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Su-Velez, Brooke
Lopez, Ivan
Ishiyama, Akira
et al.

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Human Temporal Bone Study of Vestibular Histopathology in Cochlear Implant Patients With Cochlear Hydrops

Brooke M. Su-Velez[*], **Ivan A. Lopez**[*], **Akira Ishiyama**[*], **Gail Ishiyama**[†]

[*]Department of Head and Neck Surgery, University of California, Los Angeles, California

[†]Department of Neurology, David Geffen School of Medicine, University of California, Los Angeles, California

Abstract

Hypothesis: Endolymphatic hydrops (EH) associated with cochlear implantation are associated with vestibular dysfunction.

Background: Vestibular dysfunction is a known risk after cochlear implantation (CI). CI has been shown to cause cochlear hydrops due to fibrosis surrounding the ductus reuniens. However, the association of cochlear hydrops with vestibular hydrops and the relationship to vestibular symptoms remain unknown.

Methods: Histopathological analysis and clinical evaluation of the vestibular end organs of 17 human temporal bones (HTB)s exhibiting cochlear hydrops from 15 CI recipients.

Results: Eight of 15 patients with cochlear hydrops due to CI had complaints of dizziness, vertigo, or imbalance following CI. In all 17 HTBs with cochlear hydrops, there was fibrosis, atrophy, or obstruction of the ductus reuniens, and all had straight electrode CI via cochleostomy. In one of the eight reporting postoperative dizziness, labyrinthitis ossificans was deemed causative. Six of the seven remaining patients had EH of both the saccule and utricle. Fifteen of 17 HTBs (88.2%) had saccular EH. In contrast, 8 of 17 HTBs (47.0%) in 7 patients had utricular EH, of which 6 patients had postoperative vertigo spells. It seems that hydrops of the utricle closely corresponds to postoperative vertigo spells and vestibular complaints.

Conclusion: Implantation of the CI, when complicated by ductus reuniens fibrosis, may cause both cochlear hydrops and vestibular endolymphatic hydrops. Hydrops of the vestibular periphery when involving the utricle seems to be more likely associated with disabling vertigo symptoms. This study supports the round window technique of insertion rather than cochleostomy.

Keywords

Cochlear implant; Endolymphatic hydrops; Histopathology; Temporal bone; Vestibular dysfunction

Address correspondence and reprint requests to Gail Ishiyama, M.D., UCLA Department of Neurology, 300 Medical Plaza Ste B200, Los Angeles, CA 90095; GIshiyama@mednet.ucla.edu.

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Cochlear implants (CI) provide significant benefit to both children and adults with severe or profound sensorineural hearing loss, and in recent years the indications for cochlear implantation have been expanding (1,2). Vestibular dysfunction is a well-known possible sequela after cochlear implantation, and has been reported at rates ranging from about 15% to up to 70% in different studies (1-6). Various mechanisms of vestibular injury have been proposed, such as direct surgical trauma from the electrode or from the cochleostomy (6), inflammatory reaction, implant stimulation, and endolymphatic hydrops (3,4,7).

This remarkable heterogeneity of reported incidence is due partially to the various methods of reporting and measuring vestibular dysfunction, whether subjectively reported by the patient using measures like the Dizziness Handicap Inventory, or via objective tests such as caloric testing, posturography, or vestibular evoked myogenic potentials (6-8). A study by Melvin et al. (8) in 2008 found that patient-reported levels of dizziness correlated poorly with findings from other vestibular testing. Each modality for vestibular testing is limited to only one portion of the vestibular system, with the most commonly used method, electro- or videonystagmography, assessing only the horizontal semicircular canal crista ampullaris function. However, the evaluation of the vestibular function using videonystagmography can be subject to artifact in the setting of surgical alterations to the temporal bone, which may affect the ability to induce a temperature gradient in the horizontal semicircular canal. The language with which patients describe subjective symptoms of vestibular dysfunction is also highly variable and may not be recorded or reported accurately (6). Timing of onset of vestibular symptoms is also variable and can be significantly delayed after implantation (4). This may be due to the fact that there is likely a delay in the development of endolymphatic hydrops, if hydrops is the mechanism whereby dizziness or vertigo spells occur.

Given that not all CI patients exhibit vestibular dysfunction after implantation, we sought to evaluate whether endolymphatic hydrops is a factor in CI-associated vestibular dysfunction. Additionally, patients undergoing CI can have pre-existing symptoms of vestibular dysfunction associated with the cause of their hearing loss, and some studies report that vestibular dysfunction improved following CI (3,4,6). The human vestibular system is also redundant and can compensate for unilateral abnormalities, although in the acute setting of the development of unilateral vestibulopathy, one might expect complaints of dizziness. Conversely, a slow development of a unilateral vestibulopathy may allow for compensation, making it even more difficult to assess the true incidence of vestibular damage after CI (8). The gold standard for understanding the complications of CI surgery is histopathological studies of human temporal bones (HTB) in CI patients. Only through the study of HTBs are we able to assess the evidence of vestibular injury to explain vestibular complaints following CI.

Few previous histopathologic studies of the temporal bone have described changes in the vestibular system after cochlear implantation. Tien and Linthicum (9) reported a correlation between scala vestibuli damage from electrode translocation causing cochlea and vestibular end organ damage in 11 patients. However, these histopathological findings in the vestibular periphery were not correlated with CIinical reports of balance issues (9). Handzel et al. (10) noted that while there was no change in Scarpa’s ganglion cells and the vestibular neuroepithelial hair cell layer between implanted and nonimplanted HTBs, the presence of

cochlear endolymphatic hydrops was more often observed along with saccular hydrops in the CI recipient.

In this study, we sought to better characterize the cytoarchitecture of vestibular end organs after cochlear implantation. A previous study from our group described an association between electrode insertion technique via cochleostomy and resultant fibrosis of the scala vestibuli, leading to ductus reuniens obstruction and endolymphatic hydrops of the cochlea. In that study, it is proposed that cochlear EH due to ductus reuniens fibrosis secondary to cochleostomy may be the cause of residual hearing loss after CI (11). In the present study, we hypothesize that the obstruction or fibrosis of the ductus reuniens can be associated with not only cochlear endolymphatic hydrops affecting the hearing, but also with hydrops of the vestibular system causing membrane distension and damage to the utricular and saccular maculae, as well as the semicircular canals, which may be the cause of postoperative vestibulopathy, vertigo spells, and dizziness.

METHODS

The NIDCD National Temporal Bone Laboratory at UCLA (HTB) database was used to identify bones from patients who had undergone CI with a histopathological finding of cochlear hydrops. Excluded from the study were subjects with a history of Menière's disease or a history of significant vertigo or dizziness before CI implantation.

The HTBs were prepared as has been previously described (11). The temporal bones had been removed postmortem and placed in 10% neutral buffered formalin for 3 weeks. They were then decalcified in ethylenediaminetetraacetic acid until confirmed free of calcium by X-ray imaging. The HTBs were then embedded in increasing concentrations of celloidin to allow complete penetration. To minimize any extraction movement damage, the CI electrode was removed just before the specimen being placed in hardening chloroform. Each celloidin block was cut into 20-μm sections, and every tenth section of which was mounted and processed in hematoxylin and eosin stains.

Each HTB microscopic section was examined first for evidence of cochlear endolymphatic hydrops, defined as distention of Reissner's membrane into the scala vestibuli, with the exception of the apical segment. The HTB sections were then analyzed for ductus reuniens pathology, utricular and saccular pathology, and semicircular canal and cristae ampullares pathology.

Copies of the medical record for each patient donor were reviewed for demographic information and clinical history. The medical records for patient donors were gathered from various institutions, and as a result only some had preoperative vestibular testing, most commonly in the form of electronystagmography. When available, this preoperative vestibular testing was reviewed. clinical reports of dizziness pre- and post-CI were obtained from either provider notes, patient questionnaires, or direct patient correspondence with providers archived in the medical records.

This study was approved by the Institutional Review Board (IRB) of UCLA(IRB protocol #10-001449). All methods used in this study were in accordance with NIH and IRB

guidelines and regulations. Appropriate informedconsent was obtained fromeach patient before inclusion in the study. Support for this study is through a grant from the National Institute on Deafness and Other Communication Disorders, grant number 1U24DC015910-01 (AI).

RESULTS

Demographic Information

Table 1 shows a tabular format of the demographic information, vestibular complaints and vestibular testing, and the histopathological findings within the semicircular canal cristae ampullares, the utricle and saccule, and ductus reuniens. There were a total of 17 HTBs from 15 CI patients identified for this study, implanted between 1973 and 1998. All of these HTBs were selected for further analysis of the vestibular system due to the presence of endolymphatic hydrops in the cochlea. All patients had CI electrode insertion via a cochleostomy approach confirmed on histology. There were 8 male and 7 female CI patients, and 10 left and 7 right HTBs. Two patients had missing medical records. The etiologies of the sensorineural hearing loss (SNHL) included meningitis, otosclerosis, sudden SNHL, and hereditary SNHL. In three cases, etiology was unknown. The average age at death was 76.8 years of age, and the average age at cochlear implantation was 66.9 years.

Analysis of Patients With Postoperative Vestibular Dizziness

Eight of the 15 patients (53.3%) with cochlear hydrops had complaints of dizziness, vertigo, or imbalance following CI. One patient had vertigo that was well controlled preoperatively and no documented vestibular issues following CI (HTB 6-7) and is not included in the postoperative dizziness group. Five patients had no vestibular complaints following CI (HTB 6-7, 9, 15, 16, 17). The two remaining patients had no clinical records available (HTB 8, 14).

One of the eight patients with postoperative vestibular complaints (oscillopsia in HTB 1) had evidence of labyrinthitis ossificans as the etiology of the vestibular complaints, with preoperative vestibular testing demonstrating a caloric paresis, and histopathology demonstrating ossification of the SCCs, the saccule, and the utricle. Of the seven remaining patients with postoperative dizziness, six of the seven patients had histopathology consistent with endolymphatic hydrops (EH) of the saccule and utricle (HTB 2–5, 10, 11, 12). The remaining one patient (HTB 13) who had mild dizziness and nausea postoperatively had loss of saccular nerve fibers and a normal utricle. Only two of the seven patients had EH of the semicircular canals (SCC) (HTB 2, 4–5); one patient had bilateral EH of the SCCs (HTB 4–5), while three of seven had SCC atrophy or fibrosis (HTB 3, 11, 12), and two had normal SCCs (HTB 10, 13).

Not all patients with vestibular EH exhibited dizziness; the proportion of EH of the saccule was highest, followed by the utricle, then lastly the SCC. Fifteen of 17 (88.2%) had saccular EH, with 1 having ossification (HTB1) and 1 having atrophy of nerve fibers (HTB 13). Eight of 17 HTBs in 7 patients (47.0%) had utricular EH, of whom 6 patients had postoperative vertigo spells (1 patient had unknown history). Five of the 17 HTBs (29.4%) had fibrosis,

atrophy, or hair cell loss of the utricle. Four of 17 ears (23.5%) in 3 patients had EH of the SCCs, but only 2 of these patients reporting postoperative dizziness and 1 with unknown history. Another 8 of 17 (47.0%) HTBs exhibited either fibrosis or epithelial degeneration of the SCC, with a total of 12 of 17 with pathology of the SCC (70.5%). Most of the patients with cochlear hydrops also had hydrops of the saccule; however that was not necessarily associated with postoperative dizziness. In contrast, it seems that hydrops of the utricle most closely corresponded to postoperative vertigo spells, with six out of seven of these patients reporting postoperative dizziness and one with unknown history. No patient with utricular hydrops on histopathology did not have postoperative dizziness.

Preoperative Vestibular Testing Results

Eight patients had preoperative vestibular testing results available for review (Table 1). HTB 1 was associated with unilateral left caloric weakness consistent with the history of labyrinthitis ossificans and persistent oscillopsia. HTB 2 and 3 had bilateral vestibular weakness, both with a history of otosclerosis, but both also had vertigo reported *after* CI, occurring in spells. All other five patients with preoperative vestibular testing revealed normal caloric responses, including four of the patients with postoperative recurrent vertigo spells (HTB 2, 5, 7, 8).

Histopathology in the Vestibular Periphery

In general, the cristae ampullares and the SCCs were relatively well preserved (Fig. 1, A-C), though 11 of 17 HTBs (64.7%) had some mild pathological changes of the semicircular canals ranging from membrane collapse to cristae hair cell loss and fibrosis. For comparison, a normative HTB from a subject with no audio-vestibular dysfunction and no history of CI is shown in Figures 1A, 2A, and 3A. Figure 1B and C both demonstrate thinning and atrophy of the vestibular neuroepithelium with hair cell loss. Both of these exhibited membrane distension or membrane collapse, indicative of endolymphatic hydrops within the semicircular canals. While records for the patient in 1B were not available, the patient in 1C reported recurrent episodes of vertigo starting a few months after CI.

The ductus reuniens morphology was pathological in all 17 cases of the HTBs with cochlear hydrops, exhibiting either fibrosis, atrophy, or obstruction (Fig. 2, A-C). In Figure 2B, there is obstruction of the ductus reuniens, and there is associated hydrops of the utricle and saccule. Postoperatively, this patient reported severe dizziness. In Figure 2C, there is atrophy of the ductus reuniens, and associated hydrops of the utricle and saccule, and deformity of the maculae. This patient reported sensations of imbalance after CI surgery.

Figure 3A to D demonstrates the findings of saccular and utricular pathology in this group. The most common finding was saccular degeneration, whether as membranous distension or collapse, representing hydrops or a deformed macula in 15 of the 17 HTBs (88.2%). One case had labyrinthitis ossificans with obliteration of the saccule and ossification of the utricle (HTB 1) and one had reduced saccular nerve fibers (HTB 13). In the three patients with normal utricular morphology (HTB 6, 13, 14), one had postoperative dizziness, one reported vertigo easily controlled by medications preoperatively, and one had no available records.

DISCUSSION

Cochlear implantation has been noted to be associated with postoperative vestibular complaints of vertigo, dizziness, or imbalance in some patients. In the present study, the vestibular end organs of 17 HTBs from 15 patients presenting with histopathological findings of cochlear endolymphatic hydrops after CI in patients without a history of Menière's disease were examined. There was pathology with atrophy, obstruction, or fibrosis of the ductus reuniens in 17 out of 17, indicating that ductus reuniens injury is likely playing a role in the development of hydrops. All of the HTBs had placement of CI via cochleostomy. Additionally, within this group of CI recipients with known cochlear hydrops, there was a fairly high proportion of subjects with prolonged postoperative vestibular complaints in 8 of 15 patients (53.3%). One of the subjects had evidence for labyrinthitis ossificans as causative of the dizziness, and had preoperative unilateral caloric weakness (HTB 1). Of the remaining seven with postoperative vertigo and vestibular complaints, all had endolymphatic hydrops in the saccule, except for one that reported "mild dizziness" and had only reduced saccular nerve fibers on histopathology. Within this group, it is notable for the high proportion of pathology in the saccule with all 17 saccules exhibiting pathology, and 15 of 17 exhibiting hydrops (88.2%), 1 exhibiting obliteration from fibrosis (HTB 1), and 1 exhibiting saccular nerve loss (HTB 13). Hydrops was most common in the saccule, followed by the utricle, followed by the SCCs: 15 of 17 (88.2%) exhibited saccular hydrops, 8 of 17 (47.0%) exhibited utricular hydrops, and 4 of 17 (23.5%) exhibited SCC hydrops. This suggests that vestibular endolymphatic hydrops in the presence of cochlear hydrops is most likely to affect the saccule, followed by the utricle, whereas the semicircular canals are relatively preserved.

In the present study, saccular hydrops was common in the presence of cochlear hydrops but was not predictive of postoperative dizziness or vestibular complaints. Utricular hydrops or utricular macular deformation, in the presence of coexistent saccular hydrops, was uniformly associated with postoperative vestibular complaints with eight HTBs in seven patients. One patient had no records (HTB 8). Only one patient with postoperative dizziness did not have hydrops of both saccule and utricle (HTB 13) but the dizziness was reported as mild. All three patients with hydrops of the SCC (n=4HTBs) had postoperative dizziness, except one with no records (HTB 8). Hydrops of the utricle had the highest sensitivity and specificity for postoperative dizziness and vertigo.

Vestibular dysfunction after cochlear implantation is a common complication; however, it is not well understood. There is mounting evidence regarding the role of endolymphatic hydrops relating to post-CI vestibular symptoms. Fina et al. (4) describe two distinct entities of vestibular dysfunction after CI: only a few patients have acute and transient vertigo attacks immediately postoperatively, whereas 39% of their cohort had delayed (1 mo-1 yr after CI) onset of episodic vertigo reminiscent of Menière's symptoms, suggesting a similar mechanism. Frodlund et al. compared the effects of different types of CI electrodes on the vestibular system; they reported straight electrodes were associated with sudden onset vertigo and loss of vestibular function in a delayed fashion up to 11 months after surgery, and two of these patients also lost residual hearing simultaneously, which may represent the development of endolymphatic hydrops. The cochleostomy approach and resulting

intracochlear trauma has been associated with the development of fibrosis of the ductus reuniens, and the development of cochlear endolymphatic hydrops which would likely be associated with loss of residual hearing (6,11-13). In the present study, we demonstrate that endolymphatic hydrops involving the vestibular system, especially hydrops of the utricle, is highly associated with patient report of postoperative recurrent vertigo spells or dizziness. In contrast, nearly all HTBs with cochlear hydrops due to CI have associated saccular hydrops as well, but not all of the patients with saccular hydrops have a history of postoperative vertigo spells. We think that the presence of hydrops in the SCC may fluctuate with symptoms, as it is unknown if these patients had vertigo spells near the time of HTB donation. While only four HTBs in three patients had EH of the SCC, there were an additional eight HTBs exhibiting SCC fibrosis or degeneration. Of the seven patients with postoperative vertigo after CI, all but one had utricular hydrops. Conversely, all patients with utricular hydrops had postoperative dizziness or vertigo. We propose that cochlear hydrops which occurs in the setting of cochleostomy-induced fibrosis of the scala vestibuli and ductus reuniens may be associated with postoperative recurrent spells of vertigo or dizziness when the endolymphatic hydrops is severe enough to involve the utricle. In contrast, not all patients with saccular hydrops had postoperative vertigo spells, and only a subset of the postoperative vertigo patients had SCC hydrops.

Multiple studies report evidence for saccular dysfunction after CI as documented by cervical vestibular evoked myogenic potentials testing, which is more prevalent than horizontal semicircular canal dysfunction as demonstrated by head impulse test or calorics (8,12). Anatomically, the saccule is nearest the basal turn of the cochlea where damage from electrode insertion may be most likely to occur, and is connected to the cochlea via the ductus reuniens (9). This predilection for saccular damage and saccular hydrops, and therefore an increased risk of vestibular dysfunction, in the setting of cochlear hydrops (which likely is secondary to fibrosis of the ductus reuniens) is confirmed in our findings.

A major limitation of our conclusions is due to its retrospective nature, as there are incomplete medical records and lack of consistent objective vestibular testing information for the HTB specimens. Further comparison with a larger sample size, investigation of nonimplanted HTBs, and incorporation of objective vestibular testing measures are needed. Prospective measures of vestibular symptoms accompanied by objective tests are also needed.

Our study consisted of mostly older individuals who have been shown to have poorer vestibular outcomes and more disability than younger CI patients, which may be due to comorbid conditions or decreased ability to compensate for unilateral vestibulopathy (4-6). Pediatric CI patients tend to have less vestibular issues than older patients. In the adult with equal hearing loss bilaterally, and similar length of deafness on both sides, preoperative testing of vestibular function may aid in the decision to implant the side with poorer vestibular function. The effect of fibrosis of the ductus reuniens secondary to cochleostomy-induced injury can cause endolymphatic hydrops of both the cochlea, potentially affecting hearing outcomes, and of the vestibular system, causing postoperative vestibulopathy and dizziness. To reduce the risk of damage to the ductus reuniens, we recommend the atraumatic round window approach instead of a cochleostomy for CI surgery.

CONCLUSION

Examination of the histopathology of human temporal bones with cochlear endolymphatic hydrops after CI shows significant endolymphatic hydrops in the vestibular end-organs as well, most notably in the saccule with 88% exhibiting hydrops. Of the eight patients with postoperative vertigo and dizziness, one had labyrinthitis ossificans. Of the remaining patients with postoperative dizziness, there was utricular hydrops in six out of the seven. The converse was also true, in that all patients with utricular hydrops had postoperative vertigo spells. In the present study, we demonstrate that cochlear hydrops due to ductus reuniens fibrosis and CI using the cochleostomy approach is often associated with hydrops in the vestibular end organs. When hydrops involves the utricle and ampullae of the SCC, there is a high chance for associated postimplantation vestibular symptoms of vertigo and imbalance. Therefore, based on these results the round window approach is preferred over the cochleostomy approach to prevent endolymphatic hydrops whenever possible.

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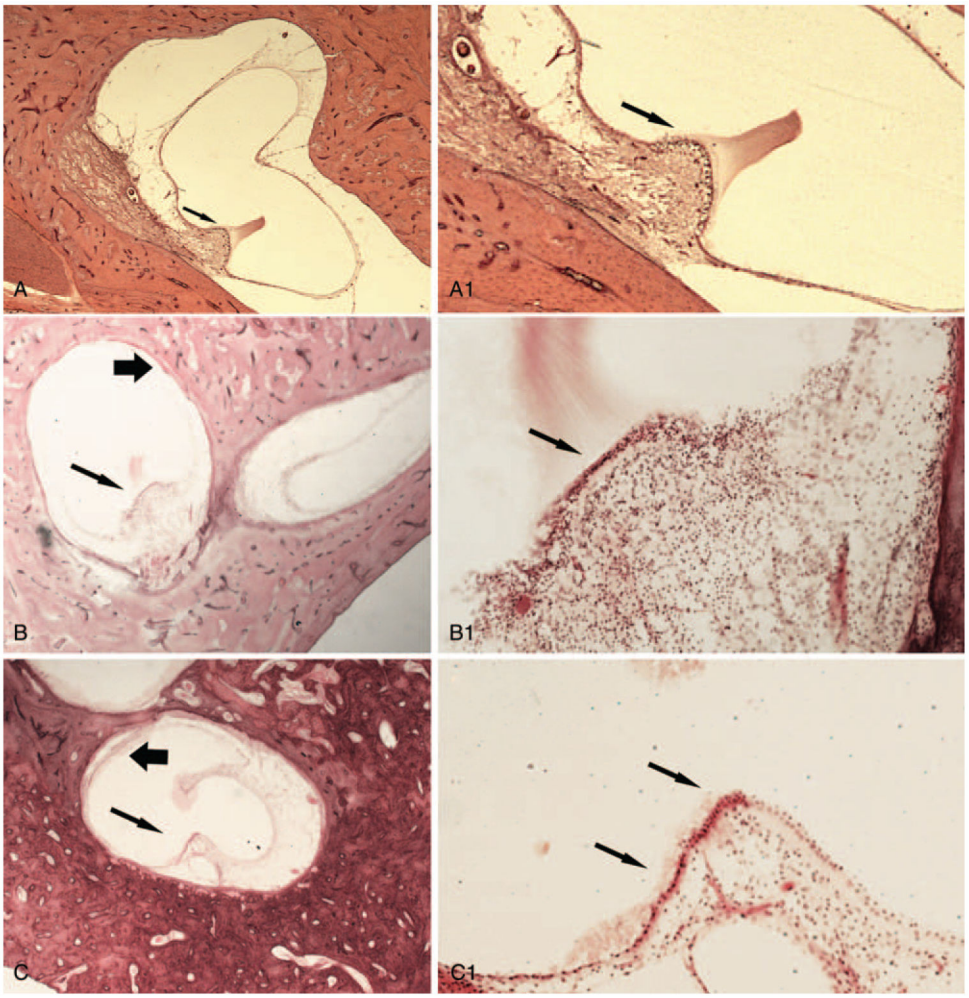

FIG. 1.

Cristae ampullares in the normative and in CI-associated cochlear hydrops. *A,* Normative horizontal semicircular canal crista sensory epithelia in a 38-year-old female without history of auditory or vestibular abnormalities, and no history of CI. *Arrow* points to the normal sensory neuroepithelia with the normal overlying cupula. There is no distension of the membrane and no endolymphatic hydrops. *A1,* On the right side is a high magnification view. HCA indicates horizontal crista ampullaris; SE, sensory epithelium. Hematoxylin & eosin staining. *B,* HTB 14: Superior semicircular canal flattening and endolymphatic hydrops in a 75-year-old male with cochlear hydrops following CI. *B*, On the left side, cochlear and endolymphatic hydrops is present (large *arrow*) with distension of the membranous labyrinth. *B1,* The superior semicircular canal demonstrates flattening of the epithelium within the apical portion (*arrows.*) The ductus reuniens exhibits fibrosis (not shown). Hematoxylin & eosin staining. *C,* HTB 5: Thinning of the crista ampullaris neuroepithelium and endolymphatic hydrops in a 67-year-old male with episodes of dizziness following CI for otosclerosis. Significant endolymphatic hydrops is present with membrane distension (*arrowhead*) in the saccule and utricle. The semicircular canal crista ampullaris neuroepithelium demonstrates loss of vestibular hair cells (*arrows*). *C1,* High magnification. Hematoxylin and eosin staining. HTB indicates human temporal bones.

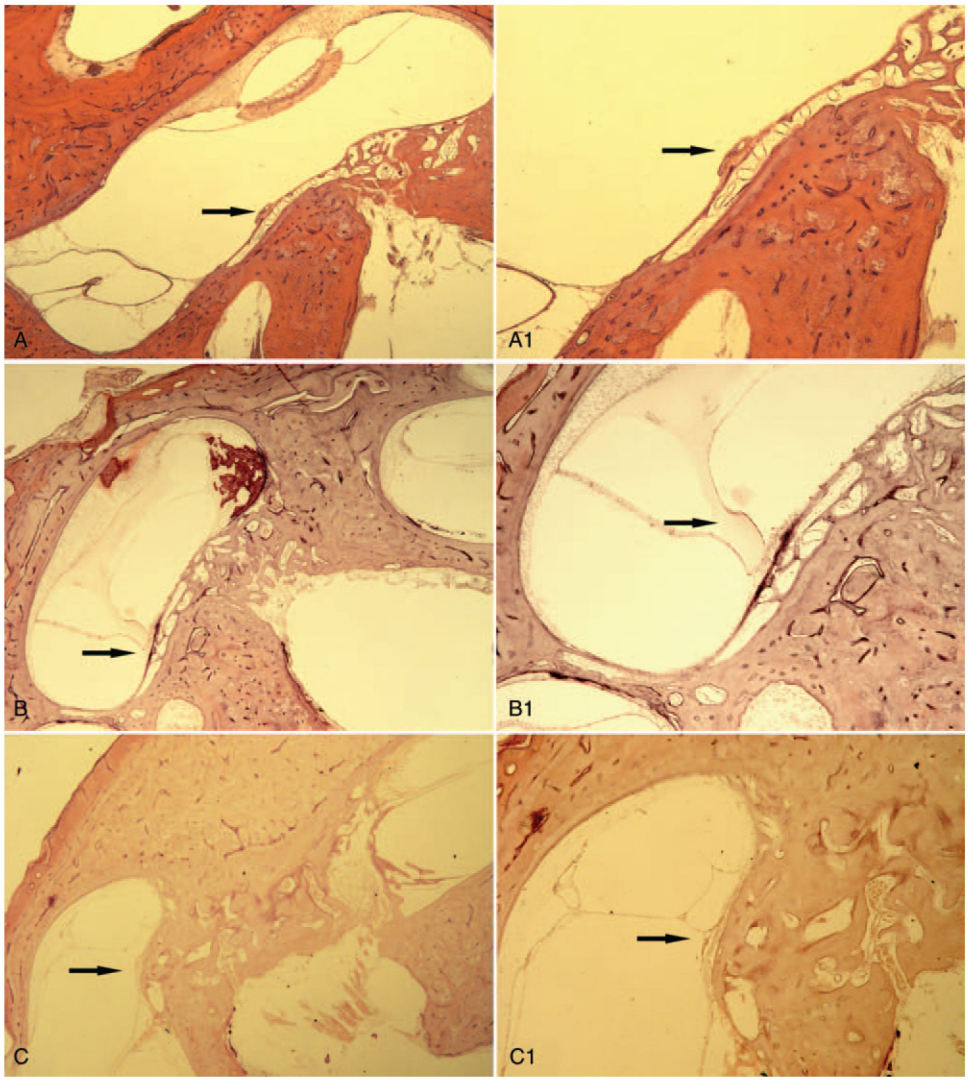

FIG. 2.

Ductus reuniens in the normative and in CI-associated cochlear hydrops. *A,* Normative ductus reuniens in a 38-year-old-female without history of auditory or vestibular abnormalities, and no history of CI (same HTB as Fig. 1A). There is no cochlear hydrops, and the organization of the ductus reuniens demonstrates patency (*arrow*). *A1,* Right side high magnification. Hematoxylin & eosin staining. *B,* HTB 12: Obstruction of the ductus reuniens in a 73-year-old female with a history of severe balance issues following CI for SNHL of undetermined cause. There is associated endolymphatic hydrops of the cochlear and the saccule, and the horizontal canal crista exhibits fibrosis (not shown). *Arrows* point to the obstruction of the ductus reuniens and fibrosis (*arrow*). *B1,* High magnification. Hematoxylin and eosin staining. *C,* HTB 11: Atrophy of the ductus reuniens in an 87-year-old female with a history of imbalance following CI for SNHL of unknown cause. There is extensive hydrops and membrane distension in the saccule. *Arrow* points to atrophy of the ductus reuniens. *C1,* Right side high magnification. Hematoxylin and eosin staining. HTB indicates human temporal bones.

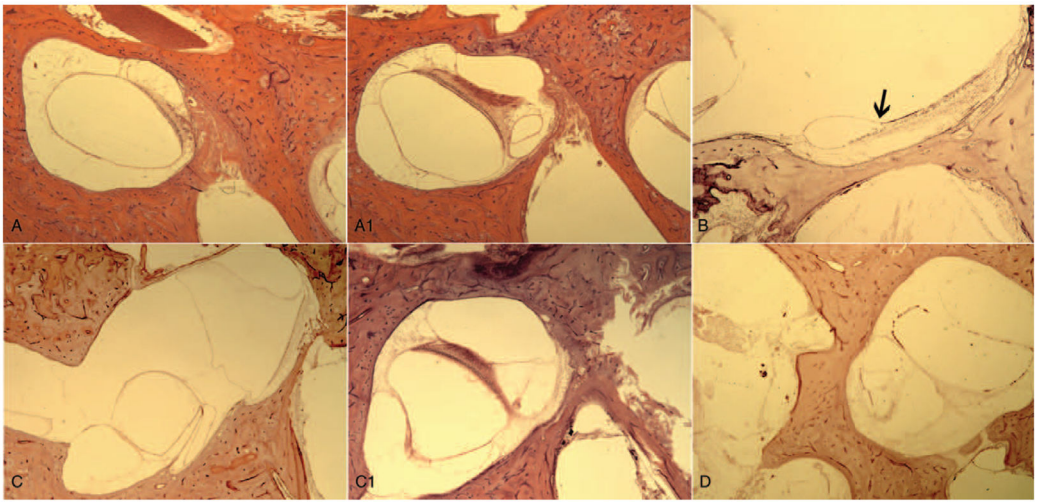

FIG. 3.

Maculae in the utricle and saccule in the normative and in CI-associated cochlear hydrops. *A,* Normative vestibular macula utricle and A1 macula saccule in a 38-year-old female without history of auditory of vestibular abnormalities, and no history of CI (same HTB as Figs. 1 and 2A). There is no endolymphatic hydrops and the vestibular neuroepithelium exhibits normal hair cells, and there is no membrane distension. Hematoxylin and eosin staining. *B,* HTB 10: Saccular hydrops in a 92-year-old female with a history of unsteadiness and dizziness following CI for otosclerosis. *B,* The saccular membrane is collapsed (*arrow*) and there is degeneration of the saccular macula. The utricular macula is deformed (not shown). There is fibrosis of the ductus reuniens (not shown). Hematoxylin and eosin staining. *C,* HTB 12: Saccular hydrops and utricular hydrops in a 73-year-old female with a history of severe balance issues following CI for SNHL of undetermined cause (same as Fig. 2B). *C,* There is saccular hydrops and distension of the saccular macula. *C1,* There is also hydrops and distension of the utricular membrane. Figure 2B demonstrates obstruction of the ductus reuniens. Hematoxylin and eosin staining. *D,* HTB 11: Hydrops and degeneration of saccule and utricle in an 87-year-old female with a history of imbalance following CI for SNHL of unknown cause (same as Fig. 2C). *D,* There is severe degeneration of vestibular end-organs with atrophy in both the utricular and saccular maculae which are both deformed. Figure 2C demonstrates atrophy of the ductus reuniens. Hematoxylin and eosin staining. HTB indicates human temporal bones.

TABLE 1.

Vestibular histopathology in HTB with cochlear hydrops

HTB	Age	Side (L, R)	Sex (M, F)	Etiology	Electrode Insertion 1 = cochleostomy 2 = RW	Electrode Type 1 = straight 2 = curved	Age at Implantation	Vestibular Symptoms	Pre-CI Vestibular Testing	SCC	Saccule	Utricle	Ductus reuniens
1	54	L	M	Meningitis	1	1	52	Oscillopsia	Unilateral left side weakness	Ossified PSCC	Obliteration	Fibrosis	Fibrosis
2	58	L	F	Ototoxicity	1	1	56	Episodic dizziness after CI for years	Normal	Collapse of membrane	Collapse of membrane	Collapse	Obstruction due to scarring
3	75	L	M	Otosclerosis	1	1	61	Acute vertigo attacks before CI, and episodic dizziness 3 years after CI	Bilateral vestibular weakness	Epithelial atrophy	Membrane distension and hydrops	Atrophy and distension	Fibrosis
4	67	R	M	Otosclerosis	1	1	65	Episodic dizziness 1–2 months after CI	Bilateral vestibular weakness	Rupture of ampulla	Membrane distension, epithelial damage	Hydrops	Fibrosis
5	67	L	M	same	1	1	65	Same	Same	Membrane Collapse	Membrane distension, epithelial damage	Hydrops	Fibrosis
6	74	R	M	Otosclerosis	1	1	64	Vertigo before CI, none postoperative	None	Normal	Membrane distension	Normal	Fibrosis
7	74	L	M	same	1	1	64	same	None	Distortion of cupulae	Collapse of saccule and distention	Partial degeneration	Fibrosis
8	71	R	F	Unknown cause of SNHL	1	1	N/A	No records	No records	Normal	Distortion of Membrane	Deformed macula	Mild fibrosis
9	87	R	M	Otosclerosis	1	1	84	No dizziness	None	Cristae hair cell loss	Deformed macula	Normal	Mild fibrosis
10	92	R	F	Otosclerosis	1	1	82	Unsteadiness a few months after CI	Normal	Normal	Distortion of Membrane	Deformed macula	Fibrosis
11	87	L	F	Unknown cause of SNHL	1	1	68	Imbalance	None	Fibrosis	Hydrops and deformed macula	Deformed macula	Atrophy
12	73	L	F	Sudden SNHL	1	1	66	Severe dizziness after CI leading to multiple falls	Normal	Posterior limb fibrosis	Severe Hydrops and	Hydrops	Complete Obstruction

HTB	Age	Side (L, R)	Sex (M, F)	Etiology	Electrode Insertion 1 = cochleostomy 2 = RW	Electrode Type 1 = straight 2 = curved	Age at Implantation	Vestibular Symptoms	Pre-CI Vestibular Testing	SCC	Saccule	Utricle	Ductus reuniens
											membrane distension		due to fibrosis
13	76	R	F	Hereditary SNHL	1	1	52	Mild dizziness	Normal	Normal	Reduced saccular nerve fibers	Normal	Fibrosis
14	75	L	M	Unknown cause of SNHL	1	1	N/A	No records	No records	Distended membrane, Flattened epithelium	Collapsed Membrane onto macula	Normal	Fibrosis
15	93	L	M	Otosclerosis	1	1	82	No dizziness	None	Fibrosis	Thickened and collapsed membrane	Diminished hair cells	Fibrosis
16	81	R	F	Postinfectious	1	1	73	No dizziness	Normal	Normal	Deformed macula	Normal	Fibrosis
17	89	L	M	Otosclerosis	1	1	73	No dizziness	None	Epithelial atrophy	Hydrops	Atrophy	Obliterated

Age indicates age in years at the time of death; C, cochleostomy; Etiology, cause of the hearing loss; F, female; HTB, human temporal bone; L, left; M, male; R, right; RW, round window insertion or extended round window; SCC, semicircular canals, including ampulla, neuroepithelium, membranous labyrinth, hair cells; Vestibular symptoms, symptoms of dizziness, vertigo, or imbalance following cochlear implantation.