

# Audiological Correlates to a Rupture of a Pontine Arteriovenous Malformation

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## Abstract

This is a report of a female patient in her midthirties who sustained a hemorrhage secondary to an arteriovenous malformation in the region of the pons. The patient's initial symptoms included hearing loss and tinnitus, which were followed by the more characteristic symptoms of headache and loss of consciousness. Results of audiological testing at three months postaccident documented the presence of a hearing loss and a central auditory processing disorder, and the patient was provided an auditory rehabilitation program. Follow-up testing over the course of an additional year documented improvement in both pure-tone threshold and central test results; however, at 15 months postaccident, some auditory deficits remained, especially in the ear ipsilateral to the primary site of lesion. The anatomical correlates of these deficits are discussed, as are the potential contributions of both the auditory rehabilitation program and spontaneous recovery mechanisms to the documented improvements in auditory function.

**Key Words:** Arteriovenous malformation, auditory processing disorder, auditory rehabilitation, brainstem disorder, central auditory processing disorder, vascular disorder

**Abbreviations:** ABR = auditory brainstem response; ALD = assistive listening device; AVM = arteriovenous malformation; CT = computerized tomography; DPOAE = distortion product otoacoustic emissions; LPFS = low-pass filtered speech; PTA = pure-tone average; SAH = subarachnoid hemorrhage

## Sumario

Este es el reporte de una paciente femenina, a mediados de su cuarta década, quien sufrió una hemorragia secundaria a una malformación arteriovenosa en la región del puente. Los síntomas iniciales de la paciente incluyeron pérdida auditiva y un acúfeno, seguidos de síntomas más característicos como la cefalea y la pérdida de conciencia. Los resultados de las pruebas audiológicas, tres meses después del accidente, documentaron la presencia de una hipoacusia y de un trastorno central de procesamiento auditivo. El paciente fue involucrado en un programa de rehabilitación auditiva. Los estudios de seguimiento en el curso del siguiente año documentaron una mejoría en los umbrales tonales puros y en los resultados de las pruebas centrales; sin embargo, algunas deficiencias permanecieron unos 15 meses después del accidente, especialmente en el oído ipsilateral al sitio primario de lesión. Se discute la correlación anatómica de estas deficiencias, al igual que las contribuciones potenciales tanto del programa de rehabilitación auditiva como de los mecanismos de recuperación espontánea, en la mejoría documentada de la función auditiva.

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**Palabras Clave:** Malformación arteriovenosa, trastorno de procesamiento auditivo, rehabilitación auditiva, trastorno del tallo cerebral, trastorno central de procesamiento auditivo, trastorno vascular

**Abreviaturas:** ABR = respuesta auditiva del tallo cerebral; ALD = dispositivo de ayuda auditiva; AVM = malformación arteriovenosa; CT = tomografía computarizada; DPOAE = emisiones otoacústicas por productos de distorsión; LPFS = lenguaje filtrado con pasa-bajo; PTA = promedio tonal puro; SAH = hemorragia sub-aracnoidea

Sudden deafness, which has been defined as a sudden or rapidly progressive hearing loss of at least 30 dB across a minimum of three contiguous audiometric thresholds, has been attributed to a number of different etiological bases, including viral infections, vascular pathologies, autoimmune diseases, substance abuse, and allergies (Schuknecht, 1993; Kanzaki, 1994; Fernandez et al, 2003). In many cases of sudden deafness, the hearing loss is related to restriction or blockage of the blood supply to the auditory end organ secondary to cochlear ischemia, and the basis of the hearing loss rests in the compromise of the peripheral end organ and/or the cochlear nerve. Tinnitus and vestibular symptoms are also common in patients with sudden hearing loss (Nakashima and Yanagita, 1993; Schuknecht, 1993; Fernandez et al, 2003). Although less common, cases of sudden hearing loss have also been associated with lesions of the central nervous system (Hansen and Sorensen, 1978; Musiek et al, 1994; Fernandez et al, 2003).

One of the potential causes of sudden deafness is a vascular accident, which is the focus of the present case study. Vascular accidents result in the hemorrhaging of the arteriovenous system subserving the peripheral and/or central auditory structures, which in turn can compromise auditory function. The incidence of all subarachnoid hemorrhages (SAH) is at around six cases per

100,000 persons per year, with the majority of patients under 60 years of age. A sudden and severe headache is considered to be a significant, but nonspecific, clinical feature in the diagnosis of SAH (van Gijn and Rinkel, 2001). In the majority of patients with SAH, the headache associated with the vascular accident is described as the “worst” headache experienced in a lifetime, and its occurrence may or may not be associated with seizures, vomiting, and loss of consciousness (Fernandez et al, 2003). The vast majority of nontraumatic SAHs (approximately 85%) have been attributed to ruptures of intracranial saccular aneurysms, whereas about 10% are reported to be due to cerebral arteriovenous malformations (AVM)—anomalous vessels that join the arterial and venous systems—and 5% to other causes (van Gijn and Rinkel, 2001; Fernandez et al, 2003). Hearing loss is reported in about 5% of the cases (Nakashima and Yanagita, 1993).

The present case study is informative in a number of aspects: (1) it adds another case to the literature that demonstrates that loss of threshold sensitivity can be associated with hearing losses of central origin as well as peripheral origin; (2) it documents a case where decreased hearing sensitivity and tinnitus were the initial symptoms of a bleed or hemorrhage associated with an AVM at the level of the pons; and (3) it highlights the roles that spontaneous recovery and intervention

may play in the recovery of auditory function in a patient with a lesion of the caudal auditory brainstem pathway.

## CASE REPORT

### History

This female patient, in her midthirties, began to notice diminished hearing and tinnitus bilaterally one day while at work. Her symptoms reportedly became more severe over a period of several minutes before she lapsed into a state of unconsciousness. At that time she was rushed to the hospital where the diagnosis of an AVM of the pons with a bleed into the pons and fourth ventricle was made. The patient was monitored, treated, and released from the hospital, but with some neurological compromise. Among the neurological sequelae of the vascular accident was difficulty hearing.

Three months following her hospital admission, the patient was seen for the first of three audiological evaluations. At this time, the patient related with considerable detail the symptoms that she experienced during her vascular accident up to the time that she lost consciousness and in the interim between her regaining consciousness and the time of her first audiologic visit. She reported communication difficulties due to a hearing loss in her right ear. She indicated that she experienced severe distortion of sounds, especially in the right ear, and that she also experienced tinnitus in the right ear, which she described as having a tonal quality. She reported that her most severe auditory problem was hearing in the presence of background noise, and that from her perspective the hearing in her left ear seemed “okay.” At the time of the first audiologic visit (three months post-incident) the patient was experiencing problems with balance but no true vertigo. She presented with right-sided facial paralysis and motor compromise on the left side and had to use a wheelchair due to her left-sided motor problems and balance difficulty. Immediately after the vascular accident, she suffered from headaches and seizures; however, by the time of her initial audiologic visit, these problems had resolved for the most part. She did, however, report that she continued to experience occasional headaches. At the end of the first audiological

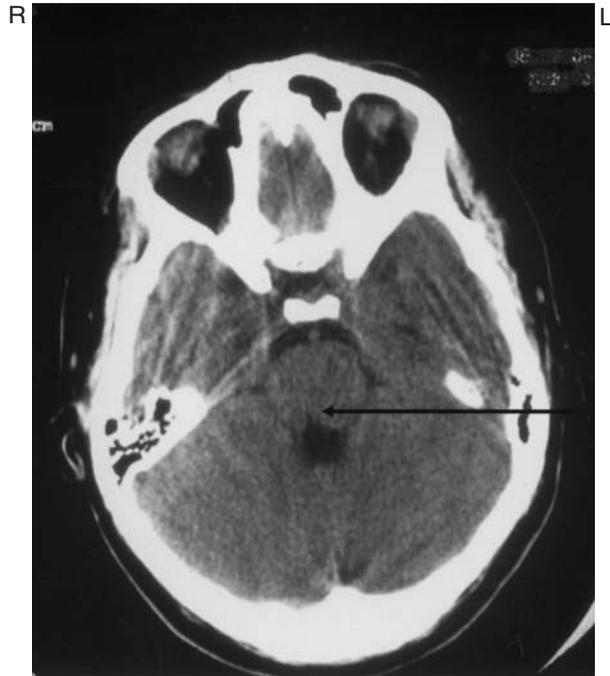
examination, the patient was placed on an auditory rehabilitation program, which will be discussed later.

The patient returned for her second visit about six months after her first audiological consultation (nine months post-incident). She had used the therapy program that was recommended but had modified it slightly to better fit her environment, needs, and resources. She reported that she had noticed considerable improvement in her hearing abilities since the time of her first visit. Specifically, she could now recognize noises and sounds around her house that she had not heard or recognized since before her vascular accident. She could hear the television and radio better, but these listening situations remained difficult for her. She had also noticed that she could discern that TV commercials were louder than the programs—something she could not do when she first returned home from the hospital. She related that hearing in background noise and tinnitus remained problematic for her. She also reported that she had begun to experience some tolerance problems for loud sounds. She was motivated by her auditory improvements and looked forward to continuing her auditory rehabilitation. She also mentioned that she was planning on undergoing gamma knife treatment in an effort to decrease the chances of future vascular problems.

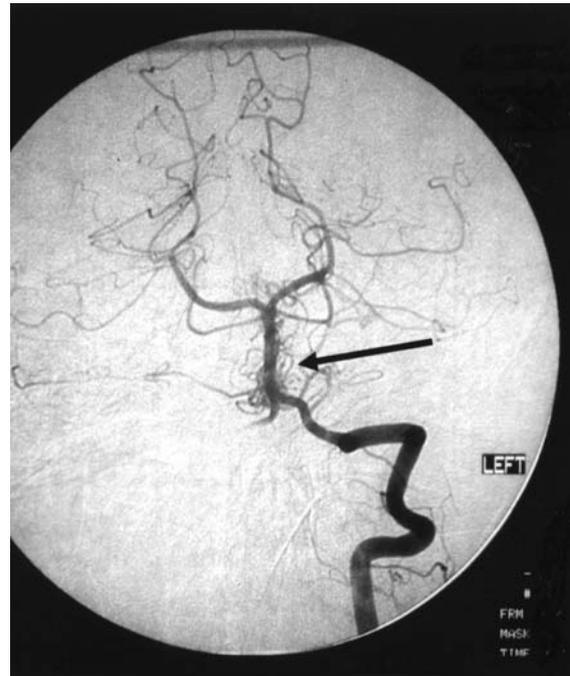
Approximately six months later (15 months post-incident), the patient returned for her third visit to the audiology clinic. At this point she had experienced only minimal subjective hearing improvement. In spite of this minimal improvement, the patient remained highly motivated and wanted to continue her auditory rehabilitation program. The mild facial paralysis and the mobility and balance impairments that were reported during the first audiologic visit persisted throughout the period of time that the patient was followed for audiologic testing.

### Radiology

Computerized tomography (CT) of the brain shortly after hospital admission showed what appeared to be a fourth ventricle hemorrhage. It also appeared, based upon information provided in the radiology report, that blood had “burrowed” into the dorsal aspect of the right side of the brainstem



**Figure 1.** A transverse view of a computerized tomography (CT) scan showing a slightly irregular-shaped fourth ventricle (the dark area immediately below the head of the arrow) and an extension of the lesion into the right posterior aspect of the brainstem (immediately beyond the tip of the arrow).



**Figure 2.** An arteriogram showing an entanglement of vessels in the caudal half of the basilar artery constituting an arteriovenous malformation.

(Figure 1) and that some hydrocephalus had been noted. An arteriogram showed an entanglement of blood vessels dorsal to, but in close proximity to, the basilar artery (Figure 2). This was considered the AVM or area of the AVM.

### **Audiological Test Procedures**

The patient was seen for audiological evaluations on three different occasions. The first evaluation was completed three months following her vascular accident. She was then reevaluated six months later (i.e., nine months post-vascular accident) and then again in another six months (i.e., 15 months post-vascular accident). All audiological testing was conducted in a sound-treated booth.

### **Pure-Tone and Speech Audiometry**

Pure-tone thresholds and speech recognition threshold tests were conducted in the conventional manner. The Northwestern University Test No. 6 served as the measure for speech recognition ability. During the

patient's first audiological assessment, speech recognition ability was tested for the right ear at a number of intensity levels in an attempt to find a level that might yield some correct responses. However, this effort did not meet with success, as the patient could not understand any of the monosyllabic words presented at any intensity level attempted. For the second and third visits, speech recognition was assessed at 35 dB sensation level (SL) in reference to the spondee threshold.

### **Distortion Product Otoacoustic Emissions**

Distortion product otoacoustic emissions (DPOAEs) were recorded with the GSI 60. The intensity levels for the two primary tones were 65 and 55 dB SPL (L1, L2), and frequencies of F2 from 1000 through 5000 Hz were tested bilaterally. Three points per octave were obtained and plotted. Noise floor, emission SPL level, and signal-to-noise floor measurements were derived for all recordings.

### ***Immittance Testing***

Tympanograms and acoustic reflexes were obtained in a conventional manner. Both ipsilateral and contralateral reflexes were measured in each ear at 500, 1000, and 2000 Hz.

### ***Auditory Brainstem Response***

The procedure for auditory brainstem response (ABR) testing has been previously reported and will not be discussed in detail here (Musiek and Lee, 1995). In each of the ABR assessments, an 80 dB nHL 100  $\mu$ sec click was presented at rates of 17.9 and 77.9 per second, and the filter band was set at 150–3000 Hz. Neuroelectrical activity was picked up from the head using a standard electrode montage with the impedance across electrodes maintained at less than 5 kohm.

### ***Central Auditory Behavioral Tests***

Two central auditory tests were administered during the three audiological visits. These included a low-pass filtered speech (LPFS) test and a monaural dichotic digits test. The LPFS test has been well described in the literature and will not be delineated here (see Musiek and Geurkink, 1982). The monaural digits test used in the present investigation was a modification of the conventional dichotic digits test (see Musiek, 1983). For this task, paired digits were routed to one ear at a time so that the digits were in competition creating a monaural low redundancy speech test. The patient was asked to repeat all the digits that she recognized. The stimuli for both tests were presented at 50 dB SL re: spondee threshold in each ear.

### ***Rehabilitation Plan***

The patient was interested in a therapy plan that might help her improve her hearing abilities. Therefore, the following suggestions for informal auditory therapy were offered. It was recommended that the patient, when necessary, wear an earplug in the right ear to reduce the amount of acoustic distortion that she was perceiving. This recommendation was made based on the patient's complaints of distortion for the right ear and her overall poor audiologic performance in that ear during

formal testing. The patient wore the earplug primarily in situations where listening with both ears became bothersome and/or confusing. As her auditory abilities improved, she used the earplug less frequently.

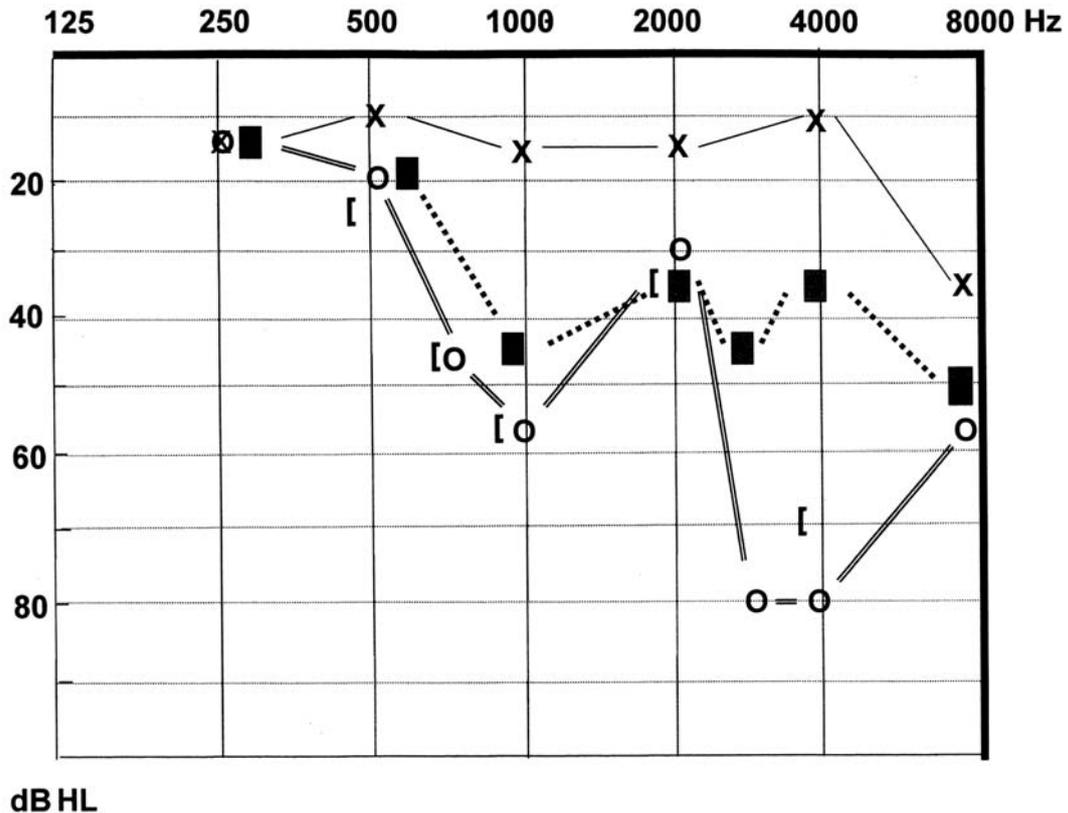
An assistive listening device (ALD) was provided to the patient along with a variety of instructions on alternative ways to use the device (a personal amplifier) in order to maximize its utility (e.g., monaurally, binaurally, etc.). The patient was encouraged to evaluate the usefulness of the ALD in a variety of listening conditions to determine how much it helped her.

Finally, the patient was encouraged to participate in an auditory training program. She worked on the discrimination of numbers, consonants, vowels, and similar sounding words and sentences. She devised an interesting and challenging way of doing auditory training, that is, over the telephone. Each day a friend of the patient would call her and say a list of numbers, words, consonants, and vowels over the phone. The patient's task was to identify and/or discriminate the acoustic stimuli. During each session, the patient would use her good ear and then her poorer ear, and her friend would speak at varied loudness levels. Usually 15 to 20 minutes per day were spent participating in these auditory training exercises, and detailed notes were kept during each session to determine which stimuli were missed. These "missed" stimuli were then the focus of the next practice session. The patient was also asked to read aloud daily, listening carefully to what she read.

### ***Test Results***

Three audiograms were obtained on the patient over a 15-month period with testing commencing at three months following the patient's vascular accident. It is important to note that the patient felt her hearing had improved over the three months that ensued between her vascular accident and her first audiologic visit.

The first audiogram (three months post-incident) revealed normal hearing sensitivity for the left ear except at 8000 Hz, where a mild deficit was evident. The hearing sensitivity for this ear did not change over the following two visits. On the initial hearing test, the right ear showed a moderate-to-severe sensorineural hearing loss primarily



**Figure 3.** An audiogram showing air-conduction thresholds for audiological visits 1 (O) and 2 (rectangles) for the right ear and visit 1 for the left ear (X). Bone-conduction thresholds are shown for the right ear (I) for visit 1 (pure-tone thresholds for the left ear were unchanged for visits 2 and 3, and for the right ear, thresholds for visit 3 were essentially the same as those recorded for visit 2).

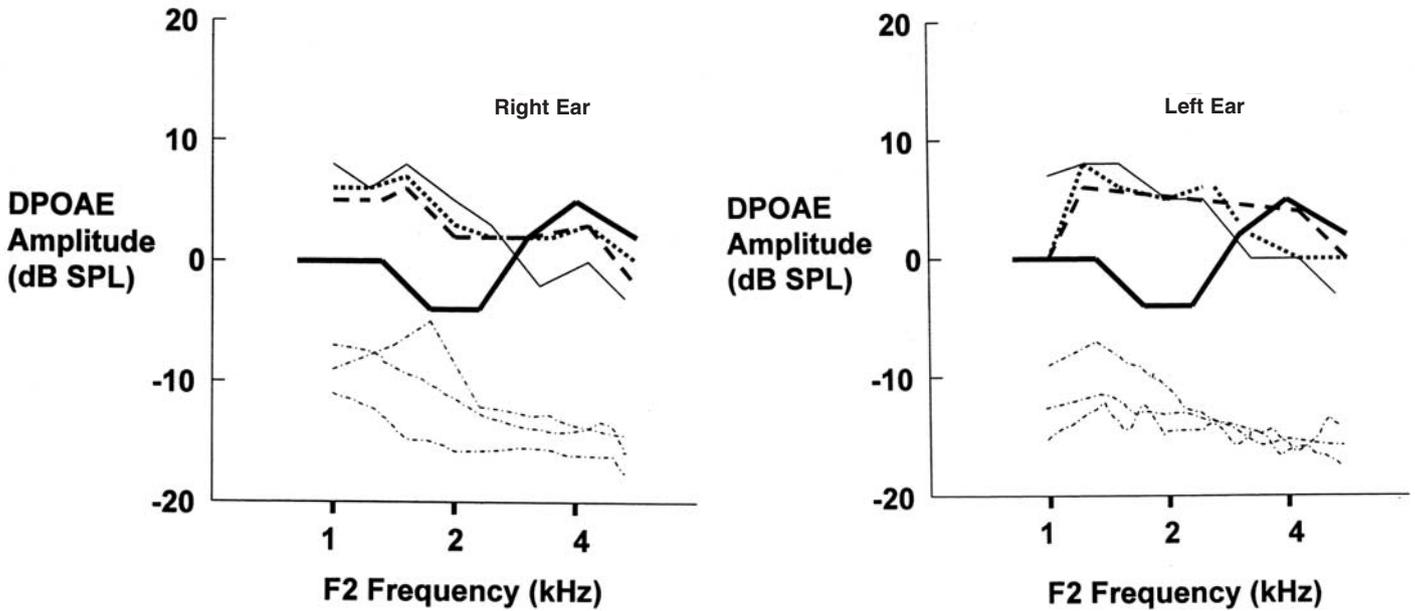
at the high frequencies (see Figure 3). At the second visit (nine months post-incident), the patient's audiogram demonstrated considerable improvement in threshold sensitivity at 3000 and 4000 Hz; however, no additional improvement was noted in threshold measures at the time of the third visit (15 months post-incident).

Speech audiometry including spondee thresholds and speech recognition testing was conducted during each of the three audiological visits previously mentioned. Speech audiometry measures for the left ear were within the normal range for all three visits. At the first visit, there was very poor agreement between the pure-tone average

(PTA) and spondee threshold for the right ear. This spondee threshold and the subsequent two spondee thresholds were established using selected spondees due to the patient's severely compromised speech intelligibility (see Table 1). Follow-up testing during the second and third visits revealed good agreement between the PTA and spondee thresholds for the right ear; however, speech recognition abilities remained severely compromised for the right ear. Some improvement in speech recognition performance was noted on the second visit (from 0% at three months post-incident to 12% at nine months post-incident); however, this improvement may not represent a

**Table 1. Pure-Tone Averages (PTA), Speech Recognition Thresholds (SRT), and Speech Recognition Scores (SR) for the Right Ear for Three Audiological Evaluations**

Visit Number	PTA dB HL	SRT dB HL	SR (% correct)
Visit #1 (3 months post-incident)	35	85	0
Visit #2 (9 months post-incident)	33	35	12
Visit #3 (15 months post-incident)	30	25	16



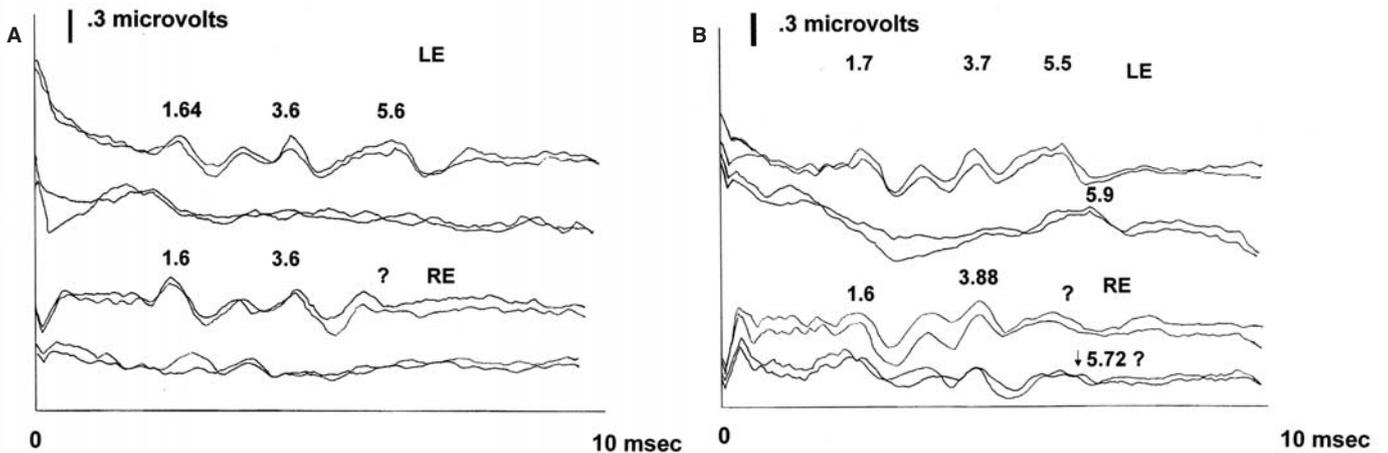
**Figure 4.** Distortion product otoacoustic emissions for the right and left ears. The thin solid line indicates the results obtained during the first audiological visit, with the dashed and dotted lines representing test results obtained during the second and third visits, respectively. The thick continuous line is the 90th percentile level. The thin dashed-dotted lines represent the noise floors for the three test sessions.

significant change in performance (Thornton and Raffin, 1978).

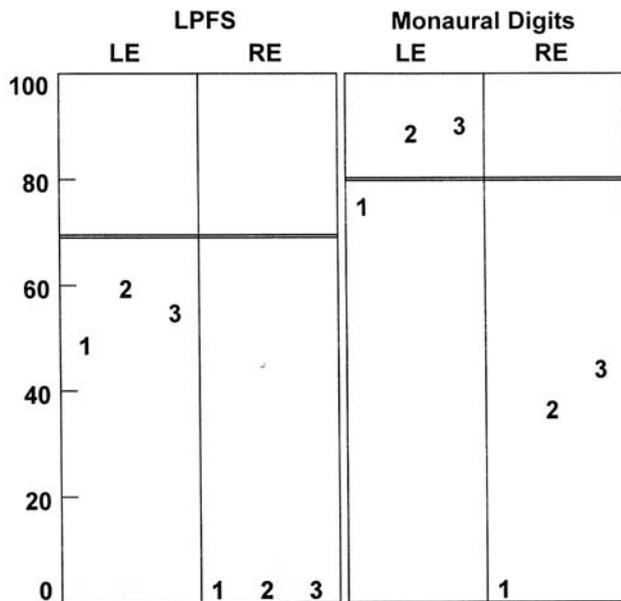
The DPOAEs were similar for right and left ears showing normal intensity levels in the 1000 to the 3000 Hz region and normal signal-to-noise floor measures throughout the frequency range tested bilaterally (Musiek and Baran, 1997). The DPOAEs derived for the right ear were better than one would have expected based on the pure-tone test results obtained at 1000 to 3000 Hz for all three visits (Figure 4). The DPOAEs for the left ear were normal and in agreement with

the audiometric test results for frequencies 1000 to 3000 Hz for all visits (Figure 4).

Tympanograms and acoustic reflex test results were similar for all three visits. The tympanograms were of normal pressure, static acoustic admittance, and shape bilaterally. Ipsilateral acoustic reflexes were present at normal levels for the left ear for frequencies 500, 1000, and 2000 Hz and were absent at the limits of the equipment (105 dB HL) for these frequencies for the right ear. Contralateral reflexes at 500, 1000, and 2000 Hz were absent for both ears at the limits of our clinical protocol (110 dB HL).



**Figure 5.** Auditory brainstem responses (ABRs) for the first (A) and second (B) audiological visits. The bottom tracing for each ear was at the high repetition rate. The numerical values are in milliseconds.



**Figure 6.** Central auditory tests results for three audiological visits (three months post-incident = 1, nine months post-incident = 2, 15 months post-incident = 3). Numbers on the left side of the graph represent percentage correct. Key: LPFS = low-pass filtered speech, RE = right ear, LE = left ear.

The initial ABR test (three months post-incident) derived for the left ear at the lower repetition rate was normal, whereas the right ear ABR at the low repetition rate appeared to have a missing wave V with wave IV present (Figure 5). In addition, the right ear amplitude of the IV-V complex was smaller than that of wave I (this measure was obviously influenced by the absence of wave V). At the high repetition rate, the left ear showed no response for any waves, and the right ear demonstrated only waves I and II. On the second visit (nine months post-incident), the ABR for the left ear at the low repetition rate remained normal, and the ABR for the right ear at this same repetition rate was determined to be normal, with the exception of a meager (reduced amplitude) IV-V complex. The amplitude of the IV-V complex for the right ear was less than one-half the amplitude of wave I. For the high repetition rate, a IV-V complex emerged with a normal latency shift for the left ear, and the right ear showed waves I-V within the normal latency range, although they were not highly replicable. The results of ABR testing conducted at the time of the third visit (15 months post-incident) were essentially

unchanged from those obtained at the second visit.

The behavioral central auditory tests demonstrated better performance for the left ear as compared to the right ear for both tests at all three visits (Figure 6). However, even though the performance of the left ear was superior to that of the right ear, the left ear scores obtained on the LPFS test during each of the three visits fell below the cut-off for normal performance. In addition, there were no obvious changes in the performance of either ear on the LPFS test over the three audiological visits. The right ear deficits on this test are not surprising given the poor speech recognition scores that were noted for the right ear during each of the three audiological visits (see Table 1); however, the LPFS scores for the left ear are not consistent with the results of speech recognition testing for the left ear (i.e., normal speech recognition test results).

The patient demonstrated some improvements on the monaural digits test on the second and third visits—especially for the right ear. There remained, however, a major difference between the ears as the right ear scores, although improved, remained well below the norms for this test at 15 months post-incident. The left ear was either near or within the normal range of performance for all three visits. The more significant improvements on the monaural digits test when compared to the negligible improvements observed for the speech recognition and LPFS tests (especially for the right ear) over the three assessment visits was likely a reflection of the relative simplicity of this closed set test.

## DISCUSSION

This case presents several interesting points worth discussion and comment. Perhaps the best starting point is with the presenting symptomatology. The initial symptoms experienced by this patient were auditory in nature. Decreased hearing and tinnitus in both ears were the first indicators that heralded a serious problem of the central nervous system. These early symptoms also provided some indication that the problem was bilateral—although formal hearing testing later indicated considerably more involvement on the right side. It is interesting to note that involvement of the central

auditory system yielded apparent problems with hearing sensitivity, an observation most often associated with involvement of the auditory periphery.

The anatomy related to the radiology and pathophysiology in this case is both instructive and curious. The radiology indicated that there were two anatomical areas of involvement in the caudal aspect of the pons. One area of compromise involved a bleed into the fourth ventricle with some extension ventrally into the right dorsal aspect of the pons. The anatomy related to this area of involvement could mean that the dorsal stria, the facial nerve nuclei and/or its fiber connections, and the vestibular nuclei, especially on the right side, may have been compromised. These anatomical lesion sites could be correlated to the patient's facial paralysis, imbalance, lack of acoustic reflexes, and perhaps to a lesser extent, the abnormal ABR and central auditory test findings. The other area of involvement was the pontine region of the AVM. A bleed from the AVM could have had effects, such as "vascular steal," beyond the anatomical locus in the pons. Since such effects frequently do not show up on CT scan, it is difficult to determine how much of an effect this vascular problem may have had beyond the anatomical region documented by the CT scan (i.e., in the region of the fourth ventricle) (De Reuck et al, 1989).

As best as could be determined, it appeared that the auditory periphery in this patient was intact for the most part. Normal hearing sensitivity was established for the left ear at all test frequencies, with the exception of 8000 Hz, during all three visits. The audiometric test findings did show a significant hearing loss for the right ear at all three visits, but these findings were inconsistent with the DPOAE test results, at least for frequencies below 3000 Hz. In addition, the ABR test results derived at each of the three visits demonstrated normal waves I and II bilaterally. The dramatic effects noted on the pure-tone threshold and speech recognition tests in this patient could be related to the severity and extent of the damage in the auditory brainstem. It has been suggested that cochlear nucleus involvement can affect ipsilateral pure-tone thresholds (Matkin and Carhart, 1966; Dublin, 1986). Masterson and colleagues (1992) have shown that mild shifts in detection thresholds can occur in animals

following sectioning of one of the lateral lemnisci. In an earlier paper in this special issue, it was shown that severe damage to the inferior colliculi resulted in major deficits in pure-tone sensitivity (Musiek et al, 2004). Therefore, it seems possible that severe involvement of the auditory brainstem pathway could result in alterations of the pure-tone audiogram. This possibility is further supported by the fact that the pure-tone thresholds in this patient improved along with noted improvements on the ABR and central auditory tests.

An alternative explanation for the pure-tone findings could be that there may have been some form of vascular disruption that indirectly restricted the blood flow to the auditory periphery through the internal auditory artery. If this were the case, the cochlea or auditory nerve may have been compromised. Although this notion is possible, it is probably unlikely as there was no reported involvement of the anterior inferior cerebellar artery or internal auditory artery, and the DPOAE results were essentially normal. Therefore, it is more likely that this case represents a situation where severe low brainstem compromise influenced pure-tone thresholds and speech recognition ability.

The acoustic reflex test results showed that only ipsilateral stimulation to the left ear yielded a response. This finding has anatomical and pathophysiological support in what is known about neural substrate of the acoustic reflex arc (Borg, 1973). Given the probable involvement in the area of the right facial nerve nuclei as well as auditory fibers that enter and exit this area, it would seem likely that the only response noted would be the left ipsilateral reflex. The right ipsilateral reflex would be absent because of the probable compromise of the right facial nerve nuclei and surrounding neural substrate given the lesion site. For the same reason, both right and left ear contralateral reflexes would be absent. Input to the left auditory nuclei/pathways and output from the left facial nerve nuclei was probably unaffected by the lesion in this case, thus permitting the activation of the left ipsilateral reflex arc.

The ABR findings in this patient are curious. At slow rates of presentation, the left ear ABR is normal but the right ear ABR appeared to have an absent (or severely attenuated) wave V. Also the V-I amplitude ratio for the right ear comes into question

(Musiek and Lee, 1995). In this case, with anatomical compromise in the area of the pons as defined by the CT, one might assume that the auditory nerve and cochlear nuclei would be sufficiently intact to generate waves I, II, and III. A previous case of a low, midline pontine lesion showed similar test findings, that is, normal waves I, II, and III with abnormal IV-V waves (Musiek et al, 1988). At the high rates (first visit), the waveforms for both ears deteriorated. Waves I and II were present for the right ear, but no waves were present for the left ear. One might question whether it is possible that the refractory period of the neurons responsible for generating the ABR in this patient might have been extended from a general neural insult to the point that at higher rates the ABR became desynchronized. At the second visit, there was some recovery noted primarily at the high repetition rate. This improvement was consistent with the subjective and measured improvement of the hearing abilities of this patient at the second visit. This noted improvement could be related to increased metabolism of relevant auditory fibers, which resulted in improved neural synchronization.

The ABR high rate study supports the possibility that there may have been some involvement on the left side of the brainstem in addition to the right side. This possibility is also supported by the LPFS test results, which showed depressed performance for both ears, but with better performance noted on the left side. The greater right-sided deficit on this test as well as the monaural digits test is consistent with previous findings that support the following interpretations. If the site of lesion is in the low/mid pons and lateralized, behavioral central tests will show the greater deficit ipsilaterally (Musiek and Guerink, 1982; Musiek et al, 1988). Conversely, if the site of involvement is in the high pons or above, the greater involvement is likely to be observed for the contralateral ear (Jerger and Jerger, 1981).

The auditory training that the patient did in her home was an activity that she actually enjoyed doing and that she was convinced was helping facilitate her recovery of auditory function. The patient also used the ALD that was recommended in a variety of listening situations, experimenting with both monaural and binaural listening conditions. The ALD did marginally improve her communication

ability during the first few months post-medical incident. However, as her hearing abilities improved over time, her use of this device decreased. Objectively, it would be difficult to determine if the auditory training did increase her auditory processing abilities because there may have been some spontaneous recovery that may have occurred over the period of time that this individual was followed. The patient was initially seen about three months after her vascular accident. Therefore, it is quite possible that spontaneous recovery played a role in her improved auditory functioning. The fact that little change was noted in auditory performance between the second and third visits even though the auditory training was continued would argue that spontaneous recovery played a significant role in the early improvement of auditory function in this patient.

## SUMMARY

This clinical research report focuses on some important audiological correlates to a lesion site in a patient who incurred damage to the caudal pons secondary to an AVM and vessel rupture. The initial signs of the vascular accident were clearly auditory in nature, with the more cardinal symptoms of headache and loss of consciousness following the auditory symptoms. Audiological assessment demonstrated an abnormal audiogram that was likely related to severe auditory brainstem pathway compromise and not peripheral hearing loss. This clinical impression was supported by the results of acoustic reflex, ABR, and central auditory testing, which were found to be consistent with those typically noted in patients with brainstem involvement. Auditory recovery was documented on both behavioral and electrophysiological tests of auditory function secondary to spontaneous recovery and possibly auditory training.

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