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Tinnitus Evoked by Speech

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Abstract: Modulation of tinnitus by a variety of somatosensory stimuli and alteration of gaze has been described. We present two cases of tinnitus induced by speech (voice). The tinnitus was troublesome in both patients, and both had hearing loss and ischemic changes in the central nervous system documented by magnetic resonance imaging. We discuss cross-modal plasticity and how it may explain the tinnitus in these two patients.

Key Words: evoked; plasticity; speech; tinnitus

Tinnitus evoked by gaze or somatosensory stimulus has raised considerable interest of late. The association of tinnitus with alteration of gaze [1], jaw clenching [2], movements of head, neck, and limbs [3], cutaneous stimulation of the upper limbs [4], electrical stimulation of the median nerve [5], and application of pressure on the temporomandibular joint has been described [6]. Gaze-evoked tinnitus typically occurs after posterior fossa tumor surgery [7]. Pinchoff et al. [8] described a variety of other orofacial movements, including pressure on the temple, modifying tinnitus.

We describe here two patients whose presenting symptoms were troublesome tinnitus induced by voice production. We have not found reference to vocalization-induced tinnitus in the literature.

CASE REPORTS

Patient 1

A 75-year-old woman presented with a symptom of hearing a harsh, rough sound on talking or singing. The perceived noise was bilateral and lasted only during the act of phonation. The patient found the phenomenon to be unpleasant. An external sound did not induce the tinnitus, and she had no spontaneous tinnitus. On questioning, she admitted to hearing loss. She had no history of ear disease.

No abnormality was evident on otolaryngologic examination apart from sensorineural deafness. An audiogram showed a flat bilateral sensorineural loss at the 50-dB level. Diagnostic auditory brainstem response testing elicited clear, repeatable responses bilaterally. The wave I–V interpeak interval was normal bilaterally. Magnetic resonance imaging (MRI) showed widespread foci of high signals on T_2 in deep white matter, consistent with ischemic changes. The posterior fossa and cerebellopontine angles were normal.

Patient 2

An 82-year-old man presented with a 2-year complaint of "horrible" buzzing in the right ear on talking. He said that he had no hearing difficulties, although he had tinnitus in both ears for many years and was habituated to it. The tinnitus caused by talking was of a different nature and was distressing to him. He said that the buzzing drowned out the sound of his own voice and lasted only when he spoke. Singing had the same effect. His medical history included right hemiparesis 13 years previously, from which he experienced good recovery. Hypertension was discovered at that time, and he had been taking antihypertensive medication ever since. He was seen again in the hospital 8 years previously for Parkinson's disease, and a computed tomography scan showed two areas of infarct: one in the left internal capsule and another in the left parietal region. He was given a prescription of selegiline and levodopa-carbidopa, which partially controlled his symptoms. He also had

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mild renal failure, generalized osteoarthritis, and diverticular disease of the colon.

Examination showed his ears to be normal. An audiogram showed his hearing to be within normal range in lower frequencies, with a moderate drop at 4 kHz and 8 kHz. Diagnostic auditory brainstem response testing showed a delay in wave I, but the interwave latencies were normal. MRI showed changes indicative of generalized cerebral atrophy. Examination revealed high-signal-intensity areas in a periventricular distribution consistent with small-vessel ischemic changes. One focal infarct involved the left occipital lobe, and another focal infarct was seen in the left cerebellar hemisphere. No expansion of the internal auditory meatus and cerebellopontine angle was found. Clinical evaluation ascertained that the tinnitus was not induced by orofacial, head, neck, limb, or eye movements in either of these patients.

DISCUSSION

The exact mechanism leading to tinnitus perception is yet to be defined. An increase in cochlear activity is unlikely to be a major factor. No increase is evident in nerve VIII potential in tinnitus patients [5], and increased activity in the auditory pathway has not been proven [9]. Division of the auditory nerve does not consistently abolish tinnitus. Increased activity of the outer hair cells has not been demonstrated consistently [10]. Although tinnitus mostly accompanies a hearing loss, it may occur without hearing loss.

In their fMRI study of normal hearing subjects with tinnitus, Melcher et al. [9] found reduced activity in the inferior colliculus contralateral to the tinnitus side in response to bilateral broadband sound stimuli. This effect was abolished by intravenous lidocaine, demonstrating an increased baseline activity in the contralateral inferior colliculus in tinnitus patients.

Moller et al. [5] suggested that the extralemniscal auditory pathway that connects with the association cortex (rather than primary auditory cortex) is responsible for tinnitus. This is partly supported by the work of Andresson et al. [11], who demonstrated increased activity in the primary and secondary cortex, among other areas, in a positron emission tomography study of patients with tinnitus.

Tinnitus can be induced or modified by a variety of somatosensory stimuli, including alteration of gaze. Some of the activities that have been held to modify or induce nystagmus are jaw clenching, jaw movement against resistance, neck movements, movements of limbs, pressure on the temple, pressure on the temporomandibular joint, touch on the dorsum of the hand, pinching together of the thumb and index fingers, and electrical stimulation of the median nerve [1,3,6,8].

Levine [3] asked all 70 patients presenting at the tinnitus clinic to perform nine sets of head and neck contractions, and the final 25 patients were asked to perform seven additional movements entailing contraction of extremities to note the effect on the tinnitus. He found that two-thirds of patients presenting with tinnitus could modulate their tinnitus with somatic activity, and he suggested that somatic modulation might be a fundamental attribute of tinnitus on a par with auditory and affective attributes of tinnitus.

In his study of brain activity mapping in patients who could modulate tinnitus by orofacial movement, Salvi et al. [2] concluded that tinnitus has a cortical origin and that cortical plasticity may contribute to certain forms of tinnitus.

Most cases of gaze-evoked tinnitus follow acoustic neuroma surgery that results in deafferentation. Gaze-evoked tinnitus has also been reported less frequently after sudden sensorineural hearing loss [7]. It has further been reported in association with metastasis of cutaneous malignant melanoma in the right cavernous sinus and multiple lesions in very superficial locations with respect to brain parenchyma [12].

Both our patients had hearing loss and central nervous system ischemic changes. The ischemic change in the white matter seen in the first case is widely present in people of similar ages but could be significant just the same. Neither of the two patients had an intracranial neoplasm or a history of intracranial surgery. The cases described by Levine [3] and Salvi et al. [2] are similar, but these authors did not describe intracranial ischemic changes. Cross-modal plasticity may explain the tinnitus in the two patients discussed here.

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REFERENCES

1. Cacace AT. Expanding the biological basis of tinnitus: Crossmodal origins and the role of neuroplasticity. *Hear Res* 175:112–132, 2003.
2. Salvi RJ, Lockwood AH, Coad ML, et al. Positron emission tomography identifies neuroanatomical sites associated with tinnitus modified by oral-facial and eye movements. In J Hazell (ed), *Proceedings of the Sixth International Tinnitus Seminar* 1999:175–180.
3. Levine RA. Somatic modulation appears to be a fundamental attribute to tinnitus. In J Hazell (ed), *Proceedings*

of the Sixth International Tinnitus Seminar. London: Tinnitus and Hyperacusis Centre, 1999:193–197.

4. Cacace AT, Cousins JP, Parnes SM, et al. Cutaneous-evoked tinnitus. *Audiol Neurootol* 4:247–257, 1999.
5. Moller AR, Moller MB, Yokota M. Some forms of tinnitus may involve the extralemniscal auditory pathway. *Laryngoscope* 102:1165–1171, 1992.
6. Rubinstein B, Axelsson A, Carlsson GC. Prevalence of signs and symptoms of craniomandibular disorders in tinnitus patients. *J Craniomandib Dis* 4:186–192, 1990.
7. Biggs NDW, Ramsden RT. Gaze-evoked tinnitus following acoustic neuroma resection: A de-afferentation plasticity phenomenon? *Clin Otolaryngol* 27:338–343, 2002.
8. Pinchoff RJ, Burkard RF, Salvi RJ, et al. Modulation of tinnitus by voluntary jaw movements. *Am J Otol* 19:785–789, 1998.
9. Melchor JR, Sigalovsky I, Levine RA. Tinnitus-related fMRI activation patterns in human auditory nuclei. In J Hazell (ed), *Proceedings of the Sixth International Tinnitus Seminar*. London: Tinnitus and Hyperacusis Centre, 1999:166–170.
10. Henry JA, Flick CL, Gilbert AM, et al. Masking curves and otoacoustic emissions in subjects with and without tinnitus. In J Hazell (ed), *Proceedings of the Sixth International Tinnitus Seminar*. London: Tinnitus and Hyperacusis Centre, 1999:345–349.
11. Andersson G, Lyttkens L, Hirvela C, et al. Regional cerebral blood flow during tinnitus: A PET case study with lidocaine and auditory stimulation. *Acta Otolaryngol* 120:967–972, 2000.
12. Caraceni A, Scolari S, Gallino G, Simonetti F. Meningeal metastases from malignant melanoma presenting with gaze-evoked tinnitus. *Neurology* 53:2207, 1999.