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CONTENTS

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 to continue and expand upon the former National Temporal Bone Banks Program. The Registry promotes research on hearing and balance disorders and serves as a resource for the public and scientific communities about research on the pathology of the human auditory and vestibular systems.

Three-dimensional length measurements of the narrow bony channels of the vestibular nerves: Clinical implications in vestibular neuritis

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A sudden onset persistent vertigo is called acute vestibular syndrome. Although it may be caused by life-threatening conditions like a cerebellar stroke, acute vestibular syndrome is more frequently caused by a sudden loss of peripheral vestibular function called vestibular neuritis (VN)[1]. Current diagnostic criteria include experiencing spontaneous vertigo and accompanying nystagmus lasting longer than 24 hours without central neurological signs [2-4].

Historically, clinicians have relied on caloric testing to identify vestibular hypofunction in patients with VN. But, with newer tests like the head impulse test, it has become clear that VN most frequently involves semicircular canals innervated by the superior vestibular nerve, sometimes all three semicircular canals and only rarely the posterior canal alone[2, 5, 6]. Vestibular neuritis involving the posterior canal is also frequently associated with hearing loss[7-9]. There have been efforts to explain why the superior nerve is more often involved. Histological measurements in both axial and coronal sections showed that the superior vestibular nerve travels longer through relatively narrower passages than the singular nerve, leading to the hypothesis that the superior division of the vestibular nerve is more susceptible to entrapment and that the nerve

continued on page 2

THE REGISTRY

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and its sensory epithelia are more susceptible to ischemia[10, 11]. Recent studies in patients with VN have included functional measurements of all sensory epithelia in the affected ear and have separately assessed responses for all semicircular canals and maculae[9, 12]. These studies found patchy involvement of the vestibular end-organs in cases of VN. In light of these publications, we aimed to revisit the theory of nerve entrapment and to measure the length of the narrow part of the bony channel (in which the nerve occupies more than 80 percent of the space), taking into account the three-dimensional (3D) trajectory of the nerves. We asked if the lengths of the narrow parts of the bony channels could provide a consistent explanation for the differential involvement of the vestibular nerves that we see clinically. This could be supported if the frequency of nerve involvement corresponded to the length of the narrow channels in which these nerves become entrapped.

Forty-six normal ears from the temporal bone library of the Otopathology Laboratory at the Massachusetts Eye and Ear Infirmary (NIDCD National Temporal Bone Registry) were sectioned in the axial plane and examined. Slice thickness was 20 µm and every tenth preparation was stained and mounted. Even by only mounting every tenth section, it was possible to render 3D reconstructions of the inner ear (Figure 1), in which the nerves and the borders of the fluid filled spaces are recognizable. To inspect the 3D paths of the nerves in greater resolution, we also reconstructed two ears for which every section was mounted and stained (Figure 2). The 3D reconstructions show that, although the superior vestibular nerve and the dual innervation of the saccule run approximate to the horizontal plane, the inferior vestibular nerve runs obliquely at a 50-to-60 degree angle with respect to the horizontal plane. Therefore, we simply applied the Pythagorean theorem to measure the inferior vestibular nerve's length, taking into account the vertical distance between slices, and calculating the hypotenuse as the length of the inferior vestibular nerve.

The regions of interest in the preparations were captured using CAPTAVISION V 3.9 + Olympus Microscope BX51 (Objective 1.25x) + Excelis Camera by Accu-Scope. Images were manually aligned using FIJI ImageJ (National Institutes of Health, Bethesda, MD, USA; *https://imagej.nih.gov/ij*) and measurements were made using the measurement tool of the software, enabling us to measure both the area and the length of curved-fitted lines. The 3D images for visualization were reconstructed using Slicer 4.10.2 (*http://www.slicer.org*).

Our results are shown in Table 1. The branch of the superior vestibular nerve to the saccule (i.e. Voit's nerve) is thin and was not observed in 14 cases (30 percent). Due to missing data and dual innervation of the saccule, the length of the bony channels for the saccule was excluded from further analysis. In 10 cases, the medial part of the singular nerve consisted of two branches in two separate bony channels (see also Figure 3).

Narrow part of the bony channel to the...->	Lateral crista	Utricular macula	Upper part of saccular macula (i.e. Voit´s nerve)	Lower part of saccular macula	Posterior crista
Average length in mm (±SD)	2.94 (±0.54) ***	1.45 (±0.36) ***	1.14(±0.48)	0.52 (±0.37)	1.95 (±0.58) ***

Table 1. Results of measurements of the narrow parts of the bony channels. (***= sign. difference between the three groups)

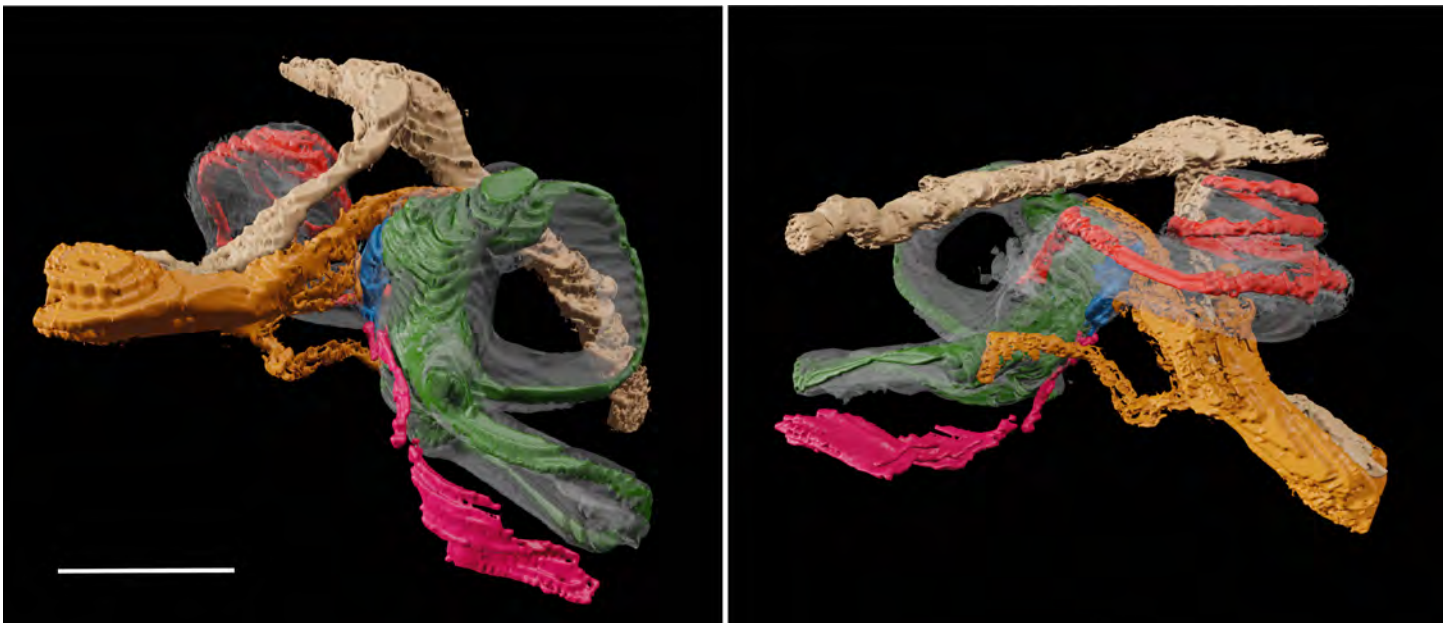

Figure 1. Raw, unmodified three-dimensional reconstructions of a right inner ear without vestibular pathology with every tenth axial section mounted and stained. The superior semicircular canal is missing. Left panel: superior view, right panel: inferior view. White: facial nerve; yellow vestibular nerves; green: utricle and semicircular canals; blue: saccule; red: cochlear duct; lilac: endolymphatic sac; perilymphatic space: semitransparent, grey. Calibration bar (white): 5 mm

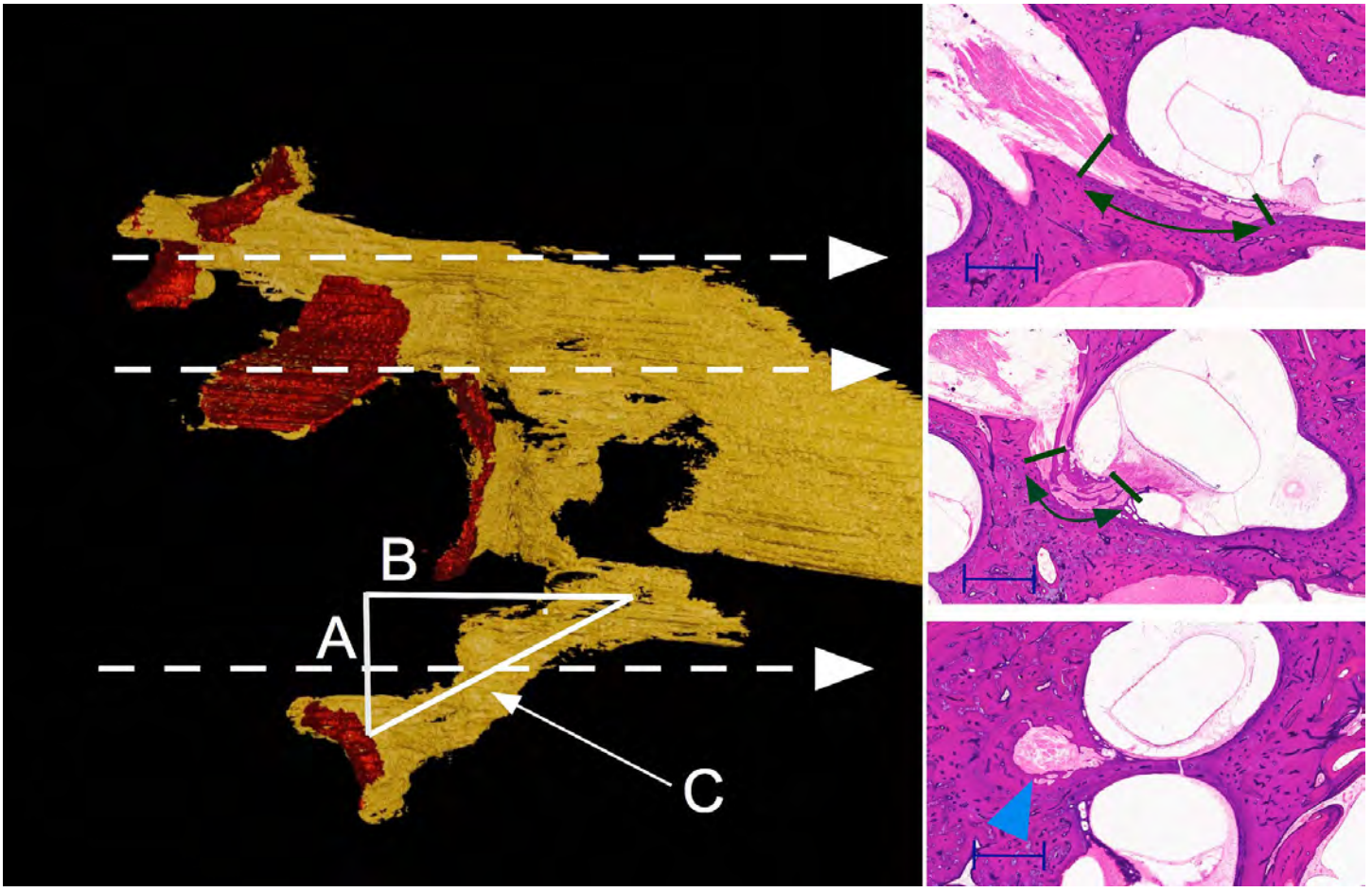

Figure 2. Left panel: High-resolution 3D reconstruction of the vestibular nerve branches viewed from posterior (based on every section stained and mounted, red: sensory epithelia). Upper dashed arrow: section in the region of the lateral ampulla and nerve. Middle dashed arrow: section in the region of the utricular nerve. Lower arrow: section in the region of the singular nerve. The triangle demonstrates the measurement principle. **'A':** vertical component= (number of sections) x (slice thickness), **'B':** horizontal distance, **'C':** calculated length of narrow part of the bony channel: (C) = $\sqrt{A^2+ B^2}$. Right panels: the curved arrows show the length of the narrow part of the bony channel. Blue arrowhead: in case of the inferior nerve one particular horizontal section only shows a cross section of the nerve. Calibration bars: 1 mm

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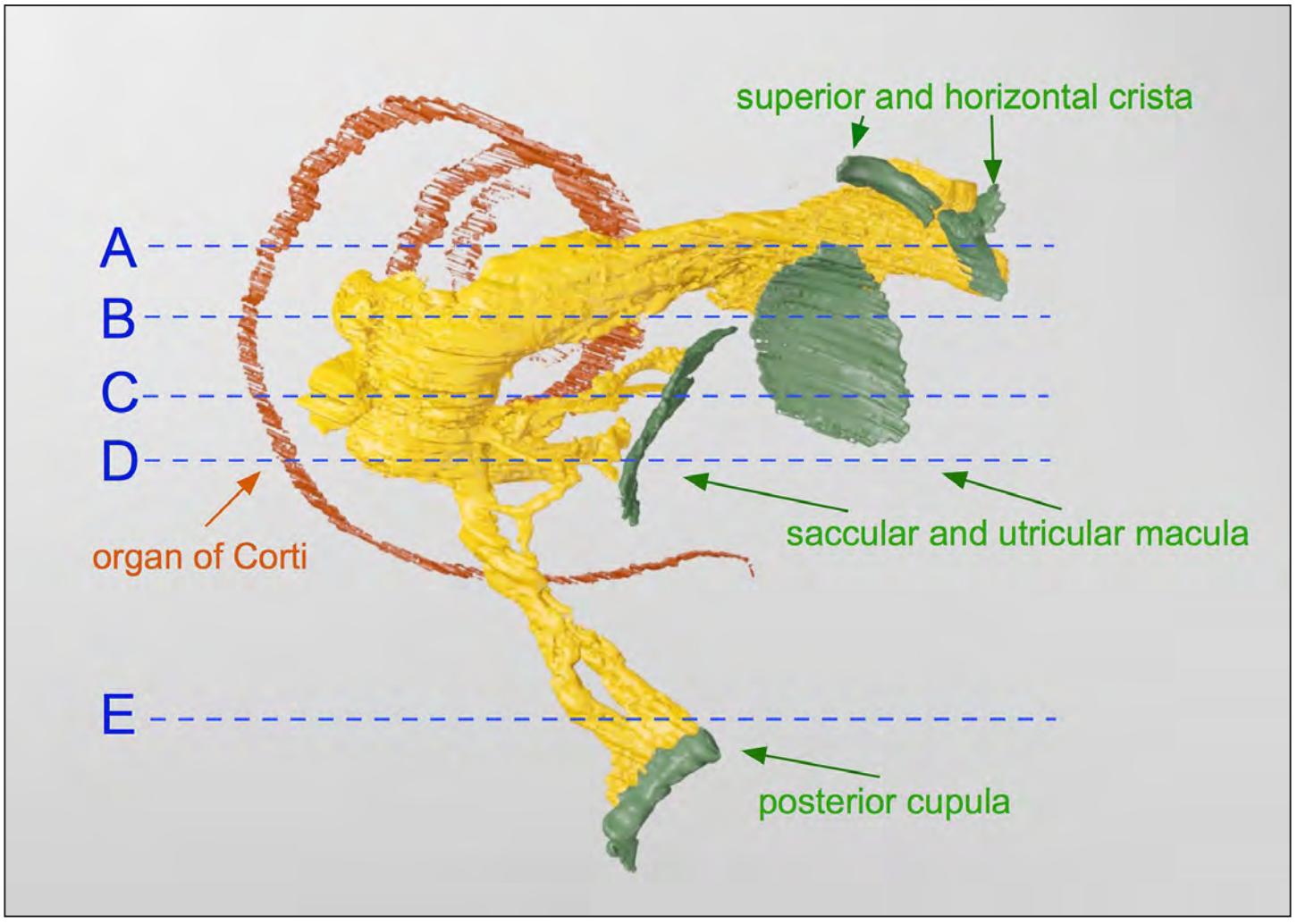

Figure 3. The bony channels for the following anatomical structures were measured: **A:** The nerve of the lateral semicircular canal. **B:** Utricular nerve; **C:** The upper branch of the saccular nerve (usually a branch of the superior vestibular nerve, i.e. Voit's nerve) **D:** The lower branch of the saccular nerve innervating the saccule (branch of the inferior vestibular nerve); **E:** The nerve of the posterior semicircular canal (i.e. the singular nerve).

Since the more caudal branch was consistently thicker and longer, the bony channel of this caudal branch was used to calculate length for these 10 cases. Using repeated measures ANOVA with a Bonferroni correction, there was a group difference for length of the narrow bony channel ($F=138.2$, $p<0.0001$). Pairwise comparisons indicated different lengths among each of the groups [lateral semicircular canals (SCC), utricle, and singular nerve ($p<0.0001$)]. The nerve to the lateral SCC traversed the longest narrow bony channel, followed by the singular nerve and then the utricular nerve ($p<0.0001$).

As reported in other publications,[10, 11,] the narrow portion through which the superior vestibular nerve travels to the lateral semicircular canal is the longest and that of the singular nerve is shorter, on average. We found, however, that the length of the narrow bony channel for the singular nerve is much longer than previously reported and exceeds the length of the utricular nerve. Differences in these length ratios may be related to the use of a 3D measurement method for the singular nerve channel. Since we now know that in vestibular neuritis the utricle is almost as frequently involved as the superior and lateral SCCs and the structures innervated by the inferior vestibular nerve are less frequently affected[12], our results show that the length of the narrow bony channels may not be the sole cause of differential involvement of the nerves in vestibular neuritis. One explanation for the discrepancy between clinical studies and the lengths of the bony channels may be related to bias in the clinical literature in which patients with hearing loss often are excluded from studies of VN, and those with hearing loss tend to be the cases that also have posterior canal impairment[9, 12]. However, among patients with hearing loss and vertigo, the utricle appears to be more frequently

affected than the posterior SCC, despite the utricular nerve traveling through a shorter portion of narrow bony channel.

An alternative hypothesis is that the cascade of the anastomoses between the nerves in the internal auditory canal may act as conduits to spread reactivated herpes viruses[13], since it has been shown that the most frequent connections exist between the facial nerve and the superior vestibular nerve[14, 15]. The sensory part of the facial nerve, the nervus intermedius, also generates anastomoses more frequently to the superior vestibular nerve than to the inferior vestibular nerve[16]. Perhaps these more frequent anastomoses between the facial and superior vestibular nerves facilitate infection and provide an additional risk factor for superior vestibular nerve involvement. ●

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Long-term histopathological findings of cochlear implant electrode translocation in humans

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Over the past decade, the indications of cochlear implantation (CI) have broadened to include profound hearing loss involving only high frequencies and single-sided deafness.[1-3] As a consequence of preserving residual hearing, there has been an increased interest in proper and atraumatic electrode placement techniques that result in better preservation of cochlear structures during CI procedures, even on those destined for pure electric stimulation.[4-6] Among the possible types of electrode insertional injury, electrode scalar translocation is often associated with severe intracochlear trauma,[7-13] and is reported to negatively affect speech perception outcomes and the residual hearing of CI recipients.[14] Additionally, preservation of residual hearing seems to be greater when the electrodes are positioned entirely within the scala tympani (ST).[15,16]

While potential mechanisms and acute consequences of insertional trauma associated with electrode translocation have been investigated in the past decade, few studies have reported long-term histopathological intracochlear changes [e.g., effects of translocation on spiral ganglion neuron (SGN) population], especially with pre-curved designs of electrode arrays.[9,10] Herein, this study aimed to assess the histopathology of human temporal bones (TBs) with CI electrode scalar translocation compared to non-translocated specimens.

To approach this aim, TB specimens from the Massachusetts Eye and Ear Otopathology Laboratory collection with history of CI and histopathological evidence of scalar translocation were identified. Specimens with a history of CI and an electrode placed entirely within the ST without evidence of scalar translocation or gross intracochlear damage served as controls. TBs from individuals with multiple CI procedures, congenital malformations of the inner ear, and severe postmortem changes or artifact were excluded. Medical records were reviewed to evaluate demographic features, details of the implantation procedure and postoperative audiometric performances. Under light microscopy, the cochlear duct and Rosenthal's canal were reconstructed by 2-dimensional (2D) methods as previously described,[17] and used to determine depth of electrode insertion, the length and angle of insertion where translocation occurred and SGN counts within four cochlear segments. These four sections were specimens with translocation from the ST to the scala vestibuli (SV), an available contralateral non-implanted TB, comparable pre-operative interaural speech detection (<15dB difference) and word recognition scores (WRS) (<15 percent difference) and similar etiology and duration of sensorineural hearing loss (SNHL) between ears. Absolute counts within specimens were normalized as a percent of age-matched controls,[17,18] and then compared between ears [SGN implanted ear of interest (percent) - SGN contralateral ear (percent)].

Image/figure courtesy of authors.

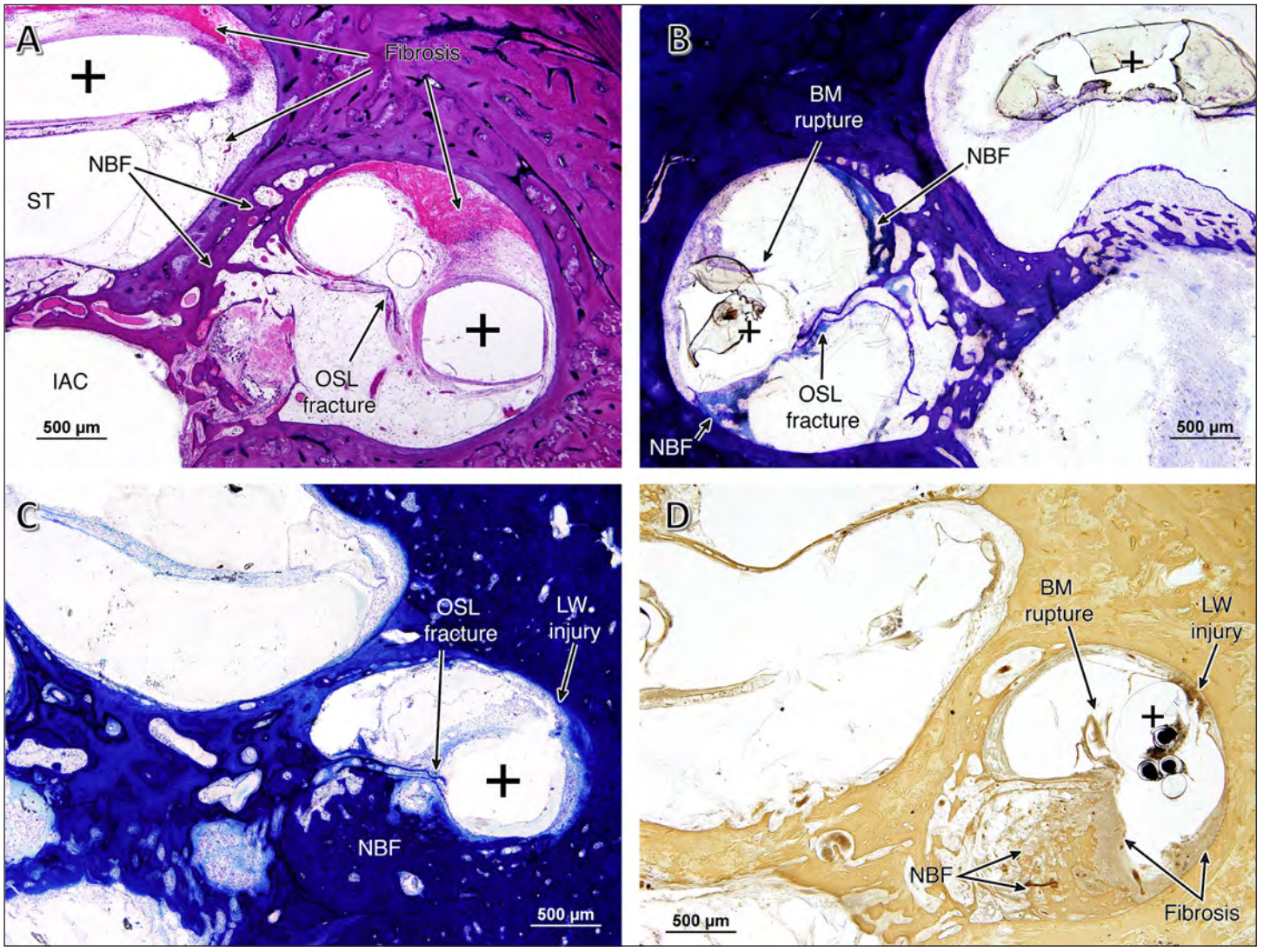

Figure 1. Photomicrographs of H&E (A), osmium (D) and toluidine blue (B and C) stained sections of human temporal bones with electrode scalar translocation. Examples of injuries caused at the translocation site are shown with fracture of the OSL (A, B and C), basilar membrane disruption (B and D) and dissection of the electrode into the LW (C and D).

Nineteen specimens with electrode translocation were identified, as were eight controls. All specimens were from patients who experienced progressive SNHL and were post-lingually deafened. Duration of deafness and the number of years of CI use were comparable between translocated and control groups (14 ± 16 vs. 7 ± 5 years; p = .531, and 7 ± 7 vs. 4 ± 3 years; p = .288, respectively). The age at implantation was greater for the control group (63.7 ± 11 vs. 74.7 ± 7 years; p = .016). A similar proportion of patients were implanted with perimodiolar (PM) and lateral wall (LW) electrode arrays between translocated (n = 5, 26 percent and n = 14, 74 percent, respectively) and control (n = 3, 37 percent and n = 5, 63 percent, respectively) groups. Among the translocated cases, 15 (80 percent) electrodes were placed with a cochleostomy (COCH) approach, and the remaining by an extended RW approach. Extended round window (RW) approach (71 percent) was more commonly used in the control group compared to COCH (29 percent). There was no significant difference in surgical approach between groups (p = .072).

Four out of the 19 translocated cases had an SV insertion, and three translocated cases had an electrode insertion into the ST that posteriorly shifted into the scala media. The average depth of insertion was comparable between translocated and control groups (20 ± 3 mm vs. 18 ± 4 mm; p = .270). Almost all translocated cases (18/19) presented moderate fibrosis and new bone formation (NBF) in the basal region of the cochlea, extending to the translocation point and/or throughout the electrode track in 42 percent. Two TBs presented NBF within the Rosenthal's canal, and fibrous tissue extending beyond the

continued on page 8

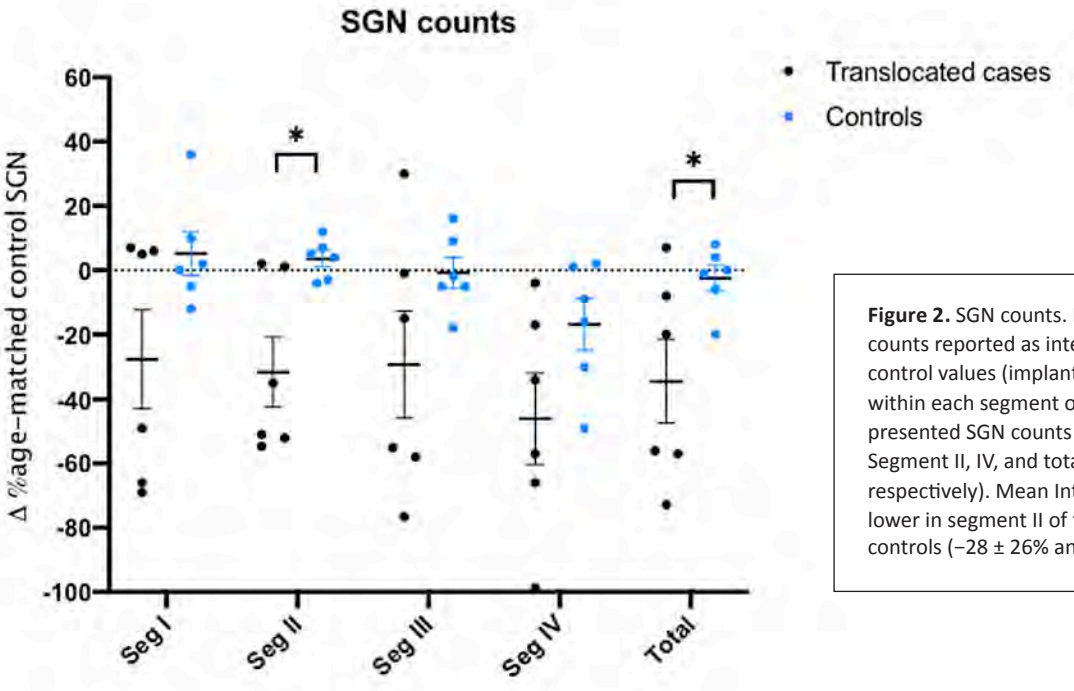

Figure 2. SGN counts. Individual and mean values of SGN counts reported as interaural differences in % of age-matched control values (implanted ear of interest – contralateral ear) within each segment of Rosenthal's canal. Translocated cases presented SGN counts significantly less than zero within Segment II, IV, and total (p = .031, p = .008 and p = .021, respectively). Mean Interaural SGN count was significantly lower in segment II of translocated cases as compared to controls (−28 ± 26% and 3 ± 6%, respectively; p = .019).

Image/figure courtesy of authors.

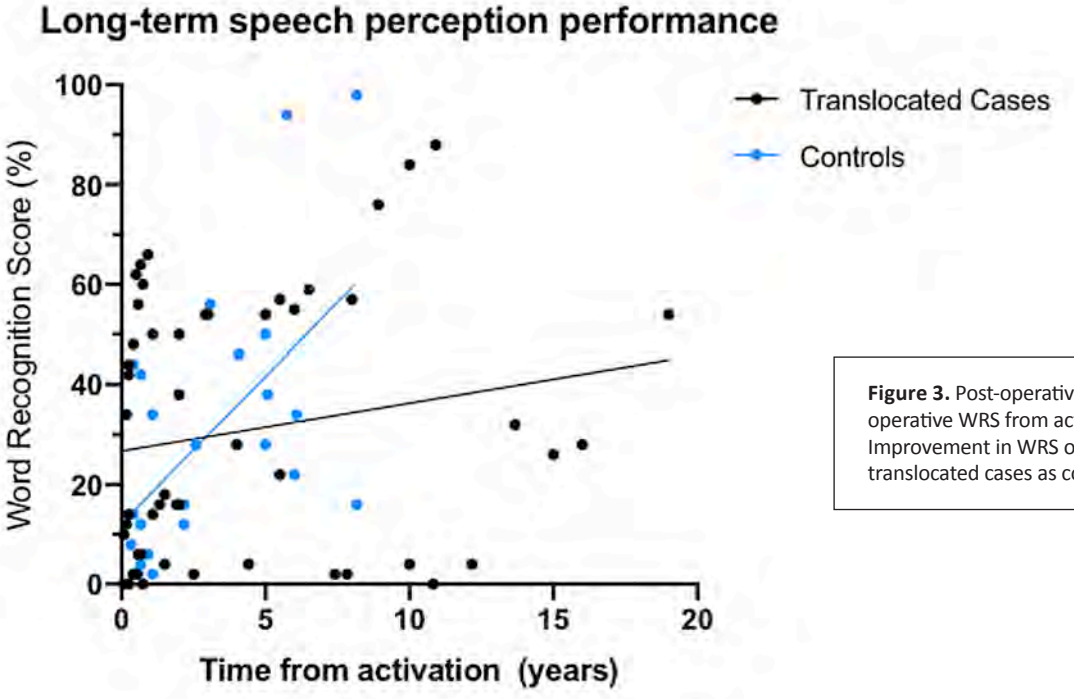

Figure 3. Post-operative hearing outcomes. Trajectories of post-operative WRS from activation (0) until last testing available. Improvement in WRS overtime was slower and decreased for translocated cases as compared to controls (p = .021).

Image/figure courtesy of authors.

tip of the electrode was found in three cases. Minimal NBF and fibrosis were found in the control group.

The most common site of translocation was the ascending limb of the basal turn (n = 14 TBs). The most common type of injury at the translocation site was disruption of the basilar membrane (79 percent, n = 15 TBs), followed by LW damage (63 percent, n = 12 TBs) and fracture of the OSL (47 percent, n = 9 TBS; Figure 1). The average length of electrode insertion at the translocation site from the entry point was 8 mm (SD ± 3 mm, range of 3 - 16 mm), without a significant difference between LW and PM electrodes (p = .483). The average angle of insertion at the point of translocation from the RW was 159º (SD ± 79º, range of 50º -370º). Straight LW electrodes tended to occur at an angle of 156º (SD ±87, range of 50º-370º), whereas PM electrodes were more commonly noted at 166º (SD ± 64º, range of 130º - 280º) from the RW with no significant difference (p = .893).

Interaural SGN counts were lower in the implanted ear of translocated cases within all cochlear segments compared to controls, with a significant difference for cochlear segment II and total SGN counts (p = .019 and p = .029, respectively; Figure 2). Although final post-operative hearing outcomes were similar between groups, translocated cases had slower rates of improvement in WRS (p = .021; Figure 3). Analysis of trajectories for those with post-operative WRS available from early follow-up (≤12 months) to late follow-up (≥ 24 months) showed comparable mean improvement in WRS over time between translocated and control cases (10.5 percent ± 24.8 percent and 11 percent ± 10.5 percent, respectively; p = .972). No correlations were found between CI performances and duration of deafness (r = -.463, p = .082), age at surgery (r = -.460, p = .084), depth of electrode insertion (r = .490, p = .063) or total absolute SGN counts (r = 040, p = .889).

This study is among the first to investigate long-term histopathological findings associated with scalar translocation in humans that includes recent PM electrode designs and a control group for comparison. Our study helps to better understand how electrode-array translocation may result in permanent intracochlear damage (e.g., extensive fibroosseous formation and neuronal degeneration), which appears to affect the rate of improvement in WRS overtime as compared to atraumatic electrode insertions and ultimately prevent successful audiometric performance.

Recent studies have focused on identification of potential factors that may influence the scalar electrode position, such as surgical approach, electrode design and electrode-array insertion depth.[19-22] While the development of more sophisticated imaging techniques and processing have helped in determining the array position within the cochlea,[12,23] irreversible micro intracochlear injuries such as isolated LW or hair cell damage may be missed and result in suboptimal auditory performance.[9] Therefore, it is imperative to develop more comprehensive methods for intraoperatively identifying and minimizing cochlear trauma during electrode insertion. ●

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Email Csilla Haburcakova, PhD, Registry Coordinator, at Mass Eye and Ear ***csilla_haburcakova@meei.harvard.edu***.

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. The visit lasts approximately one week and emphasizes the training of research assistants, technicians and junior faculty.

These fellowships are available to:

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

Up to two fellowship awards will be made each year ($1,000 per fellowship). The funds may be used to defray travel and lodging expenses. Applications will be decided on merit.

Interested applicants should submit the following:

- An outline of the educational or training aspect of the proposed fellowship (1-2 pages)
- Curriculum vitae
- Letter of support from a temporal bone laboratory director or department chairman
- Letter from the host temporal bone laboratory indicating willingness to receive the traveling fellow

Applications should be submitted to:

Felipe Santos, MD
NIDCD Temporal Bone Registry
Massachusetts Eye and Ear
243 Charles Street, Boston, MA 02114
felipe_santos@meei.harvard.edu

NIDCD National Temporal Bone, Hearing and Balance Pathology Resource Registry
Massachusetts Eye and Ear
243 Charles Street
Boston, MA 02114-3096

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Free Brochures for Your Office or Clinic about Temporal Bone Research and Donation

The Gift of Hearing and Balance: Learning About Temporal Bone Donation is a 16-page, full-color booklet that describes in detail the benefits of temporal bone research. It also answers commonly asked questions regarding the temporal bone donation process. *Dimensions: 7"x10"*

If you would like to display this brochure, please complete the form below and return it to *The Registry* by mail or fax. The brochures will be sent to you **free of charge**. Please circle the amount requested for each brochure or write in the amount if not listed.

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Name: _____

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Mail or fax this form to *The Registry* at: NIDCD National Temporal Bone, Hearing and Balance Pathology Resource Registry
Massachusetts Eye and Ear, 243 Charles Street, Boston, MA 02114
Toll-free phone: (800) 822-1327, Fax: (617) 573-3838
Email: tbregistry@meei.harvard.edu