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Tinnitus and a Linked Stomatognathic System

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1. Introduction

Most patients associate tinnitus (false sound perception) with desperation and a Sui generis awareness. Normally, it is accompanied by another otic symptoms that worsen the aversive experience. For some individuals, tinnitus occupies the majority of their attention cosmos by negative cognitive-emotional conditioning. People that suffer this condition have a considerable work-familiar-individual challenge. It also, could be shared with additional varied otic and cranial symptoms like otic fullness, otalgia, hearing loss, dizziness, and a diffuse craniofacial and stomatognathic (chewing machinery) pain with a diffuse presentation (headaches, myalgia arthralgia and cervicalgia).¹

Traditionally, tinnitus origin has been linked to conductive, sensorial or both mixed otic origins. A nervous dysfunction explanation has more adherences in the health community. With this in mind, a sensorineuronal tinnitus dysfunction has been understood as triggered by sound energy transmission complications with peripheral or central nerve damage origins. Lately, the health community has commenced understanding how a stomatognathic's (conductive) scenario has shown evidence of their links.

In this chapter the reader is invited to ingoing into an stimulating and almost new form of looking tinnitus aetiology (among other otic referred symptoms) based in the research with some biological models that permit to analyse a variety of pathophysiological tinnitus associations with the chewing apparatus.

In this sense, it must be first comprehended that the stomatognathic structure must be fatigued and in a dysfunctional state to produce tinnitus and other referred otic symptoms. This state in which the stomatognathic musculoskeletal system is hyper-functional, tender and exhausted is known as temporomandibular disorders (TMD). TMD can generate referred craniofacial symptomatology (where origin differed from its real location) that involves not only the auditive system and take influence in cervical and cranial diffuse discomfort too.

Thirty years after Costen² (1934) pioneering ideas about otic referred symptoms starting from the stomatognathic system, Myrhaug³ affirmed that the middle ear belong to the chewing system although it served the auditory system. Unfortunately for these otolaryngologists this logic was advanced for their time and was considered almost a heretic concept receiving an unjust opposition by their own medical community. Trying to rescue these clever researchers contributions some analysis about the otic/chewing linking connections will be developed.

There are fourteen possible stomatognathic models (among embryological, musculoskeletal, ligamental, vascular and neural) that offer adequate explanations for tinnitus and other

linked otic-cranio-cervico-facial referred symptoms.^{4,5,6} This analysis equally tries to support actual evidence about otic referred symptoms reliefs by stomatognathic therapeutic.⁷ Nowadays, there is no doubt that a multidisciplinary approach (otolaryngology, odontology, neurology including another health disciplines) is essential for an assertive diagnosis, treatment and prognosis of this particular symptomatology.

2. Dysfunctional stomatognathic system and Bruxism

Temporomandibular disorders (TMD) consist of musculoskeletal pain conditions, characterized by pain in the temporomandibular joint (TMJ) and/or mastication muscles. This condition involves a wide range of craniofacial conditions having multiple origins that produces a large variety of non-objective signs and symptoms.⁸ These could be primary, referred or combined from cervical muscles and associated cranial structures and appear similarly in adults, as well as, in children ^{9,10}. The prevalence of TMD is 1.5 to 4 times more common in women than in men.¹¹ After low back pain, TMD occupied the second place of musculoskeletal disability condition.

The etiology of the TMD can be isolated or the combination of macrotrauma and microtrauma (Bruxism) and this last appears to play a significant role in TMD and craniofacial referred symptoms.¹² Bruxism is an intense and subconscious rhythmic motor activity of non-functional teeth grinding and clenching. Besides exceeding the structural tolerance of the biological tissues, it triggers a cascade of primary and referred symptomatology (Figure 1).^{13,14,15,16,17}

Fig. 1. Grinding effects on teeth due to bruxism in a 45-year male patient. Consequently with his deteriorate teeth appearance; an advanced primary and referred musculoskeletal symptomatology is manifest. Photography use authorized to the author (Patient's record).

3. Stomatognathic referred otic symptoms

Since almost a century, health literature has closely perceived otic symptoms and other craniofacial complaints in TMD. However, there is little evidence for an association between the two. An integrated biological basis for otic symptoms in TMD is presented from both anatomical and physiological points of view. To accomplish a central-peripheral mechanisms involved, they are discussed along the chapter. Basic sciences let integrate diverse point of views in the understanding of common symptoms. This matter deals with perspectives of otic symptoms triggered or exacerbated by atypical stomatognathic dynamics.^{18,19}

Otic symptoms include otalgia, tinnitus, vertigo-dizziness, subjective hearing loss, and otic fullness.^{20,21,22,23,24,25} Such otic symptoms evidently can be originated in the auditory system (as a primary symptom) but are also habitually a symptom of an associated neighbored stomatognathic dysfunction (secondary or referred symptom).

Otic symptoms related to the masticatory system can be found in both adult and paediatric populations.²⁶ Monson and Wright (1920) related the position of the TMJ to hearing impairment and in 1925, Decker also related otic symptoms to stomatognathic TMJ. Costen in 1934 associated otic symptoms later named Costen's syndrome.^{27,28} Although the literature supports a connection between otic symptoms and TMD; it is still an open question as to whether this association is causal or accidental.²⁹ A screening search permit to observe apparent correlations between otic symptoms and TMD in some studies from 1933 to 2011 (Table 1).^{30,31,32,33,34,35,36,37,38,39,40,41,42,43,44,45,46,47,48,49,50,51,52,53,54,55,56,57,58,59,60,61,62,63,64,65,66} Overall, 12,732 TMD patients were mentioned in n=54 articles. Otalgia was present in 50.9 % of the patients (n=44), tinnitus in 39.1 % (n=47), otic fullness in 44 % (n=24), vertigo in 30.3 % (n=39), and hearing loss in 24.4 % (n=28). Salvetti et al.⁶⁷ found that the prevalence of otic symptoms in the general population varied from 10% to 31% and increase to 85% in TMD patients.

Taking this in mind, a relationship between otic symptoms and TMD have been found since the beginning of the last century, with some interesting findings: Kutila et al.⁴⁶ found a prevalence of tinnitus (12-17%), otalgia (12-16%), and otic fullness (5-9%) in subjects with TMD. According to Rubinstein⁶⁸ 33-67% of TMD patients reported tinnitus. Gelb et al.²² found that 42% of patients with TMD reported tinnitus, 35% reported otalgia, 18% reported dizziness, and 14% reported hearing loss. Besides, Cooper et al.²¹ found 40-70% of TMD patients experiencing dizziness and 5-40% feeling vertigo. Lam et al.⁶⁹ noted in a retrospective work that 26.4% of TMD patients had otic symptoms with a prevalence of tinnitus higher than that found in the general population. Bjorne and Agerberg⁷⁰ proved that Meniere's disease (understood as hearing loss, frequently associated with tinnitus, otic fullness, and paroxistic vertigo) is strongly related to TMD symptoms.

Recently, the somatic modulation capacity on tinnitus has served to evidence a potential connection. Lockwood et al.⁷¹ found that up to 75% of subjects were able to vary tinnitus intensity by clenching the jaw, increasing digital cranial muscle pressure, and moving the eyes (gaze-evoked tinnitus). Vernon et al. (⁷²) although expressed no association between tinnitus from TMJ origins; observed increased intensity of tinnitus when jaw-clenching or other jaw movements occurred. Levine⁷³ found that tinnitus can be modulated by isometric oro-facial and cranio-cervical manipulations.

In addition to the pain dimension, Hazell⁷⁴ reported 39% of patients suffering from tinnitus with frequent tension headaches with fatigue and muscle soreness in the facial and

Authors	N° TMD patients	% otalgia	% tinnitus	% vertigo	% hearing loss	% otic fullness
Goodfriend 1933 (30) U	91	3	14	5	26	—
Costen 1934 (2) U	400	38	13	12	13	6
Gelb-Arnold 1959 (24) U	100	13	27	5	—	—
Kelly-Goodfriend 1960 (51) U	102	—	—	75,5	—	—
Kelly-Goodfriend 1964 (52) U	105	25	44	100	44	30,4
Myrhaug 1964 (3) U	1391	21	32	28	32	—
Dolowitz <i>et al.</i> , 1964 (115) U	66	100	43	5	38	48
Gelb <i>et al.</i> , 1967 (23) U	742	36	40	20	15	—
Bernstein <i>et al.</i> , 1969 (20) U	86	93	42	14	33	62
Sharav <i>et al.</i> , 1978 (118) U	42	—	—	23	—	—
Principato-Barwell 1978 (50) U	25	100	44	44	—	—
Koskinen <i>et al.</i> , 1980 (45) U	47	47	20	26	24	26
Brookes <i>et al.</i> , 1980 (33) U	45	82	76	33	80	62
Carlsson <i>et al.</i> 1982 (37) ¥	350	38	15	17	15	—
Gelb-Bernstein 1983 (41) U	1142	—	41,5	20,6	15,4	—
Gelb-Bernstein 1983 (42) U	200	—	36	40	24,5	48
Fricton <i>et al.</i> , 1985 (40) U	164	42	42	23	17,7	—
Cooper <i>et al.</i> , 1986 (22) U	476	50	36	40	38	—
Wedel-Carlsson 1986 (53) U	148	—	14	15	14	—
Bush 1986 (35) U	105	82	33	—	—	—
Bush 1987 (36) U	35	—	33	—	—	—
Williamson 1990 (153) U	25	—	—	44	—	—
Henderson <i>et al.</i> , 1992 (119) U	21	85,7	90,5	76,1	66,6	90,5
Chole-Parker 1992 (38) ¥	338	100	59	70	—	—
Vernon <i>et al.</i> , 1992 (72) ¥	69	45	100	—	—	75
Cooper <i>et al.</i> , 1993 (21) U	996	63	51	41	25	30
Ogutcen-Toller <i>et al.</i> , 1993 (114) U	57	40	17	8,7	26	5
Ciancaglini <i>et al.</i> , 1994 (39) U	797	1,6	6,4	1,6	7,6	—
Ren-Isberg 1995 (169) ¥	53	98,1	100	54,7	—	92,5
Parker-Chole 1995 (49) ¥	338	100	59	70	—	—
Keersmaeckers <i>et al.</i> , 1996 (25) U	400	42	—	—	—	—
Manni <i>et al.</i> , 1996 (48) U	53	33,9	22,6	33,9	7,5	41,5
Wright <i>et al.</i> , 1997 (170) U	93	38	38	—	—	—
Karjalainen <i>et al.</i> , 1997 (34) ¥	63	13	—	3	—	—
Luz <i>et al.</i> , 1997 (47) U	894	10,8	—	3,1	—	—
Watanabe <i>et al.</i> , 1998 (55) U	143	—	15	10	—	11
de Felicio <i>et al.</i> , 1999 (58) ¥	30	53,3	66,6	20	20	76,6
Kuttila <i>et al.</i> , 1999 (46) U	411	14	15	—	—	7
D'Antonio <i>et al.</i> , 2000 (57) U	90	100	51,1	31,1	—	—
Wright <i>et al.</i> , 2000 (56) U	15	15	14	11	—	—
Bruto <i>et al.</i> , 2000 (65) U	40	75	17,5	—	15	17,5
Lam <i>et al.</i> , 2001 (69) U	470	32	26	18	12	—
Pascoal <i>et al</i> 2001 (66) U	126	50	50	34	10	52
Peroz 2001 (85) U	221	37	3,8	—	—	—
Tuz <i>et al</i> 2003 (54) U	200	57,2	48,3	42,7	29,4	—
de Felicio <i>et al</i> 2004 (59) U	27	59,2	74	—	—	74,1
Sobhy <i>et al</i> 2004 (86) U	30	56,7	20	—	—	—
Bernhardt <i>et al.</i> , 2004 (18) ¥	30	—	30	—	19	—
Kaygusuz <i>et al.</i> 2006 (44)	44	31,8	18,2	13,6	6,8	13,6
Abou-Atme <i>et al.</i> 2006 (32)	530	19,6	—	—	—	26,7
De Felicio <i>et al.</i> , 2008 (60) ¥	20	65	60	—	—	90
De Felicio <i>et al.</i> , 2010 (61) ¥	30	13,4	8,8	—	—	13,5
Pekkan <i>et al.</i> , 2010 (63) ¥	25	72	52	20	8	56
Bernhardt <i>et al.</i> , 2011 (64) ¥	191	—	12,6	—	—	—
Media		50,9	39,1	30,3	24,4	44,0
Total	12732					
n	54	44	47	39	28	24

U Clinical not controlled studies
¥ Epidemiological / Controlled studies

Table 1. Different TMD patient populations suffering referred otic symptoms.

masticatory muscles. Kuttilla et al.⁷⁵ showed how otic fullness, earaches, shoulder and TMD pain are risk factors for recurrent tinnitus.

TMD appear to play a relevant participation role in otic referred symptoms without misestimating another potential origins within and outside the auditory system, including viral cranial neuropathy, intracranial vascular anomalies, cerebrovascular disease, cardiovascular disease, mediastinal tumours, Meniere's disease, ear and head trauma, chronic myringitis, impacted cerumen, otic infections, ototoxic drugs, acoustic neuroma, multiple sclerosis, neuroplastic changes, noise exposure, otosclerosis, and presbycusis. In this sense, a multifactorial cause with wide cranio-cervico-facial structure participation should carefully be considered in a teamwork effort with an exclusionary diagnostic approach.

How TMD affect the auditory system must be understood in different evidence levels. A morphologic-embryologic-neurophysiologic way could help to understand the pathophysiology of these clinical events. However, clinicians should be made aware that a single cause of otic symptoms competes with a multifactorial origin. Over the last few years, the most acknowledged clarifying models have focused on central nervous system (CNS) networks. However, peripherally structures could be also an important issue to view regarding the aetiology of otic referred symptoms. An integral model concerning neurological, anatomical and physiological perspectives offer a wider angle for the multiple and dynamic enlightenments linking TMD and otic symptoms.

4. Neural explanations

Some otic experiences like tinnitus, hearing loss, vertigo, and otalgia can be explained from a multidimensional neurological point of view. Most investigators have focused on tinnitus by the multiple CNS interconnections and plasticity that actually occur.

4.1 First neural exploration

Symptoms such as vertigo, tinnitus, and subjective hearing loss can result from an auditory innervation pattern involving the trigeminal nerve. Vass et al.⁷⁶ found that trigeminal vascular system innervation in guinea pigs controls cochlear and vestibular labyrinth function. This plays an important role in regulating and balancing cochlear vascular tone and vestibular labyrinth channel and may be responsible for the symptomatic complexity of some cochlear diseases related to inner ear blood flow. Trigeminal ophthalmic fibre projection to the cochlea through the basilar and anterior inferior cerebellar arteries may play an important role in vascular tone in quick and vasodilatatory responses to intense noise. Inner ear diseases that produce otic symptoms such as sudden hearing loss, vertigo, and tinnitus can originate from reduced cochlear blood flow due to the presence of abnormal activity in the trigeminal ganglion, which is possible in patients with herpes zoster, migraines and TMD. A parallel discovery by Shore et al.⁷⁷ found that the trigeminal ganglion innervates and modulates the vascular supply of the ventral and dorsal portions of the cochlear nucleus and the superior olivary complex.

4.2 Second neural exploration

Levine et al.⁷⁸ suggested that a somatic auditory perception interference in the dorsal cochlear nucleus (DCN) and ventral cochlear nucleus by trigeminal innervation and named it "somatic tinnitus".^{78,79} Shore et al.⁸⁰ found this connectivity in the superior olivary

complex (involved in sound localisation and centrifuge reflex). Such trigeminal-auditory neural communication may cause a significant impact on the neurons from this nucleus, interfering with the auditory pathway (tinnitus), which is directed towards the auditory cortex in the presence of constant peripheral somatic signals from ophthalmic and mandibular trigeminal peripheral innervation. Young et al.⁸¹ affirm that such a stimulus is so strong that it can interfere with moderate-level acoustic stimuli.⁸² The perception originates from mechano-sensory and in a minor amount from nociceptive spinal and cranial nerve impulses in the caudal spinal nucleus and is modulated by muscle fatigue in the head and neck areas. CNS interaction between the somatosensory system and multilevel auditory tracts (including the inferior colliculus and extralemiscal auditory pathway) is a fundamental property of the auditory system.⁸³ Trigeminal somatosensory input to the inferior colliculus employs the same DCN pattern (bimodal inhibition-excitation synapses) as a reflection of its multimodal integration. Somatosensory input in TMD may explain the origin of otic symptoms such as subjective hearing loss and tinnitus when no disease is within the hearing organ.^{84,85,86}

Kaltenbach et al.⁸⁷ proposed a tinnitus model in which hyperactive neurons in the DCN can undergo different forms of plasticity (temporal, by injury, by somatosensory modulation, and by activity-dependent stimulus), becoming an important contributor to producing and modulating tinnitus depending on DCN neuron cellular membrane dynamics. Developing DCN hyperactivity seems to depend on the balance of excitatory and inhibitory input, showing that the auditory system is not an electrically peaceful system. Shore and Zhou⁸⁰ found that 70% of the DCN has a bimodal sensorial pattern consisting of approximately 2/3 suppressive 1/3 enhancing integrations. Zhou and Shore⁸⁸ and Kaltenbach⁸⁹ explained the possible function that these neural auditive and somatosensory network interactions may accomplish in the DCN. The somatosensory role of the direct or indirect neuronal network interactions in producing and modulating tinnitus has suggested that numerous inputs to the cochlear nuclear complex is a possible proprioceptive information mechanism from the spinal trigeminal nucleus amongst other nuclear complex inputs (gracile, cuneatus, reticular system, vestibular). Such proprioceptive information mechanisms are necessary for vocalisation-communication (vocal structures position), for body situation, and for pursuing a sound source (pinna position) in the eye-to-head and head-to-space orientation, which is important in environmental alerting. This neuronal network has been suggested as being a possible autogenous sound eradication mechanism, suppressing self-produced (autogenous) sounds (chewing, self-vocalisation and respiration). Kaltenbach⁸⁹ correspondingly showed that the DCN is an integral part of the brainstem's circuits and is essential in biphasically auditory stimuli attention (projection recipient and source) interconnecting segmental structures (locus ceruleus, reticular formation). The objective of this sensorineuronal attention system is to coordinate the origin of auditory stimuli for the head, eyes, and ears. Taking this in mind, it should be recalled that the middle ear stapedius and tensor tympani muscles' auditive protective and discriminative ability, together with "Kaltenbach's attention model" during a centrifuge reflex, is also important as a normal conductive selective attention (discriminative) system.

4.3 Third neural exploration

It appears that sensorineuronal tinnitus is an altered neural activity effect and may result from a lesion or dysfunction at any level of the auditory system. Tinnitus can be centrally triggered by several mechanisms: auditory nerve transaction, vascular compression,

exposure to intense sound, and/or ototoxic drugs (i.e. cisplatin, among others). Reduced auditory-nerve input (hearing loss or deafness) reduces inhibition of the DCN and spontaneously enhances central auditory pathway motion, which is experienced as tinnitus; however, some tinnitus patients do not present any trauma history, hearing loss or deafness. Corresponding, a lack of DCN inhibition can also be modulated by somatosensory (TMD) input without cortical or sub-cortical inhibition.^{78,79}

Lockwood et al.⁹⁰ consider this DCN inhibition phenomenon as being a failure of normal “cross-modal inhibition” and suggested that hearing loss causes pathways to become reorganised by neuroplasticity in the central auditory system. This leads to abnormal interactions between auditory and other central somatosensory pathways. They proved that cortical tinnitus explains an interesting scenario in which the origin of tinnitus is more complex due to false sensory perception parallel levels (from peripheral biphasic to cerebral cortex levels). Information in the auditory pathways does not solely flow from the ear to the brain; there is also considerable flow in the opposite direction mediating and filtering human auditory system gain. Jeanmonod et al.⁹¹ proved that the thalamic relay synapses explains that rhythmic discharges in the cortex are due to signal loops by thalamic lack of inhibition causes this phantom sound to be integrated in a multidimensional aetiological viewpoint. Unbalanced excitatory and inhibitory signals in the multimodal non-specific thalamic nucleus with reverberating thalamo-cortico-thalamo loops may produce tinnitus in the auditory cortex.

Cacace⁹² stated that association cortical areas (including Wernicke’s area) are of prime importance in receiving multilevel segmental cross-modal interactions between auditory, somatosensory, somatomotor, and limbic system areas and tracts which do not exclusively involve peripheral auditory input in perceiving subjective sounds. Cacace stated that there is natural cortical concurrence of different perceptual sensorial experiences (synesthesia), which combines several modalities and may explain tinnitus in other primary cross-modal cortical area integration that would involve pain (TMD). Muhlnickel et al.⁹³ hypothesised a plastic reorganisation of the tonotopic cortical map, which might present tinnitus in cochlear hair cell loss (subjective hearing loss) as being a phantom phenomenon such as with phantom limb pain in the somatosensorial cortex. Such cortical reorganisation might also begin from on-going tinnitus.

4.4 Fourth neural exploration

Behavioural dimension of tinnitus is equally important, as are emotional, cognitive and attentional models regarding pain (inhibition or facilitation) and also rhythmic oromotor activity (bruxism).^{94,95} This behavioural dimension is modulated by cortical (amygdala, cingulate, insular cortex, etc.) and subcortical levels (reticular system, locus ceruleus, etc.). Tinnitus may produce different effects on each person’s social, emotional, and physical aspects, due to the level of annoyance produced by it (de-compensation).⁹⁶ Attention and affective components of tinnitus anxiety and phobias emerge in the limbic system; moreover, the psychosocial dimension may trigger tinnitus states. Such broad limbic organisation is the most important CNS modulator component that is able to transform (positively or negatively) the whole organism’s homeostasis (pain, concentration, temperature, muscle activity, memory, motivation, autonomous tonicity, hormonal and endocrine balance, feed, sleep, and other circadian rhythms) as a response to survival instinct stimulus. Mild tinnitus could be someone’s worst experience (high priority attention) but also the least significant incident (low priority attention) for the same person

and depending on personal bio-psycho-social reaction and its negative emotional reinforcement. Curiously, these symptom limbic frontiers triggered and modulated by the cognitive-behavioural dimension are transited in a similar way for bruxistic episodes that share common territories with apparently different multilevel effects.

Kaltenbach⁸⁹ explained that reciprocal DCN connections to subcortical limbic system structures relating to anxiety stimulate the locus ceruleus and during depression state the raphe nuclear complex. Serotonergic raphe nuclear complex activity is associated with the presence of tinnitus (increased serotonin levels) and depression (lower serotonin levels). Moreover, locus ceruleus is a multifunctional noradrenergic neuronal conformation serving many reflexes and having crucial emotional limbic component work. Fascinatingly, the same limbic system, which interacts with the DCN, also intermingles with the trigeminal motor nucleus producing bruxism and a TMD effect. Kato et al.⁹⁴ stated that bruxism is an intense, spontaneous, rhythmic motor manifestation, secondary to a sequence of autonomic physiological changes expressed in accelerated heart rate and increased motor, cortical, and breathing activity, which precede bruxism stages. Such rhythmic muscular dysfunctional episodes are triggered in attention-affective stressful conditions, which seem to concomitantly initiate tinnitus. Anxiety and depression thus appear to cause otic symptoms in a varied CNS structure.

5. Embryological explanation

TMJ development and other neighbouring structures in humans (such as pharynx, Eustachian tube, and tympanic cavity) is complex and continues to be investigated.⁹⁷ The mandible is formed from the ventral part of Meckel's cartilage, which is the first branchial arch.⁹⁸ The ossicles (malleus, incus, and partially the stapes) are formed from the dorsal part of Meckel's cartilage and Reichert's cartilage (second branchial arch). The malleus has a double origin in these ossicles; the anterior process originates from mesenchymal cells (os goniale), through intramembranous ossification, and the rest form from Meckel's cartilage, through endochondral ossification^{99,100} The malleus is related to the TMJ (condylar and temporal blastemas) by fibrous connections (lateral pterygoid muscle) passing through the petrotympanic fissure, which Rees named the discomalleolar ligament in 1954. These lateral pterygoid muscular fibrous connections then form the interarticular disc in Meckel's cartilage by mechanical stimulation of this muscle¹⁰¹

Neurological, vascular, and ligamental communication between the TMJ and the middle ear is preserved during TMJ development and continues during adult life because of continuity of Meckel's cartilage through the petrotympanic fissure (causing an incomplete closing in adults). This fissure holds the chorda tympani nerve in its middle ear egress to the TMJ, amongst other ear-TMJ vestige structures. The medial pterygoid muscle and the tensor tympani muscle develop from the temporal blastema. These structures (along with the tensor veli palatine) are innervated by the trigeminal mandibular branch (V3), in turn innervating the masticatory muscles coming from the first branchial arch mesoderm.¹⁰² Myrhaug³ reasoned that the ossicular chain and middle ear muscles primarily belong to the chewing system (i.e. embryologically) but finally serve the auditory system.

The functional connection between the ear and TMJ in the adult arises from common phylogenetic establishment of both the ear and TMJ. Meckel's cartilage plays a role in organising and forming jointly-located anatomical structures (Figure 2).

Fig. 2. TMJ and middle ear nearness in an temporal bone adult sample. 1. TMJ disc, 2. Malleus head, 3. Incus body, 4. Stape, 5. Inner ear vestibule, 6. Cochlea, 7. Chorda tympani nerve cut and medially pulled, 8. TMJ and malleus common ligaments cut. Author's dissection.

6. Muscular explanations

Complex neuromuscular interactions between masticatory muscles and ear muscles were called the "Otognathic Syndrome" by Myrhaug³ and then the "Otomandibular Syndrome" by Arlen.¹⁰³ Otic symptoms in the otomandibular syndrome occur without a real source in the ear, nose, or throat but do involve one or more mastication muscles in a state of fatigue. Several interpretations exist involving multiple muscles.

6.1 First muscular exploration

Anatomically, the tensor tympani (malleus muscle; TT) and the tensor veli palatini (Eustachian tube muscle; TVP) are middle ear muscles; although, they are functionally modulated by trigeminal motor nucleus motoneurons, which also modulate six more mastication muscles larger in size. Although middle ear muscle function is far from being completely comprehended, it is possible that TT and TVP muscle participation in TMD may lead to otic referred consequences when there is a dysfunctional stomatognathic state.

The Eustachian tube connects the middle ear with the nasopharynx through the TVP, assisted by the levator palatini and salpingopharyngeus muscles during velopharyngeal movements such as swallowing and the inhaling phase of respiration, equalising external and internal pressures. Salen and Zakrisson¹⁰⁴ found that pharyngeal and laryngeal muscles simultaneously work with the TT during swallowing and assist in Eustachian tube ventilation in a similar way to that of an air pump. Normal movement patterns such as yawning, laughing, swallowing, and coughing involve pharyngeal and laryngeal muscles activating the TT and may contribute towards ventilating the middle ear. Table 2 shows common intratympanic and extratympanic movements for the TT, TVP, and stapedial muscles.

Muscles/ Movements	Tensor tympani Innervation: Trigeminal	Tensor veli palatini Innervation: Trigeminal	Stapedial Innervation: Facial
Speaking	X	X	
Chewing	X	X	
Swallowing	X	X	
Jawning	X	X	
Laughing	X	X	
Coughing	X	X	
Breathing	X	X	
Acoustic trauma	X		X
Before speaking	X		X
Palpebral reflex	X		X

Table 2. Common intratympanic and extratympanic movements.

Interestingly, otolaryngology and otology specialists have ignored the character of the TT function. Some physiologists attach to it the function of stretching the tympanic membrane for improved reception of sound energy, but in medical circles it is considered as a muscle with practically no function in sound transmission. In contrast, the stapedial muscle is recognized as a powerful muscle in sound modulation and auditory protection and there is also an awareness of how the paralysis of this muscle generates evident audiometric and clinical effects. It is also known that the stapedial muscle improves external vocalization, reducing the autogenous sound masking effect (auditory protective and discriminative function); however, the participation of the TT muscle in this remains unresolved.

Ramirez et al.¹⁰⁵ compared the length of the TT and stapedial muscles. The difference was a little more than three times greater between the length of the TT muscle and the stapedius muscle. During stapedial reflex there is a muscular movement of about 50 microns, which reduces sound transmission by approximately 50 dB bilaterally and improves perception by 50 dB. This forces us to reflect on the potential capacity of the TT in the medial mobilization of the tympanic membrane in protection and tuning mechanisms. It must be remembered that it measures three times more in length than the stapedius muscle and is connected to the ossicular chain through the malleus and almost in opposition vector to the stapedius muscle, which in theory could generate movements three times greater (approximately 150 microns) when activated. I wonder how a muscle of these dimensions can be considered as an unproductive muscle when it is known that the joint mechanics of the middle ear work with deformities around one nanometre, which explain the modulator power of the stapedial muscle. It must also be kept in mind that TT motor innervation depends entirely on the activation of the trigeminal motor nucleus, the almost exclusively neurological centre of the stomatognathic system.

TMD may produce constant (spastic) or episodic (clonic) contraction and tension in the TVP and TT muscles during a state of fatigue. Zipfel et al.¹⁰⁶ explained how in subjective tinnitus a “false sound perception” (only perceived by the person) have continuous or rhythmical contraction of the TT and stapedius muscles producing steady muscle contraction or myoclonus, which makes rhythmic movements on the stape annular membrane and in the tympanic membrane. Analogously, but excluding vascular abnormalities, a pulsate TVP muscle contraction during objective tinnitus, initiate a “true sound perception,” (perceived

by the operator too) with rhythmical opening and closing of the Eustachian tube's pharyngeal area (palatal myoclonus). An objective tinnitus can be produced by rhythmic movement during TT myoclonus or TVP myoclonus (Eustachian tube seal rhythmic opening 30 times per minute) in an individual or combined way. This involvement of these muscles can produce varying otic conductive behaviour, which may be tinnitus in fashioned forms.

6.2 Second muscular exploration

Oscicular chain equilibrium depends on the state of the opposing TT and stapedius muscle contraction (Figure 3), thus regulating the normal functioning of structures leading sound (acoustic compliance) into the middle ear.¹⁰⁷ Among incudo-malleolar and incudo-stapedial joints, it should be recognised that the ossicular chain in the middle ear is weakly supported by the tympanic membrane, some malleus and incus ligaments, the annular ligament, and stapedius and TT muscle tendons. These structures support middle ear bones in a delicate but biomechanically efficient arrangement for receiving and transmitting acoustic stimuli to the inner ear. Although differently innervated (VII pair), the stapedius muscles accompany

Fig. 3. Tensor tympani and stapedial muscles in the middle ear cavity. Modification from Bouchet & Guilleret's Anatomy. Ed. Panamericana.

the TT muscle in auditory conduction during middle ear protection and filtering mechanisms, due to tympanic membrane stiffness regulation (Figure 4). Stapedius and TT muscle contraction is produced during many normal events. Also, they can be stimulated by the CNS in centrifuge auditory inhibition control (olivocochlear efferent system), protecting and filtering auditory afferent conduction towards the CNS through contracting these muscles and by additional inner ear hair cell modification.¹⁰⁸ Such combined stapedial and TT muscle mechanisms normally work by discriminating, fine-tuning, and improving external vocalisation; reducing the masking effect of autogenous sounds (pre-vocalisation contraction); enhancing transient stimuli against continuous background noise; and responding to strong external stimuli, protecting against possible acoustic trauma. It may be activated by vocalisation, chewing, swallowing, and facial muscle movement.¹⁰⁹

Fig. 4. Human temporal specimen's middle ear and middle cranial fossae. A: 1. Tympanic membrane, 2. Malleus head, 3. Incus, 4. Stape, 5. (Arrow) Stapedial muscle's tendon emerging from Pyramidal apophysis. B: 1. Incus, 2. Malleus head, 3. Chorda tympani nerve, 4. Tensor tympani muscle, 5. Geniculate ganglion, 6. Superficial petrosal nerve. Author's dissection.

TT muscle has a high threshold (100 dB), a latency of 12 ms and large and variable amplitude, which can be masked by stapedial muscle during impedance test, due to their lower threshold (70 dB) and latency (7 ms). Through electromyography and genetic testing in other species TT muscle (as stapedial muscle) has been shown to have special contractile properties and resistant to fatigue because of its fast oxidative glycolytic fibers.¹¹⁰ The mechanical application of acoustic stimuli (70-100 dB frequency of 2 KHz) is responded immediately due to this feature.¹¹¹ According to Ochi et al.¹¹², the activity of the TT muscle (in addition to the centrifuge auditory inhibition control originated in the cochlea) can be evoked from somato-sensory or sensori-vestibular origins. With this in mind, a complex physiological activity of TT muscle cannot be forgotten, especially when hearing, protecting, discriminating, autogenous sound-enmasking and pressure equalizing multiple functions offers a paramount role in middle ear.

In 1987, Malkin¹¹³ stated that the TT serves as a barometric pressure receiver. Its proprioceptive afference signals (starting from their muscular length) can be triggered in a hypotonic situation caused by low tympanic cavity pressure (due to mucosal air exchange). Such low-pressure medially retracts the tympanic membrane and TT tendon due to great external environmental pressure accompanied by no resistance to force; its muscular

spindles perceive such new muscle elongation. The trigeminal motor nucleus produces a reflex mechanism in a polysynaptic central arrangement, beginning TT muscle contraction involving opening the Eustachian tube (through TVP activation), middle ear ventilation and pressure equilibration; such normal physiological mechanisms may be blocked by the TT fatigued and hypertonic scenario during TMD.

Klockhoff and Anderson¹¹⁴ proposed a “TT syndrome” when the above cannot function correctly during TMD. Sustained TT muscle contraction during TMD can alter the ossicular spatial position and perilymphatic and endolymphatic pressure through the transmitted changes from the oval window to the cochlea and semicircular canal walls. It should be stressed that the auditive cells are very sensitive and constantly depolarise even during rest (spontaneous otoacoustic emissions), which may be perceived as tinnitus by some patients. Sensorial spontaneous otoacoustic emissions can be mechanically increased (conductively) by the TT and stapedius spasm, or myoclonus. Moreover, the TMD pathogenic scenario regarding middle ear muscle mechanisms may abnormally reduce sonic transmitting vibration from the tympanic membrane towards the oval window, which may be expressed as a paroxistic subjective hearing loss. This anomalous muscular-ossicular relationship may also trigger abnormal mechanical sensory organ stimuli and unbalance vestibular impulses. This entire situation may be expressed as tinnitus, subjective hearing loss, and vertigo.¹¹⁵

6.3 Third muscular exploration

The shortening and thickening of the mastication medial pterygoid muscle in over-closed jaw positions (specially in edentulous patients) can produce an anatomical cross-sectional widening area that exerts a lateral pulling force on the adjacent TVP muscle and the Eustachian tube (Figure 5).¹¹⁶ Mistaken vertical dimension rehabilitation is common in edentulous patients with a varied referred symptoms presentation caused by this clinical situation (Figure 6). Goto et al,¹¹⁷ explained that an increase of medial pterygoid transverse volume can result from an open-closed position depending on muscle length and tension variation. The close proximity of both muscles can normally be observed during TVP muscle

Fig. 5. Intraoral and extraoral view of edentulous patient with referred otic symptoms originated in an advanced jaw osseous reabsorption with overclosure prosthesis (not showed) that do not regain a normal intermaxilar vertical dimension. Photography use authorized to the author (Patient's record).

contraction, which opens the Eustachian tube lumen and laterally presses on the medial pterygoid muscle (Figure 7). Sehhati-Chafai-Leuwer et al.¹¹⁸ named this contact “hypomochlia,” or fulcrum, stating that it can change muscle tension direction together with another two structures contacting the TVP (pterygoid hamulus and Ostmann’s fatty tissue), which may influence middle ear ventilation by Eustachian tube compliance.

Fig. 6. Frontal and lateral photographic registration with an incorrect (A,C) and a correct complete denture vertical dimension rehabilitation. Photography use authorized to the author (Patient’s record).

A deep overbite, medial pterygoid shortening, hypertonicity, or spasm results in muscular mass compression and lateral bunching of the adjacent TVP and associated structures.^{119,120} If masticatory muscles are hypertonic because of a TMD, the TVP and TT muscles may be hypertonic due to equal innervation by V3. Penkner et al.¹²¹ stated the opposite in a pilot-study with 16 TMD patients tested with an EMG needle and audio and tympanogram recordings, which did not provide a correlation with such dysfunction. However, in the Penkner et al. study, the patient population was small and their research design lacked a control group; moreover, it did not have an evident chronic TMD group or a satisfactory group with severe dysfunction (only two patients). Dysfunctional TVP was not present in that study, because all patients demonstrated regular and bilateral opening of the Eustachian tube.

It must be considered that the TVP and TT have gained embryological, anatomical, physiological, and neurological territory in the stomatognathic system, and research regarding them is complex and sensitive (requiring precision). A good methodological design could

Fig. 7. Jaw and pharyngeal area frontal view: Medial Pterygoid and Eustaquian tube dissection. Picture a- Jaw in normal vertical dimension: 1. Medial pterygoid muscle, 2. Sectioned frontal jaw body, *. Eustaquian tube cartilage. Picture b- Jaw in overclosure position increasing medial pterygoid transverse volume on Eustaquian tube cartilage. Author's dissections.

discard or confirm a probable cause-effect relationship between Eustachian tube dysfunction and TVP dysfunction.

McDonnell et al.¹²² stated that children with deep dental overbites were 2.8 times more likely to develop Eustachian tube dysfunction than those without deep overbites because of the muscular over-closed position. Azadani et al.¹²³ affirm that children with deep bites were 10.6 times more prone to Eustachian tube dysfunction than those without deep bites in their multivariate model.

Interestingly, although infection is the most prevalent aetiology of otitis media in children (allergic conditions or respiratory infections), TVP dysfunction (TMD) may also produce otitis media with effusion.¹²⁴ It is well known that the middle ear transudation effect is due to hypoventilation and abnormal gas exchange when the Eustachian tube becomes blocked and impeded to regulate pressure. The Eustachian tube normally maintains a closed position at rest, protecting the middle ear from retrograde nasopharynx microflora flow during rapid fluctuations in nasopharyngeal pressure associated with breathing, swallowing, coughing, sneezing, and nose-blowing. Children's Eustachian tube dysfunction plays an important role because of the anatomical configuration of the Eustachian tube (short, horizontal, and wide lumen) or the TVP open-closed dynamics is spastic or trapped by the neighbouring pterygoid muscle.¹²⁵

6.4 Fourth muscular exploration

It is reasonable to propose that if TT and TVP dysfunction can separately produce otic symptoms, then the effects of an anatomically agonistic co-working function may be more

problematic. Barsoumian et al.¹²⁶ corroborated Lupin's 1969 findings and Rood and Doyle's¹²⁷ results by discovering how the fibres of the most external TVP muscle area and the TT fibres are joined in the middle ear in adult human cadavers (Figure 8).¹²⁸ The TVP thus has an additional bone origin in the malleus manubrium. Kierner et al.¹²⁹ corroborated this functional connection in human cadavers through histological analysis. TT and TVP dysfunction in TMD can modify the malleus and tympanic membrane's medial position, individually or in combination (inner deflection). Consequently, these muscles act synergistically together and can temporarily increase a medial intra-tympanic pulling force effect with the expected otic referred symptom consequence, due to such delicate ossicular chain biomechanics. A macroscopically morphological study found these same connection fibers between TT and TVP muscles in 23 human temporal blocks (Figure 9 and 10).⁵ This proves the anterior histological and anatomical findings about the possible combined function between TVP and TT muscles with its masticatory-otic dysfunctional referred symptoms consequences. After all, understanding middle ear ventilation physiology through the Eustachian tube involves neurological territories referred from the stomatognathic system that considers TT and TVP muscle vital.

Fig. 8. TT and TVP muscle fiber connection. M. Malleus manubrium, TT. Tensor tympani muscle, TP-L. Tensor veli palatini muscle. Used with author's permission from Rood, S.R. & Doyle, W.J. (1978). Morphology of tensor veli palatini, tensor tympani, and dilatator tubae muscles. *Ann Otol Rhinol Laryngol*, 87:202-10.

6.5 Fifth muscular exploration

Several studies have investigated concomitant functional connections between TMD and cervical spine disorders.^{130,131} The functionality of cervical and masticatory systems is complemented synergistically but pathologically too in a concomitant way. Kuttilla et al.⁷⁵ reported that 45% of TMD-tinnitus patients have headaches and 54% have neck-shoulder

Fig. 9. Dissections on TT and TVP muscle fiber connections. A: AT, Auditory tube, TVP. Tensor veli palatine muscle, TM, Tensor tympani muscle, 1. Fiber connection, 2. Tensor tympani muscle's tendon, 3. Malleus head, 4. Tympanic membrane. B: 1. Tensor tympani muscle, 2. Fiber connection, 3. Tensor veli palatine muscle, 4. Osseous portion of Eustachian's tube, *. Tympanic cavity. Author's dissections.

pain. Levine et al.⁷⁸ were able to produce cranio-cervical tinnitus modulation in normal otologic patients (tinnitus and non-tinnitus patients) using isometric cephalo-cervical exercises changing the loudness, pitch, and location of tinnitus by modulating the somatosensory and acoustic central neural pathway. If cranial and cervical muscular dysfunction in TMD (producing hypertonicity and muscular spasm) can trigger tinnitus, then it can also irritate nerves and blood vessels by muscular trapping. Cervical muscular fatigue may produce tension on the vertebral artery which feeds the basilar artery and inner ear inflow, with exacerbated otic consequences.^{78,79} Additionally, it may also distort normal proprioceptive reception in the vestibular nucleus and in the cervico-oculo-vestibular muscle reflex controlling the head's postural position, thereby complicating neck-otic vascular flow and worsening the vertigo produced.

7. Temporal bone explanations

TMJ and middle ear have a bone communication (iter chorda anterior) known as Huguier's channel that is shown by dry and fresh dissections in Figure 11 and 12. The TMJ and middle

Fig. 10. Dissections on TT and TVP muscles related to left cartilaginous and mucous Eustachian tube. A: Structure in situ. B: Structure block retired from temporal bone. 1 and 2. Hemisected Eustachian tube. 3. Tensor tympani muscle, 4. Tensor veli palatine muscle, Author's dissections.

ear are small, compact structures that share through this communication vascular (anterior tympanic and deep auricular arteries), neurological (chorda tympani and auriculotemporal nerve), and ligamental: disco-malleolar ligament (DML) and anterior malleolar ligament (AML). These can be easily injured during TMJ disorder and may explain associated otic symptoms.^{132,133}

7.1 First bone common passages exploration:

Human adult and foetus dissection has confirmed an anatomical link between the TMJ, the mandibular body, and the middle ear.^{134,135,136,137,138,139,140,141,142,143,144} DML and AML are responsible for such bone communication and connection; they are attached to the ossicular chain (malleus) and may create a biomechanical connection between the middle ear and the mandible.^{145,146,147,148} These findings are corroborated in 23 human temporal bone specimens (Figure 13) that consistently show these ligamental structures.¹⁴⁹

Fig. 11. A. External view of Huguier's canal from TMJ. B. Internal view of Huguier's canal from middle ear. * Antero-lateral sample orientation. C. Internal fresh view with ossicular chain (Arrow head). 1. TMJ fossae, 2. Huguier's canal external foramen, 3. Scamo-tympanic fissure, 4. Petro-tympanic fissure, 5. Auditive tube osseous portion, 6. Huguier's canal internal foramen, 7. Huguier's canal septum, 8. Tympanic cavity. Author's dissections.

Fig. 12. Superior and postero-medial view of right middle ear and TMJ. * Postero-medial sample orientation. 1. TMJ disc, 2. Middle ear, 3. DML antero-laterally separated from the lateral side of Huguier's canal, 4. DML and AML Huguier's canal septum, 5. AML antero-medially separated from the medial side of Huguier's canal, 6. Chorda tympani nerve suprasedal trajectory, 7. TMJ bilaminar zone. Author's dissections.

These ligamental structures may be stretched by a TMJ disorder, which could affect middle ear ossicular equilibrium; although, there is controversy about their ability to disturb the ossicular chain.^{150,151,152,153} The spread of forces through cranial bone sutures was treated by Libin in 1987 and suggested that ligaments common to neighbouring structures could become tensioned during normal physiological mobilization and in abnormal temporal bone trauma.¹⁵⁴ Retrodiscal tissue elasticity can normally act as an energy buffer in spreading movement from the TMJ to the middle ear by such common ligaments; however, TMJ disc luxation or oedematous pressure from an inflammatory disorder could certainly cause tension on the malleus through Huguier's canal.^{133,155,156}

The range of tympanic membrane deformation during conducting sound energy must be understood when trying to ascertain the possibility of motion from the DML and AML on malleus ossicles. Tonndorf et al.¹⁵⁷ used time-average holography to show that an intense acoustic stimulus (111-121 dB) can deform the tympanic membrane by no more than nanometers or possibly a micrometer, depending on the frequency and place for tympanic

Fig. 13. Superior and antero-medial view of left middle ear and TMJ. * Antero-lateral sample orientation. 1. TMJ disc, 2. Malleus head, 3. Incus, 4. DML, 5. AML, 6. Chorda tympani nerve, 7. VII cranial pair geniculated ganglion, 8. tensor tympani muscle. Author's dissections.

membrane measurement.¹⁵⁸ Tympanic membrane and middle ear vibration produced by sound energy is thus on the nanometer range scale. The auditory threshold thus responds from sub-angstrom oscicular motion.^{159,160} This finding has been widely corroborated by Wada et al.^{161,162} using better-quality tympanometry equipment with low probe tone frequency (time-average speckle pattern interferometry) and finite element analysis at different frequencies and pressures in human and other species' hearing systems.

According to Eckerdal¹⁵⁰, the range of movement of these ligaments depends on the fibrous connection on the walls of the petrotympanic fissure, thereby corroborating Coleman's findings¹³⁸. Ramirez et al.,¹⁴⁹ findings concern 30.5% DML's malleolar mobility correlated well with Sato et al.¹⁶³ who showed a spacious Huguier's canal in 29.2% of samples, suggesting a wide foramen which could allow free passage of its inner structures. If the oscicular chain can transmit a nonometric tympanic vibration through two joints (from tympanic membrane to inner ear), with more than four ligaments and two muscles having an effective area and lever relationship, it would thus be potential that DML and AML have a highly probable movement effect on oscicular chain spatial disposition when TMJ-jaw traction is applied to them by the malleus.

Experiments have demonstrated that AML fixation (producing stiffness) is dominant at low frequencies. Although low frequency tinnitus has been reported to be very rare and diverse (it may vary in pitch from low to high frequency, intermittent or permanent and vary in intensity), it cannot be ruled out that it is caused by oscicular fixation and/or a low admittance pattern. The work of Nakajima et al.¹⁶⁴ has ruled out the idea that ligamental motion may not produce an auditory effect by appreciable excitation of the inner ear due to high-pass filtering by different systems (helicotrema, incudo-malleolar, incudo-stapedial and oval anullar ligament joints). They revealed reduced auditory sensitivity (8-10 dB loss)

when the AML was partially fixed and a larger loss (15-35 dB) when it was totally fixed. This proved that restraining the ossicles produced an increase in the ear's impedance. They tried to simulate an otosclerosis lesion because AML fixation clinically occurs in combination with this pathology.¹⁶⁵ This experimental model also proved the power of adjustment in sound transmission of this ligament.¹⁶⁶ In this sense, whether such motion at low frequencies involved in TMJ-jaw motion can produce enough sound to be heard at an auditory threshold close to 1 Hz (the stapes move at least one micron) is not suspicious.

Huguier's canal's morphological dimensions play a paramount role in the above possibility. Huguier's canal has a slender funnel-like form in the petrotympanic fissure (being wider near the TMJ and narrower near the middle ear). Sato et al.¹⁶³ and Eckerdal¹⁵⁰ measured such dimensions at three sagittal places (near the TMJ, the middle area and near the middle ear) agreeing a plentiful wide space dimensions. Such Huguier's canal morphology suggests permissive movement ability for the DML and AML when a force is applied to them and transmitted via ossicular chain vibration dynamics. In a partial agreement with Eckerdal¹⁵⁰ (Huguier's canal adherence restricting ligaments mobility), I do not consider such adherence able to impede nanometer movement transmission in a collagenous (and maybe elastic) ligament from TMJ anterior traction force during protrusion. Riga et al.,⁶² demonstrated an increase in stiffness of the middle ear of forty patients with TMD that could demonstrate minor conductive alterations of the middle ear by these mechanisms that generate referred otic symptoms from TMJ.

Differing explanations have arisen from these ligaments' morphology regarding Huguier's canal dimensions, strengthened by ossicular chain dynamics' physiology. Cheng¹⁶⁷ found AML good viscoelastic performance calculated for tension resistance (1.05 MPa) and stretching resistance (1.51 MPa), thereby assuring force transmission from the TMJ to the middle ear malleus. Disorganized surface collagen disposition was found regarding this structure's morphology (trespassing Huguier's canal walls), enveloping a wide and well-organized internal longitudinal collagen band ordered as a double collagen layer assembly with a thin disorganized external stratum. This characteristic, plus its width, mechanical resistance and sound energy transmission magnitude, makes Eckerdal's adherence model for Huguier's canal doubtful.

Normal excursion of the disc and condyle during mandibular movement (Figure 14) may not provoke malleus mobility and altered tympanic membrane tension; however, functional or inflammatory joint disorders such as disc luxation or secondary oedema may produce ossicular chain tension by disco-malleolar ligament traction.^{155,156,168} Ren et al.,¹⁶⁹ found a significant correlation between internal TMJ derangement and tinnitus, detecting disk luxation in the ipsilateral joint of 53 patients with unilateral tinnitus.¹⁷⁰ Kutila et al.⁷⁵ found a similar relationship between TMJ internal derangement and tinnitus. According to this hypothesis, tinnitus and vertigo may originate in the altered stapes' position due to the force being transmitted from these malleus ligaments. Likewise, otalgia may be present because of peripheral nerve stimulation in the tympanic membrane due to membrane-bonded malleus traction.

In relation to the spheno-mandibular ligament, there is agree with the findings of Abe et al.,¹⁷¹ who found that this ligament was inferiorly fixed to the mandible and superiorly fixed to the sphenoid spine and the anterior malleolar ligament as shown by personal dissections on human temporal bone specimens (Figure 15). Both may be tensed in a marked over-closure position, also stretching ossicular balance. The otic effects are latent in this tensed

Fig. 14. Superior and antero-medial view of left middle ear and the TMJ. TMJ's disc anterior traction protrusion). * Antero-lateral sample orientation. Fig. A, 1. Mastoid aditus, 2. Incudal fossa, 3. incudo-malleolar joint, 4. DML (short arrow) and AML (long arrow) emerging from the malleus anterior process, 5. DML fixing to TMJ bilaminar zone, 6. TMJ disc, 7. AML in its supra-septal medial course, 8. Internal face of tympanic membrane, 9. Chocleariform process, 10. Tensor tympani muscle tendon, 11. Incudo-stapedial joint, 12. Chorda tympani nerve over tensor tympani muscle tendon, 13. Chorda tympani nerve and AML medial course, 14. TMJ bilaminar zone stretched in excursive tractional TMJ movements, 15. TMJ disc in extreme protrusive movement. Author's dissections.

mechanical scenario, especially in edentulous patients. Alkofide et al.,¹⁴⁶ studied anterior malleolar and spheno-mandibular ligaments structural characteristics in 37 specimens, determining that the spheno-mandibular one reached the malleus (8.1%) and the middle ear (67.6%), whilst the anterior malleolar one passed through the petrotympanic fissure with the spheno-mandibular (58.3%), suggesting guaranteed connectivity between both ligaments. Burch¹⁴⁵ found that spheno-mandibular ligament relaxed during maximum jaw opening and tensed during over closure; however, it has been suggested that it can be stretched during lateral jaw movements too.¹⁷² Both ligaments can thus be tensioned in several situations, which may alter the ossicular chain,¹⁷³ although I could not observe this in my dissections.

7.2 Second bone common passages exploration

The vascular relationship between the TMJ and the middle ear may explain otic symptoms in the presence of a vascular reflex from TMJ disorders. The most medial anterior tympanic artery posterior group branches (behind the TMJ) irrigate the tympanic cavity and the

Fig. 15. Left TMJ, mandible ramus and middle ear medial view. 1. Tympanic cavity, 2. DML bilaminar area union, 3. TMJ disc, 4. Alveolar nerve sectioned and retracted over external pterygoid muscle, 5. External pterygoid muscle, 6. EML, 7. Styloid process, 8. Lingula, 9. AML fixing to EML lateral surface, 10. Sphenoid spine. Author's dissections.

external auditory meatus through the petrotympanic fissure, using the same osseous routes as the ligaments, as explained above¹⁴¹. Moreover, Merida-Velasco et al.¹⁷⁴ found how the small venous vessels from the anterior portion of the middle ear (crossing the petrotympanic fissure) reach the venous retrodiscal plexus and drain into the retromandibular vein. Interrupting normal artery flow may affect the auditory system.

7.3 Third bone communication exploration

Marasa and Ham¹²⁵ suggested that oedema produced by TMJ inflammatory disorders could spread through the petrotympanic fissure to the middle ear and produce serous otitis media. Oedema produced in the TMJ can spread collected fluid through the petrotympanic fissure to the middle ear and produce vulnerable disease via this route.¹⁷⁵ Osseous communication between the middle ear and TMJ in children may lead to pathologies such as TMJ septic arthritis (with a doubtful infectious site), in the presence of infectious otitis media.^{176,177}

8. Is TMD integrated in an otic multidimensional model?

The question is still open regarding the hierarchy of peripheral and/or central sources of otic symptoms and how they appear to interact in a simultaneous way. Rubinstein et al.,¹⁷⁸

reported that TMD patients with a longer duration of tinnitus responded worse to the TMD treatment than those with a shorter duration, suggesting peripheral acute pathology and central neuroplastic change during chronic symptoms, implying a combination of them. This dichotomy is well pictured in Abel at al.,⁷⁹ works on somatomotor-somatosensorial tinnitus modulation.⁷⁸ However, these modulations were only recognised as otic sensori-neuronal phenomenon, without allowing for an otic conductive scenario. Jaw movement needs trigeminal motor nucleus motoneuron intervention activating the TT and TVP with expected conductive effects.

It is difficult to take a solely neuroanatomical or neurophysiologic viewpoint when interpreting otic symptoms due to combined peripheral-central interactions, particularly in the absence of objective and unique neurological signs and intricate auditory and stomatognathic system connections at different CNS levels. Interspecies and human neuroanatomy provide the most important advances on the influence of some tracts and cortical areas on others and how they may be felt as secondary sensorineural otic symptoms. The protagonism of constant deep pain, trigemino-vascular auditive control, somatosensorial-auditory multimodal integration, cortical and subcortical sound broadband interpretation, corticofugal modulation, and limbic behavioural interferences as CNS phenomena has been demonstrated in referred otic symptom pathophysiology. However, CNS dynamics-initiated associated symptoms also have a relevant peripheral feedback effect on auditory cortical and subcortical connections that can be additionally produced and modified by a conductive intermediate level (middle ear).

9. Evidence regarding dental treatment outcome

There are many forms of otic treatment, such as pharmacological, surgical, instrumental, phychophysiological, counselling, electrostimulatory, physiotherapy (cervical spine mobility), acupuncture, hypnosis, thermotherapy, cryotherapy, ultrasound-laser therapy, biofeedback, and stomatognathic treatment⁶⁸. Stomatognathic treatment addressing masticatory muscle relaxation (including the TT and TVP) by using removable interocclusal plastic appliances seems to be able to eliminate or attenuate otic symptoms triggered or exacerbated by TMD.

Author	Nº patients	Otic symptoms	% relief
Gelb et al.,1967	26	O,T,V,HL	96
Bernstein et al., 1969	28	O,T,V,HL,OF	75
Gelb et al., 1975	38	T,V	82
Rubinstein et al., 1987	68	T	41
Bush 1987	14	T	40
Kerstein 1995	23	T	83
Wright 2000	15	O,T,V	80
Kuttila et al., 2002	18	O	83
Ramirez et al., 2006	23	O,T,V,HL,OF	90

Table 3. Percentage of otic symptoms relieved by muscular relaxation oral device. O: otalgia, T: tinnitus, V: vertigo, HL: hearing-loss, OF: otic fullness.

Oral devices attempt peripheral relaxation of muscle hyperactivity triggered during anxiety and depression. These devices have been used individually or as part of varied treatment including physiotherapy, counselling, and acupuncture. However, several discrepancies limit research into this management device's methodology, making them prone to error and bias due to lack of method standardisation. This variety among methods includes the sample-size, diagnostic criteria, oral appliance, combination of oral appliance with another mode of treatment, non-validated questionnaires, absence of control group, non age and gender matching, and mailing the clinical evaluation, which makes the results difficult to interpret. The placebo effect must be considered in TMD-otic symptom treatment results. Table 3 shows otic symptom relief by single oral device treatment in TMD.^{20,22,24,35,56,178,179,180} Treatment including oral devices as part of collective management were excluded due to treatment outcome non-specificity.^{45,86,170,178,181,182,183,184,185,186}

10. Conclusions

Cause and effect relations between TMD and orofacial-otic symptoms are still a polemic topic. When the link is emphasized by therapeutic results the cause-effect relations get strength. Teamwork and an exhaustive symptoms assessment based on a complete structured interview and physical examination are necessary for the diagnosis and treatment of these symptoms, closing a wide conception breach existing between health disciplines.

Interdisciplinary management, including a dental specialist in craniofacial pain, offers a key tool to medical staff during these symptoms' conservative phase. Clinical success depends on each specialist's ability to study the different aspects of the same problem. One health discipline cannot always solve patients' symptomatology by themselves unless aided by the invaluable support of a multidisciplinary management team. Every specialist contributes his/her specific knowledge towards differential diagnosis addressing a correct treatment plan.

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Up to Date on Tinnitus encompasses both theoretical background on the different forms of tinnitus and a detailed knowledge on state-of-the-art treatment for tinnitus, written for clinicians by clinicians and researchers. Realizing the complexity of tinnitus has highlighted the importance of interdisciplinary research. Therefore, all the authors contributing to the this book were chosen from many specialties of medicine including surgery, psychology, and neuroscience, and came from diverse areas of expertise, such as Neurology, Otolaryngology, Psychiatry, Clinical and Experimental Psychology and Dentistry.

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