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Journal

Journal of NeuroInterventional Surgery, 16(5)

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Publication Date

2024-04-23

DOI

10.1136/jnis-2023-020440

Peer reviewed

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Author manuscript

J Neurointerv Surg. Author manuscript; available in PMC 2024 April 25.

Published in final edited form as:

J Neurointerv Surg. ; 16(5): 516–521. doi:10.1136/jnis-2023-020440.

Clinical evaluation of pulsatile tinnitus: History and physical exam techniques to predict vascular etiology

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Abstract

Background—Pulsatile tinnitus (PT) may be due to a spectrum of cerebrovascular etiologies ranging from benign venous turbulence to life-threatening dural arteriovenous fistulas. A focused clinical history and physical examination provide clues to the ultimate diagnosis; however, the predictive accuracy of these features in determining PT etiology remains uncertain.

Methods—Patients with clinical PT evaluation and digital subtraction angiography (DSA) were included. The final etiology of PT after DSA was categorized as “shunting,” “venous,” “arterial,” or “non-vascular.” Clinical variables were compared between etiologies using multivariate logistic regression and performance at predicting PT etiology was determined by area under the receiver operating curve (AUROC).

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Contributions:

Cummins: Conceptualization, Data Collection, Analysis, Drafting Original Manuscript
Caton: Conceptualization, Data Collection, Analysis, Drafting Original Manuscript
Hemphill: Data Collection, Revising Original Manuscript
Lamboy: Data Collection, Revising Original Manuscript
Tu-Chan: Conceptualization, Oversight, Revising Original Manuscript
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Competing Interests

Amans: Board member: Mind Rhythm; Consultancy: Stryker, Neurovascular, Covidien, MicroVention, Comments: Pipeline proctor for Covidien and PHIL DSMB for MicroVention; Patents (planned, pending or issued): Cerebral venous sinus stent, Comments: provisional patent serial number 62/984,549. Editorial board: Journal of NeuroInterventional Surgery.
Narsinh: Consultancy: Stryker Neurovascular, Imperative Care
All other authors: No relevant competing interests.

Ethics approval statement

This work was approved by the University of California San Francisco Institutional Review Board (IRB #10–00936) with need for informed consent waived for this retrospective study.

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Results—There were 164 patients included. On multivariate analysis, patient-reported high pitch PT (RR = 33.81; 95%CI = 3.81–882.80) compared with exclusively low pitch PT and presence of a bruit on physical exam (RR = 9.95; 95%CI = 2.04–62.08; p = 0.007) were associated with shunting PT. Hearing loss was associated with lower risk of shunting PT (RR= 0.16; 95%CI = 0.03–0.79; p = 0.029). Alleviation of PT with ipsilateral lateral neck pressure was associated with higher risk of venous PT (RR = 5.24; 95%CI = 1.62–21.01; p = 0.010). An AUROC of 0.882 was achieved for predicting the presence or absence of a shunt and 0.751 for venous PT.

Conclusion—In patients with pulsatile tinnitus, clinical history and physical examination can achieve high performance at detecting a shunting lesion. Potentially treatable venous etiologies may also be suggested by relief with neck compression.

Introduction

Pulsatile tinnitus (PT), the auditory sensation of a pulse-synchronous sound, may be due to etiologies ranging from benign to life-threatening cerebrovascular lesions.[1] While PT itself can adversely affect mental health,[2,3] ruling out dangerous vascular lesions is the major clinical concern in evaluation of PT.[4,5] Shunting lesions such as dural arteriovenous fistulas (dAVFs) are the most concerning cause of PT, as these lesions can carry risk of hemorrhage.[6,7] Recent estimates suggest 20% of patients with cerebrovascular PT have a dangerous arteriovenous shunting source lesion.[5,8] Early recognition of shunting lesions presenting with PT can lead to effective treatment and avoidance of devastating hemorrhage.[9–11]

Non-invasive workup with MRI/MRA can provide helpful information regarding possible cerebrovascular lesions presenting with PT.[12,13] In patients in whom there is high concern for a shunting lesion or in those with debilitating symptoms, neuroangiography provides the gold standard for diagnosis and potential treatment.[1,4,5,9] Still, clinical history and physical examination provide the basis for pursuing non-invasive imaging and angiographic evaluation, when deemed appropriate. There are anecdotal reports of specific history or physical exam findings suggesting the underlying cause: For instance, a head or neck bruit on physical examination or focal neurological deficit may indicate a DAVF.[14,15] However, there has yet to be an evaluation of how well clinical history and physical examination factors can predict the cause of PT confirmed with digital subtraction neuroangiography. This study explores clinical predictors of PT etiology, including history and physical examination, and their performance at predicting the cause of PT in patients evaluated in a multidisciplinary high volume PT clinic.

Methods

Data Collection

All patients seen between 2016 and 2021 for the primary complaint of PT at the UCSF Pulsatile Tinnitus Clinic were screened and included if they underwent a previously described vascular evaluation of PT including digital subtraction angiography.[5] Indications to undergo DSA for PT evaluation included clinical findings (i.e., an audible head or neck bruit on auscultation) or non-invasive imaging suggestive of a life-threatening

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cerebrovascular etiology; or severe impact of PT on quality of life. Therefore, patients with minimal quality of life impact from PT and with low pre-test probability for dangerous cerebrovascular conditions did not undergo DSA. Patients with PT seen for a different primary complaint (including neurological deficit or hemorrhage) were excluded. Data was collected retrospectively. Demographic information and clinical data collected included patient age, gender, body mass index (BMI), hearing loss (evaluated by an audiologist), history of a mental health diagnosis (anxiety, depression, obsessive compulsive disorder, or bipolar disorder), history of prior head or neck trauma, and history of head or neck surgery. Characterization of PT included symptom duration prior to evaluation, presence of any preceding event immediately prior to PT onset, laterality (bilateral vs unilateral), subjective pitch of the sound (self-reported by the patient but usually guided by the examining physician with options of sounds created by the examiner), and alleviating or exacerbating factors. Physical exam components included evaluation for a head or neck bruit, change with occipital compression, change with venous compression, change with head turn (specifically detailed as turn your head and try to put your chin on your right or left shoulder), and change with neck flexion as well as neck extension. Assessment for an audible head or neck bruit on physical examination was performed over the carotid arteries, temporal region (pre-auricular), mastoid region (post-auricular), and orbits. Essentially, we trace the anticipated course of the dural venous sinuses with our stethoscopes with auscultation of the head. Angiographic evaluation included selective cervicocerebral arteriography of both internal and external carotid arteries, both subclavian arteries with a blood pressure cuff concurrently inflated on the upper arm and the cameras positioned over the neck, both vertebral arteries with the camera positioned over the neck as well as separately positioned over the head. In addition venography, venous manometry, and/or balloon test occlusion (BTO) was also used to isolate the underlying vascular cause, and to definitely exclude vascular causes per the previously described protocol.[5] The final etiology of PT after vascular evaluation was categorized as “arterial,” “shunting,” “venous,” or “non-vascular” as reported previously.[5] Final PT category was determined by the neurointerventional team in the context of all objective and subjective information following DSA. Principle data to determine a “venous” or “arterial” PT etiology included subjective changes in PT with BTO proximal to possible venous or arterial lesions (with the patient asked to assess PT severity while blinded to trials of balloon inflation/deflation). Supporting data such as elevated venous pressure gradients on manometry were used to determine venous sinus stenosis as the cause of PT. Venous PT included venous stenosis, diverticula, prominent jugular veins, and abnormal condylar/emissary veins. Arterial PT included arterial stenoses or aberrant arteries with PT clearly altered with proximal arterial BTO.

Statistical analysis

All statistics were performed in R version 4.2.0. Clinical variables were compared between PT etiologies using logistic regression. Each etiology was independently compared to all other etiologies to determine significant variables associated with each PT etiology. Significance values were determined using the Wald test with a cutoff of $p < 0.05$ used for significance. All variables significant on univariate analysis were included in multivariate analysis. Variables significant on multivariate analysis were used to determine a receiver operating curve (ROC) to predict each PT etiology, and the area under the ROC (AUROC)

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was reported for each etiology to determine performance at distinguishing each PT etiology from all others.

Results

Demographics and Clinical Characteristics

There were 164 patients (29.7%) included of the 552 patients seen in the PT clinic over the study period. Final PT etiologies including 34 (20.7%) shunting lesions, 79 (48.2%) venous, 11 (6.7%) arterial, and 40 (24.4%) non-vascular. Of those with a shunting lesion, 11 (32.4%) were considered benign/Borden Type 1, while 23 (67.6%) were considered Borden Type II/III. Of those with a venous cause, the majority (77%, n=61) were from venous sinus stenosis (including idiopathic intracranial hypertension). Demographics and clinical features by PT etiology are given in Table 1. Across all patients, median age was 56 (range: 25 – 89) years and 111 (67.7%) were female. Duration of PT at evaluation was a median 365.0 days (11–7665), and laterality of PT was bilateral in 23 (14.0%), right-sided in 66 (20.2%), and left-sided in 75 (45.7%). Subjective PT pitch was assessed for 122 patients, in which 71 (58.2%) had a high pitch component and 51 (41.8%) were exclusively low pitch. Seventy (42.7%) patients presented with hearing loss, 69 (42.1%) had a history of a mental health diagnosis, and 36 (22.0%) a previous history of trauma or surgery to the head or neck. A preceding event or factor at the time of PT onset was recounted by 49 (29.9%) patients, 12 (7.3%) following a flight, 11 (6.7%) following head/neck trauma or surgery, 8 (4.9%) following a sinus infection, 4 (2.4%) during pregnancy, 4 (2.4%) following a dental procedure, 2 (1.2%) following increased stress, and 2 (1.2%) following swimming. Alleviating factors of PT included changes of position in 67 (40.9%), ipsilateral lateral neck pressure in 28 (17.0%), pressure behind the ear in 12 (7.3%), Valsalva maneuvers in 10 (6.1%), pressure on the jaw in 8 (4.9%), and occipital pressure in 4 (2.4%). Exacerbating factors included positional changes in 109 (66.5%), lying down in 66 (40.2%), exercise in 60 (36.6%), Valsalva in 35 (21.3%), and standing up in 7 (4.3%). On physical examination, 46 (30.3%) patients had a bruit, 25 (15.9%) had alleviation with occipital compression, 23 (15.2%) had exacerbation with ipsilateral head turn, 23 (15.2%) had exacerbation with ipsilateral venous compression, and 13 (8.3%) had exacerbation with neck flexion or extension.

Associations with PT Etiology and Predictive Performance

All variables significant on univariate analysis for at least one etiology of PT are shown in Table 2. On univariate analysis, variables associated with increased risk of shunting PT included alleviation with pressure over the occipital artery (relative risk (RR) = 13.55; 95% confidence interval (CI) = 1.70 – 279.37; p = 0.026), high pitch PT (RR = 10.29; 95% CI = 2.81 – 66.52; p = 0.002), and presence of a bruit (RR = 9.39; 95% CI = 3.96 – 23.93; p = 8.57E-7). Variables associated with lower risk of shunting PT included greater body mass index (BMI) (RR = 0.92; 95% CI = 0.84 – 0.99; p = 0.048), presence of hearing loss (RR = 0.25; 95% CI = 0.09 – 0.60; p = 0.004), and alleviation with ipsilateral lateral neck venous pressure (RR = 0.12; 95% CI = 0.01 – 0.58; p = 0.038). On multivariate analysis, high pitch PT (RR = 33.81; 95% CI = 3.81 – 882.80; p = 0.007) and presence of a bruit (RR = 9.95; 95% CI = 2.04 – 62.08; p = 0.007) were associated with increased risk of shunting PT, while

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hearing loss was associated with lower risk of shunting PT (RR = 0.16; 95% CI = 0.03 – 0.79; p = 0.029).

On univariate analysis, variables associated with increased risk of venous PT included greater BMI (RR = 1.08; 95% CI = 1.02 – 1.15; p = 0.015), alleviation with ipsilateral lateral neck pressure (RR = 8.84; 95% CI = 3.20 – 31.32; p = 1.24E-4), and exacerbation with Valsalva (RR = 2.99; 95% CI = 1.38 – 6.84; p = 0.007). Alleviation of PT with lateral neck compression occurred more commonly with venous stenosis (44.3%, 27/61) than with other jugular (18.8%, 3/16) or condylar (13.3%, 2/15) venous sources of PT. Variables associated with lower risk of venous PT included increased age (RR = 0.96; 95% CI = 0.94 – 0.98; p = 6.76E-4) and a bruit on physical examination (RR = 0.38; 95% CI = 0.18 – 0.77; p = 0.009). On multivariate analysis, alleviation with ipsilateral lateral neck pressure (RR = 5.24; 95% CI = 1.62 – 21.01; p = 0.010) was associated with increased risk of venous PT. Location of an audible bruit on physical examination was retroauricular in 34 patients (50.0%, n=17 with a shunt), temporal in 6 patients (66.7%, n=4 with a shunt), carotid in 5 patients (60%, n=3 with a shunt), and peri-orbital in 3 patients (100.0% with a shunt). Increased age (RR = 0.97; 95% CI = 0.94 – 0.99; p = 0.022) and a bruit on physical exam (RR = 0.35; 95% CI = 0.14 – 0.81; p = 0.017) were associated with lower risk of venous PT.

On univariate analysis, only increased age was associated with risk of arterial PT (RR = 1.05; 95% CI = 1.01 – 1.12).

Using only variables significant on multivariate analysis, the area under the receiver operating curve (AUROC) using logistic regression for distinguishing each etiology respectively was 0.882 for shunting PT and 0.751 for venous PT (Figure 1). Given the strong performance of the three variables used to predict shunting PT (pitch, hearing loss, and presence of a bruit) in this cohort, a decision tree was visualized to guide likelihood of a shunt based upon these variables (Figure 2). Of patients with a high pitch component to their PT, no hearing loss, and a bruit on physical exam, 92.9% (13/14) had a shunt responsible for their PT. Of patients with exclusively low pitch PT and hearing loss present, 0% (0/16) had a shunt, and of patients with exclusively low pitch PT, no hearing loss, and no bruit on physical exam, 0% (0/20) had a shunt. Patients with other combinations of these variables had more mixed likelihood of having a shunt.

Discussion

Predicting Shunting Pulsatile Tinnitus

Triage of patients with pulsatile tinnitus often presents a challenge to providers. In 34 patients with the primary complaint of PT who were found to have a shunting lesion, 23 (67.6%) had a Borden Type II/III lesion with notable risk of hemorrhage. Determining the subset of patients with PT who may have an arteriovenous shunting cause is thus of great interest. The data presented in this work indicate that shunting lesions may be either highly suggested or improbable based on clinical history and physical examination alone. This affirms the importance of the initial clinical assessment of the patient in framing the pre-imaging probabilistic reasoning of the clinician. While neuroimaging is an essential

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component of the workup of PT, this study provides the first systematic investigation of pre-imaging clinical factors in predicting the underlying etiology of the patient's symptoms.

The finding of a bruit on auscultation, which may be referred to as "objective pulsatile tinnitus," as evidence of an arteriovenous shunt is well-described. The characterization of an arteriovenous fistula causing PT with an associated bruit on auscultation dates back to a classic report by *Fincher* over seven decades ago,[16] and bruit has been documented in over 80% of dAVFs in more recent work.[17] Even with the development of advanced imaging protocols, this component of the physical exam in patients with PT remains crucial. However, presence of a bruit was not exclusive to shunting PT, with 22.2% of venous, 33.3% of arterial, and 13.2% of patients with non-vascular PT found to have a bruit (Table 1). Presence of a bruit in the latter population with non-vascular PT could suggest a baseline error in auscultation, even in experienced clinicians. Presence of an objective bruit is also well documented in pure arterial causes of PT,[18] and has been previously reported with isolated venous stenosis that may cause venous PT.[19] More pressing, only 67.6% of patients with a shunting lesion were found to have a bruit. The precise location of bruit may inform likelihood of a shunting lesion; 100% (3/3) of peri-orbital bruits were associated with a shunt, compared with 50% (17/34) of retroaricular bruits. The presented results underscore the need for a comprehensive auscultation for bruits across the head and neck, including over the carotid, temporal, retroaricular, and peri-orbital regions.Taken together, this suggests that, in isolation, the discriminatory value of a bruit is insufficient. The current data reveal that two additional factors, PT pitch and lack of hearing loss, can substantively improve our ability to predict shunting as the cause of PT.

Previous literature has suggested *low pitch* PT commonly occurs in venous PT whereas *high pitch* supports an arterial PT,[1] although no comparative evidence between etiologies has documented this observation. Therefore, the present finding that PT pitch can differentiate etiology supports the anecdotal observation of experts who see many cases of PT. Like a bruit, it is important to note that many patients without a shunt will have high pitch PT, including 63.3% with non-vascular PT, which has been previously documented.[20] In addition to bruit on auscultation and high pitch quality, preservation of hearing increased the likelihood of shunting PT (relative risk of a shunt with hearing loss = 0.16; 95% CI = 0.03 – 0.79; p = 0.029). Compared to shunting PT (17.6%), higher rates of hearing loss were seen across venous (41.8%), arterial (45.5%), and non-vascular (60.0%) PT. This finding thus underscores the value of audiometric evaluation for patients with pulsatile tinnitus, which may also help define possible otologic, non-vascular causes of PT.[21]

Predicting Venous and Arterial Pulsatile Tinnitus

Compared to shunting PT, there was overall weaker predictive performance of potentially treatable venous causes of PT (AUC = 0.751). Alleviation of PT with patient-applied pressure on ipsilateral lateral neck was strongly associated with venous PT (RR = 5.24; 95% CI = 1.62 – 21.01; p = 0.010). We believe that, in the case of venous PT, this lateral neck pressure reflects compression of the jugular vein, and resultant occlusion of blood flow through the ipsilateral venous system causing PT. This clinical test mirrors the angiographic practice of venous balloon test occlusion of the venous system causing PT, and is a useful

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clinical tool to determine patients who may possess a treatable venous etiology causing PT. Notably, alleviation with ipsilateral jugular compression may not occur with all patients with venous PT, and appeared more commonly with PT due to venous stenosis (>40%) than with other jugular or condylar venous sources (<20%). *Raz et al* notably found a much higher sensitivity (89%) of alleviation with ipsilateral jugular compression for venous sinus stenosis. However, only a small subset of patients in this study (11%) underwent angiography,[22] which provides the more definitive diagnosis compared to non-invasive imaging. Compared to the findings by *Raz et al* using primarily non-invasive imaging, our threshold for attributing venous sinus stenosis as the cause of PT included more rigorous criteria including supporting manometry and BTO data. We believe the relationship of physical exam findings to final angiographic diagnosis may be of greater benefit to patient triage. *Tao et al* also found a higher rate of alleviation with ipsilateral neck compression (60.6%) in patients with PT who underwent DSA, but did not report on the predictive value for venous PT or venous stenosis.[23] Our findings suggest that ipsilateral neck compression can suggest venous sinus stenosis, but may not be present in all patients with venous sinus stenosis causing PT.

Arterial PT was the most difficult etiology to predict, with increased age as the only factor associated with arterial PT (RR = 1.05; 95% CI = 1.01 – 1.12; p = 0.044). This was likely due to the small number of arterial PT cases (n=11). As dangerous arterial causes of PT such as critical cervical stenosis or dissection may present with PT, Fortunately, *pure* arterial PT can typically be detected with basic non-invasive neuroimaging (e.g. CTA, MRA).

Many commonly reported and assessed aspects of PT were not significantly associated with any particular PT etiology. This included history of a mental health diagnosis, particular preceding events to PT onset, and common alleviating and exacerbating factors such as positional changes and exercise. Although these features did not alter the clinical prediction models, these components could be helpful to understand the psychosocial and cognitive effects of PT in individual patients and help guide treatment, such as through treatment of comorbid mental health conditions.

Limitations

The principle limitation of this study is its retrospective design which includes a highly selected sample of patients in whom the pre-test probability of a cerebrovascular etiology was high enough to warrant DSA. This study did not incorporate information on MR or CT imaging that may have aided triage of patients to DSA. However, the use of DSA neuroangiographic assessment as a reference standard affords a higher confidence in the final rendered diagnosis of PT etiology. Although a subset of patients had PT pitch assessed with an external frequency generator, this was not available for most patients. More standardized, objective evaluation of PT pitch may thus serve as an improved marker for PT etiology.[24] Furthermore, a number of routinely assessed variables did not prove to be significant, including factors such as PT laterality or history of trauma, that still may be helpful to triaging patients with PT.

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Conclusions

Comprehensive clinical history and physical examination are fundamental tools for evaluating patients with pulsatile tinnitus. Auscultation for bruits, audiometric evaluation, and PT pitch assessment are crucial elements to predict the likelihood of a potentially-dangerous shunting lesion in patients with PT. Conversely, alleviation of PT with ipsilateral neck compression increases the likelihood of venous PT. When combined with PT-specific neuroimaging, these clinical factors can enhance the diagnosis and risk-stratification of patients who present with a chief symptom of PT.

Funding

This work was funded in part by the National Institute of Health (NIH) National Heart, Lung, and Blood Institute (NHLBI) of the NIH under the award number R56HL149124-01 and by the Office of the Assistant Secretary of Defense for Health Affairs through the Peer Reviewed Research Program under award number PR201091.

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- **What is already known on this topic** – Clinical history and physical examination may help triage patients with pulsatile tinnitus (PT) by suggesting more benign or serious cerebrovascular causes of PT. Yet, it remains unknown how well these variables perform at predicting the true cause of PT.
- **What this study adds** – In addition to presence of a bruit, lack of hearing loss and high pitch PT are important clinical components that raise the probability of a shunt in patients with PT. Alleviation with ipsilateral lateral neck compression may indicate potentially treatable, but usually lower risk, venous causes.
- **How this study might affect research, practice or policy** – Physicians seeing patients with pulsatile tinnitus should have a high index of suspicion for a potentially dangerous shunting lesion if there is bruit on physical exam, subjective high pitch, and preservation of hearing. Treatable venous causes can be suggested by alleviation with lateral neck compression. Further work is needed to determine risk factors for potentially dangerous arterial causes of PT.

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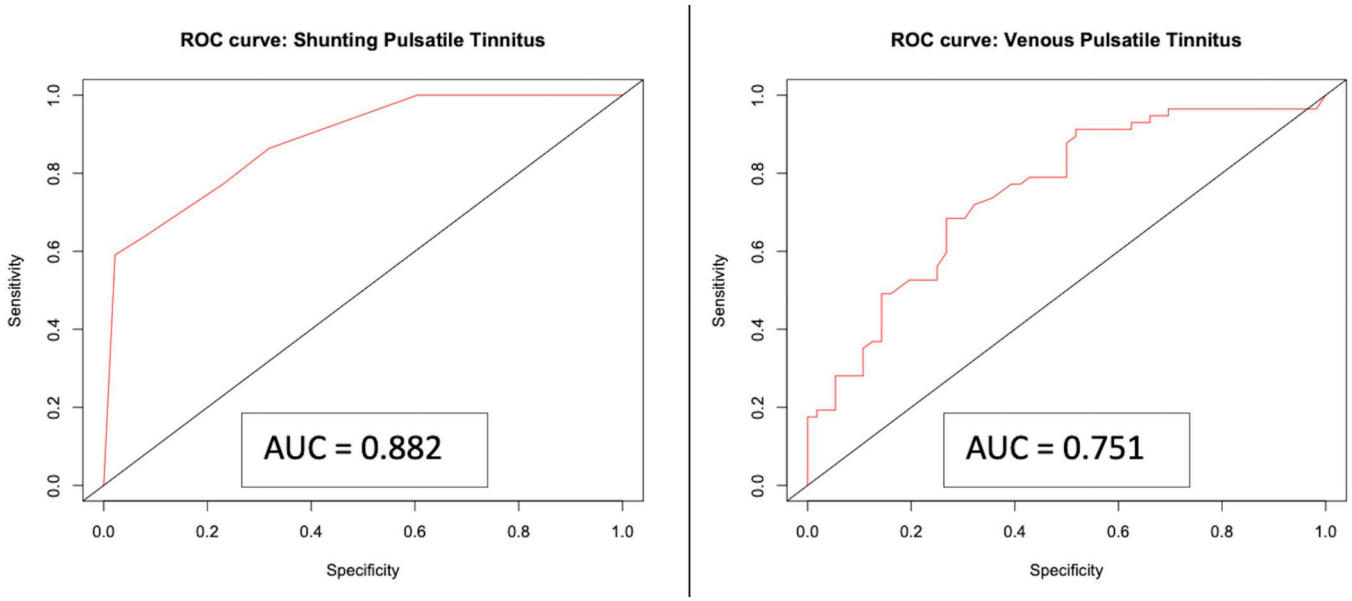

Figure 1:

Receiver operating curve (ROC) each for distinguishing pulsatile tinnitus due to **(A)** shunting and **(B)** venous etiologies determined using only variables significant on multivariate logistic regression. Area under the curve (AUC) for each ROC is reported for each: 0.912 for shunting PT and 0.786 for venous PT.

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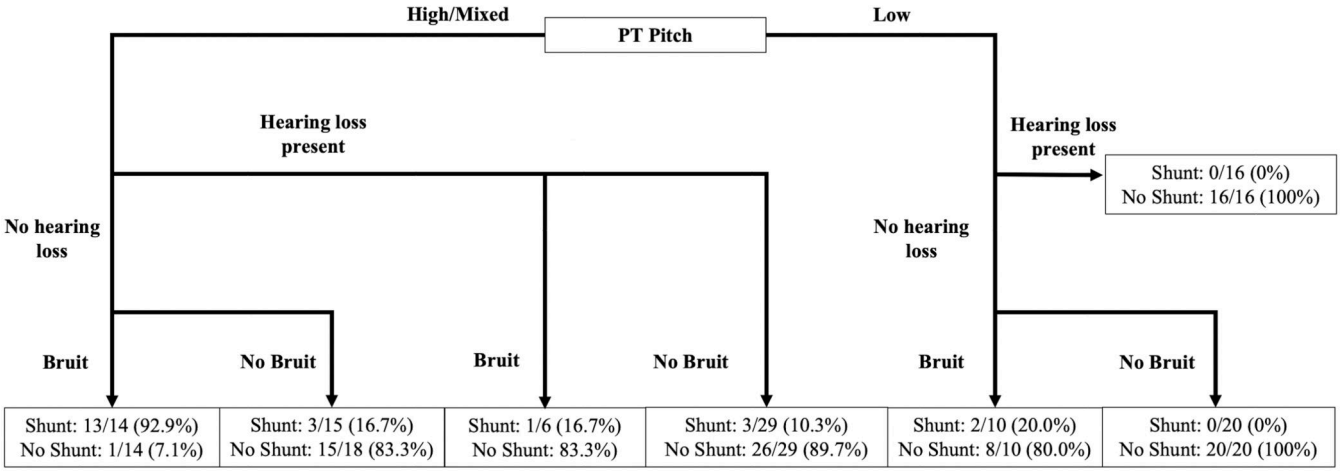

Figure 2:

A decision tree to determine presence or absence of a shunt in patients with pulsatile tinnitus (PT) based on subjective pitch, bruit on auscultation, and presence of hearing loss.

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Table 1:

Baseline Demographics and Clinical Parameters

Variable	Shunting	Venous	Arterial	Non-vascular
N (%)	34 (20.7%)	79 (48.2%)	11 (6.7%)	40 (25.6%)
PT Duration, median days (range)	165 [11 – 1095]	635 [120 – 7300]	330 [118 – 1460]	665 [60 – 7665]
Age, median years (range)	56.0 [27 – 89]	51 [25 – 77]	60 [50 – 79]	61.5 [28 – 80]
Gender, female (%)	21 (61.7%)	57 (72.2%)	7 (63.6)	27 (67.5%)
BMI, median (range)	23.8 [18.9 – 39.4]	25.8 [16.4 – 32.9]	25.8 [19.8 – 49.2]	24.5 [18.5 – 36.9]
Hearing loss, yes (%)	6 (17.6%)	33 (41.8%)	5 (45.5%)	24 (60.0%)
Mental health diagnosis, yes (%)	12 (35.3%)	34 (43.0%)	3 (27.3%)	20 (50.0%)
History of trauma or head/neck surgery	6 (17.6%)	20 (25.3%)	0	17 (42.5%)
Preceding event Flight Trauma Sinus Infection Dental Pregnancy	 2 (5.9%) 0 1 (2.9%) 1 (2.9%) 3 (8.8%)	 8 (10.1%) 4 (5.1%) 4 (5.1%) 2 (2.5%) 1 (1.3%)	 1 (9.1%) 0 0 1 (9.1%) 0	 1 (2.5%) 6 (15.0%) 3 (7.5%) 0 0
Bilateral PT, yes (%)	1 (2.9%)	15 (19.0%)	2 (18.2%)	5 (12.5%)
Alleviating factor Any positional Any neck position change Ipsilateral neck compression Jaw pressure Pressure over occipital artery Pressure behind ear Valsalva	 14 (41.2%) 8 (23.5%) 1 (2.9%) 1 (2.9%) 4 (11.8%) 2 (5.9%) 2 (5.9%)	 33 (41.7%) (29.1%) (30.4%) 4 (5.1%) 1 (1.3%) 7 (8.9%) 4 (5.1%)	 2 (18.2%) 1 (9.1%) 1 (9.1%) 0 0 0 1 (9.1%)	 19 (47.5%) 12 (30.0%) (5.0%) (7.5%) 1 (2.5%) 3 (7.5%) 3 (7.5%)
Exacerbation factors Positional Lying down Exercise Valsalva Standing	 20 (58.8%) 14 (41.2%) 11 (32.4%) 4 (11.8%) 1 (2.9%)	 57 (72.2%) 33 (41.8%) 24 (30.4%) 24 (30.4%) 5 (6.3%)	 8 (72.7%) 6 (54.5%) 6 (54.5%) 3 (27.3%) 1 (9.1%)	 25 (62.5%) 14 (35.0%) 19 (47.5%) 4 (10.0%) 0
*Subjective pitch High or mixed Exclusively low	 21 (95.5%) 1 (4.5%)	 26 (51.0%) 25 (49.0%)	 4 (50.0%) 4 (50.0%)	 19 (63.3%) 11 (36.7%)
*Physical exam Bruit present Occipital compression alleviates Ipsilateral head turn exacerbates Ipsilateral compression exacerbates Neck flexion/extension exacerbates	 23 (67.6%) 5 (15.6%) 0 0 0	 16 (22.2%) 10 (13.5%) 10 (14.7%) 10 (14.7%) 9 (12.2%)	 3 (33.3%) 2 (18.1%) 1 (9.1%) 1 (9.1%) 1 (9.1%)	 5 (13.2%) 8 (20.5%) 11 (28.2%) 11 (28.2%) 3 (7.7%)

*Parameters available for a subset of patients, which affects percentages by etiology.

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Table 2:

Significant Predictors of Pulsatile Tinnitus Etiology by Univariate and Multivariate Analysis

Clinical Variable	Significance by Etiology	
	Univariate RR [95% CI] (p-value)	**Multivariate RR [95% CI] (p-value)**
Shunting PT		
PT Duration	0.997 [0.995 – 0.999] (0.002)	0.997 [0.993 – 60.05] (0.052)
BMI	0.92 [0.84 – 0.99] (0.048)	0.90 [0.73 – 1.11] (0.324)
Hearing loss present	0.25 [0.09 – 0.60] (0.004)	0.16 [0.03 – 0.79] 0.029
Alleviation: pressure over occipital artery	13.55 [1.70 – 279.37] (0.026)	N/A (0.993)
Alleviation: ipsilateral neck compression	0.12 [0.01 – 0.58] (0.038)	1.12 [0.04 – 15.94] (0.935)
Subjective pitch: high (vs exclusively low)	10.29 [2.81 – 66.52] (0.002)	33.81 [3.81 – 882.80] (0.007)
Physical exam: bruit present	9.39 [3.96 – 23.93] (8.57E-7)	9.95 [2.04 – 62.08] (0.007)
Venous PT		
PT Duration	1.0003 [1.0000 – 1.0006] (0.031)	1.03 [0.10 – 10.67] (0.198)
Age	0.96 [0.94 – 0.98] (6.76E-4)	0.97 [0.94 – 0.99] (0.022)
BMI	1.08 [1.02 – 1.15] (0.015)	1.05 [0.98 – 1.13] (0.165)
Alleviation: ipsilateral neck compression	8.84 [3.20 – 31.32] (1.24E-4)	5.24 [1.62 – 21.01] (0.010)
Exacerbation: Valsalva	2.99 [1.38 – 6.84] (0.007)	2.23 [0.88 – 5.87] (0.094)
Physical exam: bruit present	0.38 [0.18 – 0.77] (0.009)	0.35 [0.14 – 0.81] (0.017)
Arterial PT		
Age	1.05 [1.01 – 1.12] (0.044)	1.05 [1.01 – 1.12] (0.044)