.3	80.0	7.80	0.0	35.0	56.7	85.0	6.68	0.0
60.0	60.0	90.0	Absent	6.1	100.0	100.0	100.0	Absent	0.0	100.0	100.0	100.0	Absent	0.0
25.0	51.7	85.0	6.04	0.0	35.0	55.0	80.0	6.50	0.0	45.0	61.6	71.6	6.84	0.0
20.0	45.0	78.3	6.30	3.7	26.6	46.7	73.3	6.34	0.0	18.3	43.3	71.0	6.24	0.0
16.6	40.0	70.0	Absent	2.7	20.0	36.7	60.0	Absent	0.0	20.0	41.7	73.3	Absent	2.5
95.0	96.7	83.3	7.20	10.0	90.0	93.3	86.6	7.50	11.2	85.0	91.6	88.3	7.50	0.8
40.0	42.0	60.0	7.50	0.0	40.0	50.0	65.0	7.50	0.0	40.0	55.0	75.0	7.90	0.0
20.0	35.0	58.3	6.19	0.0	20.0	35.0	55.0	6.10	0.0	21.6	38.3	58.3	6.05	0.0
20.0	30.0	70.0	6.24	14.2	15.0	23.3	60.0	6.20	14.2	15.0	35.0	76.7	6.20	14.3
30.0	35.0	80.0	Absent	0.0	30.0	35.0	80.0	Absent	0.0	30.0	35.0	90.0	Absent	0.0
48.3	58.3	81.6	6.28	0.0	28.3	43.3	65.0	6.50	0.0	41.6	55.0	70.0	6.50	0.0
30.0	35.0	35.0	6.24	0.0	25.0	45.0	53.3	6.04	0.0	26.6	36.7	41.6	6.00	0.0
15.0	42.0	50.0	7.00	0.0	25.0	50.0	60.0	7.00	0.0	25.0	50.0	60.0	6.70	0.0
32.5	33.3	61.6	6.36	0.0	36.6	36.7	58.3	6.33	0.0	20.0	41.7	73.3	6.50	0.0
10.0	11.7	16.6	5.86	10.0	15.0	20.0	25.0	5.81	10.0	10.0	18.3	28.3	5.60	3.5
15.0	20.0	30.0	6.36	12.1	21.6	23.3	48.3	6.36	12.3	15.0	10.0	36.6	5.76	12.1
15.0	20.0	46.3	6.40	10.3	20.0	26.7	65.0	6.14	0.0	25.0	40.0	68.3	6.44	0.0

crease of more than 20 dB, ABR wave V latency did not worsen in 5 of the 6 with measurable ABRs before radiosurgery. Furthermore, we found a statistically significant correlation between the 2-year decrease in LTA, PTA, and HTA hearing levels and the 2-year decrease in TEOAEs ($P<.001$, $P=.008$, and $P<.001$, respectively), but not between the 2-year decrease in hearing level and the 2-year increase in ABR wave V latency.

The decreases in LTA, PTA, and HTA hearing levels did not significantly correlate with tumor volumes, the tumor-canalicular volume ratio, the peripheral and maximal tumor doses, or the peripheral cochlear dose. We found no statistically significant correlation between the maximal cochlear dose and the 2-year decrease in the LTA or the PTA hearing level; however, a significant correlation was found with the 2-year decrease in the HTA hearing level ($P=.03$). The onset of trigeminal or facial neuropathy did not correlate with hearing loss, tumor volume, or tumor dose of radiation.



Changes in the intensity of transient evoked otoacoustic emissions (TEOAEs) after radiosurgery in patients with measurable TEOAEs before treatment. SPL indicates sound pressure level.

COMMENT

Previous studies have shown that the results of gamma knife radiosurgery in the treatment of acoustic neurinomas are, in terms of hearing preservation, generally comparable to or better than those obtained after microsurgery[11-18] and, when the series were large, a significant correlation was found between hearing loss and tumor

size or treatment dose.[14,15,17] Nevertheless, it is still not clear whether the hearing loss after radiosurgery is secondary to cochlear irradiation or to the exacerbation of the acoustic neuropathy.

The mean decrease in hearing level in our patients was less than 12 dB for all hearing levels. A deterioration of more than 20 dB in any hearing level was observed in 7 (27%) of the 26 patients with measurable hearing thresholds before radiosurgery, and only 2 became deaf in the affected ear after the treatment. These findings are partially in line with those reported in previous neuro-otologic studies. Flickinger et al[15] studied a group of 273 patients radiosurgically treated with a median minimum dose of 1500 rad and reported a 45.4% actuarial rate of auditory deterioraton, and incidences of trigeminal and facial neuropathies of 22.6% and 17.2%, respectively. Ito et al[14,17] reported a deafness rate of 18% and a PTA deterioration of more than 20 dB in 61% of 42 patients undergoing radiosurgery (a mean peripheral dose of 1680 rad) and higher incidences of facial palsy (22%) and trigeminal neuropathy after radiosurgery (30%). The discordance with our results is presumably due to the higher mean tumor dose used in those series. When a lower dose was used, the functional results are more similar to those found in our patients. Hirato et al[16] described a series of 29 patients with acoustic neurinoma treated by means of gamma knife radiosurgery and a low radiation dose (mean peripheral dose, 1210 rad) and, 2 years after treatment, found that 59% of the patients retained their preradiosurgical hearing levels, 4 patients had a PTA deterioration of more than 20 dB (3 of whom became deaf in the affected ear), 1 patient had trigeminal neuropathy, and 3 patients had facial weakness.

To our knowledge, no previous report has used TEOAE analysis to evaluate changes in cochlear function in patients with acoustic neurinoma who undergo gamma knife radiosurgery. We found a significant deterioration in TEOAE response 1 and 2 years after surgery, and the hearing loss significantly correlated with TEOAE but not ABR impairment. All of the patients with measurable TEOAEs before treatment who experienced a postsurgical decrease in hearing level of more than 20 dB lost TEOAEs within 2 years, including the 2 patients who became deaf in the affected ear.

We also found a linear correlation between the decrease in HTA hearing level and the maximal cochlear dose, which suggests that the hearing impairment after radiosurgery is more the result of cochlear irradiation than auditory neuropathy. This finding may explain why, unlike the incidences of trigeminal and facial neuropathies, the incidence of hearing impairment does not tend to be zero when the tumor dose is less than 1300 rad, as previously reported by Flickinger et al.[15] Furthermore, the existence of cochleopathy may justify the findings that the hearing deterioration is almost always irreversible, whereas trigeminal and facial neuropathies recover in a large number of cases.[15] Auditory damage seems to be related to the maximal cochlear dose, which is linked to the particular characteristics of the tumor and the geometry of the treatment plan. This relation should be considered when planning treatment to improve hearing preservation after radiosurgery.

Other studies of larger patient series are needed to establish which radiosurgical variables may influence "radiation cochleopathy" after gamma knife treatment. Our findings suggest that the analysis of TEOAEs could provide useful information and should be included in the postoperative neuro-otological evaluation of patients with acoustic neurinoma who undergo gamma knife radiosurgery.

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