*The Journal of Laryngology and Otology*
April 1980. Vol. **94.** pp. 359–366.

# Cochlear prosthesis: morphological considerations*†

By JOSEF M. MILLER, Ph.D. and DWIGHT SUTTON, Ph.D.
(Seattle, Washington)

SCHUKNECHT and colleagues conclude a recent paper with the statement 'The mandate for the implant team is to devise a method for determining ganglion cell population in patients, to develop a surgical method of implantation which will not further diminish the number of cells, and to introduce a device that will provide properly coded stimulation so that useful hearing will result' (Otte, Schuknecht and Kerr, 1978). This statement well focuses attention on two main concerns of prosthetic development and application involving temporal bone morphology. These concerns involve questions of patient selection (as previous papers in this meeting have discussed) and the damaging effects of implantation and stimulation by the prosthesis (as will be discussed subsequently at this meeting).

Re-phrasing the question: What does the patient have in terms of residual elements, and what does the prosthesis do to these? At this time the best that we can do is to examine some of the factors of concern relevant to these questions. To provide our conclusions now from review of information currently available, they are: We need more data on histopathology associated with hearing loss of various etiologies, and we must determine what elements are activated by and are essential for useful function of the implant if we are to interpret pathological data in terms of candidate selection for the prosthesis.

It seems obvious that patients identified as potential candidates must have a sufficient population of auditory neural elements to permit activation of the central auditory system. The number of elements required will vary depending upon the presumed goal of the implant and assumptions that we are willing to make regarding the similarity between system function with direct electrical stimulation and with natural acoustic activation. Intelligible speech discrimination undoubtedly requires more elements than 'putting the patient in contact with his auditory environment'. Just how much residual is needed for speech discrimination is poorly defined.

* This work was supported in part by USPH Grant NS13056 and RR00166 and the William G. Reed Fund.

† Presented at a workshop entitled 'Prosthetic and technical devices for the deaf', sponsored by the Department of Audiology at the National Technical Institute of the Deaf, August 14–18, 1978.



https://doi.org/10.1017/S0022215100088940 Published online by Cambridge University Press

On the basis of correlations between clinical tests and subsequent human temporal bone studies, varying figures have been proposed for the number of normal ganglion cells essential for speech discrimination. Absolute numbers of elements are but part of the problem, their distribution is also important. For example, the minimum number of spiral ganglion cells required for speech discrimination was estimated by Otte *et al.* (1978) as approximately 10,000, with 3,000 of this number in the apical segment. Absence of apical cells imposes a major deficit on speech discrimination. It must be noted that their estimated 'required-minimum' residual cells is a mean value derived from a number of observations. Within their material, there were patients exhibiting good discrimination where relatively few ganglion cells were found, while others exhibited poor discrimination with more than twice this mean number. Such variability is intriguing. To what extent does total number of cells and their distribution alone determine residual hearing?

Clearly of major significance is the extent to which estimates of residual function based upon natural stimulation are relevant to prosthetic stimulation. Given our limited knowledge in this field, it is conceivable that a few hundred cells optimally distributed (and optimally accessible) might carry sufficient signal information to provide for recognition of complex sounds transduced through a prosthesis. However, it is also conceivable that a full complement of functional excitable spiral ganglion cells may be insufficient for *practical* prosthetic elicited speech discrimination.

Most acquired deafness results in variable receptor loss, i.e., non-uniform (Johnsson *et al.*, 1978; Sando *et al.*, 1977). Otte *et al.* (1978) analyzed a number of cases, observing that similar etiology and audiological pathology was associated with markedly different ganglion cell losses:

(a) Structural losses in patients with similar hearing deficits ranged from severe and widespread destruction to negligible ganglion cell loss and damage to but a few outer hair cells in the cochlea.
(b) Losses of hair cells, dendrites, or spiral ganglion cells, when evident, were patchy—adjacent areas of high and low density neural elements were not uncommon.
(c) Spiral ganglion cell loss following loss of hair cells and supporting structures of the organ of Corti was variable.

In regard to this latter point one would, of course, expect secondary degeneration of nerve fibers following organ of Corti damage to vary dependent upon the survival time following initial damage. Current views concerning subsequent ganglion cells and axonal degeneration vary. The most pessimistic view, in regard to prosthesis application, is that hair cell and supporting structure damage leads to eventual degeneration of all but a few neural elements (see Schucknecht, 1974; Johnsson, 1974; Spoendlin, 1975).

https://doi.org/10.1017/S0022215100088940 Published online by Cambridge University Press

It should also be noted that in the clinical literature most concern is expressed for organ of Corti and associated spiral ganglion cell structure of the apical turn of the cochlea—since this area is principally concerned with the discrimination of low frequency speech signals. In general it seems that more basal segments of the cochlea are disproportionately affected in acquired deafness of various etiologies. Yet, given one of the principal prosthetic approaches (i.e., that involved with implants of the scala tympani), technology focuses upon the basal portion of this system. If we hope to deal with restricted stimulation of discrete segments of the system using a scala tympani prosthesis, it may be necessary to develop alternative approaches enabling stimulation of more apical segments of the cochlea. This suggestion is not based upon the goal of activating cells normally concerned with the encoding of speech frequencies, but upon the need to activate the only portion of the system that may remain.

The variation in locus and type of cell loss with etiology creates a heterogeneous population of potential implant candidates. Bergström (1975) has recently classified primary morphological changes in the temporal bone as sensory and/or neural on the basis of etiology of the disease state. She formed ten categories ranging from 'Dysplasias and genetically determined disorders causing sensory and neural pathology' through 'Traumatic lesions . . .', 'Toxins . . .', and 'Ageing . . .' to 'Neurologic disorders . . .'. In the latter category, spiral ganglion cell and VIIIth nerve degeneration is a primary characteristic. Obviously, patients with this problem would be inappropriate implant candidates. Perhaps most promising on the basis of Bergström's review are patients with pathology of genetically and traumatically determined origin. Patients with genetic or traumatically induced pathology exhibited much less spiral ganglion and VIIIth nerve loss than did those with viral histories.

Histopathological evidence for surviving ganglion cells in congenital deafness raises further considerations for the implant: (1) are the surviving ganglion cells viable, functioning elements with normal transmission capability and (2) are the associated central pathways capable of discriminative information transfer and processing? Hunter-Duvar (1976) found one-third to one-half surviving ganglion cells in the absence of any detectable hair cells in a five-year-old male with a history of infantile deafness. No supporting cell structure was present on the basilar membrane. A normal number of myelinated fibers were found in the apical turn of the cochlea with good to fair representation basally. The ventral cochlear nucleus, however, contained one-half the normal number of cells.

Central neural changes with early deafness and acoustic deprivation have also been documented in experimental animal preparations. Webster and Webster (1977) found a reduction in size of specific cell types (globular) in the ventral cochlear nucleus of acoustically deprived and deafened animals and a reduction in mean packing density of cells in the dorsal cochlear nucleus. Such results as these are consistent with the suggestion

https://doi.org/10.1017/S0022215100088940 Published online by Cambridge University Press

that there may be a need to introduce sustaining stimulation of some sort (acoustic or electrical) at the level of the periphery as early as possible in order to preserve pathways of the congenitally deaf. This suggestion may be too simplistic. An important experimental question is thus, whether such a procedure would temper conclusions from observations like Mair's (1973), that the spiral ganglion cells of albino cats inevitably disappear, given sufficient survival time.

The specification of an appropriate implant candidate on the basis of data suggesting 'typical' histopathology associated with different etiologies of deafness is made difficult by the observed variability in structure-function correlations. Perhaps the great disparity from patient to patient indicates that our complex acoustic tests are not capable of satisfactory differentation of nerve status. Unquestionably, additional studies on the morphological changes that result from various agents and diseases will help. For the purpose of the prosthesis, parametric investigations are necessary, systematically performed on a large number of specimens.

At our current rate of progress in this area and with the clear biological variability that appears to exist in this field, it is unlikely that we will have information of use in selection of candidates for a cochlear prosthesis in the near future. Can we say that same thing regarding *rejection* of candidates?

Greater emphasis in this field might be placed on development of better tests (electrophysiological) which identify peripheral auditory system function in the sensorineural deaf patient. It would be appropriate to add that with continued technical development in this field we will need to develop tests which provide not only information on the per cent of the system which is functional, but which also give specific information on the state of sensory elements of the cochlea and neural elements at various points within the geometry of the cochlea.

Finally, superimposed on these questions of candidate selection, we must consider which of the various implant approaches are to be used. It would seem that appropriate activation of the system with multipolar scala tympani electrodes, a single scala tympani wire, and multiple electrodes in the modiolus might each place different requirements upon the system. Unfortunately at this time we know little of the voltage gradients and current distribution within the periphery generated with these different stimulating systems, and we know less of what elements are essential for system activation with any of these devices.

These questions related to patient selection all bear on the morphological state of the system prior to implantation—what the patient comes with. The next question is, what will the implant do to the pre-existing complement of neural elements. Problems in regard to this question include: the traumatic effect of implantation, tissue reaction to the foreign body, and the effect of stimulation. Concerns for the effect of stimulation have been raised and addressed in a number of recent publications (see for example Tonndorf, 1977; Hambrecht and Reswick, 1977).

https://doi.org/10.1017/S0022215100088940 Published online by Cambridge University Press

Histopathological data on implanted humans and animals are limited. No data are available at this time on the effects of chronic electrode implantation in the modiolus. Of the scala tympani electrode systems, data have been reported from animals implanted with the silastic molded system and single wires (Schindler, 1975; Schindler and Merzenich, 1974; Schindler *et al.*, 1977; Simmons, 1967; Sutton *et al.*, 1978). Temporal bones from a human implanted with bare wires in the scala tympani have been studied (Johnsson *et al.* (1979)).

Schindler *et al.* (1977) reported on an experimental series of cats implanted with the molded scala tympani system for as long as 30 months. Normal animals and animals pre-treated with neomycin were used. This study showed that in such animals a significant proportion of normal spiral ganglion cells remain after such experimental manipulation. A correlation between fiber and spiral ganglion loss with supporting element damage was observed. While no evidence for osteogenesis was reported in the recent Schindler *et al.* (1977) paper, in previous reports (Schindler and Merzenich, 1974; Schindler, 1975) intracochlear bony growth was observed following damage to the endosteal tissue.

Sutton *et al.* (1978) used a multipolar electrode system in a small diameter silastic carrier which slipped relatively freely into the scala tympani, comparing this with a molded implant such as that used by Schindler *et al.* (1977). We found that inserting the molded implant is more likely to create physical trauma to the basilar membrane and osseous spiral lamina than is the free-fit implant. Comparative spiral ganglion cell loss observed with the two implants is shown in Fig. 1. Anatomical constraints of the non-human primate ear indicate that freedom in manipulation of the molded implant is limited. This may also be a consideration in designing implants for humans.

A universal challenge faced by all of the implant techniques (including the modiolar approach) stems from the need to inhibit osteogenesis resulting from endocochlear trauma. While this was not a major problem in the cats studied by Schindler *et al.* (1977), we find it to be a recurrent problem in our implants in monkeys; and Johnsson *et al.* (1979) recently documented the serious nature of this problem in a human implant case.

The material studied by Johnsson consisted of both temporal bones from a patient with bilateral implants. A single electrode had been inserted in the right cochlea approximately 1 turn, three years prior to death. In the left cochlea a cluster of five staggered-length electrodes had been inserted approximately three-fourths of a turn, eight years prior to death. The most striking findings were essentially absent neural elements in the cochlea and the presence of extensive bony growth. The osteogenesis was presumed secondary to endosteal damage associated with initial implantation. The neural degeneration was presumed to be secondary to the original pathology of the ear and further exacerbated as a result of the

https://doi.org/10.1017/S0022215100088940 Published online by Cambridge University Press

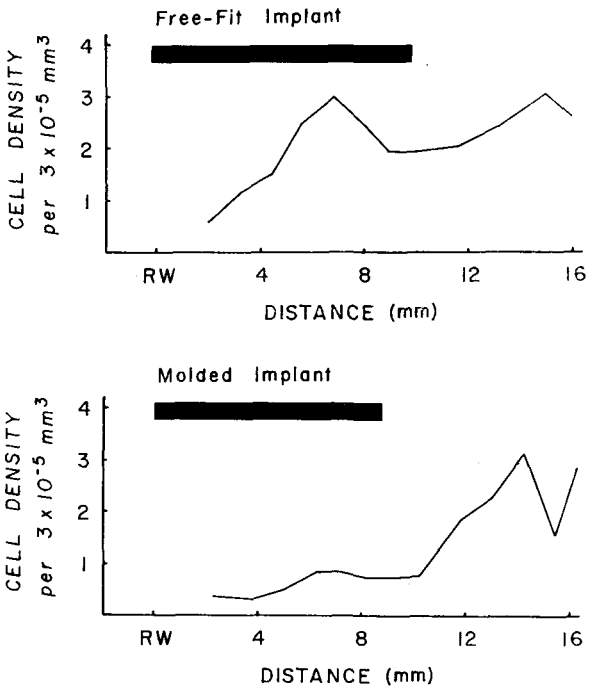



FIG. 1

Comparison of ganglion cell loss with molded and free fit scala tympani implant. Black bar indicates location of implant within the scala tympani.

factors producing the bony growth. A few hair cells remained in one ear (right). In this ear there remained approximately 4 per cent of the dendrites supplying these few hair cells. The right temporal bone 'showed two small clusters of ganglion cells in the apical and basil portions of the modiolus...' 'In the left ear a typical mid-modiolar section revealed only two single ganglion cells in the center of the modiolus plus a group of 8 cells in the base of the modiolus'. This suggests for the left ear a population of approximately 2 per cent of the normal complement. Remarkably, the patient derived functional advantage from each of these implants.

Simmons (1967) has also reported on the effects of implanting bare electrodes (not in a silastic carrier) into the scala tympani of the cat. Ears were normal at the time of surgery. Typically, two wires were inserted, one approximately 1 cm, the second less deeply. Survival time ranged up to 'over 14 months'. Simmons' report indicates that the ganglion cells tolerate the implants fairly well. Together with the observations of Schindler *et al.* (1977), these data suggest that the cat cochlea is robust and ganglion cells survive scala tympani implantation relatively well. Simmons did observe significant neural damage in some animals associated with surgery, tears in the basilar membrane (no mention is made of endosteal damage and

https://doi.org/10.1017/S0022215100088940 Published online by Cambridge University Press

bony growth) and infection and foreign body reaction. Simmons' results are presented anecdotally, but it would appear that in the majority of animals the majority of neural elements remained—particularly if infection was avoided or minimal.

While local infection was a problem in a few of Simmons' early cats, and indeed an occasional problem in some of our early monkeys, it has been a solvable problem in animal studies of the cochlear prosthesis and little problem (apparently, from reports in the literature) in humans. Foreign body reaction in general has been relatively minimal, particularly with the materials now commonly used (silastic and platinum-iridium or rhobidium). Similarly, at least for the relatively short-term studies performed to date, the effects of current on tissue viability can be controlled. The stable behavioral thresholds observed both in patients (Simmons, 1966) and in animals (Pfingst *et al.*, 1978) may be interpreted as indicative of little tissue damage from stimulation. Naturally variability exists throughout this body of data which restricts hard and fast conclusions at this time. (For clinical purposes within this field we must focus on the 'worst' findings; for experimental and developmental purposes we must focus on the 'best' observations.)

By contrast neural damage and degeneration resulting from tears in the membranous labyrinth and osteogenesis from damage to the periotic labyrinth remain as significant problems. These are technical problems—but of major importance. Moreover, their importance grows, since these problems are specifically enhanced, as we consider the potential need with the scala tympani approach to place electrodes in apical regions of the cochlea.

In regard to animal and human histopathology resulting from the implant, and in regard to the histopathological data available on sensorineural hearing loss as relevant to selection of candidates for implantation, our greatest problem appears to be interpreting the progressive nature of morphological change. Will the spiral ganglion cells and nerve fibers eventually degenerate? Major evidence suggests yes, if inner hair cells (Spoendlin, 1975) or supporting elements (Schuknecht, 1974; Johnsson, 1974) are damaged. Some data suggest that this is not necessarily so (Kerr and Schuknecht, 1968; Schindler *et al.*, 1977; Schuknecht *et al.*, 1965) but our interpretation of these findings must be conditioned by questions concerning the adequacy of survival time in this material (Mair, 1973).

There is a consensus that with cochlear pathology the last elements to degenerate are the modiolar structures (VIIIth nerve axons). This may be viewed as encouragement for the modiolar implant approach. However, given our lack of knowledge of what voltage gradients are generated and where current flows with any of these approaches and, most importantly, what elements are essential for system activation with any of these approaches, it is difficult to conclude at this time other than: we need

https://doi.org/10.1017/S0022215100088940 Published online by Cambridge University Press

more basic data in this area. Basic data are necessary on histopathology associated with sensorineural deafness. It is necessary on long-term histopathological concomitants of implantation. It is necessary on functional tests of excitability of existing elements within the periphery and central auditory system. And more basic data are necessary on current distribution and specification of essential elements for prosthetic activation of the auditory system.

## REFERENCES


BERGSTRÖM, L. (1975) *Canadian Journal of Otolaryngology*, **4,** Supplement #2, 1–28.

HAMBRECHT, F. T., and RESWICK, J. D. (1977) Marcel Dekker, New York, p. 543.

HUNTER-DUVAR, I. (1976) *Canadian Journal of Otolaryngology*, **5,** 497–504.

JOHNSSON, L.-G. (1974) *Annals of Otology*, **83,** 294–303.

JOHNSSON, L.-G., HAWKINS, J. E., and LINTHICUM, F. H. (1978) *Annals of Otology, Rhinology and Laryngology*, Supplement 48, **87,** 1–40.

JOHNSSON, L.-G., HOUSE, W. F., and LINTHICUM, F. H. (1979) *Laryngoscope*, **89,** 759–762.

KERR, A., and SCHUKENCHT, H. F. (1968) *Acta Oto-Laryngologica*, **65,** 586–598.

MAIR, I. W. S. (1973) *Acta Oto-Laryngologica*, Supplement **314,** 1–48.

OTTE, J., SCHUKNECHT, H. F., and KERR, A. G. (1978) *Laryngoscope*, **88,** 1231–1246.

PFINGST, B. E., DONALDSON, J. A., and MILLER, J. M. (1978) Proceedings of Midwinter Research Meeting, Association for Research in Otolaryngology, p. 22.

SANDO, I., HARADA, T., LOEHR, A., and SOBEL, J. H. (1977) *Annals of Otology*, **86,** 269–279.

SCHINDLER, R. A. (1975) *Journal of Laryngology and Otology*, **90,** 445–457.

SCHINDLER, R. A., and MERZENICH, M. M. (1974) *Annals of Otology*, **83,** 202–216.

SCHINDLER, R. A., MERZENICH, M. M., WHITE, M. W., and BJORKROTH, B. (1977) *Archives of Otolaryngology*, **103,** 691–699.

SCHUKNECHT, H. F. (1974) *Pathology of the Ear*, Harvard University Press, Cambridge, Massachusetts.

SCHUKNECHT, H. F., IGARASHI, M., and GACEK, R. R. (1965) *Acta Oto-Laryngologica*, **59,** 154–170.

SIMMONS, F. B. (1966) *Archives of Otolaryngology*, **84,** 2–54.

SIMMONS, F. B. (1967) *Laryngoscope*, **77,** 171–186.

SPOENDLIN, H. (1975) *Acta Oto-Laryngologica*, **79,** 266–275.

SUTTON, D., MILLER, J. M., PFINGST, B. E., and DONALDSON, J. A. (1978) Proceedings of Midwinter Research Meeting, Association for Research in Otolaryngology, p. 24.

WEBSTER, D. B., and WEBSTER, M. (1977) *Archives of Otolaryngology*, **103,** 392–396.



Josef M. Miller, Ph.D.,
Department of Otolaryngology, University of Washington,
Seattle, Washington 98195, USA.


https://doi.org/10.1017/S0022215100088940 Published online by Cambridge University Press