Ultrasonic Hearing in Humans: Applications for Tinnitus Treatment

Martin L. Lenhardt, Au.D., Ph.D. Professor
Departments of Otolaryngology and Emergency Medicine, Virginia Commonwealth University, Richmond, VA

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Reprint requests: Dr. Martin L. Lenhardt, Box 980168, Virginia Commonwealth University, Richmond, VA 23298-0168. Phone: 804-828-9687; Fax: 804-828-4454; E-mail: lenhardt@mail2.vcu.edu

Abstract: Masking of tinnitus is possible using high audio frequencies and low-frequency ultrasound. The mechanisms involved in reception and perception of both audio frequencies and ultrasound are identical with the exception that ultrasound interacts with an intermediary site, the brain. We proposed brain ultrasonic demodulation as the means of place-mapping ultrasound on the first few millimeters of the basilar membrane. We present modeling and psychoacoustic data in support of this theory.

Key Words: high-frequency hearing thresholds; masking; neuroplasticity; pitch; tinnitus

A new trend in tinnitus treatment is the use of very-high-frequency maskers, including ultrasound, delivered by bone conduction [1,2]. Masking and long-term inhibition may involve inducing plastic changes in the brain at the central level. The application of high audio frequencies (10–20 kHz), which stimulate the base of the cochlea through bone conduction, is a conventional auditory technique. The peripheral site of action of low-frequency ultrasound is more problematic but, in this study, we argue that the ultrasonic transduction is also parsimonious with conventional auditory theory, with the exception of its demodulation.

Ultrasonic Hearing Background

Human ultrasonic hearing has been independently "discovered," documented, and abandoned more than a dozen times over the last half century [3]. So outlandish is the concept that humans can have the hearing range of specialized mammals, such as bats and toothed whales, that ultrasonic hearing has generally been relegated to the realm of parlor tricks rather than being considered the subject of scientific inquiry. In one of the earliest reports [4], the experimental work of Dr. Roger Maass performed in 1946 was cited. Maass, never credited again for his original discovery, made all the essential observations in regard to ultrasonic hearing phenomenology. Ultrasonic hearing was possible in humans but only by bone conduction. Some deaf patients could hear ultrasound, albeit at levels higher than those in normal listeners, and frequency discrimination was possible, although far poorer than in the audio range.

Building on these observations, Lenhardt et al. [3] confirmed for the first time that speech discrimination in deaf subjects was possible using modulated ultrasound. More recently, the primary auditory cortex has been implicated, using magnoelectroencephalography, in processing ultrasound tonotopically, consistent with very-high-frequency hearing [5]. The same authors confirmed ultrasonic hearing and

ultrasonic speech perception in approximately one-half their deaf subjects. In the case of the deaf, a low-frequency cortical site for ultrasound was observed [6]. In contrast, this suggests an apical cochlear site or other noncochlear acoustic site that is activated in the presence of severe deafness. The implication is that different sites on the inner ear's basilar membrane are activated, depending only on the proportion of hair cells present.

In 1954, Deatherage et al. [7] were the first to warn of the potential damage (high-frequency hearing loss and tinnitus) of listening to very intense, head-coupled ultrasound; however, their studies resulted in two key observations. The pitch of the ultrasound corresponded to the highest audio frequency measured audiometrically by air conduction and the same ultrasonic tone could give rise to different pitch perception in each ear (i.e., diplacusis), if there was a hearing difference between ears. Taken together, these findings suggest the absence of a specific place of ultrasonic frequency mapping on the cochlea but a dynamic mechanism that is a function of remaining hearing or hair cells and intensity of the ultrasonic stimulation. Ultrasound would then be unique as a form of auditory stimulation that any listener with any degree of hearing loss could detect given sufficient ultrasonic energy. Ultrasound refinement could lead to new types of audio amplification, auditory orientation (echolocation), and tinnitus treatment, if the mechanism can be clearly elucidated.

The dynamic aspect of ultrasonic pitch suggests that the ear may not be directly stimulated but rather that an intermediary structure may be involved. Three lines of evidence suggest that the resonance of the brain is critical for an audible ultrasonic experience. Support for a brain ultrasound demodulation theory stems from spherical models of brain and psychoacoustic metrics of masking audio frequencies by ultrasonic noise and by matching the pitch of audible ultrasound with conventional air conduction sound.

Brain Resonance

The resonant frequency of the brain can be calculated, assuming the brain is spherical and consists of only brain matter [8–10] using the formula

$$F = {}^{c}/_{2\eth r}$$

where F is the fundamental frequency of the sound generated inside the sphere, c is the velocity of sound in brain tissue (1.46×10^5 cm/sec) [8,9], and r is the radius of the sphere. Using a 7-cm radius as a representation of the human average, the fundamental resonant frequency is calculated to be 13.4 kHz. The human head is not exactly spherical, so this is only an approximation. Further, the brain has a boundary condition: the skull and skin. Modeling the brain with such a boundary condition, the F is reported to be approximately 70% higher [8]. Boundary condition calculation, using a constant (k), results in F = 15.6 kHz. For all practicality, the exact resonant frequency of a brain with a 7-cm radius is probably a number between the free and boundary states. Applying a microwave burst to a 7-cm radius sphere containing fluid with properties similar to those of brain results in a measured peak frequency of 11.4 kHz. Microwave absorption causes a rapid heating that launches a pressure wave within the sphere. If the head is exposed to such microwave stimulation, the fundamental brain frequency is proportional to the brain size. A human infant would have a resonant frequency between 16 and 22 kHz assuming a 5-cm radius. Cats have a calculated brain resonance between 30 and 40 kHz, with head radii varying from 2.5 to 3.5 cm, and microwave exposure results in a measured

resonance of 38 kHz [11]. A hydrophone implanted in the brain of a cat recorded peak pressure at 39 kHz as a result of microwave pulses [11]. For humans, hearing in excess of 8 kHz is necessary to detect microwave absorption [8–12]. As deduced from the foregoing data, the human brain resonant frequency is likely between 11 and 16 kHz, with the exact value being determined by individual skull geometry. The human data are summarized in the upper portion of Table 1.

Ultrasonic Masking

The brain ultrasonic demodulation theory implies that the sound produced by brain resonance will propagate to the ear via fluid channels. Such channels have been proposed by Ranke [13] and confirmed experimentally by Tonndorf [14,15]. Direct vibration of the brain, but not skull, is sufficient to record auditory evoked potentials [16–18], thus verifying the brain-ear mechanism. The brain is in constant motion (i.e., it pulses approximately 2 Hz) with the contraction and relaxation of the heart. Monitoring neural vascular function with Doppler ultrasonic imaging provides unexpected support for the brain ultrasound demodulation theory. When the imaging beam was focused at the center of the brain, patients reported hearing a high audio sound, much like tinnitus. When the ultrasonic beam was directed at the ear, the sound disappeared [19]. Setting the brain into resonance resulted in a clear, high-pitch, audible sensation consistent with brain resonance in the 11- and 16-kHz range. The use of imaging ultrasound has had mixed success [20,21], but the critical feature in failure might be not focusing the beam at the brain's center to maximize brain ultrasonic demodulation.

The application of ultrasonic noise to the skull by bone conduction should produce cochlear masking in frequencies corresponding specifically to the brain's resonant frequency, raising the question of how brain resonance interacts with conventional hearing. Ultrasonic masking has been reported to suppress audio thresholds in the 8- to 12.5-kHz range by 2–29 dB [22,23]. This experiment was repeated using seven normal hearing listeners with no history of otological disease. Audiometric thresholds were obtained in the conventional audiometric frequencies and extended through the high audio frequencies (10–20 kHz) using a Virtual 310 audiometer (Portland, OR). Thresholds were repeated in the presence of ultrasonic masking noise, with peak energy centered at either 26 or 39 kHz. Both maskers were delivered at a 5-dB sensational level (SL). The ultrasonic masking data are summarized in Table 2.

Ultrasound has a maximal masking effect in the 12.5- to 16-kHz range. Masking was less for frequencies higher and lower than the resonance peak, which is characteristic of a resonance phenomena. Note also that the masker with noise centered at 39 kHz was less effective, as it is almost two octaves above the fundamental brain frequency (see Table 2). Recall that 26- and 30-kHz center noise maskers were both at an equal SL (5 dB); therefore, the relative energy is not a principle factor but only its relationship to the fundamental frequency of the brain being forced into vibration.

Pitch Match

For more than half a century, ultrasound has been reported to be associated with the pitch of air-conducted sound in the range of 8 to 16 kHz and often has been cited to be directly related to the highest frequency audible by a listener [3]. Ultrasonic hearing is unique in that even moderate changes in frequency (< 10%) are not associated with the dramatic

pitch changes as in the conventional audiometric range. Further pitch generally asymptotes approximately at the air-conduction match of some 16 kHz, rendering judgments difficult [3,24,25].

Deatherage [7] first noted an ultrasonic diplacusis; that is, the pitch associated with ultrasound could be different in each ear if hearing asymmetries were present. Pitch matches in the 8-16 kHz air conduction range and diplacusis are consistent with the hypothesis of brain ultrasonic demodulation (i.e., resonant oscillation is communicated via the vascular channels to both inner ears independently via the third windows of Ranke) [13]. Thus, the brain stimulates each ear equally, but the frequency response of each ear contributes to the perception of pitch.

A pitch-matching experiment was carried out using 10 otologically normal young adults. Each subject matched an air-conducted pure tone (variable frequency) with an ultrasonic tone of 26 or 30 kHz delivered at 5 dB SL. The mean match of five trials was tabulated for each ultrasonic frequency. The pitch data are summarized in Table 2. Note that the masker centered at 1 octave above the brain fundamental (26 kHz) produces more masking and a wider range of masking than does the masker approximately 2 octaves above (39 kHz). Thus, ultrasound will map in the high audio frequencies, because the brain is the demodulator regardless of the stimulating ultrasonic frequency. The higher the ultrasonic frequency is in regard to the brain resonance, the more energy is required to set the brain into forced resonance, accounting for the wider spread of masking for 26 versus 39 kHz (Fig. 1).

If the ultrasonic frequency does not determine the perceived pitch but rather the resonance of the brain, how is the reported pitch discrimination in the ultrasonic range possible [3,5], or is it absent [24–26]? Note in Figure 1 that a change in the center frequency of the noise, even at a constant intensity (5 dB SL), results in a different frequency or place spread of activation. The change in the area activated and the subsequent neural coding likely contributed to crude detection of pitch change. Further, the multiple coupled resonances of the brain and skull can yield subtle intensity cues [27,28] but, in any event, changes of perhaps 10% of the frequency are required in the ultrasonic range to be detected as a pitch shift [3].

The effect of airborne ultrasound on hearing has not been thoroughly studied, but young normally hearing adults seem at greater risk for very-high-frequency hearing loss. The Occupational Safety and Health Administration (OSHA) has adopted ultrasonic hearing protection working standards [29]. Ultrasonic cleaners and related technology (ultrasonic welders, etc.) are an industrial source of high audio and ultrasound of sufficient energy to induce hearing loss. The ultrasound-induced hearing loss [30] is plotted in Figure 1. Note the close correspondence of the maximal hearing loss and frequencies within the brain's resonance. The concept of brain ultrasonic demodulation as applied to industrial exposure is unique and begs for further exploration. In addition, the presence of both high audio frequencies and ultrasound in the workplace can increase the ear damage risk, as a place-coding overlap exists on the basilar membrane (see details later).

The correspondence of the results of brain resonance, ultrasonic masking, and ultrasonic pitch-matching data are summarized in Figure 2. The position of maximal stimulation of each on the basilar membrane can be calculated using a formula devised by Fay [31]:

$$P = \frac{\log 10 [\ fHz/.008\ fHz\ max] + 1^{*}}{2.1}$$

where *P* is the proportion of base to apex on the basilar membrane, *fHz* is the frequency of interest, and *fHz max* is the maximal audible frequency by air conduction in young adults.

Further, the position on the basilar membrane is determined by:

$P = 1 - P \times$ *cochlear length* (31.5 mm)

The positions on the basilar membrane for brain resonance using the boundary condition model, masking with 26 kHz center-frequency noise, and mean pitch match at 26 kHz are 15.6 kHz: 1.6 mm; 16 kHz: 1.5 mm; and 11 kHz: 3.9 mm respectively. These data clearly support the hypothesis of physical demodulation of ultrasound by brain resonance and the detection of this resonance in the base of the basilar membrane.

Alternatively, just the basal tip of the basilar membrane has been proposed as the site of ultrasonic transduction [7,32]. One-dimensional modeling of the cochlea, incorrectly assuming both scales have equal volumes, results in the prediction that ultrasound activates the first few millimeters (0–7) of the cochlear base. Although some overlap is seen with the brain ultrasound demodulation place of transduction in the cochlea, the mechanism is assumed to be direct stimulation via bone conduction. The model also predicts multipeaked or “slurred” waves higher than 20 kHz, rather than a conventional single traveling wave peak [33]. This model would not predict a peak frequency for masking or pitch matching in the range observed as does the brain ultrasound demodulation theory.

The spherical modeling, masking, and pitch data are summarized in Figure 2. Substantial agreement supports the theory that ultrasound, regardless of its frequency, stimulates an area on the basilar membrane that codes the fundamental resonant frequency on the brain. The psychoacoustical findings, despite the methodological differences, are also consistent with the brain ultrasound demodulation theory.

Applications to Tinnitus

High- and very-high-frequency stimulation were reported recently to be effective in tinnitus masking [1,2]. Plastic changes in the auditory neural axis in severe tinnitus, particularly in the auditory cortex, may play a role in the continued perception of tinnitus by adding salience to the experience [34,35]. Plastic changes reflecting neural reorganization secondary to peripheral hearing loss have also been well documented [36]. Characteristically, neurons sensitive to a damaged cochlear region will reprogram their best frequencies either higher or lower, resulting in greater neural representation of frequencies on the “edge” of the damaged peripheral region. In the only tinnitus-imaging study of neural reprogramming [34], the tinnitus frequency area expanded (more than twice the size) with some hint of lower-frequency expansion below the tinnitus frequency, as expected subsequent to hearing loss. This pattern was striking in only four of the subjects, and the mean high-frequency hearing loss for all subjects was just outside the normal range. Possibly, more hearing loss was necessary to trigger reprogramming in the subjects. What is encouraging is that neural reprogramming in tinnitus possibly can be reversed by increasing high-frequency stimulation (i.e., with frequencies above the tinnitus frequency), for auditory learning in primates has been shown indeed to expand the frequency map [37,38]. High-frequency stimulation (high audio and ultrasound) have

been shown to mask tinnitus and produce varying degrees of residual inhibition [1,2]. Because ultrasound produces high audio stimulation by virtue of brain resonance, the direct use of high audio stimulation is more economical in power requirements and still stimulates the brain at resonance.

Applications to the Deaf with Tinnitus

With progressive high-frequency hearing loss, pitch perceptual range collapses. Ultrasonic thresholds also increase with the degree of high-frequency hearing loss. Presumably, the increased ultrasonic energy increases the displacement spread on the basilar membrane toward the apex, thus accounting for the observation that the ultrasonic pitch is related to the highest frequency detectable by air conduction. In the case of severe deafness, with ultrasonic thresholds approximately at 30 dB above normal, insufficient surviving hair cells exist in the apex to detect the basilar membrane motion. Lenhardt et al. [3] argued that the saccule may be stimulated in the case of severe deafness. Assuming that maximal displacement of the basilar membrane is at the place corresponding to the brain's fundamental frequency (1.5 mm from the base for 26 kHz), bulk inner-ear fluid displacement, permitted by compliant oval and round windows at very high intensities (100 SL+), could conceivably create fluid flow in the saccule, not unlike the Tullio effect [39]. The very short cilia of saccular hair cells, not mass-loaded by gel or otoconia [40] in the striola region, are likely fluid velocity–sensitive. Stimulating the saccule, an organ having input into the auditory pathways in mammals [37,41,42], may explain ultrasonic detection in the deaf. Anecdotally, a graduate engineering subject who had no measurable hearing and who was an unsuccessful cochlear implant user described the perception of 26 kHz and an alternating knocking in her head. The cilia of hair cells have opposite orientation on either side of the saccular striola, which could neurally code the alternating phase of a sine wave as an alternating knocking. Other subjects reported unsteadiness when rising from a seated position after ultrasonic listening, which also was suggestive of saccular stimulation. Clearly, more work must be conducted to elucidate the source of nonauditory coding of ultrasound in humans, but complete loss of hearing and balance seems to preclude its detection. No published reports have cited the use of ultrasound in treating tinnitus in severely deaf individuals in spite of a high incidence of tinnitus in this population [43].

Calibration

Bone conduction calibration is always an issue when thresholds are determined. Calibration procedures are based in part on bone-anchored measurements (in the high audio frequencies of 8–20 kHz) [3]. Ultrasonic bone conduction threshold standards do not exist [44]. However, two approaches have been used: sound pressure in water and acceleration. Placing a transducer on the water surface and measuring the pressure output in a small tank can be complicated, even when brain and water impedances are similar [24]. High-frequency accelerometers have impedances closer to bone than to brain. Acceleration, measured in meters per second squared (m/sec^2), is simpler to apply than force, which is the audiometric unit of choice [45,46]. Nonetheless, the hearing threshold for bone conduction can be referenced as –30 dB relative to 1 m/sec^2 from 0.25 to 6 kHz. Zero decibel Hearing Level (HL), and this reference can be applied to higher frequencies, including ultrasound. A standard point of measurement is 1 gravity unit (g) rms (9.81 m/sec^2), a reasonable intensity mark in conducting studies with high

frequencies, and is a point of reference in the OSHA hearing protection standard for body-coupled ultrasound [29].

Speech, Echolocation, and Evolution

If speech is modulated on an ultrasonic carrier, speech monitoring and perception with a high degree of comprehension (approximately 80%) is possible [3,47], even in high ambient noise. This result opens the possibility of using ultrasonic speech for communication under poor listening conditions and in situations wherein traditional amplification is ineffective. Echolocation is also possible by direct listening to the reflected ultrasonic echoes in the high audio range, after brain ultrasonic demodulation, which, owing to its frequency separation, will not interfere with speech communication.

Deatherage swam into a 50-kHz beam and first discovered underwater audible ultrasound 50 years ago [7]. It seems reasonable to expect that any mammal can detect underwater ultrasound by brain ultrasound demodulation. The pitch would be a function of brain geometry (spheroid or tubal). In fact, what humans experience as ultrasonic perception may have been a necessary precursor in the evolution of echolocation specialization in marine mammal ears and brains.

Summary

Humans can detect ultrasound up to at least 100 kHz, but perception generally requires direct contact of the source with the body. Ultrasound sets the brain into forced vibration, and it is the brain oscillation that is detected on the base of the cochlea in normally hearing individuals. With hearing loss, greater ultrasonic energy is needed to spread the displacement on the basilar membrane toward the region of intact hair cells. Ultrasonic pitch is not related to the stimulating frequency but rather to the remaining high audio frequency ability of the listener. In the case of complete deafness, the increased ultrasonic energy likely displaces the otolith organs, resulting in saccular stimulation. Indirect high audio frequency stimulation (ultrasound) or direct high-frequency stimulation can be an effective masker in tinnitus and can contribute to long-term tinnitus relief through neural repogramming. What must be emphasized is that the auditory perception is only one component in a complex tinnitus neural circuit that Shulman [48] identified as “the final common pathway.”

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Table 1. Summary Data for the Results of Spherical Modeling, Ultrasonic Masking of Audio Frequencies, and Pitch Matching

Audio Response	**Head Radius**	
	5 cm	**7 cm**
Stress-free model	16.0	11.4 kHz
Boundary condition model	21.9	15.6
Mean	18.9	13.5
Range		8–19
Masking range		8–19
Pitch range		8–15

Note: All the values fall between 8 and 22 kHz.

Table 2. Summary of the Masking Functions for Ultrasonic Noise Centered at 26 and 39 kHz

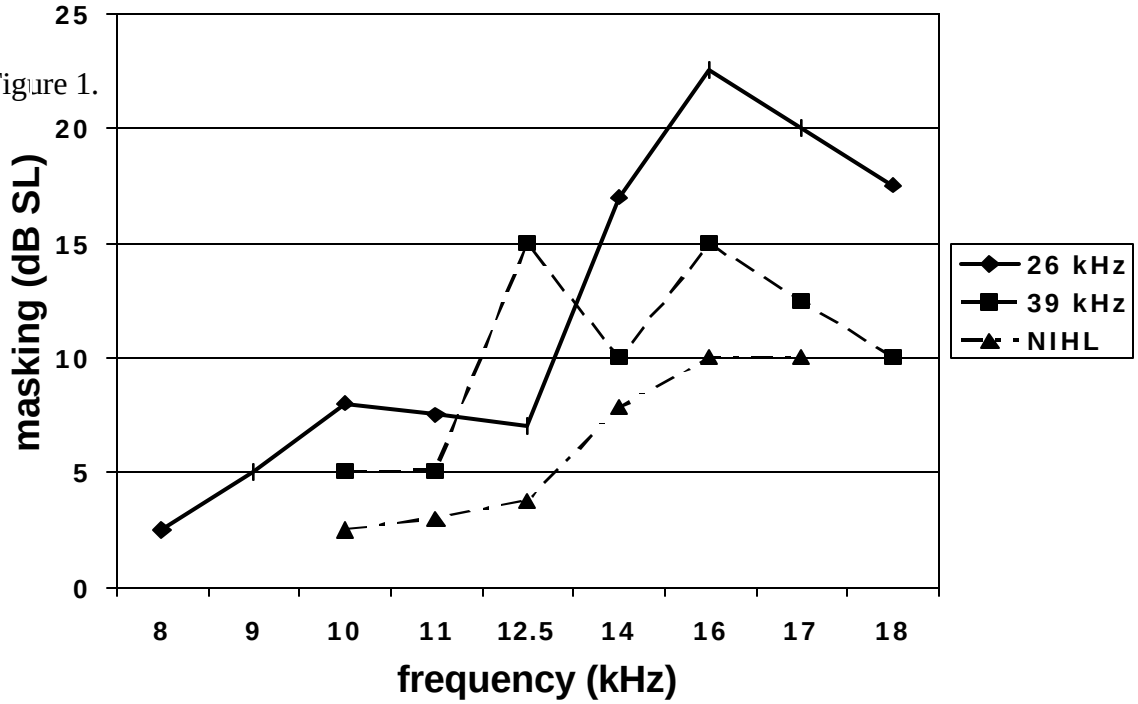
Audio Response	**Ultrasonic Center Frequency**	
	26	**39 kHz**
Maximum masking		
Frequency	16 kHz	12.5 kHz
Intensity	25 dB	15 dB
Masking range (frequency)	9–19 kHz	8.5–18 kHz
Pitch-match range (frequency)	8–15 kHz	9–14 kHz

Note: The data fell between 8 and 19 kHz.

Figure 1. The effects of ultrasound on audio frequency thresholds depicted for the two ultrasonic sources. Both were delivered at 5 dB Sensation Level (*SL*). Note that the 26-kHz noise resulted in an increase in frequencies as well as masker threshold shift as compared with 39 kHz. This effect represents the frequency distance from the fundamental resonance of the brain. Note the close correspondence of the masking curves for 26 and 39 kHz centered ultrasonic noise and the Noise Induces Hearing Loss (NIHL) composite audiograms reported for industrial worker exposed to intense ultrasound [30]. The masking, at 5 dB SL, was equal to 150 dB Sound Pressure level (SPL) re: 1 Pa (micro Pascal) in water for 26 kHz and 155 dB SPL for 39 kHz.

Figure 2. Summary of the data from modeling, spherical measurement, masking, and pitch match, supporting the theory of ultrasound brain demodulation in the high audio frequencies.

Figure 1.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frequency (kHz)
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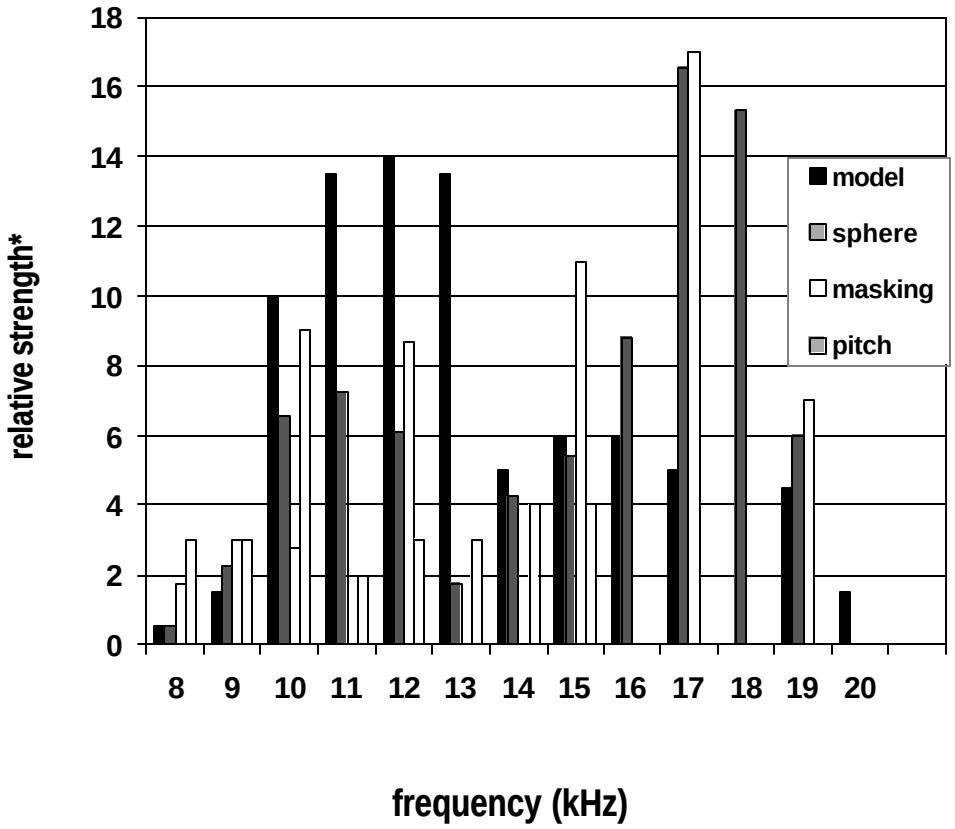
39 kHz
NIHL

Figure 2.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