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Hearing loss after neurosurgery. The influence of low cerebrospinal fluid pressure

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Abstract
A prospective study was performed to investigate the effect of neurosurgery on hearing. Thirty-two patients underwent neurosurgery while 32 patients who had surgical procedures not involving puncture or drainage of the subdural space, served as a control group. In the neurosurgical group, a significant loss of hearing was observed in the immediate post-operative period, with recovery over one week. No average threshold shift was observed in the control group. It is suggested that following neurosurgery the mechanism of hearing loss results directly from a decrease in pressure and/or volume of the cerebrospinal fluid, which is reflected within the perilymphatic fluid, comparable to a transitory endolymphatic hydrops.

Key words: Cerebrospinal fluid pressure; Hearing loss, sensorineural; Neurosurgery

Introduction

The effect on hearing of changes in the cerebrospinal fluid (CSF) has been extensively investigated. It is well known that a communication between the perilymph and CSF exists via the cochlear aqueduct (CA). The patency of the CA decreases with age (Philips and Marchbanks, 1989; Marchbanks and Reid, 1990). Pressure variations in the CSF can be transmitted to the labyrinth if the CA is open, but only small volumes of fluid are thought to move, since the cochlea is a hard-walled compartment in which only the windows are compliant.

In cases of CSF pressure changes, the transmitted pressure variations can give rise to only small displacements within the cochlea, primarily of Reissners's membrane, because there are minimal pressure differentials between the endolymph and the perilymph. Such displacements, however, may affect hearing. The present study was undertaken to evaluate systematically the extent and course of any hearing loss after neurosurgery. Similar studies after spinal anaesthesia and operations for vestibular schwannomas have indicated the occurrence of such a hearing impairment (Walsted *et al.*, 1991a, b).

Single case reports, most lacking audiometric data, have been presented after myelography (Michel and Brusis, 1992), and after spinal anaesthesia (Vandam and Dripps, 1956; Arnvig, 1963; Lee and Roberts, 1978; Panning *et al.*, 1984; Noreng and Melsen, 1986; Wang, 1986; Walsted *et al.*, 1991b; Michel and Brusis, 1992).

Material and methods

The study included 32 patients undergoing craniotomy with neurosurgery (NS) and a control group of 32 patients undergoing non-neurosurgical operations. The NS operated patients consisted of 20 males and 12 females, with a median age of 53 years (range 19–72 years). In 20 cases an intracranial tumour was removed, six had operations for hydrocephalus with normal CSF pressure, two had operations for intracranial aneurysm and four for epilepsy.

The criteria for inclusion in the study were a cooperative patient, able and willing to join in the project and the ability to attend the audiology clinic on three occasions. An existing deficit or the presence of tinnitus did not result in exclusion.

The pre-operative assessment included otoscopy, pure tone audiometry at 125, 250, 500, 1000, 2000, 4000 and 8000Hz, speech audiometry and tympanometry. The patients were asked about existing audiological symptoms. Post-operatively two tests were scheduled, one soon after operation (between Days 1 and 3) and the other later (between Days 4 and 7). At the post-operative tests, the patients were asked if any new subjective symptoms had appeared; specifically hearing loss, fullness, tinnitus and dizziness or nausea. In 27 patients all planned audiometric sessions were carried out, whereas in five patients only one could be held. At the first post-operative test 31 patients and 62 ears were tested.

The control group included 16 males and 16 females, with a median age of 48 years (range 19–73 years). These patients had the same audiological examinations as the test group, before and after the surgical procedures which did not involve puncturing the dura. The criteria for inclusion in the control group were age (18 years or more) and an uneventful general anaesthesia. The surgical procedures carried out were: 11 cases – middle ear procedures; 10 cases – other otological operations; eight – thyroid surgery; and three – parotid surgery. A single post-operative audiogram was obtained in the control group, between Days 1 and 3, as these patients were

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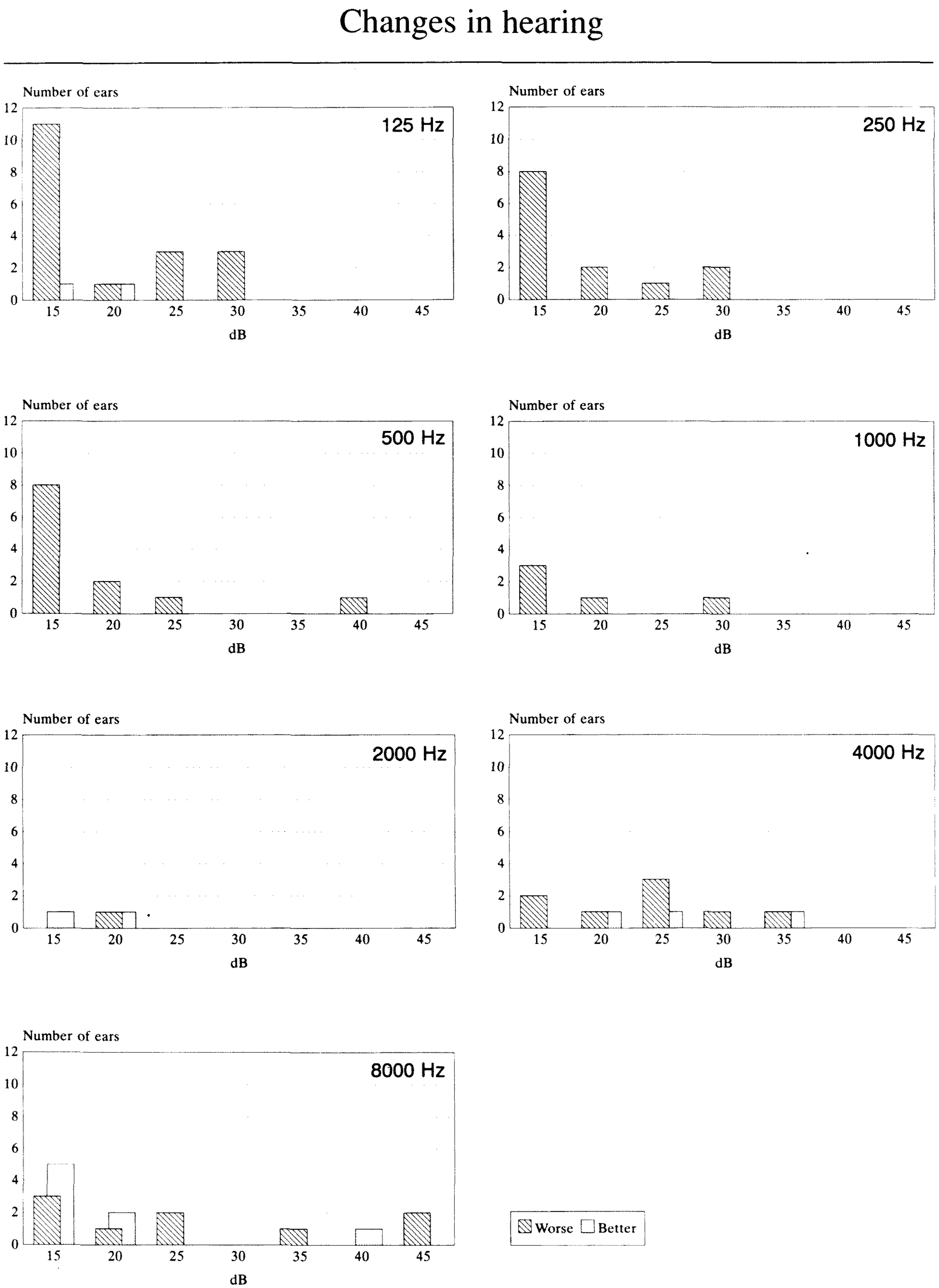

FIG. 1

The total number of threshold shifts equal to or exceeding 15 dB shown at each frequency for the neurosurgical group.

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TABLE I

PATIENTS WITH (⩾15 dB) AND WITHOUT (<15 dB) HEARING CHANGES IN THE NEUROSURGICAL (NS) GROUP AND THE CONTROL GROUP AT FIRST POST-OPERATIVE AUDIOMETRY*

	NS group (no. of patients = 31)	Control group (no. of patients = 32)
Threshold		
Unchanged	14 (45%)	27 (84%)
Worse	15 (48%)	4 (16%)
Better	5 (16%)	2 (6%)

*Three patients of the NS group and one of the control group had both ⩾15 dB better and worse thresholds.

discharged a few days after surgery. In the patients who had ear surgery or parotid surgery (if audiometry was not possible on the same side because of the surgery) only the contralateral ear was used for testing. In total 51 ears were tested in the control group.

All the audiological tests were performed under standardized conditions in soundproof booths using Madsen audiometers (OB 70 and OB 802) regularly calibrated in accordance with ISO 389 (1985) and ISO 7566–87, and supra-aural TDH 39 earphones with cushion MX41/AR, calibrated in accordance with ISO 389 (1991). The pure tone audiometry was performed by trained personnel using descending thresholds, with five dB steps in the following order: 1000, 2000, 4000, 8000, 500, 250, 125 Hz in accordance with the Hughson-Westlake method for pure tone audiometry. The speech audiometry used a Danish standard material 'Dantale', using monosyllabic numerals (digit triplets), calibrated for 50 per cent score for normal-hearing persons. The tympanometry was performed on a Madsen electroacoustic bridge (ZO 73 and ZS 77) calibrated in accordance with ISO 389 (1985).

Statistical calculations were performed for each ear separately to avoid problems of correlation between the two ears using the Rank-Spearman coefficient of concordance, with a level of significance of 0.05. According to our definition an individual patient was considered to have developed a significant hearing loss, if he or she showed a diminution in pure tone air conduction of at least 15 dB at any one of the investigated frequencies in one ear during the tests.

Results

Post-operative audiometric tests in the NS group revealed that 17 patients (53 per cent) post-operatively developed a significant hearing loss, according to our definition, during the first week. Figure 1 shows the total number and value of threshold shifts exceeding or equal to 15 dB for all the frequencies for the neurosurgery group. The threshold shift towards worse hearing is seen to be more common at the low frequencies 125, 250 and 500 Hz but also at 4 and 8 kHz, while the incidence of shifts at 1 and 2 kHz is small compared to the low and high frequencies. The distribution of the few shifts to better thresholds is skew, since nearly all appear in the treble. The results at the first post-operative audiometry are shown in Table I, for the neurosurgery and the control group.

TABLE II

NUMBER OF SIGNIFICANT THRESHOLD SHIFTS ⩾15 dB IN THE NS GROUP AND THE CONTROL GROUP AT THE FIRST POST-OPERATIVE TEST, RELATED TO NUMBER OF TESTED EARS

	NS group (62 ears)	Control group (51 ears)
Thresholds		
Worse	49 (0.79)	6 (0.12)
Better	6 (0.10)	3 (0.06)

In the whole NS group (62 ears) there were 49 significant threshold changes showing worse hearing at the first post-operative audiometry (Table II), while there were only six in the control group (51 ears).

Comparing the first post-operative threshold for all the patients with their pre-operative thresholds (separately for right and left ear), there was a significant ($p<0.05$) hearing loss in the left ear at all frequencies except 8 kHz and in the right ear a significant loss ($p<0.05$) at all frequencies except 4 and 8 kHz (Table III). The change in hearing tended to disappear when the pre-operative thresholds were compared to the second post-operative test, (Table III). The left ear showed on the whole more threshold shift towards worse hearing (nine out of 14 post-operative frequencies) when compared to the right ear (six out of 14 post-operative frequencies), suggesting that the left ear is more sensitive. In the controls no significant threshold shift could be detected from the group data ($p>0.05$) in right or left ear (Table III).

The average mean group thresholds pre- and post-operatively are shown for the 27 patients who completed all three audiometries, separately for right and left ear (Figure 2).

Changes in middle ear pressure from the pre- to first post-operative period were similar in the NS patients and the control group with significant changes ($p<0.05$), while no significant change in the middle ear pressure was present at the second post-operative test (Table IV).

In the NS group the speech reception threshold (SRT) although it had deteriorated significantly ($p<0.05$) in both ears at the first post-operative examination, was not statistically changed from the pre-operative examination at the second post-operative examination ($p>0.05$). No change was found in the control patients, Table V ($p>0.05$).

The post-operative subjective symptoms consisted, in the NS group, of complaints of post-operative nausea in two patients and in three of dizziness. Three felt fullness in the ears and eight a decreased hearing. Nine developed a new symptom of tinnitus. Three of the patients with the new post-operative tinnitus and three with the subjective hearing losses did not appear at the second check-up. Fifteen patients did not develop post-operative subjective

TABLE III

RESULTS OF COMPARISONS BETWEEN FIRST AND SECOND POST-OPERATIVE AUDIOGRAMS WITH THE PRE-OPERATIVE ONE IN THE NEUROSURGERY (NS) GROUP, AND BETWEEN POST-OPERATIVE AND PRE-OPERATIVE AUDIOGRAMS IN THE CONTROL GROUP (C)

	Frequency (Hz) 125	250	500	1000	2000	4000	8000
First post-operative audiogram							
NS, right ear	*	*	*	*	*	–	–
NS, left ear	*	*	*	*	*	*	–
Second post-operative audiogram							
NS, right ear	–	–	*	–	–	–	–
NS, left ear	*	*	–	*	–	–	–
First post-operative audiogram							
C, right ear	–	–	–	–	–	–	–
C, left ear	–	–	–	–	–	–	–

*Indicates a significant change (decrease) in hearing, ($p<0.05$); – indicates a nonsignificant change ($p>0.05$).
NS: cases undergoing neurosurgery; C: Control group.

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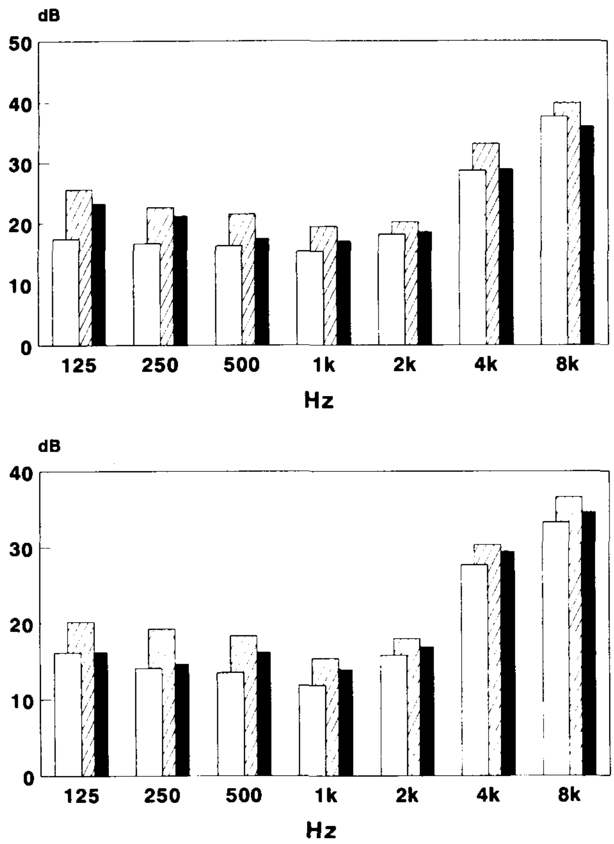

FIG. 2

Average threshold pre- and post-operatively for the neurosurgical group (right and left ear separately). Top: left ear; Bottom: right ear. White bar illustrates pre-operative thresholds, striped bar first post-operative audiogram and black bar the second post-operative audiogram.

symptoms. Only one patient in the control group had subjective symptoms, feeling her hearing was worse with a sensation of occlusion in the ears which was confirmed by audiometry and tympanometry (she was one of the patients mentioned above who developed a significant threshold decrease in both ears of 15 dB).

Discussion

This study has demonstrated audiometrically significant hearing deterioration after neurosurgery. It is presumed that this is a consequence of the loss of CSF. The change was most pronounced in the first three post-operative days, with a tendency to recover within one week.

These results confirm the findings in our previous reports (Walsted *et al.*, 1991a, b) in which it was hypothesized that the decrease in volume and pressure in the CSF, being transmitted to the perilymph via the cochlear aqueduct, could result in a relative endolymphatic hypertension, which could produce changes in hearing comparable to that of endolymphatic hydrops.

In the present study a control group was also examined audiologically before and after surgery which did not involve opening, and drainage of fluid from, the subdural space. No systematic threshold shifts occurred, which precludes the possibility that the hearing impairment is a non-specific result of surgery or anaesthesia.

Middle ear pressures were not affected by the type of surgery. The finding of a hearing loss in 45 per cent of the patient in the NS group and in 16 per cent in the control group, was comparable with an incidence of 49 deteriorated thresholds in the NS group and six in the control group which further demonstrates that the affected patients of the NS group had considerably more deterioration in their thresholds (Table II).

Besides the significant threshold deteriorations in the NS patients, a few patients improved after the operation, mostly in the treble range (Figure 1), and also in the control group a few patients had an improved threshold (Table I). No explanation for this finding is available in the literature or can be proposed. Moreover these improvements indicate that the change in hearing was not due to post-operative incapacitation after extensive neurosurgery (excluding problems caused by using a psycho-acoustic method).

The aetiology of the greater sensitivity of the left ear to that of the right ear is not known. The mean threshold of the pre-operative audiogram was slightly higher at all frequencies in the left ear when compared to the right ear (Figure 2); we do not know the reason for this since we only wanted to evaluate any changes in hearing after neurosurgery, and as the individual patient served as his own control, and we therefore included all patients without paying attention to their previous audiological condition. Asymmetry has also has been reported in tinnitus affecting the left ear one and a half times more often than the right ear (Shulman, A., 1991) which is explained by possible asymmetric noise exposure and by the relationship of tinnitus and hearing loss with localization to the poorer hearing ear. However, the greater sensitivity in the left ear in our study could indicate that unknown factors producing this asymmetry are functioning.

TABLE IV

CHANGES IN MIDDLE EAR PRESSURE IN THE NEUROSURGERY GROUP (NS) AND THE CONTROL GROUP (C)

	Middle ear pressure (mm)		
	Before op.	First post-op.	
NS, right ear	−25.5	−65.5	* (n = 29)
NS, left ear	−28.8	−67.4	* (n = 27)
	Before op.	Second post-op.	
NS, right ear	−17.8	0	– (n = 27)
NS, left ear	−35.4	−15.2	– (n = 26)
	Before op.	First post-op.	
C, right ear	−2.1	−55.2	* (n = 24)
C, left ear	−6.3	−52.8	* (n = 24)

*Indicates a significant change (p<0.05); – indicates a nonsignificant change (p>0.05).
NS: cases undergoing neurosurgery; C: control group.

TABLE V

SPEECH RECEPTION THRESHOLD CHANGES FROM PRE-OPERATIVE TO FIRST AND SECOND POST-OPERATIVE TESTS IN THE NEUROSURGICAL GROUP (NS) AND IN THE CONTROL GROUP (C)

	Speech reception threshold (dB)		
	Before op.	First post-op.	
NS, right ear	10.3	13.9	* (n = 31)
NS, left ear	12.7	17.1	* (n = 31)
	Before op.	Second post-op.	
NS, right ear	10.7	12.0	– (n = 27)
NS, left ear	12.9	13.6	– (n = 28)
	Before op.	First post-op.	
C, right ear	7.8	8.4	– (n = 24)
C, left ear	7.3	6.8	– (n = 22)

*Indicates a significant change (p<0.05); – indicates a nonsignificant change (p>0.05).
NS: cases undergoing neurosurgery; C; control group.

A hearing loss following a leak of CSF (after spinal anaesthesia, myelography or lumbar puncture) has also been reported in nine cases by Michel and Brusis (1992). Three of these patients experienced only partial or no recovery but the degree of permanent threshold change cannot be evaluated, because no pre-operative audiograms were carried out.

In our previous study (Walsted *et al.*, 1991b) which was based on audiograms of 34 patients before and after spinal anaesthesia, a minor but statistically significant general threshold shift was found at 500 Hz for the whole group. Furthermore one patient developed tinnitus, fullness and decreased hearing with recruitment in one ear. After she was given an epidural blood-patch to stop a suspected CSF leakage from the puncture site she had an immediate relief of the symptoms and the hearing was restored. This was confirmed by audiometry.

In our other study (Walsted *et al.*, 1991a) a more pronounced transitory deterioration of hearing was demonstrated in the contralateral ear after surgery for schwannomas. This was in accordance with the much greater loss of CSF during the latter operation.

The noninvasive equipment for monitoring the CSF pressure indirectly by tympanic membrane displacement (Marchbanks and Reid, 1990) was not available to us. For ethical reasons, we could not use ordinary invasive methods of measuring this pressure, and thus our conclusion that removal or bebulking of a tumour, with consequent leakage of CSF, results in lowering the intracranial pressure which directly affects the cochlea is a hypothetical one only.

The interaction in man between changes in CSF pressure and/or volume and hearing has not yet been established. Carlborg (1981) has shown that the cochlear aqueduct (CA) is the main route for the direct transmission of CSF pressure variations to the perilymph in the cat. Marchbanks and Reid (1990) and Phillips and Marchbanks (1989) observed tympanic membrane displacement as a result of variations of CSF pressure in man and were thus able to demonstrate a close relationship between the CSF pressure and the perilymphatic pressure. They have thus confirmed that the CA is patent in about 80 per cent of adults up to 40 years of age and in 30 per cent over 60 years.

The connection between hearing loss and low perilymphatic pressure has further been confirmed in some experimental studies. Funai *et al.* (1988) reported threshold elevations after perilymphatic aspiration, some combined with structural changes of Reissner's membrane, with bulging, collapse and rupture. Arenberg *et al.* (1988), using electrocochleography in guinea pigs, found summation potential changes indicating a change in the position of the membranes, which was confirmed histologically. Nomura *et al.* (1987) in histological studies showed acute hydrops after drainage of perilymph in guinea pigs. In each of the above studies, it was proposed that perilymphatic fistula and endolymphatic hydrops were closely related. In contrast Tono and Morizono (1992) could not find electrocochleographical evidence that the hearing impairment after perilymphatic aspiration was due to endolymphatic hydrops in guinea pigs.

However the hypothesis that the symptoms in perilymphatic fistulae are closely related to endolymphatic hydrops (Nomura *et al.*, 1987; Arenberg *et al.*, 1988; Funai *et al.*, 1988) is in keeping with our concept of low perilymphatic pressure as an aetiological factor for the hearing losses observed in our studies.

The conflicting results in the literature emphasize the need for further experiments to clarify the pathophysiological mechanisms in the development of hearing loss after CSF leakage.

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