Dental occlusion as one cause of tinnitus

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A B S T R A C T

There is large support in literature linking tinnitus to dental occlusion and temporomandibular joint disorders (TMD). However, there is no model to explain such a link. This hypothesis explains how the fusimotor system of the muscles innervated by the trigeminal motor nucleus is affected by inadequacies in the occlusion of the teeth that cause changes in posture and movement of the mandible. Reptile to mammal evolution shows that stomatognathic structures underwent changes related to mastication. Among several changes, there was the appearance of a new articulation between the mandible and skull: the temporomandibular joint. The bones of the old reptile joint, quadrate-articular, have detached from the mandible and are part of the middle ear bone chain. The former becomes the incus and the latter the malleus. This bone change also carried the tensor tympani and its trigeminal motor innervation. Inadequate occlusal contacts give rise to an adapted function of the malleus. This bone change also carried the tensor tympani and its trigeminal motor innervation. Inadequate occlusal contacts give rise to an adapted function of the malleus. This hypothesis explains the association of symptoms involving mandibular musculature and hearing symptoms caused by the tensor tympani. The fusimotor system hypothesis is able to explain all events related to the symptoms and helps to establish a correct diagnosis for certain types of hearing disorders.

Introduction

Tinnitus is related to various etiologies, and therefore, it would be appropriate to consider it as a symptom rather than as a disease. The determination of the cause would lead to more effective treatments. An additional problem is the simultaneous occurrence of diverse causes, which can lead to failure of treatment.

One of the causes, already well reported in literature, is related to the tensor tympani maintaining tonic activity, altering its function and causing the hearing symptoms, also known as tonic tensor tympani syndrome [1,2].

There are many articles and theories linking tinnitus to problems with dental occlusion and temporomandibular joint (TMJ), but there is no model to explain how this occurs.

In part, this difficulty originates in the deficient concept for temporomandibular disorders (TMD) that relates to the anatomic site instead of the etiology of a disease. Thus, the diagnosis TMD listed all diseases involving TMJ – with different etiologies –, as a single issue that occurs in the same anatomic structure [3]. Therefore, it has been difficult to make the correct diagnosis of changes that relate to tinnitus.

However, it is observed that, when there is inadequacy in the intercuspation of the teeth, there are disturbed mandibular movements and posture. The adapted mandibular function demands more muscular activity and the most noticeable effect is a state of hypertonia/hypo-kinesia, which seems to affect all muscles innervated by the trigeminal nerve.

Hypothesis

The inadequacies of dental occlusion that affect the mandibular function cause a reflex response in the fusimotor system involving all muscles innervated by the trigeminal motor nucleus, including the tensor tympani. This hypothesis explains the association of symptoms involving mandibular musculature and hearing symptoms caused by the tensor tympani tonic condition.

Evolution of viscerocranium

Reptile to mammal evolution shows that stomatognathic structures underwent changes related to mastication. Very concisely, there was a decrease in size of the facial bones, reduction of the number and specialization of the teeth that are inserted in sockets in the maxillary
bones and the appearance of a secondary palate. A new joint of the mandible with the skull also appeared, between the dentary bone of the mandible and the squamous of the skull: the temporomandibular joint [4].

In reptiles, the mandible is composed of multiple bones and there is a single bone in the ear. In the mammalian jaw only the dentary bone was left. The bones of the old joint detached from the mandible and are part of the middle ear bone chain. The articular (mandible) became the malleus and the quadrate (cranium) became the incus [5,6].

Interestingly, cases of hemifacial microsomia, a craniofacial developmental anomaly, show this relationship between the mandible and the middle ear, which could affect the joint, pinna, musculature, the external auditory canal and the ossicular chain of the middle ear [7]. Eventually, some clinical findings about objective tinnitus show an unusual connection between the mandibular fossa and middle ear space [8].

In this process, the muscle that was attached to this part of the mandible, accompanied the bone change and maintained its trigeminal motor innervation. It is estimated that the tensor tympani is derived from the ventral portion of the medial pterygoid musculature [9].

Another important modification, related to evolution of the mandible, was the change in the action of the musculature. In reptiles, mouth opening is performed by the depressor mandibulæ muscle, whose action is posterior to the quadrate-articular joint.

In humans, mouth opening is done by the muscles between the hyoid and mandible, when the hyoid is anchored by infrahyoid muscles. To swallow, however, the mandibular elevator muscles anchor the mandible in occlusion and the muscles between the hyoid and the mandible move the hyoid for deglutition. These muscle groups attached to the mandible and hyoid also form part of the opposition to cervical musculature to maintain head posture [10].

The mandible, during the masticatory cycle, presents two types of movement: opening and closing the mouth and lateral movement. These movements are possibly due to TMJ configuration, which should be seen as two partial joints inside the same capsule and the articular disc as a “mobile fossa” [11]. Thus, the movement of the lower compartment of TMJ (between the condyle and disc) performs mouth closing and teeth clenching, whatever the position of the mandible. The upper compartment (between the disc and temporal bone) performs the horizontal movement of the jaw (Fig. 1).

This demands remarkable specialization from the mandibular musculature. The temporal muscle [12], for instance, has fibers on several angles to the occlusal plane, where it acts (Fig. 2). In its anterior portion, the fibers are oriented at 90 degrees to the occlusal plane and act on the TMJ lower compartment; the muscle is bulky in this area and the fibers are mostly composed of strength dedicated fibers [13,14].

The middle portion of the muscle has its fibers aimed approximately at 45 degrees and shows a mixture of strength and movement devoted fibers, and act on both TMJ compartments. In the posterior part, fibers are almost parallel to the occlusal plane and only perform on the upper compartment of the TMJ. Actually, these fibers should be categorized as abductors of the condyle and are antagonist to the lateral pterygoid muscle, which should be labeled as the condyle adductor. Masseter and medial pterygoid muscles also have similar fiber arrangement to perform strength and movement, but arranged in superposed layers.

These muscles contain a large amount of muscle spindles, indicating that they need outstanding proprioceptive control, because the masticatory cycle demands refined control of movement and force applied to the mandible [15,16]. The tensor tympani also has muscle spindles, indicating that it can maintain prolonged tonic contraction [17,18].

Neuromotor control of mandibular function

Masticatory movements can be obtained by stimuli of several brain areas, which demonstrate a complex motor activity. Hence, only considerations for the demonstration of the hypothesis will be considered in this section.

Chewing involves three activities: volitional, automatic and peripheral. It first begins voluntarily when a food is placed in the mouth. After this, the individual chews unconsciously, unless some event, such as taste or hardness and texture of the food, causes the control to return to the voluntary mode for decision making. A similar situation occurs in walking when the individual has to overcome an obstacle, like stairs, returning control to volitional mode.

At the same time, peripheral mechanoreceptors continuously
influence masticatory dynamics, since it is observed that the first masticatory cycles have greater force application and are more irregular; at the last cycles, before swallowing, these cycles present a more regular pattern, indicating less influence of the peripheral impulses on motor activity. Thus, mandibular function is reflex and continuously influenced by peripheral receptors [19].

Peripheral impulses, which influence mandibular motor activity, originate in three receptor populations: in the periodontium and TMJ capsule, which reflexively control mandibular movements and tone, and in muscle spindles.

The mechanoreceptors located in the periodontium are of two groups. One part has the neurons located in the supratrigeminal nucleus and the other in the trigeminal ganglion [20]. The activity of these receptors is associated with reflexes related to the application of muscle force during mastication, and to the proprioception of the intercuspal position of the teeth [21,22].

Several studies have demonstrated neuroplasticity in response to occlusal events [23]. There is neuromotor learning, due to the occlusal information carried by the trigeminal nerve. A common example of this fact is the sensation that many patients experience after a new dental restoration, which disappears in few days, and as has been observed in cases of rehabilitation of edentulous patients with implants [24]. Other studies have also shown that there is a change in the function of the basal ganglia and alteration in the neurotransmitters balance resulting from these long lasting occlusal events, especially those that alter the mandibular function [25].

The occlusal inadequacies are dental contacts that guide the mandible to an adapted position, where the best intercuspal position of the teeth occurs, which is not always the ideal joint position and is called centric occlusion [3]. This phenomenon is governed by the mechanoreceptors of the periodontium, which are able to direct the mandible for the best relation of the dental cusps. These inadequacies may result from deficient contouring of restorations, tooth extraction, malocclusion and others. Once installed, they affect the motor performance of the mandibular tasks while they are present.

Furthermore, in response to the forces generated by the musculature, when the mandible is held in an intercuspal position, functional adaptation occurs on stomatognathic structures, such as enamel wear, remodeling of the dental sockets to move the involved teeth, remodeling of the TMJ, among others, to allow the execution of the functions [3]. This change to an adapted mandibular position, which is different from the ideal TMJ position, will bring consequences to motor task execution.

Mechanoreceptors from TMJ capsule [26] detect the mandibular position change and perform a reflex compensatory adjustment of the muscle tone. The effects of these occlusal events are immediate, not only when it starts but also when the occlusal inadequacies are removed [27-32].

The mandibular movements of the masticatory cycle require a refined control of contractions of the involved musculature, alternating between application of force and execution of movements. These events seem to be largely regulated by the basal ganglia, which generate output impulses to the brainstem, to perform the two types of muscular action: force and movement [33,34].

Changes in the function of the basal ganglia show two major groups of neuromotor disturbances: hyperkinesia (with hypotonia) and hypertonias (with hypokinesia), as observed in Huntington's chorea and Parkinson's disease [35]. Clinical observations of altered mandibular function, consequent of occlusal events, have similarity with these two situations. On the other hand, changes in mandibular function are results of long lasting peripheral impulses and not the pathology of the basal ganglia. So it is a condition that is not constant and varies its appearance throughout the day.

As a consequence of occlusal inadequacies, two types of responses in the mandibular musculature are observed: hyperkinesia or hypertonias, with a wide range of manifestations between these two extremes. However, unlike basal ganglia pathologies, it appears that these events occur in response to long lasting peripheral impulses [25], similar to the effect that a pebble in an individual's shoe would have on walking for a long distance.

Currently the diagnosis of bruxism is used for events of mandibular excessive movement, without application of force (Fig. 3) and it is observed a clinical manifestation of hyperkinesia that causes excessive tooth wear, but without alterations in periodontium and TMJ, because there is no application of force. Clearly, there is discussion about the role of occlusion in this clinical picture, but in some cases the correction of occlusion stops the manifestation.

The most common mode, in cases with occlusal inadequacy, however, is hypertonias. These are cases that present intense application of force with destructive consequences on the teeth, periodontium and TMJ (Fig. 3). These cases present an increased musculature volume, but with a loss of functional efficiency [36,37]. Women show frequent muscle pain, which makes them more likely to be affected when using the inadequate diagnosis of TMD. Testosterone gives men greater capability to support tone request, but it results in greater damage to the dental structures, as a consequence [3].

Hypertonic condition of the mandibular muscles causes a decrease in amplitude and speed of the mandible movements [38,39] and incoordination with other stomatognathic muscle groups and the individual presents tongue biting while chewing or cheek biting when sleeping. The complaint that there are errors in the execution of speech when the individual speaks fast is also very common and the occurrence of lateral movement of the mandible in speech.

Patients that present these symptoms of hypertonias complain about hearing symptoms as well, described as ear pain, tinnitus, blocked ear sensation and sudden hearing loss. These occurrences are often episodic rather than constant, and frequently appear at the same time as the other symptoms.

In the context of this hypothesis, the contraction of the tensor tympani presents the characteristics reported by patients, as described in a case of voluntary contraction of the tensor tympani [40]. On the
other hand, hypertonia of the mandibular musculature has a longer duration and seems to be due to excitation of the fusimotor system [41–43].

Clinical characteristics

The hypertonic condition of the mandibular muscles presents a group of signs and symptoms, as summarized in Table 1. One of the most interesting clinical features is muscle pain by palpation, which may be very intense, but there is no pain when moving the mandible. Palpation pain disappears immediately when performed during bimanual manipulation of the mandible [28,44].

The muscle pain originating from the fusimotor system has, as a main characteristic, regularity. It will always occur in the dermatome innervated by the trigeminal mandibular branch, which carries the motor fibers, being more common in the temporal region, and is sometimes reported as ear pain. Patients describe the pain as tightening, with features similar to cramp. The intensity and duration of pain vary little in the same individual. Frequency is also regular and sometimes associated with chewing, and very common in the morning, indicating tonic muscle activity during sleep. Hypertonic muscle pain, however, is subjective and is more common in women, and therefore, for a more objective diagnosis, bilateral palpation of the mandibular musculature is better, as shown in Fig. 4.

Temporal muscle is palpated in two areas: in the region of the temporal fossa, the superior temporal line is felt near the frontozygomatic suture (T1) and at the coronoid suture, when it crosses the superior temporal line (T2). The former is more related to movement problems, there is hypertonia when it is distinctly possible to feel the tendons of the masseter. When there is hypertonia, it is not possible to feel the internal face of the jaw.

Although innervated by the accessory nerve, the cranial portion of the sternocleidomastoid is very significant in issues concerning lateral movement of the mandible. The involvement is always unilateral and coincident with the affected side of hypertonic jaw muscle fibers dedicated to movement. When this finding is present, hypertonia of the descending part of the contralateral trapezius muscle is common.

Patients with hypertonia due to occlusal inadequacies show poor neuromotor control of mandibular movements. This is especially noted when the patient is asked to make lateral jaw movement touching the teeth. When doing this movement it is common to observe that the patient moves the lips, eyes, and even turns the head in the same direction. The report of tongue biting while chewing and cheek biting during sleep is also very common. These are non-physiological events that surpass protective reflex circuits. In addition, the report of difficulties eating hard food and making speech mistakes when the person talks fast is common.

Conclusions

The model presented in this hypothesis explains the signs and symptoms that associate the hypertonia of the mandibular musculature with the alteration of the function of the tensor tympani related to tinnitus and other symptoms.

The fusimotor system hypothesis is able to explain all events related to the problems and helps to establish a correct diagnosis for certain types of hearing disorders.

Declaration of Competing Interest

The author hereby state that have no known affiliations that present a conflict of interest.
References


