The Journal of Laryngology & Otology (2015), ***129***, *638–643*.

doi:10.1017/S0022215115001425

MAIN ARTICLE

Tinnitus and its relationship with muscle tenderness in patients with headache and facial pain

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Abstract

Objective: This study aimed to examine tinnitus prevalence in patients with different types of headache and the relationship between tinnitus and the pericranial muscle tenderness and cervical muscle tenderness scores.

Methods: A cross-sectional study was conducted of 1251 patients with migraine and/or myogenous pain, arthrogenous temporomandibular joint disorders and tension-type headache. Standardised palpation of the pericranial and cervical muscles was carried out and univariable and multivariable analysis was used to measure the odds ratio of suffering tinnitus by the different diagnoses and muscular tenderness grade.

Results: A univariable analysis showed that myogenous pain, pericranial muscle tenderness and cervical muscle tenderness scores, sex, and age were associated with tinnitus. When a multivariable model including only age, sex and a headache diagnosis was used, myogenous pain, migraine and age were found to be associated with tinnitus. When muscle tenderness scores were also included, only the cervical muscle tenderness and pericranial muscle tenderness scores were found to be significantly associated with tinnitus.

Conclusion: In a population of patients with headache and craniofacial pain, tinnitus was related to increased cervical muscle tenderness and pericranial muscle tenderness scores, rather than to any particular form of headache.

Key words: Tinnitus; Facial Pain; Headache; Temporomandibular Disorders; Epidemiology

Introduction

In recent years, there has been debate about whether the pathophysiological mechanisms that generate tinnitus depend on abnormal functioning of the cochlea or on higher structures of the auditory pathways. Some factors and mechanisms responsible for hearing loss, including noise, ageing, medication, head injury and Ménière's disease, have been shown to be related to tinnitus. In some cases, however, it is impossible to determine the cause of tinnitus.

Headache sufferers complain of numerous accompanying symptoms, including aural symptoms such as otalgia, hyperacusis, aural fullness, vertigo and tinnitus.[1] While there have been attempts to explain why these symptoms develop, no consensus has been reached on the pathogenetic mechanisms involved.[2–6]

Although the aetiology of tinnitus is not completely understood, previous studies have found significant correlations between tinnitus and temporomandibular disorders.[2,3,7] There are some reports that voluntary muscle contractions affect tinnitus onset and modulation owing to the formation of central connections between the auditory and somatosensory system.[5,8] The latter is a direct consequence of attempted compensation for hearing loss and reduced auditory input. In particular, the trigeminal system, which provides sensory innervations to temporomandibular joint (TMJ) and shares a common pathway with the auditory system along the central auditory pathways, may be implicated.[9]

A range of pathologies are currently defined as temporomandibular disorders. Temporomandibular joint disorders and craniofacial and cervical myogenous pain are distinct but overlapping clinical entities; they share symptoms and some aetiological factors. Tension-type headache and migraine are also often associated with craniofacial and cervical pain.[4]

This study aimed to assess tinnitus prevalence in patients with different types of head pain, namely migraine, tension-type headache, myogenic pain and TMJ disorder. It also aimed to investigate the relationship between tinnitus and the level of pericranial and

Accepted for publication 9 December 2014

cervical muscle tenderness in patients suffering from these diseases.

Materials and methods

A retrospective review was performed of the clinical registries of 1251 patients (1058 women and 193 men) with headache and/or facial pain who were consecutively referred to the Headache and Facial Pain Unit, Head and Neck Department, University of Turin, between 2000 and 2007 (Table I). Patients were aged 15–87 years, with a mean standard deviation ± standard deviation age of 44 ± 16 years.

The presence of tinnitus was assessed from patient interviews. The examiner asked patients whether they were suffering or had suffered from ringing or rushing noises in the ear. Only those with current tinnitus symptoms were defined as tinnitus patients. Headache was diagnosed using International Headache Society criteria,[10] and myogenic pain was diagnosed using American Academy of Orofacial Pain criteria.[11] It was possible for the same patient to have two or more diagnoses. Drug abuse patients or those with a concurrent relevant medical condition, such as an endocrine, immune, blood, neurological or circulatory disorder, were excluded.

The criteria specified that in patients with migraine, headache attacks lasted 4–72 hours and had at least two of the following characteristics: a unilateral location; a pulsating quality; moderate or severe intensity (inhibiting or prohibiting normal daily activities); and aggravated by routine physical activity. During headache, at least one of the following symptoms was present: nausea and/or vomiting, photophobia and phonophobia. In patients with tension-type headache, the pain lasted from minutes to days and produced a sense of pressure or tightening in the head. This could be mildly or moderately intense, bilateral or variable in location, and did not worsen with physical activity. Nausea was absent, but phonophobia or photophobia was sometimes present. Myogenous pain patients had pain in the projection areas of one or more facial or masticatory muscles; the pain was spontaneous but exacerbated by muscle palpation. The pain location differed according to the main muscles involved: for the lateral pterygoid and masseter muscles, pain was located in the pre-auricular and cheek areas; for the temporal muscle, pain was located in parietal, temporal and periorbital areas. Pain could be aggravated by meteorological changes or certain weather conditions (cold, damp, wind), or sport activities involving prolonged isometric contractions; mastication was not an overt aggravating factor.

To be included in the arthrogenous TMJ disorder group, a patient had to show signs of internal TMJ derangement. Most patients (95 per cent) had clinical signs of disc displacement with reduction, i.e. irregular jaw movements and a clicking noise during mouth opening and closing. Displacement without reduction, as confirmed by magnetic resonance imaging, was present only in 2 per cent of patients. A change in bone shape was also present in 3 per cent of patients. Overt arthrotic degeneration was not observed in any of these patients; the TMJ was the only or main source of pain, and mastication was always an aggravating factor.

TABLE I
PATIENT CHARACTERISTICS

Characteristic	Patients		
	Total (n = 1251)	No tinnitus (n = 917)	Tinnitus (n = 334)
Age (mean ± SD), years	44 ± 16	48 ± 16	42 ± 16
Female (n (%))	1058 (84.5)	778 (84.8)	280 (83.8)
Male (n (%))	193 (15.5)	139 (15.2)	54 (16.2)

SD = standard deviation

Examinations

A complete medical history was taken for each patient. This included a record of the pain characteristics, improving or aggravating factors, and associated symptoms. All patients underwent a clinical examination, including neurological and craniofacial examinations. Muscle palpation of the pericranial and cervical muscles was performed by an expert clinician in a standardised way, following the instructions of Dworkin and LeResche.[12] The following pericranial muscles were examined: masseter, lateral pterygoid, medial pterygoid and temporal (mandibular and cranial insertion). The following cervical muscles were examined: sternocleidomastoid (lower and cranial insertion), trapezius and nuchal. Tenderness during palpation was scored at each location on a scale of 0–3, with 0 indicating normal tone, 1 indicating mild tenderness, 2 indicating moderate tenderness, and 3 indicating severe tenderness. Pericranial and nuchal muscles were palpated by making small circular movements with pressure using the index and middle fingers; the other cervical muscles were gently pinched. Palpation of each site lasted three to four seconds. Scores for the pericranial and cervical muscles were added separately and the sums were divided by the number of sites examined. Thus, cervical muscle tenderness and pericranial muscle tenderness scores (range 0–3) were calculated for each patient. These methods represent modified versions of published techniques.[13,14] The degree of the inter-rating agreement between clinicians was assessed and the results were found to have acceptable reliability that was consistent with previous reports (intraclass correlation 0.81–0.88).

Statistical analysis

Tinnitus patients were defined as those patients who had current tinnitus symptoms and control participants were defined as those who did not; the two groups were

https://doi.org/10.1017/S0022215115001425 Published online by Cambridge University Press

compared. The odds ratios of being diagnosed with a specific condition (i.e. migraine, myogenous pain, TMJ disorder, tension-type headache), degree of pericranial and cervical tenderness, sex, and co-presence of tinnitus between patients and controls were calculated using both univariable and multivariable models with logistic regression. The association between outcome and explanatory variables was evaluated by setting the significance level in the univariate analysis to a p value of less than or equal to 0.05. Statistical analyses were performed using STATA statistical analysis software version 10.0 (StataCorp, College Station, Texas, USA).

Results

Of the 1251 patients included in the study, 327 were diagnosed with migraine, 245 with tension-type headache, 772 with myogenous pain and 260 with arthrogenous TMJ disorders (Table II). Several patients presented with symptoms of more than one disorder and were placed into two or three diagnostic categories. (Table III).

A total of 304 patients (26.6 per cent of the overall sample) suffered from tinnitus (tinnitus group) while 917 (73.4 per cent) did not. There was a female preponderance in both groups: 85 per cent of control participants and 84 per cent of tinnitus patients were female. Those with tinnitus had a mean age of 42 ± 16 years (range 14–89 years), whereas those without tinnitus had a mean age of 48 ± 16 years (range 15–87 years) (Table I). Tinnitus prevalence was higher in the presence of more than one pathology (Table III).

Univariable analysis models were performed in which tinnitus was the outcome variable and the various diagnoses (TMJ disorder, migraine, tension-type headache and myogenous pain), muscle tenderness scores, age and sex were explanatory variables. These models showed that myogenous pain, cervical muscle tenderness and pericranial muscle tenderness scores, sex and age were all significantly associated with tinnitus, whereas TMJ disorder, migraine and tension-type headache were not. A multivariable logistic regression model including only age, sex and head pain diagnosis showed that myogenous pain, migraine and age were positively associated with tinnitus. When muscle tenderness scores were also included in a multivariable logistic regression model, only the cervical muscle tenderness score (with an adjusted odds ratio of 1.25 for a one unit increase in score), pericranial muscle tenderness score (adjusted odds ratio of 1.48 for a one unit increase in the score) and age (adjusted odds ratio of 1.02 for a one year increase in age) were found to be significantly associated with tinnitus (Table IV).

TABLE II
TINNITUS PREVALENCE BY EACH PRIMARY DIAGNOSIS

Diagnosis*	No tinnitus (*n* (%))	Tinnitus (*n* (%))
M	216 (66.0)	111 (34.0)
TTH	175 (71.4)	70 (28.6)
MP	548 (70.9)	224 (29.1)
TMJ disorder†	192 (73.8)	68 (26.2)
Total population	917 (73.4)	334 (26.6)

*Patients who suffer from two pathologies were counted twice. †Arthrogenous. M = migraine; TTH = tension-type headache; MP = myogenous pain; TMJ = temporomandibular joint

Discussion

This study found that 26.6 per cent of patients with head and facial pain complain of tinnitus. The presence of tinnitus was associated with a myogenous pain diagnosis, to some extent with a migraine diagnosis (when in conjunction with myogenous pain) and with increased pericranial and cervical muscle tenderness. In contrast, no relationship was found with a diagnosis of tension-type headache or TMJ disorder.

Tinnitus is extremely common; it is associated with different conditions such as hearing loss, older age and male sex. Epidemiological studies have found that tinnitus prevalence is high in the general population, ranging from 4 to 30 per cent.[15,16] A large population study based on figures from the US National Health Survey showed that the overall tinnitus prevalence was 25.3 per cent when any episode of 'ringing, roaring, or buzzing' within the past year was included. In all, 7.9 per cent of patients experienced frequent or constant tinnitus, classified as 'chronic'.[17] In our study group, 26.6 per cent of patients stated that they were suffering from tinnitus at the time of interview. Compared with a prevalence of chronic tinnitus in the general population of about 10 per cent, our data showed a higher tinnitus prevalence in patients with headache and facial pain.

The relationship between tinnitus and TMJ disorders has been widely investigated and reported, but there are

TABLE III
TINNITUS PREVALENCE BY EACH DIAGNOSIS AND GROUP OF DIAGNOSES

Diagnosis	Total (*n*)	No tinnitus (*n* (%))*	Tinnitus (*n* (%))*
M	144	104 (72)	40 (28)
TTH	84	74 (88)	10 (12)
MP	556	425 (76)	131 (24)
TMJ disorder†	142	112 (79)	30 (21)
M+TTH	33	24 (73)	9 (27)
M+MP	76	41 (54)	35 (46)
TTH+MP	70	45 (64)	25 (36)
M+TTH+MP	28	12 (43)	16 (57)
MP+TMJ disorder†	42	25 (60)	17 (40)
TTH+TMJ disorder†	30	20 (67)	10 (33)
M+TMJ disorder†	46	35 (76)	11 (24)
Total	1251	917	334

*Presented as a percentage of the number in the second column. †Arthrogenous. M = migraine; TTH = tension-type headache; MP = myogenous pain; TMJ = temporomandibular joint

https://doi.org/10.1017/S0022215115001425 Published online by Cambridge University Press

TABLE IV
UNIVARIABLE AND MULTIVARIABLE ANALYSES MEASURING THE STRENGTH OF ASSOCIATION BETWEEN TINNITUS AND THE EXPLANATORY VARIABLES

Variable	Univariable model			Multivariable model excluding tenderness indexes			Multivariable model including tenderness indexes		
	OR	95% CI	*p* value	aOR*	95% CI	*p* value	aOR*	95% CI	*p* value
M	1.24	0.96–1.60	0.11	1.40	1.06–1.83	0.02	1.31	0.99–1.73	0.06
TTH	0.76	0.56–1.04	0.08	0.86	0.63–1.18	0.36	0.75	0.54–1.04	0.08
TMJ disorder†	1.17	0.87–1.56	0.30	1.27	0.94–1.72	0.11	1.28	0.94–1.74	0.12
MP	1.34	1.04–1.74	0.02	1.42	1.08–1.86	0.01	1.07	0.81–1.43	0.62
PTS	1.70	1.49–1.94	<0.001				1.48	1.22–1.80	<0.001
CTS	1.52	1.35–1.71	<0.001				1.25	1.04–1.48	0.01
Female sex	1.03	0.74–1.42	0.02	0.97	0.69–1.36	0.87	0.73	0.51–1.05	0.09
Age	1.02	1.01–1.03	<0.001	1.02	1.01–1.03	<0.001	1.02	1.01–1.03	<0.001

Statistical significance was set at $p \leq 0.05$. *Adjusted for all other variables in the column. †Arthrogenous. OR = odds ratio; CI = confidence interval; aOR = adjusted odds ratio; M = migraine; TTH = tension-type headache; MP = myogenous pain; TMJ = temporomandibular joint; PTS = pericranial muscle tenderness score; CTS = cervical muscle tenderness score

significant variations in tinnitus prevalence among the different studies. Tullberg and Ernberg reported that 82 per cent of patients suffering from tinnitus showed signs of TMJ disorder.[18] In a small randomised controlled trial study (20 TMJ disorder patients and 8 healthy controls), De Felício *et al.* showed that 60 per cent of TMJ disorder patients and 25 per cent of asymptomatic controls reported tinnitus which correlated with tenderness during palpation of the temporomandibular muscles and joints.[19] Lam *et al.* found that 16.4 per cent of 334 patients with TMJ disorders complained of generic aural symptoms, while 64 per cent of patients with tinnitus had a TMJ disorder.[20] In a cross-sectional study of 139 patients with tinnitus, Bernhardt *et al.* detected tenderness on palpation (of the TMJ and muscles) in 30.9 per cent of tinnitus patients, but in only 17.5 per cent of healthy participants from the general population; they calculated an odds ratio of 2.53 when more than four muscles were affected.[7] More recently, the same author showed that, after a five-year follow up, signs of temporomandibular disorders were predictors for the development of tinnitus (relative risk 2.44).[21] The largely documented correlation between tinnitus and TMJ dysfunction led researchers to investigate whether patients with both conditions comprise a special subpopulation relative to patients with isolated tinnitus. As effectively shown by Vielsmeier *et al.*, co-morbid patients are more likely to be younger females with a better hearing function.[22] This suggests a causal relationship between TMJ dysfunction and tinnitus.

TMJ dysfunction is hypothesised to be responsible for tinnitus through a mechanism involving the discomalleolar ligament connecting the middle ear with the TMJ.[2] This ligament is a developmental remnant of Meckel's cartilage that may transmit mechanical energy to the malleus. Recently, Riga *et al.* used multiple frequency tympanometry to show increased middle-ear stiffness in 34 out of 40 patients with ipsilateral TMJ disorders.[23] Other hypotheses postulate that hyperactivity of the masticatory muscles can cause aural symptoms. The masticatory muscles, in fact, share common trigeminal innervation with the tensor tympani and tensor veli palatini, the tonic contraction of which can cause hyperacusia, tinnitus, dizziness and other otologic symptoms.[3] Both tinnitus and muscle tenderness are frequently associated with stress and psychiatric disturbances.[4] Therefore, authors have suggested that psychosocial disorders (i.e. emotional burdens) may increase masticatory muscle activity and, consequently, contribute to tinnitus and vertigo.[5]

More recent theories are based on a somatic (or craniocervical) form of tinnitus that may result from interactions between the somatic and auditory pathways in the central nervous system. In some patients, tinnitus is localised to the ear ipsilateral to a head or neck somatic disorder and can be modulated by pressure and movement applied to the head and neck.[6] Based on these observations, Levine hypothesised that tinnitus perception is caused by decreased inhibitory medullary somatosensory input to the ipsilateral dorsal cochlear nuclei.

We investigated the correlation of tinnitus with the presence of cervical and cranial muscle tenderness and with the overall level of tenderness, based on examiner rating (scale of 0–3). The results show that the likelihood of suffering from tinnitus increased with the tenderness score. Moreover, when headache diagnoses and muscular tenderness scores were included in the same multivariable model, the latter was the only factor significantly associated with tinnitus. This result suggests that cervical muscle tenderness and pericranial muscle tenderness scores are actually associated with tinnitus, and that headache diagnoses may be considered confounding factors (Table IV).

We previously reported that muscle tenderness scores (cervical muscle tenderness and pericranial muscle tenderness) were related to the prevalence of anxiety in headache patients.[4] Indeed, anxiety and depression independently increased the likelihood of

https://doi.org/10.1017/S0022215115001425 Published online by Cambridge University Press

muscle tenderness, regardless of the diagnostic group.[4] It is therefore possible that the relationship between muscle tenderness and tinnitus largely depends on their association with anxiety, although it is unclear whether psychiatric co-morbidities are causes or consequences of tinnitus. The increased likelihood of suffering from tinnitus with increased muscle tenderness could also be explained by the presence of the somatic form of tinnitus, i.e. the phenomena of neural plasticity that are involved in the pathophysiology of chronic pain may also play a role in tinnitus development. Sound information, in addition to being processed along the 'classical' (or lemniscal) pathway (which is entirely auditory and tonotopic), can also travel along a 'nonclassical' (or extralemniscal) path.[24] The extralemniscal pathway is not normally involved in hearing and receives information from the somatosensory system, but may be affected in the central neural reorganisation that causes tinnitus.[25] This mechanism is similar to the one proposed for phantom limb sensation.[9] Thus, patients with hearing loss and reduced cochlear afferences may develop an extended topographical map that contributes to tinnitus (a phantom sensation) development. This mechanism could explain why most studies report that tinnitus is centred in the region of greatest hearing loss.[24] Interactions between the somatosensory, somatomotor and visual pathways of the extralemniscal pathway may also explain why some patients can alter their tinnitus perception by activating one of these pathways, as previously reported.[6]

This study did not find a correlation between tinnitus and TMJ disorders, but did identify a relationship between tinnitus and myogenous pain and, to some extent, migraine (when combined with myogenous pain in the multivariable model). This finding suggests an interaction between myogenous pain and migraine. That is, migraine becomes significantly associated with tinnitus only when myogenous pain is also present. Since, in our study, tinnitus prevalence increased when a TMJ disorder was co-morbid with other craniofacial pain types (for instance, migraine, tension-type headache or myogenous pain), it is likely that the previously reported association between TMJ disorders and otological symptoms (such as tinnitus) may be mediated by co-morbid pathologies (such as headache) which had not been investigated.

A possible association of tinnitus with migraine also deserves consideration. A previous study reported tinnitus in up to 26 per cent of patients with basilar-type migraine (an uncommon type of migraine with aura).[26] Volcy and colleagues hypothesised that migraine-associated tinnitus depends on neurogenic plasma extravasation in the inner-ear tissues due to permeability changes in the inner ear related to migraine-associated artery vasospasm.[27] They further suggested that defective ion channels (shared by the brain and inner ear) could lead to reversible hair cell depolarisation. According to this hypothesis, tinnitus development may rely on spontaneous aberrant neural activity at any level along the auditory axis, with abnormal reorganisation processes occurring in the auditory cortex following hearing receptor damage.

This study showed that 28 per cent ($n = 40$) of migraine patients referred to our pain unit reported tinnitus; this is similar to tinnitus prevalence in the general population. It is notable that when a diagnosis of migraine was considered together with other diagnoses, the percentage with tinnitus increased to 34 per cent ($n = 111$). As the univariable analysis did not show a strong association between migraine and tinnitus ($p = 0.11$), it is likely that co-morbidities, mainly psychiatric ones, can increase cervical and cranial muscle tenderness. This may partly explain the high tinnitus prevalence in patients with migraine associated with other types of headache.

There are limited data on the association between tinnitus and tension-type headache available. To our knowledge, no other studies have focused on the relation between tinnitus and tension-type headache in such a large patient sample ($n = 245$). Although tinnitus was present in 70 out of 245 patients diagnosed with tension-type headache, there was no direct association between tension-type headache and tinnitus.

Our data is limited by possible bias because it was a retrospective study based on data from patients who had been referred to the Headache and Facial Pain Unit for headache and/or facial pain, and not for complaints of tinnitus. Other diseases had previously been excluded or were excluded at the time of the visit to our unit; however, an otological examination did not form part of the standardised assessment. Moreover, a precise classification of 'ear ringing' by the use of a questionnaire such as the Tinnitus Handicap Inventory[28] was not used; therefore, a standardised tool for evaluating the severity of the patients' tinnitus was lacking.

- **Tinnitus is related to increased cervical and pericranial muscle tenderness**
- **Tinnitus is not related to any type of headache**
- **Careful muscle palpation may identify tinnitus related to craniofacial pain**
- **Treatments which decrease muscle tenderness may also alleviate tinnitus**

Nevertheless, the current and previous studies provide evidence for a correlation between positive muscle palpation and the presence of dizziness and tinnitus, indicating the clinical importance of performing careful muscle palpation in patients suffering from headache and craniofacial pain.[12] Moreover, treatment based on a musculoskeletal examination should reduce both neck pain and tinnitus, as previously demonstrated.[18]

https://doi.org/10.1017/S0022215115001425 Published online by Cambridge University Press

Conclusion

This study showed that of the different types of facial pain, only myogenous pain was significantly related to tinnitus in the univariable analysis, although migraine co-morbidity may have also played a role. Tinnitus was linked to increased cervical and pericranial muscle tenderness rather than to any particular form of headache. This study highlights the importance of correct, careful muscle palpation in patients suffering from tinnitus along with headache and craniofacial pain. Additional longitudinal studies are needed to confirm these findings, elucidate the complex interplay among craniofacial pain, muscle tenderness and psychiatric co-morbidity, and to determine whether treatments which decrease muscle tenderness can also alleviate tinnitus.

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Dr M Pezzoli takes responsibility for the integrity of the content of the paper
Competing interests: None declared