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CHAPTER 17

Evidence for a tinnitus subgroup responsive to somatosensory based treatment modalities

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Abstract: Studies have established that the somatosensory system of the upper cervical region and head can be intimately involved in tinnitus. Tinnitus can arise directly from a disorder of the head and upper neck through activation of the somatosensory system. "Somatic testing" (a series of strong muscle contractions of the head and neck) can (1) modulate the tinnitus percept of ~80% of people with ongoing tinnitus, and (2) elicit a sound percept in ~50% of people with no tinnitus. These somatic phenomena are equally prevalent among people with or without functioning cochlea. Likely neural pathways underlying both the induction and modulation of tinnitus have been revealed in animal studies. Because somatic influences are fundamental to the operation of the auditory system, in general, and to tinnitus, in particular, somatic testing should be incorporated into all evaluations of tinnitus (1) to improve understanding of the role of the somatosensory system in any individual and (2) to identify subgroups of tinnitus patients who may respond to a particular treatment modality (as has already been shown for the tinnitus associated with temporomandibular disorder). Our clinical experience and review of reports of treatment modalities directed toward the somatosensory system supports the hypothesis that these modalities can benefit individuals with symmetric hearing thresholds but asymmetric widely fluctuating tinnitus. Treatment modalities involving the somatosensory system should be re-assessed by targeting this tinnitus subgroup.

Keywords: acupuncture; trigger point injections; temporomandibular; dorsal cochlear nucleus; electrical

Introduction

As a symptom and not a disease, tinnitus has many causes (see Chapter 1). The most common neurological system associated with the abnormal auditory perception known as tinnitus is, of course, the auditory system. However, an experienced clinician once observed, "If the probability is assessed as being over 50% that a particular condition is causing the tinnitus, … most cases of tinnitus would have to be classified as 'unknown'" (i.e., idiopathic) (Coles, 1996). His statement implies that a disordered ear or auditory nerve cannot be implicated as causing tinnitus with a high level of certainty in the majority of people. On the other hand there is evidence for an association between the degree of hearing loss and the likelihood of developing tinnitus. This fact can be deduced from two well-established facts regarding tinnitus' prevalence: (a) in adults with no hearing impairment

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~10% report tinnitus (Levine et al., 2003) and (b) in adults with profound hearing loss ~80% report tinnitus (Chung et al., 1984; Levine, 1999) (see Chapter 1). Yet, frequently hearing loss per se cannot be implicated as the immediate precipitant or "trigger factor" for tinnitus onset in either of these populations. A typical example is as follows.

Case 1. A 57-year-old man reported 4 months of constant ringing or roaring tinnitus in both ears. He reported no associated symptoms with the tinnitus onset; in particular he had no dizziness, change in his hearing, or ear pressure. He denied trauma, recent dental work, or change in medications at or near the time of his tinnitus onset. An audiogram following his tinnitus onset was unchanged as compared to one from 2 years earlier. Pure tone thresholds were symmetric. He had normal hearing thresholds at 1 kHz and below. Above 1 kHz thresholds were progressively increasing reaching 45 dB HL at 8 kHz. Speech discrimination scores were normal. His otoneurological examination was unremarkable.

While in this case a symmetric mild hearing loss was present, with his tinnitus onset no other hearing or vestibular symptom were noted by the patient and his audiogram was unchanged. On the one hand, his hearing loss would appear to be a risk factor for his development of tinnitus. On the other hand, the cause for its onset 4 months prior to our evaluation could not be determined. Only when hearing loss and tinnitus develop acutely together can the two be considered causally related.

The somatosensory system and tinnitus

Of all the sensory systems other than auditory, only the somatosensory system has been shown to be closely related to tinnitus. Observations abound supporting the notion that head and neck somatic events can be associated with tinnitus (Levine, 1999). Tinnitus is generally included among the features associated with discomfort in the temporal or preauricular region that goes by various names such as Costen's syndrome, craniomandibular disorder, temporomandibular joint syndrome, or temporomandibular disorder (TMD). Studies have shown a higher incidence of tinnitus in individuals with normal hearing who have TMD as compared to controls (Chole and Parker, 1992). The same is true regarding the whiplash syndrome, where the association has been attributed to "functional disturbances of the upper cervical spine" (Tjell et al., 1999). From multiple other observations and case reports the concept of tinnitus associated with whiplash and TMD can be generalized to include tinnitus associated with any disorder of the upper cervical region and head, including dental pain (Wyant, 1979; Curtis, 1980; Levine, 1999).

Somatic modulation of tinnitus

Even before entering our clinic, ~20% of patients have already noticed that they can "somatically modulate" their tinnitus, for instance alter the loudness or pitch of their tinnitus by clenching their teeth together or pushing on various places on the head (Levine and Kiang, 1995). The following presentation of tinnitus is a case in point.

Case 2. (Part 1) A 42-year-old man with normal pure tone thresholds reported that about a year earlier he had developed a constant "choppy tone" in his left ear that he could stop momentarily by biting down hard, or poking at his ear in various ways [see p.204 (PART 2)].

Cases like this one prompted us to systematically examine our patients with a battery of isometric head, neck and jaw contractions ("somatic testing" Table 1). We found that ~80% of tinnitus patients can transiently modulate their tinnitus to variable degrees with one or more of these maneuvers (Levine et al., 2003). A variety of changes can occur. Most commonly the tinnitus gets louder, but many times the tinnitus can becomes quieter. Less frequently patients describe pitch or location changes.

To assess whether somatic modulation is a general phenomenon and not restricted to individuals

Table 1. Somatic testing: maneuvers currently being used to test for tinnitus modulation

Jaw contractions	
1.	Clench teeth together
2, 3.	Open mouth, with and without restorative pressure
4, 5.	Protrude jaw, with and without restorative pressure
6, 7.	Slide jaw to left, with and without restorative pressure
8, 9.	Slide jaw to right, with and without restorative pressure
10.	Retract jaw
Head and neck contractions	
With the head in the neutral position, contractions were made to resist pressure applied by the examiner to:	
11.	forehead
12.	occiput
13.	vertex
14.	left temple
15.	Right temple
16.	with the head turned to the left, resist the tortional force on the left zygoma
17.	with the head turned to the right, resist the tortional force on the right zygoma
18.	with the head turned to the right and tilted to the left, resist force applied to the left temple (left sternocleidomastoid)
19.	With the head turned to the left and tilted to the right, resist force applied to the right temple (right sternocleidomastoid)
Pressure on muscle insertions	
20.	right mastoid attachment of sternocleidomastoid
21.	left mastoid attachment of sternocleidomastoid
22.	right suboccipital attachment of splenius capitis
23.	left suboccipital attachment of splenius capitis
	Posterior pinna pressure
24.	right pinna attachment of posterior auricular
25.	Left pinna attachment of posterior auricular

Note: all maneuvers use maximal force applied by the examiner.

who seek help for their tinnitus, we tested somatic modulation in 62 adult volunteers (Abel and Levine, 2004). Our only exclusion criterion was that they had never sought medical care for tinnitus. Twelve subjects (19%) knew that they had tinnitus, but did not consider it a problem; another 17 (27%) were unaware that they had tinnitus until they were brought into a room with very low-level ambient noise and were asked what they heard; the remaining 33 participants had no tinnitus even in quiet and even when pointedly questioned. Among the 29 people with tinnitus, 23 (again ~80%) could modulate their tinnitus with somatic testing. Hence, somatic modulation of tinnitus is not restricted to the clinical population; its incidence (80% with our test battery) appears to be the same for all populations with tinnitus and perhaps all causes of tinnitus. This fact implies that the ability to modulate tinnitus somatically is not what makes tinnitus a clinical problem.

In the 33 people with no tinnitus whatsoever, somatic testing elicited an acoustic percept ("tinnitus") in 19, or almost 60%. This observation suggests that somatic modulation of auditory function occurs commonly but only happens to be most obvious in people with tinnitus because they have an ongoing auditory percept (Sanchez et al., 2002; Abel and Levine, 2004).

An obvious question is whether the changes in tinnitus induced by somatic maneuvers might be caused by maneuver-induced sounds or changes in the auditory periphery (e.g., middle ear muscle contractions which might modulate externally or internally generated sounds). To examine this possibility, we somatically tested people who are deaf (individuals with cochlear implants with their implant disconnected) (Levine et al., 2003). Fourteen deaf individuals, 11 with ongoing tinnitus and 3 without tinnitus were tested in our standard manner (“somatic testing”). Six of the 11 (54%) with ongoing tinnitus could modulate their tinnitus with somatic stimulation, and 2 of the 3 (67%) without tinnitus could elicit an auditory percept with somatic testing. These percentages are close to those for people with hearing (54% and 60% as compared to 80% and 60% — “close” given the relatively small sample of deaf subjects). The nature of the tinnitus changes in deaf people was also comparable to those in hearing individuals. The prevalence of somatically induced tinnitus and tinnitus changes in deaf people strongly suggests that the strikingly similar effects in hearing people are not mediated acoustically, but instead arise from neural interactions between the somatosensory and auditory systems. The fact that somatic maneuvers most commonly changed tinnitus loudness in both hearing and deaf people suggests that the changes in both groups were subserved by the same neural mechanisms.

Given this evidence, the following conclusion seems almost inescapable: that somatosensory–auditory neural interactions within the central nervous system account for most, if not all, somatic modulations of tinnitus, as well as, the development of auditory percepts with somatic testing in those with no tinnitus.

There is good evidence that somatic modulation of tinnitus may be a special case of the general principle that auditory function can be modified by somatosensory input. One line of evidence comes from animal studies showing substantial convergence of the somatosensory system on nominally “auditory” centers throughout the brain (Aitkin, 1986; Shore et al., 2000). Another comes from direct demonstrations of somatosensory–auditory interactions in people (Lewald and Ehrenstein, 1998; Lewald et al., 1999). One study, particularly relevant in the present context, used electrical stimulation of the median nerve at the wrist as the somatic stimulus to induce alterations in the perception of external sound (Møller and Rollins, 2002). Sixty percent of participants (especially children) reported changes in the perception of external sounds to the stimulus. Of those whose perceptions were altered, all perceived a change in the loudness of the external sound (83% louder, 17% quieter). In an earlier study examining the effects of the same somatic stimulus (electrical stimulation of the median nerve) in people with tinnitus, Møller et al. (1992) found that tinnitus was modulated in close to 40% of participants. This percentage is much lower than that showing tinnitus modulation with somatic maneuvers, most likely because an upper extremity was stimulated, rather than the most sensitive regions for inducing somatic modulation, the lateral upper neck and head.

If the apparent difference in efficacy of median nerve stimulation vs. somatic maneuvers for changing tinnitus generalizes to external sounds, the already substantial percentage of people experiencing a somatic influence on the perception of external sounds found by Møller and Rollins, may underestimate the prevalence of somatic influences on the perception of external sound. In other words, even more individuals would have modulated their perception of an external sound with somatosensory stimulation at other locations such as the lateral upper neck and head (see Chapter 1). Indeed, if the strength and number of projections from somatosensory to auditory system are any indication, somatic influences on everyday sound perception may be considerable (Lewald and Ehrenstein, 1998; Lewald et al., 1999).

Somatic tinnitus syndrome

Our finding that people without tinnitus (even when specifically questioned in a very low-ambient noise environment) can develop tinnitus from forceful head and neck contractions suggests that some cases of clinical tinnitus may be due to abnormal activation of somatic–auditory interactions. Case 2 (begun above) is one such example.

Case 2. (Part 2) Approximately 6–8 weeks following the onset of chronic upper left dental discomfort from treatment of left upper molar decay, a 42-year-old man with normal pure tone thresholds developed left-sided tinnitus. Because of the discomfort he would avoid biting on the left. Ultimately his tooth repair was revised and his dental discomfort resolved after which his left-sided tinnitus gradually became fainter. When evaluated in our clinic about a year following his dental revision, his tinnitus was not heard. However on examination increased muscle tension involving his left sternocleidomastoid muscle was detected independently by two different examiners, neither of whom had any knowledge of his tinnitus. With somatic testing left sternocleidomastoid contraction elicited a faint sound in his left ear only, whereas right sternocleidomastoid contraction elicited no sound perception (see Chapter 19).

Consider another such example.

Case 3. An 80-year-old woman was hearing impaired (75 dB flat loss was present at all frequencies above 500 Hz) for more than 40 years but denied ever having prior tinnitus. She reported stumbling and striking her left forehead against a doorframe after which she developed an intermittent, mild left-sided headache. Three days after the trauma, she developed very distressing intermittent left ear tinnitus [“like a loud blender”], but she noted no other change in her hearing. Her pure tone hearing loss was unchanged as compared to an audiogram from 8 months earlier. Her hearing thresholds were similar for both ears except at 500 Hz, where the left ear threshold was 25 dB better than the right. Speech discrimination scores were ~85% bilaterally. She had been using hearing aids for more than 30 years, and had a strong family history of hearing loss. On examination her left sternocleidomastoid was non-tender but was under increased tension as compared to the right. The suboccipital insertion of her left splenius capitis was, however, focally tender. Somatic testing did not modulate her tinnitus. But with a cervical exercise program her tinnitus resolved within 3 months of its onset.

These two case reports illustrate the three characteristics of the “somatic tinnitus syndrome.” First, the tinnitus begins shortly (if not immediately) after a disturbance of the lateral head or upper neck. We have never encountered patients in whom disturbances of the upper extremities, torso, or lower extremities led to tinnitus, nor have others reported such findings. Thus, there appears to be a predilection for the periauricular region and particularly the upper lateral neck muscles in tinnitus (see Chapter 1). Second, the tinnitus is always perceived as ipsilateral to the somatic event and usually in the ear. Third, the onset of tinnitus is not associated with any other, new hearing complaints, new vestibular symptoms or abnormalities on neurological examination. The syndrome can occur in people with or without hearing loss. Pure tone hearing thresholds and speech discrimination scores of the two ears are usually similar and often within normal limits. Hyperacusis is not a common feature of such individuals. In general successful treatment of the somatic disorder associated with the tinnitus can also resolve the tinnitus itself (Cases 2 and 3).

Because somatic tinnitus is commonly a part of the TMD syndrome (59% Chole and Parker, 1992), it follows that the other components of TMD, namely, ipsilateral facial pain, ear pain or pressure, occipital or temporal headaches, and facial paresthesias can be associated with the somatic tinnitus syndrome as well (Chole and Parker, 1992).

The fact that both auditory factors and somatic factors can play a role in the genesis of tinnitus implies that in some people it may be an interaction between these two factors that leads to their tinnitus (see Chapter 10). In Case 3 (above), the pre-existing hearing loss, and any resulting modifications of the central auditory system, may have set the stage for the development of tinnitus following somatic injury. Similarly, a somatic disturbance of the head and upper neck can be a predisposing factor for the development of sound-induced tinnitus as illustrated by the following.

Case 4. A 43-year-old man had had a history of bruxism and chronic left dental and sinus pain for more than a year. As a result of bruxism he had cracked his left upper canine tooth and ultimately received a dental implant. His hearing thresholds were normal. Approximately 6 months after his implant he attended a loud 3-h concert. By 1 h of the concert he developed sharp ear pains worse on the left and diminished hearing bilaterally. Both ears were felt to be equally exposed to the high-level sounds. After the concert he experienced bilateral ear pressure that resolved over an hour. Two hours following the concert he experienced left ear ''ocean wave'' tinnitus that became louder with jaw opening and protrusion. His tinnitus resolved after 36 h. Later an audiogram showed no change in hearing thresholds and jaw opening and protrusion could not induce any tinnitus.

Note that despite symmetric hearing and symmetric high-level sound exposure with symptoms of bilateral temporary threshold shift his tinnitus developed only on the side with chronic facial pain.

Dorsal cochlear nucleus inhibition hypothesis

A neurological model can account for the major features of otic [ear-related] and somatic tinnitus (Fig. 1). Recall that somatic modulation of tinnitus can occur in deaf individuals. This implies that somatic modulation occurs because of interactions between the somatosensory and auditory systems within the central nervous system. Because the tinnitus of the somatic tinnitus syndrome is usually perceived in one ear, this suggests that the somatosensory–auditory interactions occur within the afferent central auditory pathway prior to the auditory decussation, i.e., before the level of the superior olivary complex, trapezoid body. The only auditory nuclei before the auditory decussation are the dorsal cochlear nucleus and ventral cochlear nucleus.

Findings of neural hyperactivity in dorsal cochlear nucleus following acoustic trauma and cisplatin otoxicity in animals have led to the hypothesis that the dorsal cochlear nucleus is the likely site of origin of the neural hyperactivity associated with tinnitus (Kaltenbach et al., 2005)

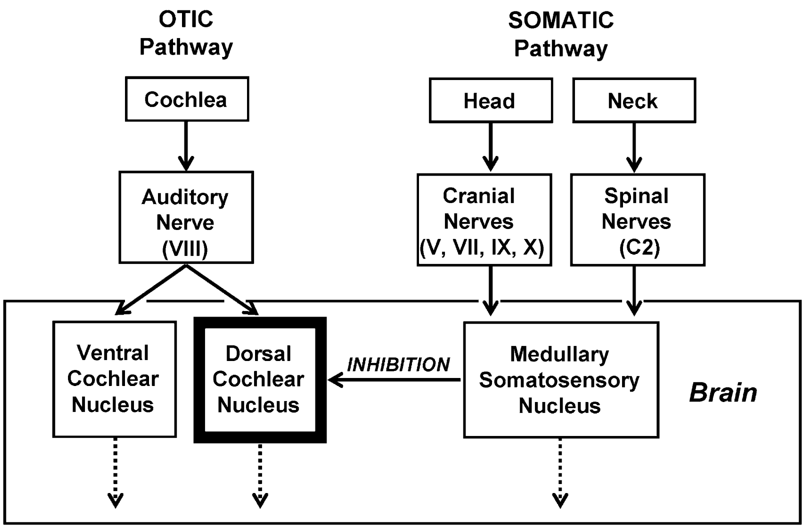

Fig. 1. Schematic depiction of the anatomic basis for the dorsal cochlear nucleus hypothesis: both somatic and otic (ear) tinnitus occurs owing to disinhibition of the dorsal cochlear nucleus. In both cases, tinnitus is due to increased activity in the output of the dorsal cochlear nucleus, which projects to the other centers and eventually leads to activation of the auditory perceptual machinery responsible for tinnitus. For somatic tinnitus sensory inputs from (1) the face via the trigeminal (V) nerve in the spinal trigeminal tract; (2) the external and middle ears via the common spinal tract of the facial (VII), glossopharyngeal (IX), and vagus (X) cranial nerves; and (3) the neck via the C2 dorsal spinal root converge to a common region of the lower part of the medulla, the medullary somatosensory nucleus, from which fibers project to the ipsilateral dorsal cochlear nucleus. Modulation of activity in the medullary somatosensory nucleus to dorsal cochlear nucleus pathway results in disinhibition of the dorsal cochlear nucleus. For otic tinnitus, loss of input (spontaneous activity) from the auditory (VIII) nerve leads to disinhibition of the dorsal cochlear nucleus.

(see Chapter 9). Somatic modulation likewise can be accounted for by invoking somatosensory influences on the dorsal cochlear nucleus. From the first observation of an anatomical connection between the somatosensory system and dorsal cochlear nucleus more and more evidence has been accumulating regarding this connection (Itoh et al., 1987; Weinberg and Rustioni, 1987). These results culminated with the Kanold and Young cat study (Fig. 2), where they showed that of all the nerves tested extending from the face to the hindlimbs, the C2 dorsal nerve root had the largest impact on the responses recorded from the cells in the ipsilateral dorsal cochlear nucleus (Kanold and Young, 2001). Furthermore it was somatosensory stimulation, muscle stretch or vibration, which was the most potent modulator of dorsal cochlear nucleus activity, as opposed to cutaneous stimulation such as light touch, brushing of hairs, or stretching of skin.

These findings obtained in the cat are perfectly concordant with our clinical findings regarding somatic modulation, namely, that (a) muscle activation is the most potent modulator of tinnitus, as opposed to cutaneous stimulation such as light touch, brushing of hairs, or stretching of skin and (b) the muscles of the upper cervical region [C2] are the most potent somatic modulators of tinnitus. Finally the projections from the somatosensory nuclei (nucleus cuneatus/medullary somatosensory nucleus) to the dorsal cochlear nucleus are only ipsilateral just as the somatic tinnitus syndrome is always ipsilateral to the cervical or dental insult. We have formalized these hypotheses by proposing that the dorsal cochlear nucleus disinhibition hypothesis can account for some forms of tinnitus on an auditory or somatosensory basis, as well interactions between the two (Fig. 1) (Levine and Kiang, 1995; Levine, 1999).

Why somatosensory projections to the dorsal cochlear nucleus?

A functional role for the somatosensory projections from the upper cervical region to the dorsal cochlear nucleus can be suggested from evidence that the dorsal cochlear nucleus is involved in up-down and front-back sound localization in animals (Sutherland et al., 1998). The convergence of the (i) acoustic and (ii) upper cervical proprioceptive information allows the dorsal cochlear nucleus to integrate (a) sound localization information extracted from the acoustic inputs reaching

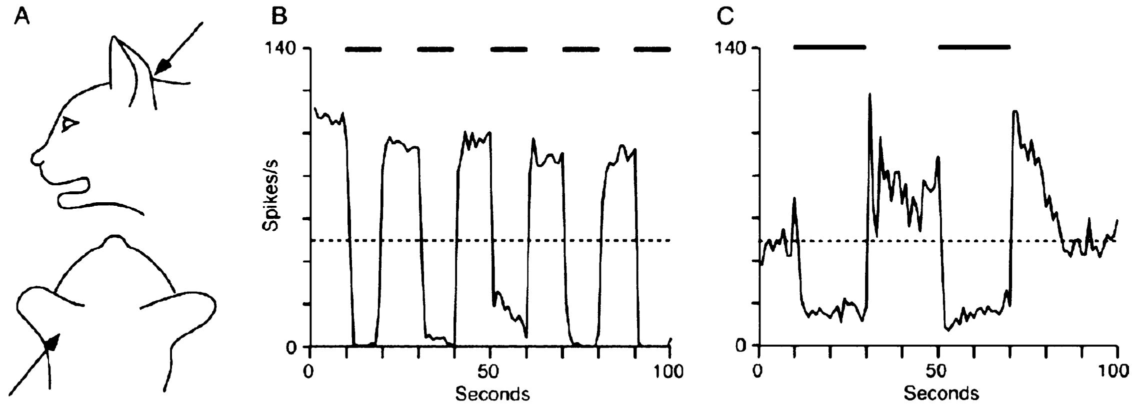

Fig. 2. Responses of dorsal cochlear nucleus (type IV) neurons to stretch of cat pinna muscles. Panels B and C show the responses of two different neurons to manual pressure (indicated by the bars above the plots) applied to the pinna as shown in A. The effect of the pinna pressure was continuous inhibition, maintained as long as the pressure was applied. The effect of pinna pressure is mediated through the following neural pathway: C2 spinal nerve to fasciculus cuneatus to medullary somatosensory nucleus to ipsilateral dorsal cochlear nucleus (Fig. 1). Muscle stretch or vibration was the most potent modulator of dorsal cochlear nucleus activity. Cutaneous stimulation (light touch, brushing of hairs, or stretching of skin) had no effect. Adapted with permission from Kanold and Young (2001).

the ear with, (b) head position information extracted from the somatosensory inputs. From integrating these two kinds of information the central nervous system can infer where in space a sound source is located.

Recently, it has been suggested that the trigeminal somatosensory inputs from the jaw muscles to the dorsal cochlear nucleus projections are present to suppress self-generated sounds such as respiration, chewing, or self-vocalizations (Shore and Zhou, 2006).

We conclude that somatosensory–auditory neural interactions within the dorsal cochlear nucleus can account for (a) somatic modulation of tinnitus, (b) inducing tinnitus in non-tinnitus individuals by strong muscle contractions of the head and neck testing, and (3) the somatic tinnitus syndrome.

Tinnitus treatments utilizing somatosensory–auditory interactions

Given that the somatosensory, as well as the auditory system, plays a role in the generation of tinnitus, the important question from a clinical standpoint is whether treatments targeting the somatosensory system can be utilized to treat tinnitus with predictable success. Such treatments include acupuncture (Hansen et al., 1982; Axelsson et al., 1994), electrical stimulation of the scalp and auricle (Engelberg and Bauer, 1985; Dobie et al., 1986; Lyttkens et al., 1986), cervical manipulation (Alcantara et al., 2002; Hulse and Holzl, 2004; Whedon, 2006), craniosacral therapy, TMD treatments (Wright and Bifano, 1997b), and trigger point treatments (Eriksson et al., 1995) including injections (Wyant, 1979; Estola-Partanen, 2000) (see Chapters 18 and 19).

Previous reports indicate that a range of such treatments can be effective, at least in isolated cases. It is likely that many of these successful treatments are mediated through central somatosensory–auditory interactions. If so, there may be a subgroup of patients whose tinnitus improves with appropriate activation of the somatosensory system.

We undertook a systematic evaluation of the literature to determine whether or not people likely to have tinnitus of somatic origin responded to treatment modalities involving the somatosensory system.

Since the examples reported in the literature rarely include all the information needed to assign a definitive somatic genesis of tinnitus, we had to rely on incomplete information. Recall that the somatic tinnitus syndrome includes (a) tinnitus that is always perceived as ipsilateral to the somatic event and usually in the ear and (b) the onset of tinnitus is not associated with any new hearing complaints. Hence tinnitus that is (a) strongly lateralized to one ear and (b) associated with symmetric hearing would be most suspects for having a strong somatic component to its etiology. Likewise people with tinnitus strongly lateralized to one ear despite symmetric hearing thresholds may be most responsive to treatment modalities mediated through the somatosensory system.

Characteristics of responders to somatosensory treatments

Cervical manipulation

Successful alleviation of tinnitus by cervical manipulation has been reported. However, the reports are strictly anecdotal and provide little detail and no information about the hearing in the patients discussed (Alcantara et al., 2002; Whedon, 2006). Thus, the characteristics of the patients responding to this treatment modality are largely unknown.

Acupuncture

Studies of acupuncture for tinnitus show a definite pattern regarding who derives benefit: people with tinnitus strongly lateralized to one side and symmetric hearing thresholds. The symmetry of hearing combined with the asymmetry of the tinnitus, makes a solely auditory origin to the tinnitus unlikely. Our suspicion is that many of the responders to acupuncture are people with somatically induced tinnitus.

The most striking published case in the acupuncture literature is that of a 60-year-old man whose tinnitus was predominantly referred to the left ear. His hearing thresholds differed by less than 10 dB between ears. He "experienced a marked improvement after the first [periauricular and extremities] acupuncture session and the reduction in tinnitus lasted several months (Axelsson et al., 1994)." In another similar case a 45-year-old woman with chronic tinnitus referred to the right ear and no hearing loss had complete resolution of her tinnitus with one session of right periauricular acupuncture (Jing Liu, LicAc, personal communication). Yet another example comes from our clinic. The following patient was seen subsequent to a dramatic response to periauricular acupuncture.

Case 5. A 77-year-old man had had 20 years of left ear tinnitus. He had symmetric, sensorineural hearing loss (SNHL) ranging from 20 dB at 250 Hz to 70 dB at 4 and 8 kHz with approximately 70% speech discrimination. For the first 15 years his tinnitus would recur intermittently at a low level with periods of no tinnitus for up to 2 weeks. Over the next 5 years his tinnitus was intermittently louder, especially after dozing in a chair. Six months prior to his visit to our clinic his tinnitus became constant, loud, and "throughout my head" leading to depression, including a suicide attempt. His hearing thresholds were unchanged.

Two months prior to his visit he started twice-weekly acupuncture (periauricular, suboccipital, and extremities). With his third treatment his tinnitus suddenly changed from bilateral to left unilateral and the tinnitus was no longer bothersome. Following that event he completed 3 weeks of acupuncture (as recommended). His tinnitus then remained in the left ear at a very low level except for a 5-day period when it was transiently louder. At his clinic visit, with somatic testing his left ear tinnitus modulated only slightly; its loudness went from 1 to 2 on a 0–10 scale with left jaw deviation or pressure on the sternocleidomastoid tendon at the left mastoid. Multiple maneuvers including right sternocleidomastoid contraction caused the development of transient right ear tinnitus that increased in loudness from 0 to 4 on a scale from 0 to 10. Over the next 12 months his tinnitus remained constant despite another 3-week course of acupuncture. However 1 year after his clinic visit his constant left tinnitus stopped abruptly and spontaneously. It then became intermittent, as it had been for his first 15 years.

Table 2. Data from Hansen et al. (1982)

Hearing	Subjects (M/F)	Improved	Not improved
Normal	2 (0/2)	2	0
Impaired bilateral	7 (4/3)	2	5
Impaired unilateral	8 (5/3)	0	8
Total	17 (9/8)	4	13

Note: Tinnitus: all unilateral; 4 intermittent.

Besides these isolated, but nevertheless striking cases, one acupuncture report described the systematic treatment of tinnitus patients all of whom had chronic unilateral tinnitus (Hansen et al., 1982). The patients were mixed in whether or not they had hearing loss. Table 2 organizes their data according to the individuals' pattern of hearing loss. Note that all periauricular acupuncture points were considered equivalent whether or not they were placed in the classical Chinese points or nearby "placebo" points. Tinnitus was intermittent in 24% of the participants, but the paper does not indicate which had intermittent tinnitus.

Two results in Table 2 are particularly noteworthy: (1) the two patients with normal hearing and unilateral tinnitus both improved. The hearing thresholds and tinnitus of these people suggests a predominantly somatic origin for their tinnitus. (2) None of the patients with unilateral hearing loss and unilateral tinnitus responded. The concordance between tinnitus and hearing loss makes an auditory, rather than somatic genesis of the tinnitus more likely in these people. Thus, the results for two categories of patients indicate that having two hallmark signs of somatic tinnitus (lateralized tinnitus, symmetric hearing thresholds) predicted a positive response to acupuncture.

The seven remaining individuals had bilateral hearing loss, but Hansen et al. do not describe

Table 3. Data from Lyttkens et al. (1986) (all with slight to moderate bilateral sensorineural hearing loss)

			Tinnitus					Match	
Subj #.	Age	Sex	Side	Yrs	Fluct?	RI	Mask	kHz	dB
1	75	M	L	20	N	N	90	0.4	45
2	68	F	L	12	Y	N	80	0.16	75
3	42	M	L	2	N	Sl	60	6.3	70
4	44	M	Both	1	N	N	70	7.0	60
5	52	M	Both	2	N	N	50	3.1	60

Fluct = fluctuating
RI = residual inhibition
N = no
Y = yes
Sl = slight
Mask = masking level.

Table 4. Data from Engelberg et al. (1985)

			Tinnitus			Match
Subj #	Age	Sex	Side	Yrs	Hearing	kHz
1	47	F	Both	15	SNHL	5.8/2.8
2	68	M	Both	38	SNHL	5.3/4.3
3	69	M	Both	8	SNHL	2.6/2.7
4	40	M	Both	20	SNHL	3.6/3.8
5	36	M	Both	3	SNHL	2.6/2.2
6	40	M	Both	20	SNHL	5.5/5.2
7	23	F	Right	1	Normal	7.0
8	34	M	Both	12	SNHL	3.1/4.0
9	32	F	Both	27	SNHL	5.0/?

SNHL = sensorineural hearing loss.

whether or not the loss was symmetric. Therefore, we do not know whether the results for this group follow those of the other two hearing groups just described (normal and unilateral loss). If they did, the two responders with bilateral hearing loss would have had symmetric hearing (and likely tinnitus triggered somatically) and the five non-responders would have had asymmetric hearing (and likely tinnitus of predominantly auditory origin). In other words, the responders would be those judged most likely to have somatically triggered tinnitus.

Electrical stimulation of the scalp and auricle

More complete case descriptions are reported in two reports on electrical stimulation. In one study electrical stimulation of the skin over the mastoid with a "Theraband" was used in five individuals with tinnitus and symmetric hearing loss (slight to moderate) (Lyttkens et al., 1986). Only participant no. 2 had any true tinnitus suppression (Table 3); for the first time in 12 years she did not hear her tinnitus. She was the only participant whose tinnitus fluctuated in addition to being unilateral. This fluctuating, unilateral tinnitus, combined with symmetric hearing strongly suggests somatically-induced tinnitus. A second report (Engelberg and Bauer, 1985) describes the effect of electrical stimulation of the earlobe in nine individuals with chronic tinnitus (Table 4). Only participant no. 7 experienced full suppression of her tinnitus. She was the only individual with unilateral tinnitus and symmetric (normal) hearing; all others had bilateral tinnitus.

A third electrical stimulation report provides less detail but used the same device, the "Theraband," and placement as Lyttkens et al. (Dobie et al., 1986). As in the Lyttkens et al. study, one of the 20 participants had a definite response. This person responded with an 85% reduction in his tinnitus loudness. His tinnitus was described as asymmetric ("left > right") and his hearing loss was "mildly asymmetric". There is no description of the direction of the asymmetry so it is unknown whether it was the same or different from the tinnitus. Both this and Lyttkens et al's "Theraband" study were effectively placebo-controlled, since nothing was heard or felt when the device was activated.

Trigger point treatments

Trigger point injections of the cervical and jaw muscles can transiently abolish tinnitus (see Chapter 18). It has been reported in a large statistical study of 178 "non-selected primary care ENT patients," (Estola-Partanen, 2000) as well as in anecdotes (Wyant, 1979). The statistical study found that within 5–10 days of injection the tinnitus disappeared in ~15% of these patients. By 6 months after the last in a series of trigger point injections, more than 30% of the patients felt improved, as compared to ~15% of a control group (Estola-Partanen, 2000). The study also found that the most cervical tension occurred on the side to which the tinnitus was referred. Women responded better than men and "chirping" tinnitus better than other descriptions.

The anecdotes include a woman with recent onset of tinnitus referred to her right ear in which she had no hearing since a labyrinthectomy 12 years earlier (Wyant, 1979). The tinnitus she referred to her right ear was associated with occipital headache radiating to the vertex and the eyes. With a steroid and lidocaine injection of her right splenius and scalenus medius trigger points her headache and tinnitus resolved promptly. The injections were repeated eight times; the relief lasted from several days to 4 weeks.

A second patient had mid-cervical pain radiating to the left face and eye, accompanied by tinnitus referred to the left ear. Injection of multiple left cervical trigger points with steroid and lidocaine provided relief of both tinnitus and pain for 4 months. No mention was made of his hearing status (Wyant, 1979).

Massage and stretching of trigger points have also eliminated tinnitus in some patients for up to 24 h (Eriksson et al., 1995).

TMD treatments

Several reports have described the effects of non-operative TMD treatment on tinnitus in TMD patients (Bernstein et al., 1969; Gelb and Tarte, 1975; Bush, 1987; Rubinstein and Carlsson, 1987; Kerstein, 1995; Wright and Bifano, 1997a). The reports are not explicit but most of the participants in these studies appear to have had normal hearing and tinnitus ipsilateral to their TMD (Curtis, 1980). Tinnitus resolved in over 50% of those who rated "their tinnitus as moderate or severe (Wright and Bifano, 1997a)," and in up to 65% of less severe cases (Wright and Bifano, 1997b). Pure tone hearing threshold data is not presented. But of those who responded to TMD treatments 70% (23 of 33) felt that their hearing ipsilateral to their tinnitus was normal, whereas all of those who did not respond to treatment felt their hearing ipsilateral to their tinnitus was not normal (Wright and Bifano, 1997a). Note the similarity to the Hansen et al. (1982) data (Table 2).

Furthermore, Wright and Bifano did some limited somatic modulation testing on the participants in their study and found a significant association between the ability to modulate tinnitus with "maximum voluntary clenching" and improvement or resolution of tinnitus with their TMD treatment program. Thus this study is consistent with the thesis that somatic testing can predict which subgroup of patients with TMD will benefit from TMD treatment for their tinnitus.

The finding of an association between response to somatic testing and the outcome of tinnitus treatment is unique among tinnitus treatment studies. No studies using auditory assessments of tinnitus such as residual inhibition, minimal masking level, tinnitus loudness, and level matching, otoacoustic emissions, evoked potentials, etc. have found any correlation between the test results and the outcome of treatment.

Conclusions

This review of the literature and our clinical experience of treatment of tinnitus patients with different modalities involving the somatosensory system show that the tinnitus of some patients improves with such treatments. Only one report made any attempt to restrict their study group by the characteristics of their tinnitus. While the Hansen et al. (1982) investigation was designed to detect a difference in tinnitus benefit depending on acupuncture location, in effect, by choosing the unilateral tinnitus subgroup, their results do identify a subgroup of individuals with tinnitus who respond to acupuncture, namely, those with unilateral tinnitus that is not caused by an ear disorder. This finding is supported by other studies employing treatments likely involving the somatosensory system (Wyant, 1979; Engelberg and Bauer, 1985; Dobie et al., 1986; Lyttkens et al., 1986; Axelsson et al., 1994; Wright and Bifano, 1997a; Estola-Partanen, 2000).

Furthermore, there is support for the notion that the subgroup of individuals with unilateral tinnitus that responds best to somatosensory treatments is those whose tinnitus is fluctuating. Two extreme kinds of fluctuating tinnitus are (a) intermittent tinnitus and (b) tinnitus that can be unilateral when quieter and non-lateralized when louder.

It is our conclusion that treatments for tinnitus that involve the somatosensory system should be restudied employing a design that tests the following hypothesis: individuals with unilateral fluctuating tinnitus and symmetric pure tone hearing significantly benefit from these treatments.

Abbreviations

SNHL	sensorineural hearing loss
TMD	temporomandibular disorder

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