

# We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

**4,800**

Open access books available

**122,000**

International authors and editors

**135M**

Downloads

Our authors are among the

**154**

Countries delivered to

**TOP 1%**

most cited scientists

**12.2%**

Contributors from top 500 universities



**WEB OF SCIENCE™**

Selection of our books indexed in the Book Citation Index  
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?  
Contact [book.department@intechopen.com](mailto:book.department@intechopen.com)

Numbers displayed above are based on latest data collected.

For more information visit [www.intechopen.com](http://www.intechopen.com)



# Tinnitus and a Linked Stomatognathic System

Luis Miguel Ramirez Aristeguieta  
*Universidad de Antioquia, Medellin  
 Colombia*

## 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).<sup>1</sup>

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 Costen<sup>2</sup> (1934) pioneering ideas about otic referred symptoms starting from the stomatognathic system, Myrhaug<sup>3</sup> 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.<sup>4,5,6</sup> This analysis equally tries to support actual evidence about otic referred symptoms reliefs by stomatognathic therapeutic.<sup>7</sup> 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.<sup>8</sup> These could be primary, referred or combined from cervical muscles and associated cranial structures and appear similarly in adults, as well as, in children <sup>9,10</sup>. The prevalence of TMD is 1.5 to 4 times more common in women than in men.<sup>11</sup> 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.<sup>12</sup> 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).<sup>13,14,15,16,17</sup>



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.<sup>18,19</sup>

Otic symptoms include otalgia, tinnitus, vertigo-dizziness, subjective hearing loss, and otic fullness.<sup>20,21,22,23,24,25</sup> 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.<sup>26</sup> 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.<sup>27,28</sup> 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.<sup>29</sup> A screening search permit to observe apparent correlations between otic symptoms and TMD in some studies from 1933 to 2011 (Table 1).<sup>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</sup> Overall, 12.732 TMD patients were mentioned in n=54 articles. Otolgia 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.<sup>67</sup> 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: Kuttilla et al.<sup>46</sup> found a prevalence of tinnitus (12-17%), otalgia (12-16%), and otic fullness (5-9%) in subjects with TMD. According to Rubinstein<sup>68</sup> 33-67% of TMD patients reported tinnitus. Gelb et al.<sup>22</sup> found that 42% of patients with TMD reported tinnitus, 35% reported otalgia, 18% reported dizziness, and 14% reported hearing loss. Besides, Cooper et al.<sup>21</sup> found 40-70% of TMD patients experiencing dizziness and 5-40% feeling vertigo. Lam et al.<sup>69</sup> 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 Agerberg<sup>70</sup> 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.<sup>71</sup> 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. Levine<sup>73</sup> found that tinnitus can be modulated by isometric oro-facial and cranio-cervical manipulations.

In addition to the pain dimension, Hazell<sup>74</sup> 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) Y	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) Y	338	100	59	70	—	—
Vernon <i>et al.</i> , 1992 (72) Y	69	45	100	—	—	75
Cooper <i>et al.</i> , 1993 (21) U	996	63	51	41	25	30
Oguteen-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) Y	53	98,1	100	54,7	—	92,5
Parker-Chole 1995 (49) Y	338	100	59	70	—	—
Keersmaekers <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	—	—	—
Katjalainen <i>et al.</i> , 1997 (34) Y	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) Y	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	—	—
Brato <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) Y	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) Y	20	65	60	—	—	90
De Felicio <i>et al.</i> , 2010 (61) Y	30	13,4	8,8	—	—	13,5
Pekkan <i>et al.</i> , 2010 (63) Y	25	72	52	20	8	56
Bernhardt <i>et al.</i> , 2011 (64) Y	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

Y Epidemiological / Controlled studies

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



masticatory muscles. Kuttilla et al.<sup>75</sup> 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.<sup>76</sup> 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.<sup>77</sup> 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.<sup>78</sup> 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".<sup>78,79</sup> Shore et al.<sup>80</sup> 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.<sup>81</sup> affirm that such a stimulus is so strong that it can interfere with moderate-level acoustic stimuli.<sup>82</sup> 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.<sup>83</sup> 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.<sup>84,85,86</sup>

Kaltenbach et al.<sup>87</sup> 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 Zhou<sup>80</sup> found that 70% of the DCN has a bimodal sensorial pattern consisting of approximately 2/3 suppressive 1/3 enhancing integrations. Zhou and Shore<sup>88</sup> and Kaltenbach<sup>89</sup> 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). Kaltenbach<sup>89</sup> 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.<sup>78,79</sup>

Lockwood et al.<sup>90</sup> 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.<sup>91</sup> 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.

Cacace<sup>92</sup> 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). Muhlntickel et al.<sup>93</sup> 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 somatosensory 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).<sup>94,95</sup> 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).<sup>96</sup> 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.

Kaltenbach<sup>89</sup> 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.<sup>94</sup> 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.<sup>97</sup> The mandible is formed from the ventral part of Meckel's cartilage, which is the first branchial arch.<sup>98</sup> 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<sup>99,100</sup> 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 muscle<sup>101</sup>

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.<sup>102</sup> Myrhaug<sup>3</sup> 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).

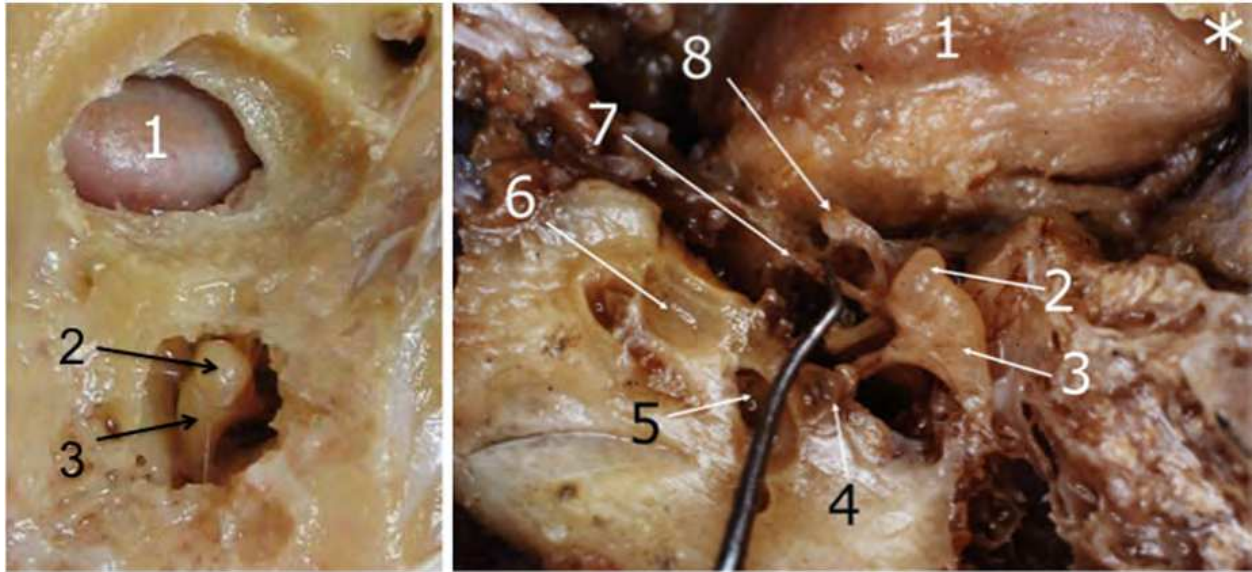


Fig. 2. TMJ and middle ear nearness in an temporal bone adult sample. 1. TMJ disc, 2. Malleus head, 3. Incus body, 4. Stape, 5. Inner ear vestibule, 6. Cochlea, 7. Chorda tympani nerve cut and medially pulled, 8. TMJ and malleus common ligaments cut. Author's dissection.

## 6. Muscular explanations

Complex neuromuscular interactions between masticatory muscles and ear muscles were called the "Otognathic Syndrome" by Myrhaug<sup>3</sup> and then the "Otomandibular Syndrome" by Arlen.<sup>103</sup> 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 Zakrisson<sup>104</sup> 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.<sup>105</sup> 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.<sup>106</sup> 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.<sup>107</sup> 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

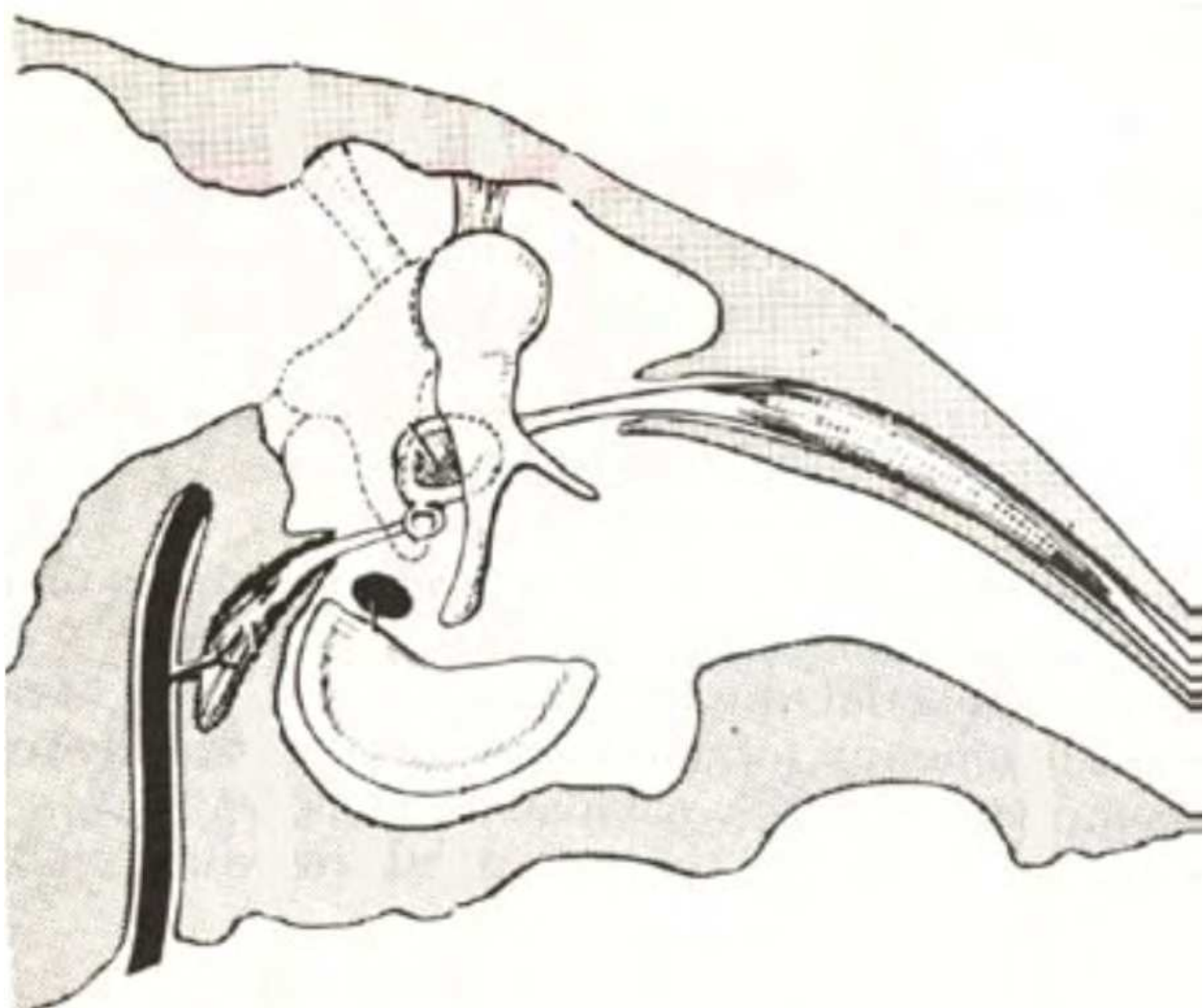


Fig. 3. Tensor tympani and stapedial muscles in the middle ear cavity. Modification from Bouchet & Guilleret's Anatomy. Ed. Panamericana.



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.<sup>108</sup> 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.<sup>109</sup>

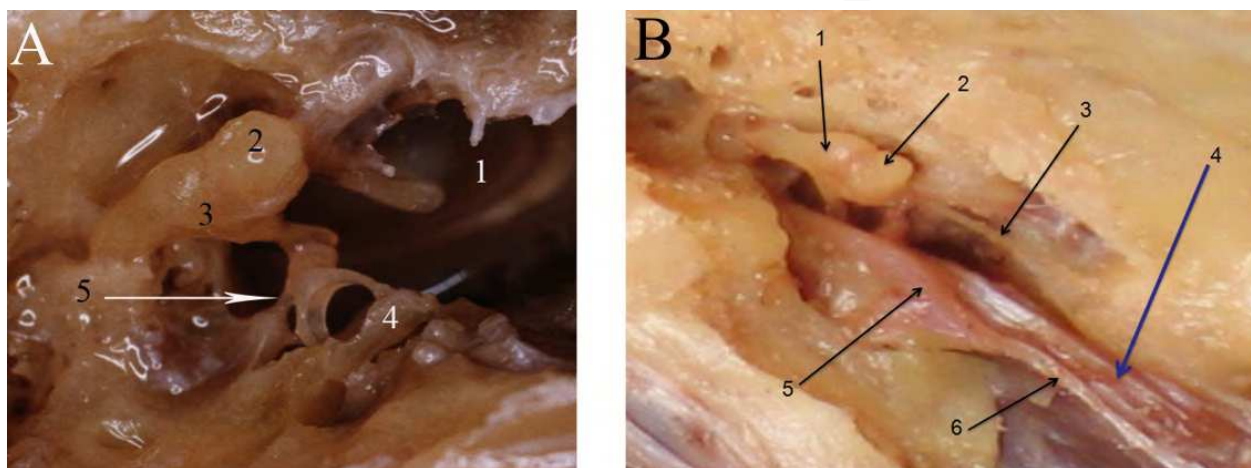


Fig. 4. Human temporal specimen's middle ear and middle cranial fossae. A: 1. Tympanic membrane, 2. Malleus head, 3. Incus, 4. Stape, 5. (Arrow) Stapedial muscle's tendon emerging from Pyramidal apophysis. B: 1. Incus, 2. Malleus head, 3. Chorda tympani nerve, 4. Tensor tympani muscle, 5. Geniculate 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.<sup>110</sup> The mechanical application of acoustic stimuli (70-100 dB frequency of 2 KHz) is responded immediately due to this feature.<sup>111</sup> According to Ochi et al.<sup>112</sup>, 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, Malkin<sup>113</sup> 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 Anderson<sup>114</sup> 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.<sup>115</sup>

### 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).<sup>116</sup> 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,<sup>117</sup> 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

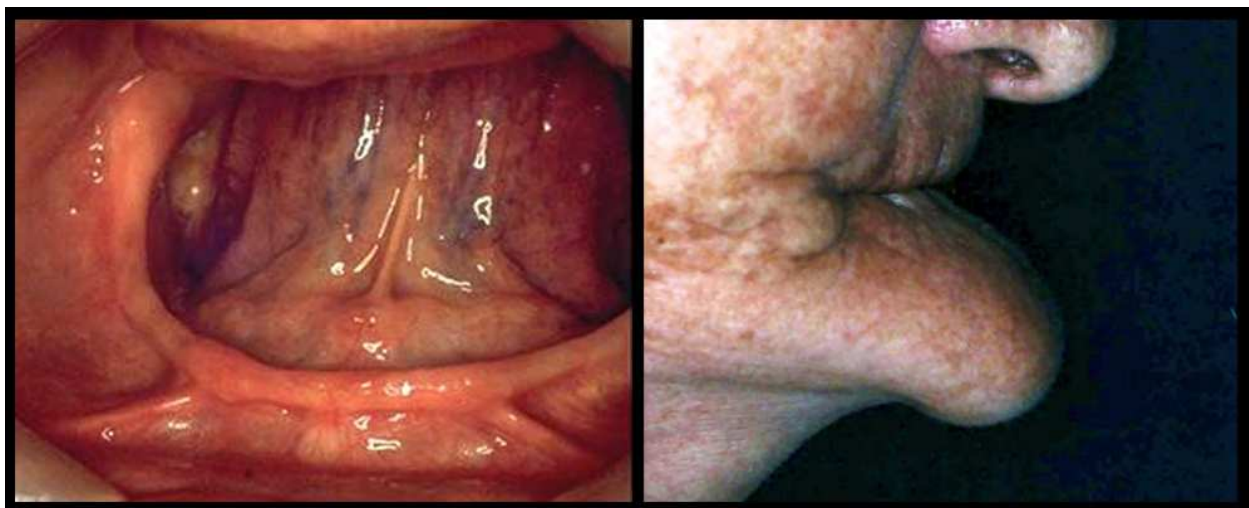


Fig. 5. Intraoral and extraoral view of edentulous patient with referred otic symptoms originated in an advanced jaw osseous reabsorption with overclosure prosthesis (not showed) that do not regain a normal intermaxilar vertical dimension. Photography use authorized to the author (Patient's record).

contraction, which opens the Eustachian tube lumen and laterally presses on the medial pterygoid muscle (Figure 7). Sehhati-Chafai-Leuwer et al.<sup>118</sup> 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.

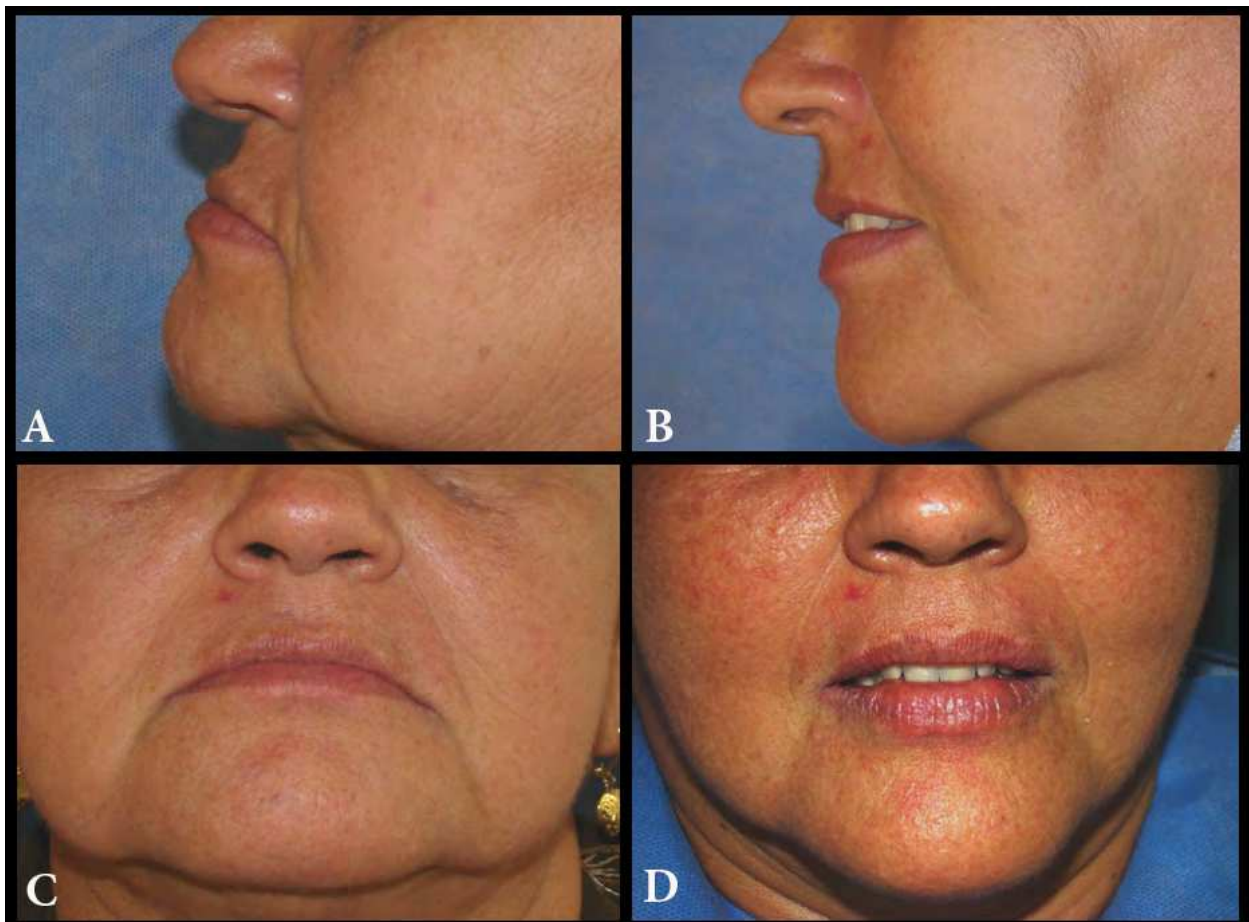


Fig. 6. Frontal and lateral photographic registration with an incorrect (A,C) and a correct complete denture vertical dimension rehabilitation. Photography use authorized to the author (Patient’s record).

A deep overbite, medial pterygoid shortening, hypertonicity, or spasm results in muscular mass compression and lateral bunching of the adjacent TVP and associated structures.<sup>119,120</sup> 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.<sup>121</sup> 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

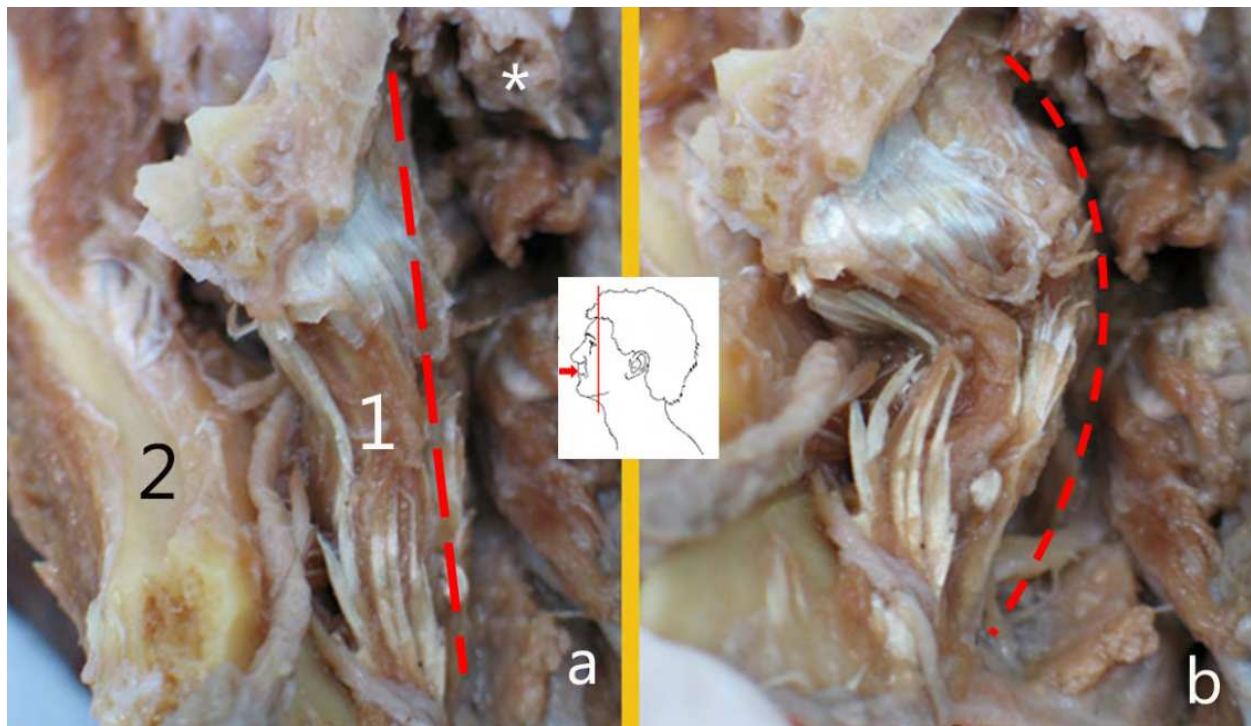


Fig. 7. Jaw and pharyngeal area frontal view: Medial Pterygoid and Eustaquian tube dissection. Picture a- Jaw in normal vertical dimension: 1. Medial pterygoid muscle, 2. Sectioned frontal jaw body, \*. Eustaquian tube cartilage. Picture b- Jaw in overclosure position increasing medial pterygoid transverse 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.<sup>122</sup> 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.<sup>123</sup> 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.<sup>124</sup> 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.<sup>125</sup>

#### 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.<sup>126</sup> corroborated Lupin's 1969 findings and Rood and Doyle's<sup>127</sup> 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).<sup>128</sup> The TVP thus has an additional bone origin in the malleus manubrium. Kierner et al.<sup>129</sup> 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).<sup>5</sup> 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.

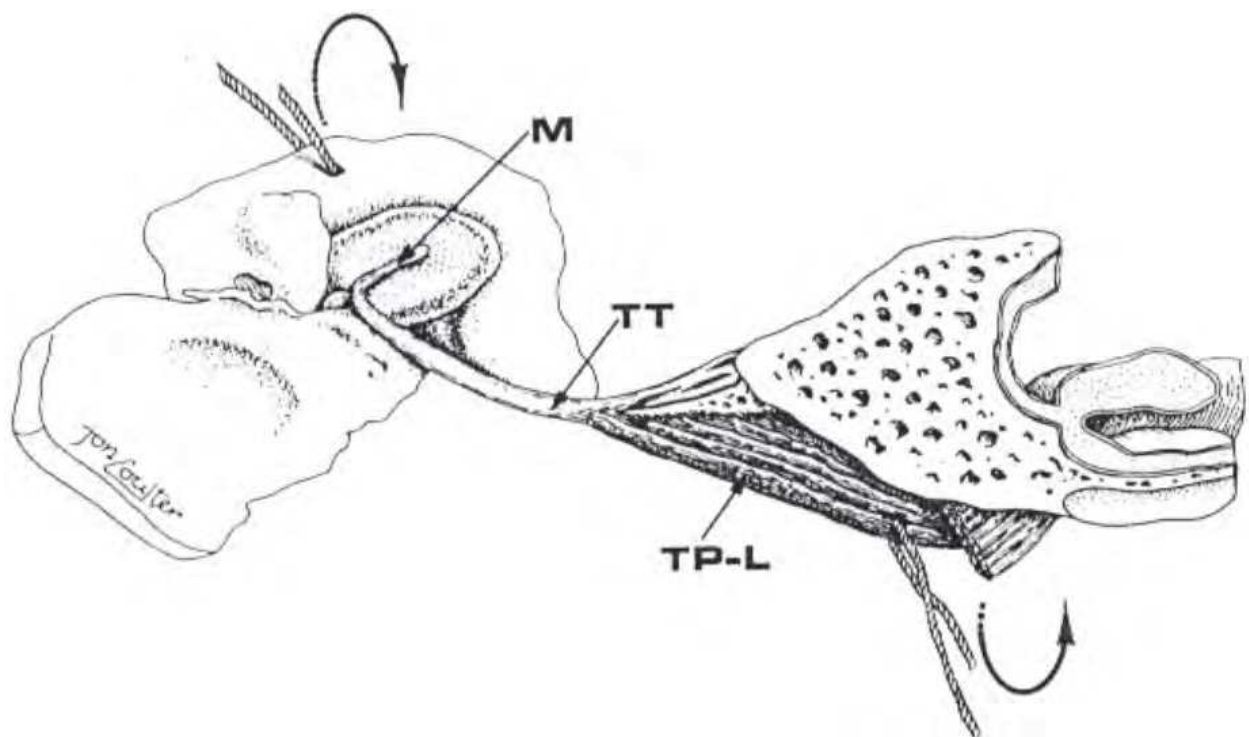


Fig. 8. TT and TVP muscle fiber connection. M. Malleus manubrium, TT. Tensor tympani muscle, TP-L. Tensor veli palatini muscle. Used with author's permission from Rood, S.R. & Doyle, W.J. (1978). Morphology of tensor veli palatini, tensor tympani, and dilatator tubae muscles. *Ann Otol Rhinol Laryngol*,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

### 6.5 Fifth muscular exploration

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

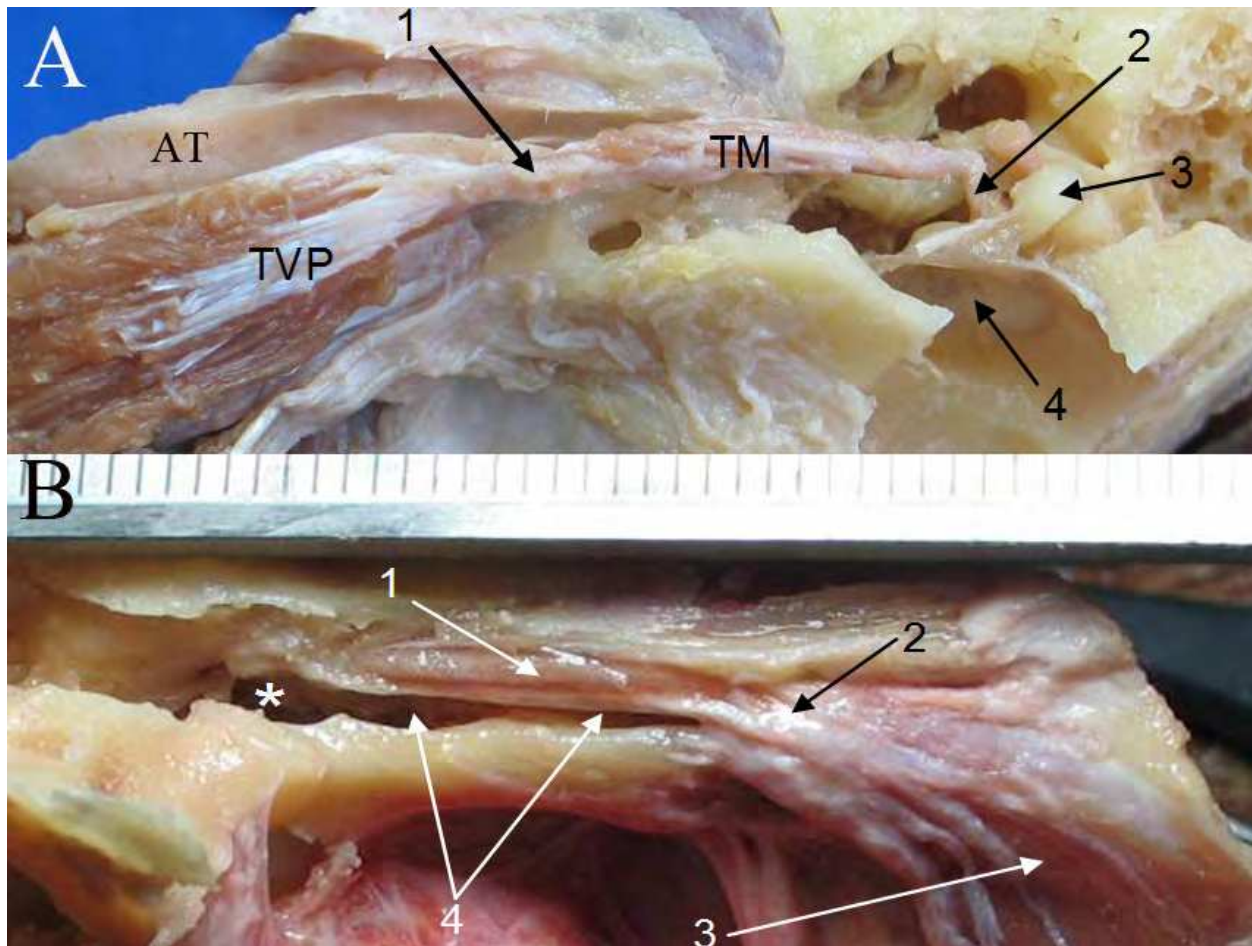


Fig. 9. Dissections on TT and TVP muscle fiber connections. A: AT, Auditory tube, TVP. Tensor veli palatine muscle, TM, Tensor tympani muscle, 1. Fiber connection, 2. Tensor tympani muscle's tendon, 3. Malleus head, 4. Tympanic membrane. B: 1. Tensor tympani muscle, 2. Fiber connection, 3. Tensor veli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

pain. Levine et al.<sup>78</sup> 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.<sup>78,79</sup> 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



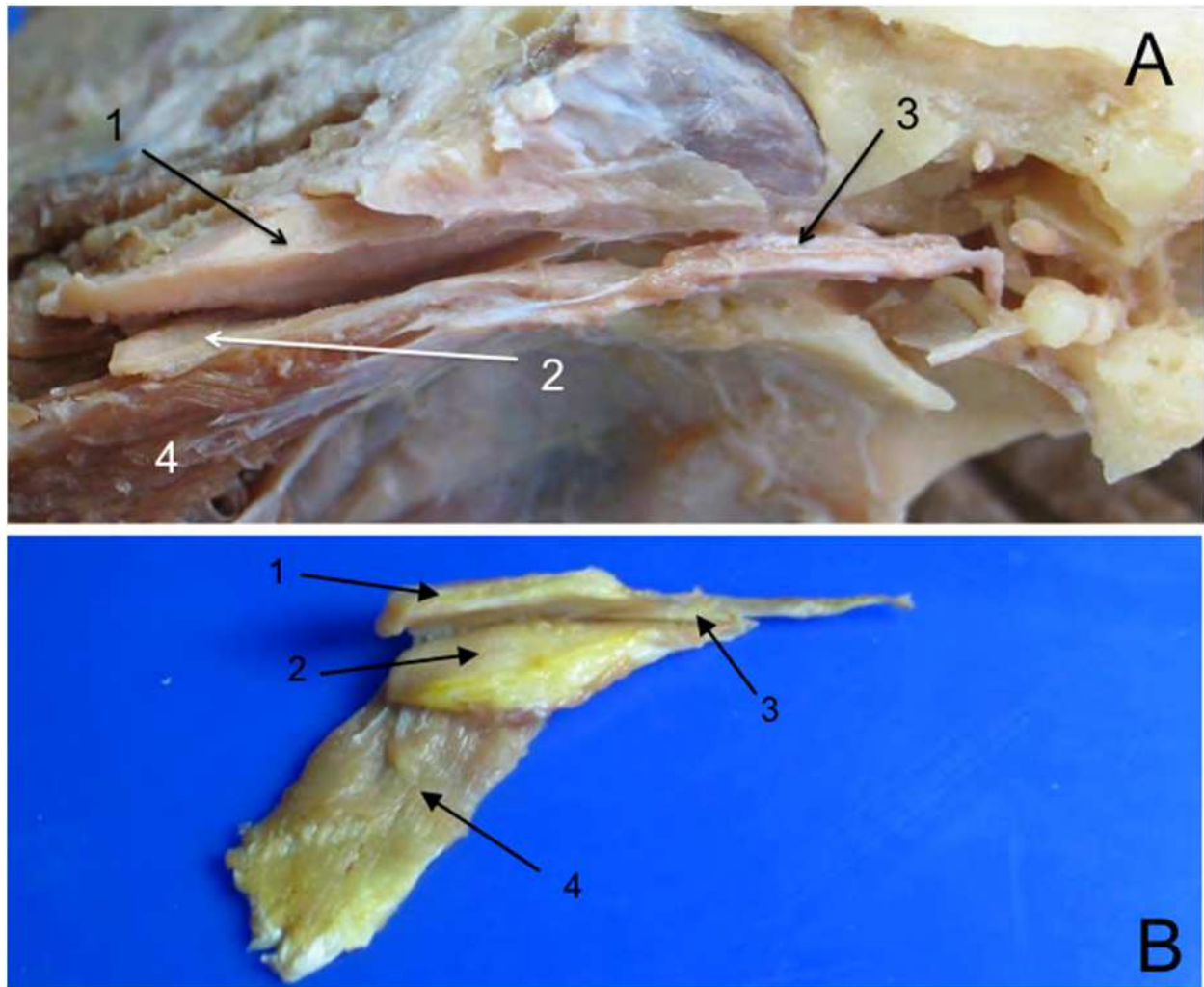


Fig. 10. Dissections on TT and TVP muscles related to left cartilaginous and mucous Eustaquian tube. A: Structure in situ. B: Structure block retired from temporal bone. 1 and 2. Hemisected Eustaquian tube. 3. Tenor tympani muscle, 4. Tensor veli palatine muscle, Author's dissections.

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.<sup>132,133</sup>

### 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.<sup>134,135,136,137,138,139,140,141,142,143,144</sup> 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.<sup>145,146,147,148</sup> These findings is corroborated in 23 human temporal bone specimens (Figure 13) that consistently show these ligamental structures.<sup>149</sup>

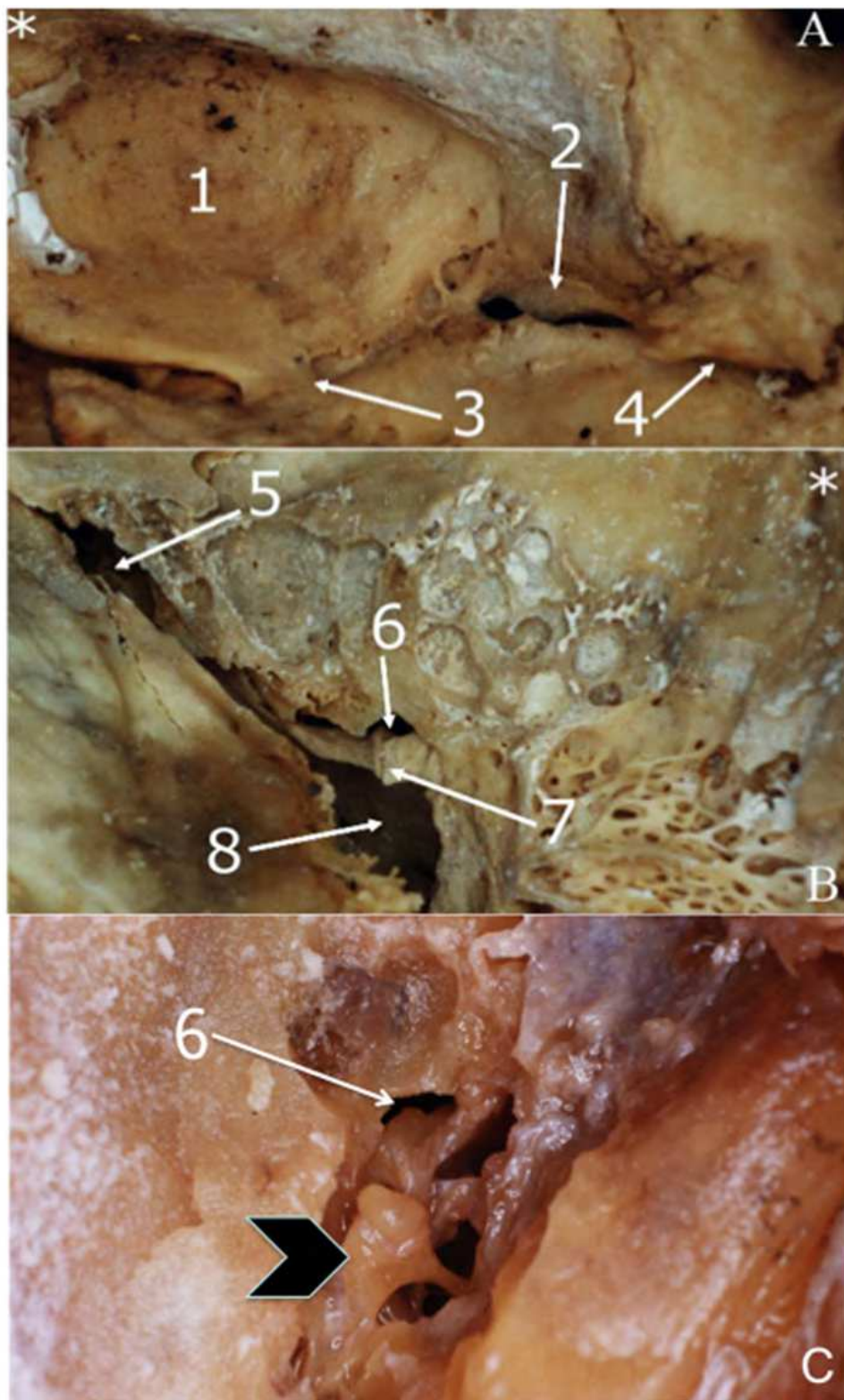


Fig. 11. A. External view of Huguier's canal from TMJ. B. Internal view of Huguier's canal from middle ear. \* Antero-lateral sample orientation. C. Internal fresh view with ossicular chain (Arrow head). 1. TMJ fossae, 2. Huguier's canal external foramen, 3. Scamo-tympanic fissure, 4. Petro-tympanic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



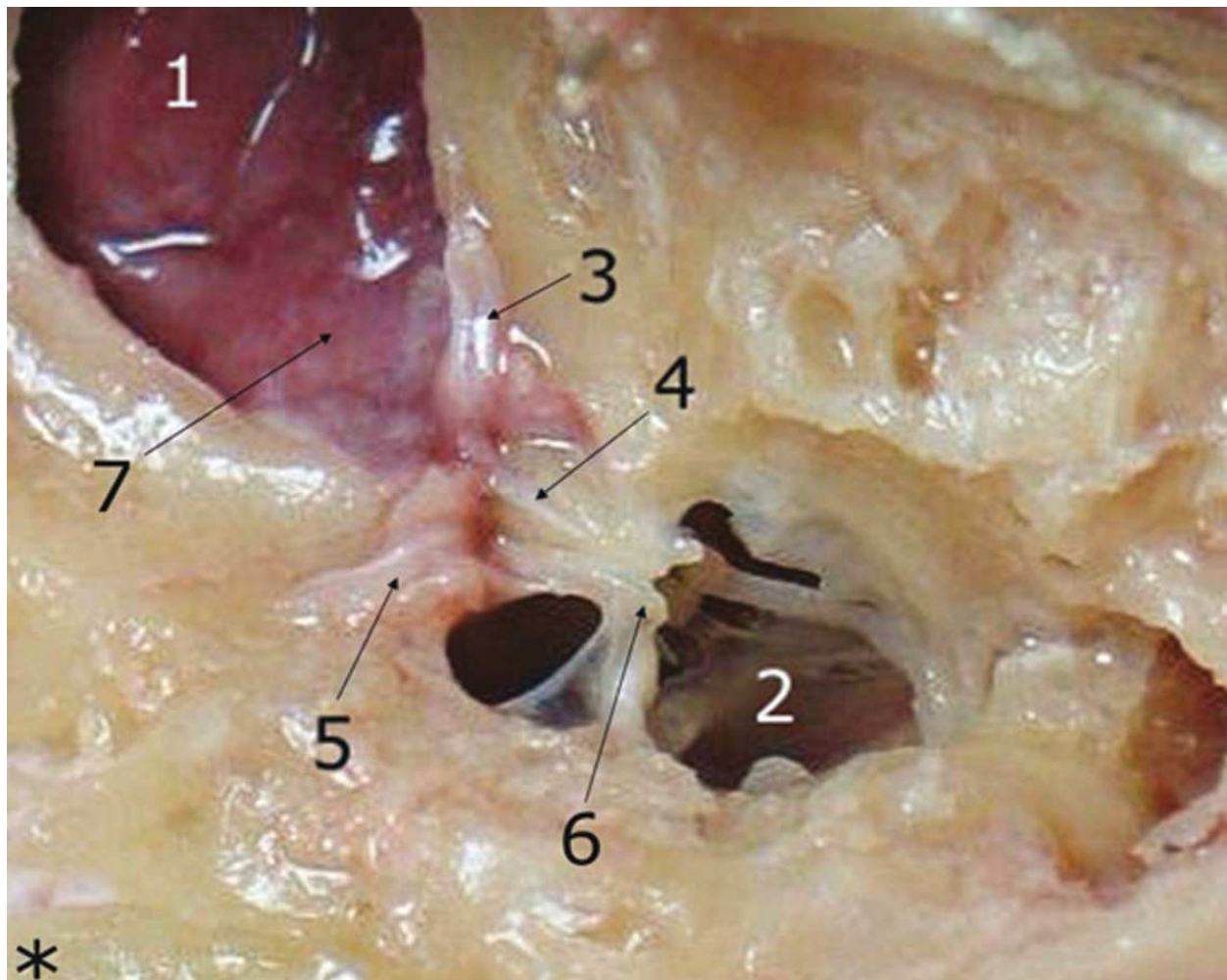


Fig. 12. Superior and postero-medial view of right middle ear and TMJ. \* Postero-medial sample orientation. 1. TMJ disc, 2. Middle ear, 3. DML antero-laterally separated from the lateral side of Huguier's canal, 4. DML and AML Huguier's canal septum, 5. AML antero-medially separated from the medial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

These ligamentous 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.<sup>150,151,152,153</sup> 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.<sup>154</sup> 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.<sup>133,155,156</sup>

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.<sup>157</sup> 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

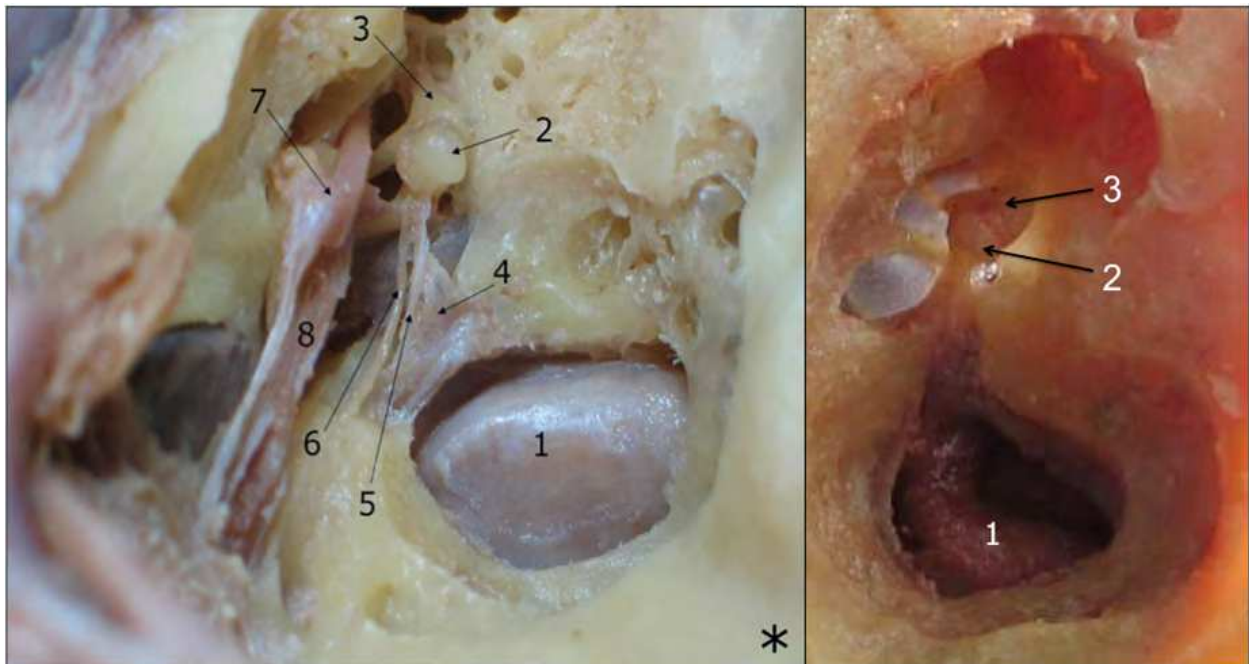


Fig. 13. Superior and antero-medial view of left middle ear and TMJ. \* Antero-lateral sample orientation. 1. TMJ disc, 2. Malleus head, 3. Incus, 4. DML, 5. AML, 6. Chorda tympani nerve, 7. VII cranial pair geniculated ganglion, 8. tensor tympani muscle. Author's dissections.

membrane measurement.<sup>158</sup> 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.<sup>159,160</sup> This finding has been widely corroborated by Wada et al.<sup>161,162</sup> 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 Eckerdal<sup>150</sup>, the range of movement of these ligaments depends on the fibrous connection on the walls of the petrotympanic fissure, thereby corroborating Coleman's findings<sup>138</sup>. Ramirez et al.,<sup>149</sup> findings concern 30.5% DML's malleolar mobility correlated well with Sato et al.<sup>163</sup> 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.<sup>164</sup> 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 annular 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.<sup>165</sup> This experimental model also proved the power of adjustment in sound transmission of this ligament.<sup>166</sup> 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.<sup>163</sup> and Eckerdal<sup>150</sup> 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 Eckerdal<sup>150</sup> (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.,<sup>62</sup> 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. Cheng<sup>167</sup> 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.<sup>155,156,168</sup> Ren et al.,<sup>169</sup> found a significant correlation between internal TMJ derangement and tinnitus, detecting disk luxation in the ipsilateral joint of 53 patients with unilateral tinnitus.<sup>170</sup> Kutila et al.<sup>75</sup> 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 sphenomandibular ligament, there is agree with the findings of Abe et al.,<sup>171</sup> 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



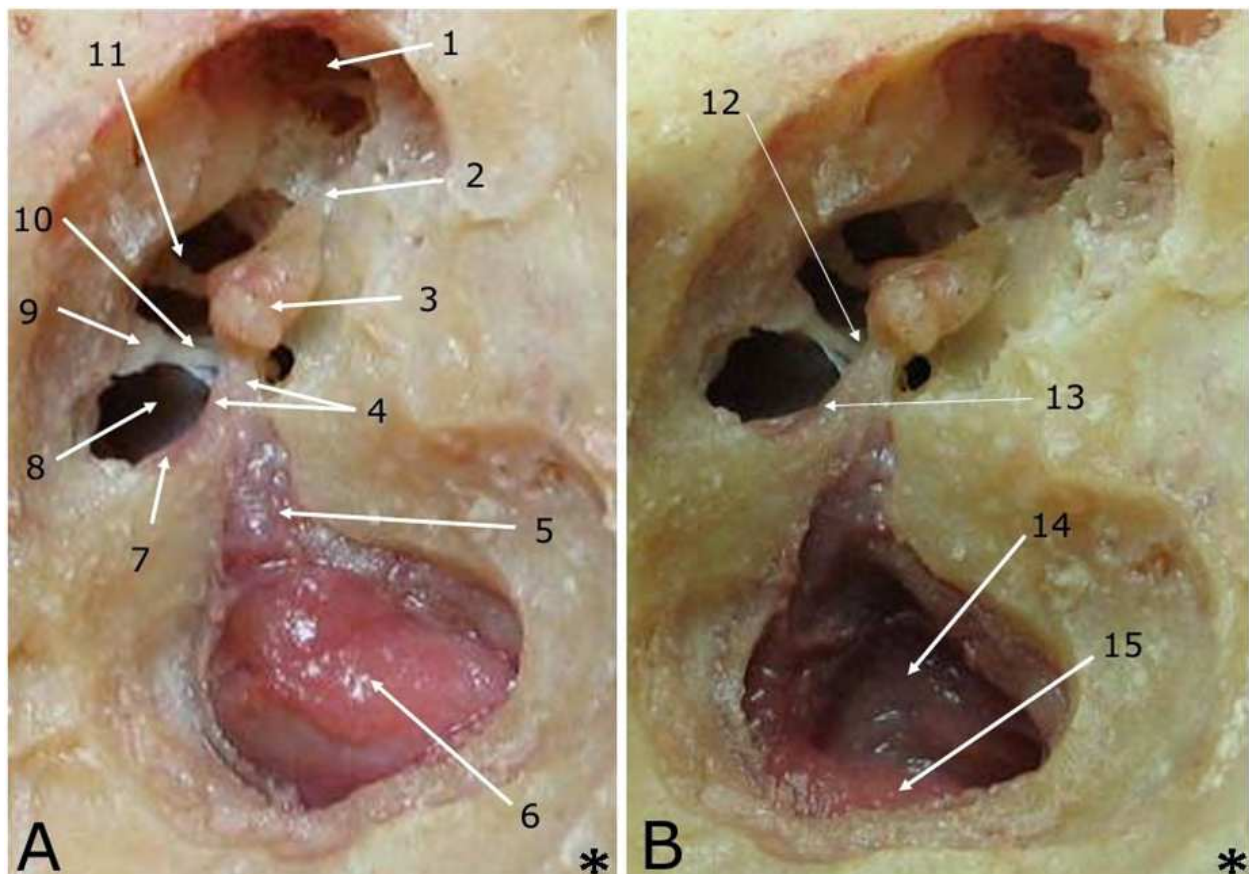


Fig. 14. Superior and antero-medial view of left middle ear and the TMJ. TMJ's disc anterior traction protrusion). \* Antero-lateral sample orientation. Fig. A, 1. Mastoid aditus, 2. Incudal fossa, 3. incudo-malleolar joint, 4. DML (short arrow) and AML (long arrow) emerging from the malleus anterior 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.,<sup>146</sup> studied anterior malleolar and speno-mandibular ligaments structural characteristics in 37 specimens, determining that the speno-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 speno-mandibular (58.3%), suggesting guaranteed connectivity between both ligaments. Burch<sup>145</sup> found that speno-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.<sup>172</sup> Both ligaments can thus be tensioned in several situations, which may alter the ossicular chain,<sup>173</sup> 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

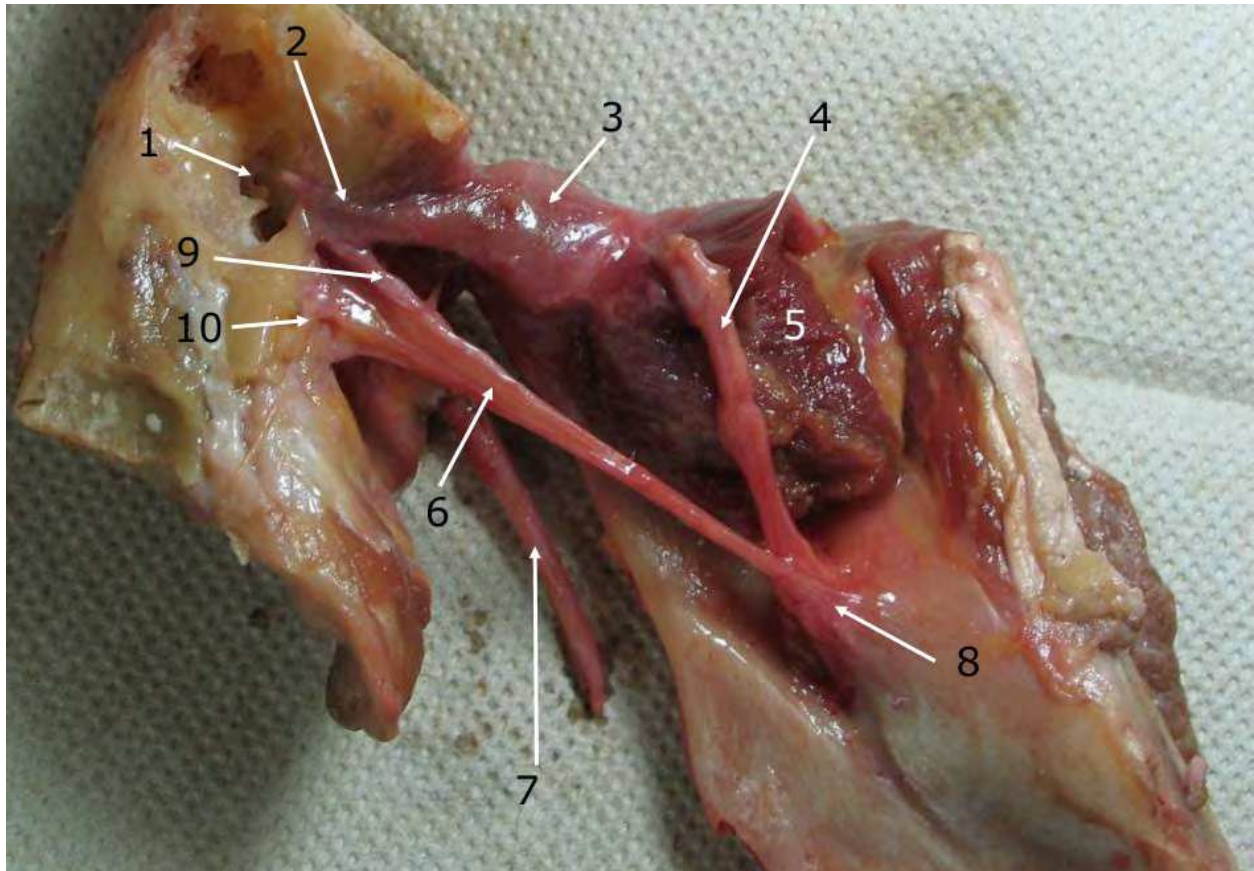


Fig. 15. Left TMJ, mandible ramus and middle ear medial view. 1. Tympanic cavity, 2. DML bilaminar area union, 3. TMJ disc, 4. Alveolar nerve sectioned and retracted over external pterygoid muscle, 5. External pterygoid muscle, 6. EML, 7. Styloid process, 8. Lingula, 9. AML fixing to EML lateral surface,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

external auditory meatus through the petrotympanic fissure, using the same osseous routes as the ligaments, as explained above<sup>141</sup>. Moreover, Merida-Velasco et al.<sup>174</sup> 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 Ham<sup>125</sup> 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.<sup>175</sup> 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.<sup>176,177</sup>

## 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.,<sup>178</sup>

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 et al.,<sup>79</sup> works on somatomotor-somatosensorial tinnitus modulation.<sup>78</sup> However, these modulations were only recognised as otic sensorineuronal 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, psychophysiological, counselling, electrostimulatory, physiotherapy (cervical spine mobility), acupuncture, hypnosis, thermotherapy, cryotherapy, ultrasound-laser therapy, biofeedback, and stomatognathic treatment<sup>68</sup>. 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	N° 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.<sup>20,22,24,35,56,178,179,180</sup> Treatment including oral devices as part of collective management were excluded due to treatment outcome non-specificity.<sup>45,86,170,178,181,182,183,184,185,186</sup>

## 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.

## 11. References

- [1] Ramirez LM, Ballesteros LE, Sandoval GP. Topical Review: Temporomandibular disorders and the multimodal nature of referred otic symptoms. *International Journal of Audiology* 2008;47:215-27
- [2] Costen, J.B. 1934. A syndrome of ear and sinus symptoms dependent upon disturbed function of the temporomandibular joint. *Ann Otol*;43:1-15.
- [3] Myrhaug, H. 1964. The incidence of ear symptoms in cases of malocclusion and temporo-mandibular joint disturbances. *Br J Oral Surg*;2:28-32.
- [4] Ramirez LM, Ballesteros LE, Sandoval GP. Morphological expression of disco-malleolar and anterior malleolar ligaments. A direct anatomical study. *Int J Morphol* 2009;27:367-79.
- [5] Ramirez LM, Ballesteros LE, Sandoval GP. Tensor veli palatini and tensor tympani: Anatomical, functional and symptomatically link. *Act Esp Otorrinolaringol* 2010;61:26-33.
- [6] Ramirez LM, Ballesteros LE, Sandoval GP. Tensor tympani muscle: Extrange chewing muscle. *Med Oral Patol Oral Cir Bucal* 2007;12:E96-E100

- [7] Ramirez LM, Sandoval GP, Ballesteros LE, Gonzales A, Muños G. Treatment and follow up of referred otic symptomatology in 23 patients with diagnosed temporomandibular disorders. *Aud Med* 2006;4:73-81
- [8] Lund JP, Lavigne GJ, Dubner R, Sessle BJ. *Orofacial pain: From basic science to clinical management*. 1st ed. Quintessence Publishing Co, Inc. Chicago, 2001
- [9] Skeppar J, DDS. Treatment of craniomandibular disorders in children and young adults. *J Orofacial Pain* 1993;7:362-369.
- [10] Youniss S, DDS. The relationship between craniomandibular disorders and otitis media in children. *The J Craniomandib Pract*. April 1991, Vol.9 No.2: 169-173
- [11] Dao TT. Gender differences in pain. *J Orofac Pain* 2000;14:169-184
- [12] Pergamalian A., Thomas R., Hussein Z., Greco C. The association between wear facets, bruxism, and severity of facial pain in patients with temporomandibular disorders. *Journal of Prosthetic Dentistry* August 2003, Vol. 90, No. 2:194-200
- [13] Okeson JP ed. *Management of temporomandibular disorders and occlusion*. Ed 4, St. Louis: Mosby;1998. p. 149-77
- [14] Major M. A controlled daytime challenge of motor performance and vigilance in sleep bruxers. *J Dent Res* 1999;78:1754-62
- [15] Kato T, Rompre R. Sleep bruxism: and oromotor activity secondary to micro-arousal. *J Dent Res* 2001;80:1940-44
- [16] Ware JC. Destructive bruxism: Sleep stage relationship. *Sleep* 1988;11:172-81
- [17] Bailey DR. Tension headache and bruxism in the sleep disordered patient. *Cranio* 1990;8:174-82
- [18] Bernhardt O, Gesch D, Schwahn C, Bitter K, Mundt T, Mack F , Kocher T, Meyer G, Hensel E, John U. Signs of temporomandibular disorders in tinnitus patients and in a population-based group of volunteers: results of the Study of Health in Pomerania. *J Oral Rehabil* 2004;31:311-9
- [19] dos Reis AC, Hotta TH, Ferreira-Jeronimo RR, de Felicio CM, Ribeiro RF. Ear symptomatology and occlusal factors: A clinical report. *J Prosthet Dent* 2000;83: 21-4
- [20] Bernstein JM, Mohl ND, Spiller H. Temporomandibular joint dysfunction masquerading as disease of ear, nose, and throat. *Trans Am Acad Ophthalmol Otolaryngol* 1969;73 1208-17
- [21] Cooper BC, Cooper DL. Recognizing otolaryngologic symptoms in patients with temporomandibular disorders. *Cranio* 1993;11:260-7
- [22] Cooper BC, Alleva M, Cooper DL, Lucente FE. Myofacial pain dysfunction: analysis of 476 patients. *Laryngoscope* 1986;96:1099-2106
- [23] Gelb H, Calderone JP, Gross SM, Kantor ME. The role of the dentist and the otolaryngologist in evaluating temporomandibular joint syndromes. *J Prosthet Dent* 1967;18: 497-503
- [24] Gelb H, Arnold GE. Syndromes of the head and neck of dental origin. I. Pain caused by mandibular dysfunction. *AMA Arch Otolaryngol* 1959;70:681-91
- [25] Keersmaekers K, De Boever JA, Van Den BL. Otagia in patients with temporomandibular joint disorders. *J Prosthet Dent* 1996;75:72-6
- [26] Youniss S. The relationship between craniomandibular disorders and otitis media in children. *Cranio* 1991;9:169-73

- [27] Costen JB. A syndrome of ear and sinus symptoms dependent upon disturbed function of the temporomandibular joint. *Ann Otol* 1934;43:1-15
- [28] Costen JB. A syndrome of ear and sinus symptoms dependent upon disturbed function of the temporomandibular joint. *Ann Otol Rhinol Laryngol* 1997;106:805-19
- [29] Camparis CM, Formigoni G, Teixeira MJ, de Siqueira JT. Clinical evaluation of tinnitus in patients with sleep bruxism: prevalence and characteristics. *J Oral Rehabil* 2005;32:808-14
- [30] Goodfriend DJ. Symptomatology and treatment of abnormalities of the mandibular articulation. *Dent Cosmos* 1933;75:844-52
- [31] Ramirez LM, Ballesteros LE, Sandoval GP. Topical review: temporomandibular disorders in an integral otic symptom model. *Int J Audiol* 2008;47:215-27
- [32] Abou-Atme YS, Zawawi KH, Melis M. Prevalence, intensity, and correlation of different TMJ symptoms in Lebanese and Italian subpopulations. *J Contemp Dent Pract* 2006;7:71-8
- [33] Brookes GB, Maw AR, Coleman MJ. Costen's syndrome'--correlation or coincidence: a review of 45 patients with temporomandibular joint dysfunction, otalgia and other aural symptoms. *Clin Otolaryngol Allied Sc* 1980;5:23-36
- [34] Karjalainen M, Le Bell BY, Jamsa T, Karjalainen S. Prevention of temporomandibular disorder-related signs and symptoms in orthodontically treated adolescents. A 3-year follow-up of a prospective randomized trial. *Acta Odontol Scand* 1997;55:319-24
- [35] Bush FM. Tinnitus and otalgia in temporomandibular disorders. *J Prosthet Dent* 1987;58:495-8
- [36] Bush FM. Tinnitus and earache: Long term studies in 105 patients with temporomandibular disorders. *J Dent Res* 1986;(special issue):185.
- [37] Carlsson GE, Kopp S, Wedel A. Analysis of background variables in 350 patients with TMJ disorders as reported in self administered questionnaire. *Community Dent Oral Epidemiol* 1982;10:47-51
- [38] Chole RA, Parker WS. Tinnitus and vertigo in patients with temporomandibular disorder. *ArchOtolaryngol Head Neck Surg* 1992;118:817-21
- [39] Ciancaglini R, Loreti P, Radaelli G. Ear, nose, and throat symptoms in patients with TMD: the association of symptoms according to severity of arthropathy. *J Orofac Pain* 1994;8:293-97
- [40] Friction JR, Kroening R, Haley D, Siegert R. Myofascial pain syndrome of the head and neck: a review of clinical characteristics of 164 patients. *Oral Surg Oral Med Oral Pathol* 1985;60:615-23
- [41] Gelb H, Bernstein I. Clinical evaluation of two hundred patients with temporomandibular joint syndrome". *J Prosthet Dent* 1983;49:234-43
- [42] Gelb H, Bernstein IM. Comparison of three different populations with temporomandibular joint pain-dysfunction syndrome. *Dent Clin North Am* 1983;27:495-503
- [43] Gelb H, Tarte J. A two-year clinical dental evaluation of 200 cases of chronic headache: the craniocervical-mandibular syndrome. *J Am Dent Assoc* 1975;91:1230-6
- [44] Kaygusuz I, Karlidag T, Keles E, Yalcin S, Yildiz M, Alpay HC. Ear symptoms accompanying temporomandibular joint diseases. *Kulak Burun Bogaz Ihtis Derg* 2006;16:205-8



- [45] Koskinen J, Paavolainen M, Raivio M, Roschier J. Otological manifestations in temporomandibular joint dysfunction. *J Oral Rehabil* 1980;7:49-54
- [46] Kuttilla S, Kuttilla M, Le Bell BY, Alanen P, Jouko S. Aural symptoms and signs of temporomandibular disorder in association with treatment need and visits to a physician. *Laryngoscope* 1999;109:1669-73
- [47] Luz JG, Maragno IC, Martin MC. Characteristics of chief complaints of patients with temporomandibular disorders in a Brazilian population. *J Oral Rehabil* 1997;4:240-3
- [48] Manni A, Brunori P, Giuliani M, Modoni M, Bizzi G. Oto-vestibular symptoms in patients with temporomandibular joint dysfunction. Electromyographic study. *Minerva Stomatol* 1996;45:1-7
- [49] Parker WS, Chole RA. Tinnitus, vertigo, and temporomandibular disorders. *Am J Orthod Dentofacial Orthop* 1995;107:153-8
- [50] Principato JJ, Barwell DR. Biofeedback training and relaxation exercises for treatment of temporomandibular joint dysfunction. *Otolaryngology* 1978;86:766-9
- [51] Kelly HT, Goodfriend DJ. Medical significance of equilibration of the masticating mechanism. *J Prosth Dent* 1960;10:496-515.
- [52] Kelly HT, Goodfriend DJ. Vertigo attributable to dental and temporomandibular joint causes. *J Prosthet Dent* 1964;1:159-73.
- [53] Wedel A, Carlsson GE. A four-year follow-up, by means of a questionnaire, of patients with functional disturbances of the masticatory system. *J Oral Rehabil* 1986;13:105-13
- [54] Tuz HH, Onder EM, Kisnisci RS. Prevalence of otologic complaints in patients with temporomandibular disorder. *Am J Orthod Dentofacial Orthop* 2003;123:620-3
- [55] Watanabe EK, Yatani H, Kuboki T, Matsuka Y, Terada S, Orsini MG, Yamashita A. The relationship between signs and symptoms of temporomandibular disorders and bilateral occlusal contact patterns during lateral excursions. *J Oral Rehabil* 1998;25:409-15
- [56] Wright EF, Syms CA, Bifano SL. Tinnitus, dizziness, and nonotologic otalgia improvement through temporomandibular disorder therapy. *Mil Med* 2000;165:733-6
- [57] D'Antonio W, Ikino C, Castro S, Balbani A, Jurado J, Bento R. Distúrbio temporomandibular como causa de otalgia: um estudo clínico. *Rev Bras Otorrinol* 2000;66:46-50.
- [58] de Felício CM, Oliveira JA, Nunes LJ, Jeronymo LF, Ferreira-Jeronymo RR. Alterações auditivas relacionadas ao zumbido nos distúrbios otológicos e da articulação temporomandibular. *Rev Bras Otorrinolaringol* 1999;65:141-46.
- [59] de Felício CM, Faria TG, Rodrigues da Silva MA, de Aquino AM, Junqueira CA. Temporomandibular Disorder: relationship between otologic and orofacial symptoms. *Rev Bras Otorrinolaringol* 2004;70:787-95.
- [60] de Felício CM, Melchior Mde O, Ferreira CL, Da Silva MA. Otologic symptoms of temporomandibular disorder and effect of orofacial myofunctional therapy. *Cranio* 2008;26:118-25
- [61] de Felício CM, de Oliveira MM, da Silva MA. Effects of orofacial myofunctional therapy on temporomandibular disorders. *Cranio* 2010;28:249-59
- [62] Riga M, Xenellis J, Peraki E, Ferekidou E, Korres S. Aural symptoms in patients with temporomandibular joint disorders: multiple frequency tympanometry provides

- objective evidence of changes in middle ear impedance. *Otol Neurotol* 2010;31:1359-64
- [63] Pekkan G, Aksoy S, Hekimoglu C, Oghan F. Comparative audiometric evaluation of temporomandibular disorder patients with otological symptoms. *J Craniomaxillofac Surg* 2010;38:231-4
- [64] Bernhardt O, Mundt T, Welk A, Köppl N, Kocher T, Meyer G, Schwahn C. Signs and symptoms of temporomandibular disorders and the incidence of tinnitus. *J Oral Rehabil* 2011; Article published online: DOI: 10.1111/j.1365-2842.2011.02224.x
- [65] Bruto LH, Kós AO, Amado SM, Monteiro C, Lima MA de. Alterações otológicas nas desordens têmporo-mandibulares. *Rev Bras Otorrinolaringol* 2000;66:327-32.
- [66] Pascoal MI, Rapoport A, Chagas JF, Pascoal MB, Costa CC, Magna LA. Prevalência dos sintomas otológicos na desordem temporomandibular: estudo de 126 casos. *Rev Bras Otorrinolaringol* 2001;67:627-33.
- [67] Salvetti G, Manfredini D, Barsotti S, Bosco M. Otologic symptoms in temporomandibular disorders patients: is there evidence of an association-relationship?. *Minerva Stomatol* 2006;55:627-37
- [68] Rubinstein B. Tinnitus and craniomandibular disorders--is there a link?. *Swed Dent J Suppl* 1993;95:1-46
- [69] Lam DK, Lawrence HP, Tenenbaum HC. Aural symptoms in temporomandibular disorder patients attending a craniofacial pain unit. *J Orofac Pain* 2001;15:146-57
- [70] Bjerne A, Agerberg G. Craniomandibular disorders in patients with Meniere's disease: a controlled study. *J Orofac Pain* 1996;10:28-37
- [71] Lockwood AH, Salvi RJ, Coad ML, Towsley ML, Wack DS, Murphy BW. The functional neuroanatomy of tinnitus: evidence for limbic system links and neural plasticity. *Neurology* 1998;50:114-20
- [72] Vernon J, Griest S, Press L. Attributes of tinnitus that may predict temporomandibular joint dysfunction. *Cranio* 1992;10:282-7
- [73] Levine RA. Somatic (craniocervical) tinnitus and the dorsal cochlear nucleus hypothesis. *Am J Otolaryngol* 1999;20:351-62
- [74] Hazell JW. Patterns of tinnitus: medical audiologic findings. *J Laryngol Otol Suppl* 1982;4:39-47
- [75] Kuttilla S, Kuttilla M, Le Bell BY, Alanen P, Suonpaa J. Recurrent tinnitus and associated ear symptoms in adults. *Int J Audiol* 2005;44:164-70
- [76] Vass Z, Shore SE, Nuttall AL, Miller JM. Direct evidence of trigeminal innervation of the cochlear blood vessels. *Neuroscience* 1998;84:559-67
- [77] Shore SE, Vass Z, Wys NL, Altschuler RA. Trigeminal ganglion innervates the auditory brainstem. *J Comp Neurol* 2000;419:271-85
- [78] Levine RA, Abel M, Cheng H. CNS somatosensory-auditory interactions elicit or modulate tinnitus. *Exp Brain Res* 2003;153:643-48
- [79] Abel MD, Levine RA. Muscle contractions and auditory perception in tinnitus patients and nonclinical subjects. *Cranio* 2004;22:181-91
- [80] Shore SE, Zhou J. Somatosensory influence on the cochlear nucleus and beyond. *Hear Res* 2006;216-17:90-9
- [81] Young ED, Nelken I, Conley RA. Somatosensory effects on neurons in dorsal cochlear nucleus. *J Neurophysiol* 1995;73:743-65

- [82] Kanold PO, Young ED. Proprioceptive information from the pinna provides somatosensory input to cat dorsal cochlear nucleus. *J Neurosci* 2001;21:7848-58
- [83] Moller AR, Moller MB, Yokota M. Some forms of tinnitus may involve the extralemniscal auditory pathway. *Laryngoscope* 1992;102:1165-71
- [84] El-Kashlan HK, Shore SE. Effects of trigeminal ganglion stimulation on the central auditory system. *Hear Res* 2004;189:25-30
- [85] Peroz I. Otaglia and tinnitus in patients with craniomandibular dysfunctions. *HNO* 2001;49:713-8
- [86] Sobhy OA, Koutb AR, Abdel-Baki FA, Ali TM, El Raffa RI, Khater AH. Evaluation of aural manifestations in temporomandibular joint dysfunction. *Clin Otolaryngol Allied Sci* 2004;29:382-5
- [87] Kaltenbach JA, Zhang J, Finlayson P. Tinnitus as a plastic phenomenon and its possible neural underpinnings in the dorsal cochlear nucleus. *Hear Res* 2005;206:200-26
- [88] Zhou J, Shore S. Projections from the trigeminal nuclear complex to the cochlear nuclei: a retrograde and anterograde tracing study in the guinea pig. *J Neurosci Res* 2004;8:901-7
- [89] Kaltenbach JA. The dorsal cochlear nucleus as a participant in the auditory, attentional and emotional components of tinnitus. *Hear Res* 2006;216-17:224-34
- [90] Lockwood AH, Salvi RJ, Burkard RF. Tinnitus. *N Engl J Med* 2002;347:904-10
- [91] Jeanmonod D, Magnin M, Morel A. Low-threshold calcium spike bursts in the human thalamus. Common physiopathology for sensory, motor and limbic positive symptoms. *Brain* 1996;119:363-75
- [92] Cacace AT. Expanding the biological basis of tinnitus: crossmodal origins and the role of neuroplasticity. *Hear Res* 2003;75:112-32
- [93] Muhlnickel W, Elbert T, Taub E, Flor H. Reorganization of auditory cortex in tinnitus. *Proc Natl Acad Sci USA* 1998;95:10340-3
- [94] Kato T, Thie NM, Huynh N, Miyawaki S, Lavigne GJ. Topical review: sleep bruxism and the role of peripheral sensory influences. *J Orofac Pain* 2003;17:191-213
- [95] Carra MC, Rompré PH, Kato T, Parrino L, Terzano MG, Lavigne GJ, Macaluso GM. Sleep bruxism and sleep arousal: an experimental challenge to assess the role of cyclic alternating pattern. *J Oral Rehabil* 2011 Feb 7 [Epub ahead of print]
- [96] Jastreboff PJ. Phantom auditory perception (tinnitus): mechanisms of generation and perception. *Neurosci Res* 1990;8:221-54
- [97] Proctor B. Embryology and anatomy of the eustachian tube. *Arch Otolaryngol* 1967;86:503-14
- [98] Strickland EM, Hanson JR, Anson BJ. Branchial sources of auditory ossicles in man. I. Literature. *Arch Otolaryngol* 1962;76:100-22
- [99] Rodriguez Vazquez JF, Merida V, Jimenez CJ. A study of the os goniale in man. *Acta Anat (Basel)* 1991;142:188-92
- [100] Rodriguez Vazquez JF, Merida V, Jimenez CJ. Relationships between the temporomandibular joint and the middle ear in human fetuses. *J Dent Res* 1993;72:62-6
- [101] Yuodelis RA. The morphogenesis of the human temporomandibular joint and its associated structures. *J Dent Res* 1966;45:182-91



- [102] Thilander B, Carlsson GE, Ingervall B. Postnatal development of the human temporomandibular joint. I. A histological study. *Acta Odontol Scand* 1976;34:117-26
- [103] Arlen H. The otomandibular syndrome: a new concept. *Ear Nose Throat J* 1977;56:60-2
- [104] Salen B, Zakrisson JE. Electromyogram of the tensor tympani muscle in man during swallowing. *Acta Otolaryngol* 1978;85:453-5
- [105] Ramirez LM, Ballesteros LE, Sandoval GP. Tensor veli palatini and tensor tympani muscles: anatomical, functional, and symptomatic links. *Acta Otorrinolaringol Esp* 2010;61:26-33
- [106] Zipfel TE, Kaza SR, Greene JS. Middle-ear myoclonus. *J Laryngol Otol* 2000;114:207-9
- [107] Pau HW, Punke C, Zehlicke T, Dressler D, Sievert U. Tonic contractions of the tensor tympani muscle: a key to some non-specific middle ear symptoms? Hypothesis and data from temporal bone experiments. *Acta Otolaryngol* 2005;125:1168-75
- [108] Chan SW, Reade PC. Tinnitus and temporomandibular pain-dysfunction disorder", *Clin Otolaryngol Allied Sci* 1994;19:370-80
- [109] Kamerer DB. Electromyographic correlation of tensor tympani and tensor veli palatini muscles in man. *Laryngoscope* 1978;88:651-62
- [110] Jung HH, Han SH, Nam SY, Kim YH, Kim JI. Myosin heavy chain composition of rat middle ear muscles. *Acta Otolaryngol* 2004;24:569-73
- [111] Van den Berge H, Kingma H, Kluge C, Marres EH. Electrophysiological aspects of the middle ear muscle reflex in the rat: latency, rise time and effect on sound transmission. *Hear Res* 1990;48:209-19
- [112] Ochi K, Ohashi T, Kinoshita H. Acoustic tensor tympani response and vestibular-evoked myogenic potential. *Laryngoscope* 2002;112:2225-9
- [113] Malkin DP. The role of TMD dysfunction in the etiology of middle ear diseases. *Int J Orthod* 1987;25:21-1
- [114] Klockhoff I, Anderson H. Reflex activity in the tensor tympani muscle recorded in man; preliminary report. *Acta Otolaryngol* 1960;51:184-8
- [115] Ogutcen-Toller MO, Juniper RP. Audiological evaluation of the aural symptoms in temporomandibular joint dysfunction. *J Craniomaxillofac Surg* 1993;21:2-8
- [116] Dolowitz DA, Ward JW, Fingerle CO, Smith CC. The role of muscular incoordination in the pathogenesis of the temporomandibular joint syndrome. *Laryngoscope* 1964;74:790-801
- [117] Goto TK, Yahagi M, Nakamura Y, Tokumori K, Langenbach GE, Yoshiura K. In vivo cross-sectional area of human jaw muscles varies with section location and jaw position. *J Dent Res* 2005;84:570-5
- [118] Sehhati-Chafai-Leuwer S, Wenzel S, Bschorer R, Seedorf H, Kucinski T, Maier H, Leuwer R. Pathophysiology of the Eustachian tube--relevant new aspects for the head and neck surgeon. *J Craniomaxillofac Surg* 2006;34:351-4
- [119] Sharav Y, Tzukert A, Refaeli B. Muscle pain index in relation to pain, dysfunction, and dizziness associated with the myofascial pain-dysfunction syndrome. *Oral Surg Oral Med Oral Pathol* 1978;46:742-7
- [120] Henderson DH, Cooper JC, Bryan GW, Van Sickels JE. Otologic complaints in temporomandibular joint syndrome. *Arch Otolaryngol Head Neck Surg* 1992;118:1208-13

- [121] Penkner K, Kole W, Kainz J, Schied G, Lorenzoni M. The function of tensor veli palatini muscles in patients with aural symptoms and temporomandibular disorder. An EMG study. *J Oral Rehabil* 2000;27:344-8
- [122] McDonnell JP, Needleman HL, Charchut S, Allred EN, Roberson DW, Kenna MA, Jones D. The relationship between dental overbite and eustachian tube dysfunction. *Laryngoscope* 2001;111:310-16
- [123] Azadani PN, Jafarimehr E, Shokatbakhsh A, Pourhoseingholi MA, Ghougeghi A. The effect of dental overbite on eustachian tube dysfunction in Iranian children. *Int J Pediatr Otorhinolaryngol* 2007;71:325-31
- [124] Holborow C. Eustachian tubal function: changes throughout childhood and neuromuscular control. *J Laryngol Otol* 1975;89:47-55
- [125] Marasa FK, Ham BD. Case reports involving the treatment of children with chronic otitis media with effusion via craniomandibular methods. *Cranio* 1988;6:256-70
- [126] Barsoumian R, Kuehn DP, Moon JB, Canady JW. An anatomic study of the tensor veli palatini and dilatator tubae muscles in relation to eustachian tube and velar function. *Cleft Palate Craniofac J* 1988;35:101-10
- [127] Rood SR, Doyle WJ. Morphology of tensor veli palatini, tensor tympani, and dilatator tubae muscles. *Ann Otol Rhinol Laryngol* 1978;87:202-10
- [128] Rood SR. The morphology of M. tensor veli palatini in the five-month human fetus. *Am J Anat* 1973;138:191-5
- [129] Kierner AC, Mayer R, Kirschhofer K. Do the tensor tympani and tensor veli palatini muscles of man form a functional unit? A histochemical investigation of their putative connections. *Hear Res* 2002;165:48-52
- [130] Eriksson PO, Zafar H, Haggman-Henrikson B. Deranged jaw-neck motor control in whiplash-associated disorders. *Eur J Oral Sci* 2004;112:25-32
- [131] de Wijer WA, Steenks MH, de Leeuw L, Bosman F, Helders PJ. Symptoms of the cervical spine in temporomandibular and cervical spine disorders. *J Oral Rehabil* 1996;23:742-50
- [132] Loughner BA, Larkin LH, Mahan PE. Nerve entrapment in the lateral pterygoid muscle. *Oral Surg Oral Med Oral Pathol* 1990;69:299-306
- [133] Williamson EH. Interrelationship of internal derangements of the temporomandibular joint, headache, vertigo, and tinnitus: a survey of 25 patients. *Cranio* 1990;8:301-6
- [134] Pinto OF. A new structure related to the temporomandibular joint and middle ear. *J Prosthet Dent* 1962;12:95-103
- [135] Komori E, Sugisaki M, Tanabe H, Katoh S. Discomalleolar ligament in the adult human. *Cranio* 1986;4:299-305
- [136] Loughner BA, Larkin LH, Mahan PE. Discomalleolar and anterior malleolar ligaments: possible causes of middle ear damage during temporomandibular joint surgery. *Oral Surg Oral Med Oral Pathol* 1989;68:14-22
- [137] Ioannides CA, Hoogland GA. The disco-malleolar ligament: a possible cause of subjective hearing loss in patients with temporomandibular joint dysfunction. *J Maxillofac Surg* 1983;11:227-31
- [138] Coleman RD. Temporomandibular joint: relation of the retrodiskal zone to Meckel's cartilage and lateral pterygoid muscle. *J Dent Res* 1970;49:626-30
- [139] Morgan DH, Goode RL, Christiansen RL, Tiner LW. The TMJ-ear connection. *Cranio* 1995;13:42-3

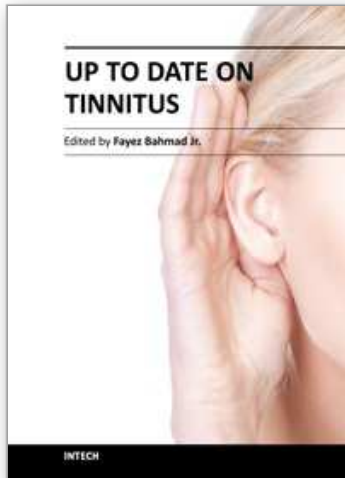
- [140] Ogutcen-Toller MO, Keskin M. Computerized 3-dimensional study of the embryologic development of the human masticatory muscles and temporomandibular joint. *J Oral Maxillofac Surg* 2000;58:1381-6
- [141] Merida V, Rodriguez Vazquez JF, Jimenez CJ. Anterior tympanic artery: course, ramification and relationship with the temporomandibular joint. *Acta Anat (Basel)* 1997;158:222-26
- [142] Perry HT, Xu Y, Forbes DP. The embryology of the temporomandibular joint. *Cranio* 1985;3:125-32
- [143] Rodriguez-Vazquez JF, Merida-Velasco JR, Merida-Velasco JA, Jimenez-Collado J. Anatomical considerations on the discomalleolar ligament. *J Anat* 1998;192:617-21
- [144] Sencimen M, Yalçın B, Doğan N, Varol A, Okçu KM, Ozan H, Aydıntuğ YS. Anatomical and functional aspects of ligaments between the malleus and the temporomandibular joint. *Int J Oral Maxillofac Surg* 2008;37:943-7
- [145] Burch JG. The cranial attachment of the sphenomandibular (tympanomandibular) ligament. *Anat Rec* 1966;156:433-7
- [146] Alkofide EA, Clark E, el-Bermani W, Kronman JH, Mehta N. The incidence and nature of fibrous continuity between the sphenomandibular ligament and the anterior malleolar ligament of the middle ear. *J Orofac Pain* 1997;11:7-14
- [147] Ogutcen-Toller M. The morphogenesis of the human discomalleolar and sphenomandibular ligaments. *J Craniomaxillofac Surg* 1995;23:42-6
- [148] Rodriguez Vazquez JF, Merida Velasco JR, Jimenez Collado J. Development of the human sphenomandibular ligament. *Anat Rec* 1992;233:453-60
- [149] Ramirez LM, Ballesteros LE, Sandoval GP. Morphological expression of discomalleolar and anterior malleolar ligaments. A direct anatomical study. *Int J Morphol* 2009;27:367-79
- [150] Eckerdal O. The petrotympanic fissure: a link connecting the tympanic cavity and the temporomandibular joint. *Cranio* 1991;9:15-22
- [151] Kim HJ, Jung HS, Kwak HH, Shim KS, Hu KS, Park HD, Park HW, Chung IH. The discomalleolar ligament and the anterior ligament of malleus: an anatomic study in human adults and fetuses. *Surg Radiol Anat* 2004;26:39-45
- [152] Cheynet F, Guyot L, Richard O, Layoun W, Gola R. Discomalleolar and malleomandibular ligaments: anatomical study and clinical applications. *Surg Radiol Anat* 2003;25:152-7
- [153] Rodriguez Vazquez JF, Merida V, Jimenez CJ. Development of the human sphenomandibular ligament. *Anat Rec* 1992;233:453-60
- [154] Libin B. The cranial mechanism and its dental implications. *Int J Orthod* 1984;22:7-11
- [155] Johansson, A.S., Isberg, A. & Isacsson, G. (1990). A radiographic and histologic study of the topographic relations in the temporomandibular joint region: implications for a nerve entrapment mechanism. *J Oral Maxillofac Surg*, 48, 953-961.
- [156] Myers, L.J. (1988). Possible inflammatory pathways relating temporomandibular joint dysfunction to otic symptoms. *Cranio*, 6, 64-70.
- [157] Tonndorf J, Khanna SM. Tympanic-membrane vibrations in human cadaver ears studied by time-averaged holography. *J Acoust Soc Am* 1972;52:1221-33
- [158] Sun Q, Gan RZ, Chang KH, Dormer KJ. Computer integrated finite element modeling of human middle ear. *Biomech Model Mechanobiol* 2002;1:109-22



- [159] Dalhoff E, Turcanu D, Zenner HP, Gummer AW. Distortion product otoacoustic emissions measured as vibration on the eardrum of human subjects. *PNAS* 2007;104:1546-51
- [160] Bekesy GV. *Experiments in Hearing*. New York, McGraw-Hill, 1960
- [161] Koike T, Wada H, Kobayashi T. Modeling of the human middle ear using the finite-element method. *J. Acoust Soc Am* 2002;111:1306-17
- [162] Wada H, Ando M, Takeuchi M, Sugawara H, Koike T, Kobayashi T, Hozawa K, Gemma T, Nara M. Vibration measurement of the tympanic membrane of guinea pig temporal bones using time-averaged speckle pattern interferometry. *J Acoust Soc Am* 2002;111:2189-99
- [163] Sato I, Arai H, Asaumi R, Imura K, Kawai T, Yosue T. Classifications of tunnel-like structure of human petrotympanic fissure by cone beam CT. *Surg Radiol Anat* 2008;30:323-6
- [164] Nakajima HH, Ravicz ME, Rosowski JJ, Peake WT, Merchant SN. Experimental and clinical studies of malleus fixation. *Laryngoscope* 2005;115:147-54
- [165] Zhao F, Wada H, Koike T, Ohyama K, Kawase T, Stephens D. Middle ear dynamic characteristics in patients with otosclerosis. *Ear Hear* 2002;23:150-8
- [166] Huber A, Koike T, Wada H, Nandapalan V, Fisch U. Fixation of the anterior malleolar ligament: diagnosis and consequences for hearing results in stapes surgery. *Ann Otol Rhinol Laryngol* 2003;112:348-55
- [167] Cheng T, Gan RZ. Mechanical properties of anterior malleolar ligament from experimental measurement and material modeling analysis. *Biomech Model Mechanobiol* 2008;7:387-94
- [168] Morgan DH. Tinnitus of TMJ origin: a preliminary report. *Cranio* 1992;10:124-29
- [169] Ren YF, Isberg A. Tinnitus in patients with temporomandibular joint internal derangement. *Cranio* 1995;13:75-80
- [170] Wright EF, Bifano SL. Tinnitus improvement through TMD therapy. *J Am Dent Assoc* 1997;128:1424-32
- [171] Abe S, Ouchi Y, Ide Y, Yonezu H. Perspectives on the role of the lateral pterygoid muscle and the sphenomandibular ligament in temporomandibular joint function. *Cranio* 1997;15:203-7
- [172] Ouchi Y, Abe S, Sun-Ki R, Agematsu H, Watanabe H et al. Attachment of the sphenomandibular ligament to bone during intrauterine embryo development for the control of mandibular movement. *Bull Tokyo Dent Coll* 1998;39:91-4
- [173] Burch JG. Activity of the accessory ligaments of the temporomandibular joint. *J Prosthet Dent* 1970;24:621-8
- [174] Merida-Velasco JR, Rodriguez-Vazquez JF, Merida-Velasco JA, Jimenez-Collado J. The vascular relationship between the temporomandibular joint and the middle ear in the human fetus. *J Oral Maxillofac Surg* 1999;57:146-53
- [175] Scolozzi P, Becker M, Richter M. Temporomandibular joint osteoarthritis: a cause of a serous otitis media? A case report. *J Oral Maxillofac Surg* 2004;62:97-100
- [176] Regev E, Koplewitz BZ, Nitzan DW, Bar-Ziv J. Ankylosis of the temporomandibular joint as a sequela of septic arthritis and neonatal sepsis. *Pediatr Infect Dis J* 2003;22:99-101

- [177] Takes RP, Langeveld AP, Baatenburg de Jong RJ. Abscess formation in the temporomandibular joint as a complication of otitis media. *J Laryngol Otol* 2000;114:373-5
- [178] Rubinstein B, Carlsson GE. Effects of stomatognathic treatment on tinnitus: a retrospective study. *Cranio* 1987;5:254-9
- [179] Ramirez LM, Sandoval GP, Ballesteros LE, Gonzales A, Muños G. Treatment and follow up of referred otic symptomatology in 23 patients with diagnosed temporomandibular disorders. *Aud Med* 2006;4:73-81
- [180] Kuttilla M, Le Bell Y, Savolainen-Niemi E, Kuttilla S, Alanen P. Efficiency of occlusal appliance therapy in secondary otalgia and temporomandibular disorders. *Acta Odontol Scand* 2002;60:248-54
- [181] Bjorne, A. & Agerberg, G. (2003a). Reduction in sick leave and costs to society of patients with Meniere's disease after treatment of temporomandibular and cervical spine disorders: a controlled six-year cost-benefit study. *Cranio*, 21, 136-43.
- [182] Bjorne, A. & Agerberg, G. (2003b). Symptom relief after treatment of temporomandibular and cervical spine disorders in patients with Meniere's disease: a three-year follow-up. *Cranio*, 21, 50-60
- [183] Erlandsson, S.I., Rubinstein, B. & Carlsson, S.G. (1991). Tinnitus: evaluation of biofeedback and stomatognathic treatment. *Br J Audiol*, 25:151-61.
- [184] Kerstein RB. Treatment of myofascial pain dysfunction syndrome with occlusal therapy to reduce lengthy disclusion time--a recall evaluation. *Cranio* 1995;13:105-15
- [185] Tullberg M, Ernberg M. Long-term effect on tinnitus by treatment of temporomandibular disorders: a two-year follow-up by questionnaire. *Acta Odontol Scand* 2006;64:89-96
- [186] Kaygusuz I, Karlidag T, Keles E, Yalcin S, Yildiz M et al. Ear symptoms accompanying temporomandibular joint diseases. *Kulak Burun Bogaz Ihtis Derg* 2006;16:205-8

IntechOpen



### **Up to Date on Tinnitus**

Edited by Prof. Fayeze Bahmad

ISBN 978-953-307-655-3

Hard cover, 186 pages

**Publisher** InTech

**Published online** 22, December, 2011

**Published in print edition** December, 2011

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.

#### **How to reference**

In order to correctly reference this scholarly work, feel free to copy and paste the following:

Luis Miguel Ramirez Aristeguieta (2011). Tinnitus and a Linked Stomatognathic System, Up to Date on Tinnitus, Prof. Fayeze Bahmad (Ed.), ISBN: 978-953-307-655-3, InTech, Available from:  
<http://www.intechopen.com/books/up-to-date-on-tinnitus/tinnitus-and-a-linked-stomatognathic-system>

**INTECH**  
open science | open minds

#### **InTech Europe**

University Campus STeP Ri  
Slavka Krautzeka 83/A  
51000 Rijeka, Croatia  
Phone: +385 (51) 770 447  
Fax: +385 (51) 686 166  
[www.intechopen.com](http://www.intechopen.com)

#### **InTech China**

Unit 405, Office Block, Hotel Equatorial Shanghai  
No.65, Yan An Road (West), Shanghai, 200040, China  
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元  
Phone: +86-21-62489820  
Fax: +86-21-62489821



© 2011 The Author(s). Licensee IntechOpen. This is an open access article distributed under the terms of the [Creative Commons Attribution 3.0 License](#), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

IntechOpen

IntechOpen