

Theories on Otic Symptoms in Temporomandibular Disorders: Past and Present

Teorías de los Síntomas Óticos en los Trastornos Temporomandibulares: Pasado y Presente

*Luis Miguel Ramirez Aristeguieta; **Germán Pablo Sandoval Ortiz & ***Ballesteros, L. E.

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SUMMARY: For many years otic symptoms have been closely observed in TMD clinical practice. This review includes trials that meet eligibility criteria in the field of otic symptoms diagnosed and treated by a stomatognathic focal point. The aim is centered on the implications of the debated cause-effect connection between otic symptoms and TMD offering a probable bond between the TMJ, ear and adjacent structures. The rationalization is based on an embryological and anatomical approach that offers explanations on the otic pathogenic mechanisms symptoms interconnected with TMD. The accompanying reciprocity among disciplines of odontology and otolaryngology is brought together by TMD and otic referred mutual symptomatology as is showed in multiple trials. This research is an attempt that targets the integration of these two health specialties in the diagnosis and treatment of this type of population.

KEY WORDS: Tinnitus; Otic fullness; Vertigo; Temporomandibular Disorders; Referred Pain.

INTRODUCTION

Temporomandibular disorders (TMD) are a sub-classification of musculoskeletal disorders and include a wide range of orofacial conditions with multiple etiologies that mask a great variety of subjective signs and symptoms. These can be referred isolated or combined from the temporomandibular joint (TMJ), the masticatory muscles, the cervical muscles and associated structures. TMD and its referred symptomatology in their acute-chronic condition can seriously affect quality of life. The causal relationship between heterotopic pain and the masticatory system is found in large numbers of adult patients as well as in the infant population. These symptoms appear similarly in adults as in children, (Youniss, 1991; Lipton *et al.*, 1993; Skeppar & Nilner, 1993; McNeill, 1997) but the prevalence of TMD pain can range between 1.5 to 2 times higher in women than in men. (Solberg *et al.*, 1979; Klausner, 1994; Dao & LeResche, 2000; Lund *et al.*, 2001).

TMD usually manifests itself as symptoms in the head

and neck, occurring as articular, facial, otic and craniosinusal pain as well as cephalalgic symptoms. These symptoms are also associated with shoulder and back pain, (Bernstein *et al.*, 1969; Hellstrom *et al.*, 2000) which can generate referred orofacial symptomatology by involving the anterior, lateral and suboccipital cervical muscles in maximum occlusion. (Curtis, 1980; Edmeads, 1988; Cooper & Cooper, 1993; Mense, 1993; Santander *et al.*, 1994; Keersmaekers *et al.*, 1996; Dos Reis *et al.*, 2000).

There appears to be a cause and effect relationship between orofacial disorders and dysfunctional masticatory activities such as bruxism and emotional disorders. (Greene & Marbach (1982); Greene & Laskin, 2000). Therefore bruxism (microtrauma resulting from a subconscious non-functional clenching and grinding of the teeth) (Kato *et al.*, 2001) plays a meaningful role in TMD and referred orofacial symptoms although many researchers consider the involvement of bruxism and TMD inconclusive. (Pergamalian *et al.*, 2003).

* Doctor of Odontology with a degree in Prosthetic Dentistry and Temporomandibular Disorders. Professor of Morphology in the Department of Basic Sciences of the Medicine Faculty - Universidad Industrial de Santander (UIS) - Bucaramanga - Colombia.

** Medical Doctor with degree in Otolaryngology. Otolaryngology and Chief Surgeon of Head and Neck Surgery Department in Carlos Ardila Lule Clinic - Bucaramanga - Colombia. Surgery Professor of the Medicine Faculty - Universidad Industrial de Santander (UIS) - Bucaramanga - Colombia. Centro Médico Carlos Ardila Lule, Bucaramanga- Colombia.

*** Medical Doctor with Degree in Anatomy. Basic Sciences Department Director, Professor of Morphology in the Department of Basic Sciences of the Medicine Faculty - Universidad Industrial de Santander (UIS) - Bucaramanga - Colombia.

Although people with TMD suffer local orofacial muscle pain that affects the muscles, they can also suffer referred pain that can affect the cervical and the middle ear muscles with varied symptomatology that ranges from otic symptoms to headache and neck pain. These symptoms can be associated with TMD from muscle hyperactivity that produces primary and referred pain symptomatology and an otic non-painful expression. (Gelb & Arnold, 1959; Okeson, 1998). It is important to know that symptoms of the ear are not present in over 50% of the patients who express otic complaints such as retroauricular and/or preauricular pain, tinnitus, vertigo, hearing impairment, otic fullness, and ear popping sensations. (Gelb *et al.*, 1959; Gelb & Tarte, 1975; Kuttilla *et al.*, 1999; Kuttilla *et al.*, 2001). Bernhardt *et al.*, (2004) found a significant correlation between tinnitus and TMD, affirming that TMD might cause otologic symptoms.

Monson and Wright in 1920 related the position of the jaw and the TMJ to impairment of hearing in child and adult populations. Decker in 1925 and Goodfriend in 1933 related otic symptoms with TMJ. (Decker, 1925; Parker & Chole, 1995. Costen (1934) associated auricular and craniosinusal symptoms with articular disorders called Costen's Syndrome. Costen was the first one to describe otic symptoms in total or partial edentulous patients and the reflex muscular contraction (Shapiro & Truex, 1943) of muscles innervated by the mandibular branch of the trigeminal nerve (V3). Most of the populations are not aware of the relationship existing between symptoms such as headache and otalgia to TMD and the masticatory system. It can be observed in the clinic how functional and inflammatory disorders of TMJ in their acute state are recognized by patients as "ear pain". (Rubinstein *et al.*, 1990; Ash & Pin-to, 1991).

Prevalence and evidence

Patients with otologic symptoms usually have more painful orofacial muscles than in people without otic symptoms. The otic pain in individuals with TMD without a pathological condition in hearing or in the nasopharynx can be explained as a referred pain from the masticatory muscles, TMJ and associated structures. (Miller & Wyrwa, 1992).

There appears to be a connection between otic symptoms and the muscular and articular dysfunction in the head and neck. (Koskinen, *et al.*, 1980) Chart 1 widely demonstrates how otic symptoms are intimately associated with TMD. (Goodfriend, 1933; Principato & Barwell, 1978; Brookes *et al.*, 1980; Gelb & Bernstein, 1983a,b; Friction *et al.*, 1985; Bush, 1986; Wedel & Carlsson, 1986; Chole & Parker, 1992; Kisinisci, 1999; Wright & Syms, 2000; Tuz *et al.*, 2003). It is important to mention that in this chart the

researchers at the beginning of this century recognized TMD patients based on the clinician's preferred etiology hypothesis but the multifactorial nature of TMD began to be acknowledged in 1960 due to a demand for its diagnostic criteria. Between 1990 and 1996 TMD diagnostic criteria has reached a consensus. (Dworkin & LeResche, 1992; McNeill, 1990, 1993; Sessle *et al.*, 1995; Okeson, 1996 and National Institute of Health Technology Assessment Conference on Management of Temporomandibular Disorders).

It is still a mystery the unique cause of otic symptoms. Among a several suggested origins that include Meniere's disease, acoustic neuroma, ear trauma, noise exposure, otosclerosis, ototoxic drugs and presbycusis, TMD appear to play a relevant role. This factor could generate some conflict because the wide range of other causes that may express otic symptoms, cases that are frequently observed in the otolaryngology daily practice. For example Tinnitus and vertigo, as well as other otic symptoms, have been implicated in patients with a compromised systemic condition like systolic and diastolic pressure levels but with no correlation demonstrated (Weiss, 1972; Chatellier, *et al.*, 1982).

The prevalence of dizziness in patients with TMD ranges from 40% to 70% and that of vertigo from 5% to 40%. (Cooper *et al.*, 1986). In relation to tinnitus, it is not clear yet if TMD contributes to its development or if they are the same phenomenon. (Bush, 1987). Lockwood *et al.*, (1998) found up to 75% of patients had alternate intensity of tinnitus with voluntary muscular orofacial movement. Vergara (1996) also found a solid relationship between facial and masticatory muscle movements and the variations in the intensity of tinnitus. (Yamamoto *et al.*, 1985). Morgan (1992) finds that when the muscles of patients with otic symptoms are palpated 75% have trigger points. Rubinstein & Carlsson, 1987) reasoned that tinnitus is not a disease but a symptom in the otologic spectrum. Rubinstein found patients with chronic tinnitus responding less to stomatognathic treatment than those with acute tinnitus. Vernon *et al.* (1992) expressed that in tinnitus the greater the quantity of different sounds then the more complex the tinnitus and TMD. Lam *et al.* (2001) noted that the prevalence of referred otic pain and other otic symptoms of non otologic origin in patients with TMD vary from 3.5% to 42% and that the prevalence of tinnitus in patients with TMD seems to be higher than that found in the general population. The frequency of tinnitus in patients with TMD varies from 33% to 76%. Rubinstein, 1993. Hazell (1981) reported that 39% of the patients with tinnitus showed frequent tension headaches with fatigue and muscle soreness in facial and masticatory muscles. Bjerne *et al.* (Bjerne & Agerberg, 1996;

Bjorne *et al.*, 1998) expressed that the hearing damage and auditive loss frequently associated with tinnitus is strongly related to emotional tension such as headaches, cervical and pericranial muscular tension, as well as dreaming disorders that produce suboccipital muscular tension and vertigo experience.

Rationalized approach to pathogenic mechanisms:

Several authors have tried to find an explanation between otic symptoms and TMD with some interesting answers. How TMD affects otic health may sound illogical to some clinical researchers but must be understood and observed in an anatomic-embryologic-physiologic form to elucidate more than a few possibilities in its potential connection.

Embryology link

Focusing on the articulation and the masticatory muscles as a potential origin of some otic effects it is undeniable that this common embryologic beginning of both the ear and TMJ at least proclaims them as next door neighbors.

In humans the development of TMJ and structures such as the pharynges, the pharyngotympanic tube, and the tympanic cavity is complex and still a controversial subject. Meckel's cartilage, which is the first branchial arch, forms the jaw and the maxillar. The first and second arches form the ossicle chain. In this chain the malleus has a double origin: the anterior process originates from mesenchimal cells (os goniale) through intramembranous ossification, and the rest from Meckel's cartilage, through endochondral ossification. (Rodríguez *et al.*, 1991, 1993; Whyte *et al.*, 2002). The middle auricular bones relate the malleus (hammer), to the condylar blastema and the temporal blastema by fibrous connections passing through the petrotympanic fissure that Rees (Rees, 1954) named the discomalleolar ligament. These fibrous connections of the lateral pterygoid muscle in the Meckel's Cartilage form the interarticular disc by mechanical stimulation of this muscle although there isn't total agreement (Yuodelis, 1966). A neurological, vascular and ligament connections between the TMJ and the middle ear persist in the course of articulation development because of the continuity in the Meckel's Cartilage through the petrotympanic fissure that in adults shows an incomplete closing. The medial pterygoid muscle and the tensor tympani muscle develop from the temporal blastema. These structures along with the tensor veli palatini are innervated by V3 through the otic ganglion, which innervates the masticatory muscles coming from the mesoderm of the first branchial arch. (Baume, 1962; Furstman, 1963; Misurya, 1976;

Thilander *et al.*, 1976; Wong *et al.*, 1985; Ohta *et al.*, 1993; Perry *et al.*, ; Sadler, 1998; Schames *et al.*, 2002). Myrhaug (1964) reasoned that the ossicular chain and the middle ear muscles belong embryologically to the chewing system and that after this they become and serve as the hearing sense.

Muscular involvement

The muscles tensor tympani and tensor veli palatini are the first focus of the functional origin of the otic disorders with symptoms like tinnitus, otic fullness, subjective hearing loss and vertigo. The center of attention of their involvement starts when it is understood that anatomically they are muscles of the middle ear although they are really muscles of mastication because they are modulated by motoneurons coming from the trigeminal motor nucleus (V3). (Campbell *et al.*, 1982; Myrhaug; Eckerdal, 1991; Ciancaglini *et al.*, 1994; Greene, 2001; Travell & Simons, 2002) TMD high muscular activity can be exacerbated originating in this nucleus with its possible otic consequences from the tensor tympani and tensor veli palatine muscles participation.

These complex neuromuscular interactions between the masticatory muscles and the ear were named "Otognatic Syndrome" by Myrhaug in 1964 and then "Otomandibular Syndrome" by Bernstein in 1969 and Arlen in 1977. The patients with otomandibular syndrome have one or more otic symptoms without having a real source in the ear, nose or throat but with one or more mastication muscles in a state of constant spasm.

The stapedial muscles accompany the tensor tympani muscle in the middle ear mechanism. The contraction of the stapedial and tensor tympani muscle is produced during high sounds and immediately before speaking. (Klockhoff & Anderson, 1960). These muscles are involved in the "centrifuge auditory inhibition" control from the Central Nervous System (CNS) that protects and filter the auditory afferent conduction to the CNS by the contraction of these muscles. The stapedial muscle improves external vocalization reducing the masking effect of autogenous sounds. The tensor tympani responds to strong external stimuli and is activated by vocalization, chewing, swallowing and movement of the facial muscles. (Kamerer, 1978; Gray, 1983). It is imperative to recognize that the ossicular chain in the middle ear is practically sustained in a fragile way by the tympanic membrane, some ligaments of the malleus and incus and the tendons of the stapedial and tensor tympani muscles. These structures sustain the bones of the middle ear in an efficient biomechanical function on the acoustic stimulus movement. Myrhaug noted that the equilibrium of the ossicular chain depends on the tonic state of tensor tympani and stapedial muscle (opposing muscles) regulating

the normal functioning of the structures that lead sound into the middle ear. In TMD the sustained contraction of tensor tympani muscle can alter the ossicular spatial position and the endolymphatic pressure through the transmitted changes from the oval window to the labyrinth walls. This can unchain and unbalance the vestibular impulses expressed in vertigo. Additionally, the same middle ear pathogenic muscle mechanism can diminish the sonic transmitting vibration from the tympanic membrane toward the oval window through the ossicular chain which is expressed as a hearing impairment. Ogutcen-Toller & Juniper (1993) suggested that the structures that lead to sound in the middle ear could be affected by the contraction of the tensor tympani muscle produced by TMD. Considering that TMD can produce an episodic or constant muscle contraction or spasm depending on its stage, the otic symptoms appear as an episodic or constant presence.

The dysfunction of the tensor tympani muscle in conjunction with tensor veli palatini muscle also plays an important role in the relationship of TMD and otic symptoms. Neuromuscular dysfunctions of these muscles may trigger alterations in the sound-conducting apparatus. TMD produces contraction and tension in the tensor veli palatini and tensor tympani muscles that can generate different abnormal muscular behaviors: in subjective tinnitus there is a contraction of the tensor tympani muscle and in objective tinnitus where there is a rhythmical opening and closing of the pharyngeal area of the pharyngotympanic (palatal myoclonus), the contraction is present in the tensor tympani and tensor veli palatini muscles. (Virtanen, 1983). Zipfel *et al.* (2000) noted that the most common mechanical origin of objective tinnitus is palatal myoclonus and middle ear myoclonus (rhythmic movement of the tympanic membrane secondary to repetitive contraction of the tensor tympani and stapedial muscles).

The normal function of the pharyngotympanic tube is to equalize the pressure of the middle ear and it is regulated by the tensor veli palatini muscle. The pharyngotympanic tube communicates with the middle ear to the nasopharynx by contracting the tensor veli palatini muscle assisted by the levator palatini muscle during velopharyngeal movements such as swallowing and the inhaling phase of respiration. (Misurya, 1976; Swarts, 1990; Spauwen *et al.*, 1991; Su *et al.*, 1993; Ishijima *et al.*, 2002). Movement patterns such as yawning, laughing, swallowing and coughing involve pharyngeal and laryngeal muscles that activate the tensor tympani. Salen & Zakrisson (1978) found that pharyngeal and laryngeal muscles work simultaneously during swallowing with the tensor tympani and assist in Eustachian tube ventilation similar to an air pump. During its reciprocal contraction generates an inner deflection of the tympanic

membrane that breaks the seal of the mucosal membranes of the pharyngotympanic tube isthmus that expels air and contributes to the ventilation of the middle ear. The alteration of its function can change the intratympanic pressure and can generate tinnitus, vertigo, sensations of hearing loss of low tones, otic fullness. Its dysfunction can also produce otitis media with effusion, especially in children. (Alper *et al.*, 1997). Marasa & Ham (1988) as well as Youniss think the pharyngotympanic tube dysfunction plays an important role in otitis media with effusion. Children are more susceptible to chronic otitis media with effusion especially during respiratory system infections because their pharyngotympanic tubes are configured as a short, horizontal and wide lumen. (Holborow, 1975).

Costen noted an interesting explanation why the Eustachian tube dysfunction would occur by neighboring compression. The compression comes about because of the relaxation of the upper head of the lateral pterygoid and medial pterygoid muscles that occurs in a loss of vertical dimension from edentulism. (Dolowitz *et al.*, 1964). McDonnell *et al.* (2001) express in the same vertical scenario that children with deep dental overbites were 2.8 times more increased risk for developing pharyngotympanic tube dysfunction than those without deep bites. The result of this is the compression and bunching of the muscular mass (tensor veli palatini) and associated structures in repose (muscular hypotension) by lateral pterygoid and medial pterygoid muscles. Normally the pharyngotympanic tube is maintained closed by the collapsing pressure of an external mass. The pharyngotympanic tube maintains a closed position at rest, which protects the middle ear from retrograde flow of nasopharynx microflora during rapid fluctuations in nasopharyngeal pressure associated with breathing, swallowing, coughing, sneezing, and nose blowing. Under normal conditions the contraction of the tensor veli palatini muscle opens the lumen of the pharyngotympanic tube while it presses laterally on the medial pterygoid muscle. (Leuwer *et al.*, 2002). Kelly & Goodfriend (1964) found a possible union between vertigo, nausea, vomit and syncope in 105 patients with over-closed dental relations because the medial pterygoid muscle enlarges and compresses, and he found the complete resolution of their symptomatology in 89% of these patients by increasing their vertical dimension. Another picture with the same medial pterygoid muscle and pharyngotympanic tube proximity is the hypertonicity and spastic contraction of medial pterygoid muscle due to muscular dysfunction in TMD. Also, this could externally compress the tensor veli palatini muscle and generate anatomical interferences with the normal function of the pharyngotympanic tube. In this scenario the contraction of the tensor veli palatini muscle can be interrupted and causes difficulty in its opening. (Ross, 1971). If the mastication

muscles are hypertonic because of TMD it is also possible that the tensor veli palatini and tensor tympani muscles can be hypertonic because of the common innervation by V3. As the tensor veli palatini remains spastic its normal function of opening and closing the pharyngotympanic tube will cease generating otic fullness among other otic symptoms.

At this point, the complexity of the middle ear pressure aeration mechanisms can be observed. If the tensor tympani and tensor veli palatine muscle dysfunction can generate otic symptoms each one separately, then the effects of an agonist co-working are anatomically more problematic. Barsoumian, Kuehn *et al.* (1998) corroborate Lupin's 1969 findings and those later of Rood & Doyle (1978) findings by discovering in adult cadavers how the fibers of the most external zone of the tensor veli palatini muscle and the fibers of the tensor tympani are joined in the middle ear. (Rood, 1973; Williams *et al.*, 1989; Prades *et al.*, 1998). Kierner *et al.* (2002) also found this functional connection between the tensor veli palatini and the tensor tympani muscles in human cadavers by histological analysis. (Proctor, 1967). Because of this, the tensor veli palatini has an additional bone origin in the manubrium of the malleus. Consequently these muscles act synergistically together and temporary can increase intratympanic pulling force with otic symptoms like otalgia, tinnitus, vertigo and subjective hearing loss. The dysfunction of them in TMD can either individually or in combination modify the medial position of the malleus and the tympanic membrane since the spatial disposition of the ossicular chain can be modified by muscle force. Schames *et al.* found a possible myofibrotic contraction of the tensor tympani and tensor veli palatini muscles due to dysfunction generated by TMD. This complicates this anatomical and functional relationship as a consequence of reduced muscle fiber length.

TMJ and middle ear adjoining

Dissections of human adults and fetuses carried out by Pinto (1962) Komori *et al.* (1986) and other researchers (Rees, 1954; Coleman, 1970; Ioannides & Hoogland, 1983; Perry *et al.*, 1985; Loughner *et al.*, 1989; Rodríguez & Vásquez, *et al.*, 1993; Morgan *et al.*, 1995; Mérida-Velasco *et al.*, 1997; Rodríguez-Velásquez, *et al.*, 1998) proved a specific anatomical link between the TMJ, the mandibular body and the middle ear. This connection is made by the discomalleolar and the anterior malleolar ligaments that attach to the malleus of the ossicular chain in its anterior process and create a biomechanical connection between the middle ear and the mandible. (Hoshino, 1988).

The anterior malleolar ligament is a continuation of the sphenomandibular (tympano-mandibular) ligament noted by Burch (1966) and according to Komori *et al.* and

Ogutcen-Toller (1995) inserts on the ventral surface of the malleus coming out to form a horizontal "V" with the discomalleolar ligament in the petrotympanic fissure. According to Pinto, the discomalleolar ligament is found more external and is inserted in the medio-posterior-superior border of the capsular ligament and the disc. According to Komori *et al.* it is inserted only in the medio-posterior-superior zone of the retrodiscal area and in the articular capsule. The anterior malleolar ligament is more medial than the discomalleolar ligament and connects the sphenomandibular ligament along with the Chorda Tympani nerve (VII) in the petrotympanic fissure (Iter Chorda Anterioris) trespassing together within the Huguier Channel. The sphenomandibular ligament is inserted in the lingula or mandibular spix spine and originates in the malleus, part of the sphenoid spine and the most medial zone of the petrotympanic fissure. (Rodríguez *et al.*, 1992) The malleolar anterior ligament is an embryological remainder of the Meckel's cartilage and the disc-malleolar ligament is a remainder of the lateral pterygoid muscle.

These structures can be stretched in a TMJ functional or inflammatory disorder affecting the middle ear structures in some patients. The otic symptomatology in this scenario can be expressed as tinnitus, vertigo and otalgia. The first two symptoms start in the altered position of the stape because the force applied to the middle ear bones by these ligaments. Otalgia can be present by peripheral nerve stimulation in the tympanic membrane due to the traction of the malleus that is bonded to the membrane. Ren & Isberg (1995) found a significant correlation between internal derangement of the TMJ and tinnitus. In their research disk displacement was found to be present in the ipsilateral joint in all 53 patients who had unilateral tinnitus. In 1933 Bernstein *et al.* observed how in extreme jaw movements some patients could change the intensity and quality of tinnitus. According to Eckerdal, the range of movement of these ligaments depends on the fibrous connection on the walls of the petrotympanic fissure, corroborating Coleman's findings in 1970.

How these ligaments can start a force in the middle ear bones is the question. This ligament link between the TMJ and the middle ear can be biomechanically subjected to functional or inflammatory articular disorders since the luxation or displacement of the disc and the secondary edema from inflammation (Sessle *et al.*; Myers, 1988; Johansson *et al.*, 1990) can produce anterior tension on the discomalleolar ligament because of the disc disposition and the additional increase of intrarticular pressure by the edema. The sphenomandibular and malleolar anterior ligaments only can be tensed in a position of marked over-closure also stretching the ossicular position, explaining the increase of

tinnitus in exaggerated intermaxilar positions. (Bernstein *et al.*, 1969; Myers, 1988; Rubinstein *et al.*, 1990). Bernstein, observed how in extreme jaw movements some patients could change the intensity and quality of tinnitus. Loughner *et al.* (1989) reasoned that in surgeries for TMJ where condyle is distracted the ossicular chain could be injured by the extreme traction of these ligaments over the anterior process of the malleus. Additionally in relation to the TMJ-Middle ear bone communication (Huguier Channel) Marasa & Ham suggested that the inflammation produced by inflammatory disorders of TMJ can spread through the petrotympanic fissure to the middle ear and generate otitis media. The osseous communication between the middle ear and TMJ is capable subsequently to start pathologies with doubtful origins like a TMJ septic arthritis with an occult original infectious site, but in presence of infectious otitis media. (Leighty *et al.*, 1993; Regev *et al.*, 2003). Using the same bone connection, the edema produced by inflammatory or functional disorders from the TMJ can spread through the petrotympanic fissure to the middle ear a fluid collection and generate vulnerable infection (otitis media) via this route. (Marasa & Ham, 1988; Moses *et al.*, 1998).

In a similar way the proximity of TMJ and the middle ear can be explored in a vascular and lymphatic point of view. These components go through the petrotympanic fissure in the same form like the ligaments explained above. Merida-Velasco *et al.* (Mérida- Velasco *et al.*, 1999) confirmed Bleicker's findings that in newborns the small vessels of the anterior portion of the middle ear cross the petrotympanic fissure and reach the venous retrodiscal plexus that drains into the retromandibular vein, which is a part of the articular vascular plexus. In adults the most medial branches of the posterior group of the tympanic anterior artery (behind TMJ) irrigate the tympanic cavity and the external auditory meatus. The most medial branches are in intimate contact with the discomalleolar ligament that enters the middle ear through the petrotympanic fissure and with the external ear through the most external branches in the scamotympanic fissure. This vascular relationship between TMJ and the middle ear can explain the referred otic symptoms in presence of secondary vascular reflex from TMJ disorders. (Williamson, 1990). Gelb *et al.* (1997), in relation to the labyrinth lymphatic drainage mechanisms and based on Young (1952) discoveries, suggested that the mechanical obstruction of the pharyngotympanic tube by TMJ inflammation or muscular cause could involve the lymphatic channels coming from the labyrinth to the middle ear and the retro-naso-pharyngeal lymph nodes which can diminish the outflow of perilymph. This increases the volume and pressure of the labyrinthine fluid and "hydrops of the inner ear" and consequently expressing an otic symptomatology. In the same way functional or

inflammatory disorders of TMJ could exert pressure on the lymphatic contents coming from the middle ear through the petrotympanic fissure.

As can be seen, the TMJ is a small compacted structure with vascular and neurological tight components that can easily be injured during TMJ disorders. Ash & Pinto reasoned how the otic symptomatology can be generated by injury of the parasympathetic nerve fibers of the auriculotemporal nerve that travel from the otic ganglion and tympanic plexus (glosopharyngeal nerve). When irritated can produce a reflex vascular spasm in the labyrinthine system secondary to abnormal stimulation of these fibers. (Kopp, 2001). The irritated auriculotemporal nerve can produce otalgia because it profusely innervates articulation as well as the tympanic membrane, the anterosuperior zone of the external ear, the tragus and the external part of the ear among other structures that can explain the auricular pain experienced. (Fernández *et al.*, 2003 and Schmidt *et al.*, 1998). Johansson explained that in the nerve entrapment etiology (auriculotemporal nerve) is important not only to anatomical mobility and bone deformities but also inflammation of vascular and muscular structures that can injure closely situated nerves since inflammation can alter and reduce the normal contour and size of the anatomical passages. Loughner *et al.* (1990) confirmed that during lateral pterygoid spasm and hypertrophy, it can injure auriculotemporal nerve

Finally Myers in 1988, found that TMJ inflammation disorders produce a pattern of edema leakage and fibrosis, subsequently, that extends into neighboring areas following the patterns of least resistance through the muscular masses in medial and anterior-medial areas of TMJ. This inflammatory course can compress nerves: alveolar, lingual and chord tympani. Nerve entrapment origin is important not only because of the anatomical mobility and bone deformities but also inflammation of vascular and muscular structures that can injure nerves in close proximity since inflammation can alter and reduce the normal contour and size of the anatomical passages. The fibrosis and adhesions in progress by this inflammation leakage can produce tension in the carotid sheath during jaw and neck movements. This tension is transmitted to the jugular foramen and to the endolymphatic sack, generating pressure over the hair cells of the cochlea producing tinnitus and vertigo. Another pattern of outflow is described which passes between the origin of the levator palatini muscle and the tensor veli palatini muscle toward the most vulnerable area of the Eustachian tube (isthmus). This pressure can generate otic fullness sensation. Myers demonstrates the fragility of the extrabony extensions of the middle ear in the Eustachian Tube and of the internal ear in the endolymphatic sack.

Peripheral and Central Nervous System contribution

Otalgia can be expressed like referred (heterotopic) pain with an orofacial origin that is different to the ear. Miller & Wyrwa (1992) suggested that in the "Theory of Convergence" by Sessle, (Sessle, 2000) the pain referred to the ear (which is secondary to orofacial soreness (Svensson & Graven-Nielsen, 2001) and chronic painful stimulus) begins by chronic irritation of the Peripheral Nervous System (PNS) which alters the normal physiologic functioning of the brain and sensitizes the CNS. These nerves send peripheral nociceptive impulses to second-order neurons through the trigeminal subnucleus caudalis. The sensorial innervation of the ear and periauricular region is derived from cranial nerves V, VII, IX, X, C2, and C3. The neurons of the spinal nucleus of the trigeminal nerve in the brain stem particularly in the subnucleus caudalis and the transition zone between caudal and interpolar subnucleus, receive these orofacial and neck afferents (nociceptive) signals. The central sensitization also involves spinal motor neurons, the thalamus and the somatosensory cortex, which makes a facilitator effect on referred pain and muscle reflex spasms. The "convergence" of these afferent nerves toward the spinal nucleus of the trigeminal, the thalamus and the cortex can confuse the brain as to the location of the sources of peripheral chronic pain by the sensitization of afferent interneurons not related.

Okeson (1998) noted an accumulation of neurotransmitter substance in the interneuronal synapses when pain stimulus is continuous leading to the leakage of neurotransmitter toward groups of adjacent interneurons. The adjacent interneurons are modulated by the action of these neurotransmitters, generating the perception that these impulses are coming from the same pain area. This explains how the originally excited neuron transmits information about the real origin of pain (primary pain) and the other excited neurons show central excitement which means that the perceived pain from these neurons is heterotopic pain, i.e. referred pain.

Woolf & Thompson (1991) reasoned that the pathogenicity of pain is in part a reflex of the CNS neurons capacity to suffer dynamic alterations in the properties of response and that the nociceptive fibers can trigger this functional plasticity. Chronic pain brings about, as a consequence, a reduction in the threshold of the cutaneous afferent ways and an expansion of the peripheral response. The repeated peripheral stimuli of primary afferent fibers produces a progressive increase in the action potential discharged and in this same manner a prolonged enlargement in the excitability of spine and brain stem neurons posterior to deep and chronic pain stimulus. Coderre *et al.* (1993) found

that it is possible that the normal pain inhibition process (Wall, 1978) in the brain stem is altered by peripheral pain reception and that the central sensitization process generates expansion of the perceived peripheral pain.

Taking into account the "Pain Adaptation Model," by Lund *et al.* (1991) and how the chronic musculoskeletal pain present in TMD produces a protective adaptation to avoid further muscular pain, then it can be expected that this mechanism perpetuate the muscle fatigue cycle dysfunction because the larger muscle agonist-antagonist contractions which generate more chronic pain and continues to sensitize the peripheral orofacial receptors. These receptors become involved in the pathophysiology of the central excitatory effect by constant afferent muscle stimulation continuing the recurrent TMD cycle. (Hu *et al.*, 1992, 1993; Svensson *et al.*, 1996, 1998).

The orofacial pain can express its constant input to the CNS in a systemic way. The motor nucleus of various cranial nerves like the facial (VII), glossopharyngeal (IX), vagus (X), and the hypoglossal (XII) including the trigeminal (V) are stimulated by the ventral trigemino-thalamus tract carrying to the cortex nociceptive and sensorial signals in an ascending "divergent" manner coming from the contralateral spinal and principal sensory trigeminal nucleus. (Affifi & Bergman). The importance of this resides on the provoked neural "abnormal muscular reflex behaviors" influenced by chronic muscular-articular-mucosal deep pain toward all the skeletal muscles innervated by this pathway: tongue, intrinsic and extrinsic larynx, pharynx and palatal, tensor and levator veli palatini, some of the styloid process, facial, stapedius (possible stapedial intratympanic mioclonus), tensor tympani, digastrics, mylohyoid and all the agonist muscles of mastication. It will be interesting to determine the coverage of the collateral branch stimulation of the trigemino-thalamus tract on the vital centers (respiratory, metabolic and cardiovascular) and secretomotor activity of the motor nucleus of the X, IX and VII cranial nerves in deep and chronic pain situation.

Symptoms like vertigo, tinnitus and subjective hearing loss can result from a new auditory innervation pattern that involves the trigeminal nerve. Vass *et al.* (1998). found that in animals the trigeminal innervation of the vascular system controls the cochlear and the vestibular labyrinth function. The projection of the ophthalmic fibers of the trigeminal Gasser ganglion to the cochlea through the basilar and anterior inferior cerebellar arteries can play an important role in the vascular tone in quick vasodilatory responses to metabolic stresses like intense noise. The inner ear disease which produces otic symptoms such as sudden hearing loss, vertigo and tinnitus, can originate from the

reduction of cochlear blood flow due to the presence of abnormal activity in the trigeminal ganglion in patients with herpes zoster, migraine or by the central excitatory effect originated in a chronic or deep pain produced by TMD. Vass *et al.* also found that the trigeminal sensorial innervation plays an important role in the regulation and balance of the cochlear vascular tone and in the vestibular labyrinth channel that can be responsible for the symptomatic complexity of some cochlear diseases related to inner ear blood flow. Furthermore the headaches, tinnitus, hearing loss, vertigo and Meniere's syndrome can be explained by this innervation model. A parallel discovery by Shore *et al.* (2000) found that the trigeminal ganglion innervates the ventral cochlear nucleus and the superior olivary complex. This can interfere with the auditory pathways which lead to the auditory cortex in the presence of constant peripheral somatic signals from the ophthalmic and mandibular trigeminal peripheral innervation in TMD. Levine (1999) suggests a similar somatic interference or inhibition of the auditory perception in the dorsal and ventral cochlear nucleus. Young *et al.*, (1995). suggests that this stimulus is so strong that it can interfere with moderate-level acoustic stimuli. The perception originates from spinal and cranial nerves impulses in the medullary somatosensory nucleus and is modulated by muscle fatigue in the head and neck areas. These CNS interactions between the somatosensory system and the auditory system (including the extralemiscal auditory pathway (Moller *et al.*, 1992) can explain the origin of otic symptoms without existing disease in the ear, nose or throat. (Sobhy *et al.*, 2004).

Forceful muscle contraction in TMD can elicit modulations in the neurological auditory perception and body balance. Bjorne *et al.* (1996, 1998) describe a type of vertigo and tinnitus without a cochlear and labyrinth origin. They found that these symptoms can be initiated in the facial and cervical muscular dysfunction in TMD which generates hypertonicity and muscular spasm irritating nerves and blood vessels by muscular trapping. When this compression develops in the neck, it distorts the normal proprioceptive reception to the vestibular nucleus in the oculo-vestibular muscle reflex that controls the postural position of the head. (Cowin & Bryner, 2002). The cervical muscular compression could generate tension on the vertebral artery which feeds the basilar artery and the inner ear inflow. Bjorne suggested that this tension can produce temporary or permanent hearing loss. They reasoned that constant muscular tension is associated with tinnitus and vertigo by neuroplasticity originating in the irritation and posterior peripheral sensitization. (Bjorne & Agerberg, 2003; Salvinelli, *et al.*, 2003).

Tinnitus can result from the interaction between the somatic and otic nervous pathways inside the CNS without

involving the peripheral structures (cochlea and cochlear nerve). Levine suggested that the reduction in the auditory-nerve input leads to desinhibition of the dorsal cochlear nucleus and an increase in spontaneous activity in the central auditory system which is experienced as tinnitus and can be modulated by somatosensory input. Lockwood *et al.* (2002) named this phenomenon "Cross-Modal Inhibition" and suggests that hearing loss leads to reorganization (neuroplasticity) of the pathways in the central auditory system and leads to abnormal interactions between auditory and other central pathways, showing similarities between neuropathic pain and tinnitus with a TMD origin.

DISCUSSION

The development of TMD is multifactorial and is centered in local, systemic and recent psychological factors. Predisposition, initiation and perpetuating factors increase the biomechanic and the neuromuscular aspect of the dysfunction. The psychological foundation in patients with acute and chronic TMD shows how anxiety, depression, and personality alterations add to the complexity of this disorder. Understanding these disorders is developed by fusing the traditional biomedic model with psychological, social and behavioral dimensions of the patients. (Mc Neill, 1997; Greene & Lakin, 2000). An important and potent modulator of TMD are the emotional disorders. (Dworkin *et al.*, 1987; 1987; Dworkin, 1994; Kinney *et al.*, 1992; Gatchel *et al.*, 1996; Molin, 1999). Treatment should emphasis physical management as well as behavior management in relation to this problem. This cascade of events that can start with emotional stress can be expressed in articular-muscular orofacial and cervical pain finally represented like otic symptoms. (Goodfriend, 1933; Myrhaug, 1964; Bernstein *et al.*, 1969; Williamson, 1990; Kutilla *et al.*, 1999) Meniere's syndrome or Endolymphatic Hydrops expressed like tinnitus, vertigo, hearing loss and otic fullness could be explained partially or totally by this research that open a possibility in the therapeutics of this disease. (Myrhaug, 1964; Myers, 1988; Hu *et al.*, 1993; Bjorne & Agerberg, 1996). In orofacial pain the medical team rarely has interest in the muscular or articular orofacial evaluation and its crucial connection with referred cranial symptoms expressed. Otolaryngology and odontological exams are vital to establish differential diagnosis of the orofacial symptoms by TMD. These should incorporate both intraoral and extraoral muscle explorations of the head and neck (Kim *et al.*, 1995) as well as palpation of the muscles and TMJ. (Conti *et al.*, 2002). Also, a physical and visual examination of associated structures such as the pterygoid hamulus of the soft palate, the stylohyoid complex, the temporalis tendon, the sphenopalatinum ganglion,

Table I. Percent of TMD-otic symptoms in different populations

Researchers	N° TMD Patients	% Oralgia	% Tinnitus	% Vertigo	% Hearing Loss	% Ear Fullness
Goodfriend, 1933	91	3	14	5	26	---
Costen, 1934	400	38	13	12	13	6
Gelb-Arnold, 1959	100	13	27	5	---	---
Kelly-Goodfriend, 1960	102	---	---	75.5	---	---
Kelly-Goodfriend, 1964	105	25	44	100	44	30.4
Myrhaug, 1964	1391	21	32	28	32	---
Dolowitz <i>et al.</i> , 1964	64	100	43	5	38	48
Gelb <i>et al.</i> , 1967	742	36	40	20	15	---
Bernstein <i>et al.</i> , 1969	86	93	42	14	33	62
Sharav <i>et al.</i> , 1978	42	---	---	23	---	---
Principato-Barwell, 1978	25	100	44	44	---	---
Koskinen <i>et al.</i> , 1980	47	47	20	26	24	26
Brookes <i>et al.</i> , 1980	45	82	76	33	80	62
Gelb-Bernstein, 1983a	1142	---	41.5	20.6	15.4	---
Gelb-Bernstein, 1983b	200	---	36	40	24.5	48
Friction <i>et al.</i> , 1985	164	42	42	23	17.7	---
Cooper <i>et al.</i> , 1986	476	50	36	40	38	---
Wedel-Carlsson, 1986	148	---	14	15	14	---
Bush, 1986	105	82	33	---	---	---
Bush, 1987	35	---	33	---	---	---
Williams, 1990	25	---	---	44	---	---
Henderson <i>et al.</i> , 1992	21	85.7	90.5	76.1	66.6	90.5
Chole-Parker, 1992	338	100	59	70	---	---
Cooper <i>et al.</i> , 1993	996	63	63	41	25	30
Ogutcen-Toller <i>et al.</i> , 1993	57	40	17	8.7	26	5
Ciancaglini <i>et al.</i> , 1994	797	1.6	6.4	1.6	7.6	---
Ren-Isberg, 1995	53	98.1	100	54.7	---	92.5
Parker-Chole, 1995	338	100	59	70	---	---
Wright <i>et al.</i> , 1997	93	38	38	---	---	---
Kisnisci <i>et al.</i> , 1999	152	66.7	55.5	55.5	27.8	---
De Felicio <i>et al.</i> , 1999	30	53.3	66.6	20	20	76.6
Kuttila <i>et al.</i> , 1999	411	12-16	12-17	---	---	5-9
Wright <i>et al.</i> , 2000	15	15	14	11	---	---
D'Antonio <i>et al.</i> , 2000	90	100	51.1	31.1	---	---
Bruto <i>et al.</i> , 2000	40	75	17.5	---	15	17.5
Lam <i>et al.</i> , 2001	470	32	26	18	12	---
Pascoal <i>et al.</i> , 2001	126	50	50	34	10	52
Tuz <i>et al.</i> , 2003	200	57.2	48.3	42.7	29.4	---
De Felicio <i>et al.</i> , 2004	27	59.2	74	---	---	74.1
Sobhy <i>et al.</i> , 2004	30	56.7	20	---	---	---

paranasal sinuses, the stylomandibular ligament, the parotid gland and the dental structures as well as possible cervical lesions should be included. (Hertz, 1968; Kopstein, 1975; Correl *et al.*, 1979; Shankland, 1987; Ernest *et al.*, 1991; Raustia, 1993; Kunachak, 1995; Peterson *et al.*, 1995; Koelbaek *et al.*, 1999; Farella *et al.*, 2002). These exams are necessary in order to make a certain exclusionary diagnostic approach because of the similarity of orofacial symptoms. The pain present in TMD is a subjective symptom which possesses a relevant diagnostic difficulty, especially under the absence of objective and unique signs which is why a differential diagnosis including the associated structures is imperative.

Solving these problems should be approached carefully and conservatively. Reversible therapies should be the first treatment elected. Appropriate handling of TMD should implement non-invasive therapies. Physical self-regulation can eliminate negative oral habits and maintain more relaxed muscles, avoiding the ischemia and accumulation of algescic intramuscular and articular substances. Patients must be dedicated to their physical treatment and the management of their harmful habits and behavior in relation to their TMD. Conscious lifestyle changes that eliminate the muscular hyperactivity must be implemented. Prevention or reduction of factors such as bruxism, muscle hyperactivity inflammation has vital

importance in any therapeutic treatment that gives the body the opportunity to repair itself and adapt to positive habits. In our daily practice we can observe how this pattern of referred symptomatology continuously repeats and how ultraconservative and conservative treatments restore the original state of health. (McNeill, 1997; Clark *et al.*, 1999; Lockwood *et al.*, 2002).

The orofacial symptoms present in TMD are like a mystery where the symptoms characterize the clues and the current epidemiological information and a good clinical examination constitute the evidence of where it is collected and takes form. Sometimes it is necessary to be ready to renounce some of the procedures and theories accepted as paradigms in professional practice. Only the flexible use of refreshing new knowledge and adoption of a new direction will benefit our patients, and diminish any unavoidable uncertainty in our daily practice.

Although the cause-effect relationship of the referred symptoms has an alliance with TMD continuous learning is still necessary. Many of these rationalization theories are not possible to prove using measuring instruments, but in daily clinical practice they are constantly applied. Costen's 1934 positions are regularly applied and appear to be one of the correct visions of TMD and their connection with referred symptomatology. Even after 68 years these theories give the impression of being applicable in the explanation of some of the orofacial symptoms. The clinicians that lean on anatomical and physiological exploration in their daily practice have given form to each one of these theories. These focuses should be kept in mind as diagnostic tools as they are the understanding we must have about the possible pathogenic approach of these disorders in order for successful treatment. Now then, all this takes in account a deep position when we observe the wide groove that exists on this topic between health disciplines like dentistry and medicine.

RAMIREZ, A. L. M.; SANDOVAL, O. G. P. & BALLESTEROS, L. E. Teorías de los síntomas óticos en los trastornos temporomandibulares: Pasado y presente. *Int. J. Morphol.*, 23(2):141-156, 2005.

RESUMEN: Por muchos años, síntomas óticos observados en la práctica clínica se han relacionado estrechamente con trastornos temporomandibulares (TTM). Esta revisión incluye juicios que plantean criterios específicos en el campo de los síntomas óticos diagnosticados y tratados desde un punto de vista estomatognático. El objetivo está centrado en las implicancias debatidas causa-efecto entre síntomas óticos y TTM estableciendo un probable vínculo entre la ATM, oído y estructuras adyacentes. La revisión está basada en una aproximación embriológica y anatómica que explica los mecanismos y síntomas de patogenia ótica interconectados con TTM. El acompañamiento recíproco entre disciplinas de odontología y otorrinolaringología estableció un equilibrio entre los TTM y la mutua sintomatología ótica remitida, como está mostrado en múltiples diagnósticos. Esta investigación constituye un intento de alcanzar una integración entre estas dos especialidades médicas en el diagnóstico y tratamiento de este tipo de patologías.

PALABRAS CLAVE: Tinnitus; Oído; Vértigo; Trastornos Temporomandibulares; Dolor referido.

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Correspondence to:
 Prof. Dr. Luis Miguel Ramírez Aristeguieta
 Department of Basic Sciences
 Faculty of Medicine
 Universidad Industrial de Santander (UIS)
 Bucaramanga
 Calle 45 No 33-17. 702B.
 Bucaramanga
 COLOMBIA

Email: lmra3@yahoo.com

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