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Somatic tinnitus prevalence and treatment with tinnitus retraining therapy

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Abstract. *Somatic tinnitus prevalence and treatment with tinnitus retraining therapy*. *Objectives*: Somatic tinnitus originates from increased activity of the dorsal cochlear nucleus, a cross-point between the somatic and auditory systems. Its activity can be modified by auditory stimulation or somatic system manipulation. Thus, sound enrichment and white noise stimulation might decrease tinnitus and associated somatic symptoms. The present uncontrolled study sought to determine somatic tinnitus prevalence among tinnitus sufferers, and to investigate whether sound therapy with counselling (tinnitus retraining therapy; TRT) may decrease tinnitus-associated somatic symptoms.
Methods: To determine somatic tinnitus prevalence, 70 patients following the TRT protocol completed the Jastreboff Structured Interview (JSI) with additional questions regarding the presence and type of somatic symptoms. Among 21 somatic tinnitus patients, we further investigated the effects of TRT on tinnitus-associated facial dysesthesia. Before and after three months of TRT, tinnitus severity was evaluated using the Tinnitus Handicap Inventory (THI), and facial dysesthesia was assessed with an extended JSI-based questionnaire.
Results: Among the evaluated tinnitus patients, 56% presented somatic tinnitus -including 51% with facial dysesthesia, 36% who could modulate tinnitus by head and neck movements, and 13% with both conditions. Self-evaluation indicated that TRT significantly improved tinnitus and facial dysesthesia in 76% of patients. Three months of TRT led to a 50% decrease in mean THI and JSI scores regarding facial dysesthesia.
Conclusions: Somatic tinnitus is a frequent and underestimated condition. We suggest an extension of the JSI, including specific questions regarding somatic tinnitus. TRT significantly improved tinnitus and accompanying facial dysesthesia, and could be a useful somatic tinnitus treatment.

Introduction

Somatic tinnitus is defined as a tinnitus that is somatically modulated in intensity and pitch by head and neck movements, or that is associated with facial dysesthesia (i.e. abnormal facial sensation, such as tingling, electric shocks, or sensation of swelling, pain or burning). It is hypothesized that tinnitus has a central subcortical aetiology. Peripheral cochlear damage leads to re-organisation of the auditory cortex tonotopic map, with overrepresentation of adjacent tinnitus-related frequencies and increased neural synchronisation and spontaneous neural firing rate.[1] Furthermore, auditory deafferentation may disrupt inhibitory and regulating top-down mechanisms in auditory central areas, as well as in nonauditory central areas like the fronto-parietal cortex, the insula, and the limbic system.[2] Tinnitus generation is particularly linked to peripheral cochlear damage that triggers inhibition release and thus increased activity of the dorsal cochlear nucleus (DCN).[3-6] The same behaviour was recently described for the ventral cochlear nucleus (VCN).[7-8]

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Compliance with Ethical Standards

Disclosure of potential conflicts of interest: The authors declare that they have no conflict of interest.

Research involving Human Participants and/or Animals:

Ethical approval: All procedures in this study involving human participants were performed in accordance with the ethical standards of the institutional research committee (CHU Saint Pierre, Brussels, Belgium, O.M. 007) and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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This article does not contain any experiments with animals performed by any of the authors.

Informed consent: Informed consent was obtained from all individual participants included in the study.

The association between tinnitus and somatic symptoms suggests the presence of neural connections between the somatosensory and auditory systems. It has been described that tinnitus and DCN activities can be modulated by somatic system manipulation. Shore *et al.* revealed projections from the trigeminal ganglion to both CN.[9-10] Electric stimulation of trigeminal ganglion induces VCN neural activation, and both excitation and inhibition are observed in different DCN units, with inhibitory preponderance creating an overall imbalance of neural activity. Dehmel *et al.*[11] reported increased DCN neural excitation in animals suffering from tinnitus. Interestingly, deaf animals showed higher DCN neuron activity upon trigeminal stimulation than normal hearing control animals, suggesting that hearing loss may influence the excitation-inhibition balance induced by somatosensory stimulation, thus enhancing DCN activity and tinnitus generation.

Analysis of the subcortical distribution of the vesicular glutamate transporters VGLUT1 and VGLUT2 revealed increased glutamatergic projections from the trigeminal and cuneate nucleus (somatosensory system) to both CN after induced unilateral hearing loss.[12] VGLUT1 is prominent in the VCN magnocellular zone, which receives afferent auditory fibres. Three weeks after induced hearing loss, VCN exhibits decreased VGLUT1, which can be explained by auditory deafferentation. On the other hand, both CN show increased VGLUT2, which is especially prevalent in the granular zone of DCN receiving neural projections from trigeminal and cuneate nucleus. These observations suggest that hearing loss leads to the development of new and compensating somatic projections to both CN, which could result in increased susceptibility for tinnitus that then will be more easily influenced by somatic stimulation.

It is hypothesized that tinnitus might be decreased by restoring DCN inhibition via either somatic or auditory stimulation, such as electrical stimulation of the auditory nerve, and cochlear and auditory brainstem implants.[13] Hearing aids are also reportedly effective in patients with mild hearing loss.[14] Several studies show effective tinnitus treatment using tinnitus retraining therapy (TRT),[16-18] although there remains a lack of randomised studies and conclusive evidence.[15] The underlying mechanism could be two-fold, involving weakening of tinnitus-related neuronal hyperactivity by auditory stimulation, along with habituation of negative emotional reactions to tinnitus via combined counselling and cognitive behavioural therapy. The limbic system is strongly involved in both tinnitus perception and anxiety generation.[17,19] Thus, sound enrichment combined with counselling therapy could further decrease tinnitus-related facial dysesthesia by restoring normal neural activity in DCN.

The present study aimed first to establish the prevalence and characteristics of somatic tinnitus, particularly of associated facial dysesthesia, among tinnitus sufferers at an ENT outpatient department. Secondly, as little is known about the specific treatment of somatic tinnitus, we also investigated the effect of TRT on tinnitus and associated facial dysesthesia.

Material and methods

In the first part of this study, we investigated the prevalence of somatic tinnitus within a current ENT outpatient population. This part of the study included every tinnitus patient with or without facial dysesthesia consulting our outpatient department over a five-month period. A total of 70 patients with complaints of uni- or bilateral tinnitus agreed to follow the TRT protocol and were enrolled. To undergo TRT, patients had to fulfil the following criteria: THI score of >40 (including moderate, severe, and catastrophic tinnitus, and excluding slight and mild tinnitus according to Newman's THI classification[20]), age of <70 years, no associated pathology (acoustic neuroma, Ménière's disease, etc.), and no objective tinnitus (perceivable by another person or caused by other internal body noises, for example of vascular origin). The mean patient age was 49.39 years.

To investigate the presence and characteristics of tinnitus-associated somatic symptoms, we developed an extension of the Jastreboff Structured Interview (JSIe) that includes additional questions regarding facial dysesthesia presence and severity, type of abnormal sensation (e.g. tingling, tension, heat, or pain), ability to modulate tinnitus via somatic modification, and any link between tinnitus and facial dysesthesia intensity (Table 1, items 1d and 6-8). One positive answer to the facial dysesthesia questions was necessary and sufficient for inclusion in the group of somatic tinnitus

Table 1

Extended questionnaire based on Jastreboff Structured Interview (JSIe)

1) Indicate the importance of each problem in your daily life.

	No problem					Very important problem
a. Tinnitus	0	1	2	3	4	5
b. Hearing loss	0	1	2	3	4	5
c. Hyperacusis	0	1	2	3	4	5
d. Facial dysesthesia	0	1	2	3	4	5

2) What is the intensity of your tinnitus?
0 – 1 – 2 – 3 – 4 – 5 – 6 – 7 – 8 – 9 – 10

3) What percentage of time are you disturbed by your tinnitus?
0 – 10 – 20 – 30 – 40 – 50 – 60 – 70 – 80 – 90 – 100%

4) What impact does your tinnitus have on your daily life?
0 – 1 – 2 – 3 – 4 – 5 – 6 – 7 – 8 – 9 – 10

5) How much are you disturbed by your tinnitus?
0 – 1 – 2 – 3 – 4 – 5 – 6 – 7 – 8 – 9 – 10

6) Do you feel any facial sensation that you did not experience before the occurrence of your tinnitus? Yes / No

If yes, which sensation?

– Tingling sensation on the side of tinnitus or bilaterally
– Sensation of swelling on the side of tinnitus or bilaterally
– Tension, electric shocks, or pain on the side of tinnitus or bilaterally
– Sensation of heat or burning on the side of tinnitus or bilaterally
– Sensation of breath on the face on the side of tinnitus or bilaterally

7) Can you modulate your tinnitus by acting on your face? Yes / No

If yes, how?

– Increase tinnitus by pressure on the face or by biting on the teeth
– Decrease tinnitus by pressure on the face or by biting on the teeth
– Frequency variation by pressure on the face or by biting on the teeth

8) Are those facial sensations linked to you tinnitus (more intense on days when tinnitus increases)? Yes / No

Totally – Frequently – Regularly – Sometimes – No, it is independent

To identify and define somatic tinnitus, we included additional questions regarding the presence and severity of facial dysesthesia, type of abnormal sensation, the possibility of modulating tinnitus by any somatic action, and the presence of a link between tinnitus intensity and facial dysesthesia (items 1d and 6-8).

sufferers. To evaluate the correlation between two dichotomous variables, we used statistic tables based on the "Phi" coefficient. To measure the degree of linear correlation between two numeric variables, we calculated the Pearson product-moment correlation coefficient "r".

In the second prospective part of our study, the effects of acoustic stimulation and counselling (TRT protocol) on tinnitus and somatic symptoms were investigated. From our initial tinnitus patient sample, 21 patients with a primary complaint of tinnitus (THI >40%) associated with facial pain or

dysesthesia were enrolled in this portion of the study. The mean age was 48.67 years. Patients with known temporomandibular joint dysfunction were excluded from this group. For a three-month period, all patients underwent a TRT protocol that included daily use (mean of 12 hours) of uni- or bilateral white noise generators (depending on whether the patient suffered from uni- or bilateral tinnitus) set to the mixing point, with or without amplification depending on the patient's audiometric profile. The patients also completed four structured counselling sessions, which included explanation and discussion of the neurophysiological model of tinnitus proposed by Jastreboff, cochlear physiology, tinnitus aetiology with cortical and subcortical mechanisms, the habituation process, the vicious circle of tinnitus, stress, hyperacusis and facial dysesthesia, the role of white noise stimulation, and the aims of therapy.

Before and after TRT, tinnitus severity was assessed using the Tinnitus Handicap Inventory (THI) and both tinnitus and facial dysesthesia were examined using the JSIe. To evaluate not only the presence and type of facial dysesthesia but also its severity and evolution before and after TRT, we added two more questions to the JSIe questionnaire, which related to facial dysesthesia awareness and annoyance and were answered using a visual analogic scale (Table 2, items 9 and 10). Statistical analyses were again performed using the "Phi" and "r" coefficients. Our patient sample was not normally distributed (qualitative characteristic of the data); thus, we used the non-parametric Wilcoxon signed-rank test to compare the numeric responses to the THI and to items 1, 9, and 10 of the JSIe before and after treatment.

Results

Investigating somatic tinnitus prevalence in our ENT outpatient population revealed two types: tinnitus that could be modulated in intensity and/or frequency by manipulating regions of the head and neck, and tinnitus associated with facial pain or dysesthesia. Patients with at least one of these types were considered somatic tinnitus sufferers. Based on this definition, 56% of our patient sample presented somatic tinnitus (39 patients; Figure 1), including 20 patients (51%) suffering from associated facial dysesthesia, 14 (36%) who could modulate tinnitus by head and neck movements, and 5 (13%) with both conditions (Figure 2).

Among the 25 patients suffering from tinnitus-associated facial dysesthesia, 14 (56%) indicated substantial discomfort or pain with a score of three or more on the five-point facial dysesthesia analogic scale (item 1 of JSIe). Furthermore, 80% of the patients suffering from tinnitus-associated facial dysesthesia stated that subjective tinnitus intensity was correlated with facial dysesthesia intensity, with 38% answering totally, 21% frequently, 17% regularly, and 4% sometimes on item 8 of the JSIe. For these patients, facial dysesthesia or pain was more intense with increased tinnitus, and vice versa. We found no evidence that facial dysesthesia intensity impacted tinnitus severity (items 2-5 of JSIe and THI). Facial dysesthesia occurred before tinnitus onset in 50% of patients, tinnitus started before facial dysesthesia onset in 43% of patients, and both started simultaneously in 7% of patients. Among 93% of patients, bilateral facial dysesthesia co-existed with bilateral tinnitus, and ipsilateral discomfort co-existed with unilateral tinnitus.

Table 2

Extended questionnaire based on Jastreboff Structured Interview (JSIe) – facial dysesthesia awareness and annoyance

9) What percentage of time are you disturbed by your facial dysesthesia (facial dysesthesia awareness)?

0% 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%

10) What impact does facial dysesthesia have on your daily life (facial dysesthesia annoyance)?

0 1 2 3 4 5 6 7 8 9 10

No impact Unbearable

To evaluate the severity and evolution of facial dysesthesia before and after three months of tinnitus retraining therapy, we added to the questionnaire two more questions related to facial dysesthesia awareness (item 9) and annoyance (item 10) using a visual analogic scale.

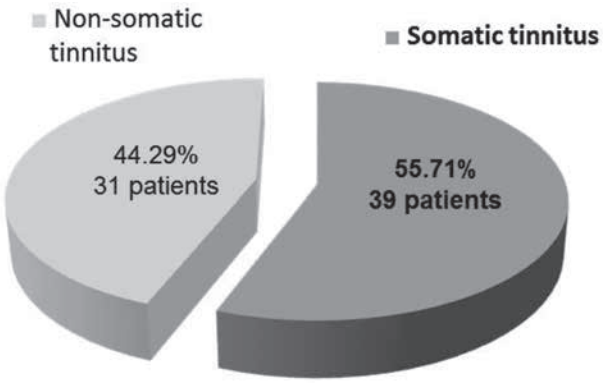

Figure 1
Population of somatic tinnitus patients within the whole tinnitus patient sample.

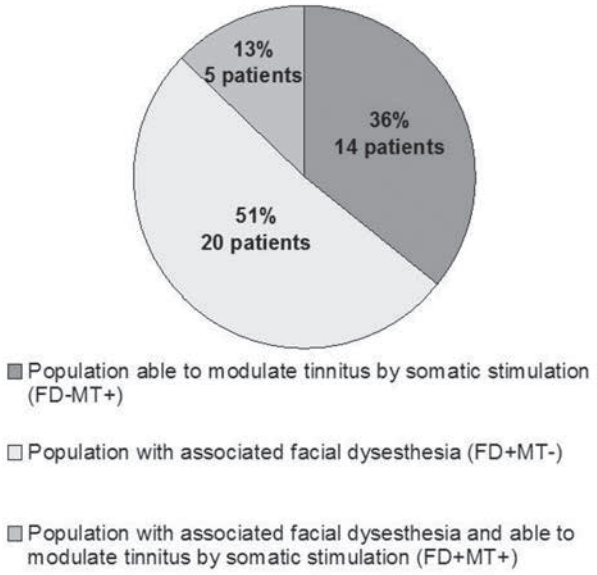

Figure 2
Composition of somatic tinnitus patient population

Among the 19 patients who could modulate tinnitus through head and neck movements, 95% (18 patients) reported that somatic stimulation led to increased tinnitus perception or variation in tinnitus frequency, while only 5% of patients (1 patient) could decrease tinnitus intensity.

Having established these characteristics of our tinnitus population, we further investigated the effect of TRT on tinnitus and associated facial dysesthesia or pain. Self-evaluation revealed that TRT significantly improved tinnitus and facial dysesthesia in 76% of patients (16 of 21). After three months of TRT, the mean THI score decreased from 67.14% to 32.19%, with a mean decrease of 34.95% ($P<0.05$) (Figure 3a). Only one patient showed a worsened THI score. TRT also decreased the subjective complaints of tinnitus, hearing loss, hyperacusis, and facial dysesthesia evaluated by item 1 of the JSIe. Interestingly, facial dysesthesia complaint showed the most substantial decrease: 2.81 mean points pre-TRT and 0.88 mean points post-TRT, with a mean decrease of 38.57% ($P<0.05$) (Figure 3b). The mean JSIe score for facial dysesthesia awareness (item 9 of the JSIe) was reduced from 37.38% to 12.38%, with a mean decrease of 25% ($P<0.05$). The mean JSIe score for facial dysesthesia annoyance (item 10 of the JSIe) was reduced from 3.5 to 1.12, with a mean decrease of 23.81% ($P<0.05$) (Figure 3c).

Discussion

Somatic tinnitus is not a rare condition: more than half of our tinnitus patient sample presented associated somatic symptoms. Despite this high prevalence, patients rarely link somatic complaints to their tinnitus, and currently existing questionnaires do not include items regarding complaints of facial dysesthesia. Therefore, we developed an extension of the Jastreboff Structured Interview (JSIe) that includes additional questions about somatic symptoms. This questionnaire could represent an essential tool for evaluation and documentation of somatic tinnitus presence and characteristics. The questionnaire should be integrated in current tinnitus evaluations and ENT practice to identify somatic tinnitus.

Use of our extended questionnaire revealed two types of somatic tinnitus: tinnitus associated with facial dysesthesia, and tinnitus that can be induced and modulated by somatic stimulation. We found that 27% of our tinnitus population could modulate tinnitus via head and neck movements, confirming the previously described influence of the somatosensory system on tinnitus.[21] However, our results differ from those of Levine *et al*.[24] who reported that 80% of their tinnitus patients could modulate tinnitus by somatic stimulation. As mentioned above, Shore *et al*.[10] identified afferent excitatory projections from the spinal trigeminal nucleus to both DC. Moreover, somatic stimulation influences CN activity balance – especially in cases of associated hearing loss – and thus may evoke tinnitus. Prior studies have investigated multiple

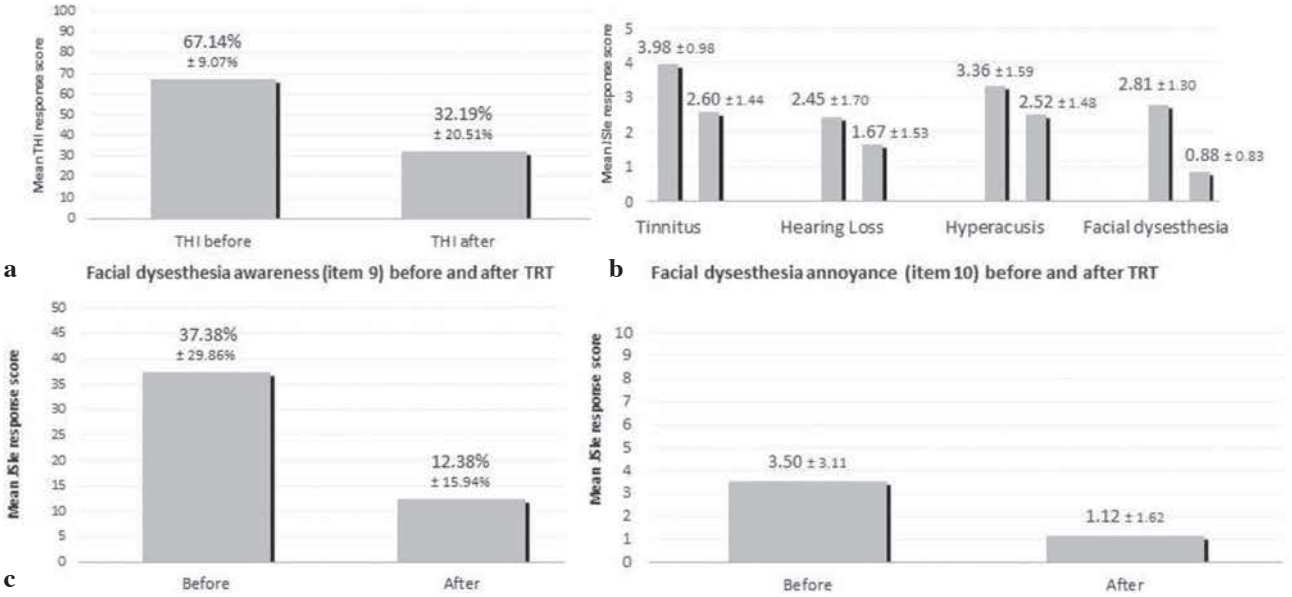

Figure 3

(a) After three months of tinnitus retraining therapy (TRT), the mean Tinnitus Handicap Inventory (THI) score decreased from 67.14% to 32.19%. (b) Item 1 of the extended Jastreboff Structured Interview (JSIe) evaluated the subjective complaints of tinnitus, hearing loss, hyperacusis, and facial dysesthesia. After three months of TRT, the score on item 1 decreased, especially regarding the complaint of facial dysesthesia, with a mean of 2.81 points before TRT and 0.88 points after TRT and a mean decrease of 38.57% ($P<0.05$). (c) Evolution of facial dysesthesia awareness and annoyance (items 9 and 10 of the JSIe) before and after TRT.

treatment options that are based on the concept that tinnitus is a consequence of a somatic disorder, and can thus be modified through somatic input. For example, local lidocaine injection into head and neck muscles and myofascial trigger points, acupuncture, and manual therapies aiming to relax jaw and neck muscle tension have seemed to be effective in some tinnitus patients.[22-23]

Associated facial dysesthesia is a less explored phenomenon. Thus, it is notable that about half of our somatic tinnitus patients presented associated facial dysesthesia that occurred before (in 50% of patients) or after (in 43% of patients) tinnitus onset. Our clinical study showed that TRT reduced both tinnitus and associated facial dysesthesia. Interestingly, complaints of facial dysesthesia decreased first and more substantially than complaints of tinnitus. The underlying mechanism could involve acoustic stimulation inducing partial re-establishment of normal neural activity in the DCN – the cross-point between auditory and somatic pathways – or at a higher central level. The post-TRT improvements may indicate the existence of neural pathways descending from the central nervous system to the peripheral somatosensory system, potentially via the medulla and parallel to the trigeminal afferent pathway. If such efferent nerve fibres exist, they could be regulated by the interconnection of auditory and nonauditory areas. Moreover, in the case of neural imbalance and hyperactivity as observed in tinnitus, they could generate and influence somatic perception.

Major limitations of our study include the absence of a control group and the relatively small sample size. It would have been interesting to compare the TRT-treated group with a group treated in another manner – for example by local lidocaine injection into head and neck muscles and myofascial trigger points, manual therapies, or counselling only. Additionally, the measurement of response to treatment was subjective, and dependent on the patient's self-evaluation, which may have been subject to a placebo effect. Moreover, the extended JSI questionnaire remains to be validated. Despite these limitations, our study results showed a positive effect of TRT on complaints of facial dysesthesia among somatic tinnitus patients, which warrants further investigation.

Conclusion

Somatic tinnitus is a much more common condition than previously believed. Here we identified two types of somatic tinnitus: tinnitus that can be modulated in intensity and/or frequency by manipulating regions of the head and neck, and tinnitus associated with facial pain or dysesthesia. We developed an extension of the Jastreboff Structured Interview that includes specific questions regarding somatic tinnitus, and we suggest the use of this JSIe in ENT practice.

Moreover, we found that tinnitus retraining therapy significantly improved tinnitus and accompanying facial dysesthesia within our somatic tinnitus patient sample. Despite the limited sample size, it was evidence that sound enrichment combined with counselling had positive effects on both symptoms. Thus, tinnitus retraining therapy might be an effective specific treatment for somatic tinnitus patients.

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