

Original Article

The Effect of Degree of Temporal Bone Pneumatization on Sound Transmission of Pulsatile Tinnitus Induced by Sigmoid Sinus Diverticulum and/or Dehiscence: A Clinical and Experimental Study

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BACKGROUND: Although many studies have suggested that air cells may play an important role in sigmoid sinus diverticulum and/or dehiscence (SSDD)-induced pulsatile tinnitus (PT), the exact effects remain unclear. This study aims to quantitatively investigate the effect of different degrees of pneumatization of temporal bone on sound transmission from the sigmoid sinus to the tympanic cavity.

METHODS: In the clinical study, 25 patients were enrolled to measure the sound intensity and frequency of SSDD-induced PT. In the experimental study, sound inputs at different frequencies at the dehiscence sigmoid plate were measured and compared among realistic object models of different degrees of pneumatization, when sound intensity in the tympanic cavity was fixed as the level of the PT sound intensity sensed by patients.

RESULTS: The sound intensity sensed by PT patients was 34.0 ± 13.0 dB SPL, which represented the sound intensity in the tympanic cavity transmitted from the dehiscence sigmoid plate. In the experimental study, when sound received in the tympanic cavity was fixed at 34 dB SPL, the mean inputs of sound intensity at the dehiscence sigmoid plate were 46.9, 46.2, 45.2, 47.1, 57, 57.4, and 74.1 dB SPL in a hypo-pneumatized model; 42.6, 43, 41.5, 43.2, 47.3, 58.2, and 78.8 dB SPL in a moderately pneumatized model; 52.6, 52.8, 48.1, 61, 64.2, 82.4, and >87.3 dB SPL in a well-pneumatized model; and 47.2, 46.2, 45.4, 49.4, 54.9, 66.6, and 77.7 dB SPL in hyper-pneumatized model, with increased sound frequencies (125–8000 Hz). The mean sound transmission distances were 41.7 mm, 45.2 mm, 47.6 mm, and 48.4 mm in successively better pneumatized models.

CONCLUSION: Sound reduces while passing through air cells and attenuation is lowest in the moderately pneumatized temporal bone, followed by the hypo- and hyper-pneumatized temporal bones, and the highest in the well-pneumatized temporal bone. Lower-frequency sound attenuation (≤ 1000 Hz) is less than that of higher-frequency sound (> 1000 Hz).

KEYWORDS: Pulsatile tinnitus, pneumatization, temporal bone, sigmoid sinus, sound transmission, model

INTRODUCTION

Tinnitus is a common condition, constituting a serious public health problem that affects 10–30% of the population worldwide, and can be divided into non-pulsatile and pulsatile subtypes.¹ Approximately 4% of patients have pulsatile tinnitus (PT), defined as a rhythmical noise that is synchronous with the patient's heartbeat in the absence of an external acoustic stimulus.² The psychological impact of tinnitus on many patients is so severe that it can lead to depression or even suicide.³

PT is a multifactorial disease and can be attributed to arterial, venous, and non-vascular causes. Sigmoid sinus diverticulum and/or dehiscence (SSDD) have recently been found to be the common and treatable causes of PT. The prevalence is about 20% in PT

patients.^{4–6} Schoeff described that 33% had SSDD, among PT patients with definable etiologies.⁷ In another study, Dong reported that 86.4% of venous PT is associated with SSDD.⁸

In general, PT sound associated with SSDD originates from the sigmoid sinus.^{5,9–16} According to anatomical structures, the sound from the sigmoid sinus must pass through the pathway of air cells in the temporal bone to propagate into the tympanic cavity, and is ultimately sensed as PT. Therefore, the air cells in the temporal bone would be important contributors to SSDD-induced PT. However, the magnitude of temporal bone pneumatization necessary for triggering PT remains contentious. Some studies suggest that small air cells may constitute an acoustic barrier preventing sound transmission. In contrast, large air cells may increase the resonance of sound by prolongation, amplification, and filtering when sound transmits through the extensively pneumatized temporal bone. Increased transmission of sound caused by blood flow to the cochlea may be experienced as PT.¹¹ However, Duvillard proposed that all kinds of air cells, in temporal bones pneumatized to varying degrees, form air cushions and constitute an acoustic barrier that impedes sound transmission from the sigmoid sinus to the cochlea.¹⁷ However, our previous studies revealed that: (1) although 80% of PT patients with sigmoid sinus diverticulum exhibited a hyper- or well-pneumatized temporal bone, 20% of the patient sample exhibited moderately pneumatized temporal bone;¹² and (2) the degree of temporal bone pneumatization was not significantly different between PT patients with SSDD and healthy controls, when comparing 302 control subjects with 199 unilaterally persistent PT patients with SSDD.¹⁸ Above all, the effects of air cells on PT sound transmission proposed by previous authors are contradictory. Furthermore, these effects of temporal bone air cells on the occurrence of PT induced by SSDD were suggested only on the basis of observation of PT patients' imaging features. The exact underlying effects remain unclear and have not been systematically evaluated.

The current study sought to systematically and quantitatively evaluate the effects of air cells in the temporal bones pneumatized to varying degrees, on sound transmission from the sigmoid sinus to the cochlea, combining applied clinical data and highly realistic object simulation models. Similar to most previous studies, we also hypothesized that extensively pneumatized temporal bone is essential for inducing PT in patients with SSDD, by increasing sound transmission. If this hypothesis is correct, sound from the sigmoid sinus would be expected to be present in tympanic cavity only in the hyper-pneumatized model, and the sound intensity received in the tympanic cavity would be higher than input sound at the dehiscence sigmoid plate.

MAIN POINTS

- Air cells in the temporal bone play an important role in sound transmission in patients with SSDD-induced PT.
- Compared with low-frequency sound (≤ 1000 Hz), high-frequency sound (> 1000 Hz) attenuation is significantly increased while passing through air cells, which supports the observation that SSDD-induced PT sound is generally of low-frequency.
- Sound reduces while passing through air cells, and attenuation is lowest in the moderately pneumatized temporal bone, followed by the hypo- and hyper-pneumatized temporal bones, and the highest in the well-pneumatized temporal bone.

METHODS

Patients

The study was approved by the Ethics Committee of our hospital and written informed consent was obtained from patients who participated in this study. A total of 25 patients with unilaterally persistent PT were enrolled in this study. The PT transiently diminished in response to compression of the ipsilateral cervical vascular structures or rotation of the head toward the affected side in all patients, which suggested venous causes. All patients underwent preoperative dual-phase contrast-enhanced CT to exclude other possible causes of PT besides SSDD, in addition to pure-tone audiometry to test the hearing, and tinnitus matching test to determine the PT sound intensity and frequency sensed by patients. In the study, 9 of 25 patients underwent surgical reconstruction of the dehiscence sigmoid plate, and the details of operation have been reported in our previous study.¹⁹ In brief, the mastoid was opened and the dehiscence region of the sigmoid plate was exposed and skeletonized. The temporalis fascia and autologous bone powders were used to resurface the dehiscence area in sequence, and the graft was fixed with a medical adhesive (FAL, Beijing Fuaile Co., Beijing, China).

Development of Highly Realistic Object Simulation Models

Eight temporal cadaveric head specimens were used from our medical university to develop highly realistic object simulation models. None of the tissue donors were from a vulnerable population and all donors provided written informed consent that was freely given. These cadaveric head specimens were divided into hypo-pneumatized, moderately pneumatized, well-pneumatized, and hyper-pneumatized groups according to the degree of temporal bone pneumatization (Figure 1).²⁰ In each group, there were 2 temporal cadaveric head specimens with the same degree of temporal bone pneumatization. A round defect was milled at the superior curve of the sigmoid plate with a 6.5 mm diameter (Figure 2A). An earphone (H210, Edifier, Beijing, China) was tested in the audiometry room before the study to confirm that the sound intensity from both the earplugs of the earphone was the same, and then used to connect the computer and the cadaveric head specimen. One earplug of the earphone was inserted into the defect in the sigmoid plate (Figure 2B). The plug of the earphone was connected to a computer (MS2332, Acer, Taipei, China). Soundwave generator software (SineGen, V2.1) was used to create sound that was transmitted to the defect of the sigmoid plate via the earphone, and input sound frequency was recorded. The external auditory canal was milled to remove any materials blocking the pathway between the external auditory canal and the tympanic cavity, such as the tympanic membrane (Figure 2C). Two noise-measuring instruments (HT-825, Hcjyet, Guangzhou, China) were used (Figure 2D), with the following characteristics: precision ± 1.5 dB SPL, frequency response 31.5–8000 Hz, measuring range of intensity 30–130 dB SPL, and data updation 2 times/sec. The first noise-measuring instrument was used to measure the sound intensity from the other earplug of the earphone, representing the input sound intensity at the dehiscence sigmoid plate. The tip of another noise-measuring instrument was inserted via the external ear canal to measure the sound intensity in the tympanic cavity. The models were embedded in paraffin in order to more accurately measure the input and received sound intensity at the dehiscence sigmoid plate and the tympanic cavity, respectively (Figure 2E).

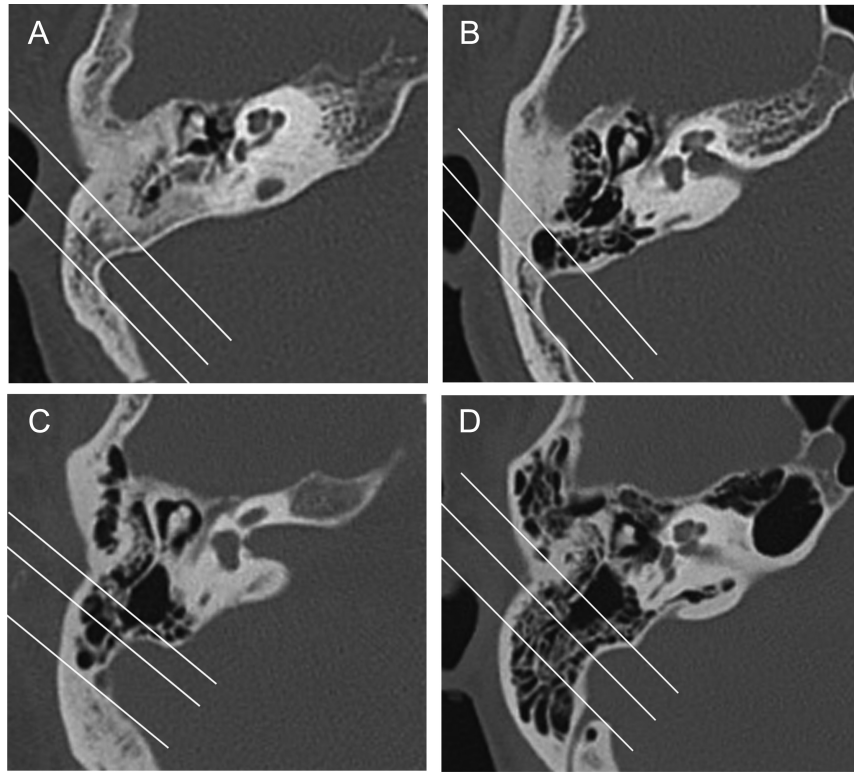


Figure 1. Four types of temporal bone pneumatization. On axial CT image where the malleoincudal complex appeared as an ice cream cone-shaped section, 3 parallel straight lines angled at 45° in the anterolateral direction passed through the most anterior point, the most lateral aspect, and the most posterior point of the sigmoid sinus respectively. (A) Hypo-pneumatized temporal bone and air cells confined to the anteromedial region of the medial line. (B) Moderately pneumatized temporal bone and air cells located between the medial and middle lines. (C) Well-pneumatized temporal bone and the air cells were between the middle and lateral lines. (D) Hyper-pneumatized temporal bone and air cells extended beyond the lateral line.

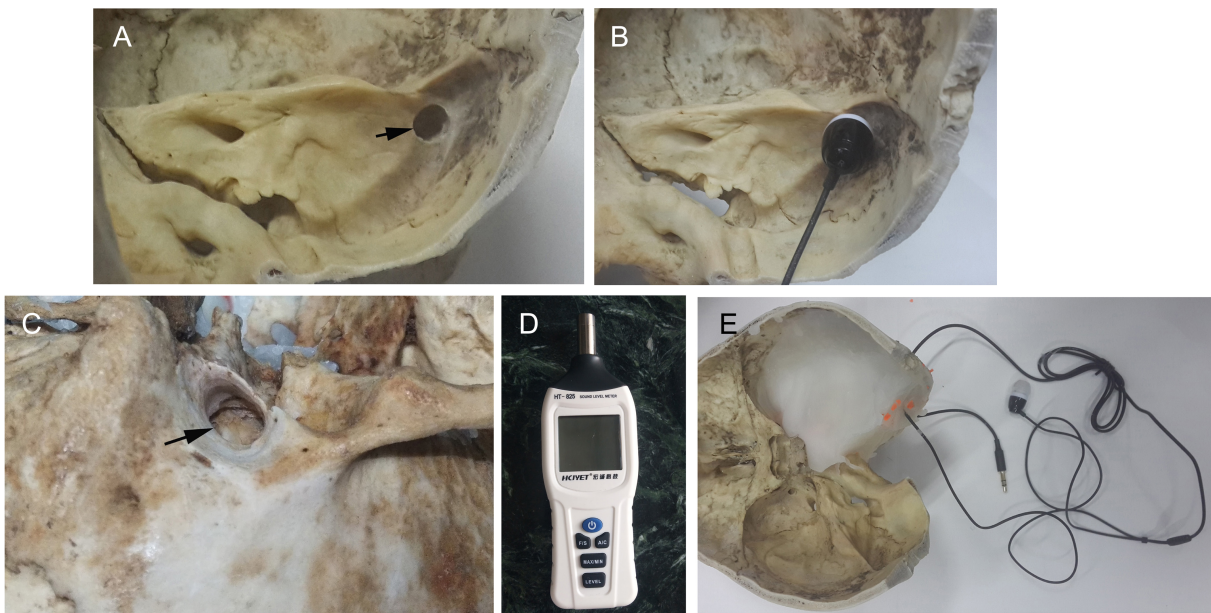


Figure 2. Highly realistic object simulation model. (A) A round 6.5 mm defect in the superior curve of sigmoid plate of a temporal cadaveric head specimen (black arrow). (B) One earplug of the earphone inserted into the defect in the sigmoid plate to input sound. (C) The milled external auditory canal, showing that any materials blocking the pathway between the enlarged external auditory canal and the tympanic cavity were removed (black arrow). (D) A noise-measuring instrument with a long neck and tip to measure sound intensity. (E) shows a highly realistic object simulation model embedded in paraffin.

CT Imaging

All object simulation models underwent HRCT on a 64-slice multi-detector scanner (Brilliance 64, Philips Medical Systems, Best, The Netherlands) to measure the distance of sound transmission from

the dehiscent sigmoid plate to the tympanic cavity. The scanning parameters were as follows: 140 kV and 120 mAs/slice; matrix, 512 × 512; field of view, 18 × 18 cm; detector configuration, 16 × 0.625 mm; gantry rotation time, 0.75 s; and pitch, 0.891. Axial images were

Table 1. Input Sound Intensity in the Dehiscent Sigmoid Plate of All the Highly Realistic Object Simulation Models

Model	Input Sound Intensity (dB SPL)						
	125 (Hz)	250 (Hz)	500 (Hz)	1000 (Hz)	2000 (Hz)	4000 (Hz)	8000 (Hz)
Hypo-pneumatized group							
Model 1	46.1	46.2	45.4	46.5	68.5	55.6	84.2
Model 2	47.7	46.2	45.2	47.6	45.4	59.2	63.9
Mean	46.9	46.2	45.2	47.1	57.0	57.4	74.1
Moderately pneumatized group							
Model 1	40.8	41.0	40.2	41.0	40.8	56.7	74.7
Model 2	44.3	44.9	42.8	45.3	53.7	59.7	82.9
Mean	42.6	43.0	41.5	43.2	47.3	58.2	78.8
Well-pneumatized group							
Model 1	51.3	51.6	45.8	57.9	61.0	81.0	82.9
Model 2	53.9	54.0	50.4	64.1	67.3	83.8	>91.6
Mean	52.6	52.8	48.1	61.0	64.2	82.4	>87.3
Hyper- pneumatized group							
Model 1	44.1	43.4	43.3	46.2	48.1	65.6	79.2
Model 2	50.2	48.9	47.4	52.6	61.6	67.6	76.1
Mean	47.2	46.2	45.4	49.4	54.9	66.6	77.7

reconstructed using bone algorithms with a section thickness of 0.67 mm and a -0.33 mm section interval.

RESULTS

Clinical Characteristics of PT Patients

The sample group of 25 PT patients with SSDD comprised 3 men and 22 women, with a mean age of 41 years (range 26-71 years). Thirteen patients exhibited right-sided PT and 12 exhibited left-sided PT. The duration of PT ranged from 1 month to 26 years. The audiograms showed bilateral normal hearing. The mean intensity of PT noise sensed by patients was 34.0 ± 13.0 dB SPL (range 15-60 dB SPL). The sound frequency was 125 Hz in 14 patients, 250 Hz in 10 patients, and 1000 Hz in only 1 patient. Among the 9 patients who underwent surgical reconstruction of the dehiscent sigmoid plate, PT disappeared

completely in 7 patients, and significantly improved in 2 patients. The other 16 patients refused the operation because they found the PT bearable.

Sound Transmission Through Models of Temporal Bone with Different Degrees of Pneumatization

According to the sound intensity experienced by the PT patients in the above clinical study, the measured sound in the tympanic cavity was fixed at 34.0 dB SPL during the study on object simulation models. The maximum input sound intensity in the dehiscent sigmoid plate generated by the computer was 91.6 dB SPL. Table 1 shows the input sound intensity at the site of the dehiscent sigmoid plate in every model of each pneumatized group. Figure 3 showed the change tendency of input sound intensity with increased sound frequency of the different pneumatized temporal bones.

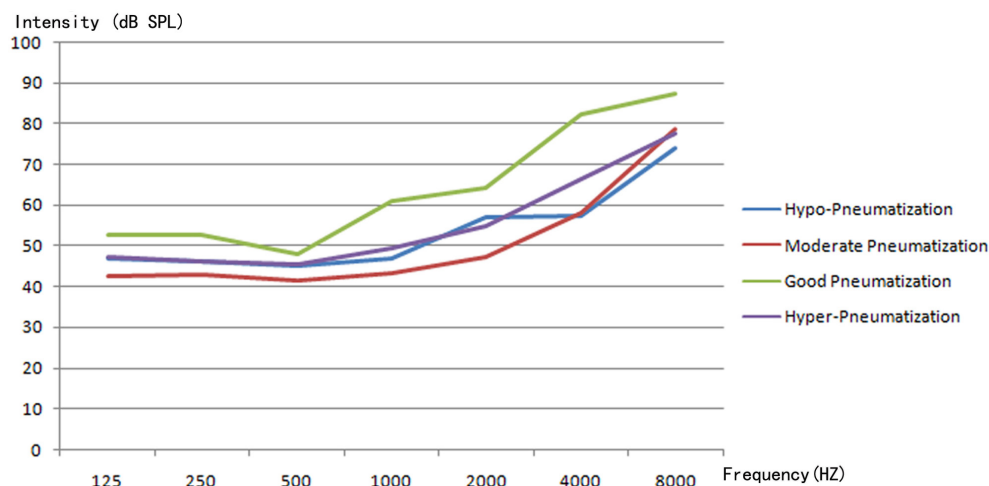


Figure 3. The change tendency of input sound intensity with increased sound frequency, with respect to the 4 different degrees of pneumatization of the temporal bone.

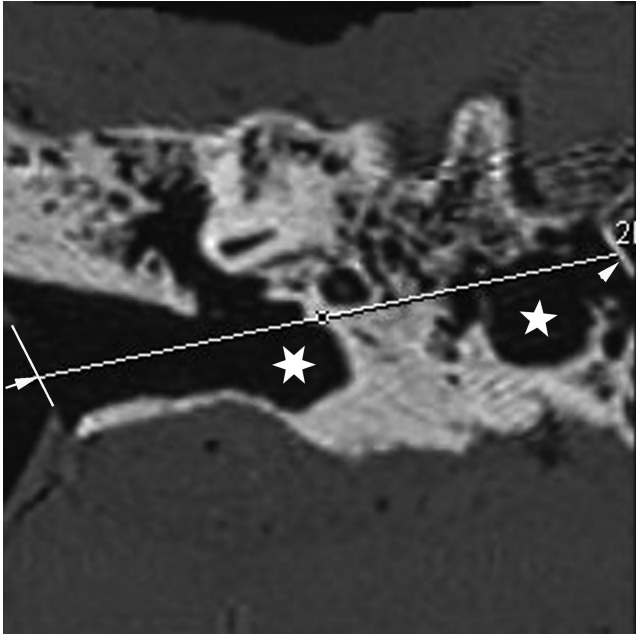


Figure 4. Curve CT image of the PT patient was performed to measure the distance of sound transmission from the dehiscence sigmoid plate (white arrow head) to the tympanic cavity (6-pointed star) passing through the air cells of the temporal bone (5-pointed star).

The sound transmission distance from the earplug of the earphone in the dehiscence sigmoid plate to the noise-measuring instrument in the tympanic cavity was measured on curve-reformed CT images (Figure 4). The mean distance was 41.7 mm in the hypo-pneumatized group, 45.2 mm in the moderate pneumatized group, 47.6 mm in the well-pneumatized group, and 48.4 mm in the hyper-pneumatized group, respectively.

DISCUSSION

Although many studies have suggested that the air cells in the temporal bone play an important role in the occurrence of PT, the proposed effects were different and contradictory. Our findings in the current study differ from previous hypotheses in several ways. First, sound was reduced by air cells, regardless of the degree of pneumatization of the temporal bone. Second, the lowest sound attenuation occurred in the moderately pneumatized temporal bone, followed by the hypo- and hyper-pneumatized temporal bones, and the capitation of sound attenuation was highest in the well-pneumatized temporal bone. Third, compared with higher-frequency sound (>1000 Hz), the sound attenuation of lower-frequency sound (≤ 1000 Hz) was less.

Three factors may impact the changes in sound intensity when it passes through air cells in the temporal bone, which include the distance of sound transmission, magnitude of air cells, and sound frequency. It has been well established that longer propagation routes diminish sound intensity more than shorter routes. Furthermore, increased propagation routes may be associated with more sound reflection on the wall of the air cells, which would also be expected to weaken sound intensity. The distance from the sigmoid plate dehiscence to the tympanic cavity was gradually lengthened, with increased pneumatization of the temporal bone. Thus, attenuation of sound intensity would be greater with increased pneumatization of the temporal bone, if only the sound transmission distance were

considered. However, although sound transmission distance was the shortest in the hypo-pneumatized temporal bone and the longest in the hyper-pneumatized temporal bone, and sound attenuation was similar in both pneumatized models, which suggests that sound attenuation is lower or sound amplification is higher in larger air cells. In another words, the sound was transmitted more easily when the magnitude of air cells was greater, keeping the sound transmission distance the same. Compared with other pneumatized models, the combined action of medium sound transmission distance and size of air cells results in the lowest sound attenuation in the moderately pneumatized temporal bone. In the same way, the coaction of a longer sound transmission distance and larger air cells causes the sound attenuation to be the highest in the well-pneumatized temporal bone. In addition, our study revealed that increased sound frequency was associated with greater sound intensity attenuation, which may be due to diffraction. Low-frequency sound has a longer wavelength and can easily pass barriers in propagation routes through air cells.²¹ The result of this experimental study are consistent with clinical findings, which showed that the most common PT sound frequency in the 25 patients in this study was 125 Hz, followed by 500 Hz. It is also supported by the recent study by Kim who reported that the venous sound is generally of low frequency.²²

In addition to air cells in the temporal bone, blood flow in the sigmoid sinus and sigmoid plate were the 2 other important factors in the induction of PT for patients with SSDD. It is well known that blood flow abnormalities in the sigmoid sinus are the primary factors inducing SSDD-PT.⁹ Many authors have suggested that flow disturbances can induce vibration of the sigmoid sinus wall or noise which is sensed by the cochlea as PT, because blood flow in the sigmoid sinus is normally laminar and silent.^{5,10-13} Liu et al.¹⁵ and Li et al.¹⁴ illustrated flow velocity and regurgitation changes in SSDD-PT patients. In addition, Mu et al.¹⁶ proposed the high pressure of the vessel wall on anomalous areas of the sigmoid sinus wall as one of the causes of SSDD-PT. In recent studies, some authors have found that changes of blood flow in the sigmoid sinus of SSDD-PT patients may be associated with idiopathic intracranial hypertension.²³⁻²⁵ An intact sigmoid plate is thought to operate as an insulator, and may impede the sound from the sigmoid sinus being transmitted to the mastoid air cells. The insulating properties of the sigmoid plate might be destroyed in sigmoid plate dehiscence. The sound from the sigmoid sinus could easily be transmitted to the mastoid air cells through the dehiscence sigmoid plate.^{12,13,26,27}

In order to increase the accuracy and reliability of the current results, we developed highly realistic object simulation models to evaluate the effects of air cells in PT caused by SSDD. Because this study cannot be performed in humans in vivo, we used temporal cadaveric head specimens. Compared with other material, the physical properties of cadaveric head specimens are the most similar to in vivo human samples. In our previous studies of 15 PT cases with sigmoid sinus diverticulum and 23 PT cases with dehiscence sigmoid plate, the average maximum anteroposterior and vertical diameters of sigmoid plate dehiscence were 6.5 mm; 73.9% of patients had only a single defect on sigmoid plate; and approximately 73% of dehiscence sigmoid plates were located at the superior curve.^{12,13} Therefore, we created a round defect with ≤ 6.5 mm diameter at the superior curve of the sigmoid plate, similar to that found in most PT patients with SSDD.

Limitations of the Study

There were 2 major limitations of this study. First, we were not able to evaluate the possible maximum sound intensity caused by abnormal flow in the sigmoid sinus. Second, we could not elucidate the detailed mechanism of different-sized air cells on sound transmission. We plan to conduct future studies to further clarify this issue, using highly realistic computer models.

CONCLUSION

Compared with low-frequency sound (≤ 1000 Hz), high-frequency sound (> 1000 Hz) attenuation is significantly increased when passing through air cells, which suggest that PT sound induced by SSDD is generally low frequency. Although sound intensity attenuation differs with different degrees of pneumatization of the temporal bone, PT sound at the dehiscence sigmoid plate can transmit to tympanic cavity, which suggests that it is not rational to consider the degree of temporal bone pneumatization as one of indicators for etiologic diagnosis of SSDD-induced PT.

Ethics Committee Approval: Ethical committee approval was received from the Ethics Committee of Capital Medical University, Beijing Tongren Hospital (Approval No: V2017003).

Informed Consent: Written informed consent was obtained from all participants who participated in this study.

Peer Review: Externally peer-reviewed.

Author Contributions: Concept – L.Z., L.W.; Design – L.Z.; Supervision – L.Z.; Resource – L.Z., L.W.; Materials – L.W.; Data Collection and/or Processing – H.X., L.W., Z.L.; Analysis and/or Interpretation – L.Z.; Literature Search – L.W., H.X.; Writing – L.Z.; Critical Reviews – L.W.

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Conflict of Interest: The authors have no conflict of interest to declare.

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