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Functional Dizziness: Diagnostic Keys and Differential Diagnosis

Thomas Brandt[1], Doreen Huppert[1], Michael Strupp[2], Marianne Dieterich[2,3]

In the 1990s, 9 % of neurological inpatients were found to have functional (then called psychogenic or somatoform) rather than structural neurological disorders of the nervous system as the primary cause of admission [1]. This is a conservative figure, since secondary and minor pseudo-neurological symptoms were not included; other studies later found up to 18–20 % [2]. In a further study, it was reported that 61 % of patients referred to a neurology service had at least one medically unexplained symptom, and 35 % fulfilled the diagnostic criteria for an ICD-10 somatoform disorder [3].

Functional dizziness is one of the most frequent functional disorders in adult in- and outpatients. In a tertiary referral dizziness unit, it accounted for 19.5 % of 17,700 adult outpatients; thus, it is the second most common diagnosis after benign paroxysmal positional vertigo [4]. The frequencies vary for different countries and study designs: 2.5 [5], 16 [6], and 23 % [7] have been reported. Functional dizziness is also a relevant diagnosis in children. Its frequency peaks at 21 %, and it is an important differential diagnosis for migrainous vertigo, which has the highest frequency (39 %) in childhood [8].

The term "functional dizziness" is not a nosological entity but comprises several diagnoses. To complicate matters, a functional component enhances, overlaps, or prolongs the symptomatology of vestibular or central nervous system disorders in many patients. A typical development is, for example, the evolution of vestibular neuritis into phobic postural vertigo, which was documented by artificial neural network posturography [9]. Therefore, it was proposed to clinically differentiate between primary and secondary somatoform (now functional) dizziness [10, 11]. This is particularly relevant for unpredictable episodic vertigo syndromes (like vestibular migraine or Menière's disease), especially when they remain undiagnosed or are poorly explained to the patient. Sometimes it is clinically difficult to differentiate primary vestibular from secondary functional symptoms.

Many clinicians hesitate to use the diagnostic label "functional" because they fear the consequences of a potential misdiagnosis and the danger of overlooking a vestibular, neurological, or psychiatric disorder. In the following we draw on our experience in the German Center for Vertigo and Balance Disorders to provide both typical positive criteria and unlikely signs and symptoms in patients presenting with functional dizziness syndromes. In Table 1, we define key signs that may sometimes even uncover deliberate aggravation and malingering.

With regards to the above features, it is our expert opinion that chronic subjective dizziness [12] without associated neurological or otoneurological dysfunction has no relevant vestibular or neurological differential diagnosis. Features 2–5 have one characteristic in common—a dissociation of subjective complaints and objective findings. Anxiety is a central feature, but it is often revealed only by carefully taking the patient's history. Vestibular and neurological dizziness syndromes do not improve with alcohol consumption. The combination of dizziness with non-vestibular complaints refers to the association of abdominal or heart symptoms, muscle weakness, dysesthesia, pain syndromes (except headache), or sleep disturbances. In contrast to causes of chronic dizziness in bilateral vestibulopathy or downbeat nystagmus syndrome, patients with phobic postural vertigo show a circadian rhythm with minimal symptoms in the morning [13]. Situational or social triggers of dizziness and their avoidance are not typical for vestibular or neurological disorders. If a patient complains about an acute rotational vertigo during the physical examination, but the physician cannot observe a spontaneous nystagmus, then a vestibular cause is unlikely. Finally, a number of functional disorders of posture and gait are characterized by unusual and bizarre motor patterns like excessive slowness, hesitation, momentary fluctuations, improvement of Romberg test values during distraction, "walking on ice" gait pattern, or sudden buckling [14]. Here, ostentatious behaviour such as mannered posturing of the hands, a facial expression of

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Table 1: Features typical for a functional dizziness syndrome.

1.	Chronic spontaneous dizziness or unsteadiness lasting for months or longer
2.	Dissociation between objective balance tests and subjective imbalance
3.	Fear of falls without a history of falls
4.	Improvement during bodily activity, mental distraction or after alcohol consumption
5.	Inappropriate excessive anxiety or fear of impending doom
6.	Dizziness combined with non-vestibular or non-balance symptoms
7.	Situational or social events as triggers of dizziness and avoidance behavior
8.	Rotational vertigo without concurrent spontaneous nystagmus
9.	Unusual or bizarre postural and gait patterns
10.	Chronic unsteadiness and dizziness following transportation in vehicles

suffering, or moaning, supports the suspicion of a functional disorder. Psychiatrically, the functional disorders of stance and gait are classified among dissociative disorders and somatic symptom and related disorders [15, 16]. Mal de debarquement syndrome refers to a sensation of swinging, swaying, unsteadiness, and disequilibrium that is preferably experienced in sea travel (less often other forms of travel) immediately on disembarking and may persist for weeks to years in some individuals.

With the Table 2 of unlikely or atypical features [17] we would like to stress their non-functional origin. For example, spontaneous episodic vertigo or dizziness attacks with symptom-free intervals are rarely functional, but are a criterion of vestibular disorders such as Menière's disease, vestibular migraine, or vestibular paroxysmia. Nausea and emesis most often indicate an acute peripheral labyrinthine, vestibular nerve, or vestibular nucleus lesion. Rotational vertigo with a directional pulsion, deviation of gait, and a tendency to fall also points to vestibular disorders. A combination of dizziness with hearing loss, tinnitus, or fullness of the ear is typical for peripheral vestibular dysfunction, especially Menière's disease and less frequent in vestibular migraine. Head and body movements relative to gravity indicate benign paroxysmal positional vertigo; head rotations may indicate vestibular paroxysmia due to neurovascular cross-compression. Patients presenting with functional dizziness may spontaneously blame physical (rarely psychological) stress as the cause of their dizziness.

Dizziness or vertigo may be the primary cause of admission of outpatients suffering from a psychiatric disorder. The most

Table 2: Features atypical for a functional dizziness syndrome.

1.	Frequent episodic vertigo/dizziness attacks with symptom-free intervals
2.	Nausea and emesis
3.	Rotational vertigo with directional pulsion or falls
4.	Dizziness/vertigo with concomitant auditory symptoms
5.	Head rotation or head tilt as specific triggers
6.	Spontaneous suspicion of patients that psychological (not physical) stress is causative

frequent of these are listed below according to DSM-V and the ICD-10 classification [15, 16]:

- Anxiety disorders (specific phobias, panic disorders),
- Depressive disorders,
- Somatic symptom and related disorders,
- Posttraumatic stress disorders.

Dizziness and vertigo are not keys that help establish a specific psychiatric diagnosis. Here, psychopathological symptoms are the main indicators. This is also true for specific phobias and panic disorders in which dizziness is mentioned as one among many bodily symptoms. It is well acknowledged that dizziness may be a complaint in many other psychiatric conditions; however, those patients will rarely be seen by physicians who manage dizzy patients.

These vestibular and neurological disorders may be misdiagnosed as functional (Table 3).

Attacks of vestibular migraine with dizziness and postural stability of various durations [18] manifest without concomitant headache in about 30 % of patients [19]. Episodic vertigo syndromes in childhood are associated with migraine in about 50 % of the cases [20]; the frequent benign paroxysmal vertigo of childhood—a migraine equivalent—manifests typically without

Table 3: Differential diagnoses of functional dizziness.

1.	Vestibular migraine without concomitant headache
2.	Sensory polyneuropathy
3.	Bilateral vestibulopathy
4.	Side effects of medication
5.	Alcohol or substance abuse
6.	Orthostatic hypotension or hypertensive crises
7.	Major and mild cognitive impairment
8.	Mild degenerative cerebellar ataxia and downbeat nystagmus syndrome
9.	Orthostatic tremor
10.	Superior canal dehiscence syndrome

headache. The physical examination of dizzy patients should include sensory testing for polyneuropathy; it frequently occurs in elderly patients and is often combined with visual deficits and bilateral vestibulopathy. Patients with bilateral vestibulopathy are symptom-free if they do not move, but they typically complain of oscillopsia with head movements (due to the defective vestibulo-ocular reflex) and imbalance during locomotion in darkness or on uneven ground when vision and proprioception cannot substitute for the vestibular deficit. Numerous medications, alcohol, or substance abuse cause dizziness and numbness; thus, the patient history must clarify this point. Fluctuating alertness, slowing of cognitive processing, and motor performance, especially postural imbalance and falls, are hints of an abuse. Hypotensive and hypertensive blood pressure dysregulation may also cause dizziness, which is often described as numbness or lightheadedness. Dizziness may be the leading complaint of patients with mild cognitive impairment or dementia. The same is true for beginning degenerative cerebellar ataxias, which can be identified by ocular motor and other cerebellar signs of motor performance. Rare orthostatic tremor is characterised by a 13–18 Hz body sway which can sometimes be visually observed but has to be confirmed by posturography or electromyography. Superior canal dehiscence syndrome is also rare and characterized by spells of vertigo and oscillopsia induced by coughing, Valsalva maneuver, and sometimes even loud sounds. It is caused by a bony apical defect of the superior semicircular canal, which can be shown by thin-slice CT of the petrous bone.

We would like to encourage those who manage dizzy patients to discuss with the patient the functional genesis of the mechanisms causing the sometimes frightening dizziness and subjective postural instability. The readiness of most patients, who experience much stress as a result of their suffering, to understand the functional mechanism and to overcome it by desensitization is a positive experience for both the physician and the patients [4]. However, physicians should avoid a dichotomous thinking of functional versus organic disorders for at least two reasons: first, both conditions frequently overlap, and second, a vestibular condition can change into a functional condition. Various cognitive, educational, behavioural, physical, and medical therapies have to be applied for the individual patient and the different conditions summarized under the umbrella term "functional dizziness."

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Rocking Pneumonia

Ger T. Rijkers*, Maria Rodriguez Gomez

Abstract

Ever since Chuck Berry coined the term "rocking pneumonia" in his 1956 song "Roll over Beethoven," pneumonia has been mentioned frequently in modern blues and rock songs. We analyzed the lyrics of these songs to examine how various elements of pneumonia have been represented in popular music, specifically the cause of pneumonia, the risk groups, comorbidity (such as the boogie woogie flu), the clinical symptoms, and treatment and outcome. Up to this day, songwriters suggest that pneumonia is caused mainly by the cold and rain and that treatment is hardly possible, aside from a shot of rhythm and blues.

Keywords: Rocking pneumonia, Lyrics, Symptoms, Comorbidity, Treatment

In 1926, Virginia Woolf published an essay "On being ill," in which she wondered why "illness has not taken its place with love, battle and jealousy among the prime themes of literature" [1]. Illness has not been a prime theme of literature, or music lyrics, for that matter, yet it has a significant impact on the human condition. Pondering this oversight, Woolf specifically called for odes to pneumonia, novels devoted to influenza, epic poems to typhoid, and lyrics to toothache.

Now, more than 90 years after Virginia Woolf's essay, this question regarding illness's representation in literature can be addressed with modern search techniques. The lyrics database www.lyrics.com considers itself "the web's largest resource for music, songs and lyrics" and it is the latter functionality that was used for this research into how illness—specifically pneumonia—has been represented in music lyrics.

Search results revealed that the word "pneumonia" is found in 239 song lyrics, which is a rather modest number when compared to the word "jealous," which is found in 3879 lyrics, and "love," which found in 324,016 lyrics. Pneumonia, however, clearly outperforms the other diseases suggested by Virginia Woolf: (novels devoted to) influenza, 29; (epic poems to) typhoid, 11; and (lyrics to) toothache, 73. In her essay she suggested "love and jealousy are states that spark the language but that the sufferer has the pain in his head but the language runs dry" [1]. This suggestion of the experience of pain overruling the ability to of language to adequately describe it warrants an analysis of the language that is used in songs about pneumonia.

"I got a rocking pneumonia, I need a shot of rhythm and blues" [2] is a famous line from the song "Roll over Beethoven", a Chuck Berry original (1956) which has been covered by many others, including The Beatles (1963) and Electric Light Orchestra (1973). Briefly, to highlight one way in which an understanding of pneumonia in lyrics can be utilized in an academic setting, Chuck Berry's lyric about pneumonia could be used in a creative multiple-choice question in an immunology or microbiology exam, structured as follows:

Streptococcus pneumoniae can cause: a) otitis media, b) meningitis, c) pneumonia and d) bacteremia. Which one of these four manifestations of a pneumococcal infection did Chuck Berry have for which he needed a shot of rhythm and blues?

We have put this question to bachelor students at the University College Roosevelt (The Netherlands) and the frequency distribution of the answers is an almost perfect 25% for each possibility, so the general knowledge of university students of classical rock and roll song texts appears to be limited. It can only be hoped that their knowledge of the pneumococcus is better.

Chuck Berry's "Roll over Beethoven" was recorded in 1956 (Fig. 1). The top position the song reached in the Billboard Hot

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Fig. 1: Chuck Berry's "Roll over Beethoven" and Huey "Piano" Smith's "Rockin Pneumonia & the Boogie Woogie Flu."

100 was a modest 29th place. About a year later, "A Rockin' Pneumonia and the Boogie Woogie Flu" by Huey Smith and his Clowns made its first appearance on Billboard's rhythm and blues chart. In his biography, Huey Smith admits that he had heard Chuck Berry sing "I got the rocking pneumonia, I need a shot of rhythm and blues" [3]. He was inspired to use this term and added "the boogie woogie flu" himself.

As indicated above, a number of songs have been published since Chuck Berry's "Roll over Beethoven", dealing with many aspects of pneumonia. Below, we analyze lyrics that deal with the cause of pneumonia, risk groups, comorbidity (the boogie woogie flu), the clinical symptoms, and treatment and outcome.

Cause of Pneumonia

In most songs about pneumonia, standing in the rain or in the cold is indicated as cause of the disease. For example, Rod Stewart in "Lost Paraguayos" sings: "it appears to be raining again,... honey hurry I'm catching pneumonia" (from the album *Sing It Again Rod*, 1972). The Easybeats in "Come In You'll Get Pneumonia" sing "Standing in the rain, [...] all right, come in you'll get pneumonia" (from the album *The Easybeats*, 1981). As recently as 2013, Pusha T in his song "Amen" (from the album *Still Ya Pusha*) sings/raps "And you might get pneumonia, I'm colder than an elf on a sleigh". The last example is Elvis Presley, from the movie *GI Blues* the song "Didja' Ever" (from the album *G. I. Blues* 1960), who sings, "Ya get up in the morning and turn the shower on, you're gettin' pneumonia, the hot water is gone."

In fact, there is only one song in which an infectious nature of pneumonia is described, "I'll Never Fall in Love Again," with the lyrics: "What do you get when you kiss a girl? You get enough germs to catch pneumonia," originally composed by Burt Bacharach in 1969. At the time he composed the song, Bacharach himself was in hospital with pneumonia. There are many different artists who have recorded this classical song. Among them, Dionne Warwick who sings, as all female interpreters do: "What do you get when you kiss a guy?" All male singers kiss a girl. In none of the versions is there a single same sex relation. We can debate on the mode of transmission implied in the lyrics. True, *S. pneumoniae* is readily detectable in saliva. Wyllie *et al*., using molecular techniques, found 88% of primary school children tested positive for *S. pneumoniae* in saliva [4]. In 1932, Gundel and Okura, using conventional culture techniques, reported 66% of saliva samples from teenagers to be positive for *S. pneumoniae* with point prevalence as high as 85% for boys and 71% for girls [5]. The actual volume of saliva exchanged during intimate kissing is less than 0.5 ml, but it can contain up to 80×10^6 (total) bacteria [6]. Still, unlike the Epstein Barr virus, kissing as route of transmission hasn't been firmly established for *S. pneumoniae*.

Risk Groups

Pneumonia can affect people of all ages, but the age groups with highest risk of infection are infants below 2 years of age, and people over 65. This is reflected in popular music, with the lyrics in "Streets of New York" from Wolfe Tones describing one of the risk groups when they sing, "little baby daughter… has pneumonia". Both "Angel of Mercy" from Black Label Society (2014) and "Speed Break" from Abscess also refer to this age group (infants below 2 years of age) as having pneumonia. The older age category is described in "Swing 'em High" from The Tiger Lillies (1999) "and the old lady's pneumonia did rage". Unlike causative agents, as far as risk groups are concerned, these song lyrics accurately describe a fact about pneumonia.

Comorbidities

The intricate association between pneumonia and influenza is mentioned in a number of songs. The classical "Rocking pneumonia and the boogie woogie flu", by Huey Smith already alludes to this association. Also Otis Redding in his 1966 song "Hawg for You" sings, "I got rockin' pneumonia, asiatic flu, I got something to tell you baby!" The Squirrel Nut Zippers in their song "La Grippe" (from the album *The Inevitable*, 1995) also sing, "There's an Asian influenza, infecting us all by the scores, and it's turning into pneumonia." In more recent songs, in fact since the onset of the AIDS epidemic, pneumonia as a manifestation of an underlying HIV infection is also indicated. This is seen in the song "The Kids" by Eminem (from the album *The Marshall Mathers LP*, 2000) where he sings, "Mr. Kaniff is out with pneumonia (HE'S GOT AIDS!)."

Clinical Symptoms

The clinical symptoms of pneumonia (high temperature, pleuritic pain and a dry cough, next to shortness of breath, fatigue, confusion, hypothermia, nausea, vomiting or diarrhea) are not at all adequately described in song lyrics. Lightnin' Hopkins in "Pneumonia Blues" (1964) sings that his "head hurt me so bad till I'm almost to go blind." Although a few cases have been described in the medical literature, blindness as a complication is a rare event [7, 8].

One of the prominent clinical signs of pneumonia, fever, is rarely mentioned in songs about pneumonia. Danko Jones, in his 1999 song "My love is bold" sings, "I got a fever baby's givin' me pneumonia." He is implying that the fever is the cause of the pneumonia, rather than a consequence.

Just a few songs have the single word "pneumonia" as a title. For the purposes of this discussion, which focuses on lyrics, we can ignore "Pneumonia" by Kool and the Gang, because that is an instrumental. Perhaps the most intriguing is Joe Tex's song entitled "Pneumonia." In his early career, Joe Tex claimed he was the author of another song, "Fever," but that he sold it for 300 dollars. According to the label, "Fever" was written by Otis Blackwell in 1956 and first recorded by Little Willie John but made famous by Peggy Lee, Etta James, Madonna, Beyoncé, the Black Keys, and many others. Joe Tex was never recognized as the writer, but 2 years after "Fever" was released, he published his song "Pneumonia." The melody bears strong resemblance to "Fever," and even the lyrics are related (see Table 1). At the place where you would intuitively sing "Fever", he sings "Pneumonia." There never has been a court case for plagiarism, so maybe Joe Tex actually did write "Fever." A direct comparison of the lyrics of "Pneumonia" and "Fever" (Table 1) shows how close the relation between pneumonia and fever is.

Table 1: Comparison of the lyrics of pneumonia (Joe Tex) with fever (Peggy Lee).

Pneumonia	Fever
Oh baby you know that I never did need you	Never know how much I love you
You know that I never did care	Never know how much I care
And if you put your cotton picking arms around me	When you put your arms around me
I'm gonna hit you with this rocking chair	I get a fever that's so hard to bear
You give me...pneumonia	You give me...fever when you kiss me
Yes, you give me pneumonia baby	Fever when you hold me tight
You know the love you gave me has grown so cold	Fever in the morning
I've got pneumonia in my heart and soul	A fever all through the night

Treatment and Outcome

In most songs, pneumonia is described as a serious disease for which no treatment is available. In the 1949 musical romantic comedy "Neptune's Daughter," made during a time when antibiotics were not yet generally available, Ricardo Montalbán and Esther Williams sing the Academy Award winning song "Baby it's cold outside," which includes the line, "If you got pneumonia and died." The only intervention that could help is a shot of rhythm and blues: "I got a rocking pneumonia, I need a shot of rhythm and blues." Other treatment options put forward in songs about pneumonia are even less evidence-based; for example, treatments such as "soy bologna", according to Nellie McKay's song "Suitcase Song" (2004), and "prayers" from Rod Stewart's "Lost Paraguayos."

In the song "Lost Weekend" (2004), Lloyd Cole mentions treatment but complains about the costs of antibiotics ("It took a lost weekend in a hotel in Amsterdam, and double pneumonia in a single room, and the sickest joke was the price of the medicine"). Lloyd Cole must have travelled on a tight budget, because the price of a course of amoxicillin is well below €60, less than a single night in a cheap Amsterdam hotel. This shows us that it took almost half a century of pneumonia being mentioned in song lyrics before the ability to treat pneumonia with antibiotics also found its way into popular music. We may have to wait another 50 years before songwriters will realize that pneumonia can be prevented by vaccination, rather than by an umbrella, or refraining from kissing girls.

Editor-in-Chief's Challenge

Rather than waiting for current knowledge of vaccine efficacy to filter down to popular culture, perhaps the next step in raising awareness about pneumonia is to take our knowledge of the disease and pen a song about pneumonia prevention—who knows, it might just be a hit!

Thus, the *Pneumonia* journal has a holiday challenge for you: to compose lyrics for a song that highlights vaccination as a means of preventing pneumonia. The most fitting submissions will be published as responses to this commentary.

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All references to songs are listed by year of release. If the song is published as part of an album, the name of the album is also indicated.

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The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Authors' contributions
GTR conceived the original idea for this commentary. GTR and MRG analyzed and interpreted the lyrics data and co-wrote the manuscript. Both authors read and approved the final manuscript.

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GTR is a medical immunologist, studying the interaction between *S. pneumoniae* and the immune system and is an editorial board member of Pneumonia. In his private life he is a big fan of classic blues and rock music. MRG is a bachelor student of the premedical program at UCR, Middelburg.

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The authors declare that they have no competing interests.

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Inner Ear Function in Patients with Obstructive Sleep Apnea

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Abstract

Objective: Because of their high metabolic activity and low-resting oxygen tension, the organs of the inner ear are vulnerable to hypoxia, a condition that occurs repetitively in obstructive sleep apnea-hypopnea syndrome (OSAHS). The present study aimed to investigate the inner ear function of patients with OSAHS.

Methods: A total of 58 patients with OSAHS (116 ears) and 20 adults without OSAHS were enrolled in the present study. The clinical features, such as air-conduction thresholds, auditory brainstem response (ABR, 11 times/s and 51 times/s stimulation rates), and distorted products otoacoustic emission (DPOAE), were evaluated and compared between these two groups.

Results: Air-conduction thresholds at 4 kHz and 8 kHz were higher in patients with OSAHS compared with controls ($P < 0.001$). At the rate of 11 times per second, biauricular wave I latencies and wave V latencies in the OSAHS group were longer than those in the control group (1.51 ± 0.13 vs. 1.33 ± 0.07 ms, $P < 0.001$; 5.65 ± 0.23 vs. 5.53 ± 0.23 ms, $P = 0.0016$). At the rate of 51 times per second, biauricular wave I latencies and wave V latencies in the OSAHS group were longer than those in the control group (1.64 ± 0.12 vs. 1.44 ± 0.06 ms, $P = 0.0001$; 5.92 ± 0.26 vs. 5.80 ± 0.18 ms, $P = 0.0077$). However, there was no significant difference in the wave I and wave V interval between these two groups ($P = 0.10$). DPOAE amplitude was significantly reduced in OSAHS patients, although no hearing loss was observed.

Conclusion: High-frequency hearing loss was detected in adults with severe OSAHS, and wave I latencies and wave V latencies of ABR were prolonged.

Keywords: OSAHS, ABR, DPOAE, Inner ear

Introduction

Deafness is a major problem that affects quality of life. In recent years, clinical surveys have indicated that the clinical prevalence of obvious hearing loss is 7–10%. [1–3]. Hence, there is an urgent need to prevent deafness. Ischemic hypoxia is an important factor of sudden hearing loss. The cochlea is an organ that requires a large amount of energy but the PO_2 level near the cochlear organ of Corti is among the lowest throughout the body. Therefore, when blood PO_2 is reduced or blood transport to the cochlea is insufficient, cochlear function may be severely damaged [4].

Studies using animal models have reported that after acute ischemic hypoxia, animals exhibit a prolonged latency of auditory brainstem response (ABR) as well as a reduction in amplitude and a prolonged phase of distorted products otoacoustic emission (DPOAE). However, at present, there is a paucity of data on the effects of inner ear function in people with OSAHS. The present study aimed to investigate the inner ear function of patients with OSAHS by pure tone audiometry, ABR, and otoacoustic emission (OAE).

Methods

A convenience sample of fifty-eight patients with OSAHS (116 ears) was recruited from the Sleep Monitoring Center, Department of Otolaryngology Head and Neck Surgery, Changgung Hospital from January 2016 to February 2017, while

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20 subjects without OSAHS were recruited from the Health Examination Center in Changgung Hospital as healthy controls. Study subjects (patients with OSAHS and controls) with profound hearing loss, tinnitus, vertigo, history of ear surgery, and family genetic history of deafness were excluded from participation.

Test Methods

All patients underwent the examinations to ensure the patency of the external auditory canal, the integrity of the tympanic membrane, and the absence of ear cerumen and secretions before electronic otoscopy. Pure tone audiometry was performed using an Astera pure tone meter, and air and bone conduction thresholds at 125, 500, 1000, 2000, 4000, and 8000 Hz were recorded. The auditory-evoked potential system was also applied for the ABR test. The amplifier-bandpass filter was set at 100–3000 Hz, with 100 k gain and 1024 times of stacking. An ER-3A plug-in air guide headphone was used as the transducer. A short click was employed using the maximum output of 95 dB nHL. Each patient was tested at 11/s and 51/s. The latencies of wave I, III, V, and I-III and I-V were recorded twice, and there were two stimulus rates: 11 times/s and 51 times/s. The test environment was in line with GB/T16403 (1996) standards. A Capella OAE analyzer was used to detect binaural OAEs at 750 Hz, 1 kHz, 2 kHz, 4 kHz, 6 kHz, and 8 kHz. The ICS auditory-evoked potential system was used to place the electrodes on the skin. The recording electrodes were placed at the middle of the forehead, while the reference electrode was placed in the bilateral earlobe or mastoid. The ground electrode was placed at the nasal root, and the inter-pole impedance was ≤ 3 kΩ. The background noise was < 24 dB (A).

Polysomnography

Polysomnography (PSG) was performed on all patients in the OSAHS group and control group by monitoring nighttime sleep for 7 h or more using the Philips Wei Kang A6 (USA) or Kangdi lead multi-function sleep-monitoring system (Australia).

PSG recordings were collected and analyzed offline (Grael HD PSG system, Compumedics, Victoria, Australia) during light-off to light-on. The overnight standard PSG protocol included synchronous measurement of electroencephalogram (EEG) with scalp electrodes in the standard 10–20 configuration, electrooculogram (EOG), genioglossus muscle electromyogram (EMG) using an intra-oral surface electrode, electrocardiogram (ECG), nasal flow pressure (thermistor and nasal pressure cannula), respiratory (thoracic and abdominal) movements, oxy-hemoglobin saturation (pulse oximeter), mandibular and tibialis anterior EMGs, and body position.

Apnea was defined as an airflow reduction of 90% or more for at least 10 s. Hypopnea was defined as the peak signal drop by ≥ 30% of pre-event baseline for at least 10 s associated with either oxygen desaturation of at least 3% or arousal. Apnea-hypopnea index (AHI) was calculated by dividing the total number of apneas and hypopneas by the number of hours of sleep. The oxygen desaturation event index (ODI) was defined as the number of events per hour in which oxygen saturation decreased by 3% or more. "The evaluation of sleep stages and events was based on guidelines of the American Academy of Sleep Medicine (AASM)."

Statistical Analysis

We used the software program SAS 9.2 to conduct the statistical analysis. All results were expressed as mean ± SD. For two group comparisons, each value was compared by *t* test when data conformed to normal distribution, while the non-normally distributed continuous data were compared using non-parametric tests. A value of $P < 0.05$ was considered statistically significant.

Results

Basic characteristics of patients

The 58 patients included 46 men (79%). Patient age ranged from 20 to 65 years (40.4 ± 11.3 years) and body mass index (BMI) ranged from 22.8 to 42.6 kg/m^2 (23.5 ± 9.4 kg/m^2). Of the 58 patients, 40 (69%) had normal BMI (< 25 mg/K^2), and 18 (31%) were obese (> 30 mg/K^2). Patients with OSAHS lasting for a disease course of 8 to10 years accounted for 41% of patients. Patients with an apnea-hypopnea index (AHI) > 30 accounted for 67%, while patients with decreased blood oxygen saturation accounted for 57%. The AHI grading was moderate in 17 patients (29%) and severe in 41 patients (71%), but this was normal in two patients (3%), mild in 15 patients (26%), moderate in 33 patients (57%), and severe in eight patients (14%). The course of snoring was 0–3 years in 11 patients (19%), 3–5 years in 11 patients (19%), 5–8 years in six patients (10%), 8–10 years in 24 patients(41%), and over 10 years in six patients (10%).

Table 1 shows that air-conduction thresholds at 500 Hz, 1 kHz, and 2 kHz frequencies between the two groups were not significantly different ($P = 0.38$). Furthermore, the high-frequency (4 kHz and 8 kHz) air-conduction thresholds were higher in the OSAHS group than in the control group (4 kHz, $t = 3.664$, $P < 0.001$; 8 kHz, $t = 3.875$, $P < 0.001$).

The average air-conduction thresholds at the four frequencies of 500 Hz, 1 kHz, 2 kHz, and 4 kHz were not significantly

different between the two groups. The average air-conduction thresholds of 116 OSAHS group were 37 ± 12.00 ($\bar{x}$ ± s). The average air-conduction thresholds of 40 control group were 15. 14 ± 7.74, $t = 1.347$, $P = 0.18$.

Table 2 shows the OAE results at various frequencies. There was a significant difference in the rate of abnormal OAEs between the two groups ($P < 0.001$).

As shown in Table 3, at 11 times/s and 51 times/s stimulation rates, the binaural wave I and wave V latencies of these patients were longer, when compared with those of the controls ($P < 0.05$), while there was no significant difference in the other wave latencies between these two groups ($P > 0.05$). Furthermore, there was also no significant difference in the wave I and wave V interval between these two groups ($P > 0.05$).

As shown in Table 4, there were 32 patients who underwent uvulopalatophar-yngoplasty surgery (UPPP) and were reexamined for ABR 6 months after surgery. Of these patients, 16 (32) ears selected to compare their PL and IPL before and after UPPP at high and low stimulation rates. Results showed that there was no significant difference in PL and IPL before and after UPPP at both 11 times/s and 51 times/s ($P = 0.58$).

There was no significant correlation between ABR wave latencies and AHI and the minimum oxygen saturation under different stimulation rates, as revealed by Spearman rank correlation. Besides, DPOAE amplitude was significantly reduced in OSAHS patients, although no hearing loss was observed.

Table 1: Comparison of pure tone thresholds between the two groups ($\bar{x} \pm s$).

Group	Case (*n*)	500 Hz	1 kHz	2 kHz	4 kHz	8 kHz
OSAHS group	116	13.88 ± 8.16	15.86 ± 10.84	16.42 ± 13.70	23.32 ± 20.11	23.99 ± 22.10
Control group	40	15.09 ± 7.71	15.50 ± 8.84	15.89 ± 10.25	16.24 ± 8.12	16.49 ± 13.12
t		0.875	0.454	0.725	3.664	3.875
P		0.38	0.65	0.47	<0.01	<0.01

Table 2: Otoacoustic emission under different stimulation frequencies (*n*[%]).

Frequency	Abnormal rates OSAS group	Abnormal rates control group	χ^2	*P*
750 Hz	82/116(71%)	0/40	72.1838	<0.01
1 kHz	54/116(47%)	0/40		
2 kHz	30/116(26%)	0/40		
4 kHz	38/116(33%)	0/40		
6 kHz	33/116(28%)	0/40		
8 kHz	40/116(34%)	0/40		

Abnormal rates of OSASH group: the abnormal number in 116 patients
Abnormal rates of control group: no abnormal number in 40 healthy control group

Table 3: Binaural PL, IPL, and Δ IPL under different stimulation frequencies between two groups($\bar{x} \pm s$, ms).

Group	*n*	11/s			51/s			Different stimulation frequency ΔI~V
		I	V	I~V	I	V	I~V	
Control	40	1.33 ± 0.07	5.51 ± 0.23	4.18 ± 0.21	1.44 ± 0.06	5.80 ± 0.18	4.36 ± 0.17	0.18 ± 0.09
OSAHS	116	1.51 ± 0.13	5.65 ± 0.24	4.15 ± 0.21	1.64 ± 0.12	5.92 ± 0.26	4.30 ± 0.21	0.25 ± 0.61
t		8.338	3.215	0.779	10.099	2.702	1.631	0.722
P		<0.01	<0.01**	0.44	<0.01**	0.01**	0.10	0.47

$P < 0.05$; **$P < 0.01$
PL, peak latency: the time from the beginning of sound feeding to the recording of a group of action potential wave peaks was given a certain sound intensity stimulus
IPL, interpeak latency: the time difference between two peaks in a set of action potentials
ΔIPL: the difference between the 51/s IPL and 11/s IPL

Table 4: PL, IPL, and ΔIPLPL of patients before and after treatment at different stimulation rates ($\bar{x} \pm s$, ms).

Group	*n*	11/s			51/s			Different stimulation rates ΔI~V
		I	V	I~V	I	V	I~V	
Before treatment	32	1.52 ± 0.14	5.66 ± 0.32	4.16 ± 0.21	1.64 ± 0.12	5.90 ± 0.27	4.25 ± 0.23	0.12 ± 0.08
After treatment	32	1.55 ± 0.12	5.71 ± 0.25	4.19 ± 0.25	1.66 ± 0.11	5.93 ± 0.24	4.27 ± 0.19	0.11 ± 0.07
t		1.259	1.075	0.669	0.636	0.850	0.512	−0.555
P		0.22	0.29	0.51	0.53	0.40	0.61	0.58

PL, peak latancy of ABR
IPL, interpeak latency from I–V of ABR

Discussion

The present study revealed that air-conduction thresholds of patients with moderate and severe OSAHS are similar to those of healthy controls. However, there was a significantly higher rate of abnormality in OAE in patients with OSAHS. Wave I and wave V latencies at high and low stimulation rates of OSAHS patients were higher, when compared with controls, indicating that an oxygen-rich blood supply to the cochlea was compromised. Though the oxygen supply in patients with OSAS was probably compromised compared with control subjects, a substantial impact on cochlear function was not found.

Auditory brainstem responses reflect the bioelectrical potential arising from the auditory nerve and brainstem in response to acoustic short-duration sounds, such as a click [5–8]. It typically consists of a series of peaks within the first 10 ms after the onset of a stimulus. These peaks are labeled with Roman numerals I–VII, in which wave I, III, and V are more stable and distinct, while wave V is the most stable component. Therefore, the latency and amplitude of wave V, the interpeak latencies among wave I, III, and V, and the amplitude ratio of wave I and wave V are often used to localize the lesions of auditory nerve pathways [9].

OAE is a kind of sound energy produced in the cochlea, which can be recorded in the external auditory canal. It can also reflect the functional status of outer hair cells in the cochlea. According to the presence of external stimulus signal induction and the source of acoustic stimulation induction, OAE can be categorized as spontaneous OAE (SOAE) and evoked OAE (EOAE). SOAE can be obtained from test ears under silent stimulation. EOAE can be induced through a certain acoustic stimulation on the test ears, and the most common OAEs are transient evoked OAE (TEOAE) and distorted products OAE (DPOAE). DPOAE is common in clinical practice, which is a kind of acoustic energy produced by cochlear outer hair cells induced by two pure tones (F1 and F2), and recorded in the external auditory canal. Matsumura E *et al* [10, 11] showed that the inner ear damage caused by severe OSAHS was manifested as the decrease of DPOAE amplitude. The present study revealed that DPOAE amplitude was significantly reduced in OSAHS patients, although no hearing loss was observed. The possible reason for the phenomenon could be that chronic hypoxia of the cochlea in patients with OSAHS may damage the outer hair cells in the cochlea, thereby affecting cochlear function [5–7]. It has also been observed that the change in DPOAE amplitude was earlier than that of hearing thresholds [12]. Thus, DPOAE may be used to monitor the cochlear function of OSAHS patients [8].

In conclusion, high-frequency hearing loss was detected in adults with severe OSAHS, and the wave I latencies and wave V latencies of ABR were prolonged. Furthermore, the changes in DPOAE amplitude occurred earlier, when compared with those for the hearing thresholds.

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Compliance with ethical standards

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Research involving human participants and/or animals Yes

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Diagnostic Significance of Vestibular Evoked Myogenic Potentials for Different Types of Vertigo

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Abstract

Objectives: To study the parameters of vestibular evoked myogenic potentials in patients with various types of vertigo.
Materials and methods: A total 77 patients (35 men, 42 women) aged 43.7 ± 12.5 years with vestibular vertigo and postural instability were studied. Surface electrode myography was used to record the activity of the sternocleidomastoid muscle arising in response to sound stimulation of the ipsilateral ear.
Results and discussion: As compared with controls, patients with signs of impairment to the central compartment of the vestibular analyzer showed a tendency to an increase in the latent period of the N2 wave and a significant reduction in the amplitude of the P1–N2 complex on both sides ($p < 0.005$), without any significant change in the coefficient of asymmetry. Patients with signs of damage to the peripheral compartment of the vestibular analyzer showed significant increases in the latent period of P1 as compared with healthy subjects ($p < 0.005$). In addition, there was a statistically significant ($p = 0.0007$) increase in the coefficient of asymmetry of latent period P1. Patients with postural instability showed no significant differences in P2 and N2 latencies as compared with controls.
Conclusions: The study results showed that patients with vertigo of both peripheral and central genesis have impairments to the formation of the vestibulospinal reflex at different levels of the vestibular analyzer.

Keywords: Vestibular evoked myogenic potentials, Central vertigo, Peripheral vertigo, Instability

Vertigo is one of the most widespread symptoms in neurological and general medical practice and arises as a result of damage to the vestibular system, at any level [1–5]. The cause is generally damage to the peripheral compartment of the vestibular analyzer – the labyrinth of the inner ear and the vestibular nerve. Pathology is accompanied by the development of nystagmus and impairments to balance and autonomic reactions. Otoneurological investigations allow signs of vestibular dysfunction to be detected in more than 80% of patients suffering from sensorineural deafness without complaints of loss of balance or vertigo [6–8]. Contemporary instrumented methods of investigating the vestibular analyzer allow the level and possible causes to be identified. Results from classical heat and rotation tests in many patients complaining of vestibular disorders are found to be within the normal range [9–11]. The search for effective methods for instrumented investigation of the state of the vestibular system is very relevant.

Investigations of vestibular evoked myogenic potentials (VEMP) are used to study the vestibulospinal reflex and provide an objective method for assessing the state of the vestibular nerve, which is inaccessible to investigation by other instrumented methods. The method is based on the unique ability of part of the vestibule of the labyrinth to perceive sound stimuli and, by means of a complex reflex arc, to elicit involuntary contractions of the sternocleidomastoid muscle [12–17]. The VEMP reflex arc is multineuronal, involves a large number of synaptic connections, and includes the sacculus, the vestibular nerve, right vestibular nucleus, the medial vestibulospinal tract, the accessory nucleus, the XI cranial nerve, and the sternocleidomastoid muscle. Changes in VEMP can be caused by impairment to any part of this pathway. An obligatory condition for forming VEMP is unhindered conduction of sound through the middle ear to the inner ear.

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Data from various authors indicate that in diseases involving damage to the peripheral compartment of the vestibular analyzer, changes to VEMP are apparent as decreases in the amplitude of the P1–N2 complex to the level of its complete disappearance, while diseases of the central compartment are linked with increases in the latent periods (LP) of the P1 and N2 complexes [12, 18–20]. Investigations of VEMP provide an objective method for assessing the state of the sacculus and vestibular nerve, including parts inaccessible to investigation by other clinical methods [21].

The aim of the present work was to study VEMP parameters in patients with different types of vertigo.

Materials and Methods. The study included 77 patients (35 men and 42 women) aged 43.7 ± 12.5 years, who were divided into three groups. The differential diagnosis of peripheral and central vertigo was made using the ALGORITM neurovestibular testing system developed by ourselves [22]. Group 1 consisted of 40 patients with clinical impairments to the peripheral compartment of the vestibular analyzer, group 2 consisted of 21 patients with impairments to the central compartments of the vestibular analyzer, and group 3 consisted of 16 patients with postural instability (Table 1). The control group included 15 healthy subjects of comparable gender and age as patients of the study groups, i.e., 10 men and five women (mean age 38.6 ± 8.5 years) with no signs of vestibular disorders, hearing impairments, or complaints of vertigo.

Surface electromyography was used to record the activity of the sternocleidomastoid muscle arising in response to ipsilateral sound stimulation of the ear with tonal bursts at 500 Hz, duration 0.1 msec, intensity 100 dB. The active electrode was placed on the upper part of the sternocleido-mastoid muscle and the reference electrode was placed on the muscle at the lateral margin of the upper part of the sternum; the ground electrode was placed on the lip. Averaging of muscle responses formed VEMP. Studies were performed using a Viking Quest multifunctional 40-channel system for clinical neurophysiological studies (Nicolet Biomedical, USA). The investigation protocol included evaluation of the LP of P1 and N2, the amplitude of the P1–N2 peaks, and the percentage relationship between vestibular asymmetry (VA) of the P1–N2 peak amplitudes and P1 latency, which were calculated as:

$$\text{Percent ratio of VA by amplitude} = 100\% \cdot (\text{Amp}[\text{P1–N2 left}] - \text{Amp}[\text{P1–N2 right}]) / ([\text{Amp}[\text{P1–N2 left}] + \text{Amp}[\text{P1–N2 right}]);$$

$$\text{Percent ratio of VA by P1 latency (\% asymmetry of vestibulospinal conduction)} = 100\% \cdot (\text{Lat}[\text{P1 left}] - \text{Lat}[\text{P1 right}]) / ([\text{Lat}[\text{P1 left}] + \text{Lat}[\text{P1 right}]),$$

where Amp is peak amplitude and Lat is latency.

Results were analyzed statistically using Statistica version 12.0. Mathematical statistics methods were used: calculation of numerical characteristics of random values (mean, median, standard deviation, significant interval), methods for confirming statistical hypotheses and assessing relationships identifying significant differences between mean values. The threshold significance level for statistical hypotheses was taken as 0.05. Data for representative sets consisted of mean (*M)* and standard deviation (σ). The distributions of all values for one or more groups differed from the normal law, as evidenced by the Shapiro–Wilk test. Quantitative signs not following the normal distribution

Table 1. Distribution of patients by types of vestibular disorder.

Diagnosis	Group 1 (*n* = 40)	Group 2 (*n* = 21)	Group 3 (*n* = 16)
Benign paroxysmal positional vertigo	28	–	–
Ménière's disease	10	–	–
Vestibular neuronitis	2	–	–
Vestibular migraine	–	16	
TIA in the vertebrobasilar system	–	3	
Vestibular Schwannoma	–	1	–
Cerebellar neoplasm	–	1	–
Diabetic polyneuropathy	–	–	4
Generalized anxiety disorder	–	–	7
Parkinson's disease		–	2
Phobic postural instability after acute otolaryngological diseases	–	–	3

law were described by the median (Me) and quartiles [Q1; Q3]. Groups were compared using the Kruskal–Wallis test.

Results. Parametric distribution analysis demonstrated the absence of any statistically significant differences in VEMP in patients with different types of balance impairments (Table 2). Nonetheless, particular trends to changes in VEMP parameters were found in patients with different types of vertigo and healthy subjects. Thus, group 1 showed a significant increase in the LP of P1 compared with controls ($p < 0.005$). In addition, there was a statistically signif- icant ($p = 0.0007$) increase in the coefficient of asymmetry of the LP of P1, pointing to slowing of vestibulospinal conduction on one side; this was objective evidence of unilateral impairment to the functioning of the peripheral compartment of the vestibular analyzer (see Table 2).

Analysis of VEMP in patients of group 2 identified a tendency to increases in the LP of N2 as compared with controls, as well as a significant reduction in P1–N2 amplitude on both sides ($p < 0.005$) without any significant changes in the coefficient of

Table 2. Measures of VEMP in patients of the study and control groups (Me [Q1; Q3]).

Measure	Group 1 (*n* = 40)	Group 2 (n = 21)	Group 3 (*n* = 16)	*p**	Control group (*n* = 15)	Between-group comparisons, *p***
LP, msec						
P1 left	17.5 [15.6; 20.4]	15.6 [15.1; 18.6]	16.3 [15.0; 22.2]	0.32	15.1 [14.2; 15.8]	Group 1 – control, $p = 0.0008$
						Group 2 – control, $p = 0.045$
						Group 3 – control, $p = 0.034$
N2 left	26.3 [23.0; 31.1]	26.9 [24.3; 32.1]	24.7 [23.3; 32.2]	0.68	25.6 [22.6; 26.4]	Group 1 – control, $p = 0.095$
						Group 2 – control, $p = 0.022$
						Group 3 – control, $p = 0.13$
P1 right	17.1 [15.4; 22.1]	17.2 [16.4; 19.3]	16.3 [15.8; 18.8]	0.66	15.2 [14.2; 15.7]	Group 1 – control, $p = 0.006$
						Group 2 – control, $p = 0.023$
						Group 3 – control, $p = 0.073$
N2 right	26.1 [23.8; 31.0]	30.7 [26.2; 32.0]	28.0 [22.7; 33.1]	0.24	25.1 [23.4; 27.9]	Group 1 – control, $p = 0.057$
						Group 2 – control, $p = 0.020$
						Group 3 – control, $p = 0.085$
Amplitude of P1–N2 peaks, μV						
left	79.5 [33.9; 113.5]	83.9 [55.5; 118.0]	47.3 [34.9; 80.3]	0.32	120.0 [85.0; 167.5]	Group 1 – control, $p = 0.055$
						Group 2 – control, $p = 0.062$
						Group 3 – control, $p = 0.001$
right	71.0 [46.0; 112.0]	55.0 [44.3; 89.1]	64.7 [32.9; 96.8]	0.62	132.0 [100.1; 164.4]	Group 1 – control, $p = 0.11$
						Group 2 – control, $p = 0.036$
						Group 3 – control, $p = 0.025$
Coeffi cient of asymmetry for						
peak amplitudes	24.0 [5.4; 44.1]	10.6 [3.5; 30.1]	11.3 [6.5; 26.3]	0.21	7.9 [4.2; 13.9]	Group 1 – control, $p = 0.032$
						Group 2 – control, $p = 0.28$
						Group 3 – control, $p = 0.38$
LP, msec	11.3 [4.6; 17.1]	5.9 [4.5; 9.6]	11.2 [4.3; 19.0]	0.60	2.9 [2.0; 5.3]	Group 1 – control, $p = 0.0007$
						Group 2 – control, $p = 0.026$
						Group 3 – control, $p = 0.019$

Data are presented at medians and lower with upper quartiles; **p* – significance of differences, Kruskal–Wallis test, rank analysis of variance between the three study groups; ***p* – significance of differences compared with control group.

asymmetry (see Table 2). In our view and as indicated by published data [14, 23, 24], this is highly likely to be evidence of bilateral impairments to the function of the central compartment of the vestibular analyzer.

Analysis of VEMP in patients of group 3 did not identify any significant differences in P1 and N2 latency as compared with controls (see Table 2). However, none of the three groups showed any significant reduction in the amplitude of the VEMP response below the normal P1–N2 amplitude ($p < 0.005$), which was less than 70 μV for young people. This probably indicates elevated noise sensitivity of the peripheral compartment of the vestibular analyzer in patients with both vestibular vertigo of peripheral and central origin and postural instability.

Discussion. The diagnostic potentials of the method based on recording VEMP has been studied since 1964, though clear indications for its use remain to be defined and no single protocol for running investigations has yet been approved and developed [21, 25–27]. The number of publications on the use of VEMP in different diseases of the vestibular system has increased rapidly in recent years. Decreases in VEMP amplitude on the side of the impaired ear have been shown to be a characteristic feature of impairment to the peripheral compartment of the vestibular analyzer [28, 29], and is seen particularly in Ménière's disease. There are reports addressing studies of VEMP in CNS lesions. Thus, decreases in the amplitude of potentials and increases in the LP of P1–N1 have been demonstrated in patients with progressive supranuclear palsy, multiple sclerosis, and brainstem stroke [12, 23, 30–33]. Changes in VEMP parameters are detected in patients with migraine.

The results of the present study show that patients with vertigo of both peripheral and central origin have increases in the LP of P1, which is evidence of possible impairment to the formation of the vestibulospinal reflex and may be due to damage to the inner ear, vestibular nerve, and brainstem at different levels. In our view, the measure of the asymmetry of VEMP amplitude has important diagnostic value, particularly when it is elevated by factors of two or more or decreased to less than 70 μV in young patients. The former situation is significant evidence of dehiscence (fissure) of the superior semicircular canal or can reflect hyperexcitability of the hair cells of the maze, while the latter situation is due to trauma to the vestibular system or demyelination due to use of gentamicin.

Thus, investigation of VEMP is a potential method for the diagnosis of vestibular system pathology and requires further study and determination of normal values for the parameters recorded.

The authors have no conflicts of interests.

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Sudden Deafness without Vertigo as a sole Manifestation of AICA Infarct

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Introduction

Sudden sensorineural hearing loss is a striking clinical condition characterised by a sudden onset of unilateral or bilateral loss of hearing, frequently associated with tinnitus, a sensation of pressure in the ear and disequilibrium or vertigo. Even if numerous aetiologies have been postulated as an explanation for this syndrome, viral and vascular causes have been considered the most important.

Case report

Eight days after an uncomplicated operation of abdominal surgery a 55-year-old hypertensive man developed transient light-headedness, followed after some minutes by tinnitus with deafness on the left-side. On admission, he complained of a subjective total loss of hearing on the left-side. The remainder of the neurological examination proved normal, including the absence of nystagmus (also absent after use of Frenzel glasses) and ataxia.

Hearing impairment was severe and shown to be localized to the left cochlea from the findings of pure tone audiometry (PTA: 80 dB of hearing loss), speech discrimination testing (roll-over: 30%), stapedial reflex testing (positive Metz-test on the left side) and ABR (typical cochlear intensity-latency). Cold caloric responses were normal bilaterally, no nystagmus was observed after head shaking test.

Magnetic resonance revealed high intensity areas on T2-wighted images, within the left medial cerebellar peduncle and left cerebellum – the lateral pons was not involved (Fig. 1). These lesions were compatible with a partial AICA (anterior inferior cerebellar artery) infarct. Transthoracic echocardiography, electrocardiography and Doppler sonography of neck arteries were normal. After eight months pure tone audiometry showed no improvement of hearing loss.

Discussion

Sudden sensorineural deafness is often considered an idiopathic disease because no aetiology can be given in the majority of cases. Even though it has been defined that sudden deafness occurs within 72 hours, most patients suffer acute hearing-loss within minutes: hence a vascular mechanism should be taken into consideration, at least in such cases. In recent years sudden deafness has frequently been described in the anterior inferior cerebellar artery and as presenting symptoms of, or being associated with, other brainstem or cerebellar signs.

Vertigo is the clinical manifestation most frequently associated with this because of contemporary damage to the vestibular labyrinth. Adams was the first to describe the syndrome as being associated with an AICA occlusion. His patient had vertigo, tinnitus and bilateral hearing- loss as early symptoms. An acute ischaemic stroke in the distribution of the anterior inferior cerebellar artery (AICA) is associated with facial weakness, hypalgesia, ataxia, vertigo, hearing loss and nystagmus [1]. However, it is only in recent reports that there has been a focus on the hearing-loss that occurs with AICA infarction. In our patient neurological symptoms and signs were absent, probably because of limited extension of infarction, but the ischaemic lesions revealed by MRI are sufficiently evident to demonstrate AICA territory involvement.

The internal auditory artery arises from the anterior cerebellar artery (AICA) and supplies the inner ear, usually dividing into two main branches – the common cochlea artery and the anterior vestibular artery. The auditory artery and its subdivisions are end arteries with minimal collateral branches from

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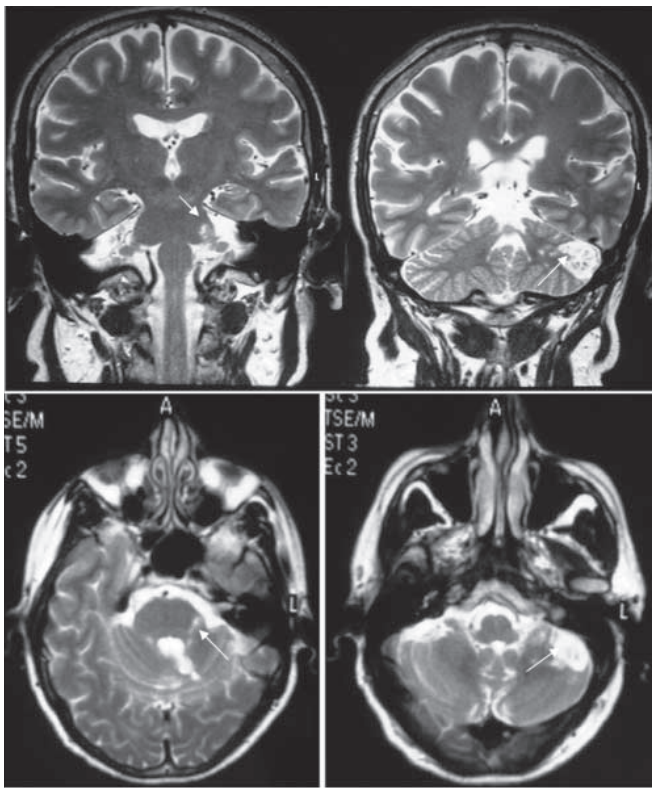

Fig. 1: Axial and coronal T2 MRI images showing left partial AICA infarction (*arrow*).

other major arterial branches [2]. We can therefore surmise that a posterior circulation event should be evident only in the cochlea or inner ear and either embolic or haemodynamic in nature, because it concerns a terminal territory. In this patient we considered a haemodynamic mechanism, probably related to transient orthostatic hypotension.

In the inner ear ischaemic lesions cannot be shown by current magnetic resonance techniques and so the diagnosis of cochlear infarct must be based on indirect indications of involvement from the posterior circulation territory [3]. Clinicians should consider performing MRI on all patients showing sudden deafness, although as sole manifestation, especially where there are vascular risk factors and a very acute onset of the condition exists. MRI is extremely useful in detecting small asymptomatic vascular lesions within AICA territory or alteration of the perfusion, which starting from the vertebral artery may have repercussions on the circulation of the inner ear.

Patients showing a sudden loss of hearing accompanied by vertigo have been described as showing these symptoms in association with an AICA infarction [4].

In the patient here described sudden deafness was the only manifestation of an ischaemic lesion that involved AICA territory.

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Notes