INTRODUCTION

Vascular tinnitus is the most common form of pulsatile tinnitus, especially when the sound corresponds well with the patient’s pulse. Vascular tinnitus is an uncommon otologic symptom; the incidence has been reported to be approximately 4% in patients with tinnitus [1]. It originates from vascular etiologies and may occur by the result of turbulence in the blood flow. The sound produced by the turbulence is transmitted to the inner ear and causes tinnitus percept. According to the vascular structures of origin, vascular tinnitus can be classified as arterial or venous type [2]. In the clinical practice of treating tinnitus patients, the diagnosis of vascular tinnitus is challenging due to its rarity and no standardized diagnostic protocol. Therefore, the otolaryngologist is likely to misdiagnose it as idiopathic subjective tinnitus. Because significant intracranial conditions can evoke vascular tinnitus, a diagnostic failure may lead to a serious outcome.

In the present study, we summarized the 10-year data on vas-
cular tinnitus of our tinnitus clinic to investigate the frequency of the underlying etiologies, to introduce a diagnostic protocol, and to evaluate the treatment outcomes.

MATERIALS AND METHODS

Patients
The clinical data on 57 patients with pulsatile tinnitus who presented to our tinnitus clinic between April 2001 and December 2011 were retrospectively reviewed by a single tinnitus specialist (SNP). This study was approved by the Institutional Review Board of Seoul St. Mary’s Hospital (KC12RISE0250).

History and physical examination
A detailed history of pulsatile tinnitus was taken, and the standardized interview form and validated tinnitus questionnaires were administered to the patients to score the subjective tinnitus severity. These self-rating scores included awareness (AW, percent of time aware of tinnitus during the waking hours), 10-point visual analogue scale for tinnitus loudness (LD)/annoyance (AN)/effect on daily life (EOL), and tinnitus handicap inventory (THI). The initial diagnostic approach depended primarily on the tinnitus quality described by the patient. A throbbing, rushing, or humming sound suggested a vascular etiology. Thorough physical examinations were performed through otoscopy, auscultation of the ear canals, and a full head and neck examination. A tympanic mass could be visible on otoscopy. When the patient showed a normal eardrum with bruises on the auscultation around the head and neck area, the patient’s pulse was checked if it was synchronous with the tinnitus sound. To discriminate the vascular structures of origin, the ipsilateral internal jugular vein (IJV) was gently compressed and any changes in tinnitus loudness were examined. Head turning test was also conducted to confirm the compression maneuver. In tinnitus of venous origin, light digital pressure over the ipsilateral IJV or head turning towards the tinnitus side allowed tinnitus to diminish or subside. When those maneuvers exerted no effect on tinnitus loudness, tinnitus of arterial origin was suspected.

Audiometric, laboratory, and imaging tests
All patients underwent pure tone audiometry, speech audiome-

![Fig. 1. Diagnostic algorithm for vascular tinnitus. TBCT, temporal bone computed tomography; TBMRI, temporal bone magnetic resonance imaging; HBP, hypertension; IJV, internal jugular vein; TN, tinnitus; MRA, magnetic resonance angiography; MRV, magnetic resonance venography; TCD, transcranial Doppler sonography; BIH, benign intracranial hypertension; ACAD, atherosclerotic carotid artery disease; AVF, arteriovenous fistula.](image-url)
try, and tinnitogram. Pure tone average (PTA) was determined at 0.5, 1, 2, and 3 kHz. We defined the hearing as low-frequency hearing loss when the low tone average at 125, 250, and 500 Hz was ≥20 dB higher than the high tone average at 1, 2, and 3 kHz. Blood pressure was measured to rule out hypertension, and blood tests were performed to exclude anemia and hyperthyroidism, which could increase cardiac output and cerebral blood flow.

In patients with a tympanic mass medial to the eardrum, temporal bone computed tomography (TBCT) and/or magnetic resonance imaging were obtained to demonstrate jugular bulb anomalies, glomus tumor, and the other tumorous lesions. In case of a glomus tumor, carotid angiography was also conducted to evaluate the cerebral collateral circulation as well as for embolization of the feeding artery before surgical removal. If the otoscopic finding was normal, and the venous or arterial origin could be differentiated by the physical maneuver, various imaging studies—TBCT, brain magnetic resonance angiography/venography (MRA/MRV), and transcranial Doppler sonography (TCD)—were performed subsequently to find the specific etiologies.

In patients with potential venous-origin vascular tinnitus, TBCT was the first choice to detect venous anomalies. Once jugular bulb anomaly was diagnosed, no further imaging study was not performed. Otherwise, MRV was additionally obtained to exclude the other vascular or intracranial pathology. In cases of normal imaging, we consulted a neurologist to exclude benign intracranial hypertension (BIH). When BIH was excluded by the neurologist, we finally diagnosed the tinnitus as venous hum.

In patients with potential arterial-origin vascular tinnitus, TCD was the first choice along with MRA or brain CT angiography. Atherosclerotic carotid artery disease (ACAD), dural arteriovenous fistula (AVF), and intracranial aneurysm could be found through these tests. In cases of normal imaging, we diagnosed the tinnitus as idiopathic. The flow chart is presented in Fig. 1. We provided the patients with appropriate treatment according to the specific etiology and tinnitus severity. The treatment options included surgical intervention, tinnitus retraining therapy (TRT), reassurance, and medications. The outcomes were assessed using patient’s subjective reports at the follow-up interviews.

### RESULTS

Of the total 57 patients (10 males and 47 females; age range, 18 to 85 years; mean age, 48.9 years) included, high jugular bulb was the most common cause (27 patients) of vascular tinnitus followed by venous hum (10 patients), dural AVF (5 patients), ACAD (4 patients), hypertension (4 patients), and intracranial aneurysm (3 patients) (Table 1). The mean duration was 25.9 months. Tinnitus was unilateral in 55 patients, and bilateral in two. The left ear was affected in 23 patients and the right in 32 patients. A female predominance was clearly observed in vascular tinnitus. Interestingly, there was no male patient who had venous hum or hypertension-related vascular tinnitus in our case series. High jugular bulb involved the right ear three times more frequently than the left.

The mean PTA of all patients was 18.8±12.2 dB (mean±standard deviation). In only one patient with high jugular bulb, air-bone gap of 30 dB was found in the affected ear. In tinnitogram, the mean tinnitus loudness was 31.0±19.7 dB (range, 6 to 90 dB); the mean pitch, 1,883±3,108 Hz (range, 125 to 8,000 Hz). Although the mean pitch was in the high frequency range, the low pitch was predominant except 1,000–3,000 Hz: 125, 250, 500, 4,000, 6,000, and 8,000 Hz was reported by 51.7%, 10.4%, 13.8%, 3.4%, 3.4%, and 17.2% of patients, respectively. Low-frequency hearing loss was shown in 38%/50%/60%/25%/75%

### Table 1. Relative frequency of the etiologies of vascular tinnitus

| Etiology                                           | No. of cases |
|---------------------------------------------------|--------------|
| High jugular bulb                                 | 27           |
| Venous hum                                        | 10           |
| Dural arteriovenous fistula                       | 5            |
| Hypertension                                      | 4            |
| Atherosclerotic carotid artery disease             | 4            |
| Intracranial aneurysm                             | 3            |
| Temporal cavernous hemangioma                     | 1            |
| Aberrant carotid artery                           | 1            |
| Glomus tympanicum                                | 1            |
| Benign intracranial hypertension                   | 1            |
| Total                                             | 57           |

### Table 2. Demographic data and tinnitus severity scores in patients with 5 major etiologies of vascular tinnitus

| Etiology               | Age (year) | Sex (M:F) | Duration (month) | Affected side (L:R:B) | LFHL    | AW     | LD     | AN     | EOL    | THI    |
|------------------------|------------|-----------|-----------------|-----------------------|---------|--------|--------|--------|--------|--------|
| High jugular bulb (n=26) | 46.7±16.6  | 6.20      | 36.1±55.2       | 6.200                 | 10(38)  | 55.4±32.0     | 4.9±2.6 | 5.2±3.1 | 4.8±2.8 | 36.7±22.5 |
| Venous hum (n=10)       | 40.7±16.5  | 0.10      | 13.0±8.4        | 6.3±1                 | 5(50)   | 70.0±27.7     | 5.6±2.3 | 6.4±2.6 | 4.9±3.4 | 40.0±9.2  |
| DAVF (n=5)              | 53.2±12.8  | 2.3       | 29.3±51.6       | 4.10                  | 3(60)   | 73.3±23.1     | 5.0±2.6 | 5.0±0.0 | 4.0±1.0 | 25.0±1.4  |
| ACAD (n=4)              | 51.7±19.2  | 1.3       | 5.0±5.5         | 1.30                  | 1(25)   | 50.0±50.0     | 3.7±1.2 | 6.3±2.3 | 5.7±1.2 | 51.3±25.3 |
| Hypertension (n=4)      | 73.7±9.9   | 0.4       | 30.0±35.5       | 3.10                  | 3(75)   | 63.3±30.6     | 6.0±3.5 | 4.3±1.5 | 4.3±1.5 | 19.0±19.8 |

Values are presented as mean±SD or number (%). L, left; R, right; B, both; LFHL, low-frequency hearing loss; AW, awareness (percent of time aware of tinnitus during the waking hours); LD/AN/ EOL, 10-point visual analogue scale for loudness/annoyance/effect on daily life; THI, tinnitus handicap inventory; DAVF, dural arteriovenous fistula; ACAD, atherosclerotic carotid artery disease.
Table 3. Treatment options and outcomes for vascular tinnitus

| Diagnosis (n) | Treatment (n) | Response (n) |
|--------------|---------------|--------------|
| High jugular bulb (27) | TRT with medication (17) | Gradual, incomplete (15) |
| | TRT with sound generator (1) | Gradual, incomplete (1) |
| | Reassurance with medication (7) | Gradual, incomplete (3) |
| | Surgical ligation (2) | Gradual, complete (1) |
| Venous hum (10) | TRT with medication (10) | Gradual, incomplete (8) |
| | Follow-up loss (1) | |
| DAVF & aneurysm (8) | Embolization (6) | Prompt, complete (6) |
| | TRT with medication (2) | Gradual, incomplete (2) |
| Hypertension (4) | Antihypertensives (4) | Gradual, complete (4) |
| ACAD (4) | Angioplasty (1) | Prompt, complete (1) |
| | TRT with medication (3) | Gradual, incomplete (3) |
| Hemangioma (1) | TRT with medication (1) | Gradual, incomplete (1) |
| Aberrant carotid artery (1) | TRT with medication (1) | Gradual, incomplete (1) |
| Glomus tympanicum (1) | Surgery (1) | Prompt, complete (1) |
| BIH (1) | TRT with medication (1) | Gradual, incomplete (1) |

TRT, tinnitus retraining therapy; DAVF, dural arteriovenous fistula; ACAD, atherosclerotic carotid artery disease; BIH, benign intracranial hypertension.

DISCUSSION

In this study, we have summarized the data of our large case series of vascular tinnitus to provide the practitioners with some helpful information about the diagnosis and treatment of this challenging symptom in otology clinics. It is suggested that imaging studies are particularly important in the diagnostic process of vascular tinnitus. When venous origin was suspected, TBCT should be checked first to rule out the most common jugular bulb anomalies. If arterial origin was suspected, TCD along with MRA should be obtained. TCD was useful to evaluate the functional degree of intracranial vascular stenosis, especially ACAD. In addition, consultation with neurologists or neurosurgeons is frequently critical in vascular tinnitus. Because tinnitus patients who first visit otolaryngology clinics usually do not have identifiable neurologic signs and symptoms, high index of suspicion is needed. For example, before a neurologic consultation for BIH, other presenting symptoms such as headache, dizziness, and visual disturbances also require careful attention.

In our case series, high jugular bulb was the most common cause of vascular tinnitus, and the second, venous hum. Arterial causes...
were less common. Glomus tympanicum, intracranial cavernous hemangioma, aberrant carotid artery, and BIH were identified in only one case, each. Sismanis reported a 15-year study of 145 patients with vascular tinnitus [3]. In his study, BIH was the most common diagnosis (56 patients) followed by ACAD (24 patients). It has been reported that the incidence of BIH is increased in women who are 10% or more over ideal weight [4], and varies according to the prevalence of obesity in the respective region [5]. In our study, the incidence of BIH was very low; only one patient among 54 patients. These different incidences may be attributed to the different patient populations. Furthermore, the chief complaint of our patients is almost always tinnitus, not neurologic symptoms. If we collaborate with neurologists for the study, more intriguing data would be available on vascular tinnitus caused by intracranial vascular abnormalities.

High jugular bulb has been defined as a jugular bulb extending above the inferior bony annulus [6]. The incidence of high jugular bulb has been reported from 3.5% to 22.6% [7]. High jugular bulb is usually asymptomatic, but when symptoms are present, tinnitus is known to be a common complaint [8]. The initial treatment method for high jugular bulb could be TRT, medication, and sound therapy [9]. In cases of the failure of initial treatment, surgical therapy can be considered. Surgical treatment options for high jugular bulb include jugular vein ligation, transcatheter endovascular coil embolization, transvenous stent-assisted coil embolization, surgical lowering through subfacial and infralabyrinthine approaches, and surgical covering and reinforcement using fascia, perichondrium, or autologous cartilage [10-14]. However, surgical outcomes of these methods were not sufficient to be considered as successful, and have not been evaluated properly. Moreover, potential complications of the surgery are intracranial hypertension, facial nerve injury, and conductive hearing loss. Thus, the surgery is not recommended as an initial therapy for tinnitus patients caused by high jugular bulb [9]. The authors initially provided our patients with TRT and medications: alprazolam (0.25 mg) and ginkgo biloba extract (80 mg) daily for 3 months. In only two cases among the 27 high jugular bulb patients, failure of the conservative treatment led to IJV ligation. One showed complete relief of tinnitus, and the other showed partial improvement.

Venous hum can be defined as an idiopathic vascular tinnitus of venous origin [15-17]. It is common in patients between 20 and 40 years with a marked female preponderance. A turbulent blood flow produced in the IJV is supposed to be the cause of venous hum [18]. Diagnosis of venous hum can only be made after the exclusion of the other disorders. In our clinic, all patients with this second most common cause of vascular tinnitus were treated with TRT and medication, because surgical treatment of venous hum has not been popular to date. The treatment responses were good in 80% of the patients, although no one showed complete cure. Dural AVF and aneurysm are relatively common intracranial vascular abnormalities. Tinnitus can be one of the most frequent symptoms in dural AVF. Cerebral venous thrombosis has been proposed to be the major pathogenic factor in the formation of dural AVF. As the thrombosed segment recanalizes, regeneration of dural arteries results in artery-to-sinus anastomosis. In a recent report, the overall mortality rate was 4.8% within the first 12 months [19]. An aneurysm is a focal dilatation of an arterial vessel caused by high blood pressure, shearing forces, turbulence, and morphological or biological characteristics within the vessel wall [20-22]. Overall prevalence was estimated to be 3.2% in a population with an mean age of 50 years [23]. Aneurysm of the internal carotid artery, particularly in the petrous portion, can manifest with pulsatile tinnitus alone [24]. Because the lesion is life-threatening when ruptured, interventions such as surgical clipping or endovascular coiling should be conducted if indicated. In our cases, all tinnitus patients with aneurysms were treated with embolization, and the pulsatile tinnitus was cured immediately after the treatment.

In conclusion, vascular tinnitus could be alleviated or cured in most of the patients after the individualized treatment according to the causes. Many patients with vascular tinnitus have treatable underlying etiologies. We suggest that establishing the correct diagnosis through a regular diagnostic protocol and early selected treatment will help patients to be relieved from their tinnitus effectively.

CONFLICT OF INTEREST
No potential conflict of interest relevant to this article was reported.

REFERENCES
1. Struiffer JL, Tyler RS. Characterization of tinnitus by tinnitus patients. J Speech Hear Disord. 1990 Aug;55(3):439-33.
2. Liyanage SH, Singh A, Savundra P, Kalan A. Pulsatile tinnitus. J Laryngol Otol. 2006 Feb;120(2):93-7.
3. Sismanis A. Pulsatile tinnitus: A 15-year experience. Am J Otol. 1998 Jul;19:472-7.
4. Durcan FJ, Corbett JJ, Wall M. The incidence of pseudotumour cerebri: Population studies in Iowa and Louisiana. Arch Neurol. 1988 Aug; 45(8):875-7.
5. Radhakrishnan K, Thacker AK, Bohlaga NH, Maloo JC, Gerryo SE. Epidemiology of idiopathic intracranial hypertension: a prospective and case-control study. J Neurol Sci. 1993 May;116(1):18-28.
6. Overton SB, Ritter FN. A high placed jugular bulb in the middle ear: a clinical and temporal bone study. Laryngoscope. 1973 Dec;83(12): 1986-91.
7. Subotic R. The high position of jugular bulb. Acta Otolaryngol. 1979 Mar-Apr;87(3-4):340-4.
8. Weiss RL, Zahltz G, Goldofsky E. Parnes H, Shikowitz MJ. High jugular bulb and conductive hearing loss. Laryngoscope. 1997 Mar; 107(3):321-7.
9. Yoo HJ, Park SN, Kim DK, Park KH, Kim MJ, Kim JE et al. Incidence and clinical characteristics of patients with tinnitus according to diag-

Bae SC et al. Diagnosis and Treatment Outcomes of Vascular Tinnitus 11
nostic classification. Korean J Otorhinolaryngol-Head Neck Surg. 2011 Jun;54(6):392-8.
10. Huang BR, Wang CH, Young YH. Dehiscent high jugular bulb: a pitfall in middle ear surgery. Oto Neurotol. 2006 Oct;27(7):923-7.
11. Buckwalter JA, Sasaki CT, Virapone C. Pulsatile tinnitus arising from jugular megabulb deformity: a treatment rationale. Laryngoscope. 1983 Dec;93(12):1534-9.
12. Yoon BN, Lee TH, Kong SK. Management of high jugular bulb with tinnitus: transvenous stent-assisted coil embolization. Otolaryngol Head Neck Surg. 2008 Nov;139(5):7401.
13. Couloigner V, Grayeli AB, Bouccara D, Julien N, Sterkers O. Surgical treatment of the high jugular bulb in patients with Meniere’s disease and pulsatile tinnitus. Eur Arch Otorhinolaryngol. 1999;256(5):224-9.
14. Kondoh K, Kitahara T, Mishiro Y, Okumura S, Kubo T. Management of hemorrhagic high jugular bulb with adhesive otitis media in an only hearing ear: transcatheter endovascular embolization using detachable coils. Ann Otol Rhinol Laryngol. 2004 Dec;113(12):975-9.
15. Arenberg IK, McCreary HS. Objective tinnitus aurium and dural arteriovenous malformations of the posterior fossa. Ann Otol Rhinol Laryngol. 1971 Feb;80(1):111-20.
16. Chandler JR. Diagnosis and cure of venous hum tinnitus. Laryngoscope. 1983 Jul;93(7):892-5.
17. Engstrom H, Graf W. On objective tinnitus and its recording. Acta Otolaryngol Suppl. 1950;95:127-37.
18. Hentzer E. Objective tinnitus of vascular type: A follow-up study. Acta Otolaryngol. 1968 Oct;66(4):273-81.
19. Piippo A, Laakso A, Seppa K, Rinne J, Jaaskelainen JE, Hernesniemi J, et al. Early and long-term excess mortality in 227 patients with intracranial dural arteriovenous fistulas. J Neurosurg. 2013 Jul;119(1):164-71.
20. Hashimoto T, Meng H, Young WL. Intracranial aneurysms: Links among inflammation, hemodynamics and vascular remodeling. Neurol Res. 2006 Jun;28(4):372-80.
21. Mizutani T, Kojima H, Asamoto S, Miki Y. Pathological mechanism and three-dimensional structure of cerebral dissecting aneurysms. J Neurosurg. 2001 May;94(5):712-7.
22. Tulamo R, Frosen J, Hernesniemi J, Niemela M. Inflammatory changes in the aneurysm wall: a review. J Neurointerv Surg. 2010 Jun;2(2):120-30.
23. Juvela S. Prevalence of risk factors for intracranial aneurysms. Lancet Neurol. 2011 Jul;10(7):595-7.
24. Kim DK, Shin YS, Lee JH, Park SN. Pulsatile tinnitus as the sole manifestation of an internal carotid artery aneurysm successfully treated by coil embolization. Clin Exp Otorhinolaryngol. 2012 Sep;5(3):170-2.