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Journal

World Journal of Otorhinolaryngology-Head and Neck Surgery, 3(4)

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Publication Date

2017-12-01

DOI

10.1016/j.wjorl.2017.12.008

Peer reviewed

World Journal of Otorhinolaryngology-Head and Neck Surgery (2017) 3, 211–213

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.keaipublishing.com/WJOHNS; www.wjent.org

Research Paper

Cochlear implant histopathology

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Received 1 November 2017; accepted 12 December 2017
Available online 6 March 2018

KEYWORDS
Cochlear implant

Abstract The microscopic examination of fifty-five serially sectioned implanted temporal bones has provided insight into what is being stimulated; and the changes that are the result of the insertion and presence of the implant. The ganglion cell bodies (neurons) are structures being stimulated (two laboratories have reported an inverse relationship of the number of neurons and performance). Insertion through the round window, verses a cochleostomy, produces the least fibrosis and new bone. Fibrosis and new bone do not affect the implant function unless they form in the scala vestibuli in the region of the ductus reuniens, and, block it; and produce cochlear hydrops resulting in a delayed low tone loss of hearing in hybrid implants. Animal models cannot be applied to humans because of the difference in size and myelination of the neurons.

The histopathology of fifty-five human temporal bones, with various types of cochlear implants, were analyzed to determine how much initial and late damage to residual structures occurred, what structures were stimulated, and how many surviving elements were necessary for function. The temporal bones used in this study were from deaf or near deaf individuals who had the foresight to pledge their ears for postmortem study so that scientists might better understand how the implants worked and possibly improve their function. Each individual; signed a pledge willing their inner ears, hearing nerves, and related structures for scientific evaluation. The temporal bones were removed in an autopsy room and placed in 10% buffered formaldehyde, as soon as possible, preferably; after immediate embalming, to avoid postmortem degeneration. Decalcification and then embedding in celloidin require several months in order

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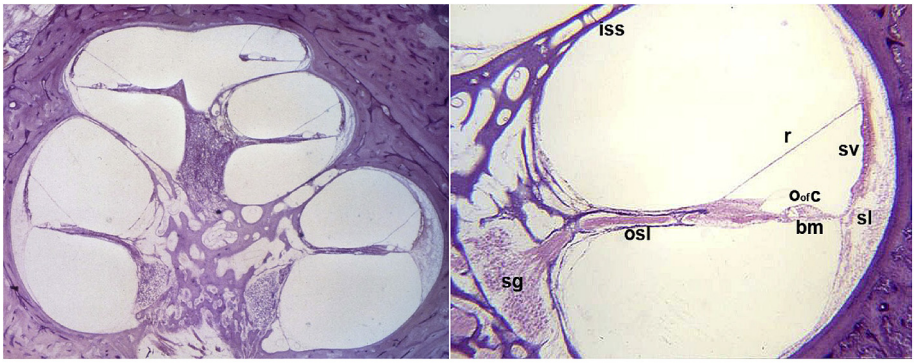
Peer review under responsibility of Chinese Medical Association.

Production and Hosting by Elsevier on behalf of KeAi

https://doi.org/10.1016/j.wjorl.2017.12.008



A B

Figure 1 A. Normal cochlea. B. Labelled anterior basal segment sg: spiral ganglion in Rosenthal's canal; iss: interscalar septum; osl: osseous spiral lamina; r: Reissner's membrane; o $_{of}$ c: organ of Corti; bm: basilar membrane; sv: stria vascularis; sl: spiral ligament (H&E ×20 & ×63).

to make sure of complete penetration of all tissues. The celloidin block is cut-down to 3 cm × 4 cm to fit into the sliding microtome that cuts them into 20-micron sections. Every tenth section was stained with hematoxylin, and eosin (H&E) and mounted on a numbered glass slide; the other nine, of each ten, are stored in alcohol for use in other analysis such as immunohistochemistry and electron microscopy. The patients' clinical records, including audiometric studies, and a quantitative neuron count, are stored in the area near the prepared bones. Clinical and histopathological findings are entered into a database that facilitates the retrieval of various histopathological findings alone or in combination. Images of significant observations are kept in a separate database.

Results

The insertion of a cochlear implant electrode through the round window, verses a cochleostomy, causes the least reaction within the cochlea shown in Fig. 1. Fig. 2 shows peri- and endolymphatic spaces inbones with a cochleostomy contained fibrous tissue that is localized or widely

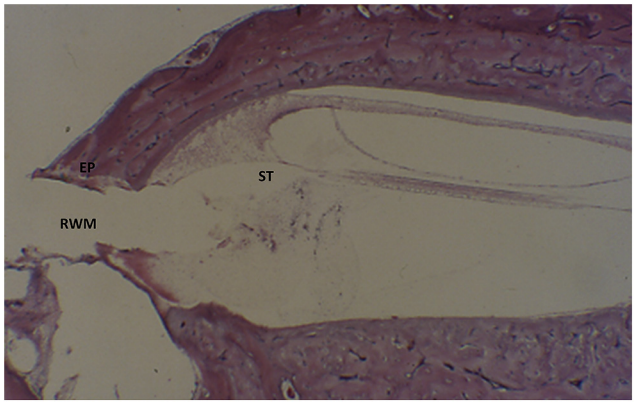


Figure 2 Round window membrane (RWM) in cochlear implant case without a cochleostomy. Electrode path (EP) is directly into the lower basal scala tympani through the round window membrane. The operculum was drilled off (H&E ×200).

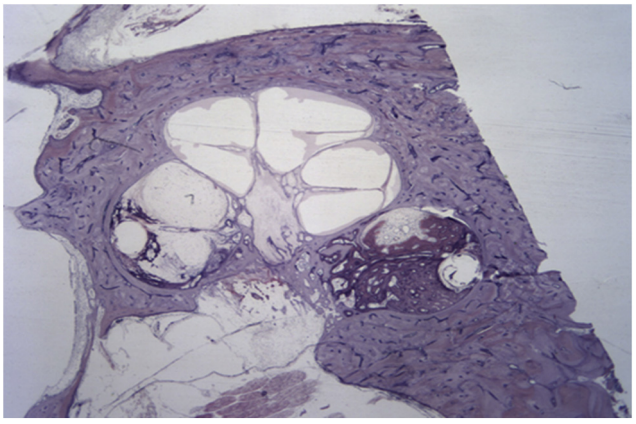


Figure 3 Midmodiolar section of a cochlea that had a cochleostomy insertion of an electrode. The electrode path is visible as two round holes in the ectopic bone. In spite of the extensive fibrosis and new bone, performance was good (H&E ×10).

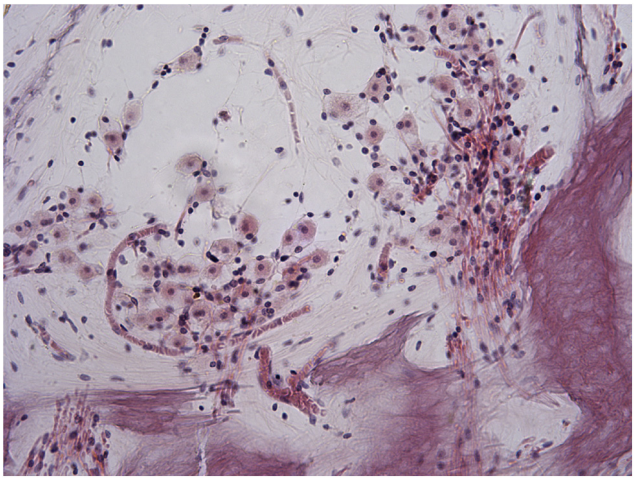


Figure 4 Unipolar neurons in a woman who successfully wore an implant until her death at age 76. Note the axons extending into the modiolar bone below but no afferent neurites from above (H&E ×200).

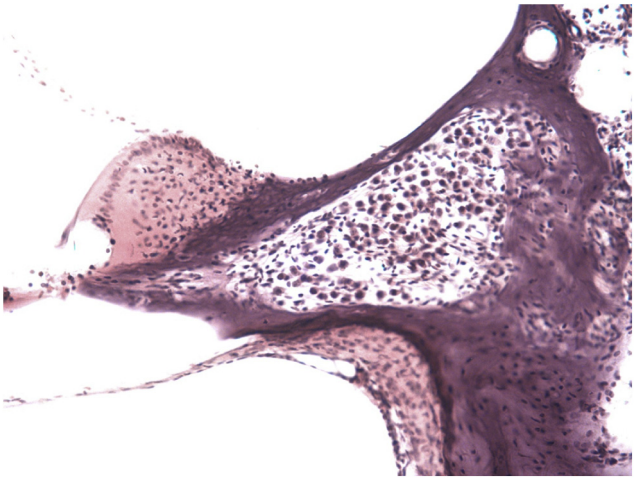


Figure 5 Image of segment of a mouse spiral ganglion, taken at the same magnification as in Fig. 4, that illustrates why animal models are not good predictors of human response. Human neurons are encased in a single layer of myelin supplied by satellite cells whereas the mouse spiral ganglia neurons are covered by multiple layers of myelin. (H &E ×200).

spread and ossified. Fig. 3 shows that there is no evidence of an inflammatory process.

Analysis of the surviving structures indicates that the spiral ganglion neurons are the structures being stimulated by the implant (Fig. 4). There was no difference in function between individuals with or without peripheral processes (neurites), erroneously called dendrites by some; the true dendrites are very small and extend from the habenula perforata, at the edge of the osseous spiral lamina, to the hair cells. Human cochlear neurons degenerate at a far slower rate than those of animals when deprived of an afferent input, Fig. 5 shows the spiral ganglia neurons in the mouse cochlea.

Discussion

The cochlear fibrosis and ossification produced by cochleostomies does not affect implant performance except in the cases of some hybrid implants in which there is some residual low tone hearing. If the insertion involves the scala vestibuli it may cause fibrosis and ossification that blocks the ductus reuniens and result in a delayed endolymphatic hydrops because the endolymph passage from the cochlea to the saccule is blocked.[1] There was no relationship between speech understanding and the number of neurons. In fact, there was a slightly reversed association. One individual, a retired pilot, used his implant for seventeen years before death and was found to have only 943 ganglion cells; the cause of his loss was advanced cochlear otosclerosis. Examination of these temporal bones suggests that the formation of fibrosis and subsequent ossification are due to damage to the endosteum by a cochleostomy or by longer electrode as they encounter the anterior bend to the first cochlear segment.

Funding

This work was supported by NIDCD U24 1U24DC015910-01(AI)

Conflicts of interest

The authors have no conflicts of interest to disclose.

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Edited by Jing Li