



Tinnitus in patients with temporo-mandibular joint disorder: Proposal for a new treatment protocol



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ABSTRACT

The present study was designed to verify the correlation between tinnitus and temporomandibular joint dysfunction. 86 consecutive patients were enrolled in the study, all affected by subjective tinnitus without hearing impairment, from both genders, age between 18 and 60 years old.

The final number of patients included in the study was 55. All patients received a temporo-mandibular joint examination. All the patients were asked to rate the severity of their symptoms before and after treatment using a VAS scale and the Tinnitus Handicap Inventory (THI) and they followed a standardized protocol for the investigation of tinnitus. All the subjects were monitored by the same researcher and they underwent the same splint treatment. The comparison between pre- and posttreatment phase scores showed in patients with predisposition of TMD and with TMD a statistically significant decrease of THI and VAS values. The characteristics of tinnitus and the degree of response to treatment confirmed the relationship between tinnitus and TMD. The authors believe that, when the most common causes of tinnitus, such as otologic disorders and neurological diseases are excluded, it is correct to evaluate the functionality of the temporo-mandibular joint and eventually treat its pathology to obtain tinnitus improvement or even resolution.

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1. Introduction

Tinnitus is the perception of sound when no actual sound is present; it is a subjective symptom the severity of which is directly related to the description of the patient (Mc Fadden, 1982). Tinnitus is usually described as a ringing noise, but in some patients it takes the form of hissing, humming or whistling sound. The noise is often continuous, in which case it can be the cause of great distress and leads the patient to feel sick or affected by a disease (Douek, 1981). In recent years, scientific research has dealt with the etio-pathogenesis of tinnitus without concrete results, and has reactivated the debate about a patho-physiological mechanism for subjective tinnitus. The most-well accepted theory is that the cause of tinnitus should be sought in an anomaly of

one or more elements of the neural tube that constitute the auditory system (Douek, 1981). Tinnitus can be a symptom of otologic disorders such as an external ear infection, chronic otitis media or otosclerosis, and Meniere's syndrome (Gelb et al., 1997). It can also be a symptom of neurological disorders such as acoustic neuroma, and multiple sclerosis; or alteration in blood pressure and temporo-mandibular disorder (TMD). The relationship between the etiology of tinnitus and TMD is not well known. Muscles of the middle ear as well as chewing and facial muscles share a common embryological origin and functional relationship (Tullberg and Ernberg, 2006). A dysfunction of the Eustachian tube can also lead to diseases of the middle ear, and these can often be related to pathologies of temporo-mandibular joint (TMJ) (Franz and Anderson, 2007; Shehhati-Chafai-Leuwer et al., 2006). Some authors believe that the TMD can be related to the onset of tinnitus through a neuro-anatomical interaction between the neural input of the trigeminal system and dorsal cochlear nucleus (Rubinstein, 1993). Pinto (Pinto, 1962), in 1962, proposed another

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hypothesis for the correlation among tinnitus, cranio-cervical dysfunction, and mandibular dysfunction: the presence of a tiny ligament that connects the malleus head to the upper and postero-medial portion of the capsule and articular disc of the TMJ. Another study (Paparo et al., 2008) on fresh adult cadavers showed that the mandibular nerve is close to the TMJ capsule, particularly at its anterior and medial aspect. At this level, irritating or compressive occurrences acting on the mandibular nerve might cause trigeminal-like symptoms even extended to the entire orofacial region, which could even chronicize at a central nervous system level in some predisposed subjects.

2. Materials and methods

The present prospective study was performed by enrolling 86 consecutive patients, all affected by subjective tinnitus without hearing impairment, from both genders, and between 18 and 60 years of age. Twenty patients did not give the consent for the study, 11 patients were lost to follow-up, and the final number of patients included in the study was therefore 55. The main inclusion criterion was the presence of chronic subjective tinnitus that had lasted at least for the last 12 months. Exclusion otologic criteria were: objective tinnitus, hearing loss, pharmacologic treatment of tinnitus within 4 weeks prior to study entry, any otologic disorder that could cause tinnitus, neurological and oncological diseases, and drug abuse. After applying the exclusion/inclusion otologic criteria all patients underwent a baseline evaluation including detailed medical history, audiometric test, and otorhinolaryngologic examination. In addition, patients received TMJ examination, which consisted in an anamnestic evaluation, objective examination, first instrumental examination (X-Ray orthopantomography, kinesiograph recordings) and possibly a second-level instrumental examination (computed tomography, dynamic magnetic resonance imaging), and they were classified according the Wilkes Classification Table 1 (Wilkes, 1989).

All patients with history of facial trauma, with dental alteration (absence of many or all of the teeth) or a patient with TMD with

Wilkes Classification Stages III, IV, and V were excluded. Patients who had advanced stages of the Wilkes classification, presented a disc dislocation or degenerative joint disease, and would not benefit from the use of a neuromuscular device (Cascone et al., 2013; Rinna et al., 2013; Spallaccia et al., 2013; Tuncel, 2012) were also excluded.

At this point, three groups of patients were identified: without TMD, with a predisposition to TMD, and with TMD (Filiaci et al., 2012). Group I comprised 10 patients with a mean age of 43.9 years (standard deviation [SD] \pm 7.87 years); group II consisted of 30 patients with a mean age of 44.5 years (SD \pm 12.4 years); and the third group consisted of 15 patients with a mean age of 35 years (SD \pm 6.72).

Patients without TMD were patients without a history of facial pain and absence of restriction of TMJ motion; patients with pre-disposition are patients belonging to Wilkes Classification Stage I (painless clicking, no restricted motion); and patients with TMD belong to Wilkes Classification Stage II (occasional painful clicking, intermittent locking, and headaches).

All the patients were asked to rate the severity of their symptoms using a 10-point visual analogue scale (VAS) and the Tinnitus Handicap Inventory (THI) (Table 2), and they followed a standardized protocol for the investigation of tinnitus.

THI is a self-reported questionnaire developed by Newman in et al. (Newman et al., 1996) for the evaluation of tinnitus impact on patients' quality of life. It is a 25-item questionnaire divided into three subscales: a functional subscale (12 items), an emotional subscale (8 items), and a catastrophic response subscale (5 items) that address role and physical functioning, psychological distress, and desperation and loss of control, respectively. These multiple-choice items, presented only three possible answers: yes, sometimes, and no, scoring respectively 4, 2, and 0. Higher scores are related to more serious tinnitus handicap and its impact on quality of life. The THI has been proved to be a robust, psychometrically adequate measure of the impact of tinnitus on everyday life (Zeman et al., 2011).

All of the subjects were examined by the same researcher and underwent the same treatment therapy with neuromuscular

Table 1
Wilkes stage (11).

Early stage	Clinical: no significant mechanical symptoms, other than reciprocal clicking (early in opening movement, and soft in intensity). No pain and limitation of motion Radiologic: slight forward displacement, good anatomical contour of disk, and normal tomograms Surgical: normal anatomical form, slight anterior displacement, and passive incoordination (clicking) demonstrable
Early/intermediate stage	Clinical: first few episodes of occasional pain, joint tenderness and related temporal headaches, beginning of major mechanical problems, increase in intensity of clicking sounds, joint sounds later in opening movements, and beginning of transient subluxations or joint catching and locking Radiologic: slight forward displacement, slight thickening of posterior edge or beginning of anatomical deformity of disk, and normal tomograms Surgical: anterior displacement, early anatomical deformity of disk (slight to mild deformity thickening of posterior edge) and well-defined central articulating area
Intermediate stage	Clinical: multiple episodes of pain, joint tenderness, temporal headaches, major mechanical symptoms—transient catching, locking and sustained locking (closed locks) restriction of motion and difficulty (pain) with function Radiologic: anterior displacement with significant anatomical deformity/prolapse of disk (moderate to marked thickening of posterior edge) and normal tomograms Surgical: marked anatomical deformity with displacement, variable adhesions (anterior, lateral, and posterior recesses), and no hard tissue change
Intermediate/late stage	Clinical: characterized by chronicity with variable and episodic pain, headaches, variable restriction of motion, and undulating course Radiologic: increase in severity over intermediate stage, abnormal tomograms, and early to moderate degenerative remodeling hard tissues changes Surgical: increase in severity over intermediate stage, hard tissue degenerative remodeling changes of both bearing surfaces, osteophytic projections, multiple adhesions (lateral, anterior, and posterior recesses) and no perforation disk attachment
Late stage	Clinical: characterized by crepitus on examination, scraping, grating, grinding symptoms variable and episodic pain, chronic restriction of motion, and difficulty of function Radiologic: anterior displacement, perforation with simultaneous filling of upper and lower compartments, filling defects, gross anatomical deformity of disc and hard tissues, abnormal tomograms as described, and essentially degenerative arthritic changes Surgical: gross degenerative changes of disc and hard tissues, perforation of posterior attachments, erosions of bearing surfaces, and multiple adhesions equivalent to degenerative arthritis (sclerosis, flattening, anvil-shaped condyle, osteophytic, projections, and subcortical cystic formation)

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