

THE REGISTRY

Newsletter of the NIDCD National Temporal Bone, Hearing and Balance Pathology Resource Registry



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CONTENTS

Featured Research

Histologic Analysis of Folded Cochlear Implant Electrode Arrays....1

Labyrinthine Concussion: Reexamination of a Historic Diagnosis Using Otopathology Techniques4

Registry News

Upcoming Events6

Otopathology Mini-Travel Fellowship7

Order Form for Temporal Bone Donation Brochures.....8

MISSION STATEMENT

The NIDCD National Temporal Bone, Hearing and Balance Pathology Resource Registry was established in 1992 by the National Institute on Deafness and Other Communication Disorders (NIDCD) of the National Institutes of Health (NIH) to continue and expand upon the former National Temporal Bone Banks (NTBB) Program. The Registry promotes research on hearing and balance disorders and serves as a resource for the public and the scientific community about research on the pathology of the human auditory and vestibular systems.

Histologic Analysis of Folded Cochlear Implant Electrode Arrays

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A traumatic placement of cochlear implant (CI) electrodes is a desired surgical technique as studies have shown that insertional trauma, over-insertion, and interscalar electrode translocation lead to poorer audiometric outcomes.^{1,2} Acute consequences of insertional trauma may reduce functional results from disruption of intracochlear structures with traumatic loss of spiral ganglion neurons (SGNs).¹ Studies of human temporal bones (TBs) of patients having undergone CI additionally show that implantation frequently results in chronic changes, such as intracochlear fibrosis and ossification.^{3,4} These chronic post-implant sequelae may intensify in cases with acute traumatic electrode placement and are correlated with worse hearing outcomes.^{3,4}

Recently developed electrodes with precurved conformations allow for perimodiolar placement within the scala tympani (ST) and may be placed atraumatically; however, they also present a greater potential for folding of the electrode array than with straight designs.^{3,5} Electrode folding may cause damage to intracochlear structures leading to both acute and chronic intracochlear changes. Herein, we examine the histologic findings of human CI cases where folding of the implant electrode array is observed.

Materials and Methods

TB specimens from patients having undergone CI during life were evaluated. Inclusion criteria were: 1) insertion of the electrode through an extended round window approach and 2) similar etiology, duration, and magnitude of hearing loss between ears. “Folded” specimens exhibited folding of the intracochlear electrode array and “control” specimens exhibited electrode placement entirely within the ST without evidence of electrode folding.

‘SGN count’ was quantified as the difference in normalized SGN (% age-matched controls) between the implanted ear of interest and contralateral ear within each patient:⁶

continued on page 2



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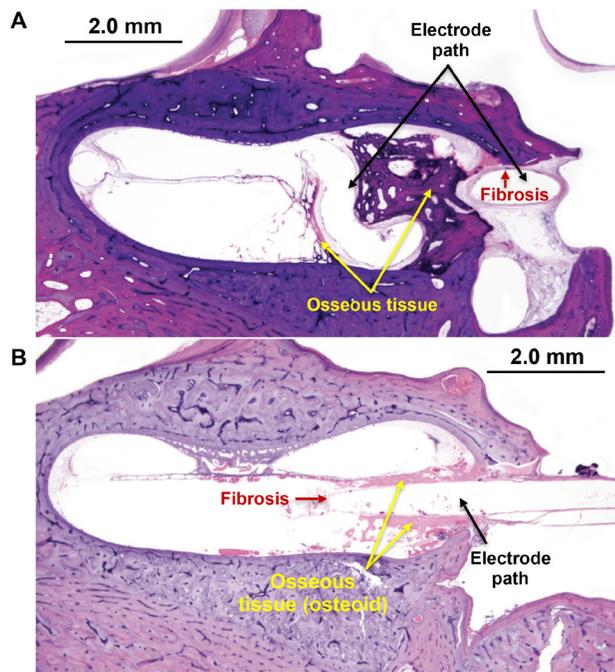


Figure 1: Histology of TB specimens. A: Example of hematoxylin and eosin (H&E) stained folded case showing an electrode (black arrows) entering the cochlea and folding over within the basal turn. Extensive new osseous tissue (yellow arrows) is seen within the cochlear lumen and fibrous tissue (red arrow) surrounds the electrode path. B: Example of H&E stained control case showing an electrode (black arrow) entering the ST of the basal turn. Minimal osseous tissue (yellow arrows) is observed within the cochlear lumen and a thin layer of fibrous tissue (red arrow) surrounds the electrode path.

Human otopathologic findings in cases of folded cochlear implant electrodes. Trakimas DR, Kozin ED, Ghanad I, Nadol JB Jr, Remenschneider AK. Otol Neurotol. 2018 Jul 13.

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p<0.05), which corresponds to the region of array folding (Figure 2). Little to no osseous tissue formation was observed within the basal turn of 4 of 5 control cases and intracochlear structures remained identifiable and relatively normal in appearance within this group (Figure 1B). The length of proximal array folding varied between the four folded cases (range: 2.8–6.5 mm) and was strongly correlated with the degree of ossification ($r=0.97$, $p<0.05$). We quantified the amount of fibrosis within the cochlea similarly to osseous tissue and found no difference in fibrosis between folded cases and controls (Figure 3).

Overall, interaural SGN counts were decreased in folded cases (-14.1 ± 13.4 percent), while controls showed no interaural SGN count differences (0.7 ± 5.2 percent) (Figure 4). Within segments of Rosenthal's canal, folded cases showed the greatest difference in SGN counts in Segment I and II (-21.6 ± 16.5 and -28.3 ± 17.0 percent, respectively), which correspond to the locations of folding of the electrode array in each case. In contrast, SGN counts in Segments I and II were similar or slightly higher for all control cases (7.3 ± 14.6 and 4.8 ± 4.9 percent). Additionally, within the folded group, the specimen with the greatest length of array folding showed the greatest SGN count difference in Segment I and II (-44.7 percent, -52.3 percent).

Both ossification and lower SGN counts were more pronounced in folded cases than controls. SGN counts in Segments I, II, and overall were negatively correlated with the amount of ossification within these areas ($r=-0.72$, $p<0.05$; $r=-0.71$, $p=0.05$; $r=-0.87$, $p<0.01$, respectively).

(SGN implanted ear of interest (%) – SGN contralateral ear (%) $\times 100\%$). 3-D reconstruction of the cochlea, as described by others, was used to quantify volumes of new fibrous or osseous tissue.⁴

Results

Four cases with folding of the electrode array within the basal turn and five controls with unilateral, normal electrode conformation were identified. All patients experienced progressive sensorineural hearing loss and the duration of hearing loss (3.8 ± 1.7 vs. 9.6 ± 5.3 yr, age at implantation (70 ± 7 vs. 73 ± 7 yr), and duration of implant use before death (8.3 ± 4.3 vs. 10.3 ± 2.2 yr) were comparable between the folded and control groups.

Throughout the ascending basal turn of the cochlea, folded cases qualitatively showed more osseous tissue compared to controls (Figure 1A).

Quantitatively, there was greater neo-ossification within Segment I of folded cases versus controls (29.2 ± 12.2 vs 6.6 ± 4.9 percent,

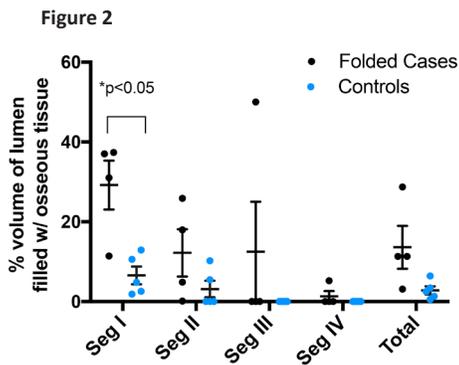


Figure 2: Intracochlear osseous tissue. Individual and mean values of percent volume of the cochlear lumen filled with osseous tissue for folded cases and controls within Segments I–IV. Black and blue symbols correspond to folded and control cases, respectively. Error bars represent standard error of the mean.

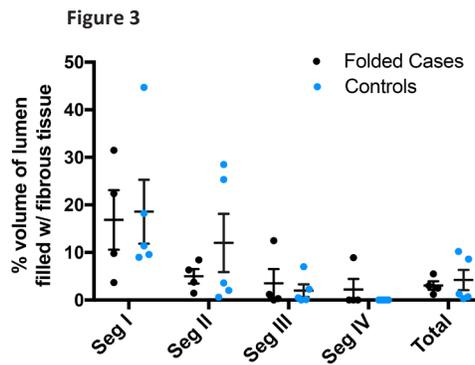


Figure 3: Intracochlear fibrous tissue. Individual and mean values of percent volume of the cochlear lumen filled with fibrous tissue for folded cases and controls within Segments I–IV. Black and blue symbols correspond to folded and control cases, respectively. Error bars represent standard error of the mean.

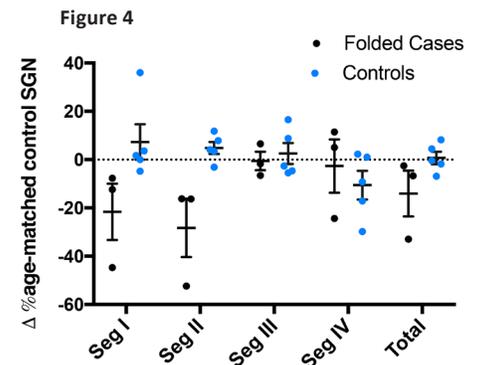


Figure 4: Interaural SGN counts. Individual and mean values of SGN counts reported as interaural differences in percent of age-matched control values (implanted ear of interest – contralateral ear) within each segment of Rosenthal’s canal. *SGN counts were significantly less than zero within Segment I, II, and overall in folded cases. Black and blue symbols correspond to folded and control cases, respectively. Error bars represent standard error of the mean.

Human otopathologic findings in cases of folded cochlear implant electrodes. Trakimas DR, Kozin ED, Ghanad I, Nadol JB Jr, Remenschneider AK. Otol Neurotol. 2018 Jul 13. Volume and issue number not available as at the moment of reuse the article was still in Publish Ahead of Print, 2018, Wolters Kluwer®. All Rights Reserved

Discussion

In this study, specimens with folding of the CI electrode showed significantly greater volumes of intracochlear osseous tissue than controls, which was most prominent in areas adjacent to sites of folding. In these same areas, folded cases showed lower SGN counts when compared to the contralateral ear, whereas controls showed stable interaural SGN populations. Interestingly, the degree of intracochlear fibrosis was similar between cases and controls. Additionally, our cases demonstrate a negative correlation between intracochlear ossification and local SGN counts.

Previous studies in human TBs have shown a positive association between gross intracochlear trauma and new intracochlear osseous tissue formation.⁴ We additionally demonstrate that folding of a CI electrode leads to local intracochlear osseous change that is dependent on the degree of electrode folding. While the effect of CI on SGN remains under debate, others have shown a local protective effect of CI on segments of the cochlea receiving stimulation.⁷ In line with these studies, we show that in the absence of electrode folding and with minimal intracochlear damage, stable populations of SGNs are observed. Therefore, loss of SGNs in our cases of electrode folding may result from acute trauma and chronic osseous deposition. Although unknown, this mechanism may be attributed to osseous occlusion of the inferior cochlear vein, which is proximal to the round window membrane and receives drainage from the SGN and ST through the posterior spiral vein.⁸

Our findings show that folding of CI electrodes causes significant acute intracochlear damage. Insertional damage from misplaced electrodes may stimulate immediate ossification, but chronic placement of a folded array also may exert pressure on intracochlear structures leading to a progressive osteitic reaction. If the latter is true, then withdrawal and reinsertion of a CI electrode in the proper configuration could prevent further degeneration, which is particularly relevant for precurved devices.⁹ These findings highlight

the need for proper, atraumatic initial insertion of CIs. Further analysis is necessary to determine if intracochlear degeneration may be prevented via immediate identification with intraoperative imaging⁹ and rapid correction of CI electrode placement. ●

This article is adapted from: Human otopathologic findings in cases of folded cochlear implant electrodes. Trakimas DR, Kozin ED, Ghanad I, Nadol JB Jr, Remenschneider AK. *Otol Neurotol*. 2018 Jul 13.

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Labyrinthine Concussion: Reexamination of a Historic Diagnosis Using Otopathology Techniques

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Head injury is a major cause of worldwide morbidity and death.¹ Although not commonly discussed in the trauma literature, head injury may also result in significant auditory dysfunction, such as hearing loss and tinnitus.^{2,3} Following a temporal bone (TB) fracture, hearing loss is commonly thought to be due to direct anatomic disruption of the middle ear or inner ear sensory neuroepithelium.^{4,5}

While hearing loss following TB fracture is well described, the epidemiology and pathophysiology of auditory dysfunction in the setting of head injury without TB fracture remains underinvestigated. In a recent review by Chen et al.⁶, the studies with the highest level of evidence reported hearing loss after head injury without associated TB fracture in 0.9 to 58 percent of the patients. When looking specifically at concussions in children, Thompson et al. suggested that the ability to understand speech in noise, particularly over extended periods of background noise, might be compromised even in the absence of hearing loss.⁷

Over the past century, various terms such as “labyrinthine concussion” and “inner ear concussion” have been used to describe sensorineural hearing loss (SNHL) following head trauma without TB fracture. There are several proposed mechanisms for traumatic auditory injury in the literature: 1) pathologic inner ear fluid wave⁸, 2) trauma to the cochleovestibular nerve⁹, 3) direct injury to membranous labyrinth⁸, 4) endolymphatic hydrops¹⁰, and/or 5) injury to the central auditory pathway.¹¹ A lack of data has made it difficult to determine the underlying mechanism by which head injury results in auditory pathology. Treatment options also remain largely nonexistent.

Taken together, while SNHL secondary to head injury without TB fracture is a recognized clinical phenomenon, the underlying pathophysiology remains unknown. We hypothesize that a unique set of changes occurs in the inner ear as a result of head injury that can be detected by otopathology techniques. Human otopathologic

Table 1. Study inclusion and exclusion criteria

Inclusion criteria

- 1) Individuals with a history of head injury without TB fracture

Exclusion criteria

- 1) History of noise exposure
- 2) Clinical, radiographic, or histologic TB fracture through the inner ear
- 3) Otologic surgery involving the middle and/or inner ear
- 4) Hearing loss prior to the head trauma or due to other otologic disorders (eg, chronic otitis media)
- 5) Meniere’s syndrome or sudden sensorineural hearing loss prior to head injury
- 6) Severe postmortem changes (eg, compression artifact or autolysis)

Table 2. Clinical History

Case/Side	Age (yr.)/ Sex	Type of trauma (Age, yr.)	HL at last audiogram (Age, yr.)
1/R	92/M	Concussions (NA, child)	Moderate (79)
1/L	92/M	Concussions (NA, child)	Profound (79)
2/L	71/F	Concussion (34)	Profound (71)
3/R	66/M	Fall (61)	Mild (63)
4/R	96/M	Fall (78)	Moderate (95)
5/L	72/M	MVA (57)	Moderate (69)

L: left; R: right; yr.: year; HL: hearing loss; NA: not applicable; M: male; F: female, MVA: motor vehicle accident

Modified from Ishai R, Knoll RM, Chen JX, et al. Otopathologic changes in the cochlea following head injury without temporal bone fracture. *Otolaryngology–Head and Neck Surgery*. March 2018.

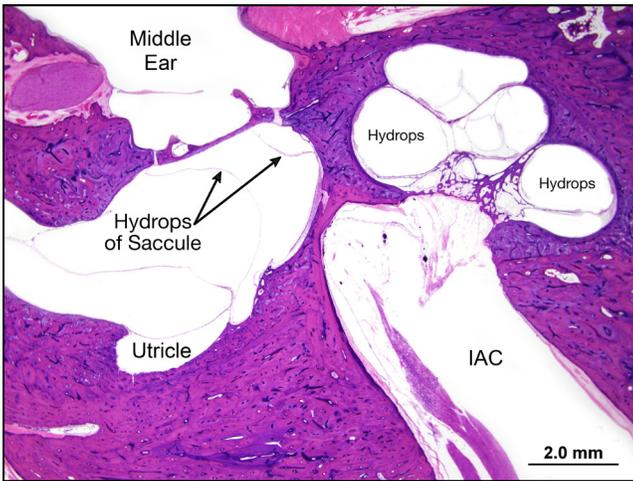


Figure 1: Low-power view of the left TB in case 1L. Midmodiolar section of the cochlea demonstrates severe cochlear and vestibular hydrops (sacculle/arrows). The stapes footplate does not show any abnormalities (arrow head). IAC: internal auditory canal

Modified from Ishai R, Knoll RM, Chen JX, et al. Otopathologic changes in the cochlea following head injury without temporal bone fracture. Otolaryngology–Head and Neck Surgery. March 2018.

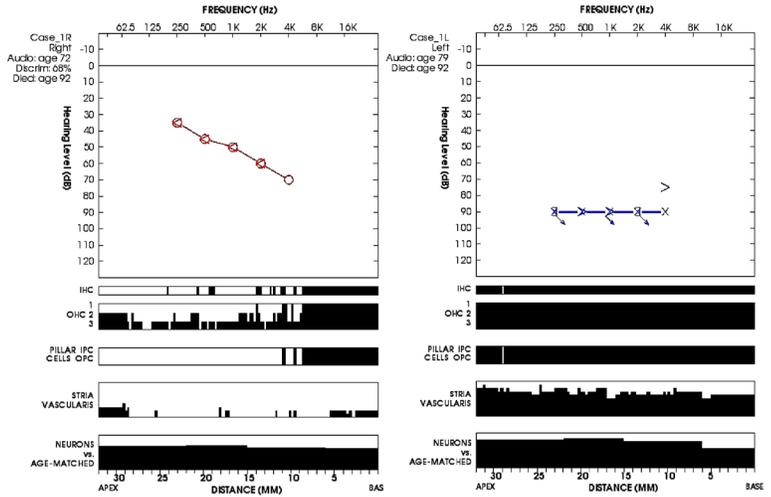


Figure 2: Audio-cytocholeogram of the TBs in case 1. Right ear (left panel): Loss of hair cells and pillar cells predominantly at the lower basal turn of the cochlea. Left ear (right panel): Severe loss of hair cells, pillar cells, stria vascularis, and spiral ganglion neurons. IHC: inner hair cell; IPC: inner pillar cell; OHC: outer hair cell; OPC: outer pillar cell

Modified from Ishai R, Knoll RM, Chen JX, et al. Otopathologic changes in the cochlea following head injury without temporal bone fracture. Otolaryngology–Head and Neck Surgery. March 2018.

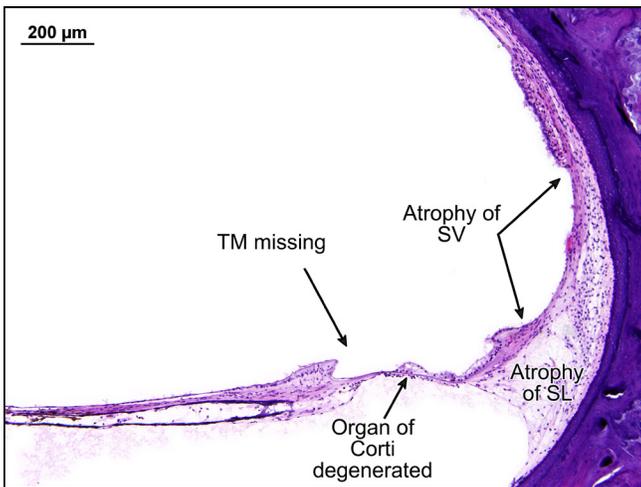


Figure 3: High-power view of the basal turn of the cochlea in case 1L. Photomicrograph shows severe endolymphatic hydrops, degeneration of the organ of Corti, missing tectorial membrane (TM), and severe atrophy of the stria vascularis (SV) and spiral ligament (SL).

Modified from Ishai R, Knoll RM, Chen JX, et al. Otopathologic changes in the cochlea following head injury without temporal bone fracture. Otolaryngology–Head and Neck Surgery. March 2018.

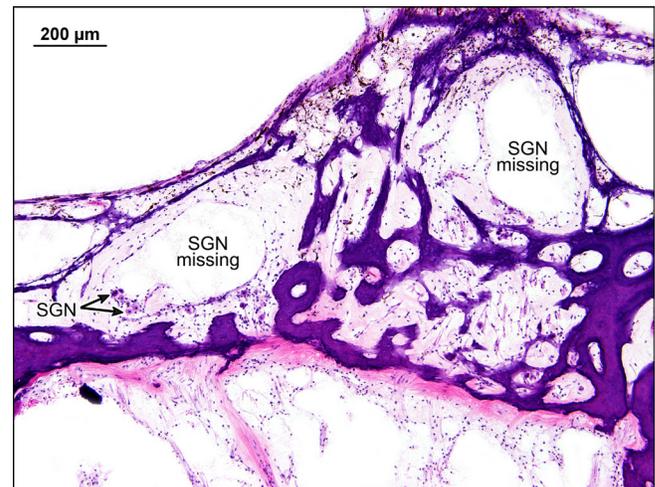


Figure 4: High-power view of the modiolus in case 1L. Photomicrograph shows that Rosenthal’s canal contained very few spiral ganglion neurons (SGNs) (arrows).

Modified from Ishai R, Knoll RM, Chen JX, et al. Otopathologic changes in the cochlea following head injury without temporal bone fracture. Otolaryngology–Head and Neck Surgery. March 2018.

studies may indicate potential future avenues of research for prevention and treatment algorithms in this clinical setting.

As a proof of concept study, the National Temporal Bone, Hearing and Balance Pathology Resource Registry was used to identify individuals with a history of head injury without TB fracture. Inclusion and exclusion criteria are summarized in Table 1. Inner ear anatomy of all TBs was evaluated by light microscopy, including condition of the stria vascularis, presence or absence of endolymphatic hydrops, and counts of spiral ganglion neurons (SGNs), which were compared to historical age-matched controls.¹²

Six TBs from five patients (4 men and 1 woman) met initial inclusion and exclusion criteria (Table 2). All subjects had an available post-head injury audiogram that demonstrated mild to profound SNHL. As a representative individual, Case 1 had a history of multiple concussions of unknown etiology during childhood. The patient subsequently experienced hearing loss that was bilateral and progressive, and experienced total loss of hearing by age 17. The patient died at age 92. Postmortem computed tomography of TBs were reviewed, and there was no evidence of TB fracture or other middle and inner ear pathology, such as enlarged vestibular aqueduct. When both TBs were

continued on page 6

analyzed, the left TB had presence of vestibulocochlear hydrops (Figure 1). The left TB demonstrated severe loss of inner and outer hair cells and pillar cells along the length of the cochlea, while the right TB showed moderate to severe loss at the basal turn of the cochlea and mild to moderate loss at the middle turn of the cochlea to the apex (Figure 2). Severe atrophy of the stria vascularis and spiral ligament were found throughout the cochlea in the left TB, while the right TB showed only mild degeneration of the stria vascularis in the basal and apical turns of the cochlea (Figure 3). Furthermore, both sides demonstrated decreased SGN populations (21 and 33 percent of historical age-matched controls, respectively) (Figure 4).

Additional review of four other patients with unilateral TBs available also demonstrated inner ear changes after head trauma. All four cases showed decreased total SGN populations with an average of 57 percent of historical age-matched controls (range, 21–79 percent of historical age-matched controls). In addition, there was loss of HC and mild to moderate degeneration of the stria vascularis. Cochlear endolymphatic hydrops was found in 50 percent (n=2) of cases.

The histopathologic changes in the present study are thought provoking and may partially explain auditory symptomatology that may develop following head trauma. Limited related human^{10,13,14} and animal studies^{14,15} have shown some similarity in the findings of our study. Lindsay and Zajtchuk¹³ reported a case in which both TBs demonstrated degeneration of organ of Corti, stria vascularis, and spiral ligament predominantly in the middle and apical turns of the cochlea as well as reduced SGN populations. Paparella et al.¹⁰ described a case that developed Meniere's syndrome following a head trauma, in which otopathologic analysis showed vestibulocochlear hydrops, HC and SGN loss in the basal turn of the cochlea, and an eosinophilic precipitate in the endolymphatic duct. In a cat model of head trauma, Schuknecht and Davison¹⁵ found pathologic changes located within the basal turn of the cochlea, with damage of the outer hair cells. A feline model of head trauma developed by Wittmaack¹⁴ demonstrated severe degeneration of the membranous labyrinth and SGNs in the middle turn, with mild to moderate changes in the apical and basal turns of the cochlea.

There are limitations to otopathologic evaluation of the auditory periphery following head injury. The time of death is often years after the insult and additional unknown factors, including undocumented noise exposure, may contribute to the observed pathology. Although the study has distinct limitations, review of human TB may augment our understanding of auditory symptoms following head injury. Future prospective studies in animal models may elucidate whether or not these otopathologic findings are directly attributable to trauma. ●

Portions of this manuscript have been accepted for publication in *Otolaryngology–Head and Neck Surgery*.

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Otopathology Mini-Travel Fellowship Program

The NIDCD National Temporal Bone Registry's mini-travel fellowships provide funds for research technicians and young investigators to visit a temporal bone laboratory for a brief educational visit, lasting approximately one week. The emphasis is on the training of research assistants, technicians, and junior faculty.

These fellowships are available to:

- U.S. hospital departments who aspire to start a new temporal bone laboratory
- Inactive U.S. temporal bone laboratories who wish to reactivate their collections
- Active U.S. temporal bone laboratories who wish to learn new research techniques

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- Letter of support from temporal bone laboratory director or department chairman
- Letter from the host temporal bone laboratory, indicating willingness to receive the traveling fellow

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