Review Article

Soft Tissue Conduction: Review, Mechanisms, and Implications

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Abstract

Soft tissue conduction (STC) is a recently explored mode of auditory stimulation, complementing air (AC) and bone (BC) conduction stimulation. STC can be defined as the hearing induced when vibratory stimuli reach skin and soft tissue sites not directly overlying skull bone such as the head, neck, thorax, and body. Examples of STC include the delivery of vibrations to the skin of parts of the body by a clinical bone vibrator, hearing underwater sounds and free field air sounds, while AC hearing is attenuated by earplugs. The vibrations induced in the soft tissues are apparently transmitted along soft tissues, reaching, and exciting the ear. Further research is required to determine whether the mechanism of the final stage of STC hearing involves the excitation of the ear by eliciting inner ear fluid pressures that activate the hair cells directly, by the induction of skull bone vibrations, or by a combination of both mechanisms, depending on the magnitude of each mechanism.

Keywords

soft tissue conduction, air conduction, bone conduction, hair cells, cochlear amplifier, basilar membrane, threshold, acoustic impedance

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Introduction

Hearing by Air Conduction

In most situations, hearing involves air conducted (AC) sounds reaching the tympanic membrane and the ossicular chain. The established view is that during AC stimulation the vibrations of the stapes footplate in the oval window are accompanied by opposite phase vibrations of the round window, producing a pressure difference across the basilar membrane, which initiates the passive traveling wave along the basilar membrane (Oghalai, 2004; von Bekesy, 1960). In the clinic, AC is used together with bone conduction (BC) for the differential diagnosis between a conductive and a sensorineural hearing loss.

Hearing by BC

The generally accepted view is that BC is elicited when the clinical bone vibrator is applied with a static force of 5 Newton (5 N) to the mastoid or forehead overlying skull bone, inducing vibrations of the underlying bone, which are transmitted along skull bone to the temporal-petrous bone, leading to vibrations of the walls of the inner, middle, and outer ears (Stenfelt, 2011; Stenfelt & Goode, 2005). As a result, the multiple, coexisting mechanisms of BC (inner ear fluid inertia, compression of inner ear bone, ossicular chain inertia, and the occlusion effect) are induced. Each of these BC mechanisms (perhaps each with different onset intensities and different phases, and dominating at different frequency regions) also lead to pressure differences across the basilar membrane and to a passive traveling wave, as in AC hearing (Chhan, Roosli, McKinnon, & Rosowski, 2013; Stenfelt, 2011; Stenfelt & Goode, 2005; Tonndorf, 1968). This hypothesis is supported by the existence of interactions between AC with BC, such as cancellation (Chordekar, Kriksunov, Kishon-Rabin, Adelman, & Sohmer, 2012), mutual beats, masking

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(Adelman, Fraenkel, Kriksunov, & Sohmer, 2012), and otoacoustic emissions (Watanabe, Bertoli, & Probst, 2008).

Hearing by Soft Tissue Conduction

Soft tissue conduction (STC) is a recently explored mode of auditory stimulation, complementing the better known modes of AC and BC stimulation. A form of STC can be easily demonstrated by occluding the external auditory meatus with a finger to block AC masking sounds and then gently stroking the cheek (or stubble or an earring) on the same side of the face as the occluded ear. The auditory sensation then perceived is STC, since AC has been blocked, and the gentle stroking of the cheek was likely too weak to elicit actual vibrations of skull bone (which are assumed required for BC hearing). Furthermore, the stroked cheek is not even over skull bone, but rather over the oral cavity, and it is generally agreed that a prerequisite for the initiation of the BC mechanisms is the induction of vibrations of skull bone (Stenfelt, 2011; Stenfelt & Goode, 2005). The vibrations induced in the soft tissues of the face by the gentle stroking presumably reach and activate the inner ear.

Forms of Soft Tissue Condition

A clinical bone vibrator can also elicit STC hearing when, in the presence of earplugs to block AC, it is used to deliver low-intensity vibratory stimulation to sites on the head not directly overlying skull bone such as the eye (Ito et al., 2011; Sohmer, Freeman, Geal-Dor, Adelman, & Savion, 2000; Watanabe et al., 2008), and to skin sites on the neck, thorax, and body that are not near or over skull bone, and even quite distant from the ear (skin over the lower thoracic vertebra; Adelman, Yehezkely, Chordekar, & Sohmer, 2015; Kaufmann, Adelman, & Sohmer, 2012). Interestingly, in superior canal dehiscence patients, vibratory stimuli delivered even to the ankle-elicited hearing so that the tissue vibrations induced at the ankle are likely transmitted over a greater body distance to the ear. It is suggested that the opening (dehiscence) between perilymph and cerebrospinal fluid (CSF) at the superior semicircular canal probably facilitates activation of the inner ear (Brantberg, Verrecchia, & Westin, 2016). Furthermore, attempts to maximize hearing protection in especially noisy environments (e.g., weapon systems where sound can reach 150 dB SPL, and during functional magnetic imaging which reach 123 to 138 dB SPL) have led to the term *body conduction* for the hearing even in the presence of deeply inserted ear plugs and helmets (Berger, Kieper, & Gauger, 2003; Ravicz & Melcher, 2001). Body conduction is therefore a synonym for STC since the helmets shielded the head and skull from the sound field, preventing the initiation of BC mechanisms. In these situations, the vibrations induced in the tissues are likely transmitted along soft tissues (hence STC; Brantberg et al., 2016), eventually reaching and stimulating the inner ear.

It has also been shown that in the presence of customized deeply inserted earplugs coupled with earmuffs (which together attenuate free field AC sounds by 40 to 46 dB), the threshold to free field sound from a loudspeaker of normal hearing participants was 46 to 54 dB HL (Chordekar, Adelman, Sohmer, & Kishon-Rabin, 2016). On the other hand, effective BC mechanisms can be initiated when the free field sound intensity is greater than about 60 dB HL (Steiger, 2015), that is, the thresholds of the participants were lower than those which could elicit BC mechanisms. In these conditions, the free field sound in air apparently induced vibrations of skin and soft tissues of the body, which reached the ear.

An additional example of STC is hearing of threshold level sound from an underwater source, even when only the forehead is under water, and the ears equipped with ear plugs are in air above the water. Therefore, the subject is not responding to AC sounds. Also, as a result of the differences in physical properties (density and sound velocity) between water and skull bone, it is not likely that the threshold level sound in water could induce vibrations of skull bone (Chordekar, Kishon-Rabin, Kriksunov, Adelman, & Sohmer, 2015). The sound field underwater is actually in direct contact with the soft tissues of the head and body. Hearing is then likely induced by the vibrations initiated in the soft tissues of the body, which reach the ear (Chordekar et al., 2015).

STC Without Direct Physical Contact Between Sound Source and Skin

In addition, a threshold auditory sensation can also be elicited even without direct contact of the bone vibrator with the skin, as when the clinical bone vibrator is applied to fluid or to a layer of ultrasound gel on the skin of the head, in the presence of earplugs (Geal-Dor, Chordekar, Adelman, & Sohmer, 2015). Furthermore, the delivery of vibratory stimulation to fluid applied to the external auditory meatus also elicits hearing (Perez, Adelman, & Sohmer, 2016; Ronen et al., 2017). In these latter examples, the vibrations induced by the sound source are coupled to the skin only by the surrounding media such as water, air, and ultrasound gel (Chordekar et al., 2015; Geal-Dor et al., 2015; Perez et al., 2016; Ronen et al., 2017; Shupak et al., 2005), without direct physical contact between the sound source and the skin (i.e., 0 N application force, and not the traditional 5 N force), and over a larger skin area than that during stimulation by the clinical bone vibrator. In general, hearing is then elicited when audio frequency vibrations are induced in soft tissues of the body, irrespective of the way these vibrations are initiated, either by direct

stimulation to the skin (e.g., by a clinical bone vibrator), from vibrations in the surrounding media (in the presence of hearing protection devices), for example, water—as in underwater hearing or air—as in free field hearing, or even intrinsic body sounds such as pulsatile tinnitus induced by vibrations of the heart and major arteries (De Ridder, Vanneste, & Menovsky, 2013) and one's own vocalizations induced by vibrations of the vocal cords and additional sound producing tissues. Since hearing had been elicited in each of these situations, it is obvious that the ear had been activated. Therefore, in each of these examples of STC, vibrations had apparently been induced in the tissues, which were transmitted along a path of least resistance (i.e., with minimal differences in acoustic impedance between the various tissues along the path) through a series of soft tissues, reach the ear and elicit auditory sensation (see Figure 1). In fact, the transmission of audio frequency vibrations (between 0.25 to 20 kHz) initiated by a bone vibrator applied to the wrist through the soft tissues of the body has been demonstrated directly by detecting the tissue vibrations with an accelerometer placed at various sites on the body, over a distance of up to 30 cm (Zhang et al., 2017).

The mechanism of the final stage of STC hearing, enabling the tissue vibrations initiated in response to STC stimulation to activate the ear, is not immediately apparent. In most of these experimental examples of STC, threshold (the routine, quantitative, method for hearing assessment) was determined under conditions in which AC sounds had been ruled out, for example, by earplugs and other hearing protection devices. The conclusions and implications (see later section) of this review reflect the tissue vibrations induced by low stimulus intensities (threshold). Other factors may be involved when the tissue vibrations that are induced are higher in magnitude.

In studies in which STC responses were assessed as a function of stimulus frequency, it was found that the thresholds to higher frequency stimuli delivered to the eye were elevated 10 to 15 dB compared with the lower frequencies (Ito et al., 2011; Watanabe et al., 2008). Furthermore, it has been shown in cadavers that higher frequency stimuli delivered to soft tissue were more attenuated than those to lower frequencies (Roosli et al., 2016). In addition, thresholds to higher frequency stimuli delivered to the neck and to fluid applied to the external auditory meatus were elevated 15 dB compared with the lower frequencies. These results provide evidence that the STC pathway may be less efficient at the higher frequencies.

STC or BC?

The possibility that the soft tissue vibrations could lead to hearing by inducing actual vibrations of skull bone (i.e., BC-related mechanisms), leading to opposite phase movements of the oval and round windows, has been studied. Such displacements of the two windows would cause a pressure difference across the basilar membrane and the initiation of a passive traveling wave along the basilar membrane. However, there is a large difference between the acoustic impedances (defined as the product of the density of the medium and the velocity of sound in that medium) of the media and tissues involved: bone (7.8×10^6 kg/m^{2}sec); water and gel (1.5×10^6 kg/m^{2}sec); typical soft tissues (1.6×10^6 kg/m^{2}sec); air (0.0004×10^6 kg/m^{2}sec; Baun, 2004; Blakley & Siddique, 1999; Wever & Lawrence, 1954). In the presence of a large difference in acoustic impedance between two media (referred to as a mismatch), a major part of the vibrations is not transmitted, but rather reflected (i.e., attenuated) at the boundary between the two media. For example, in view of the large acoustic impedance mismatch between air and skull bone (70 dB attenuation), it is highly unlikely that vibrations in free field air below 60 dB HL in intensity could induce vibrations of skull bone directly, leading to hearing by BC mechanisms. On the other hand, in such conditions, and in the presence of hearing protection devices, hearing may then be elicited by air, inducing vibrations in the skin (soft tissue) exposed to the sound field (the air-soft tissue mismatch is smaller than that between air and bone; Chordekar et al., 2016).

Finally, the nature of the excitatory events taking place at the boundary between the soft tissues and skull bone must be considered. Given the acoustic impedance mismatch between soft tissue and bone, one would theoretically expect that about 70% of the vibratory energy would be reflected (equivalent to 7 dB attenuation) and not transmitted at the boundary between soft tissue and bone. However, in experimental studies in which the actual attenuation (damping) at the interface between soft tissue and bone was assessed quantitatively, the damping was 10 to 28 dB, depending on frequency (Hakansson, Tjellstrom, & Rosenhall, 1985; Tjellstrom et al., 1980). Therefore, it seems that the acoustic impedance mismatch between soft tissue and bone (about 7 dB) is probably only one of the mechanisms of the overall attenuation (10 to 28 dB). In other words, it is possible that the vibrations induced in the skin and soft tissues of the neck, body, and thorax during soft tissue stimulation at low stimulus levels may not have been able to induce vibrations of the much more compact and dense skull bone because of the acoustic impedance mismatch and additional damping. Nevertheless, hearing was elicited. Therefore, the hearing induced by STC stimulation at threshold may not involve vibration of skull bone (i.e., BC-related mechanisms), and alternative modes have to be considered. Furthermore, following the induction of several

experimental manipulations (immobilization of the ossicular chain, discontinuity of the chain, fixation of the cochlear windows), unchanged BC thresholds were seen. In addition, in these same animals (ossicular chain manipulations and window fixation), auditory responses could be elicited following the delivery of vibratory stimulation to a pool of saline in the surgical area (Perez, Adelman, & Sohmer, 2011), and to the CSF and to fluid (saline) applied to the middle ear cavity (Perez, Adelman, & Sohmer, 2015). These experimental manipulations would hinder the initiation of a passive traveling wave as a result of the severe impediments to the BC mechanisms of ossicular chain inertia and to the changes induced in the load impedance of the cochlear windows to the inner ear BC mechanisms (compression of the cochlea and fluid inertia). These results confirm that hearing can be elicited by inducing vibrations in fluids and that the resulting hearing may not involve BC mechanisms. Even in patients (Ronen et al., 2017) and animal models (Perez et al., 2016) of postradical mastoidectomy (lacking an ossicular chain), hearing was elicited in response to a form of STC (inducing vibrations in fluid applied to the mastoidectomy cavity). In addition, it has been reported that a form of STC (stimulation to the dura in cadavers) had a minor effect on vibrations of the promontorium (Sim et al., 2016).

The Final Stage: Inner Ear Excitation

Following the initiation of vibrations in the soft tissues at each of the many possible stimulation sites and conditions on the body (head, neck, thorax) by extrinsic sources (e.g., underwater sound and free field sound in the presence of hearing protection devices; a bone vibrator applied to the skin) or by intrinsic sounds (e.g., cardiovascular origin; vocalizations), the vibrations are likely transmitted along a series of soft tissues having similar acoustic impedances via multiple parallel soft tissue and fluid channels (see Figure 1). The tissue vibrations presumably reach the ear and elicit hearing.

The resulting hearing occurs in the apparent absence of traditional AC and osseous BC mechanisms, both of which have been thought to lead to a passive traveling wave in the cochlea. The occlusion effect (pressure variations in the air in the occluded external canal induced by the vibrations of the soft tissue, resulting from the STC stimulation; the pressure variations could induce displacements of the tympanic membrane, as in AC) may have contributed to the hearing in those examples

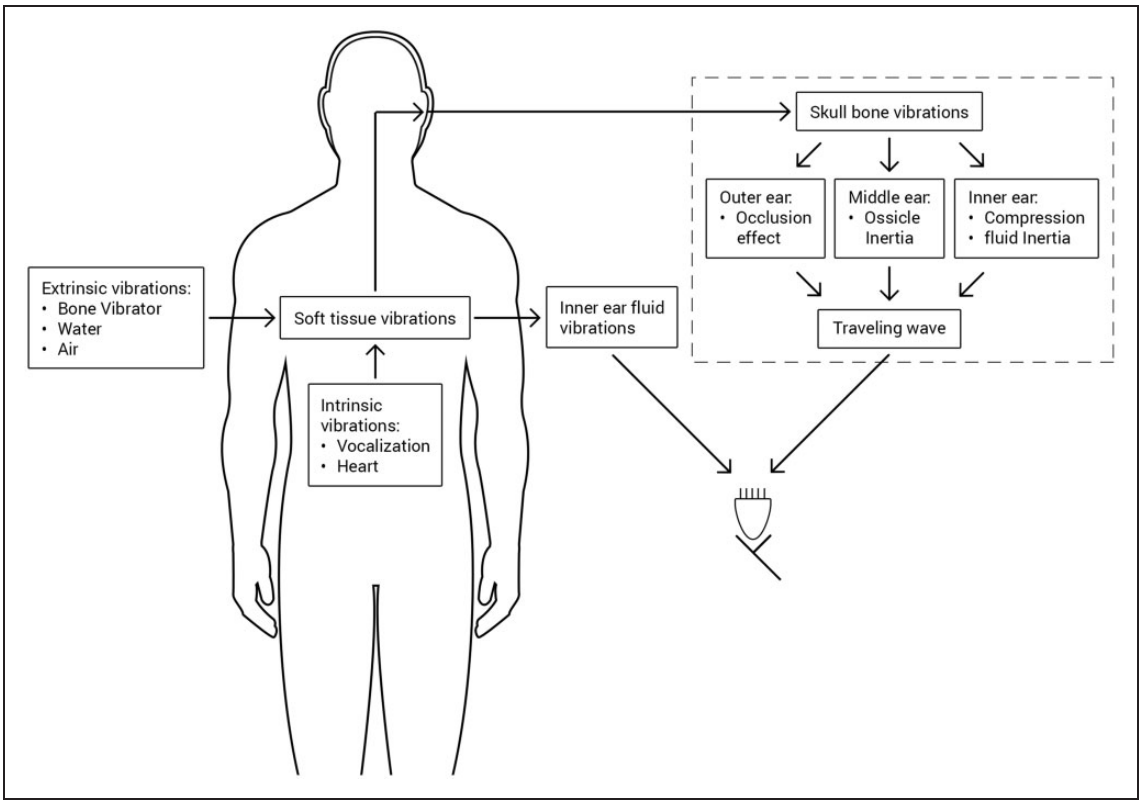

Figure 1. Diagram outlining the suggested pathway of vibrations during STC hearing, beginning with their initiation in the soft tissues of the body, their transmission through the tissues, and culminating in the final stage—end point in the ear, leading to hearing.

of possible STC in which the external canal was occluded with standard earplugs. However, the occlusion effect was not involved in the studies in which customized, deeply inserted earplugs were used (Chordekar et al., 2016), or the ear canal had been filled with fluid (Ronen et al., 2017), or in experiments in which the middle ear conducting mechanisms had been impeded (Perez et al., 2011, 2015). Alternatively, the soft tissue vibrations may give rise to inner ear fluid pressures that activate the outer hair cell cochlear amplifier directly, which, in turn, excite the inner hair cells and auditory nerve, without the involvement of a passive basilar membrane traveling wave. Since the soft tissues and fluids have similar acoustic impedances, the vibrations of the soft tissues may induce vibrations of the inner ear fluids, perhaps via fluid channels similar to those which allow large molecules applied to the middle ear cavity to reach perilymph (Mikulec, Plontke, Hartsock, & Salt, 2009), or through fluid channels communicating between the CSF in the cranial cavity and the inner ear (e.g., aqueducts and perineural and perivascular channels). The round window itself may serve as such a channel. This direct excitation of the inner ear hair cells by the fluid pressures is somewhat similar to the situation in frogs and lizards (Manley & Köppl, 2008; Smotherman & Narins, 1999), and even in humans, where AC and BC auditory stimuli activate the vestibular hair cells in the absence of a basilar membrane in the vestibular end organs and elicit the vestibular evoked myogenic potential (Sohmer, 2006). A dehiscence in the superior semicircular may facilitate such communication between CSF and inner ear fluids (Brantberg et al., 2016).

On the other hand, there are studies providing evidence that the delivery of more intense STC vibratory stimuli may nevertheless induce vibrations of skull bone, leading to hearing. For example, it has been shown that soft tissue stimulation led to vibrations of promontory bone in cadaver heads (Roosli et al., 2016). The enhanced stimulus intensities likely overcome attenuating mechanisms such as the impedance mismatch (Roosli et al., 2016).

Moreover, there may be differences in the mechanisms of STC, depending on the exact site, intensity and area of STC stimulation, and the nature of the coupling media and application forces. For example, it is possible that with low-intensity stimulation in response to which the magnitude of the induced tissue vibrations is small, the final stage of STC activation of the inner ear may involve direct fluid pressure activation of the hair cells. On the other hand, with higher intensity stimulation when the tissue vibrations are greater in magnitude, they may be able to overcome any damping (e.g., acoustic impedance mismatch) and initiate transition to vibrations of skull bone, leading to osseous BC mechanisms and a passive traveling wave, as the final stage (end point) of STC hearing (see Figure 1).

This is reminiscent of the long-standing controversy as to whether the cochlea is activated by the fast fluid pressure waves or by the slow mechanical traveling waves (Cooper & Rhode, 1996; Olson, 2013a, 2013b). The present review has provided evidence for the possibility that when low-level vibratory stimulation is delivered to soft tissue sites, the final stage (end point) may involve inner ear fluid pressures that propagate rapidly through the fluid and directly activate the hair cells. For example, it has been shown that motility of isolated outer hair cells, which serve as the mechanical basis for the cochlear amplifier, can be elicited by fluid pressures delivered to the surrounding fluid (Brundin, Flock, & Canlon, 1989). In addition, support for the activation of the cochlear amplifier without the involvement of passive displacements of the basilar membrane comes from studies designed to estimate the magnitudes of the passive and active (cochlear amplifier) components of basilar membrane displacements. The magnitude of the basilar membrane traveling wave displacements in the live animal at threshold (presumably equal to the sum of the passive and active components) is about 1 nm (Oghalai, 2004), while shortly postmortem (leading to loss of the cochlear amplifier active component resulting from cessation of metabolism) it is so small, that stimulus intensity has to be elevated by 60 to 80 dB to once again achieve a displacement of 1 nm (Robles & Ruggero, 2001; Ruggero, Rich, Recio, Narayan, & Robles, 1997). This result is interpreted as evidence that in the live animal at threshold, the passive displacement of the basilar membrane was 60 to 80 dB smaller than 1 nm, and the overwhelmingly major component of basilar membrane displacement in the live animal was the active (cochlear amplifier) component. Therefore, the magnitude of the passive displacement of the basilar membrane traveling wave at threshold was of the order of 0.001 to 0.0001 nm; too small to initiate a traveling wave and thereby activate the cochlear amplifier. In such a situation at threshold, the cochlear amplifier (outer hair cells) may be activated directly by the fluid pressures. As shown by Brundin et al. (1989), such direct activation by fluid pressures of the outer hair cells in the absence of the basilar membrane is possible.

At higher intensity stimulation, it is possible that there may be a transition to activation by the slow mechanical traveling wave propagating along the basilar membrane. Further study is required to determine the exact mechanism of the final stage of STC hearing (see Figure 1).

Implications

In our daily lives, most hearing is by AC so that STC is not the chief mode of hearing. However, STC and its mechanisms as presented here, may explain several low level auditory phenomena. In addition to the

forms of STC discussed earlier (e.g., hearing under water, in free field air in the presence of hearing protection devices), STC may be responsible for the following auditory phenomena:

1. The scratch test, in which following tympanomastoid surgery, the patients are asked whether they hear the gentle scratching of their head bandage. Hearing is then a sign that the inner ear was not damaged by the surgery (Iacovidou, Giblett, Doshi, & Jindal, 2014). It is feasible that the scratching of the bandage delivered STC stimulation to the skin.
2. The liquid test, the hearing in response to the delivery of vibratory stimuli to liquid applied to the cavum conchae and external meatus (Tabuchi et al., 2005). We suggest that the stimulation to the fluid delivered STC stimulation to the skin.
3. The cartilage hearing aid—hearing vibratory stimuli applied to the skin over cartilage of the external ear (Shimokura, Hosoi, Nishimura, Yamanaka, & Levitt, 2014).
4. In some patients in whom a transcutaneous BC device had been implanted, the floating mass transducer was placed in contact with the dura and compressed the dura or the sigmoid sinus 2 to 5 mm. It has been suggested that in these patients, the additional transmission of vibratory energy to the CSF and blood of the sinus (i.e., STC) may be beneficial (Vyskocil et al., 2016).

Furthermore, it is possible that STC may also explain several auditory phenomena that had previously (before STC had been described and understood) been ascribed as being the result of BC stimulation:

1. The fetus in utero after 18 to 20 weeks gestation hears maternal sounds (Gerhardt et al., 1996; Sohmer & Freeman, 2001). Although suggested that this is by BC mechanisms, the fetal skull bones are then soft and are separated from each other by membranous spaces (sutures) and are not continuous (Opperman, 2000). Therefore, the initiation of vibrations of skull bones by amniotic fluid, which are transmitted to the temporal-petrous bone (a prerequisite for BC mechanisms), is not likely. The vibrations of the amniotic fluid may excite the inner ear by an STC mechanism.
2. It has been suggested that a speaker hears his own voice both by AC and by BC (Stenfelt & Goode, 2005), the latter resulting from the vibrations of the speech producing organs and the air in the oral cavity. However, since these are soft tissues and air, with acoustic impedances very different from that of bone, it is not likely that their vibrations could induce vibrations of skull bone, and STC may be more likely.
3. Finally, in pulsatile tinnitus (De Ridder et al., 2013), the soft tissues of the thorax and neck provide the connection between the heart and major arteries and the ear.

Therefore, while AC hearing is dominant in our daily lives, hearing involving STC is probably more prevalent than previously thought. Considering the mismatch between the acoustic impedance of the various media involved in each of these auditory phenomena and that of bone, it is likely that several low-level auditory phenomena hitherto thought to be elicited by BC may involve vibrations that are transmitted through soft tissues (STC) to the ear.

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