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Authors

Hodge, Sarah
Ishiyama, Gail
Lopez, Ivan
et al.

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Histopathologic Analysis of Temporal Bones with Otosclerosis following Cochlear Implantation

Sarah E. Hodge, MD[1], **Gail Ishiyama, MD**[2], **Ivan A. Lopez, PhD**[3], **Akira Ishiyama, MD**[3]

[1]House Ear Clinic, Los Angeles, California USA, David Geffen School of Medicine at UCLA, Los Angeles California, USA

[2]Department of Neurology, David Geffen School of Medicine at UCLA, Los Angeles California, USA

[3]Department of Head and Neck Surgery, David Geffen School of Medicine at UCLA, Los Angeles California, USA

Abstract

Objective: Analyze changes in osteoneogenesis and fibrosis following cochlear implant (CI) surgery in patients with otosclerosis and compare differences based on insertion technique.

Background: When advanced otosclerotic disease extends to the otic capsule, severe and profound sensorineural hearing loss necessitates consideration of a cochlear implant. Histopathological analysis of the human temporal bone after implantation in the patient with otosclerosis may reveal important variables that predict CI success.

Methods: Histopathological evaluation of archival human temporal bones from subjects with a history of CI for cochlear otosclerosis. A total of 17 human temporal bones (HTB) were analyzed, 13 implanted and 4 contralateral non-implanted controls.

Results: Histopathological studies revealed extensive osteoneogenesis and fibrosis which was more prominent at the cochleostomy insertion site in the basal turn of the cochlea often obliterating the scala tympani in the basal turn, and in some cases extending to the scala media and scala vestibuli. Cochlear hydrops was nearly universal in these cases. This contrasted with the round window insertion, which exhibited minimal osteoneogenesis within the cochlear duct. In addition, in the contralateral, unimplanted control ears, there was otosclerosis at the stapes footplate, fissula ante fenestrum but no osteoneogenesis within the cochlear duct.

Conclusion: Cochleostomy approach to CI insertion in otosclerosis patients is associated with significant fibrosis, osteoneogenesis, and cochlear hydrops. A round window insertion technique can be utilized to help minimize these histopathologic findings whenever feasible.

INTRODUCTION

Otosclerosis is a bony disease process found in the otic capsule. It is characterized by abnormal replacement of endochondral bone with spongiotic and ultimately sclerotic

Sarah Hodge, MD, House Ear Clinic, 2100 W 3rd St, Unit 111, Los Angeles, CA 90057, (213) 483-9930, shodge@houseclinic.com.

bone through a continuous cycle involving repeated osteolysis and osteogenesis. The mean clinical prevalence of otosclerosis is reported to be approximately 0.3–0.4% though histologically, the prevalence ranges from 2.5–12% based on cadaveric temporal bone studies (1, 2). Therefore, otosclerosis is thought to be inherited in an autosomal dominant nature with incomplete penetrance (3). The classic clinical presentation of otosclerosis is a progressive conductive hearing loss, though up to a third of patients can present with a mixed loss (2).

When looking at the process of otosclerosis histologically, the bone is populated by a high number of active osteoblasts and osteoclasts which contribute to the formation of otospongiotic and otosclerotic lesions (2,4). These lesions are most commonly found anterior to the oval window, as 96% of temporal bones with otosclerosis demonstrated lesions in this region (5). In 49–60% of temporal bone specimens reviewed in two separate studies, however, otosclerotic lesions were found in more than one site in the otic capsule (2, 5–6). A less frequent, though clinically significant site of otosclerosis is within the cochlear wall itself (2). Previous histologic analysis has proven that when otosclerosis involves the cochlear wall and invades the endosteum, it leads to hyalinization of the spiral ligament (2,4). This more advanced otosclerosis can lead to severe and ultimately profound sensorineural hearing loss necessitating consideration of a cochlear implant for optimal hearing rehabilitation (7).

Cochlear implantation in patients with otosclerosis poses important considerations for the surgeon as this disease process is a known potential negative prognostic factor with regards to postoperative speech performance scores (1,7–8). Patients additionally may experience an increased rate of complications including electrode dislocation and facial nerve stimulation (1,7–8). Histologically, cochlear implantation in a non-otosclerotic ear shows formation of intracochlear fibrosis and new bone formation thought to be induced by the trauma of insertion and as a reaction to the presence of the electrode, a foreign material, within the cochlea (9). Multiple studies have investigated surgical techniques to minimize this initial trauma, especially when emphasis is placed on preserving residual hearing (9–12). These investigations postulate that implantation via the round window, when compared to the standard promontory cochleostomy, can minimize initial intracochlear trauma and help to limit the amount of consequent new tissue formation (9,10). The advantages of a round window insertion includes a reduction in the amount of drilling required for electrode insertion thereby decreasing the amount of bone dust that enters the cochlea (9,10). These techniques to limit bony trauma may be particularly important in patients with otosclerosis, where the bone itself is the primary location of the inherent disease process. This study thus aims to investigate the histopathologic changes in the cochlea after cochlear implant surgery in patients with otosclerosis to ultimately highlight important surgical considerations in this patient population.

METHODS

Selection of temporal bones included those with a diagnosis of otosclerosis who had undergone cochlear implantation as well as 2 temporal bones with a cochlear implant which did not have otosclerosis which were used for further comparison. A total of 17

human temporal bones were analyzed. Three of the psatients had bilateral implantation, so each side was analyzed separately. A total of 11 implanted human temporal bones (HTBs) with otosclerosis were analyzed in addition to 4 contralateral, non-implanted HTBs. Demographic data is summarized in Table 1. When analyzing the temporal bones, a particular focus of review was on the electrode insertion site, the path of the electrode through the cochlea, and on the surrounding areas of otosclerosis and new tissue formation. The Institutional Review Board (IRB) approved this study (IRB protocol #10–001449). All methods used in this study are in accordance with NIH and IRB guidelines and regulations. The temporal bone donors were part of a National Institute of Health funded National Temporal Bone Laboratory through the National Institute on Deafness and Other Communication Disorders.

The processing of the temporal bones occurred as described in (13). The temporal bones had been removed postmortem and placed in 10% neutral buffered formalin for 3 weeks, decalcified in ethylenediaminetetraacetic acid until shown by x-ray to be free of calcium. Embedding was done in increasingly concentrated celloidin to allow complete penetration. To minimize extraction movement, the electrode was removed just before the specimen was placed in hardening chloroform. The celloidin block was cut into 20-mm sections of which every tenth was mounted and stained with hematoxylin and eosin (H&E)

RESULTS

Demographic information is summarized in Table 1. There are a total of 17 human temporal bones (HTBs) which were analyzed from 8 patients with otosclerosis and 2 patients without otosclerosis. Eight out of ten of the patients were male. All otosclerosis patients had a diagnosis of bilateral otosclerosis. 13 of the HTBs had undergone cochlear implantation (CI), with three patients having bilateral CI (HTBs 1–6), and six patients with unilateral CI. In one case (HTB 10), the contralateral HTB was not sectioned in a manner to facilitate comparison. Eleven of the CIs were placed using cochleostomy, and two HTB underwent CI by round window insertion (HTB 8 and 17). There were four ears (HTBs 12–15) of the unimplanted, contralateral ears which were available for within subject comparison of the effect of the CI in otosclerosis. Of the implanted HTBs studied, there were 5 on the right, and 8 on the left. Of the implanted ears, 6 were implanted with a short 3M/House 6mm single channel electrode while the remaining 7 were implanted with the Nucleus 22 multi-channel electrode.

Histopathological studies of the HTBs with otosclerosis revealed varying degrees of osteoneogenesis from the site of insertion extending throughout the temporal bone along the electrodes, more prominent in the basal turn when cochleostomy insertion technique had been used. In cases of cochleostomy insertion of CI, there was concomitant fibrosis and tissue formation near the ductus reuniens, which was associated with cochlear hydrops. Osteoneogenesis and/ or fibrosis was much more prominent at the cochleostomy insertion site in the basal turn of the cochlea, when compared with the osteoneogenesis and / or fibrosis in the case of round window insertion. See Figures 1a–d. Within subject comparison was performed with HTB 9 and 14 (see Figure 2a–d). Additionally, comparison with HTBs was performed to assess the histologic changes seen with both cochleostomy and round

window insertion in the absence of otosclerosis to further emphasize the role this inherent disease may have on post-implantation fibrosis and neo-ossifcation (see Figure 3a–b).

DISCUSSION

Human temporal bone histopathological studies of cochlear implantation in patients with otosclerosis reveal the formation of an intracochlear foreign body reaction to the electrode and new bone formation i.e. osteoneogenesis within the cochlea itself (2). Additionally, a fibrous capsule is known to develop along the length of the electrode array (2). Detailed histopathological studies of human temporal bones investigating histologic changes based on electrode insertion technique have not yet been done in otosclerosis patients.

Aiming to decrease the amount of fibrosis and osteoneogenesis in the cochlea has important clinical implications, as extensive osteoneogenesis and new tissue formation is associated with poor speech outcomes in CI patients (7). Histopathological studies of human temporal bones after cochlear implantation demonstrate new tissue growth centered along the insertion site, the basal turn of the cochlea, and along the electrode (2, 9). In the present study, all otosclerosis patients with CI placed using cochleostomy showed extensive osteoneogenesis at the site of cochleostomy, often extending throughout the cochlear duct following the path of the electrode. In many cases, there was complete obliteration of the scala tympani in the basal turn. In contrast, in the otosclerosis patient with CI placed using the round window approach, there was no osteoneogenesis within the cochlear duct, and only a fibrous capsule surrounding the electrode. The scala tympani remained patent throughout including in the basal turn. Of note, the contralateral unimplanted ear in the patient with the CI placed by the round window approach, there was no osteoneogenesis within the cochlear duct. In three cases, the contralateral unimplanted ear could be compared with the ear implanted using the cochleostomy approach. The differences were stark in all cases as noted in Figure 2. Within a given patient, the effect of CI placed using the cochleostomy approach triggered moderate to severe osteoneogenesis often with near obliteration of the scala tympani. An additional comparison was made with implanted patients without otosclerosis (Figure 3). When a cochleostomy was performed (Figure 3a), there was noted fibrosis and osteogenesis along the path of the electrode with consequent new bone formation along the insertion site with fibrosis in the scala tympani extending into the scala vestibuli in the basal turn. However, when compared to the bony and fibrotic changes in the otosclerosis bones, there was notably less new bone formation and reactive tissue formation. The round window insertion in the non-otosclerotic bone (Figure 3b) showed no new bone formation and only mild fibrosis along the electrode capsule. Choi and Oghalai created cochlear models which demonstrated that the presence of fibrosis and scar can dampen vibrations along the basilar membrane, which may explain why the speech outcome is affected in the setting of new tissue formation (14). This osteoneogenesis and new tissue formation can have a direct impact on electrode impedance level which can cause raised stimulus thresholds of the cochlear implant (15). Additionally, Seyyedi et al., when reviewing HTBs with otosclerosis, found that the majority exhibited invasion of the cochlear endosteum by otosclerotic plaques in at least one turn (16). These changes in the cochlear endosteum due to otosclerosis has also been cited to influence additional postoperative

concerns regarding unwanted facial nerve stimulation following implantation in this patient population (16).

An additional adverse effect of new bone and fibrous tissue formation after cochlear implantation is the consequent obstruction, atrophy, or fibrosis of the ductus reuniens (13, 17). This has previously been reported to cause endolymphatic hydrops of the cochlea, possibly impacting the preservation of residual hearing and ultimately impacting audiometric outcomes (13). Additionally, obstruction of the ductus reuniens can lead to hydrops of the vestibular system, causing postoperative recurrent vertigo spells and dizziness (17). Previous reports have therefore stressed that the atraumatic round window electrode insertion is preferred over the cochleostomy insertion technique as the round window approach triggers less osteoneogenesis and fibrosis after cochlear implantation (13, 17, 18). We hypothesized that the effect of cochleostomy in patients with otosclerosis would be magnified given that damage to the endosteum can trigger osteoneogenesis in otosclerosis. Thus, it would be advised to implant at the earliest possible time given that the development of narrowing of the cochlear duct on CT may indicate more difficulty to successfully place the electrodes and may also make it more challenging to use the round window approach. This would help to avoid cases where otosclerosis involves the round window, or in fact, obliterates it completely (2), necessitating drilling of the round window which in the case of otosclerosis would similarly trigger osteoneogenesis. Additionally, earlier identification of narrowing of the cochlear duct may allow for more successful placement of the electrodes, and less likelihood for the need for cochleostomy and the more unusual measure taken in far-advanced otosclerosis which may include cochleostomy into the scala vestibuli due to complete ossification of the scala tympani (19).

It is important to note that the available example of a definitive round window insertion involved a short, single-channel electrode. The consequent osteoneogenesis and fibrosis was therefore compared to a short, single-channel electrode inserted via cochleostomy to maintain as much validity within the comparison as possible. Longer electrodes can lead to even more robust intracochlear damage as it travels longer lengths through the cochlea. It has previously been reported that the greatest amounts of new tissue formation is localized at the electrode insertion site and within the basal turn with a decrease as one progresses apically and little beyond the tip of the electrode (9). Longer electrodes therefore, when compared to the House/3M 6mm single-channel electrode depicted in this study, have the potential to cause damage along longer lengths of the cochlea. This highlights the importance of minimizing electrode insertion trauma whenever possible.

A notable limitation of this study is our one histologic example of a round window insertion with which to make comparison to the cochleostomy insertion in otosclerosis patients. Therefore, to help aid our investigation, multiple additional comparisons were done including with round window insertion techniques in non-otosclerosis patients, and in un-implanted otosclerosis bones to help highlight and differentiate the histopathologic changes in this patient population. Despite this, however, additional HTB specimens in otosclerosis patients who have undergone cochlear implantation via round window insertion would be extremely useful.

Future areas of research would involve direct comparison with longer, multi-channel electrodes in this patient population. Moreover, future studies outlining hearing outcomes in otosclerosis patients with each insertion technique would be of paramount importance. Previous techniques to better quantify the amount of new bone formation following cochlear implantation using 3D temporal bone modeling has been performed (20). Using this technique in this subset of patients will be a future endeavor to further characterize the post-implantation histologic changes.

CONCLUSION

Round window insertion when performing cochlear implant surgery in otosclerosis patients was associated with minimal new bone growth while the cochleostomy approach to implant insertion uniformly had extensive amounts of osteoneogenesis and fibrosis, findings which have been associated with poorer hearing outcomes (7, 13–15). The round window technique should therefore be prioritized, when feasible, in this patient population.

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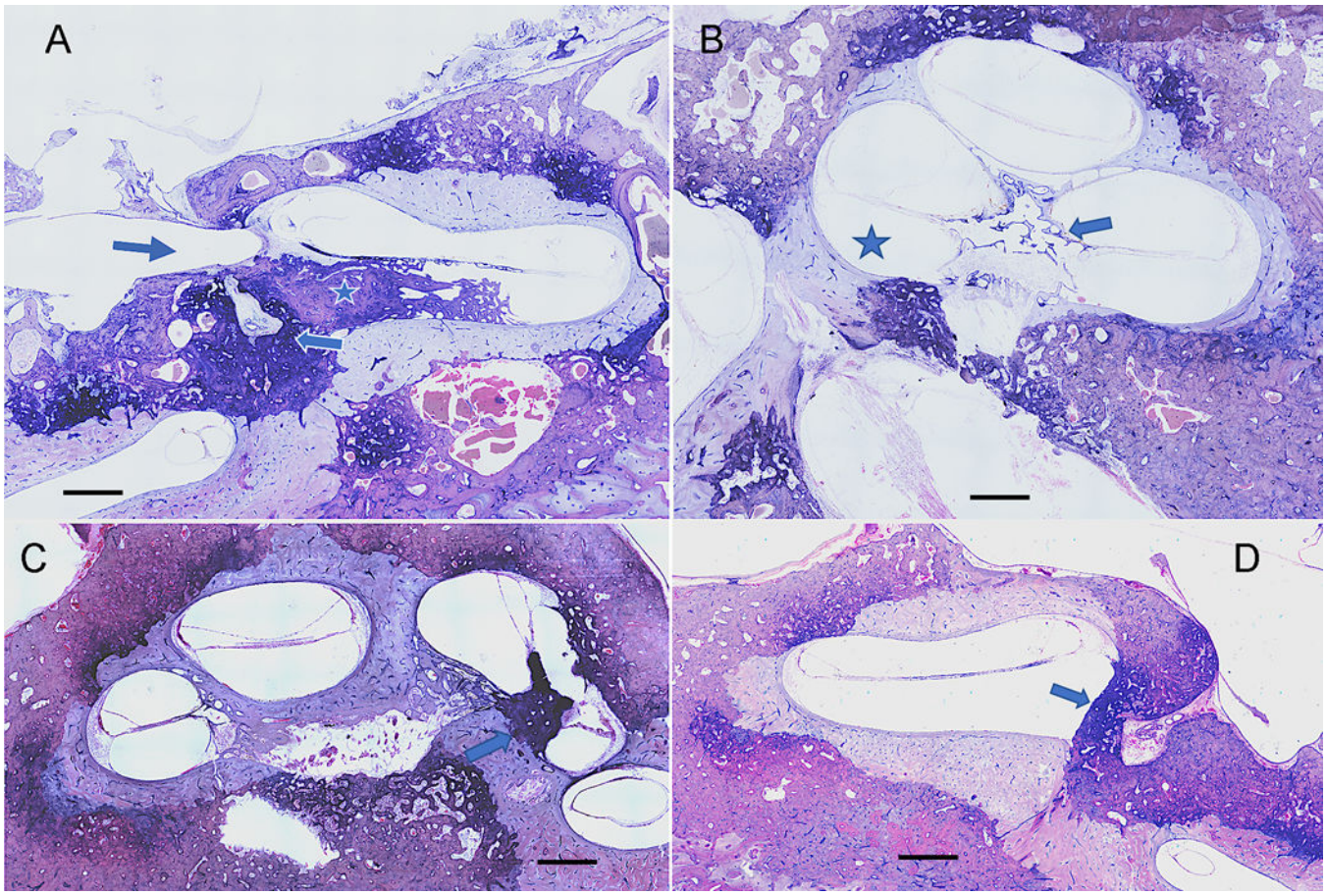

Figure 1: 1a&b: Right cochlear implant via cochleostomy with a House/3M 6mm single-channel electrode (HTB 3, H&E stain).

67-year-old male with otosclerosis with cochlear implantation 2 years prior via cochleostomy. **1a.** Electrode insertion path via cochleostomy (long, thin arrow) with surrounding extensive osteoneogenesis (short, thick arrow) extending throughout the scala tympani, with near obliteration at the basal turn. **1b.** Midmodiolar view of same ear showing fibrosis along the path of the electrode in the scala tympani (star) and otoscelorosis plaques along the modiolus (arrow).

1c&d: Cochlear implantation via Round Window Approach with a House/3M 6mm single-channel electrode (HTB 8, H&E stain). 74 year-old male with a long history of otosclerosis who underwent left-sided cochlear implantation via round window approach. **1c.** The insertion site of the electrode via the round window (star) shows minimal reactive tissue formation and very little neo-ossification. The crista fenestra is intact and the scala tympani patent despite a thin fibrous capsule along the length of the electrode. There is a small foci of otosclerosis (arrow). **1d.** Cross-sectional view of the cochlea with further demonstration of electrode path with some surrounding osteogenesis (arrow). Magnification bar is 500 microns.

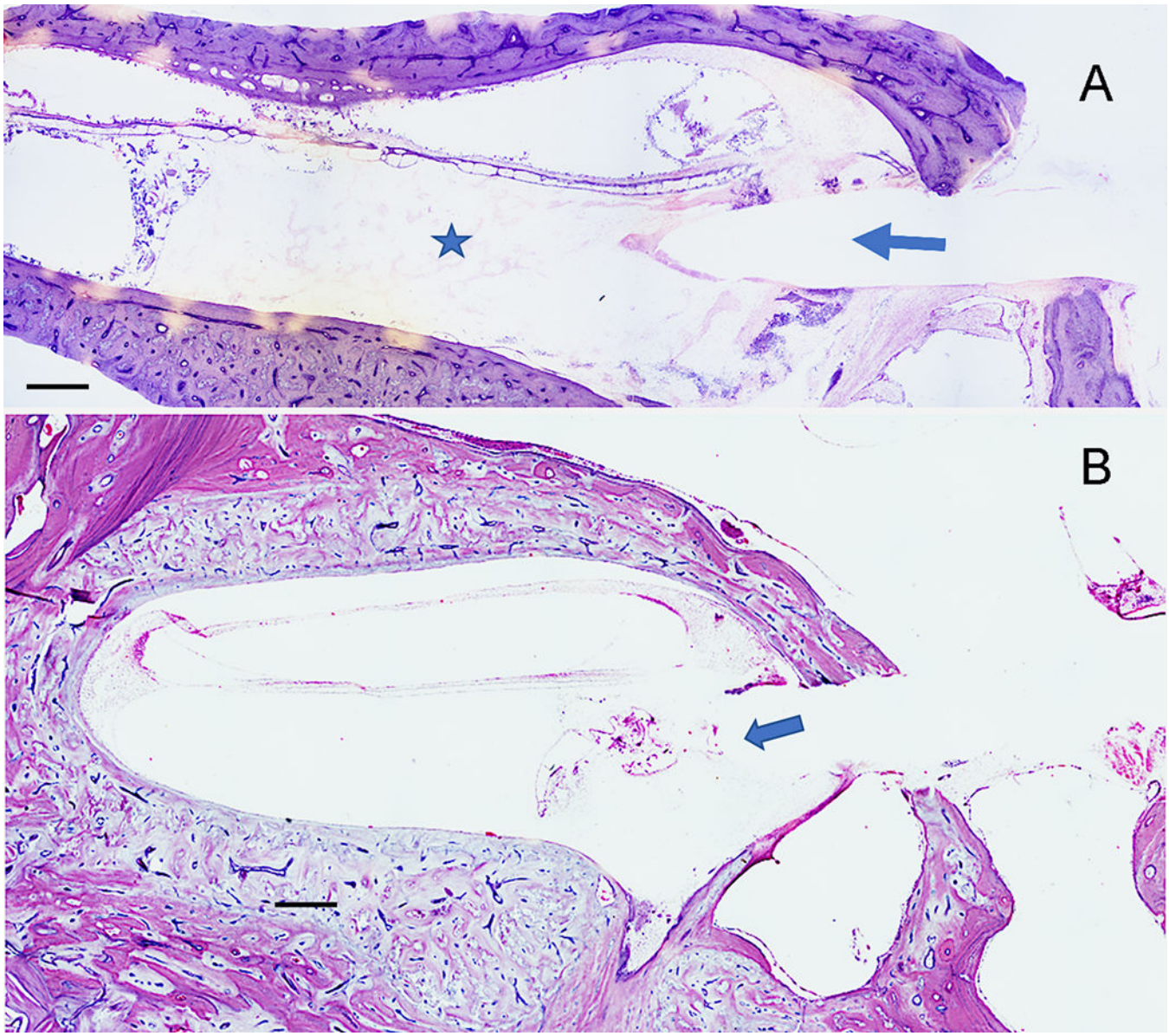

Figure 2: Figure 2a&b: Cochlear implantation via cochleostomy with a House/3M 6mm single-channel electrode (HTB 9, H&E stain):

92 year-old female with a long history of otosclerosis who underwent right-sided cochlear implantation 10 years prior via cochleostomy insertion. **2a.** The cochleostomy site shows extensive fibrosis and scarring around a large otosclerosis plaque (short arrow) near the basal turn. Surrounding the path of the electrode (long arrow) is intracochlear new bone formation (star) causing obliteration of the sinus tympani and of the scala media. **2b.** Midmodiolar view of same ear showing a patent scala tympani (star) though present otoscelorosis plaques along the modiolus (arrow). **2c&d: Contralateral, non-implanted (HTB 14, H&E stain). 2c.** Moderately-severe cochlear otosclerosis, with an intense focus along the basal turn of the cochlea (arrow). However, the scala appear patent throughout and there is no intracochlear new bone formation. **2d.** Basal turn of the cochlea showing an intense focus along the basal turn and at the round window (arrow). Magnification bar is 500 microns.

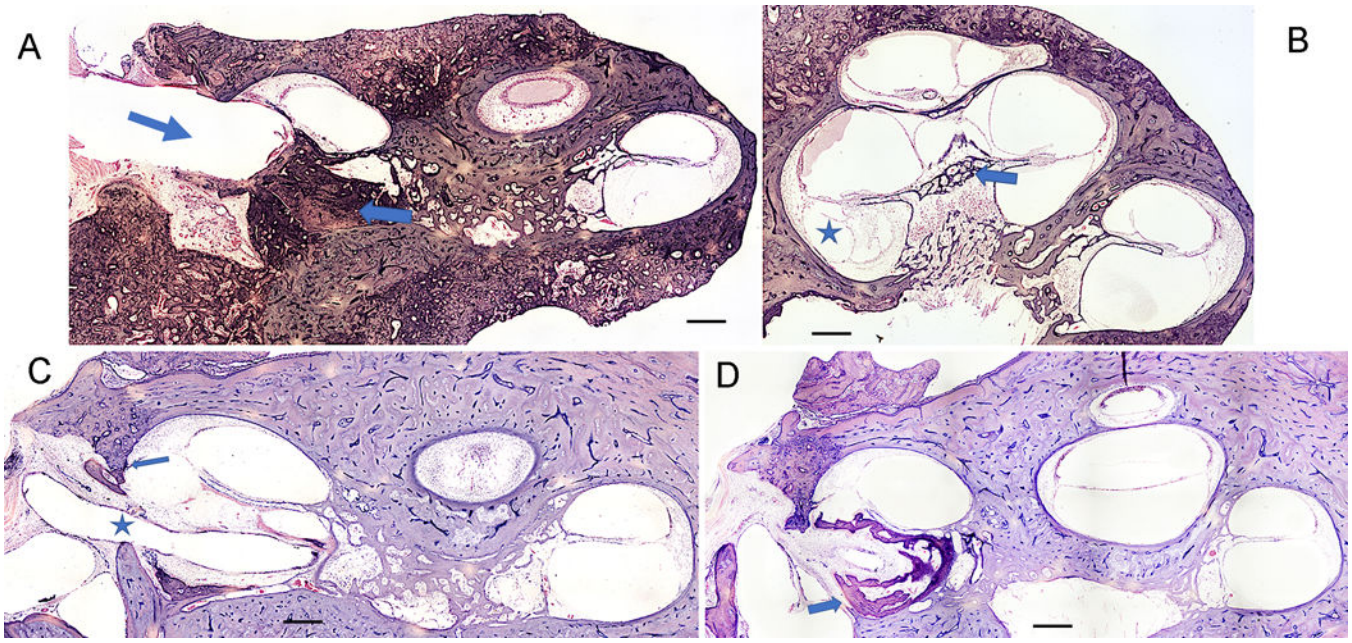

Figure 3: Cochlear implantation via cochleostomy with a House/3M 6 mm single-channel electrode (HTB 16, H&E stain):

Figure 3a: 58 year-old female with a history of streptomycin ototoxicity. There is mild fibrous tissue throughout the scala tympani (star) with surrounding areas of new bone formation along the periphery. There is additional fibrous tissue extending into the scala media and scala vestibuli**. 3b. Cochlear implantation via Round Window Approach with a House/3M 6mm single-channel electrode (HTB 17, H&E stain).** 80 year-old male with a previous history of progressive SNHL spanning the previous 40 years who underwent a left-sided cochlear implantation 8 years prior via round window insertion. Mild fibrous tissue around insertion site with some loose areolar fibrous tissue in the scala tympani in the inferior basal turn extending only half way the length of the inferior basal turn. Magnification bars is 500 microns.

Table 1:

Summary of Demographic Data

HTB #	Age	Age at Implantation	Side	Sex	Etiology of Hearing Loss	Implant Device
1	75	61	L	M	Otosclerosis	Nucleus 22
2	75	61	L	M	Otosclerosis	Nucleus 22
3	67	65	R	M	Otosclerosis	House/3M
4	67	65	L	M	Otosclerosis	House/3M
5	74	64	R	M	Otosclerosis	Nucleus 22
6	74	64	L	M	Otosclerosis	Nucleus 22
7	87	84	R	M	Otosclerosis	Nucleus 22
8	74	N/A	L	M	Otosclerosis	House/3M
9	92	82	R	F	Otosclerosis	House/3M
10	93	82	L	M	Otosclerosis	Nucleus 22
11	89	73	L	M	Otosclerosis	Nucleus 22
12	87	N/A	L	M	Otosclersosis	N/A
13	74	N/A	R	M	Otosclersosis	N/A
14	92	N/A	L	F	Otosclersosis	N/A
15	89	N/A	R	M	Otosclersosis	N/A
16	58	N/A	R	F	Ototoxicity	House/3M
17	80	72	L	M	Unknown	House/3M

Table 2:

Summary of Histopathologic Findings in Otosclerosis Temporal Bones

HTB #	Type of Insertion	Osteoneogenesis and Fibrosis	Ductus Reuniens
1	Cochleostomy	Extensive, obliterates scala tympani in basal turn and partially in mid turn	Fibrosis
2	Cochleostomy	Extensive, follows path of electrode in scala tympani in basal turn	Fibrosis
3	Cochleostomy	Extensive, obliterates scala tympani in basal turn	Fibrosis
4	Cochleostomy	Extensive, obliterates scala tympani in basal turn	Fibrosis
5	Cochleostomy	Extensive, obliterates scala tympani in basal turn	Fibrosis
6	Cochleostomy	Extensive, follows path of electrode in scala tympani in basal turn	Fibrosis
7	Cochleostomy	Extensive, follows path of electrode in scala tympani in basal turn	Fibrosis
8	RW	Patent scala tympani, minimal neo-ossification	Fibrosis
9	Cochleostomy	Extensive, obliterates scala tympani, media and vesitbuli in basal turn	Fibrosis
10	Cochleostomy	Extensive, basal turn to mid turn of cochlea, following path of electrode	Fibrosis
11	Cochleostomy	Extensive, obliterates scala tympani, media and vesitbuli in basal turn	Obliterated
12 (7)	Not Implanted	Otosclerosis focused along stapes flootplate, fissula ante fenestrum, & region of basal turn of cochlea. Scala patent.	Normal
13 (8)	Not Implanted	Moderate otosclerosis focused along stapes flootplate, fissula ante fenestrum. Scala patent.	Normal
14 (9)	Not Implanted	Moderately-severe otosclerosis focused along stapes flootplate, fissula ante fenestrum, and basal turn. Scala patent.	Normal
15 (11)	Not Implanted	Moderately-severe otosclerosis focused along stapes flootplate, fissula ante fenestrum, & region of basal turn of cochlea. Scala patent.	Normal

*HTB 10 did not have contralateral histologic slides available for comparison.

*( )represents corresponding, implanted side.