Int. Adv. Otol. 2010; 6:(2) 167-172

ORIGINAL ARTICLE

Tinnitus in Temporomandibular Disorders: Electrophysiological Aspects

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Objective: To evaluate the brainstem auditory evoked potentials (BAEPs) and the middle latency responses (MLRs) obtained in the patients with TMD and tinnitus in the hope of clarifying the electrophysiological aspects of this association.

Materials and Methods: The study group consisted of 22 adults with unilateral TMD and ipsilateral tinnitus. Two were men, and twenty were women, with a mean age of 34.4 years, ranging between 18 and 54 years. Fifteen adults with normal hearing, free of tinnitus and neurologic disease, served as the control group. There were 5 men and 10 women, with a mean age of 29.4 years, ranging from 22 to 35 years. Bilateral BAEP and MLR were recorded and statistically compared between the groups.

Results: BAEPs were not significantly different between these two groups ($p>0.01$). As for MLRs, the mean values of the latencies of the wave Pb and the wave Nc were significantly smaller than those of the control group ($p<0.01$). The mean values of the amplitudes of the waves Na, Pa and Pb were significantly greater in comparison with those of the control group ($p<0.01$).

Conclusion: These findings indicate that TMDs cause electrophysiological changes in the higher brain centers, while classical lemniscal pathway is not affected. It might be concluded that, by the stimulation of the somatic pain, extralemniscal pathway might be involved in tinnitus perception in the patients with TMDs.

Submitted : 07 January 2010 *Revised : 05 April 2010* *Accepted : 15 April 2010*

Introduction

Tinnitus is the perception of sound that results exclusively from activity within the nervous system without any corresponding mechanical, vibratory activity within the cochlea, and not related to external stimulation of any kind [1]. It can be objective, such as a tinnutus generated from an abnormal pattern of blood flow near the cochlea; or subjective, when there is no such physical sound attributable to the tinnitus. When the subjective tinnitus can be ascribed to a disorder of the ear or the acoustic nerve, it is referred to as "otic tinnitus". In the absence of such an otoneurologic disorder, the tinnitus is named as "non-otic tinnitus".

Temporomandibular disorders (TMDs) are among the etiological factors for non-otic tinnitus. Goodfriend [2], an American dentist, is credited as the first to report this relationship in 1933. Since then, the pathogenesis of tinnitus in TMDs has been attempted to be explained by various mechanisms: the eustachian tube hypothesis [3], the tensor tympani hypothesis [4], the oto-mandibular ligament hypothesis [5,6], the excessive somatic concern hypothesis [7]. However, all of these mechanisms have been regarded as speculative, and there exists no supporting data in the literature.

Recently, observations on the modulation of tinnitus perception by somatosensory system have lead to a new hypothesis, proposing that the somatosensory interactions with auditory system may play a role in the pathogenesis of tinnitus in TMDs [8]. Various explanations have been postulated to explain the role of this hypothesis in tinnitus generation in patients with somatosensory disorders, such as TMDs, whiplash syndrome, etc. Two different pathways, through which these interactions occur, have been put forward: lemniscal and extralemniscal auditory pathways [9-11].

The purpose of the present work was to study the brainstem auditory evoked potentials (BAEPs) and the middle latency responses (MLRs) obtained in the patients with TMD and tinntius, and to compare these results with those obtained from normal individuals. Thus, it was hoped that the possible locus or loci of interaction between the somatosensory system and the auditory system would be found out.

Materials and Methods

The study group consisted of 22 adults with unilateral TMD and ipsilateral tinnitus. Two were men, and twenty were women, with a mean age of 34.4 years,

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ranging between 18 and 54 years. The severity of TMD of each patient was graded according to Helkimo's Clinical Dysfunction Index [12]. The patients, in whom occurrence of the pain in the region of TMJ and the tinnitus were coinciding chronologically, were included in the study group. Patients with bilateral TMD were excluded from the study in order to identify the possible effects of unilateral lesion to the contralateral neural pathways. To avoid the possibility of neurotologic etiologies for tinnitus, TMD patients with abnormal otoscopic findings and audiograms (hearing thresholds poorer than 15 dB) were not included in the study. Patients with any kind of otological disorder in their history were also excluded. Fifteen adults with normal hearing, free of tinnitus and neurologic disease, served as the control group. There were 5 men and 10 women, with a mean age of 29.4 years, ranging from 22 to 35 years.

The auditory evoked response testing was performed by using a Nicolette Spirit System (Madison, Wisconsin, USA) with subjects reclining comfortably in a lounge chair. The surface electrodes were attached to Cz, C3, C4, A1, A2 and Fpz (ground). The recordings were performed in terms of the ear stimulated. When the stimulus was presented to the left ear, the BAEP data were obtained from channel Cz-A1, and the MLR data were obtained from channel C4-A2. For the right ear, the channels were Cz-A2 and C3-A1, respectively. Interelectrode impedance was balanced and maintened at less than 5 kOhm. The auditory stimuli consisted of click sounds delivered through a TDH-39 headphone at a rate of 9.7 pps at an intensity of 90 dB (nHL). The BAEP data and the MLR data were obtained by bandpass filtering 30 Hz-3.0 kHz and 10-250 Hz, respectively. Four averages of 1,000 stimuli repetitions were recorded for each ear.

The latencies and the amplitudes of waves I, III, V for BAEPs and Na, Pa, Nb, Pb, Nc for MLRs were analyzed, both for the ear ipsilateral to the TMD and for the ear contralateral to the TMD with comparison to the mean values of these waves in the control group.

The statistical analysis was undertaken with the statistics program SPSS® for Windows. The independent samples t-test was used for comparison of means. The difference would be accepted as statistically significant, if the value of p was smaller than 0.01.

Results

Of the 22 patients, 11 had TMD on the right side, while 11 patients complained of this disorder on the left side. In all patients, the severity of pathology was grade 2 according to the Helkimo's Clinical Dysfunction Index. Thus, the study group was homogeneous on this issue, so that the effects of the joint pathology would be also similar.

When the acoustic stimulation of the ears ipsilateral to the side of TMD was considered, it was noted that the mean values of the latencies and the amplitudes of the waves I, III and V in BAEPs were not significantly different in comparison to the mean values of these waves in the control group ($p>0.01$). Similarly, no significant differences were found for the mean values of these waves, when the contralateral ears were stimulated ($p>0.01$).

As for the MLRs obtained after the stimulation of the ipsilateral ears, the mean values of the latencies of the wave Pb and the wave Nc were significantly smaller than those of the control group ($p<0.01$). The mean values of the amplitudes of the waves Na, Pa and Pb were signficantly greater in comparison with those of the control group ($p<0.01$). The amplitudes and the latencies of the remaining waves revealed no significant difference for the stimulation of the ipsilateral ears.

When the stimuli were presented to the contralateral ears, similar statistical results were obtained as for the ipsilateral ears. That is, the mean latencies of the waves Pb and Nc were significantly shortened, while the mean amplitudes of the wave Na and the wave Pa were significantly increased when compared with the mean latencies and the mean amplitudes of the control group ($p<0.01$). No significant differences were encountered for the mean latencies of the waves Na, Pa, Nb, and the mean amplitudes of the waves Nb, Pb and Nc ($p>0.01$).

Table 1 summarizes the mean±2SD values of the latencies and the amplitudes of the waves for the BAEPs and the MLRs for the study group and the control group. Figure 1 shows the MLR waveforms obtained from a representative subject (no:21) in the study group with repect to the temporal range of the latencies of the control group. Figure 2 shows the MLR amplitudes of the same subject in comparison with the mean±2SD amplitudes of the control group.

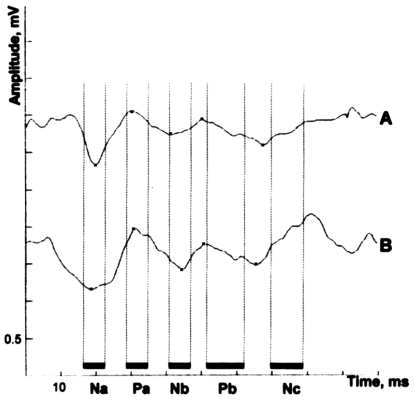

Figure 1. Two MLR tracings for subject 21 of the study group were the average of four runs. Trace A and B were obtained form the contralateral ear and ipsilateral ear, respectively. Temporal range of wave latencies of the control group is shown just above the axis of the diagram.

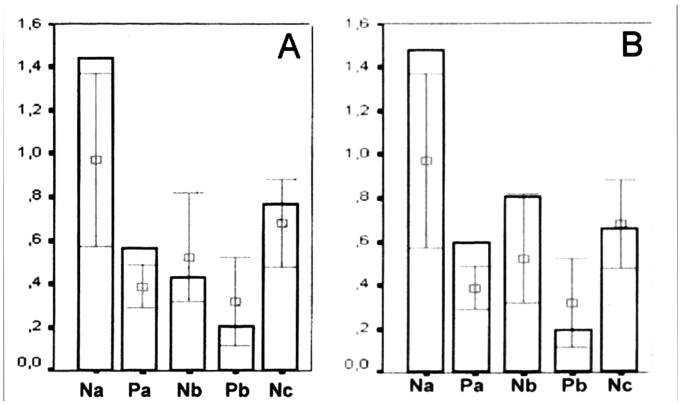

Figure 2. MLR amplitudes of subject 21 of the study group. The mean amplitudes for the control group are displayed as mean±2SD.

Table 1. The mean±2SD values of the latencies and the amplitudes of the waves for the BAEPs and the MLRs for the study group and the control group.

Wave	Control Group		Ipsilateral Ear		Contralateral Ear	
	Amplitude	Latency	Amplitude	Latency	Amplitude	Latency
I	0.24±0.2	1.54±0.1	0.29±0.1	1.57±0.1	0.28±0.1	1.58±0.1
III	0.38±0.1	3.35±0.1	0.39±0.2	3.64±0.3	0.39±0.3	3.59±0.1
V	0.37±0.1	5.45±0.1	0.40±0.2	5.46±0.2	0.37±0.3	5.49±0.3
Na	0.97±0.4	19.43±2.2	1.25±0.3	18.6±0.2	1.34±0.4	18.8±2.2
Pa	0.39±0.1	30.89±3.3	0.68±0.5	31.11±2.8	0.62±0.3	30.36±2.4
Nb	0.52±0.3	43.57±2.8	0.66±0.4	43.48±3.6	0.48±0.2	43.53±3.7
Pb	0.32±0.2	56.73±4.9	0.58±0.4	53.28±4.2	0.48±0.4	54.01±4.5
Nc	0.68±0.2	74.56±5.4	0.75±0.6	70.77±4.0	0.79±0.5	71.41±4.4

Discussion

The somatosensory system seems to be the only sensory modality that can significantly modulate tinnitus perception. Systematic studies estimate that more than 30% of tinnitus patients can somatically modulate their tinnitus with face, head and neck movements [8]. Positron emission tomography mapping of the brain regions has shown that orofacial movements not only alter the loudness of tinnitus, but cause unilateral changes in the cerebral blood flow of tinnitus patients, as well [13]. Modulation of tinnitus perception by somatosensory changes also occurs in the physiologically normal individuals. However, there have been no reports of visual, gustatory or olfactory associated tinnitus [9].

The results of the study by Møller et al. [11] have shown that the perception of loudness of certain sounds, as well as certain forms of tinnitus, can be manuplitated by stimulation of the somatosensory system. The fact, that the stimulation of the somatosensory system can increase the loudness of the tinnitus, indicates that somatic pain may aggravate a patient's tinnitus [11]. As for tinnitus in TMDs, there exist an association between tinnitus and pain in the region of ear and/or temporomandibular joint [9]. Some investigators emphasize the joint's role and refer to the syndrome as "temporomandibular joint syndrome" or "craniomandibular disorder" [4-14]. Others stress the muscle tension as the key to the syndrome and describe it as "myofacial pain – dysfunction syndrome" [15-17].

Levine [9] has proposed that the non-otic tinnitus, such as in TMDs, can arise from somatic – auditory interactions in the lemniscal pathway of the central nervous system. Considering the observation that tinnitus complaint is generally lateralized to the side with pain in patients with TMDs [15], Levine has suggested that non-auditory interaction with the auditory system for this non-otic tinnitus is occuring at the level of the cochlear nucleus. This is because it is the only part of the lemniscal pathway before the trapezoid body, which is the first auditory decussation important for sound lateralization [18].

However, the results of the present study did not reveal any supporting data for the above mentioned hypothesis. The latencies and the amplitudes of the waves I, III and V in the recordings of BAEPs of the TMD patients with unilateral tinnitus were not significantly different when compared with those of the control group. Assuming the existence of a relationship between the somatosensory inputs and cochlear nucleus, a shortening of the latency and/or an increase of the amplitude of the wave III would be expected, since, according to Levine, increased activity in the cochlear nucleus is associated with tinnitus.

On the other hand, the present study revealed significantly shortened latencies of the wave Pb and the wave Nc, and significantly increased amplitudes of the Na, Pa in MLRs of the study group in comparison with the control group. These significant differences were obtained bilaterally, that is, by stimulating both the ipsilateral ears and the contralateral ears. In addition, the mean amplitude of the wave Pb was significantly increased, when the stimulation was presented to the ipsilateral ears. These data point out to the presence of hyper-responsive loci in the subcortical and cortical portions of the auditory system. However, since a consensus is still lacking regarding the origin of MLRs in humans [19,20], it is impossible to name the exact loci with these data.

Recently, a number of reports have made it clear that injury to or stimulation of the peripheral portion of the auditory system can result in hyperactivity in more centrally located structures [21]. Another observation is that tinnitus involves a number of regions of the auditory system [13,22,23]. Supporting these data, the results of the present study indicates that unilateral somatosensory stimulation in patints with TMDs result in multifocal hyperactive reponses in the subcortical and cortical auditory system as demonstrated by MLRs. However, since normal responses were acquired in BAEPs, an alternative pathway, which provides the interaction between the somatosensory system and auditory system, should exist in the central nervous system.

In their study with the patients with intractable tinnitus, Møller et al. [10] have noted that there is no difference for the patients with or without tinnitus in the latencies of wave III, which has been assumed to be generated by the cochlear nucleus. This study of Møller et al. has revealed slight but statistically significant shortening of latency of wave V of the BAEPs. Authors have concluded that the location of the physiological abnormality that resulted in tinnitus in these patients might be between the cochlear and the inferior colliculus. However, in their latter study, they have noted that these data must be interpreted cautiously, because of the small number of patients studied and the small difference in the latency of wave V between the patients with tinnitus and those without tinnitus, who had the same degree of hearing loss. Therefore, they have suggested that the extralemniscal pathway might be involved in generating the tinnitus that these patients experienced [11].

The extralemniscal portion of the ascending auditory system transmits auditory information to higher brain centers in conjunction with the classical lemniscal pathway. Although this system is believed to branch off from the classical ascending lemniscal pathway at the level of the inferior colliculus [24], the system is scattered throughout the core of the brainstem [25,26]. Thus, it does not contribute noticeably to BAEPs. Many neurons of the extralemniscal pathway receive inputs from other sensory modalities, such as the somatosensory system [24]. It is also known from animal experiments that the responses of neurons in the extralemniscal pathway to auditory stimulations can be modulated by stimulating another system, such as the somatosensory system [28,29]. The cortical areas to which the extralemniscal system projects are therefore also known as “the polysensory areas” [11].

The lack of the electrphysiologic data for the evaluation of the extralemniscal system might be an explanation to the presence of normal responses in the BAEPs of the TMD patients with tinnitus in the

present study. The presence of hyper-responsive loci in the cortical and subcortical auditory system detected in MLRs, are probably the result of active neural inputs to more central auditory system from the somatosensory system via the extralemniscal pathway. Bilateral occurrence of these hyper-responsive loci point out the fact that the somatosensory stimulation by-pass the peripheral auditory system, where binaural interactions does not exist. Thus, in the light of the fact that auditory and somatosensory informations interact by the extralemniscal pathway, but not by the lemniscal system; it might be concluded that, by the stimulation of the somatic pain, the extralemniscal pathway might be involved in tinnitus perception in the patients with TMDs.

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