Bilateral Idiopathic Sensorineural Hearing Loss following Dental Surgery

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Abstract

Background: This is a case study of an 18-year-old female who suffered a bilateral idiopathic sensorineural hearing loss that was coincident with the removal of four impacted wisdom teeth. Throughout childhood the patient had normal hearing for pure tones bilaterally as measured at the pediatrician’s office. One month prior to dental surgery (May) the patient volunteered to participate in an auditory experiment at which time her pure-tone audiogram was normal. Immediately following surgery (June), the patient had substantial swelling of the face and complained of some hearing loss with no other auditory/vestibular complaints. The following month (July) during the course of a routine physical examination a pure-tone audiogram revealed bilateral, air-conduction thresholds of 30–35 dB HL (500–4000 Hz) and 20 dB HL (8000 Hz). Because bone conduction was not tested, it is impossible to know whether the hearing loss was conductive, mixed, or sensorineural. The pediatrician thought that the hearing loss was conductive and would resolve as the edema subsided.

A month later (August) the subject again volunteered for an auditory experiment at which time her hearing again was tested.

Purpose: The purpose of this report is to detail the dental procedures involved in the extraction of the wisdom teeth, to report the results of a variety and series of post-op hearing tests, and to discuss the possible mechanisms that might be involved in the “idiopathic” bilateral sensorineural hearing loss.

Research Design: Case report.

Results: During the August visit to the laboratory, hearing for pure tones bilaterally was 0 to 5 dB HL at 250–1000 Hz with a 40–45 dB HL notch at 2000 Hz with a return to 10 dB HL at 8000 Hz. Air conduction and bone conduction thresholds were equivalent. Word recognition in quiet was ≥92 percent correct for both ears, whereas the signal-to-noise ratio (SNR) hearing loss measured with the Words-in-Noise test was high normal in the left ear with a mild SNR hearing loss in the right ear. Tympanometry and acoustic reflex thresholds were normal. Distortion product otoacoustic emissions were reduced in the 1000–3000 Hz region for both ears, which is consistent with cochlear hearing loss. The hearing loss has remained unchanged for the past 19 months.

Conclusions: The possible etiologies, including insults to the cochlea by vibration trauma and through alterations in the blood supply to the cochlea, are considered.

Key Words: Auditory perception, hearing loss, idiopathic bilateral sudden sensorineural hearing loss, speech perception, word recognition in multitalker babble

Abbreviations: AC = air conduction; ART = acoustic reflex threshold; BC = bone conduction; BPPV = benign paroxysmal positional vertigo; DPOAE = distortion product otoacoustic emissions; ISSHL = idiopathic sudden sensorineural hearing loss; SNHL = sensorineural hearing loss; SNR = signal-to-noise ratio; TMJ = temporomandibular joint; WIN = Words-in-Noise test

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idiopathic sensorineural hearing loss is a term applied to a hearing loss whose etiology is unknown. A sudden sensorineural hearing loss (SNHL) typically occurs either instantaneously or over a period of hours. Typically, the idiopathic sudden sensorineural hearing loss (ISSHL) is unilateral, with a flat pure-tone threshold configuration around 60 dB HL and poor word-recognition performance in quiet (e.g., <20% correct). Hearing loss of this type is associated with a variety of causes including virus, vascular problems, membrane rupture, otologic surgical procedures, and nonotologic surgical procedures, most notably heart bypass surgery (Millen et al, 1982). Sudden-onset SNHL is not a disease but, rather, a symptom of an insult to the cochlea (Hallberg, 1956; Fetterman et al, 1996).

HISTORY

This is a case report of an 18-year-old female who had bilateral ISSHL coincident with or following extraction of her wisdom teeth. The history is unremarkable except that the patient was diagnosed with a seizure disorder when she was 12 years old. For the past six years the seizures have been controlled with Lamictal. The patient experienced the usual childhood illnesses including repeated ear infections that always were treated successfully. A series of audiograms from the pediatrician documented that hearing for pure tones over the years was normal. The patient was aware of the harmful effects that noise can have on the auditory system and behaved accordingly. The patient is a flautist and as such has some history of exposure to loud sounds generated in the wind ensemble and band (McBride et al, 1992). On May 4, 2007, the patient participated in an auditory experiment involving computerized recognition/scoring of verbal responses to word-recognition materials. This experiment was monaural, which is why only the right-ear audiogram is available from that date. As can be seen in Figure 1, the hearing in her right ear was normal (≤20 dB HL [American National Standards Institute, 2004]).

DENTAL PROCEDURE

On June 22, 2007, the patient underwent light general anesthesia and removal of four impacted wisdom teeth. In the operating theater the patient was placed in the appropriate position with her head, neck, and back in a virtual straight line throughout the procedure. The operating table allowed for flexion at the waist, which ranged from 0 to 30 degrees for the entire procedure. The patient was prepped and draped in the usual fashion while breathing 7 l/min of oxygen via a nasal mask. Nitrous oxide was titrated over several minutes until it reached 70 percent concentration. Vital signs (EKG, respiration, pulse, and blood pressure) were monitored and remained within normal limits. An IV then was started in the right antecubital area with a 22 gage angiocath. At this point an adult rubber bite block was placed between the left maxillary and mandibular dentition to allow access to her airway. Then Versed, Phenergan, Decadron, and morphine sulfate were titrated separately over the next several minutes. A gauze throat pack then was placed to protect the airway and lungs from blood, irrigation, and debris. The nitrous oxide was discontinued, and oxygen was increased to 7 to 8 l/min. Next a local anesthesia (2% Xylocaine with epinephrine times eight carpules, each with 1.7 ml of local anesthesia) were injected into the four operative areas (one carpule in each maxillary area and three carpules in each mandibular area). Versed continued to be titrated, with propofol subsequently added. The patient was calm and in a light state of general anesthesia, that is, twilight sleep. The right maxillary and mandibular wisdom teeth then were removed in the usual and customary fashion using the #15 scalpel blade, a #9 periosteal elevator, and a Hall Drill with a #8 round burr. The drilling lasted <30 sec/tooth. The teeth were sectioned and removed completely without incident and with minimal bleeding. The operative areas were irrigated thoroughly, and sutures were placed with 3.0 chromic. The retractor was removed, and the throat pack was placed over the operative area. In preparation for a mirror image procedure on the left maxillary and mandibular impacted wisdom teeth, the rubber bite block was moved to the left side and another throat pack was inserted. The results on the left side were equally excellent, and closure was by the same method.

Figure 1. A right-ear audiogram obtained on May 4, 2007, prior to participation in a computerized word-recognition experiment.
The throat pack and all instrumentation from the oral cavity were removed, and bilateral gauze packs were placed over the operative sites. A head and neck ice pack was placed to help decrease postoperative swelling. The patient was tapped gently on the forehead, at which time she began to respond. After a satisfactory time for recovery she was discharged in satisfactory condition. The total medications used were 10 mg Versed, 4 mg Decadron, 12 mg Phenergan, 5 mg morphine sulfate, 130 mg propofol, and eight carpules of 2 percent Xylocaine with epinephrine. The patient’s vital signs were stable throughout and upon discharge (130–145 systolic, 70–85 diastolic; pulse 62–80/min; respiration 17 to 22/min). There was less than 50 cm³ of blood loss. There were no untoward effects, and the patient tolerated the procedure well. During the immediate post-op period several medications were prescribed for pain and swelling, including Oxycodo/apap (10–325 mg), Tramadol HCL (50 mg), Medrol (4 mg), and Promethazin gel (25 mg).

AUDIOLOGIC TEST RESULTS

Following the surgery there was noticeable edema, with the main complaints related to pain and discomfort with an occasional mention of hearing loss. There were no other auditory/vestibular complaints, including no complaint of tinnitus. On July 3, 11 days following surgery, an air-conduction audiogram was obtained during a routine physical examination by the pediatrician (see Figure 2). The audiogram revealed bilateral, air-conduction thresholds of 30–35 dB HL (500–4000 Hz) and 20 dB HL (8000 Hz). Unfortunately, bone conduction was not tested. At that time the edema was still apparent but substantially reduced, which led the pediatrician to attribute the hearing loss to a conductive problem that she thought would resolve with dissipation of the edema. Although the pure-tone configurations in the July 3 audiogram are consistent with conductive hearing loss bilaterally, it is possible that the hearing losses at that time were mixed or even sensorineural. Over the next several weeks, the patient reported that her hearing improved, and by the end of July the edema was gone and she was not aware of a hearing loss.

On August 2, the patient volunteered to participate in another auditory experiment at which time her audiograms (Grason-Stadler, Model 61), which are depicted in Figure 3, revealed 0 to 5 dB HL thresholds at the frequency extremes with a notched SNHL of 40 to 45 dB HL from 1500 to 3000 Hz. Serial audiograms over the next 19 months (August 2007 to March 2009) indicated no change in hearing. Admittance, 226 Hz tympanograms were normal, and ipsilateral acoustic reflex thresholds at 500, 1000, and 2000 Hz were present bilaterally at normal levels (Grason-Stadler, Model TympStar).

The results from word-recognition testing in quiet and in noise on the Northwestern University Auditory Test No. 6 materials (Department of Veterans Affairs, 2006) are shown in Figure 4. In quiet, performances were excellent, ranging from 92 to 100 percent correct at 60 and 84 dB HL. The Words-in-Noise (WIN) test was used to evaluate recognition performance in

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**Figure 2.** Air-conduction audiograms for the right (O) and left (X) ears obtained by the pediatrician during a routine physical examination 11 days following the dental procedure (July 3, 2007). Bone conduction was not tested.

**Figure 3.** The audiogram from August 2, 2007, that is representative of the serial audiograms over a ten-month period from August 2, 2007, through June 16, 2008. The ∕ represents the ipsilateral acoustic reflex thresholds.
multitalker babble (Wilson, 2003; Wilson and McArdle, 2007). The WIN assesses performance at seven signal-to-noise ratios (SNRs) from 24 to 0 dB in 4 dB decrements. The SNR at which the 50 percent correct point occurs is the metric of interest that is used to define the SNR hearing loss. The 50 percent point for the WIN, which is calculated with the Spearman–Kärber equation (Finney, 1952; Wilson et al, 1973), on young adult listeners with normal hearing is \( \leq 6 \) dB SNR (Wilson et al, 2003). The shaded regions in Figure 4 indicate the ranges of normal performance on the WIN at the various SNRs. The performance by the patient on the WIN indicated normal function (albeit high normal) on the materials presented to the left ear (50% point = 6.0 dB SNR) and a mild SNR hearing loss with the materials presented to her right ear (50% point = 7.6 dB SNR). Interestingly, the recognition performances by the patient on materials presented (independently) to both ears were excellent at the higher SNRs with poorer-than-expected performances at the less favorable SNRs. Typically a young adult with normal hearing has a 50 percent point in the 0 to 3 dB SNR range with the WIN datum points at the respective SNRs well within the shaded regions on the graphs in Figure 4.

During an evaluation on August 17, 2007, distortion product otoacoustic emissions (DPOAEs) were measured (Biologic, Model Navigator Pro) using two frequencies/octave, two levels (\( L_1 = 65 \) dB SPL, \( L_2 = 55 \) dB SPL), and an \( f_1/f_2 \) of 1.22. The \( 2f_1 – f_2 \) DPOAE amplitudes obtained at eight \( f_2 \) frequencies from 750 to 8000 Hz are depicted as circles in Figure 5 for the left ear (top panel) and right ear (bottom panel). The triangles represent the associated noise floors. The abnormally low DPOAE amplitudes bilaterally in the 1500–3000 Hz region are consistent with cochlear hearing loss.

**DISCUSSION**

Although the hearing loss incurred by the patient could be unrelated to the dental procedure, the likelihood is that the hearing loss is in some way linked to the removal of the wisdom teeth, which was the only seminal event that occurred between the May 4 audiogram (normal hearing) and July 3 audiogram (hearing loss).\(^1\) The exact cause of the hearing loss is impossible to determine, hence the “idiopathic” descriptor. Two aspects of the hearing loss are intriguing. First, the hearing loss is bilateral and almost symmetrical. The incidence of bilateral sudden-onset SNHL is low, typically \(<5\) percent of the reported sudden-onset cases (Shaia and Sheehy, 1976; Mattox and Simmons, 1977; Oh et al, 2007). Second, the pure-tone thresholds exhibit a “signature” configuration with a sensorineural notch in the 1500 to 3000 Hz range. The question is, Whose signature, or what is the etiology?
Because of the bilateral and symmetrical nature of the hearing loss that affected the patient, the causal mechanism must be one that occurred concurrently to the ears. The two general mechanisms that are possible are hearing loss induced by noise (Schuknecht and Tonndorf, 1960) and hearing loss related to some aspect of the blood supply to the cochleae (Pearlman et al, 1959; Mom et al, 2005).

Typically noise-induced hearing loss is caused by a rapid, substantial change in barometric pressure that affects the basal end of the cochlea via the normal transmission route from the outer ear through the tympanic membrane and middle ear to the cochlea. The abnormally large pressure of the traveling wave propagated along the basilar membrane damages the hair cells in the basal end of the cochlea that is manifested by a “noise notch” in the air-conduction audiogram in the vicinity of 4000–6000 Hz. Over the years, noise in the dental office has been the topic of a plethora of studies, which for the most part have been directed at the hearing of dentists and the various dental assistants (e.g., Forman-Franco et al, 1978; Zubick et al, 1980; Setcos and Mahyuddin, 1998; Hyson, 2002). The relationship between noise in the dental office from drilling and so on and hearing loss in dental personnel is tenuous at best. Brusis et al (2008) recently reported that the average noise level in dental offices was between 70 and 78 dB(A), with some rare peaks >80 dB(A). Interestingly, Brusis et al found that aspirators produced the most intense noise in dental offices, not drilling instruments.

In the current case, in which the “notch” in the audiogram is in and around 2000 Hz, the route of the possible “acoustic trauma” (that is, actually vibration induced) is via bone conduction from the teeth through the skull bones to the cochleae. Cochlear hearing losses purportedly caused via this bone-conduction route are evident in the literature, with most related to drilling during various otologic surgeries (Schuknecht and Tonndorf, 1960; da Cruz et al, 1997). There is some agreement among the studies that drilling on the skull bones, most notably the temporal bone, can produce SNHL with the characteristic 4000 Hz notch in the air-conduction audiogram. The combinations of variables involved in this type of hearing loss are numerous, including duration, drill pressure, drill speed, drill bit type, and so on. Kylén and Arlinger (1976) measured the “equivalent air-borne sound levels” using accelerometer data generated on cadavers by a 20,000 rpm drill with a 6 mm burr without irrigation. Their ipsilateral data indicated frequency-dependent levels that ranged from 90 to 100 dB equivalent airborne sound levels with the higher levels in the higher frequencies, namely, 2000 and 4000 Hz. Contralateral measures were only a few decibels lower. In contrast to those studies that report some relationship between drilling on the skull bones and hearing loss, Tos et al (1989) report no contralateral SNHL in 50 consecutive patients who underwent surgery for acoustic neuromas using the translabyrinthine technique. Similarly, in a prospective study of 40 patients, Urquhart et al (1992) conclude that any SNHL following mastoid surgery was not caused by drill-generated trauma to the cochlea.

Sensorineural hearing loss has been generated in animal models using vibrating probes of various characteristics to induce skull bone vibration that in turn produces hearing loss (e.g., Zou et al, 2001; Sutinen et al, 2007). Although hearing loss has been demonstrated in these models, the results are inconclusive with respect to the many variables that are involved.

Several lines of reasoning, however, do not support the notion in the current case that bone-conducted transmission of vibrations produced hair cell damage. First, the notch in the air-conduction audiogram is at 2000 Hz, not at the characteristic 4000 Hz. Here, the unanswered question is: In humans, does “acoustic trauma” transmitted via bone conduction produce hair cell damage in the same region of the cochlea that is affected by acoustic trauma transmitted via the traditional air-conduction route to the cochlea? Second, the noise induced by the drilling was minimal, lasting <30 sec/tooth. Although the effective amplitude of the bone-conducted vibration from the teeth through the skull bones to the cochlea is unknown, it is doubtful that the amplitude and duration of the vibrations mediated through a tooth exceeded the amplitude of the vibrations caused by drilling directly on the temporal bone. Third, the effects of hearing loss (at 2000 Hz) induced in this manner should be reported widely as the number of patients exposed to dental drills is substantial, but these reports are lacking.

In general, ISSHL affects one ear and the hearing loss is extensive in both the spectrum and level domains (Schuknecht and Donovan, 1986). Characteristic of a sudden-onset SNHL is a flat pure-tone threshold configuration around 60 dB HL with poor word-recognition performance in quiet, typically <20 percent. The three most commonly suggested etiologies for ISSHL are vascular lesions, membrane breaks, and viral infection. Schuknecht and Donovan suggest that partial obstruction of arterial or venous flow to the cochlea could produce anoxia that would result in fluctuations in cochlear function. Simmons (1968) championed membrane breaks that produced widespread cochlear damage. Finally, the effects of viral infections on auditory function are well documented, especially with mumps, measles, maternal rubella, and upper respiratory infections. Of the three etiologies, vascular seems the most logical in the current case, with possible factors including anoxia owing to reduced blood flow to the cochlea through head and jaw positioning during the procedure or a blood-borne
agent that reduced the supply of oxygen to the cochleae possibly through the general anesthesia or the dental procedure itself. Ototoxicity was considered, but the medications involved both alone and in combination were not considered potentially ototoxic.

Mid-frequency SNHL (1000–2000 Hz) also has been associated with temporomandibular joint (TMJ) problems. Baldursson and Blackmer (1987) report that of the 50 patients studied with TMJ problems, 60 percent had bilateral mid-frequency notches. Further, many patients with TMJ problems had substantial sensorineural notches while not admitting to any hearing loss. No causal relation between TMJ problems and hearing loss are advanced by the authors. The potential relation between these described TMJ problems and the current case is that during the dental procedure the head was positioned slightly back and the mouth was held open with a rubber bite block. There is a possibility that while in this position the blood supply to the cochleae could have been restricted. Tan et al (2007) report one case of unilateral sudden SNHL following a dental procedure. Initially the air conduction thresholds were somewhat flat at 45 to 60 dB HL with a low-frequency air–bone gap. Three days later some recovery had occurred characterized by a 50 dB HL sensorineural notch at 1000 Hz with a continued low-frequency air–bone gap. Although this is a case of idiopathic hearing loss, the authors felt that the most probable cause was a circulatory disruption to the cochlea caused by microemboli. Evan et al (1997) indicate that at the time of their article there were only 15 cases of sudden-onset SNHL reported in which general anesthesia was the reported culprit (heart bypass cases were excluded). The Evan et al study adds three cases, all of whom exhibited unilateral SNHL, with two of the cases having predominately hearing loss in the low and mid frequencies.

Four cases of unilateral “deafness” following dental surgery are reported by Farrell et al (1991), who suggest that a relation between dental surgery and sensorineural hearing deficits had not been previously reported. Farrell et al indicate that the only commonalities among the four patients were “opening the jaw widely and undergoing a dental procedure.” Several causes of the hearing loss are offered, including the release of microemboli that potentially could include pathogenic bacteria, the use of vasoconstrictors that could cause vasospasm of the cochlear branch of the internal auditory artery, and a neurogenic reflex involving the trigeminal nerve that Brennan (1991) suggests might originate from the TMJ owing to prolonged opening of the mouth during dental procedures. The bilateral, symmetrical characteristic of the hearing losses in the current case, however, argue against microemboli as the etiology. The possibility of having two emboli independently affect (almost identically) the cochlea would appear to be remote.

This report ends in the same manner in which it started, namely, with bilateral ISSHL. The case may be the first such case reported with the bilateral hearing loss concurrent with or following a dental procedure. The documentation in this case of the hearing loss is very good, with a normal audiogram one month prior to surgery, a patient complaint of hearing loss immediately after surgery, and an abnormal audiogram 11 days after surgery. For the low-frequency range, hearing returned to normal within six weeks of surgery, leaving the patient with the false impression that she had no hearing problem when, in fact, thresholds in the vicinity of 2000 Hz were 40 to 45 dB HL. The mid-frequency notch characteristic of the hearing losses produced functional handicaps that are subtle and essentially unnoticeable by the patient. Hopefully this report will alert audiologists to the existence of such idiopathic hearing loss, which in turn will be reported in future literature.

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NOTES

1. Although there are numerous references in the literature about sudden-onset hearing loss, the majority of reports, which are mostly in the medical literature, simply use the terms hearing loss and deafness to describe the auditory deficits of their sample. Unfortunately, few audiograms are shown or described. In the early literature, hearing loss and deafness were used interchangeably, making it difficult to assume a degree of hearing loss, except when total deafness was used as the descriptor.

2. Although it was difficult to establish a substantial cause-and-effect link, recently, Chiarella et al (2008) has reported that following certain types of dental surgery (e.g., tooth extractions), benign paroxysmal positional vertigo (BPPV) was of “suspected iatrogenic origin.” Traumatic insult in close proximity to the posterior semicircular canal was thought to be the culprit that caused the BPPV.

REFERENCES


