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How Often Does Stapedectomy for Otosclerosis Result in Endolymphatic Hydrops?

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Objectives: 1) To evaluate the long-term (≥10 year) clinical incidence of endolymphatic hydrops (EH) after stapedectomy for otosclerosis, using low-frequency sensorineural hearing loss (LFSNHL) as a marker for EH. 2) To determine the histologic incidence of EH in human temporal bone specimens (TBS) with a history of stapedectomy for otosclerosis. 3) To determine the histologic incidence of EH in a control group of human TBS.
Study Design: Retrospective review and temporal bone study.
Setting: Tertiary medical center and temporal bone pathology laboratory.
Patients: Patients with otosclerosis, human TBS with otosclerosis, and human TBS with presbycusis as the control group.
Intervention: Pure-tone audiometry, temporal bone pathology.
Main Outcome Measures: 1) LFSNHL, defined as >10 decibel elevation of bone conduction thresholds at 250 and 500 Hz, after correcting for age-related hearing loss (per ISO 7029). 2) Histologic assessment of EH.
Results: In patients with otosclerosis, 8 of 110 (7.3%) operated patients versus 3 of 123 (2.4%) nonoperated patients developed LFSNHL ($p = 0.08$). No patients with LFSNHL had other symptoms of EH. In TBS with otosclerosis, 11 of 93 (11.8%) operated TBS versus 3 of 156 (1.9%) nonoperated TBS had evidence of EH ($p \leq 0.001$). In the control group of TBS with presbycusis, 9 of 253 (3.5%) had EH.
Conclusion: The long-term incidence of LFSNHL, a marker for EH, in patients with otosclerosis was not significantly higher in those who underwent stapedectomy. The histologic incidence of EH, however, was significantly higher in TBS that had undergone stapedectomy compared with nonoperated TBS or a control group of TBS.
Key Words: Endolymphatic hydrops—Stapedectomy—Temporal bone pathology.
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An association between otosclerosis and endolymphatic hydrops (EH), based on the study of human temporal bone pathology, was first noted more than 50 years ago (1). The causal relationship between the two disorders has been investigated through clinical and radiologic studies, animal models, and further human otopathology studies (1–7). In human temporal bone studies, EH has been associated with otosclerotic lesions that extend to the cochlear endosteum or that involve the vestibular aqueduct and obstruct the longitudinal flow of endolymph (2,4). In addition, the surgical procedures for otosclerosis may result in immediate or delayed iatrogenic EH (3,8–14). Primary EH is the pathologic correlate of Ménière's disease, in which the cause is unknown. Secondary EH is the pathologic correlate of Ménière's syndrome, which occurs secondary to known causes (e.g., surgical trauma, inflammatory or infectious conditions, or otosclerotic foci). Ménière's disease and syndrome are clinically characterized by symptoms of fluctuating sensorineural hearing loss, episodic vertigo, tinnitus, and aural fullness (15). Although any symptom may precede the others by months or years, the initial symptom is hearing loss in more than 40% of patients (16,17). The hearing loss associated with Ménière's is typically a low-frequency sensorineural hearing loss (LFSNHL), which manifests as an upward–sloping curve on the audiogram. The hearing loss may also involve the high frequencies, such that 250 to 1000 Hz and 3000 to 4000 Hz are predominantly affected and 2000 Hz is less affected (a ''peaked'' curve) (18–20).

Although the development of EH (or Ménière's syndrome) immediately after stapedectomy has been described (3), little is known about the delayed onset of EH over the years after stapedectomy. Therefore, the main goal of this manuscript was to determine whether surgery for otosclerosis increases the incidence of EH over a long period of time. We performed both a retrospective clinical audiologic outcomes study and a study

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of human temporal bone pathology, and compared the results. The first aim of this study was to determine the clinical incidence of secondary EH after surgery or otosclerosis by evaluating hearing thresholds in the low frequencies over a long follow-up period. The second aim was to determine the histologic incidence of EH in temporal bones from patients who had undergone a stapedectomy during life, and compare this to both the histologic incidence in a group of control temporal bones and the clinical incidence.

METHODS

Clinical Study

Subjects

All aspects of this study were reviewed and approved by the Human Studies Committee and the Institutional Review Board (697293-2). All patients who underwent stapedectomy (including both small fenestra stapedotomy and partial and total stapedectomy) at a single tertiary referral hospital between August 1993 and February 2015 were identified. Patients with $\geq$10 years postoperative follow-up were included. Pertinent medical information was extracted from charts and electronic medical records. Patients with a history of additional otologic diagnoses (including chronic otitis media with or without cholesteatoma, congenital or genetically determined hearing loss, sudden deafness, superior semicircular canal dehiscence, vestibular or facial schwannoma), and additional otologic surgical procedures (tympanoplasty, mastoidectomy, cochlear implantation) in the past were excluded. For a control group, nonoperated patients with otosclerosis with $\geq$10 years of follow-up were identified, and the same exclusion criteria were applied.

Audiometry

Audiological assessment was performed using the Harvard Audiometer Operating System software (21) with real-time automated calculation of appropriate masking levels, which were recorded for each ear, except when there were no air bone gaps on either side ($\leq$10 dB) where the unmasked BC threshold can be used for both sides. When the BC values were out at limits, data were conservatively extrapolated to the next audiometric step (+5 dB). The limits of the BC measurements were: 45, 65, 70, 75, and 75 dB at 250, 500, 1000, 2000, and 4000 Hz, respectively. Evaluation of the sensorineural hearing (SNH) was based on the level of BC threshold at the five frequencies (250 Hz, 0.5 kHz, 1 kHz, 2 kHz, and 4 kHz) obtained in the audiograms at three time points: preoperation, postoperation, and at least 10 years after the operation. A low-frequency BC threshold average consisting of the 250 and 500 Hz thresholds was calculated, since low-frequency hearing loss is most commonly associated with EH. The median hearing threshold change in a group of normal hearing individuals (ISO7029), matched for age and sex, was determined for each patient at the initial (preoperative audiogram) and follow-up time point ($\geq$10 yr later) (22). Low-frequency sensorineural hearing loss (LFSNHL) was defined as >10 dB hearing change compared with the age and sex-matched normal hearing thresholds (per ISO7029) at both 250 and 500 Hz, and this definition was used to determine whether each ear fit the criteria for LFSNHL. If there was no available threshold data at 250 Hz, then the criteria were applied using the 500 Hz threshold alone.

Otopathology Study

The histologic incidence of EH was determined in three groups of temporal bone specimens: operated otosclerosis, nonoperated otosclerosis, and presbycusis. The operated and nonoperated otosclerosis groups were comprised of all consecutive temporal bone specimens in the collection at the MEEI Otopathology Laboratory that had been catalogued with a diagnosis of otosclerosis, with or without a history of surgery for otosclerosis. The operated ears had undergone the following types of operations for otosclerosis during life: stapedectomy or stapedotomy, mobilization and lateral canal fenestration surgery. Consecutive temporal bone specimens with a diagnosis of presbycusis, including sensory, neuronal, strial or mixed histologic types, and no histologic evidence of otosclerosis, were used as a control group.

For each temporal bone specimen in each of the three groups (operated, nonoperated, and presbycusis groups), we evaluated the histologic sections by light microscopy for the presence of EH in the vestibule (including the saccular and utricular membrane wall) and in the cochlear duct. The degree of the hydrops in the cochlear duct was described using the grading scale from Cureoglu et al. (23). This scale is based on the position of Reissner's membrane (RM) and includes: 1) slight hydrops (slight bulging of RM), 2) moderate hydrops (less than 90-degree angle of RM with the osseus spiral lamina), and 3) profound hydrops (greater than 90-degree angle of RM with the osseus sprial lamina(. Since mild hydrops in the apical turn alone are frequently observed in temporal bone specimens (24), this was not considered pathologic and not counted as EH. Lastly, for each temporal bone with EH, we evaluated all histologic sections containing the vestibular aqueduct (VA) to determine whether the VA was obstructed and whether the VA was involved with otosclerosis. The prevalence of EH was compared between the three groups. Clinical history, when available, was evaluated and correlated with the otopathology findings.

RESULTS

Clinical Study

There were 110 operated ears with otosclerosis that met the inclusion and exclusion criteria, and 123 nonoperated ears with otosclerosis that were identified for the control group. The operated group was 72% female compared with 60% in the nonoperated group, but this difference was not statistically significant ($p = 0.05$, Fisher's exact test). The patients in the operated group were younger (mean $\pm$ SD of 46 $\pm$ 11 yr (range 23–82) versus 53 $\pm$ 14 yr (range 25–71), unpaired t test, $p < 0.05$), and had a shorter follow-up time compared with the nonoperated group (mean $\pm$ SD of 14 $\pm$ 9 years (range 10–20) versus13 $\pm$ 3 years (range 10–21), unpaired t test, $p < 0.05$).

The BC threshold means at the first visit and last follow-up is presented in Table 1. On average, patients in both the operated and nonoperated groups had normal bone conduction hearing in the low frequencies, and mild hearing loss in the high frequencies at the initial audiogram. At the final audiogram, both groups had mild bone conduction hearing in the low frequencies, and moderate hearing loss in the high frequencies.

TABLE 1. *Mean BC thresholds at initial and final follow-up audiogram*

	250 Hz	0.5 kHz	1 kHz	2 kHz	4 kHz
Operated, initial	10	19	24	36	29
Nonoperated, initial	11	21	23	33	38
Operated, final	17	27	27	40	42
Nonoperated, final	19	31	35	48	42

BC indicates bone conduction; for operated group, N = 110 ears; for nonoperated group, N = 123 ears.

The 250 and 500 Hz bone conduction threshold data were available for 222 ears (99.6%); there was one operated ear for which the 250 Hz threshold was not available. For the one ear with missing data, the determination was based on the 500 Hz threshold alone, and this patient did meet the criteria for significant LFSNHL. For all other ears, the 250 and 500 Hz frequency thresholds were used to determine whether the ear fit the criteria for LFSNHL, as defined in the methods. In the operated group, 8 of 110 (7.3%) ears developed significant LFSNHL, compared with only 3 of 123 patients (2.4%) in the nonoperated group. There was no difference in the incidence of LFSNHL over the ≥10 year follow-up period in the operated group compared with the nonoperated group ($p = 0.08$, Fisher's exact test), although there was a trend toward a higher incidence of LFSNHL in the operated group. The average BC threshold change, after adjustment for expected age-related hearing loss, in the LFSNHL group ranged from 20.1 to 26.4 dB (Table 2). None of the 11 patients with significant LFSNHL had symptoms suggestive of Ménière's disease, such as fluctuating hearing loss or episodic vertigo.

Otopathology Study

There were 249 temporal bone specimens with otosclerosis, including 93 operated specimens and 156 nonoperated specimens. Most of the operated temporal bones (71/93, 77%) had undergone a single operation, but 20% had undergone two operations and 3% had undergone three operations. Stapedectomies were performed in 72 specimens, stapes mobilization in 22 specimens, and lateral canal fenestration operation in 16 ears.

TABLE 2. *Change in hearing over long-term follow-up*

		Operated	Nonoperated
Total	No.	110	123
	Δ BC, age-adjusted at 250 Hz (dB)	5.7	2.6
	Δ BC, age-adjusted at 500 Hz (dB)	6.7	8.1
LFSNHL	No. (% of total)	8 (7.3%)	3 (2.4%)
	Δ BC, age-adjusted at 250 Hz (dB)	21.4	22.5
	Δ BC, age-adjusted at 500 Hz (dB)	20.1	26.4
	Initial WR (%)	84.0	85.0
	Final WR (%)	74.0	68.0

Initial refers to the first audiogram; Final refers to last available follow-up audiogram.

Δ BC indicates BC difference between final and initial follow-up times (≥10 yr); LFSNHL, low-frequency sensorineural hearing loss; No., number; WR, word recognition score (monosyllabic).

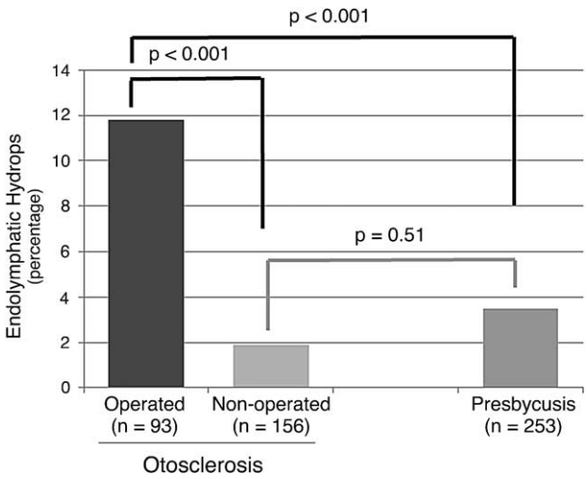

FIG. 1. Prevalence of endolymphatic hydrops in operated otosclerosis and nonoperated otosclerosis, and presbycusis.

There were 276 temporal bone specimens with presbycusis, which were evaluated for use in the control group. Of these, we excluded 23 specimens because of a concomittant histologic diagnosis of otosclerosis, leaving a control group of 253 specimens.

Evidence of EH was found in 11 of 93 (11.8%) operated temporal bones, 3 of 156 (1.9%) nonoperated temporal bones, and 9 of 253 (3.5%) presbycusis temporal bones (Fig. 1). The incidence of EH in operated otosclerosis temporal bones was statistically significantly higher compared with nonoperated otosclerosis temporal bones ($p \leq 0.001$, χ^2 test), and compared with presbycusis temporal bones ($p \leq 0.001$, χ^2 test). There was no difference in the incidence of EH between nonoperated otosclerosis and the presbycusis groups ($p = 0.51$). A representative patient with bilateral otosclerosis can be observed in Figure 2 (see also Table 3), which shows a profound EH on the operated side (Patient 2L, Table 4).

The demographic and relevant clinical characteristics of the operated, nonoperated otosclerosis, and presbyacusis groups (including the type of operation and symptoms of episodic vertigo and hearing fluctuation) are shown in Table 4. Notably, increasing numbers of surgeries (i.e. revision surgeries) on each ear were correlated with increasing risk of developed delayed EH. All three ears that had undergone three operations developed EH, compared with 21% (4/19) of ears status post two operations, and compared with 5.6% (4/71) for whom who had only single operation.

Of the 253 specimens in the control group, 9 (3.5%) specimens had histologic evidence of EH. All of these nine specimens had EH in the cochlea, which was

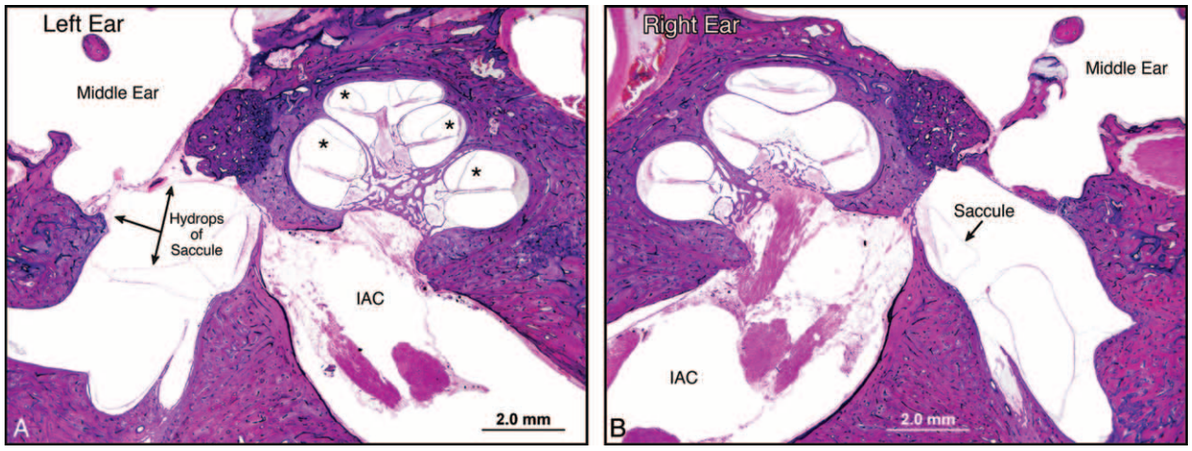

FIG. 2. A 70-year-old woman with bilateral otosclerosis, who had undergone stapedectomy followed by mobilization surgery on the left side and no surgery on the right side. *A*, On the operated side, the inner ear has severe endolymphatic hydrops affecting the saccule, which balloons out to reach the level of the oval window. There are profound hydrops in the basal turn and moderated hydrops in the middle and apical turns of the cochlea (*). *B*, On nonoperated right ear, there is no endolymphatic hydrops (Patient 2L, Table 3). IAC indicates internal auditory canal.

profound in seven of nine specimens (Table 4). Six of the operated temporal bones had evidence of EH in both the cochlea and vestibule (saccular/utricular membrane wall), three specimens had EH only in the vestibule, and one specimen had EH only in the cochlea. For temporal bone specimen 1L, EH could not be assessed in the vestibule because of new bone formation, but EH was found in the cochlear duct. The otosclerosis in the operated temporal bones was more extensive, with more foci, and more patients in whom the foci of otosclerosis reached the endosteal layer in the cochlear duct, compared with nonoperated otosclerosis specimens (Table 4). The hearing evaluation was available in all the patients except one patient of the presbycusis group. The

TABLE 3. *Location and degree of endolymphatic hydrops*

			Hydrops			Degree of Cochlear Hydrops		
Group	No.	Patient/Side	Sc.	U	C	Slight	Moderate	Profound
Operated	1	1/L	[a]	[a]	Y	–	–	Y
	2	2/L	Y	Y	Y	–	–	Y
	3	3/L	N	N	Y	Y	–	–
	4	4/R	Y	Y	Y	Y	–	–
	5	5/R	Y	N	Y	Y	–	–
	6	6/R	Y	N	Y	–	–	Y
	7	7/R	Y	N	N	–	–	Y
	8	7/L	Y	Y	Y	Y	–	–
	9	8/R	Y	N	Y	Y	–	–
	10	9/L	Y	N	N	–	–	–
	11	10/L	Y	N	N	–	–	–
Nonoperated	1	11/L	Y	N	Y	–	Y	–
	2	11/R	Y	N	Y	Y	–	–
	3	12/R	Y	Y	Y	–	–	Y
Presbycusis	1	13/L	N	N	Y	–	–	Y
	2	14/L	Y	Y	Y	–	–	Y
	3	15/L	Y	N	Y	–	–	Y
	4	16/L	Y	N	Y	–	–	Y
	5	17/R	Y	Y	Y	–	–	Y
	6	18/R	Y	Y	Y	–	–	Y
	7	18/L	Y	Y	Y	–	–	Y
	8	19/L	Y	N	Y	Y	–	–
	9	20/L	Y	N	Y	–	Y	–

[a]Cannot assess hydrops because of a new bone formation.
C indicates cochlea; L, left; N, no; No., number; R, right; Sc., saccule; U, utricule; Y, yes.

TABLE 4. *Operated, nonoperated, and presbycusis patients with endolymphatic hydrops*

								Foci of Otosclerosis			
Group	No.	Patient/ Side	Sex	Operation	Episodic Vertigo	Hearing Fluctuation	Hearing Loss at Last Audiogram	AOW	C	RW	VA
Operated	1	1/L	F	M, M, S	Y	N	Profound, flat SNHL	Y[a]	Y[b]	N	N
	2	2/L	F	M, S	Y	N	Severe-profound, flat mixed HL	Y[a]	Y[b]	Y	N
	3	3/L	F	M	N	N	Severe-profound, down-sloping mixed HL	Y	N	N	N
	4	4/R	F	M, S	Y	N	Moderate, down- sloping mixed HL	Y[a]	Y	Y	N
	5	5/R	F	S	N	N	Severe- profound, down-sloping mixed HL	Y[a]	Y[b]	N	N
	6	6/R	F	S,S	Y	N	Moderate-severe, down- sloping mixed HL	Y[a]	Y[b]	Y	N
	7	7/R	M	M	N	N	Profound, down-sloping mixed HL	Y[a]	Y[b]	Y	N
	8	7/L	M	S, S	N	N	Profound, down- sloping mixed HL	Y[a]	Y[b]	Y	N
	9	8/R	M	Fn, S, S	N	N	Moderate, down-sloping mixed HL	Y[a]	Y[b]	N	N
	10	9/L	M	S	N	N	Moderate-severe, down- sloping SNHL	N	N	N	N
	11	10/L	F	M, S, S	Y	N	Severe, down- sloping mixed HL	Y[a]	N	N	N
Nonoperated	1	11/L	F	None	Y	N	Sever-profound, flat mixed HL	Y	N	N	N
	2	11/R	F	None	Y	N	Severe-profound, flat SNHL	Y	N	N	N
	3	12/R	F	None	N	N	Profound, flat SNHL	Y	N	N	N
Presbycusis	1	13/L	M	None	N	N	Severe, flat SNHL	_	_	_	N
	2	14/L	F	None	Y	N	Severe-profound, down-sloping SNHL	_	_	_	N
	3	15/L	M	None	Y	N	NA	_	_	_	N
	4	16/L	F	None	Y	N	Mild, flat SNHL[c]	_	_	_	N
	5	17/R	M	None	N	N	Severe SNHL	_	_	_	N
	6	18/R	F	None	Y	N	Moderate-severe, down-sloping SNHL	_	_	_	N
	7	18/L	F	None	Y	N	Severe-profound, flat mixed HL	_	_	_	N
	8	19/L	M	None	N	N	Severe-profound, down-sloping SNHL	_	_	_	N
	9	20/L	F	None	N	N	Severe-profound, down-sloping SNHL	_	_	_	N

[a]Replacement of the vestibular endosteum.
[b]Replacement of the cochlear endosteum.
[c]Hearing test was made 20 years before death.
AOW indicates anterior oval window; C, cochlea; F, female; Fn, fenestration; HL, hearing loss; L, left; M, male; M, mobilization; N, no.; N/A, nonavailable; No., number; R, right; RW, round window; S, stapedectomy; SNHL, sensorineural hearing loss; VA, obstruction of the vestibular aquaduct; Y, yes.

audiograms demonstrated down-sloping or flat moderate-profound sensorineural hearing loss. Notably, the severity of hearing loss was more pronounced among the nonoperated patients (Table 4). Most patients did not have recorded BC thresholds on the last available audiogram, and likely bone conduction values would have been out-of-limits of the audiometer given the severity of the recorded air conduction thresholds. The medical chart review of the patient histories for the 11 ears with EH found that 5 (45%) of the operated temporal bones had episodic vertigo, of them three had objective evidence of vestibular dysfunction based on vestibular testing in the office or laboratory. Ménière's disease was diagnosed clinically in two of the three patients with objective vestibular dysfunction.

DISCUSSION

The objective of this study was to determine whether stapedectomy causes a delayed onset of EH, by comparing a clinical study evaluating LFSNHL as a marker for EH and a histopathologic human temporal bone study to evaluate for the histologic incidence of EH after stapedectomy. In the clinical study, we did not find a higher incidence of LFSNHL in operated otosclerosis ears over the $\geq$10 years after stapedectomy compared with nonoperated ears over a similar time period (7.2% versus 2.4%, $p = 0.08$). The otopathology study did, however, support the hypothesis that the operation for otosclerosis may cause damage to the inner ear resulting in EH. Specifically, the results demonstrated that operated ears had a significantly higher incidence of EH compared with the nonoperated ears (11.8% versus 1.9%, $p \leq 0.001$) and to the reference or age-related hearing loss group (11.8% versus 3.5%, $p \leq 0.001$). It is possible that stapedectomy does carry a risk of developing delayed EH many years after the operation, but based on our clinical study, this EH may not result in clinically significant LFSNHL.

Rauch et al. (25) conducted a double-blinded temporal bone study to assess the association between the histologic occurrence of EH and the clinical diagnosis and symptoms of Ménière's disease. Six of 19 patients (31%) with idiopathic EH did not meet the clinical criteria for a diagnosis of Ménière's disease based on clinical history and audiologic data (in strict accordance with American Academy of Otolaryngology—Head and Neck Surgery guidelines), highlighting that, in some patients, the finding of EH is not associated with specific symptoms.

Merchant et al. (26) studied EH in human temporal bones with or without Ménière's syndrome. They analyzed 35 patients diagnosed with idiopathic EH and 44 patients with secondary EH (surgical trauma, temporal bone fracture, infectious process, etc.). Nine patients (25%) of the idiopathic hydrops and 10 patients (22%) of the secondary hydrops were without a history of episodic vertigo. The authors summarized that EH (of the cochlea and/or vestibular system) did not always coincide with history of episodic vertigo, or specific low-frequency or fluctuating-type sensorineural hearing loss. The findings by Rauch et al., Merchant et al., and the comparison between histologic and clinical findings in the present study all suggest that histologic EH may not correlate 1:1 with the presence of a specific amount or type of hearing loss (i.e. may not always present as a LFSNHL).

Our clinical study describes very long-term audiologic outcomes ($\geq$10 yr) and a relatively large sample size (N = 223), with 99.6% of relevant data (i.e. low-frequency masked bone conduction thresholds) available in the study group. There are few reports of low-frequency hearing outcomes over long follow-up periods in operated otosclerosis patients, and none to the best of our knowledge that specifically evaluate age-adjusted low-frequency BC thresholds over this extended time period. Vincent et al. (27) evaluated the BC hearing threshold change in poststapedotomy patients at four frequencies (500, 1000, 2000, 4000 Hz), not including 250 Hz, and found that the mean all-frequency BC threshold did not change significantly over time in the 180 patients followed for $\geq$10 years. In this study, there was no adjustment for age-related hearing loss or comparison to control group of nonoperated otosclerosis patients. Our results of age-adjusted BC threshold change of 5.7 dB at 250 Hz and 6.7 dB at 500 Hz were similar to Vincent et al., but still were slightly higher (more hearing loss) than Vincent et al., especially given that the age correction had already been applied to our study. Redfors et al. (28) followed the hearing results after surgery for over 30 years. He summarized that the sensorineural hearing loss measured by BC was significantly greater long after the operation (matching to ISO 7029). When evaluating his results in low frequency at 500 Hz (the frequency of 250 Hz was not tested), there was no significant difference, similar to the findings in our study.

The Carhart effect, which is an overestimation of BC thresholds because of an audiological artifact of stapes fixation, is most pronounced at 2000 Hz (29,30). Since the Carhart effect is less pronounced at lower frequencies and the Carhart effect correction for 250 Hz is undefined, we decided not to apply any Carhart correction to the clinical study.

Our clinical study has several limitations, including selection bias because of the retrospective design. Given the differences in age and follow-up time between the operated and nonoperated groups, it is possible that the younger age and slightly shorter follow-up time contributed to better audiologic outcomes and the lack of finding more LFSNHL in the operated group. Nevertheless, the comparison of the temporal bone study results to the clinical study helps us understand the clinical relevance of pathologic findings, and the clinical study provides data on a large number of patients over a very long follow-up period.

Although emerging radiologic techniques make some imaging for EH of the inner ear possible during life (6,7), human temporal bone pathology remains the gold standard for diagnosing pathology such as EH. This was a relatively large number of temporal bone specimens, but there is inherent selection bias using the temporal bone collection; use of the collection may select for more involved or severe patients of otologic disorders since these patients are particularly motivated to donate their ears.

Additionally, relevant information of symptoms (ear's trauma, noise exposure, unsteadiness, vertigo, ear fullness, etc.) may be missing for the clinical history charts in both subsets of studies. By definition the symptoms of EH are mostly consistent with episodic vertigo and fluctuate hearing loss; therefore, the entirely episode may be missed.

CONCLUSIONS

Long-term audiologic follow-up data in the clinical study showed no difference in LFSNHL between operated ears with otosclerosis and nonoperated ears with otosclerosis (used as a control), suggesting that stapedectomy does not significantly increase the risk of developing LFSNHL. The histologic incidence of delayed endolymphatic hydrops was significantly higher in operated temporal bones with otosclerosis (11.8%) compared with nonoperated temporal bones with otosclerosis (1.9%) or compared with a control group of temporal bones with presbycusis (3.5%). Although endolymphatic hydrops are more common after stapedectomy based on this temporal bone study, the comparison to the clinical study shows that delayed EH may not translate to clinical symptoms, such as LFSNHL.

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