Effects of hematoma-induced changes in auricular thickness on the propagation components of cartilage conduction

Akane Tamura[1†‡], Sho Otsuka[2,3], and Seiji Nakagawa[2,3,4*] ([1]Dept. of Medical Eng., Grad. School of Sci. & Eng., Chiba Univ.; [2]Ctr. for Frontier Medical Eng., Chiba Univ.; [3]Grad. School of Eng., Chiba Univ.; [4]Med-Tech Link Ctr., Chiba Univ. Hospital)

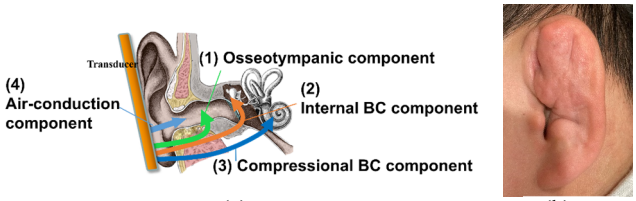
1. Introduction

Bone conduction (BC) is a method of perceiving sound through biological tissues, such as the skin, bone, and muscle, by presenting a transducer against the skull. The sound is transmitted via 4 components[1)]: (1) the osseotympanic component, which involves sound radiated into the ear canal; (2) the inertial BC component, based on the relative motion between the middle ear ossicles and temporal bone; (3) the compressional BC component, resulting from the compression and expansion of the cochlear shell; and (4) the air-conduction component, which radiates from the transducer into the air and enters the ear canal. Since some components, such as (2) and (3), directly reach the middle or inner ear, BC has been employed in hearing aids for conductive hearing loss. However, the conventional BC requires the transducer to be pressed strongly against the skull, causing discomfort with prolonged wearing.

As a solution to this problem, cartilage conduction (CC) has been proposed, in which the transducer is placed on the auricular cartilage[2)]. The auricular cartilage is a soft and elastic tissue that is less likely to cause pain. The CC sound is transmitted as the same 4 components as BC (**Fig. 1(a)**). CC has been applied in hearing aids for ear canal atresia[3)] and smartphone screens[4)].

CC is also effective in diseases such as chondrodysplasia and auricular hematoma, where the auricular morphology is altered, making the use of conventional earphones or hearing aids difficult. However, it has been reported that hearing of CC is affected by individual auricular characteristics, such as size and hardness[5)]. The auricular hematoma is characterized by swelling and deformation of the auricle caused by internal bleeding due to pressure/friction stimulation and fibrosis of the accumulated blood[6)] (**Fig. 1(b)**). Therefore, while CC devices are effective for the auricular hematoma, changes in morphology and hardness can affect CC hearing.

We previously reported that the hearing threshold of CC in the auricles with hematoma was increased in the low-frequency range and decreased in the high-frequency range compared to normal-morphology auricles[5)]. However, there were large individual differences in the extent of auricular hematoma and onset site, and these differences are likely to cause differences in CC propagation. In this study, we classified auricular hematomas based on location and thickness and examined in detail how these factors affect CC propagation components.



(a) (b)

Fig. 1(a) Four propagation components of cartilage conduction. (b) Auricular hematoma in Judo athletes.

2. Experiments

This study involved 10 subjects with auricular hematoma (Males, 18-59 years), referred to as "Hematoma." Each subject had at least one affected auricle, and 18 affected auricles were tested in a total. In addition, 15 participants with normal auricle morphology (10 males and 5 females, 21-28 years) comprised "Normal" group and 30 ears were represented in a total.

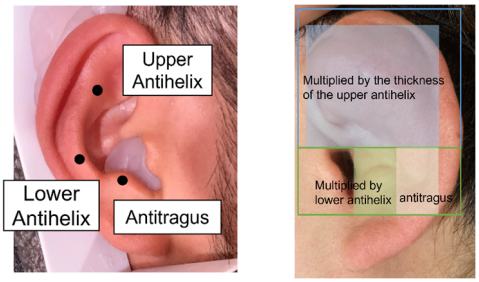
A sound transducer consisted of a piezo-electric device (R-11-244018-01, TOKIN) placed on a flat acrylic plate with 50 mm × 50 mm dimensions. The plate was positioned to cover the entire pinna.

The ear canal sound pressure (ECSP) was measured using a probe tube microphone (ER-7C, Etymotic Research). Tone bursts at 250-8000 Hz with 10-s duration were used as stimuli. The input voltage to the transducer was set to 0.54 Vpp.

The thickness of the upper antihelix, lower antihelix, and antitragus were measured (**Fig. 2(a)**).

The auricular hematomas were analyzed in four ways, including auricular thickness and volume: (A) the thickness of the upper antihelix, (B) the thickness of the lower antihelix, (C) the thickness of the antitragus, (D) the hematoma volume. For (D), the hematoma volume was estimated based on the size and thickness of the auricle (**Fig. 2(b)**). The area of the auricle occupied by the hematoma was estimated and simply multiplied by the measured thickness to obtain the volume. The upper part of the auricle (blue area in **Fig. 2(b)**) was multiplied by the thickness of the upper antihelix, half of the lower part (green area in **Fig. 2(b)**) by the thickness of the lower antihelix, and the other half by the thickness of the

E-mail: †akane_tamura@chiba-u.jp, *s-nakagawa@chiba-u.jp



(a) (b)

Fig. 2(a) Measurement points of thickness. (b) (D): Hematoma volume. The solid line shows the actual measured size of the pinna, and the area of the hematoma (filled area) was estimated. The volume was estimated by multiplying the thickness of the pinna.

antitragus. All classifications (A-D) were divided into tertiles (Class 1-3).

3. Results

Analysis of variance showed significant main effects on the ECSP for all the thickness measurement points (A-C) ($p<0.05$). In the Hematoma group, significant main effects were observed only for (A) and (D) ($p<0.001$).

Fig. 3 shows the ECSP classified by (A). At 250-1000 Hz, the ECSP decreased as the degree of hematoma increased, and significant differences were confirmed between Normal and Class 3 ($p<0.05$). For (D), the ECSP similarly decreased with increasing class at 250-1000 Hz, with significant differences observed between Normal and Class 3 at 250 and 1000 Hz ($p<0.05$).

On the other hand, for (B), the ECSP of Class 2 was lower than Class 3(**Fig. 4**). For (C), both Class 2 and 3 showed similar decreases.

4. Discussion

A decrease in the ECSP indicates reduction in the (1) osseotympanic and (4) air-conduction components. In a previous study, significant decreases in thresholds were observed in the Hematoma group at 125-1000 Hz[5]. The significant difference in thresholds is likely due to the reductions in the sum of the (1) osseotympanic and (4) air-conduction components in subjects with a thick upper antihelix or large volume of hematoma (Class 3 of (A) and (D)).

Conversely, in (B) and (C), Class 2 and 3 showed similar or reversed. For (B) and (C), which are closer to the ear canal, the hypothesis that greater the swelling would block the ear canal entrance and decrease the air-conduction component was not supported. Changes in tissue due to hematoma, in addition to increased thickness, may affect the CC propagation.

While the thickness of the auricle influences CC propagation at any site, this study demonstrates that the thickness of the upper antihelix and

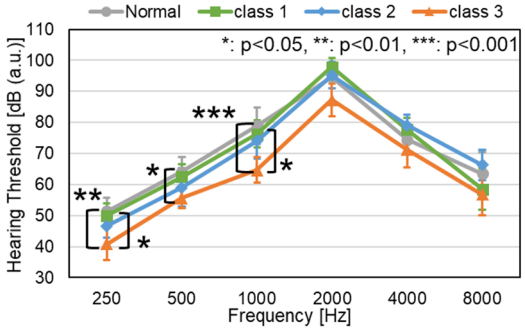


Fig. 3 ECSP classified by the thickness of the upper antihelix (A).

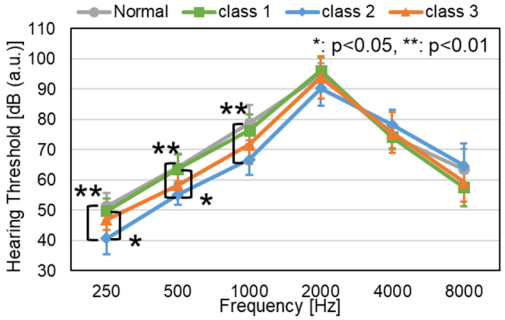


Fig.4 ECSP classified by the thickness of the lower antihelix (B).

hematoma volume are significantly involved in reducing the sum of the (1) osseotympanic and (4) air-conduction components at 250-1000 Hz.

5. Conclusion

Auricular hematomas were classified by severity (thickness) and site, and it was revealed that the greater the thickness of the upper antihelix and the larger hematoma volume, the greater decrease in the air-conduction and osseotympanic components. In contrast, fluctuations in the lower antihelix and antitragus may be more pronounced in moderate cases than in severe cases, depending on frequency.

Acknowledgment

This work was supported by the JSPS KAKENHI Grant Number JP24K03260 and a Research Grant from the Canon Foundation for SN.

References

1) S. Stenfelt, N. Hato, and R. L. Goode, J. Acoust. Soc. Am., **111**, 947, (2002).
2) T. Sakaguchi, Y. Hosoi, Audiol. Jpn., **51**, 375 (2008). [in Japanese]
3) T. Nishimura, H. Hosoi, O. Saito, R. Miyamae, R. Shimokura, T. Matsui, and T. Iwakura, Auris Nasus Larynx, **40**(5), 440 (2013).
4) S. Nakagawa, T. Hotehama, and K. Ito, Proc. Life Eng. Sympo., 431 (2013).
5) A. Tamura, Irwansyah, S. Otsuka, and S. Nakagawa, Annu. Int. Conf. IEEE EMBC, (2023).
6) J. D. Greywoode, E. A. Pribitkin, H. Krein, Facial Plastic Surg., **26**(6), 451 (2010).