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ORIGINAL RESEARCH

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Evidence of air-conduction transmission pathway and strategized transtemporal operative techniques for venous pulsatile tinnitus: Combining water occlusion test and operative sensing applications

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Abstract

Objectives: (1) To establish evidence of the transmission pathway of venous pulsatile tinnitus (PT) associated with sigmoid sinus wall anomalies (SSWAs) and (2) quantify the efficacy of transtemporal surgery.

Methods: This retrospective study included 33 surgical cases of PT associated with SSWAs and 15 controls with venous PT without SSWAs. Quantitative water occlusion test (q-WOT) and imaging data were acquired for preoperative evaluation prior to strategized transtemporal osteovascular reconstruction surgery. A condenser microphone and hydrophone were intraoperatively deployed to assess and monitor in vivo amplitude variations of the PT in eight participants.

Results: A total of 23 (69.6%) participants with SSWA responded to the q-WOT with a median solution volume of 1.3 (1.1/1.6), which significantly differed from that observed in controls ($p < 0.01$). The change in the operative peak amplitude of the acoustic data was statistically significant ($p < 0.01$), from a median of 57.6 (55.5/57.9) dB SPL to 34.3 (33.4/38.8) dB SPL.

Conclusion: Intraoperative application of acoustic sensors revealed that PT associated with SSWAs is predominantly transmitted via the air-conduction pathway. If objective findings such as q-WOT and sensing applications suggest that the transmission of venous PT is involved in middle ear air conduction, the reconstruction technique should be prioritized; if less involvement of middle ear air-conduction is indicated, addressing flow pathologies may be imperative for resolving venous PT.

Level of Evidence: 4

KEYWORDS
pulsatile tinnitus, sensor, sigmoid sinus wall anomalies, ultrasound, water occlusion test

Yue-Lin Hsieh and Xiaobing Xu are considered as joint first authors.

1 | INTRODUCTION

Vascular pulsatile tinnitus (PT) is the abnormal perception of blood flow somatosound.[1-3] Venous PT represents the most common type of vascular PT. Unlike PT with arteriovenous and arterial etiologies, venous PT is categorized by immediate cessation, reduction, or sometimes augmentation of pulse-synchronous vascular sounds during the modulation of bilateral jugular hemodynamics.[4] The sensation of venous PT is closely associated with flow kinetic energy inside the dural venous sinus.[5] In addition to altered intrasinus hemodynamics, various anatomical anomalies adjacent to the hearing apparatus and increased intracranial pressure have been observed in patients with venous PT (see Figure 1 for characteristic radiological findings and corresponding physical examination results).[1,6-9]

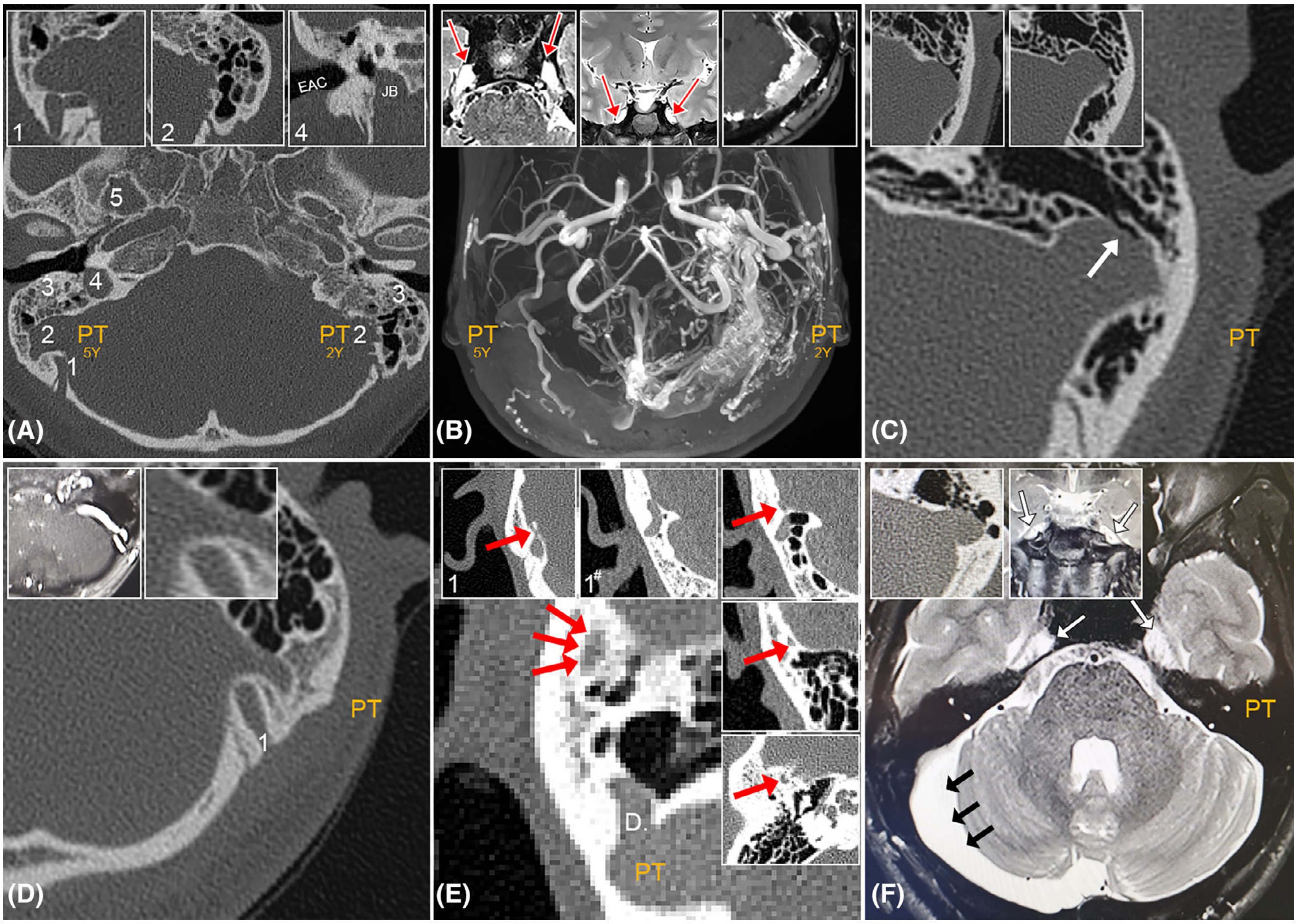

FIGURE 1 Characteristic radiological findings and corresponding physical examinations of venous pulsatile tinnitus (PT). (A) High-resolution temporal bone-computed tomography (CT) and (B) magnetic resonance angiography (MRA) of a patient with bilateral PT caused by severe left-sided arteriovenous fistula (AVF) and increased intracranial pressure (38 cm H_2O). Only right-sided PT disappeared when right ipsilateral internal jugular vein (IJV) is compressed. Water occlusion test (WOT) was ineffective on either ear. (1) Circuitous enlarged emissary vein; (2) bilateral sigmoid plate dehiscence speculative due to long-term strong flow anomalies caused by AVF; (3) right-sided jugular bulb (JB) dehiscence (EAC, external auditory canal); and (4) widening of foreman ovale. Red arrows indicate meningoceles involving both of Meckel caves (axial and coronal T2 scans). (C) A case with left-sided multiple dehiscence (white arrow) who responded to ipsilateral internal jugular vein (IJV) compression and water occlusion test (WOT). Notice that multiple sites of dehiscence situate near level of middle ear ossicles which opens directly to the antrum. (D) A patient with right-sided dehiscence (white arrow) adjacent to the junction of sigmoid sinus and emissary vein. The volume of PT initially reduced but immediately restored during the IJV compression possibly due to excessive flow disturbance at the dehiscent region. WOT was ineffective. (E) An interesting case with right-sided PT caused by an ipsilateral diverticulum and an anastomosing petrosquamous sinus [(1, axial CT), and (1#, coronal CT)] that exits from the diverticulum (2, coronal CT), courses over the lateral superior surface of the petrous bone (3, coronal CT), and drains anteroinferiorly into the retromandibular vein (4, axial CT). Red arrows indicate petrosquamosal sinus; D, diverticulum. Patient's PT disappeared during IJV compression and WOT test. (F) T2 axial scan demonstrating a patient with right-sided intracranial arachnoidal cyst (black arrows), and meningoceles involving both of Meckel caves (white arrows). Her PT presented on the left-side with ipsilateral dehiscence and positive response to IJV compression. Her opening lumbar pressure was 14 cm H_2O and WOT was ineffective

Sigmoid sinus wall anomalies (SSWAs), such as sigmoid sinus wall dehiscence and diverticulum, are the most identified anomalies during initial radiologic examination screening for venous PT.[8] Gradual thinning of the sigmoid plate results in failure to insulate the intrasinus vascular sound, rendering the motion of blood flow audible.[1,10,11] In addition to SSWAs, hemodynamic perturbations in the transverse-sigmoid sinus are considered to contribute to increases in the volume of vascular sound.[10] Such perturbations include: (1) excessive flow kinetic energy in the form of transverse-sigmoid sinus enlargement (TSSE) or sigmoid sinus ectasia,[5,10,12,13] (2) flow obstructions such as transverse sinus stenosis (TSS) and arachnoid granulation,[14-18] and (3) idiopathic intracranial hypertension (IIH).[8] Despite a convoluted relationship among PT, SSWAs, and flow pathologies, the incidence of SSWAs is higher in patients with IIH than in healthy controls and the general PT population.[7] Researchers have also suggested that spontaneous cerebrospinal fluid leakage can occur in the late stage of SSWA development if increases in intracranial pressure remain unresolved.[19] Furthermore, a recent study provided striking evidence suggesting that addressing flow pathologies can reverse the process of SSWA formation.[20] SSWAs may therefore represent anatomical abnormalities that develop due to pathologic accumulation of flow perturbations.[10]

SSWA-induced air pulsation inside the mastoid cavity has recently been identified as a causative factor of PT.[21] A positive response in the water occlusion test indicates that aerial transmission of PT energy from the dehiscent/thinned sigmoid plate to the inner ear via the antrum is highly plausible.[21,22] To that effect, transtemporal resurfacing of dehiscence has been highly successful in isolating the transmission of PT.[23] However, because there may be multiple sites of dehiscence on the ipsilateral temporal bone, it remains imperative to fully examine the locations of dehiscence. Additionally, patients with bilateral SSWAs are not rare. Contralateral emergence of soft PT in patients with bilateral dehiscence can occur during ipsilateral jugular vein compression or after surgery, which can be explained by the Stenger principle.[8]

The association between venous PT and middle ear mechanics remains unaccentuated. The purpose of this study was to quantify the transmission pathway and amplitude of PT using sensing engineering techniques. The previous extraluminal compression technique was modified to achieve higher surgical efficacy and safety based on objective patient-specific findings. Considering these objective findings in conjunction with the expected surgical benefits may allow surgeons who utilize transtemporal approaches to resolve venous PT safely and effectively.

2 | MATERIALS AND METHODS

2.1 | Study design and radiologic measurements

This study recruited 33 participants (4 men and 29 women) who had undergone strategized transtemporal osteovascular reconstruction

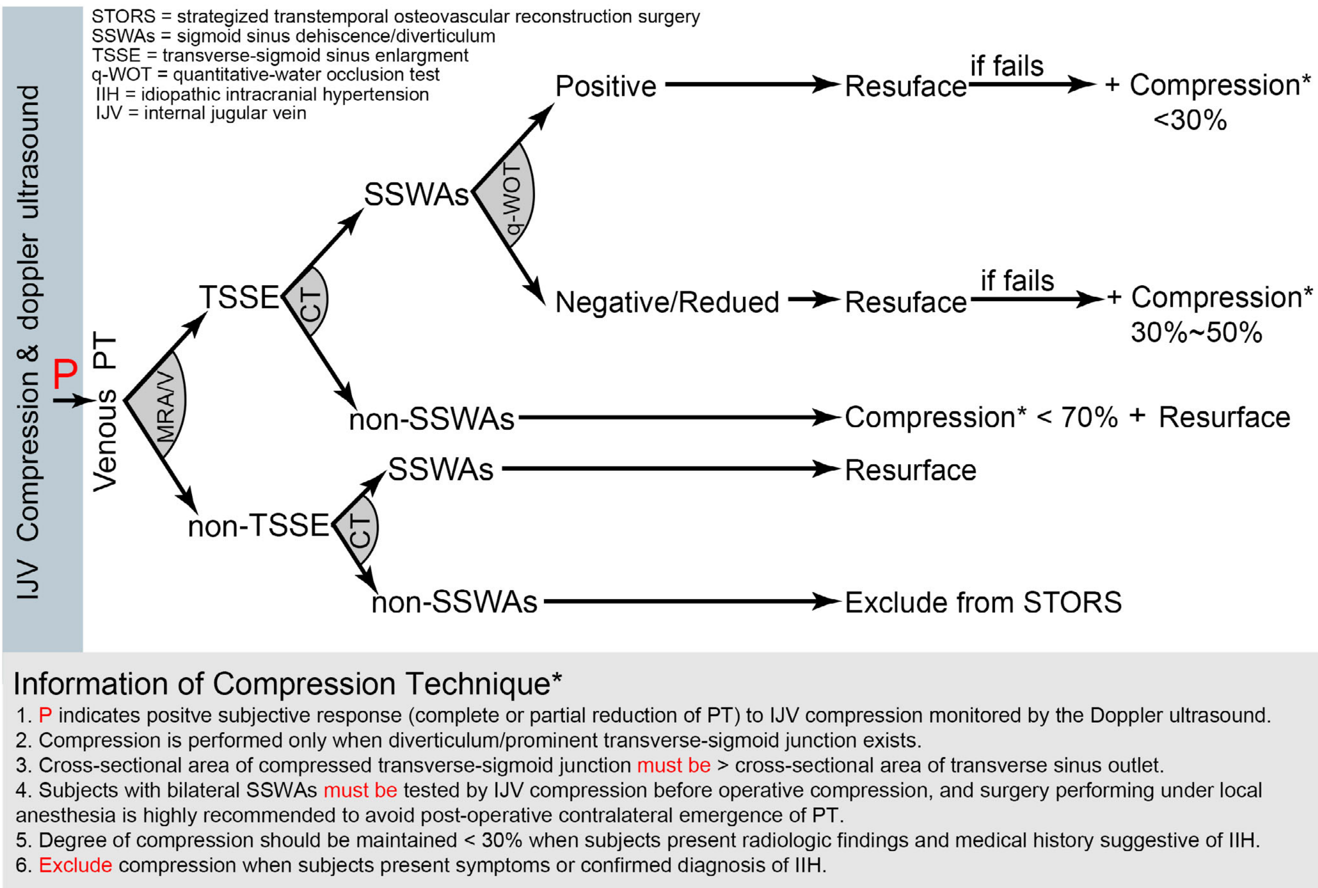

FIGURE 2 Flowchart of strategized transtemporal osteovascular reconstruction surgery (STORS)

surgery with complete operative Doppler ultrasound and quantitative water occlusion test (q-WOT) data. All participants were treated at the Otology and Skull Base Surgery Center of the Eye, Ear, Nose, and Throat Hospital at Fudan University from July 2020 to August 2021. Jugular vein compression and radiological high-resolution computed tomography (CT)/contrast-enhanced CT venography, magnetic resonance angiography/venography, cervical Doppler ultrasound, and audiometric examinations were performed prior to transtemporal surgery for complete evaluation. The study adopted strict inclusion criteria of a diagnosis of venous PT and radiologic evidence of ipsilateral SSWAs. PT of venous origin was determined based on a positive response (completely or partially reduced sensation of PT) to compression of the ipsilateral internal jugular vein by the transducer, which was monitored using Doppler ultrasound. In addition, 15 controls without SSWAs were enrolled to examine the efficacy of q-WOT. CT and q-WOT were performed for the initial diagnosis of venous PT at the PT clinic.

Radiological analyses were performed using a workstation NUMARIS/4 (SYNGO MRB17, Siemens AG) and Mimics 19.0 software (Materialize, Belgium). TSSE was defined as 150% enlargement when compared to the contralateral transverse-sigmoid sinus.[5,10] Transverse sinus hypoplasia was defined as stenosis in 40% of the entire length and/or discontinuity of the transverse sinus.[10,24] Dehiscence was defined as the absence of boney plate overlying the sigmoid sinus on at least three consecutive 0.6-mm axial CT slices and was intraoperatively verified by surgeons in all patients.[1] Diverticulum was defined as the outpouching of the sigmoid sinus vessel wall protruding into the mastoid air cell(s) and/or cortex with an irregular trajectory of the vascular silhouette.[1] A normalized scale developed by dividing the largest cross-sectional area by the smallest was used to determine the degree of TSSE.[10]

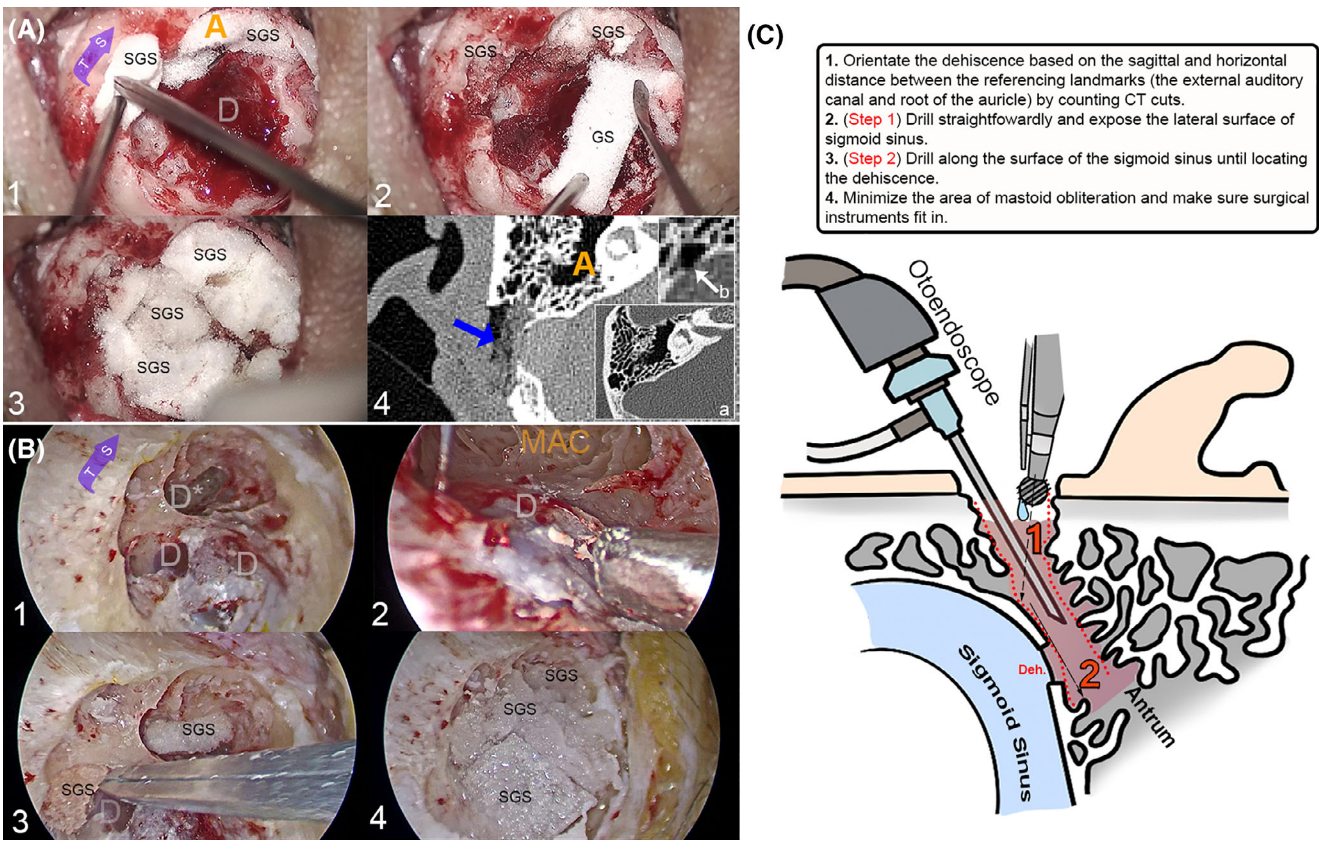

FIGURE 3 (A) Intraoperative photograph showcasing the blockage of sound transmission pathway of pulsatile tinnitus (PT) using gelatin sponge smeared with surgical glue (solidified gelatin sponge) of a case with right-sided PT. These solidified gelatin sponges sufficed to isolate PT from surface of dehiscence and antrum transmitting to the cochlea permanently. Soft gelatin sponges were utilized to temporarily fill dead spaces and fixate operative fillers if necessary. D, dehiscence; A, antrum; SGS, solidified gelatin sponge; GS, gelatin sponge; T-S arrow, transverse to sigmoid sinus flow direction. (1) The blockage of antrum. (2) Covering dehiscence. Photograph showcasing soft gelatin sponge placed above the dehiscence region prior to surgical glue fixation. (3) Reinforcement of the sigmoid plate. (4) Postoperative contrast-enhanced computed tomography showcasing the blockage of antrum using SGS (blue arrow). (B) STORS performed using an otoendoscope on a case with right-sided PT. D, dehiscence; *, same location marked. (1) Thinned wall/dehiscence observed at three distinct locations at the transverse-sigmoid junction. (2) A close observation at the thinned sigmoid plate situating at medial portion of sigmoid sinus. (3) Resurfacing of dehiscence with less spaces utilized. (4) Reconstruction of the defected sigmoid plate. (C) Schematic demonstration of an otoendoscope technique in reducing the area of mastoid obliteration (an optional technique for STORS)

2.2 | Strategized transtemporal osteovascular reconstruction surgery

All surgical participants in this study presented with PT and ipsilateral SSWAs and met the surgical inclusion criteria. The inclusion criteria for surgical candidates were refined as follows: (1) complete elimination of PT or complete/partial reduction of PT during Doppler diagnostic jugular compression in patients with SSWA; (2) pulse-synchronicity of PT confirmed via Doppler ultrasound auscultation of the ipsilateral jugular vein. The exclusion criteria and designated operative strategy for compression/reconstruction techniques are presented in Figure 2.

The original extraluminal angioplasty technique was improved and re-established as "strategized transtemporal osteovascular reconstruction surgery" based on clinical experience with over 100 surgical cases from 2009 to 2020.[5,10,12] The current technique is performed under general and local anesthesia as appropriate. Modifications to intraoperative procedures included the following: (1) isolating sound transmission at the antrum and air cells via fixation of solidified gelatin sponges smeared with surgical glue; (2) prioritizing the resurfacing of the sigmoid plate and antrum/air cells and performing extraluminal compression if the resurfacing procedure failed to eliminate PT; and (3) (optional) using an otoendoscope to reduce the bone-burring area of the fenestration window. As the drilling reaches the lateral surface of sigmoid sinus, the bone burring pathway can be tunneled through the surface of sigmoid sinus using an otoendoscope, which allows the reduction in the area of mastoid obliteration, typically suitable for a medially situated dehiscence in comparison to using a microscope. See Figure 3 for operative details.

Notably, iatrogenic causes of IIH and contralateral emergence of PT should be addressed using the reversal compression technique. Removing operative fillers and restoring the volume of venous return using sutures to suspend the compressed sinus wall

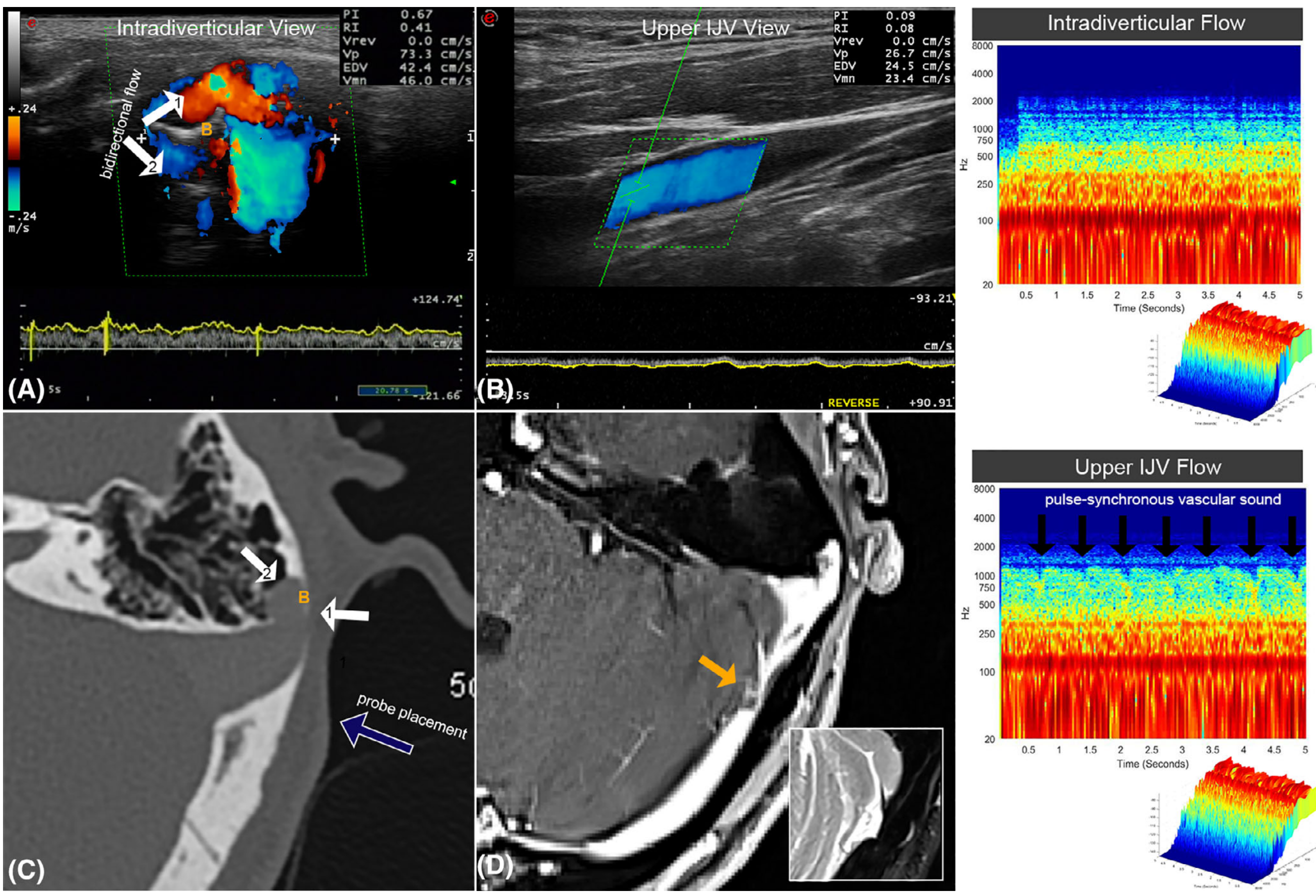

FIGURE 4 The insonation and sonification of intradiverticular flow in a case with large pedunculated diverticulum anterolaterally protruding under the skin tissue highlighting that the overlying dehiscence rather than the intradiverticular flow is the cause of pulsatile tinntius. (Right panel) observation of pulse-synchronicity differences of the vascular sound inside the diverticulum (audio data shown in Data S1) and internal jugular vein (Data S2) using the spectro-temporal analysis. (A) The intradiverticular hemodynamic characteristics and Doppler ultrasonographic view. Arrow #1 and arrow #2 indicate opposite directions of flow. (B) indicates the remnant bone structure separating the diverticular vascular walls. Notice differences of blood flow velocity and pulse-synchronicity between the diverticular region and upper internal jugular vein (IJV). See also graph C for presence of relative structures on axial CT. (B) The hemodynamics of upper IJV. (C) The axial CT demonstrating the anterolateral protrusion of a large diverticulum and probe placement for insonation. Relative structures observed via the ultrasonographic insonation (arrow #1, arrow #2, and B, see also graph A). (D) Image of axial T1-3D volumetric interpolated breath-hold sequence (VIBE). Notice that the intrinsic transverse sinus stenosis (TSS) caused by the arachnoid granulation (yellow arrow, see also T2-turbo spin echo image)

should be performed timely to reverse the impaired flow condition. A detailed medical treatment process and the reversal compression technique has been introduced in our previous work in detail.[10]

2.3 | Doppler ultrasound flow analysis

Ultrasonographic examination was performed using a color-coded Doppler ultrasonographic system (MyLab ClassC, ESOATE SpA, Genoa, Italy), in line with previously described methods.[6,22] The hemodynamics of the upper segment of the bilateral IJV were gauged at the region of the mandible extending to the jugular bulb at the level of the skull base using a sonographic transducer (LA-523) with a center frequency of 4–13 MHz. Bilateral hemodynamic parameters including mean flow velocity, peak velocity, and flow volume were recorded. See Figure 4 for details regarding postauricular measurements and sonification of the intradiverticular flow.

2.4 | Quantification of PT loudness and annoyance

A visual analog scale (VAS) was used to quantify the loudness of PT in all 33 patients with SSWAs before operation and to assess short-term postoperative changes in PT within 1 week after the procedure. The level of tinnitus was rated on a scale from 0 to 10 and classified as follows: 1–3, mild tinnitus; 4–7, moderate tinnitus; 8–10, severe tinnitus. Additionally, the broadly applied questionnaire Tinnitus Handicap Inventory (THI) for was also implemented to investigate the operative changes of PT pertaining to the handicap nature of tinnitus.

2.5 | Quantitative-water occlusion test (q-WOT)

Participants were confirmed to be free of ear drum perforation and otitis media using an otoendoscope and preoperative CT prior to the q-WOT. The q-WOT was performed as follows: warm saline was injected into the external auditory canal at a moderately slow rate using a 5-ml

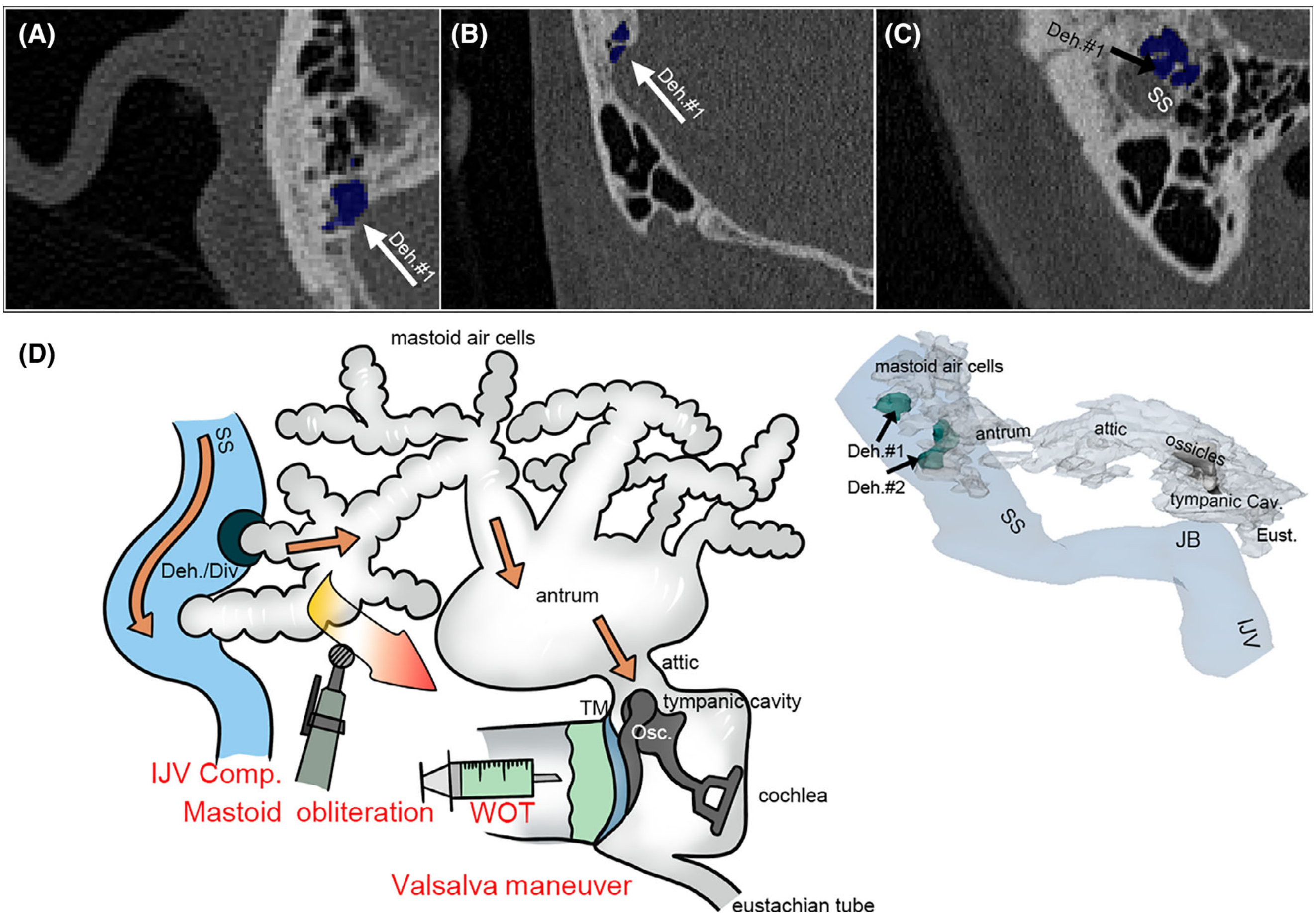

FIGURE 5 Schematic diagram introducing influence factors of air-transmission route of PT (left panel) and patient-specific 3D reconstruction of multiple dehiscence-related air transmission pathways (right panel) using high-resolution computed tomography (CT) (graphs A–C). Deh., indicate dehiscence; Div., diverticulum; SS, sigmoid sinus; JB, jugular bulb; IJV, internal jugular vein; Comp., compression; WOT, water occlusion test; osc., middle ear ossicles; Cav., cavities. (A) Axial plane of dehiscence no. 1. Aerial space marked with blue color. (B) Coronal plane of dehiscence no. 1. Aerial space marked with blue color. (C) Sagittal plane of dehiscence no. 1. Aerial space marked with blue color

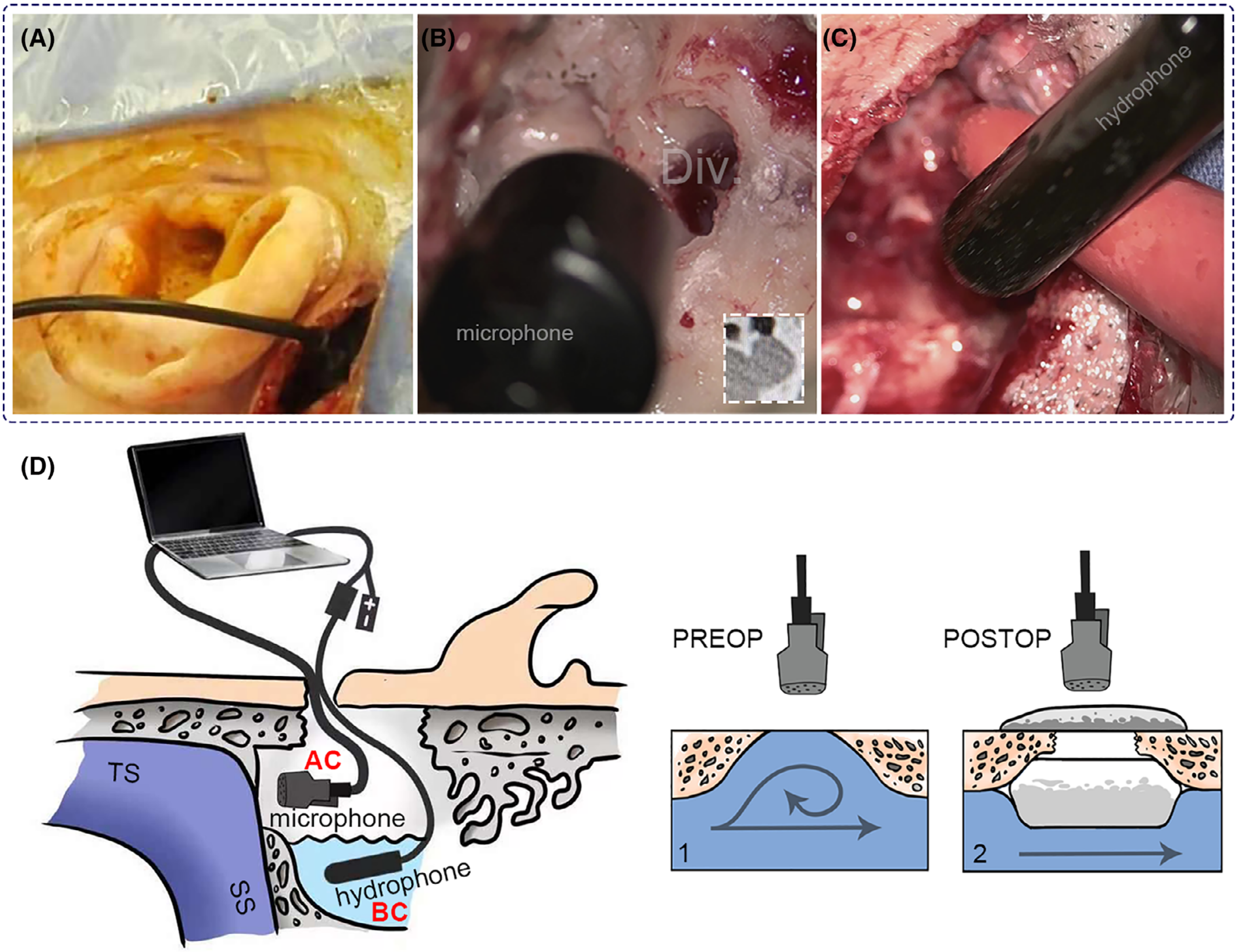

FIGURE 6 Intraoperative setup of a condenser microphone and hydrophone acoustic sensors to verify preoperative (1) and postoperative (2) air-conduction (AC) and bone-conduction (BC) transmission pathways of pulsatile tinnitus (PT). (A) Photograph of the condenser microphone inserted into the mastoid cavity with reapproximated periosteal pedicle and skin flap. The incision was later covered by stainless-steel plate to deflect external sound interference during PT recording. (B) Condenser microphone recording of case with small diverticulum anterolaterally protruding into mastoid air cells. (C) Placement of hydrophone after administration of warm saline solution

TABLE 1 Clinical characteristics of 33 participants with sigmoid sinus wall anomalies. Variables of continuous data are expressed as median and interquartile range

	Dehiscence ($n = 21$)	Diverticulum ($n = 12$)	p value
Normalized transverse sinus stenosis degree			
Ipsilateral side	4.1 (3.4/4.7)	5.3 (4.5/7.5)	.319
Contralateral side	3.4 (2.5/4.7)	6.0 (4.7/8.6)	.025
Operative data			
Operative changes of PT (E/R/N)[a]	18/3/0	11/1/0	>.999
Cross-sectional area reduction (%)	29.5 (15.1/57.5)	59.0 (50.5/59.6)	.009
Median postoperative follow-up duration (Mos)	4 (2/8)		
q-WOT			
Responders (E & R)/nonresponders (N & I)[b]	14/7	9/3	.710
Responders' injection volume (ml)	1.3 (0.9/2.3)	1.3 (1.1/1.5)	.949

[a]E, elimination; R, reduction; N, no change.
[b]Responders indicates elimination (E) or reduction (R) of pulsatile tinnitus; non-responsive indicates no change (N) or increase (I) of pulsatile tinnitus.

syringe. The injection was paused the moment PT disappeared; if PT persisted, a total of 5 ml of solution was injected until depletion, filling the external ear canal and auricular concha. Participants were asked to carefully distinguish the changes in PT and were instructed to provide immediate feedback on whether the injection was effective (disappearance/reduction) or noneffective (no change/increase) in altering PT. The association between q-WOT results and the intactness of the sigmoid plate was also investigated (Figure 5).

2.6 | Intraoperative installation of sensors

A condenser microphone and a hydrophone were deployed to detect the vascular sound, as shown in Figure 6. Both sensors were disinfected using low-temperature plasma sterilization. Of the 33 participants, eight participants agreed and were enrolled for the microphone examination. The hydrophone was only applicable on two participants due to its size unfittable into the mastoid bone of other six participants. To verify whether PT transmits via air conduction, a microphone recording was performed with a reapproximated auricle. The recording took place before and after the fixation of sound insulation materials and was monitored intraoperatively to ensure surgical efficacy. To clarify whether bone conduction contributes to PT transmission, the hydrophone was submerged in a warm saline solution near the sigmoid sinus wall. See Data S5. for sensor specifications, methods for post-analysis of the recorded acoustic data).

2.7 | Statistical analysis

Statistical analysis was performed using RStudio version 1.1.383 (RStudio, Boston, MA, USA). Descriptive data were analyzed using Fisher's exact test. After the Shapiro–Wilk test was performed to assess the normality of continuous data, the Mann–Whitney *U* test was used to examine differences in continuous data, as appropriate. Spearman correlation coefficients were computed to examine the correlations among the objective measurements. Correlation strength was defined as very strong (0.5–1.0), moderate (0.3–0.5), or weak (<0.3) according to the criteria provided by Cohen.[25] The significance level was set at $p < .05$.

3 | RESULTS

3.1 | Descriptive/radiological characteristics

Among the 33 patients with SSWAs, there were 21 cases of dehiscence and 12 cases of diverticulum. The mean age and body max index were 33.7 ±9.5 years (range: 23–69 years) and 26.03 ± 4.24 kg/m^2 (range: 19.5–35.1 kg/m^2). Empty sellae were found in seven participants (dehiscence/diverticulum = 4/3), whose compression degrees were all less than 30%. Of all 33 participants, PT of 32 participants presented on their dominant side of transverse-sigmoid sinus; only one participant whose PT was observed on the smaller side of the sinus system. In those 32 subjects whose PT presented on the dominant side of the sinus system, contralateral transverse sinus hypoplasia was found in 8 participants. The median values of the ipsilateral and contralateral normalized degrees of the TSS were 4.5 (3.6/6.1) and 5.4 (4.6/8.1), respectively (Table 1). There was a significant difference in the contralateral side of the TSS between the dehiscence and diverticulum cohorts (Mann–Whitney *U* test, $p = .025$).

3.2 | Outcomes of the q-WOT

Twenty-three participants with SSWA (dehiscence/diverticulum = 14/9) responded to the q-WOT (disappearance/reduction = 16/7) (Table 2). Ten participants did not respond to the q-WOT after a full 5-ml injection. Additionally, PT did not disappear after the depletion of 5 ml in those exhibiting reduced PT. The median injection volume among all responders was 1.3 (1.1/1.6) ml (range: 0.8–3.1 ml), wherein the median values for the dehiscence and diverticulum groups were 1.3 (0.9/2.3) and 1.3 (1.1/1.5) ml, respectively (Table 1).

Thirteen of the 15 controls were nonresponders, two of whom reported an increase in PT perception. Two controls reported a reduction in PT after injections of 1.4 and 2.1 ml of solution, respectively.

TABLE 2 Characteristics of q-WOT between 33 participants with sigmoid sinus wall anomalies and 15 controls without sigmoid sinus wall anomalies

	Participants	Controls	Total[a]
Responders (E & R)[b]	23	2	25
nonresponders (N & I)	10	13	23
Total[b]	33	15	48
Responders' injection volume (ml)[c]	1.3 (1.1/1.6)	1.7 (1.5/1.9)	0.420

Note: Variables of continuous data are expressed as median and interquartile range.
[a]Responses to the q-WOT significantly differed between participants with sigmoid sinus wall anomalies (SSWAs) and controls, $p < .001$.
[b]Responders indicate elimination (E) or reduction (R) of pulsatile tinnitus; nonresponders indicate no change (N) or increase (I) of pulsatile tinnitus.
[c]Comparison of responder's injection volume between participants versus controls, Mann–Witney *U* test.

TABLE 3 Hemodynamic parameters of 33 participants and correlation with degree of compression

	Units	Preoperative		Postoperative		Operative hemodynamics *p* value		Degree of compression[a]
		Ipsilateral	Contralateral	Ipsilateral	Contralateral	Ipsilateral	Contralateral	Ipsilateral/Contralateral
Flow volume	(g/s)	16.8 (11.8/24.5)	7.0 (4.0/11.6)	16.4 (11.6/23.1)	6.5 (3.6/11.1)	0.719	0.787	−0.178/0.226
Peak velocity	(m/s)	0.35 (0.30/0.42)	0.34 (0.28/0.41)	0.35 (0.27/0.47)	0.33 (0.25/0.39)	0.847	0.460	−0.191/0.261
Mean velocity	(m/s)	0.28 (0.22/0.35)	0.24 (0.20/0.31)	0.25 (0.19/0.36)	0.27 (0.16/0.29)	0.648	0.792	−0.181/0.278
Pulsatility index	NA	0.58 (0.32/0.69)	0.35 (0.61/0.81)	0.45 (0.28/0.57)	0.61 (0.34/0.71)	0.222	0.714	0.112/0.245
	Unit		**Bilateral**		**Bilateral**	**Operative *p* value**		**Degree of compression[a]**
Flow volume$_{BTOV}$[b]	(g/s)		26.0 (18.1/35.7)		23.9 (16.8/31.6)	.758		−0.06

Note: Variables of continuous data are expressed as median and interquartile range.
[a]Spearman correlation: degree of compression versus difference between operative ipsilateral hemodynamic variables.
[b]BTOV, bilateral total outflow volume.

TABLE 4 Operative subjective outcomes of 33 participants

	Visual analog scale (VAS) loudness	THI score
Preoperative[a]	7 (6/8)	48 (36/58)
Postoperative[a]	0 (0/0)	0 (0/4)
p value	<0.01	<0.01

Note: Variables of continuous data are expressed as median and interquartile range.
[a]Mann–Whitney *U* test.

Responses to the q-WOT significantly differed between participants with and without SSWAs (Fisher's exact test, $p < .001$) (Table 2).

3.3 | Operative surgical outcomes

All 33 participants with SSWAs in this study underwent strategized transtemporal osteovascular reconstruction surgery. PT completely was resolved in 29 participants. Four patients experienced postoperative recurrence of PT during the first 2–7 days of follow-up. All four of these patients had intermittent reductions in PT. The median decrease in the cross-sectional area of the enlarged transverse-sigmoid junction was 29.5% (15.1/57.5%) in the dehiscence group and 59.0% (50.5/59.6%) in the diverticulum group. The difference between the two groups was significant (Mann–Whitney *U* test, $p < .01$) due the need to fully eliminate the diverticulum. Five patients without a prominent transverse-sigmoid junction were free of compression. Neither iatrogenic increases in intracranial pressure nor contralateral emergence of PT was observed during the mean postoperative follow-up period of 5.1 months (range: 0.5–13 months).

3.4 | Operative alterations of ultrasound hemodynamics and subjective outcomes

The median value for preoperative bilateral upper jugular outflow was 26.0 (18.1/35.7) g/s (range: 3.1–53.8 g/s), while that for postoperative outflow was 23.9 (16.8/31.6) g/s (range: 4.7–63.4 g/s). Complete data for the ultrasound hemodynamic variables are shown in Table 3.

Results of operative assessment of subjective outcomes are shown in Table 4 and Figure 7. There was a significant difference between the median preoperative (rating: 7; $n = 6/8$) and postoperative (rating: 0, $n = 0/0$) (Mann–Whitney *U* test, $p < .01$). A significant difference was also found between the preoperative (score: 48, $n = 36/58$) and postoperative (score: 0, $n = 0/4$) Tinnitus Handicap Inventory (THI) scores (Mann–Whitney *U* test, $p < .01$).

3.5 | Outcomes of intraoperative sensor detection

There was a statistically significant difference between the median preoperative and postoperative RMS amplitudes (Mann–Whitney *U* test, $p < .01$), which were 0.0011 (0.00081/0.0018) and 0.00014

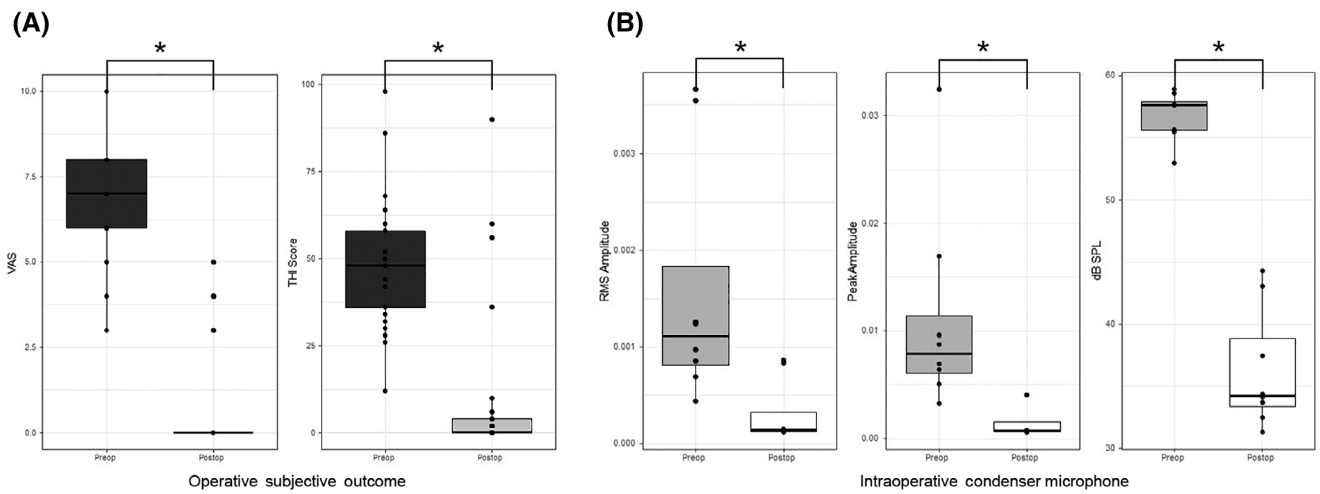

FIGURE 7 Comparison of operative subjective characteristics and intraoperative sensed amplitudes of pulsatile tinnitus (PT). *, statistical significance (p value <.05) by the Mann–Whitney U test

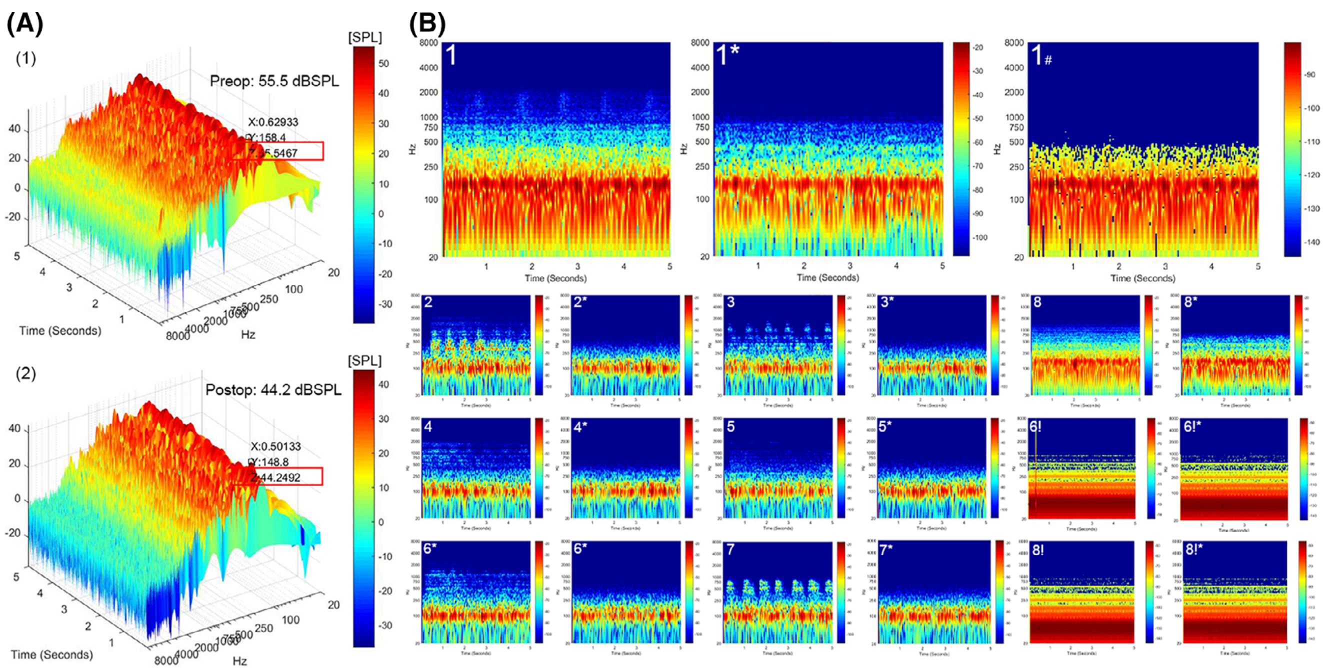

FIGURE 8 Results of intraoperative recordings of pulsatile tinnitus (PT) of eight participants. (A) Comparison of pre-and post-operative RMS, peak amplitudes, and dB SPL. *, statistical significance by Mann–Whitney U test. (B) Spectro-temporal analysis of operative recordings of eight participants. The preoperative, postoperative, and the filter of lower 50%-loudness of representative case (case 1) is highlighted above. *, postoperative results; #, the filter of lower 50%-loudness;!, hydrophone results. (See Data S3 for preoperative PT and Data S4 for postoperative recording.) (C) Display of operative peak SPL amplitude of representative case (case 1) using waterfall diagram (marked with red rectangles)

(0.00012/0.00032), respectively (Figure 7). The median preoperative and postoperative peak amplitudes were 0.0078 (0.006/0.011) and 0.00073 (0.00063/0.0015), and the difference between the two was significant (Mann–Whitney U test, $p < .01$). The median preoperative and postoperative peak amplitudes were 57.6 (55.5/57.9) dB SPL and 34.3 (33.4/38.8) dB SPL, with a postoperative drop of 20.4 dB SPL on average. However, no pulse-synchronous vascular sounds were observed in the microphone data of one participant or in the hydrophone data of both participants (Figure 8).

4 | DISCUSSION

The present study aimed to identify the transmission pathway and amplitude of PT using sensing engineering techniques. As evidenced by the sensing techniques and intraoperative monitoring data, the sound wave pressure of PT permeates via the dehiscent sigmoid plate. Fortifying the dehiscent sigmoid wall and aerial cavities using solid sound-insulating materials is a major procedure for eliminating PT.[1,23] As the hydrophone did not detect pulse-synchronous signals underwater, this suggests that the dominant transmission pathway for PT is the air-conduction route in patients with SSWAs. Because water is a more efficient intermedium for transmitting sounds and vibrations than air, hydrophone results obtained without direct contact with the vessel wall indicate that the effect of the bone-conduction pathway is probably limited, which verifies the viewpoint proposed by Eisenman and colleagues.[11]

Another crucial factor related to surgical efficacy is the ability to maintain airtight middle ear pressure. When invasion of the mastoid is deliberately avoided, resurfacing dehiscence alone may not be sufficient to eliminate PT.[5] However, PT disappears the moment that the mastoid air cells are cut open. This indicates that, aside from intraoperative emotional disturbances, the dissipation of vascular sound wave pressure into the atmosphere, mastoid obliteration, and the entry of fluids likely interfere with the transmission of PT. Therefore, the destruction of aerial spaces between the tympanum, antrum, and air cells and/or redistribution of middle air pressure may in part serve a secondary therapeutic effect, particularly in q-WOT responders.

The q-WOT reflects the integrity of the sigmoid plate and aerial middle air cavities. Park et al. proposed air-pulsation or turbulence conduction as prominent causes of venous PT based on the water occlusion test.[21] Although the air-pulsation theory may explain why the sinus vessel wall was completely exposed to air, approximately 70% of subjects with SSWA in our study responded to the q-WOT. In fact, intraoperative application of laser displacement sensors indicated that the displacement of the dehiscent/diverticular wall was not pulse-synchronous in eight of nine SSWA cases prior to the surgical separation of vascular and osseous walls.[22] Most dehiscence predetermined by CT scans is covered by a thin layer of boney plate tightly fixated to the sigmoid sinus, with the periosteal membrane above and dura mater underneath. While the air-pulsation theory may be true, the underlying mechanism is likely more relevant to the flow sound pressure emanating from the sinus flow via SSWAs,[26] whereby external pressure is added to the eardrum to pause or reduce PT. This may be due to an increase in the internal pressure of the middle ear and mechanical restriction of the ossicular chain movements. It also explains why performing the Valsalva maneuver may seize PT temporarily in a few participants, and why sound distortion may occur after the injection of the solution.

The compression technique is crucial when the reconstruction technique fails to eliminate PT. This can be observed in patients with SSWAs who are irresponsive to q-WOT but sensitive to the modulation of jugular hemodynamics. Although some authors have suspected a need to decompress the sigmoid sinus,[27] other studies have reported that eradicating flow pathologies at the prominent sinus junction reduces flow amplitude.[5,17,18] Furthermore, the operative change in jugular outflow volume is insignificant based on current Doppler outcomes. This may be due to the regulation of flow dynamics via the external jugular and vertebral pathways. Although it is highly possible that some degree of flow anomaly/increase in intracranial pressure promotes osteolytic effects,[19,20] increased cerebrospinal opening pressure is not a definitive finding in patients with PT associated with SSWAs.[28] Except for neuro-ophthalmologic signs or symptoms of IIH, neuroimaging findings are not required for the diagnosis of definite IIH.[29] Furthermore, the debate over diagnostic criteria for IIH (e.g, 20 or 25 cm H_2O cerebrospinal fluid pressure) and nomenclature persists.[30,31] After prudent preoperative assessments, reshaping the convex contour of the prominent transverse-sigmoid junction is safe, which has also been corroborated by other authors.[2,13,32]

Aside from the physical characteristics of PT, emotional disturbances caused by PT also require clinical attention. Recent studies have demonstrated that anxiety, depression, and sleep disorders are common in patients with PT.[6,33] Although THI scores were significantly reduced after the surgical intervention, emotional disturbances persisted in some of our participants. This may be because most participants experienced a "plugged" feeling in the ear due to intraoperative fluid retention and ear numbness after surgery. Concern regarding these symptoms may have influenced emotional outcomes following the procedure. Therefore, surgeons should aim to provide sufficient preoperative education to patients prior to surgery, and postoperative care should be implemented to reduce the influence of emotional experiences on postoperative results.

The main limitation of this study was its insufficient sample size for the intraoperative studies due to patients' unwillingness to participate in this additional test. Collecting intraoperative acoustic data can be arduous when the sigmoid sinus is laterally placed, although the current study provides strong evidence that PT transmission occurs via air. Additionally, as we aimed to avoid unnecessary expansion of the area of mastoid obliteration, we were unable to utilize the hydrophone in six patients due to improper fitting. Therefore, we were unable to fully expose outliers or represent those without SSWAs due to the small sample size. Further comparisons involving those without SSWAs using the hydrophone technique are warranted. Second, the radiologic data of the controls were mostly incomplete. Further studies are warranted to compare the ultrasonographic hemodynamics between patients with SSWAs and those without. Lastly, additional studies are required to determine whether fluid retention in the middle ear contributes to recurrence of PT within 2 months after surgical intervention. In most patients with postoperative fluid retention identified using an otoendoscope, PT will disappear once the fluid drains and the area is refilled with air.

5 | CONCLUSION

The current method for strategized transtemporal osteovascular reconstruction surgery highlights the importance of both the compression and reconstruction techniques. Patient-based individual mechanics and radiological characteristics are integrated to avoid adverse postoperative complications while maximizing surgical efficacy. Through the intraoperative installation of acoustic sensors, our findings indicated that venous PT associated with SSWAs is predominantly transmitted via the air-conduction route. If objective findings such as q-WOT results and data obtained using intraoperative sensing techniques suggest that the transmission of venous PT is involved in middle ear air conduction, the reconstruction technique should be prioritized; if less involvement of middle ear air-conduction is indicated, addressing flow pathologies may be imperative for resolving venous PT.

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CONFLICT OF INTEREST

The authors declare no conflicts of interest.

AUTHOR CONTRIBUTIONS

Yue-Lin Hsieh designed the study, collected the dataset, and ran the statistical and acoustic analyses. Xiaobing Xu performed the Doppler examinations. Yongzhen Wu performed audiometric/psychoacoustic testing. Wuqing Wang and Yue-Lin Hsieh provided clinical and post-clinical online consultations. Wuqing Wang was the chief otologists who performed all surgeries and supervised this study. Yue-Lin Hsieh co-supervised this study. All the authors gave their agreement to the full submission.

ETHICS STATEMENT

This study was approved by Ethical Committees of the Eye, Ear, Nose, & Throat Hospital in Shanghai, China (no. 2020010-1). All 33 participants and 15 controls who participated in this study provided written informed consent, and all experimental measurements were conducted in accordance with the Declaration of Helsinki.

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

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