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Main Article

Vestibular evoked myogenic potentials in patients with fibromyalgia syndrome

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Abstract

Objective: To assess vestibular evoked myogenic potentials in patients with fibromyalgia syndrome.

Methods: Twenty-four patients with fibromyalgia syndrome (two men and 22 women) and 21 female controls were included in the study. All patients underwent vestibular evoked myogenic potential testing.

Results: Statistical comparison of fibromyalgia patients with control subjects showed a significant difference with respect to n23 latencies and interpeak latencies ($p < 0.05$). There was no significant difference in p13 latencies, nor in p13 amplitudes, n23 amplitudes or interpeak amplitudes ($p > 0.05$).

Conclusions: Although patients with fibromyalgia syndrome generally have subjective neurotological symptoms, clinical and laboratory assessments usually fail to detect any objective abnormality. However, it is possible to detect abnormalities on vestibular evoked myogenic potential testing in such patients, indicating dysfunction in the vestibulospinal pathway, possibly in the saccule. Elongation of the n23 latency and of the interpeak latency of waves p13–n23, during vestibular evoked myogenic potential testing, may be a useful, objective indicator demonstrating neurotological involvement in fibromyalgia syndrome patients. Future research investigating the mechanisms of this latency elongation may help increase understanding of the pathogenesis of fibromyalgia syndrome.

Key words: Fibromyalgia; Vestibular Function Tests; Vestibular Evoked Myogenic Potential

Introduction

Fibromyalgia is a non-inflammatory disease characterised by chronic, widespread musculoskeletal pain, stiffness and tenderness.[1] In 1990, the American College of Rheumatology defined specific fibromyalgia syndrome criteria.[2] These criteria include the presence of widespread, chronic pain and mild or greater tenderness to digital palpation of at least 11 of 18 specified tender points.

Patients with fibromyalgia may experience various neurovegetative disorders, such as constipation, chilliness, low blood pressure, dermatographia and headaches.[3] Fibromyalgia is generally associated with psychological symptoms including fatigue, poor sleep, cognitive difficulties (such as memory problems, diminished mental clarity and concentration difficulties) and psychological distress.[4] Fibromyalgia may also be associated with a number of physical conditions including spastic colon, mitral valve prolapse, bursitis, chondromalacia, temporomandibular joint dysfunction, vertigo, sinus and thyroid problems, sensory symptoms, swollen glands, tinnitus, chronic cough, tachycardia, and weakness.[5] Although fibromyalgia is predominantly found in women aged 45 to 60 years, it can affect anyone at any age.

The aetiological and triggering factors for the chronic, widespread musculoskeletal pain of fibromyalgia syndrome are unknown. However, there is evidence implicating both genetic and environmental factors. Environmental factors may trigger the development of the disease; the onset of symptoms often follows a number of conditions including trauma, emotional stress, catastrophic events, serious infection, autoimmune disorders and other pain conditions.[6,7] Genetic factors may play a role in the development of the disease. A possible genetic linkage of fibromyalgia to certain human leukocyte antigen regions has been demonstrated by sib-ship analysis.[8]

Despite significant developments in the understanding of fibromyalgia pathophysiology in recent years, its aetiology and pathogenetic mechanisms are still unclear. However, several potential mechanisms for chronic, widespread pain have been proposed: central sensitisation, abnormalities of descending inhibitory pathways, neurotransmitter abnormalities, neurohumoral abnormalities and comorbid psychiatric conditions.[9]

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Aberrant central pain mechanisms have been suggested to explain the pathogenesis of fibromyalgia.[10] Increased excitability of the spinal cord neurons that transmit nociceptive information to the brain (i.e. central sensitisation) may be a possible underlying mechanism for heightened pain sensation in fibromyalgia. Patients with fibromyalgia also have increased sensation to various stimuli including touch, heat, cold, chemical stimuli, light, sound and smell.[11] The cause for this enhanced sensitivity is unknown, but dysfunctional central and peripheral pain processing may play a role.

Dysfunction of descending inhibitory pathways may also contribute to the chronic pain of fibromyalgia. Several neurotransmitters have been studied for their possible roles in the pathogenesis of the disease, including serotonin and dopamine. Since serotonin is an important modulator of pain perception, sleep and mood in healthy individuals, abnormalities in the metabolism and transmission of serotonin may be important in the pathogenesis of fibromyalgia syndrome.[12] The levels of primary metabolites of neuroepinephrine and 5-hydroxytryptamine are reduced and the level of substance P is elevated in the cerebrospinal fluid of patients with fibromyalgia.[13,14] It was shown that the hypothalamic–pituitary–adrenal axis is affected in fibromyalgia patients.[15] Alteration of central nervous system (CNS) blood flow is another suggested mechanism. Abnormalities in several areas of the CNS have been reported in fibromyalgia patients, including the thalamus, caudate nucleus, inferior pontine tegmentum, superior parietal cortex and gyrus rectalis.[16] Patients with fibromyalgia have a relatively higher incidence of comorbid psychiatric conditions, suggesting psychopathology as a possible predisposing factor for the disease.[17]

Vestibular evoked myogenic potentials are short-latency muscular responses evoked by high level acoustic stimulation. The electrophysiological techniques used currently to assess the vestibular system, such as electronystagmography and posturography, do not evaluate all the clinically relevant structures and pathways of the vestibular system. Vestibular evoked myogenic potential testing has become an attractive tool for the diagnosis of peripheral vestibular disorders, especially in patients with saccular pathology, as a complement to conventional vestibular tests. Vestibular evoked myogenic potentials are mediated by a pathway consisting of the saccular macula, its primary neurons, the vestibulospinal neurons from the lateral vestibular nucleus, the medial vestibulospinal tract, and finally the motor neurons of the ipsilateral sternocleidomastoid muscle. The intense sound stimulus delivered to the ear evokes vestibular-dependent, biphasic potentials (p13n23) recordable from the ipsilateral sternocleidomastoid muscle. The short onset latency of these responses (about 8 ms) suggests that they are mediated by an oligosynaptic arc, which probably consists of the primary vestibular afferents synapsing at the vestibular nuclei, the second order vestibulocollic neurones, and the neck motor neurones innervating the sternocleidomastoid muscle.

Vestibular evoked myogenic potential responses are considered to reflect the state of the vestibulocollic reflex arc, and such testing is used to assess the functional status of the otolith organ (i.e. the saccule), the inferior vestibular nerve and the vestibulospinal tract.[18] Some studies suggest the occurrence of neuro-otological abnormalities in fibromyalgia syndrome patients, which can occur in up to 30 per cent of the patients as dizziness is the most common complaint of fibromyalgia patients.[19,20]

Despite the high incidence of dizziness amongst fibromyalgia patients, no previous study has assessed such patients using vestibular evoked myogenic potential testing. Therefore, the present study aimed to evaluate fibromyalgia patients using vestibular evoked myogenic potential testing, in an attempt to evaluate the network formed by the saccule, inferior vestibular nerve and vestibulospinal tract.

Materials and methods

Twenty-four patients with fibromyalgia syndrome and 21 controls were included in the study. After taking a clinical history, a thorough otolaryngological examination was performed and blood tests undertaken to determine whether patients were appropriate for the study.

The diagnosis of fibromyalgia syndrome was made based on the American College of Rheumatology 1990 criteria.[2] Briefly, these criteria comprise diffuse aches and stiffness in muscle and tendon insertions upon digital palpation with an approximate force of 4 kg (the amount of pressure required to blanch a thumbnail), lasting for at least three months. Pain must be present in 11 or more of the 18 specified tender point sites. The fibromyalgia questionnaire was administered to all patients.[21] The patients had no symptoms other than pain.

We excluded from the study any patient with objective signs of articular or periarticular disease, an erythrocyte sedimentation rate of more than 10 mm/hour, a positive latex fixation test, elevated creatine phosphokinase values, or obvious underlying disease such as diabetes mellitus, chronic renal insufficiency, epilepsy, chronic psychiatric disorder, multiple sclerosis or hypothyroidism. Other exclusion criteria included: any anatomical or functional problem of the external or middle ear; a history of otosclerosis, chronic otitis media, neurotological or otological surgery; any neuropsychiatric problems; an age of less than 20 or more than 60 years; poor general condition; any neck problems; any central nervous system, systemic, metabolic or autoimmune disease; and an air–bone gap on pure tone audiometry.

Vestibular evoked myogenic potential testing

The sternocleidomastoid muscle was selected as the target for recording the vestibular evoked myogenic potential. Patients were positioned supine on a bed, and were instructed to elevate and turn their head contralaterally towards the ear being tested, to achieve maximal contraction of the sternocleidomastoid muscle. Surface electromyographic (EMG) responses

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TABLE I

VESTIBULAR EVOKED MYOGENIC POTENTIAL PARAMETERS IN FIBROMYALGIA PATIENTS AND CONTROLS

Parameter	Fibromyalgia pts* (mean ± SD)	Control group† (mean ± SD)	*t*	*p*
p13 latency	15.16 ± 0.89	14.73 ± 0.69	−2.52	0.15
n23 latency	24.42 ± 3.40	22.54 ± 1.62	−2.55	0.002
Interpeak latency	9.25 ± 2.98	7.83 ± 1.41	−2.03	0.003
p13 amplitude	126.90 ± 98.83	162.50 ± 115.84	0.79	0.39
n23 amplitude	173.65 ± 145.80	209.22 ± 174.57	0.48	0.62
Interpeak amplitude	300.49 ± 239.89	383.79 ± 281.59	0.61	0.50

*$n = 48$; †$n = 42$. Pts = patients; SD = standard deviation

were recorded with superficial electrodes. The active electrode was fixed over the middle third of the sternocleidomastoid muscle, the reference electrode over the sternoclavicular joint (where the sternocleidomastoid muscle attaches to the sternum) and the passive electrode over the middle of the forehead. The acoustic stimuli had an intensity of 95 dB HL and a frequency of 5 Hz, were of 5 ms duration with 1 ms rise and fall, and were conducted to the ears monoaurally with an insertion-type earphone. Electromyographic responses from each side were amplified and bandpass-filtered (10 Hz to 3 KHz). The analysis time was 50 ms. Responses were acquired by calculating the average of 128 stimuli. Two trials were obtained for each test in order to confirm the accuracy of responses.

A total of 48 ears of fibromyalgia patients and 42 ears of healthy controls were tested. After acoustic stimulation, the first positive myoelectrical peak was considered to be p13 and the first negative peak n23. The p13 and n23 latencies for the two trials were averaged to represent the latencies of each test. In addition, the difference between the amplitudes of p13 and n23 (i.e. the interpeak amplitude difference) and the difference between the latencies of p13 and n23 (i.e. the interpeak latency difference) were calculated.

Statistical analysis

The paired *t*-test was used to compare dependent variables (i.e. the right and left ears), and the independent samples *t*-test was used to compare the results of patients and controls.

Results

Twenty-four patients with fibromyalgia syndrome and 21 controls were included in the study. The fibromyalgia group included two men and 22 women, with a mean age of 37.41 ± 12.76 years (range 20–54).

Three patients complained of dizziness which could not be attributed to a specific disease. No other patients reported dizziness or vertigo.

The control group comprised 21 women, with a mean age of 32.71 ± 8.24 (21–50). There was no significant difference between the ages of the patients and controls ($p > 0.05$).

A total of 48 ears of patients with fibromyalgia and 42 ears of healthy controls underwent vestibular evoked myogenic potential testing. There was no statistically significant interaural difference for latencies or amplitudes, for either patients or controls ($p > 0.05$). The latencies and amplitudes for vestibular evoked myogenic potential responses obtained from fibromyalgia patients and control subjects are shown in Table I.

In the fibromyalgia patients, the mean latencies of p13 and n23 were 15.16 ± 0.89 ms and 24.42 ± 3.40 ms, respectively. The mean interpeak latency for the fibromyalgia patients was 9.25 ± 2.98 ms. The mean amplitudes of p13 and n23 in the fibromyalgia patients were 126.90 ± 98.83 mv and 173.65 ± 145.80 mv, respectively, while the mean interpeak amplitude was 300.49 ± 239.89 mv. The vestibular evoked myogenic potential abnormalities observed in the fibromyalgia patients are listed in Table II.

In the control group, the mean latencies for p13 and n23 were 14.73 ± 0.69 ms and 22.54 ± 1.62 ms, respectively, while the interpeak latency was 7.83 ± 1.41 ms. The mean amplitudes of p13 and n23 in the control group were 162.50 ± 115.84 mv and 209.22 ± 174.57 mv, respectively, while the mean interpeak amplitude was 383.79 ± 281.59. mv

Statistical comparison of fibromyalgia patients with control subjects showed a significant difference in n23 latencies and interpeak latencies ($p < 0.05$). There was no significant difference in p13 latencies, or in p13 amplitudes, n23 amplitudes or interpeak amplitudes ($p > 0.05$) (Figures 1 and 2).

Discussion

Animal studies have shown that intense acoustic stimulation activates otolith afferents.[22,23] This response is presumed to originate in the saccule.[24] Vestibular evoked myogenic potential responses arise from modulation of background muscle electrical activity, and differ from neural potentials in that

TABLE II

VESTIBULAR EVOKED MYOGENIC POTENTIAL ABNORMALITIES DETECTED IN FIBROMYALGIA PATIENTS*

Elongation of n23 & p13–n23 interpeak latencies in both ears in 6 pts
Elongation of n23 & p13–n23 interpeak latencies in left ear in 2 pts
Elongation of n23 latency in left ear in 4 pts
Elongation of n23 latency in right ear in 1 pt
Elongation of n23 & n23–p13 interpeak latencies in right ear in 1 pt

*as per normative clinical data. Pts = patients

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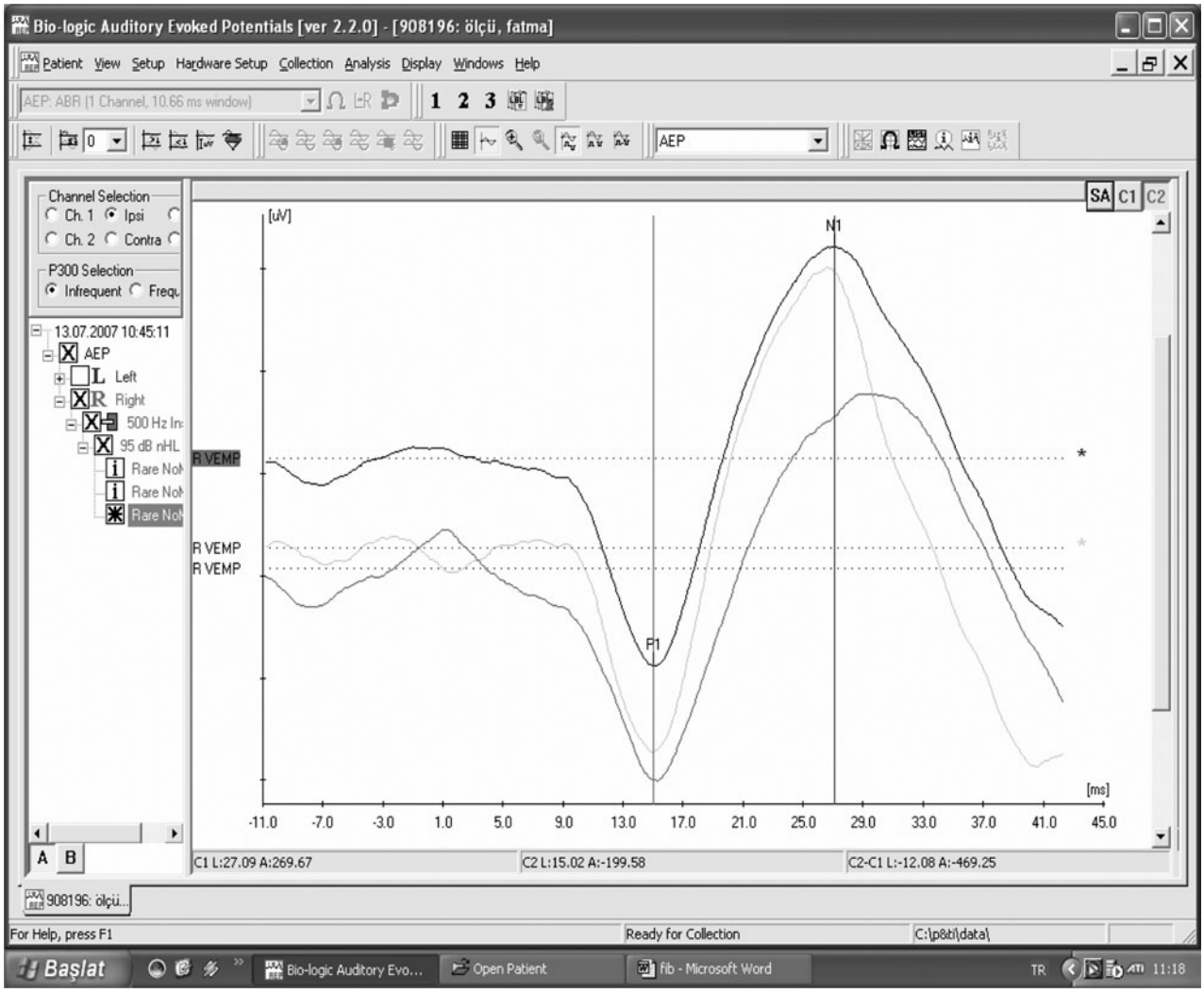

FIG. 1

Screengrab showing vestibular evoked myogenic potential recording of the right ear of a fibromyalgia patient. The amplitudes and the p13 latency are similar to those for control patients, whereas the n23 latency is prolonged compared with control patients.

they require tonic contraction of the sternocleidomastoid muscle.[25] The vestibular evoked myogenic potential response is assumed to reflect the state of the vestibulocollic reflex, which originates in the saccule and travels via the inferior vestibular nerve, lateral vestibular nucleus and medial vestibulospinal tract before finally terminating in the motor neurons of the sternocleidomastoid muscle. Thus, prolongation of the vestibular evoked myogenic potential indicates abnormal function of the saccule, vestibular nerve or vestibulospinal tract. During vestibular evoked myogenic potential testing, an intense auditory stimulus is introduced into the ear and short-latency myoelectrical responses are recorded from surface electrodes fixed over the ipsilateral, tonically contracted sternocleidomastoid muscle.[26]

The vestibular evoked myogenic potential response reflects the integrity of the lower brain stem and vestibulospinal tract.[27] Therefore, this response can be used to evaluate the function of the peripheral vestibular nerve, as well as to investigate central vestibulospinal tract abnormalities.[28] Because the vestibulospinal reflex has a descending pathway passing through the brain stem, vestibular evoked myogenic potential testing may be helpful in demonstrating brain stem involvement and determining whether aberrant central mechanisms affect the vestibulospinal reflex pathway, in fibromyalgia syndrome patients.

Prolonged vestibular evoked myogenic potential latencies, especially of p13, would suggest lesions in the vestibulospinal tract.[29] The vestibular evoked myogenic potential results of the current study indicated no significant difference in p13 latencies between the fibromyalgia patients and controls. However, n23 and interpeak latencies were significantly elongated in the fibromyalgia patients, compared with the controls. Despite the presence of a normal p13 wave, elongation of the n23 and interpeak latency suggests that the fibromyalgia pathogenetic mechanisms may have affected the sacculocollic reflex arc. The p13n23 potentials are thought to originate from the saccule.[30] Therefore, a saccular

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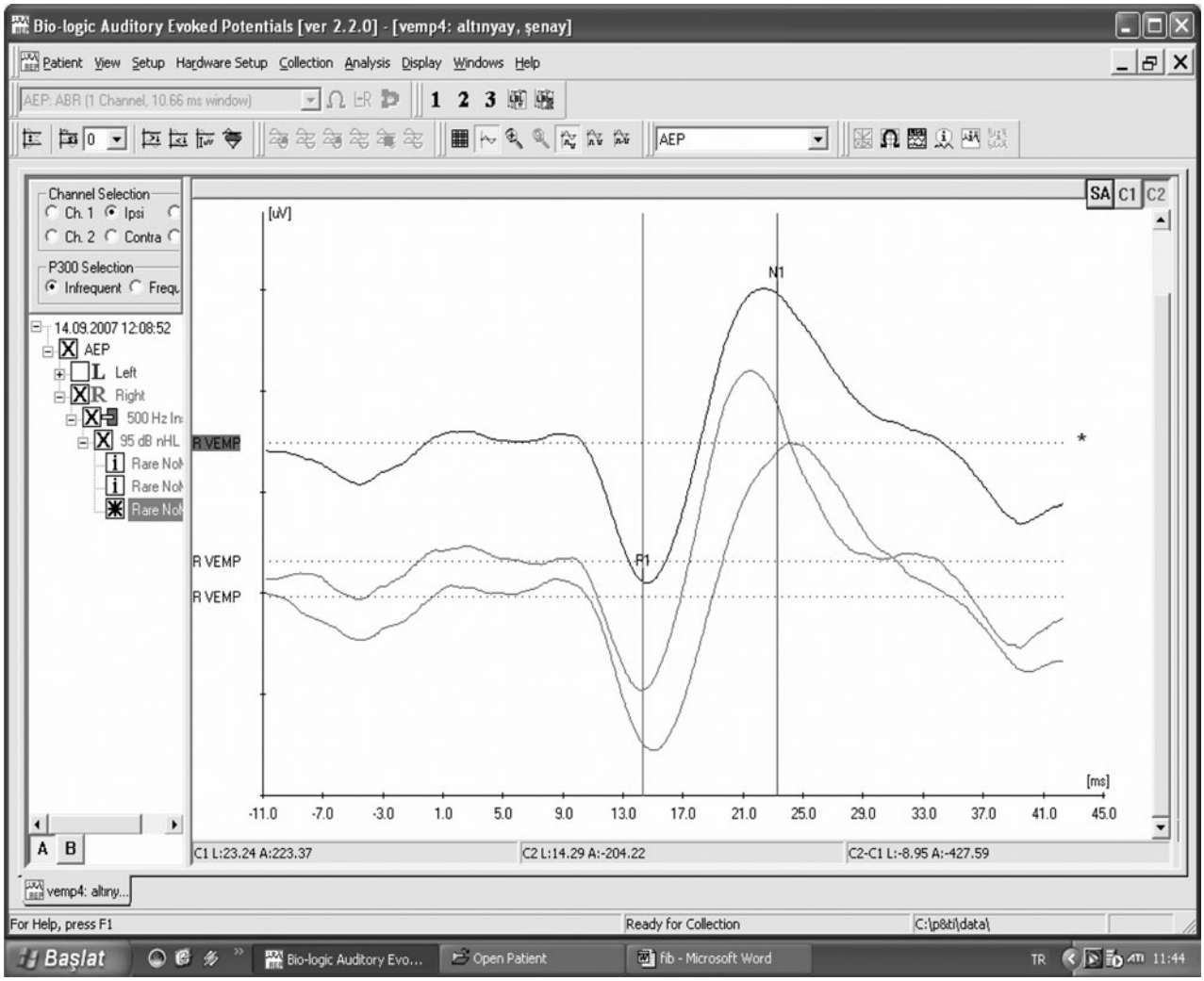

FIG. 2

Screengrab showing vestibular evoked myogenic potential recording of the right ear of a control subject. The amplitudes and the p13 latency are similar to those for fibromyalgia patients.

dysfunction is likely in fibromyalgia syndrome as far as elongation in n23 latency is concerned.

- **This study aimed to assess vestibular evoked myogenic potential responses in patients with fibromyalgia syndrome**
- **Fibromyalgia is a non-inflammatory disease characterised by chronic, widespread musculoskeletal pain, stiffness and tenderness**
- **Although fibromyalgia patients generally have subjective neurotological symptoms, clinical and laboratory assessments usually fail to detect any objective abnormality**
- **However, it is possible to detect vestibular evoked myogenic potential abnormalities in such patients, indicating vestibulospinal pathway dysfunction, possibly in the saccule**

Despite its clinical value, many factors can influence the results of vestibular evoked myogenic potential testing, including insufficient muscular contraction and altered tonicity of the cervical muscles.[28] Low tonicity of the sternocleidomastoid muscle generates small amplitudes, while excessive contraction generates higher amplitudes. Many studies have investigated p13 and n23 latencies and wave thresholds in patients with otological disease. However, few have investigated amplitude differences in cases of specific otological pathology. Such amplitude-related data vary greatly between individuals, laboratories and different degrees of sternocleidomastoid muscle contraction, and it is therefore difficult to directly apply such findings to clinical decisions.[31] For this reason, vestibular evoked myogenic potential amplitudes may be less reliable parameters for the evaluation of the vestibulocollic reflex, compared with latency data. This contention would appear to be supported by the findings of the current study: that is, the absence of any significant difference in amplitudes between fibromyalgia patients and controls, in the presence of elongation of n23 and interpeak latencies.

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Conclusion

Although patients with fibromyalgia syndrome generally have subjective neurotological symptoms, clinical and laboratory assessments usually fail to detect any objective abnormality. However, it is possible to detect abnormalities in vestibular evoked myogenic potential responses in such patients, indicating dysfunction of the vestibulospinal pathway, possibly involving the saccule. The elongation of n23 latency and of p13–n23 interpeak latency, seen on such testing, may be a useful, objective sign demonstrating neurotological involvement in patients with fibromyalgia syndrome. Future studies investigating the mechanisms of this latency elongation may improve understanding of the pathogenesis of fibromyalgia syndrome.

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