Ultrasonic Hearing

M. L. Lenhardt et al. (1) suggest that "bone-conducted, ultrasonic stimulation may provide an alternative therapeutic approach for the rehabilitation of severe hearing loss." They argue that when speech signals are used to modulate the amplitude of an ultrasonic carrier, people detect and recognize speech sounds by physiological mechanisms other than those that normally transduce audible speech.

There is an alternative explanation for the findings of Lenhardt et al. that is consistent with classic auditory physiology, but does not support the possibility of using this approach to bypass a damaged cochlea. Even a slight, even-order (rectifying) nonlinearity in the transfer path from transducer to skull would result in demodulation. The speech signals, which had been modulated onto the ultrasonic carrier, could be converted back into audio frequency bone-conducted stimuli, capable of being transduced by conventional cochlear processes.

The stimuli used by Lenhardt et al. were intense compared with those used for bone conduction testing in the audio frequency range. Lenhardt et al. specify their stimuli as acceleration, in decibels with reference to $10^{-3}$ m/sec$^2$, whereas the American National Standards Institute S3.26-1981 standard (2) for zero hearing level for the B-71 bone vibrator averages 30.5 dB with reference to 1-μN force at 2 kHz. If this force is applied to a 5-kg head moving as a rigid body, an acceleration of $6.7 \times 10^{-6}$ m/sec$^2$, or 43 dB below Lenhardt's reference level (1), would result. Response thresholds to ultrasound are reported (1) at ±82 to ±112 dB (with reference to $1 \times 10^{-3}$ m/sec$^2$) or 125 to 155 dB above threshold accelerations at audio frequencies. If only 10% of the head mass were to effectively move in response to bone-conducted ultrasound, their thresholds would still be 105 to 135 dB more intense than audio-frequency thresholds.

A slight nonlinearity, resulting in demodulation of audible speech signals, might be difficult to observe in spectral analysis with only a 60-dB dynamic range. A better test would be to measure cochlear potentials. Foster and Wiederhold (3) showed that, in cats, pulsed ultrasound produced cochlear microphonics and compound action potentials that were indistinguishable from those produced by audible transient stimuli. We suspect that, were a study to be performed similar to (3) that used the stimuli presented by Lenhardt et al., it would reveal that these stimuli were present in the cochlear microphonics and thus available in the audible range within the cochlea.

The results obtained by Lenhardt et al. in human subjects were not clearly better than could have been achieved by presenting the same speech stimuli in the normal audio range. Even their "deaf" subjects had mean thresholds [(1), figure 1A] that were within about 55 dB of the normal threshold at 250 Hz [we assume that the abscissa of figure 1A in (1) should have been labeled from 0 to 10,000 Hz for the air conduction thresholds, and that the lowest frequency tested was about 250 Hz]. Thus, some of these subjects could probably discriminate some speech stimuli when presented with adequate stimulus intensity. Two of the nine "deaf" subjects displayed accuracy scores of 20 and 30%, respectively, on a closed-set test for which random performance would produce a score of 16%. Even if these two performances had been significantly better than chance, presentation of the same speech materials as high-intensity audio stimuli might have given similar results.

One critical control experiment would be quite simple. If speech signals were reaching the cochlea as audio-range signals after demodulation, they would be maskable by audio-range noise maskers.

Until more conventional mechanisms can be excluded, it appears premature to suggest a separate ultrasonic receptor, particularly when the structure suggested to detect ultrasound, the saccule, responds to vibrations from zero to only 2 kHz in the squirrel monkey (4).

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Response: Dobie and Wiederhold postulate that the relatively intense ultrasonic signals used in our study (1) could allow demodulation of ultrasonic speech into the audio frequency range. The normal hearing ear is an excellent demodulation detector. With modulation, all listeners in our study reported hearing the sidebands and the carrier, which would have been impossible had the signals been demodulated. With deaf subjects we again found no evidence for following exception: We defined the "signal" in each section by taking a measurement from a 75 by 75 μm area within the SCN and subtracting a background measurement from an area of equal size lateral to the SCN.

27. The AP-1 consensus oligonucleotides used in binding assays were oligo 1 (Oncogene Science), double-stranded 36-nucleotide, 5'-GATCCATCGTGAC- TCAGCCCTCGCCAATTCACCGGG-3'; oligo 2 (Promega), double-stranded 21-nucleotide, 5'- CGCTTGATGAGTCAGCCGGAA-3'. (Boldface indicates consensus AP-1 binding sites.) Oligonucleotides were radiolabeled with [32P]deoxyctydine 5'-triphosphate and terminal deoxynucleotidyl transferase and gel-purified by electrophoresis on a 15% polyacrylamide gel. Whole-cell extracts of brain tissue were prepared by sonication of each SCN sample (17) at 4°C in 50 μl of buffer [20 mM Hepes (pH 7.8), 5 mM spermidine, 15 mM MgCl2, 30% glycerol, 0.2 mM EDTA, bovine serum albumin (BSA) (1 mg/ml), 0.1% Nonidet P-40, 5 mM dithiothreitol (DTT), 0.5 mM phenylmethylsulfonyl fluoride, leupeptin (0.5 μg/ml), pepstatin (0.7 μg/ml), aprotinin (1 μg/ml), and benzamid (40 μg/ml)]. Extracts were then centrifuged at 15,600g for 10 min (at 4°C) and the supernatants collected. Binding assays were performed with 10 μl of SCN cell extract, 1.5 μg of poly[dI:C]-p-labeled probe (50 mM Hepes (pH 7.8), 5 mM spermidine, 15 mM MgCl2, 30% glycerol, 3 mg/ml), 0.3% Nonidet P-40, and 15 mM DTT], and 30,000 cpm of 32P-labeled oligonucleotide, with water added to a final volume of 25 μl. Reactions were incubated for 15 min on ice before addition of 32P-labeled oligonucleotide, then for an additional 15 min at 22°C. When competition with unlabeled oligonucleotide was performed, a 50-fold molar excess of oligonucleotide relative to the radiolabeled probe was added to the binding assay. When antibodies were included, 1 μl was added to the reaction 15 min before the addition of the probe. The antibody to Fox is a rabbit antiserum made against the synthetic peptide corresponding to amino acids 127 to 156 of mouse Fox; rabbit antiserum against bovine neuron-specific enolase (Accurate) was used as a control antibody. Samples were subjected to electrophoresis on 4% nondenaturing polyacrylamide gels, dried, and autoradiographed. 28. We thank T. Qurran, D. Linzer, and J. Mordacq for advice on AP-1 gel mobility-shift assays, and R. J. Disel and B. M. Spiegelman for use of the antibody to Fox. Supported by grants from the NIH, NSF, and McKnight Endowment Fund for Neuroscience (K.E.M.) and from the NIH, NSF, and the National Institute of Mental Health (J.S.T.).
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