International Tinnitus Journal 1, 127-137 (1995)

Objective Pulse-Synchronous "Essential" Tinnitus due to Narrowing of the Transverse Dural Venous Sinus

Eric J. Russell,[1,2,3] M.D., Brian J. De Michaelis,[2] M.D., Richard Wiet,[2,3] M.D., Joel Meyer,[1,3] M.D.

Departments of Radiology[1] and Otolaryngology/Head and Neck Surgery[2], Northwestern Memorial Hospital and Northwestern University Medical School[3], Chicago, Illinois, USA

Abstract: Subjective tinnitus is a common problem with many etiologies. Objective tinnitus, in which the sound is perceived by both the patient and the examiner, is less common. Objective tinnitus of the vascular type, in which a pulse synchronous bruit is heard by an independent observer, is frequently related to an underlying arterial or arteriovenous malformation, most commonly a dural arteriovenous fistula (DAVF) involving the transverse and sigmoid sinuses. The remaining cases are usually termed "essential" vascular tinnitus, and are presumed to have a venous etiology. In these cases, the audible noise is generally assumed to be produced within the sino-jugular connection, or within an enlarged jugular bulb.

We present four documented cases of objective pulse synchronous tinnitus due to focal narrowing (acquired and developmental) of the mid-portion of the transverse dural sinus. In all cases, a bruit was audible directly over a focal constriction in the sinus, demonstrated by cerebral angiography or direct catheter venography. In one case, selective venography revealed a distensible sinus narrowing, associated with a jet of contrast marking fast flow within a developmental sinus segmentation. In another case, a loud pulse synchronous bruit was heard directly over a focal transverse sinus stenosis, which was detected by angiography at the site of a vascular surgical clip. In this case, magnetic resonance (MR) falsely predicted sinus occlusion. In two other cases, an audible bruit was also heard directly overlying a narrowed transverse sinus, seen in the venous phase of angiography.

Transverse sinus stenosis is an unappreciated cause of objective pulsatile tinnitus, and we believe that this mechanism may underlie many cases of "essential" or venous etiology tinnitus not otherwise anatomically explained. Non-invasive testing, computed tomography (CT) and MR and non-directed angiography may overlook it. Conventional catheter arteriography or venography should be performed in such cases, with attention to the dural sinuses, if other tests fail to define the anatomic basis of the audible bruit.

INTRODUCTION

Subjective tinnitus is a common problem with many etiologies. Objective tinnitus, in which the sound is perceived by both the patient and the examiner, is less common. Objective tinnitus of the vascular type, in which a pulse synchronous bruit is heard, is often related to an underlying arterial anomaly (ectopic tympanic carotid), dural arteriovenous fistula (DAVF), cerebral arteriovenous malformation, arterial aneurysm, or a vascular neoplasm such as paraganglioma.[1-6] Arterial causes of objective pulsatile tinnitus are readily diagnosed by CT or MRI, although selective catheter angiographic investigation may be required to fully delineate the nature of the underlying process.[1]

Roughly half of the patients with vascular tinnitus are presumed to have a venous etiology of sound production. These have been referred to as cases of "essential" or venous tinnitus.[7] Venous tinnitus is predominantly a disease of women, and carries an excellent prognosis, although cases have been noted to persist for many

Reprint requests: Eric J. Russell, M.D., Department of Radiology, Northwestern Memorial Hospital, Olson Pavilion 3420, 710 N. Fairbanks Ct., Chicago, Illinois 60611, USA

years.[7-11] The sounds producing the symptoms of tinnitus are generally attributed to cervical jugular venous distortion (cervical venous hum), or to anatomical variants of the sino-jugular axis and the jugular bulb.[8-10] Three years ago, we were the first to observe a patient with objective tinnitus due to narrowing of the transverse venous sinus,[12] and we have since found three additional patients with similar findings. Our four cases illustrate a previously unappreciated cause of objective tinnitus: stenosis or segmentation of the transverse dural sinus. We believe that this entity may be commonly related to noise production in cases of pulse synchronous tinnitus, and that its previous lack of description is due to the difficulty of making the diagnosis with non-invasive imaging. Detection requires a high index of suspicion and a meticulous imaging approach.

Case Reports

Case 1 (GM)

A 45-year-old-African-American female with chronic depression and headaches presented with a "swishing" sound in her left ear of 18 months duration. The sound was heard initially only at night, later becoming continual. A bruit was heard beginning in July 1983. The sound changed with position, becoming louder when supine and decreasing with the head turned to the left or right. No history of trauma, exposure to loud noise, hearing loss, ototoxic medications or hypertension was elicited. Physical examination revealed an easily heard left postauricular bruit, synchronous with systole and obliterated by manual ipsilateral jugular compression. Neurologic exam was normal, and no evidence for hyperdynamic state was found on examination or laboratory investigation.

Selective cerebral angiography was undertaken and demonstrated no arterial abnormality. However, left sided dural sinus venous drainage was poorly visualized. Prompted by the objective findings and the venous phase of the angiogram, a femoral venous puncture was performed and a selective catheter was placed through the jugular vein into the distal left sigmoid sinus

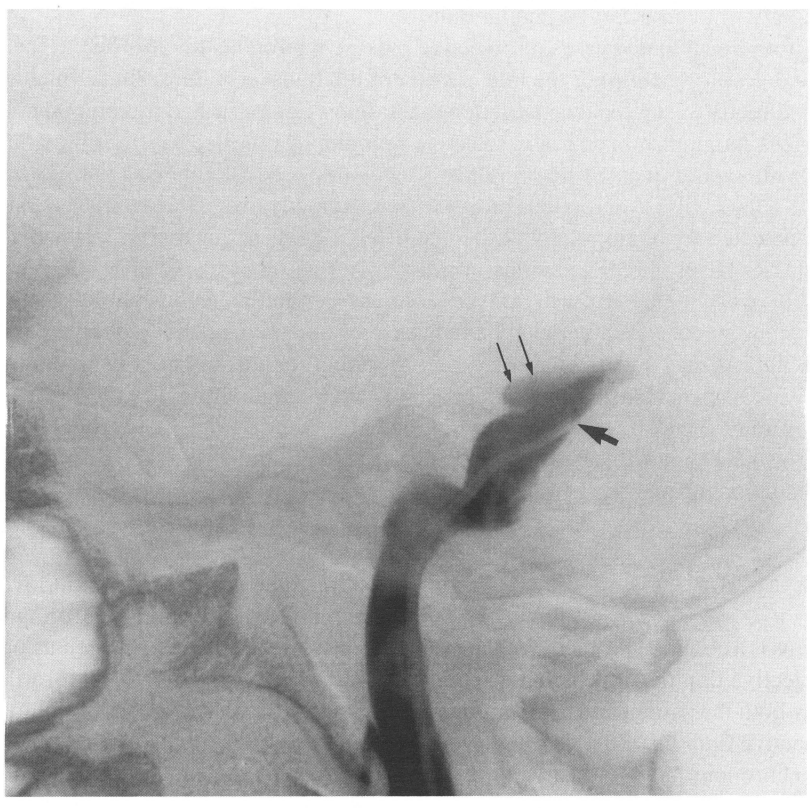

Figure 1a. Obstruction of retrograde flow of contrast in left sigmoid sinus during selective catheter venography. The catheter tip has been positioned in the sigmoid sinus (arrow). Contrast fails to reflux into the transverse sinus. Note excellent definition of a diverticulum of the superior margin of the sinus (arrows).

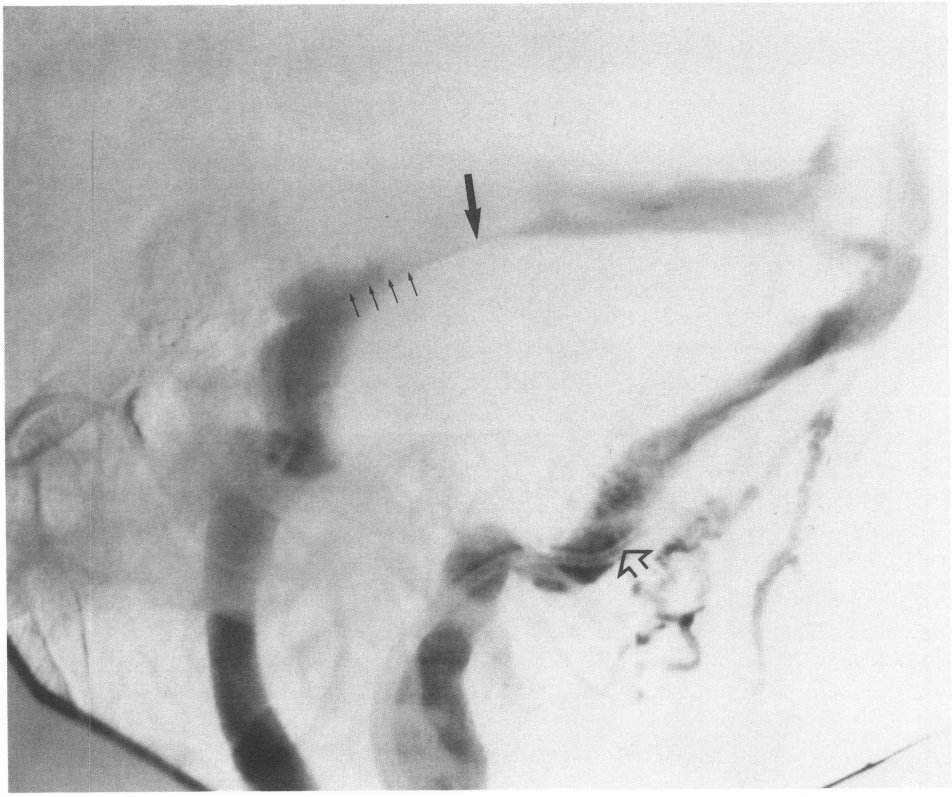

Figure 1b. Demonstration of stenotic left transverse sinus by contralateral right catheter venography. The catheter tip has been advanced into the right sigmoid sinus (open arrow). Contrast injection fills the narrowed left transverse sinus (large arrow) by reflux around the torcula. Note the jet of contrast (small arrows) existing the stenotic segment, impacting the diverticulum of the sigmoid sinus beyond it (compare to 1a).
(Image reprinted from Russell EJ, et al., in Huckman MS, et al: Highlights of the 28th annual meeting of the ASNR, Los Angeles, March 19-23, 1990, AJNR 1990;11:1057-1068, with permission).

A selective retrograde injection into the sigmoid sinus revealed the presence of a sigmoid diverticulum; however, the contiguous transverse sinus was narrowed and could not be filled by reflux (Fig. 1a).
Next, the catheter was directed into the contralateral right jugular vein and then into the right sigmoid sinus. Forceful injection of contrast filled the contralateral left sigmoid sinus by reflux around the torcula, and demonstrated that the mid/distal left transverse sinus was markedly narrowed (Fig. 1b). A "jet" of contrast was seen to traverse the stenotic portion of the transverse sinus, and impact the more distal diverticulated portion of the sigmoid sinus. In an attempt to further define the nature of the narrowed segment, the catheter was carefully reintroduced into the left jugular bulb, and this time gently advanced, without difficulty, through the left transverse sinus. The narrowed segment easily dilated to accommodate the catheter, and injection of contrast outlined a narrowed segment with a wider diameter than could be appreciated from the contralateral injection (Fig. 1c). Upon withdrawal of the catheter, the sinus again collapsed to its original diameter, implying the presence of a pliable stenosis.
Considering the clear relationship of the audible bruit to the underlying venous stenosis, possible treatment options were discussed, including ligation, attempted dilatation, or close observation. She chose the latter.
One year after the onset of the tinnitus, and shortly after angiography and venography, the tinnitus and bruit disappeared. The temporal relationship with the procedure suggests the possibility that the sinus thrombosed, perhaps due to local catheter trauma or contrast effect.

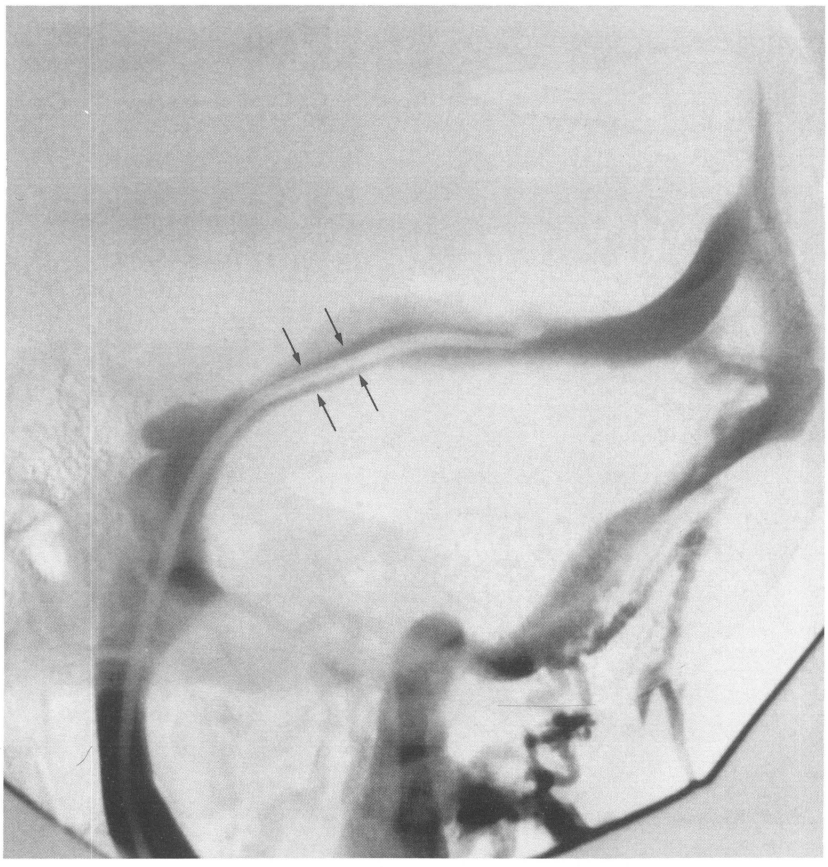

Figure 1c. Distension of narrowed segment by catheter introduction. The catheter was repositioned within the left sigmoid sinus, and carefully advanced over a guide wire, into the stenotic segment of the transverse sinus. The sinus dilated appreciably to accommodate the catheter (arrows). This segment collapsed once more following catheter withdrawal.

Two months later, the tinnitus returned at a lower volume; however, the objective bruit could not be heard. This may represent partial recanalization, or venous rerouting around a thrombosed transverse sinus. Over the following six years the subjective tinnitus persisted with much less severity and the objective bruit has not reappeared.

Case 2 (LW)

A 30-year-old female presented with a two and one-half year history of increasing right-sided pulsatile tinnitus, beginning during pregnancy. The noise began with a faint sound and became progressively louder over the course of several months. The MRI and carotid doppler studies were entirely normal. In view of the presence of an easily heard pulse synchronous bruit in the right post-auricular region, conventional cerebral angiography was performed in October 1989. The arterial phase was normal. In the venous phase of both the right internal carotid and left vertebral artery injections, a marked constriction of mid-and-distal portion of a dominant right transverse sinus was appreciated. A contrast jet through the stenotic segment more distally impacted a sigmoid sinus diverticulum (Figs. 2 a, b). The right superior petrosal sinus was seen to enter the stenotic segment at its midpoint.

A discussion of therapeutic alternatives ensued, but no intervention was performed. Although after a year the noise had diminished. Follow-up four years later revealed no change in the intensity of the sound, which recently was described at auscultation by an independent physician observer as similar to the "sound of Canadian geese flying in the distance."

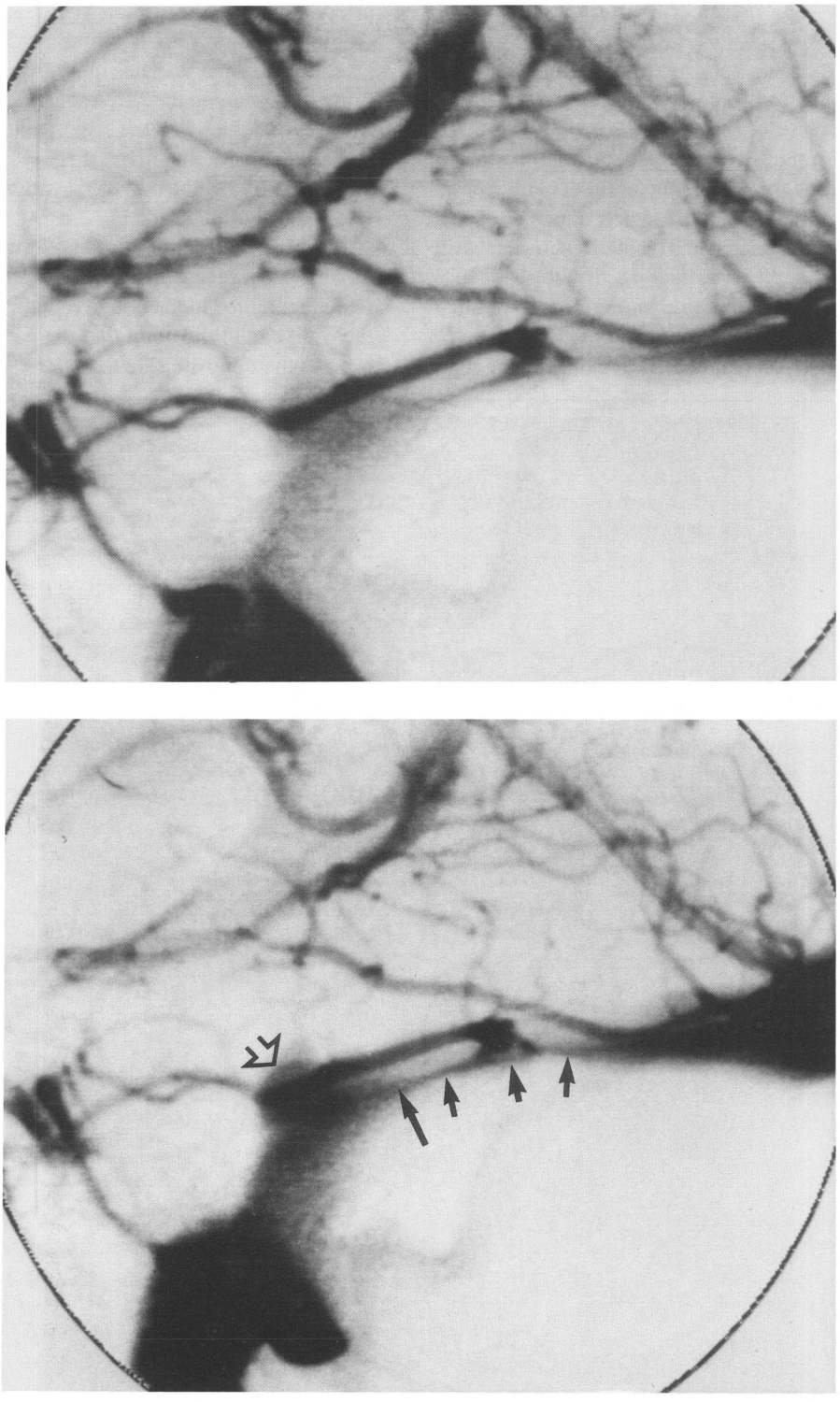

Figure 2a & b. Mid (a) and the late (b) venous phase images from right carotid angiogram reveals marked narrowing of the dominant right transverse sinus (arrows, b), with a jet of contrast (long arrow) traversing this segment to impact a diverticulum of the more distal sigmoid sinus (open arrow, b).

Case 3 (LS)

A 33-year-old woman presented with right-sided pulse synchronous tinnitus. The noise began seven months prior to admission and became constant after a month. Upon admission, the patient complained of a recent increase in sound intensity, with occasional pain in the right ear, which was intermittently severe. There was no sign of hypertension, although the patient was moderately obese. On physical examination, a pulse synchronous bruit was heard best behind the right ear. The pitch increased with Valsalva and the sound level decreased with ipsilateral neck compression. An outside magnetic resonance angiography (MRA) was interpreted to be suspicious for a dural arteriovenous fistula, with prominence of the right transverse sinus and prominent branches of the occipital artery visualized in the scalp (Fig. 3a).

Angiography performed in June 1993 failed to reveal any arterial abnormality. The right occipital artery was prominent, as seen on the MRA, but was otherwise normal. In the venous phase, the right transverse and sigmoid sinuses were dominant and there was moderate narrowing of the mid-right transverse sinus (Fig. 3b), directly under the audible bruit.

No surgical intervention was undertaken. Follow-up six months later revealed no interval change in the noise, which remained loud enough to interfere with hearing in the right ear, to the point that the patient would turn her head to the opposite side to hear better.

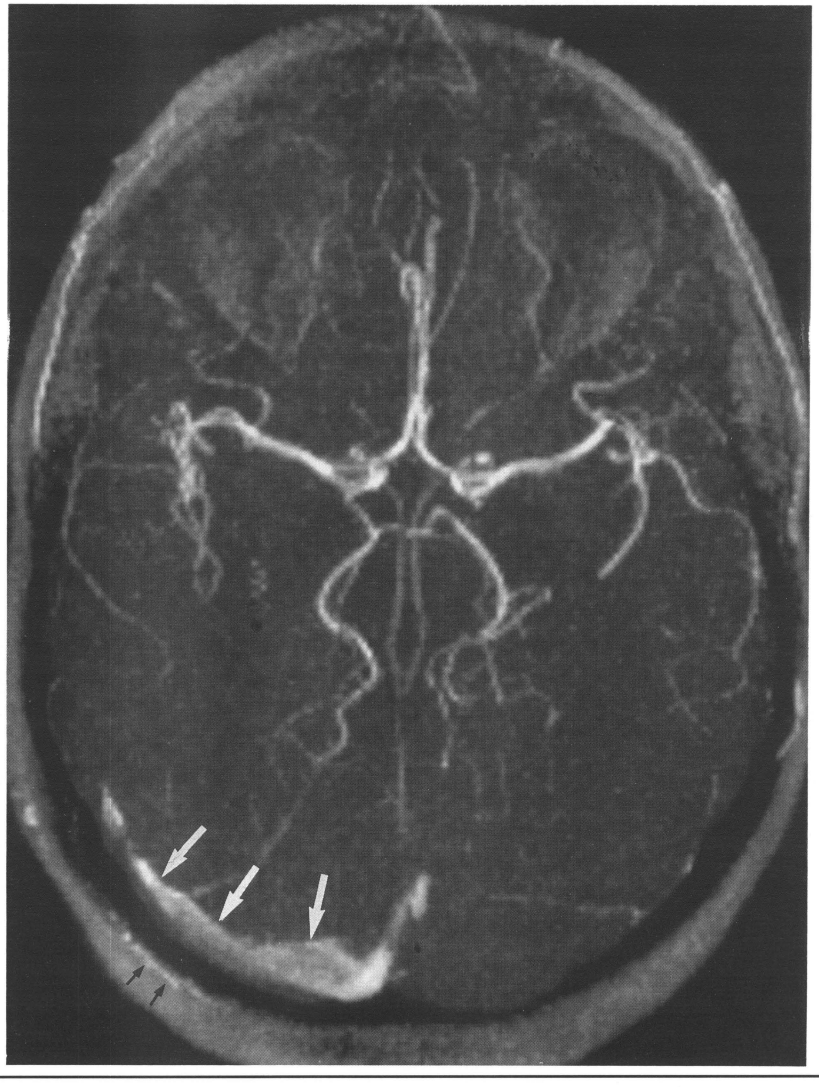

Figure 3a. Collapsed view of MR angiogram (basal projection) clearly shows a normal Circle of Willis. No arterial abnormalities are seen. Note some prominence of scalp branches of the occipital artery (arrows) and relatively rapid flow (high signal) in the right transverse sinus (white arrows).

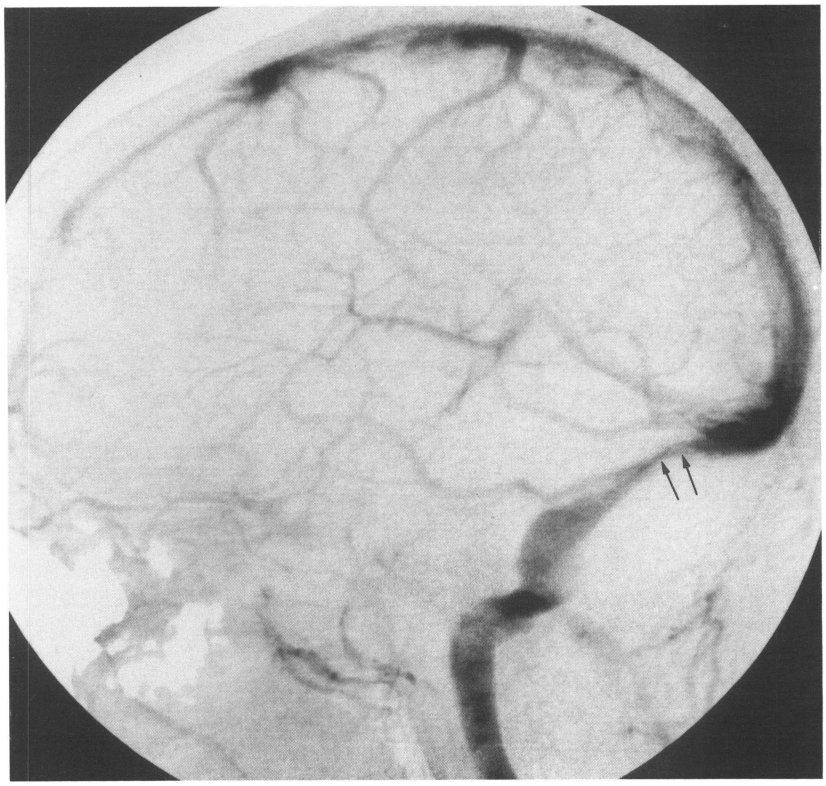

Figure 3b. Venous phase of carotid arteriogram reveals moderate narrowing of the mid-right transverse sinus, (arrows), just beneath the audible bruit observed clinically.

Case 4 (MU)

A 69-year-old woman presented with subjective tinnitus in the left ear for several months. She had a long history of hypertension. Three years previously she had undergone resection of a left acoustic schwannoma by retrosigmoid approach. The procedure was complicated by bleeding at the junction of the transverse and superior petrosal sinuses, requiring the placement of several vascular clips in this region. On physical examination, a loud bruit was clearly audible in the left post-auricular region, centered over the location of the left transverse venous sinus vascular clips. The pre-angiographic laboratory work-up revealed an unexpected low hematocrit 25 (HCT).

MR scans revealed post-operative changes with surgical clips seen in the retromastoid region. The left transverse sinus was not visualized and appeared to be thrombosed. MRA was not performed.

Four vessel angiography performed in July 1993 revealed no evidence of vascular tumor or arteriovenous fistula. In the venous phase of the left carotid injection, a large patent right transverse sinus and jugular were seen. Venous drainage from the left hemisphere was primarily directed toward the right, (Fig. 4a). A relatively hypoplastic left transverse sinus filled, and appeared to be further narrowed near the surgical clips at its mid-portion (Figs. 4a,b). The most proximal segment of the left transverse sinus appeared hypoplastic (Fig. 4a). The diagnosis was objective tinnitus related to constriction of the transverse sinus, probably related to turbulent blood flow near a post-operative stricture along the developmentally hypoplastic venous sinus.

We considered that a hyperdynamic state derived from the severe anemia accentuated flow-related sounds through the stenotic segment of the sinus and recommended re-evaluation after treatment. The most recent follow-up, four months after angiography, revealed that the noise persisted, although it had significantly diminished and that with iron therapy the HCT has risen to 13 from 8.

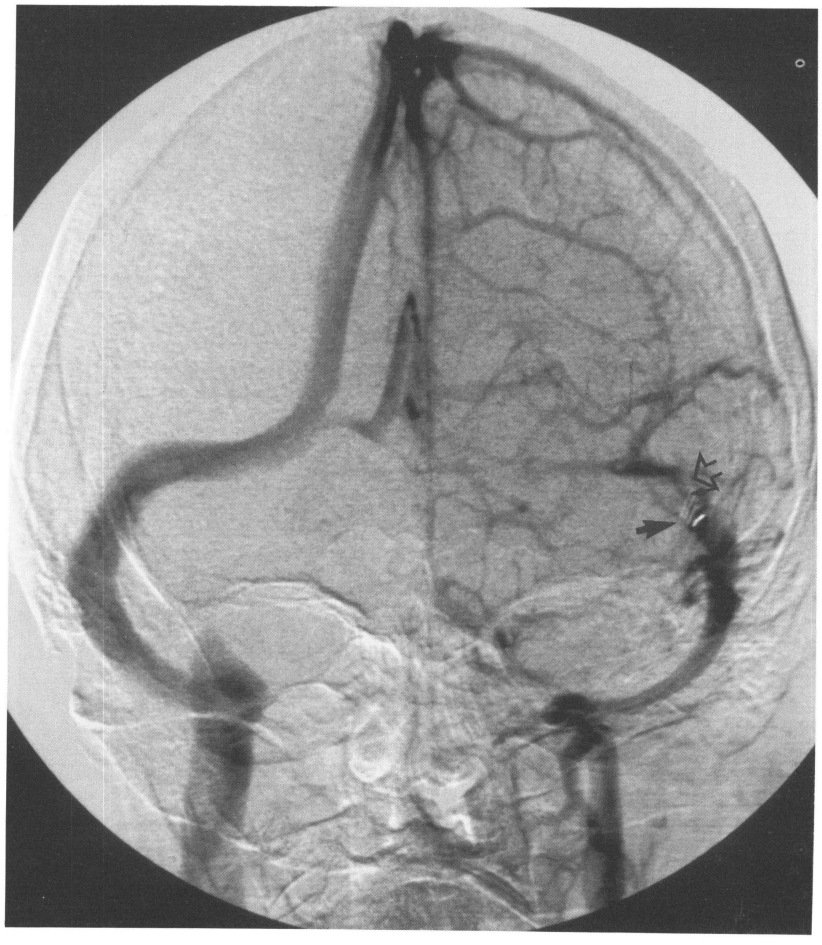

Figure 4a. Frontal view of the venous phase of carotid angiogram reveals metallic surgical clips adjacent to the narrowed mid-portion of the left transverse sinus (open arrow). The sinus is slender, and is further narrowed by the clips (arrow), which were placed to control intraoperative hemorrhage at the site of entrance of the superior petrosal sinus.

CONCLUSION

About 30% of the adult population has reported experiencing tinnitus of some kind.[9] The effects of tinnitus can vary from inconsequential to psychologically debilitating, and suicides have been reported.[7] Up to half of all objective vascular tinnitus is classified as idiopathic, or "essential" and is presumed to have a venous etiology.[7,8] If selective cerebral angiography fails to reveal an acquired or congenital arterial disorder such as an acquired dural arteriovenous fistula (DAVF), it has been assumed that the noise likely originates in the jugular bulb, or at the junction of the bulb and the sigmoid sinus. An elevated position of the jugular bulb in relation to the floor of the hypotympanum has been cited as a potential cause of pulsatile tinnitus.[8] So-called high or dehiscent jugular bulbs may protrude into the middle ear and cause an audible and characteristic "white noise" turbulence. Graham[13] cites both expansion and diverticulation of the jugular bulb as causes for pulsatile venous tinnitus. Other factors have been implicated, including angulation of the internal jugular vein at the transverse process of the atlas or axis.[9]

Venous tinnitus may be clinically confirmed based on response to specific physical maneuvers. Venous tinnitus is usually eliminated by ipsilateral jugular pressure or turning the head toward the uninvolved side, resulting in cervical venous narrowing and a decrease in flow.

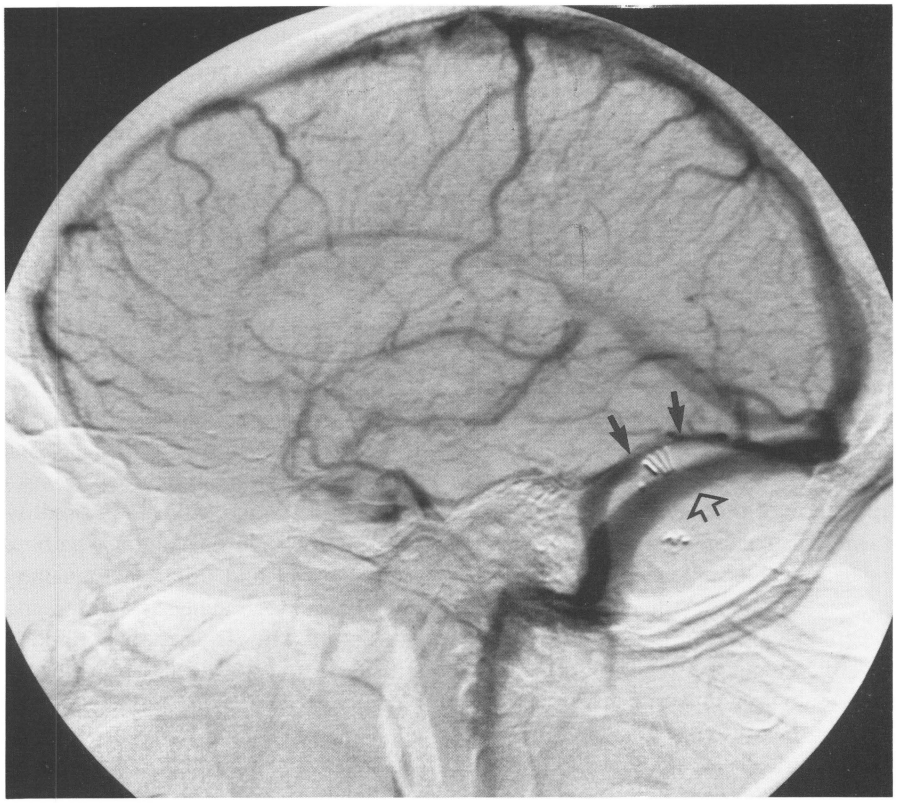

Figure 4b. Lateral view of venous phase showing narrowed left transverse sinus (arrows), and normal contralateral right transverse sinus (single arrow). After medical treatment for anemia (hematocrit level from 25 to 33), the subjective noise improved but did not fully disappear.

Turning the head toward the involved side, Valsalva maneuver, and deep inspiration may increase flow, and the noise.[11] Our first patient had several of these responses. The anatomic substrate of noise production in cases of objective venous tinnitus has, however, generally been impossible to pin down.

Our cases generally fit the pattern described first by Graf,[14] who in 1947 and 1952 first described the entity of essential tinnitus. He found an age range from 20-40 years, and a strong female predilection. Except for our Case 4, in which a surgical procedure likely played a role in generating the noise, our patients were 30, 33 and 45-years-old, and all were women. The prognosis, while said to be excellent, was mixed in our group. In the two patients with long term study, one experienced a decrease in noise level after several years, and one had no change in severe tinnitus after five years. All patients did however choose not to pursue surgical attempts to obliterate the sound, probably because they were indirectly encouraged not to do so by a full presentation of the possible risks and unproven nature of such endeavors. In most cases of venous tinnitus, the patient is assured of the benign nature of the condition after appropriate work-up, and no therapy is recommended. Electronic masking devices may be employed.[15] A removable cervical prosthesis which compresses the cervical jugular vein and redirects flow to reduce the noise level has been advocated.[16]

However, in cases where the noise is debilitating, treatment can be considered to lessen the bruit. Ligation of the internal jugular vein has been undertaken,[11] but this procedure has a high initial and delayed failure rate presumably due to the rapid development of collateral venous flow around the ligated level, particularly through

mastoid and condylar emissary veins.[7] Some authors have advocated obliteration of the sigmoid sinus in intractable cases.[7] Definitive localization of the site of noise production may be aided by the inflation of a non-detachable balloon catheter at the suspected site of turbulent blood flow (jugular vein) and by observing the resultant disappearance of the tinnitus.[10] This technique may also guide treatment in severe cases, by suggesting the optimal level for surgical ligation.[2,10] However, it may be dangerous to ligate a dominant jugular vein, particularly if the contralateral drainage system is hypoplastic.[10] Proposed alternatives include the placement of a synthetic graft anastomosis between the lateral sinus and internal jugular vein, to bypass the noise producing segment and allow continuation of venous outflow.[10]

Despite extensive prior investigations into the source of sound in cases of venous tinnitus, and all currently available testing, a **specific** anatomic explanation for the source of the sound has not and is often still not obtained, either by clinical methods or by imaging techniques. If a specific diagnosis could be made, further expensive and potentially invasive diagnostic testing would become unnecessary. Our current cases illustrate an etiology of pulsatile tinnitus which has previously gone unrecognized. We believe that the transverse sinus may frequently be the source of objective vascular tinnitus, when other etiologies are not found.

The pathophysiology of the narrowing is uncertain and likely variable. The distensibility of the "stenotic" segment in Case 1 may indicate the presence of a weak dural interface between the lumen of the sinus and the intracranial compartment, allowing the intracranial contents to "sag" on the sinus, producing reversible narrowing. In other cases, this segment of the sinus may be developmentally hypoplastic or segmented, a common condition given the tremendous variations known to occur in this region.[17,18] Huang,[17] fully described narrowing and atresia of the transverse sinus, while observing that the proximal portion of the sinus was more frequently affected. They also illustrated examples of distal sinus stenosis. Dora and Zileli[18] studied 192 retrograde venograms and observed that transverse sinuses were well formed bilaterally in only 47% of their cases, and that an occipital sinus is usually well developed when a transverse sinus is hypoplastic.

In other cases, sinus thrombosis and recanalization may underlie the stenosis. This well known phenomenon has also been proven to be associated with the production of transverse sinus dural arteriovenous fistula (DAVF),[4-6] where sinus stenosis and occlusion often precedes the development of the fistula.

The circumstances observed bring into question the etiology of the acute onset of tinnitus in patients who may have a fixed developmental sinus stenosis. Why do noises develop and then wax and wane in such cases? Cyclical changes in vascular resistance, blood viscosity and oxygen carrying capacity may play a role, such as in Case 4, where the noise abated with correction of anemia. In other cases, sinus thrombosis may produce relative obstruction and turbulence, or narrowing contralateral to the side of tinnitus may increase flow toward a moderately narrowed ipsilateral sinus segment. In either case, changes in flow may increase flow related sounds which may then be sufficient to cross the threshold above which sound becomes noticeable to the patient or an observer.

Normal sounds are likely present in normal individuals who have developmentally narrow or hypoplastic transverse sinuses; however, these sounds are generally sublimated by the structure of the ear, cochlear thresholds and other adaptations.[2] Breakdown in the patient's adaptation to low level noise perhaps due to changes in mood level and other factors, may explain the high incidence of clinical depression in patients with essential tinnitus. In essence, since tinnitus is often relatively acute in onset, and the presumed anatomic substrate relatively static, noise is likely to be related to an increase in local or systemic blood flow from a variety of possible conditions.

Many cases of pulsatile tinnitus may be adequately investigated with CT, MRI or MRA. If the tympanic membrane is abnormal (vascular), high resolution CT should be performed first, since it is effective at differentiating jugulotympanic paraganglioma from aberrant carotid artery and jugular dehiscence.[19] With a normal tympanic membrane, MR should be the first study, because of its improved contrast resolution. MRA increases the sensitivity of standard MR for the detection of dural arteriovenous fistula, the most common cause of objective tinnitus in the adult population.[20]

Despite the ability of special MR techniques to visualize the dural sinuses with excellent resolution, and even permit the distinction of sinus hypoplasia from thrombosis in some patients,[21] we believe that current **routine** MR imaging strategies may fail to diagnose a narrowed transverse sinus as the cause of objective tinnitus. The variable resolution of MRA also limits its accuracy for the reliable detection of a narrowed sinus segment, although detection may improve with a high index of suspicion, and perhaps slow velocity encoded MR venography.

Concerning angiography, investigation is based on the premise that many causes of objective tinnitus, such as DAVF, have potentiallyneurological sequelae and are treatable. Therefore considering this and the lack pitfalls of MR and MRA, selective arteriography should be performed when the source of objective tinnitus is not found

with non-invasive imaging. However, unless careful attention is paid to the venous circulation or subsequent venography is performed, dural sinus stenosis may still go undetected without a high index of suspicion.
It appears from our experience, that segmentation or stenosis of the dural sinuses may be responsible for a portion of the previously termed "idiopathic" cases of objective pulsatile tinnitus. Increased awareness of this condition should permit a firm diagnosis to be made in such cases, halting further pointless and expensive diagnostic workup, and allowing appropriate therapeutic options to be fully addressed.

REFERENCES

1. Willinsky RA: Tinnitus: Imaging algorithms. Can Assoc Radio J 43(2):93-99, 1992.

2. Ward PH, Babin R, Calcaterra TC, Konrad HR: Operative treatment of surgical lesions with objective tinnitus. Ann Otol 84:473-482, 1975.

3. Arenberg IK, McCreary HS: Objective tinnitus aurium and dural arteriovenous malformations of the posterior fossa. Ann Otol 80:111,1971.

4. Chaudhary MY, Sachdev VP, Cho SH, et al: Dural arteriovenous malformation of the major venous sinuses: An acquired lesion. AJNR 3:13-19, 1982.

5. House OW, Campbell JK, Campbell RJ, et al: Arteriovenous malformation affecting the transverse dural sinus - an acquired lesion. Mayo Clin Proc 54:651-661, 1979.

6. Nishijima M, Takaku A, Endo S, et al: Etiological evaluation of dural arteriovenous malformations of the lateral and sigmoid sinuses based on histopathological examinations. J Neurosurg 76(4):600-606, 1992.

7. Hentzer E: Objective tinnitus of the vascular type. A follow-up study. Acta Oto-laryngologica 66:273-281, 1968.

8. Adler JR, Ropper AH: Self-audible venous bruits and high jugular bulb. Arch Neurol 43:257-259, 1986.

9. Hazell JWP: Tinnitus. In: Alberti PW, Ruben RJ, (eds.) Otologic Medicine and Surgery. New York, Churchill Livingstone, 1988.

10. George B, Reizine D, Laurian C, Riche MC, Merland JJ: Tinnitus of venous origin. J Neuroradiology 10:23-30, 1983.

11. Tyler RS, Babin RW: Tinnitus. In: Cummings CW, et al. (eds.) Otolaryngology - Head and Neck Surgery. St. Louis: CV Mosby, 1986.

12. Russell EJ, Kim KS, Mulopoulos G: Segmentation of the lateral-sigmoid sinus junction: An etiology of objective tinnitus of venous origin. Presented at the 28th annual meeting of the ASNR, Los Angeles, CA., March 1990. Cited in Huckman MS, et al., Highlights of the 28th annual meeting of the American Society of Neuroradiology, AJNR 11:1057-1068, 1990.

13. Graham MD: The jugular bulb: Its anatomic and clinical considerations in contemporary otology. Laryngoscope 87:105-125, 1977.

14. Graf W, Moeller T, Mannheimer E: Continuous murmur. Acta Med Scand (Suppl) 196:167,1947.

15. Hazell JWP, Wood SM: Tinnitus masking -A significant contribution to tinnitus management. Br J Audiol 15:223, 1981.

16. Cary FH: Symptomatic venous hum. Report of a case. NEJM 264(17):869-870, 1961.

17. Huang YP, Okudera T, Ohta T, Robbins A: Anatomic variations of the dural venous sinuses. In: Kapp JP, Schmidek HH. (eds.) The Cerebral Venous System and its Disorders. Orlando, Grune and Stratton, 1985, pp.109-167.

18. Dora F, Zileli T: Common variations of the lateral and occipital sinuses at the confluens sinuum. Neuroradiology 20:23-27, 1980.

19. Remley KB, Coit W, Harnsberger HR, et al: Pulsatile tinnitus and the vascular tympanic membrane: CT, MR, and angiographic findings. Radiology 174:383-389, 1990.

20. Chen JC, Tsuruda JS, Halbach VV: Suspected dural arteriovenous fistula: Results with screening MR angiography in seven patients. Radiology 183(1):2271, 1992.

21. Mas JL, Meder JF, Meary E, Bousser MG: Magnetic resonance imaging in lateral sinus hypoplasia and thrombosis. Stroke 21(9):1350-1356, 1990.

ACKNOWLEDGMENT

The article was presented in January 1994 at the Middle Section meeting of the Triological Society, Mayo Medical Center, Rochester, MN.