Hearing Research 329 (2015) 41–51

Contents lists available at ScienceDirect

Hearing Research

journal homepage: www.elsevier.com/locate/heares

Review

# Inner ear contribution to bone conduction hearing in the human

Stefan Stenfelt*

*Department of Clinical and Experimental Medicine, Linköping University, 581 85 Linköping, Sweden*

ARTICLE INFO

*Article history:*
Received 15 September 2014
Received in revised form
5 November 2014
Accepted 8 December 2014
Available online 18 December 2014

ABSTRACT

Bone conduction (BC) hearing relies on sound vibration transmission in the skull bone. Several clinical findings indicate that in the human, the skull vibration of the inner ear dominates the response for BC sound. Two phenomena transform the vibrations of the skull surrounding the inner ear to an excitation of the basilar membrane, (1) inertia of the inner ear fluid and (2) compression and expansion of the inner ear space. The relative importance of these two contributors were investigated using an impedance lumped element model. By dividing the motion of the inner ear boundary in common and differential motion it was found that the common motion dominated at frequencies below 7 kHz but above this frequency differential motion was greatest. When these motions were used to excite the model it was found that for the normal ear, the fluid inertia response was up to 20 dB greater than the compression response. This changed in the pathological ear where, for example, otosclerosis of the stapes depressed the fluid inertia response and improved the compression response so that inner ear compression dominated BC hearing at frequencies above 400 Hz. The model was also able to predict experimental and clinical findings of BC sensitivity in the literature, for example the so called Carhart notch in otosclerosis, increased BC sensitivity in superior semicircular canal dehiscence, and altered BC sensitivity following a vestibular fenestration and RW atresia.

*This article is part of a Special Issue entitled <Lasker Award>.*



## 1. Introduction

In most cases, sound heard by the human is transmitted in the air through the ear canal and the middle ear ossicles to the inner ear. In the inner ear, specifically in the cochlea, the sound pressure creates a travelling wave on the basilar membrane and the sensory cells in the organ of Corti transforms the vibration to a neural representation in the auditory nerve. This route for sound transmission is known as the air conduction (AC) route. However, the sound can also be transmitted as vibrations in the skull bone, cartilage, and soft tissues that finally cause the bone around the cochlea to vibrate and result in a perception of sound (Stenfelt and Goode, 2005a). This route is often referred to as bone conduction (BC) even if it involves other structures than bone *per se*.

The route of BC is most often excited by a transducer pressed onto the skin covered skull bone, but in some cases the vibration is coupled directly to the skull. However, an airborne sound is also transmitted as BC vibrations, but that route is some 40–60 dB less sensitive than the AC route for a person with normal sound transmission through the outer and middle ear (Reinfeldt et al., 2007).

The way BC sounds causes a sound perception has been debated during the last century and there is no consensus of the relative importance of the pathways resulting in a perception of BC sound. Herzog (1926) and Krainz (1926) presented a theory of BC as originating in two phenomena. One was the compression and expansion of the cochlear boundary due to the compressional waves in the bone forcing a fluid flow between the scalae in the cochlea that excites a travelling wave on the basilar membrane. The other phenomenon was the relative motion of the middle ear ossicles due to inertial effects. von Békésy (1932) extended this theory and concluded that BC sound in the human was caused by three phenomena. At low frequencies, the relative motion of the middle ear ossicles dominated the BC sound perception while above 1.5 kHz compressional waves in the bone became apparent and the response was attributed to compression and expansion of the cochlear space. He also suggested the relative motion between mandible and the skull as a third contributor to BC sound.

The number of possible sources for BC sound perception increased and Tonndorf (1966), from his seminal studies in cats,

*Abbreviations:* BC, Bone conduction; AC, Air conduction; SSCD, superior semicircular canal dehiscence; SV, Scala vestibuli; ST, scala tympani; OW, val window; RW, Round window; CA, Cochlear aqueduct; VA, vestibular aqueduct

* Tel.: +46 13 28 47 98.
*E-mail address:* Stefan.stenfelt@liu.se.

http://dx.doi.org/10.1016/j.heares.2014.12.003
0378-5955/

proposed as much as seven different sources for BC sound. Lately, Stenfelt and Goode (2005a) and Stenfelt (2011) listed five sources as possible contributors for BC sound perception in the human. These were (1) sound pressure generation in the ear canal, (2) inertial forces on the middle ear ossicles causing a relative motion between the stapes footplate and the cochlear promontory bone, (3) inertial forces acting on the cochlear fluid, (4) alteration of the cochlear space, and (5) sound pressure transmission from the skull interior. The sound pressure generation in the ear canal seem to be about 10 dB lower than other contributors for BC sound perception in the normal ear (Stenfelt, 2006; Stenfelt et al., 2003) but can be important at frequencies below 2 kHz when the ear is occluded (Reinfeldt et al., 2013; Stenfelt et al., 2007). The relative motion of the middle ear ossicles increases with frequency at the low frequencies and resonates at around 1.5–1.7 kHz (Homma et al., 2009; Stenfelt et al., 2002). In the normal ear, it is unlikely that this motion is important for BC sound perception at low frequencies but can be of importance at around its resonance frequency (Homma et al., 2009; Stenfelt, 2006).

As indicated above, the status of the outer and middle ear seems to be of little importance to the sensitivity to BC sound. This fact is used in audiology where the uninvolvement of the outer and middle ear in the BC sound perception leads to nearly unaltered BC thresholds for a conductive impairment located in the outer or middle ear, but the conductive impairment affects the AC thresholds (Stenfelt, 2013). Consequently, comparing the AC and BC thresholds is used to classify between conductive and sensorineural hearing losses. This also means that the cochlea can be seen as the dominating part for BC perception. One of the direct cochlear stimulation pathways is sound pressure transmission from the skull interior. This transmission is hypothesized to rely on sound pressure transmission from the cerebrospinal fluid through compliant pathways to the cochlea (Sohmer et al., 2000) and can be excited by, for example, applying a vibrating transducer to the eye (Perez et al., 2011). However, several clinical findings such as BC sensitivity change due to superior semicircular canal dehiscence (SSCD) indicate that it is not the most important pathway for BC hearing in the human (Rosowski et al., 2004; Songer et al., 2010).

Accordingly, the two most important mechanisms for BC sound perception in the human are alteration of the cochlear space and fluid inertia. However, their relative importance for BC sound perception is not clarified and is often debated. The effect of the alteration of the cochlear space will hereafter be referred to as the compression response. This mode of stimulation is based on the compression and expansion of the cochlear bone during wave motion in the skull bone. The idea is that the fluid is incompressible or nearly incompressible and that during the compression phase the cochlear space is reduced forcing the excess fluid towards the compliant in and outlets of the cochlea, primarily the oval and round windows. During the expansion phase the fluid flow is in the opposite direction. This action of forcing fluid flow in the cochlea excites the basilar membrane causing a hearing perception. The fluid inertia relies on the inertial forces acting on the cochlear fluid during vibration of the bone surrounding the cochlea. This force induces a pressure gradient across the basilar membrane that initiates the travelling wave and subsequently a hearing perception.

It has been argued that the compression response is not a significant contributor at low frequencies for BC sound perception in the human (Stenfelt, 2011; Stenfelt and Goode, 2005a). This is based on the notion that wave transmission at low frequencies are close to rigid body motion where expansion and compression of the bone is almost non-existent (Stenfelt and Goode, 2005b). According to finite element simulations of the bone surrounding the cochlea, the compression of the bone is around 25 dB lower than the translational motion of that same bone during BC stimulation up to 5 kHz (Hudde, 2005). However, the compression response may be of importance at higher frequencies, above 4–5 kHz (Kim et al., 2011; Stenfelt and Goode, 2005a; Taschke et al., 2006).

As indicated above, models have been used to understand perception of BC sound in the human (Homma et al., 2009; Kim et al., 2011; 2014a; Taschke et al., 2006). For example, based on Zwislocki's model of the middle ear (Zwislocki, 1962), Williams et al. (1990) devised a circuit model to estimate the middle ear inertial component of BC sound. A more comprehensive finite element model of the middle ear for BC was presented by Homma et al. (2009). The occlusion effect have been investigated by both lumped element models (Schroeter et al., 1986; Stenfelt et al., 2007) and finite element models (Brummund et al., 2014). The most comprehensive work of a finite element model of a whole human head was presented by Taschke et al. (2006). This model was developed for a complete human head including different structures as soft tissue, fluids, solids, or a mixture thereof. They concluded that the inner ear was the primary contributor to the perception of BC sound whiles the outer and middle ear played a secondary role in a healthy ear. They also investigated the different modes for inner ear stimulation and concluded that at low frequencies the inertial effect dominated while at frequencies above 1.4 kHz, deformation of the cochlear bony shell contributed to the excitation of the inner ear when stimulation is by BC.

There have been several models that have simulated the inner ear or cochlear response to BC excitation. An advanced inner-ear lumped element model was developed with the aim to explore differences in hearing loss with BC and AC stimulation (Schick, 1991, 1992). However, that model was not verified with human physiological data, and some of the assumptions have later been found erroneous. Bohnke et al. (2006) presented a three-dimensional finite element cochlear model for BC stimulation using the assumption that the BC stimulation was a harmonic pressure at the cochlear bony shell and was directed toward the cochlea. Such stimulation is not correct for BC excitation of the cochlea where motion of the bony shell or inertial effects distributed in the fluid is believed to constitute the mechanical excitation. Even if the above model was able to replicate some clinical findings, including sensitivity increase due to stapes footplate removal and the ability to cancel BC sound with AC sound, the incorrect geometry and erroneous stimulation mode limits its usability. In a series of studies, Kim et al. developed a finite element model of the inner ear (Kim et al., 2011; Kim et al., 2013b; Kim et al., 2014b). The model evolved from a tapered box-model used to investigate inertial BC cochlear excitation (Kim et al., 2011) to an anatomically correct model with a coiled cochlea including the vestibular system with the semicircular canals and middle ear (Kim et al., 2014b). Such models are powerful for simulations of BC sound but are complex and require specialized software.

Regardless of model complexity, it is important to provide valid excitation pattern for BC sound. Several of the above mentioned models have used a pressure excitation at a rigid boundary, e.g. (Bohnke et al., 2006; Schick, 1991). This type of excitation means that an in-phase pressure is distributed at the boundary but the boundary itself is immobile. Such excitation is very different from a real BC excitation where the excitation itself depends on the motion of the boundary, both in-phase and out-of-phase. Consequently, for simulations of BC cochlear stimulation the excitation should rely on the motion of the cochlear boundary. Moreover, both the compression and the inertial responses depend on the whole structure of the inner ear. It is therefore not enough to only include the cochlea but the vestibular system should also be included in the simulations as it influences both the inertial forces as the compression of the fluid.