Otopathology in a Case of Multichannel Cochlear Implantation

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The histopathology of the temporal bones of a patient who died of unrelated causes 10 weeks following cochlear implantation using a Richards Ineraid® device is presented. Deafness was caused by a prolonged course of intravenous gentamycin therapy 5 years prior to implantation.

The electrode array of the cochlear implant was left in situ throughout histologic preparation and sectioning. Despite displacement and disruption of supporting structures of the inner ear, particularly in the 6-to-15-mm range as measured from the round window, there was no significant difference in the mean densities of spiral ganglion cells in the implanted and unimplanted sides.

This case is presented as evidence that despite significant disruption of supporting elements of the inner ear, which is common during cochlear implantation, there appears to be little effect on the residual spiral ganglion cell count, at least in the short term.

INTRODUCTION

Histologic studies of the temporal bones of patients who had previously undergone cochlear implantation have provided information concerning trauma to supporting elements of the cochlea due to electrode insertion, tissue reaction to the presence of implanted electrodes, and, perhaps most important, secondary degeneration of spiral ganglion cells. Previous studies have demonstrated significant trauma to the spiral ligament and basilar membrane, particularly at the round window near the site of insertion and in the 8-to-15-mm region of the upper basal turn. A variable tissue response, ranging from perielectrode fibrosis to new bone formation, has been reported.

The effects of implantation on the spiral ganglion cell population are less clear. Some studies have found a decrease in the number of spiral ganglion cells on the implanted side. Other reports have demonstrated no significant effect of implantation or stimulation on survival of spiral ganglion cells in either humans or experimental animals. Still other reports have shown an improved survival of residual spiral ganglion cells on the implanted versus nonimplanted side in deafened experimental animals.

The temporal bones from a patient who died 10 weeks following implantation of a Richards Ineraid® device were studied by light microscopy. This represents the second case report of the histopathologic findings following cochlear implantation using the Richards Ineraid device and the first report following implantation of the currently used “single bundle” electrode array. In addition, this study was performed using a new embedding and sectioning strategy allowing the implanted cochlea to be sectioned with the electrode array in situ, avoiding artifacts caused by removing the electrode array prior to embedding.

CASE REPORT

This 62-year-old woman became deaf following a pro-
longed course of intravenous gentamycin therapy 5 years prior to implantation. Her preoperative audiogram (Fig. 1) demonstrated a bilateral severe to profound sensorineural loss with no measurable speech discrimination. She underwent cochlear implantation in the right ear, using the Richards Ineraid device and a round window scala tympani approach. She died 10 weeks following the procedure of unrelated causes. Psychophysical data gathered just prior to her death was consistent with sound awareness and pitch perception. However, since a speech processor had not been fitted, there was no opportunity to test speech perception. Both temporal bones were removed 12 hours after death and fixed in 4% buffered formalin leaving the electrode array in situ. After decalcification in EDTA, the specimens were trimmed with a razor blade and postfixed in 2% osmium tetroxide, dehydrated in graded ethanol, exchanged with propylene oxide, and embedded in araldite. Registration holes were drilled in the specimen block using an excimer laser. The specimens were serially sectioned in the horizontal (axial) plane using a tungsten carbide blade on a LKB historange microtome at an average thickness of 35 μm. Selected sections were used for three-dimensional reconstruction and other sections were remounted on Epon blocks and sectioned at 5 μm with a glass knife. The sections were stained in toluidine blue, O, for light microscopic study.

The density of spiral ganglion cells in both the im-

Fig. 2. Unstained 35-μm axial section of the implanted right ear. The electrode array (EA) was sectioned in situ. Disruption of the basilar membrane (BM) is shown on the left, approximately 11 mm from the round window membrane, and displacement of the basilar membrane is shown on the right, approximately 18 mm from the round window membrane (original magnification x 28).}

Fig. 3. Unstained 35-μm axial section of the contralateral, unimplanted left cochlea. Normal supporting elements are seen (original magnification x 28).
planted and unimplanted cochleae was determined in multiple 5-mm sections at known distances from the round window. Using the MOP-3 (Carl Zeiss, Inc., Thornwood, NY) morphometry system and the segmental density technique, spiral ganglion cells containing a nucleolus were counted and the density of spiral ganglion cells per 0.001 mm$^3$ was calculated using a correction factor of 0.283 at a magnification of 430×.

RESULTS

Insertional Trauma From Cochlear Implantation

A detailed evaluation of the electrode position and insertional trauma in this ear is available in another study. Light microscopic evaluation of the morphology of the organ of Corti clearly demonstrated marked displacement of disruption of supporting structures of the inner ear (Fig. 2), whereas the morphology of supporting elements of the unimplanted ear was normal (Fig. 3). The most severe disruption occurred in the 6-to-15-mm range as measured from the round window. In this region the basilar membrane, spiral ligament, stria vascularis, and Reissner's membrane were disrupted (Figs. 2, 4). In the 16-to-24-mm range, the supporting elements of the organ of Corti were not disrupted, but were significantly displaced toward the scala vestibuli, with the electrode array positioned in the normal location of the scala media (Fig. 2). The supporting elements of the more apical portions of the cochlea were unaffected.

Survival of Spiral Ganglion Cells in the Implanted and Unimplanted Cochleae

The cochleae were divided for histologic study into six segments as measured in millimeters from the round window: 0 to 5, 6 to 10, 11 to 15, 16 to 20, 21 to 25, and 26 to the apex. The mean density of remaining spiral ganglion cells within each segment was determined from 2 to 14 sections in each cochlear segment for both the implanted (right) and unimplanted (left) ears (Table I, Fig. 5). An estimate of the total segmental spiral ganglion cells was calculated for this patient using the mean total segmental spiral ganglion count from normal hearing controls and the percentage of normal of the density measurements (Table I, Fig. 6).

As can be seen in Figure 5, the implanted cochlea had a higher density of spiral ganglion cells in each segment except in the 11-to-20-mm segments which coincide with the area with greatest disruption due to insertional trauma as evaluated by serial section light microscopy. In Figure 6 the segmental spiral ganglion cell count of normative controls, the average segmental spiral ganglion cell count from patients deafened by ototoxic injury, and the segmental spiral ganglion cell counts predicted for this patient derived from a regression analysis are also displayed. The total and segmental spiral ganglion cell counts for the left and right ear were not significantly different from each other. The total spiral ganglion cell counts of both ears were significantly smaller than those of normal hearing individuals ($P = .003$), but there was no significant difference for either ear of this patient from the distribution predicted for this patient using a regression analysis based on cause of hearing loss, age, duration of deafness, and sex.

DISCUSSION

Trauma to Supporting Elements of the Cochlea Caused by Electrode Implantation

The histologic findings in this case are similar to previous reports, both of human temporal bones implanted during life and in cadaveric specimens. In this case displacement or disruption of the spiral ligament and basilar membrane occurred along the electrode path and particularly in the 11-to-15-mm range.
Survival of Spiral Ganglion Cells in the Implanted Cochlea

Earlier reports by Zappia, et al. and Marsh, et al. attributed a decreased ganglion cell count on the implanted side to insertional trauma. Zappia, et al. reported a loss of 90% of normal spiral ganglion cell density adjacent to the implant on the implanted side compared to 46% loss on the unimplanted side, but in all other areas of the cochlea the segmental spiral ganglion cell counts were higher in the implanted ear. However, the etiology of deafness in the two ears of that patient was not comparable, and the onset was not simultaneous, making comparisons between the two ears tenuous. Marsh, et al. reported that the implanted side showed approximately 57% fewer spiral ganglion cells than the unimplanted side. Most damage occurred in the turns containing an electrode. In an individual who had lost hearing 42 years earlier secondary to meningitis and who had undergone unilateral Nucleus multichannel implantation 2½ years before death, Clark, et al. found equal numbers of ganglion cells in the spiral ganglia adjacent to as well as apical to the multichannel implant device. This was interpreted as evidence that the implantation and stimulation did not result in significant damage to the remaining ganglion cells. However, the residual number of spiral ganglion cells in the 0-to-25-mm range was consistently higher in the contralateral unimplanted cochlea than in the implanted cochlea. In their study of 22 temporal bones from 13 patients who had undergone cochlear implantation during life, Linthicum, et al. found no difference in counts of remaining spiral ganglion cells in the implanted and unimplanted sides.

In cochlear implants in monkeys and in the cat, no significant difference in residual spiral ganglion cell counts between implanted and unimplanted cochleae was found. In the cochleae of implanted ears, the average density of spiral ganglion cells (cells per 0.001 mm²) for normal adult human cochlea (Nadol, 1988) and the implanted and unimplanted side in the reported case. Error bars represent standard deviation in each segment.

<table>
<thead>
<tr>
<th>Cochlear Segment*</th>
<th>Density SPG (No. of Cells/0.001 mm² ± SD)</th>
<th>Density (± SD) in Normative Controls†</th>
<th>% Normal SPG Density‡</th>
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</thead>
<tbody>
<tr>
<td>0–5 mm</td>
<td>11.72 (±2.17)</td>
<td>39.0 ± 6.7</td>
<td>27.9%</td>
</tr>
<tr>
<td>6–10 mm</td>
<td>15.06 (±5.57)</td>
<td>45.0 ± 4.0</td>
<td>33.5%</td>
</tr>
<tr>
<td>11–15 mm</td>
<td>23.95 (±4.92)</td>
<td>49.0 ± 1.7</td>
<td>48.9%</td>
</tr>
<tr>
<td>16–20 mm</td>
<td>25.02 (±7.89)</td>
<td>47.0 ± 6.7</td>
<td>53.2%</td>
</tr>
<tr>
<td>21–25 mm</td>
<td>23.35 (±4.99)</td>
<td>46.0 ± 4.0</td>
<td>50.7%</td>
</tr>
<tr>
<td>26 mm–apex</td>
<td>17.22 (±4.39)</td>
<td>35.0 ± 5.9</td>
<td>49.2%</td>
</tr>
</tbody>
</table>

% Normal SPG density (average entire cochlea)

*Distance from round window.
‡% normal SPG density = \( \frac{\text{density SPG (implanted or unimplanted)}}{\text{density SPG (in normative controls)}} \times 100\% \).

SD = standard deviation.
planted deafened guinea pigs, Lousteau et al. demonstrated a significantly greater number of spiral ganglion cells in the stimulated implanted ears compared to the contralateral unimplanted control. Lousteau and Hartshorn, et al. both concluded that cochlear implantation and stimulation may slow degeneration of spiral ganglion cells in deafened ears.

There was no statistically significant difference in the residual spiral ganglion cell counts in the implanted and unimplanted ears of the patient reported here. Since the patient had been deafened by intravenous gentamycin therapy, a nearly equal, bilateral, preoperative complement of spiral ganglion cells would be expected. While counts were lower in the basal region in which most trauma occurred, given the large standard deviation in these data, the densities of the spiral ganglion cells were not significantly different. Furthermore, densities in the implanted side exceeded those of the unimplanted side in three of the four segments where the electrode array was located. There was no evidence of significant degeneration of the spiral ganglion cells despite significant disruption of the supporting elements. Because the patient died approximately 10 weeks after implantation, these results represent only short-term effects, and long-term results of mechanical disruption and stimulation could not be evaluated.

BIBLIOGRAPHY