Cervical and ocular vestibular evoked myogenic potentials in epileptic patients

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Abstract
Background: Vertigo and dizziness are very common complaints that may be related to epilepsy. The purpose of this study was to assess vestibulo-spinal and linear vestibulo-ocular function in epileptic patients in the inter ictal period. The current observational study was carried out in audio-vestibular unit Menoufia University. Subjects in the current study were divided into two groups: The control group included 30 normal individuals not complaining from any dizzy symptoms and the epileptic cases group included 30 epileptic patients. All subjects in the study were submitted to cervical and ocular vestibular evoked myogenic potential.

Results: There was no significant difference between the control and epileptic group regarding the age and sex distribution. Sixty-seven percent of epileptic cases had dizzy symptoms. There was statistically significant difference in the latency and amplitude of c and o VEMP between the control and the epileptic group, 39/60 ears (65%) in the study group had cVEMP abnormalities, 32/60 ears (53%) had oVEMP abnormalities. Abnormal c and o VEMP were reported in 28/60 ears (46.7%). There was statistically significant relationship between VEMP abnormalities and duration of seizures, frequency of epileptic attacks, and type of therapy.

Conclusion: Vestibular abnormalities were frequently reported in epileptic patients in the current study which may be related to the severity and control of epilepsy.

Keywords: Cervical, Dizziness, Epilepsy, Ocular VEMP

Background
Epilepsy is a brain dysfunction characterized by recurrent disturbances in the normal electrical activity of the brain that results in seizures [1]. Vertigo and dizziness are very common complaints that occur either due to peripheral vestibular or central nervous causes. Among the latter, epilepsy should be taken into consideration [2]. Vestibular symptoms are common accompanying symptoms in seizure but it is rare to be the sole symptoms [3]. If vestibular symptoms occurred predominantly in seizure, this condition is called vestibular epilepsy, vestibulogenic seizure epileptic vertigo, and vertiginous epilepsy [4].

Vestibular evoked myogenic potentials (VEMP) is a short latency electromyographic response to sound stimuli that is used to assess ipsilateral sac- cular and inferior vestibular nerve functions (cervi- cal: c VEMP), as well as contralateral utricular and superior vestibular nerve functions (ocular: o VEMP) [5].

VEMP also reflect functions of the central otolithic pathways and can be applied for the diagnosis of central nervous system disorders [6].

Many studies previously assessed the semicircular canals (SCCs) and vestibulo ocular function in these patients, but to our knowledge no studies assessed vestibulo spinal and otolith function in those patients. So, this study aimed at assessment of vestibulo spinal and linear vestibule-ocular functions in epileptic patients using c and o VEMP in the inter ictal period.

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Methods
The current observational study was carried out in audio-vestibular unit (tertiary referral center), ENT department from April 2019 to May 2020.

Ethical approval from the hospital committee (according to Helsinki Declaration) and written consent from all the patients were obtained. Epileptic patients were recruited from outpatient neurology clinic.

Subjects in the current study were divided into two groups:

Control group
Included 30 normal individuals not complaining from any dizzy symptoms with age range between 20 and 40 years.

Cases group
Included 30 subjects with confirmed diagnosis of epilepsy through neurology specialist according to the criteria of international league against epilepsy (7), with age range between 20 and 40 years with exclusion of patients suffering from other neurological disorders, systemic diseases, history of otovestibular disorder, e.g., Meniere’s disease, conductive hearing loss, and cervical spine problems.

Equipment
The following equipment was used; two channel audiometer model (Madsen, orbiter 922), middle ear analyzer (Interacoustic AT235), and vestibular evoked myogenic potential (Eclipse/EP25 system, Interacoustics Inc., Middlefart, Denmark).

Methods
All subjects in the study group were submitted to full history taking (duration and frequency of epileptic attack, treatment, and audiovestibular symptoms), neurological examination, otoscopic, and basic audiological evaluation in the form of pure tone audiometry in the frequency range of 250 to 8000 HZ for air conduction and 500-4000 Hz for bone conduction, speech reception threshold (SRT), word discrimination score (WD) [7], and immittance to exclude conductive hearing loss before VEMP testing.

VEMP
Electrode montage
The skin was cleaned carefully to ensure impedance less than 5kohm. For cVEMP, active surface electrode was placed on the upper half of sternocleidomastoid muscle (SCM) of the stimulated side, the reference electrodes were placed over mastoid bone ipsilateral to stimulation side. For oVEMP, active electrodes were placed on the face just inferior to eye contralateral to the stimulated side, reference electrodes were placed 2-3 cm below active electrode, and the ground electrode was placed on the forehead for c and o VEMP.

Instruction to the patient
For cVEMP, the subjects were asked to turn their head to the opposite side of recording with slight head flexion approximately 30 degrees to contract the SCM. For oVEMP, the subject was asked to look upward at distant target in the midline and the patient eye position was kept at a vertical visual angle of approximately 30-35° above horizontal.

Stimulus and recording parameters
Two hundred sweeps of 500 Hz tone burst (2:1:2 cycle) were delivered to the subjects via insert earphones at a rate of 5.1 Hz and intensity of 95 dBNHL. The filter was adjusted to be 10-750 Hz at a time window: -20 to 80 ms.

Wave analysis
The equipment system was monitoring the electromyography (EMG) levels throughout the testing. EMG scaling was done to compensate for the uneven contraction of SCM that may occur in both sides to allow for more accurate comparison between the individual right and left VEMP response. For all the recorded traces (in c and o VEMPs), the positive and negative peaks were identified according to their latencies followed by measuring of base to peak rectified amplitude of each wave. At least two traces were taken to ensure wave reproducibility.

Interaural amplitude ratio was also calculated for each type of VEMPs recording. The AR (asymmetry ratio) value was calculated as follows: (Amplitude of unaffected side – Amplitude of affected side)/(Amplitude of unaffected side + Amplitude of affected side) x 100, ratio more than 34% was considered pathological [8].

Statistical analysis
Results were analyzed using SPSS statistical package version 20. Two types of statistics were done: descriptive statistics, e.g., number (no.), percent (%) for qualitative data, mean (x), and standard deviation (SD) for quantitative data. Analytic statistics, comparison between quantitative data in two groups was done using Student’s t test (parametric test) and Mann-Whitney test (non-parametric test). Chi-squared test ($\chi^2$) was used to study association between two qualitative variables. P value of <0.05 was considered statistically significant.

Results
There was no significant difference between the control and study groups regarding the age and sex distribution (Table 1).
Audiovestibular symptoms

Vestibular symptoms were evident in 20/30 patients (67%), 11/30 (37%) of epileptic cases had imbalance (3 cases had sense of imbalance as an aura symptom and 8 cases had imbalance which was represented on starting administration of antiepileptic drugs). 9/30 (30%) of cases had vertigo that occurred spontaneously as aura symptoms in 4 patients and was related to starting administration antiepileptic therapy in 5 patients. Aural fullness was reported in 2 patients. The mean duration of seizures was 12.33±4.30 min. Frequency of attacks was as follows: one attack per week in 12/30 cases (40%), once per month in 11/30 cases (37%), and more than once per month in 7/30 cases (23%). 17/30 cases (57%) received only one drug and 13/30 cases (43%) received more than one drug. All subjects in the control and study groups had normal otoscopic examination, pure tone audiometry, speech discrimination, tympanometry and neurological assessment. VEMP Results: results of the amplitude or the latency beyond mean ± 2 SD of the normal values for the same age group in our laboratory were considered abnormal. All subjects in the control group had normal c and o VEMP results. There was statistically significant difference in the latency and amplitude of c and o VEMP between the control group and the study group (Table 2).

The current study showed that 39/60 ears (65%) in the study group had cVEMP abnormalities either prolonged latencies in 22/60 ears (37%), high amplitude in 28/60 ears (47%), low amplitude in 9/60 ears (15%), and amplitude asymmetry in 5/30 patient (17%). In oVEMP, 32/60 ears (53%) had abnormalities either prolonged latency in 19/60 ears (32%), high amplitude 14/60 ears (23%), low amplitude in 3/60 ears (5%), and amplitude asymmetry in 3/60 ears (10%). Abnormal c and o VEMP was reported in 28/60 ears (46.7%). All patients with vestibular symptoms had also abnormal VEMP results either ocular or cervical potentials. There was no statistically significant relationship between c and o VEMP abnormalities and age, gender (p > 0.05). Table 3 demonstrates relationship between abnormal VEMP results and duration of seizure, frequency of attacks, and type of therapy among the epileptic cases.

Discussion

Vestibular symptoms in epileptic patients may occur as an aura symptom or as a side effect of anti-epileptic drugs (AEDs) or may be related to a second comorbid disease (e.g., migranous vertigo) [9]. Focal intermittent epileptic discharge may cause episodic vestibular symptoms which are called epileptic vertigo [10]. Cortical stimulation of the vestibular cortex, e.g., superior lip of intraparietal sulcus may be the cause of epileptic vertigo.

Table 1 Demographic data of the control and study groups

| Demographic data | Control group (n=30) | Study group (n=30) | Test of significance | P value |
|------------------|----------------------|-------------------|---------------------|---------|
| Age (year)       | Mean ± SD 31.56±4.32 | 33.86±10.00       | t=1.16              | 0.15    |
| Sex no. (%)      | Male 14 (46.7)       | 10 (33.3)         | χ²=1.11             | 0.29    |
|                  | Female 16 (53.3)     | 20 (66.7)         |                     |         |

This table shows that there is no statistical significant difference between the studied groups regarding age and gender distribution

SD standard deviation, t student’s t test, χ² chi-square test

Table 2 The peak latencies and amplitudes of o and c VEMP

|                      | Control group (n=60 ears) mean ±SD | Study group (n=60 ears) mean ±SD | Test of significance* | P value |
|----------------------|------------------------------------|----------------------------------|----------------------|---------|
| cVEMP                |                                    |                                 |                      |         |
| Latency (ms) P1     | 13.83±1.50                         | 15.20±3.07                       | t=3.11               | 0.002*  |
| Latency (ms) N1     | 22.40±1.98                         | 24.36±3.94                       | t=3.44               | <0.001* |
| P1N1 amplitude (μv) | 15.57±2.63                         | 17.18±3.77                       | t=2.71               | 0.008*  |
| Amplitude ratio (n=30) | 18.86±5.68                        | 22.50±7.01                       | t=2.02               | 0.04*   |
| oVEMP                |                                    |                                 |                      |         |
| Latency (ms) N1     | 10.98±1.99                         | 14.64±3.20                       | t=7.52               | 0.003*  |
| Latency (ms) P1     | 15.78±3.91                         | 17.70±2.72                       | t=3.12               | 0.005*  |
| N1P1 amplitude (μv) | 10.52±3.65                         | 11.81±2.89                       | t=2.15               | 0.02*   |
| Amplitude ratio (n=30) | 16.86±4.38                        | 21.07±9.99                       | U=2.11               | 0.03*   |

This table shows that there is statistical significant difference between the studied groups regarding the recorded parameter in c and o VEMP

cVEMP cervical evoked myogenic potential, oVEMP ocular evoked myogenic potential, SD standard deviation, t student’s t test, U Mann-Whitney test

* = statistical significance
Vertigo which may occur as aura symptoms reflects central vestibular dysfunction due to epilepsy [11]. AEDs modulate the activity of cerebral neurons so dizziness may occur as side effect of these drugs [12]. Dizziness is one of the most frequently reported side effects of AEDs [13].

The vestibular system consists of SCCs and otolith organs (Utricle and Saccule) that respond to angular and linear velocity changes. The VOR is a reflex that generates eye movement matching the head movement; the angular VOR receives sensory input from the SCCs and compensate for rotation, the linear VOR receives sensory input from the otolith and compensate for translation. The vestibular spinal reflex (VSR) also receives otolith input, in response to linear motion changes [14]. The current study assessed the vestibulospinal function using cVEMP and linear VOR using oVEMP in epileptic patients. VEMP are useful tools in detecting central vestibular dysfunction when combined with oculomotor test of the VNG [15].

The auditory symptoms as hearing impairment and tinnitus may be reported as side effects of AEDs [16]. Hamed [17] reported that vestibular manifestations, including dizziness (62.22%) and sense of imbalance (44.44%) are frequent in patients with epilepsy, (24.44%) suffering from central vestibular dysfunction, 9 (20%) suffering from combined vestibular dysfunction and one (2.22%) had peripheral vestibular dysfunction. Gandelman-Marton et al. [18] also reported that dizziness and unsteadiness are common complaints in epilepsy patients. Also, epileptic patients may experience attacks of benign paroxysmal positional vertigo (BPPV) [19]. Fohner et al. [20] reported that long-term treatment with some antiepileptic drugs (AEDs) may cause dizziness, ataxia, imbalance, nystagmus, abnormalities in oculomotor functions, and their study revealed delayed conduction within auditory pathway (cochlea, auditory nerve, and brainstem) evidenced by abnormalities in the auditory brainstem response and nystagmography recordings indicating central and/or peripheral vestibular dysfunctions. In the current study, the vestibular symptoms (imbalance or vertigo) were reported in more than two-thirds of the epileptic cases, these symptoms may be related to the attack itself or starting the administration of AEDs.

VEMP responses are not only affected by peripheral vestibular end-organ dysfunction or vestibular nerve pathology but also can be affected by central neurological disorders [6]. The otolith organs are cortically represented in both hemispheres. The input from the SCCs and the otolith is converged at the vestibular nuclei within the brain stem and integrated through the vestibular cortical areas [16]. Lesions affecting the vestibular nuclei can cause abnormalities of both c and o VEMPs [16]. Abnormal ocular and cervical VEMP (which reflects vestibular affection in epilepsy) was reported in near than half of the epileptic patients in the current study.

### Table 3 Relationship between abnormal VEMP and duration of seizure, frequency of attacks, and type of therapy among the epileptic cases

|                          | Cervical VEMP | Ocular VEMP |
|--------------------------|--------------|------------|
|                          | Abnormal (n=39 ears) | Normal (n=21 ears) | Abnormal (n=32 ears) | Normal (n=28 ears) |
| Duration of seizure (minute) mean ±SD | 12.92 ± 3.53 | 11.13 ± 2.59 | 13.88 ± 2.71 | 11.73 ± 3.62 |
| P value                  | 0.04* (t=2.4) | 0.01* (t=2.62) |
| Frequency of attack      | n (%)        | n (%)      | n (%)        | n (%)        |
| Once/week                | 20 (51.30)   | 4 (19.00)  | 15 (46.90)   | 9 (32.10)    |
| Once/month               | 14 (35.90)   | 8 (38.10)  | 14 (43.70)   | 8 (28.60)    |
| > Once/month             | 5 (12.80)    | 9 (42.90)  | 3 (9.40)     | 11 (39.30)   |
| P value                  | 0.01* (χ²=8.84) | 0.02* (χ²=7.47) |
| Epileptic therapy        | n (%)        | n (%)      | n (%)        | n (%)        |
| Monotherapy              | 16 (41.00)   | 18 (85.70) | 11 (34.40)   | 23 (82.10)   |
| Poly therapy             | 23 (59.00)   | 3 (14.30)  | 21 (65.60)   | 5 (17.90)    |
| P value                  | 0.002* (χ²=9.36) | 0.002* (χ²=13.88) |

This table shows that there is significant statistical relationship between c and o VEMP abnormalities and duration of seizures, frequency of epileptic attacks, and type of therapy.

SD standard deviation, t student’s t test, χ² chi-square test
* = statistical significance
Sixty-five percent of the ears in the epileptic group had abnormal cVEMP either prolonged latency (36.7%), high amplitude (46.7%), and low amplitude (15%). Prolonged latencies are signs of central disorders [22]. Also brainstem lesions, especially those in the vestibulospinal tract may cause prolongation of p13 [23]. Some studies have shown that brainstem involvement in epilepsy is related to generalized seizures; however, it is possible to have focal epilepsy with brainstem disorders [24, 25].

The effect of epilepsy on brain stem and vestibulo-spinal tract may be because of demyelination [26]. Slinger et al. [27] reported significant white mater changes in corticospinal tract, vestibulo-spinal tract in epilepsy. The AEDs may cause vestibular abnormalities through the delayed conduction in the brain stem pathway due to inhibitory GABA neurotransmitter and channel blockage which interfere with the firing of CNS neurons [28]. So, brain stem involvement and vestibulospinal tract affection may be the cause of abnormalities in cVEMP in the current study.

VOR is processed via the brain stem and cortical vestibular circuits especially parietal-vestibular nuclei circuits. Cortical control of the VOR has been demonstrated in electrical stimulation and behavioral studies, additionally cortical disorders affect the vestibulo-ocular function [29]. So, cortical stimulation of the vestibular cortex in epilepsy may interfere with VOR. Fifty-three percent of the ears, in the epileptic group in this study, had abnormal oVEMP either prolonged latency (31.7%), high amplitude (23.3%), and low amplitude (5%). oVEMP reflects the function of the vestibular nuclei and the crossed VOR circuits in the medial longitudinal fasciculus (MLF) [16]. Brainstem disorders that may occur in epilepsy can affect the VOR due to involvement of the vestibular nucleus, nucleus prepositus hypoglossi [30]. MLF can be responsible for the fast-epileptic propagation to lateral temporal area, reported audiovestibular symptoms like tinnitus; dizziness can be due to the epileptic spread to the superior temporal gyrus through the MLF fibers [30]. Lesions involving MLF can present abnormalities of both c and oVEMPs [16].

In the current study, high amplitude of o and c VEMP response was frequently reported finding which represent vestibular hyperexcitability and hyperactive response from the otolith organs. The AEDs have side effects on the vestibular system, AED therapy for long time may cause vestibular symptoms due to central or peripheral vestibular affection [28].

Abnormal vestibular function among epileptic patients may be also attributed to the presence of a common cerebral disorder responsible for both seizures and vestibular dysfunction [31]. Gandelman-Marton et al. [20] evaluated balance function in epileptic patients (generalized tonic clonic seizure) using computerized dynamic platform posturography (CDPP) and revealed poorer Sway Index (SI) in the epileptic patients than the controls. Also, El-Gohary et al. [13] assessed vestibular functions in epileptic patients using videonystagmography (VNG); they reported abnormal saccadic testing, central vestibular affection with focal EEG activity, and peripheral vestibular affection with generalized EEG activity.

In the current study, there was no significant statistical relationship between VEMP abnormalities (ocular and cervical) and age, gender among the epileptic cases, but these abnormalities were increased with higher frequency of the seizure as frequent attacks may represent lack of control of the disease with associated increase in the vestibular abnormalities (Table 3). There was significant statistical relationship between VEMP abnormalities and the duration of seizure, the increase in seizure duration is related to the severity of the disease (Table 3) [32]. So, vestibular abnormalities may be related to the severity of the disease.

Co-administration of two or more AEDs may cause adverse effects like dizziness through blockage of sodium channels [33]. These side effects depend on the type and the dose of the drugs [15]. In the current study, the vestibular abnormalities were more in patients who received polytherapy than who received monotherapy (Table 3). This may be due to more side effects reported in polytherapy than monotherapy. Further studies should be conducted on the relation between vestibular dysfunction and different types and doses of AEDs, also studies comparing oculomotor test and VEMP to detect which is more sensitive in detecting central vestibular dysfunction.

The authors concluded that abnormal VEMP results (which reflect vestibular affection in epilepsy) were frequently reported in epileptic patients which may be interpreted by the effect of epilepsy on vestibular cortex and brainstem or as adverse effects of antiepileptic drugs. Vestibular abnormalities may be related to the severity and control of epilepsy.

Conclusion

Our study concluded that vestibular abnormalities were frequently reported in epileptic patients in the current study which may be related to the severity and control of epilepsy.

Abbreviations
VEMP: Vestibular evoked myogenic potentials; SCCs: Semicircular canals; SCM: Sternocleidomastoid muscle; EOG: Electromyography; AR: Asymmetry ratio; AEDs: Anti-epileptic drugs; VSR: Vestibular spinal reflex; MLF: Medial longitudinal fasciculus

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Nil.

Authors’ contributions
AHK searched the literature, analyzed, and interpreted the patient data, prepared, and reviewed the manuscript. KHA searched the literature and reviewed the manuscript. SME gathered and analyzed the patient data and
prepared the manuscript. ASM reviewed the manuscript. The authors read and approved the final manuscript.

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

Declarations

Ethics approval and consent to participate
Ethical approval from the ethical Committee of Faculty of Medicine, Menoufia University (according to Helsinki Declaration) with approval number (1055/9/5/2018) and written consent from all the patients were obtained.

Consent for publication
Not applicable.

Competing interests
There are no conflicts of interest to disclose.

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