Assessment of Objective Pulsatile Tinnitus in a Patient with Syringohydromyelia

James R. Steiger[*]
Patricia A. Saccone[†]
Karen N. Watson[†]

Abstract

We examined a 38-year-old male with syringohydromyelia and concomitant symptoms suggestive of intracranial hypertension including unilateral low-frequency sensorineural hearing loss and objective pulsatile tinnitus. The tinnitus was heard by the authors (through a hearing aid stethoscope tube), measured objectively (with a probe microphone), measured subjectively (as minimum masking levels and with fixed frequency Bekesy), and altered by changes in ear canal pressure (subjectively reported and measured objectively with a probe microphone). The audiologic symptoms were likely associated with elevated cerebrospinal fluid pressure that traveled to the cochlea through the cochlear aqueduct. The tinnitus may have originated from pulsations of central vascular structures that traveled through the cochlear aqueduct or the endolymphatic duct. Hearing loss likely resulted from tinnitus masking or a stiffening of the cochlear partition or stapes footplate.

Key Words: Intracranial hypertension, objective tinnitus, pulsatile tinnitus, syringohydromyelia

Sumario

Examinamos un hombre de 38 años con siringohidromielia y con síntomas concomitantes sugestivos de hipertensión intracraneana, que incluían una hipoacusia sensorineural unilateral de bajas frecuencia y un acúfeno pulsátil. El acúfeno fue escuchado por los autores (por medio de un estetoscopio para auxiliares auditivos), medido objetivamente (con una sonda micrófono), medido subjetivamente (como niveles mínimos de enmascaramiento y con un Bekesy de frecuencia fija), y alterado por cambios en la presión del canal auditivo (reportados subjetivamente y medidos objetivamente por una sonda micrófono). Los síntomas audiológicos estuvieron asociados con elevaciones en la presión del líquido cefalorraquídeo que viaja a la cóclea a través del acueducto coclear. El acúfeno debe haberse originado a partir de pulsaciones en las estructuras vasculares centrales, que viajaban por el acueducto coclear o el ducto endolinfático. La hipoacusia debe haber resultado del enmascaramiento del acúfeno o de un aumento en la rigidez en la partición coclear o en la platina del estribo.

Palabras Clave: Hipertensión intracraneana, acúfeno objetivo, acúfeno pulsátil, siringohidromielia

*School of Speech–Language Pathology and Audiology, University of Akron; †West Palm Beach Veterans Affairs Medical Center

Jim Steiger, School of Speech–Language Pathology and Audiology, The Polsky Building 181, Akron, OH 44325-3001; Phone: 561-523-0454; E-mail: jsteiger5@aol.com

LITERATURE REVIEW

Syringomyelia, or syringohydromyelia, occurs rarely (8.4 per 100,000; Schliep, 1978) and involves the formation of a syrinx (cavity) in the spinal column (Buettner and Caplan, 1996; Saremi, 2003). More specifically, syringomyelia is the accumulation of cerebrospinal fluid in a syrinx causing distention of the central canal of the spinal cord, and syringohydromyelia indicates communication with the subarachnoid space (Buettner and Caplan, 1996) or dissection of surrounding white matter (Saremi, 2003). Syringohydromyelia can be caused by trauma, tumor, arachnoiditis, subarachnoid hemorrhages, meningitis, Chiari malformations, or multiple sclerosis (Park et al 1989; Saremi, 2003; Padmanabhan et al, 2005). Maldevelopment leading to Chiari malformation accounts for approximately 80% of cases (Buettner and Caplan, 1996). The definitive test for syringohydromyelia is magnetic resonance imaging, and treatment depends on the cause, duration, and severity of symptoms. Surgical treatment options include decompression or shunting to relieve cerebrospinal fluid pressure (Buettner and Caplan, 1996).

Syringohydromyelia has been associated with intracranial hypertension and abnormal cerebrospinal fluid flow (Buettner and Caplan, 1996; Mazzola and Fried, 2003; Saremi, 2003; Owler et al, 2004). Symptoms of intracranial hypertension, also called pseudotumor ceribri, benign intracranial hypertension, and idiopathic intracranial hypertension (Digre and Corbett, 2001; Digre, 2003), include objective whooshing pulsatile tinnitus, unilateral low-frequency hearing loss, headache, retroocular pain, diplopia, vision impairment, and neck and back pain (Sismanis, 1987; Orcutt, 1988; Round and Keane, 1988; Giuseffi et al, 1991; Wall and George, 1991; Chiu et al, 1998; Celebisoy et al, 2002; Digre, 2003; Rudnick and Sismanis, 2005). Objective pulsatile tinnitus and low-frequency hearing loss can be the major or only symptoms of intracranial hypertension (Sismanis, 1987).

Intracranial hypertension is the most common cause of objective pulsatile tinnitus, especially when vascular disorders such as carotid artery abnormalities, atherosclerosis, and glomus tumors have been ruled out (Sismanis and Smoker, 1994; Sismanis et al, 1994; Sismanis, 1998a; Sismanis, 1998b). Tinnitus might originate from vascular pulsations of the walls of the dural sinuses (Rudnick and Sismanis, 2005) transmitted to the cochlea though the cochlear aqueduct (Rudnick and Sismanis, 2005) or the endolymphatic duct (Fishman, 1992). Hearing loss might result from either tinnitus masking (Weider et al, 1990; Sismanis, 1998b; Rudnick and Sismanis, 2005) or by stiffening of the cochlear partition or stapes footplate caused by elevated fluid pressure (Sismanis, 1987) that reaches the cochlea through the cochlear aqueduct (Marchbanks et al, 1990). Diagnosis of intracranial hypertension is made by lumbar puncture (Sismanis, 1998b). The lumbar puncture can also relieve cerebrospinal fluid pressure and thus serve as treatment. Other treatments include shunting, drug therapy with diuretics and steroids, and weight loss (Wullner and Corbett, 1996).

Doxycyclines, drugs used to treat malaria and acne, have also been associated with intracranial hypertension (Monaco et al, 1978; Stuart and Litt, 1978; Walter and Gubbay, 1981; Lander 1989; Gardner et al, 1995; Goulden et al, 1996; Chiu et al, 1998; Nagarajan and Lam, 2000; Weese-Mayer et al 2001; Digre, 2003). The mechanism for doxycycline-induced intracranial hypertension could be interference with cerebrospinal fluid absorption at the arachnoid granulations (Chiu et al, 1998; Arjona et al, 2003). Symptom onset of doxycycline-induced intracranial hypertension is typically two weeks to six months post–treatment initiation (Klasco, 2006).

PATIENT HISTORY

A 38-year-old obese male was referred to the tinnitus clinic at the West Palm Beach Veterans Affairs Medical Center for evaluation of his pulsatile tinnitus. This patient reported childhood head traumas including a right facial fracture at age 16 years and a head injury with loss of consciousness at age 12 or 13 years. He served in the military but denied significant military noise exposures. He also denied any history of hearing loss or tinnitus prior to the symptoms that are the topic of this paper. Vascular disorders such as carotid artery abnormalities, atherosclerosis, and glomus tumor were ruled out after extensive examinations and imaging. His history is summarized below.

His first suspicious symptom was right retroocular pain reported to his ophthalmologist in 2000. Headaches and right retroocular pain were reported several times subsequently at

emergency room visits and appointments with his primary care physician and neurologist. In 2001 he complained to his neurologist of acute right retroocular pain with light flashes and photophobia. His neurologist's diagnosis was migraine headaches.

In 2003 the patient was prescribed tetracycline (a doxycycline) for acne, a prescription he filled twice. Approximately one month later he reported more frequent headaches to his neurologist. In 2004 he reported headache, neck pain, and right arm numbness to his primary care physician. His primary care physician ordered magnetic resonance imaging in 2005. His radiologist identified a small syrinx 1.6 millimeters in diameter at the conus medullaris consistent with syringohydromyelia. His neurologist opined that the syrinx was a variant of normal. The definitive test for intracranial hypertension, lumbar puncture, was not conducted.

Later in 2005 tetracycline was again prescribed for acne, and the prescription was filled once. Approximately three months later, he reported diplopia to his ophthalmologist who diagnosed binocular diplopia of unknown etiology. He reported right aural fullness to his primary care physician and he was referred to the otology clinic where he also reported right pulsatile tinnitus. A magnetic resonance angiogram was ordered to investigate the pulsatile tinnitus. The findings were essentially normal, with a mildly tortuous basilar artery and a variation of normal circulation in the circle of Willis, both on the right side. At a neurology follow-up in 2006, he again reported diplopia and pulsatile tinnitus.

The patient, at the request of his otologist, was seen in the Audiology Department's tinnitus clinic. He complained of right pulsatile whooshing tinnitus that was less intense with his right ear plugged. He was seen three times in a two-week period, and all testing was done in an audiologic testing booth. The following are the essential findings:

1. Physical examination. The patient's ear canals were clear, and his tympanic membranes appeared normal. His pulsatile tinnitus was identified as objective tinnitus using a hearing aid stethoscope tube inserted into the right ear canal. The authors heard tinnitus pulsing at a rate consistent with the carotid pulse rate. The tinnitus could not be heard through the skull around the pinna using an amplifying stethoscope.

2. Audiometrics. A right ear low-frequency sensorineural hearing sensitivity loss was documented (Table 1, Figure 1).

3. Tympanograms. Tympanograms showed right middle ear static compliance greater than left middle ear static compliance (3.4 ml for the right ear and 1.2 ml for the left ear). The right middle ear resonance of 600 Hz was lower than the left middle ear
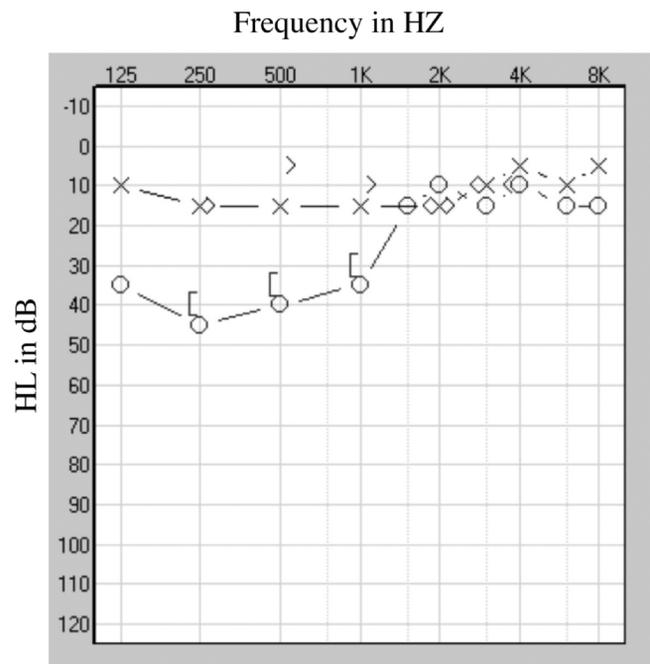
Frequency in HZ

Figure 1. Pure-tone audiogram in dB HL. O = right ear; X = left ear.

Table 1. Audiometric Air Conduction (AC) Thresholds Converted to dB SPL

	Hz									
	125	250	500	1000	1500	2000	3000	4000	6000	8000
Right AC	61	60	46	35	15	13	18	10	12	11
Left AC	37	30	21	15		18	13	5	8	1

resonance of 1000 Hz. These asymmetric findings were not explained by history.

4. Probe microphone measures. Noise levels in each ear canal were measured in 1/3-octave bands using an Audioscan Verifit analyzer with the tip of the probe microphone tube inserted 33 ml past the tragal notch. The measures were made with the ears open and with the ears plugged (with a foam audiometric insert earphone and with silicone impression material) to investigate the patient's report that tinnitus was softer with the right ear plugged. The noise levels measured in the right ear canal are shown in Figure 2, and the noise levels measured in the left ear canal are shown in Figure 3. The numerical data is given in Table 2. Noise intensity was greater in the right ear canal where tinnitus is heard. Noise intensity was greater with the ears plugged, which was not consistent with the patient's perception. Also, the authors noted 3–5 dB intensity fluctuations that corresponded to the pulsatile tinnitus.

5. Fixed frequency Bekesy. Tinnitus loudness measures are routinely made
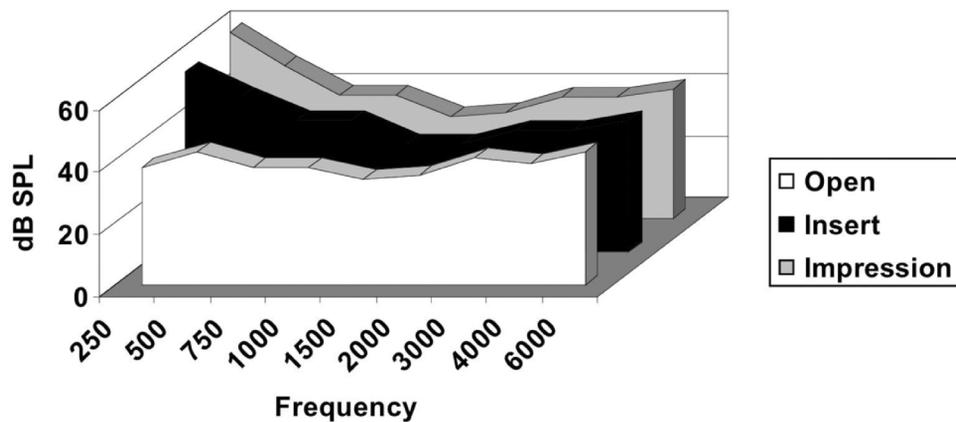
Probe Microphone Measurements - Right Ear

Figure 2. Probe microphone measures of the right ear canal. The data series with horizontal markings was obtained with the ear canal open; the data series with vertical markings was obtained with the ear canal plugged with a foam audiometric insert earphone; and the data series without markings was obtained with the ear canal plugged with silicone impression material.
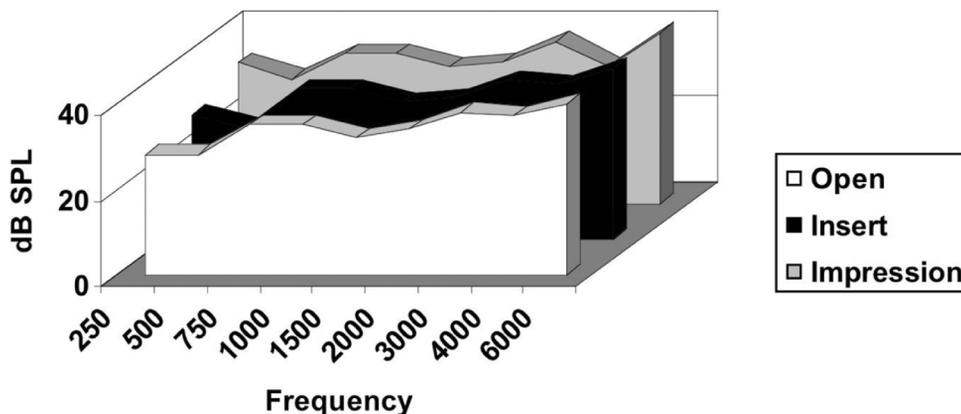
Probe Microphone Measurements - Left Ear

Figure 3. Probe microphone measures of the left ear canal. The data series with horizontal markings was obtained with the ear canal open; the data series with vertical markings was obtained with the ear canal plugged with a foam audiometric insert earphone; and the data series without markings was obtained with the ear canal plugged with silicone impression material.

with tinnitus patients (Henry and Zaugg, 2005). We used fixed frequency Bekesy, a nontraditional method, to match tinnitus loudness. The comparison tone was a 1000 Hz continuous tone presented to the left (nontinnitus) ear for one minute. The patient used a handheld button to keep the loudness of the tone equal to the loudness of his tinnitus. The results are shown in Figure 4. The bottom-most tracings were with the right ear canal open, and as shown, the subjective loudness matches roughly ranged from 50 to 55 dB HL (57 to 62 dB SPL). With his right ear plugged with silicone impression material (uppermost traces in Figure 3), his loudness matches were generally 5–10 dB better. The loudness matches were in rough agreement with the patient's hearing loss (Table 1) and the low-frequency portion of the 1/3-octave probe microphone noise measures given in Table 2. It should be noted that Bekesy test-retest differences can be seen. This could reflect practice effect or test-retest variability inherent with fixed frequency Bekesy tinnitus loudness matching.

6. Minimum masking levels. Minimum masking levels were measured using white noise. The white noise was presented through an insert earphone to the right ear, and minimum masking levels were measured using an ascending procedure (Henry and Zaugg, 2005). The minimum masking levels (Table 3) roughly agreed with the patient's hearing loss (Table 1) and the right ear probe microphone measures (Table 2).

Table 2. 1/3-Octave Probe Microphone Measures of Each Ear in dB SPL

					Hz				
Right Ear	250	500	750	1000	1500	2000	3000	4000	6000
Open dB SPL	38	43	38	38	34	35	41	39	43
Insert dB SPL	58	50	42	42	35	35	39	39	42
Impression Material dB SPL	60	49	40	40	33	34	39	39	42
					Hz				
Left Ear	250	500	750	1000	1500	2000	3000	4000	6000
Open dB SPL	28	28	35	35	32	34	38	37	40
Insert dB SPL	29	26	35	35	32	33	37	36	40
Impression Material dB SPL	33	29	35	35	32	33	38	32	40
					Hz				
Right-Left Ear Difference	250	500	750	1000	1500	2000	3000	4000	6000
Open dB SPL	10	15	3	3	2	1	3	2	3
Insert dB SPL	29	14	7	7	3	2	2	3	2
Impression Material dB SPL	27	20	5	5	1	1	1	7	2

Note: Data was obtained with ears open, ears plugged with foam audiometric insert earphones, and ears plugged with silicone impression material.
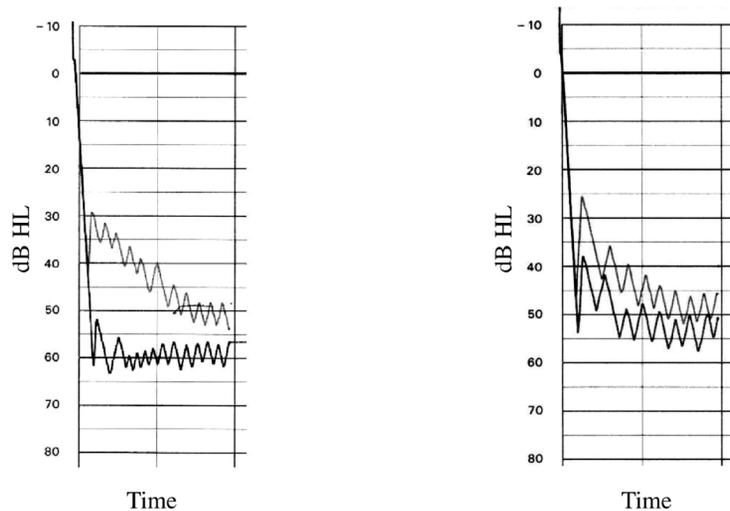
Figure 4. Right ear fixed frequency Bekesy tinnitus loudness matches, test (left) and retest (right). Lower traces are with the ear canal open, and upper traces are with the ear canal filled with silicone impression material. The comparison signal was a 1000 Hz pure tone delivered through a foam audiometric insert earphone to the left ear.

7. Loudness judgments with right ear canal pressure changes. This patient's report of reduced tinnitus loudness with earplug use warranted further evaluation. Typically, tinnitus becomes louder when the ear is plugged because the sound is trapped in the ear. We suspected that earplug insertion might have forced pressure changes at the tympanic membrane that inhibited the pulsatile tinnitus. We therefore asked the patient to judge tinnitus loudness while right ear canal pressure was controlled using a Grason Stadler 33 tympanometer with the probe tone tube disconnected and plugged. The authors are not aware that this procedure has ever been conducted, and we arbitrarily chose pressures within the range typically used during tympanometry. The pressure was changed in 100 daPa steps from positive 200 daPa to negative 300 daPa, and the patient was asked to indicate whether tinnitus loudness changed with each pressure change. Tinnitus loudness decreased with negative ear canal pressure (Table 4).

8. Probe microphone measures with ear canal pressure changes. Probe microphone measures were made in the right ear as described above but with the probe microphone in place. Adhesive clay of the type typically used in hearing aid test boxes was used to help seal the ear around the tympanometry probe and probe microphone. The authors are not aware that this procedure has ever been conducted, and we arbitrarily chose pressures within the range typically used during tympanometry. Only three pressures were tested because of the difficulty maintaining a hermetic seal. The tinnitus noise levels in the right ear canal were lowest with negative pressure (Table 5, Figure 5) consistent with the subjective judgments (Table 4).

DISCUSSION

Our patient had unilateral objective pulsatile tinnitus that was heard by the authors

Table 3. Minimum Tinnitus Masking Levels for White Noise Presented through an Insert Earphone to the Right Ear

Test	Retest	Mean
63 dB SPL	61 dB SPL	62 dB SPL

Note: Test, retest, and mean minimum masking levels are given in dB SPL.

Table 4. Tinnitus Loudness Judgments with Changes in Right Ear Canal Pressure

Ear Canal Pressure	Loudness Judgments
+200 daPa	No change
-100 daPa	No change
0 daPa	No change
-100 daPa	Softer than at 0 daPa
-200 daPa	Softer than at -100 daPa
-300 daPa	Softer than at -200 daPa

(through a hearing aid stethoscope tube), measured objectively (with a probe microphone), measured subjectively (as minimum masking levels and with fixed frequency Bekesy), and altered by changes in ear canal pressure (subjectively reported and measured objectively using a probe microphone). He also had unilateral hearing loss. We concluded the following based on our observations and data: First, the noise measured in the right ear canal was the tinnitus heard by the patient. This we conclude because the noise intensity measures and fluctuations in the right ear canal were similar to the tinnitus loudness measures and the fluctuations the authors and patient heard in that ear. Second, the noise (tinnitus) could have masked right ear low-frequency hearing. This we conclude because the noise (tinnitus) intensity and frequency composition measured in the right ear canal was similar to the degree and configuration of the low-frequency hearing loss.

The etiology of this patient's objective pulsatile tinnitus and hearing loss remains undiagnosed; his physicians reported only the symptoms. Vascular disorders such as carotid artery abnormalities, atherosclerosis, and glomus tumors were ruled out after extensive examinations and imaging. Intracranial hypertension is the most common cause of objective pulsatile tinnitus for such patients (Sismanis and Smoker, 1994; Sismanis et al, 1994; Sismanis, 1998a; Sismanis, 1998b). His radiologist identified syringohydromyelia, and this patient was therefore at risk for intracranial hypertension (Buettner and Caplan, 1996; Mazzola and Fried, 2003; Seremi et al, 2003; Owler et al, 2004). The apparent tetracycline exacerbation of this patient's symptoms is also suspicious (Stuart and Litt, 1978; Walter and Gubbay, 1981; Gardner et al, 1995; Nagarajan and Lam, 2000), as is his obesity (Sismanis, 1998a; Rudnick and Sismanis, 2005). However, the patient's neurologist opined that the syrinx (syringohydromyelia) was a variant of normal and did not order the definitive test for
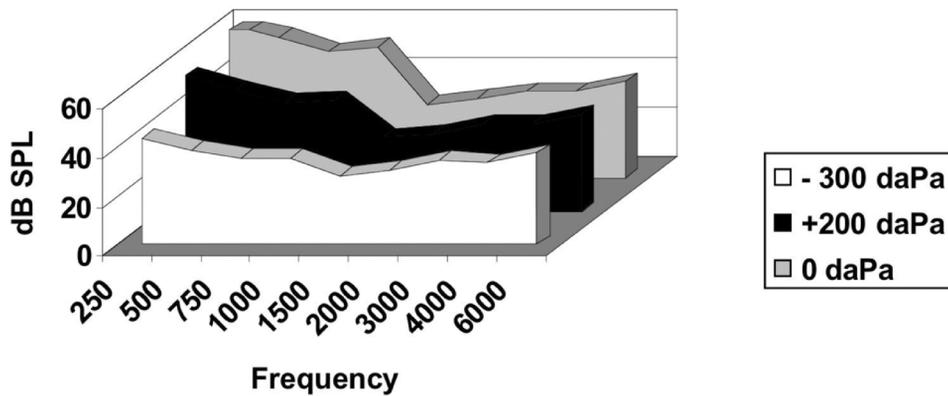

Figure 5. Probe microphone 1/3-octave noise measures of the right ear canal. The data series with horizontal markings was obtained with -300 daPa ear canal pressure; the data series with vertical markings was obtained with +200 daPa ear canal pressure; and the data series without markings was obtained with 0 daPa ear canal pressure.

Table 5. Probe Microphone 1/3-Octave Noise Measures in dB SPL with Changes in Right Ear Canal Pressure

	Hz								
	250	500	750	1000	1500	2000	3000	4000	6000
+200 daPa	55	49	44	45	30	31	35	35	39
0 daPa	62	57	51	53	30	32	35	35	39
-300 daPa	43	38	35	35	28	30	34	33	37

intracranial hypertension, lumbar puncture. Finally, the patient's primary care physician reviewed his history and imaging and determined that follow-up was needed. Follow-up plans were not made because of the patient's plan to move out of the state.

We suspect, as suggested in the literature, that this patient's tinnitus might have originated as central vascular pulsations from the walls of the dural sinuses (Rudnick and Sismanis, 2005) and in turn traveled through the cochlear aqueduct (Rudnick and Sismanis, 2005) or endolymphatic duct (Fishman, 1992) to the cochlea, middle ear, and ear canal. According to this scenario, his hearing loss might have been caused by tinnitus masking as discussed above (Weider et al, 1990; Sismanis, 1998b; Rudnick and Sismanis, 2005) or elevated fluid pressure within the cochlea that stiffened the cochlear partition or stapes footplate (Sismanis, 1987). Therefore, cochlear hair cells were likely intact, and restoration of this patient's hearing and elimination of his tinnitus appears possible through medical treatment or by spontaneous recovery (Weider et al, 1990, Sismanis, 1998b).

An alternative possibility is that elevated cochlear fluid pressure oriented the apex of the cochlear partition downward because of the stiffness differential between the oval and round cochlear windows. Such a downward orientation could inhibit cochlear hair cell functioning (Davis, 1958) causing the low-frequency hearing loss. In this scenario, the tinnitus origin could be central vascular structures or a tortuous blood vessel in the disoriented cochlear partition (Sismanis, 1998a). This hypothesis was supported by the reduction in tinnitus noise levels and tinnitus loudness that was induced by negative ear canal pressures. Negative ear canal pressure could have pulled middle ear structures and the oval window outward and the cochlear partition upward to a more normal position. However, this is contradicted by the finding that positive pressures also reduced the noise levels, albeit by a lesser amount that the patient could not detect. Perhaps, instead, changes in ear canal pressure reduced noise (tinnitus) levels by stiffening the middle ear and raising its resonance. One of the more puzzling findings, decreased tinnitus loudness with the right ear plugged, might also be explained by resonance changes. The insertion of an earplug would shorten the ear canal and raise ear canal resonance. Middle ear resonance might also be raised by the compression of the air mass stiffening the tympanic membrane. The higher resonance might inhibit the low-frequency pulsations.

It is unclear why this patient had symptoms only in his right ear. It is interesting that his mildly tortuous basilar artery and abnormal circle of Willis blood flow were on the right side, but it is not known whether the tinnitus could have originated at either of these sites. There might also be unknown anatomic variations such as a larger right cochlear aqueduct or endolymphatic duct that served as a more effective pathway for the pulsations on the right side rather then the left side. Pulsations on the right side might also have been enhanced in the right middle ear because of its low resonant frequency.

CLINICAL IMPLICATIONS

Objective pulsatile tinnitus and low-frequency hearing loss can be the major or only symptoms of intracranial hypertension (Sismanis, 1987). Audiologists should therefore be aware of the symptoms and be prepared with an otology or neurology referral source. An audiologic test protocol need not be as elaborate as the one we conducted. Identification of pulsatile tinnitus alone is sufficient to warrant referral, and this can be accomplished by listening through a hearing aid stethoscope tube inserted into the ear canal and quantified with open ear probe microphone measurements.

FUTURE RESEARCH

We evaluated our patient's objective pulsatile tinnitus with unconventional procedures. First, we measured tinnitus loudness using fixed frequency Bekesy. Researchers may wish to evaluate the utility, reliability, and validity of tinnitus loudness judgments obtained with this procedure. Finally, we obtained subjective and objective measures of tinnitus loudness and intensity while inducing pressure changes in the ear canal. Both loudness and intensity changed with ear canal pressure, and the authors proposed possible explanations including changes in middle ear resonance and basilar membrane orientation. Researchers could explore these possibilities.

REFERENCES

Arjona A, Delgado F, Fernandez-Romero E. (2003) Intracranial hypertension secondary to giant arachnoid granulations. *J Neurol Neurosurg Psychiatry* 74:418.

Buettner UW, Caplan LR. (1996) Syringomyelia and syringobulbia. In: Brandt T, Caplan LR, Dichgans J, Diener HC, Kennard C, eds. *Neurologic Disorders.* San Diego: Academic Press, 653–658.

Celebisoy N, Secil Y, Akyurekli O. (2002) Pseudotumor ceribri: etiological factors, presenting features and prognosis in the western part of Turkey. *Acta Neurol Scand* 106:367–370.

Chiu AM, Chuenkongkaew WL, Cornblath WT, Trobe JD, Digre KB, Dotan SA, Musson KH, Eggenberger ER. (1998) Minocycline treatment and pseudotumor ceribri syndrome. *Am J Opthalmol* 126:116–121.

Davis H. (1958) A mechano-electrical theory of cochlear action. *Ann Otol Rhinol Laryngol* 67:789–801.

Digre KB. (2003) Not so benign intracranial hypertension. *BMJ* 326:613–614.

Digre KB, Corbett JJ. (2001) Idiopathic intracranial hypertension (pseudotumor ceribri): a reappraisal. *Neurologist* 7:2–67.

Fishman RA. (1992) Anatomical aspects of the cerebrospinal fluid. In: Fishman R, ed. *Cerebrospinal Fluid in Disease of the Nervous System.* Philadelphia: WB Saunders, 20.

Gardner K, Cox T, Digre KB. (1995) Idiopathic intracranial hypertension associated with tetracycline use in fraternal twins: case reports and review. *Neurology* 45(1)6–10.

Giuseffi V, Wall M, Spiegel PZ. (1991) Symptoms and disease associations in idiopathic intracranial hypertension (pseudotumor ceribri): a case control study. *Neurology* 41:239–244.

Goulden V, Glass D, Cunliffe WJ. (1996) Safety of long term high-dose minocycline in the treatment of acne. *Br J Derm* 134:693–695.

Henry J, Zaugg TL. (2005) Clinical guide for audiologic tinnitus management II: treatment. *Am J Audiol* 14:49–70.

Klasco RK, ed. (2006) DRUGDEX® System. Thomson Micromedex, Greenwood Village, Colorado. Vol. 127 [3/2006].

Lander CM. (1989) Minocycline induced BIH. *Clin Exp Neurol* 26:161–167.

Marchbanks RA, Burge DM, Martin AM, Bateman DE, Pickard J, Brightwell AP. (1990) The relationship between intracranial pressure and tympanic membrane displacement. *Br J Audiol* 24(2):123–129.

Mazzola CA, Fried AH. (2003) Revision surgery for Chiari malformation decompression. *Neurosurg Focus* 15(3):Article 3.

Monaco F, Agnetti V, Mutani R. (1978) Benign intracranial hypertension after minocycline therapy. *Eur Neurol* 17:48–49.

Nagarajan L, Lam GC. (2000) Tetracycline-induced benign intracranial hypertension. *J Paediatr Child Health* 36:82–83.

Orcutt JC. (1988) Vision loss in pseudotumor ceribri. *West J Med* 148(6):691–692.

Owler BK, Halmagyi GM, Brennan J, Besser M. (2004) Syringomyelia with Chiari malformation; 3 unusual cases with implications for pathogenesis. *Acta Neurochir (Wein)* 146(10):1137–1143.

Padmanabhan R, Crompton D, Burn D, Birchall D. (2005) Acquired Chiari 1 malformation and syringomyelia following lumboperitoneal shunting for pseudotumour cerebri. *J Neurol Neurosurg Psychiatry* 76(2):298.

Park TS, Cail WS, Broaddus WC, Walker MG. (1989) Lumboperitoneal shunt combined with myelotomy for treatment of syringohydromyelia. *J Neurosurg* 71(6):950–953.

Round R, Keane JR. (1988) The minor symptoms of increased intracranial pressure: 101 patients with benign intracranial hypertension. *Neurology* 38:1461–1464.

Rudnick E, Sismanis A. (2005) Pulsatile tinnitus and spontaneous cerebrospinal fluid rhinorrhea: indicators of benign intracranial hypertension syndrome. *Otol Neurol* 26(2):166–168.

Saremi F. (2003) Syringohydromyelia. eMedicine from WebMD. http://www.emedicine.com/radio/topic670.htm.

Schliep G. (1978) Syringomyelia and syringobulbia. In: Vinken PJ, Bruyn GW, eds. *Handbook of Clinical Neurology.* Amsterdam: Elsevier, 255–327.

Sismanis A. (1987) Otologic manifestations of benign intracranial hypertension syndrome. *Laryngoscope* 97(8, Pt. 2, Suppl. 42):1–17.

Sismanis A. (1998a) Pulsatile tinnitus. In: Vernon J, ed. *Tinnitus Treatment and Relief.* Boston: Allyn and Bacon, 28–33.

Sismanis A. (1998b) Pulsatile tinnitus: a 15-year experience. *Am J Otol* 19(4):472–477.

Sismanis A, Smoker WRK. (1994) Pulsatile tinnitus: recent advances in diagnosis. *Laryngoscope* 104:681–687.

Sismanis A, Stamm MA, Sobel M. (1994) Objective tinnitus in patients with atherosclerotic carotid artery disease. *Am J Otol* 15(3):404–407.

Stuart BH, Litt IF. (1978) Benign intracranial hypertension with tetracycline therapy. *J Paediatr* 93:901.

Wall M, George D. (1991) Idiopathic intracranial hypertension: a prospective study of 50 patients. *Brain* 114:155–180.

Walter BN, Gubbay SS. (1981) Tetracycline and benign intracranial hypertension: report of five cases. *Br Med J (Clin Res Educ)* 282(6257):19–20.

Weese-Mayer DE, Yang RJ, Mayer JR, Zaparackas Z. (2001) Minocycline and pseudotumor ceribri: the well known but well-kept secret. *Pediatrics* 108(2):519–520.

Weider DJ, Kuo A, Spiegel PK, Musiek FE. (1990) Objective tinnitus of vascular origin with hearing improvement after treatment. *Otol Neurol* 11(6):437–443.

Wullner U, Corbett JJ. (1996) Pseudotumor ceribri. In: Brandt T, Caplan LR, Dichgans J, Diener HC, Kennard C, eds. *Neurologic Disorders.* San Diego: Academic Press, 637–639.