

Aural Symptoms in Temporomandibular Disorder Patients Attending a Craniofacial Pain Unit

David K. Lam
DDS Candidate
Faculty of Dentistry
University of Toronto
Toronto, Ontario, Canada

Herenia P. Lawrence, DDS, MSc, PhD
Assistant Professor
Discipline of Community Dentistry
Faculty of Dentistry
University of Toronto
Toronto, Ontario, Canada

Howard C. Tenenbaum, DDS, PhD,
FRCD(C)
Professor and Head
Discipline of Periodontology
Faculty of Dentistry
University of Toronto

Craniofacial Pain Unit
Wasser Pain Management Centre
Mount Sinai Hospital

Toronto, Ontario, Canada

Correspondence to:
Dr Herenia P. Lawrence
Faculty of Dentistry
University of Toronto
124 Edward Street
Room 515D
Toronto, Ontario, Canada M5G 1G6
Fax: (416) 979-4936
E-mail: herenia.lawrence@utoronto.ca

This material was presented at the 78th General Session and Exhibition of the International Association for Dental Research, Washington, DC, April 7, 2000.

Aims: To determine (1) the prevalence of aural symptoms in orofacial pain patients and (2) a potential association between temporomandibular disorders (TMD) and aural health, while controlling for covariates known to be associated with TMD or auditory dysfunction. **Methods:** In a retrospective study, health questionnaires, medical histories, clinical findings, diagnoses, and treatments were systematically retrieved from the charts of 776 patients. The dates of initial assessment ranged from May 1987 to June 1999. Of the included subjects, 39.7% were female; the median age was 39 years; 16.4% displayed only aural symptoms (otalgia, tinnitus, vertigo, or perceived hearing loss); 26.4% had both TMD and aural symptoms; 17.8% had TMD but no aural complaints; and 39.4% had neither TMD nor aural symptoms. **Results:** Of the 344 subjects who had TMD, 59.9% complained of aural symptoms, versus 29.2% of the 432 patients without TMD. Of the subjects with otalgia, tinnitus, vertigo, or perceived hearing loss, 67%, 64.1%, 65.2%, and 62.2% had TMD, respectively. Subjects with aural symptoms were significantly more likely to be female; to consider themselves in poor health; to smoke; or to have TMD, orofacial pain, headaches (temporal, occipital, or frontal), neck and shoulder pain, altered vision and sensation, sleep disturbances, loss of appetite, memory loss, or low energy. Clinical findings indicated that pathognomonic signs of TMD were associated with an increased risk of aural complaints in this patient population. A significantly greater negative impact on normal life functions was found in subjects exhibiting aural symptoms versus those who only had TMD complaints. **Conclusion:** These findings indicate that TMD is significantly correlated to aural health, although no cause-and-effect relationship has yet been demonstrated. Aural symptoms were also found to have a measurable impact on the subjects' quality of life.

J OROFAC PAIN 2001;15:146-157.

Key words: temporomandibular disorders, otalgia, tinnitus, vertigo, perceived hearing loss, quality of life

Temporomandibular disorders (TMD) are characterized by various signs and symptoms of pain and dysfunction in the temporomandibular joint (TMJ) and/or the masticatory musculature. The signs and symptoms of TMD can manifest in areas of the face and neck; the temporal, occipital, and frontal areas of the head; and the preauricular and auricular areas. In addition to pain and dysfunction, many patients with TMD also complain of aural symptoms. The most commonly reported aural symptoms in TMD patients are otalgia, tinnitus, vertigo/dizziness, and subjective hearing loss. Otalgia arises from otologic causes

(otitis media, otitis externa, mastoiditis) as well as from non-otologic causes, which include dental conditions, tonsillitis, neoplasms, neuralgia, and TMD. The prevalence of non-otologic aural symptoms, or referred otalgia, in TMD patients varies from 3.5% to 42%.¹⁻⁵ The prevalence of tinnitus in the TMD population appears to be greater than that found in the general population. Studies of the general population have revealed a median prevalence of all forms of tinnitus in the range of 15% to 20%, with the prevalence increasing with age.^{6,7} The frequency of tinnitus among patients attending TMD clinics has been reported as varying from 33% to 76%.^{5,7-9} Dizziness and vertigo are common complaints in TMD patients that are often associated with facial, head, and neck pain. The prevalence of dizziness in TMD patients is of the order of 40% to 70%,^{9,10} whereas vertigo is less commonly reported (5% to 40%).¹¹ To date, few studies have focused on the prevalence of audiometrically tested hearing loss in TMD patients, and yet subjective complaints of hearing impairment have been reported in this group of patients (prevalence ranging from 23% to 57%).¹²⁻¹⁵ Hearing loss among TMD sufferers is generally correlated with sensorineural hearing loss, usually in the mid- and higher-frequency ranges.¹⁵⁻¹⁷

Several researchers have investigated the basis for the putative connection between aural symptoms and TMD symptoms. As early as 1934, aural symptoms, such as otalgia, stuffiness, tinnitus, vertigo, and hearing impairment, were included among the symptoms Costen related to TMD.¹⁸ Costen claimed that hearing impairment was secondary to Eustachian tube compression resulting from mandibular overclosure. However, the etiologic relationship first mentioned by Costen has since been questioned by a number of authors. Anatomic dissections carried out by Sicher¹⁹ refuted Costen's ideas about the etiology of TMD-related ear symptoms by stating that the Eustachian tube could not be compressed during mandibular overclosure. Shapiro and Truex²⁰ suggested that tonic spasm of the tensor tympani and stapedius muscles associated with TMD may cause a loss of ability to hear low tones. Toller and Juniper¹⁵ noted that the sound-conducting structures of the middle ear could be affected by the reflex spasm of the tensor tympani muscle. Further dissections of human cadavers, initially by Pinto²¹ and later by others,²²⁻²⁶ established a specific anatomic link between the TMJ and the middle ear through a tiny ligament, the mandibular-malleolar or discomalleolar ligament. This originates on the anterior process of the malleus and penetrates the

petrotympanic fissure, attaching itself to the TMJ capsule and disc. Pinto noted that movement of the capsule and disc caused movement in the middle ear ossicles, subsequently affecting the inner ear structures.²¹

Other studies support the contention of a neuromuscular interrelationship between the TMJ and the middle ear. Bernstein et al¹³ reported that a neurologic association could exist between the muscles of mastication, the muscle opening, the Eustachian tube, and the middle ear muscle on the basis that nerves to the medial pterygoid, tensor palatini, and tensor veli tympani muscles, respectively, arise from a common branch of the mandibular nerve. This complex local neuromuscular interaction between the chewing muscles and the hearing apparatus has given rise to what is termed "otomandibular syndrome."^{27,28} Patients with this syndrome may present one or more aural symptoms with no pathology identified by an ear, nose, and throat (ENT) examination, but with one or more of the muscles of mastication in a state of constant spasm. A neurophysiologic basis for the TMJ-ear connection has also been postulated by Miller and Wyrwa,²⁹ who illustrated how the "convergence theory"^{30,31} can be used to hypothesize referred pain to the ear that is secondary to dental pathosis. There is evidence that many brain stem neurons in the spinal nucleus of the trigeminal nerve, especially the subnucleus caudalis, receive afferent nociceptive input from the orofacial region and also from other cranial and cervical nerves. The convergence of these different afferent fibers upon the subnucleus caudalis may lead to perceptual errors in the brain that confuse identification of the pain source.

Studies also have demonstrated the association of aural symptoms with TMD by reporting that aural symptoms were alleviated following TMD or dental treatment.^{14,32,33} Morgan^{34,35} showed that joint surgery could correct tinnitus and vertigo. House et al³⁶ found that TMJ surgery resulted in the improvement or complete elimination of ear pain, vertigo, ringing in the ears, subjective hearing loss, and fullness, pressure, or blockage in the ears in 30% to 61% of patients. The impact of TMJ arthroscopy on the symptoms of dizziness and tinnitus continues to be demonstrated,³⁷ but at the same time, non-surgical TMD therapy has also been shown to resolve or improve aural symptoms; eg, internal derangements of the TMJ and concomitant vertigo were treated successfully by reduction of the dislocation using mandibular repositioning.³⁸ Keersmaekers et al⁴ showed that conservative treatment of TMD resulted in a

marked reduction of otalgia 1 year after the first examination and the start of treatment. Dolowitz et al¹² reported that tinnitus was eliminated in 40 of 43 patients after treatment with a regimen of jaw muscle exercises. Similarly, Principato and Barwell³⁹ reported elimination of dizziness and tinnitus 64% and 71% of the time, respectively, in patients with TMD treated with electromyographic biofeedback.

However, reports in the literature of patients treated for TMJ dysfunction and accompanied by tinnitus relief are still few in number.^{40,41} It appears that patients with more severe or disabling tinnitus are less likely to experience improvement after TMD therapy.^{32,41,42} This may be explained by recent evidence that at least some forms of tinnitus may have a central component (as opposed to cochlear tinnitus) but can be modified both by voluntary orofacial movements (including tooth clenching) and purely sensory stimuli.^{43,44} These findings support the need to identify the clinical type of tinnitus, as it appears that there can be multiple etiologies for tinnitus.

Despite a long history of research surrounding TMD, there remains a great deal of uncertainty about the biologic basis for the connection between these disorders and auditory dysfunction. An understanding of the relationship between the TMJ and auditory health is required to determine how improvements in TMJ function can, if possible, lead to improvements in aural health. Hence, the purpose of this study was twofold: (1) to determine the prevalence of aural symptoms in orofacial pain patients, and (2) to investigate the potential association between TMD and aural health. The hypothesis of this study was that aural symptoms were significantly more prevalent in TMD patients than in non-TMD patients attending a tertiary pain referral center, after adjustments were made for covariates known to influence both TMD and hearing.

Materials and Methods

Study Population and Design

The study population consisted of 776 orofacial pain patients referred for diagnosis and treatment to the Craniofacial Pain Unit of the Wasser Pain Management Centre at Mount Sinai Hospital in Toronto. The sociodemographic characteristics of the study population were: 39.7% female, median age 39 years, 51% married, and 64.2% working in skilled occupations. Of the 776 subjects, 85% per-

ceived their health as good, 41% currently were under health care, 26% were smokers, and only 12% were on, or had been on, potentially ototoxic drugs, such as aminoglycoside antibiotics, diuretics (furosemides), or aspirin.

The study used a retrospective observational design on all new patients seen consecutively in the unit over a period of approximately 12 years. A standardized Patient Health Questionnaire, Case History Form, and Clinical Examination Form were used consistently in the study for all 776 patients over the 12-year period. The history and clinical examinations were conducted by 1 of 4 investigators for the establishment of a clinical diagnosis. All 4 clinicians remained the same over the 12-year study period, thereby providing consistency with regard to treatment recommendations. A recent study showed that there was no difference in treatment outcomes for the 4 clinicians in this study.⁴⁵

With regard to the diagnosis of TMD, the investigators used criteria analogous to the Research Diagnostic Criteria (RDC) introduced in 1992,⁴⁶ and accordingly 3 groups of TMD patients were classified as follows:

1. TMD with myogenous pain only, based on the presence of one or more masticatory muscle groups being painful to palpation
2. TMD with TMJ pain only (no demonstrable muscle pain)
3. TMD with combined muscle and joint pain

The authors' diagnostic classification was based not only on diagnostic subsets similar to the RDC, but it also incorporated patient findings analogous to those of the RDC (ie, demographics, self-reported patient characteristics, axis I diagnosis, and axis II profile); it was non-hierarchical; and it allowed for the possibility of multiple diagnoses for a given subject.

The TMD signs and symptoms were recorded at the time of the subjects' clinical exam, by both self-report and by the examining dentist's assessment. Signs related to TMD pain were elicited by examiner palpation of selected masticatory structures within the head and neck region. As firm pressure was applied to these structures, subjects were asked whether pain was developing or not. Examiners calibrated their finger pressure by applying pressure to the arm muscle to the point where obvious fingernail blanching occurred.

The occurrence of specific aural symptoms was obtained through self-report. The standard practice among the 4 examiners was simply to ask subjects whether they had aural complaints, rather

than to “probe” for a particular response. All examiners based their questions on a standard case history form (see below) that had a section dedicated to aural complaints.

Health questionnaires, medical histories, clinical findings, and treatments were systematically retrieved from the patients’ charts. The dates of initial assessment ranged from May 1987 to June 1999. A complete set of dental, medical, and audiologic data required for this analysis was available for 470 of the 776 patients. From the chart review, 2 groups of patients were selected for comparison: the first group included those individuals with a history of aural symptoms ($n = 332$), and the other was composed of subjects having no aural complaints ($n = 138$). From the self-administered health questionnaire, information on variables known to be associated with TMD and potentially associated with auditory outcomes was obtained. Since age, sex, diabetes, hypertension, renal disease, medications, medical treatments, and occupational noise exposure have been linked to hearing problems, each of these variables was used as a covariate in the statistical comparison of TMD with aural symptoms.

Case History Form

A standard case history form was used to record self-reported symptoms, including ear and TMD-related symptoms. This form contained information on the patient’s demographics and was divided into 6 parts, which detailed the following information:

1. Chief complaint.
2. History of complaint.
3. Present symptoms. These included pain; limitation of mandibular movement; TMJ noises; headaches; neck pain; altered sensation (numbness or tingling, hyperesthesia or hyperalgesia, or allodynia); other pain; altered vision (loss of peripheral vision [left/right], diplopia, blurring, or blindness); sleep disturbance; loss of appetite; loss of energy; loss of memory/concentration; mood factors (patient’s predominant mood and anhedonia). These and other symptoms (gait disturbance, loss of coordination, muscle weakness, change in libido, and other chronic illnesses either personally or familial) were noted with respect to site(s), pain description, frequency, duration, triggering mechanisms/points, time of day when pain was most acute, precipitating event, onset, effect on life, and method of relief. Patients were also asked about the presence of

otalgia (“earaches”), perceived hearing loss, tinnitus (“noise in ears or head”), and vertigo (“spinning or things spinning”) and about the site, frequency, duration, associated factors, precipitating events, and progression of these aural symptoms.

4. Any parafunctional habits. This included such habits as bruxism, clenching, and nail-biting, along with the time of day (nocturnal or daytime) and the presence of any precipitating events related to these behaviors.
5. The possible impact of these symptoms on the patient’s normal functioning. This question was assessed by the use of an ordinal scale ranging from 0 (no impact) to 10 (incapacitating impact) in increments of 1. The interference of craniofacial symptoms on eating, talking, sleeping, sports, and work/occupation also was evaluated.
6. Previous treatment for craniofacial symptoms and results or effect of treatment. This addressed items such as physiotherapy; chiropractic; drugs (anti-inflammatories, analgesics, anti-depressants, anti-convulsants, muscle relaxants, and others); bite appliances; bite adjustment; dental reconstruction; orthodontics; surgery; and psychiatric/psychologic treatment, when applicable.

Clinical Examination Form

The clinical assessment examined the following:

- Facial swelling and asymmetry
- Tenderness to pressure (extraorally and intraorally with a graded pain response scale from 0 to 3)
- Joint noises
- Condylar translation
- Mandibular movement (maximum opening with/without pain, deviation on opening/closing, and lateral movement with/without pain)
- State of dentition
- Presence and fit of dentures
- Vertical dimension
- Periodontal status
- Occlusion in terms of Angle Class relation, overbite, overjet, centric occlusion relative to centric relation, and functional relation
- Other pertinent findings

In addition, when applicable, dental sensitivity to percussion/pressure, trigger points, decreased sensation, other alterations of sensation, and anesthetic tests were recorded. Also included were a note on whether the patient’s pain was exacerbated by the clinical examination; a brief summary of any radiographs and reports brought by the

patient or taken as part of the exam; a provisional differential diagnosis; any arranged consultations, radiographs, or lab investigations; and patient management information.

Data Analysis

Chi-square tests were used to measure differences in proportions between the 2 comparison groups (ie, presence/absence of reported aural symptoms), while independent-samples *t* tests were used to compare covariate means between the 2 groups. Odds ratios and 95% confidence intervals were calculated for the risk of subjective aural complaints based on TMD-related symptoms, sociodemographic factors, general health measures, and self-rated health. The same group comparisons were performed for subjects with and without evidence of impaired TMJ function by the use of logistic regression analysis to control for different subsets of covariates. Stepwise logistic models were initially adjusted for sex, age, dental insurance, occupation, marital status, and offspring. Variables were allowed to enter the models at $P < 0.15$ and to remain at $P < 0.05$. Comparisons were again carried out, with diabetes, renal problems, self-rated health, smoking, under physician's care, and use of ototoxic medications added to the original covariate set. The effect of the use of oral contraceptives on the aural health of females was examined by entering the variable into the model in the final step of the analysis, so as not to restrict the final model to females only.

Results

The primary diagnoses of the orofacial pain patients were as follows: (1) musculoligamentous (eg, TMD), $n = 389$, 49% females, median age 34 years; (2) neurologically based (eg, migraine, trigeminal neuralgia, tension-type headache, cluster headache, and atypical facial pain), $n = 67$, 43% females, median age 46 years; and (3) dentoalveolar pain (eg, reversible/irreversible pulpitis, periapical periodontitis, periodontal abscess), $n = 103$, 28% females, median age 45 years. The remaining 217 patients had either a miscellaneous diagnosis, or the diagnosis was unknown but the patient complained of aural symptoms. Of the 776 patients attending the Pain Unit, 39.7% were female, the median age was 39 years, 16.4% had only aural symptoms, 26.4% had TMD and aural symptoms, 17.8% had TMD with no aural complaints, and 39.4% had neither TMD nor aural symptoms. Thus, the non-TMD contrast group

was composed of those who presented complaints of tooth or periodontal pain, trigeminal neuralgia or atypical facial pain, or aural symptoms. Patients with combined muscle and joint pain were classified under the umbrella diagnosis of TMD.⁴⁶

Subsequent analyses included only 470 of the 776 records because of missing data resulting from incomplete/partial recording of information in some patients' charts. A non-response bias analysis was carried out to evaluate the extent to which missing information could have biased the representativeness of the sample and potentially biased the results of the study. This analysis indicated that subjects with incomplete records were significantly more likely to be older (*t* test, $P < 0.001$, mean age \pm standard error 44 ± 0.86 years vs. 38 ± 0.69 years); male (χ^2 test, $P < 0.001$, 71.2% vs. 53.2%); and with identified dentoalveolar pathology or a miscellaneous diagnosis (χ^2 test, $P < 0.001$, 83.7% vs. 16.4%) than those with complete records. For patients who had primarily dentoalveolar or miscellaneous diagnoses, some clinical data were missing. This can be explained by the fact that when such diagnoses were identified, further assessment of other structures (eg, TMJs, muscles of mastication) was generally not conducted and was in fact not required, especially when the diagnosis was readily apparent.

The prevalence of aural symptoms in TMD patients was almost 60%, compared to about 29% in non-TMD patients (Fig 1). There was a significant association between TMD and aural symptoms (odds ratio = 3.6; 95% confidence interval 2.7 to 4.9) in this patient population. Specifically, of the subjects with otalgia, tinnitus, vertigo, or perceived hearing loss, 67%, 64.1%, 65.2%, and 62.2%, respectively, had TMD (χ^2 tests, $P < 0.01$) (Fig 2).

Comparison of sociodemographic characteristics between subjects with or without aural symptoms indicated that the aural symptoms subjects were more likely to be female, work in unskilled jobs, and have children (Table 1). In addition, of the few patients with reported diabetes mellitus ($n = 11$), all were in the aural symptoms group (Table 2). More smokers and fewer women on oral contraceptives were among those with aural complaints. Similarly, a significantly higher proportion of subjects with aural symptoms were under the care of a physician and rated their health as being poor (Table 2).

Subjects who experienced pain in the orofacial region and other parts of their body were more likely to complain of ear symptoms. Subjects with aural symptoms were significantly more likely to

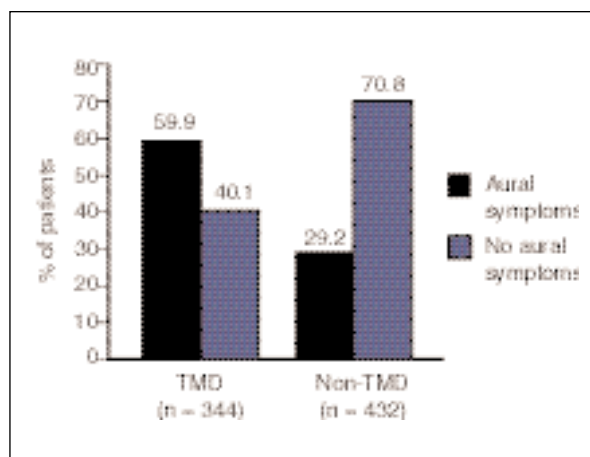


Fig 1 Prevalence of aural symptoms in TMD patients (n = 344) and in non-TMD patients (n = 432). Odds ratio = 3.6 (95% confidence interval 2.7 to 4.9); Chi-square test, $P < 0.001$.

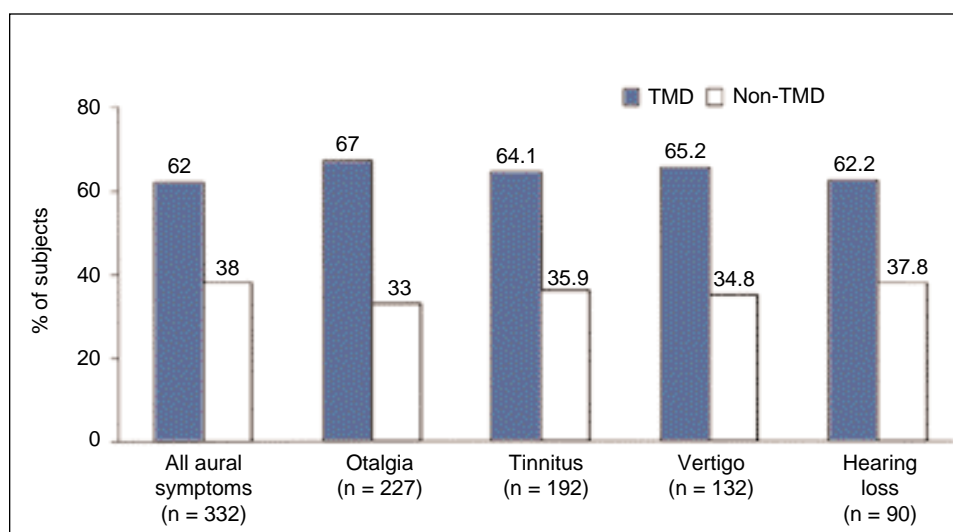


Fig 2 Prevalence of TMD in subjects with different types of aural symptoms (Chi-square test, $P < 0.01$).

Table 1 Comparison of Sociodemographic Characteristics Between Subjects With and Without Aural Symptoms

Characteristics	Aural symptoms		No aural symptoms		P value*
	%	Total no.	%	Total no.	
Female (n = 308)	46.4	332	34.7	444	0.001
Unskilled occupation (n = 121)	39.6	230	27.8	108	0.035
Married (n = 181)	54.1	244	44.1	111	0.082
Offspring (n = 169)	58.2	225	38.0	100	0.001
Dental insurance (n = 299)	66.6	302	75.4	130	0.068

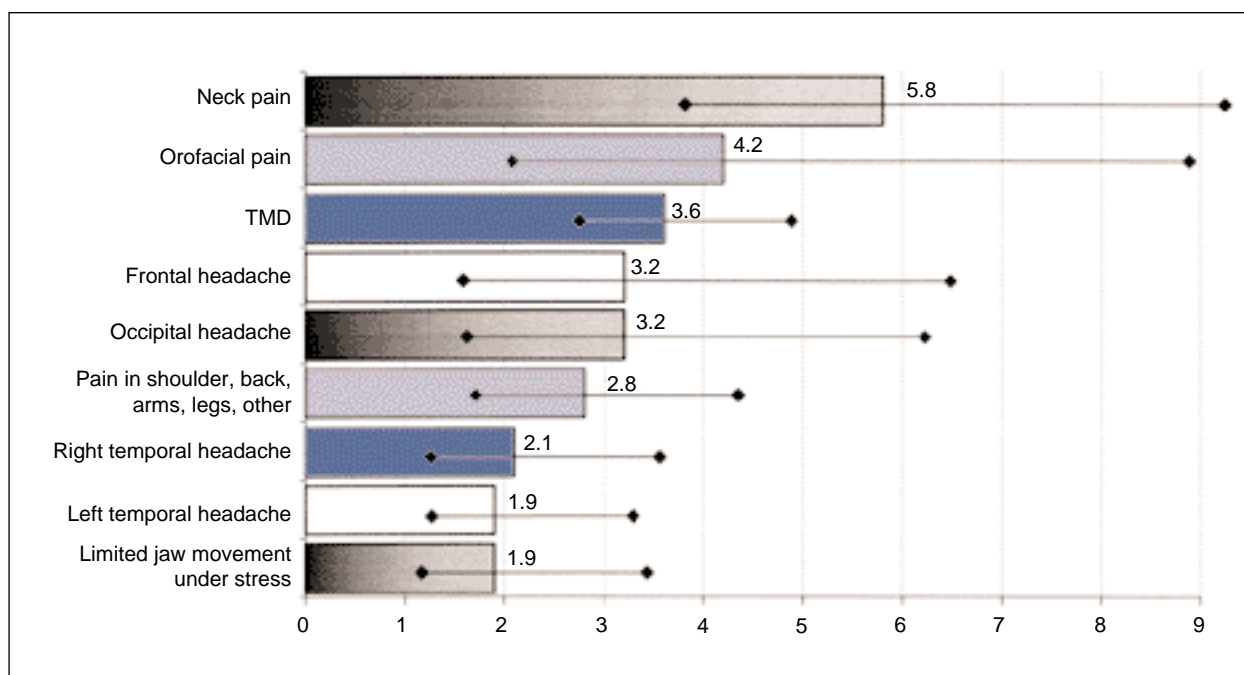
Mean age (y) \pm SD: Aural symptoms group, 40.0 \pm 14.9 (n = 332); No aural symptoms group, 40.6 \pm 15.5 (n = 441). $P = 0.595$ (t test).

*Chi-square test (2-tailed).

Table 2 Comparison of General Health Characteristics Between Subjects With and Without Aural Symptoms

Characteristic	Aural symptoms		No aural symptoms		P value*
	%	Total no.	%	Total no.	
Diabetes mellitus (n = 11)	3.3	332	0.0	138	0.039
Renal problems (n = 23)	5.7	332	2.9	138	0.196
Smoker (n = 88)	30.6	235	15.7	102	0.004
Oral contraceptives (n = 31)	12.7	126	41.7	36	< 0.001
Ototoxic medications (n = 56)	13.3	331	8.7	138	0.162
Under physician's care (n = 176)	48.7	302	22.1	131	< 0.001
Self-rated poor health (n = 65)	19.7	305	3.8	133	< 0.001

*Chi-square test or Fisher's exact test (2-tailed).

**Fig 3** Odds ratio and 95% confidence interval for aural symptoms in subjects with and without TMD/pain ($P < 0.05$).

have neck pain; orofacial pain; TMD; supraorbital, frontal, or occipital headaches; shoulder, back, arm, or leg pain; temporal headaches; and limited jaw movement when the patient was under stress (χ^2 test, $P < 0.05$) (Fig 3). Subjects reporting aural symptoms were also at increased risk for memory loss, appetite loss, altered vision, sleep disturbances, loss of energy, altered sensation, and mood swings (Fig 4). The risk of aural symptoms was greater for subjects who had a poor perception of their general health, were under a doctor's care, were smokers, had children, were blue-collar workers, or were female (Fig 4).

Figure 5 depicts the results of the clinical assessments. The risk of aural symptoms was significantly higher for patients with tenderness to extraoral pressure on the TMJ preauricular region, sternocleidomastoid, temporalis, and medial pterygoid muscles, as well as tenderness to intraoral pressure of the medial pterygoid muscles, lateral pterygoid region, and the zygomatic attachment of the masseter. Subjects with aural symptoms also were significantly more likely to experience pain during maximum opening and lateral movements of the mandible, dental sensitivity to percussion, and orofacial pain exacerbated by the dental exam.

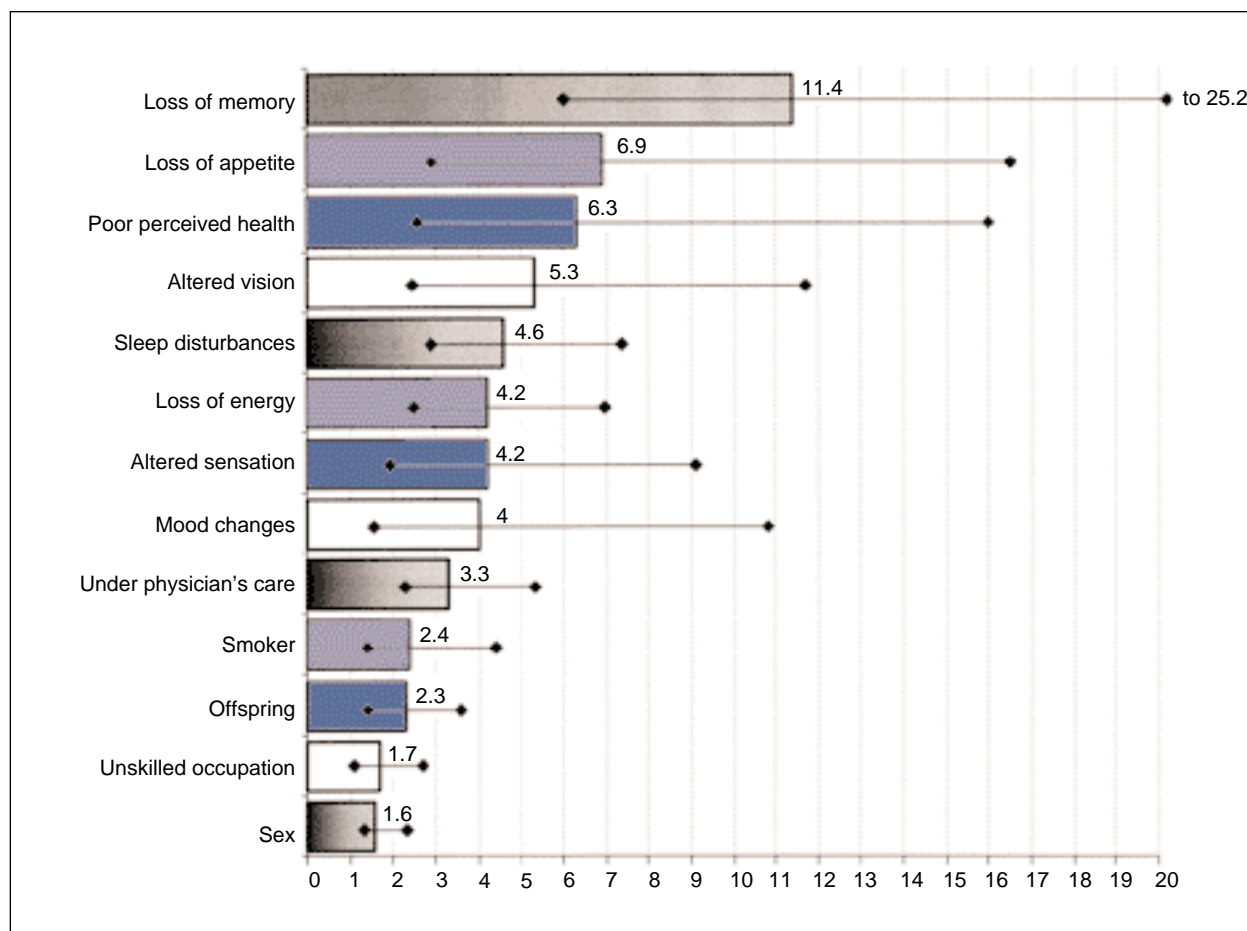


Fig 4 Odds ratio and 95% confidence interval for aural symptoms and patient history ($P < 0.05$).

The score for impact of symptoms on the subject's normal life functions was significantly higher for those with aural complaints than those without (6.6 ± 0.16 [mean \pm standard error] vs. 4.9 ± 0.24 , respectively; t test, $P < 0.001$).

When the significant covariates known to influence auditory and/or TMJ function (Tables 1 and 2) were accounted for in logistic regression, the odds ratio for aural symptoms between TMD and non-TMD subjects increased from 3.6 to 3.7 (95% confidence interval 2.7 to 5.0) and remained statistically significant.

Discussion

Studies that have evaluated the prevalence of aural symptoms in TMD patients vary, both in symptoms reported and in the method of evaluation. For example, Gelb et al⁴⁷ reviewed 742 patients, in

whom the incidence of aural symptoms was found to be tinnitus 42%, otalgia 35%, dizziness 18%, and deafness/hearing loss 14%. Kuttilla et al⁵ reviewed 411 TMD patients classified according to their treatment need in a 2-year follow-up study. Otagia without infection varied between 12% and 16%, while the prevalence of tinnitus was 12% to 17% and fullness of ears was 5% to 9%. Although the prevalence rates differed in magnitude, the rank order in our study was similar to that of Gelb et al—otalgia, vertigo, and tinnitus were reported most commonly, and perceived hearing loss was reported least often. For the purpose of this exploratory analysis, we chose simply to group the aural symptoms. However, analyses carried out by comparison of aural symptoms separately indicated the results did not change the associations found for the pooled data. These analyses indicated that TMD were significantly associated with otalgia, tinnitus, vertigo, and perceived hearing loss at similar rates.

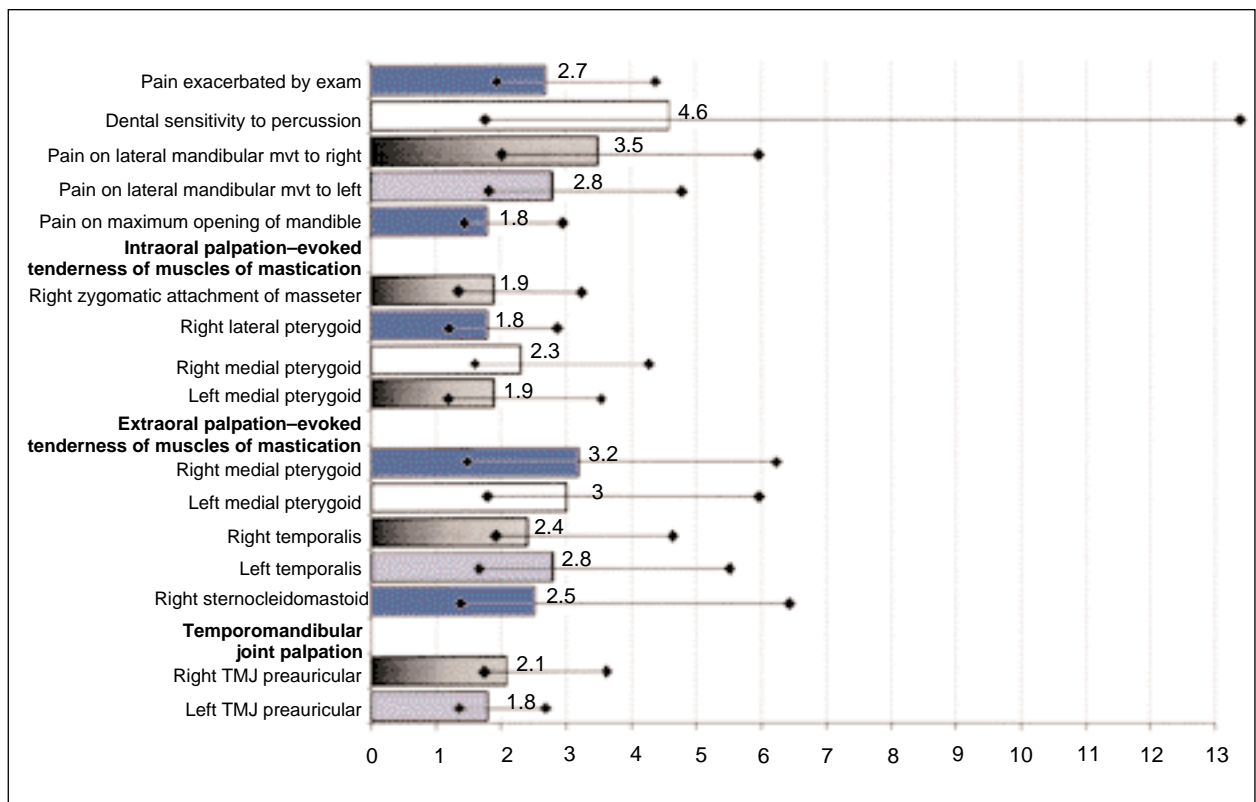


Fig 5 Odds ratio and 95% confidence interval for aural symptoms and clinical findings ($P < 0.05$).

The findings of this study indicate that there is a highly significant association between aural symptoms and TMD in this patient population, with a prevalence of aural symptoms in TMD patients of almost 60%. This association remained after significant covariates, such as general health-related variables, were accounted for in the analyses. However, since this was a retrospective study, the temporal relationship between exposure and disease may be difficult to establish because it is dependent upon patient recollection for the history of their chief complaint. Thus, a randomized controlled study of TMD therapies on auditory function is needed, since well-controlled, randomized clinical trials will provide the best evidence that TMD and aural health are directly related. It is noteworthy that if there is a relationship between TMD and aural symptoms, it is not necessarily a cause-and-effect association. Nonetheless, if this relationship does exist, it could imply cause and effect, which might therefore have implications regarding treatment of either condition. It is also notable that, following well-designed prospective

analyses, there may be no or at best a poor association or causal relationship between aural symptoms and TMD. This information could be used to prevent often needless investigation and treatment of patients with either condition, particularly those with aural complaints alone. In this regard, it is not unusual, in the authors' experience, for patients with aural complaints (other than pain) to be referred needlessly for a TMD assessment.

Despite the pitfalls of a retrospective design, this study has raised issues for further investigation, as outlined above. We found that subjects who experienced pain in the orofacial region and other parts of their body were more likely to complain of ear symptoms. This is in accordance with other studies of facial pain patients, in whom facial pain was influenced by painful comorbidity in body parts other than the face.^{48,49} While our study population consisted of patients in a tertiary referral center, who were more likely to be labeled as "complex" cases and have multiple comorbidities, further prospective studies are underway enrolling control subjects from the general population.

The gender ratio in the clinic was different from others in North America, with only 40% of patients being female. A possible explanation may be that the clinic also treats patients with advanced dental and periodontal infections, which are known to be more prevalent among males, as females are more likely to visit the dentist regularly.⁵⁰ In other words, all patients presenting to the Mount Sinai Hospital Department of Dentistry were surveyed, including those with TMD. When patients presenting with only TMD were examined, we noted that the ratio of males to females (1:1) was more in line with what has been reported elsewhere.⁵¹

Ash and Pinto⁵² have suggested that some subjective aural complaints, such as tinnitus, may affect the patient's quality of life more than complaints about limited jaw opening, chewing difficulties, and headaches. Not surprisingly, aural symptoms were found to have a measurable negative impact on the subjects' normal life functions when the 2 groups were compared on an ordinal rating scale.

When compared to the non-aural symptoms group, the subjects with aural symptoms more often had masticatory muscles that were tender to palpation. Kuttilla et al⁵ also reported that subjects with aural symptoms had more clinical signs of TMD, such as pain on palpation of TMJs or masticatory muscles. It would have been interesting to compare the intensity of muscular pain with that of the aural symptom; however, the severity of aural symptoms was not estimated in our study or in that of Kuttilla et al. Similarly, the potential for clinician bias on patients' reports of referred pain was not estimated in these studies. A recent study has found that bias induced by instructions from the clinician can result in an over-report of the presence and intensity of referred pain upon muscle palpation in TMD patients.⁵³ The study indicated that factors such as the patients' attention, their expectations, and their level of anxiety or response bias affected pain reports. Thus, in an effort to reduce clinician bias, the 4 clinicians who worked on this study consistently relied on standardized histories and examination forms when making their assessments of patients' pain reports throughout the 12 years of the study.

Our study also found that patients with dental percussion sensitivity were more likely to suffer from aural symptoms (odds ratio = 4.6). This association may be related to a central trigeminal connection linking oral pain with aural pain, although it is not entirely clear how the local neural network in the TMJ region sends afferent nociceptive

input to cortical areas of the brain.³¹ Moreover, dental percussion causes vibrations similar to those caused by the mechanics of the stomatognathic system during oral function, and these vibrations of a dental origin are conducted most effectively to the ear (dentaural hearing).⁵⁴

It should also be noted that patients with TMD have the propensity for referred craniofacial pain; this may explain the findings in the present study of a significantly greater frequency of aural complaints in TMD than in non-TMD pain conditions. Referred aural pain is prevalent in the craniofacial region, as reported in a recent retrospective study of patients with TMD.⁵⁵ The greater frequency of aural complaints in TMD versus non-TMD pain conditions is significant because it suggests the possibility that TMD nociceptive mechanisms differ from those of other craniofacial pain conditions. There may be differences in the primary nociceptive afferents innervating the TMJ (capsule, disc, and associated musculature) or in the relay and processing of noxious stimuli in the spinal nucleus of the trigeminal nerve, particularly the subnucleus caudalis, in terms of nociceptive receptors and/or mediators. Clearly, further study of the nociceptive mechanisms involved in TMD pain is necessary. Likewise, clinical trials of TMD therapies also would aid in establishing a cause-and-effect relationship. Such evidence may help individuals suffering from ear symptoms to isolate the cause of their symptoms and also preclude the need for unnecessary otologic surgical procedures.

The findings of this study have important clinical implications for the treatment and management of patients suffering from orofacial pain and aural symptoms. In light of the findings that an association exists between TMD and auditory function, and that the aural symptoms associated with TMD have a negative impact on the patient's life functions, it is important to undertake further research into the biologic mechanisms of TMD. Such research will allow us to understand this association and to determine a cause-and-effect and/or a functional relationship. Assuming the latter can be established, the efficacy of various therapeutic modalities for TMD and aural symptoms must be tested.

Acknowledgments

The study was supported by a Canadian MRC Summer Scholarship and NIH-NIDCR Grant DE13330. The authors wish to thank Ms Susan Deshmukh for her help with the preparation of the graphics for this paper.

References

1. Myrhaug H. Clicking ear and pharyngeal tic associated with functional disturbances of the jaw. *Acta Otolaryngol Suppl* 1958;188:430-433.
2. Posselt U. The temporomandibular joint syndrome and occlusion. *J Prosthet Dent* 1971;25:432-438.
3. 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;8:293-297.
4. Keersmaekers K, De Boever JA, Van Den Berghe L. Otagia in patients with temporomandibular joint disorders. *J Prosthet Dent* 1996;75:72-76.
5. Kuttilla S, Kuttilla M, Le Bell Y, Alanen P, Jouko S. Aural symptoms and signs of temporomandibular disorders in association with treatment need and visits to a physician. *Laryngoscope* 1999;109:1669-1673.
6. Coles RRA. Epidemiology of tinnitus: (1) Prevalence. *J Laryngol Otol* 1984;(Suppl 9):7-15.
7. Rubinstein B. Tinnitus and craniomandibular disorders: Is there a link? *Swed Dent J* 1993;95(Suppl):1-46.
8. Chan SWY, Reade PC. Tinnitus and temporomandibular pain-dysfunction disorder. *Clin Otolaryngol Allied Sci* 1994;19:370-380.
9. Parker WS, Chole RA. Tinnitus, vertigo, and temporomandibular disorders. *Am J Orthod Dentofac Orthop* 1995;107:153-158.
10. Cooper BC, Alleva M, Cooper DL, Lucente FE. Myofascial pain dysfunction: Analysis of 476 patients. *Laryngoscope* 1986;96:1099-1106.
11. Chole RA, Parker WS. Tinnitus and vertigo in patients with temporomandibular disorder. *Arch Otolaryngol Head Neck Surg* 1992;118(8):817-821.
12. 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.
13. Bernstein JM, Mohl ND, Spiller H. Temporomandibular joint dysfunction masquerading as disease of ear, nose, and throat. *Trans Am Acad Ophthalmol Otolaryngol* 1969;73:1208-1217.
14. Koskinen J, Paavolainen M, Raivio M, Roschier J. Otolological manifestations in temporomandibular joint dysfunction. *J Oral Rehabil* 1980;7:249-254.
15. Toller MÖ, Juniper RP. Audiological evaluation of the aural symptoms in temporomandibular joint dysfunction. *J Craniomaxillofac Surg* 1993;21:2-8.
16. Baldursson G, Blackmer ER. Temporomandibular joint symptoms in patients with midfrequency sensorineural hearing loss. *Ear Hear* 1987;8:63-67.
17. Vernon J, Griest S, Press L. Attributes of tinnitus that may predict temporomandibular joint dysfunction. *Cranio* 1992;10:282-288.
18. Costen JB. A syndrome of ear and sinus symptoms dependent upon disturbed function of the temporomandibular joint. *Ann Otol Rhinol Laryngol* 1934;43:1-15.
19. Sicher H. Temporomandibular articulation in mandible overclosure. *J Am Dent Assoc* 1948;36(2):131-139.
20. Shapiro HH, Truex RC. The temporomandibular joint and the auditory function. *J Am Dent Assoc* 1943;30:1147-1168.
21. Pinto OF. A new structure related to the temporomandibular joint and middle ear. *J Prosthet Dent* 1962;12(1):95-103.
22. Komori E, Sugisaki M, Tanabe H, Katoh S. Discomalleolar ligament in the adult human. *Cranio* 1986;4:300-305.
23. 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.
24. Manni A, Brunori P, Giuliani M, Modoni M, Bizzi G. Oto-vestibular symptoms in patients with temporomandibular joint dysfunction. Electromyographic study [in Italian]. *Minerva Stomatol* 1996;45:1-7.
25. 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.
26. Rodriguez-Vásquez JF, Mérida-Velasco JR, Mérida-Velasco JA, Jiménez-Collado J. Anatomical considerations on the discomalleolar ligament. *J Anat* 1998;192:617-621.
27. Arlen H. The otomandibular syndrome: A new concept. *Ear Nose Throat J* 1977;56:60-62.
28. Arlen H. The otomandibular syndrome. In: Gelb H (ed). *Clinical Management of Head, Neck and TMJ Pain and Dysfunction*. Philadelphia: Saunders, 1985:171-180.
29. Miller DA, Wyrwa EB. Ear pain: A dental dilemma. *Compend Contin Educ Dent* 1992;13:676-684.
30. Sessle BJ, Hu JW, Amano N, Zhong G. Convergence of cutaneous, tooth pulp, visceral, neck and muscle afferents onto nociceptive and non-nociceptive neurons in trigeminal subnucleus caudalis (medullary dorsal horn) and its implications for referred pain. *Pain* 1986;27:219-235.
31. Sessle BJ. Acute and chronic craniofacial pain: Brain stem mechanisms of nociceptive transmission and neuroplasticity and their clinical correlates. *Crit Rev Oral Biol Med* 2000;11:57-91.
32. Erlandsson SI, Rubinstein B, Carlsson SG. Tinnitus: Evaluation of biofeedback and stomatognathic treatment. *Br J Audiol* 1991;25:151-161.
33. Kempf H-G, Roller R, Mühlbradt L. Correlation between inner ear disorders and temporomandibular joint diseases [in German]. *HNO* 1993;41:7-10.
34. Morgan DH. Dysfunction, pain, tinnitus, vertigo corrected by mandibular joint surgery. *South Calif Dent Assoc J* 1971;39:505-534.
35. Morgan DH. Temporomandibular joint surgery. Correction of pain, tinnitus, and vertigo. *Dent Radiogr Photogr* 1973;46(2):27-39.
36. House LR, Morgan DH, Hall WP, Vamvas SJ. Temporomandibular joint surgery: Results of a fourteen-year implant study. *Laryngoscope* 1984;94:534-538.
37. Steigerwald DP, Verne SV, Young D. A retrospective evaluation of the impact of temporomandibular joint arthroscopy on the symptoms of headache, neck pain, shoulder pain, dizziness, and tinnitus. *Cranio* 1996;14:46-54.
38. Williamson EH. The interrelationship of internal derangement of the temporomandibular joint, headache, vertigo, and tinnitus: A survey of 25 patients. *Cranio* 1990;8:301-306.
39. Principato JJ, Barwell DR. Biofeedback training and relaxation exercises for treatment of temporomandibular joint dysfunction. *Otolaryngology* 1978;86:766-769.
40. Rubinstein B, Carlsson GE. Effects of stomatognathic treatment on tinnitus: A retrospective study. *Cranio* 1987;5:254-259.

41. Wright EF, Bifano SL. Tinnitus improvement through TMD therapy. *J Am Dent Assoc* 1997;128:1424-1432.
42. Bush FM. Tinnitus and otalgia in temporomandibular disorders. *J Prosthet Dent* 1987;58:495-498.
43. 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;50:114-120.
44. Pinchoff RJ, Burkard RF, Salvi RJ, Coad ML, Lockwood AH. Modulation of tinnitus by voluntary jaw movements. *Am J Otol* 1998;19:785-789.
45. Grossi ML, Goldberg MB, Locker D, Tenenbaum HC. Reduced neuropsychological measures as predictors of treatment outcome in patients with temporomandibular disorders. *J Orofac Pain* (in press).
46. Dworkin SF, LeResche L. Research Diagnostic Criteria for Temporomandibular Disorders: Review, Criteria, Examinations and Specifications, Critique. *J Craniomandib Disord* 1992;6:301-355.
47. 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.
48. Dao T, Reynolds WJ, Tenenbaum HC. Comorbidity between myofascial pain of the masticatory muscles and fibromyalgia. *Alpha Omegan* 1998;91(2):29-37.
49. Türp JC, Kowalski CJ, Stohler CS. Generic pain intensity scores are affected by painful comorbidity. *J Orofac Pain* 2000;14:47-51.
50. U.S. Department of Health and Human Services. Oral Health in America: A Report of the Surgeon General. Rockville, MD: U.S. Department of Health and Human Services, National Institute of Dental and Craniofacial Research, National Institutes of Health, 2000.
51. LeResche L. Epidemiology of temporomandibular disorders: Implications for the investigation of etiologic factors. *Crit Rev Oral Biol Med* 1997;8:291-305.
52. Ash CM, Pinto OF. The TMJ and the middle ear: Structural and functional correlates for aural symptoms associated with temporomandibular joint dysfunction. *Int J Prosthodont* 1991;4:51-57.
53. Branch MA, Carlson CR, Okeson JP. Influence of biased clinician statements on patient report of referred pain. *J Orofac Pain* 2000;15:120-127.
54. Lawrence HP, Garcia RI, Essick GK, Hawkins R, Krall EA, Spiro III A, et al. A longitudinal study of the association between tooth loss and age-related hearing loss. *Spec Care Dent* (in press).
55. Wright EF. Referred craniofacial pain patterns in patients with temporomandibular disorder. *J Am Dent Assoc* 2000;131:1307-1315.