

# Electrical Suppression of Tinnitus

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## HISTORY

Tinnitus is sufficiently common and distressing that it has been described in medical texts for more than three millennia. According to Feldmann, the first mention of tinnitus in archival materials derived from Egyptian papyri dates to the second or third century BCE.<sup>1,2</sup> It is not unexpected that efforts to treat tinnitus would be comparably ancient and would attempt to use all available technologies of a given era. Thus, the topical herbal treatments of ancient Egypt made way for the application of the battery at the dawn of the nineteenth century.<sup>3</sup> Volta first determined that current flowed from his battery by touching it to his tongue; it is not surprising

that he subsequently applied it to his own external auditory canals.<sup>3</sup> The 50-volt shock he applied to himself reportedly knocked him down, plausibly owing to vestibular stimulation, and produced a loud and persistent tinnitus-like auditory percept. Volta's experiment arguably heralded the twin birth of cochlear implantation and electrical manipulation of tinnitus, but, apparently, he chose not to repeat the experience himself.

Within 1 year of Volta's publication describing the battery, the first reliable source of ongoing electrical current, a subsequent paper detailed a complex apparatus for electrical stimulation of the external ears (Figure 24-1).<sup>4</sup> Throughout the nineteenth century, various authors described multiple methods for such

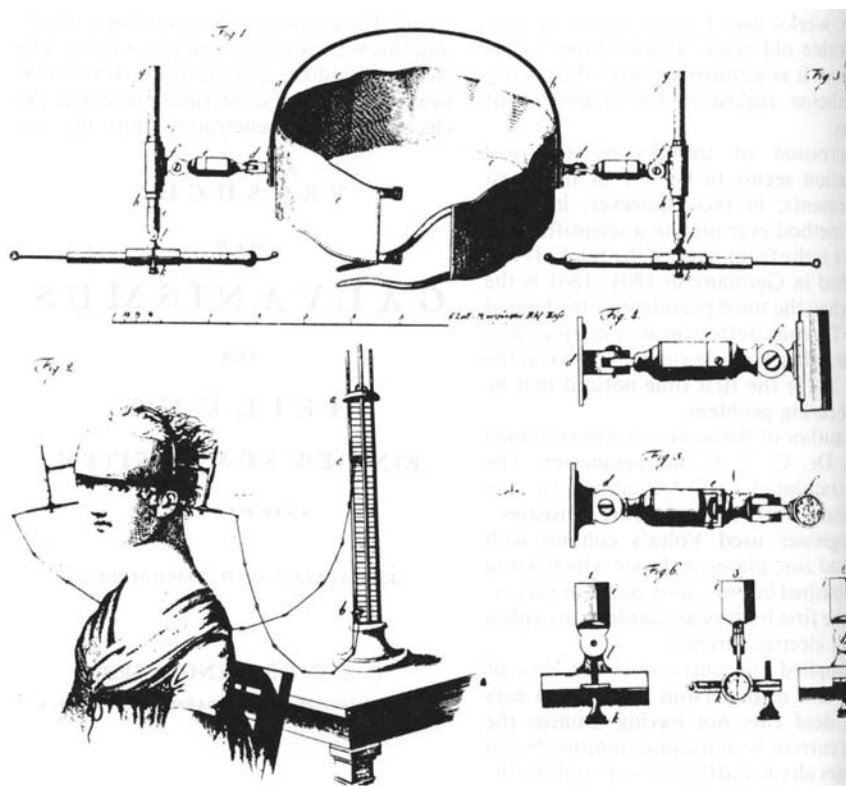


FIGURE 24-1. Apparatus for electrical stimulation of external auditory canals for treatment of deafness and tinnitus. Reproduced with permission from Grapengiesser CJC.<sup>4</sup>

stimulation as a treatment for deafness and tinnitus. Stimulation sites included the external auditory canal, auricle, zygoma, tragus, mastoid, and eustachian tube orifice. Electrodes included balls, wicks, and, in one case, needle electrodes applied simultaneously to the eustachian tube transnasally and the promontory transtympanically. Although, in the first half of the nineteenth century, all such stimulation used direct current, known as galvanization, the invention of the induction coil spawned the use of alternating current, or faradization. Such treatments were apparently so widespread that an entire book by Brenner was devoted to electrical stimulation of the ears: *Investigations and Observations on the Effect of Electric Current on the Hearing Organ in the Healthy and Ill*.<sup>5</sup> The subtitle of the book, *Attempts to Create a Rational Electro-Otology*, suggests a response to the appropriate skepticism with which claims of cures of deafness and tinnitus were met. By the early twentieth century, therapeutic electrical stimulation of the ears had fallen out of favor.

Despite the uncontrolled nature of the experiments reported during the nineteenth century, multiple authors noted a number of consistent observations. They frequently observed that anodal direct current applied to the ipsilateral mastoid or zygoma could temporarily suppress or eliminate tinnitus in some patients, whereas cathodal stimulation more commonly caused auditory percepts and an increased intensity of tinnitus. In this century, Hatton and colleagues replicated these findings using electrodes placed on the zygomatic arches bilaterally.<sup>6</sup> Similar findings were also noted in multiple publications from Bordeaux, France.<sup>7-10</sup> The latter group applied electrical stimuli via a transtympanic electrode to the round window and promontory.

The 200-year-old finding that anodal direct current can suppress tinnitus in some cases has, unfortunately, not resulted in a useful clinical intervention. This is due to limitations in the ability to stimulate biologic tissue with direct current chronically. Such stimulation leads to tissue necrosis from multiple mechanisms, including hydrolysis and other toxic electrochemical reactions.<sup>11</sup> Only specific frequencies and intensities of alternating current, such as those used in cochlear implants, are safe for chronic stimulation.

With the increasing use of cochlear implantation as a treatment for profound hearing impairment, a substantial literature documented that tinnitus suppression is a common beneficial side effect of this modern form of electrical stimulation.<sup>12-20</sup> Although

the reported efficacy rate varies from 28 to 79%, it is quite unusual, even in a large cochlear implant center, to have implant recipients complain of disabling tinnitus. This is in contrast to individuals with moderate to severe hearing impairment using hearing aids, for whom marked and bothersome tinnitus is quite common. One might expect that patients with implants would suffer similarly if they were deprived of their speech processors for a period of time.

The promising early results with anodic direct current stimuli, the knowledge that direct current cannot be applied continuously without causing tissue injury, and the success with cochlear implants have led to numerous studies of alternating current stimulation for suppression of tinnitus.<sup>21-27</sup> These studies have used external and middle ear electrodes and a variety of stimulus waveforms and again suggest that in some patients, tinnitus can be suppressed and occasionally eliminated. The results of these studies are variable and confusing, however, and none of them were placebo controlled, limiting the conclusions that can legitimately be drawn. A tinnitus suppression device, the Audimax Theraband, initially produced considerable enthusiasm<sup>28</sup> but was relegated to historical interest by a carefully designed, double-blind, placebo-controlled trial.<sup>29</sup> It is of note, however, that this device produced a verifiable, repeatable, and clinically relevant tinnitus suppression in one subject using electrodes located on the mastoid.

## PHYSIOLOGIC MECHANISMS

As described in the previous section, a wide variety of stimulus waveforms, electrode locations, and patients with tinnitus have been tested in an empiric and uncontrolled manner. Our current understanding of tinnitus pathophysiology is not much more advanced than it was in Volta's era, and well-executed empiric studies are still important and potentially powerful tools. It is, however, important to understand the possible mechanisms by which electrical stimulation can suppress tinnitus. It should be noted that although all of the mechanisms to be discussed presume a peripheral cause of tinnitus, none of them exclude the possibility of secondary central dysfunction, such as an increase in central auditory "gain" in response to loss of peripheral input. Many recent data point to such central dysfunction as an important physiologic correlate of tinnitus.<sup>30-34</sup>

An electrical stimulus to a normally functioning cochlea can evoke a variety of possible responses, including electrophonic activation of the basilar membrane, direct polarization of hair cells with modulation of transmitter release, and direct polarization of spiral ganglion cells with modulation of spontaneous firing or induction of spike activity.<sup>35</sup> Thus, if tinnitus were due to some abnormal increase in peripheral activity, a stimulus that decreased this activity could potentially suppress tinnitus without producing audible percepts. Alternatively, a stimulus that increased peripheral activity could potentially “mask” the tinnitus but would presumably also be audible, offering little advantage over acoustic masking.

Because tinnitus is most commonly associated with sensorineural hearing loss, the most plausible hypothesis is that tinnitus is due, at least initially, to a pathologic decrease in peripheral spontaneous activity.<sup>36,37</sup> If this is the case, an electrical stimulus could potentially suppress tinnitus without producing audible percepts through the restoration of normal levels of peripheral spontaneous activity. This intriguing concept is explored further in this chapter.

## **SITE OF EXCITATION**

It is clear from work with galvanic stimulation of the vestibular system that small (~ 1 mA) currents applied to the external ear can excite the labyrinth.<sup>38–40</sup> Such stimuli can, however, also excite the somatosensory system. Stimuli on the zygoma will evoke trigeminal activity, whereas those posterior to the auricle will stimulate the cervical roots. Recent clinical work documents craniocervical somatosensory modulation of tinnitus,<sup>41</sup> and neurophysiological studies have documented the mechanisms by which such modulation can occur.<sup>42</sup> It seems likely that at least some of the electrical stimulations in studies discussed earlier, particularly those that demonstrate differences between anodal and cathodal stimuli applied to the skin around the ear, achieved their effects through modulating the somatosensory system and its connections to the cochlear nuclei. It is known that anodal electrical stimulation of fiber terminals hyperpolarizes the terminal but depolarizes the fiber more centrally. This would lead to increased excitability of a somatosensory axon<sup>43</sup> and possible tinnitus suppression via somatosensory modulation. A similar mechanism would lead to decreased excitability with cathodal stimuli.

These effects are precisely the opposite of what is seen in auditory nerve fibers with intracochlear electrical stimulation.<sup>44</sup>

The previous discussion indicates that “electrical stimulation of the ear” implies only inner ear stimulation when the electrodes are intracochlear, and thresholds for auditory nerve fibers are typically well below those of other cranial nerves. Electrical stimulation in the middle ear allows the potential for stimulation of Jacobson’s nerve or of somatic afferents from the tympanic membrane or external auditory canal. All have the theoretic potential to modulate tinnitus without altering firing in the auditory nerve directly. Thus, “middle ear stimulation,” including round window and promontory electrode placements, suffers from similar ambiguity as “external ear stimulation” with regard to the neurons affected by the stimulus.

The possibility of multiple sites of excitation with both external and middle ear electrodes may be partly responsible for the variety of outcomes observed both within and across studies of electrical stimulation for tinnitus. This possibility is strengthened by contrasting this extraordinary variability with the far more consistent observation of tinnitus suppression in patients with cochlear implants. Studies in recipients of cochlear implants are complicated only by the variability of tinnitus and its perception, not by ambiguity as to the site of excitation. Although the variability of tinnitus perception is itself a significant experimental problem, psychoacoustic studies demonstrate that it is a tractable one.<sup>45,46</sup>

## **PLACEBO CONTROL**

It is unclear to what degree placebo responses have played a role in earlier trials of electrical stimulation for tinnitus. Some investigators report minimal placebo effects,<sup>22</sup> whereas in other studies, placebo was more frequently effective than the treatment.<sup>29</sup> It should be noted, however, that even in the latter study, placebo effects were rare enough that 75% of subjects had no tinnitus improvement with the placebo or the test condition. Owing to substantial placebo effects in drug trials for tinnitus,<sup>47,48</sup> any potential therapy for tinnitus, including electrical stimulation, should undergo a rigorous placebo-controlled trial prior to widespread clinical use. Depending on the relationship between any percepts produced and the tinnitus

suppression achieved by a given electrical stimulation paradigm, adequate placebo control may or may not be possible. If the stimulation is perceived by the subject, it is necessary to devise some sort of placebo stimulus that is identically perceived but has a different effect or no effect on the tinnitus. If this is not possible, other procedures can be used to minimize the likelihood of placebo responses. The time course of tinnitus suppression and recovery can be monitored and repeatedly measured. Repeatable tinnitus suppression with a consistent time course argues against placebo effects. The stimulus waveform, frequency, and intensity can be modified to search for some “optimal” parameter. If, for example, a “best intensity” exists, this argues against placebo effects.<sup>22</sup> Lastly, false trials without a stimulus presented can be implemented randomly without feedback to the subject.

### “RATIONAL ELECTRO-OTOLOGY?”

As discussed earlier, one possible mechanism for tinnitus associated with sensorineural hearing loss is the loss of normal spontaneous activity that occurs with damage to inner hair cells.<sup>36,37</sup> Normally, the peripheral auditory nerve fibers are spontaneously active in quiet.<sup>49</sup> This spontaneous firing of the auditory nerve is due to continuous, undriven release of neurotransmitter at the inner hair cell synapse.<sup>50</sup> This release process and the resulting firing patterns of the spiral ganglion have been extensively observed, modeled, and analyzed in multiple species. For durations on the order of seconds, transmitter release reflects a Poisson process, a specific pattern of random vesicle release.<sup>51</sup> Refractory properties of the auditory neurons modify the transmitter release process, resulting in spike times reflecting what is known as a renewal process or a Poisson process with dead time. Early theories for the peripheral origin of tinnitus suggest that loss of this normal pattern of spontaneous activity can lead to abnormal central auditory activity perceived as sound.<sup>36,37</sup> In this light, spontaneous activity may be viewed as the “code for silence.” This theory is consistent with the fact that most tinnitus is associated with hearing loss and most hearing loss is associated with loss or alteration of spontaneous activity.<sup>52</sup> It also explains why cochlear nerve section is usually ineffective and, not uncommonly, makes tinnitus worse.<sup>12</sup> Although investigators using animal models associate tinnitus with increased spontaneous activity in the dorsal cochlear nucleus, the presence of lateral

inhibition in the cochlear nuclei could produce this finding in response to decreased spontaneous activity in the periphery.<sup>53,54</sup> The efficacy of acoustic masking with white noise can then be explained through the effect of such noise on the periphery; it evokes spike intervals similar to those of spontaneous activity. The primary difference between spontaneous activity and noise-evoked responses is the across-fiber correlation that acoustic noise produces in neurons innervating adjacent locations on the basilar membrane.

It has been demonstrated with a computational model that an appropriate electrical stimulus may produce a spontaneous-like renewal process in the auditory nerve, a process that is uncorrelated across fibers.<sup>55</sup> Much of the underlying theory has been confirmed in animal studies,<sup>56–58</sup> which demonstrate uncorrelated, Poisson-like activity across a population of auditory nerve fibers activated by an unmodulated 5,000 pulse/s stimulus. A rational hypothesis is that such stimuli can suppress tinnitus without producing an auditory percept by restoring the “code for silence.” No prior studies have effectively examined such stimuli for tinnitus suppression owing to the engineering difficulties of delivering such high rates; these rates have become available only in cochlear implant stimulators recently.<sup>59</sup> Because most tinnitus sufferers do not have severe to profound hearing loss, such a hypothesis should be tested, at least initially, with stimuli applied outside the cochlea. As mentioned earlier, this then leads to ambiguity as to exactly what is stimulated, so a parallel test of the hypothesis should involve patients with severe to profound hearing impairment managed with cochlear implants. The recent advent of “hybrid” or “electroacoustic” cochlear implantation allows partial intracochlear insertion of an electrode array with preservation of residual low-frequency hearing.<sup>60</sup> If the hypothesis is correct, such technology creates the future potential for intracochlear stimulation for tinnitus suppression even in patients with mild hearing loss.

### RESULTS

The hypothesis that unmodulated high-rate pulse trains can suppress tinnitus without producing a percept has now been tested using transtympanic stimulation in 13 subjects with mild to moderate hearing loss and intracochlear stimulation in four subjects with profound hearing loss who were managed with cochlear implants.<sup>27</sup>

#### NO RESPONSE OR LIMITED BY PAIN (FOUR TRANSTYMPANIC SUBJECTS, ONE COCHLEAR IMPLANT SUBJECT)

In two transtympanic subjects, no perception of the stimulus, pain, or effect on tinnitus could be elicited up to the maximal current output of the stimulator ( $> 1.1$  mA at  $80 \mu\text{s}/\text{phase}$ ). In two other subjects, stimulation above approximately  $400 \mu\text{A}$  evoked pain. No significant sound percept or tinnitus effect was noted below this pain threshold. Jacobson's nerve was found to be posteriorly displaced in one of these subjects. It was resting on the anterior lip of the round window niche and was likely responsible for the atypical pain percept in this subject. Multiple electrode placements were attempted to avoid pain perception without success. The cochlear implant subject noted no change in her tinnitus with current levels up to most comfortable loudness on multiple electrodes.

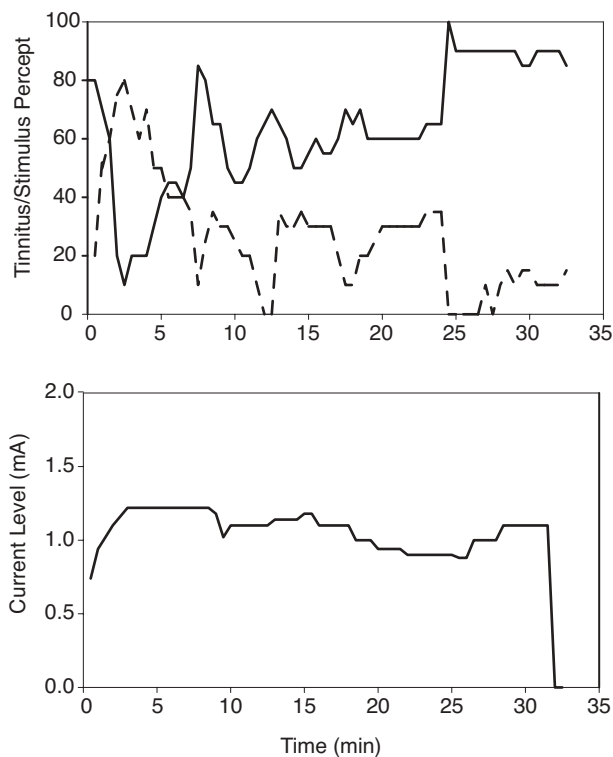


FIGURE 24-2. Tinnitus and stimulus perceptions of subject with transtympanic electrical stimulation of the round window. Stimulus frequency 4,800 Hz,  $80 \mu\text{s}/\text{phase}$ . Dashed line is stimulus percept; solid line is tinnitus percept. The perception of the stimulus appeared to produce a masking effect on the perception of tinnitus in this subject.

#### ELECTRICAL TINNITUS “MASKING” (THREE TRANSTYMPANIC SUBJECTS, ONE COCHLEAR IMPLANT SUBJECT)

Three transtympanic subjects showed tinnitus suppression only in the presence of a stimulus percept. All noted that the stimulus percept sounded similar to their underlying tinnitus. Figure 24-2 illustrates the tinnitus and stimulus percepts over time, along with the stimulus current presented for one of the subjects. The response pattern was similar for all three subjects and did not demonstrate any significant residual inhibition. No particular subject preference for the stimulus percept over the underlying tinnitus was noted in response to questioning. Figure 24-3 demonstrates a similar “masking” pattern in the cochlear implant subject. Tinnitus is suppressed only during perception of the stimulus and virtually mirrors this perception. There was no residual inhibition. Although this phenomenon is clearly different from acoustic masking, we call it a “masking” pattern to differentiate it from tinnitus suppression in the absence of a percept. It is virtually identical to the results reported by Dauman.<sup>61</sup>

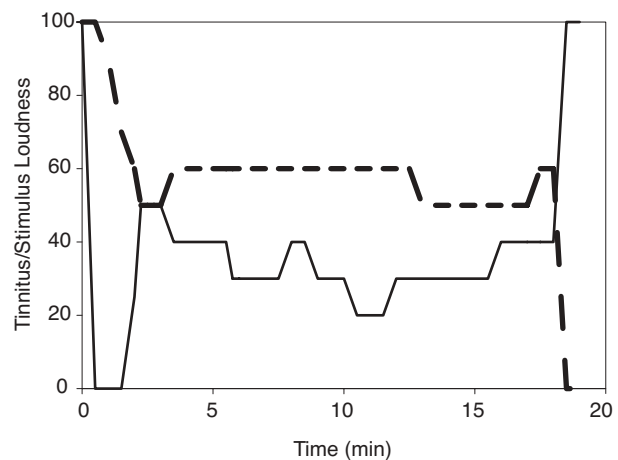


FIGURE 24-3. Tinnitus and stimulus perceptions of a subject with activation of his Cochlear Corporation CI-24M cochlear implant. Stimulus frequency 4,800 Hz,  $25 \mu\text{s}/\text{phase}$  using electrode 14 in monopolar mode. Stimulus onset at time 0, offset at 18 minutes. Dashed line is stimulus percept; solid line is tinnitus percept. The perception of the stimulus appeared to produce a masking effect on the perception of tinnitus in this subject.

### ELECTRICAL TINNITUS SUPPRESSION (SIX TRANSTYMPANIC SUBJECTS, TWO COCHLEAR IMPLANT SUBJECTS)

Six subjects showed suppression of tinnitus in the absence of a stimulus percept or after complete or nearly complete adaptation to the stimulus percept. In the development of speech processing strategies for cochlear implants, we noted that there is a dramatic degree of adaptation to the unmodulated high-rate pulse trains used in this study.<sup>62</sup> Round window stimulation demonstrated this phenomenon in four of the subjects, who initially perceived loud tinnitus-like sounds when the stimulus was ramped up. After several minutes, a time course comparable to that observed in our cochlear implant subjects, the stimulus perception adapted to zero or near-zero followed shortly after by a decrement in the perceived tinnitus. There was a residual inhibition lasting from 45 minutes to 72 hours in five of the subjects. Five of the six patients who suppressed in this manner found clinically significant relief from the annoyance of their tinnitus and were very pleased with the result. Figure 24-4 demonstrates the tinnitus and stimulus percepts from one of these subjects. In all of these individuals,

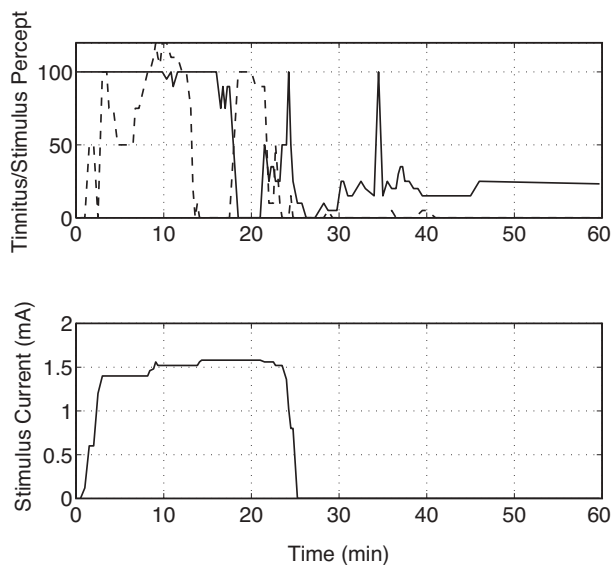


FIGURE 24-4. Tinnitus and stimulus perceptions of subject T. S. with transtympanic electrical stimulation of the round window. Stimulus frequency 4,800 Hz, 80  $\mu$ s/phase. Dashed line is stimulus percept; solid line is tinnitus percept. The perception of the stimulus was followed by rapid adaptation and subsequent suppression of tinnitus.

stimulation at lower pulse rates and/or at lower current levels did not evoke tinnitus suppression.

Figure 24-5 demonstrates tinnitus suppression in one of the four cochlear implant subjects. It verifies that tinnitus suppression in this subject was repeatable. There appeared to be a level-dependent effect, with higher currents evoking more rapid and complete tinnitus suppression. The perceptions produced by the two stimuli were identical, and the tinnitus suppression was therefore effectively single-blinded because no feedback was given to the subject. Residual inhibition lasted approximately 45 minutes.

### CASE STUDY

R. H. is a 63-year-old woman with a past history of Meniere's disease in the left ear. She underwent an endolymphatic shunt 10 years previously, which eliminated her incapacitating vertigo but was complicated by profound deafness and bothersome tinnitus in the involved ear. After informed consent was obtained, she underwent transtympanic round window stimulation following the protocol above,<sup>27</sup> and nearly complete tinnitus suppression was obtained for a period of

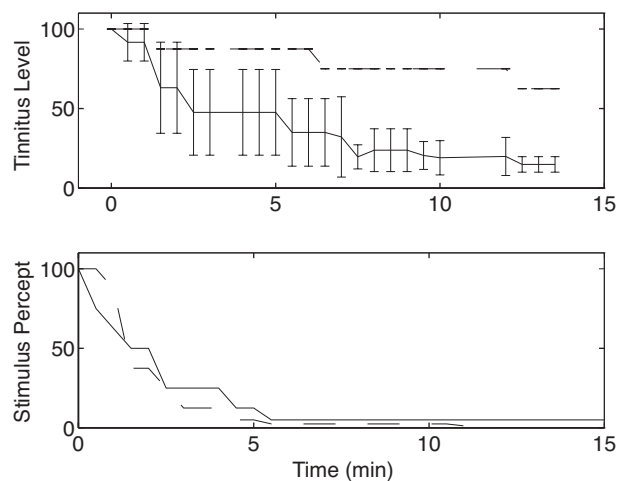


FIGURE 24-5. Tinnitus and stimulus perceptions of a subject with activation of her Cochlear Corporation CI-24M cochlear implant. Stimulus frequency 4,800 Hz, 25  $\mu$ s/phase using electrode 7 in monopolar mode. The dashed line represents a stimulus intensity of 134 clinical units; the solid line represents 142 clinical units. Error bars represent the range of three repetitions on three separate days.

about 45 minutes. After further informed consent was obtained, she elected to undergo cochlear implantation in the deaf ear with the intention of determining whether chronic tinnitus suppression could be achieved with the same stimulus protocol. Surgical implantation of a CI-24 contour (Cochlear Corporation, Lane Cove, Australia) was uneventful.

One month postoperatively, her device was activated, and an apical electrode was pitch-matched to her low-frequency tinnitus. This electrode was then stimulated with a 4,800 pps unmodulated pulse train that was slowly increased while tinnitus and stimulus percepts were noted. Her transtympanic suppression was replicated with intracochlear stimulation, but, unfortunately, the effect was not long lasting. Although the tinnitus was nearly completely suppressed without any lasting percept of the stimulus, it would slowly rise back to prestimulation levels after approximately 45 minutes of continuous stimulation. After many sessions, we demonstrated that tinnitus suppression without a stimulus percept can be maintained over at least a 5-hour period by turning the stimulus on and off at several different levels every few minutes. Our current studies are focused on development of a placebo-controlled paradigm that produces similar transient percepts but does not suppress the tinnitus. R. H. is extremely gratified that she is able to exercise some level of control over her tinnitus. Her subsequent artwork provides her depiction of the distress caused by her tinnitus (Figure 24-6).

## DISCUSSION

At this time, the only clinically effective treatment that decreases the loudness of tinnitus is acoustic masking. Although this is helpful for some patients, for others it simply replaces one undesirable sound with another. An ideal therapy for tinnitus would create the perception of silence or at least decrease the loudness of tinnitus without introducing any new sounds. We demonstrated in 6 of 13 subjects that this is possible, at least temporarily. Although placebo control in the current experimental paradigm is not ideal, we doubt that these six subjects are demonstrating significant placebo effects given the similarity of their responses to those of the cochlear implant subjects, for whom single-blinded placebo control was possible. In addition, multiple stimulus frequencies and intensities failed to suppress tinnitus in the process of data ac-



FIGURE 24-6. Painting by chronically implanted subject R. H. depicting her perception of the impact of tinnitus on her life. She performed this work after undergoing the studies described in the text.

quisition for these subjects. Because of the prolonged residual inhibition of tinnitus produced by the stimulus, test–retest variability could not be assessed in the transtympanic subjects because this would have required a second myringotomy at a later testing session.

Although the theory underlying the hypothesis tested would suggest rapid (< 20 milliseconds) onset and offset of spontaneous-like activity in the auditory nerve, tinnitus suppression as induced in the seven subjects appears to be a slow process. Onset of tinnitus suppression typically required between 5 and 15 minutes of stimulation, and residual inhibition lasted from minutes to days. This suggests involvement of an unknown central process and is in contrast to the immediate “masking” effects in four subjects. The prolonged residual inhibition is puzzling but must also represent some central effect and does not support or refute the underlying theory. Gibson reported that 8% of 27 cochlear implant patients with tinnitus note residual inhibition lasting from 30 to 60 minutes.<sup>15</sup>

One of our cochlear implant patients, who is not in this study, reported that his tinnitus returns only when his implant is turned off for more than 24 hours. Although unusually long relative to residual inhibition with acoustic masking,<sup>63</sup> the residual inhibition reported here may, in fact, make clinical application of a tinnitus suppression device easier.

Our findings support the hypothesis that high-rate electrical pulse trains applied to the round window can result in tinnitus suppression without an ongoing stimulus percept. Two problems must be addressed before a clinical device becomes feasible. First, with the notable exception of two cochlear implant subjects, it is still not clear whether the effects we describe are repeatable within a subject. Repeatability is critical for the long-term use of a tinnitus suppression device and for placebo control during a clinical trial. Second, it is not clear whether the stimulation has an effect on acoustic thresholds. On questioning, none of our subjects noted decreased hearing during stimulation. In fact, one subject spontaneously noted improved clarity of speech perception during the stimulation. Nevertheless, increased hearing thresholds and/or decreased speech recognition are a possibility owing to either a mechanical effect of the round window electrode or interaction between electrical stimulation and acoustically evoked hair cell activity.<sup>35</sup> Answering these questions will require that more human subjects undergo surgical implantation of a modern, cochlear implant-like device in the cochlea or at the round window that can be tested repeatedly and used chronically. Although electrical stimulation is clearly not a panacea, it will likely play a future role in the management of some patients with severe tinnitus.

## REFERENCES

1. Feldmann H. History of tinnitus research. In: Shulman A, editor. *Tinnitus diagnosis/treatment*. San Diego (CA): Singular Publishing Group, Inc.; 1997. p. 3–37.
2. Feldmann H. Suppression of tinnitus by electrical stimulation: a contribution to the history of medicine. *J Laryngol Otol Suppl* 1984;9:123–4.
3. Volta A. On the electricity excited by the mere contact of conduction substances of different kinds. *Philos Trans R Soc Lond B Biol Sci* 1800;90:403–31.
4. Grapengiesser CJC. *Versuche den Galvanismus zur heilung einiger Krankheiten anzuwenden*. Berlin: 1801.
5. Brenner R. *Untersuchungen und Beobachtungen über die Wirkung elektrischer Strome auf das Gehörorgan im gesunden und kranken Zustande*. Leipzig: 1868.
6. Hatton DS, Erulkar SD, Rosenberg PE. Some preliminary observations on the effect of galvanic current on tinnitus aurium. *Laryngoscope* 1960;70:123–30.
7. Aran JM. Neural correlates of electrically induced cochlear dysfunction. *Clin Otolaryngol* 1977;2:305–10.
8. Aran JM. Electrical stimulation of the auditory system and tinnitus control. *J Laryngol Otol Suppl* 1981;4:153–62.
9. Aran JM, de Sauvage R, Cazals Y. Observation of an electrically evoked whole-nerve response using the same stimulating and recording electrode. *Hear Res* 1980;2:343–6.
10. Aran JM, Cazals Y. Electrical suppression of tinnitus. *Ciba Found Symp* 1981;85:217–31.
11. Agnew WF, McCreery DB, Bullara LA, Yuen TGH. Effects of prolonged electrical stimulation of peripheral nerve. In: Agnew WF, McCreery DB, editors. *Neural prostheses: fundamental studies*. Englewood Cliffs (NJ): Prentice Hall; 1990.
12. House JW, Brackmann DE. Tinnitus: surgical treatment. *Ciba Found Symp* 1981;85:204–16.
13. House JW. Effects of electrical stimulation on tinnitus. *J Laryngol Otol Suppl* 1984;9:139–40.
14. Tyler RS, Kelsey D. Advantages and disadvantages reported by some of the better cochlear-implant patients. *Am J Otol* 1990;11:282–9.
15. Gibson WPR. The effects of electrical stimulation and cochlear implantation on tinnitus. In: Aran JM, Dauman R, editors. *Tinnitus 91: proceedings of the Fourth International Tinnitus Seminar*. Amsterdam: Kugler; 1992. p. 403–8.
16. Zwolan TA, Kileny PR, Souliere CR, Kemink JL. Tinnitus suppression following cochlear implantation. In: Aran JM, Dauman R, editors. *Tinnitus 91: proceedings of the Fourth International Tinnitus Seminar*. Amsterdam: Kugler; 1992. p. 423–6.
17. Bredburg G, Walden J, Lindstrom B. Tinnitus after cochlear implantation. In: Aran JM, Dauman R, editors. *Tinnitus 91: proceedings of the Fourth International Tinnitus Seminar*. Amsterdam: Kugler; 1992. p. 417–22.
18. Dauman R, Tyler RS, Aran JM. Intracochlear electrical tinnitus reduction. *Acta Otolaryngol (Stockh)* 1993;113:291–5.

19. Tyler RS. Tinnitus in the profoundly hearing-impaired and the effects of cochlear implants. *Ann Otol Rhinol Laryngol Suppl* 1995;165:25–30.
20. Ruckenstein MJ, Hedgepeth C, Rafter KO, et al. Tinnitus suppression in patients with cochlear implants. *Otol Neurotol* 2001;22:200–4.
21. Vernon JA, Fenwick JA. Attempts to suppress tinnitus with transcutaneous electrical stimulation. *Otolaryngol Head Neck Surg* 1985;93:385–9.
22. Kuk FK, Tyler RS, Rustad N, et al. Alternating current at the eardrum for tinnitus reduction. *J Speech Hear Res* 1989;32:393–400.
23. Hazell JWP, Jastreboff PJ, Meerton LE, Conway MJ. Electrical tinnitus suppression: frequency dependence of effects. *Audiology* 1993;32:68–77.
24. Okusa M, Shiraishi T, Kubo T, Matsunaga T. Tinnitus suppression by electrical promontory stimulation in sensorineural deaf patients. *Acta Otolaryngol Suppl (Stockh)* 1993;501:54–8.
25. Ito J, Sakakihara J. Tinnitus suppression by electrical stimulation of the cochlear wall and by cochlear implantation. *Laryngoscope* 1994;104:752–4.
26. Matsushima JL, Sakai N, Uemi N, et al. Evaluation of implanted tinnitus suppressor based on tinnitus stress test. *Int Tinnitus J* 1997;3:123–31.
27. Rubinstein JT, Tyler RS, Johnson A, Brown CJ. Electrical suppression of tinnitus with high-rate pulse trains. *Otol Neurotol* 2003;24:478–85.
28. Shulman A. External electrical stimulation in tinnitus control. *Am J Otol* 1985;6:110–5.
29. Dobie RA, Hoberg KE, Rees TS. Electrical tinnitus suppression: a double-blind crossover study. *Otolaryngol Head Neck Surg* 1986;95:319–23.
30. Salvi RJ, Lockwood A, Burkard R. Neural plasticity and tinnitus. In: Tyler RS, editor. *Tinnitus handbook*. San Diego (CA): Singular Publishing Group, Inc.; 2000. p. 1–24.
31. Lockwood AH, Salvi RJ, Coad ML, et al. The functional neuroanatomy of tinnitus: evidence for limbic system links and neural plasticity. *Neurology* 1998;50:114–20.
32. Lockwood AH, Wack DS, Burkard RF, et al. The functional anatomy of gaze-evoked tinnitus and sustained lateral gaze. *Neurology* 2001;56:472–80.
33. Melcher JR, Sigalovsky IS, Guinan JJ Jr, Levine RA. Lateralized tinnitus studied with functional magnetic resonance imaging: abnormal inferior colliculus activation. *J Neurophysiol* 2000;83:1058–72.
34. Plewnia C, Bartels M, Gerloff C. Transient suppression of tinnitus by transcranial magnetic stimulation. *Ann Neurol* 2003;53:263–6.
35. Miller CA, Abbas PJ, Rubinstein JT, et al. Effects of remaining hair cells on cochlear implant function. Fifth quarterly progress report NO1-DC-9-2106. Neural Prosthesis Program. Bethesda (MD): National Institutes of Health; 2000.
36. Kiang NYS, Moxon EC, Levine RA. Auditory-nerve activity in cats with normal and abnormal cochleas. In: Wolstenhome GEW, Knight J, editors. *Sensorineural hearing loss*. London: JEA Churchill; 1970. p. 241–73.
37. Kiang NYS, Liberman MC, Levine RA. Auditory nerve activity in cats exposed to ototoxic drugs and high-intensity sounds. *Ann Otol Rhinol Laryngol* 1976;75:752–68.
38. Jahn K, Naessl A, Schneider E, et al. Inverse U-shaped curve for age dependency of torsional eye movement responses to galvanic vestibular stimulation. *Brain* 2003;126:1579–89.
39. MacDougall HG, Brizuela AE, Curthoys IS, Halmagyi GM. Three-dimensional eye-movement responses to surface galvanic vestibular stimulation in normal subjects and in patients: a comparison. *Ann NY Acad Sci* 2002;956:546–50.
40. Watson SR, Welgampola MS, Colebatch JG. EMG responses evoked by the termination of galvanic (DC) vestibular stimulation: ‘off-responses.’ *Clin Neurophysiol* 2003;114:1456–61.
41. Levine RA. Somatic (craniocervical) tinnitus and the dorsal cochlear nucleus hypothesis. *Am J Otolaryngol* 1999;20:351–62.
42. Shore SE, El Kashlan H, Lu J. Effects of trigeminal ganglion stimulation on unit activity of ventral cochlear nucleus neurons. *Neuroscience* 2003;119:1085–101.
43. Rubinstein JT. Axon termination conditions for electrical stimulation. *IEEE Trans Biomed Eng* 1993;40:654–63.
44. Miller CA, Robinson BK, Rubinstein JT, et al. Auditory nerve response to monophasic and biphasic electric stimuli. *Hear Res* 2001;151:79–94.
45. Tyler RS, Conrad-Arnes D. Masking of tinnitus compared to masking of pure tones. *J Speech Hear Res* 1984;27:106–11.
46. Tyler RS. Psychoacoustical measurement. In: Tyler RS, editor. *Tinnitus handbook*. San Diego (CA): Singular Publishing Group, Inc.; 2000. p. 149–80.

47. Duckert LG, Rees TS. Placebo effect in tinnitus management. *Laryngoscope* 1984;92:697–9.
48. Dobie RA, Sakai CS, Sullivan MD. Antidepressant treatment of tinnitus patients: report of a randomized clinical trial and clinical prediction of benefit. *Am J Otol* 1993;14:18–23.
49. Liberman MC. Auditory nerve responses from cats raised in a low-noise environment. *J Acoust Soc Am* 1978;75:442–55.
50. Sewell WF. The relation between the endocochlear potential and spontaneous activity in auditory nerve fibers of the cat. *J Physiol* 1984;347:685–96.
51. Johnson DH. Point process models of single-neuron discharges. *J Comput Neurosci* 1996;3:275–99.
52. Liberman MC, Dodds LW. Single-neuron labeling and chronic cochlear pathology. II. Stereocilia damage and alterations of spontaneous discharge rates. *Hear Res* 1984;16:43–53.
53. Kaltenbach JA, Godfrey DA, Neumann JB, et al. Changes in spontaneous neural activity in the dorsal cochlear nucleus following exposure to intense sound: relation to threshold shift. *Hear Res* 1998;124:78–84.
54. Zhang JS, Kaltenbach JA. Increases in spontaneous activity in the dorsal cochlear nucleus of the rat following exposure to high-intensity sound. *Neurosci Lett* 1998;250:197–200.
55. Rubinstein JT, Wilson BS, Finley C, Abbas PJ. Pseudospontaneous activity: stochastic independence of auditory nerve fibers with electrical stimulation. *Hear Res* 1999;127:108–18.
56. Runge-Samuels C. Response of the auditory nerve to sinusoidal electrical stimulation: effects of high-rate pulse trains [thesis]. Iowa City (IA): University of Iowa; 2002.
57. Litvak L, Delgutte B, Eddington D. Auditory nerve fiber responses to electric stimulation: modulated and unmodulated pulse trains. *J Acoust Soc Am* 2001;110:368–79.
58. Litvak L. Towards a better speech processor for cochlear implants: auditory nerve responses to high rate electric pulse trains [thesis]. Boston (MA): Massachusetts Institute of Technology; 2002.
59. Rubinstein JT, Hong RS. Signal coding in cochlear implants: exploiting stochastic effects of electrical stimulation. *Ann Otol Rhinol Laryngol Suppl* 2003;191:14–9.
60. Gantz BJ, Turner CW. Combining acoustic and electrical hearing. *Laryngoscope* 2003;113:1726–30.
61. Dauman R. Electrical stimulation for tinnitus suppression. In: Tyler RS, editor. *Tinnitus handbook*. San Diego (CA): Singular Publishing Group, Inc.; 2000. p. 377–98.
62. Hong RS, Rubinstein JT, Wehner D, Horn D. Dynamic range enhancement for cochlear implants. *Otol Neurotol* 2003;24:590–5.
63. Tyler RS, Conrad-Armes D, Smith PA. Postmasking effects of sensorineural tinnitus: a preliminary investigation. *J Speech Hear Res* 1984;27:466–74.

## **EDITORIAL COMMENTARY**

Rubinstein and Tyler review the evidence that tinnitus is initially due to decreased spontaneous activity in the auditory nerve. They point out the ambiguity that exists in electrical stimulation of the middle ear as to whether the effect is due to auditory nerve stimulation or somatosensory stimulation that is known to be capable of modulating tinnitus.

In stimulating the inner ear, that ambiguity is avoided. A high rate of electrical pulses (5,000/s) re-

stores spontaneous-like activity in the auditory nerve, and this may be the way in which it suppresses tinnitus.

Rubinstein and Tyler demonstrated that high-rate electrical pulse trains can suppress tinnitus without producing an audible percept. The remaining problems of repeatability, placebo control, and hearing effect require further research with surgical implantation of advanced cochlear implant-like devices in the cochlea to determine the future role of electrical suppression in the management of tinnitus.

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