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REVIEW

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CNS somatosensory-auditory interactions elicit or modulate tinnitus

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Abstract Evidence has accumulated linking clinical tinnitus to the somatosensory system. Most clinical tinnitus patients can change the psychoacoustic attributes of their tinnitus with forceful head and neck contractions. The significance of such somatic modulation of tinnitus was assessed by testing non-clinical subjects. Like clinical tinnitus patients, about 80% of non-clinical subjects who had ongoing tinnitus at the time of testing (whether or not they had been previously aware of it) could modulate their tinnitus with head and neck contractions. Almost 60% of those with no tinnitus at the time of testing could elicit a tinnitus-like auditory percept with head and neck contractions. Because similar results were found in the profoundly deaf, we conclude that somatosensory-auditory interactions within the central nervous system account for most, if not all, somatic modulation of tinnitus as well as the development of auditory percepts with somatic testing. Other observations implicate the muscle spindle as initiating the neural activation that ultimately modulates the central auditory pathway, including the dorsal cochlear nucleus. Somatic influences upon auditory perception are not limited to tinnitus subjects but are a fundamental property of the auditory system.

Keywords Tinnitus · Somatosensory · Auditory · Muscle spindle · Cochlear nucleus

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Introduction

Tinnitus is a many faceted symptom. It may be due to a sound generated by an acoustic source within the body (objective tinnitus) or it may have no acoustic source (subjective tinnitus), in which case it is a purely neural phenomenon. Subjective tinnitus has been characterized by (1) assessing its psychoacoustic attributes such as with pitch and loudness matching, as well as with the effect of external sounds upon its perception (masking properties), or (2) its affective features through psychological profiles of the patient.

Another aspect of subjective tinnitus that has received much less attention is the somatic component. We have previously (1) described for normal hearing subjects the "somatic tinnitus syndrome": unilateral tinnitus temporally associated with, and ipsilateral to, a somatic disorder involving the head or upper neck and (2) suggested a physiological mechanism involving somatosensory interactions with the dorsal cochlear nucleus to account for the somatic tinnitus syndrome (Levine 1999a).

It has long been known, almost as a curiosity, that some people can modulate their tinnitus somatically. Møller et al. showed that median nerve stimulation could modulate tinnitus in close to 40% of subjects (Møller et al. 1992). Rubinstein found that about a third of her subjects could influence their tinnitus with jaw movements or pressure on the temporomandibular joint (Rubinstein 1993). When interviewed, about 20% of our tinnitus clinic patients report they can somatically modulate the acoustic properties of their tinnitus by head and neck movements or muscle contractions such as clenching the teeth together (Levine and Kiang 1995). In a systematic study of 70 consecutive patients seen in our tinnitus clinic we found that 68% could somatically modulate their tinnitus (Levine 1999b). However, the significance of this finding is uncertain, since there have been no reports of the effects of muscle contraction upon auditory perception in non-clinical subjects.

To understand the significance of somatic modulation in our tinnitus clinic patients, we studied the effect of somatic contractions upon spontaneous auditory perceptions in (1) a non-clinical population and (2) the profoundly deaf. Our results support the view that somatic modulation is more than a curiosity restricted to tinnitus patients, but is a fundamental property of the human auditory system. Furthermore our findings suggest that it is somatosensory-auditory interactions within the central nervous system which account for somatic modulation of tinnitus, just as they account for the somatic tinnitus syndrome.

Materials and methods

Subjects

Sixty-two adults recruited from our personal contacts comprised the non-clinical population of this study (39 males, 23 females, ages 18–74 years, mean age = 41 years), while 13 adults recruited from the Massachusetts Eye and Ear Infirmary cochlear implant research laboratory comprised the profoundly deaf group (7 males, 6 females, ages 37–69 years, mean age = 51 years). The cochlear implant subjects were postlingually profoundly deaf bilaterally. All had received a unilateral cochlear implant between 9 months and 16 years earlier (mean = 8 years).

Prior to somatic testing, the following questions were asked of each subject:

1. Do you have normal hearing?
2. Do you ever have a brief ring in one ear, lasting about a minute or less?
3. Do your ears ever ring after a loud sound?
4. Do you have tinnitus?

Procedure

Subjects were asked to report any changes in their auditory perception, including changes in loudness (louder or softer), pitch or location. They were instructed to rate the loudness of their ongoing or elicited tinnitus on a numerical scale where zero corresponds to no tinnitus and ten corresponds to a sound at the loudest level they could tolerate.

Somatic testing consisted of 25 brief, forceful contractions made by each subject while sitting in a room with low-level ambient noise. Duration of contractions was only a few seconds—just enough time for the subject to judge whether any change occurred in his/her auditory perceptions. If loudness changed with a contraction, the subject provided a new number corresponding to the tinnitus loudness at the time of the change. Pitch and location changes were recorded qualitatively. The contractions chosen for testing were motivated by (1) reports from our patients that they could change their tinnitus with pressure on the head (Levine and Kiang 1995) and (2) the reports of Møller and Rubinstein which implicated the jaw and extremities as sources of somatic modulation (Møller et al. 1992; Rubinstein 1993).

Ten contractions involved the jaw as follows:

1) Clench the teeth together as forcefully as possible,

2, 3) Maximally open the mouth, with and without maximal restorative pressure applied by the heel of the subject or examiner's hand,

4, 5) Maximally protrude forward the jaw, with and without maximal restorative pressure,

6, 7) Maximally slide the jaw to the left, with and without maximal restorative pressure,

8, 9) Maximally slide jaw to right, with and without maximal restorative pressure, and

10) Maximally retract jaw.

Ten involved head and neck contractions as follows:

With the head in the neutral position, contractions were made to resist maximal pressure applied by the examiner to the:

11) Occiput,
12) Forehead,
13) Vertex,
14) Mandible (upward),
15) Right temple, or
16) Left temple.
17) With the head turned to the right, resist maximal tortional force applied by the examiner to the right zygoma.
18) With the head turned to the left, resist maximal tortional force applied by the examiner to the left zygoma.
19) With the head turned to the right and tilted to the left, maximally resist full force applied by the examiner to the left temple (left sternocleidomastoid)
20) With the head turned to the left and tilted to the right, maximally resist full force applied by the examiner to the right temple (right sternocleidomastoid)

Five involved extremity contractions as follows:

21) Locking the subject's flexed fingers of the two hands together and pulling them apart as forcefully as possible.

Contractions were made to resist maximal pressure applied by the examiner to the subject's

22) Abducted right shoulder,
23) Abducted left shoulder,
24) Flexed right hip, or
25) Flexed left hip.

Results

A. Non-clinical subjects

Nine subjects (15%) reported a mild hearing loss (audiograms were not obtained). Forty-eight subjects (77%) had recalled experiencing a tonal ring in one ear lasting less than a minute. Thirty-three subjects (53%) reported that they had experienced tinnitus after a loud sound.

Twelve of the 62 subjects (19%) reported prior to testing that they had been aware of ongoing tinnitus (Table 1). The ratings of their tinnitus loudness ranged between 0.5 to seven with a mean of 2.5. Almost half of these subjects (5) perceived their tinnitus as lateralized to one ear; the tinnitus loudness ratings of these five spanned the entire range.

Another 17 subjects (27%) were unaware of any ongoing tinnitus, but, immediately after entering and sitting in a quiet room prior to somatic testing, when questioned again they reported hearing something that no one else in the room heard (i.e. tinnitus). The ratings of their tinnitus loudness ranged from between less than one to five with more than 80% at one or less. Only two of these subjects perceived their tinnitus as lateralized to one

Table 1 Subtypes of non-clinical subjects

Subject type	Number
Previously aware of tinnitus	12 (19%)
Not previously aware of tinnitus	17 (27%)
Without tinnitus	33 (53%)
Total	62

ear but these two subjects had the largest tinnitus loudness ratings of this group.

From combining these two tinnitus groups together, overall nearly half (47%) of all the non-clinical subjects had tinnitus in the quiet at the time of testing.

Somatic modulation

A variety of descriptions were used by the subjects to describe the change in the hearing percept that occurred with somatic testing. These included "ring, high-pitched ring, or tone." We did not consider descriptions of "clicking" as tinnitus, since this probably represented joint or muscle generated sounds such as those coming from the temporomandibular joint or Eustachian tube. Descriptions such as "wind" or "snowstorm" were also not considered tinnitus since these likely represent tensor tympani contraction as has been described for tinnitus associated with forced eye closure (Rock 1995).

Overall 23 of the 29 subjects (79%) who had ongoing tinnitus in the quiet test room modulated their tinnitus with somatic testing (Table 2). Whether or not the subjects had been previously aware of their tinnitus, the incidence of somatic modulation was essentially no different (83% and 76% respectively). In contrast, of the 33 subjects who had no ongoing tinnitus in the quiet, only 19 (58%) developed an acoustic perception (tinnitus) with somatic testing.

Alterations in tinnitus perception that occurred with somatic testing included changes in loudness, pitch or location. By far the most common were changes in tinnitus loudness that could be either louder or softer, or louder for some maneuvers and quieter for others in the same subject. Of these increased loudness alone was the most frequent (Table 3). Five of the subjects with ongoing tinnitus at the time of testing could increase their tinnitus loudness with some contractions and decrease it with others; in four of these five their tinnitus was not perceptible with at least one of the maneuvers. Pitch changes were described by 13 of the subjects; in all but one of them loudness changes also occurred. Four of the subjects described the pitch change as a new sound in addition to their baseline tinnitus percept, which continued to be present unchanged.

Sometimes the effects of somatic testing were prolonged. In 21% of subjects who could change or induce tinnitus with a somatic manipulation, the effect persisted after the contraction was released. This effect could be for a few seconds or up to 10 min. The two longest ones (5 and 10 min) occurred in subjects who had had no tinnitus before the somatic modulation testing.

Table 2 Somatic modulation in non-clinical subjects as related to whether or not they were experiencing tinnitus at the time of testing

Subject type	Somatic modulation		Totals
	Present	Not present	
With ongoing tinnitus	23 (79%)	6 (21%)	29
Without tinnitus	19 (58%)	14 (42)	33
Totals	42 (68%)	20 (32%)	62

Table 3 Loudness changes with somatic testing reported by subjects with ongoing tinnitus using the zero to ten loudness scale

Direction of change	No. subjects	Range		Mean
		Minimum	Maximum	
Increase in loudness	20	0.5	5	2.2
Decrease in loudness	7	0.1	3	1.0

The maneuvers that altered a subject's tinnitus varied from subject to subject. On average seven different maneuvers altered a subject's auditory perception (range 1–20). This average and range was about the same whether or not a subject had tinnitus at the time of testing.

Head and neck contractions changed tinnitus more effectively than extremity contractions. Without exception whenever extremity maneuvers modulated or elicited tinnitus, head and neck maneuvers also did in the same manner, but the reverse was not always true. In fact, twice as many subjects could modify or elicit tinnitus with head and neck contractions as with extremity contractions. Whichever the direction of the loudness changes, those elicited by head and neck maneuvers in any subject were always equal to or larger than those from extremity maneuvers of the same subject.

B. Profoundly deaf subjects

The 13 profoundly deaf subjects were tested with their cochlear implant processor disconnected from the patient. At the time of testing ten subjects had ongoing tinnitus and three did not. Five of the ten (50%) with ongoing tinnitus could modulate their tinnitus with somatic testing, while two of the three (67%) without tinnitus could elicit an auditory percept with somatic testing. As with hearing subjects loudness changes were the most common type of somatic modulation. Of the five with ongoing tinnitus three increased and two decreased their tinnitus loudness with somatic testing. No subjects of this group did both. Likewise the effects of somatic testing could persist; the longest was for a subject whose tinnitus disappeared for 4 min. Pitch changes also occurred, but only in one subject. For this subject, loudness increased for some maneuvers, and pitch for others. In all cases head and neck alterations in auditory perception were more effective than those induced by extremity maneuvers.

Discussion

This two part study demonstrates that somatic modulation of tinnitus is widespread and is not restricted to clinical patients. Our previous study in our clinic population found that 68% of 70 consecutive tinnitus patients could modulate their tinnitus with somatic testing (Levine 1999b). The present study is not exactly comparable because it included more types of maneuvers, particularly involving the jaw. When these additional maneuvers are included in testing our clinic patients (unpublished results), we have found virtually identical incidence of somatic modulation as in the non-clinical group. In both groups about 80% of subjects with ongoing tinnitus at the time of testing could modulate their tinnitus with somatic testing. Other characteristics were likewise similar in the two groups; these include (a) loudness changes were much more common than pitch or location changes, (b) changes were usually transient with the contraction, but occasionally could persist for as long as 10 min after the maneuver, and (c) head and neck contractions were always more effective than those of the extremities. Thus, somatic modulation is as common in the non-clinical tinnitus subjects as in clinical tinnitus patients. The ability to somatically modulate is therefore not what makes tinnitus a clinical problem.

Furthermore in the present report we have demonstrated that an auditory perception (tinnitus) could be elicited in 58% of non-clinical subjects who in the quiet were hearing nothing at the time of somatic testing. This result suggests that it is likely that some cases of clinical tinnitus may be due to activation of latent somatic-auditory interactions. Such activations could account for some cases of clinical tinnitus including the somatic tinnitus syndrome (Levine 1999a). One such example is as follows.

A 29-year-old woman with normal audiometry had for 7 months highly distressing right ear tinnitus, which had resolved about 2 months prior to her visit to our tinnitus clinic. On physical examination there was increased muscle tension and tenderness in her right sternocleidomastoid as compared to the left. At the time of somatic testing she was hearing slight constant ringing of both ears (1/10), which was much fainter than her prior right ear tinnitus. With somatic testing, each time her right sternocleidomastoid muscle was forcefully contracted she reported hearing right ear tinnitus identical to her prior distressing tinnitus (Table 4). The right ear tinnitus did not persist after her somatic testing.

Our findings can account for the fact that the incidence of change in auditory perception with somatic testing is significantly lower in subjects with no tinnitus than in subjects with tinnitus. From our observations of clinical and non-clinical tinnitus subjects, it is clear that somatic testing can increase and/or decrease tinnitus loudness and pitch. If such changes in auditory perception are happening equally for all groups of subjects, then one would expect the incidence of a change in auditory perception to be less in people without tinnitus than in those who have an ongoing auditory perception (tinnitus) for at least two reasons: (1) If somatic modulation decreases the loudness of auditory perception, then this would not be detected in the non-tinnitus subjects; similarly for any change of pitch. (2) If somatic testing causes only a small degree of change in the level of activity in the auditory system, such as might be perceived in tinnitus subjects as a small change in their tinnitus loudness, then subjects without tinnitus might raise the overall activity in their auditory pathways, but not enough to cross the threshold of auditory perception. Thus, for these two reasons the incidence of change in auditory perception will be lower in the non-tinnitus groups than the tinnitus groups.

Several possible underlying mechanisms for changes in auditory perception with somatic testing can be considered. One is that the changes in auditory perception are due to changes in otoacoustic emissions (OAEs). We cannot totally exclude this possibility, but some of our subjects who have spontaneous OAEs heard nothing in the quiet and it is well established that the vast majority of subjects with spontaneous OAEs never hear them (Wilson 1986).

Since somatic testing consists of forceful muscle contractions, the generation by a muscle of an acoustic sound that is heard by the ear is an obvious possibility. This study has excluded from consideration elicited sounds that have been described with such terms as "wind" or "snow storm" since these are highly likely to be generated by tensor tympani and/or pharyngeal muscle contractions (Rock 1995). Nonetheless, a case has been described of "continuous high-frequency unilateral [objective] tinnitus" that disappeared with sectioning of the stapedius and tensor tympani tendons (Bento et al. 1998). If a similar phenomenon occurs with somatic testing, then this could account for many of our findings. However, in none of our subjects was the modulation "objective," since a high pitched sound coming from the ear has never been heard by others during somatic testing. Moreover we have made ear canal microphone recordings in some subjects during somatic modulation of their tinnitus and detected no signal corresponding to the change in auditory perception described by the subject. Also, subjects who can decrease the loudness of their tinnitus with somatic testing are highly unlikely to be doing so with muscular generation of an acoustic sound.

Our findings in the cochlear implant subjects with and without tinnitus, who describe changes in their auditory perceptions with somatic testing, clearly indicate that acoustic sounds are not responsible for their results, since these subjects are profoundly deaf and their implant was not activated at the time of somatic testing. Furthermore the similarities in the characteristics of the changes in auditory perception that occur for all groups suggest that the mechanism operating in the profoundly deaf is likewise operating for most, if not all, of the other groups. Thus we conclude that somatosensory-auditory neural

Table 4 Somatic testing of 29-year-old woman with prior right ear tinnitus

Condition[a]	Right ear tinnitus	Left ear tinnitus
Baseline (0–10 scale)	1	1
Forehead pressure	4	1
Mandibular pressure	4	1
Right sternocleidomastoid	4	1

[a]Only maneuvers that modulated her tinnitus are shown

interactions within the central nervous system are accounting for most, if not all, somatic modulation of tinnitus and the development of auditory percepts with somatic testing.

The neural system responsible for somatic modulation of auditory perception (somatic modulation of tinnitus) is likely the motor system and not the cutaneous sensory system. Never in our clinical experience or from clinical reports have intact patients reported that light touch can modify auditory perception. The only report of cutaneous stimulation causing modulation of auditory perception is in two patients who had been deafened by posterior fossa surgery (Cacace et al. 1999). It is almost exclusively non-noxious but forceful muscle contractions that elicit modulations of auditory perception. Such contractions involve (a) the entire voluntary efferent motor system (motor cortex, corticospinal and corticobulbar tracts, and primary motor neurons), and (b) the motor afferent system, which begins with the deep muscle receptors such as the muscle spindles and tendon organs.

Further insights implicating the motor afferents have come from two clinical observations. In one subject muscle fatigue abolished somatic modulation of auditory perception.

A 57-year-old man with lifelong non-lateralized tinnitus could increase the loudness of his tinnitus with jaw protrusion from an estimated loudness of 4/10 to 6/10. After being a subject of an fMRI experiment in which he repeatedly modulated his tinnitus with jaw protrusion for about 1 h, for the next 3–4 days he could not modulate his tinnitus. His ability to modulate with jaw protrusion then gradually returned over about a day.

Muscle fatigue is principally due to fatigue (weakening) of the muscle fibers. Unlike the motor efferent system, the motor afferent system requires contraction of the muscle fibers to be activated. Therefore, muscle fatigue inactivates the motor afferent system.

The other observation points specifically to the muscle spindle.

A 69-year-old woman with tinnitus for about 2½ years described her tinnitus as hissing and varying in location and intensity. When soft, it was heard in the right ear and when loud throughout the head. It was never heard in the left ear only. At the time of her examination her tinnitus was very loud and perceived throughout the head. She rated its loudness as 8 (on a 0 to 10 scale). On examination her left sternocleidomastoid muscle was taut and tender; her right sternocleidomastoid was normal. Vibration (with a hand-held massager) applied to her right sternocleido-mastoid did not alter her tinnitus. However, when applied to her left sternocleidomastoid, she noticed that the tinnitus gradually became quieter (4/10) over about 5 min and shifted its location from throughout the head to the right ear only. It remained quieter and in the right ear for about 30 min after the massage treatment.

Because vibration is known to be a potent activator of muscle spindles (Brown et al. 1967), this result provides further support for the hypothesis that muscle spindle activation is a mediator of somatic modulation.

Hence, these observations support the hypothesis that the muscle spindles of the motor afferent system are responsible for somatic modulation of auditory perception. This conclusion is consistent with Kanold and Young's findings (2001), which implicate activation of the cat's pinna muscle spindles as the likely source of neural activity affecting ultimately the dorsal cochlear nucleus of the central auditory pathway.

Possible sites of neural somatosensory-auditory interactions include the inferior colliculus, since it is known to exhibit tinnitus-related abnormalities (Melcher et al. 2000) and it receives somatosensory inputs (Aitkin et al. 1981). Experimentally, the firing of all units in the cat central nucleus of the inferior colliculus can be somatically modulated (Davis 1999).

Dorsal cochlear nucleus appears to be critical when tinnitus is due to ear disorders (Kaltenbach and McCaslin 1996) and is an established site of somatosensory-auditory interaction (Kanold and Young 2001). Furthermore, such interactions within the dorsal cochlear nucleus can account for the clinical features of the somatic tinnitus syndrome (Levine 1999a).

If the change in auditory perception is unilateral, then the dorsal cochlear nucleus becomes a highly likely site for the site of somatic-auditory interaction, as we have proposed previously to explain the unilateral tinnitus of the somatic tinnitus syndrome. Non-lateralized tinnitus suggests either bilateral cochlear nucleus or some higher center such as the inferior colliculus.

We conclude that (a) somatic modulation of tinnitus demonstrates the interaction of the central auditory nervous system with the central somatosensory system, (b) an auditory sensation can be created by somatosensory stimulation alone, (c) somatosensory modulation of auditory perception probably originates peripherally from muscle spindles, and (d) because somatic testing can suppress some subjects' tinnitus, understanding somato-sensory-auditory interactions could lead to more effective tinnitus treatments. Somatosensory-auditory neural interactions are critical not only for understanding clinical tinnitus, but also for understanding auditory perception in general.

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