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Research Paper

Does hearing in response to soft-tissue stimulation involve skull vibrations? A within-subject comparison between skull vibration magnitudes and hearing thresholds

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ABSTRACT

Hearing can be elicited in response to bone as well as soft-tissue stimulation. However, the underlying mechanism of soft-tissue stimulation is under debate. It has been hypothesized that if skull vibrations were the underlying mechanism of hearing in response to soft-tissue stimulation, then skull vibrations would be associated with hearing thresholds. However, if skull vibrations were not associated with hearing thresholds, an alternative mechanism is involved. In the present study, both skull vibrations and hearing thresholds were assessed in the same participants in response to bone (mastoid) and soft-tissue (neck) stimulation. The experimental group included five hearing-impaired adults in whom a bone-anchored hearing aid was implanted due to conductive or mixed hearing loss. Because the implant is exposed above the skin and has become an integral part of the temporal bone, vibration of the implant represented skull vibrations. To ensure that middle-ear pathologies of the experimental group did not affect overall results, hearing thresholds were also obtained in 10 participants with normal hearing in response to stimulation at the same sites. We found that the magnitude of the bone vibrations initiated by the stimulation at the two sites (neck and mastoid) detected by the laser Doppler vibrometer on the bone-anchored implant were linearly related to stimulus intensity. It was therefore possible to extrapolate the vibration magnitudes at low-intensity stimulation, where poor signal-to-noise ratio limited actual recordings. It was found that the vibration magnitude differences (between soft-tissue and bone stimulation) were not different than the hearing threshold differences at the tested frequencies. Results of the present study suggest that bone vibration magnitude differences can adequately explain hearing threshold differences and are likely to be responsible for the hearing sensation. Thus, the present results support the idea that bone and soft-tissue conduction could share the same underlying mechanism, namely the induction of bone vibrations. Studies with the present methodology should be continued in future work in order to obtain further insight into the underlying mechanism of activation of the hearing system.

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1. Introduction

Traditionally, air conduction (AC) and bone conduction (BC) are described as the two major paths of sound transmission to the inner ear. Recently, however, studies in humans and experimental animals demonstrated that hearing can also be elicited in response

Abbreviations: STC, Soft-tissue conduction; BAI, Bone-anchored implant

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to soft-tissue stimulation. The results support the existence of a complementary pathway to AC and BC, namely soft-tissue conduction (STC) (Sohmer, 2015), also known as nonosseous BC (Vento and Durrant, 2009). For example, STC hearing can be elicited by applying a clinical bone vibrator with a 5 Newton (N) application force to many skin and soft-tissue sites on the neck and thorax (Kaufmann et al., 2012) and even when the stimuli is applied to the ankle (Brantberg et al., 2016). While there is a general agreement that hearing can be elicited in response to soft-tissue stimulation, its underlying mechanism is inconclusive. Some researchers suggest that STC does not require actual skull bone vibrations (and thus is different from BC) (Sohmer, 2015) or that additional mechanisms are involved besides bone vibrations (Ito et al., 2011; Watanabe et al., 2008). Others suggest that STC results from the initiation of skull bone vibrations and therefore is similar in its mechanism to BC (thus reflecting BC hearing) (Rosowski, 2009; Roosli et al., 2016). To date, the experimental findings from human models (dry skulls, cadaver heads) and animal models are equivocal. One way to settle this ongoing debate is to measure both behavioral thresholds and direct skull bone vibrations in response to BC and STC stimulation in the same participants. The use of the bone-anchored implant (BAI) for auditory rehabilitation of conductive or mixed hearing loss offers a unique opportunity to measure both skull vibrations and behavioral thresholds in the same living individuals (see Appendix A for a description of the BAI). Skull vibration measurements are possible because one side of the BAI is osseointegrated in the skull (Tjellström and Håkansson, 1995), while the other side is visually accessible above the skin for optical measurements (Majdalawieh et al., 2006).

The suggestion that STC leads to hearing sensation without the initiation of skull vibrations was related to the acoustic impedance mismatch of soft-tissue and bone (Sohmer, 2015, 2017). Studies that evaluated the influence of skin imposed between the vibration transducer and skull found attenuation of 10–25 dB, depending on the frequency (Tjellström et al., 1980; Håkansson et al., 1984; Stenfelt and Goode, 2005). While it is possible that bone vibrations are involved at high-intensity levels of stimulation, it was suggested that at threshold levels, soft-tissue stimulation could initiate hearing via an alternative pathway. Such a pathway from the site of stimulation to the inner ear may include fluids and soft-tissue channels with similar acoustic impedance (Sohmer, 2017).

An alternative explanation for STC is that soft-tissue stimulation eventually leads to vibrations of skull bones, as in BC. The phenomenon of BC is complex and occurs when a vibratory stimulus is applied to sites on the head overlying skull bones, usually the mastoid or forehead. The stimulus initiates skull vibrations, which are thought to propagate over bone to the bony walls of the outer, middle, and inner ears, leading to the initiation of several mechanisms: (1) compression-distortion of the cochlear shell, (2) inertia of the inner ear fluids, (3) inertia of the middle ear ossicles, and (4) sound radiation into the external auditory meatus (Stenfelt and Goode, 2005; Stenfelt, 2011). These mechanisms lead to a mechanical wave along the basilar membrane in the inner ear (traveling wave), which activates the outer hair cells and eventually leads to hearing sensation (Stenfelt, 2011). The hypothesis that a BC mechanism underlies STC is supported by a study that showed soft-tissue stimulation led to promontory vibrations in cadaver heads (Roosli et al., 2016).

To date, measuring skull bone vibrations directly in live individuals was difficult because bones are covered by skin and other soft-tissues that dampen the vibrations reaching the bone (Eeg-Olofsson et al., 2013). Therefore, most studies measured skull vibrations by using models of the human head such as animals, dry

human skulls, and cadaver heads. The use of such models was limited because mechanical vibrations were measured without the assessment of behavioral (psychoacoustic) measurements of audition, such as threshold. The exception is a study that used an accelerometer that was placed between the teeth (not direct skull vibration measurements) and measured both hearing thresholds and skull vibrations (in response to BC and STC stimuli) (Ito et al., 2011). It is questionable, however, how well the teeth reflect skull or inner-ear vibrations (Reinfeldt et al., 2013; Sim et al., 2016). In another study, recordings in patients with common cavities of the outer and middle ears allowed the measurement of inner-ear vibrations using a laser-Doppler vibrometer (LDV) and their correlation with hearing thresholds (Eeg-Olofsson et al., 2013). This study, however, did not assess thresholds and bone vibrations in response to soft-tissue stimulation. A more recent study that compared hearing-threshold differences (in response to stimulation at different sites compared to the mastoid) of humans to bone vibration differences in cadaver heads found that these measurements (threshold and vibration difference) were comparable (Dobrev et al., 2016).

A unique opportunity to overcome this limitation is by measuring skull vibrations in hearing-impaired individuals in whom a percutaneous BAI has been surgically implanted as a mean of auditory rehabilitation due to hearing loss. Because one side of the titanium implant is osseointegrated in the mastoid or the parietal skull bones (Eeg-olofsson et al., 2008; Mudry and Tjellström, 2011; Tjellström and Håkansson, 1995) while the other side is visually accessible above the skin, it is possible to directly assess vibrations of the skull by measuring the vibrations of the implant. Therefore, the vibrations of the implant are assumed to approximate the motion of the skull. It should be noted that BAI vibrations have been previously measured successfully by an accelerometer (Håkansson et al., 1996) and LDV (Majdalawieh et al., 2006) in response to BC stimuli. No previous study however, has used BAI to measure skull vibrations in response to soft-tissue stimulation.

The present study was designed to obtain further insight into the underlying mechanism of hearing via STC in living humans in order to ascertain whether sound stimulation via STC involves actual skull bone vibrations (osseous BC) or an alternative non-osseous pathway. For the purposes of this study, hearing thresholds and skull vibrations were measured in participants with BAI in response to stimuli on bone (mastoid) and soft-tissue (neck) sites and then compared. The rationale for the study was that if the differences between the thresholds at the two sites were equal to the differences in the magnitude of vibrations in response to stimulation to these two sites, then it is likely that there is a causal relationship between them, suggesting that the vibrations induced the auditory sensations. If however, the threshold difference between stimulation at both sites is not the same as the difference in skull vibration magnitude following stimulation at these same two sites, an alternative nonosseous mechanism could be involved.

2. Materials and methods

2.1. Participants

Two groups participated in the present study. The BAI group included five postlingual hearing-impaired adults, two males and three females, mean age 56.2 ± 10.8 years, implanted with BAI (unilaterally) due to acquired conductive or mixed hearing loss. The unaided AC pure tone average (PTA) threshold (at 500, 1000, and 2000 Hz) of the ear on the same side as the implant was 67.3 ± 12 dB HL, for these participants, and the BC PTA was

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