Analysis of hearing loss after shunt placement in patients with normal-pressure hydrocephalus

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Object. Following shunt placement for treatment of normal-pressure hydrocephalus (NPH), several patients suffered hearing loss. The authors undertook a study to analyze this outcome.

Methods. Sixteen patients in whom NPH was diagnosed were treated by placement of a ventriculoperitoneal shunt. Their hearing was assessed pre- and postoperatively by using pure tone audiometry. Two thirds of the ears tested showed a postoperative hearing loss of more than 10 dB. Recovery of the hearing loss occurred 6 to 12 weeks after shunt placement in 75% of the ears examined.

Conclusions. Although shunt insertion for treatment of NPH results in a decrease in hearing, most of the loss can be recovered.

KEY WORDS • hearing loss • shunt • normal-pressure hydrocephalus • subdural hygroma

After several patients who had undergone shunt placement for treatment of presumed NPH complained about postoperative hearing loss, a study was initiated to investigate this finding further. Freshshunt and postshunt placement hearing was analyzed in patients with NPH. It has been hypothesized that changes in the CSF pressure affecting cochlear physiology may be caused by shunt insertion, and hearing disturbances after lumbar puncture have previously been described. Recently, Stoeckli, et al. reported one case of hearing disturbances after shunt implantation.

Clinical Material and Methods

Patient Population

This pilot study included 16 consecutive patients (nine men and seven women, ranging in age from 70 to 84 years of age) with the classic clinical triad of NPH and hydrocephalus, based on CT results. They were all treated with ventriculoperitoneal shunt placement. Different shunt types were used, as in the ongoing Dutch NPH study (Holter medium pressure; Phoenix Biomedical Corp., Valley Forge, PA. PS Medical medium pressure; Medtronic, Goleta, CA. Medos Hakim low and medium pressure; Codman, Raynham, MA).

Clinical Assessment

The clinical status of the patients was assessed using the Mini-Mental State Examination and gait scores before and 1, 6, and 12 weeks after operation. Computerized tomography scanning was performed before and 6 or 12 weeks after surgery as well as in cases of neurological deterioration. The bicaudate index was used to quantify and compare ventricular enlargement on the CT scans. Preoperatively, a lumbar infusion test was performed to measure the opening pressure and the CSF outflow resistance.

Hearing was assessed by pure tone audiometry preoperatively and 3 days and 1, 6, and 12 weeks postoperatively. Based on the assumption that changes in the ICP might influence inner ear function we analyzed only bone conduction. Bone conduction is the most appropriate test for inner ear function and is not affected by air conduction. Air conduction was not analyzed because it represents middle ear function and because it may be disturbed by intubation and consequent temporary eustachian tube dysfunction or fluids such as iodine solution or blood entering the external auditory canal during operation.

Audiometry revealed preoperative hearing loss in the higher frequencies in most patients, which is consistent with presbyacusis. At frequencies of 1000 Hz and higher no significant postoperative hearing losses were found in any patients examined, and therefore it was decided to analyze hearing only at 250 Hz and 500 Hz. These frequencies are not the most important for functional hearing; however, because of the preexisting hearing loss in the higher frequencies this additional hearing loss in the lower frequencies became relatively more important.

Statistical Analysis

For statistical analysis the mean of both frequencies

J. Neurosurg. / Volume 95 / September, 2001
Hearing loss after shunt placement for normal-pressure hydrocephalus

(250 and 500 Hz) was computed to obtain one value for each ear. Hearing losses before and after the operation were compared using the Wilcoxon matched-pairs signed-rank test. Different patient characteristics were tested for their effect on hearing loss with the Mann–Whitney U-test and the Wilcoxon rank-sum W-test.

Results

Only 31 of 32 ears could be evaluated; one ear could not be evaluated because of preexisting severe hearing loss. Because of randomization as part of ongoing trials, low-pressure valves with an opening pressure of 2 to 6 mm Hg were implanted in three patients; the remaining 13 patients received valves with opening pressures of 6 to 10 mm Hg. Eight patients developed subdural effusions; three of these patients were symptomatic and required evacuation of the effusion and temporary clipping of the drain. The bicaudate index, seen on control CT scanning, decreased in 70% of the patients. Sixty percent of the patients showed clinical improvement in the first week after shunt placement. This figure dropped to only 40% after 12 weeks.

The number of patients who could not undergo complete follow-up evaluation is considerable. After 12 weeks, six patients could not be evaluated properly because of complications related to shunt placement (overdrainage in three, intracerebral hematoma in one, and deep venous thrombosis in one) and intercurrent diseases or accidents (intracerebral hematoma in one, vital depression in two, and spinal fracture due to a fall in one). This high incidence of complications could be explained by the older age of this group of patients.

Figure 1 shows the difference in hearing loss in all ears before and after shunt placement. The decrease in hearing in the first week after shunt insertion was highly significant ($p < 0.005$). The mean difference was $11 \text{ dB}$, standard deviation $\pm 10 \text{ dB}$, maximum $30 \text{ dB}$, minimum $-5 \text{ dB}$. In the following weeks the mean difference decreased due to recovery of hearing losses. Looking more closely at the course of the postoperative hearing loss, different patterns could be identified. Four patterns were found based on the following criteria for change in hearing loss and consecutive recovery: 1) change in hearing loss was defined as an additional hearing loss of $10 \text{ dB}$ or more; and 2) a recovery was defined as a recovery of at least $10 \text{ dB}$ from the worst postoperative hearing loss. Figure 2 shows these four patterns with the absolute values of hearing loss. Twenty ears (64%) showed additional hearing loss, whereas hearing in 11 (35%) remained at the preoperative level. Hearing in six ears was recovered postoperatively, whereas in two it was not. The remaining 12 ears did not recover within the first 6 weeks. Hearing loss was not tested at 12 weeks for various reasons, such as overdrainage necessitating temporary clipping of the shunt, additional disease, or the patient’s recent entry into the study.

Overall, there was no significant difference in hearing between the left and right ears. In more than half of the patients the changes in both ears were related. Nine pair of ears showed an identical pattern on both sides with no hearing loss, hearing loss with recovery, or hearing loss without recovery. Four pair of ears showed asymmetrical patterns and three pair of ears showed asymmetrical recovery.

Three patient characteristics (receipt of a low-pressure valve, subdural effusions, and clinical improvement) were evaluated for their effect on the frequency of hearing loss. None attained a correlation sufficient to be statistically significant. Also, the preoperative level of hearing loss failed to be of prognostic value. The numbers were too small to test for factors influencing recovery of the hearing loss; however, Fig. 2 shows that the recovery of hearing loss is less likely to occur in cases with a large drop in hearing level after shunt placement.

Discussion

Although the number of patients in this study is small, we are confident in stating that shunt placement in patients with NPH causes hearing loss in more than half of the ears. The effect we found is obvious and involves incidence as well as extent of postoperative hearing loss. Recovery of hearing loss occurs 6 to 12 weeks, sometimes even longer, after shunt placement. A part of the hearing

J. Neurosurg. / Volume 95 / September, 2001
loss cannot be recovered, thus leaving the patient with seriously impaired hearing. Two patients were considered for hearing aids based on the results of their postoperative audiometry.

Because in nine patients both ears showed the same changes, whereas in seven they did not, one might assume that factors located in the ear itself as well as factors working on both ears, such as ICP, are important to establish the observed effect.

The number of patients was too small to identify statistically significant factors influencing the occurrence of hearing loss; however, there seemed to be a correlation between the occurrence of hearing loss and patient characteristics consistent with excessive drainage of CSF. This is consistent with earlier findings reported in the literature. Hearing loss has been described after lumbar puncture and also in various neurological conditions involving raised ICP. Several reports on hearing loss in patients who have received epidural anesthetic agents entailed a discussion about the pathophysiological mechanisms of this phenomenon, especially the issue of whether the hearing loss was caused by high or low ICP. It is probably the change in ICP that is causing the effect. Intracranial pressure changes can be conducted to the inner ear, if the cochlear aqueduct is patent. Results of anatomical as well as radiological studies show that the cochlear aqueduct is open and sometimes even enlarged, although its patency probably diminishes during the patient’s life. A study in cats showed a round membrane bulging into the middle ear if the ICP was artificially raised.

It is known that a balance exists between the pressure in the perilymphatic space and the endolymphatic space in the cochlea, as well as mechanisms to maintain this balance. If the cochlear aqueduct is patent, an ICP drop will be conducted to the perilymphatic space, causing an overpressure in the endolymphatic space, with subsequent dysfunction of the basilar membrane producing hearing loss. Recovery of hearing loss may be established by the usual mechanisms involved in maintaining or restoring the pressure balance in the cochlea. Persistent hearing loss could be explained by irreversible damage to the basilar membrane due to sudden and/or severe pressure drops or fluctuating ICP, as in effusions or drain dysfunction, hence preventing restoration of the pressure balance in the inner ear.

Older patients may be at risk than younger patients for postshunt placement hearing loss. First their pre-existing hearing level is frequently impaired and second their ears may be more vulnerable to pressure changes. Postshunt placement hearing loss was never observed in younger age groups. This is remarkable because the cochlear duct is more frequently patent at a younger age. It is possible that the same phenomenon exists in younger patients, but that it is not noticed. Younger patients, in general, have better (preoperative) hearing. A hearing loss of less than 30 dB is usually not noticed. If, however, a preoperative mild hearing loss exists, as is frequently the case in older patients, postoperative hearing may drop below the noticeable 30 dB. Preexisting hearing loss in the older age group is usually attributed to basilar membrane dysfunction or sclerosis of the hair cells on it. This may increase the vulnerability to pressure changes of the inner ear in the older patient.

Conclusions

The consequences for clinical practice of this postshunt placement hearing loss are limited, as most of the loss can be recovered. A small portion of the patients in whom a shunt is placed to treat NPH will develop a severe persistent hearing loss. It is, therefore, important to identify these patients before shunt placement or to know how to prevent hearing loss from occurring at all. Further investigations will be conducted to solve these problems.

References


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