PNEUMOCOCCAL MIDDLE EAR INFECTION AND COCHLEAR IMPLANTATION

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Supported by the Deafness Foundation of Victoria.

A limited study for the experimental induction of pneumococcal otitis media is presented. It is a useful model to study the effects of otitis media in the implanted and nonimplanted cochlea of the cat. Pneumococcal otitis media caused minor pathological changes in two nonimplanted cochleas and more widespread changes together with significant loss of neural elements in two implanted cochleas. However, the small number of animals used in this study did not allow us to distinguish between the effects of electrode insertion trauma, infection, or the combination of both.

Intracochlear implantation via the round window membrane breaks down the physiologic seal between the inner ear and the middle ear. The new round window seal following the insertion of a cochlear implant electrode must be effective in preventing infection entering the cochlea from the middle ear. This is especially important in pediatric cochlear implantation because children have a high incidence of otitis media.

Streptococcus pneumoniae is the commonest cause of acute bacterial otitis media in humans, accounting for over one third of all cases. Streptococcus pneumoniae is a gram-positive encapsulated diplococcus. Its virulence is the result of the presence of a type-specific capsular polysaccharide that is nontoxic, but acts to neutralize an antibody before it can bind the pneumococcus, and thus promote infectivity. The pneumococcus also elaborates a hemolysin and a neuraminidase, but there is no clear evidence that these are related to pathogenicity.

Cats are very resistant to pneumococcal infection. Experimentally induced pneumococcal otitis media has been described in susceptible animals such as the rat and the chinchilla, but the literature does not describe similar experiments in cats. Because of the successful technique in inducing otitis media with S pneumoniae in a pilot study, and because of its far greater clinical relevance compared to the rat and the chinchilla, pneumococcus was selected for use in this model to study the effects of delayed otitis media following implantation.

METHODS

Four healthy cats weighing at least 2 kg underwent unilateral insertion of a round window electrode and contralateral insertion of a control electrode placed in the bulla without opening the round window membrane. At 12 weeks postimplantation, a period considered an adequate time to allow a round window seal to be achieved, the cats underwent bilateral bulla inoculation with S pneumoniae group 2. We introduced 0.2 to 1.0 mL of a broth containing 10⁶ organisms/mL directly into the surgically opened bulla. The organisms were retained in the bulla by Gelfoam. Antibiotics were not administered during this procedure. The virulence of the pneumococcus was enhanced by intraperitoneal inoculation in mice before its use in the cat. The electrodes consisted of a Silastic shaft with a diameter of 0.6 mm and three platinum rings at the tip. The cats were killed 10 days following inoculation. Swabs of the bullae were taken just prior to killing. The cochleas were prepared for light microscopic histological examination using both hematoxylin and eosin and Gram's stain.

RESULTS

Of the four cats, two died before any histologic data could be obtained. A postmortem examination did not disclose signs of septicemia. Cultures taken from the bullae at the time of killing grew the inoculated organisms from each of the remaining four bullae. All these bullae showed unequivocal histopathologic inflammatory changes with the presence of inflammatory exudate, fibroblasts, polymorphs, monocytes, and lymphocytes. Organisms were occasionally seen within epithelial cells of the mucosa. The round window membrane, which was markedly thickened, showed in places the presence of respiratory-type epithelium on the bulla surface of the membrane. This was characterized by ciliated pseudostratified columnar and goblet cells.

The overall extent and severity of the otitis media produced was comparable for all cochleas, regardless of whether an electrode had been inserted before inoculation. However, this was not the case for changes in the inner ear. More severe damage was seen in the implanted than the control cochleas. However, the control group did show the presence of a number of inner ear pathologic features, which were generally restricted to the basal turn. These included collapse of Hensen's and Claudius' cells, collapse of inner sulcus cells, loss of some hair cells in the organ of Corti, absence of supporting cells, some ganglion cell losses, mild congestion and atrophy of the stria vascularis, fibrin precipitate in the cochlear duct, and connective tissue fibers in the perilymph adjacent to the round window.

The implanted cochleas showed, in addition, more widespread and severe changes. There was extension of the inflammatory process from the bulla into the scala tympani adjacent to the round window membrane, together with evidence of traumatic electrode insertion. This was represented by local fractures of the osseous spiral lamina, together with new bone formation, and concomitant local reduction in the number of nerve fiber and ganglion cells. More significantly, however, these cochleas also showed a more widespread loss of nerve fibers and ganglion cells in regions of the cochlea distant to the site of trauma.

DISCUSSION

The technique of packing the cat bulla with Gelfoam and inoculating with a large number of pneumococci was shown reliably to induce an acute otitis media in all four cochleas, despite the fact that the organism was reputed not to be virulent in cats. The technique is relatively quick, simple, and should be repeatable in a major series.

Gelfoam served to retain the organisms in the bulla and prevent discharge through the eustachian tube. In addition, the large number of organisms inoculated no doubt contributed to the success of the model, which is expected...
from previous studies showing that the greater the size of the inoculum, the greater the incidence of otitis media.\textsuperscript{7}

Pneumococcal otitis media was shown to induce mild pathologic inner ear changes in the nonimplanted cochlea. This is in contradistinction to the group A β-hemolytic streptococcus that was shown to be nonvirulent even to the implanted cochlea, despite having produced an adequate otitis media.\textsuperscript{4} These changes do not appear to be artifactual because they are reproduced in different cochleas, whereas other elements of cochlear microstructure are well preserved. The changes may be a manifestation of the severity of the otitis media produced.

Similar inner ear pathologic changes have been described following otitis media.\textsuperscript{7} Their clinical significance is uncertain, and the question of whether they resolve with time, or persist and lead subsequently to some degree of sensorineural hearing loss, is unanswered.

The pathologic inner ear effects of pneumococcal otitis media appear to be greater in the implanted than in the nonimplanted cochleas. In the implanted cochleas, the sites of nerve fiber losses were scattered throughout the basal turn and not simply localized to the sites of trauma. Trauma, in the absence of infection, has been shown to induce loss of neural elements, but this is confined to the region of the injury.\textsuperscript{4,10}

The histopathologic changes in the cochlea may be caused by the synergistic effect of severe pneumococcal otitis media, with either electrode implantation alone or with traumatic implantation. If the former is correct, then young children, given their susceptibility to otitis media, could be considered as unfavorable candidates for cochlear implantation because they would be at risk of developing recurrent pneumococcal otitis media and thereby sustaining significant nerve fiber losses, if they were fitted with an intracochlear electrode. Ongoing loss of neural elements would ultimately lead to a poor result from cochlear implantation.

However, before this conclusion can be drawn, other factors to explain the fiber and ganglion cell losses must be considered. The otitis media produced was extraordinarily severe, as evidenced by the inner ear pathologic changes seen even in the nonimplanted control cochleas. The severity of the infection was the result of the virulence enhancement of the pneumococcus, the large size of the inoculum, the presence of foreign material in the bulla, and because antibiotics were withheld. All of these factors would not be present in a clinical situation.

Other considerations include the possible inadequacy of the round window seal achieved by the technique of electrode insertion, the difference in biological response between cats and humans, and the small number of animals used in this study.

**CONCLUSIONS**

A severe pneumococcal otitis media in the cat was achieved by direct inoculation of a concentrated suspension of organisms, their retention in the bulla by Gelfoam, and virulence enhancement by intraperitoneal inoculation in mice before its application in the cat. In the nonimplanted cochlea pneumococcal otitis media caused minor pathologic changes in the inner ear and enhanced pathologic changes in the implanted cochlea. The small number of animals, however, did not permit us to separate the effects of insertion trauma, infection, or the combination of both.

**ACKNOWLEDGMENTS** — We thank Dr Roy Robbins-Browne of the Department of Microbiology, University of Melbourne, and Dr Peter Cole of the Veterinary Research Department of the Commonwealth Scientific and Industrial Research Organization, together with respective personnel for the preparation and supply of the bacteria used in this study. We are also grateful to them for their advice and suggestions.

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Title:
Pneumococcal middle ear infection and cochlear implantation

Date:
1987

Citation:

Persistent Link:
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