Comparison of Clinical Characteristics and Somatic Modulation between Somatic Tinnitus and Otic Tinnitus

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Key Words
Auditory responses · Cochlear nucleus · Neck · Somatosensory signals · Temporomandibular joint · Tinnitus

Abstract
Aims: To compare the clinical characteristics of tinnitus among patients with somatic tinnitus (ST), otic tinnitus with somatic modulation (OT+), and otic tinnitus without somatic modulation (OT–), and to examine differences in somatic modulation between patients with ST and OT+.

Methods: We retrospectively reviewed the medical records of 65 patients with unilateral tinnitus and classified the patients into three groups: ST (n = 24), OT+ (n = 21) and OT– (n = 20).

Results: Only one difference in clinical characteristics was found between the OT+ and OT– groups. Maneuvers related to the temporomandibular joint showed the highest frequency of somatic modulation in both the ST and OT+ groups. There was no significant difference between the ST and OT+ groups with regard to the rate of somatic modulation or the rate of increase or decrease of tinnitus among all aspects of somatic testing.

Conclusions: The presence or absence of somatic modulation does not influence the clinical characteristics of otic tinnitus, and the somatosensory-auditory interaction is a general auditory physiologic phenomenon.

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Introduction

Most cases of subjective tinnitus result from injury to the peripheral auditory organs [Kiang et al., 1970; Hazell and Jastreboff, 1990]. A decrease in afferent signals following cochlear injury causes a decrease in lateral inhibition in the area of the relevant frequency in the central auditory system, such as the dorsal cochlear nucleus (DCN) or inferior colliculus, followed by the development of hyperactivity in auditory neurons at the edge of this frequency [Eggermont, 2003; Ochi et al., 2003; Kaltenbach et al., 2005]. In some patients with tinnitus, the loudness or pitch changes with specific physical actions or in a specific posture, i.e. when contracting particular muscles. This phenomenon is termed ‘somatic modulation of tinnitus’ [Levine, 2004]. In a previous study, 40% of tinnitus patients reported an alteration in their tinnitus following stimulation of the median nerve [Moller et al., 1992]; in another study, a third of tinnitus patients described the same type of change when they clenched their jaw or temporomandibular joint (TMJ) [Rubinstein, 1993]. In another study, patients with TMJ disorder were highly likely to experience tinnitus [Wright and Bifano, 1997]. Levine et al. [2007] presented the hypothesis that somatosensory nerves such as the trigeminal nerve, vagus nerve, glossopharyngeal nerve and second cervical nerve (C2) are connected to the nerves of the DCN, and that alterations in signals coming from the somatosensory nerve cause tinnitus. This type of tinnitus is termed ‘somatic tinnitus’, whereas tinnitus based on the traditional hypothesis (including cochlear injury) is termed ‘otic tinnitus’. Although it has no fixed definition, somatic tinnitus must exclude otic tinnitus caused by cochlear injury. Therefore, the diagnosis of somatic tinnitus is limited to cases where the patient experiences tinnitus in the ipsilateral ear to somatic modulation, with normal hearing on the side with tinnitus, and to those who demonstrate symmetrical hearing loss with aging [Levine et al., 2007]. Patients with a history of noise exposure or ototoxic drug administration prior to the onset of tinnitus should be excluded from a diagnosis of somatic tinnitus; in contrast, if tinnitus occurs after TMJ impairment, dental treatment, or head-neck trauma, a diagnosis of somatic tinnitus may be considered. Because the pathophysiology differs between somatic tinnitus and otic tinnitus, different treatment methods should be applied.

No previous study has compared the clinical characteristics or somatic modulation between patients with somatic and otic tinnitus. Therefore, in the present study, we compared the clinical characteristics of tinnitus among patients with somatic tinnitus (ST group), otic tinnitus with somatic modulation (OT+ group) and otic tinnitus without somatic modulation (OT– group), and examined differences in somatic modulation between the ST and OT+ groups.

Materials and Methods

Subjects

The study group consisted of 65 patients with unilateral tinnitus who met the study criteria for somatic or otic tinnitus, and the participants were selected from 140 patients who visited the Department of Otolaryngology (Eulji Hospital, Korea) complaining of tinnitus and who underwent somatic testing between March and December 2010. We reviewed the medical records of the subjects and classified them into three groups: ST (n = 24), OT+ (n = 21) and OT– (n = 20).

Criteria for Somatic Tinnitus. All of the following criteria must be met for a diagnosis of somatic tinnitus.

1 A hearing threshold ≤20 dB HL in both ears in pure tone audiometry (PTA) at 0.25, 0.5, 1, 2, 4 and 8 kHz, and symmetric hearing ability within 10 dB HL difference of both ears.
in whole frequency PTA at 10, 12, 14, 16, 18 and 20 kHz, or symmetric sensorineural hearing loss (air-bone hearing gap of <10 dB HL) within 10 dB HL difference at 0.25, 0.5, 1, 2, 4 and 8 kHz.

(2) A history of TMJ disorder, head-neck disease, dental treatment immediately prior to experiencing tinnitus, or neck or TMJ tenderness on physical examination.

(3) Somatic modulation during more than one maneuver on the ipsilateral side to the tinnitus during somatic testing, with tinnitus confirmed on tinnitogram.

(4) Excluded were patients who experienced objective tinnitus, those with otologic disease, a family history of hereditary hearing impairment, excessive noise exposure and a history of ototoxic drug administration.

Criteria for Otic Tinnitus. All of the following criteria must be met for a diagnosis of otic tinnitus.

(1) A hearing threshold ≥20 dB HL in the ear with tinnitus compared with the opposite side in at least two consecutive frequencies in PTA performed at frequencies of 0.25, 0.5, 1, 2, 4 and 8 kHz, and sensorineural type of hearing loss (air-bone hearing gap ≥10 dB HL).

(2) Complaining of tinnitus in the ear with worse hearing, with the tinnitus confirmed at tinnitogram.

(3) Excluded were patients who experienced objective tinnitus, those with otologic disease and those with a family history of hereditary hearing impairment.

Procedure

Somatic Testing. To investigate whether the patients could modulate their tinnitus, the patient was seated in a soundproof room while an otolaryngologist performed 25 maneuvers designed to contract the cervical and TMJ muscles (table 1). The duration of the contractions was only a few seconds – just enough time for the subject to judge whether any change had occurred in their auditory perceptions. The patient was asked to classify the loudness of their tinnitus initially and throughout testing, using a scale of 0–10 (0 = no tinnitus and 10 = the loudest tolerable level). Somatic modulation was defined as an increase in loudness ≥1 [Levine et al., 2007].

Outcome Measures. All subjects were asked to complete a tinnitus questionnaire regarding the following: site of tinnitus, duration of tinnitus, subjective tinnitus loudness score (10-point scale), tinnitus awareness score indicating the percentage of time that the patient hears tinnitus while awake, the presence of vertigo, hyperacusis, ear fullness, TMJ disorder, recent dental treatment, herniated cervical disc, posterior neck pain and traffic accident. The Tinnitus Handicap Inventory (THI; 25 questions in total; range, 0–100) of Newman et al. [1995] was used to evaluate the degree of impairment caused by tinnitus in everyday life. The Beck Depression Inventory (BDI; total 21 questions; range, 0–63) was used to evaluate the degree of depression caused by tinnitus. A physical examination was performed to detect whether palpation in the following areas caused an increase in muscle tension or pain: both temples, jaw, cheek, tragus and retroauricular/cervical area. All subjects underwent audiological tests including PTA, impedance audiometry, tinnitogram, auditory brainstem responses and otoacoustic emissions. All patients underwent PTA at 0.25, 0.5, 1, 2, 4 and 8 kHz. If the hearing thresholds of both sites were <20 dB HL up to 8 kHz, PTA was performed at extended frequencies of 10, 12, 14, 16, 18 and 20 kHz.

Statistical Analysis

We compared the clinical characteristics among the ST, OT+ and OT− groups using one-way ANOVA, the Kruskal-Wallis test and the χ² test, with the Statistical Package for Social Sciences (SPSS 15.0; SPSS, Chicago, Ill., USA). A χ² test was used to compare the results of somatic testing. A value of p < 0.05 was taken to indicate statistical significance.
Results

Comparison of Clinical Characteristics among the Three Groups

Statistically significant differences among the three groups were found for mean tinnitus awareness score, mean tinnitus loudness score, mean THI score, mean BDI score, mean pure tone average, posterior neck pain and traffic accident (table 2; p < 0.05). In contrast, no statistical significance was found among the three groups for age, sex, site, mean duration of tinnitus, mean tinnitus pitch, past history of TMJ disorder, recent dental treatment, cervical herniated disc, or the presence of vertigo, hyperacusis, or ear fullness (table 2; p > 0.05).

The mean tinnitus awareness score was 64.8 ± 32.1% in the ST group, 73.3 ± 25.7% in the OT+ group, and 89.5 ± 20.4% in the OT− group; it was significantly lower in the ST group than in the OT− group (p < 0.05). Comparison of mean tinnitus loudness score between the ST group (5.1 ± 1.5) and the OT+ group (6.1 ± 1.8) showed no significant difference (p > 0.05), while that of the ST group was significantly less than that of the OT− group (6.7 ± 2.3; p < 0.05). The THI score was 33.1 ± 25.7 in the ST group and 45.7 ± 26.9 in the OT+ group (no significant difference; p > 0.05); THI score was significantly less in the ST group than in the OT− group (52.9 ± 21.6; p < 0.05). BDI score was 10.7 ± 7.6 in the ST group, 10.0 ± 7.8 in the OT+ group and 17.1 ± 7.5 in the OT− group; BDI score was significantly less in the ST
Comparison of the Results of Somatic Testing

The results of analysis of somatic modulation experienced during each of the somatic testing maneuvers revealed that of the 24 patients in the ST group, modulation was most common while protruding the jaw against resistance (test 5; 9/24, 37.5%). For each of the following maneuvers, 8 patients (33.3%) experienced somatic modulation: clenching the teeth (test 1), protruding the jaw (test 4), and turning the head to the left and tilting to the right (test 19; fig. 1).

Of the 21 patients in the OT+ group, 8 patients (38.1%) experienced somatic modulation when turning the head to the right and when resisting torsional force on the right zygoma (test 17). For each of the following maneuvers, 7 patients (33.3%) experienced somatic modu-

and OT+ groups compared with the OT– group (p < 0.05). Mean pure tone average was 16.0 ± 13.1 dB HL in the ST group, which was significantly less than that in the OT+ (35.0 ± 23.4 dB HL) and OT– (47.1 ± 25.4 dB HL) groups (p < 0.05). Posterior neck pain was reported by 54.2% (13/24) in the ST group, 19.0% (4/21) in the OT+ group and 10.0% (2/20) in the OT– group; a significantly higher percentage of the ST group reported posterior neck pain compared with the other two groups (p < 0.05). A history of a recent traffic accident was 20.8% (5/24) in the ST group but 0% in the other two groups; a history of a recent traffic accident was significantly higher in the ST group than in the other two groups (table 2; p < 0.05).

**Table 2.** Comparison of the clinical characteristics of tinnitus in the ST, OT+ and OT– groups

|                          | ST group (n = 24) | OT+ group (n = 21) | OT– group (n = 20) | p value |
|--------------------------|------------------|-------------------|-------------------|---------|
| Mean age, years          | 47.6 ± 16.6      | 52.5 ± 15.2       | 58.5 ± 14.5       | 0.077   |
| Males/females            | 8/16             | 11/10             | 10/10             | 0.371   |
| Site, right/left         | 11/13            | 10/11             | 7/13              | 0.676   |
| **Questionnaire**         |                  |                   |                   |         |
| Mean duration of tinnitus, months | 25.6 ± 38.6   | 25.1 ± 77.4       | 45.2 ± 64.2       | 0.485   |
| Mean tinnitus awareness score, % | 64.8 ± 32.1  | 73.3 ± 25.7       | 89.5 ± 20.4       | 0.018a  |
| Mean tinnitus loudness score (0–10) | 5.1 ± 1.5    | 6.1 ± 1.8         | 6.7 ± 2.3         | 0.024a  |
| Mean THI score (0–100 scores) | 33.1 ± 25.7 | 45.7 ± 26.9       | 52.9 ± 21.6       | 0.042a  |
| Mean BDI score (0–63 scores) | 10.7 ± 7.6  | 10.0 ± 7.8        | 17.1 ± 7.5        | 0.008b, c |
| **Audiologic test**      |                  |                   |                   |         |
| Mean tinnitus pitch, kHz | 5.4 ± 2.9        | 3.5 ± 2.9         | 4.2 ± 3.4         | 0.379   |
| Mean pure-tone average (affected ear), dB HL | 16.0 ± 13.1  | 35.0 ± 23.4       | 47.1 ± 25.4       | 0.000b, b |
| **Past history**         |                  |                   |                   |         |
| TMJ disorder             | 2/24             | 3/21              | 0/20              | 0.227   |
| Recent dental treatment  | 1/24             | 3/21              | 0/20              | 0.150   |
| Cervical herniated disc  | 3/24             | 4/21              | 2/20              | 0.684   |
| Posterior neck pain      | 13/24            | 4/21              | 2/20              | 0.003b, b |
| Traffic accident         | 5/24             | 0/21              | 0/20              | 0.010b, b |
| **Associated symptoms**  |                  |                   |                   |         |
| Vertigo                  | 9/24             | 8/21              | 8/20              | 0.965   |
| Hyperacusis              | 7/24             | 7/21              | 4/20              | 0.671   |
| Ear fullness             | 9/24             | 10/21             | 9/20              | 0.813   |

a p < 0.05, ST group vs. OT– group; b p < 0.05, ST group vs. OT+ group; c p < 0.05, OT+ group vs. OT– group (χ² test, Kruskal-Wallis test and one-way ANOVA).
lation: clenching the teeth (test 1), protruding the jaw (test 4) and protruding the jaw against resistance (test 5; fig. 1).

The results of analysis of the number of maneuvers that caused modulation during somatic testing for each of the 24 patients in the ST group revealed the highest frequency in 4 patients who experienced somatic modulation during 12 of the total 25 maneuvers. Eight patients showed somatic modulation during only 1 maneuver. Of the total 25 maneuvers, the average number of maneuvers that caused somatic modulation was 5.0 ± 4.3 per patient (fig. 2).

Analysis of the number of maneuvers that caused modulation during somatic testing for each of the 21 patients in the OT+ group revealed the highest number in 1 patient who experienced somatic modulation during 15 maneuvers, while 6 patients showed somatic modulation in only 1 maneuver. Of the total 25 maneuvers, the average number of maneuvers that caused somatic modulation was 4.0 ± 3.8 per patient (fig. 3). There was no difference in the average frequency of somatic modulation in each patient between the ST and OT+ groups (p > 0.05).

Analysis of somatic testing of the ST group (24 patients) and OT+ group (21 patients) indicated that in the ST group, 480 of the total 600 maneuvers (25 maneuvers × 24 patients; 80.0%) caused no change in tinnitus, 51 maneuvers (8.5%) caused a decrease in tinnitus, and 69 maneuvers (11.5%) caused an increase in tinnitus. In the OT+ group, 440 of the total 525 maneuvers (25 maneuvers × 21 patients; 83.8%) caused no change in tinnitus, 44 maneuvers (8.4%) caused a decrease in tinnitus, and 41 (7.8%) an increase in tinnitus. There was no difference between the two groups in terms of the total frequency of somatic modulation and
the total rates of increase or decrease in tinnitus for all 25 maneuvers taken together (table 3; p > 0.05). For each maneuver alone, comparison of the frequency of somatic modulation and the rates of increase or decrease between the ST and OT+ groups revealed a significant difference only for pressure applied to the forehead (test 11): 5 patients in the ST group (20.8%) experienced a decrease in tinnitus, while 2 patients in the OT+ group (9.5%) experienced an increase in tinnitus (table 3; p < 0.05).
Discussion

The present study examines the hypothesis that because somatic tinnitus and otic tinnitus result from different mechanisms, they may have different clinical characteristics, and that there are two subgroups in otic tinnitus: with and without somatic modulation. The present results showed no significant difference between the clinical characteristics of the OT+ and OT− groups except for mean BDI score, which indicates that there is no difference in the mechanism between the two types of otic tinnitus. Factors showing a significant difference between the ST and OT− groups were mean pure tone average, presence of posterior neck pain and a history of a recent traffic accident. These results reflect the characteristics of somatic tinnitus, which is generally diagnosed with normal hearing ability, the presence of cervical pain and a history of cervical disease or injury.

Many factors showed significant differences between the ST and OT− groups: significantly increased values were found for the OT− group in terms of mean tinnitus awareness score, mean tinnitus loudness score, mean THI score, mean BDI score and mean pure tone average, while the ST group showed significantly increased values for frequency of posterior neck pain and a recent traffic accident. This finding suggests that OT− patients experience a longer period in time and louder tinnitus than do ST patients, resulting in a higher degree of handicap and depression in their daily life. In the case of otic tinnitus, abnormal spontaneous neural activity of the auditory system occurs constantly because the auditory stimulus delivered from the cochlea to the central auditory system decreases constantly. In contrast, a change in the

Table 3. Results of somatic testing of the ST and OT+ groups

|        | ST group (n = 24) | OT+ group (n = 21) | p value |
|--------|------------------|-------------------|---------|
|        | no change | decreased | increased | no change | decreased | increased |         |
| 1 Clench | 16 (66.7%) | 1 (4.2%) | 7 (29.2%) | 14 (66.7%) | 3 (14.3%) | 4 (19.0%) | 0.415   |
| 2 Open jaw | 20 (83.3%) | 1 (4.2%) | 3 (12.5%) | 18 (85.7%) | 3 (14.3%) | 0 (0%) | 0.141  |
| 3 + resistance | 18 (75.0%) | 1 (4.2%) | 5 (20.8%) | 18 (85.7%) | 3 (14.3%) | 0 (0%) | 0.054  |
| 4 Protrude jaw | 16 (66.7%) | 1 (4.2%) | 7 (29.2%) | 14 (66.7%) | 3 (14.3%) | 4 (19.0%) | 0.415  |
| 5 + resistance | 15 (62.5%) | 2 (8.3%) | 7 (29.2%) | 14 (66.7%) | 3 (14.3%) | 4 (19.0%) | 0.652  |
| 6 Right jaw | 21 (87.5%) | 2 (8.3%) | 1 (4.2%) | 17 (78.0%) | 1 (4.8%) | 3 (14.3%) | 0.458  |
| 7 + resistance | 19 (79.2%) | 1 (4.2%) | 4 (16.7%) | 15 (71.4%) | 4 (19.0%) | 2 (9.5%) | 0.253  |
| 8 Left jaw | 18 (75.0%) | 2 (8.3%) | 4 (16.7%) | 18 (85.7%) | 2 (9.5%) | 1 (4.8%) | 0.448  |
| 9 + resistance | 17 (70.8%) | 2 (8.3%) | 5 (20.8%) | 16 (76.2%) | 4 (19.0%) | 1 (4.8%) | 0.204  |
| 10 Retract jaw | 19 (79.2%) | 3 (12.5%) | 2 (8.3%) | 20 (95.2%) | 0 (0%) | 1 (4.8%) | 0.205  |
| 11 Forehead | 19 (79.2%) | 5 (20.8%) | 0 (0%) | 19 (90.5%) | 0 (0%) | 2 (9.5%) | 0.033* |
| 12 Occiput | 21 (87.5%) | 2 (8.3%) | 3 (12.5%) | 19 (90.5%) | 1 (4.8%) | 1 (4.8%) | 0.889  |
| 13 Vertex | 23 (95.8%) | 0 (0%) | 1 (4.2%) | 20 (95.2%) | 0 (0%) | 1 (4.8%) | 0.923  |
| 14 L temple | 18 (75.0%) | 3 (12.5%) | 3 (12.5%) | 18 (85.7%) | 2 (9.5%) | 1 (4.8%) | 0.605  |
| 15 R temple | 22 (91.7%) | 2 (8.3%) | 0 (0%) | 17 (81.0%) | 2 (9.5%) | 2 (9.5%) | 0.293  |
| 16 L turn | 19 (79.2%) | 2 (8.3%) | 3 (12.5%) | 16 (76.2%) | 2 (9.5%) | 3 (14.3%) | 0.972  |
| 17 R turn | 18 (75.0%) | 4 (16.7%) | 2 (8.3%) | 13 (61.9%) | 4 (19.0%) | 4 (19.0%) | 0.528  |
| 18 Turn R tilt L | 18 (75.0%) | 4 (16.7%) | 2 (8.3%) | 16 (76.2%) | 1 (4.8%) | 4 (19.0%) | 0.302  |
| 19 Turn L tilt R | 16 (66.7%) | 5 (20.8%) | 3 (12.5%) | 16 (76.2%) | 3 (14.3%) | 2 (9.5%) | 0.778  |
| 20 Right SCM | 19 (79.2%) | 2 (8.3%) | 3 (12.5%) | 20 (95.2%) | 1 (4.8%) | 0 (0%) | 0.187  |
| 21 Left SCM | 18 (75.0%) | 3 (12.5%) | 3 (12.5%) | 19 (90.5%) | 1 (4.8%) | 1 (4.8%) | 0.399  |
| 22 Right SC | 21 (87.5%) | 2 (8.3%) | 1 (4.2%) | 21 (100%) | 0 (0%) | 0 (0%) | 0.245  |
| 23 Left SC | 21 (87.5%) | 1 (4.2%) | 2 (8.3%) | 20 (95.2%) | 1 (4.8%) | 0 (0%) | 0.400  |
| 24 Right post. auri. | 24 (100%) | 0 (0%) | 0 (0%) | 21 (100%) | 0 (0%) | 0 (0%) | 1.000  |
| 25 Left post. auri. | 24 (100%) | 0 (0%) | 0 (0%) | 21 (100%) | 0 (0%) | 0 (0%) | 1.000  |

Total 600 480 (80.0%) 51 (8.5%) 69 (11.5%) 525 440 (83.8%) 44 (8.4%) 41 (7.8%)“

For comparison between two groups, a χ² test was used (* p < 0.05). For further information, see table 1. L = Left; R = right; + resistance = against resistance; SCM = sternocleidomastoid muscle; SC = splenius capitus; post. auri. = posterior auricle.
tension in the cervical muscles, which is assumed to be a cause of somatic tinnitus, is affected by various postures and by physical conditions, and can thus alter the degree of tinnitus to reduce the inconvenience caused by tinnitus in the ST group. A limitation of the present study is that the small sample size may have led to no differences being detected with regard to the factors of a history of TMJ disorder, recent dental treatment or cervical herniated disc.

The loudness of tinnitus is changed when the DCN is stimulated electrically [Soussi and Otto, 1994], and spontaneous neural activity of the DCN is increased when agents that cause tinnitus are administered [Kaltenbach and Zhang, 2007]. In addition, hyperactivity of the DCN is generated after intense sound exposure [Kaltenbach and Afman, 2000]. These previous studies support the hypothesis that the DCN plays a role in the generation and modulation of tinnitus. It was previously shown that a change in the tension in cervical muscles and TMJ disorder are related to tinnitus [Rubinstein, 1993], and that this phenomenon suggests a certain connection between the central auditory pathway and the somatic nerve originating from the head-and-neck region.

A previous study of deaf patients with cochlear implants who were tested with their implants disconnected showed that 50% (5/10) of patients with ongoing tinnitus could modulate their tinnitus with somatic modulation, while 67% (2/3) of patients without tinnitus could elicit an auditory percept during somatic testing [Levine et al., 2003]. This result indicates a somatosensory-auditory neural interaction within the central nervous system without any interference from auditory inputs, and because somatically modulated tinnitus is most commonly perceived in one ear, this finding suggests that somatosensory-auditory interactions occur within the afferent central auditory pathway prior to the auditory decussation, before the level of the superior olivary complex. The only auditory nuclei before the auditory decussation are the DCN and ventral cochlear nucleus [Levine et al., 2007]. Levine et al. [2007] suggest the following DCN disinhibition hypothesis of tinnitus: sensory input from the upper cervical dorsal roots and 4 cranial nerves (V, VII, IX and X) converge to a common region in the lower part of the medulla, where fibers project from the medullary somatosensory nuclei to the ipsilateral DCN. The changes in somatosensory signals depending on head-and-neck conditions or following trauma reduce the inhibitory regulation of the DCN, eventually accelerating spontaneous activity within the DCN [Levine, 1999a]. A recent neuroanatomical study demonstrated that the DCN actually receives signals from auditory and somatosensory nerves [Shore et al., 2007]; according to this study, pyramidal cells in layer II of the DCN receive inputs on their basal dendrites from auditory nerve fibers and apical dendrites from granule cells innervated from the trigeminal ganglion, and at the same time receive an inhibitory signal from cartwheel and stellate cells. Changes in somatosensory signals generated in cervical muscles accelerate spontaneous activity within the DCN by reducing inhibitory signals from cartwheel and stellate cells through weakened excitatory signals from granule cells.

In the present study, we used somatic testing [Levine et al., 2007], including 25 maneuvers that included jaw and head-and-neck contractions, and pressure on muscle insertions. In a previous study of 18 patients who showed normal hearing in PTA up to 8 kHz, 12 patients (66.7%) with tinnitus experienced a higher hearing threshold on the affected side in the extended high-frequency band compared with that in a control group of the same age and sex [Shim et al., 2009]; therefore, it is necessary to test PTA beyond 8 kHz to obtain an exact diagnosis of somatic tinnitus. For this reason, patients included in the ST group were only those with symmetric hearing ability (within 10 dB HL difference) in whole frequency of PTA that included 10, 12, 14, 16, 18 and 20 kHz, if the hearing threshold was below 20 dB HL in both ears at less than 8 kHz.

A previous study of 82 subjects and 25 maneuvers, including extremity contraction, found that somatic modulation occurred most commonly in protruding the jaw against resistance (40.5%) and during application of pressure to the forehead (40.3%) [Abel and Levine,
In another study, 45 tinnitus patients underwent somatic testing consisting of 42 maneuvers including movement of the eyes, extremities, head, neck and shoulder; the results showed that among the 1,890 total maneuvers (42 movements × 45 subjects), 15.9% (300 of 1,890) resulted in somatic modulation. The maneuvers that most commonly modulated tinnitus perception were midline jaw thrust (35.6%), right jaw clench (31.1%), active neck extension (28.9%) and right shoulder rotation with resistance (28.9%) [Simmons et al., 2008]. In the ST group of the present study, protruding the jaw against resistance was the most common maneuver to cause somatic modulation (9/24, 37.5%). Although the most common maneuver causing somatic modulation in the OT+ group was turning the head to the right (test 17; 8/21, 38.1%), the next most common were three maneuvers associated with jaw movement (7/21, 33.3% in each). Clearly, maneuvers related to jaw movement were most likely to cause somatic modulation in both groups, in agreement with the results of previous studies, confirming that the pterygoid muscle is the main muscle involved in somatic modulation and explaining why tinnitus is common in patients with TMJ disorder.

The number of patients with tinnitus reported to experience modulation in somatic testing varies among previous reports. Studies of systematic somatic modulation reported somatic modulation of tinnitus in 68.6–80.5% of tinnitus patients [Levine, 1999b; Abel and Levine, 2004; Simmons et al., 2008]. Although it is difficult to determine how often general somatic modulation occurred in the tinnitus patients in the present study, we calculated the frequency of somatic modulation for each maneuver: of the total maneuvers, somatic modulation occurred in 20.0% of patients in the ST group and in 16.2% in the OT+ group.

Recent treatments for tinnitus that target the somatosensory system include cervical manipulation [Alcantara et al., 2002], acupuncture [Hansen et al., 1982], electrical stimulation of the scalp and auricle [Engelberg and Bauer, 1985; Lyttkens et al., 1986], treatment of TMJ disorder [Wright and Bifano, 1997], treatment utilizing myofascial trigger points [Rocha and Sanchez, 2007] and transcutaneous electrical nerve stimulation [Herraiz et al., 2007; Vanneste et al., 2010]. Some studies have confirmed the efficiency of these treatments, but few long-term results have been reported. Therefore, further research of treatment related to the somatosensory system, performed in subjects with somatic tinnitus, is necessary to evaluate and analyze the effectiveness of the treatment.

We conclude that (a) in otic tinnitus, the presence or absence of somatic modulation does not influence the clinical characteristics of otic tinnitus; (b) in somatic tinnitus, symptoms are weaker and of shorter duration than those in otic tinnitus, and therefore somatic tinnitus patients feel a lesser degree of handicap and depression in daily life; (c) TMJ-related maneuvers showed the highest frequency of somatic modulation in both somatic tinnitus and otic tinnitus patients, and (d) there was no significant difference in somatic modulation between the otic tinnitus and somatic tinnitus groups in terms of the frequency of somatic modulation or the rate of increase or decrease of tinnitus over all of the somatic testing maneuvers, implying that the somatosensory-auditory interaction is a general auditory physiologic phenomenon. Based on these conclusions, in addition to the somatic testing performed when diagnosing somatic tinnitus, it is necessary to determine any previous history of head-and-neck disease, to examine the head-and-neck region and to perform a hearing test (including extended high-frequency testing).

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