MECHANISMS OF BONE CONDUCTION: A REVIEW OF THE LITERATURE

By

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Final approval and acceptance of this Audiology Doctoral Project is contingent upon the candidate's submission of the final copies of the Audiology Doctoral Project to the Graduate College.

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Table of Contents

Abstract

Clinical bone conduction during a routine audiological evaluation is a vital tool for audiologists to differentiate a sensorineural hearing loss from a conductive hearing loss. However, the mechanisms that give rise to responses during bone conduction stimulation are poorly understood. This paper will cover the results of a review of the literature regarding the mechanisms of bone conduction to attempt to aid in the understanding of the mechanisms underlying bone conduction testing. Various proposed mechanisms of bone conduction will be discussed, including mechanisms involving the outer ear, middle ear, and inner ear, along with newly proposed mechanisms such as soft tissue conduction and cerebrospinal fluid conduction, along with evidence found in the literature supporting the existence of each mechanism of bone conduction. The purpose of this paper is to further the reader's understanding of bone conduction stimulation and the mechanisms that compose this response, which will then lead to improved clinical decision making.

Introduction

The peripheral hearing mechanism is traditionally divided into three parts: the outer, middle, and inner ear. The outer ear consists of the auricle, or pinna, which is the visible portion of skin and cartilage, that captures and funnels sound into the ear canal, a tunnel of skin, cartilage and bone that terminates at the tympanic membrane, or eardrum. The tympanic membrane is the lateral border of the middle ear space, and is a multilayered “sandwich” of epithelial, fibrous, and mucosal tissue. On the medial side of this membrane lie the ossicles, an interconnected “chain” of three tiny bones of the middle ear that couples the tympanic membrane to the oval window and conduct sound energy to the cochlea. The malleus is the largest and first of these bones and is attached to the tympanic membrane. Next is the incus, rigidly attached to the malleus, and finally the stapes, the smallest bone in the human body, connected to the incus via a flexible joint. The stapes is attached to the oval window of the cochlea via a fenestration called the oval window. The cochlea marks the beginning of the inner ear. Its sensory structures are contained in a membranous coiled duct housed in the temporal bone. The cochlea is divided into three portions, the scala tympani, scala vestibuli and scala media, which is sandwiched between the first two scalea mentioned. The scala media contains the organ of Corti, where pressure waves conducted by the ossicular chain result in the depolarization of hair cells, which ultimately leads to the activation of auditory nerve fibers (Musiek & Baran, 2018).

The process of sound perception begins when an object is set into motion, causing periodic alternating patterns of rarefaction and compression of air particles. These sound pressure waves then are conducted into the ear canal via the pinna, where they continue to the tympanic membrane. The vibration of air particles sets the tympanic membrane into motion, which then causes motion in the ossicles. The stapes, which is connected to the oval window of the cochlea, is moved in a rocking motion. This piston like motion creates pressure waves of rarefaction and condensation of the fluid in the scala media of the cochlea, which then allows depolarization of inner hair cells in the cochlea. Depolarization of the inner hair cells is then transmitted to the connecting auditory nerve fibers, which is then conducted to the brain. These nerve action potentials within the auditory structures of the brain are then perceived as sound (Musiek & Baran, 2018).

The typical process of hearing, which is detailed above, is by air-conduction (AC). That is, perception of sound requires the transmission of sound waves (rarefactions and condensations, or “pressure waves” of air molecules) through the ear canal. A disorder in any part of the ear (i.e., outer, middle, inner) can cause hearing loss. Audiologists measure hearing by presenting air conducted pure tone signals to a patient’s ear using supra-aural or insert earphones. According to the Hughson Westlake procedure, air-conducted pure tone thresholds, the lowest hearing levels needed for the patient to respond to the pure tones 50% of the time, are then recorded on an audiogram (Katz et al., 2014).

There is another medium through which sound can be perceived, which is bone conduction. Bone conduction (BC) hearing occurs naturally when air-conducted sounds are of a high enough intensity to vibrate the bones of the skull. BC hearing involves vibration of the bones of the skull, which are conducted through a variety of proposed mechanisms to the inner ear, theoretically bypassing the outer and middle ear. BC hearing has also been utilized for diagnostic purposes in clinical settings for centuries. By using BC hearing compared to air conduction (AC) hearing, it is possible to differentiate between hearing loss originating from the outer and/or middle ear (conductive hearing loss), and hearing loss originating from the inner ear (sensorineural hearing loss). However, the exact mechanisms of BC and how sound can stimulate the cochlea via the bones of the skull is a more complicated mechanism than it seems.

There are many theoretical mechanisms by which bone vibration provides sound perception. To aid in understanding how the cochlea is activated via BC, four proposed major mechanisms (i.e., outer ear, middle ear, inner ear, and soft tissue conduction) will be briefly described.

Mechanisms Involving the Outer Ear

Jürgen Tonndorf (1968) proposed a mechanism of BC that is generally referred to as osseotympanic BC. This type of BC involves the outer ear. When air conducted sound is strong enough to induce BC hearing, or when a BC transducer is used, the bony portion of the temporal bone that forms the ear canal and the cartilaginous portion of the outer ear are set into vibration. Most of this sound energy escapes out the unoccluded ear canal. However, some of that energy reaches the tympanic membrane, setting it into motion, thus stimulating the inner ear. This is also assumed to be the source of the occlusion effect. The occlusion effect describes a phenomenon in which occluding the ear canal in normal ears increases the perceived volume of a bone conducted stimulus. This effect was first described in 1891 by Albert Bing (Csovanyos, 1961). This effect will be discussed later in this document in the section entitled “Conclusions regarding the external auditory meatus (EAM).”

Mechanisms Involving the Middle Ear

One of the first writers to describe the mechanisms of BC was Ernst Barany (1938) within his doctoral thesis entitled “A contribution to the physiology of bone conduction.” In his document, Barany describes the physics and mechanisms of two main types of BC, that are now known as inertial and compressional BC. Compressional BC involves the inner ear, and therefore will be described under "Mechanisms Involving the Inner Ear."

Inertial BC involves the inertia of the ossicles. Since the ossicular chain is not rigidly attached to the middle ear space, its inertia causes the ossicles to lag behind the motion of the middle ear space around it, resulting in motion of the ossicular chain relative to the middle ear space and cochlea. This motion of the ossicles causes the stapes to create pressure waves in the cochlea, resulting in a traveling wave in the cochlea in the same way air conducted sound does.

Mechanisms Involving the Inner Ear

Another mechanism proposed by Barany in 1938 was a process he called compressional BC. Compressional BC describes compressional forces induced by sound pressure waves that act on the otic capsule and cause the volume of the cochlea to decrease, displacing fluid into the pliable round window. Then the restoring forces cause the cochlea to return to its "resting volume". Consequently, inertia then causes overshoot, increasing the volume in the cochlea, making the round window retract. Finally, the cochlea returns to its resting volume and the whole cycle begins again as long as the stimulus is present. This periodic motion of compression (condensation) and expansion (rarefaction) causes movement of the perilymph and endolymph in the cochlea, thus causing displacement of the basilar membrane as air conducted sound does.

Mechanisms Involving Soft Tissue Conduction (STC) and CSF Conduction

Recently, another mechanism has been proposed as a result of experiments starting in 2011, written mainly by the authors Adelman, Chordekar, and Sohmer. The authors proposed a new type of sound conduction mechanism that they termed soft tissue conduction (STC). In this mechanism, vibration is passed through the soft tissues (i.e. skin, muscle, blood, and other supporting connective tissue) of the head to the inner ear, where it creates basilar membrane motion similar to what occurs traditionally for air conducted stimuli. The exact path to which vibrations are conducted to the inner ear is still being studied, however current evidence suggests that STC vibrations are eventually conducted to cerebrospinal fluid (CSF), where vibrations can directly interface with perilymph via the cochlear aqueduct and create pressure waves in the cochlea. The easiest way to demonstrate evidence for STC is to plug one ear canal to eliminate masking by air conducted noise (this can also be done in a soundproof room or a deeply inserted earplug if the potential confound of the occlusion effect is a concern). Next, lightly scratch the cheek on the side of the plugged ear. Sound perception is elicited; however, the vibrations of the scratching are likely not powerful enough to create vibrations in the bone. Thus, STC must be the modality through which the vibrations are traveling to stimulate the inner ear. Further evidence will be discussed in the section addressing STC later in this paper.

While these five mechanisms of BC make logical sense, understanding the nature of the individual BC mechanisms and how they may contribute to the bone conducted hearing thresholds is important, as this information can help guide interpretation of test results in the context of pathologies that specifically alter the function of the outer, middle, and/or inner ear. A secondary goal of this review was to provide a comprehensive record of the experimental research studies that have contributed to our knowledge of the mechanisms of BC.

Methods

This paper will be using a scoping style of literature review. Scoping literature reviews are often used "to map rapidly the key concepts underpinning a research area and the main sources and types of evidence available, and can be undertaken as stand-alone projects in their own right, especially where an area is complex or has not been reviewed comprehensively before" (Mays, Roberts, & Popay, 2001, p. 194). The fact that the body of literature regarding BC hearing is vast and complex makes this research more suitable for a scoping literature review than any other kind of literature review.

A scoping review of the literature was done using EndNote to compile the sources. The sources for this document were drawn from Scopus, Embase, Medline, and PsychInfo. The research term used in all sources was "(mechanism OR mechanisms)" AND "bone conduction" AND "hearing". The resulting articles were then compiled into one list so that duplicates could be deleted. Then, the titles and abstracts were reviewed to remove irrelevant articles. Because this paper is attempting to compile evidence for mechanisms of BC, non-experimental articles were excluded. In other words, articles merely speculating about the mechanisms of BC or giving theoretical mechanics without evidence were not included. However, articles that compiled other experimental articles, then drew conclusions independent of the conclusions reached by the original authors (as opposed to merely summarizing) were included. There were also no restrictions placed on the article's date of publishing. This is because many of the most compelling and useful articles regarding BC mechanisms were written as far back as the 1920's. Although not recent, the experimental evidence remains relevant as the mechanisms have not changed.

After reviewing the articles obtained from the initial search, it was decided to exclude the following topics: mechanisms of underwater hearing, ultrasonic BC, and math/3D models of the ear. Underwater hearing and ultrasonic BC were excluded because the focus of this paper is to evaluate mechanisms of BC as they apply to everyday hearing and in clinical settings using a bone vibrator, and articles pertaining to underwater hearing and ultrasonic BC do not provide information towards this goal. Regarding articles that use math/3D models of the ear to approximate BC, these were excluded because, as this paper will show, mechanisms of BC involve several incredibly complex mechanisms, all of which are not considered with these models. Indeed, to truly account of all potential sources of BC, one would likely need to model the entirety of the head including the musculature and vasculature at bare minimum to account for contributions due to STC, which are not modeled in the articles obtained.

Results

Using the search terms outlined in the methods section, 262 articles were gathered from Scopus, 205 from Embase, 174 from Medline, and 36 from PsychInfo. These were then combined into a common list, and duplicates were removed, leaving 349 articles. These articles were then analyzed based on reading the title and abstract to eliminate unrelated articles. After this, the full article was read, then deleted if it did not provide info to address the research question or included and categorized according to which section of the ear the mechanism purported to act on: inner ear, middle ear, outer ear, or other. Occasionally, these articles addressed more than one anatomical area, and thus were included in more than one category. After this process, 60 articles in total were included with 26 addressing the inner ear, 34 addressing the middle ear, 12 addressing the outer ear, and 26 articles that addressed STC, and 10 articles addressing CSF conduction.

A total of five categories were developed to facilitate the review and discussion of all articles identified for this review. These categories include: mechanisms involving the outer ear, mechanisms involving the middle ear, mechanisms involving the inner ear, mechanisms involving STC, and mechanisms involving CSF conduction. A table organizing which of these five categories were addressed in the article is included in appendix A.

Along with the 60 articles obtained with the initial search, 10 more sources were gathered to supplement information obtained in the sources from the original search. Appendix A contains only the 60 sources initially obtained, while the bibliography contains all 70 sources used in this paper.

Discussion

Mechanisms Involving the Outer Ear

The Occlusion Effect

The EAM as it is used in this paper refers to the portion of the ear canal between the tympanic membrane (TM) and the outer aperture of the ear canal as it exits the pinna. The EAM is involved during BC most obviously when the occlusion effect occurs. As explained previously, air conducted sound of sufficient amplitude to set the bones/cartilage into motion, or bone conducted sound stimulated via a BC transducer, vibrates the bony/cartilaginous portions of the EAM. This sound energy generally escapes through the unoccluded ear canal. However, when the ears are occluded (usually via the use of insert earphones), then the acoustic energy generated in the EAM can't radiate out, and instead reaches the TM, which is set into motion and stimulates the inner ear in the same way air conducted sound does.

Evidence for the Occlusion effect

A clinically relevant utilization of the occlusion effect is the Bing test. The Bing test was first described by its namesake Alfred Bing in 1891 in his article titled "A new tuning fork test. A contribution to the differential diagnosis of the diseases of the conductive and perceptive hearing apparatus." He described a test involving a tuning fork (or BC transducer in modern times) being placed on the mastoid process on the ear to be tested. The person is first tested with the ear canal unoccluded, and the perceptual loudness is noted. Then the ear canal is plugged using a foam plug or a finger blocking the ear canal. In those with a normal middle ear system, the perception of loudness increases. In those with an abnormal middle ear system, the perception of loudness remains unchanged (Csovanyos, 1961). Even though there is an enhanced signal reaching the TM in this case, the signal is not transmitted efficiently through the abnormal middle ear system. Numerous authors have tested this theory and have all confirmed the tests' validity (Clemente, 1951; Onchi, 1954). This effect can also be measured objectively by performing a bone conducted ABR. For example, when the ear canal is occluded and 1,024 Hz tonebursts are used, the amplitude of the bone-conducted ABR waves will increase and the latencies will decrease in those with normal middle ears (Bochenek & Bochenek, 1972).

It should be noted that the sound energy created in the EAM, although generated using a BC transducer, ends up reaching the TM through AC. This is evident by the fact that occlusion increases loudness perception in those with normal hearing or sensorineural hearing loss, and not in those with conductive hearing losses, as previously discussed in the above paragraph. Further experiments concluded that traditional bone transducers conduct vibration energy to the bony portion of the ear canal primarily which may travel to the cartilaginous portion coincidentally due to its close proximity (Nishimura et al., 2015).

Frequency Characteristics of the Occlusion Effect

The occlusion effect does not happen equally across frequencies. Békésy found that occluded BC has a resonance peak at 200 Hz. At the time, he postulated that this was due to contribution of the mandible. Shibui (1959) demonstrated that EAM resonant peaks in individuals without mandibles that were tested using Békésy's method during occluded BC did not change (Shibui, 1959). This therefore confirms that the EAM has a resonance peak of 200 Hz with no contribution of the mandible. In other words, the EAM resonates around 200 Hz and therefore increases sound pressure levels in the ear canal which improve thresholds (if the ear canal is occluded). Accordingly, the occlusion effect improves thresholds most for low frequencies (average of 23 dB HL improvement around 200 Hz) with diminishing returns as frequency increases until there is little change at 2000 Hz (Aazh et al., 2005).

Mechanisms Involving the Middle Ear

Although BC hearing, in theory, is thought to be an indication of cochlear hearing status, studies have shown that mechanisms that alter the admittance of the middle ear can alter BC thresholds. In this section, I review evidence for inertial BC. For simplicity, I include mechanisms related to the TM and the middle ear as anything that alters the admittance of the TM will also inevitably alter the admittance of the middle ear and its structures. Recall that the inertial BC, for a healthy middle ear, relies on a lag between the inertia of the ossicles and the inertia of the middle ear space. Thus, most evidence for this mechanism has relied on examining the changes in BC that occur when the admittance of the TM and middle ear is altered thus changing the inertial lag between the ossicles and middle ear space.

Evidence for Inertial Bone Conduction: TM stiffening mechanisms

Pressure changes have been shown to cause changes in BC thresholds. This phenomenon is the basis of a test for conductive hearing loss that is similar to the Bing test called the Gellé test. Named after Marie Ernst Gellé, who invented the test in 1881, the premise of the test is first the individual makes note of the subjective loudness of a bone conducted tone (at the time this was done using a tuning fork held against the mastoid). Then pressure is introduced into the EAM using a pneumatic otoscope. If the person has normal hearing or sensorineural hearing loss, the loudness of the tone should decrease. Traditionally, a 512 Hz tuning fork is used to administer the Gellé test. If the person has a middle ear pathology that is causing a conductive hearing loss, then the loudness of the tone will not change. Since its invention, the mechanism behind the Gellé test has been determined to likely be because pressure changes that are introduced into a healthy ear stiffen the tympanic membrane and therefore the ossicular chain. This stiffening of the ossicular chain then interferes with its ability to contribute to BC (Arnold & Schindler, 1963).

The effect of pressure on BC results, like the occlusion effect, differs across frequencies. A study by Homma and colleagues (2010) determined that when -400 mmH_2O is introduced to the adult ear canal, ossicle velocities decreased (which therefore decreased sound pressure levels in the cochlea) the most (compared to the control condition of 0 mmH_2O) by about 8.6 dB at 1.6 kHz, with decreasing effects as the frequency deviated from 1.6 kHz both higher and lower. The authors concluded that the reason this maximum decrease in ossicle velocity occurs at 1.6 kHz is because the resonant frequency range of the ossicles is around 1.6 kHz. Therefore, when the ossicles are fixed by stiffening the TM, the contribution of the resonant frequency of the ossicles is eliminated, resulting in a decrease in ossicular motion compared to normal conditions. This finding is consistent with findings from many other studies (Aoki et al., 1996; Baisakhiya & Singh, 2019; Browning & Gatehouse, 1989; Chhan et al., 2016; Goodhill, 1966; Hempel et al., 2012; Holcomb, 1958; Kitahara et al., 1994; Lee et al., 2008; Marres, 1965; Nakai, 1975; Smyth et al., 1981; Zhang et al., 2016).

Evidence for Inertial Bone Conduction: Otitis Media and Otosclerosis

Stiffening of the TM and ossicular immobilization also occur in otitis media. As such, otitis media often causes a decline in BC thresholds particularly around 2000 Hz. Placement of tympanostomy tubes or resolution of the ear infection also has been noted to reverse the decrease in BC threshold associated with otitis media (Aoki et al., 1996; Browning & Gatehouse, 1989; Lee et al., 2008; Nakai, 1975; Ohhashi et al., 1980). Thus, it can be concluded that the decrease in BC in these cases must be due to a transient change in middle ear mechanisms of BC.

Similarly, many clinical audiologists are familiar with the concept of the “Carhart’s notch,” which describes the presence of a relative decrease in bone conducted hearing ability compared to neighboring frequencies in those with otosclerosis (Katz et al., 2014). Generally, this notch is seen at 2 kHz during routine clinical audiometry. Combining this information with the findings from the previous paragraph, one can conclude that Carhart's notch is likely due to the same phenomenon. In both cases, the ossicular chain has been essentially immobilized Thus, it can be concluded that the decrease in BC in these cases must be due to a transient change in a middle ear mechanism of BC.

Evidence for Inertial Bone Conduction: TM Changes

Regarding TM perforations, similar findings as those mentioned above have been recorded. In those with a large TM perforation, deficits are seen in high frequency BC, especially at 2000 Hz. Generally, frequencies below 1000 Hz are not affected by the presence of a perforation. Also, the amount of decline in BC results varied, however they generally declined further the greater the size of the perforation was. After the perforation was fixed or healed, the decreased BC results almost always recovered completely to match the contralateral ear (Hempel et al., 2012; Holcomb, 1958).

In cases where the TM has been too flaccid, a similar pattern of decreased BC results around 2000 Hz appears (between 1000 and 3000 Hz). Once the TM was surgically stiffened, BC results improved substantially between 1000 and 2000 Hz (Proctor & Proctor, 1970). This loss in BC results that improve once the problem is remedied is further evidence that decreased BC is not always indicative of permanent sensorineural hearing loss.

Frequency Characteristics of Inertial Bone Conduction

Interestingly, while BC results were significantly different (especially around 2000 Hz) in the cases mentioned previously, in those with stapes immobilization due to otosclerosis, many times BC does not change significantly after stapedectomy (Hall & Rytzner, 1958; Hüttenbrink et al., 2004; Révész et al., 2014). Among those that do report an improvement in BC results, it is reported to be at 500 and 1000 Hz, not 2000 Hz like previously discussed (Ohbuchi et al., 2012).

In fact, while a large number of articles support the conclusion that inertia of the ossicles contributes the most to bone conducted hearing around 2000 Hz, Röösli and colleagues in 2012 reached a different conclusion. These authors measured the velocity of the umbo compared to the velocity of the outer rim of the TM (which approximated the motion of the skull). In this experiment, the authors found that the difference in velocities during BC stimulation was greatest below 500 Hz. This difference in velocity was concluded to be differences in the velocity of the skull and the velocity of the ossicles due to the inertia of the ossicles, so the authors concluded that the inertia from the ossicles must contribute the most to BC below 500 Hz. They also noted that the difference in velocity during BC was significantly smaller than the umbo velocity during AC stimulation at threshold levels. The velocity of the umbo at threshold levels during AC is assumed to be the smallest amount of ossicle motion possible that can elicit hearing. Therefore, the authors concluded that the inertia of the ossicles must be too small to elicit hearing sensation. Legouix and Tarab (1959) also reported similar results to Röösli, stating that fixation of the ossicles led to the largest change in the cochlear microphonic of guinea pigs at 500 Hz.

However, Röösli and colleagues noted that there was a large amount of variability between subjects along with a noise floor that potentially masked out certain responses. In fact, a similar study conducted by Irvine, Yates, and Johnstone (1979) encountered similar issues. When they tried to measure the motion of the ossicles during AC as well as during BC to approximate the contribution of ossicular inertia, their measurements were too small to rise above the noise floor.

Comparing the previously discussed articles in which 2000 Hz was the most affected frequency range and these articles reporting that 500 Hz is the most affected, there is one obvious difference between the two groups of articles. In general, the articles that found that 2000 is the most affected were based on changes in audiometric measures. In contrast, the other group of articles measured 500/1000 Hz as the most affected through objective measures, like measuring the amount of displacement of the ossicles during BC stimulation. It seems that a possible explanation for this discrepancy in measured frequency characteristics may be explained by considering that objective measures of ossicular displacement may not necessarily directly translate to equivalent changes in loudness perception. In other words, although ossicular displacement was largest at 500/1000 Hz, it may be that other unknown factors come into play regarding subjective perception of sound. Therefore, although ossicular displacement was largest at 500/1000 Hz, the largest perceived difference in loudness during BC may still occur at 2 kHz.

Another consideration is that there were many fewer articles that concluded 500/1000 Hz was the most affected as opposed to those that concluded 2000 Hz is the most affected. More research needs to be done to determine the exact nature of inertial BC and how much the ossicles truly contribute to the overall precept of sound during BC stimulation.

Summary

At first, it may seem like the EAM and the TM/ossicular chain have little impact on BC, since BC is often described as a test that bypasses the outer and middle ear, testing the cochlea independently. However, many studies, such as those reviewed in this paper, have shown that the EAM (osseotympanic mechanism) and the middle ear (inertial mechanisms) contribute to BC hearing. This is an important conclusion, as it shows that BC thresholds do not necessarily represent true “cochlear potential,” and can be influenced by middle or outer ear pathology.

While the EAM and middle ear obviously contribute to BC overall, it is important to note that not this contribution seems to be relatively small compared to other mechanisms. In fact, a study by Yehezkely and colleagues (2019) examined BC hearing thresholds in individuals who had radical mastoidectomies and/or subtotal petrousectomies. Despite not having a functioning TM or ossicles, 30% and 8% (respectively) had BC thresholds better than or equal to 15 dB HL, despite missing traditional mechanisms for inertial and osseotympanic components of BC. This article reconfirmed findings from an earlier article regarding those with labyrinthine destruction (Ohhashi et al., 1980). Evidently, the EAM, TM, and ossicles are not the sole contributor to BC sound perception, and in fact may play a relatively small role in BC overall. This concept was confirmed when an article by Chhan and colleagues was published in 2013 in which the ossicles were decoupled from the cochlea. This essentially separated the outer and middle ear from the inner ear, thus preventing them from contributing to BC responses. When changes in pressure in the scala tympani and scala vestibuli were measured, AC pressures decreased about 30-40 dB, while BC pressures decreased only 10-20 dB. This supports the conclusion that BC responses are largely due to other mechanisms. These other mechanisms will be discussed in the following sections.

Mechanisms Involving the Inner Ear

Many researchers have suggested that BC does not only rely on the middle ear structures. They suggest considering the contribution from the two windows of the cochlea, the round window and the oval window. The oval window is where the stapes connects to the cochlea and allows pressure waves to be conducted from the ossicles into the cochlear fluid or perilymph/endolymph. Since fluid cannot be compressed, this pressure needs a way to displace the fluid somehow, otherwise the impedance would be too high and most of the sound energy would be reflected. The round window is a pliable membrane covered aperture through which the fluid can be displaced, allowing pressure waves to be conducted into the cochlea for sound perception. Therefore, if the cochlea is involved in BC hearing, it would be safe to assume that two mobile windows must play some role.

Evidence for Compressional Bone Conduction

When the mechanisms of BC were first explored, they were poorly understood. It was unclear what role the cochlea played in BC perception, if any. This question was answered when Wever and Lawrence (1954) found that a tone delivered via BC could be cancelled out when a tone of the same frequency, but 180° out of phase was delivered via AC. Because the tone was able to be cancelled out by shifting the phase, they were able to conclude that BC sound must be delivered to the cochlea. Further evidence for a cochlear site of activation during BC was found by Watanabe, Bertoli, and Probst in 2008 when distortion product otoacoustic emissions could be generated with f1 being delivered by AC and f2 being delivered by BC. Once it was determined that the cochlea is involved in BC, the next question was how the cochlea functioned during BC stimulation. Herzog and Krainz proposed the concept of compressional BC in 1926. These writers described compression and expansion of the cochlear capsule. However, at this time, but it was unknown the route through which this compression occurred.

Subsequent experiments determined that functional cochlear windows are an important contributor to BC responses. In these subsequent articles, either the round window was blocked, the stapes was immobilized in the oval window, or the windows were affected by cochlear atresia (and therefore not as mobile), and BC responses were measured. In these cases, BC results were decreased when compared to control conditions (Chhan et al., 2016; Linder, Ma, & Huber, 2003; Perez, Adelman, & Sohmer, 2011a; Perez, Adelman, & Sohmer, 2011b; Sawashima, 1958; Stieger et al., 2018).

While summarizing the conclusions from these articles, an interesting pattern emerged. Some articles reported a relatively small change in bone conducted responses (about 5 dB) (Chhan et al., 2016; Perez, Adelman, & Sohmer, 2011a), while others reported a large decrease in bone conducted responses, when the two cochlear windows were blocked (about 20 dB)

(Linder, Ma, & Huber, 2003; Sawashima, 1958). Upon further investigation, it seems the main differences in these two groups of articles is that those that found a small change were using low intensity stimulation to find changes in thresholds, while the others used a higher intensity and measured the change in amplitude of the response to the stimulus. In other words, it seems that perhaps low intensity bone conducted stimulation relies less on needing two mobile windows in the cochlea than higher intensity stimulation.

While immobilization of the cochlear windows negatively influences BC responses, AC responses always declined more than bone (Linder, Ma, & Huber, 2003; Perez, Adelman, & Sohmer, 2011a; Chhan et al., 2013). In fact, a study by Stieger and colleagues in 2018 found that while measuring motion at the round and oval windows during AC and BC, the velocities of the two windows were not equal, while they were for AC. Traditionally, whatever input is delivered to the oval window should be able to be measured equally at the round window since the pressure has nowhere else to release. Therefore, measuring different velocities between the two windows suggests there must be another mechanism of input besides the oval window. This conclusion is also supported by the fact that BC simulation still could elicit hearing perception even when both windows have been immobilized (Chhan et al., 2016; Perez, Adelman, & Sohmer, 2011b; Sawashima, 1958). Therefore, not only does BC have another mechanism of input besides the oval window, but one can also conclude that BC does not necessarily completely rely on two functioning cochlear windows. In fact, a 3rd window mechanism has been proposed as contributing to compressional BC.

Bone Conduction Mechanisms with a 3rd Window

As previously discussed, the cochlea has two windows that allow for pressure release during stimulation. The term “third window lesions” refers to pathologies that create a weakness or dehiscence in the bony labyrinth, effectively creating a third “window.” In other words, these third windows allow another way for the sound pressure in the cochlea to be released other than the oval and round windows. Third window lesions include large vestibular aqueduct syndrome (LVAS) and superior semicircular canal dehiscence (SSCD), among many others. Usually, third window lesions are focal in nature, but may also be pathologically diffuse, like in Paget's disease, where general bone density of the otic capsule is decreased (Monsell, 2004). In these pathologies, a low frequency air-bone gap is often recorded (Attias et al., 2012; Chien et al., 2007; Kitahara et al., 2011; Kubota et al., 2015; Merchant et al., 2007; Merchant & Rosowski, 2008; Rosowski et al., 2004). When audiograms of these patients are compared over the progression of the pathology, it becomes clear that the cause for this air-bone gap is not necessarily just the worsening of AC thresholds, but also the improvement of BC thresholds. The origin of this air bone gap seems counterintuitive, an air-bone gap is almost always indicative of a conductive hearing loss, one that affects the middle ear. If this is the case, then why would a pathology whose site of lesion is the cochlea cause an air-bone gap?

As it turns out, the differences in AC and BC in individuals with a third window lesion is likely due to the difference in how BC and AC stimulate the cochlea. Studies that have measured the displacement of the round window/ossicles found that during AC, velocities at the round window were decreased, while velocities of the ossicles or oval window is increased (Chien et al., 2007; Merchant & Rosowski, 2008; Rosowski et al., 2004). As discussed before, the movement of the windows should be equal and opposite in normal systems. Therefore, these measurements suggest that energy that arrives at the oval window must be being redirected to another place other than the oval window. In other words, a third window lesion allows for pressure to be routed away from the round window into the dehiscence during AC. Several articles seem to come to this same general conclusion (Attias et al., 2012; Chien et al., 2007; Kitahara et al., 2011; Kubota et al., 2015; Merchant et al., 2007; Merchant & Rosowski, 2008; Rosowski et al., 2004). Consequently, some of the sound energy is routed away from the basilar membrane, decreasing displacement and therefore sound perception, worsening AC thresholds. This hypothesis that sound energy is shunted into the dehiscence was shown to be correct when sound pressure was able to be measured through the dehiscence (Chien et al., 2007; Rosowski et al., 2004). This shunting of energy occurs during air conducted stimulation but doesn't occur, or occurs to a lower extent, during BC.

Now the question is why bone conducted stimulation does not cause sound energy to flow through the dehiscence to the same degree as AC stimulation does. An article by Merchant and Rosowski entitled "Conductive Hearing Loss Caused by Third-Window Lesions of the Inner Ear" (2008) hypothesizes that, while air conducted sound coming in through the oval window gets redirected into the third window, BC behaves differently. Because of compressional mechanisms of BC, energy does not necessarily have to enter the cochlea through the oval window. Compressional forces "squeeze" the cochlea equally over the entirety of the structure. Because the impedances of the round window and oval window are different, the impedances between the scala tympani and scala vestibuli are also different. Therefore, a pressure difference forms across the cochlear partition resulting in motion of the basilar membrane, and therefore sound perception. However, when a third window is present, the difference in impedances between the scala tympani and scala vestibuli is increased because the density of the liquid in the scala vestibuli is lowered due to an increase in volume from the third window. When the impedances between the scalas is increased, then compressional BC can create a large pressure difference and cause greater displacement of the basilar membrane, therefore improving BC thresholds.

According to this hypothesis, improved BC in those with third window lesions are evidence supporting the mechanism of compressional BC. However, the exact cause has not been definitively determined, and more study needs to be done.

Mechanisms Involving Soft Tissue Conduction

As its name suggests, soft tissue conduction (STC) entails conduction of vibrations generated by either AC or BC stimuli. Perez, Adelman, and Sohmer in 2011 and again in 2012 conducted studies in which the ossicles were removed and the windows of the cochlea immobilized in fat sand rats. In these cases, BC ABR thresholds remained unchanged or only shifted about 4 dB, even without the ability to induce inertial nor compressional BC. Inertial BC could not be induced because the ossicles were immobilized, and compressional could not be induced because the cochlear windows were immobilized, therefore eliminating the aperture for pressure release, thereby making it theoretically impossible to displace the fluid in the cochlea. In addition, an ABR threshold could be measured at a level of 23.5 dB, even when the BC transducer was applied to the eye without touching bone (Perez, Adelman, and Sohmer, 2011a). Thus, these authors concluded that sound perception can be elicited without the use of inertial nor compressional BC, and suggested a mechanism of conduction not involving osseous components (i.e. skull vibration, inertial motion of the ossicles, compressional forces on the cochlea). These results were replicated by Perez and colleagues when the same alterations were made to the ossicles and cochlear windows in fat sand rats, and AC responses worsened while BC and STC responses did not change (Perez et al., 2014).

In fact, a simple way to demonstrate STC is to plug the ears and lightly stroke the cheek with a finger. Sound can be heard in the ipsilateral ear, despite the fact that AC sound generated cannot reach the plugged ear, and that the stimulation is not strong enough to initiate vibrations in the bone (Sohmer, 2017). Since this discovery, further studies have been conducted to try to determine the nature of this stimulation and the mechanisms of conduction.

Evidence for Soft Tissue Conduction

In clinical BC testing, application force of the BC transducer is considered important to be able to transfer the vibrations to the skull bones. However, in a study by Chordekar, Adelman, and Sohmer in 2013, a BC transducer was applied to gel on the skin using 0 grams of application force, and the same thresholds were obtained when compared to the same transducer on the forehead with 500 grams of application force. This study was replicated by Geal-Dor and colleagues (2015), and similar results were found. In short, thresholds obtained to a BC transducer with and without application force are similar, suggesting that a large proportion of clinical BC thresholds are contributed by a non-osseous mechanism, which is likely STC. While these studies found that BC thresholds did not change with changes in application force, it should be considered that other articles have found that application force of the BC transducer in neonates using ABR does influence threshold (Yang et al., 1991).

Further testing has been performed to try to elucidate the mechanisms of STC. At first, it was hypothesized that STC utilized the same mechanisms as traditional osseous BC. When drugs were introduced that decrease the cochlear amplifier by De Jong and colleagues in 2011, AC, BC, and STC responses universally decreased. This suggests that STC hearing perception is based in cochlear activation, as are AC and BC. This was confirmed when it was determined that AC, BC, and STC are able to cancel one another when phase shifted (Chordekar et al., 2012).

However, when the outer ear canal was occluded, only BC responses increased (using the same mechanisms as previously described in the Bing test) (De Jong et al., 2011). This suggests that, while STC and BC are similar, they have different mechanisms of activation. In another study, laser vibrometry was conducted to measure skull vibration during both BC and STC stimulation. The authors in this study concluded that “skull vibration magnitude differences can adequately explain hearing threshold differences and are likely to be responsible for the hearing sensation" (Chordekar et al., 2018). In other words, STC and BC likely share a similar mechanism, skull vibration.

In fact, it seems that STC and BC may share several similar mechanisms. When conducting the Weber test on STC sites, STC is lateralized to the side of a conductive pathology, like BC (Adelman, Lotem, & Sohmer, 2011). Also, when a conductive hearing loss is induced, like using deeply inserted earplugs or making alterations to the ossicular chain, AC thresholds became poorer, while BC and STC did not change significantly (Adelman et al., 2015; Perez, Adelman, & Sohmer 2011a; Perez, Adelman, & Sohmer, 2011b; Perez, Adelman, & Sohmer 2012; Ronen et al., 2017). In addition, BC transducers with no application force on skin still generate hearing perception that is largely unchanged when compared to a BC transducer with 5 N of force, as previously mentioned. Therefore, it seems that traditional BC and STC share many mechanisms of activation. This may be explained by considering that BC may actually be largely composed of STC. However, more investigation into the phenomenon of STC needs to be done before a conclusion can be made.

While many mechansism appear to be similar for BC and STC, they likely do not share the exact same mechanisms. Evidence for this difference in mechanisms was shown when masking was introduced to a STC site and ABR thresholds were measured to BC stimulation. Then, the roles were switched, masking was introduced to the BC site while ABR thresholds were measured to STC stimulation. Results showed a difference in the amount of elevation in thresholds between the two situations. This suggests that the two types of stimulation are not variations of the exact same mechanisms (Perez, Adelman, & Sohmer, 2011b).

Like previously mentioned, STC is a relatively newly discovered mechanism, and as such needs to have much more research conducted to determine the exact mechanism of stimulation. STC and BC may share mechanisms, and BC may also be largely composed of STC. The amount of this contribution is heretofore unknown. However, this newly discovered mechanism is an important factor to consider, particularly during clinical BC testing.

Mechanisms Involving CSF Conduction

Along with the previously mentioned mechanisms, there is some evidence for other mechanisms of BC transmission that do not fall into the other categories. The most prominent example of these other mechanisms is the concept of cerebrospinal fluid (CSF) transmission. CSF transmission describes the way through which sound can travel through CSF, where it then interfaces with the cochlear aqueduct and creates basilar membrane motion.

Freeman, Sichel, and Sohmer (2000) were the first to conduct experiments that touched on this topic. They first delivered a BC signal to animals and measured an ABR. Then they extensively removed skull bone and determined that this did not affect the measured ABR significantly. Subsequently, they drained CSF from the animal, and ABR thresholds to BC stimuli increased, while ABR thresholds to AC stimuli did not. Therefore, they concluded that not only is CSF important to BC responses, but also that the amount of CSF can also influence BC ABR thresholds.

Then, Sohmer and colleagues (2000) conducted a study in which BC stimulation was delivered in neonates over the fontanelle, and an ABR was obtained. The authors of this article concluded that stimulation over the fontanelle was unlikely to induce vibration in the bone, and thus would not be able to induce inertial nor compressional BC, and therefore the ABR measured would likely be the result of CSF conduction. In another test, audiometric thresholds were also measured in neurosurgical patients on the skin over a craniotomy. These thresholds were no different than those obtained with the transducer over bone in the same patients. Finally, audiometric responses were obtained with the BC transducer on the eye, which acts like a natural craniotomy. These responses were no different than those obtained with the transducer on various parts of the skull.

Sohmer and Freeman in 2004 conducted one of the first experiments to investigate the phenomenon of CSF conduction thoroughly. In their experiment, two rats' CSF were coupled together via a plastic tube connected to a craniotomy in each rats' skull. When one rat was delivered BC stimulation, an ABR was able to be obtained from the other rat. Various alterations to the experimental parameters determined that this response was due to CSF conduction, not any other means like AC contamination or conduction through the plastic of the tubes.

Finally, there have also been theories that pulsatile tinnitus is largely due to a combination of a bony weakness (like a 3rd window) along with transmission of vibrations through bodily fluids to the cochlea (Adelman et al., 2014; Bertholon et al., 2018; Kim et al., 2016). Or in other words, the theoretical path of transmission for pulsatile tinnitus is the same as the path of transmission described for CSF conduction.

It is unclear if STC and CSF are due to the same mechanisms of conduction. It may be that the two mechanisms of conduction actually describe the same phenomenon, which gives rise to possible confounds. For example, in articles claiming to give evidence to STC and those that claim to give evidence for CSF conduction, both use the eye as a site of stimulation. As both are newly discovered mechanisms of sound conduction, not many experiments have been conducted in general, and as such, further experimentation is needed.

Conclusion

In summary, BC transmission is composed of several intricately related mechanisms involving several different sites and methods of transmission. In fact, it has been postulated that various mechanisms of BC may be at odds with one another. In other words, one mechanism of BC may be out of phase with another, and therefore may create destructive interference, thereby decreasing the overall BC response. This idea was the main argument in a 2002 article by Sichel, Freeman, and Sohmer, in which they attempted to explain the mechanism of lateralization behind the Weber test. In this experiment, they measured ABR thresholds in normal fat sand rats, then again after ossicular fixation. After ossicular fixation, AC ABR thresholds worsened, while BC ABR thresholds improved. Their theory behind this improvement in BC thresholds was that inertial BC is out of phase with the BC responses generated by other mechanisms. Therefore, by eliminating inertial BC by fixating the ossicles, BC responses can increase, because there is no longer destructive interference. They also used this logic to explain the pattern of lateralization during the Weber test (a tuning fork held to the forehead will lateralize to the better ear in SNHL or to the poorer ear in a CHL).

Therefore, it may be that any changes made to attempt and isolate one mechanism of transmission will inevitably influence the overall response in ways not intended. Attempting to isolate one mechanism may actually improve the ability of another mechanism to impart sound conduction to the cochlea. This is an unavoidable confound of investigating mechanisms of BC. Not only this, but truly isolating one mechanism of conduction may be impossible, given how interconnected they all are. For example, a previously discussed experiment attempted to eliminate inertial and compressional BC by immobilizing the ossicles and the cochlear windows (Perez, Adelman, & Sohmer, 2011a). They concluded that sound perception after these alterations must be due to STC, however other experiments have suggested that immobilization of the cochlear windows does not necessarily inhibit the ability for cochlear activation to occur through compressional BC, particularly in the presence of a 3rd window (Chhan et al., 2016; Linder, Ma, & Huber, 2003; Rosowski et al., 2004; Sawashima, 1958; Stieger et al., 2018).

It also should be mentioned that clinical BC using a BC headband is fraught with issues regarding calibration as well as differences between subjects' skulls and individual anatomical differences. For example, it is not uncommon to measure BC thresholds as higher than AC thresholds, which should be theoretically impossible. In reality, there are many technical considerations that likely influence BC results. In almost all articles discussed, a clinical BC transducer was used, meaning that the same calibration and technical limitations that also apply to clinical BC testing.

In short, further testing needs to be done to determine not only the mechanisms of BC, but how they interact, what the frequency characteristics are, and how they contribute to the overall percept of BC sound. As this paper has demonstrated, clinically measured BC thresholds may not be an accurate representation of cochlear function, since it is influenced by other structures. However, understanding the mechanisms of clinical BC is important for understanding how certain pathologies can influence BC, and therefore inform how BC results may be affected. Also, for individuals with BC hearing instruments, measurement of BC may be all the more important, since BC is the way sound is delivered into the system. In other words, while clinically measured BC may not be an accurate measure of cochlear function, it does accurately represent how BC sound is conducted into the system, and therefore is vital in assessing BC hearing instrument candidates.

Appendix A

Full Citation	OE	ME	IE	STC	CSF
Aazh, H., Moore, B., Peyvandi, A. A., & Stenfelt, S. (2005). Influence of ear canal occlusion and static pressure difference on bone conduction thresholds: Implications for mechanisms of bone conduction [Article]. *International Journal of Audiology, 44*(5), 302-306. https://doi.org/10.1080/14992020500060669	X	X-			
Adelman, C., Chordekar, S., Perez, R., & Sohmer, H. (2014). Investigation of the mechanism of soft tissue conduction explains several perplexing auditory phenomena [Review]. *Journal of Basic and Clinical Physiology and Pharmacology, 25*(3), 269-272. https://doi.org/10.1515/jbcpp-2014-0037				X	
Adelman, C., Cohen, A., Regev-Cohen, A., Chordekar, S., Fraenkel, R., & Sohmer, H. (2015). Air conduction, bone conduction, and soft tissue conduction audiograms in normal hearing and simulated hearing losses [Conference Paper]. *Journal of the American Academy of Audiology, 26*(1), 101-108. https://doi.org/10.3766/jaaa.26.1.11			X-	X	
Adelman, C., Lotem, S., & Sohmer, H. (2011). Lateralization determined by weber test on bone and on soft tissue [Conference Abstract]. *Journal of Basic and Clinical Physiology and Pharmacology, 22*(3), 83. https://doi.org/10.1515/JBCPP.2011.016	X-			X	
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Baisakhiya, N., & Singh, G. (2019). Study the Effect of Tubotympanic Disease on Bone Conduction Threshold [Article]. *Indian Journal of Otolaryngology and Head and Neck Surgery, 71*, 1357-1363. https://doi.org/10.1007/s12070-018-1424-2		X			
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Chordekar, S., Adelman, C., & Sohmer, H. (2013). The non-osseous (soft tissue) component of bone conduction thresholds [Conference Abstract]. *Journal of Basic and Clinical Physiology and Pharmacology, 24*(3), A5.				X	
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Freeman, S., Sichel, J. Y., & Sohmer, H. (2000). Bone conduction experiments in animals - Evidence for a non-osseous mechanism [Article]. *Hearing Research, 146*(1-2), 72-80. https://doi.org/10.1016/S0378-5955(00)00098-8					X
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Hall, A., & Rytzner, C. (1958). Stapedectomy and cochlear function [Article]. *Acta Oto-Laryngologica, 49*(1), 282-293. https://doi.org/10.3109/00016485809134757		X			
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