

Some Forms of Tinnitus May Involve the Extralemniscal Auditory Pathway

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It has previously been shown that the click-evoked responses recorded from the intracranial portion of the eighth nerve in patients with incapacitating tinnitus are not abnormal, nor is the latency of peak III of the click-evoked brainstem auditory-evoked potentials significantly altered; however, the latency of peak V is slightly (but significantly) shortened in comparison to that of patients with the same degree of hearing loss but no tinnitus. In this study the hypothesis that the extralemniscal auditory system is involved in the generation of tinnitus is tested. We made use of the fact that neurons of the extralemniscal auditory system also receive input from the somatosensory system, and that stimulation of the somatosensory system can influence the processing of auditory information in the extralemniscal system. In 4 of 26 patients with mild-to-severe tinnitus whose median nerve was stimulated electrically, the tinnitus increased noticeably during stimulation, in 6 the intensity of the tinnitus decreased noticeably, and in the remaining 16 there was no noticeable change in the tinnitus. In some of the patients the character of the tinnitus changed in a complex way. There were no significant differences in hearing thresholds in these three groups of patients. Electrical stimulation of the median nerve in 12 individuals with normal hearing who did not have tinnitus either had no effect on the loudness of sounds or it caused a slight increase in the loudness.

INTRODUCTION

It has been shown that certain forms of tinnitus can be relieved by microvascular decompression (MVD) of the eighth nerve.¹⁻³ It was therefore concluded that certain forms of severe (incapacitating) tinnitus may belong to a family of disorders that are caused by vascular irritation of a cranial nerve (CN) (such as trigeminal neuralgia [TN], CN V; hemifacial

spasm [HFS], CN VII; disabling positional vertigo [DPV], CN VIII; and glossopharyngeal neuralgia [GPN], CN IX). It has been hypothesized that the symptoms and signs of at least one such disorder (HFS) are the result of hyperactivity of more central structures (presumably the facial motonucleus).⁴ This hypothesis was supported by results from intraoperative neurophysiological recordings in patients who had been operated on to relieve HFS using the MVD technique, which showed that the anatomical site of the physiological abnormality in patients with HFS is most likely located in the facial motonucleus, and that the signs of HFS are a result of hyperactivity of the facial motonucleus.⁵⁻¹¹ The hypothesis that this hyperactivity of the facial motonucleus is brought about by a mechanism similar to the kindling phenomenon has been presented.^{5,7,8} (The kindling phenomenon is associated with the experimental findings of Goddard,¹² who showed that daily electrical stimulation of the amygdala in rats for several weeks results in epileptic seizures (*cf.* Wada¹³). In HFS, it is believed that such hyperactivity in the facial motoneurons develops over a period of time as a result of neural activity in the facial nerve that is generated by irritation of the facial nerve from the close contact with a blood vessel.^{8,9} We have, in fact, shown by the results of studies in animals that it is possible to produce signs similar to those seen in patients with HFS by stimulating the facial nerve electrically over a period of time.^{14,15}

The results of recent animal studies by Gerken, *et al.*¹⁶ support the assumption that injury to (or stimulation of) the peripheral portion of the auditory system can result in hyperactivity in more centrally located structures. These investigators demonstrated that overstimulation of or injury to the ear may cause hyperactivity in auditory nuclei. It is generally assumed that such hyperactivity develops over a period of time and is not present immediately when the injury or overstimulation occurs.

There is evidence, however, that some forms of tinnitus that are less severe may in fact be the direct result of neural activity in the auditory nerve that is caused by irritation or injury to specific structures in the ear or the auditory nerve.¹⁷ Thus, acute injury to

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the eighth nerve from surgical manipulation, such as may occur in operations to remove acoustic tumors or in MVD procedures to relieve vascular compression of lower cranial nerves, may result in tinnitus that is noted immediately after the operation.

If severe tinnitus, at least in some cases, is a result of hyperactivity in the ascending auditory pathway, the structures involved would likely be located between the cochlear nucleus and the inferior colliculus. However, we have reported on the compound action potentials (CAP) that can be recorded from the exposed intracranial portion of the eighth nerve: in 19 patients with intractable tinnitus who were operated on by MVD² to decompress the intracranial portion of the eighth nerve to relieve incapacitating tinnitus, the CAPs were not noticeably different from the CAPs that were recorded from the exposed eighth nerve in patients without tinnitus but with the same degree of hearing loss.¹⁸⁻²⁰ Also, the latency of peak III of the brainstem auditory-evoked potentials (BAEP) in these 19 patients with tinnitus was not significantly different from that of peak III of the BAEP in patients with similar hearing loss but no tinnitus (peak III of the BAEP may be regarded to be generated by the cochlear nucleus^{21,22}). However, the latency of peak V of the BAEP in the patients with tinnitus was slightly (but statistically significant) shorter than that of peak V in the BAEP in patients with similar hearing loss but no tinnitus.¹⁸⁻²⁰ The sharp vertex-positive tip of peak V is assumed to be generated by the lateral lemniscus.²³

These results¹⁸⁻²⁰ indicate that the location of the physiological abnormality that resulted in tinnitus in these patients was not on the eighth nerve or in the cochlear nucleus but might, as shown by the slight (but statistically significant) shortening of the latency of peak V of the BAEP, be between the cochlear nucleus and the inferior colliculus. Because of the small number of patients (19) studied and the small difference in the latency of peak V between patients with tinnitus and those without tinnitus who had the same degree of hearing loss, these data must be interpreted cautiously. Therefore, the classical lemniscal system may not be involved in this form of tinnitus, and we have suggested that the extralemniscal auditory system may be involved in generating the strong sound sensations that these patients experienced.^{18,20}

The extralemniscal portion of the ascending auditory pathway transmits auditory information to higher brain centers in conjunction (parallel) with the classical lemniscal system. Whereas the classical lemniscal system projects to the primary auditory cortical areas, the extralemniscal system projects to association cortical areas. The extralemniscal system has been named the "adjunct system" by investigators who have studied the anatomy and physiology of that system.²⁴ Although some investigators have shown evidence that the extralemniscal system branches off

more peripherally,²⁴ this system is believed to branch off from the classical ascending lemniscal system at the level of the inferior colliculus²⁵ (Fig. 1). The lemniscal system of the auditory pathway specifically conducts and processes timing and frequency information in the sounds that reach the ear, but the neurons of the extralemniscal system do not seem to conduct such information.

Many neurons of the extralemniscal system receive input from other sensory modalities such as the somatosensory system,²⁵ and it is known from animal experiments that the responses of neurons in the auditory extralemniscal system to auditory stimulation can be modulated by stimulating another system such as the somatosensory system.^{26,27} The cortical areas to which the extralemniscal system projects are therefore also known as the polysensory areas.

Since auditory and somatosensory information interacts in the extralemniscal system (but not in the lemniscal system), it was assumed that it would be possible to find out if the extralemniscal system was involved in tinnitus by stimulating the somatosensory system in such patients. Therefore, in the present study, we investigated whether electrical stimulation of the median nerve in patients with varying degrees of tinnitus could alter the tinnitus. The results were compared with those obtained from similar electrical stimulations in individuals who did not have tinnitus and to whom different types of sounds were presented. The purpose of the latter part of the study was to ascertain if the extralemniscal system might play a role in the perception of normal sounds.

Patients and Test Subjects

The patients were those who came to our clinic for initial evaluation, treatment, and postoperative visits during a period of 8 months. Standard audiological testing, which included pure-tone audiometry, speech discrimination testing using recorded speech material, and acoustic middle ear reflex testing, was performed on each patient. For the study, patients were divided into three groups based on the degree of tinnitus. The tinnitus of patients in whom daily activities or sleep were not affected was categorized as mild; the tinnitus of patients in whom daily activities were affected, but who were generally able to concentrate and usually able to sleep was characterized as moderate; the tinnitus of patients in whom both daily activities and sleep were affected was characterized as severe. In the group of 26 patients who underwent electrical stimulation of the median nerve, the tinnitus was severe in 14, moderate in 5, and mild in 7. Five of the patients in this group had either previously undergone MVD to relieve their tinnitus or did so during the time of this study.

The experimental subjects were employees and students. All of the subjects who participated in these tests had normal hearing; none of them had prior

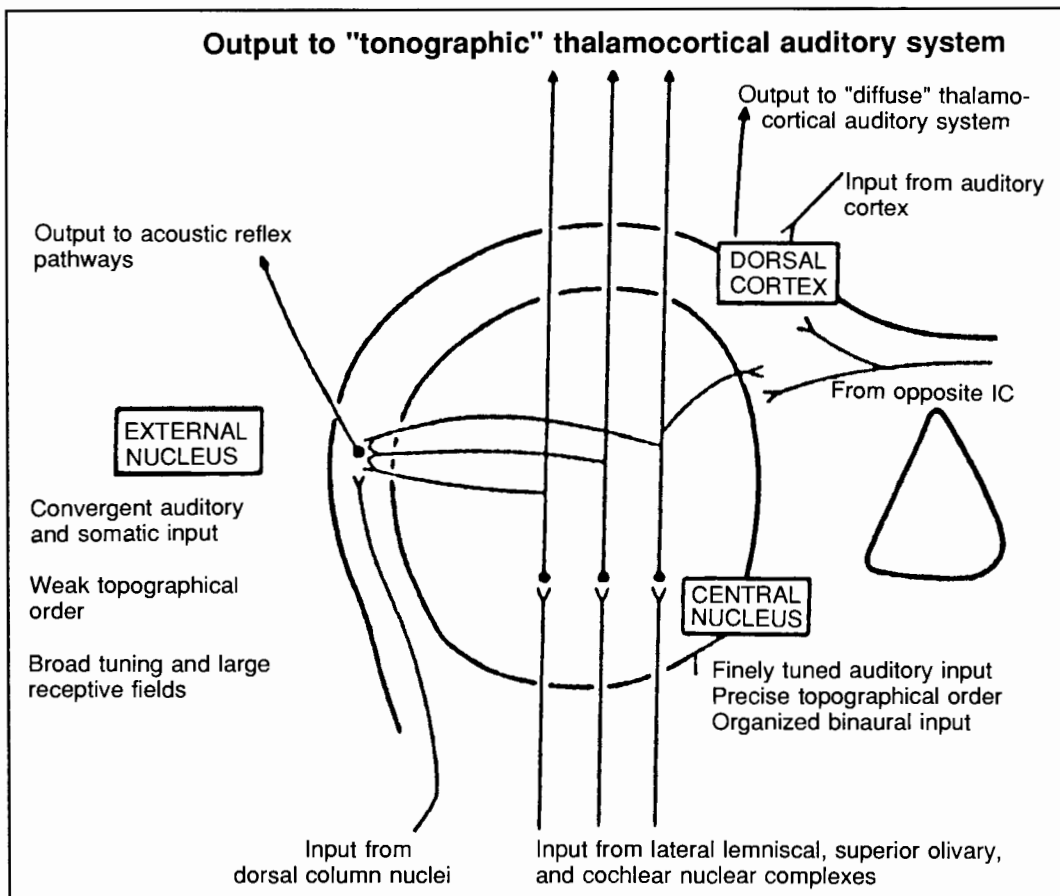


Fig. 1. Schematic of the connections of the three divisions of the inferior colliculus showing how the extralemnisal system is believed to branch off from the classical ascending lemniscal system. (From Aitkin, L.: *The Auditory Midbrain, Structure, and Function in the Central Auditory Pathway*. Humana Press, Clifton, NJ, 1986. Reprinted with permission.)

experience in participating in psychoacoustic testing. The testing procedure was approved by the Biomedical Institutional Review Board of the University of Pittsburgh, and all patients gave informed consent.

METHODS

The median nerve was stimulated by applying rectangular electrical impulses of 100- μ s duration through miniature electrocardiographic (EKG) pads at a rate between 1 and 10 pulses per second (pps) (Type SD9 stimulator, Grass Instrument Co., Quincy, Mass.). The stimulation was first applied at 2 pps at a level below threshold, and then the stimulus strength was increased until a strong tingling sensation, but no pain, was felt by the individual.

The patients, while supine in a sound-insulated room, were asked to listen carefully to their tinnitus. The electrical stimulation was then applied for 10 to 15 seconds and the patients were asked to report any change in the quality or intensity of their tinnitus. The patients were not given any indication of what changes to expect. This procedure was repeated 3 to 5 times and, if inconsistent results were obtained, repeated further. The median nerve on the side of the patient on which the tinnitus was worse was stimulated first; the median nerve on the other side was stimulated second.

The median nerve of individuals who did not have tinnitus was stimulated using the same technique. Sounds were presented monaurally using miniature stereo ear-

phones (Realistic microstereo earphones, Radio Shack, Division of Tandy Corporation, Ft. Worth, Tex.). These sounds were 1. trains of click sounds delivered at a rate of 40 pps by an audiostimulator (Type S10 CTCMA, Grass Instrument Co.), or 2. broadband noise, generated by the same audiostimulator. The stimulus intensity was calibrated by a 1/4-inch condenser microphone (Type 4135, Bruel & Kjaer, Naerum, Denmark). The sound was presented for 20 to 30 seconds before the ipsilateral and then the contralateral median nerve was stimulated for a period of 10 to 15 seconds in the way described previously. The subjects were asked to report any change in the character or loudness of the sound. If they found the electrical stimulation caused the sound to seem louder (or less loud), the intensity of the stimulus sound was varied in 2-dB steps until it matched the intensity of the sound as they perceived it during the electrical stimulation. Changes that were noticeable but less than 2 dB were disregarded. All of the tests were performed with the subjects seated in a sound-insulated room.

RESULTS

All Patients With Tinnitus

Four of 26 patients reported that the intensity of their tinnitus increased as a result of electrical stimulation of the ipsilateral median nerve. Of the 4 patients who experienced an increase in tinnitus, 2 had severe tinnitus, 1 had moderate tinnitus, and 1 had mild tinnitus. Six of 26 patients tested reported a distinct decrease in their tinnitus when the ip-

silateral median nerve was stimulated. Of these 6 patients, 4 had severe tinnitus, 1 had moderate tinnitus, and 1 had mild tinnitus. One of these patients who experienced a decrease in the intensity of tinnitus had been previously operated on to remove an acoustic tumor and was deaf in the ear that had been operated on; however, despite the deafness, the tinnitus was located in that same ear. Three of 5 patients who experienced a decrease in the intensity of tinnitus when the median nerve was stimulated electrically also experienced a distinct change in the character of the tinnitus.

A mean audiogram was calculated for each of the three groups of patients (increase in tinnitus, decrease in tinnitus, no change in tinnitus as a result of median nerve stimulation); these mean audiograms are shown in Figure 2. Although the group that reported no effect from electrical stimulation of the median nerve had lower hearing thresholds at low frequencies, Wilcoxon's test showed no statistically significant differences between the hearing thresholds at any frequency in these three groups. The average speech discrimination score of the patients who experienced an increase in tinnitus when their median nerve was stimulated was 77.0 ± 8.25 . In those who experienced a decrease in tinnitus it was 72.8 ± 28.34 , and in those who did not experience any effect from median nerve stimulation it was 89.1 ± 15.64 . There were no statistically significant differences between these values either. (The patient who was operated on to remove an acoustic tumor was deaf in the ear that was operated on and that had tinnitus; another patient was also deaf in the ear with tinnitus. These two patients were excluded when calculating the average audiograms shown in Figure 2, and their hearing thresholds and speech discrimination values were not included in the statistical analysis.)

Patients Who Underwent MVD to Relieve Tinnitus

In the 5 patients with severe tinnitus who underwent MVD specifically to treat their tinnitus, electrical stimulation of the median nerve resulted in more complex effects than were noted in any of the other patients. In 2 of these 5 patients there was a distinct decrease in tinnitus as a result of stimulation of the median nerve, in 1 there was a questionable decrease, in 1 there was no change at all, and in 1 there was a considerable worsening of tinnitus.

Patient 1. This patient had a vascular compression of the eighth nerve on the right side where the tinnitus was strongest, which was confirmed intraoperatively. Electrical stimulation of the median nerve resulted in complex effects on tinnitus. Before the operation the patient reported that her tinnitus changed "dimension" and became much less unpleasant as a result of electrical stimulation of the median nerve on the right side. The most effective stimulus intensity was just below the threshold of pain; at pain threshold the stimulation was less effective. Upon

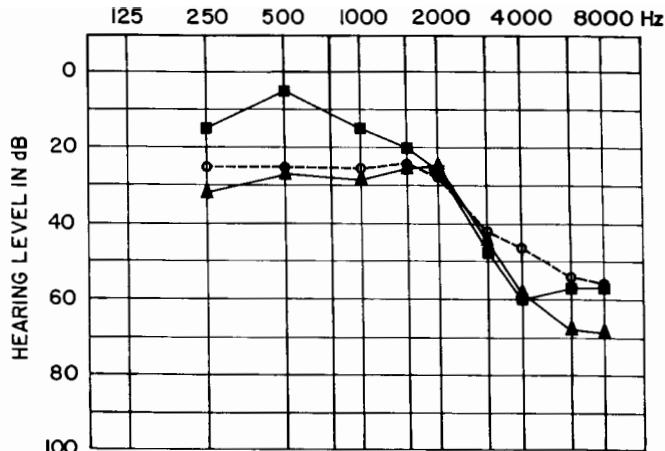


Fig. 2. A mean audiogram for each of the three groups of patients: those who experienced an increase in tinnitus (filled squares), those who experienced a decrease in tinnitus (filled triangles), and those with no change in tinnitus (open circles) as a result of median nerve stimulation.

stimulation of the left median nerve, the tinnitus in the right ear increased, but the milder tinnitus in the left ear decreased.

One month after MVD on the right side, this patient reported that the tinnitus was much improved. At that time, electrical stimulation of the right median nerve again removed some components of the tinnitus. At one point during testing she asked that the ceiling light in the testing room be turned off so that she could better concentrate on her remaining tinnitus. In fact, the tinnitus became undetectable during electrical stimulation and for 1 to 2 minutes after electrical stimulation had ended.

By the time this patient was tested 3 months after operation, the tinnitus had further improved. Electrical stimulation of the right median nerve at a rate of 2 pps had no effect on the remaining tinnitus, but when the right median nerve was stimulated at rates of 4 pps and 10 pps the tinnitus became more severe. And when the median nerve was stimulated at a rate of 15 pps, the tinnitus increased further, although after stimulation ended the tinnitus became much less than it was before stimulation had begun.

This patient also noted that she had had difficulty in picking up small objects with the fingers of her right hand before the operation, but after the operation this problem did not exist. Finally, she noted that she could hear more clearly after the operation.

Patient 2. Another patient had previously been operated on to relieve tinnitus on the left side. The operation resulted in a considerable decrease in tinnitus, but the tinnitus returned to its previous level after an accident. Electrical stimulation of this patient's left median nerve affected her tinnitus in a way similar to that noted by patient 1. Patient 2 described

the change being "as if something had come between me and the tinnitus; the sound got muffled." Some components of this patient's tinnitus disappeared when her median nerve was being stimulated. Stimulation of this patient's right median nerve was equally or more effective than stimulation of the left median nerve, and caused the tinnitus to totally disappear for a period of time. In this case, a stimulus rate of 2 pps was optimal; at a stimulus rate of 6 pps the tinnitus was practically unaffected.

Patient 3. In this patient, who had been operated on to relieve tinnitus and had experienced some improvement, electrical stimulation of the median nerve on the operated side had little and uncertain effect on tinnitus. This patient had taken diazepam immediately before the test was performed.

Patient 4. This patient, who was tested before a MVD operation for Meniere's disease and who had severe tinnitus, reported no change in tinnitus as a result of electrical stimulation of the median nerve. However, this patient did report considerable relief from tinnitus when she was examined 3 months after the operation.

Patient 5. This patient, who also had previously undergone an MVD operation to relieve tinnitus, was tested 15 months after the first operation to relieve tinnitus. He suffered a surgically induced injury to the auditory nerve that caused hearing loss and increased tinnitus. Electrical stimulation of either the left or the right median nerve resulted in an increase in tinnitus. This patient also reported that tinnitus increased when he moved his left arm. Stimulation of the median nerve on the left side caused tinnitus to increase, and it took considerable time for the tinnitus to decrease to the value it had before the stimulation.

Other Patients With Tinnitus

Another patient with severe tinnitus had acquired it in connection with a neck injury and had not been operated on. She experienced a 50% decrease in the severity of tinnitus following electrical stimulation of the median nerve at an optimal stimulus rate of 2 pps, but also noted a distinct increase in the pitch of tinnitus. Contralateral stimulation was just as effective as ipsilateral stimulation.

In another patient, tinnitus was related to an acoustic tumor on the right side. After an operation to remove the tumor, the patient experienced worsening of the tinnitus on the operated side. This patient reported a substantial decrease in tinnitus following electrical stimulation of the right median nerve. The pitch of the tinnitus was lowered and the patient reported that it felt "like there was something in between me and the tinnitus" when his right median nerve was being stimulated electrically. Stimulating the left median nerve had much less effect than stimulating the right median nerve. A stimulus rate of 2 pps was found to be optimal, and the effect was considera-

TABLE I.
Change in Perceived Loudness (dB) From Electrical Stimulation of the Median Nerve.*

40 pps Clicks, 2 pps Median Nerve Stimulation								
Ipsilateral Stimulation				Contralateral Stimulation				
75 dB	85 dB	95 dB	105 dB	75 dB	85 dB	95 dB	105 dB	
0	0	2	2	0	0	2	2	
2	2	2	2	2	4	2	2	
0	0	0	2	0	0	-2	0	
0	2	2	2	0	2	2	4	
2	2	4	2	0	0	0	0	
4	4	2	4	2	4	4	4	
0	0	0	2	0	0	0	0	
0	0	0	2	0	0	0	0	
2	2	2	2	2	2	2	2	
0	2	4	4	0	2	2	2	
0	0	0	0	0	0	0	0	
0	0	2	2	0	4	2	4	
Mean	0.833	1.167	1.667	2.167	0.500	1.500	1.167	1.667
SD	1.337	1.337	1.435	1.030	0.905	1.732	1.568	1.670

Noise Stimulation, 2 pps Median Nerve Stimulation								
Ipsilateral Stimulation				Contralateral Stimulation				
60 dB	70 dB	80 dB	90 dB	60 dB	70 dB	80 dB	90 dB	
0	0	-2	0	0	0	0	0	
0	0	0	0	0	0	0	0	
0	0	0	0	0	0	0	0	
0	0	2	2	0	0	0	0	
0	0	0	0	0	0	0	0	
2	2	2	4	2	2	2	2	
0	0	0	0	0	0	0	0	
0	0	0	0	0	0	0	0	
0	0	0	0	0	0	0	0	
0	0	0	0	0	0	0	0	
0	0	0	0	0	0	0	0	
0	0	0	0	0	0	0	0	
0	0	0	0	0	0	0	0	
0	0	0	0	0	0	0	0	
Mean	0.167	0.167	0.167	0.500	0.167	0.167	0.167	0.167
SD	0.577	0.577	1.030	1.243	0.577	0.577	0.577	0.577

4 pps Median Nerve Stimulation				10 pps Median Nerve Stimulation				
40 pps Clicks		Noise		40 pps Clicks		Noise		
IPSI	CONT	IPSI	CONT	IPSI	CONT	IPSI	CONT	
0	4	-2	0	0	2	0	0	
4	2	0	0	2	2	0	0	
0	0	0	0	0	0	0	0	
4	2	2	2	4	2	2	2	
2	0	0	0	2	0	0	0	
2	4	2	2	4	4	2	2	
0	0	0	0	0	0	0	0	
0	0	0	0	0	0	0	0	
2	0	0	0	2	0	0	0	
4	2	0	0	4	4	0	0	
0	0	0	0	0	0	0	0	
2	4	0	0	4	2	0	0	
Mean	1.667	1.500	0.167	0.333	1.833	1.333	0.333	0.333
SD	1.670	1.732	1.030	0.778	1.801	1.557	0.778	0.778

*Level of 40-pps clicks is given in dB PeSPL (105 dB ~ 68 dB HL); the level of the noise is in dB SPL.

IPSI = ipsilateral; CONT = contralateral.

bly less when a stimulus rate of 4 pps or 1 pps was used. Despite the fact that this patient was deaf on the side of the tinnitus, the tinnitus was worsened by external sounds as well as by the patient's own speech. Electrical stimulation of the right median nerve made the sound-elicited tinnitus return much faster to its baseline value than it did without such stimulation.

Individuals Without Tinnitus

Electrical stimulation of the median nerve in some individuals who did not have tinnitus increased the perceived loudness of some of the sounds that were used for stimulation. This effect was greatest for click sounds presented at the highest intensity (105 dB Pe SPL) (Table I), at which the average increase in perceived loudness corresponded to an increase in the stimulus intensity of $2.167 \text{ dB} \pm 1.030$ when the ipsilateral median nerve was being stimulated. The perceived loudness of clicks of lower intensity and of broadband noise was less affected by median nerve stimulation, and contralateral median nerve stimulation affected the loudness of sounds less than ipsilateral stimulation. Rates of 4 pps and 10 pps of the median nerve stimulation were nearly equally effective in influencing the perceived loudness of clicks and noise (Table I). Two of 12 subjects reported a decrease in the loudness as a result of median nerve stimulation, but only at one stimulus intensity, and some of the subjects did not experience any change in loudness at all. However, several of the subjects reported that the loudness was reduced immediately after the termination of electrical stimulation.

Several of the 12 subjects tested reported that the perception of the sound changed as a result of stimulating the median nerve, not only with regard to intensity but also with regard to the character of the sound. The subjects' judgments of changes in the character of the sound had considerable individual variation, and some of these individuals reported that the sound became muffled or that the pitch increased as a result of median nerve stimulation. One of the individuals tested reported that his perception of the sound's location moved from lateral to the middle of the head with stimulation of the median nerve.

DISCUSSION

The results of this study show that the perception of loudness of certain loud sounds, as well as certain forms of tinnitus, can be manipulated by stimulation of the somatosensory system. However, one important difference between the effect of electrical stimulation of the median nerve in patients with tinnitus versus individuals without tinnitus was noted: in some patients with tinnitus, electrical stimulation of the median nerve caused a consistent decrease in the loudness of the tinnitus, but the only consistent effect (if any) of similar stimulation on subjects' perceptions of sounds was an increase in the perceived loudness. This indicates that there may be a difference between

neural activity that gives rise to tinnitus and neural activity that is evoked by sound. Also, it is notable that two of the patients used the same expression to describe the change in their perception of tinnitus ("something came in between me and the tinnitus" and "that a component of the tinnitus disappeared"), despite the fact that patients were only instructed to report any change in tinnitus and that they were not given any suggestions regarding what to expect.

These findings are interpreted to indicate that the perception of loudness of certain forms of tinnitus, as well as that of certain sounds, involves not only the classical (lemniscal) auditory pathways, but also the extralemniscal auditory system. This, however, does not mean that the anatomical site of the physiological abnormality that gives rise to tinnitus in these patients is necessarily located within the extralemniscal system, but only that the extralemniscal system is involved in generating tinnitus.

The results of this study do not contradict the hypothesis that close contact between the auditory nerve and a blood vessel may give rise to abnormal neural activity¹⁷ and that this can lead to the development, in some central neural structures, of hyperactivity that can cause incapacitating tinnitus.

The finding in one patient who underwent MVD to relieve tinnitus that her preoperative difficulty in picking up small objects with the fingers on the side of the tinnitus improved after the operation is another indication that the somatosensory system is affected by vascular compression of the auditory nerve. The most likely mechanism for this effect is interaction between auditory and somatosensory information; the most likely location for this to occur would be in the extralemniscal somatosensory pathways.

The fact that stimulation of the somatosensory system can increase the loudness of tinnitus indicates that somatic pain may aggravate a patient's tinnitus. The finding that the perception of certain types of sounds in individuals who do not have tinnitus is affected by stimulation of the somatosensory system may be taken to indicate that the perception of intense, and perhaps unpleasant, sounds may involve the extralemniscal system.

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