

# Vestibular assessment in children with sensorineural hearing loss using both electronystagmography and vestibular-evoked myogenic potential

Eman Abdel-Fattah Said

Audiology Unit, Department of Otolaryngology, Assiut College of Medicine, Assiut, Egypt

Correspondence to Eman Abdel-Fattah Said, Audiology Unit, Department of Otolaryngology, Assiut College of Medicine, Assiut, Egypt  
Tel: 01007238234; Fax: +0882333327  
e-mail: emanelgandy40@yahoo.com

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## Background

The clinical course and prognosis in sensorineural hearing loss (SNHL) may be even worse if the vestibular system is also involved, especially because of near location of the anatomic structures in the inner ear. However, vestibular function in children with SNHL appears to be under-reported. The aim of the study was to assess the vestibular function and to determine the prognostic value of some etiological, audiological, and demographic (age, sex) factors in predicting a possibility of vestibular impairment for very early identification of children with vestibular deficits.

## Materials and methods

The control group consisted of 30 children with normal hearing (17 girls and 13 boys) and the study group consisted of 50 children with varying degrees of bilateral SNHL, aged between 5 and 15 years. All of them were subjected to the following: basic audiological evaluation (pure-tone, speech audiometry), immittanceometry and auditory brainstem responses, electronystagmography (ENG), and vestibular-evoked myogenic potential (VEMP).

## Results

Hearing impaired (HI) children showed bilateral SNHL of various degrees ranging from moderate to profound hearing loss (HL) [moderate-severe (32%), severe (18%), and profound (50%)] and of different etiologies [heredofamilial (46%), acquired (38%), not known (16%)]. Abnormal ENG findings were recorded in 64% of HI children. Abnormal caloric test findings were found in 56% of the HI children with heredofamilial cause of HL, in 84.2% with acquired HL, and in 37.5% with unknown cause. These results according to the degree of HL were 37.5, 55.5, and 76% for moderate, severe, and profound HL, respectively. It was noticed that HI children with profound degree and acquired etiology of HL had the highest abnormal caloric findings. Abnormal VEMP was found in 72% of HI children, but this percentage varied according to the different etiologies, 56.5, 84, and 75% for heredofamilial, acquired, and unknown, respectively. Bilateral saccular affections were more common than unilateral. HI children with profound HL had the highest percentage of both bilateral and unilateral saccular affections in the absence of VEMP.

## Conclusion

Vestibular deficits occur in a significant percentage of HI children. Abnormal ENG and VEMP findings in HI children varied according to the etiology and or the degree of HL; those with acquired etiology and/or profound degree of HL had the highest abnormal scores.

## Keywords:

electronystagmography, prognostic factors, sensorineural hearing loss, vestibular-evoked myogenic potential

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## Introduction

From birth onwards, auditory stimulation directs and intensifies visual orientation behavior. The infant's earliest responses to auditory stimuli include the visual-motor behavior of moving the eyes or head to localize sound. Therefore, it has been suggested that the lack of early auditory input could contribute to motor delays in deaf and hard-of-hearing children [1].

Recent studies [2] reported that the importance of studying the relationship between peripheral vestibular function and the setting of deafness is underlined by the fact that the cochlea and vestibule are the peripheral sensory organs

of the auditory and vestibular system, respectively. They are anatomically (close proximity of the cochlea and the vestibular apparatus also in terms of innervation and vascular supply), phylogenetically, and functionally related. Disturbances in cochlear function, which can result in sensorineural hearing loss (SNHL), could accompany vestibular impairment because the cochlea and vestibule share continuous membranous labyrinth of the inner ear. Therefore, prenatal, perinatal, or postnatal injury or trauma may cause damage to one or both the systems [3-5].

In 2006, National Center for Hearing Assessment and Management [6] reported that SNHL is the

most common congenital sensory impairment, occurring in three of every 1000 live births. Although a small proportion of these individuals with SNHL exhibit concurrent vestibular involvement, this would still account for a large number of individuals with vestibular dysfunction, requiring identification, education, and therapy.

A previous author [7] reported that comprehensive testing of the vestibular function should include tests of canal and otolith function for diagnostic purposes and functional evaluation to identify impairments warranting therapeutic intervention. Horizontal semicircular canal function was abnormal in response to a caloric stimulus in 50% (16/32) children with severe to profound SNHL [8]. Other studies reported similar findings [3,9,10].

The amplitudes of vestibular-evoked myogenic potential (VEMP) were lower in children with SNHL compared with children with normal hearing [11]. Previous authors [8] studied 40 children with SNHL and reported that 40% demonstrated saccular dysfunction on the basis of the absence of a VEMP response, either bilaterally or unilaterally.

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### Aim of this study

The aim of the study was as follows:

- (1) To evaluate the vestibular function using both electronystagmography (ENG) and VEMP for examination of horizontal semicircular canal (SCC) and saccular dysfunction in a cohort of hearing impaired (HI) children.
- (2) To determine the prognostic value of some etiological and audiological factors associated with SNHL in predicting a possibility of vestibular dysfunction for very early identification of children with vestibular deficits. This allows proper counseling and recommendation to their parents.

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### Participants and methods

#### Participants

##### *Control group*

Thirty children with normal hearing and normal middle ear function were included, 17 of them were girls and 13 were boys.

##### *Study group*

Fifty-three children with bilateral hearing loss of more than 45 dB hearing level (HL) were included, with varying degrees and cause of HI (hereditary, acquired,

and unknown). Three children did not complete the test battery; hence, only 50 children – 27 girls and 23 boys – were enrolled. Forty-two children had been fitted with bilateral conventional hearing aids, five children had been fitted with monaural hearing aids, and three children had no hearing aid.

All children were between 5 and 15 years of age, with normal intelligence (a score of 80 or higher on a standard Stanford Binet test of intelligence). They were recruited from the Auditory Department, Assiut Medical University, with bilateral HI, during the period from November 2011 to January 2012.

#### *Exclusion criteria*

Children with neuromotor or orthopedic dysfunctions or on medication affecting the central nervous system were excluded.

Informed consent was obtained from the parents of all participants. The study was approved by the Ethics Committee of Assiut Medical University.

### Methods

*Each child was subjected to the following*

- (1) Careful systematic history-taking procedure with focus on the vestibular and hearing complaints and physical otoneurological development was carried out. Only 11 cases of HI children had delayed physical developmental milestones as their parents reported.
- (2) Basic audiological evaluation: A recent audiogram was available for all children with HI. Behavioral (pure tone) audiometry, which included air and bone conduction thresholds, speech audiometry, and tympanometric measurements, was performed to confirm normal middle ear pressure and mobility before VEMP testing. Auditory brainstem response was also used when deemed necessary to establish or confirm the hearing loss.

The examination was carried out in a standard sound-proof room. SNHL was classified according to the degree of hearing loss [12]. A previous study had proposed an etiologic classification that clarifies the interaction between time of insult, causation, and time of expression of hearing loss [13,14].

#### *Equipments*

- (1) Dual channel clinical audiometer (Madsen OB 922, GN Otometrics, Copenhagen, Denmark).
- (2) Immittancemeter (impedance audiometer AZ 26, Denmark).
- (3) Sound-treated booth (IAC model 1602-A-t; Industrial Acoustic Company, USA).

- (4) NicoLit Spirit equipment, USA, used to perform electrophysiological testing (auditory brainstem responses).
- (5) Four-channel ENG (ENG version Micromedical ENG device, USA, version 8.1 R).
- (6) Navigator Pro-evoked potential system made by Bio-logic (Mundelein, Illinois, USA) was used for VEMP.

#### Tests of vestibular end organ function

ENG and VEMP results were categorically defined as either normal or abnormal.

#### Electronystagmography test

Essentially, ENG consists of three parts [15]:

- (1) Oculomotor function: gaze, fixation, saccade (accuracy, latencies, and velocities), tracking (pursuit) (gain, phase, acceleration), and optokinetics (gain, phase) were evaluated.
- (2) Positioning/positional testing tests were recorded in five positions, and nystagmus, if present, was classified into direction-changing or direction-fixed.
- (3) Caloric Test: The abnormalities of low frequency horizontal canal function, asymmetry of reaction and unilateral weakness were calculated according to the formula of Jongkees and colleagues [16,17]. Fixation suppression during the caloric test was also observed.

After calibration, ENG can detect nystagmus (involuntary rapid eye movement) in response to various stimuli, which allowed to distinguish between central and peripheral vertigo.

Silver–silver chloride electrodes are affixed to the patient's skin with adhesive collars after brisk cleaning of the skin with an alcohol wipe to clear off oils and makeup and assure good electrical and physical contact. One ground electrode is placed on the forehead and one pair is placed above and below the right eye to detect blinks and vertical movements. A pair of electrodes is placed just lateral to each eye, horizontally aligned with the pupil, to maximally sense horizontal movement and reject vertical movement. The individual undergoes various forms of stimulation, and the other ocular response is recorded by means of corneoretinal potential [18].

All ears were inspected and debrided of wax before caloric testing. The ENG results were classified as normal, peripheral, and central vestibular lesions.

Testing instructions were explained using total communication to assure understanding of the activities required, with the help of the parents. Total

communication consisted of sign and oral language as well as demonstration. Instructions were repeated until the participant knew what was to be expected. Initially, the test was demonstrated directly by performing it on normal hearing participant and then on HI children.

#### Indicators of normal vestibular system

The vestibular system was considered normal when all subtests were normal.

Peripheral vestibular lesions were considered [15]:

- (1) If spontaneous nystagmus was horizontal or horizontal rotary, suppressed by visual fixation, nondirection changing, and exacerbated by gazing in the direction of the fast phase.
- (2) On the basis of the positioning Dix-Hallpike Maneuver (which should be completed before any other positional testing): delayed onset, observe patient for at least 20 s, transient burst of nystagmus, lasts about 10–15 s, subjective report of vertigo and fatigability.
- (3) On the basis of positional tests: direction-fixed, geotropic direction changing in different positions, latency of onset and fatigable.
- (4) If during the caloric test, unilateral or bilateral weakness almost always peripheral vestibular Disease (rule out vestibular suppressants in cases of bilateral vestibular weakness). The patient should be able to suppress caloric-induced nystagmus by opening.

Central vestibular lesions were considered [15]:

- (1) If spontaneous nystagmus was vertical, not suppressed by fixation and direction changing.
- (2) If abnormal findings were found in oculomotor tests (saccades test, smooth pursuit and optokinetic tests).
- (3) On the basis of positional tests: ageotropic direction changing in different positions, direction changing in a single position, immediate onset and not fatigable.
- (4) Failure of fixation suppression during the caloric test, in patients with central nervous system pathologic conditions who show little difference in the velocity of caloric-induced nystagmus whether their eyes are open or closed.

To facilitate adequate alertness throughout the test, one hearing aid was left on and functioning during caloric testing, whereas the other was held away from the pinna and protected against water.

The factors that predispose children to false-negative results in tests of vestibular function are malposition of irrigation tubing, presence of cerumen, crying, agitation, and inadequate performance in mental

tasks [19]. In addition to these issues, inattention and frequent random eye movement are more common in children and create difficulties in the analysis of the ENG traces obtained during a caloric stimulus, which often require a trace by trace analysis [20].

### VEMP test

Quick, easy, objective, and noninvasive nature of this procedure has led to some clinical study. However, a few studies with pediatric participants are available to reflect saccular function.

The sternocleidomastoid (SCM) muscle was chosen as a target to record the VEMPs. The VEMP test was performed in a sitting position. Older children could sit upright by themselves while being tested. They were instructed to turn their head toward the contralateral side of the ear being tested to activate the SCM. For younger children, an assistant was used to attract the patients' attention, allowing them to make a head turn focusing on a cartoon character to maintain head turn for muscle contraction [21]. If head turning did not yield a tracing, the child was positioned in a head lift position for bilateral/simultaneous activation of both SCMs to increase the yield of testing [22,23].

The skin over the SCM muscles and sternum was cleaned with an alcohol wipe. The active electrodes were attached on the midhalf of the SCM muscles on both sides; attention was given to place bilateral electrodes on the symmetrical site and a reference electrode on the suprasternal notch.

At stimuli level of 90 dB nHL, the stimulus rate was 4/s. Stimulus was presented monaurally through earphones (Telephonics TDH4, Denmark). Potentials were amplified and band pass was filtered (50–500 Hz). Analysis time was 80 ms. Recordings were obtained averaging 200 stimuli and two traces from each side were obtained to assess the reproducibility of peaks p13 and n23; thus, VEMP responses were termed 'present'. Conversely, VEMP responses were absent when reproducibility of the biphasic p13–n23 waveform was lacking. Thereby, the latencies of the peak p13, peak n23 (based on their respective latencies), and p13–n23 amplitude were measured. Patients were given 30–60 s to relax between each recording [24].

### Statistical analysis

Data were collected and analysis was performed using computer program SPSS, version 17 (SPSS Inc., Chicago, Illinois, USA). Data were expressed as mean and SD using the *t*-test to determine the significance for quantitative variable. The  $\chi^2$ -test was performed to determine the significance for qualitative variable (number, percentage).

*P*-value greater than 0.05 was considered nonsignificant, *P*-value less than 0.05 was considered significant, and *P*-value less than 0.001 was considered highly significant.

## Results

### Basic audiological evaluation

All children in the control group showed bilateral normal hearing. Meanwhile, HI children showed bilateral SNHL of various degrees, ranging from moderate to total hearing on audiometry, and had various etiologies (Table 1).

### ENG

Vestibular impairment of peripheral type was noted in 64% of HI children, whereas 10% (5/50) of HI children had central type of impairment (latency in saccade) and gain in optokinetic; only one of these five children had abnormal gain in the tracking (pursuit) test. Only 10% of normal hearing children had abnormal ENG response. There was statistically significant difference in the percentage of abnormal ENG response between normal hearing children (NH) children and HI children ( $P = 0.001$ ).

**Table 1 Demographic characteristics of HI children**

Features	n (%)
Age (years)	
Group 1: 5–7	12 (24)
Group 2: 8–10	15 (30)
Group 3: 11–15	23 (46)
Sex	
Male	22 (44)
Female	28 (56)
Degree of SNHL	
Moderate-severe	16 (32)
Severe	9 (18)
Profound	25 (50)
Etiology of sensorineural hearing loss	
Heredofamilial	23 (46)
Nonsyndromic	21 (42)
Syndromic	2 (4)
Acquired	19 (38)
Prenatal	2 (4)
Perinatal	6 (12)
Postnatal	12 (24)
Not known	8 (16)
Hearing aid fitting	
Binaural	42 (84)
Monaural	5 (10)
Not fitted	3 (6)
History of delayed motor development, clumsiness, or unsteadiness	11 (22)

HI, hearing impaired; SNHL, sensorineural hearing loss.

Abnormalities of low-frequency horizontal canal function were observed in 32 children (64%) as measured by caloric testing. The majority of HI children, 72% (23/32), had bilateral horizontal canals dysfunction. However, some asymmetries did exist in 28% (9/32) HI children (Table 2).

**The impact of SNHL etiology on the results of caloric test (peripheral vestibular test)**

Abnormal horizontal canal function was found in 56, 84.2, and 37.5% of patients with hereditary, acquired, and unknown etiologies, respectively (Table 3).

As noticed, although children in the acquired hearing loss group had the highest score of abnormalities when compared with the other etiologies, this difference did not reach the level of significance ( $P = 0.292$ ).

**The impact of the degree of SNHL on the results of caloric test**

To avoid overestimation of the results, we analyzed the results of the caloric test according to the degree of hearing loss. Abnormal caloric test findings were noted in 88% (22/25) of HI children with profound SNHL, whereas the percentage in the remaining groups was 25% (4/16) and 66.6% (6/9) in moderate-severe and severe SNHL, respectively.

As noticed from Table 4, there was no statistically significant difference in the percentage of abnormal caloric test findings between the groups with different degrees of SNHL ( $P = 0.128$ ).

**VEMP**

Disruption of saccular function, as measured by abnormalities of VEMP response, was found in 72% (36/50) of HI children. There was statistically significant difference in the percentage of abnormal VEMP response between NH children (10%) and HI children ( $P = 0.001$ ) (Table 2).

VEMP response was bilaterally absent in 21/50 (42%) and unilaterally in 12% (6/50) and was delayed in 9/50 (18%) patients.

**The impact of etiology of SNHL on VEMP response**

The highest abnormal VEMP response score was recorded in 16/19 (84.2%) of children with SNHL because of acquired cause when compared with children with other etiologies: 56.5% (14/23) of children with hereditary SNHL and 75% (6/8) of children with unknown etiology. There was no statistically significant difference in the results of VEMP response between HI children with different etiologies of SNHL (Table 5).

**Table 2 Comparison between NH and HI children on the basis of their results in ENG and VEMP tests**

	Control (n = 30) [n (%)]	HI (n = 50) [n (%)]	P-value
<b>ENG results</b>			
Normal findings	27 (90)	18 (36)	0.001**
Spontaneous nystagmus	0 (0)	1 (2)	0.345
Positional and/or positioning nystagmus	3 (10)	16 (32)	0.02*
Gaze nystagmus	0 (0)	0 (0)	–
<b>Oculomotor test</b>			
Smooth pursuit eye movement	1 (3.3)	1 (2)	0.495
Saccade	0 (0)	5 (10)	0.471
Optokinetic	1 (3.3)	5 (10)	0.593
<b>Caloric test (canal paresis)</b>			
Unilateral	0 (0)	9 (18)	0.04*
Bilateral	0 (0)	23 (46)	0.001**
Total abnormalities	0 (0)	32 (64)	0.01*
<b>VEMP results</b>			
Normal findings	27 (90)	14 (28)	0.01*
Unilateral	1 (3.3)	6 (17)	0.351
Bilateral	2 (6.6)	21 (58)	0.000***
Latency±amplitude abnormalities	0 (0)	9 (18)	0.04*
Total abnormalities	3 (10)	36 (72)	0.001**

ENG, electronystagmography; HI, hearing impaired; VEMP, vestibular-evoked myogenic potential; \*Significant; \*\*moderately significant; \*\*\*Highly significant.

**Table 3 Caloric test (peripheral vestibular test) results in hearing impaired children according to their etiologies of SNHL**

	Caloric test results in HI children [n (%)]		
	Normal	Unilateral canal paresis	Bilateral canal paresis
<b>Etiology of SNHL</b>			
Heredofamilial (n=23)	10 (43.4)	4 (17)	9 (39)
Total		13 (56)	
Acquired (n=19)	3 (16%)	5 (26)	11 (58)
		16 (84.2)	
Not known (n=8)	5 (62.5)	0 (0)	3 (37.5)
		3 (37.5)	
Total abnormalities		32 (64)	
P-value		0.292	

$\chi^2$ -test was used. HI, hearing impaired; SNHL, sensorineural hearing loss.

**Table 4 Caloric test (peripheral vestibular test) results in hearing impaired children according to their degrees of SNHL**

	Caloric test results in HI children [n (%)]		
	Normal	Unilateral canal paresis	Bilateral canal paresis
<b>Degree of SNHL</b>			
Moderate-severe (n=16)	12 (75)	1 (6)	3 (19)
		4 (25)	
Severe (n=9)	3 (33)	1 (11)	5 (55.5)
		6 (66.6)	
Profound (n=25)	3 (12)	7 (8)	15 (76)
		22 (88)	
P-value		0.128	

$\chi^2$ -test was used. HI, hearing impaired; SNHL, sensorineural hearing loss.

**The impact of the degree of SNHL on VEMP response**

Fourteen of 25 (56%) HI children with profound SNHL had bilaterally absent VEMP response versus 4/9 (44%) and 3/16 (19%) of HI children with severe and moderate SNHL, respectively. Meanwhile, unilateral absence of saccular function was demonstrated in 6% of HI children with moderate SNHL, 11% of HI children with severe SNHL, and 16% of HI children with profound SNHL (Table 6); this indicate that bilaterally absent response was more common than unilateral, and the differences in the degree of SNHL may be a predictor of VEMP response. However, there was no statistically significant difference in the percentage of abnormal VEMP findings between groups with different degrees of SNHL ( $P = 0.279$ ).

In this study, there was no significant relationship between abnormalities of caloric testing and VEMP testing.

The latencies of P13 and n23 in normal children showed earlier than those in normal adults, indicating that the structural factors such as neck length, conduction velocity, or head size may affect the p13 latency [25]. Mean latencies of peaks p13 and n23 in 30 healthy children were  $11.7 \pm 0.99$  and  $18.12 \pm 1.53$ , respectively, whereas P1 and N1 latencies in HI children were delayed ( $12.28 \pm 1.9$  and  $20.08 \pm 1.28$  ms). However, this difference did not reach statistical significance (Table 7). Considerable statistically significant difference ( $P < 0.001$ ) was noted in VEMP p13–n23 amplitude between NH ( $5.12 \pm 0.74$ ) and HI children ( $3.76 \pm 0.45$ ) (Table 7).

Lowenstein [25] had given the anatomic compartmentalization of the saccule and cochlea. One might predict that saccular function may be more likely affected than utricular or semicircular canal function in the presence of an inner ear injury, leading to SNHL.

**Discussion**

SNHL still remains a problem with respect to the factors that might predict the extent of handicap and prognosis. Previous study suggested plausibly that lesions leading to SNHL could also contribute to dysfunction of the vestibular end organs [26]. In addition, other authors suggested that the clinical course of SNHL may be aggravated if the vestibular system is also involved [27,28]. It may even result in delayed motor development in children [29].

**ENG test**

This study demonstrated vestibular deficits in HI children using ENG test. Abnormal ENG was noted in (32/50) 64% of peripheral type patients with

**Table 5 Comparison between HI children with different etiologies of SNHL on the basis of their results in VEMP**

Etiology of SNHL	Normal [n (%)]	Latency $\pm$ amplitude abnormalities [n (%)]	Absence [n (%)]	
			Unilateral	Bilateral
Hereditary (n=23)	9 (39)	2 (9)	3 (13)	9 (39)
Total		14 (56.5)	12 (52)	
Acquired (n=19)	3 (16)	5 (26)	3 (16)	8 (42)
Total		16 (84.2)	11 (58)	
Not known (n=8)	2 (25)	2 (25)	0 (0)	4 (50)
Total abnormalities		6 (75)	4 (50)	
P-value		0.429		

HI, hearing impaired; SNHL, sensorineural hearing loss; VEMP, vestibular-evoked myogenic potential.

**Table 6 Comparison between HI children with different degrees of SNHL on the basis of their results in VEMP**

Degree of SNHL	Normal [n (%)]	Latency $\pm$ amplitude abnormalities [n (%)]	Absence [n (%)]	
			Unilateral	Bilateral
Moderate-severe (n=16)	9 (56)	3 (19)	1 (6)	3 (19)
Total		7 (43.7)	4 (25)	
Severe (n=9)	3 (33)	1 (11)	1 (11)	4 (44)
Total		6 (66.6)	5 (55.5)	
Profound (n=25)	2 (8)	5 (20)	4 (16)	14 (56)
Total		23 (92)	18 (72)	
Total abnormalities		36 (72)		
P-value		0.279		

HI, hearing impaired; SNHL, sensorineural hearing loss; VEMP, vestibular-evoked myogenic potential.

**Table 7 Comparison between the means ( $\pm$ SDs) of VEMP measurements in NH and HI children**

VEMP outcomes	NH children (n = 27)	HI children (n = 9)	P-value
P1 latency (ms)	$11.7 \pm 0.99$	$12.28 \pm 1.9$	0.421
N1 latency (ms)	$18.12 \pm 1.53$	$20.08 \pm 1.28$	0.147
Amplitude, ( $\mu$ V)	$5.12 \pm 0.74$	$3.76 \pm 0.45$	0.000

HI, hearing impaired; VEMP, vestibular-evoked myogenic potential.

horizontal canal dysfunction on caloric testing, either absent or abnormal caloric responses, and in 10% (5/50) of central type impairment patients (Table 2). This was in agreement with an other author who noted abnormal ENG in 88% of patients (22 ears); in nearly all of them (20 ears), vestibular impairment was of peripheral type [3].

Some investigators have reported a high incidence of vestibular hypofunction in children with SNHL [10,30]. In children with SNHL, concomitant damage to vestibular structures is commonly reported [10,31,32].

This is also consistent with another research in which 22 children were tested. Their age was 2–16 years, and bithermal caloric irrigation, rotational chair testing, and computerized dynamic posturography were performed. They found that 68% of the children had vestibular hypofunction or areflexia [33].

An overall incidence of horizontal canal dysfunction on caloric testing was found in 50% children with SNHL [8]. Published reports showed that vestibular dysfunctions were found in ~30–70% of children with hearing loss [11,34,35]. Other author reported that two-third of HI patients had significant vestibular deficits [33].

Vestibular dysfunction in children with SNHL had been widely prevalent [36]; 20–70% of children with SNHL demonstrate an element of vestibular end organ dysfunction [37–39]. In addition, Rosenblüt *et al.* [40] found either absent or abnormal caloric responses in 49% of 107 deaf children. This wide range is likely a reflection of the variety of methods used to quantify vestibular dysfunction and the characteristics of the study populations, particularly with respect to the degree and etiology of the HI [41].

However, other investigators reported that a vestibular response was present in 90% of patients. Nevertheless, this may be because of the small number of HI children (only 25) [42].

#### *Impact of etiology of SNHL on the results of ENG*

In this study, it was noticeable that the HI children with acquired etiology had the highest abnormal ENG scores [16 (84.2%)] compared with the other etiologies, hereditary [13 (56%)] and unknown [3 (37.5%)] (Table 3). A tendency toward parallelism between the etiology and the vestibular function reflected that the etiology of SNHL may be a useful predictor of caloric hypoactivity.

This was consistent with the results in a recent study [26] in which some parallels were discovered between the etiology of hearing loss and the vestibular function. In addition, this was in agreement with the study by other authors who suggested that SNHL following bacterial meningitis is often accompanied by profound vestibular dysfunction and associated with bilateral vestibular loss [43–47].

A previous study [37] also found that loss of vestibular function in children is associated less often with a genetic cause than with acquired deafness. In addition, other authors reported that the prevalence of vestibular test abnormalities is higher in acquired deafness (meningitis, labyrinthitis) [32,38,48,49].

The variability in vestibular responsiveness among different etiologies of SNHL was readily apparent in the available literature, and this incredible variability is present even within a single etiologic category such as deafness secondary to meningitis even though the relationship between auditory and vestibular function in this setting is thought to be better understood. Children with SNHL due to meningitis demonstrate a disproportionately large degree of vestibular nonresponsiveness; however, the degree of nonresponsiveness does range from unilateral weakness to bilateral areflexia of the horizontal canals [50].

The prevalence of vestibular disorders among these other etiologies is highlighted in studies that excluded children whose deafness was secondary to meningitis or other severe infantile infection. Despite this exclusion, Rosenblüt *et al.* [40] reported that a large proportion (43%) of the remaining cohort demonstrated vestibular dysfunction [40].

This was in disagreement with the study by Rapin [19] who documented that nearly half of the children with profound SNHL due to meningitis demonstrated normal horizontal canal function as measured by a caloric stimulation. Another author found a range of vestibular responsiveness that spanned from absent to normal across their designated etiologic subgroups as well as across different categories of residual hearing [51].

#### *Impact of the degree of SNHL on the results of ENG*

In this study, it was noticed that the percentage of abnormal ENG results varied according to the degree of hearing loss (37.5, 55.5, and 76% for moderate to moderately severe, severe, and profound hearing loss, respectively). It was noted that HI children with profound hearing loss had the highest percentage of abnormal ENG (88%), and a tendency toward parallelism between the extent of hearing loss and the vestibular function was found in this study. However, there was no statistically significant difference between the results of abnormal ENG among different degree of SNHL (Table 4).

This was in agreement with the study by Cushing *et al.* [8] who reported that horizontal semicircular canal function was abnormal in response to a caloric stimulus in 50% (16/32) children with severe to profound SNHL. Rosenblüt *et al.* [40] concluded that

vestibular function was proved to be normal in 80% of individuals with hearing loss of less than 90 dB as opposed to only 20% of those whose hearing loss exceeded 98 dB. Vestibular function appears, therefore, to be normal up to a point at which acoustic function has been almost entirely lost.

This was also supported by other studies, which showed that vestibular dysfunction has a high frequency (up to 80%) in children with severe HI [10,11,30,34,40–42]. Another author [3] noted that the percentage of abnormal ENG was significantly greater in the residual hearing/deafness group compared with the remaining group of 158 ears (88 vs. 60%, respectively). Previous studies [9,10] had the same note.

The prevalence of vestibular test abnormalities is higher in profound SNHL [34,38]. A number of studies have shown that, at least on a group level, the likelihood of a vestibular impairment relates to the degree of the hearing loss [9,36,50].

In a previous study, the authors [10] divided 57 patients into three groups on the basis of average auditory threshold (<90, 91–97, >98 dB), with vestibular deficits (caloric) detectable in 20, 50, and 80% of patients, respectively [42]. It was noted that the prevalence of vestibular test abnormalities is higher in profound SNHL.

Other investigators [40] concluded that the relationship between auditory and vestibular function is certainly complex. The intricacy of this interaction is particularly evident in patients with well-preserved vestibular function in the presence of even the most severe auditory dysfunction and in instances where apparently minor losses of auditory function are accompanied by complete vestibular dysfunction. Although the relationship between vestibular and auditory function is not simple, they do appear to be associated. Rosenblüt *et al.* [40] investigated vestibular function in 107 deaf children. It emerged that the vestibular and auditory functions were not mutually independent, that is the greater the hearing loss, the greater the vestibular impairment. Hearing function was, however, classified on the basis of the type of audiometry curve and not assigned a definitive, quantified degree of hearing loss. It is striking that, in 16.1% of the children with relatively good auditory sensitivity, no vestibular responses were demonstrable, whereas normal responses occurred in 43.3% of the children with the poorest sensitivity. The authors conclude that, although there is a correlation between auditory function and vestibular response, this link is not sufficiently clear to allow predictions to be made on a case-by-case basis [28,40].

This was in agreement with the study by Christensson and Garwicz [52] and Grillner *et al.* [53] who found that the prevalence of vestibular dysfunction seemed to correlate also with the severity of the cochlear loss. In addition, Schwab and Kontorinis [26] concluded that the extent of hearing loss alone does not enable conclusions be drawn about either vestibular function or dynamic balance performance.

#### *Assessment of saccular function using VEMP*

In the present study, it was found that most children with SNHL had reduced saccular function, which was observed in the form of abnormal VEMP in 72% of HI children (Table 2). This was in agreement with the study by Zhou *et al.* [11] who found that abnormal VEMP was found in 21 of 23 children (91%) with SNHL.

Previous authors [8] also studied 40 children with SNHL and found that 40% demonstrated saccular dysfunction on the basis of the absence of VEMP response, either bilaterally or unilaterally. No VEMP was evoked bilaterally in 42% of the HI children; this was consistent with other studies published previously [38,54,55].

In this study, the normative mean data for p13 and n23 latencies of VEMP obtained with a click are 11.7 ( $\pm 0.99$ ) and 18.12 ( $\pm 1.53$ ) ms, respectively (Table 7). This was in agreement with the results of other authors [22] who found that normative data for p13 and n23 latencies of VEMP obtained with a click in young children were 11.3 ( $\pm 1.3$ ) and 17.6 ( $\pm 1.4$ ) ms, respectively.

In contrast to the results in this study, other investigators [11] reported that normative mean data for p13 and n23 latencies of VEMP obtained with a click in young children are 16.14 ( $\pm 2.81$ ) and 21.38 ( $\pm 3.04$ ) ms, respectively. The differences between these studies and our results could be justified by the different stimulus used.

In this study, it was found that most children with SNHL had delayed P1 and N1 latencies compared with children with normal hearing. However, this difference did not reach statistical significance. This was in agreement with a previous research [11] in which there were no differences in the P1 and N1 latencies between the two groups. They reported that most children with SNHL had reduced amplitude in N1–P1.

In this study, the prevalence of abnormal VEMP responses varied according to the etiology of SNHL, 56.5, 84, and 75% for hereditary, acquired, and



unknown, respectively (Table 5). Most HI children with acquired cause of hearing loss had abnormal VEMP response.

The degree of SNHL in this study also affected the absence VEMP responses (both unilateral and bilateral) in various percentages: 25% for moderate, 56% for severe, and 72% for profound hearing loss. In this study, most HI children with profound hearing loss had abnormal VEMP responses (Table 6). These findings are consistent with a study by other authors who reported that 50% of children with profound SNHL had abnormalities of saccular function and VEMP responses [56].

In this study, there was no significant relationship between abnormalities in saccular function (measured by VEMP test) and abnormality in horizontal canal function (measured by caloric test). This was in agreement with the study by Cushing *et al.* [8].

It remains unclear why many HI children with abnormal VEMP outcomes do not have complaints of vestibular symptoms. Possible explanations include the following: (a) young children are not able to describe dizziness or vertigo to their parents and physicians, (b) saccular impairment alone is not enough to cause clinically significant vestibular disturbance, (c) chronic peripheral vestibular deficit may generate central compensation, and (d) less attention is paid to subtle manifestations of vestibular dysfunction by caregivers.

## Conclusion

Vestibular deficits occur in a significant percentage of HI children as reflected by a high score of abnormal findings in both ENG and VEMP test results (abnormal ENG findings were recorded in 64% of HI children and abnormal VEMP findings were recorded in 72% of them). HI children with acquired cause and/or profound degree of hearing loss had highest score of abnormal findings. Thus, a combination of caloric and VEMP tests may serve as supplementary diagnostic tools in evaluating HI children.

Vestibular dysfunction in very young children may have significant detrimental effects on motor development. Therefore, information regarding identification and treatment of these balance issues is very important, particularly in young children.

## Recommendations

The high incidence of vestibular dysfunction in HI children without other handicaps is important information for therapists who are concerned in

evaluating and treating HI children. Therefore, to minimize the adverse effects of them on normal development, it is crucial to carry out vestibular screening examinations and vestibular testing in all children with SNHL. Furthermore, once vestibular abnormalities were identified, appropriate interventions of balance and motor deficits warranted that functional improvement can be achieved through participation in vestibular rehabilitation focused on substitution and adaptation exercises. However, additional study is needed to examine the long-term effects of intervention.

## Acknowledgements

### Conflicts of interest

None declared.

## References

- 1 Savelsbergh GJP, Netelenbos JB, Whiting HTA. Auditory perception and the control of spatially coordinated action of deaf and hearing children. *J Child Psychol Psychiatry* 1991; 32:489–500.
- 2 Rajendran V, Roy FG. An overview of motor skill performance and balance in hearing impaired children. *Ital J Pediatr* 2011; 37:33.
- 3 Pajor A, Jozefowicz-Korczyńska M. Prognostic factors for vestibular impairment in sensorineural hearing loss. *Eur Arch Otorhinolaryngol* 2008; 265:403–407.
- 4 Wilson VJ, Peterson BW. In: Mountcastle VBed. The role of the vestibular system in posture and movement. *Medical physiology* 14 ed. 1980; St. Louis, MO: CV Mosby Co; 1:813–836.
- 5 Kaga K. Vestibular compensation in infants and children with congenital and acquired vestibular loss in both ears. *Int J Pediatr Otorhinolaryngol* 1999; 49:215–224.
- 6 Centers for Disease Control and Prevention. (2008). *Summary of 2006 national EHDI data*. Retrieved July 21, 2008, from HYPERLINK "http://www.cdc.gov/ncbddd/ehdi/data.htm" www.cdc.gov/ncbddd/ehdi/data.htm.
- 7 Rine RM. Growing evidence for balance and vestibular problems in children. *Aud Med* 2009; 7:138–142.
- 8 Cushing SL, Papsin BC, Rutka JA, James AL, Gordon KA. Evidence of vestibular and balance dysfunction in children with profound sensorineural hearing loss using cochlear implants. *Laryngoscope* 2008; 118:1814–1823.
- 9 Goldstein R, Landau WM, Kleffner FR. Neurologic assessment of some deaf and aphasic children. *Ann Otol Rhinol Laryngol* 1958; 67:468–479.
- 10 Sandberg LE, Terkildsen K. Caloric tests in deaf children. *Arch Otolaryngol* 1965; 81:350–354.
- 11 Zhou G, Kenna MA, Stevens K, Licameli G. Assessment of saccular function in children with sensorineural hearing loss. *Arch Otolaryngol Head Neck Surg* 2009; 135:40–44.
- 12 Clark JG. Uses and abuses of hearing loss classification. *Asha* 1981; 23:493–500.
- 13 Espeso A, Owens D, Williams G. The diagnosis of hearing loss in children: common presentations and investigations. *Curr Paediatr* 2006; 16:484–488.
- 14 Davidson J, Hyde JM, Alberti PW. Epidemiology of hearing impairment in childhood. *Scand Audiol Suppl* 1988; 30:13–20.
- 15 Jacopson GP, Newman CW, Kartush J. *Handbook of balance function test*. London: Singular Publishing Group Inc.
- 16 Jongkees LB, Maas JP, Philipszoon AJ. Clinical nystagmography. A detailed study of electro-nystagmography in 341 patients with vertigo. *Pract Otorhinolaryngol (Basel)* 1962; 24:65–93.
- 17 Jongkees LB. Vestibular tests for the clinician. *Arch Otolaryngol* 1973; 97:77–80.
- 18 Carl JR. In: Jacopson GP, Newman CW, Kartush Jeds. *Principles and techniques of electro-oculography*. Chapter 4. *Handbook of balance function test* London: Singular Publishing Group Inc., 69–82.

- 19 Rapin I. Hypoactive labyrinths and motor development. *Clin Pediatr (Phila)* 1974; 13:922–937.
- 20 Snashall SE. Vestibular function tests in children. *J R Soc Med* 1983; 76:555–559.
- 21 Valente LM. Adaptation of adult techniques for evaluating vestibular function in children. *Hear J* 2007; 60:34–44.
- 22 Kelsch TA, Schaefer LA, Esquivel CR. Vestibular evoked myogenic potentials in young children: test parameters and normative data. *Laryngoscope* 2006; 116:895–900.
- 23 Wang C-T, Young Y-H. Comparison of the head elevation versus rotation methods in eliciting vestibular evoked myogenic potentials. *Ear Hear* 2006; 27:376–381.
- 24 Henry K. The dizziness and balance center. Incorporating VEMP testing into your vestibular assessment protocol. Chicago: American Academy of Audiology April 6–9, 2011.
- 25 Lowenstein O. The equilibrium function of the vertebrate labyrinth. *Biol Rev Camb Philos Soc* 1936; 11:113–145.
- 26 Schwab B, Kontorinis G. Influencing factors on the vestibular function of deaf children and adolescents – evaluation by means of dynamic posturography. *Open Otorhinolaryngol J* 2011; 5:1–9.
- 27 Narozny W, Kuczkowski J, Kot J, Stankiewicz C, Sicko Z, Mikaszewski B. Prognostic factors in sudden sensorineural hearing loss: our experience and a review of the literature. *Ann Otol Rhinol Laryngol* 2006; 115:553–558.
- 28 Nakashima T, Yanagita N. Outcome of sudden deafness with and without vertigo. *Laryngoscope* 1993; 103:1145–1149.
- 29 Angeli S. Value of vestibular testing in young children with sensorineural hearing loss. *Arch Otolaryngol Head Neck Surg* 2003; 129:478–482.
- 30 Brookhouser PE, Cyr DG, Beauchaine KA. Vestibular findings in the deaf and hard of hearing. *Otolaryngol Head Neck Surg* 1982; 90:773–777.
- 31 Goodman J, Hopper C. Hearing impaired children and youth: A review of psychomotor behavior. *Adapt Phys Act Q* 1992; 9:214–236.
- 32 Sule B, Guvin N, Bal S, Metin N, Artan I. A comparison study of grossmotor developmental skill normal, hearing-impaired and Down syndrome children. Paper presented at 18<sup>th</sup> International Congress on Education of the Deaf, Tel Aviv, Israel 1995; pp. 16–20.
- 33 Buchman CA, Joy J, Hodges A, Telischi FF, Balkany TJ. Vestibular effects of cochlear implantation. *Laryngoscope* 2004; 114:1–22.
- 34 Selz PA, Girardi M, Konrad HR, Hughes LF. Vestibular deficits in deaf children. *Otolaryngol Head Neck Surg* 1996; 115:70–77.
- 35 Anthrop J, Allison MT. Roll conflict and the high school female athlete. *Res Q Exerc Sport* 1983; 24:104–111.
- 36 Solan HA, Shelley-Tremblay J, Larson S. Vestibular function, sensory integration, and balance anomalies: A brief literature review. *Optom Vis Dev* 2007; 38:13–17.
- 37 Arvig, J. Vestibular function in deafness and severe hardness of hearing. *Acta Otolaryngol* 1955; 45:283–288.
- 38 Tribukait A, Brantberg K, Bergenius J. Function of semicircular canals, utricles and saccules in deaf children. *Acta Otolaryngol* 2004; 124:41–48.
- 39 Telian SA. Comments about the value of vestibular testing in young children with sensorineural hearing loss. *Arch Otolaryngol Head Neck Surg* 2003; 129:483–484.
- 40 Rosenblut B, Goldstein R, Landau WM. Vestibular responses of some deaf and aphasic children. *Ann Otol Rhinol Laryngol* 1960; 69:747–755.
- 41 Potter CN, Silverman LN. Characteristics of vestibular function and static balance skills in deaf children. *Phys Ther* 1984; 64:1071–1075.
- 42 Swisher LP, Gannon RP. A comparison of auditory and vestibular responses in hearing-impaired children. *Acta Otolaryngol* 1968; 66:89–96.
- 43 Kaplan SL, Goddard J, van Kleecck M, Catlin FI, Feigin RD. Ataxia and deafness in children due to bacterial meningitis. *Pediatrics* 1981; 68:8–13.
- 44 Kumar A, Fishman G, Torok N. Vestibular and auditory function in Usher's syndrome. *Ann Otol Rhinol Laryngol* 1984; 93:600–608.
- 45 Karjalainen S, Terasvirta M, Karja J, Kaariainen H. Usher's syndrome type III: ENG findings in four affected and six unaffected siblings. *J Laryngol Otol* 1985; 99:43–48.
- 46 Samuelson S, Zahn J. Usher's syndrome. *Ophthalmic Paediatr Genet* 1990; 11:71–76.
- 47 Otterstedde CR, Spandau U, Blankenagel A, Kimberling WJ, Reisser C. A new clinical classification for Usher's syndrome based on a new subtype of Usher's syndrome type I. *Laryngoscope* 2001; 111:84–86.
- 48 Wiegersma PH, Van der Velde A. Motor development of deaf children. *J Child Psychol Psychiatry* 1983; 24:103–111.
- 49 Horak FB, Shumway-Cook A, Crowe TK, Black FO. Vestibular function and motor proficiency of children with impaired hearing, or with learning disability and motor impairments. *Dev Med Child Neurol* 1988; 30:64–79.
- 50 Huygen PLM, Van Rijn PM, Cremers CWRJ, Theunissen EJJM. The vestibulo-ocular reflex in pupils at a Dutch school for the hearing impaired; findings relating to acquired causes. *Int J Pediatr Otorhinolaryngol* 1993; 25:39–47.
- 51 Guilder R, Hopkins L. Auditory function studies in an unselected group of pupils at the Clarke School for the Deaf. *Laryngoscope* 1936; 46:190–197.
- 52 Christensson M, Garwicz M. Time course of postnatal motor development in ferrets: Ontogenetic and comparative perspectives. *Behav Brain Res* 2005; 158:231–242.
- 53 Grillner S, Hongo T, Lund S. The vestibulospinal tract. Effects on alpha-motoneurons in the lumbosacral spinal cord in the cat. *Exp Brain Res* 1970; 10:94–120.
- 54 Shinjo Y, Jin Y, Kaga K. Assessment of vestibular function of infants and children with congenital and acquired deafness using the ice-water caloric test, rotational chair test and vestibular-evoked myogenic potential recording. *Acta Otolaryngol* 2007; 127:736–747.
- 55 Zagólski O. Vestibular system in infants with hereditary nonsyndromic deafness. *Otol Neurotol* 2007; 28:1053–1055.
- 56 Jin Y, Nakamura M, Shinjo Y, Kaga K. Vestibular-evoked myogenic potentials in cochlear implant children. *Acta Otolaryngol* 2006; 126:164–169.