

Somatic Tinnitus

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When the quality of tinnitus is nonspecific (such as buzzing, tonal, hissing, humming, ringing, roaring, rushing, whistling and whooshing, crickets), establishing a cause for the tinnitus is a challenging problem. An experienced clinician has 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’” (ie, idiopathic).¹

Among the reasons why establishing the cause for nonspecific tinnitus is so difficult is the fact that tinnitus is common in the general population presumably because it can be a normal physiologic phenomenon. At one extreme is the study by Heller and Bergman, who found that 94% of people they studied had tinnitus.² A more recent study found that although 19% of adults, when questioned, reported tinnitus, when the same individuals were then taken to a quiet room, another 28% described some tinnitus, so that a total of 47% described nonspecific tinnitus.³

Another reason contributing to the difficulty in establishing a cause for nonspecific tinnitus is that for any pathologic process associated with tinnitus, not all subjects with this diagnosis will, in fact, develop tinnitus. An extreme case is individuals who are deaf; 80% have tinnitus, but 20% have no tinnitus.⁴ Hence, the presence of tinnitus and the existence of a pathologic process by themselves do not imply that the two are related.

Another consideration in attempting to establish a diagnosis to account for a patient’s tinnitus is that the cause of the tinnitus may be multifactorial. A closely related idea is that tinnitus can be considered a threshold phenomenon,⁵ such that whereas any one factor, such as chronic progressive hearing loss, may not be sufficient to elicit a tinnitus complaint, two or more factors may synergistically lead to the tinnitus becoming symptomatic. For example, it is well established that the prevalence of tinnitus increases with

increasing hearing loss for any type of chronic progressive sensorineural hearing loss, such as presbycusis, chronic acoustic trauma, or a hereditary hearing loss. The recent onset of tinnitus in a patient with a known chronic progressive sensorineural hearing loss suggests that some other factor might be combining with the sensorineural hearing loss to lead to the tinnitus. Along a similar vein, several authors have put forth the concept of “triggering factors” that can lead to symptomatic chronic tinnitus.^{1,6}

What might be some of these triggering factors? First, any pathologic process associated with tinnitus can be a triggering factor. Some classes of medications probably can be triggering factors, although this has not been well established for any medication. From our clinical experience, α_1 -selective adrenoreceptor blocking agents, benzimidazole inhibitors of gastric acid secretion, and angiotensin-converting enzyme inhibitors appear to be triggering factors in some cases. Finally, any factor that has been associated with changes in tinnitus loudness would also qualify as a triggering factor. At least three factors often are associated with changes in the loudness of tinnitus, namely psychosocial stress, high-intensity sound exposure, and head and neck “somatic” factors. People with clinical tinnitus commonly describe one or more of these three factors as altering their perception of their tinnitus.

If a patient reports that his or her tinnitus is intermittent or has wide fluctuations in loudness or other qualities, and there is neither exposure to intense sound nor evidence of stress, then somatic modulation must be suspected. Consider the following case: *Case 1.* A 77-year-old woman, who had two major thyroid operations at age 23 and 26 years, reported 5 months of non-lateralized intermittent tinnitus. The tinnitus was always present upon awakening but at times would disappear during the day. Pure-tone thresholds were normal for both ears except at 8 kHz where both

ears had thresholds of 60 dB HL and at 4 kHz for the left ear where the threshold was 30 dB HL. Brief firm pressure at the insertion of her right sternocleidomastoid (SCM) muscle onto the mastoid process abolished her right ear tinnitus for more than 8 minutes and reduced her left ear tinnitus loudness from 5/10 to 3/10 on a 0 to 10 loudness scale for less than a minute.

A history of variations in tinnitus loudness raises the suspicion of a somatic factor modulating the percept's loudness (Table 9-1). At an extreme are patients who describe that they have periods when their tinnitus cannot be heard, even in the quiet. Others report wide variations in the loudness of their tinnitus. For still others, their tinnitus is unilateral when it is relatively quiet but becomes nonlateralized when the tinnitus is louder (see case 6 below). Such phenomena suggest that there are ongoing somatically mediated factors modulating the tinnitus percept.

Diurnal fluctuations in the tinnitus percept also suggest that somatic modulation is operative. Patients who describe their tinnitus as louder on awakening raise the possibility that somatic factors such as bruxism (grinding of the teeth) are active during sleep and are causing an increase in tinnitus loudness. Others report that their tinnitus has usually vanished by the time they awaken and then returns a few hours into the day; this scenario suggests that, during the day, they are reactivating their tinnitus through somatic mechanisms, such as the tonic muscle contractions required to support the head in an upright position or clenching the jaws related to the stress of daily activities. Finally, others report that their tinnitus is louder after awakening from a nap in a chair; this may relate to somatic factors such as stretching of the neck muscles when their head passively falls forward while dozing in a sitting position.

In this chapter, I explore further this "somatic" factor. I review the effects of muscle contractions and other closely related somatic factors on auditory perception in general and tinnitus in particular. The evidence is reviewed for (1) how somatic factors are commonly responsible for fluctuations in tinnitus

perception, (2) how somatic factors can act as a trigger factor and thereby lead to the somatic tinnitus syndrome, and (3) how somatic factors can combine with other conditions to cause tinnitus on a multifactorial basis. Finally, I provide a hypothesis that can serve as a conceptual framework to account for the interactions between hearing and a variety of nonotic factors, including the somatic factor.

SOMATIC FACTORS ARE COMMONLY RESPONSIBLE FOR FLUCTUATIONS IN TINNITUS PERCEPTION

It has long been known, almost as a curiosity, that some people can modulate their tinnitus somatically. Møller and colleagues showed that median nerve stimulation could modulate tinnitus in close to 40% of subjects.⁷ Rubinstein found that approximately one-third of her subjects could influence their tinnitus with jaw movements or pressure on the temporomandibular joint (TMJ).⁸ When interviewed, approximately 20% of patients in our tinnitus clinic reported that they can somatically modulate the acoustic properties of their tinnitus by head and neck movements or muscle contractions such as clenching the teeth together.⁹ In a systematic study of 70 consecutive patients in my tinnitus clinic, my colleagues and I found that 68% could somatically modulate their tinnitus with head or neck maneuvers.¹⁰ With the same maneuvers, Sanchez and colleagues reported almost identical results (65%).¹¹ When we added jaw maneuvers to our test battery (Table 9-2), the percentage of those who could somatically modulate their tinnitus rose to 80%.³ Thus, somatic modulation of tinnitus is very common in clinical tinnitus subjects. A typical pattern of somatic modulation is illustrated by the following case.

Case 2. A 50-year-old man had nonlateralized high-pitched tinnitus that had begun with an upper respiratory infection 9 months earlier. Prior to his visit, he had noticed that jaw protrusion increased the loudness of his tinnitus, and he suspected that his tinnitus was louder on awakening when he had consumed modest quantities of wine the previous night. His audiogram was symmetric, and all pure-tone thresholds were 25 dB HL or better. His pattern of modulation is the most common type (Table 9-3): his tinnitus only became louder, and the change in loudness did not persist following the release of a forceful contraction. Also typical is the pattern of tinnitus mod-

TABLE 9-1. Tinnitus Properties Suggesting a Somatic Component

Intermittency
Large fluctuations in loudness
Variability of location
Diurnal pattern
No hearing loss but head, neck, or dental insult

TABLE 9-2. Maneuvers Currently Being Used to Test for Somatic Modulation (All Use Maximal Force)*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 are made to resist pressure applied by the examiner to

11. The forehead
12. The occiput
13. The vertex
14. The left temple
15. The right temple
16. The left zygoma, with the head turned fully to the left
17. The right zygoma, with the head turned fully to the right
18. The left temple, with the head turned to the right and tilted to the left (left sternocleidomastoid muscle)
19. The right temple, with the head turned to the left and tilted to the right (right sternocleidomastoid muscle)

Extremity Contractions

20. Pulling apart the locked fingers of the two hands
- Resisting pressure to
21. Left hip flexion
 22. Right hip flexion
 23. Left shoulder abduction
 24. Right shoulder abduction

ulation that occurred with the maneuvers that can be attributed to the contraction of a single muscle; namely, lateral deviation of the jaw (subserved by the contralateral medial pterygoid muscle) is usually associated with changes in the contralateral ear only, whereas SCM muscle contractions usually affect only the ipsilateral ear.

To assess whether somatic modulation is a general phenomenon and not restricted to clinical tinnitus subjects, my colleagues and I tested somatic modulation in 62 nonclinical subjects (friends and relatives).³ Our only exclusion criterion was that they had never sought any medical care for tinnitus. Twelve (19%) knew that they had tinnitus, but it was not a problem, and 17 (27%) were unaware that they had tinnitus until we brought them into a low-ambient noise room and asked them what they heard. In this group of 29 nonclinical subjects with tinnitus, 23

(again about 80%) could modulate their tinnitus. Hence, somatic modulation of tinnitus is not restricted to the clinical population; its incidence (80% with our test battery) is the same for all populations with tinnitus and probably all causes of tinnitus. This implies that the ability to modulate tinnitus somatically is not what makes tinnitus a clinical problem.

Finally, when we tested the other 33 nonclinical subjects who in the quiet had no tinnitus whatsoever, even when pointedly questioned, somatic testing elicited an acoustic perception (“tinnitus”) in 19, or almost 60%. Thus, somatic modulation of auditory perception is a fundamental property of the auditory system that happens to be most obvious in tinnitus subjects because they have spontaneous ongoing auditory perception. In fact, it has been shown that the perception of external sounds can also be somatically modulated. Møller and Rollins reported that the perception of ex-

TABLE 9-3. Somatic Testing for Case 2

<i>Maneuver*</i>	<i>Right Ear Tinnitus Loudness</i>	<i>Left Ear Tinnitus Loudness</i>
Baseline (0–10 scale)	7	7
2. Open mouth	8.5	8.5
3. Open mouth, against resistance	9	9
4. Protrude jaw	9	9
5. Protrude jaw, against resistance	9	9
9. Slide jaw to right, against resistance	7	9
11. Forehead	7.5	7.5
18. Sternocleidomastoid muscle, left	7	7.5
19. Sternocleidomastoid muscle, right	8	7

*Only maneuvers that modulated the patient's tinnitus are shown.

ternally generated sounds can be modulated by a non-physiologic type of somatosensory stimulation, namely median nerve stimulation at the wrist.¹² Of those who could modulate, 83% perceived the external sound as louder and 17% as softer. Although they found that 60% of subjects could modulate their perception of the external sound, they did not use a physiologic stimulus and did not stimulate the most sensitive region for somatic modulation of tinnitus, the head and upper neck (see below). It is likely that even more subjects would modulate their perception of an external sound with other types of somatosensory stimuli.

Now that it has been established that subjects without tinnitus can somatically modulate their auditory perception, the lower incidence of change in auditory perception with somatic testing in subjects with no tinnitus than in subjects with tinnitus is readily explained. From our observations of clinical and nonclinical tinnitus subjects, it is clear that somatic testing can increase and/or decrease tinnitus loudness and pitch (see below). If such changes in auditory perception are happening equally for all groups of subjects, 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 nontinnitus subjects, similarly for any change of pitch, and (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, 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 nontinnitus groups than in the tinnitus groups.

A similar result was obtained for the effect of loud sounds on tinnitus. We asked all 62 nonclinical subjects whether they had ever experienced tinnitus after a loud sound; 32% of those without tinnitus in the quiet reported affirmatively compared with 63% of those experiencing tinnitus at the time.⁵ This highly significant difference ($p < .01$) can be explained in a similar way using the threshold idea. If loud sounds cause 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, subjects without tinnitus might raise the overall activity in their auditory pathways but not enough to cross the threshold of auditory perception.

Alterations in tinnitus perception that occurred with somatic testing of the clinical and nonclinical tinnitus subjects were similar and included changes in loudness, pitch, or location. By far, 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, as shown in Table 9-4 for nonclinical subjects. 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 except 1 subject, 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.

TABLE 9-4. Loudness Changes with Somatic Testing Reported by Subjects with Ongoing Tinnitus Using the 0 to 10 Loudness Scale

<i>Direction of Change</i>	<i>Number of Subjects</i>	<i>Range</i>		<i>Mean</i>
		<i>Minimum</i>	<i>Maximum</i>	
Increase in loudness	20	0.5	5	2.2
Decrease in loudness	7	0.1	3	1.0

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 minutes. The two longest ones (5 and 10 minutes) occurred in subjects who had had no tinnitus before the somatic modulation testing. 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 were 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 so, 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. Although Cullington reported a single subject who could somatically modulate his left ear tinnitus with active movement of his left long finger, the report does not describe the effect of head and neck movements, so it is unclear what head and neck movements would have done for this subject.¹³ In any case, we have never encountered such a subject.

We have probed further into our understanding of somatic modulation of tinnitus by testing individuals who are deaf (cochlear implant subjects with their implant disconnected). At present, 13 deaf subjects have been tested in the standard manner ("somatic testing"). At the time of testing, 10 subjects had ongoing tinnitus and 3 did not. Five of the 10 (50%) with ongoing tinnitus could modulate their tinnitus with somatic testing, whereas 2 of the 3 (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 minutes. Pitch changes also occurred but only in one subject. For this subject, loudness increased for some maneuvers, whereas pitch increased for others. In all of these subjects, head and neck maneuvers were more effective in altering auditory perception than extremity maneuvers.

Consider the following two examples. In case 3 somatic testing elicited tinnitus in a deaf subject who had no ongoing tinnitus. In case 4 somatic testing modulated tinnitus in a deaf subject who had ongoing tinnitus. *Case 3.* This 52-year-old woman had received a left cochlear implant 12 years earlier. She had no tinnitus with her processor connected or disconnected. Three months prior to testing, she experienced 45 minutes of left ear tinnitus three times over a week but not subsequently. The effects of somatic testing are shown in Table 9-5.

Case 4. This 52-year-old man had received a right cochlear implant 16 years earlier. He had no tinnitus with his processor connected but heard "wind" in his right ear with the processor disconnected. His tinnitus persisted louder for up to 2 minutes with forehead pressure and 1 minute with right temple pressure (Table 9-6).

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 because these subjects are 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 individuals who are deaf is likewise operating for most, if not all, of the other groups. Thus, I

TABLE 9-5. Somatic Testing for Case 3

<i>Maneuver*</i>	<i>Right Ear Tinnitus Loudness</i>	<i>Left Ear Tinnitus Loudness</i>
Baseline (0–10 scale)	0	0
2. Open jaw	0	1
5. Protrude jaw against resistance	0	0.5
10. Retract jaw	0	0.5

*Only maneuvers that elicited the patient's tinnitus are shown.

conclude that somatosensory-auditory neural interactions within the central nervous system account for most, if not all, somatic modulations of tinnitus and the development of auditory percepts with somatic testing.

Some of our observations provide insights into the neural system responsible for somatic modulation of auditory perception (somatic modulation of tinnitus). The cutaneous sensory system is unlikely. 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.¹⁴ On the other hand, the motor system is likely because the actions that elicit modulations of auditory perception are almost exclusively non-noxious but forceful muscle contractions. Such contractions involve (1) the entire voluntary efferent motor system (motor cortex, corticospinal or corticobulbar tracts, and primary motoneurons) and (2) the motor afferent system, which begins with the deep muscle receptors such as the muscle spindles and tendon organs.

One fortuitous occurrence has shown that muscle fatigue can abolish somatic modulation of auditory perception.

Case 5. A 57-year-old man with lifelong nonlateralized tinnitus could increase the loudness of his tinnitus with jaw protrusion from an estimated loudness of 4 of 10 to 6 of 10. After being a subject of a functional magnetic resonance imaging (fMRI) experiment in which he repeatedly modulated his tinnitus with jaw protrusion for approximately an hour, for the next 3 to 4 days, he could not modulate his tinnitus, following which his ability to modulate with jaw protrusion gradually returned over about a day.

Muscle fatigue is principally due to fatigue (weakening) of the muscle fibers per se and not the motor efferent system. Unlike the efferent system, the motor afferent system requires contraction of the muscle fibers to be activated. Therefore, muscle fatigue inactivates the motor afferent system but not the motor efferent system. Hence, the motor afferent system (from muscle spindles and Golgi tendon organs) is responsible for at least some, if not all, somatic modulation of auditory perception.

The Golgi tendon organ senses muscle tension, and the muscle spindle senses muscle length. Because somatic testing is done principally with isometric muscle contractions (ie, little change in muscle length), our results favor the Golgi tendon organ as the source for somatic modulation of auditory perception.

TABLE 9-6. Somatic Testing for Case 4

<i>Maneuver*</i>	<i>Right Ear Tinnitus Loudness</i>	<i>Left Ear Tinnitus Loudness</i>
Baseline (0–10 scale)	4	0
3. Open jaw against resistance	5	0
5. Protrude jaw against resistance	5	0
11. Forehead pressure	5	0
15. Right temple pressure	6	0
16. Left turn	5	0
18. Left sternocleidomastoid muscle	5	0

*Only maneuvers that modulated the patient's tinnitus are shown.

On the other hand, another observation favors the muscle spindle.

Case 6. A 69-year-old woman with tinnitus for about 2½ years described her hissing tinnitus as 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 one of her visits, 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 SCM muscle was taut and tender; her right SCM muscle was normal. Vibration with a handheld massager applied to her right SCM muscle did not alter her tinnitus. However, when applied to her left SCM muscle, she noticed that the tinnitus gradually became quieter (4 of 10) over about 5 minutes and shifted its location from throughout the head to the right ear only. It remained quieter and in the right ear for about 30 minutes.

Because vibration is known to be a potent activator of muscle spindles, this result provides support for the hypothesis that muscle spindle activation is a mediator of somatic modulation.

Hence, our observations support the hypothesis that both the muscle spindles and the Golgi tendon organs of the motor afferent system may be responsible for somatic modulation of auditory perception. This conclusion is consistent with Kanold and Young's findings, which implicate activation of the cat's pinna muscle spindles and/or Golgi tendon organs as the likely source of neural activity ultimately affecting the dorsal cochlear nucleus (DCN).¹⁵

Possible sites of neural somatosensory-auditory interactions include the inferior colliculus because it is known to exhibit tinnitus-related abnormalities and it receives somatosensory inputs.¹⁶ Experimentally, the firing of all units in the cat central nucleus of the inferior colliculus can be somatically modulated.¹⁷ The DCN appears to be critical when tinnitus is due to ear disorders and is an established site of somatosensory-auditory interaction.^{15,18}

If the change in auditory perception is unilateral, the DCN becomes a highly likely site for somatic-auditory interaction. Nonlateralized tinnitus suggests either the bilateral cochlear nucleus or some higher center, such as the inferior colliculus.

SOMATIC FACTORS CAN TRIGGER THE 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 it is likely that some cases of clinical tinnitus may be due to activation of latent somatic-auditory interactions. One such example follows.

Case 7. A 29-year-old woman with normal audiometry had highly distressing right ear tinnitus for 7 months, which had resolved approximately 2 months prior to her visit to our tinnitus clinic. On physical examination, she had increased muscle tension and tenderness in her right SCM muscle compared with the left. At the time of somatic testing, she was hearing slight constant ringing of both ears (1 of 10), which was much fainter than her prior right ear tinnitus. With somatic testing, each time that her right SCM muscle was forcefully contracted, she reported hearing right ear tinnitus identical to her prior distressing tinnitus (Table 9-7). The right ear tinnitus did not persist after her somatic testing.

In some of our clinical cases, a well-described event occurred that precipitated the tinnitus. Many of these people had normal audiograms as well. We refer to such cases as examples of the somatic tinnitus syndrome.

Case 8. A 52-year-old woman underwent a right interscalene block to have manipulation of her frozen shoulder performed. With the injection, anesthesia of the shoulder did not occur; rather, she developed anesthesia of her right ear, right postauricular region,

TABLE 9-7. Somatic Testing for Case 7

<i>Maneuver*</i>	<i>Right Ear Tinnitus Loudness</i>	<i>Left Ear Tinnitus Loudness</i>
Baseline (0–10 scale)	1	1
Forehead pressure	4	1
Mandibular pressure	4	1
Right sternocleidomastoid muscle	4	1

*Only maneuvers that modulated the patient's tinnitus are shown.

and slightly right side of the face, with a dull ache in the same distribution. There was no facial weakness or dizziness. The numbness resolved within 14 hours. But immediately on injection of the local anesthetic (15 mL of 1.5% mepivacaine), she developed right ear tinnitus that has persisted unchanged for more than 10 years. She described her tinnitus as a high-pitched ringing in the right ear that sounds “like the brakes of a bus.” An otolaryngologic evaluation 2 weeks later noted right occipital spasm. The audiograms for both ears were normal at the six standard audiometric frequencies. Her tinnitus was matched to a 3 kHz tone. Tympanograms and the stapedial reflexes were normal. Two subsequent audiograms in the next month remained similar but unlike the first audiogram; in these later audiograms, 6 kHz was also tested, and her thresholds for 6 kHz were 25 dB HL for both ears. All other frequencies tested were approximately 10 dB HL. On two of these occasions, her tinnitus was matched to a 6 kHz tone at 10 and 5 dB SL. Spontaneous otoacoustic emissions were not detected. A bolus of intravenous lidocaine abolished the tinnitus for 10 minutes. Oral mexiletine provided marginal benefit. Contrast magnetic resonance imaging (MRI) a year following the onset showed in the posterior part of the right cerebellar hemisphere two small regions of chronic infarction estimated to be more than 6 months old. A magnetic resonance angiogram of the neck arteries was normal. Her neurologic examinations have always been normal.

Case 9. A 45-year-old, right-handed man developed left dental pain and left-sided high-pitched tinnitus at about the same time. Treatment of an abscessed left upper molar with analgesics and antibiotics followed by a root canal procedure resolved his dental pain in a few days, whereas his tinnitus remained unchanged. An audiogram 3 months following the onset of the tinnitus was normal. Over the next several months, his tinnitus slowly became quieter but never totally resolved. Its pitch was matched to a 10 kHz tone. After 8 years, the tinnitus is still heard only in the left ear but is generally barely perceptible except for episodes of abrupt growth in loudness followed by a gradual return to its baseline loudness over the ensuing few days to weeks. Sleeping with the left ear down may precipitate such an episode. He described a vague strange feeling in the left periauricular region since the onset of his tinnitus.

Case 10. A 34-year-old, right-handed man presented to an otolaryngologist complaining of 5 days of high-pitched, left ear tinnitus and a history of 3 to 4 months

of left-sided facial pain, which, more recently, had been associated with left facial swelling and mild pain. An abscessed left upper molar had been surgically treated 1 day previously. His examination and audiometry were unremarkable. The facial pain and swelling resolved, and the tinnitus improved for approximately a week following the dental surgery but then worsened again. When evaluated 2 months later, his tinnitus was present about 80% of the time. His left posterior cervical muscles were under increased tension compared with the right but were nontender. Clenching of the teeth on the left or pressure on the left mastoid abolished his tinnitus. After another 3 months of nearly continuous tinnitus, the tinnitus stopped. At no time did he have any vestibular complaints.

Case 11. A 6-year-old girl fell off her bicycle, fracturing the left mandibular ramus and dislocating the left TMJ. Within 4 months, the fracture and dislocation healed without surgery, but she had some persistent discomfort in the left preauricular and infra-auricular regions. She never complained of tinnitus or hearing loss after the accident, but 2 years later, she failed a routine school hearing test and did poorly on some subsequent audiograms because of the left ear. Otoacoustic emissions were normal. Temporal bone computed tomographic scans and contrast MRIs were normal. Once it was realized that she had left ear tinnitus, she was taught the difference between her tinnitus and the audiometer tones. Subsequent audiograms have been normal. Her left ear tinnitus was described as buzzing like a dial tone. She had noticed that her tinnitus became quieter with tilting her head to the left and louder with tilting to the right. When examined 3 years after the accident, she had full range of motion of her neck, but her left SCM muscle was rope-like in consistency and tender. Her baseline tinnitus was 8 of 10 in the left ear; by tilting to the right, it became 9 of 10, and by tilting to the left, it became 5 of 10. With somatic testing, contracting the left SCM muscle, such as with forehead pressure, left temple pressure, or left SCM muscle testing, her tinnitus became much louder, as high as “13 of 10.” Other maneuvers caused pain but did not change the loudness of her tinnitus.

Case 12. A 39-year-old, right-handed woman described hearing a high-pitched ringing principally in the right ear since at least her teens. Her tinnitus has been unchanged over the years, with the exception of becoming louder during the last months of her two pregnancies, and returned to baseline within 3 months of parturition, despite nursing both infants

for a year. On one of these occasions, she was treated with physical therapy for “stiffness” of her neck, but her tinnitus was unchanged. Head position has always modulated her tinnitus loudness. On a 0 to 10 loudness scale, she rates her tinnitus as 3 of 10. With turning the head to either side or tilting to the left, loudness increases to 5 of 10, whereas with tilting to the right, the loudness was barely perceptible (1 of 10). Clenching her teeth increased the loudness only slightly (4 of 10). On examination, two regions of increased muscle tension and tenderness were noted in the right neck compared with the corresponding regions on the left, namely the upper part of the SCM muscle and the medial part of the suprascapular region. Otherwise, her otoneurologic examination was unremarkable. An audiogram was normal. Her tinnitus matched to an 11 kHz tone at 10 dB SL.

Case 13. As a 50-year-old man was swallowing some sleeping pills, he sneezed and developed acute right ear pain. The next day he noticed right ear tinnitus. An otolaryngologic evaluation 2 days later detected an abrasion of his right side of his nasopharynx, which resolved uneventfully. His audiogram was normal. He matched his tinnitus to a 10 kHz tone. His right ear tinnitus has persisted for more than 2 years. With somatic testing, his tinnitus went from a baseline of 3 of 10 up to 4 of 10 with forceful contraction of his right SCM muscle.

CLINICAL FEATURES OF SOMATIC TINNITUS SYNDROME

These seven cases illustrate the characteristic features of the somatic tinnitus syndrome. First, the tinnitus is closely associated temporally with factors relating to the head or upper neck. We have never encountered patients with tinnitus similarly associated with the upper extremities, torso, or lower extremities, nor have others reported such findings. There appears to be a predilection for the periauricular region and particularly the upper part of the SCM muscle in many cases. Second, the tinnitus is always described as coming from the ear ipsilateral to the somatic event. The tinnitus is usually described as a high-pitched constant ringing. Third, there are no other associated hearing or vestibular complaints and no abnormalities on the neurologic examination. The syndrome can occur in people with no hearing loss. Pure-tone and speech audiometry of the two ears is symmetric and often within normal limits.

Hyperacusis is not a feature of any of these cases. Note that successful treatment of the associated disorder may resolve the tinnitus in some cases but not in others.

REVIEW OF PRIOR REPORTS

There has been little description in the literature of the clinical features of nonotic somatic tinnitus. The association between whiplash and tinnitus has been well described, particularly in the German literature, and has been attributed to “functional disturbances of the upper cervical spine.”^{19,20} Beside being frequently associated with other elements of the whiplash syndrome (dizziness, pain, and nausea) and unrelated to hearing loss, rarely is any detail about the tinnitus provided. Wyant described intermittent unilateral tinnitus in a presumably normal-hearing man that was associated with neck pain radiating to the ipsilateral side of the face and eye.²¹

Many articles describe an association between tinnitus and pain in the region of the ear or TMJ. Some authors emphasize the joint’s role and refer to the syndrome as TMJ syndrome, Costen’s syndrome, or craniomandibular disorder. Others stress muscle tension as the key to the syndrome and describe it as myofascial pain-dysfunction syndrome. A recent report of tinnitus and TMJ syndrome associated the tinnitus with muscle dysfunction and not joint dysfunction.²² The fact that somatic tinnitus would appear to have been previously described as part of TMJ syndrome suggests that somatic tinnitus not only is limited to the craniocervical regions but may more likely be from the lateral craniocervical regions, the periauricular regions.

Although virtually all reports include tinnitus as part of TMJ syndrome, detailed characteristics of the location of the tinnitus are few. Three reports describe some features of tinnitus consistent with our cases of somatic tinnitus. Curtis reported the tinnitus as lateralized to the side with the pain in 14 of the 17 patients in whom the pain was unilateral, whereas the three other patients reported bilateral tinnitus.²³ Of the 28 patients with bilateral but asymmetric pain, the tinnitus was lateralized to the side of greater pain in 13 and was bilateral in the other 15 patients. Ten other patients had symmetric otalgia, and all had bilateral tinnitus. Travell and Simons described a patient who had tinnitus ipsilateral to a trigger point in the upper posterior part of the masseter muscle.²⁴

A controlled study of tinnitus and TMJ syndrome defined the syndrome as “both clicking in the joint and pain in the region of the ear (joint).”²⁵ Based on questionnaire data, the authors found that tinnitus was significantly more prevalent in the patients with TMJ syndrome than in two control groups.

MULTIFACTORIAL TINNITUS: SOMATIC FACTORS AND HEARING LOSS INTERACT

Three cases illustrate that factors predisposing individuals toward otic tinnitus can interact with factors predisposing individuals toward somatic tinnitus.

Case 14. A 50-year-old woman carried the diagnosis of unilateral otosclerosis manifested by a left, predominantly sensorineural, hearing loss. Her hearing loss predated her intermittent left ear tinnitus by more than 5 years. She reported that her tinnitus had begun following neck manipulation a few months prior to being seen in our clinic. When initially examined, she was not having tinnitus. Her left suboccipital muscles, however, were noted to be tender and under increased muscle tension compared with the corresponding muscles on the right side. Within an estimated 5 minutes of examining the cervical musculature, she reported that her left-sided tinnitus had started. On reexamination, her left suboccipital muscle tension had become much more pronounced. Within another 5 minutes, her tinnitus abated, and her suboccipital muscles were again more relaxed.

Case 15. A 50-year-old man reported that he had noticed very faint tinnitus in his left ear for many years. On a 0 to 10 loudness scale, he rated it as 1 of 10. An audiogram at age 41 revealed normal thresholds bilaterally except 25 dB HL at 4 kHz for the left ear. At age 45, 5 to 6 days after placement of a permanent crown on a left lower molar, his left ear tinnitus became much louder (4 to 5 of 10). At about the same time, he had also attended a loud concert. His tinnitus then remained unchanged until age 48, when it vanished (0 of 10) following placement of a temporary inlay on the tooth that occludes with the left lower molar crown. Two weeks later, while leaving the dentist’s office after the placement of the permanent gold inlay, his tinnitus suddenly became very loud (10 of 10) and remained that way for the next 6 months. A repeat audiogram revealed normal thresholds except again at 4 kHz in the left ear, in which the threshold was now 40 dB HL. Over the last 2 years,

his tinnitus loudness has gradually decreased to the 6 to 7 of 10 range.

Case 16. A 62-year-old man had been doing “facial exercises” for about 20 years to improve his scowl. One evening, while vigorously contracting his facial muscles, he developed right face and lateral neck discomfort associated with right ear tinnitus. He had had an upper respiratory infection at the time. He was evaluated at our clinic 8 months later. The tinnitus and facial discomfort persisted. Facial massage could temporarily resolve the facial pain and lower the intensity of the tinnitus. On examination, his right SCM muscle was tender. An audiogram revealed normal thresholds bilaterally except for his right ear at 4 kHz (35 dB HL) and 8 kHz (25 dB HL). He matched his tinnitus to a 15 dB SL narrowband noise centered at 8 kHz. His auditory brainstem responses were normal. His right ear tinnitus has persisted for more than 10 years.

These observations are dramatic examples of how tinnitus of presumably otic origin can interact with craniocervical somatic factors. Although these cases, like the cases of purely somatic tinnitus, are all restricted to the head or upper cervical region, somatic modulation of tinnitus is likewise predominantly a head and neck phenomenon. With the possible exception of the Cullington case report, there have been no reports of somatic modulation outside the head and upper neck in physiologically intact individuals using physiologic stimuli.¹³ Somatic modulation of tinnitus can easily be accounted for by central nervous system interactions between the auditory and somatic systems, such as is proposed by our neurologic model (presented below). Otic-somatic interactions may account for (1) why some patients with a hearing disorder develop tinnitus and others with an otherwise identical hearing disorder do not, (2) why some patients with chronic progressive hearing loss develop tinnitus at some point in time, and (3) why patients with symmetric hearing loss can develop tinnitus in only one ear.

NEUROLOGIC MODEL OF SOMATIC TINNITUS AND AUDITORY INTERACTIONS

Because somatic tinnitus syndrome is closely related to somatic modulation of auditory perception, and I have shown that somatic modulation occurs because of central nervous system interaction, I propose a neurologic model of somatic tinnitus that will account for all

features of somatic modulation of auditory perception (Figure 9-1). This model follows directly from the clinical characteristics of somatic tinnitus.

In the afferent auditory pathway, although binaural interaction can occur at the cochlear nucleus, it is probably at the level of the superior olivary complex, where the binaural interaction necessary for sound lateralization first occurs.^{26,27} At these higher levels of the auditory pathway, all degrees of lateralization are probably represented. Accordingly, activation of these regions would not likely result in lateralization of the percept to one ear exclusively. On the other hand, at lower levels such as the cochlear nucleus, auditory nerve, or inner ear, lateralization of the percept exclusively to one ear would be expected. In fact, clinically, it is known to be the case that tinnitus is lateralized exclusively to the ipsilateral ear

for disorders of the auditory nerve and cochlea. The unilateral characteristic of somatic tinnitus suggests that nonauditory interaction with the auditory system for somatic tinnitus occurs at the level of the cochlear nucleus because it is the only part of the afferent central auditory pathway before the trapezoid body, where the first auditory decussation important for sound lateralization is located.²⁸ The fact that our defining cases of somatic tinnitus always report their tinnitus as coming from one ear suggests that the cochlear nucleus is the site on the auditory pathway where the nonauditory inputs interact with the auditory system to initiate the neural discharge patterns that are ultimately interpreted as tinnitus.

That the defining cases of somatic tinnitus are associated only with processes that involve the ipsilateral head and upper neck also must be accounted

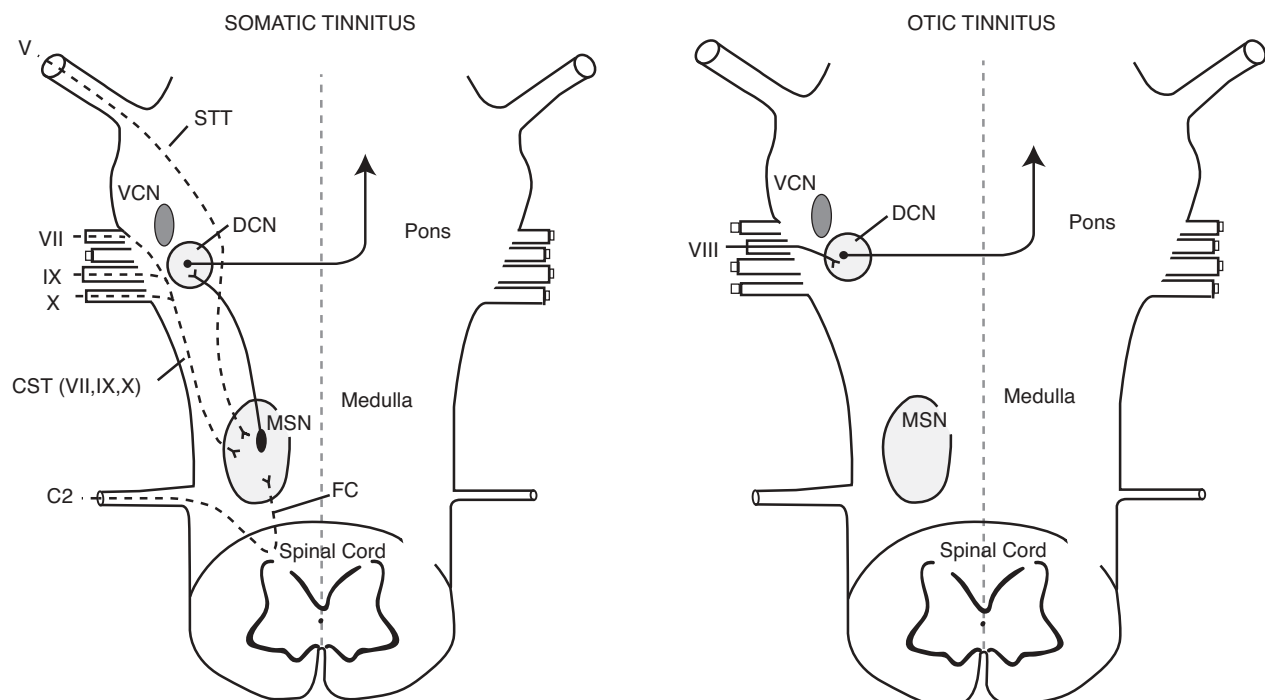


FIGURE 9-1. Schematic diagram of the brainstem and upper cervical part of the spinal cord, depicting the anatomic basis for the dorsal cochlear nucleus (DCN) hypothesis: both somatic and otic tinnitus occur owing to disinhibition of the DCN. In both cases, tinnitus is due to increased activity in the output of the DCN (*curved arrow*), which projects to the other centers and eventually leads to activation of the auditory perceptual machinery responsible for tinnitus. For somatic tinnitus (*left panel*), sensory inputs (*long dashed lines*) from (1) the face via the trigeminal (V) nerve in the spinal trigeminal tract (STT); (2) the external and middle ears via the common spinal tract of the facial, glossopharyngeal, and vagus nerves [CST (VII, IX, X)]; and (3) the neck via the C2 dorsal root and the fasciculus cuneatus (FC) converge to a common region of the lower part of the medulla, the medullary somatosensory nuclei (MSN), from which fibers project to the ipsilateral DCN (*solid line*). Modulation of activity in the MSN-DCN pathway results in disinhibition of the DCN. For otic tinnitus (*right panel*), loss of input (spontaneous activity) from the auditory (VIII) nerve leads to disinhibition of the DCN. VCN = ventral cochlear nucleus.

for by any hypothesis regarding the neuroanatomic basis of this type of tinnitus. If we assume that all cases of somatic tinnitus have a similar neuroanatomic substrate, the possible neuroanatomic regions involved are limited. Sensation of the face is subserved principally by the trigeminal nerve, but the second cervical root also contributes to the sensation of the auricle and, to some extent, the nearby face. Parts of the auricle, ear canal, and tympanic membrane are innervated by branches of the facial, glossopharyngeal, and vagus nerves. Sensation of the upper neck is via the upper cervical roots, namely C2 and C3. The branches of cranial nerves VII, IX, and X that innervate the ear join the spinal tract of cranial nerve V most medially (Figure 9-2), where they come to assume a position adjacent to the most lateral fibers of the fasciculus cuneatus.²⁹ Kunc suggests that this distinct bundle should be called the “common spinal tract of the facial, glossopharyngeal, and vagus nerves” [CST(VII, IX, X)].²⁹ In awake patients, according to Kunc, mechanical stimulation of CST(VII, IX, X) elicits pain in the “auditory passage, the pharynx and the tonsil. Stimulation of the later-

al portion of this small tract evokes pain in the area served by the third division of the trigeminal nerve. Stimulation of the medial portion causes pain over the area innervated by the second cervical spinal root.”²⁹ Thus, despite the fact that somatic nontic tinnitus is associated with the upper cervical dorsal roots and four cranial nerves (V, VII, IX, and X), these primary sensory pathways associated with somatic tinnitus all converge to the region of the CST (VII, IX, X), namely the ipsilateral dorsolateral lower medulla and upper cervical spinal cord. This region has been referred to as the “medullary somatosensory nuclei” (MSN).³⁰ I hypothesize that this region of anatomic convergence is involved in somatic tinnitus.

For this hypothesis to be reasonable, there must be a connection between MSN and the primary auditory pathway. In fact, both experimental anatomic and electrophysiologic studies provide support for such a pathway between this location and the ipsilateral cochlear nucleus, principally the DCN. Anatomic and physiologic studies of the cat and rat demonstrate a direct projection between MSN and the ipsilateral DCN.^{31,32} Although the initial effect of activation of this pathway may be to excite the DCN granule cells, the overall effect appears to be inhibition of the DCN projection neurons, the pyramidal cells, through a multisynaptic system within the DCN. There is evidence that stimulation of the granule cells excites the cartwheel cells, which, in turn, inhibits the pyramidal cells. These authors go on to argue that this pathway may be important in sound localization because pinna position can modify the activity within the ipsilateral DCN via this pathway. I hypothesize that somatic tinnitus occurs because of inappropriate excitation of the auditory pathway, which is due to pathology within a somatic pathway that is normally present and innervates the DCN.

Another line of reasoning based on our somatic modulation observations also implicates the DCN. The fact that somatic modulation appears to originate from the proprioceptive muscle receptors (Golgi tendon organs and muscle spindles) suggests interaction with the part of the auditory system requiring proprioceptive information, that is, for sound localization. Furthermore, because somatic modulation can affect only one ear, then the auditory structure involved must be involved with the mode of sound localization requiring only one ear,

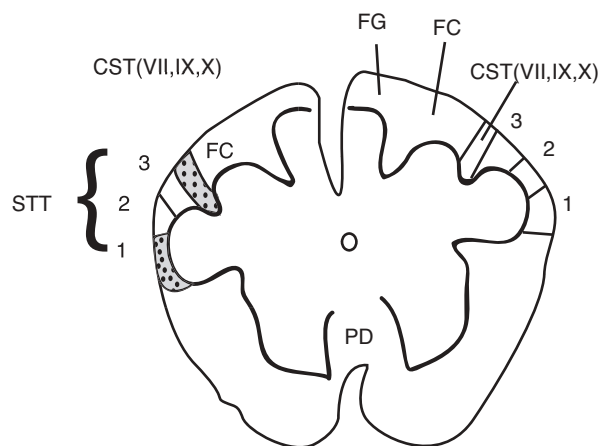


FIGURE 9-2. Cross-section of the lower part of the medulla at the level of the decussation of the pyramidal tract (PD). Stippled on the left is the position of the fibers from the facial (nervus intermedius), glossopharyngeal, and vagus nerves making up the common spinal tract [CST(VII, IX, X)], as shown in relationship to (1) the positions of the fibers of the first, second, and third divisions (1, 2, 3) of the trigeminal nerve in the spinal trigeminal tract (STT) and (2) the fasciculus cuneatus (FC). FG = fasciculus gracilis. Adapted from Kunc Z.²⁹

that is, vertical and front-back sound localization. Finally, animal ablation experiments have implicated the DCN as being involved in vertical and front-back sound localization.³³ Thus, our somatic modulation results also converge on the DCN as the locus of somatic-auditory interactions related to tinnitus.

In summary, consideration of the clinical features of somatic tinnitus syndrome and the properties of somatic modulation of tinnitus, along with experimental neuroanatomy and electrophysiology, leads to the hypothesis that somatic modulation of tinnitus and the somatic tinnitus syndrome occur through modulation of the pathway from the MSN to the ipsilateral DCN. If increased activity in the primary output cells of the DCN is associated with tinnitus, as has been suggested for noise-induced tinnitus, our speculation can be taken a step farther to suggest that inhibition of the MSN to DCN pathway could lead to tinnitus through disinhibition of the DCN fusiform cells.³⁴ The findings in case 8 are consistent with this hypothesis because her tinnitus began with an injection of a local anesthetic to her upper neck, which, presumably, reduced activation of the lateral part of the fasciculus cuneatus, which, in turn, might result in less DCN inhibition. As will be shown, cases of otic tinnitus also give us reason to think that disinhibition in the DCN is involved in the origin of otic tinnitus.

RELATIONSHIP TO OTIC TINNITUS

Multiple theories have been put forward to account for tinnitus related to disturbances of the peripheral part of the auditory system, auditory nerve, and cochlea. Based on the observation that aminoglycoside-induced hair cell loss was associated with loss of spontaneous activity of auditory nerve fibers innervating the region of hair cell loss, one proposal was that tinnitus was a consequence of decreased neural input to the cochlear nucleus.³⁵ It was hypothesized that the absence of active neural input from the auditory nerve to the central nervous system resulted in increased neural activity within the auditory pathway, leading to perception of sound (tinnitus).

Support for this theory also comes from patients who have received auditory nerve electrical stimulation. Although reports vary in the degree of tinnitus improvement with cochlear implants, in our experience, about 80% of these deaf subjects report tinnitus

just prior to receiving their cochlear implants. Following a multichannel cochlear implant, the tinnitus associated with the ear that received the implant improved in 88% (M. A. Jalaludin, D. K. Eddington, R. A. Levine, M. Whearty, unpublished data). Rubinstein and colleagues also reported that, in about half of subjects with high-frequency sensorineural hearing loss of varying degrees, high-rate pulse trains applied to the cochlea suppressed tinnitus, with no perception of the stimulus.³⁶ These observations are consistent with the theory that tinnitus in individuals who are deaf owing to a cochlear disorder is due to the absence of neural input from the auditory nerve because reestablishment of auditory nerve activity with electrical stimulation abolishes or decreases the tinnitus.

Further support for this theory has come from reports of the effect of inner ear lesions on DCN spontaneous activity. In experimental animals with cochlear hearing losses (acoustic trauma or ototoxic drugs), which are known to be associated with loss of type I auditory nerve fiber spontaneous activity, increased spontaneous activity was found in the regions of the DCN tonotopically corresponding to the regions of cochlear injury (particularly outer hair cells) and the associated loss of auditory nerve fiber spontaneous activity.³⁷ The original suggestion that a decrease in spontaneous activity in auditory nerve fibers can lead to increased spontaneous activity from higher levels of the auditory pathway (and thereby tinnitus) is supported by multiple DCN studies.^{18,34,37} In fact, these studies support the idea that the DCN may play an important role in otic tinnitus, possibly through its projections to the inferior colliculus, ventral cochlear nucleus, or medial geniculate body.³⁸

Our model (see Figure 9-1) now generalizes this theory for otic tinnitus to somatic tinnitus by proposing that tinnitus can also occur from a somatic source of DCN disinhibition via a pathway originating from the MSN.

More recent anatomic studies of the DCN have shown that not only do the auditory nerve and MSN provide inputs to the DCN, but multiple other regions of the central nervous system likewise project to the DCN (Figure 9-3).³⁹ Thus, our hypothesis can be further generalized to include the vestibular system, pontine nuclei (including the medial olivocochlear efferent system), and inferior olive. Such inputs, many of which are not readily measured or observed, could account for (1) the high incidence of idiopathic tinnitus and (2) the fact that tinnitus is multifactorial.

OTHER CASES

Although I selected for presentation some of our most clear-cut cases with somatic tinnitus, other cases that, at first, might appear to be obvious cases of otic tinnitus, viewed from this new perspective, may actually be cases of somatic tinnitus. The following case is an example.

Case 17. A 25-year-old, left-handed woman developed an upper respiratory infection with ear discomfort, particularly on the right. As her physician irrigated her right ear canal, she developed excruciating ear pain, hearing loss, and bleeding from the external auditory meatus. By the next day, she was aware of right ear tinnitus as well. A 20% central perforation of the posterior part of the right tympanic membrane was identified, and audiometry revealed a 10 to 20 dB conductive hearing loss. Within 2 weeks, her perforation had healed, and her audiogram returned to normal. However, the right ear tinnitus has remained unchanged for over 2 years. It is described as a high-pitched ring and was matched to a 7 kHz tone at 5 dB SL. She had no other hearing complaints.

Considering that at no time did this patient have any auditory or vestibular complaints attributable to the inner ear, another possible mechanism that would account for her tinnitus is somatic, namely originating from the somatic innervation of the tympanic

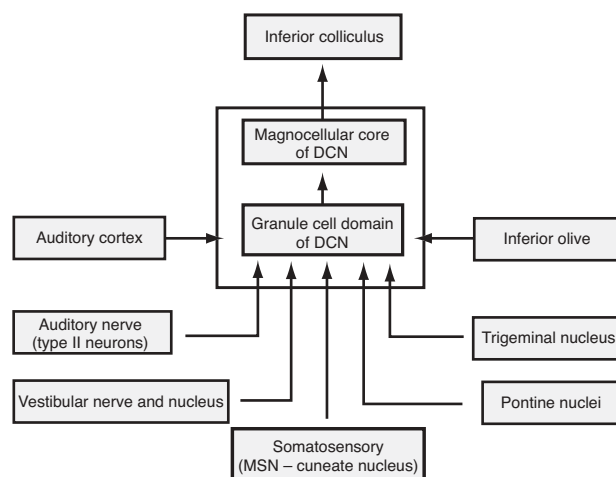


FIGURE 9-3. Block diagram that summarizes the multiple inputs to the granule cell domain of the dorsal cochlear nucleus (DCN). Such organization is consistent with the multifactorial nature of tinnitus. Adapted from Ryugo DK, et al.³⁹

membrane in a manner analogous to cases of facial or dental pain (cases 9, 10, and 13). Coles described several cases of tinnitus following ear canal cleaning that could be on a similar basis.¹

INDIVIDUAL DIFFERENCES IN SUSCEPTIBILITY TO TINNITUS

The fact that for the same otic or somatic insult some individuals will develop tinnitus but others will not suggests that there are differences among individuals in their susceptibility to tinnitus. For example, tooth abscesses are very common, but the development of tinnitus with a tooth abscess is rare (cases 9 and 10). We must conclude that there is something different about the neuroanatomy and/or physiology of individuals who develop tinnitus compared with those who do not develop tinnitus. What these differences may be will require further study but may relate to the wide variety of influences on the DCN. That the cerebellum, which projects to the pontine nuclei, could be involved is suggested by case 8, whose MRI showed small cerebellar infarcts. However, a similar situation obtains for otic tinnitus. Patients with similar diseases of the auditory periphery and indistinguishable audiometry may or may not have tinnitus. Given apparently similar insults to hearing, some people will develop tinnitus, whereas others will not. Nothing about the changes in hearing that occur from an insult have revealed which human subjects have tinnitus and which do not. However, animal models of tinnitus suggest that outer hair cell rather than inner hair cell dysfunction is more likely to cause tinnitus.³⁷ Furthermore, transection of the auditory nerve in patients with otic tinnitus does not reliably diminish the tinnitus.^{40,41}

TINNITUS TREATMENT

From our experience and those of others, it is not clear whether somatic tinnitus can generally be treated successfully by addressing what appears to be the associated condition, such as TMJ syndrome or myofascial pain syndrome involving the craniocervical region. At least two possible explanations can be offered for this experience. First, as can be seen from cases 9, 10, and 13, as well as case 8, there would appear to be a general principle regarding all types of subjective tinnitus that removal of what would appear to be the initial source for the tinnitus does not guarantee that the tinnitus will resolve. Second, in general, treatment for

TMJ syndrome or cervical myofascial pain syndrome has mixed results.

On the other hand, our hypothesis suggests that restoration of DCN inhibition through either the auditory or the somatic inputs to the DCN could suppress some types of unilateral tinnitus (Table 9-8). In fact, there is ample evidence that increasing the inhibition from the ear-DCN pathway through electrical stimulation of the auditory nerve or DCN can suppress tinnitus, for example, with (1) cochlear implants (M. A. Jalaludin, D. K. Eddington, R. A. Levine, M. Whearty, unpublished data), (2) auditory brainstem implants, or (3) high-rate pulse trains applied to the cochlea.^{36,42} Likewise, there are reports suggesting that somatic stimulation of the head or upper neck can suppress tinnitus through this somatic pathway. For example, placebo-controlled studies have shown that mastoid to mastoid electrical stimulation can suppress tinnitus in some patients.^{43,44} Chouard and colleagues suggested that such effects were due to “direct action on sensitive cutaneous fibres, rather than direct action on the cochlea.”⁴⁵ Likewise, reports of acupuncture suppressing tinnitus could be mediated by activation of this somatic pathway.⁴⁶

An altogether different approach to treating this type of tinnitus, namely reduction of DCN output, also follows from our hypothesis. If tinnitus is due to increased neural activity projecting from the DCN to higher centers, interruption of this pathway might abolish the tinnitus. Such a procedure (ablating the DCN or transecting the dorsal acoustic stria) is likely to have little effect on hearing because the behavioral evidence from chronic ablation of DCN outflow pathways in experimental animals suggests that, beside orienting to an elevated sound source, loss of the DCN has no detectable effect on hearing.³³ Patients who would have elected to have their auditory nerve sectioned for control of their unilateral

tinnitus may have derived more benefit from a DCN procedure because sectioning of the auditory nerve guarantees deafness and, in general, has about as much likelihood to worsen as to improve tinnitus; on the other hand, for patients with strictly lateralized otic or somatic tinnitus, our hypothesis suggests a more promising tinnitus treatment with little impact on hearing.

ALTERNATIVE HYPOTHESES

The proposed model for somatic tinnitus is based on combining our clinical observations and known anatomy and physiology. Undoubtedly, there are other possible models that would be consistent with our current state of knowledge. For example, the inferior colliculus receives somatic inputs, and all units of the central nucleus of the inferior colliculus of the cat can be somatically modulated.¹⁷ So the inferior colliculus is another possible site of auditory-somatic interaction. Because the inferior colliculus occurs after the auditory chiasm, activation from this center might not be expected to result in a perception that is consistently fully lateralized. It is for this reason that the inferior colliculus was felt to be a less likely site in the auditory pathway for the initial somatic-auditory interaction.

Another reservation about this model pertains to the DCN. Not only have there been no human studies regarding a pathway from the cuneate or spinal tract of cranial nerve V to the ipsilateral DCN, but the architecture of the human DCN differs from that of many other mammals.^{47,48} The two most superficial layers (granular and molecular layers) are said to be vestigial in adult humans. Nonetheless, some report that, although the relative numbers may differ, in humans, all classes of DCN neurons are found as in other mammals.⁴⁹ Further advances, such as with fMRI, may provide more insights into the neurology of somatic tinnitus that will allow refinement of this model and, ultimately, effective treatment.

TABLE 9-8. Treatments Suggested by the Dorsal Cochlear Nucleus Disinhibition Hypothesis

<i>Restoration of DCN Inhibition</i>	<i>Reduction of DCN Output</i>
Electrical stimulation of auditory nerve	Transect the output tracts of the DCN
Electrical or mechanical stimulation of somatic pathway	Lesion the DCN

DCN = dorsal cochlear nucleus.

CONCLUSION

In this chapter, I reviewed the evidence that the somatosensory system plays a major role in clinical tinnitus. Not only can somatic events cause tinnitus (somatic tinnitus syndrome), but changes in the somatosensory system can account for changes in ongoing tinnitus loudness, pitch, and location (somatic modulation of tinnitus). These somatic-auditory interactions occur within the central nervous system and are not limited to tinnitus but are a fundamental property of the auditory system. The somatosensory inputs appear to originate from motor afferents (Golgi tendon organs and muscle spindles) and almost exclusively from the upper cervical and head regions. Among the principal muscles involved are the SCM and the pterygoids. When somatic-auditory interactions are restricted to one ear, the DCN appears to be the site of interaction. The somatosensory system is probably only one of several nonauditory neural systems that modulate the DCN activity.

These multiple influences on the DCN, many of which are not readily measured or observed, may account for much of the difficulties in determining the cause of and devising effective treatments for tinnitus. The challenge for the future is to understand these relationships so that improved tinnitus treatments can be developed.

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