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Method for hearing preservation in cochlear implant surgery

Peter S. Roland, MD,[a] Wolfgang Gstöttner, MD,[b] Oliver Adunka, MD[b]

From the [a]Department of Otolaryngology–Head and Neck Surgery, The University of Texas Southwestern Medical Center at Dallas, Dallas, Texas; and the
[b]Department of Otolaryngology, Johann Wolfgang Goethe University, Frankfurt, Germany.

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Individuals with useful hearing in the lower frequencies (less than 1000 cycles per second) but with severe to profound losses in the higher frequencies often have poor speed discrimination scores, especially in noise. Conventional hearing aids only provide marginal increases in word discrimination for this class of individuals. Consequently, interest has developed in electrically stimulating those regions of the cochlear (apical) that subserve higher frequencies while permitting the individual to continue to hear (with or without a hearing aid) lower frequency sound acoustically. To successfully implement such a strategy, residual hearing must be maintained for lower frequency sounds. Technical alterations in the operative technique of cochlear implantation designed to preserve hearing include: (1) avoidance of acoustic trauma using low speed drills; (2) careful placement of the cochleostomy anterior and inferior to the round window membrane to avoid damage to the basilar membrane and ossea spiral lamina; (3) the use of steroids to protect against injury to the organ of Corti at the cellular level; (4) the use of shorter, thinner, atraumatic electrodes; and (5) a small cochleostomy to prevent buckling of the electrode and escape of perilymph.

The extent to which inserting an electrode into the cochlea produces endocochlear damage and injury to neural structures has been a topic of interest since the inception of cochlear implant technology. Interest in cochlear implant array insertional trauma has recently increased because cochlear implant surgeons now wish to preserve residual hearing. Until recently, complete loss of residual hearing was the expected outcome of cochlear implantation. However, recent efforts at hearing conservation have shown that residual hearing can be preserved in the majority of cases if the operating surgeon pays very close attention to minimizing surgical trauma while inserting the electrode array. However, even when great care is taken, residual hearing is completely lost in at least 10% to 20% of recipients.[1] The precise cause of hearing loss in this group of cochlear implant recipients remains obscure. We believe that a careful assessment of the details of electrode insertional trauma will help eliminate or ameliorate hearing loss in cochlear implant recipients. Questions about the extent to which mechanical insertion of a cochlear implant electrode array damages anatomic structures within the cochlea is a separate question from whether or not chronic electrical stimulation results in injury to residual neural elements. The latter question will not be addressed in this article but has been extensively investigated by other researchers who have concluded that chronic electrical stimulation sustains, rather than injures, spiral ganglion cells.[2]

A number of approaches have been used to determine the extent to which and the mechanism by which mechanical introduction of an electrode array damages endocochlear elements. Traditional methods that have been used to evaluate insertional trauma have included[3-10]:

1. Electrophysiologic assessment of animals into which electrode arrays have been inserted in vivo.
2. Postmortem histopathologic evaluation of animals, principally rodents and cats, into which cochlear implant electrode arrays have been inserted. Animal studies provide useful information but do not allow for control of

Address reprint requests and correspondence: Peter S. Roland, MD, The University of Texas Southwestern Medical Center at Dallas, 5323 Harry Hines Boulevard, Dallas, TX 75390-9035.
E-mail address: peter.roland@utsouthwestern.edu.

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individual variability, cause of deafness. or surgical technique.
3. Postmortem evaluation of human temporal bones taken from cochlear implant recipients. Such evaluations are especially useful for determining long-term changes associated with cochlear implantation, especially the assessment of endocochlear new bone formation and fibrosis.
4. Cochlear implant electrode array insertion into cadaveric temporal bones from individuals who have never been implanted. Such temporal bones can be either fresh or formalin fixed. Human cadaveric temporal bones can be used in a variety of different ways. Electrodes can be inserted during fluoroscopy, and the passage of the electrode into the bony structures in the cochlea can be visualized. However, fluoroscopic evaluation does not allow good visualization of soft tissues and does not permit assessment of subtle soft tissue trauma. Electrodes can first be inserted into human temporal bones, fresh or fixed, and then submitted for histopathologic evaluation. Histopathologic evaluation of temporal bones into which cochlear implant electrode arrays have been inserted require special techniques because sectioning of the relatively hard electrode array cannot be performed after simple decalcification. Freezing techniques produce artifacts, and fixation using plastic produces significant swelling artifact.

Opening the cochlea and inserting a cochlear implant electrode array can produce trauma from a variety of mechanisms: (1) opening the cochleostomy, (2) damage can occur as a result of passing the electrode into scala tympani, (3) insertional trauma can lead to subsequent new bone formation and fibrosis within scala tympani, and (4) opening the cochlea is a portal of entry for infection and the development of infectious labyrinthitis.

To avoid injury, a promontory cochleostomy must be placed inferior to spiral ligament, the basilar membrane, and osseous spiral lamina. Scala tympani dips inferiorly and anteriorly as it passes forward from the round window membrane. Because the round window membrane itself cannot generally be visualized when performing a classic cochleostomy, the point on the promontory where drilling can be performed without risking damage to the basilar membrane osseous spiral lamina is unclear. The frequency with which injury to the basilar membrane and ossea spiral lamina occurs has not been reported, but careful study of the relevant anatomy suggests that such injuries may be relatively common. Animal data suggest that localized injuries to the basilar membrane or osseous spiral lamina in and of themselves probably do not result in total hearing loss.[6] However, the effect of penetration into scala vestibule with the attendant introduction of perilymph into scala media is likely to result in significant and widespread injury to the organ of Corti because perilymph is toxic to hair cells.

Passing the electrode array through the cochleostomy can also produce injury by a variety of mechanisms:

1. Endosteal injury. Trauma to the endosteum will increase postoperative fibrosis and osteoneogenesis. At UT Southwestern, we have not been able to show significant endosteal injury in temporal bones, either fixed or fresh, as a result of electrode array insertion. The lateral wall of scala tympani is laced with venules. Injury to these venules with subsequent bleeding has been hypothesized, but damage to venules in the lateral wall has not been verified in our laboratory. However, one must hasten to emphasize that these are postmortem specimens and post-insertional bleeding may be undetectable.
2. The electrode array can become kinked within the temporal bone. We have not seen folding over of the tip with doubling back of the electrode array onto itself in our investigations, but other investigators have. Such kinking occurs when the tip of the electrode array impacts the modiolus, fixing it. Continued insertion then causes the portions of the electrode array just beneath the tip to continue advancing as the electrode array doubles over itself.
3. Fraction and dislocation of the ossea spiral lamina has been seen by most investigators.[3-5] It can occur directly as a result of the tip impacting the osseous spiral lamina and fracturing it or, as seen in our dissections, can occur from buckling of the more proximal portions of the electrode in scala tympani after the tip becomes fixed. The extent to which fracture or dislocation of the ossea spiral lamina produces hearing loss is unclear. Damage to the osseous spiral lamina and basilar membrane in animals appears to produce only a discreet loss of those spiral ganglion cells subserving the fractured or torn segments. Widespread loss of ganglion cells is not seen in animals unless damage to the osseous spiral lamina or basilar membrane involves large portions of the cochlea.
4. Basilar membrane perforation occurs occasionally, and appears to be at least partially related to the stiffness and rigidity of the electrode. However, it appears perforation of the basilar membrane may occur with any electrode, and it may not be possible for the surgeon to prevent basilar membrane perforation reliably in every case. It seems that injury to the basilar membrane itself will only produce a discreet loss of spiral ganglion cells. However, it is hypothesized that if there is mixing of endolymph and perilymph, more widespread hearing loss can occur.
5. Displacement of the basilar membrane appears to be common. Although it is not known with surety whether or not this produces damage to neural elements, it is believed that minor displacements of the basilar membrane inward toward scala media probably have no adverse effect on auditory function.
6. Dissection of the spiral ligament away from the lateral wall of the cochlea appears to occur more frequently than was previously recognized. Again, the extent to which this impacts hearing is unclear, but it probably depends on the extent to which it occurs. Whether or not this is more or less likely in older individuals with age related changes to the spiral ligament is also uncertain.

Hearing preservation

Surgical technique

During the last 5 years, a new method, the combined electricacoustic stimulation (EAS) of the auditory sys-