REVIEW

Bone conduction: An explanation for this phenomenon comprising complex mechanisms

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Summary Bone conduction hearing inevitably involves vibration of the basilar membrane in response to a pressure gradient on either side of the membrane. The propagated wave that symbolizes this vibration of the basilar membrane can be triggered intentionally, when a bone vibrator is placed on the mastoid bone, or inadvertently when testing hearing of one ear by air conduction while disregarding transmission of the sound to the other side. When hearing is tested with a bone vibrator, the pathways leading to the basilar membrane can be divided into two main categories. The first type of pathway short-circuits the middle ear and comprises three distinct mechanisms: cochlear fluid inertia, compression of the cochlear walls, and pressure changes exerted via cerebrospinal fluid. In the second type of pathway, the stimulus reaches the basilar membrane via the middle ear, either directly or via the outer ear. Although it is difficult to precisely determine the contribution of each of these pathways to the basilar membrane, bone conduction remains the clinically most reliable way of directly testing cochlear function.
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Introduction

The mechanisms by which an individual is able to perceive sound by bone conduction (BC) have been studied for many decades [1,2]. Such intense interest in these mechanisms can be essentially explained by the growth of otological surgery and the need to inform patients about the chances of success of the proposed operation. Progress in middle ear surgery has therefore been closely associated with increasingly reliable audiometry and rigorous study of BC thresholds that provide essential information on the functional status of the cochlea.

However, the interest in BC is not limited to the distinction between conductive hearing loss and other forms of hearing loss. More recently, the good hearing results obtained in patients with mixed hearing loss after implantation of a vibratory transducer on the round window [3] raised new questions concerning the role of BC in the observed improvement of hearing [4].

Furthermore, the observation of unusually low BC thresholds in subjects with surgically documented dehiscence of the superior semicircular channel [5] has led to new hypotheses concerning the possible mechanisms of BC.

A final common pathway: stimulation of the basilar membrane

Georg von Békésy [6] was the first to raise the question of whether hearing by BC involved stimulation of the cochlea or whether it was mediated by another peripheral organ [7]. To address this essential question, he adopted the following rationale: if a sound stimulates the basilar membrane in

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an identical way whether it is transmitted by air conduction (AC) (thereby passing via the middle ear) or by BC, it should be possible to suppress perception of one (sound perceived by air conduction) by perception of the other (sound perceived by bone conduction) and he demonstrated this effect by asking a normal subject to listen to two signals of equal amplitude and frequency (0.4 kHz) but dephased by 180 degrees with respect to each other.

Using a different process, but with the same objective to elucidate the mechanism of BC, Weaver and Bray [8] completed the hypothesis proposed by von Békésy. It was already known, at that time, that by placing an electrode close to the auditory nerve in the cat, it was possible to record an electrical signal accurately reproducing the shape of the acoustic signal presented to the animal's ear [9]. This potential was therefore called the cochlear microphonic potential. Weaver and Bray therefore confirmed that a sound transmitted by BC was able to evoke the same cochlear microphonic potential as the sound transmitted by AC.

In a psychoacoustic study, Khanna et al. [10] demonstrated that a weak 1 kHz sound transmitted by AC was no longer heard when a sound of the same frequency was simultaneously presented by BC between 40 and 70 dB hearing level (HL).

After this brief historical review, let us now examine what actually happens in the basilar membrane. The basilar membrane is a fibrous structure attached medially to the osseous spiral lamina and laterally to the spiral ligament [11]. Under physiological conditions (transmission of sound by air conduction), movement of the stapes footplate in the oval window induces pressure fluctuations, which are transmitted to the scala vestibuli and scala media and then to the scala tympani via the cochlear partition. Pressure changes in the scala tympani are compensated by a movement of the round window in the opposite direction, without which movement of cochlear fluid would be impossible. The pressure changes induced across the cochlear partition vary as a function of time according to the vibrations of the stimulating sound. The basilar membrane is rigid adjacent to the base of the cochlea and gradually loses its stiffness towards the apex. This rigidity gradient of the basilar membrane is the result of three factors: the width of the membrane, which increases towards the apex; its thickness, which decreases towards the apex; and the general anatomical structure of the membrane. Due to the rigidity gradient of the basilar membrane, the pressure fluctuations induced by a pure tone give rise to a propagated wave, which travels towards the apex, with a waveform presenting a peak followed by a rapid decline at a precise point of the basilar membrane determined by the frequency of the tone. von Békésy [12] was awarded the Nobel Prize in physiology and medicine for his research leading to the discovery of these physiologically important phenomena. In this review on the mechanisms of BC, it must be remembered that propagation of the wave is identical regardless of the site and modality (AC or BC) of stimulation of the cochlea.

The exceptional quality of the research conducted by von Békésy was confirmed half a century later by direct laser Doppler measurements of the movement of the basilar membrane, demonstrating the similarity of the effects of AC and BC [13].

It should also be stressed that differences are observed according to whether the initial conduction is air conduction or bone conduction [14]. When hearing is tested with a bone vibrator (for example Radioear B71) placed on the mastoid, at low frequencies and high amplitudes, the transducer does not just stimulate BC, but also induces vibrotactile excitation, thereby producing multimodal perception [15]. This loss of auditory specificity of BC at low frequencies has important consequences for paediatric audiometry, as children with profound hearing loss may present true behavioural reactions at low frequencies (from 45 dB HL at 250 Hz and 65 dB HL at 500 Hz), while these reactions have a vibrotactile origin [16]. It is important to identify these reactions in young children with hearing loss, as they validate the quality of the conditioning ensured by the examiner and allow more reliable measurement of BC thresholds beyond 1000 Hz.

Bone vibrators can also stimulate the vestibular system and this phenomenon can be observed both in large vestibular aqueduct syndrome and superior semicircular canal dehiscence syndrome [17].

Another difference was observed between AC and BC in a study comparing progression of loudness in the two types of conduction [18]. This study showed that loudness increased more rapidly by BC than by AC, particularly at low frequencies. This could be explained by the multimodal nature of BC perception, allowing loudness to increase more rapidly than by AC despite the similarity of physical loudness levels.

The electrophysiological thresholds measured by AC and BC must also be interpreted cautiously, as suggested by a comparative study conducted on the V wave of brainstem auditory evoked potentials in the same individuals [19]. Three sources of disparity between electrophysiological responses produced by the two types of conduction have been identified [14]: frequency component differences between the listener and the bone vibrator; hearing threshold differences (sound pressure for AC and force for BC); and lower dynamics with the bone vibrator (which presents more nonlinear distortions). Hence the value of using corrections when comparing auditory evoked potentials recorded with the two types of stimulation [20].

A final argument indicating that vibration of the basilar membrane constitutes the main mechanism of hearing by BC is provided by the recording, under certain technical conditions, of distortion products by otoacoustic emissions in response to a pure tone stimulus transmitted by BC, showing comparable results to those obtained with a stimulus transmitted by AC [21].

Pathways to the basilar membrane in bone conduction hearing

If all sounds perceived by BC are mediated by the basilar membrane, how do these sounds reach this membrane?

First of all, it must be stressed that activation of BC mechanisms during audiometry, when the subject's hearing is supposedly tested by AC, can be misleading. When testing a subject by AC using earphones, a sort of filter situated in the middle of the skull base prevents the sound from being transmitted from one cochlea to the other until the intensity of the stimulus exceeds a certain threshold. This

threshold is called *interaural attenuation* [22]. This phenomenon can be illustrated by performing an audiogram in a subject with profound hearing loss in one ear. By stimulating the deaf ear with TDH39 supra-aural earphones, without masking the contralateral ear, beyond a certain threshold the subject will hear a pure tone that cannot be perceived, as the ''tested'' ear is totally deaf. As emphasized by Lehnhardt and Laszig [23], the ''intense'' sound transmitted by earphones towards the deaf ear does not travel around the head in air, but reaches the contralateral ear by BC. This major finding can be summarized by the following formula: beyond a certain level, AC is transformed into BC. This effect can be misleading to the examiner, as, in practice there is no interaural attenuation by BC and the examiner always records the response from the better ear [24]. In practice, interaural attenuation is not the same in all individuals (in particular, it depends on the conformation of the skull base), and it also varies from one frequency to another. As far as AC is concerned, most authors agree on a mean interaural attenuation value of 50 dB, but some authors consider that a risk of transmission to the other side may be observed at values of only 40 dB with TDH39 supra-aural earphones [22]. The risk would be lesser, i.e. interaural attenuation would be more effective, with ER-3A insert earphones, at least for low frequencies, less than or equal to 1000 Hz [25]. Description of the technique of effective masking of the contralateral ear designed to avoid the phantom curve is beyond the scope of this review.

While bearing in mind this audiometric trap, a sound transmitted by a bone vibrator maintained behind the ear can reach the basilar membrane via multiple pathways, which can be divided into two groups for the purposes of clarity, as perception of a sound by BC reflects a pressure difference between the scala vestibuli and the scala tympani, resulting in displacement of the basilar membrane, and the BC test therefore essentially verifies the functional status of the organ of Corti.

Pathways not involving the middle ear

This first group corresponds to the more classical pathways and comprises pathways that short-circuit [7] the transmission apparatus, i.e. the outer ear and middle ear by means of three mechanisms that, each in its own way, acts on the fluids of the inner ear: cochlear fluid inertia, compression of the cochlear walls, pressure changes exerted via cerebrospinal fluid (CSF).

Cochlear fluid inertia
When the bone surrounding the cochlea starts to vibrate, cochlea fluid is submitted to inertia forces [14]. These forces lead to the creation of a pressure gradient across the basilar membrane, resulting in a propagated or travelling wave. However, displacement of cochlear fluid as a result of inertia forces cannot occur in an incompressible medium such as the labyrinth unless a membrane compensates for fluid movements on either side of the basilar membrane. Under physiological conditions, this compensatory membrane role is played by the oval window on the scala vestibuli side and the round window on the scala tympani side.

However, other structures can also relieve pressure on the cochlea. These structures are collectively called the ''third window'' [14]. According to this concept, fluid movement induces a propagated wave along the basilar membrane for as long as a pressure gradient is maintained across this membrane. Obstruction of the round window [26] was one of the first diseases in which a hypothesis of a so-called ''third window'' mechanism was considered. Two mechanisms have subsequently been proposed to explain this phenomenon. The first mechanism is that of displacement of cochlear fluid in response to cochlear fluid inertia. The second mechanism will be described below, as it is based on a different concept from that of cochlear fluid inertia.

Two teams, one in Baltimore and the other in Boston, have been particularly interested in the third window concept related to fluid inertia, and both of these teams have been working on superior semicircular canal dehiscence syndrome (SSCDS). Minor et al., the first team to identify SSCDS [5], showed that, in addition to vestibular symptoms [17], these patients also presented excessive sensitivity to BC [27]. In an experimental model of SSCDS in the chinchilla, Songer et al. initially reproduced the loss of AC on low frequencies [28], then confirmed the presence of an apparent conduction hearing loss at the same low frequencies due to unusually good sensitivity of BC hearing [29].

Compression of cochlear walls
When a transverse wave is propagated in skull bones, these bones undergo compression and distension [14]. In other words, the structure of the bone is deformed. When a propagated wave reaches the cochlea, these deformities affect the cochlear space, producing a fluid movement and a pressure gradient. This phenomenon has been called ''compression'' by von Békésy [30] and ''distortion component'' by Tonndorf [31]. The theory of BC by compression is based on asymmetry of the cochlea: the scala vestibuli occupies a considerably larger space (about 50% larger) than the scala tympani, and the impedance of the oval window is higher (more rigid) than that of the round window (which therefore presents greater compliance) [31]. Consequently, when the cochlea is compressed, fluid displacement towards the scala tympani and round window is observed, while movement in the opposite direction, from the scala tympani to the scala vestibuli, is observed when the cochlea becomes distended, resulting in stimulation of the basilar membrane.

The rolled-up cochlea can be compared to a sphere about 10 mm in diameter. If the limit of efficacy for a mechanism of BC by compression corresponds to a wavelength less than or equal to 10 times the size of the cochlea, the lowest frequency at which such a mechanism would be observed would be 4 kHz, which is consistent with other estimations of the importance of this mechanism in normal humans [7].

Pressure changes exerted via CSF
This approach has been especially studied by Sohmer in Jerusalem. After several animal experiments [32], this team formulated the hypothesis that the vibratory energy transmitted by a bone vibrator applied to the skull could reach the inner ear without passing directly by the bones, but by conduction by the contents of the skull, especially

CSF. This hypothesis was then verified by recording evoked potentials in older children undergoing neurosurgery with craniotomy and neonates tested via the fontanelle [33]. Recently, the same team recorded BC evoked potentials in animals although the entire contents of the middle ear (ossicles, stapes, round window) had been blocked by glue, thereby suggesting that soft tissues could also represent a pathway for transmission of bone vibrations to the inner ear [34].

Pathways to the basilar membrane via the middle ear

Although these modes of conduction are quantitatively limited, they may be involved in certain pathological conditions and help to explain the audiometric changes induced in these conditions.

Outer ear and basilar membrane

The transducer placed on the mastoid skin can induce vibration of soft tissues and these vibrations can be transmitted to the outer ear, especially the cartilaginous part of the external auditory canal [35]. The sound produced in the external auditory canal is inevitably transmitted to the middle ear by air conduction. When the external auditory canal is open, this canal contribution to BC hearing can be considered to be minor, situated at least 10 dB below the other contributions. However, this participation can become considerably greater, particularly at low frequencies, when the external auditory canal is closed [36]. This so-called occlusion effect can be easily illustrated by talking while occluding the ear. It must be noted that the natural voice produces bone vibration in the speaker and this vibration is accentuated when the external auditory canal is closed. This effect is related to reinforcement of the low frequency component of BC, together with decreased perception of the voice by AC due to occlusion of the ear. This phenomenon probably plays a considerable role in subjects who poorly tolerate hearing aids in the auditory canal, as they often experience unnatural perception of their own voice. The outer ear can also receive vibrations transmitted by the temporal bone [7].

Middle ear and basilar membrane

The middle ear can also be stimulated directly by the temporal bone, in response to the ossicle inertia force [14], as the ossicles are suspended in the middle ear cavity by the tympanum, ligaments and tendons. The most important of all these elements in terms of BC hearing are the tympanum and the annular ligament that maintains the stapes footplate in place in the oval window. Arranged in this way, these two elements act like real springs to which the ossicles are attached. When the skull base surrounding the middle ear cavity starts to vibrate as a result of BC, the spring effect causes the ossicles to vibrate with the skull at low frequencies. At higher frequencies, ossicle vibration becomes dissociated from the surrounding bone vibration. This distinct behaviour of the ossicles according to the frequency of stimulation has been confirmed experimentally [37]: ossicles vibrate with the surrounding bone at low frequencies and vibrate less at frequencies above the resonant frequency of the ossicular chain, situated at 1.5 kHz.

Inertia of the middle ear ossicles appears to play an important role in BC hearing, when the stimulating sound is situated in the vicinity of the resonant frequency of the ossicular chain. The frequent alteration of BC thresholds around 2 kHz observed when the ossicles are immobilized by stapes otosclerosis (Carhart effect) provides support for this theory.

Conclusion

The study of the mechanisms of BC shows that complex phenomena are involved. Although now better understood, these mechanisms still raise many questions concerning their respective roles in various clinical settings.

Nevertheless, BC audiometric tests have an essential role, provided they are conducted according to established guidelines, especially masking the contralateral ear.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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