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Review

Fundamental Principles of Bone Conduction Hearing in Humans

Georgios K Panagiotopoulos*

Athens Balance & Hearing. Private ENT practice. Greece

***Correspondence to:** Georgios K Panagiotopoulos; OtoRhinoLaryngologist Neurotologist, Leoforos Pentelis 37A Vrilissia, PC 15235 Athens, Greece; Tel: +30 2108100770; Email: gporl@hotmail.com

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ABSTRACT

Bone conduction involves sound transmission through bone oscillations of the skull or neighboring body areas, resulting in auditory perception. Its significance is not confined to differential diagnosis of hearing loss only. It represents a secondary auditory pathway supplementing air conduction process co instantaneously. Known bio-mechanical mechanisms involved in bone conduction in humans are analyzed and summarized in a most concise way including most recent updates that improve current clinical routine practice. Nevertheless, longstanding assumptions still need further research in order to establish a thorough bone and tissue conduction understanding.

Keywords: *Bone conduction;Vibration;Resonance; Impedance.*

Abbreviations

AC: Air Conduction; BC: Bone Conduction; BM: Basilar Membrane; HC: Hair Cell; HF: High Frequency; HL: Hearing Loss; IA: Interaural Attenuation of Sound; IE: Inner Ear; LF: Low Frequency; ME: Middle Ear; OC: Ossicular Chain; OE: Outer Ear; OW: Oval Window; PTA: Pure Tone Audiometry; RW: Round Window; SSCD: Superior Semicircular Canal Dehiscence Syndrome; ST: Scalae Tympani; SV: Scalae Vestibuli; TA: Trans-cranial Attenuation of Sound; TD: Transcranial Delay of Sound; TM: Tympanic Membrane; ZS: Skin Impedance; ZT: Skull Impedance.

INTRODUCTION

Both AC and BC pathways transmit sound to the IE although normally, the AC pathway is the most dominant.[1,2]

Regardless of the way of transmission, the sense of hearing undoubtedly encompasses movement of the BM as a feedback to a sound related pressure change, on either side of it.[2] Although PTA AC thresholds are most routinely measured, only when comparing them to BC thresholds, site of lesion information can be securely established.

UNDERLYING PHYSIOLOGY STILL UNCLEAR

Sound energy through BC, stimulates the IE, bypassing OE and ME, theoretically unaffected. However, this doesn't happen without some loss of energy. In depth understanding of the contribution of each part of the ear to the establishment of BC thresholds is essential to accurately diagnose between the various etiologies of HL.

The significance in BC is not confined to differential diagnosis of HL only. New surgical perspectives with adequate hearing outcomes achieved in subjects with mixed HL, after placement of a vibratory transducer on the RW, along with the well-established detection of atypical low BC thresholds in patients with in vivo diagnosed SSCD, raised new interest and queries regarding the role of BC. Furthermore, new assumptions involving potential processes of BC have arisen.[2-3]

BC PATHWAYS

The AC pathway involves sound propagating in the air, through the external, middle, and inner part of the ear. Energy is transmitted in a unidirectional way, entering the external auditory canal, vibrating the TM, traversing across the OC and dislocating the stapes towards the OW of the cochlea, thus stimulating the HCs of IE. Neural responses born in the cochlea, reach the auditory brain and are perceived as hearing sensation.[1]

BC involves sound transmission through bone oscillations of the skull or neighboring body areas, resulting in auditory perception.

Each anatomical element of the human head (bony skull, cartilages, tissues and cerebral fluids) takes part in BC. It represents a secondary auditory pathway supplementing AC process coinstantaneously.[1]

Three Classic Pathways Bypass the ME Acting on the Cochlea Fluids

1. Cochlear fluid inertia: Due to oscillations of the surrounding bones, inner ear fluid is prone to inertia forces[4] creating a pressure gradient across BM and subsequently a travelling wave.[2]

2. Compression of cochlear walls: The framework of the bone is mangled during BC, further affecting the inner ear space, producing fluid displacement and pressure change.[2,4]

3. Pressure gradients exerted via CSF: The energy transmitted by oscillation may short-circuit the bones, reaching IE through CSF.[2,5]

Pathways through the ME

1. Via OE: Oscillator on the mastoid induces vibration of soft tissues of the cartilaginous part of the OE. The sound produced there is transmitted to the ME by AC.[2]

2. Via ME: BC through temporal bone due to the spring effect created by the tympanum and the annular ligament which holds the stapes footplate in position. This, forces the ossicular chain to oscillate with the skull at LF. At HF, this oscillation becomes dissociated from the neighboring bone movement.[2] Moreover, based on the distribution of vibration, the stapes remains relatively stable or oscillates with certain time delay due to its own inertia.[1]

VIBRATION MODES OF BC

There are seven BC mechanisms.[6] These derive of two elementary types of oscillation of the human skull that take place at LF and HF:[1]

1. Inertial BC Mode

Inertial BC mode, where the skull oscillates as one piece, vibrating towards the direction of a present force.[1] It impersonates the result of variations in amplitudes of motion and time lags in the displacements of each IE parts.[1] There are two mechanisms of inertial BC.

Inertial IE mechanism: Oscillations of the cranium are directed straight to the IE through the oscillations of the surrounding temporal bone (osseous pathway).[1]

Inertial ME mechanism: Oscillations from the cranium are inducing relative dislocations of the OC because of the differences in inertia of the participating bones (Osseo-tympanic pathway). Moreover, as the cranium vibrates as a unit, the oscillation of the OC is further set back because of the inertia originated by their suspension with springy ligaments.[1]

LF translational oscillations of the cranium as a whole are directly transmitted to the bony cochlea, where its walls move relatively to the fluid compartment because of the fluid own inertia.[1] The bony walls also move in relation to the stapes, which has its own inertia, while the RW membrane tends to move conjointly with the cochlea.[1] All these relative movements result in the phase shift between the displacement of the OW-RW and the displacement of the inner ear fluids that ultimately moves the BM.[1]

2. Compressional Mode

The cranium is divided into numerous segments that vibrate in opposite directions, generating pulsating translocations of the whole unit. It depends on the BC functions of the inner and outer parts of the ear, which are:

Compressional IE mechanism: Where compressional oscillations of the temporal bones displace the cochlear fluids inside their bony segments (osseous pathways). It derives from the interchanging compression-expansion of the inner ear bony frame, in synchroneity with compression and rarefaction of the colliding sound waves.[1]

Two different sub-mechanisms are considered,[6] rising from the axiom that inner ear fluids are incompressible and therefore buckle under the domination of the opposite displacements of the cochlear bony frame.[2,6]

1st mechanism: 1st mechanism derives from the different compliances between OW/RW[1,6] (the ratio OW/RW is about 1:5 because the displacement of the OW is constrained by the stapes footplate). Since fluids are not compressible, back and forth oscillations of the inner ear frame should induce 180° out-of-phase displacements of the OW-RW mucosae so as to accommodate fluid pressure alterations. This asymmetry, forces RW to be displaced to a greater degree than the OW creating momentary pressure differences across scala media. These pressure differences create BM displacement.[1]

2nd compressional mechanism: 2nd compressional mechanism results from the fact that SV and ST have different relative volumes of 22 and 29mm3 respectively.[1] As SV is linked to the perilymphatic compartment of the vestibular apparatus, the culminating mass of fluid motion attributable to the alternate oscillations of the inner ear frame is larger than in ST.[1] The ratio reaches 5:3, thus increasing the gradient of pressure and displacement on each side of the BM.[1]

Interestingly enough, although the contraction of the stapedius muscle increases OC stiffness, protecting the cochlea from excessive inertial ME stimulation, it doesn't safeguard the hair cells from overstimulation due to the compressional BC mechanism of the IE.[1]

Compressional OE mechanism: Oscillations from the bony part of the external meatus are mediated back to the IE, parallel to the AC pathway (osseo-tympanic pathways).[1,7] This mechanism is attributed to the disparities in the displacements of the mandible and those of the cranium.[1] The temporal-mandibular joint is situated under the cartilaginous part of the outer canal. The delay in the motion of the mandible creates oscillations of the walls of the canal. These compressions of the canal wall produce sound pressure alterations in it and subsequently move towards the TM and the OC.[1] Although an non occluded canal doesn't contribute much to BC hearing (as the sound energy is mostly radiated outwards), if occluded, energy cannot escape anymore, radiating inwards.[1,7]

RESONANCES

Resonance is a high intensity oscillation of any system provoked by an exterior periodic force, whose frequency is close to or equal to the natural frequency of the system.[1] Opposed to resonance, antiresonance (parallel resonance) results when the impedance of a system reaches infinity and any shift in stimulation frequency induces an augmented feedback of the system. The specific frequency defining the antiresonance of the system is the 'antiresonance (notch) frequency'.[1]

The mechanical system of the ear has a resonance frequency from 800-1200Hz.[1] At LF; BC causes it to vibrate as a unit (inertial mode) parallel to the direction of the applied force. At HF; sound vibrations travel as complex waves, involving transverse and longitudinal oscillations, causing skull to vibrate as a series of subunits (compressional mode).

The first natural (compressional) resonance of the head is around 800Hz.[1,7] In this pattern, the cranium oscillates as a front-back oriented dipole.[1] Above 800Hz, the one way front back mode of oscillation progressively turns into the second compressional pattern (1600Hz) where skull starts to oscillate as 2 out-of-phase pairs of segments drifting along the medial and lateral axes.[1,7]

Although these findings are confirmed by several other authors, the reported resonance frequencies vary widely.[1] These differences reflect the fact that resonance frequencies rely, besides anatomical and physical characteristics (i.e. age-related bone density and flexibility), on the exact point of excitation.[1]

In conclusion, human skull vibrates in various oscillation patterns with 2 main types of oscillation; at 800-1000Hz (compressional) and 1500-1600Hz (inertial).[1] Moreover, an intense LF inertial antiresonance is reported in the 150-400Hz range,[1] attributable to skull-oscillator coupling.[1] Finally, the antiresonance of the skull reported at 2KHz is related to the resonant characteristics of the OC. The fixation of the stapes i.e., can induce an acute shift in the BC threshold at 2000Hz (Carhart's notch).[1]

MECHANICAL IMPEDANCES

The mechanical impedance (Z) of the head reflects its total resistance to exterior forces acting upon it.[1] Its basic components are resistance (friction related) and reactance (mass and stiffness related).

There are two separate impedance measures of the head, referred as skin (ZS) and skull impedance (ZT). At LF, the magnitude of the ZT escalates with frequency, demonstrating a mass-controlled system.[1] ZT reaches highest values at the inertial antiresonance of the head (150 to 400 Hz).[1] Beyond the resonance frequency, ZT diminishes with frequency and the phase angle becomes negative, indicating a stiffness-controlled system.[1] In general, the impedance status far from the inertial resonance frequency ranges between 30dB-50dB for most frequencies 100-8000Hz.[1]

ZS decreases as frequency increases, reaching minimum at the resonance frequency of 3kHz. Similarly, the phase angle is negative up to 3kHz, in accordance with the stiffness-controlled status of ZS in this frequency spectrum. Beyond the resonance frequency, ZS grows up slightly and the phase angle reaches positive values demonstrating the mass-controlled aspect of the impedance.[1]

ZS is 10-30dB lower than ZT. The greatest differences are in the 150-400Hz and 2000-3000Hz regions.[1] These numbers are relatively big considering the magnitude of the acoustical impedance of air,[1] and this is why AC thresholds differ from those measured by BC for free-field sounds.

TRANS-CRANIAL ATTENUATION OF SOUND

The ability of sound to convey through the cranium is noticeable when estimating thresholds for ears with different auditory sensitivity. IA (amount of sound isolation provided by the head when delivered through the ears) for AC, with properly inserted earphones, reaches 100dB at 250-500Hz and 80dB at 2000-4000Hz.[1] With supra-aural earphones IA reaches 50dB at LFs to 60dB at HFs[1,7] meaning that sound >40-60dbHL, can cross the head and stimulate the opposite ear.[1,7]

In free-field conditions, IA is greater when the sound source is placed along a lateral axis on one side of the skull, varying from 0dB for <200Hz to 20dB for >10kHz.[1]

For BC sounds, the term TA, is utilized suggesting cranial rather than aural stimulation. When the oscillator is situated in the median plane of the cranium, TA is practically zero because sound attenuates symmetrically. If the vibrator is placed off midline, the TA differs from zero due to different attenuation paths. The amount of difference is frequency dependent; the TA for a oscillator on the lateral side is <5 dB in the 250-500Hz range, reaching 15-20dB in the 2000-4000Hz range and above.[1,8]

TRANSCRANIAL DELAY OF SOUND

TD for BC relies on the mechanical characteristics of the skull and the place of stimulation, mainly determined by the speed of sound through the structures of the cranium.[1] The speed of sound through bones is about 2600m/s.[1] Bekesy first measured the speed of sound through the head, reporting values of 570 m/s.[1,7] Other authors using phase cancellation techniques measured phase velocities reaching 250 m/s and 400 m/s at the cranial vault and the skull base of the cadaver head, respectively.[1,8] The speed of sound at LFs<400 Hz is lower (50-100 m/s) than at HFs.[1,8]

Placing the vibrator on the frontal bone, a time delay of 2.0ms at 500Hz, and 0.8ms at 2000Hz, 4000Hz is noted. Alternatively, on the mastoid process, the time delay diminished to 1.5 ms at 500Hz and 0ms at 2000Hz.[1] These frequency/location-dependent time lags of BC sounds imply a frequency-dependent velocity of sounds crossing the cranium.[1]

The decreased speed of sound measured in vivo compared to that of a dry skull can be partially due to the low stiffness of the live skull compared to dry bones.[1] Moreover, wave transmission through live skull and tissues is quite complex, including longitudinal and latitudinal components, additionally reduced by the high viscosity of brain matter and skin elasticity.[1] In such circumstances, phase and group velocities are frequency dependent, possibly resulting in audible nonlinear distortions of high intensity BC sounds, decelerating wave propagation.

NONLINEAR BEHAVIOR OF BC

Various mechanisms were proposed, involving nonlinear behavior of skin and soft tissue through nonlinearity of cochlear response.[1] Second (symmetrical) and third (asymmetrical) harmonic distortions at LF were reported for BC by various studies.[1]

When the linearity of sound propagation through the living skull was investigated, no significant indications of nonlinearities from 100-10,000Hz and levels up to 77dB HL were noted.[1] Moreover, skin impedance over the mastoid was also found not to have nonlinearities.[1] Previously reported results were supposed to be confounded by nonlinearities of the transducers and the measuring procedures indicating that perception through BC can be considered as a linear mechanism.[1,8]

However, LF signals are prone to transient distortions due to inertial vibration of the head. Moreover, LF skull vibrations exceeding 77dBHL are not rare, provoking nonlinear behavior of the skull. To avoid these potential distortions, high intensity BC signals should include mid and high frequencies only.[1]

TACTUAL PERCEPTION

Oscillators can create tactual sensations in addition to or independently of the auditory ones. These tactile sensations are limited to signals < 1000Hz and impulse stimuli. Both perceptions can co-exist when BC sound is not masked.[9] The magnitude of the tactual perception, analogous to the auditory, changes (depending on the surface area, static pressure of the contact and exact position).[1,7]

Pacinian corpuscles, are the receptors responding to changes in pressure and vibrations <1000 Hz. They are unevenly scattered over the skin. The greater their density, the more sensitive the skin to tactile stimulation. The greatest sensitivity is found at the fingertips and various head locations, gradually diminishing at stimulation sites closer to the abdomen.[1]

The force level at which the vibrotactile thresholds are reached, increase with frequency from 125-500Hz, but remain constant up to 2kHz. Vibrotactile thresholds can be confounded with BC thresholds measurements up to 500Hz, particularly when measured on the forehead.[9]

CLINICAL APPLICATIONS

The inertial mechanism of the ear reaches maximum values in the lateral direction when the axis of oscillation parallels the axis of the position of the cochlea. Therefore, placing an oscillator on the mastoid is a very efficacious stimulation place for the intact cochlea.[1,10]

The closure of the canal and concomitant increased perception of loudness (occlusion effect) is related to the volume of trapped air.[1] The larger the volume, the greater the occlusion effect, mostly due to the compressional OE mechanism. Controversially, the deep, firm closure of the canal may not be as effectual in increasing the perceived loudness of BC sound.[1] This is attributed to the increased impedance of the TM, the reduced volume of air and the concomitant decreased mobility of the TM-OC.[1] In such cases, a small controlled leakage can increase the loudness of the sound, simply by loosing the TM.[1]

During BC testing, the relatively low TA demands masking when there are interaural threshold differences. However, sealing the non-test ear with an earphone, enhances the BC signal and creates an additional problem.[1] Isolating the non-test ear with a firmly inserted earphone and narrow-band noise represents the most efficient way of masking.[1]

CONCLUSIONS

BC process is far from being thoroughly understood. Recent findings of the transmission pathways through the skull and through the oral cavity have expanded our comprehension.[11] Taking into account the fact that the oscillation of the soft tissue or the skull bone has an effect of low-pass filtering, whereas the sound radiation in the outer canal has an effect of 2–3 kHz bandpass filtering, transmission to the outer ear may not be a dominant contributor to BC speech perception during vocalization.[11]

Moreover, recent evidence suggests that even different dentition may have impact on the transmission of acoustic vibrations through bone conduction. In such cases, proper dental treatment can decrease hearing thresholds through improved bone conduction.[12] Therefore, longstanding assumptions need further research in order to establish a thorough bone and tissue conduction understanding.

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