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Review Article

Pulsatile Tinnitus without an Acoustic Source: Somatosensory from Head and Neck Myofascial Dysfunction (SSPT) or Auditory Nerve Compression, or Both?

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Abstract

The cardiac synchronous tinnitus of patients for whom no acoustic source has been detected, despite a thorough investigation, is referred to as neuropulsatile tinnitus, since the tinnitus is not on an acoustic basis but on a neurological basis. Review of our patients with neuropulsatile tinnitus has led to the identification of two etiologies: (i) auditory nerve compression and (ii) head and neck myofascial dysfunction (also known as somatosensory pulsatile tinnitus syndrome or SSPT). Both are amenable to treatment: decompressive surgery for the former, dry needling and/or intramuscular botulinum toxin injections for the latter.

Keywords: somatic testing; somatic tinnitus; recumbent; modulation

Abbreviations:

SSPT : somatosensory pulsatile tinnitus syndrome

NPT : neuropulsatile tinnitus

ANC : auditory nerve compression

ST : somatic testing

AES : auricular electrical stimulation

DCN : dorsal cochlear nucleus

Introduction

Despite an exhaustive effort to detect an acoustic source for pulsatile tinnitus, in some cases none is found [1]. Such cases will be referred to as neuropulsatile tinnitus (NPT). It is very likely that ear canal recordings, analyzed using Spectro-Temporal Analysis, will be able to identify patients with NPT without requiring an exhaustive imaging and metabolic evaluation. However, the definitive study establishing the utility of this technique has yet to be performed [2, 3].

Auditory nerve compression: Auditory nerve compression (ANC) is one of the two possible etiologies for unilateral NPT. That VIIIth nerve vascular compression can cause unilateral NPT has been well established by Ryu and colleagues. They made their observations in subjects with unilateral tinnitus whose primary complaint was ipsilateral hemifacial spasm, not tinnitus. In half of these subjects their tinnitus was pulsatile; all described their NPT as low-pitched. Surgical decompression of the facial nerve in the cerebellopontine angle was performed to treat their hemifacial spasm. At surgery they found that 100% of those with preoperative tinnitus had arterial ANC, while only 6% of those without tinnitus had ANC. Furthermore surgical decompression of the auditory nerve resolved the NPT of 80% [4]. In a second report of surgical decompression of the auditory nerve within its cisternal segment for tinnitus, it was concluded that low pitch pulsatile and high pitch continuous tinnitus are probably due to ANC within the cistern and, if preoperative hearing is well preserved, can be resolved by surgical decompression [5]. These reports lead to the conclusion that ANC can cause unilateral NPT and in such cases surgical decompression of the auditory nerve is highly effective.

Somatosensory pulsatile tinnitus syndrome (SSPT) from head and neck myofascial dysfunction: The other cause of NPT is SSPT [6, 7]. Unlike ANC which is always unilateral, SSPT can be lateralized or non-lateralized. Thirty-three (45%) of our 73 NPT patients had unilateral NPT; forty (55%) had non-lateralized NPT. Unlike ANC the evidence for SSPT causing NPT is more circumspect and relies upon three lines of evidence: how NPT is affected by (i) intense activation of head and neck muscles, tendons and joints, which is referred to as "somatic testing (ST)," (ii) treatments directed toward SSPT, and (iii) recumbency [7].

A. How NPT is affected by somatic testing (ST)

Our studies of 73 patients with NPT has found that 100% could modulate their tinnitus with ST, whether or not their NPT was (i) lateralized, (ii) present at the time of ST, or (iii) suppressible by ST [6, 7]. This contrasts with only 80% from 99 subjects with non-pulsatile tinnitus ($p < .00001$) [8].

Another striking difference between these two groups is the number who could abolish their tinnitus completely with ST: 62% for the NPT group vs. 14% for the non-pulsatile tinnitus group ($p < .00001$).

When NPT is Intermittent ---

Of the nineteen subjects with intermittent NPT, nine were hearing their tinnitus at the time of ST. Eight of the nine (88%) suppressed their pulsations with ST; in all but one the NPT was totally abolished.

Of the 11 subjects who underwent ST when their intermittent NPT was not present, seven (64%) elicited their NPT (Table 1). Pulsatile tinnitus was never elicited by ST in any of the 99 non-pulsatile tinnitus subjects.

When NPT is Constant ---

Of the fifty-four subjects (74%) with constant NPT, fifty (93%) could suppress their pulsations with ST. The suppression was total for about ¾ of these subjects. For the other ¼ the pulsatile quality only was suppressed; some residual non-pulsatile tinnitus was still heard.

Combining the intermittent cases of NPT with the constant cases, 63 had NPT at the time of ST and 58 (92%) of them suppressed their pulsations with ST (table 1).

NPT Present at time of testing?	Effect of ST upon NPT	# of cases	%
Yes	Suppressed	58	79
	NOT Suppressed	5*	7
No	Elicited	7*	10
	NOT Elicited	4	6
	Total	73*	

*One subject is included in the table twice. Her NPT was intermittent and she was tested with ST twice. At the first encounter when her NPT was not present, ST elicited NPT. At the second encounter NPT was present but was not suppressed.

Table 1: How Somatic Testing (ST) Modulates Neuro pulsatile Tinnitus (NPT)

B. How NPT is affected by treatments directed toward SSPT

In four cases of NPT there has been a major response to treatment. While anecdotal they have implications for SSPT causing NPT.

Dry needling of cervical trigger points:

Case 1: About twelve times over four years, a 70-year-old otolaryngologist developed severe right-sided neck pain and constant right-sided NPT. Each time his NPT recurred he received dry needling of his right sternocleidomastoid and adjacent muscles. After about 3 weekly sessions, his NPT and pain resolved completely.

Case 2: A 67-year-old woman developed non-lateralized, non-pulsatile constant tinnitus together with constant NPT that was usually but not always left-sided. Her exam revealed multiple left posterolateral cervical trigger points. Seven months after the onset of her NPT and within a span of six weeks, she received four sessions of dry needling of her cervical trigger points, after which her constant NPT resolved while her other tinnitus persisted.

Botulinum toxin injections of cervical trigger points:

A colleague has reported that intermittent unilateral NPT resolved for 4 to 6 months in 11 patients following botulinum toxin injections of the ipsilateral splenius capitis at the craniocervical junction [9].

Auricular electrical stimulation (AES):

Case 3: A 58-year-old woman had had constant right-sided NPT for one year; with ST the pulsatile quality of her NPT was transiently totally suppressed. Following 7 weekly applications of AES consisting of 3 days of continuous 1 per sec, 1 msec pulses, her NPT gradually attenuated and by four months she had no tinnitus [10, 11].

Case 4: After 10 weekly AES applications for constant, non-lateralized NPT that had been present for more than a year, this 59-year-old man's NPT stopped and was replaced by intermittent non-pulsatile tinnitus that was not heard about a third of the time and was quieter than his NPT had been. The benefit presently has persisted for more than four years. Prior to AES he had found that sternocleidomastoid and splenius capitis massage would inconsistently provide total relief for 1-2 days.

Dry needling of cervical trigger points and botulinum toxin injections of cervical trigger points are clearly acting upon the neck muscles and, thereby, support the hypothesis that head and neck myofascial dysfunction can cause NPT. The mechanism for AES is less straightforward, but the auricular innervation consists of branches of the trigeminal and vagus cranial nerves and the spinal dorsal roots of C2 and C3 all of which have been implicated in tinnitus generation since (a) they converge upon the medullary somatosensory nucleus of the brainstem which only projects to the dorsal cochlear nucleus (DCN) and (b) the DCN is the presumptive source of tinnitus [12, 13].

How NPT is associated with recumbency

Of our 78 patients, nine had NPT that was intermittent with no other types of non pulsatile tinnitus. Eight of these nine reported a close relationship between their intermittent NPT and recumbency, as did another two who in addition to their intermittent NPT also heard a non-pulsatile tinnitus (Table 2). Ten of the eleven were women. The one man (M/81) had lateralized intermittent NPT. For ten their intermittent NPT occurred almost exclusively with prolonged recumbency and disappeared upon arising from bed. F/40 at times did not hear her P when recumbent, but always upon awakening, even if not heard overnight. Only F/70 was examined when recumbent and having NPT.

Case 5 (F/70). This 70 year old woman reported having 7 months of non-lateralized intermittent NPT ("motor") that occurred daily and almost exclusively after recumbent in bed for several minutes. Audiogram, MRA and MRV were normal. At her initial visit her NPT was not present despite being supine for about five minutes. No bruits were detected. ST elicited no tinnitus. At her next visit after lying awake and supine for 30 minutes, her NPT began and was localized to her occiput. Her silent 30-second count of her pulsations and the examiner's simultaneous count of her radial pulse were identical. No bruits were detected. Right, left, or bilateral jugular compression did not alter her NPT. Active turning of her head to the left increased the loudness of her NPT. Her NPT was unchanged by active or

passive neck forward flexion, but her NPT was abolished by neck forward flexion against resistance. When contacted five years later her "recumbent" NPT was rarely occurring.

The relationship between some cases of NPT and recumbency (Table 2 and case 5) again is supportive of the hypothesis that somatosensory inputs from the head and neck account for many cases of NPT. As observed for case 5, her NPT was not related to sleep but rather to recumbency, since her NPT appeared after 30 minutes and yet she was awake. With recumbency a major change occurs in the activity of the somatosensory afferents originating from the cervical muscles, tendons and joints. When upright these cervical structures are supporting the 10-pound head but, when recumbent, these same structures are in a more relaxed state. In these eleven cases it is likely that the change in the activity of the somatosensory afferents originating from the cervical muscles, tendons and joints is responsible for the change in NPT. Thus the association between recumbency and NPT again supports the concept that NPT is closely related to the somatosensory afferents arising from the head and neck.

Is there a relationship between the unilateral NPT from ANC and the unilateral NPT from SSPT?

Imaging presently cannot reliably identify symptomatic ANC [14]. However "typewriter tinnitus" especially if confirmed by its suppression by carbamazepine is a reliable sign of the presence of ANC [15, 16]. On the other hand suppression of NPT by ST is a reliable sign of SSPT. The following case raises intriguing questions about the relationship between these two NPT etiologies.

Case 6. A 60-year-old woman reported at her first visit that her right ear tinnitus began three months earlier as a few occurrences of a foghorn lasting a few seconds; a week later it became constant and cardiac synchronous. When her tinnitus was very quiet, she heard only pulsatile clicking, when loud only pulsatile "whooshing." No bruits were detected in the cervical or periauricular regions. Jugular compression did not alter her tinnitus. With ST all tinnitus was abolished by resisted neck forward flexion. Her audiogram showed normal pure tone thresholds at 3 kHz and below. Above 3 kHz was a sloping loss reaching 40 dB at 8 kHz. Thresholds were identical at the two ears except for 15 dB poorer for the right ear at 1 and 3 kHz. Her CISS MRI scan detected ANC of the right VIIIth nerve in its cisternal segment (figure 1). A trial of carbamazepine was aborted after 3 days due to a rash. Within less than a year from its onset her clicking had stopped. Now nine years from its onset her whooshing NPT persists.

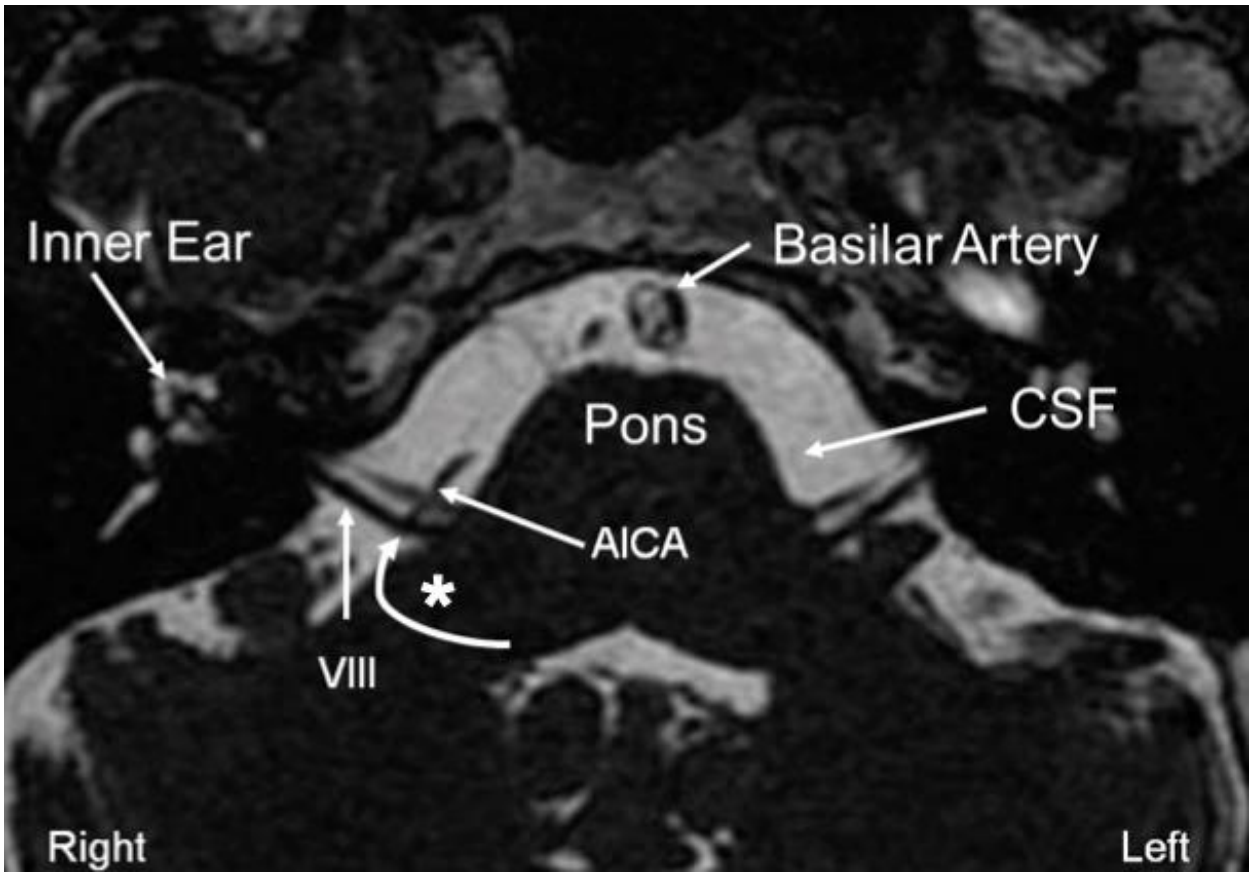

Figure Legend 1: Case 6. MRI scan of NPT subject with features of both ANC and SSPT. In addition to her MRI showing right cisternal ANC (curved arrow, next to asterisk) she had intermittent clicking (typewriter tinnitus) pathognomonic for ANC. Characteristic of SSPT her NPT was totally abolished by resisted neck forward flexion. AICA = anterior inferior cerebellar artery, VIII = eighth cranial nerve, CSF = cerebrospinal fluid. From Levine RA, Oron Y. Tinnitus [23].

The above case of lateralized NPT has ANC unquestionably based upon her imaging as well as her pathognomonic clicking [17]. In addition her NPT is low pitched as has been reported for cases of surgically verified ANC [4, 5]. Another one of our cases of lateralized NPT was felt by the radiologist to have a vascular loop within his left internal auditory canal "where there may be neurovascular contact." The patient described his NPT as constant, left and high-pitched; it was abolished by right jaw deviation against resistance. A third subject (M/81 of Table 2) with lateralized NPT described two types of left tinnitus: (a) low-pitched NPT, present only when recumbent and (b) constant humming. He was never tested while having his NPT. His MRI scan was highly suspicious for vascular compression of his left auditory nerve within the medial cistern where the auditory nerve enters the brainstem at the pontomedullary junction.

Sex/Age	Lateralized?	Relationship to recumbency
F/62	No	Primarily in bed. Stops immediately when she sits up
F/70	No	Hear in bed almost exclusively
F/47	No	Always in bed before arising; and again late in day
F/40	No	Always upon awakening. Gradually quiets during day
F/48*	No	Always in bed but not upon awakening
M/81*	Yes	Only after recumbent 5-10 minutes
F/41	Yes	Only when recumbent to L
F/22	Yes	Only when recumbent to R
F/34	Yes	Only when recumbent to side with headache
F/64	Yes	Only when recumbent to L

* Cases with constant non-pulsatile tinnitus in addition to their NPT

Table 2: Ten cases with intermittent NPT closely related to recumbency

In all three of these cases the NPT was "fully lateralized." It was perceived in the ear itself, not in the vicinity of the ear or the side of the head and with no tinnitus in the contralateral ear. From case 6 with definite ANC, when taken together with the two other cases with imaging suggestive of ANC, it is tentatively concluded that NPT from ANC (a) is "fully lateralized" (perceived in the ear itself), (b) can be abolished by somatic testing, (c) can be constant or intermittent, and (d) can be accompanied by other types of non-pulsatile tinnitus, such as "typewriter" or humming.

It is abundantly clear then that suppression of unilateral NPT with ST does not distinguish between unilateral NPT's two etiologies, SSPT and ANC. In other words ANC and SSPT can co-exist in the same patient. Unilateral SSPT does not exclude the possibility of ANC.

Discussion & Conclusion

Mechanism of NPT

Auditory Nerve Compression

It has previously been hypothesized that NPT from ANC is actually a soma to sound and not a neuro sound [18]. The suggestion was made that an arterial loop within the internal auditory canal is causing a sound generated by "a resonance effect in the petrous bone transmitted to the cochlea." Case 6 involves ANC but not within the internal auditory canal rather within the cerebellopontine angle cistern where there is no contact with the petrous bone. Further more for the NBT cases of Ryu et al., ANC was confirmed visually from the surgical exposure of the auditory nerve and its neighboring blood vessels with no reference to any petrous bone contact. Like case 6, Ryu's cases of low pitch pulsatile tinnitus were found to have ANC in the cisternal segment of the auditory nerve [5, 19]. None of these cases are consistent with the resonance hypothesis.

These surgically confirmed cases of cisternal ANC with low pitch NPT and "mildly disturbed hearing" suggest that their pulsations are from pulsatile modulation of the auditory nerve single unit activity from the pulsatile pressure upon the cisternal segment of the auditory nerve.

That NPT from ANC can be suppressed by ST raises questions about the mechanism of its suppression. Consider the following two facts: (i) the auditory nerve projects only to the cochlear nucleus and (ii) cervical and head somatosensory afferents do not project directly or indirectly to the auditory nerve. When taken together with the proposition that NPT from ANC is due to pulsatile modulation of the single unit activity within the cisternal auditory nerve, these two facts suggest that the ST suppression of ANC-related NPT is occurring at the DCN or some higher level of the auditory CNS [13].

SSPT - from Head and Neck Myofascial Dysfunction

Unilateral SSPT As described above, when exclusively unilateral and located to the ear itself, even if suppressed by ST, ANC is a distinct possibility. In such cases the pulsations likely are from arterial compressions of the auditory nerve. Other unilateral cases are not reported as localized to the ear itself but more diffusely one-sided such as temporally, occipitally, or variably on one side of the head. Some will even describe at times involvement of the contralateral side (e.g. case 2). Such cases are unlikely to be from ANC, but related to head and neck myofascial dysfunction.

Head and neck myofascial dysfunction has been proposed to cause lateralized SSPT in one of two ways[6]. Both involve a modification of the original somatic tinnitus theory which hypothesized that tinnitus results from head and neck muscle afferents disinhibiting the activity of the ipsilateral dorsal cochlear nucleus (DCN) via the medullary somatosensory nucleus, a component of the trigeminal-cervical-complex [13]. One way proposes that the neural activity of head and neck somatosensory afferents to the auditory CNS is cardiac synchronous and thereby causes cardiac synchronous disinhibition of the DCN. A second way is that the head and neck somatosensory afferents disrupt one of functions of the DCN, namely, the suppression of self-generated sounds. Besides respirations, chewing, and vocalizations the DCN functions to suppress our internally generated cardiovascular sounds [20, 21]. Disruption of the DCN's ability to suppress cardiovascular sounds would result in NPT. A related consideration is some combination of these two proposed mechanisms.

Non-lateralized SSPT: From considering several possibilities to account for non-lateralized SSPT, the only viable hypotheses were either the above two mechanisms occurring bilaterally or cardiac synchronous modulation of the auditory CNS either (a) in a locus rostral to the trapezoid body (the auditory decussation) or (b) from interactions between neural structures from both sides either above or below the trapezoid body, such as through the reciprocal connections between the cochlear nuclei of the two sides [6, 22].

Conclusions

NPT when non-lateralized is caused by CNS auditory dysfunction mediated by somatosensory inputs from the muscles, tendons and joints of the head and neck (SSPT), including the 8% of cases who cannot suppress their pulsations by ST. When NPT is lateralized, in addition to SSPT ANC must be considered, particularly if the NPT is localized to the ear. Also when lateralized, suppression of pulsations by ST does not always distinguish between these two considerations, since some cases of ANC can suppress their pulsations by ST.

Surgical decompression of the auditory nerve can often abolish NPT when the compression is cisternal. Needling of head and neck muscles (dry needling and/or trigger point injections), auricular electrical stimulation, and botulinum toxin injections of head and neck muscles can abolish NPT due to SSPT in some.

Conflict of Interest

None

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None

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