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Eyes as Fenestrations to the Ears: A Novel Mechanism for High-Frequency and Ultrasonic Hearing

Martin L. Lenhardt

Departments of Biomedical Engineering, Otolaryngology, and Emergency Medicine, Virginia Commonwealth University, Richmond, Virginia

Abstract: Intense airborne ultrasound has been associated with hearing loss, tinnitus, and various nonauditory subjective effects, such as headaches, dizziness, and fullness in the ear. Yet, when people detect ultrasonic components in music, ultrasound adds to the pleasantness of the perception and evokes changes in the brain as measured in electroencephalograms, behavior, and imaging. How does the airborne ultrasound get into the ear to create such polar-opposite human effects? Surprisingly, ultrasound passes first through the eyes; thus, the eye becomes but another window into the inner ear.

Key Words: eye; hearing loss; music; tinnitus; ultrasound

It has long been recognized that intense sound can distort vision and reduce visual acuity, presumably by overpressure of the lens [1–3]. Infrasound often is considered a primary source; however, any frequency with sufficient energy could theoretically produce the same effect, including sound beyond the traditional audible limit (i.e., ultrasound). Further, it is well documented that acoustic energy, including ultrasound, can pass into the eye [3]. What has not been examined in any of these studies is the possibility that the acoustic energy that passes through the eye may also serve as a source of auditory perception.

The eye is a mechanical structure capable of sound excitation under certain conditions and constraints. Eye conduction of airborne sound could explain hearing loss and tinnitus in young workers exposed to intense ultrasound (typically cleaners) [4–6] and could explain the perplexing phenomenon that ultrasonic energy influences judgments of music quality and induces changes in electroencephalographic and positron emission tomographic examinations [7,8]. The implications of the latter are enormous for the commercial music industry. It has long been recognized that live music has a wider (upper and lower) dynamic range than can be precisely captured by analog and 44.1 kHz/16-bit digital recordings: Could it be that the eye acts as an acoustic lens for the ear, passing frequencies beyond the impedance-matching capacity of the eardrum (>20 kHz) to the ear? Further, could airborne ultrasonic energy entering the eye secondarily induce tinnitus and hearing loss?

The appreciation value of very-high-frequency music energy has triggered a provocative debate over the use of high digital sampling rates to capture such energy in recordings. Conventional wisdom [9] since the 1930s has been that high-frequency energy to only 14 kHz was needed to recreate music faithfully. This assumption, some 75 years later, is only slightly modified in that useful hearing does not exceed 20 kHz; therefore, digital sampling of audio frequencies at 44.1 kHz/16 bit is sufficient to meet the high-frequency needs of most listeners. The upper frequency (UF) limit or the Nyquist frequency is represented as: UF = DS/2, where DS is the digital sampling rate. Thus, if the sampling rate is 44.1 kHz, the upper listening range is 22.05 kHz. Nonetheless, this sampling rate of 44.1 kHz introduces high-frequency distortions (i.e., quantizing granularity) detectable by some listeners, which could be eliminated by sampling at 96 kHz/24 bit. In effect, the conservative estimate of 20 kHz as the upper range of hearing has resulted in a too-slow digital sampling to support

Reprint requests: Martin L. Lenhardt, AuD, PhD, Department of Biomedical Engineering, Virginia Commonwealth University, Box 980168 MCV, Richmond, VA 23298-0168. Phone: +1 804 828 9687; Fax: +1 804 828 4454; E-mail: lenhardt@hsc.vcu.edu

high fidelity without distortions. If frequencies higher than 20 kHz are audible or useful, clearly sampling at 96 kHz is a necessity for true high fidelity.

The question can be asked another way: Does the ear run out of musicality at 20 kHz? Many instruments produce energy well beyond 20,000 Hz at high levels [10]; even the human voice is capable of ultrasonic output if sufficient vocal effort is mustered (personal observation, 1990). Further electroencephalographic and positron emission tomographic changes were documented when frequencies beyond 22 kHz were presented along with conventional audio frequencies [7,8] to human listeners. No ultrasonic route to the ear was proposed in these studies, but certainly possible is that the ultrasonic energy in music—and for that matter, industrial noise—could pass through the eye and be detected by the ear. Is the eye pathway the reason that live music sounds better than recordings or the reason why ultrasonic instruments can cause hearing loss and tinnitus? That proposition is examined in this study.

FLUID PATHWAYS TO THE EAR

Extratympanic auditory pathways exist, as Ranke [11] proposed in 1930. The endolymphatic and cochlear aqueducts (or, frankly, any vascular connection to the cochlea) could act as acoustic fluid pathways, much as the oval or round windows act as aerial pathways. These fluid pathways were termed *the third window of Ranke* by Tonndorf and Tabor [12], who later demonstrated cochlear function after sealing both the round and oval windows; thus, for the basilar membrane to be displaced, some compliant window is necessary. More recently, it has been shown that direct vibration of the brain [13,14] could elicit evoked responses from the auditory nerve, confirming that a brain can be a passive "fluid" conductor of sound, even an amplifier near resonance [15] and, at the same time, a physiological responder to the sound propagated through it. Thus, the pathway through the brain to the ear has been established. The ear link to the eye has yet to be established.

The eye is a fluid globe that will offer an impedance mismatch to sound in air, much like that of an ear with a fluid-filled middle-ear space. Hearing with a fluid-filled middle ear is not only possible but the degree of loss is small (on the order of 30 dB). After the impedance mismatch is overcome with sufficient energy, is the eye, like the fluid-filled middle ear, capable of transmitting the absorbed sound to the brain?

METHODS

Five normal-hearing adults (ages 20–59 years) with no history of ocular, otic, or neural disease served as our subjects. All had normal hearing at the time of testing. We established stimulating parameters and recording techniques over a 2-year period using six additional subjects. High-frequency stimulating transducers were custom-made ceramic aluminum bimorphs attached with tape to the skin of the skull. We attached eye-recording piezoelectric film sensors to the closed eyelids with tape. We also mounted calibration accelerometers either to the transducer sensors or to the skin of the skull for mass-loaded measurements. All attached devices were well tolerated. Testing time did not exceed 1 hour; most subjects completed testing in three sessions. The study received local institutional review board approval.

Experiment 1: Determining the Frequency Response of the Eye

We determined the frequency response of the eye by driving sound through the head via transducers on the skin of the head and recording from the eye. Specifically filtered white noise (10–100,000 Hz) was applied through a custom-designed aluminum ceramic transducer placed on the skin of the head (mastoid, occiput, and forehead). We measured the input to the head with a Buel and Kjaer (B&K) high-frequency accelerometer placed on the transducer while mass-loaded to the head. We subsequently fed the signal into a B&K pulse system for calibrating the stimulating level (42 dB re: 1 m/sec^2), the result of which is depicted in Figure 1A. To record the sound spectra from the eye, we taped a piezoelectric film sensor, constructed of a piezoelectric polymer polyvinylidene fluoride [16], over the closed eyelid, being careful not to make contact with the orbit, to detect the frequency response of the globe to white noise propagating around the skull and through the brain. This sensor has the acoustic impedance of water. We found the frequency transfer from the brain to the eye to be in the passband from 25 to 60 kHz.

Experiment 2: Determining the Transmission from the Eye to the Brain

In the next experiment, we applied airborne noise within the passband of the eye (25–60 kHz, as depicted in Fig. 1B) as sound anterior to the center of the globe and recorded the response from the skull in one subject. We modified the same type of transducer that delivered contact sound to the skull to produce a narrow beam of airborne ultrasound by fabricating a foam gasket with a 2-mm aperture. We placed the transducer 10 mm from the center of the eye and maintained the sound pressure at 96.5 dB sound pressure level (SPL) or $10^{-6.4}$ W/cm^2. The eye was in the near field (aperture diameter^2/4$\times$ wavelength), so theoretically the beam size was relatively

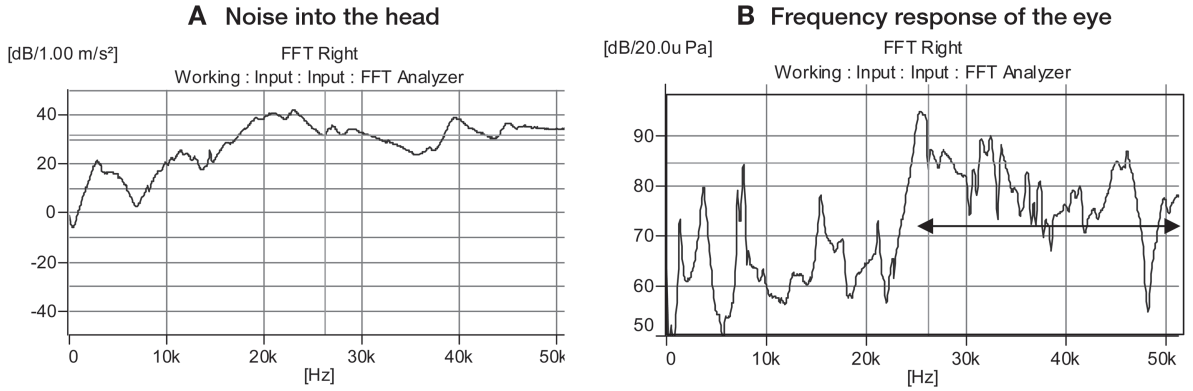

Figure 1. Ultrasonic skull-brain stimulus, eye response. (A) Noise spectrum delivered to the head. (B) Transfer function of the eye. The effective band of eye frequency response is from 25 to approximately 60 kHz (*double-headed arrow*). Both graphs are referenced to acceleration at 1 m/s^2.

constant, although there is near-field intensity uncertainty. The piezoelectric film transducer acted as a sensor for calibration trials but was removed during measurements of ocular transmission. The film has the acoustic impedance of water and a flat frequency response in the range of interest, allowing relative monitoring of the peak SPL. The threshold for hearing at 25 kHz is approximately 125 dB SPL [17], and the present stimulus was inaudible, but the induced head vibration was recordable. The transmission loss (TL) from bone to brain or eye is predictable on the basis of the acoustic properties related to tissue density and propagation speed [18].

As a control, the experiment was repeated with the subjects wearing acrylic goggles. The ultrasonic beam was directed at the forehead or into the ear canal as an additional control. We measured skull-brain vibration under all three control conditions. As a precaution, the mouth remained closed, and the beam was not directed at the nares. Tactile responses to airborne ultrasound have been reported in the oral and nasal cavities if the mouth is open, likely owing to a standing wave phenomenon [4] that directly stimulates epithelial cilia.

RESULTS

Broadband noise (5–70 kHz) delivered to the skull at the mastoid, occiput, or forehead excites vibration in the eye. The frequency response of eye excitation ranged from 25 to 60 kHz. When that band of noise was presented as an airborne sound directed at the eye, we measured vibration of the brain and skull at the mastoid, occiput, and forehead in the same frequency range of 25–60 kHz. The assumption is that the eye dimensions are small in regard to the near field, and so scattering within the eye may not be anticipated. Some scattering might arise in the brain, adding to the attenuation [19].

The predicted transmission loss was determined by the acoustic mismatch between the impedance of sound in air with that of sound in ocular tissues. Specifically, acoustic impedance (Z) is the product of density, expressed in kilograms per cubic meter, and ultrasonic velocity, expressed in expressed in meters per second. The transmission coefficient (TC) of a medium can be calculated by $4(Z_1Z_2)/(Z_1 + Z_2)$. The TC for air to the body is 0.0011 or a TL of 59 dB. Placing the aluminum transducer on the skin results in a TC of 0.3 and a transmission difference of 10 dB. The propagation speeds and densities of the relative tissues for this study are depicted in Figure 2.

We did not measure the TL from the transducer to the eye; however, the air between the transducer and the eye acts as a coupling medium, and the TL may be less than air to body by an estimated 7–17 dB (Table 1). Any reflected waves from the eye are rapidly absorbed

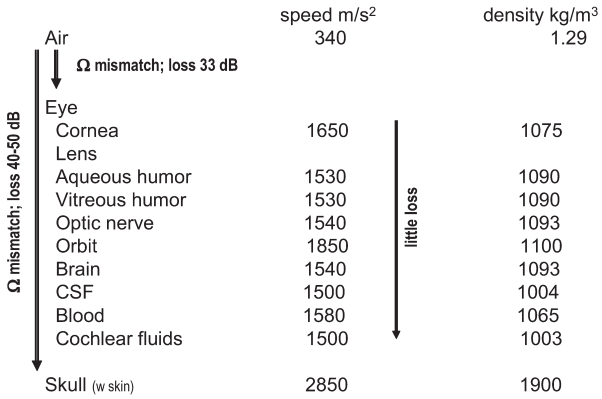

Figure 2. Physical properties of the eye, brain skull, blood, and fluids. There is considerable acoustical similarity among the soft tissues suggesting little impedance mismatches contributing to transmission loss. Air coupling to the eye might reduce the transmission loss to about 33 dB, whereas skin-brain-skull transmission loss might be as high as 50 dB (depending on frequency).

Table 1. Dominant Frequency and Attenuation in the Head for Ultrasound

	Measured Values			Predicted Values:
Recording Site	Frequency (kHz)	Peak (kHz)	Attenuation (dB)	Attenuation (dB)
Mastoid to eye	25–60	35	14	—
Mastoid to forehead	2–63.5+	15	4–6	—
Eye to forehead	25–60	30–40	8	7
Eye to mastoid	25–60	35	14	17

in air. Data in Figure 2 would predict little attenuation for sound passing from the eye to the brain in regard to changes in substrate density; only distance would determine transmission loss. Thus, the energy leaving the eye should be more intense at the forehead than at the mastoid, correcting for impedance changes from fluid to bone. The predicted difference was 10 dB (see Table 1); the measured difference was 6 dB, given the short distance along the skull. Skull-to-skull measurements are wider in frequency response, with a peak at approximately 15 kHz, presumably reflecting brain resonance as well as less attenuation.

Finally, to demonstrate that the response recorded from the skin of the head represented the vibrations in the brain, we asked the subjects to perform a Valsalva maneuver, which increases cerebrospinal fluid [20]. The head is driven with filtered white noise with a 2- to 50-kHz passband; eyelid recordings are obtained from piezoelectric film sensors as before. It is argued that an increase in cerebrospinal fluid will damp the vibration in the brain while not substantially altering the spectrum. Data from two subjects are presented in Figure 3.

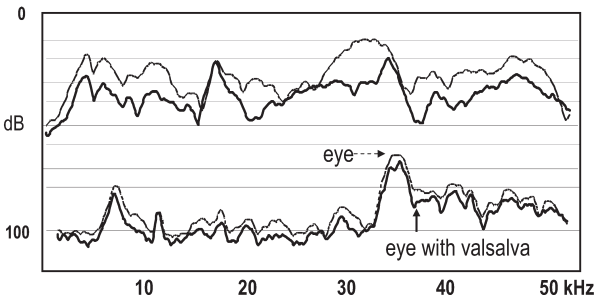

Figure 3. Frequency response of two subjects with increased cerebrospinal fluid pressure after performance of a Valsalva maneuver. Increased cerebrospinal fluid increases the damping in the brain, which would lower the intensity of the spectrum recorded from both skull and brain. This damping, of some 5 dB, is clearly visible in both records. These data represent the extremes in variability encountered. Pre– and post–Valsalva maneuver records were essentially the same. The Valsalva maneuver supports the interpretation that the eye data reflect the acoustic properties of the brain-skull.

The damped tracing during the Valsalva maneuver (solid line) is some 5 dB lower in intensity in both subjects; the variability between subjects is also evident, yet consistent with the concept of the eye as an acoustic sensor for ultrasound. The brain, skull, and eye are coupled acoustically; with the skull essentially rigid, the compression of the brain against the skull is the source of change in Figure 3.

DISCUSSION

Direct vibration of the brain can be communicated to the cochlea via intracranial fluid conduction [13,14], a pathway proposed by Ranke [11] more than 60 years ago and termed *the third window* by Tonndorf and Tabor [12]. Thus, transmission of airborne ultrasonic frequencies through the eye to the ear via intracranial fluid conduction helps to explain two mysteries in the human extended range of hearing. A number of authors [4–6] have documented harmful, intense airborne ultrasound effects on people. Analysis of data to date suggests that progressive loss of hearing in the very high frequencies (<12 kHz) and tinnitus are somehow related to an increased, intense airborne ultrasonic exposure. "Subjective nonauditory effects" or "ultrasonic sickness" characterized by nausea, vomiting, headaches, dizziness, and fullness in the ears [4–6,21,22] are also reported to exist after components of intense airborne ultrasonic exposure. The eye window is a functional vibration pathway through the brain to the ear via intracranial fluid conduction and could account for hearing loss, tinnitus, and the nonauditory subjective effects. Consistent with this concept, displacement of the oval and round windows as release mechanisms for fluid conduction to the cochlea could also account for the feeling of middle-ear fullness.

It was von Gierke [23] who first proposed that audible subharmonics of ultrasound and the middle ear (air volume or ossicular displacement or both) were somehow involved in the response of workers to ultrasonic exposure. Displacement of the round and oval windows as release mechanisms for eye-induced brain vibration could certainly interact with the ossicular chain, producing a detectable audio component. Such a middle ear–induced audio component would "sound" like a demodulation and could be misinterpreted as demodulation of the primary tone. There are, nonetheless, airborne sources as intrusion alarms, which do not have audible subharmonics that can induce audible and subjective effects. In a related report, swimming into a 50-kHz beam produced an audible perception, and merely immersing the jaw into water with a 50-kHz beam present produced a lower sensation [24]. A parsimonious explanation (not suggested by the authors) is that the eyes were responsive

for the lowered threshold of ultrasound detection when the head is submerged by providing an efficient route to the ear via the brain, as the acoustic impedance of the eye and seawater are very similar (see Table 1). An aftereffect of listening to the intense water-coupled tone was persistent tinnitus [24].

Tinnitus has commonly been associated with intense airborne ultrasound exposure [4–6], but quantification details are lacking. Prevention of hearing loss and tinnitus involves remaining uncoupled from ultrasonic vibrations. Sometimes the uncoupling can be difficult but, in the case of the ultrasonic eye window, closing it to dangerous levels of airborne ultrasound can be readily accomplished by the use of an acrylic barrier (i.e., goggles). This personal protection should provide ample safety; nonetheless, the current Occupational Safety and Health Administration regulations [25] regarding hearing safety in airborne ultrasound should be reviewed. Certainly our study sets forth a method of quantifying the transmission of airborne ultrasound into brain vibrations. Though no acoustic standards have been promulgated for measuring ultrasonic sound in human bone or fluid, guidance is available [26,27]. The lack of a mechanism for airborne sound to act on the ear directly has, no doubt, influenced the lack of research. Animal studies are limited in that the ratio of surface area to mass is small and that fur readily absorbs ultrasound.

Musical airborne energy beyond 22 kHz has been shown to alter the electroencephalogram and evoke physiological activity in the brainstem and thalamus but only when ultrasonic musical frequencies are combined with the musical spectrum below 22 kHz. The effect is based on the combination of two coherent acoustic routes, one conventional and one solely ultrasonic [7,8]. Each signal stimulates a separate area on the basilar membrane that would be integrated into a whole as any conventional complex auditory pattern. A case is made here for a separate airborne ultrasonic input, but the final pathway is the same because ultrasound activates the auditory cortex in normal-hearing and deaf listeners. Clearly, the eye, with its ultrasonic passband of 25–60 kHz, could transmit energy from instruments with ultrasonic energy (e.g., cymbals) to the ear and would activate both the auditory thalamus and the other nuclei in the auditory pathway. Very-high-frequency recordings (6–21 kHz) have activated the thalamus and other regions in the brain [28] in patients who have high-frequency tinnitus; thus, the thalamus plays a role in high-frequency and ultrasonic hearing. Musical instruments that have high-frequency and ultrasonic components are, for the most part, percussive; thus, the high audio and ultrasonic spectra would complement conventional audio frequencies consistent with the findings that the full spectrum is a better activator of the auditory system than is the ultrasound alone [7,8]. Ultrasound may contribute to pitch perception by extending the spectrum upward in frequency and by enhancing temporal cues. A simple test of the eye window's role in concert music would be to assess music quality with and without goggles. Goggles, as used as a control in this study, eliminated the eye window for airborne ultrasound.

In regard to music recording and reproduction, more than doubling the sampling rate (95 kHz/24 bits) will increase the frequency range, and a secondary effect likely will be an improvement in the phase linearity within the extended audio range; reducing quantizing errors, both factors will improve the quality of music. Personal headphones could be supplemented or replaced with bone conduction transducers with frequency responses >50 kHz. Such transducers are already in use for medical treatment of tinnitus [29,30] and can be readily modified for personal musical use (Fig. 4).

The eye is an airborne ultrasonic window to the ear and, as such, extends the range of human hearing. The mechanics of ocularly transmitted ultrasound should be similar in all other respects to ultrasound delivered to the skin of the head or neck as vibration [15], with the eventual activation of the auditory cortex in normal-hearing and deaf individuals [31,32]. If ultrasound is demodulated in the brain [15], the ear would be stimulated at or near brain resonance (14–15 kHz); thus, the auditory cortex that codes high audio frequencies will be involved. If the ultrasound acts directly on the base of the cochlea, just the tip of the basilar membrane would be stimulated [33], also resulting in high-frequency area activation of the auditory cortex [31]. In terms of region on the basilar membrane, there is not much difference: Brain resonance would produce a peak displacement

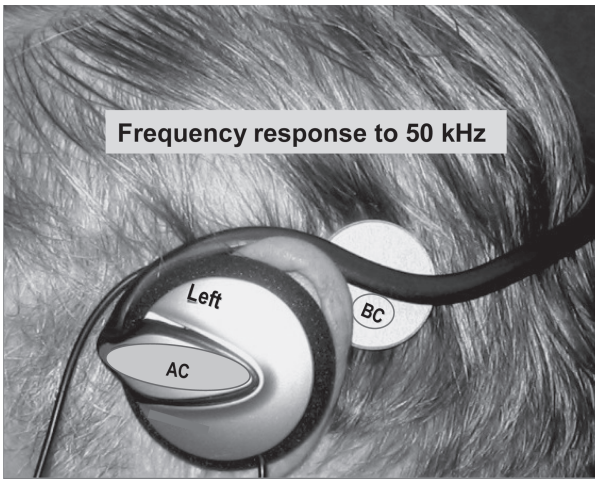

Figure 4. Frequency response to 50 kHz. (*AC* = air conduction; *BC* = bone conduction.)

1.9 mm from the base, and the tip displacement would move the first 0.5–1 mm [15]. The cilia of hair cells in the base of the basilar membrane are very short (5–10 μ), are immersed in fluid, and resonate in the ultrasonic frequencies. Cilia directly driven by ultrasound have been proposed as another possible mechanism in humans [34], and the concept was demonstrated in isolated turtle hair cells [35]. All methods would result in ultrasound's having a high pitch, as documented by studies over the last 60 years [15].

An intriguing concept in cortical audio processing is that some neurons with best frequencies and frequency responses in the mid-audio range may be activated by much higher frequencies [36]. Specifically, amplitude modulation (AM) outside of a neuron's response can be sufficient for activation. Because ultrasound is coded as high-frequency sound, there is no reason to believe that this is not a potential mechanism. Assuming the music scenario, conventional audio and ultrasound may drive some units, supporting the perception that ultrasonic components of music are important for apprehension.

An auditory cortical neuron's sharply tuned frequency response (inverted V shape) is depicted in Figure 5A. The frequency to which the neuron is most sensitive is the center frequency. Stimulation of high frequencies outside the frequency response has been shown to cause some neurons to fire. The stimulation in this case is in a form of AM that is a process in which one sound is multiplied by another. One sound is termed the carrier (C) and the other the modulator (M). The product of the multiplication is the carrier, the carrier plus the modulator (C+M), and the carrier minus the modulator (C–M). Simply put, three tones beyond the frequency range of some auditory cortical neuron will cause it to fire. AM stimulation may not be necessary to obtain this effect; it was merely the mode of stimulation in our study. Musical harmonic information is coded by place both on the basilar membrane and temporally in neural firing. Ultrasound might contribute to the musical harmonic structure but may just provide more high-frequency energy to emphasize the treble in such instruments as the cymbals, triangles, trumpets, violins, and oboes (see Fig. 5B).

Nonlinear distortion produced anywhere in the bone conduction (BC) transmission path could demodulate the AM ultrasound to audible frequencies [37,38]. However, BC vibration in the human head was found to be linear for levels equal to 77 dB HL for the frequencies [39], which argues against demodulation due to skull-brain nonlinearities, although no findings beyond 77 dB HL or 10 kHz have been reported. Ultrasonic skull vibration does vary from individual to individual on the basis of geometries [26], but this does not necessarily imply nonlinearities. The hair cells could act as a demodulator, but the most likely demodulator is the brain itself [15]. As mentioned earlier, the ossicles can be displaced by the oval-window movement with brain vibration, resulting in an audible tone near middle-ear resonance of 3 kHz, which is not primary demodulation of the ultrasound [23]. Finally, a tactile response to airborne ultrasound has been reported in the oral and nasal cavities if the mouth is open, likely owing to standing wave phenomena [4].

Propagating ultrasonic components of music toward the body induces brain and behavioral changes [7,8] but, when the body is sound-shielded, these changes are not observed [8]. Consistent with the current finding is that the eye is the input window into the ear for high-frequency airborne sound and music. There is no need to postulate an additional unknown somatosensory route to the ears [8]; nonetheless, the concept of multisensory coding in music perception is intriguing.

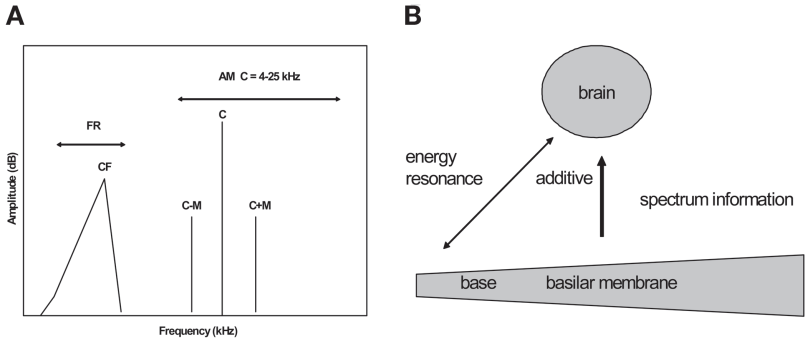

Figure 5. (A) Theoretical support for ultrasonic modulation of cortical neurons, based on the animal work of Schulze and Langer [37]. (B) This simple model is applied to musical encoding on the basilar membrane in which the spectral cues are coded by place of peak basilar membrane displacement, and the ultrasonic energy passed from the eye through the brain to the ear is coded as high-frequency energy. Both components act as complex sounds that will be summed in the neural axis in a conventional fashion (i.e., the airborne energy is added to the audiofrequency spectrum). Simply put, three tones beyond the frequency range of some auditory cortical neuron will cause it to fire. (*AM* = amplitude modulation; *C* = carrier; *CF* = center frequency; *FR* = frequency response; *M* = modulator.)

Is there any evolutionary advantage in having an eye that responds to light and intense ultrasound? A considerable body of literature reports on sound-vision interactions in humans with forward-facing eyes [40, 41]. Hearing is more accurate if the eyes look in the direction of the sound source. An auditory cue can trigger a visual shift in attention, and a louder cue is more effective than a softer one. Abrupt onset of sound can improve the detection of a subsequent flash, the opposite of masking [42]. So it appears that intense high frequencies that pass through the eye window as opposed to just surpassing the impedance barrier to the body [43] might indeed lead to multisensory coordination and, hence, survivability for humans, but there is a limit [24] that, if exceeded, may result in hearing loss and tinnitus.

CONCLUSIONS

The eye can serve as an acoustic window to the ear via the intracranial soft tissues. The frequency response is in the low ultrasonic range, and this type of hearing is termed *eye conduction*. Auditory and vestibular coding is postulated.

In regard to music recording and reproduction, more than doubling the sampling rate (95 kHz/24 bits) will extend the audible frequency range that can be coded in the eighth nerve and will result in a gain in linearity and reduction in quantizing errors, factors that will improve music quality.

Personal headphones could be supplemented or replaced with bone conduction transducers, with frequency responses extending to at least 50 kHz. Such transducers are already in use for medical treatment of tinnitus and can be readily modified for personal musical use (see Fig. 4).

Musical harmonic information is coded by place on the basilar membrane and temporally in neural firing. Ultrasound might contribute to the musical harmonic structure and provide more high-frequency treble emphasis in instruments, such as the cymbals, triangles, trumpets, violins, and oboes.

Eye conduction may contribute to industrial high-frequency loss and tinnitus because ultrasound and high audio frequencies overlap in terms of basilar membrane displacement patterns, especially if high audio subharmonics are generated by intense industrial devices. Simultaneous activation of the saccular and cochlear nerves likely contributes to the symptoms of "ultrasonic sickness."

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REFERENCES

1. Parker PD, Tubbs RL, Littlefield VM. Visual field displacements in human beings evoked by acoustic transients. *J Acous Soc Am* 63(6):1912–1918, 1978.
2. Jones PD, Loeb M, Cohen A. Effects of intense continuous- and impact-type noise on pupal size and visual acuity. *J Am Aud Soc* 2(6):202–207, 1977.
3. Gorig C, Varghese T, Stiles T, et al. Evaluation of acoustic wave propagation velocities in the ocular lens and vitreous tissues of pigs, dogs, and rabbits. *Am J Vet Res* 67(2):288–295, 2006.
4. Parrack HO. Effects of airborne ultrasound on humans. *Int Aud* 5:294–308, 1966.
5. Grzesik J, Pluta E. High-frequency hearing risks of operators of industrial ultrasonic devices. *Int Arch Occup Environ Health* 53(1):77–78, 1983.
6. Grzesik J, Pluta E. Dynamics of high-frequency hearing loss of operators of industrial ultrasonic devices. *Int Arch Occup Environ Health* 57(2):137–142, 1986.
7. Oohashi T, Nishina E, Honda M, et al. Inaudible high frequency sounds affect brain activity: Hypersonic effect. *J Neurophysiol* 83(6):3548–3558, 2000.
8. Oohashi T, Kawai N, Nishina E, et al. The role of biological system other than auditory air-conduction in the emergence of the hypersonic effect. *Brain Res* 1073:339–347, 2006.
9. Snow WB. Audible frequency ranges of music, speech and noise. *J Acoust Soc Am* 3(1):44–51, 1931.
10. Boyk J. There's life above 20 kilohertz! A survey of musical instrument spectra to 102.4 kHz. Obtainable from www.cco.caltech.edu/~boyk.
11. Ranke OF. Physiologie des Gehors. In OF Ranke and L Lullies (eds), *Gehor Stimme Sprache*. Berlin: Springer, 1953:3–110.
12. Tonndorf J, Tabor JR. Closure of the cochlear windows. *Ann Otol Rhinol Laryngol* 71:5029–5035, 1962.
13. Sohmer H, Freeman S, Geal-Dor M, et al. Bone conduction experiments in humans—a fluid pathway from bone to ear. *Hear Res* 146:81–88, 2000.
14. Sohmer H, Freeman S. Further evidence for a fluid pathway during bone conduction auditory stimulation. *Hear Res* 193:105–110, 2004.
15. Lenhardt ML. Ultrasonic hearing in humans: Applications for tinnitus treatment. *Int Tinnitus J* 9(2):69–75, 2003.
16. Harris G. Piezoelectric polymer probe for ultrasonic applications. *J Acoust Soc Am* 69(3):853–859, 1981.
17. Herbertz J. Loudness of airborne ultrasonic airborne noise. In *Ultrasonics International Conference Proceedings* 1984:S226–S231.
18. Duck FA. *Physical Properties of Tissues: A Comprehensive Reference Book*. New York: Academic, 1990.
19. Zemanek J. Beam behavior within the near field of a vibrating piston. *J Acoust Soc Am* 49(1):181–191, 1971.
20. Richards DG, Lenhardt ML, Madsen AG. Non-invasive

acoustic measurement of intracranial pressure. In J Vossoughi (ed), *Biomedical Engineering: Recent Developments*. Washington, DC: Medical and Engineering Publishers, 2002:41–42.

21. Davis H. Biological and physiological effects of ultrasonics *J Acoust Soc Am* 20:605–607, 1948.
22. Crabtree RB, Forshaw EE. Exposure to Ultrasonic Cleaner Noise in the Canadian Forces. DCIEM Technical Reports No 77x45. Ottawa Department of National Defense, 1977.
23. von Gierke HE. Subharmonics generated in human and animal ears by intense sound. *J Acoust Soc Am* 22:675–679, 1950.
24. Deatherage BH, Jeffers LA, Blodgett HC. A note on the audibility of intense ultrasonic sound. *J Acoust Soc Am* 26:582, 1954.
25. Occupational Safety and Health Administration. *Technical Manual*, sec. III, chap. 5, subchap. V: Ultrasonics. Washington, DC: US Department of Labor, 2002.
26. Cai Z, Richards DG, Lenhardt ML, Madsen AG. Response of human skull to bone conducted sound in the audiometric to ultrasonic range. *Int Tinnitus J* 8(1):1–8, 2002.
27. Lenhardt ML, Richards DG, Madsen AG, et al. Measurement of bone conduction levels for high frequencies. *Int Tinnitus J* 8(1):9–12, 2002.
28. Shulman A, Strashun AM, Avitable J, et al. Ultra-high-frequency acoustic stimulation and tinnitus control: A positron emission tomography study. *Int Tinnitus J* 10(2): 113–125, 2004.
29. Lenhardt ML, Goldstein BA, Shulman A, Guinta R. Use of high frequency and muscle vibration in the treatment of tinnitus. *Int Tinnitus J* 9(1):32–36, 2003.
30. Goldstein BA, Lenhardt ML, Shulman A. Tinnitus improvement with ultra-high-frequency vibration therapy. *Int Tinnitus J* 11(1):14–22, 2005.
31. Hosoi H, Imaizumi S, Sakaguchi T, et al. Activation of the auditory cortex by ultrasound *Lancet* 351:496–497, 1998.
32. Imaizumi S, Hosoi H, Sakaguchi T, et al. Ultrasound activates the auditory cortex of profoundly deaf subjects. *Neuroreport* 12(3):583–586, 2001.
33. Tonndorf J, Kurman B. High frequency audiometry. *Ann Otol Rhinol Laryngol* 93:576–582, 1984.
34. Grant JW, Lenhardt ML. Distributed ultrasonic resonance in hair cells. *Assoc Res Otolaryngol Abstr* 413, 1992.
35. Lenhardt ML, Grant JW, Merkle A. Vibration induced cilia bundle movement. *Assoc Res Otolaryngol Abstr* 345, 1999.
36. Schulze H, Langer G. Auditory cortical responses to amplitude modulations with spectra above frequency receptive fields: Evidence for wide spectral integration. *J Comp Physiol [A]* 185:493–508, 1999.
37. Westervelt PJ. The theory of steady forces caused by sound waves. *J Acoust Soc Am* 23(4):312–314, 1951.
38. Westervelt PJ. Parametric acoustic array. *J Acoust Soc Am* 35(4):532–537, 1963.
39. Hakansson B, Brandt A, Carlsson P, Tjellstrom A. Resonance frequency of the human skull *in vivo*. *J Acoust Soc Am* 95:1474–1481, 1994.
40. McDonald JJ, Teder-Salejarvi WA, Hillyard SA. Involuntary orientation to sound improves visual perception. *Nature* 407:906–908, 2000.
41. Stein BE, London N, Wilkinson LK, Price DD. Enhancement of perceived visual intensity by auditory stimuli: A psychophysical analysis. *J Cogn Neurosci* 8:497–506, 1996.
42. Shams L, Kamitani Y, Shimojo S. Visual illusion induced by sound. *Cogn Brain Res* 14:147–152, 2002.
43. Zwislocki J. In search of the bone conduction threshold in a free sound. *J Acoust Soc Am* 29:795–804, 1957.