TRANSMISSION OF MICROWAVE-INDUCED INTRACRANIAL SOUND TO THE INNER EAR IS MOST LIKELY THROUGH CRANIAL AQUEDUCTS

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Abstract

The most frequently cited sequence of events used to explain auditory sensations resulting from microwave pulses, or "microwave hearing", starts with transduction of microwave energy to sound in the head. In this explanation, the sound is then transmitted through cranial bones, i.e., by bone conduction, to stimulate hair cells in the inner ear. Recently reported experiments with animals and humans indicate that sound conduction through bone itself is not necessary in bone-conduction hearing. Instead, sound generated inside the cranium is most efficiently transmitted through holes in the cranium that form channels to the inner ear: vestibular aqueduct, cochlear aqueduct, and/or perivascular and perineural spaces. The short latency of cochlear microphonics reported for microwave hearing and the oscillation of the microphonics at the calculated brain resonant frequency are consistent with transmission through the channels. Thus, the channels are the most likely pathway for transmission of sound to the inner ear in microwave hearing. Consideration of this transmission pathway may be useful in reconciling results from various microwave hearing experiments.

Introduction

Microwave hearing is the auditory perception of microwave pulses impinging on the head, which has been reviewed in the literature [1-3]. The chain of events described in these reviews starts with generation of acoustic energy (sound) in the head. The sound produced is transmitted by bone conduction to the inner ear where it stimulates auditory receptors in the cochlea. The resulting neural signal is then processed normally by the auditory nervous system. Understanding processes involved in microwave hearing is important because of the use of microwave hearing thresholds in setting limits for human exposure to microwave pulses [4].

Fig. 1 shows principal components of the mammalian inner ear for reference and locations of the components within the temporal bone. The figure is adapted from images created by Alec N. Salt, Washington University [http://oto.wustl.edu/cochlea/intro1.htm].

Fig. 2 emphasizes acoustic pathways in a schematic diagram of the inner ear and nearby tissues. The cochlear aqueduct connection with the cochlea is much closer to the middle ear than depicted in this figure. Cranial contents are represented by brain tissue and cerebral spinal fluid (CSF) in Fig. 2. Distance between CSF and the temporal bone is exaggerated in both figures for clarity.

The most likely mechanism for transduction of a pulse of microwave energy to sound in tissue is thermoelastic expansion during the pulse [1-3,5-7]. Mathematical models of thermoelastic expansion in spherical heads having the dielectric properties of brain tissue predict that the generated sound has a fundamental resonant frequency determined only by head size [3,6]. Characteristics of sound measured in spherical tissue models and animal heads of different sizes are consistent with the prediction [7-9]. In addition, the round-window cochlear microphonic, which represents the acoustic waveform in the cochlea, recorded in animals in response to a microwave pulse oscillates near the calculated resonant frequency for the head being exposed [2,3,10,11].

Reports of and reviews on microwave hearing that mention bone conduction of sound to the inner ear [1-3,5,12] do not distinguish among the several known mechanisms of bone conduction. Stimulation of auditory receptors in the cochlea by bone conduction, differentiated from stimulation by air condution through the external meatus, was previously thought to occur through the following three pathways: (1) relative motion of inner ear contents due to inertial lag, (2) relative motion of inner ear contents due to distortion of the bony cochlear shell, and (3) coupling of energy to air in the external meatus [13,14]. These pathways, all of which depend on sound transmission in bone, are depicted in Fig. 2 by two curved arrows originating near the bone stimulator, a device commonly used to elicit auditory responses by bone conduction. The solid arrow directed to the cochlea represents the first two pathways. The dashed arrow directed to the external meatus-middle ear region is dashed to indicate the minor contribution of the third pathway to bone-conduction hearing unless the

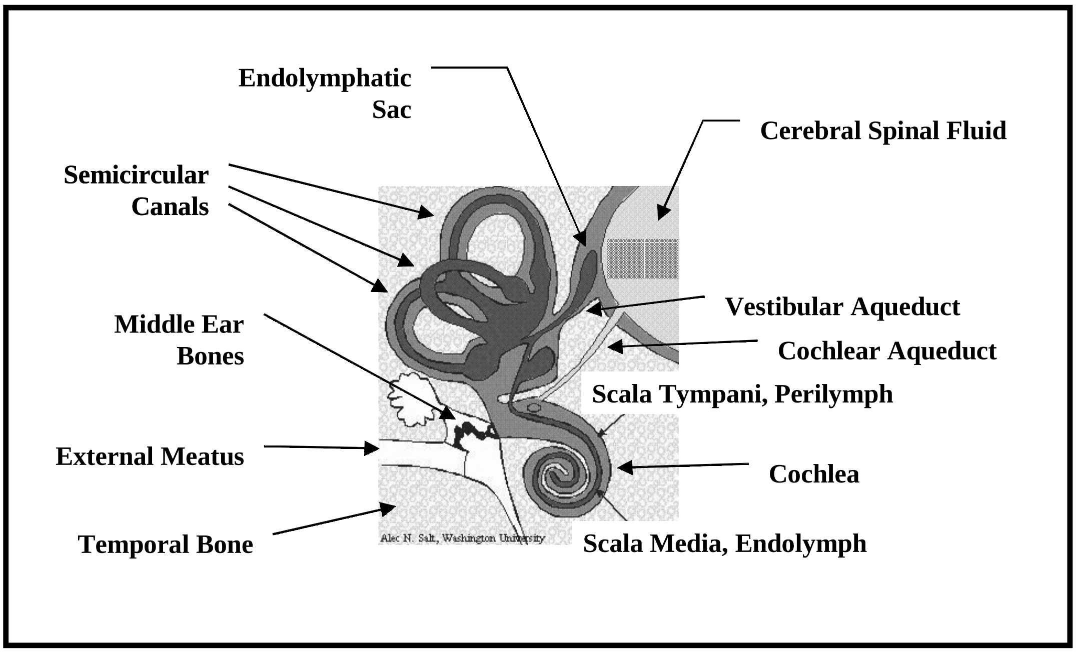

Figure 1. A diagram of the mammalian inner ear.

external meatus is blocked. Because microwave hearing literature sites work on bone-conduction hearing tested with a bone stimulator [15], a pathway through bone of the type represented by the solid arrow from the bone stimulator and to the cochlea in Fig. 2 was probably intended. Of course, when the source of sound is inside the cranium, the pathway through bone originates at the interior surface of temporal bone rather than the exterior.

An additional pathway for sound transmission to the inner ear by bone conduction has recently been discovered in experiments on rodents and humans [14,16]. This pathway is not through bone tissue itself but through channels that connect cranial contents with the inner ear. This pathway is shown in Fig. 2 as a curved

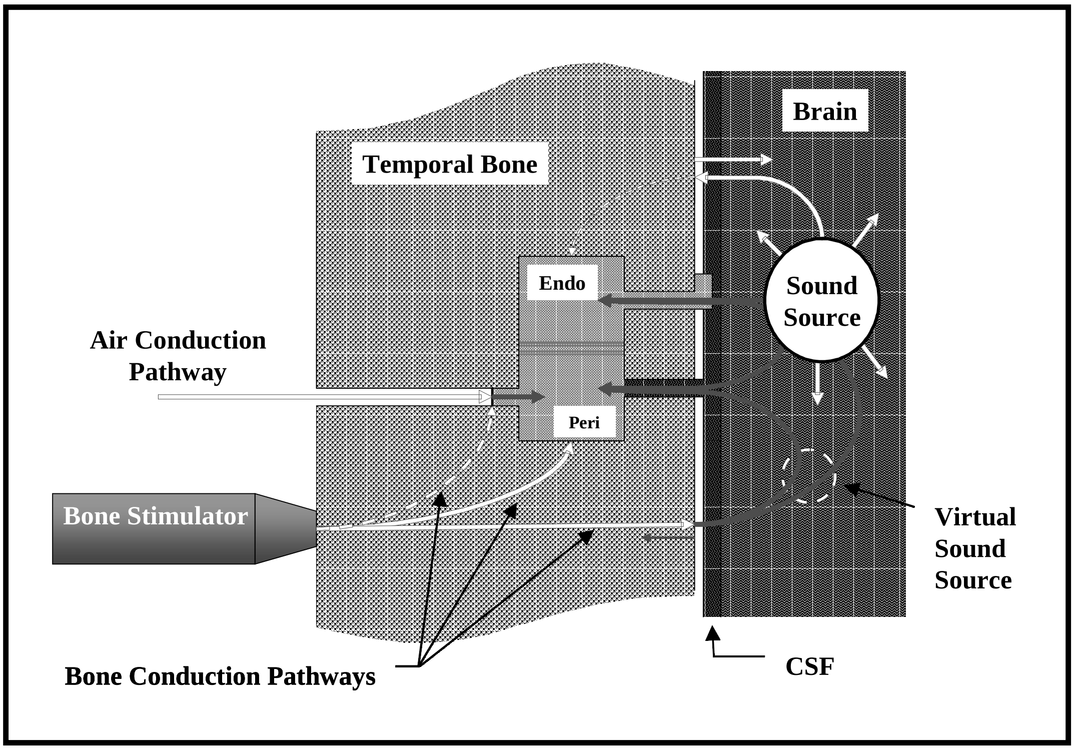

Figure 2. Sound pathways in air- and bone-conduction hearing.

arrow originating near the bone stimulator, passing as two branches (one partially obscured) into cranial contents, and then through channels in the bone into the inner ear. The channels likely contributing most to transmission in this pathway are the vestibular and cochlear aqueducts, as shown Fig. 2. Channels through which nerves and blood vessels travel provide additional possible paths for sound transmission in parallel with the aqueducts. Together these channnels constitute a "non-osseous" pathway that accounts for most of the sound transmitted to the cochlea in bone-conduction hearing [14,16].

An experimental finding in the recent bone-conduction experiments is that sound in cranial contents capable of stimulating the inner ear does not produce detectable vibration of bone [14,16]. This was tested in a number of ways that included placing a bone stimulator directly on the dura mater, which covers the CSF. Stimulation of the amphibian inner ear has also been observed by tapping exposed dura [17]. We can expect that sound generated by absorption of microwave pulses inside the cranium travels to the inner ear by the same pathway. Sound generation within the cranial contents, say, by microwave absorption, is represented in Fig. 2 by a sound source located in the brain. The pathway to the inner ear is shown by curved arrows originating at the sound source and passing through the aqueducts. An arrow from the sound source to bone is shown to represent generated sound that is transmitted into bone. Because of the difference in acoustic impedance at the CSF-bone interface [14], most of the sound is reflected back into intracranial soft tissues (straight arrow) and very little is transmitted into bone (dashed curved arrow).

At least two observations in microwave hearing research provide support for the proposed direct pathway for sound transmission. One is the correspondence between the frequency of cochlear microphonic oscillations and the calculated resonant frequency of the brain [2,3,11]. If sound generated intracranially were to couple to bones of the skull we would expect resonant vibration of the skull to be reflected in the cochlear microphonic. The resonant frequency of the adult human skull is 1-2 kHz [18-20]. This is about one-tenth of the predicted brain resonant frequency of 11 kHz for microwave hearing in adults [3]. In the absence of data on animal skulls, we might expect a similar ratio between resonant frequencies of brain and skull for other mammals. However, only the higher frequency of brain resonance is observed in cochlear microphonic oscillations. Lack of skull vibration is consistent with vibration not being detected with exposure to microwave pulses [21].

Another relevant observation is the short delay of less than 40 μs between onset of microwave pulse and start of cochlear microphonic in animals [2,10,11]. Auditory responses to bone stimulation can be expected to be delayed 0.1-0.5 ms (100-500 μs) from stimulus onset, depending on type of animal, location of stimulus, and other factors [22-23]. We might also expect a delay due to mass inertia of the skull and the relatively low resonant frequency of the skull. Propagation delays of sound travelling between points on the skull and low-pass filtering by the skull have been measured with bone stimulation [23]. One might suggest that a later, slower component of the microwave cochlear microphonic might have been overlooked in microwave hearing experiments because of the short time window used to study the high-frequency cochlear microphonic at the round window. However, one would expect that a microphonic component of comparable or larger amplitude, as well as being less impacted by the early microwave-pulse-induced artifact, would be easily identified before detailed observations of the high-frequency component were made. The short latency of the only or, perhaps, the most dominant microwave cochlear microphonic is inconsistent with forms of bone conduction that involve vibration of the bone.

Summary

Based on a number of considerations, we can reasonably conclude that the pathway for transmission of sound from intracranial tissues to the inner ear in microwave hearing is through various channels in bone that connect to the intracranial space to the inner ear. This pathway appears to dominate over other pathways in bone-conduction hearing and can be driven by bone-conducted sound, but the pathway through bone does not require that sound actually travel in the bone itself. The previously proposed pathway for sound transmission to the cochlea in microwave hearing that includes bone vibration is most likely not the pathway. This observation should be useful in reconciling results from various experiments on microwave hearing. Results from future microwave hearing experiments to test for non-osseous bone conduction can be considered in setting exposure limits for microwave pulses.

Acknowledgments

This work is supported by U.S. Army Medical Research and Materiel Command contract DAMD17-94-C-4069 awarded to McKesson BioServices Corporation. The views, opinions and/or findings contained in this report are those of the author(s) and should not be construed as an official Department of the Army position, policy or decision unless so designated by other documentation. This article is a US Government work and, as such, is in the public domain in the United States of America. WRAIR is the Walter Reed Army Institute of Research in Washington, DC USA. The author is grateful to Dr. Clifford Sherry for various stimulating discussions on microwave hearing topics.

References

[1] Lin JC. 1980. The microwave auditory phenomenon. Proc IEEE 68(1):67-73.

[2] Chou CK, Guy AW, Galambos R. 1982. Auditory perception of radio frequency electromagnetic fields. J Acoust Soc Am 71(6):1321-1334.

[3] Lin JC. 1990. Auditory perception of pulsed microwave radiation. In Gandhi OP (ed): Biological Effects and Medical Applications of Electromagnetic Fields. New York: Prentice-Hall, pp 277-318.

[4] International Commission on Non-Ionizing Radiation Protection. 1998. Guidelines for limiting exposure to time-varying electric, magnetic, and electromagnetic fields (up to 300 GHz). Health Phys 74(4):494-522.

[5] Foster KR, Finch ED. 1974. Microwave hearing: Evidence for thermoacoustic auditory stimulation by pulsed microwaves. Science185(147):256-258.

[6] Lin JC. 1978. Microwave Auditory Effects and Applications. Springfield,Illinois: Charles C. Thomas.

[7] Lin JC, Su JL, Wang Y. 1988. Microwave-induced thermoelastic pressure wave propagation in the cat brain. Bioelectromagnetics 9(2):141-147.

[8] Olsen RG, Lin JC. 1981. Microwave pulse-induced acoustic resonances in spherical head models. IEEE Trans Microwave Theory and Tech 29(10):1114-1117.

[9] Olsen RG, Lin JC. 1983. Microwave-induced pressure waves in mammalian brains. IEEE Trans Biomed Eng 30(5):289-294.

[10] Chou CK, Galambos R, Guy AW, Lovely RH. 1975. Cochlear microphonics generated by microwave pulses. J Microwave Power 10(4):361-367.

[11] Chou CK, Guy AW, Galambos R. 1977. Characteristics of microwave-induced cochlear microphonics. Radio Sci 12(6S):221-227.

[12] Chou CK, Galambos R. 1979. Middle-ear structures contribute little to auditory perception of microwaves. J Microwave Power 14(4):321-326.

[13] Tonndorf J. 1972. Bone conduction. In Tobias JV (ed): Foundations of Modern Auditory Theory. New York: Academic Press, pp 197-237.

[14] Freeman S, Sichel JY, Sohmer H. 2000. Bone conduction experiments in animals: Evidence for a non-osseous mechanism. Hear Res 146(1-2):72-80.

[15] Corso JF. 1963. Bone-conduction thresholds for sonic and ultrasonic frequencies. J Acoust Society Am 35(11):1738-1743.

[16] Sohmer H, Freeman S, Geal-Dor J, Adelman C, Savion I. 2000. Bone conduction experiments in humans: A fluid pathway from bone to ear. Hear Res 146(1-2):81-88.

[17] Seaman RL. 2002. Non-osseous sound transmission to the inner ear (letter). Hear Res 166(1-2):218-219.

[18] Håkansson B, Carlsson P, Tjellström A. 1986. The mechanical point impedance of the human head, with and without skin penetration. J Acoust Soc Am 80(4):1065-1075.

[19] Kosugi Y, Ikebe J, Hara S, Takakura K. 1987. Detection and analysis of cranial bruit. IEEE Trans Biomed Eng 34(3):185-191.

[20] Stenfelt S, Håkansson B, Tjellström A. 2000. Vibration characteristics of bone conducted sound *in vitro*. J Acoust Soc Am 107(1):422-431.

[21] Frey AH, Coren E. 1979. Holographic assessment of a hypothesized microwave hearing mechanism. Science 206(4415):232-234.

[22] Sohmer H, Freeman S. 2001. The latency of auditory nerve brainstem evoked responses to air- and bone-conducted stimuli. Hear Res 160(1-2):111-113.

[23] Durrant JD, Hyre R. 1993. Observations on temporal aspects of bone-conduction clicks: Real head measurements. J Am Acad Audiol 4(3):213-219.