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## **Somatic (Cranio-cervical) Tinnitus and the Dorsal Cochlear Nucleus Hypothesis**

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## **ABSTRACT**

Of all non-auditory sensory systems only the somatosensory system appears to be related to tinnitus (e.g. temporomandibular joint syndrome and whiplash).

**Purpose:** To describe the distinguishing characteristics of tinnitus associated with somatic events and to use these characteristics to develop a neurological model of somatic tinnitus

**Materials and Methods:** Case series

**Results:** Some patients with tinnitus but no other hearing complaints share several clinical features including (1) an associated somatic disorder of the head or upper neck, (2) localization of the tinnitus to the ear ipsilateral to the somatic disorder, (3) no vestibular complaints, and (4) no abnormalities on neurologic examination. Pure tone and speech audiometry of the two ears is always symmetric and usually within normal limits.

Based on these clinical features, it is proposed that somatic (craniocervical) tinnitus, like otic tinnitus, is due to disinhibition of the ipsilateral dorsal cochlear nucleus. Nerve fibers whose cell bodies lie in the ipsilateral medullary somatosensory nuclei mediate this effect. These neurons receive inputs from nearby spinal trigeminal tract, fasciculus cuneatus, and facial, vagal and glossopharyngeal nerve fibers innervating the middle and external ear.

**Conclusions:** Somatic (craniocervical) modulation of the dorsal cochlear nucleus may account for many previously poorly understood aspects of tinnitus and suggests novel tinnitus treatments.

Tinnitus is common; estimates of its prevalence range up to 80 per cent of all adults<sup>1</sup>. About ten per cent of people complain of chronic tinnitus, while 0.5% of adults describe it as interfering with their ability to lead a normal life<sup>2</sup>. When tinnitus can be ascribed to a sound being generated within the head such as from an abnormal pattern of blood flow near the ear, it is often referred to as objective tinnitus. This report will be restricted to subjective tinnitus, i.e. tinnitus not accountable by the presence of physical sounds.

From a neurological perspective the clinical problem of subjective tinnitus can be conceptualized as having two components. The first is a mechanism for the generation of neural signals somewhere in the auditory pathway that ultimately results in the higher centers critical for auditory perception receiving a neural pattern similar to those generated by external sounds. The second is the affect elaborated by the forebrain in reaction to this activation of the auditory cortex<sup>3,4</sup>. When disorders of the inner ear or auditory nerve are temporally associated with tinnitus lateralized to the affected ear, it is generally considered that the ear/nerve disorder is in some way related to the tinnitus; we shall refer to such tinnitus as "otic" tinnitus. However there are many other patients who have either no detectable ear/nerve disorder or there is no close temporal relationship between such a disorder and tinnitus, so that the initiating event of their "non-otic" tinnitus is obscure<sup>5</sup>. In this report we argue from clinical materials that there is a type of non-otic tinnitus due to central nervous system interactions between the somatosensory system and the auditory system, which may also account for many other previously poorly understood aspects of tinnitus.

The somatosensory system appears to be the only sensory modality that can significantly modulate tinnitus perception in physiologically intact individuals. It has long been observed, almost as a curiosity, that tinnitus can be modulated somatically with face, head, and neck movements. In fact recently such phenomena have been used to provide insights into tinnitus using functional imaging<sup>6</sup>. Systematic studies estimate that

more than 30% of tinnitus patients can somatically modulate their tinnitus<sup>7</sup>. Similarly, tinnitus has been associated with two somatic disorders, temporomandibular joint syndrome and whiplash, both of which are limited to the head and neck<sup>8,9</sup>. While eye movements can modulate tinnitus in a few patients following cerebellopontine angle surgery, the nervous systems of these individuals can not be considered physiologically intact because of their surgery; their tinnitus probably represents an intermodality crosstalk that is related to inappropriate postoperative reinnervation<sup>10</sup>. There have been no reports of visual, gustatory, or olfactory associated tinnitus.

In order to reduce the number of hypotheses that can account for this relationship between tinnitus and the somatosensory system, we report here our clinical observations of cases in which the temporal association between the development of tinnitus and involvement of the somatosensory system argue for the entity "somatic (cranio-cervical)" tinnitus. From these subjects, we will (1) define the principle attributes of somatic tinnitus and (2) propose a specific hypothesis to account for these attributes. Our hypothesis is consistent with experimental data regarding otic tinnitus and is supported by anatomically and physiologically established central somatic-auditory interactions. Furthermore our proposal is testable and predictions regarding specific treatments for somatic and otic tinnitus follow.

## **Case Reports**

The cases described herein have been seen in the Tinnitus Clinic of the Massachusetts Eye and Ear Infirmary.

Case 1. In June 1993 at the age of 52, this right-handed woman injured her right shoulder . In March 1994 the shoulder was repaired under general anesthesia. However a frozen shoulder developed. In order to perform manipulation of the shoulder under anesthesia, on April 22, 1994 she received a right interscalene block. Immediately upon injection of the local anesthetic (15 ml of 1.5% mepivacaine), she developed tinnitus of

her right ear that has persisted unchanged. With the injection, anesthesia of the shoulder did not occur, rather she complained of numbness involving the right ear, right postauricular region, and slightly onto the right side of her face with a dull ache in the same distribution. There was no facial weakness or dizziness. The numbness resolved within 14 hours. Later that day shoulder manipulation was performed under general anesthesia. An otolaryngological evaluation two 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 reflex were normal. Two subsequent audiograms in the next month remained similar, but unlike the first audiogram, these later audiograms also tested 6 kHz and found that for both ears her thresholds (25 dB HL) were slightly worse than all other frequencies tested which were all approximately 10 dB HL. On one of these occasions her tinnitus was matched to a 6 kHz tone at 10 dB SL. She described her tinnitus as a high-pitched ringing in the right ear "like the brakes of a bus." A bolus of intravenous lidocaine abolished the tinnitus for ten minutes. Oral mexilitine provided marginal benefit. A contrast MRI scan in June 1994 reported in the right posterior cerebellar hemisphere two small regions of chronic infarction estimated to be more than six months old. An MRA scan of the neck arteries was normal. Her neurological examinations have always been normal. Repeated matching tests found her tinnitus matched to a 6 kHz tone at 5 dB SL. Spontaneous otoacoustic emissions were not detected.

Case 2. 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 month 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 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 3/10. With turning the head to either side or tilting to the left, loudness increases to 5/10, whereas with tilting to the right the loudness was barely perceptible (1/10). Clenching her teeth increased the loudness only slightly (4/10). On examination, two regions of increased muscle tension and tenderness were noted in the right neck as compared to the corresponding regions on the left, namely the upper sternocleidomastoid and the medial suprascapular regions. Otherwise her otoneurological examination was unremarkable. An audiogram was normal. Her tinnitus matched to a 11 kHz tone at 10 dB SL.

Case 3. This 50 year old right-handed man yawned and within a few minutes noticed a loud noise in his head. The noise lessened over the next 3-4 hours but has persisted over the next twelve years. It was described as a very high-pitched ringing on the right side of the head ("like power lines in the summer"). Manipulation of the head could modify the tinnitus. Pushing inward on the mid-mandible reduced the tinnitus by 90%. Backward flexion of the neck could increase the loudness of the tinnitus, as could firm pressure on any of the following points: the forehead, the right postauricular region, or the right temple. Pressure on the left temple did not alter the tinnitus. When pressure on the right temple was combined with left temple pressure, his tinnitus did not increase. His right tympanic membrane had some old scarring, the remainder of his otoneurological examination was unremarkable. His audiogram was symmetric. Thresholds were normal for 0.5 to 2 kHz, but at 4 and 8 kHz, a 50 to 60 dB hearing loss was present. He had been born with a double-cleft palate and had had multiple reconstructive procedures, but none within the 12 years prior to the tinnitus onset.

Case 4. 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 root canal resolved his dental pain in a few days, while his tinnitus remained unchanged. An audiogram three months following the tinnitus onset 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 three years, the tinnitus is still heard only in the left ear but is generally barely perceptible except for episodes of abrupt growth in loudness of the tinnitus 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. Since the onset of his tinnitus, he describes a vague strange feeling in the left periauricular region.

Case 5. A 27 year old woman reported pain from an upper molar. Tinnitus of the ipsilateral ear began at about the same time. A tooth abscess was diagnosed and treated. Her tinnitus resolved with treatment of the dental abscess. Audiometry was within normal limits.

Case 6. A 34 year old right-handed man presented to an otolaryngologist complaining of five days of high-pitched left-ear tinnitus and a history of three to four 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 on the day before. His examination and audiometry were unremarkable. The facial pain and swelling resolved and the tinnitus improved for about a week following the dental surgery, but then worsened again. When evaluated two months later his tinnitus was present about eighty percent of the time. His left posterior cervical muscles were under increased tension as compared to the right, but were non-tender. Clenching on the left or pressure on the left mastoid abolished his tinnitus. After another three months of nearly continuous tinnitus, the tinnitus stopped. At no time did he have any vestibular complaints.

## Discussion

### *Clinical features of somatic tinnitus*

These six cases illustrate the characteristic features of somatic tinnitus. (1) 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. (2) 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. (3) There are no other associated hearing or vestibular complaints and no abnormalities on the neurologic exam. Pure tone and speech audiometry of the two ears is symmetric and often within normal limits\*. Hyperacusis was 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 such non-otic 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 <sup>9</sup>." Besides 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<sup>11-13</sup>. Wyant describes one case of intermittent unilateral tinnitus in a presumably normal hearing man that was associated with neck pain radiating to the ipsilateral face and eye<sup>14</sup>.

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\* Only the six standard frequencies were tested routinely in these subjects. While it is possible that testing of other frequencies could have been abnormal in some of these subjects (e.g. above 8 kHz), the temporal association between the tinnitus and a somatic factor makes this consideration unlikely.

Many papers describe an association between tinnitus and pain in the region of the ear/temporomandibular joint [we shall refer to this "syndrome" as TMJ]. Some authors emphasize the joint's role and refer to the syndrome as temporomandibular joint syndrome, Costen's syndrome, or craniomandibular disorder<sup>8, 15</sup>. Others stress muscle tension as the key to the syndrome and describe it as myofascial pain-dysfunction syndrome<sup>16-18</sup>. The fact that somatic tinnitus would appear to have been previously described as a part of TMJ suggests that somatic tinnitus not only is limited to the cranial-cervical regions but may be more likely from the lateral cranial-cervical regions, the periauricular regions.

While virtually all reports include tinnitus as part of TMJ, detailed characteristics of the tinnitus are few. Three reports describe some features of such tinnitus consistent with our cases of somatic tinnitus. Bernstein et al. described the tinnitus of his 36 patients as "usually high-frequency hissing, rarely roaring"<sup>19</sup>. Curtis reports the tinnitus as lateralized to the side with the pain in 14 of the 17 patients in whom the pain was unilateral, while the other 3 cases reported bilateral tinnitus. 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<sup>18</sup>. Travell described a patient who had tinnitus ipsilateral to a trigger point in the upper posterior masseter muscle<sup>20</sup>. On the other hand Shulman writes that there was no relationship between the location of the tinnitus and ear pain, temporomandibular joint pain or head pain<sup>11</sup>; however, no supporting data are presented.

A controlled study of tinnitus and the TMJ syndrome defined TMJ as "both clicking in the joint and pain in the region of the ear (joint)<sup>21</sup>." Based on questionnaire data the authors found tinnitus significantly more prevalent in the patients with TMJ than in two control groups. This paper also reviewed and rejected for a variety of reasons previous theories proposed to account for such tinnitus (eustachian tube dysfunction, hyperactivity of the tensor tympani muscle, direct mechanical stimulation of the malleus

by way of a ligament that goes between the malleus and the temporomandibular joint, and "excessive somatic concern.") The authors concluded that the mechanism accounting for the association between TMJ and tinnitus is unknown.

*Neurological Model for Somatic (Craniocervical) Tinnitus*

Rather than attempting to account for somatic tinnitus by involving the middle ear or cochlea, we suggest a neurological model of somatic tinnitus (figure 1) which postulates that tinnitus can arise from somatic-auditory interactions occurring within the central nervous system. This model follows directly from the clinical characteristics of somatic tinnitus. It will be apparent that this model will also account for tinnitus associated with neck pain as described by Wyant<sup>14</sup> as well as the tinnitus of TMJ origin to the extent it is as described by Curtis and Travell<sup>18, 20</sup>.

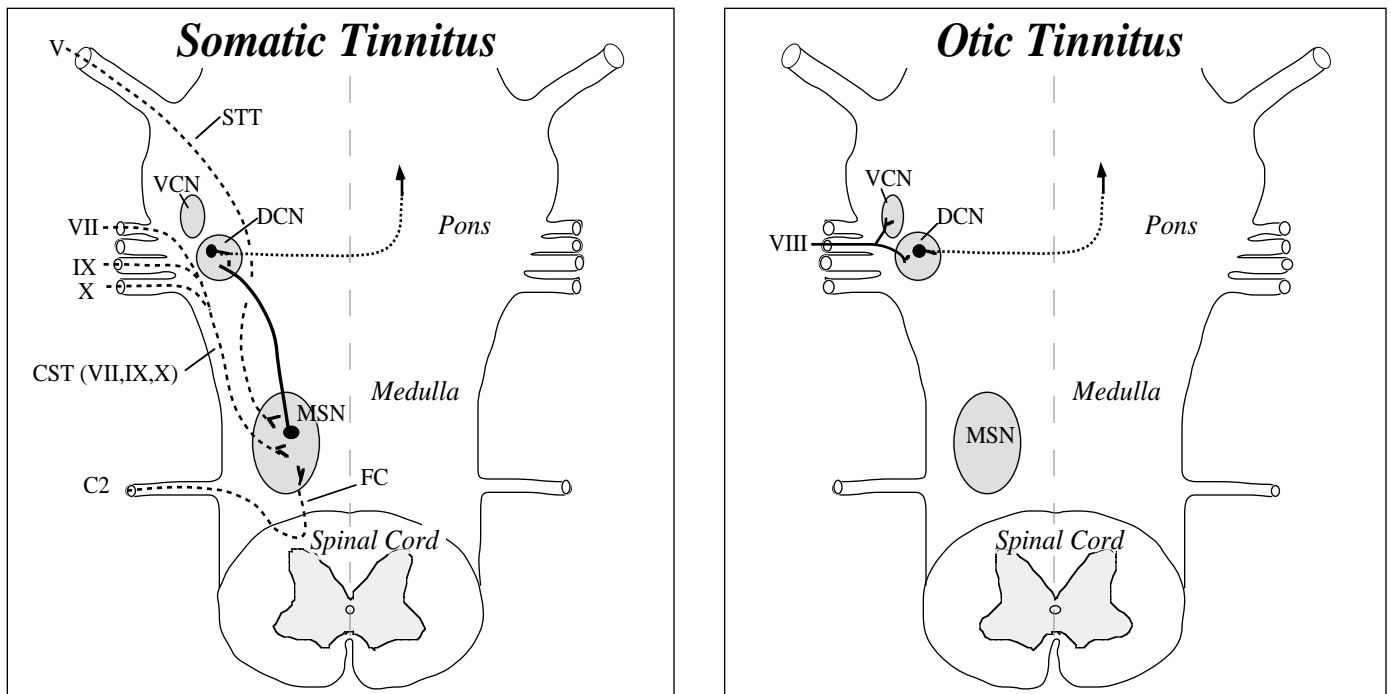


Figure 1. Schematic diagram of brainstem and upper cervical spinal cord, depicting the anatomic basis for the dorsal cochlear nucleus (DCN) hypothesis: both somatic and otic tinnitus occur due to disinhibition of the

DCN. In both cases tinnitus is due to increased activity in the output of the DCN (curved arrow), which then projects to other centers and eventually leads to activation of the auditory perceptual machinery responsible for tinnitus.

For somatic tinnitus (left panel), sensory inputs [shown as long-dashed lines] from (1) the face [via the trigeminal nerve (V) and 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 medulla, the medullary somatosensory nuclei (MSN), from which fibers project to the ipsilateral DCN [solid line]. Modulation of the activity in the MSN to DCN pathway results in disinhibition of DCN. For otic tinnitus (right panel), loss of input (spontaneous activity) from the auditory nerve (VIII) leads to disinhibition of DCN. VCN= ventral cochlear nucleus

In the afferent auditory pathway, while binaural interaction can occur at the cochlear nucleus<sup>22, 23</sup>, it is probably at the level of the superior olivary complex where the binaural interaction necessary for sound lateralization first occurs. 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 then that non-auditory interaction with the auditory system for somatic tinnitus is occurring at the level of the cochlear nucleus, since it is the only part of the afferent central auditory pathway before the trapezoid body, the first auditory decussation important for sound lateralization<sup>24</sup>. 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

non-auditory inputs interact with the auditory system to initiate the neural discharge patterns that are ultimately interpreted as tinnitus<sup>3</sup>.

That the defining cases of somatic tinnitus are associated only with processes which involve the ipsilateral head and upper neck also must be accounted for by any hypothesis regarding the neuroanatomical basis of this type of tinnitus. If we assume that all cases of somatic tinnitus have a similar neuroanatomic substrate, then the possible neuroanatomical 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<sup>25</sup>. Parts of the auricle, ear canal and tympanic membrane are innervated by branches of the facial, vagus and glossopharyngeal nerves<sup>26</sup>. Sensation of the upper neck is via the upper cervical roots, namely C2 and C3<sup>25</sup>. The branches of cranial nerves VII, IX, and X that innervate the ear join the spinal tract of V most medially (figure 2) where they come to assume a position adjacent to the most lateral fibers of the fasciculus cuneatus<sup>27</sup>. Kunc suggests that this distinct bundle should be called the "common spinal tract of the facial, glossopharyngeal, and vagus nerves" [CST(VII,IX,X)]. 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 lateral 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." Thus, despite the fact that somatic non-otic tinnitus is associated with the upper cervical dorsal roots and four different 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)<sup>28</sup>." We hypothesize then that this region of anatomical convergence, MSN, is involved in somatic non-otic tinnitus.

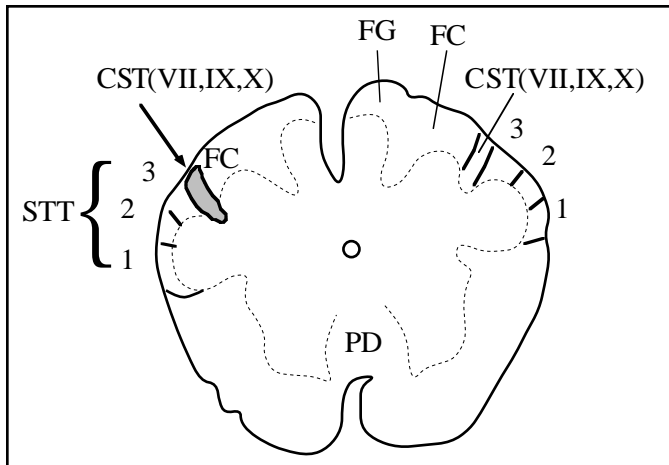


Figure 2. Cross section of lower medulla at level of decussation of the pyramidal tract (PD). Stippled on the left is the position of the fibers from the facial (nervus intermedius), glossopharyngeal and vagus nerves (CST(VII,IX,X)), as shown in relationship to (a) the positions of the fibers of the first, second and third divisions (1, 2, 3) of the trigeminal nerve in the spinal trigeminal tract (STT) and (b) the fasciculus cuneatus (FC). FG= fasciculus gracilis. From Kunc (1965).

For this hypothesis to be reasonable there must be a connection between MSN and the primary auditory pathway. In fact both experimental anatomical and electrophysiological studies provide support for such a pathway between this location and the ipsilateral cochlear nucleus, principally the dorsal cochlear nucleus (DCN). Anatomical tracing studies in the cat<sup>29</sup> and rat<sup>30, 31</sup> demonstrate a direct projection between MSN and the ipsilateral DCN. Physiological studies of the cat confirm such a pathway<sup>28, 32</sup>. While the initial effect of activation of this pathway may be to excite the DCN granule cell, 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 cell excites the cartwheel cell which in turn inhibits the pyramidal cell<sup>31</sup>. 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<sup>33</sup>. We hypothesize, then, 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.

In summary, consideration of the clinical features, along with experimental neuroanatomy and electrophysiology leads to the hypothesis that cranio-cervical somatic tinnitus occurs through modulation of the pathway from MSN to the ipsilateral DCN. If increased activity in the DCN is associated with tinnitus, as has been suggested for noise-induced tinnitus<sup>34</sup>, then our speculation can be taken a step further to suggest that inhibition of the MSN to DCN pathway could lead to tinnitus through disinhibition of the DCN. Our case 1 above is consistent with this hypothesis since her tinnitus began with an injection of a local anesthetic to her upper neck which presumably reduced activation of the lateral cuneate, which in turn might result in less DCN inhibition. As we will see, 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 auditory system, the auditory nerve and cochlea<sup>35</sup>. 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<sup>36</sup>. 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 pathways leading to perception of sound (tinnitus).

Support for this theory also comes from patients who have received cochlear implants. While reports vary in the degree of tinnitus improvement with cochlear implants<sup>37-40</sup>, in our experience, about eighty per cent of these profoundly 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% (Jalaludin MA, Eddington DK, Levine RA, and Whearty M., unpublished data).

These observations are consistent with the theory that tinnitus of the profoundly deaf due to a cochlear disorder is due to the absence of neural input from the auditory nerve, since reestablishment of auditory nerve activity with electrical stimulation (via a cochlear implant) abolishes or decreases the tinnitus.

Further support for this theory has come from reports of the effect of inner ear lesions upon DCN spontaneous activity<sup>34, 41</sup>. In hamsters with cochlear hearing losses (acoustic trauma), which is known to be associated with loss of auditory nerve fiber spontaneous activity, increased spontaneous activity was found in the regions of the DCN tonotopically corresponding to the regions of cochlear injury and the associated loss of auditory nerve fiber spontaneous activity.

The original suggestion that 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 this DCN study. 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<sup>42</sup> or ventral cochlear nucleus<sup>43</sup>. Our model (figure 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 MSN.

### *Other cases*

While we have 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.

Case 7. 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 twenty per cent central perforation of the posterior right tympanic membrane was identified and audiometry revealed a 10 to 20 dB conductive hearing loss. Within two weeks her perforation had healed and her audiogram returned to normal. However, the right-ear tinnitus has remained unchanged for over two years. It is described as a high-pitch 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 otherwise 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 membrane in a manner analogous to the cases of facial or dental pain (cases 2,4, & 5). Coles has described several cases of tinnitus following ear canal cleaning that could be on a similar basis <sup>13</sup>.

#### *Somatic-otic interactions*

Similarly, it is likely that factors predisposing toward otic tinnitus can interact with factors predisposing toward somatic tinnitus. Two cases illustrate this point.

Case 8. A 50 year old patient 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 as compared to the corresponding muscles on her right side. Within an estimated five minutes of examining the cervical musculature, she reported that her left-sided tinnitus had started. On re-examination her left suboccipital muscle tension had become much more

pronounced. Within another five minutes her tinnitus abated and her suboccipital muscles were again more relaxed.

Case 9. A now 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 1/10. A 1990 audiogram revealed normal thresholds bilaterally except 25 dB HL at 4 kHz for the left ear. In 1994 5-6 days after placement of a permanent crown on a left lower molar, his left ear tinnitus became much louder (4-5/10). At about the same time he had also attended a loud concert. His tinnitus then remained unchanged until 1997, when it vanished (0/10) following placement of a temporary inlay on the tooth that occludes with the 1994 crown. Two weeks later while leaving the dentist office after the placement of the permanent gold inlay, his tinnitus suddenly became very loud (10/10) and remained that way for the next 6 months. A repeat audiogram revealed normal thresholds except again for the left ear at 4 kHz whose threshold was now 40 dB HL. Over the last 2 years his tinnitus loudness has gradually decreased to the 6-7/10 range.

These observations are dramatic examples of how tinnitus of presumably otic origin can interact with cranio-cervical somatic factors. Wyant describes a similar case of a woman, who developed head pain and unilateral tinnitus on the side with an ear that had been deafened 12 years earlier by a labyrinthectomy<sup>14</sup>; her tinnitus and head pain responded transiently to trigger point injection. According to Rubinstein<sup>7</sup> about one third of patients could modulate their tinnitus by jaw movements or pressure on the temporomandibular joint. Sixty-five percent of thirty-seven consecutive patients seen in our clinic could modulate their tinnitus with isometric head/neck contractions. Like the cases of purely somatic tinnitus, somatic modulation of tinnitus is restricted to the head and neck. There have been no reports of such somatic modulation outside of the head

and upper neck in physiologically intact individuals using physiological stimuli\*. Somatic modulation of tinnitus can easily be accounted for by central nervous system interactions between the auditory and somatic systems, such as has been proposed by our neurological model.

### *Individual differences in susceptibility to tinnitus and response to treatment*

The fact that for the same otic or somatic insult some individuals will develop tinnitus and others will not suggests that there are differences amongst individuals in their susceptibility for tinnitus. For example, tooth abscesses are very common, but the development of tinnitus with a tooth abscess is rare (cases 4, 5 & 6). We must conclude that there is something different about the neuroanatomy and/or physiology of individuals who develop such tinnitus as compared to those who do not develop such tinnitus. Similarly there must be differences between individuals who develop a transient tinnitus from a tooth abscess (cases 5 & 6) and those who develop a permanent tinnitus (case 4). What these differences may be will require further study. That the cerebellum could be involved is suggested by case 1, whose MRI scan reported 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 and 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<sup>46, 47</sup>. Furthermore transection of the auditory nerve in patients with otic tinnitus does not reliably diminish the tinnitus<sup>48-51</sup>.

### *Tinnitus treatment*

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\* Cacace has described a subject who had a cerebellopontine angle tumor removed and with cutaneous stimulation of the dorsum of the hand hears a sound in the ear that had been operated upon<sup>44</sup>. Møller was able to modify some subjects' tinnitus with electrical stimulation of the median nerve<sup>45</sup>.

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 temporomandibular joint syndrome<sup>52</sup> or myofascial pain syndrome involving the cranio-cervical region<sup>14, 53</sup>. At least two possible explanations can be offered for this experience. First as can be seen from cases 4, 5 and 6, as well as case 1, 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. Secondly, in general, treatment for temporomandibular joint syndrome or cervical myofascial pain syndrome has mixed results.

Table 1.

<b>Treatments suggested by the dorsal cochlear nucleus disinhibition hypothesis</b>	
<b>Restoration of DCN inhibition</b>	<b>Reduction of DCN output</b>
-Electrical stimulation of auditory nerve -Electrical or mechanical stimulation of somatic pathway	-Transect the output tracts of DCN -Lesion DCN

On the other hand, our hypothesis suggests that restoration of DCN inhibition through either the auditory or somatic inputs to the DCN could suppress some types of unilateral tinnitus (table 1). 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, e.g. with cochlear implants (Jalaludin MA, Eddington DK, Levine RA, and Whearty M., unpublished data) or with the auditory brainstem implant<sup>54</sup>. 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<sup>55, 56</sup>. Chouard et al. suggested that such effects were due to "direct action on sensitive cutaneous fibres, rather than direct action on the cochlea"<sup>57</sup>. Likewise reports of acupuncture suppressing tinnitus could be mediated by activation of this somatic pathway<sup>58, 59</sup>.

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, then 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 upon hearing because the behavioral evidence from chronic ablation of experimental animal DCN outflow pathways suggests that, besides orienting to an elevated sound source, loss of the DCN has no detectable effect upon hearing<sup>60</sup>. Patients who would have elected to section their auditory nerve for control of their unilateral tinnitus may derive more benefit from a DCN procedure, since sectioning of the auditory nerve guarantees deafness and, in general, has about as much likelihood to worsen as 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 upon 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<sup>61</sup> and all units of the cat central nucleus of the inferior colliculus can be somatically modulated<sup>62</sup>. 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/spinal tract of V to the ipsilateral DCN, but the architecture of the human DCN differs from that of many other mammals. The two most superficial layers (granular and molecular layers) are said to be vestigial in adult humans<sup>63-65</sup>. Nonetheless some report that, while the relative numbers may differ, in man all classes of DCN neurons are found as in other mammals.<sup>63</sup> Further advances such as with functional imaging may provide more insights into the neurology of somatic tinnitus that will allow refinement of this model and ultimately more effective treatments<sup>66, 67</sup>.

### *Testing the somatic hypothesis*

Finally, our hypothesis can be systematically investigated. For example by appropriately selecting subjects and applying somatic stimulation to the craniocervical region by various techniques (electrical, motor), such subjects can be studied for modulation of their tinnitus and be compared to appropriate controls. Case 1 (and others not reported herein) suggest that somatic tinnitus may be particularly responsive to intravenous lidocaine<sup>68</sup>.

### *Conclusions*

Whether or not the proposed model for somatic (craniocervical) tinnitus is correct in all its details, it represents a focus for future systematic studies of somatic tinnitus and a framework for approaches to treatment. Moreover, we have presented a series of patients in whom the evidence argues for a craniocervical, non-otic basis for their tinnitus. As such it seems likely that some cases of somatic tinnitus may result from

interactions between the somatic and auditory pathways within the central nervous system with no involvement of the auditory periphery (cochlea, or auditory nerve).

It may be that central somatic-auditory interactions will have even broader implications for tinnitus than the "pure" cases of somatic tinnitus reported above. We have also presented cases in which both otic and somatic factors appear to interact to cause tinnitus. Such otic-somatic interactions may account for (1) why some patients with a hearing disorder develop tinnitus and others do not with an otherwise identical hearing disorder, (2) why some patients with chronic progressive hearing loss develop tinnitus at some point in time, (3) why patients with symmetric hearing loss can develop tinnitus in only one ear, or (4) how cranio-cervical movements can modulate tinnitus.

The concept of somatic modulation of the central auditory pathway opens up a whole new way of studying such questions<sup>45</sup>. It may be that these cases of "pure" somatic tinnitus and somatic-otic tinnitus can point to a significant role for intermodality "masking" (somatic modulation) in the treatment of tinnitus.

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