Assessment of saccular function using cervical vestibular-evoked myogenic potentials in children with sensorineural hearing loss

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Objectives
The aim of this work was to examine saccular function using cervical vestibular-evoked myogenic potentials (cVEMPs) in children with congenital or acquired sensorineural hearing loss (SNHL).

Study design
Descriptive cross-sectional study.

Participants and methods
This study was carried out on 30 children with different degrees of SNHL with normal middle ear function and 25 age-matched and sex-matched typically developing children. All the participants' age ranged from 5 to 18 years. For each child, the following were administered: history taking, an otologic examination, a basic audiological evaluation in the form of pure tone audiometry and immittancemetry, air-conducted cVEMPs, and computed tomography scan of the temporal bone. The results obtained from the two groups were then compared. In addition, correlation studies between all the results obtained were carried out.

Results
There was a statistically nonsignificant difference between the patients and the controls in cVEMP P13 and N23 latencies, whereas the cVEMP P13–N23 amplitude and threshold results showed a highly significant difference. cVEMP measurements in relation to sex within the patient group showed that the cVEMP threshold was the only positive outcome that showed a significant difference, and was higher in females. There was a highly significant positive correlation between hearing loss and the cVEMP threshold among the patient groups with different degrees of hearing losses. In addition, there was a significant positive correlation between age and cVEMP P13 and N23 latencies among the patient group.

Conclusion
SNHL is associated with saccular dysfunction in the pediatric population in the form of decreased cVEMP P13–N23 amplitudes and elevated cVEMP thresholds. Females presented with higher cVEMP threshold values than men, and cVEMP latencies appear to increase with age. An increase in hearing loss is associated with an increase in the cVEMP threshold and cVEMP amplitude appears to decrease with hearing loss irrespective of its degree. We recommend the inclusion of cVEMPs in the battery of evaluation of children with SNHL to detect early subtle changes in saccular function. Future research focusing on the genes causing SNHL and its relation to VEMP findings should be carried out.

Keywords:
cochlea, pure tone audiometry, saccule, sensorineural hearing loss, sternocleidomastoid muscle, vestibular-evoked myogenic potentials

Introduction
Epidemiological studies have shown that one in 1000 children are born with or present in early childhood with severe or profound hearing impairment [1]. More lose their hearing later during childhood. The lack of auditory input from environmental sounds and speech during early childhood interferes with the normal development of the auditory system and impedes the development of speech and language abilities [2].

Children with deafness are at risk of vestibular dysfunction, because in some forms of inner ear deafness, the damage extends to the vestibular receptors as well. There are reports of peripheral vestibular dysfunction and delayed postural control in some types of congenital and acquired deafness such as meningitis, viral labyrinthitis, and some forms of hereditary deafness [3].

The cochlea and the vestibule are the peripheral sensory organs of the auditory and vestibular systems, respectively.

They are anatomically and functionally connected [4]. The disturbance of cochlear function, which can result in sensorineural hearing loss (SNHL), could accompany vestibular impairment, because the cochlea and the vestibule share the continuous membranous labyrinth of the inner ear [5].

The vestibular-evoked myogenic potential (VEMP) is a short latency-negative response that is induced by brief pulses of air-conducted sound, bone-conducted vibration, or electrical stimulation [6]. Cervical VEMP (cVEMP) records the poststimulatory tonical contraction of the sternocleidomastoid (SCM) muscle [7]. VEMPs occurring in the cervical muscles (cVEMP) after an intense acoustic stimulation of the ear are polysynaptic responses of otolith–vestibular nerve origin and have become reliable tests to assess the function of the saccule [8].

cVEMP originates in the saccular macula and then moves to the neurons of Scarpa's ganglion, through the inferior vestibular nerve, the lateral vestibular nucleus, the medial vestibulospinal tract, and onto the motor neurons of the SCM muscle. cVEMPs are independent of the presence of SNHL, whereas they are absent in patients in whom vestibular deafferentation has been performed [9].

At present, the general consensus is that short latency potentials with an initial positivity (P13 and N23) recorded from ipsilateral SCM muscle in response to air-conducted sounds are saccular dependent [10]. cVEMP is a well-tolerated test for screening vestibular function in young children, performed with minimal test time and with reproducible results [11].

Hearing loss is usually diagnosed early in life. Although an early intervention focusing on the development of communication skills is initiated, vestibular function deficits in hearing-impaired children are overlooked and have not been investigated thoroughly. In contrast to the adult population, early detection of peripheral vestibular dysfunction in the pediatric population can not only help clinicians and parents understand the problem but also facilitates children's learning of compensation strategies for balance control [12].

Focusing on this rationale, the aim of this study was to examine saccular function using cVEMPs in children with congenital or acquired SNHL.

Patients and methods

This study was carried out on 55 children ranging in age from 5 to 18 years. The patient group included 30 children, mean age 11.16 ± 3.95 years, with different degrees of SNHL with normal middle ear function. Patients were recruited from the Audiology Outpatient Clinic of Kasr El-Aini Hospitals. The exclusion criteria included the presence of inner ear anomalies, conductive, mixed hearing loss or otitis media, ear drum perforations, and any complaints of vertigo or dizziness.

Twenty five healthy typically developing children, mean age 11.57 ± 3.89 years, with normal hearing sensitivities (age and sex matched with the patient group) were included in the control group. They were recruited among relatives of patients attending the Audiology Outpatient Clinic of Cairo University Hospitals.

The audiological assessment was carried out at the Audiology Outpatient Clinic of Kasr El-Aini Hospitals. This research was carried out in the period between July 2010 and December 2011 and the study protocol was approved by the Otolaryngology Department Council of Cairo University. A written consent to participate in this research was obtained from the children's parents before commencement of the study. Thereafter, each child under study was subjected to the following:

Full history taking
This was performed in the presence of the parents of the child and included information on personal history, consanguinity, prenatal, natal, and postnatal and developmental history. Detailed history of the hearing loss and any associated balance problems and hearing aid (HA) use were discussed extensively. This was followed by a full otologic examination.

Basic audiological assessment
Pure tone audiometry was performed at frequencies of 250, 500 Hz, 1, 2, 4, and 8 kHz using a dual-channel clinical audiometer (MADSEN Orbiter 922, version 2; GN Otometrics, Taastrup, Denmark) with TDH 39 earphones. Immittancemetry including tympanometry and acoustic reflex threshold measurement (ipsilateral and contralateral at frequencies 500 Hz, 1, 2, and 4 kHz) was also performed using a GSI 33 (Grason Stadler) middle ear analyzer (Viasys Healthcare Inc., Eden Prairie, Minnesota, USA) calibrated according to the ISO standards. Immittancemetry was performed to exclude patients with middle ear pathologies.

Hearing loss was divided (according to the pure tone audiometry average) [13] into the following categories:

Mild SNHL: between 25 and 40 dB HL.

Moderate SNHL: between 41 and 55 dB HL.

Moderately severe SNHL: between 56 and 70 dB HL.

Severe SNHL: between 71 and 90 dB HL.

Profound SNHL: greater than 90 dB HL.

Cervical vestibular-evoked myogenic potentials
This was performed using a Synapsys Auris one-channel auditory-evoked potential and VEMP (Synapsys, Marseille, France). First, the skin was cleaned carefully before application of the electrodes to ensure that the impedance was less than $5 k\Omega$. The surface electrodes were placed as follows: active electrode on the middle-third of the SCM muscle, with the reference electrode on the upper sternum and the ground electrode on the forehead. Children were placed in a supine position and asked to raise and turn their heads contralateral to the ear being tested to achieve maximal contraction of the SCM

muscle ipsilateral to the side of stimulation. For the recording of the cVEMP response, clicks were presented through the TDH 39 headphones (Audiometrics Inc., Oceanside, California, USA). Stimuli were presented monaurally at an intensity of 100 dB normalized HL (nHL) at a frequency of 5 Hz. The analysis time window was 50 ms and the responses of 250 stimuli were averaged. The stimulus intensity level of 100 dB nHL was used as a default starting intensity; two trials were carried out at each intensity to ensure reproducibility. The stimulus intensity was decreased in 10 dB steps or increased in 5 dB steps depending on the presence or absence of cVEMP response, respectively. The lowest intensity, at which a clear and repeatable biphasic wave was observed, was recorded as the cVEMP threshold. If no reliable response was obtained, the cVEMP threshold was considered as absent but the threshold was recorded as 10 dB higher than the maximal intensity of the stimulus (110 dB nHL). This was used for statistical analysis. The cVEMP amplitude (peak to peak), P13 and N23 latencies were measured at a stimulus level of 100 dB nHL. Waves were evaluated as present or absent; P13 and N23 latencies, P13–N23 amplitude, and cVEMP threshold were assessed. Positive cVEMP was defined as an initial positive polarity (P13), ~13 ms after stimulus onset, and subsequent negative polarity (N23), nearly at 23 ms, giving rise to a biphasic P13–N23 wave.

Radiologic evaluation

Computed tomography (CT) scan of the temporal bone was carried out for all patients. Any patient with any inner ear anomaly or pathology was excluded from the study. CT imaging was performed using a GE light speed plus multi-slice machine (four slices) (General Electric Company, Fairfield, Connecticut, USA). A noncontrast CT scan of the petrous temporal bone in the axial plane was performed. The radiological dimensions of the vestibular aqueducts were measured at both the operculum and the midpoint on both the right and the left sides. CT scans were also checked as a whole for any congenital anomalies or pathologies.

Statistical analysis

An International Business Machine Corp. (IBM, Armonk, New York, USA) compatible personal computer was used to store and analyze the data. Analysis of data was carried out using Statistical Package for the Social Sciences (SPSS) 17 for Windows (SPSS, Armonk, New York, USA).

Description of the variables was presented as follows:

(1) Description of quantitative variables was in the form of mean, SD, minimum, and maximum.
(2) Description of qualitative variables was in the form of numbers and percentage (%).

Data were explored for normality using the Kolmogorov–Smirnov test of normality. The results of the Kolmogorov–Smirnov test indicated that some data were normally distributed (parametric data) and some data were not normally distributed (nonparametric data); thus, both parametric and nonparametric tests were used for comparisons.

(1) Comparison between parametric quantitative variables was carried out using the Student t-test of two independent samples. Results were expressed as P-values.
(2) Comparison between nonparametric quantitative variables was carried out using the Mann–Whitney U-test. Results were expressed as P-values.
(3) Comparison between qualitative variables was carried out using the χ^2-test. The Fisher exact test was used instead of the χ^2-test when one expected cell or more were 5 or less.
(4) Binary correlation was carried out by the Pearson correlation test in most of the cases or the Spearman correlation test in the case of categorical ordinal variables 'hearing level'. Results were expressed as correlation coefficients (R) and P-values.

The significance of the results was assessed as a P-value that was differentiated into the following:

(1) Nonsignificant when P-value more than 0.05.
(2) Significant when P-value 0.05 or less.
(3) Highly significant when P-value 0.01 or less.

Results

This study included 30 patients (60 ears) and 25 controls (50 ears), representing 54.5 and 45.5% of the entire study group, respectively. Of the 30 patients (60 ears), 18 (60%) were females and 12 (40%) were males, and of the 25 controls (50 ears), 12 (48.0%) were females and 13 (52.0%) were males. There was no significant difference between the patient and the control groups in terms of sex ($P = 0.208$).

The age of the patients and the controls included in this study ranged between 5 and 18 years, with a mean age of 11.16 ± 3.95 and 11.57 ± 3.89 years of the patients and the controls, respectively ($P = 0.589$).

The entire control group had normal hearing threshold levels (100%; 25 controls; 50 ears). The patient group was divided into mild SNHL (11.7%; seven ears), moderate SNHL (18.3%; 11 ears), and moderately severe SNHL (18.3%; 11 ears). Severe SNHL was 30.0% (18 ears) and profound SNHL was 21.7% (13 ears) (Fig. 1). There was a highly significant difference between the control and the patient groups in terms of the hearing levels ($P < 0.0001$).

The percentage of positive consanguinity was 63.3% (19 patients) in the patient group and 20.0% (five controls) in the control group ($P < 0.0001$). The cause of hearing loss in all of the patients included in this study was not identified. The presence of a large percentage of patients with positive consanguinity suggests that the cause of the hearing loss might be congenital nonsyndromic SNHL in most of the patients.

Forty percent of the patients were using HAs (24 ears); 10 children were wearing binaural HAs (20 ears) and four children were wearing it only on the right side (four ears), whereas 60% of the patients did not use HAs (36 ears).

On comparing the different cVEMP measurements in the patient and the control groups, the results showed that there was a statistically nonsignificant difference between the patients and the controls in P13 and N23 latencies ($P > 0.05$). The P13–N23 amplitude and cVEMP threshold results between the patients and the controls showed a highly significant difference ($P < 0.0001$). The P13–N23 amplitude was lower in the patient group and the cVEMP threshold was higher in the patient group compared with the control group (Table 1).

cVEMP measurements in relation to sex within the patient group showed no significant difference between males and females in the different cVEMP outcomes ($P > 0.05$), except the cVEMP threshold, which showed a statistically significant difference ($P = 0.03$). Females had higher cVEMP thresholds than males (Fig. 2).

Correlation between different parameters and cervical vestibular-evoked myogenic potential outcomes

Correlation between hearing level and different cervical vestibular-evoked myogenic potential outcomes among the study group
Table 2 shows a highly significant negative correlation between hearing level and the P13–N23 amplitude and a highly significant positive correlation between hearing level and the cVEMP threshold among the study group ($P < 0.0001$). Hence, an increase in the hearing level leads to a decrease in the P13–N23 amplitude and an increase in the cVEMP threshold.

Correlation between hearing loss and different cervical vestibular-evoked myogenic potential outcomes within the patient group
Table 3 shows a highly significant positive correlation between hearing loss and the cVEMP threshold among the patient groups with different degrees of hearing losses ($P = 0.002$). The cVEMP threshold increases with an increase in hearing loss. There was no significant difference between the other cVEMP outcomes and the different hearing loss patient groups ($P > 0.05$).

Correlation between age and different cervical vestibular-evoked myogenic potential outcomes among the patient group
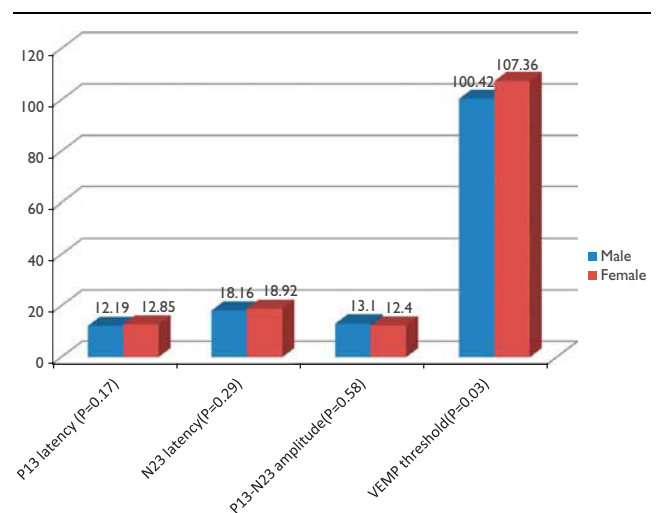Table 4 shows a significant positive correlation between age and P13 latency and a highly significant positive correlation between age and N23 latency among the patient group ($P = 0.049$ and 0.002, respectively). P13

Figure 1
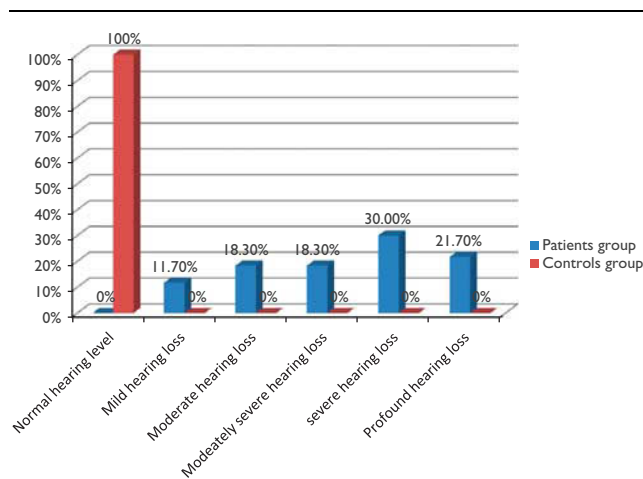
Distribution of hearing threshold levels among the patient and the control groups.

Figure 2

Cervical vestibular-evoked myogenic potential (cVEMP) outcome in relation to sex within the patient group. Latency = ms; amplitude = μV; threshold = dB (nHL).

Table 1 Cervical vestibular-evoked myogenic potential outcome distribution among the patient and the control groups

cVEMP outcomes	Patient group		Control group		Test	P value	Significance
	Mean	SD	Mean	SD			
P13 latency	12.52	1.50	12.19	1.76	t-Test 0.936	0.352	NS
N23 latency	18.53	2.24	18.67	2.70	t-Test −0.262	0.794	NS
P13–N23 amplitude	12.75	5.26	24.84	15.76	Mann–Whitney 517.5	<0.0001	HS
cVEMP threshold	104.58	12.12	91.30	3.00	Mann–Whitney 480	<0.0001	HS

Latency = ms; amplitude = μV; threshold = dB (nHL).
cVEMP, cervical vestibular-evoked myogenic potential; HS, highly significant.

Table 2 Correlation between the hearing level and the different cervical vestibular-evoked myogenic potential outcomes among the study group

cVEMP outcomes	Patient group mean	Control group mean	Correlation coefficient 'r'	P value	Significance
P13 latency	12.52	12.19	0.163	0.125	NS
N23 latency	18.53	18.67	0.022	0.838	NS
P13–N23 amplitude	12.75	24.84	−0.393	<0.0001	HS
cVEMP threshold	104.58	91.30	0.691	<0.0001	HS

Latency=ms; amplitude=μV; threshold=dB (nHL).
cVEMP, cervical vestibular-evoked myogenic potential; HS, highly significant.

Table 3 Correlation between hearing loss and different cervical vestibular-evoked myogenic potential outcomes within the patient group

cVEMP outcomes	Mild HL	Moderate HL	Moderately severe HL	Severe HL	Profound HL	Correlation coefficient 'r'	P value	Significance
P13 latency	12.01	13.36	12.07	12.10	13.15	0.065	0.691	NS
N23 latency	17.62	18.92	18.12	17.65	20.96	0.238	0.144	NS
P13–N23 amplitude	11.14	16.33	8.71	13.73	12.17	0.015	0.927	NS
cVEMP threshold	92.86	100.00	107.27	106.94	109.23	0.392	0.002	HS

Latency=ms; amplitude=μV; threshold=dB (nHL).
cVEMP, cervical vestibular-evoked myogenic potential; HL, hearing loss; HS, highly significant.

Table 4 Correlation between age and different cervical vestibular-evoked myogenic potential outcomes among the patient group

cVEMP outcome	Correlation coefficient 'r'	P value	Significance
P13 latency	0.314	0.049	S
N23 latency	0.473	0.002	HS
P13–N23 amplitude	−0.009	0.956	NS
cVEMP threshold	0.210	0.108	NS

Latency=ms; amplitude=μV; threshold=dB (nHL).
cVEMP, cervical vestibular-evoked myogenic potential; HS, highly significant; S, significant.

and N23 latencies increase with age. There was no significant correlation between age and P13–N23 amplitude and the cVEMP threshold ($P > 0.05$).

Discussion

Over the last decade, increasing emphasis has been placed on the need for the early identification of children with hearing impairments, with the target of having such children aided by the age of 6 months, a goal that is now being achieved routinely. In the setting of hearing rehabilitation, there is a clear relationship between early intervention and outcome. Although difficult to estimate because of the impact of etiology and residual thresholds, for all-comers with SNHL, the incidence of a simultaneous vestibular lesion is likely around 30% [14].

Despite this correlation, we have not adopted the same aggressive approach for the identification and rehabilitation of the concurrent vestibular impairment. There are a number of reasons why we should strive to do so. First, identification of peripheral vestibular dysfunction prevents the false labeling of children as having a global delay, central lesions, or multiple handicaps. Second, different therapeutic approaches can be used for the rehabilitation of children with either loss of vestibular sensitivity or deficits in sensory organization [15].

Documentation of vestibular function and dysfunction in children with hearing impairment has a long and rich history, which has indicated that 20–85% of children with hearing loss have some form of vestibular end organ dysfunction [15–17].

This study was carried out on 30 children (54.5%) with different degrees of SNHL (from mild to profound) and 25 children (45.5%) with normal hearing levels as the control group (Fig. 1). cVEMP was carried out to detect saccular function; all parameters of the cVEMP response were examined (P13 and N23 latencies, P13–N23 amplitude, and cVEMP threshold).

cVEMP outcome distribution among the patient and the control groups showed that there was no significant difference between the two groups in terms of P13 and N23 latencies (Table 1). Children with SNHL presented with normal P13 and N23 latencies. Kelsch et al. [11] have reported normative data of cVEMPs obtained with a click in young children. Their latency values of P13 and N23 were 11.30 ± 1.30 and 17.60 ± 1.40 ms, respectively. Picciotti et al. [7] reported mean latencies of P13 and N23 of 16.14 ± 2.81 and 21.38 ± 3.04 ms, respectively.

In our study, there was a highly significant difference between the patient and the control groups in the P13–N23 amplitude and the cVEMP threshold (Table 1). Rosengren et al. [6] reported that patients with SNHL had higher cVEMP thresholds than the normal controls. Zhou et al. [5] reported that abnormal cVEMPs were found in 21 of 23 children (91%) with SNHL. The thresholds of cVEMP were significantly higher ($P < 0.001$) and the amplitudes were lower in children with SNHL than those in children with normal hearing. There were no differences in the P13 and N23 latencies between the two groups.

The relation between sex and cVEMP outcomes was examined in the present research. Females with SNHL presented with significantly higher cVEMP thresholds than males (Fig. 2). This is in agreement with other studies that reported no sex-related differences in

cVEMP amplitudes or P13 and N23 latencies [18,19]. Ochi and Ohashi [18] reported no significant sex-related differences both in the threshold and in the P13–N23 amplitude. Felipe *et al.* [20] reported that males presented with higher cVEMP amplitudes than females and attributed this to the difference in muscle tone between both sexes. This postulation might also explain the presence of significantly lower cVEMP thresholds in males compared with females.

The correlation between the hearing levels with the different cVEMP outcomes showed that in the study group as a whole, both the P13–N23 amplitude and the cVEMP threshold were highly significantly correlated to the hearing level. In the patient group, however, only the cVEMP threshold showed a highly significant correlation to the hearing loss. This suggests that the cVEMP P13–N23 amplitude decreases with hearing loss irrespective of its degree (Tables 2 and 3).

In contrast, the correlation between hearing level and P13 and N23 latencies in this study showed no significant differences (Tables 2 and 3). This is in agreement with the results of Sazgar *et al.* [21], who compared the results of cVEMP outcome in 50 patients with SNHL with 32 volunteers with normal hearing and found that no significant differences exist between the two groups in the P13 and N23 latencies.

The correlation between the degree of hearing loss and different cVEMP outcomes in children with SNHL showed a highly significant positive correlation between the degree of hearing loss and the cVEMP threshold (Table 3). The cVEMP threshold increases with the increase in the severity of hearing loss. Our results confirm that cVEMPs can be recorded using acoustic stimulation despite the presence of severe degrees of hearing loss.

Although the relationship between vestibular and auditory function is not simple, they do appear to be associated. A number of studies have shown that, at least on a group level, the likelihood of a vestibular impairment relates to the degree of hearing loss [17]. It is generally believed that nonsyndromic recessive causes of deafness, of which the most common are defects in the gap junction B2 (*GJB2*) gene, are not typically associated with concurrent deficits in vestibular end organ function [22]. This emphasizes the need to carry out further studies on the genetic causes of hearing loss and its relation to the VEMP response.

Tribukait *et al.* [23] reported that if the hearing level was better than 90 dB (pure tone average of 0.5, 1, and 2 kHz), vestibular function was often normal. For hearing levels of 100–120 dB, otolith function decreased significantly. Similarly, the proportion of individuals with vestibular impairment is significantly lower (20–36%) in children with a hearing threshold of less than 90 dB and higher (80%) in those with more severe hearing loss [17]. However, hearing thresholds are not predictive for the individual case. A study has reported that 16% of children with relatively good auditory sensitivity showed complete absence of vestibular function, whereas 43.3% of the children with the poorest auditory sensitivity had normal responses. The relationship between auditory and vestibular function is certainly complex. The intricacy of this interaction is particularly evident in cases where vestibular function is well preserved 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 [15]. Zhou *et al.* [5] have reported that they did not find a clear relationship between the degree of hearing loss and the severity of saccular dysfunction.

A study examining vestibular responses in children attending a school for the deaf noted that with the exception of meningitis, it was absolutely impossible to predict the vestibular response of a child on the basis of the etiology of deafness. They observed 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 [14].

Understanding the correlation between vestibular function and hearing impairment is important as vestibular dysfunction may lead to delays in reaching motor milestones that may provide an indication of either a progressive or a missed hearing loss. Proper assessment of a gross motor delay (sitting, walking) in the absence of deficits in fine motor function could lead to an earlier identification than would occur with the failure to develop language, the true hallmark of hearing loss [7].

During early development, the otic vesicle divides into several chambers, including a utricular chamber, which gives rise to the utricle and the semicircular canals (SSCs), and a saccular chamber, which gives rise to the saccule and the cochlea. The inner ear is often viewed to consist of two separate divisions. The superior division consisting of the three SSCs and the utricle and the inferior division includes the saccule and the cochlea [14].

Given the anatomic compartmentalization of the saccule and the cochlea, one might predict that saccular function may be more likely affected than utricular or SSC function in the presence of an inner ear injury, leading to SNHL. It is therefore reasonable to theorize that in some instances, lesions or insults that lead to auditory dysfunction may also lead to dysfunction of the auditory end organs. In turn, dysfunction of the vestibular end organs may cause disruption in the ability to maintain static and dynamic balance [14].

Although peripheral vestibular function is an important consideration in the evaluation of children with SNHL, what is likely more important clinically is their ability to maintain balance to a sufficient degree to carry out their activities of daily living [15]. It remains unclear why many hearing-impaired children with abnormal cVEMP 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. Hence, future studies with a more comprehensive investigation, including a standardized balance questionnaire for the children and their parents or caregivers and other vestibular evaluation procedures, are required to address these issues [5].

The variability in the relationship between auditory and vestibular function can certainly be linked to differences in the etiology of the inner ear disorder. In addition to etiology, however, the degree of SNHL may also aid in predicting the likelihood of an associated loss of vestibular function [15].

Children may be candidates for unilateral or bilateral cochlear implantation (CI). We need to be certain that our baseline measures are accurate and adequately reflect functional outcome. An understanding of baseline vestibular function may also allow physicians to experiment with the properties of CI in an effort to increase the quality of the sensory information provided to children with concurrent lesions of the cochlea and the labyrinth [24]. Compared with the horizontal SSC, the saccule may be more susceptible to damage than the utricle or SSCs because of its proximity to the insertion path of the implant's electrode array [25].

Basta *et al.* [26] reported that chronic, persisting dizziness after CI surgery is largely based on a dysfunction of the saccular macula, which is an integral component of the otolith system. This saccular impairment is induced, most likely, by the insertion trauma of the CI electrode when advancing it into the inner ear. A possible coactivation of the inferior vestibular nerve by the electrical stimulation might play an additional role in the pathogenesis of the persisting postsurgical dizziness. cVEMP testing will allow parents to be more completely informed of the risk of vestibular impairment should the implant be carried out on a functional vestibule. Furthermore, this non-negligible risk of permanent vestibular dysfunction would argue against bilateral CI in a single surgical procedure without a previous vestibular assessment [25].

Our results showed that cVEMP P13 and N23 latencies were positively correlated with age; aging leads to an increase in P13 and N23 latencies (Table 4). This positive correlation has also been reported by other investigators [5,27]. In contrast, Basta *et al.* [19] and Welgampola and Colebatch [28] reported no difference in latency measurement across age groups. The latter authors, however, reported a weak correlation between N23 latency and age. Knowing that the degree of SCM contraction does not affect cVEMP latencies, the disagreement among the reported findings may be related to differences in recording techniques such as variations in stimuli and filter settings. In relation to the frequency of the stimuli, P13 latencies were shown to be prolonged as the frequency of the stimulus decreased [29,30].

This study showed no significant correlation between age and cVEMP P13–N23 amplitude and cVEMP threshold (Table 4). This is in agreement with the results of Janky and Shepard [31], who reported no significant correlation between age and the cVEMP threshold in response to click stimulation.

Reductions in the cVEMP amplitude with increased age, however, have been reported by some investigators [19,27,28,32]. They attributed this to the fact that cVEMP amplitudes are linearly dependent on the tonic activity of the SCM. This may be caused by the decrease in vestibular hair cells, Scarpa's ganglion cells, and cells of the vestibular brain-stem during the aging process [27]. Others have attributed this to a subsequent decline in overall neuroanatomy and physiological function as, with age, there is a decrease in the number of otoconia, specifically within the saccule, as well as decreased number of neurons within the medial vestibular nucleus [33].

In summary, the cVEMP test represents a useful diagnostic tool for the diagnosis of vestibular function, alone or in combination with conventional vestibular tests, in order to comprehensively evaluate all the vestibular systems in children. In fact, it has major advantages: it is an objective, noninvasive, low-cost, quick exam that is easy to perform, with no discomfort for the individual being evaluated [20]. By measuring all parameters of the cVEMP response, the sensitivity of detecting minor changes in saccular function is enhanced.

Conclusion

From this study, the authors could conclude that SNHL is associated with saccular dysfunction in the pediatric population, as indicated by the presence of abnormal cVEMP responses, namely, decreased P13–N23 amplitudes and elevated cVEMP thresholds compared with their normal peers.

Females presented with higher cVEMP threshold values compared with their male counterparts and cVEMP latencies appear to increase with age. An increase in hearing loss is associated with an increase in the cVEMP threshold and the cVEMP amplitude appears to decrease with hearing loss irrespective of its degree. Our research confirmed the assumption that cVEMPs could be recorded despite the presence of severe degrees of SNHL.

Inclusion of cVEMPs in the battery of evaluation of children with SNHL to detect early subtle changes in saccular function is highly recommended, as minor vestibular deficits can be easily overlooked in children. Future researches focusing on the genes causing SNHL and its relation to VEMP findings should be carried out. We also suggest the inclusion of cVEMP testing in the battery of investigations for CI candidates.

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