Audio-Vestibular Manifestations in Radiologically Confirmed Enlarged Vestibular Aqueduct in Congenital Non-Syndromic Sensorineural Hearing Loss in Egypt

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Abstract

Background: Enlarged vestibular aqueduct (EVA), the commonest radiological finding in children with sensorineural hearing loss is associated with variable auditory and vestibular symptoms.

Aim of the Work: Is to determine vestibular findings in patients with EVA.

Patients and Methods: 20 patients diagnosed as having sensorineural hearing loss and EVA as the study group, 20 healthy subjects matched for age and sex participated as the control group. Both groups underwent: Full history taking, General and neurological examination, Full ENT examination, pure tone audiometry, tymanometry, high resolution computed tomography (HRCT), vestibular evoked myogenic potentials (VEMP), caloric test and computerized dynamic posturography.

Results: EVA was bilateral in 14 patients and unilateral in 6 patients. The VEMP amplitude of wave p13-n23 was higher in ears with EVA and it correlated positively with its size, p13-n23 latencies did not differ significantly from the control group, caloric test showed absent response in 4 patients, unilateral weakness in 6 patients and normal results in 10 patients, there was no statistical significant difference regarding computerized dynamic posturography results.

Conclusion: Results showed larger VEMP amplitude, which is indicative of third window lesion. Some patients showed Caloric test abnormalities which indicates a peripheral vestibular lesion. VEMP, caloric test and computerized dynamic posturography testing are effective in evaluating patients with EVA.


Introduction

MORE than 200 years ago, temporal bone dissection by Carlo Mondini revealed wide vestibular aqueducts in patients with cochlear dysplasia (Mondini’s dysplasia). In 1978, Valvassori and Clemis [1] were the first to use imaging, and to note an association between enlargement of the vestibular aqueduct and sensorineural hearing loss (SNHL).

The vestibular aqueduct (VA) is a bony canal in the posterior temporal bone that runs from the vestibule to the posterior cranial fossa, and contains the endolymphatic duct that runs from the vestibule to the endolymphatic sac and the vestibular aqueduct vein [2].

The clinical presentation of EVA varies markedly [3,4]. Hearing loss is not always present at birth, it is usually diagnosed in childhood. Hearing loss is usually sensorineural but it is could be conductive or mixed, it is usually progressive but it may be sudden and fluctuating, it could be unilateral or bilateral [1].

Vestibular symptoms also varies, its incidence is reported to be 4-48%. It may be precipitated by minor head trauma or vigorous physical activity [4]. The characteristics of hearing affection have been studied extensively in patients with EVA but not much information exists regarding vestibular affection in those patients [5].

Aim of this study is to detect vestibular findings in patients with EVA.

Subjects and Methods

The study was performed in Audiology Outpatient Clinic, Kasr El-Aini Hospital, in the period
12 Audio-Vestibular Manifestations in Radiologically Confirmed between May 2009 and December 2010. The study was performed on 2 groups, the study group consisted of 20 children suffering from non-syndromic sensorineural hearing loss and diagnosed as having EVA by high resolution computed tomography (HRCT). The control group consisted of 20 healthy volunteers matched for age and sex.

Both groups were subjected to:
- Full history taking: To determine the onset of symptoms.
- General and neurological examination.
- Full ENT examination with careful otoscopic examination.
- Audiological evaluation:

All patients included in this study was subjected to:
- Pure tone audiometry: Using two channels clinical audiometer: Orbiter 922 calibrated according to ISO standards.
  - Air conduction in the frequency range of 250-8000 Hz,
  - Bone conduction in the frequency range of 500-4000Hz.
- Speech audiometry:
  - Speech reception threshold (SRT) using Arabic spondee words.
  - Word discrimination score (WDS) using Arabic phonetically balanced words.
- Immittancemetry: Using GSI 33 version II (Grason Stadler middle ear analyzer) calibrated according to ISO standards. Single component, single frequency tympanometry with a probe tone of 226 Hz. To exclude middle ear abnormalities. Testing of acoustic reflex threshold, for the ipsilateral and contralateral elicited reflexes, using pure tones at frequencies 500, 1000, 2000, 4000 Hz.
- Radiological evaluation:
  - GE Light Speed Plus multislice machine (4 slices), slice thickness was 1.25mm, with interslice gap 0mm and bone window setting with bone filter (edge enhancement ± reconstruction). Non contrast computerized tomography (CT) scan of the petrous temporal bone in axial and coronal planes was done to all patients included in the study. The radiological dimensions of the vestibular aqueduct (VA) were measured at both the operculum and the midpoint on the right and the left sides. CT scans were also checked as a whole for any other congenital anomalies or pathologies.
- Vestibular testing:
  a- VEMP testing: Using AURIS Synapsys calibrated according to ISO standards. Patients were placed in the supine position on a gurney. They were instructed to turn and hold their heads as far as possible towards the side contralateral to the stimulated ear. Surface EMG activity was recorded by means of Ag\AgCl electrodes. The active electrode was placed over the middle portion of the ipsilateral SCM muscle. The reference and the ground electrodes were placed over the upper sternum and the on the midline forehead, respectively. Auditory stimuli consisted of clicks, presented to the ear ipsilateral to the contracted SCM muscle. The intensity was 100 dB nHL. EMG responses from each side were amplified, bandpass-filtered (20 Hz to 2 k Hz) and averaged by a Neuro-ropack evoked potential recorder. Analysis time was 1 00ms. The responses of 250 sound presentations are averaged. Thereafter, the mean peak latency (in milliseconds) and peak- to-peak amplitude (in microvolts) of each peak (p13 and n23) were measured. The potentials (p13-n23) are known to be of vestibular origin [6,7].
  b- Computerized Dynamic Posturography testing: Using NEUROCOM SMART Equi Test Balance Master (NeuroCom International, Inc., Clackamas) calibrated according to ISO standards. The sensory organization test (SOT) procedure requires patients to stand on a pressure-sensitive, dynamic tilted force plate facing a sway-referenced visual surround, instructed and strapped into a safety harness to prevent injury in the event of a loss of balance. The SOT comprises 3 trials for each of 6 conditions representing different aspects of balance. SOT 1, eyes open, fixed surface and visual surroundings; SOT 2, eyes closed, fixed surface; SOT 3, eyes open fixed surface, sway referenced visual surround; SOT 4, eyes open, sway referenced surface, fixed visual surround; SOT 5, eyes closed, sway referenced surface, fixed visual surround; SOT 6, eyes open, sway referenced surface and visual surroundings [7].

Statistical analysis:

Statistical analyses were done using SAS software, version 6.0 (SAS Institute, Cary, North Carolina). The Fisher extract was used to compare percentages and 95% CIs were calculated. All p-values less than 0.05 was considered statistically significant.

Results

The study group included 20 patients, 12 males (60%) and 8 females (40%), suffering from non-syndromic sensorineural hearing loss since child-
hood with enlarged vestibular aqueduct which was diagnosed by high resolution computed tomography (HRCT). Their age ranged from 6 to 28 years with mean age of 13.20 years ± 6.07.

The control group comprised 20 subjects, 10 males (50%) and 10 females (50%), with normal auditory function and normal radiological dimensions of the vestibular aqueduct. Their age ranged from 10 to 27 years with mean age of 15.70 years ± 5.03. There was no statistical significant difference between the age of the case and control groups (p-value=0.210) Table (1).

Table (1): Mean, standard deviation (S.D) and range of age in years in the whole study group.

<table>
<thead>
<tr>
<th>Group</th>
<th>Age</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
</tr>
<tr>
<td>Case</td>
<td>13.20</td>
</tr>
<tr>
<td>Control</td>
<td>15.70</td>
</tr>
<tr>
<td>p-value</td>
<td>0.210</td>
</tr>
</tbody>
</table>

There was no statistically significant difference between the case group and normal controls as regards the age (p>0.05).

Audiological findings:

All the patients in the study group had SNHL. They had normal tympanometry results reflecting normal middle ear function. The severity of hearing loss as determined by pure tone averages are presented in Table (2) (Fig. 1).

Table (2): Number of ears included in each degree of hearing loss in patients with EVA.

<table>
<thead>
<tr>
<th>Degree of SNHL</th>
<th>Slight</th>
<th>Mild</th>
<th>Moderate</th>
<th>Ms</th>
<th>THL</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>4</td>
<td>2</td>
<td>10</td>
<td>8</td>
<td>10</td>
<td>34</td>
</tr>
<tr>
<td>%</td>
<td>11.76%</td>
<td>5.88%</td>
<td>29.41%</td>
<td>23.54%</td>
<td>29.41%</td>
<td>100%</td>
</tr>
</tbody>
</table>

Radiographic findings:

The 20 patients with vestibular aqueduct abnormality were diagnosed by HRCT scan, the EVA was bilateral in 14 (70%) and unilateral in 6 (30%) patients. So the number of ears with EVA was 34 and the number of unaffected ears 6. HRCT did not reveal any associated congenital anomalies.

The range of the dimensions of the vestibular aqueduct at the midpoint (MP) and the operculum (OP) in ears with EVA, unaffected ears and the control group are presented in Table (2). There was a high statistical significant difference between the ears with EVA, and the control group as regards the mean of vestibular aqueduct dimensions at the midpoint (MP) and operculum (OP) (p-value=0.000), which were larger in the ears with EVA than the control group, Tables (3,4) respectively (Fig. 2).

Right VA operculum = 6.0mm and midpoint = 3.8mm. Left VA operculum = 5.2mm and midpoint = 3.8mm

![CT scan of a female child aged 10 years with bilateral EVA.](image)

Fig. (2): CT scan of a female child aged 10 years with bilateral EVA.

Table (3): t-test results of the dimensions of the vestibular aqueduct at both the midpoint (MP) and the operculum (OP) in millimeters (mm) between the ears with EVA of the cases group and the control group.

<table>
<thead>
<tr>
<th></th>
<th>Ears with EVA No=12</th>
<th>Unaffected No=6</th>
<th>Control No=40</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>VA Mp (mm)</td>
<td>Min</td>
<td>Max</td>
<td>Min</td>
<td>Max</td>
</tr>
<tr>
<td></td>
<td>0.50</td>
<td>3.80</td>
<td>0.70</td>
<td>0.70</td>
</tr>
</tbody>
</table>

Table (4): t-test results of the dimensions of the vestibular aqueduct at both the midpoint (MP) and the operculum (OP) in millimeters (mm) between the ears with EVA of the cases group and the control group.

<table>
<thead>
<tr>
<th></th>
<th>Ears with EVA No=34 ears</th>
<th>Control group No=40 ears</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MP (mm)</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td></td>
<td>1.49</td>
<td>0.93</td>
<td>0.63</td>
</tr>
<tr>
<td>OP (mm)</td>
<td>2.42</td>
<td>1.33</td>
<td>1.01</td>
</tr>
</tbody>
</table>

*p-value highly significant <0.01.

Fig. (1): Number of ears included in each degree of hearing loss.
Vestibular findings:

In the 20 patients included in the study, there were 6 (30%) patients reported vestibular symptoms represented by frequent episodes of vertigo and nausea related mainly to physical hyperactivity in 4 (20%) patients and the other 2 (10%) patients suffered from imbalance.

a- VEMP findings: p 13-n23 latencies and VEMP amplitude were recorded. There was statistically significant difference between the ears with EVA and the control group as regards the mean of VEMP amplitude which was larger in ears with EVA, p13-n23 latencies was not statistically significant different between ears with EVA and the control group (p = 1.000 and 0.095 respectively), Fig. (3) and Table (5).

Table (5): *t*-test results of P 13, N 23 latencies and VEMP amplitude between the ears with EVA and control group.

<table>
<thead>
<tr>
<th></th>
<th>Ears with EVA</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No=34</td>
<td>No=40</td>
</tr>
<tr>
<td>p13 latency (msec)</td>
<td>12.57 2.34</td>
<td>13.17 1.66</td>
</tr>
<tr>
<td>n23 latency (msec)</td>
<td>18.99 3.19</td>
<td>21.00 2.25</td>
</tr>
<tr>
<td>Amplitude (uv)</td>
<td>72.12 48.67</td>
<td>29.85 9.98</td>
</tr>
</tbody>
</table>

**p*-value statistically significant <0.05.

b- Computerized Dynamic Posturography testing:

When comparing the SOT of computerized dynamic posturography between the cases and control There was no statistically significant difference between the study group and the control group, Table (7).

Table (7): *t*-test results of computerized dynamic posturography between the cases and control groups.

|              | Cases group No=20 | Control group No=20 |  | p-value |
|--------------|--------------------|---------------------|  |         |
|              | Mean SD            | Mean SD             |  |         |
| SOT1         | 92.93 2.01         | 94.18 3.23          | 0.161 |
| SOT2         | 91.40 2.99         | 92.59 4.07          | 0.272 |
| SOT3         | 89.22 3.56         | 91.94 3.57          | 0.172 |
| SOT4         | 83.77 4.62         | 84.97 5.18          | 0.596 |
| SOT5         | 67.37 10.82        | 74.27 12.49         | 0.088 |
| SOT6         | 64.61 10.95        | 68.44 11.01         | 0.570 |
| CS           | 78.50 4.73         | 81.20 4.24          | 0.323 |

*p*-value significant <0.05.

There was significant positive correlation between vestibular aqueduct diameter at the midpoint and operculum and amplitude of VEMP (r=0.461, p=0.041 and r=0.588, p=0.006 respectively). There was no correlation between the size of the VA and the latency of P13-N23, degree of hearing loss, caloric test results and computerized dynamic posturography results.

Discussion

EVA is a distinct entity in the spectrum of congenital inner ear anomalies and known to be associated with hearing loss and vestibular symptoms in the pediatric population [7].

In this study we investigated 20 patient diagnosed as having sensorineural hearing loss and EVA.

The number of males 12 was greater than the number of females 8. Some studies found female
preponderance, but others reported the opposite [8]. Our study was in favour with the study group comprised 16 patients (32 ears) suffering from non-syndromic SNHL since childhood. The study group was divided according to VA size into three groups: Group A, B and C. Group A included 6 ears (4 patients) with EVA, group B included 11 ears (7 patients) with borderline EVA and group C included 15 ears (9 patients) with normal VA size. Similarly, there were no statistically significant differences between the three groups as regards laterality, degree of hearing loss and audiometric configuration. There was no correlation between VA midpoint & operculum and different variables (age and average pure tone thresholds). However, a significant correlation between VAmidpoint & operculum was found. EVA was diagnosed in 6 out of 32 ears (18.75%) in the study sample. EVA size was not related to the degree of hearing loss or configuration [9].

In our patients the severity of hearing loss ranged from slight to total with 11.76% slight, 5.88% mild 29.41% moderate, 23.54% moderately severe and 29.41% total SNHL (Table 1). Arjmand and webber [10] found that the threshold hearing level in patients with EVA ranged from none to profound, with 65% of ears having mild or moderate hearing impairment. The mechanism of hearing loss is a breach in homeostasis of inner ear electrolytes and the transmission of cerebrospinal fluid pressure fluctuations via the patent VA [11].

The definition of EVA is controversial, authors have defined VA enlargement as a diameter greater than or equal to 1.4, 1.5, 2.0, or 4.0mm [12-14]. The place at which the VA is measured is also variable, from midway between the common crus and external aperture to the external pore [15]. Other authors have defined the EVA by comparing it to the posterior SCC [16] or the diameter of the facial nerve [17].

In this study EVA was bilateral in 14 (70%) patients and unilateral in 6 (30%) patients. Studies have demonstrated that the most frequent finding is bilateral EVA. In different studies the incidence of bilateral EVA ranged from (55% to 94%) [1, 5, 18, 19]. Was also in favor with Abou Elew et al. [20] had bilateral sensorineural hearing loss of variable degrees ranging from slight SNHL to total hearing loss. EVA was found in HRCT of four patients. Of the 4 patients 2 patients had bilateral EVA and 2 patients had unilateral EVA. Seven patients had borderline EVA, 4 of them had bilateral borderline EVA and the remaining 3 patients had 6 of them had bilateral normal VA and the other 3 had unilateral normal VA.

As regards rotary chair testing results of the current study, all patients had within normal results for phase and asymmetry at all frequencies, all patients had normal gain results at all frequencies except only one patient who had abnormal gain results at 0.16 Hz, 0.32 Hz and 0.64 Hz.

While Ishiyama et al. [21] found that rotary chair testing in enlarged vestibular aqueduct (EVA) patients revealed impaired VOR on low frequency sinusoidal rotation and a shortened VOR time constant in some patients with normal responses at higher frequencies of sinusoidal rotation indicating that there was subtotal loss of vestibular function. Some other patients revealed normal VOR to rotational testing.

In the current study 6 (30%) patients had vestibular symptoms in the form of vertigo and nausea in 4 (20%) patients and imbalance in 2 (10%) patients. Many authors found that vestibular symptoms ranged from 12% to 46.9%, the complaints were vertigo, imbalance and motor delay [22,23,3]. Yetiser et al. [24] found only two patients with Meniere-like vertigo. Faye et al. [25] found that one patient complained of positional vertigo and imbalance may be caused by an underdeveloped vestibular [24]. Al Attia et al., reported an 18-year-old Egyptian male with Pendred Syndrome (PDS). [3] was in favor with our study.

In our patients VEMP findings were larger VEMP amplitude in ears with EVA (34 ears) when compared to either control group (40 ears) or unaffected ears (6 ears) (Tables 6,7,8 and 9). There was no statistical significant difference between ears with EVA when and the control group regarding p13-n23 latencies, (Table 6).

Zhou and Gopen [9] found that ears with EVA had higher VEMP amplitude with no difference in p 13-n23 latencies between ears with EVA and ears without EVA.

Many authors found this pattern of VEMP outcome in other third window lesions as superior semicircular dehiscence syndrome, in which high VEMP amplitude [26-29].

Yetiser [24] postulated that vestibular dysfunction in all patients with EVA may be due to vestibular end-organ pathology.

When comparing the computerized dynamic posturography regarding SOT parameters between the study group and the control group. There was
no statistically significant difference between the two groups, unfortunately, these results obtained could not be compared to others for the scarcity of researches in this point [30].

In this study there was significant positive correlation between the size of EVA and VEMP amplitude but there was no correlation between the size of EVA and degree of SNHL. VEMP latencies, caloric test results and computerized dynamic posturography. Antonelli et al. [17] reported significant correlation between VA size and severity of hearing impairment in patients with EVA and cochlear dysplasia, several other reports have failed to demonstrate a relationship between VA size and the degree of sensorineural hearing loss [31,32].

The pathogenesis of hearing loss may be due to increased cerebrospinal fluid pressure causing hair cell damage. Others postulate that hyperosmolar fluid may reflux into the cochlea causing damage to the hair cells. Vestibular dysfunction may have a similar etiology and some argue that reflux of hyperosmotic fluid into the basal end of the cochlear duct may directly elicit vertigo. Degeneration of vestibular hair cells due to osmotic and chemical imbalance may be another mechanism of injury [23].

In the present study, the study group composed of 20 patients suffering from non-syndromic sensorineural hearing loss since childhood with enlarged vestibular aqueduct were diagnosed by HRCT scan. The number of males 12 was greater than the number of females 8. Some studies found female preponderance, but others reported the opposite [11].

In this study, 14 patients (70%) had bilateral EVA (28 ears) and 6 patients (30%) had unilateral EVA (6 ears) so the study comprised of 34 ears affected with EVA (Table 3). Studies have demonstrated that the most frequent finding is bilateral EVA. In different studies the incidence of bilateral EVA ranged from (55% to 94%) [11,23]. As regards the HRCT scan study of the affected 34 ears, the VA operculum ranged from 1.5mm to 6mm with the mean of about 2.42mm+1.33 and the midpoint range was from 0.5mm to 3.8mm with average of 1.49mm+0.93 (Table 4).

The patients in the study were evaluated by axial and coronal HRCT, Dimopoulos et al. [12] Pollock [19] found that the axial transverse view was sufficient for visualization of the VA. On the other hand, Murray et al. [13] reported that coronal CTs allowed better visualization and it gave consistent measurement of the VA.

The definition of EVA is controversial, authors have defined VA enlargement as a diameter greater than or equal to 1.4 mm [33], 1.5mm [14], 2.0mm [14], or 4.0mm 31.

The age ranged from 6 to 28 years with a mean age of 13.20 years+6.07 (Table 1).

As regards the audiometric results of the current study, all patients included in the study were diagnosed with sensorineural hearing loss. The severity of hearing loss as determined by pure tone averages (of 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz) were; 4 ears (11.76%) demonstrating slight SNHL, 2 ears (5.88%) with mild SNHL, 10 ears (29.41%) with moderate SNHL, 8 ears (23.53%) with moderately severe (MS) SNHL and 10 ears (29.41%) with profound SNHL.

There was no statistically significant correlation between size of the operculum or mid point with pure tone thresholds at different frequencies (p>0.05). These results are in accordance with Wageih et al. [5] who found that there was no statistically significant correlation between size of the operculum or mid point with pure tone thresholds at different frequencies. And also in accordance with the results of Pinto et al. [29] who found that there is no correlation between the level of hearing loss and width of the aqueduct.

Also in accordance with the results of Benton et al. [15], who measured the diameters of EVA cases in the axial planes of the temporal bone. Who that the linear regression analysis between the EVA and hearing loss showed no correlation (p>0.05).

Among the 20 patients with EVA included in the study, there were 6 patients (30%) reported history of vestibular symptoms represented by frequent episodes of vertigo and nausea related mainly to physical hyperactivity in 4 patients (20%) and the other 2 patients (10%) suffered from imbalance.

This finding is in agreement with the study of Grimmer and Hedlund [23] who found that the overall incidence of vestibular symptoms in patients with EVA ranges from 12 to 71%. Emmet [34] reviewed 26 patients with EVA, one patient presented with episodic vertigo and two patients reported imbalance giving a 12% incidence of vestibular symptoms.

Jackler and De la Cruze [35] reported a 30% incidence of vestibular symptoms in a series of 17 patients with EVA. Three adult patients presented
with episodic vertigo and two children presented with imbalance and incoordination.

Zhou and Gopen [6] found that ears with EVA had higher VEMP amplitude with no difference in p13-n23 latencies between ears with EVA and ears without EVA.

Also Yetiser et al. [24] reviewed ten patients with EVA and reported that three patients (30%) had episodic vertigo. The highest incidence of vestibular symptoms was reported by Berrettini et al. [11] reviewed 15 patients, seven (47%) complained of vestibular disturbance. Also Grimmer and Hedlund, [23] reported 15 patients (47%) of 32 patients with EVA had vestibular symptoms.

Concerning the VEMP amplitude in the current study, 34 ears affected with EVA, 18 ears (53%) of them had VEMP amplitude records higher than those records for control group and 16 ears (47%) had within normal VEMP amplitude records. VEMP amplitude records were statistically significant higher in the affected ears with EVA in the case group than those records for the control group (p<0.05).

Furthermore, patients with unilateral EVA in the study group, 6 patients, (6 ears with EVA), showed VEMP amplitude records in the affected ears with EVA higher than those records for the non-affected ears, there was statistically significant difference between the affected ears with EVA and non-affected ears in the case group as regards the mean of VEMP amplitude (p<0.05).

As regards P 13 and N 23 latencies, VEMP records showed within normal latencies in the affected ears with EVA, there was no statistically significant difference between the affected ears with EVA and normal controls (p>0.05).

These findings are in accordance with the findings of Sheykholeslami et al. [8] who found that the VEMP amplitude records in patients with EVA were higher than those records both for normal subjects and for non-affected ears in patients with unilateral EVA.

Sheykholeslami et al. [8] and Merchant et al. [26] found that VEMP findings were indication of third window lesion that causes the air conducted sounds to be shunted away from cochlea to the vestibule causing larger deflection of the vestibular sensors within the membranous labyrinth, which make peripheral vestibular organs more sensitive to sound/pressure changes.

Among 15 patients with enlarged vestibular aqueduct, Berrettini et al. [11] reported 13 patients (86%) who had hypofunction as a response to caloric testing but there was no strict correspondence between the side of the vestibular deficit and the side of enlarged vestibular aqueduct in these patients and these results are not in accordance with this study results.

Yetiser et al. [24] postulated that vestibular dysfunction in patients with EVA may be due to degeneration of vestibular hair cells due to osmotic changes or injury due to chemical imbalance.

As regards rotary chair testing results of the current study, all patients had within normal results for phase and asymmetry at all frequencies, all patients had normal gain results at all frequencies except only one patient who had abnormal gain results at 0.16 Hz, 0.32.

References


