Acta Oto-Laryngologica

Publication details, including instructions for authors and subscription information:
http://www.tandfonline.com/loi/ioto20

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Published online: 06 Jun 2015.

To cite this article: Munehisa Fukushima, Tadashi Kitahara, Arata Horii & Hidenori Inohara (2013) Effects of endolymphatic sac decompression surgery on endolymphatic hydrops, Acta Oto-Laryngologica, 133:12, 1292-1296

To link to this article: http://dx.doi.org/10.3109/00016489.2013.831480

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ORIGINAL ARTICLE

Effects of endolymphatic sac decompression surgery on endolymphatic hydrops

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Abstract

Conclusions: The present findings suggest that complete control of vertigo after endolymphatic sac decompression surgery (ESDS) does not always depend on improved vestibular function or reduced endolymphatic hydrops. Vertigo control is, however, associated with hearing stability.

Objective: Among surgical treatments for intractable Meniere’s disease, ESDS is performed to preserve and improve inner ear function. We examined the correlation between changes in vertigo frequency and neuro-otologic function to understand the condition of the inner ear in patients whose vertigo was completely controlled after undergoing ESDS.

Methods: This was a retrospective cross-tabulation study. Between 1997 and 2001, we treated 52 patients with intractable vertigo using ESDS and followed the patients regularly for 2 years. Postoperatively we evaluated and recorded changes in vertigo attack frequency, maximum slow phase eye velocity, worst hearing level, and glycerol test results according to modified American Academy of Otolaryngology–Head and Neck Surgery 1995 criteria.

Results: We found no correlation between vertigo control and vestibular function. There was also no correlation between vertigo control and negative conversion of the glycerol test. There was a significant correlation between vertigo control and hearing control.

Keywords: Intractable Meniere’s disease, endolymphatic sac drainage, surgical results, vertigo control

Introduction

Meniere’s disease, characterized by recurrent vertigo, fluctuating hearing loss, tinnitus, and aural fullness, is a common disease with an incidence of 17–46 cases per 100 000 population [1]. Despite the use of various medications, some of those patients are frequently prevented from participating in activities of daily life owing to frequent vertigo attacks and progressive sensorineural hearing loss. Patients with these severe symptoms are said to have intractable Meniere’s disease.

When conservative medical treatment has failed, surgical strategies such as endolymphatic sac decompression surgery (ESDS), vestibular neurectomy, and labyrinthectomy are considered depending on the conditions in each patient. ESDS, first performed by Portmann in 1927 [2], is still commonly used worldwide. One reason for its reputation as a mainstay surgical treatment for intractable Meniere’s disease is that – unlike other, ablative procedures – ESDS is nondestructive. Its purpose is to preserve and improve inner ear function.

Temporal bone studies in 1938 revealed that the otopathology of Meniere’s disease is endolymphatic hydrops [3,4]. ESDS drains the excessive endolymph and decompresses membranous labyrinthine pressure. It has been reported that ESDS accomplishes complete vertigo control in 42–88% of patients [5–10]. To date, no evidence has been presented that elucidates the mechanisms that produce the symptomatic relief resulting from shunting or decompressing the endolymphatic sac. Does ESDS improve inner ear function, resulting in good vertigo results? Alternatively, is...
it no more effective than a placebo operation [11]? To understand the inner ear conditions in patients for whom ESDS provided complete vertigo control, we examined the correlation between changes in vertigo frequency and neuro-otologic function.

**Material and methods**

**Patients**

The Ethics Committee of Osaka University Hospital approved this study (certificate no. 0421). The study is registered with ClinicalTrials.gov of the US Food and Drug Administration (certificate no. NCT00500474).

A total of 52 patients with a clinical diagnosis of Meniere’s disease according to the 1995 American Academy of Otolaryngology–Head and Neck Surgery (AAO-HNS) criteria were eligible for enrollment in the study [12]. Intractable Meniere’s disease was designated in cases where at least 6 months of various forms of medical and psychological management had been applied and had failed [1]. Between 1997 and 2001, we performed ESDS on 52 patients (19 men and 33 women; 42 unilateral and 10 bilateral) with a mean age of 47.1 ± 15.9 years (range 23–80 years). The duration of disease was a mean of 60.4 ± 8.9 months (range 6–256 months). The surgery was performed at Osaka Rosai and Osaka University Hospitals. Postoperatively, the patients were followed regularly for at least 2 years.

**Treatment**

The technical details of ESDS are as follows [10]. Simple mastoidectomy was performed, clearly exposing the endolymphatic sac in the area between the sigmoid sinus and the inferior margin of the posterior semicircular canal. If possible, the sac was exposed to include the rugose portion. It was opened with an L-shaped incision made along the posterior and distal margins of the lateral wall. It was then filled with 20 mg of prednisolone. While it was dissolving, we prepared a bundle of absorbable gelatin film with fan- and stick-shaped ends. These films were tied to each other with biochemical adhesive at the stick-shaped end. The fan-shaped end was then inserted into the sac. Small pieces of absorbable gelatin sponge soaked in a high concentration of dexamethasone (32 mg/4 ml) were placed inside and outside the sac lumen, which expanded with the bundle. The dexamethasone-containing sponges placed outside the sac were coated with the adhesive so dexamethasone was slowly delivered into the sac over a long period of time. The sponges comprised a natural sustained-release vehicle. The stick-shaped end extending out of the sac was fixed to the front edge of the mastoid cavity with the same adhesive so the incision into the sac remained expanded for as long as possible. The mastoid cavity was filled with relatively large pieces of absorbable gelatin sponge dipped in a steroid-antibiotic solution, after which the wound was closed with skin sutures.

**Functional examinations**

A definitive dizzy spell lasting more than 20 min was regarded as a Meniere’s vertigo attack according to the modified 1995 AAO-HNS criteria [12]. The frequency of vertigo was calculated based on the number of vertigo attacks during the 6 months before surgery. Frequency after surgery was calculated based on the number of vertigo attacks during the 6 months between 18 and 24 months after surgery. The data could be obtained for all the 52 cases.

Vestibular function was measured by a caloric test using electronystagmography (ENG) twice, during the 6 months before surgery and during the 6 months between 18 and 24 months after surgery. For the test, the external auditory canal was irrigated, in turn, with 30°C cold water and 44°C hot water (20 ml) for 10 s each. The induced nystagmus was recorded using ENG in a dark, open-eyes situation. Based on the mean maximum slow-phase eye velocity (max-SPEV) on the treated side, the max-SPEV before treatment/ max-SPEV after treatment ratio was calculated. Values > 1.1 were recognized as an improvement in vestibular function during the second follow-up year [10]. It is not easy to evaluate vestibular improvement using changes in caloric responses. Using the furosemide test criteria to detect vestibular endolymphatic hydrops, changes of ± 10% were considered positive [13]. The data could be obtained from 50 of 52 cases, because of the lack of paired examinations in two cases.

Hearing function was measured by a pure-tone audiometer and was evaluated based on the four-tone average formulated by (a + b + c + d)/ 4 (a, b, c, d are hearing levels at 0.25, 0.5, 1.0, and 2.0 kHz, respectively) according to the modified 1995 AAO-HNS criteria [12]. The worst hearing level during the 6 months before surgery was adopted as the hearing level before treatment (‘before’). The worst hearing level during the 6 months between 18 and 24 months after surgery was adopted as the hearing level at the second follow-up year (‘after’). Differences in hearing levels > 10 dB before and after treatment were regarded as ‘better.’ Differences smaller than –10 dB were regarded as...
worse and values in between better and worse as no change. The data could be obtained for all the 52 cases.

We performed a glycerol test to detect endolymphatic hydrops twice: during the 6 months before surgery and during the 6 months between 18 and 24 months after surgery [14]. This test is considered positive in pure-tone audiometry if there is a $\Delta 10$ dB improvement at two or more frequencies between 0.25 and 2.0 kHz. The data could be obtained for 25 of 50 cases, because negative results at the first glycerol test before surgery were found in 15 cases and a lack of paired examinations was found in another 10 cases. Therefore, the positive ratio at the first glycerol test was 62.5% (25/40).

**Table I.** Two-way table of the ratio of control of vertigo and vestibular function.

<table>
<thead>
<tr>
<th>Factors</th>
<th>Complete control</th>
<th>Others</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Improved max-SPEV</td>
<td>20</td>
<td>3</td>
</tr>
<tr>
<td>Unchanged and worse max-SPEV</td>
<td>19</td>
<td>8 (NS)</td>
</tr>
<tr>
<td>(b)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Improved and unchanged max-SPEV</td>
<td>37</td>
<td>10</td>
</tr>
<tr>
<td>Worse max-SPEV</td>
<td>2</td>
<td>1 (NS)</td>
</tr>
</tbody>
</table>

max-SPEV, maximum slow-phase eye velocity; NS, not significant.

**Table II.** Two-way table of the ratio of control of vertigo and negative conversion of endolymphatic hydrops.

<table>
<thead>
<tr>
<th>Glycerol test</th>
<th>Complete control</th>
<th>Others</th>
</tr>
</thead>
<tbody>
<tr>
<td>+    → −</td>
<td>14</td>
<td>3</td>
</tr>
<tr>
<td>+    → +</td>
<td>4</td>
<td>4 (NS)</td>
</tr>
</tbody>
</table>

NS, not significant.

**Statistical analysis**

Data were analyzed using a $2 \times 2$ contingency table method. Each correlation was assessed using the $\chi^2$ test or Fisher’s test. All the data from treatment results were treated statistically with the use of StatView, version 4.0 (SPSS, Chicago, IL, USA). All reported $p$ values were two-sided, and those $<0.05$ were considered significant.

**Results**

Surgical results, including the number of patients with complete control of vertigo (41/52: 77.8%), those with vestibular improvement (23/50: 46.0%), those with negative conversion of glycerol test (17/25: 68.0%), and those with hearing improvement (25/52: 48.1%) are shown in Figure 1. Duration of disease indicates the period between the onset of symptoms and the date of surgery.

As shown in Table I, there was no significant correlation between complete control of vertigo and improvement ($p = 0.1895$) or improvement/preservation ($p = 0.5337$) of max-SPEV (i.e. vestibular function). There was also no significant correlation between complete control of vertigo and negative conversion of the glycerol test ($p = 0.1563$) (Table II). Finally, there was no significant correlation between complete control of vertigo and hearing improvement ($p = 0.1199$) (Table IIIa). Table IIIb shows significant correlation between complete control of vertigo and hearing improvement/preservation ($p = 0.0075$). Additionally,
there were tendencies in relationships between vestibular and glycerol test improvements \((p = 0.0538)\) (Table IVa) and statistical significances in relationships between vestibular and hearing improvements \((p = 0.0217)\) (Table IVb).

**Discussion**

We examined correlations between the frequency of vertigo and neuro-otologic function in our patients after ESDS to elucidate the mechanisms of the ESDS effects on symptomatic vertigo relief. Judging from Tables I and II, vertigo could be completely controlled after ESDS regardless of the improvement of vestibular function or reduction of endolymphatic hydrops. Uno et al. reported that some patients were still hydrops-positive through the inner ear MRI with intratympanic administration of gadolinium despite complete control of vertigo after ESDS [15]. This finding supports our results that complete control of vertigo does not always depend on improving vestibular function or diminishing endolymphatic hydrops.

We suggest that there are at least three steps by which ESDS can cure intractable Meniere’s disease: 1) prevent progression of endolymphatic hydrops; 2) reduce endolymphatic hydrops; 3) improve the function of vestibular hair cells and/or neurons. All three steps have the potential to stop vertigo attacks, but only the third step can facilitate recovery of the caloric response and hearing level. Roughly speaking, according to the present data, ESDS can help 80% of intractable Meniere’s patients achieve results via one of these three steps, but in only half of them can the third step be successfully accomplished.

Judging from the data in Table III, complete control of vertigo after ESDS was accomplished following improvement/preservation of the hearing level. Huang and Lin [5], Moffat [6], Gibson [7], Gianoli et al. [8], Kitahara and Goto [9], and Kitahara et al. [10] reported 2-year results with their modified ESDS. They achieved complete control of vertigo in 89%, 42%, 57%, 60%, 85%, and 88% of patients, respectively, and hearing improvement/preservation in 33/54, 15/56, 5/39, 60/22, 32/55, and 49/44% of patients, respectively. These findings indicate that the proportion of patients with hearing improvement/preservation is likely to be similar to the proportion of those with complete vertigo control, except for Moffat’s study [6]. After ESDS, the first and second steps outlined earlier would take place most often in the inner ear, resulting in complete control of vertigo and hearing preservation. Judging from the data in Table IV, hearing improvement after ESDS could be accomplished by the direct effect of ESDS on the third step and/or its effect on it indirectly. This might be explained by the diminishing vertigo-induced disease stress and stress-induced plasma vasopressin, resulting in the good condition of the inner ear [16–18]. Altogether, intractable vertigo attacks and progressive hearing loss are controlled when ESDS prevents or reduces the severity of endolymphatic hydrops. The caloric response and hearing level undergo recovery only when ESDS facilitates functional improvement of the inner ear hair cells and/or neurons.

Our study has limitations. First, we have modified the original ESDS [2] to what we used in the present study [10]. Our ESDS includes several interventions not found in the original ESDS protocol. These changes were needed to accomplish an appropriate study design. In later communications, we plan to include several controls to increase data reliability. Second, we have not yet found the best way to evaluate vestibular improvement that obtains international consensus. Based on a previous report of the furosemide test criteria [13], we deemed changes in caloric responses of ± 10% as positive. The third limitation is that both the glycerol test and electrocochleogram have approximately 60% sensitivity at most [14,19]. In recent reports, endolymphatic hydrops imaging was demonstrated using gadolinium-enhanced inner ear MRI [15,19,20]. The sensitivity of this imaging analysis for endolymphatic hydrops was more than 90%, better than that of neuro-otologic exams. Further studies including gadolinium-enhanced inner ear MRI could evaluate the effects of Meniere’s treatments on endolymphatic hydrops.

**Conclusion**

According to the surgical results and neuro-otologic data, complete freedom from vertigo after ESDS does not always depend on improved vestibular function or a reduction of the severity of endolymphatic hydrops.
Acknowledgments

The authors wish to thank Dr Michiko Shuto, a registered statistician (certificate no. 62720218), for helpful advice on statistical analysis. This study was supported in part by a Health Science Research Grant for Specific Disease from the Ministry of Health, Labour and Welfare, Japan (2011-2013).

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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