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Auditory symptoms in subjects with Occipital neuralgia and concomitant Craniomandibular Disorders: A comparison study.

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Abstract
Introduction: *Ear symptoms constitute a subgroup of very complex disorders in the field of Orofacial Pain, Neurology, Ear, Nose and Throat disorders. They are currently considered a diagnostic challenge for clinicians and even for specialists in the field.* ***Goals:*** *Evaluate frequencies of dizziness, vertigo, ear stuffiness, diminished hearing and tinnitus in subjects with Craniomandibular Disorders and Occipital Neuralgia and in those without.* ***Methods:*** *History of the chief complain, evaluation of signs and symptoms, self-reported questionnaires, clinical examination, palpation of joint and muscles, self-report and biomechanical tests were used to gather data about signs and symptoms of Craniomandibular Disorders, bruxing behavior, occipital neuralgia and ear symptoms. Clinical records of 90 subjects with Craniomandibular Disorders and Occipital neuralgia, 80 with Craniomandibular Disorders and no Occipital Neuralgia and 30 with no Craniomandibular Disorders and no Occipital Neuralgia were retrieved from a database, evaluated retrospectively and compared regarding some auditory signs and symptoms. Data were analyzed using Kruskal-Wallis statistics, Fisher´s exact test and Chi-squared for independence and trends.* ***Outcome:*** *The frequency of dizziness was about 84,4%, 40% and 23,3% in the Craniomandibular Disorder and Occipital Neuralgia, Craniomandibular disorder no Occipital Neuralgia and control subgroup, respectively. The frequency of vertigo was about 34,4%, 11,3%, and 3,3% in these same subgroups. Ear stuffiness was reported by 60%, 35% and 16,7%, diminished hearing by 54,5%, 22,5% and 3,3% and tinnitus by 68,9%, 25% and 16,7% subjects respectively in the same subgroups. Chi-squared for independence ($p<0,0001$) and for trends ($p<0,0001$) showed that all groups were independent and that the frequency of every specific ear symptom increased from the control no Craniomandibular Disorder and No Occipital neuralgia subgroup to the Craniomandibular Disorder but no Occipital neuralgia and then to the Craniomandibular Disorder with Occipital neuralgia subgroup. Higher frequencies of ear disorders were observed in the Craniomandibular Disorder subgroup with Occipital Neuralgia when compared to the other subgroups.* ***Conclusion:*** *Higher frequencies of dizziness, vertigo, diminished hearing, ear stuffiness and tinnitus were observed in the Craniomandibular Disorder and Occipital neuralgia subgroup as compared to the control subgroups. Hearing disorders should be accepted as part of the pathological profile of subjects presenting with occipital neuralgia with or without Craniomandibular Disorders.*

Key Words*: Occipital Neuralgia. Aural Symptoms. Craniomandibular Disorders. Signs. Symptoms*

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I. Introduction

Craniomandibular Disorders (CMDs) or Temporomandibular joint dysfunction (TMJD) is a common condition affecting up to 33% of the general population and is characterized by the presence of unilateral or bilateral pain in the temporomandibular joints (TMJs) and adjacent masticatory muscles[1]. CMDs also present other clinical features including a complaint of pain, different joint noises, tenderness to palpation of the masticatory muscles and impaired jaw movements. Many CMDs individuals also complain of different types of headaches[2].

Occipital Neuralgia (ON) is a refractory and disabling pain disorder described as "recurrent episodes of moderate to severe pain localized in the occipital region but with frequent radiation to the face and neck[3].

ON patients usually complain of episodes of throbbing, stabbing, shooting and burning pain unilaterally or bilaterally in the back of the head and neck, sometimes described as dull but referred to the anterior part of the head and sometimes to the face[4]. Burning, shooting or stabbing, the presence of paraesthesia and a pain generating zone located medially and below the mastoid process are the hallmarks of ON. Neuralgia is pain occurring in one or more nerves caused by compression and/or irritation of peripheral nerve structures. Chronically contracted muscles and spondylosis of the upper cervical spine may compress and/or irritate the greater occipital nerve (GON) or the lesser occipital nerve (LON), thus, causing pain and dysfunction[5].

ON is also known as Arnold´s neuralgia. This disorder closely resembles the symptoms of migraine and in which some ear disorders including dizziness, vertigo and others, are frequently described by patients[4] In the medical areas of Neurology and Ear, Nose and Throat is very common to see patients who present with migraine-like symptoms and at the same time, dizziness, vertigo, aural fullness, auditory symptoms and tinnitus[6]. One problem with this description is that even though the headache symptoms seem to be those of migraine, in actuality, what patients describe is usually a set of signs and symptoms of ON in which, aural symptoms occur more frequently. Due to many connections of the GON with some cranial nerves including the VIII, IX, X and even with the cervical sympathetic ganglion, a number of ear symptoms associated with ON have been reported in the medical literature[5].

Ear symptoms including ear stuffiness, dizziness, tinnitus and ear fullness sensation have been reported frequently in the dental literature. These symptoms have been anecdotally related to TMDs and the occlusion. The true is that during decades it has been very difficult for the dental and medical profession to extricate the mechanisms and etiology of such disorders. For instance, such symptoms have been associated with CMDs, but other superimposed disorders including migraines and ON have not been considered in the evaluation of CMDs subjects. In the last two decades and during the examination process we have observed that ON occurs much more frequently than migraine in samples of CMDs individuals as ON may mimic both migraine and CMDs. Because of such observations, this studied was designed to:

1.Evaluate the frequency of some aural symptoms in a selected population of subjects with CMDs and ON;
2.Because it has been accepted that the prevalence of ear disorders in CMDs and BB subjects is high and recent reports point to ear disorders as part of the pathological profile of ON subjects, we expect to observe a higher frequency of some ear disorders in CMDs and ON subjects as compared to CMDs subjects without ON and to control subjects with neither CMDs nor signs and symptoms of ON.
3.Discuss the most common mechanisms implicated in ear disorders and referred pain to distant anatomic structures in CMDs and ON subjects.

II. Methods

Patients referred to the Orofacial Pain (OFP) Department, School of Dentistry Gurupi University are evaluated using a strict and comprehensive protocol described as follows: Description and analysis of the chief complaint, evaluation of signs and symptoms, use of self-report and clinical examination, a set of questionnaires, self-report and clinical examination to assess severity and type of BB, biomechanical tests to evaluate type and severity of internal derangement of the TMJs (TMJs-ID) and questionnaires to examine the most common types of headache, for instance, tension-type headache, migraine, myofascial headache and occipital neuralgia. Further, another instrument is also used to gather information about oral jaw habits, and psychological tests are also used if deemed appropriate. Following comprehensive evaluation, subjects are classified as bruxers or non bruxers, mild, moderate, severe, extreme bruxism, daytime, nighttime or mixed bruxism, CMDs or non CMDs, migraine, TTH, Myofascial headache, ON and migraine sufferers based on widely accepted clinical criteria.

Once the comprehensive evaluation is completed, the medical and dental records of every referral is stored in a database for future evaluation of a variable of scientific or clinical interest, for instance, nausea and vomiting in chronic tension-type headache as compared to acute TTH. In the last two months, we consecutively retrieved the medical and dental records of 90 subjects demonstrating signs and symptoms of CMDs and ON, 80 records of subjects demonstrating signs and symptoms of CMDs without ON (Control group 1) and a subgroup of 30 subjects with no CMDs and no ON (Control group 2). Criteria explained as follows were used to classify subjects in subgroups presenting CMDs and ON, CMDs No ON, and No CMDs No ON:

Criteria for CMDs: A complain of pain in the masticatory system, joint noises based on self-report and clinical examination, tenderness to palpation of the masticatory muscles and/or TMJs, difficulties to perform normal jaw movements and headache of musculoskeletal origin.

Criteria for ON: Pain in the cervical region radiating inside or above the orbit, pain described as moderate severe or very severe, shooting, stabbing, jabbing, continuous and/or intermittent, burning, presence of a pain generating zone medially to the mastoid process, presence of ear disorders including dizziness, vertigo, ear stuffiness, tinnitus and hearing deficiency, numbness in the vertex area and/or in the face.

Exclusion criteria: Subjects were excluded and a comprehensive medical and dental evaluation was not carried out if they presented with severe psychological and/or psychiatric disorders, motor disturbances, some type of epilepsy including but not restricted to Parkinson Disease and learning and/or cognitive difficulties. The medical and dental records of such subjects were not stored in the database and thus, they were not retrieved to take part in any retrospective study.

Statistical Analysis

Kruskal-Wallis nonparametric statistics was used to carry out an analysis of variance (ANOVA) when the three subgroups (CMDS + ON, CMDS No ON and Controls no CMDs no ON) were compared regarding age. Fisher´s exact test was used to assess significance when the frequency of a symptom or disorder was compared in two subgroups. Finally, Chi-squared for independence and for trends were used when the frequency and trend of a specific disorder (for instance ear stuffiness) was compared in three different subgroups organized in a certain order, for instance the normal subgroup (no CMDs No ON), compared to the dysfunctional subgroup (CMDs No ON) , compared to the most dysfunctional subgroup (CMDs and ON).

III. Outcome

This investigation evaluated a subgroup of CMDs and BB subjects with ON (n=90), a subgroup of subjects with CMDs and No ON (n=80) and a subgroup of subjects with no CMDs and no ON (n=30). The group with CMDs but no ON was used as a Control group to be compared with the CMDs + ON subgroup whereas the Non CMDs and No ON was used to form a gradient to evaluate the behavior of some ear disorders by the degree of severity, for instance CMDs + ON versus CMDs No ON versus No CMDs No ON. Mean age in these subgroups is described as follows: CMDs + ON (Mean=40,5, SD=10,1, range=15-75); CMDs No ON (Mean=32,0, SD=12,4, range=17—73) and Controls no CMDs no ON (Mean=44,5, SD=14,1, range=17-73). There was a statistically and significant difference when age was compared in these three groups (Kruskal-Wallis statistics p<0,0001): CMDs + ON versus CMDs No ON (p<0,001); CMDs + ON versus Controls No CMDs No ON (p<0,01); CMDs No ON versus No CMDs No ON (p>0,05). See Table 1 for additional details.

The frequencies of dizziness, vertigo, ear stuffiness, ear deficiency and tinnitus in the subgroups with CMDs + ON, CMDs No ON and No CMDs No ON or Control, as described as follows: dizziness 76/90=84,4%, 32/80=40% and 7/30=23,3%, respectively. Vertigo: 31/90=34,4%, 9/80=11,3%, and 1/30=3,3%, respectively. Ear stuffiness: 54/90=60%, 28/80=35%, and 5/30=16,7%, respectively. Ear deficiency: 49/90=54,4%, 18/80=22,5%, and 1/30=3,3%, respectively. Tinnitus: 62/90=68,9%, 20/80= 25%, and 5/30=16,7%, respectively. See Table 2 for further details. The frequencies of dizziness, vertigo, ear stuffiness, ear deficiency and tinnitus were compared in different subgroups to evaluate for possible statistical significance once a difference in frequency was detected. Statistical significance in the frequencies of pair of subgroups was assessed using Fisher´s exact test. With the exception of dizziness in the subgroups CMDs no ON versus Controls (Fisher´s exact test p=0,12), vertigo in the subgroups CMDs No ON versus Controls (p=0,28), ear stuffiness in the subgroups CMDs No ON versus Control (p=0,06), and tinnitus in the subgroups CMDs No ON versus Control (p=0,44), comparison between other symptoms in different pairs of subgroups yielded statistically significant differences. See table 3, for additional details.

When the three subgroups were ordered from the Control one (No CMDs No ON) to the CMDs No ON and to the CMDs + ON subgroups, that is, from the normal subgroup (No CMDs No ON) to that with more severe symptoms (CMDs + ON) in order to form a gradient of severity, with the assumption that the frequencies of ear symptoms would increase from the normal subgroup in the direction of the more impaired CMDs subgroup, the following observations should be mentioned:

1: Chi-squared for independence yielded a p-value=0,0001. Consequently we can state that the subgroups were independent.

2.Chi-squared for trends yielded a p-value=0,0001. Thus, there was a trend to a higher frequency of ear symptoms from the No CMDs no ON to the group with CMDs and no ON and then to the subgroup with CMDs and ON. See table 4 for further details.

IV. Discussion

1.Subjects with CMDs and ON demonstrated higher frequencies of specific ear symptoms.

In the current investigation a prevalence of 84,4% dizziness was observed in the subgroup of CMDs and ON subjects. Thus, dizziness was the ear disorder with the highest prevalence among subjects in the experimental subgroup. The prevalence of dizziness was quite different from the frequency of 40% to 70% reported in a previous investigation in TMDs patients[7]. The difference is explained by the fact that we examined patients with CMDs and concomitant ON that obviously inflates the frequency of dizziness. Kusdra and associates[8] reported a frequency of 30,5% of dizziness in a large sample of CMDs subjects. Again, this

difference in frequency can be attributed to the fact that we examined CMDs and ON subjects following a strict selection criteria for both disorders. Further, it is not a rule for investigators in CMDs to examine the presence of ON in CMDs subjects. Kuhn, Kuhn and Gilberstad[9] evaluated a small sample of subjects presenting with signs and symptoms of ON but without CMDs. Researchers reported a frequency of 50% dizziness together with other ear disorders. This difference is undoubtedly explained by the fact the higher frequency reported in the current investigation yields frequencies of two concomitant disorders (ON + CMDs) presenting with similar ear disorders, thus increasing the prevalence in those presenting with both CMDs and ON. Molina and associates[10] reported a frequency of 40.6% of dizziness in a group of ON subjects with concomitant CMDs. This lower frequency is probably associated to a methodological difference during sample evaluation, to the fact that the sample was not so large and probably to the fact that newer and probably more chronic and complicated cases were referred with time to the same facility thus, inflating the frequency of dizziness. Cervicogenic dizziness is most frequently associated with flexion and extension injuries on the cervical structures and such symptom has also been reported in patients with severe cervical arthritis, herniated cervical disks and head trauma[11]. Thus, it is apparent that dizziness may be associated with a number of musculoskeletal disorders.

In the current study, a frequency of 34,4% **vertigo** was reported by subjects in the CMDs + ON subgroup. Thus, this outcome is consonant with one investigation[7] in which researchers reviewed the current literature and indicated that in CMDs patients, vertigo can be reported by 5% to 40% of the cases.. Mota and colleagues[12] reported a frequency of 9,5% vertigo un a group of 21 patients presenting with CMDs sign and symptoms. The low prevalence may be due to the fact that the sample was small, very likely researchers did not evaluate the presence of ON signs and symptoms and probably the method they used to collect the information about vertigo. Lam and associates[7] in their experimental investigation in 204 subjects presenting with TMJ and aural symptoms reported a frequency of vertigo of about 64,7%. It is likely that this higher prevalence was associated with the fact that in Lam and associates´ study, patients were referred to a tertiary unit to which the most complex cases are referred. Vasaghi-Gharamaleki and Naser[13] evaluated a group of 42 subjects presenting with cervical spine injury. They reported a frequency of 48,5% vertigo in the participants of the experimental study. It is likely that such a higher frequency may be attributed to severer damage to cervical structures including the GON and LON in the group they evaluated.

In the current research we report a frequency of 60% ear stuffiness in the subgroup presenting with CMDs and ON. This frequency was lower in the CMDs No ON and in the control subgroup. In one investigation[8] researchers evaluated a large sample of CMDs subjects and reported a lower frequency of ear stuffiness (39%) probably due to the absence of subjects presenting with ON signs and symptoms. The frequency we report in the current investigation was more similar to the frequency of 74% ear stuffiness reported in one investigation[14] in CMDs no ON subjects. Ferendiuk and associates[15] reported that many ear disorders occur at the same time in the same individual. For instance, in some patients they evaluated, they reported that tinnitus was accompanied by an ear plugging sensation. Mota and associates[12] evaluated a subgroup of 21 subjects presenting with signs and symptoms of CMDs and reported a frequency of 81% ear fullness, a frequency which is a little higher as compared with the outcome in the current study. Such difference may be attributed probably to the lower sample they evaluated and/or to methodological differences.

Hearing deficiency was reported by 54,5% of CMDs and ON subjects in the current investigation (22,5% in the CMDs no ON and 3,3% in the no CMDs no ON subgroup). **Lam and** associates[7] evaluated a sample of 344 subjects referred consecutively to an Orofacial Pain Unit. They reported that aural symptoms were more frequently observed in the CMDs as compared to the Control subgroup, thus, the outcome in their study is in line with findings in the current investigation. They reported that even though they did not evaluate the prevalence of ON (if present), they reported that 62,2% of the subjects they assessed had both CMDs and perceived hearing loss a figure which compares favorably with the outcome in the current investigation. CMDs patients often complain of associated disorders including diminished hearing which may be present in 18% of the group[16]. This difference may be explained by methodological reasons and probably by the fact that we evaluated CMDs subjects with ON and Fricton and coworkers evaluated subjects with myofascial pain (MPDS). MPDs not necessarily occurs together with ON and temporomandibular disorders.

The combination of CMDs and ON with the enormous amount of signs and symptoms leads clinicians and specialists to diagnose and treat these cases. **Kitsoulis** and associates[17] evaluated 340 CMDs subjects and reported that the frequency of ear disorders including hearing difficulties increased with the severity of CMDs. Such observation is consonant with findings in the current study. They also reported that number of aural symptoms was directly related to the severity of CMDs. This observation reinforces data in the current investigation as we found higher frequencies of ear symptoms when the experimental subgroup (CMDs + ON) was compared with the CMDs No ON and with the Control subgroup with neither CMDs nor ON. ON is considered a very disabling headache or craniomandibular disorder. Because Kitsoulis and associates[17] reported a prevalence of 48,3% hearing loss in the CMDs subgroup their data compare favorably with the

outcome observed in the current investigation. Nowaczewska and coworkers[18] evaluated ear disorders in a large sample of subjects with tinnitus and headache compared to another subgroup of tinnitus no headache subjects. Both subgroups were not typical CMDs patients. They found that some ear disorders occurred more frequently in the subgroup with tinnitus and headache than in the subgroup with tinnitus and no headache. Researchers in such a study reported a prevalence of 32,6% hearing loss as compared to the frequency of 54,4% observed in the current investigation. The higher frequency in the current study is explained by the fact that all subjects in the current investigation had signs and symptoms of ON and also that the higher frequency of reported hearing loss included unilateral and bilateral hearing loss thus inflating the result. Subjective hearing disorder that occurs frequently in patients with functional disorders of the masticatory system has been broadly reported in the current literature[15] However, data on ear disorders in subjects with both CMDs and ON are extremely scarce.

Tinnitus was reported by **68,9%** subjects in the subgroup presenting with ON and CMDs. This prevalence of tinnitus observed in the current investigation is higher as compared to 42% of tinnitus reported in a previous study[8]. This lower prevalence is explained by the fact that Kusdra and associates evaluated TMDs rather than TMDs and ON subjects. One investigation in CMDs subjects**[7]** reported a frequency of 64,1% of tinnitus, a frequency which is quite similar to that reported in the current study. This similarity of data is in some way influenced by the fact, that Lam and associates evaluated CMDs subjects, and the frequency of ON in CMDs subjects is high, thus, contributing to a high frequency of tinnitus. Tinnitus is the acoustic sensation heard in one or both ears or inside the patient´s head when no acoustic stimuli are coming from the outside[15]. In the current study, we found that tinnitus was the second most common ear disorder in the subgroup demonstrating signs and symptoms of both CMDs and ON. Thus, findings are endorsed by one study[15] about ear, nose, throat symptoms in patients with TMDs but no ON reporting that tinnitus was the second most common ear symptom in the sample researchers evaluated. Finally, Kuhn, Kuhn and Gilberstadt[9] evaluated a small group of medical records of subjects referred to an university- based hospital presenting with signs and symptoms of ON. They reported a frequency of 33% tinnitus in the sample. Even though the subjects they evaluated had clear signs and symptoms of ON, the small sample and the fact they were not CMDs subjects may explain the lower frequency of tinnitus in the group. When a larger sample of CMDs subjects without ON was evaluated researchers[14] reported a frequency of 74% tinnitus, a figure which is very similar to what it was found in the current study,

2.CMDs and ON subjects demonstrated the highest frequencies in those ear symptoms evaluated in the current study as compared to those presenting CMDs but no ON.

If we consider how the presence of ear disorders can be influenced by variables related with anatomic structures in the face, occlusion and TMJ, by the presence of CMDs and dysfunctional masticatory muscles and TMJs, by trauma-related dysfunctional cervical structures, independent of the presence of ON and by the presence of ON itself, then we can better understand the high prevalence of many ear disorders reported by the subgroup with CMDs and ON in the current investigation. These observations and assumptions are strongly supported by many studies reporting that:

1.Dizziness, vertigo and loss of balance occur in 20-58% of individuals who have sustained a whiplash injury[11]

2.There is a correlation between migraine headache and tinnitus[19]

3.Four cranial nerves (V, VIII, IX, X), two upper cervical nerves (C2, C3), and the upper cervical sympathetic ganglion may contribute with the sensory innervations of the ear[20]. Connections of cranial nerves VIII, IX and X and the upper cervical sympathetic ganglion contribute with the development of tinnitus and dizziness[5] and congested nose observed frequently in cases of ON.

4.ON has many migraine-like characteristics and migraine is associated with auditory vestibular dysfunction[6].

5.Severer temporomandibular disorders are associated with greater number of ear complaints[17]

7.The prevalence of tinnitus is very high in patients with tension-type headache and migraine[18]. Tension-type headache is observed frequently in subgroups of subjects presenting with Craniomandibular Disorders.

8.Injury or pathology of the neck may cause malfunction of proprioceptors and joint receptors.There are strong connections between the cervical dorsal roots and the vestibular nuclei thus contributing to the development of dizziness[11].

3.The neurophysiology of ear disorder is as complex as the complexity of the sophisticated an varied innervations to the ears.

The complexity of internal ear structures and their connections with the central nervous system can only be measured not only by the complex neurophysiological and very specific functions they carry out but also by the complex innervations reaching minute anatomical structures within the ear.

Compression and damage to the nerve is by far the most common mechanism and/or etiological factors related to ON and the development of ear disorders. Compression on the nerve, pressure, irritation and/or entrapment are the most common elements causing damage to the GON[21]. The proximity of the muscle tendon membrane,

a swelling lymph node, and/or the pressure from an artery in the peripheral course of the nerve [22] may be one of the commonest mechanisms causing GON pain and dysfunction. Pathological vascular contact of the GON is the mechanism responsible for the development of hemifacial sensory changes, hemifacial pain involving the three distributions of the trigeminal nerve[23] and ear symptoms.

Patients with neck pain, limited range of motion or headache, cervical arthritis, herniated cervical disks and head trauma complain of cervicogenic dizziness associated with flexion extension injuries[11] which may affect the disk, local nerves and other anatomical structures of the first cervical nerves. Pathological vascular contact followed by transient or persistent damage or lesion may result in chronic continuous and strong afferent input in ON[24], thus causing prolonged and severe referred pain, neuropathic and ear disorders. The most common trigger in ON is compression of the GON and the LON, a disorder reported in approximately 90% and 10% of clinical cases respectively[5]. Son and Choi[24] studies reported a clinical case of a lady in her seven decade of life in which the GON was entrapped by pathological contact with the occipital artery. The patient complained of severe right-sided ON and tenderness over the trunk of the right GON with a strong pulsation of the occipital artery branch.

The innervations of the ear is one of the most complex of the human body. There are four cranial nerves and two upper cervical nerves that contribute to sensory innervation of the ear: Nerve V or trigeminal, nerve VII or facial, nerve IX or glossopharyngeal and nerve X or vagus and the upper cervical nerves C2 and C3[20]. **Choi and Jeong[5]** defend the notion that due to connections of the GON with nerves VIII, IX and X cranial nerves and with the upper cervical ganglion, symptoms including tinnitus, dizziness, vision impairment and ocular pain occur relatively frequently.

Vass and associates[25] used iontophoretic injections of biocytin into the trigeminal ganglion of guinea-pigs to assess the innervation of the inner ear vasculature in order to explain clinical disturbances including hearing loss, tinnitus, imbalance and headache. The reported that labeled fibers from the injection site were observed as bundles around the ipsilateral spiral modiolar blood vessels and individual labeled fibers in the interscala septae and ipsilateral stria vascularis. The dark cell region of the cristae ampullaris in the vestibular labyrinth was also labeled. Researchers were then the first investigators that using the method described above documented the trigeminal innervations of the inner ear blood vessels. Based on the studies of Wrisley and colleagues[11], it is now believed that damaged joint receptors in the upper cervical region may lead to abnormal afferent input to the vestibular nucleus thus, resulting in cervicogenic dizziness.

One of the first clinical studies about the relationship between ear symptoms and CMDs was carried out and published by Costen. He strongly believed that CMDs could cause damage to the auriculotemporal nerve or lead to improper adjustment of intratympanic pressure by blocking the Eustachian tube, producing otological symptoms[26]. At the time of publication, Costen had been deeply influenced by previous studies by Monson and Goodfriend who defended the notion that distal and upper displacement of the mandibular condyle could cause impingement of neural elements in the posterior zone of the TMJ. It has also been reported that ear symptoms could be explained on anatomical and neural basis. Nerves to the medial pterygoid muscle, tensor palatine, and tensor veli tympani muscles arise from a common branch of the mandibular nerve. Thus, there exists a neurologic association between the muscles opening the Eustachian tube and the middle ear muscles[7] that may explain ear symptoms associated with CMDs. A similar anatomic and neural theory defends the role played by anatomic structures located in the proximities of the TMJ. According to some researchers, anatomic structures including the disco-malleolar ligament, the chordae tympany nerve and the tympanic artery may be compressed or distended, thus transferring some type of interference to the middle ear and contributing to some ear disorders[27].

Embryology and development

The reported causes for the presence of both CMDs and ear symptoms include a common embryonic origin of the ear and the masseter muscles and the compression of vessels, nerves and ligaments by posteriorly translocated articular heads of the mandible in the middle and inner ear regions[15].

Sensitization is one of the most frequent complex mechanism used to explain persistent and protracted pain and some peripheral symptoms similar to those that occur in neuropathy. In the most simple form, sensitization may be defined as "the phenomenon in which nerves become more sensible to transmit painful information even when the stimulus is of lesser intensity usually associated with more intense and frequent pain". Filatova and coworkers[28] define sensitization as "the process whereby the stimulus needed to generate a response decreases over time , while the amplitude of the response to any given stimulus increases". In line with this simple definition, one investigation**[23]** asserts that sensitization of central nociceptors in the trigeminocervical complex occur in response to strong dural noxious inputs seen for instance in secondary headache syndromes. There is no doubt that increased sensitization of trigeminocervical neurons causes failed integration and spread of nociceptive and other sensory information to different anatomic areas, for instance, the face and the ear. With strong connections and "sensitization" between the cervical receptors and balance

function, it becomes more clear that injury or pathology of the neck may be associated with a sensation or perception of dizziness or disequilibrium**[11].** Pathological vascular contact with the greater occipital nerve may be associated with sensitization and hypersensitivity of the second-order-neurons in the trigeminocervical complex in the C2 dorsal horn that receives significant converging input**[24]**

Chronic noxious afferent input from a compressed nerve like in cases of ON, may lead to central sensitization of the trigeminocervical complex, resulting not only in the development of ear symptoms but also in referred hemifacial pain mediated by the trigeminal nerve**[29].** In many subjects with chronic headache including CMDs and ON, pain is not confined to the known anatomic region but may be observed in areas very distant from the true source of innervation. This phenomenon occurs because of facilitation of central neurons[30]. Clinical manifestations in ON occur by the central sensitization of nociceptive second order neurons induced by strong nociceptive input and a progressively decreased threshold for[30] nociceptive information. It has been reported recently[31] that prolonged nerve contact, pressure and deformation of the nerve, may cause superficial cutaneous sensitization which is currently considered an indicator of central sensitization and longer duration of ON headache[31].

It is not necessary for an anatomic structure to present nerve damage to facilitate the development of central and peripheral sensitization. In the case of three common headaches, TTH, migraine and ON, several clinical observations indicate that the development of sensitization at the level of the spinal dorsal horn and the trigeminal subnucleus caudalis is the result of prolonged continuous nociceptive input from pericranial myofascial tissues[28], and from a damaged and/or entrapped GON.

Repeated episodes of migraine, tension-type headache and/or ON attacks and severe toothache, may have over the years, cumulative adverse consequences on the function of the trigeminovascular pathway[32], including the presence of central sensitization characterized by a lower threshold for pain and readiness to respond to a minor nociceptive stimulation. It has been reported recently[33] that subjects who suffer from chronic OFP demonstrate a generalized reduction in pain thresholds not only in facial anatomic sites but also in areas not involved by pathology, for instance, the upper trapezius muscle[33]. It seems that the transition from an acute TMD state to a chronic one is accompanied by a reduction in the pain threshold restricted to the orofacial region[34].

Convergence

ON is a neuropathic pain disorder usually presenting in the chronic form in which the phenomena of convergence and central sensitization are observed more easily. Following a number of neurophysiological and clinical studies, it has become more clear in the last two decades, that the subnucleus caudalis and C2 and C3 constitute anatomic areas where nociceptive information associated with different types of headaches, including TTH, migraine and ON, converge. Thus, chronic and recalcitrant pain, patient´s poor response to pharmacological treatment and physical/emotional impairment, allows the astute researcher to more clearly observe the phenomena of sensitization and convergence of afferent nociceptive information.

Convergence of nociceptive afferent neurons and sensitization of neurons in the trigeminocervical complex have enormous clinical importance in the interpretation of some common phenomena including chronic pain, patient´s resistance to respond favorably to the use of common analgesics, longer duration and more intense pain. These considerations are echoed by the investigation carried out by Lee and Son[23] reporting that convergence of nociceptive afferents and sensitization of trigeminocervical neurons have important clinical correlates including hypersensitivity, spread and referral of **pain[23]** frequently observed in patients with chronic headache, facial paraesthesias and ear disorders. In this regard, it has been reported that cervicogenic headache including ON, which involves pain referral from cervical structures to other areas of the head and face, is produced by convergent excitation evoked by stimulation of C1,C2,C3 and trigeminal nerve resulting in increased excitability of second order neurons in the trigeminocervical **complex[5]**

Trigeminocervical complex or simply TCC is a population of neurons in the C2 dorsal horn that receives convergent input from facial skin corresponding to the trigeminal nerve and cervical structures innervated by cervical roots C1, C2, C3[23]. Convergence and sensitization are parallel neurophysiological phenomena that occur when the information converging to such population of neurons is intense, frequent and disturbing in such a way that the information cannot be integrated properly and relied directly to higher centers in the CNS. The convergence of upper cervical nerves and trigeminal sensory afferent in the trigeminocervical complex has been well documented in the last two decades and there is neurophysiological coupling between craniofacial structures and the cervical system**[35].** It has been reported that the close association between sensory and motor fibers of some spinal nerves and sensory fibers or adjacent nuclei of the TN allows for exchange of sensory and motor information from the trapezius, sternomastoid and other cervical muscles to converge in the trigeminal and cervical nucleus resulting in the referral of pain from cervical and from masticatory structures including face and teeth**[36]. Thus, not** only those neurons associated with TTH, Migraine and ON pain may be sensitized with time, but both convergence and sensitization also

explains the transformation of an apparently innocuous trigger point in a chronic one capable of producing pain distant from the original source of pain, that is, the trigger point.

V. Conclusion

Dizziness, vertigo, ear stuffiness, hearing deficiency and tinnitus were observed much more frequently in the CMDs and ON subgroup as compared to the CMDs No ON and to the subgroup with no CMDs and No ON. Thus, in CMDs and BB individuals the presence of ON contributes not only to a higher frequency of ear disturbances but to referred pain and paraesthesia to the face and other structures innervated by the trigeminal nerve. Additional studies are now needed using different research models in order to investigate the mechanism responsible for the development of ear disorders associated with ON.

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Table 1: Social and demographic data in subjects presenting with CMDs + ON (n=80), Controls CMDs No ON (n=90) and Controls no CMDs no ON subjects (n=30).

	CMDs + ON n=90	CMDs No ON n-80	No CMDs No ON n=30
AGE			
Mean	40,5	32,0	44,5*
SD	10,1	12,4	14,1
Range	15—75	17--73	17--73
GENRE			
Females	87=96,7%	73=91,3%	21=70%
Males	3=3,3%	7=8,7%	9=30%
Totals	90=100%	80=100%	30=100%

*****Kruskal-Wallis statistis (p<0,0001: CMDs + ON versus CMDs No ON (-<0,001); CMDs + ON versus No CMDs No ON (p<0,01); CMDs No ON versus No CMDs No ON (p>0,05).

Table 2: Frequencies of Dizziness, vertigo, ear stuffiness, ear deficiency and tinnitus in subjects with CMDs + ON (n=90), CMDs No ON (n=80) and Controls No CMDs, No ON (n=30).

	SUBGROUPS					
	CMDs + ON n=90		CMDs No ON n=80		No CMDs No ON n=30	
Ear Disorders	n	%	n	%	n	%
Dizziness						
Yes	76	84,4	32	40	7	23,3
No	14	15,6	48	60	23	76,7
Total	90	100	80	100	30	100
Vertigo						
Yes	31	34,4	9	11,3	1	3,3
No	59	65,6	71	88,7	29	92,7
Total	90	100	80	100	30	100
Ear Stuffinness						
Yes	54	60	28	35	5	16,7
No	36	40	52	65	25	83,3
Total	90	100	80	100	30	100
Ear deficiency						
Yes	49	54,4	18	22,5	1	3,3
No	41	45,6	62	77,5	29	96,7
Total	90	100	80	100	30	100
Tinnitus						
Yes	62	68,9	20	25	5	16,7
No	28	31,1	60	75	25	83,3
Total	90	100	100	100	30	100

Table 3: Data about significance when comparing frequencies of ear symptoms in different subgroups

SYMPTOMS	**SUBGROUPS**	**p-value**	**Significant?**
Dizziness	CMDs + ON vs CMDs No ON	<0,0001	Yes
	CMDs + ON vs Control	<0,0001	Yes
	CMDs No ON vs Control	0,12	No
Vertigo	CMDs + ON vs CMDs No ON	<0,0005	Yes
	CMDs + ON vs Control	<0,0006	Yes
	CMDs No ON vs Control	0,28	No
Ear stuffiness	CMDs + ON vs CMDs No ON	<0,001	Yes
	CMDs + ON vs Controls	<0,0001	Yes
	CMDs No ON vs Controls	O,06	No
Ear deficiency	CMDs + ON vs CMDs No ON	< 0,0001	Yes
	CMDs + ON vs Controls	<0,0001	Yes
	CMDs No ON vs Control	<0,02	Yes
Tinnitus	CMDs + ON vs CMDs No ON	<0,0001	Yes
	CMDs + ON vs Controls	<0,0001	Yes

	CMDs No ON vs Controls	0,44	No

Table 4: Use of X-squared for independence and for trends regarding every symptom in three different subgroups from the least or no disordered to the more severely disordered one. Controls=30, CMDs No ON=80, CMDs + ON=90.

	Control	CMDs No ON	CMDs + ON	CHI-SQUARED:	
SYMPTOM	n %	n %	n %	Independence	Trends
Dizziness	7/30=23,3	32/80=42	76/90=84,4	p<0,0001	p<0,0001
Vertigo	1/30=3,3	9/80=11,3	31/90=34,4	p<0,0001	p<0,0001
Ear Stuffiness	5/30=16,7	28/80=35	54/90=60	p<0,0001	p<0,0001
Hearing deficiency	1/30=3,3	18/80=22,5	49/90=54,5	p<0,0001	p<0,0001
Tinnitus	5/25=16,7	20/80=25	62/90=68,9	p<0,0001	p<0,0001

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