Patterns of Hearing Loss Following Retrosigmoid Excision of Unilateral Vestibular Schwannoma

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Abstract

Objectives To determine the pattern of auditory responses, time-course of hearing deterioration, and possible site of lesion following retrosigmoid excision of unilateral vestibular schwannomas.

Design Prospective, nonrandomized, observational pilot study.

Setting Tertiary referral medical center.

Main outcome measures Preoperative and postoperative pure-tone and speech audiometry, auditory brainstem response testing, and distortion product otoacoustic emissions were performed in 20 patients. Testing was conducted every 24 hours for the duration of hospitalization. Transtympanic electrocochleography was performed if delayed deterioration of auditory responses was documented.

Results Of the 20 patients, 7 had no discernible cochlear nerve at the end of the procedure. Of the 13 patients with an intact nerve, 6 retained hearing, 3 with evidence of reduced neural function. Of the 7 who lost hearing despite an intact nerve, 5 lost at least cochlear and possibly also neural function, and 1 had reduced neural function but retained cochlear function. There were two examples of delayed deterioration of cochlear nerve responses.

Conclusions Hearing loss following retrosigmoid removal of vestibular schwannomas most often involves loss of at least cochlear function, possibly in addition to neural damage. In a smaller number of cases anacusis or hearing deterioration can be attributed to purely neural trauma.

Keywords

- ▶ acoustic neuroma
- ▶ vestibular schwannoma
- ▶ hearing preservation

Introduction

Anacusis following vestibular schwannoma excision may occur despite apparent cochlear nerve preservation. It is believed to result from trauma to the cochlea or cochlear nerve or interruption of the vascular supply to either structure.¹ Electrophysiological measures of both hair cell and cochlear nerve responses can be used to differentiate between the cochlear and neural components of hearing loss

and determine the likely site of operative trauma causing hearing loss. Such information is vital in developing therapeutic interventions that minimize hearing loss by targeting the sites most vulnerable to trauma.

Separation of the sensory and neural components of hearing loss in patients with vestibular schwannomas has been previously accomplished using evoked otoacoustic emission (OAE) testing.^{2,3} OAEs have been shown to reflect preneural outer hair cell mechanisms; therefore, their

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presence in an ear with sensorineural hearing loss suggests, by exclusion, that the loss is of primarily neural, as opposed to cochlear, etiology. Following tumor removal, a loss of outer hair cell function may result from damage to the otic capsule or vascular compromise of the internal auditory artery or smaller arterial vessels supplying the cochlea.^{1,4,5}

Postoperative neural hearing loss, demonstrated by an absence or prolongation of latency of the peaks of the auditory brainstem response (ABR), may result either from an inability to maintain an intact cochlear nerve or through more subtle intraneural hemorrhaging and axonal disruption.⁶ Although the nerve may appear to be anatomically preserved at the conclusion of surgery, structural changes resulting from manipulation of the nerve may cause total loss or deterioration of hearing acuity.⁶ Transient or permanent interruption of the vascular supply to the cochlear nerve may also cause functional deterioration.^{1,4,6-8}

The objective of this pilot study was to determine the site of any surgical trauma following retrosigmoid removal of vestibular schwannomas. We aimed to assess the functional integrity of the cochlea and cochlear nerve using behavioral and electrophysiological measures of auditory function. These data were recorded in sufficiently fine time resolution to document any progressive changes in auditory function, especially in the early postoperative period. We report data from 20 patients and discuss two cases in detail that illustrate a deterioration of cochlear nerve responses in the early postoperative period.

Methods

Ethical approval was obtained from the Upper South A Regional Ethics Committee (Ref: URA/07/03/017) and the University of Canterbury Human Ethics Committee (Refs: HEC 2007/45 and HEC 2008/44).

Patients

Twenty patients (6 male, 14 female) undergoing unilateral vestibular schwannoma removal via the retrosigmoid approach at Christchurch Public Hospital between July 2007 and December 2008 participated in this study. The patients' ages ranged from 29 to 67 years with a mean age of 49.9 years. All patients had measurable hearing preoperatively, and cochlear nerve preservation was attempted in each case. Mean tumor size was 16.3 mm (standard deviation [SD] = 10.2 mm, range = 0 [i.e., intracanalicular] to 35.6 mm) based on the maximal axial diameter of the portion of the tumor in the cerebellopontine angle (CPA) on magnetic resonance imaging (MRI). The extent of lateral extension of the tumor was measured as the distance of the tumor from the fundus of the internal auditory canal (IAC) on MRI. The mean tumor distance from the fundus was 1.8 mm (SD = 2.3 mm, range = 0 to 8.1 mm).

Procedure

Preoperative and postoperative assessments included pure-tone audiometry (air- and bone conduction at 0.25 to 8 kHz), speech audiometry using the New Zealand

Recording of the Consonant Vowel Consonant (Revised AB) Word Lists (National Audiology Centre, Auckland, New Zealand), distortion product otoacoustic emissions (DPOAEs), and ABR. Preoperative assessments were performed the day prior to surgery in sound-treated rooms. Initial postoperative assessments were performed 0.5 to 3 hours postoperatively and included ipsilateral ABR and DPOAEs. ABR and DPOAE testing were then repeated at the bedside for both ears, where possible, ~24-hourly for the duration of hospitalization (range = 6 to 8 days, mean = 6.5 days). Pure-tone and speech audiometry were performed at the bedside as soon as the patient was able and, when possible, were repeated at 24-hour intervals for the remaining period of hospitalization.

The maximum word recognition score (WRS) was recorded as the percentage of words repeated correctly when stimuli were presented at a level no greater than 50 dB hearing level (HL) above the average threshold at 1 and 4 kHz. Pure-tone average (PTA) was calculated according to the guidelines of the American Academy of Otolaryngology-Head and Neck Surgery, as the average threshold at 0.5, 1, 2, and 3 kHz.⁹ Hearing was considered "serviceable" if the average PTA was 50 dB HL or less and the WRS was 50% or more.¹⁰ Statistical analyses of audiometric data were performed using a computer-based statistical software package (SPSS for Windows version 17.0, SPSS, Inc., Chicago, Illinois, USA).

The ABR was elicited using rarefaction clicks presented at a rate of 21.3/second via an Ear Tone ABR insert earphone (Etymotic Research, Inc., Elk Grove Village, Illinois, USA). Differential recordings were made between the high midline forehead and the ipsilateral earlobe using self-adhesive silver/silver chloride (Ag/AgCl) surface electrodes. Two averaged waveforms of 2,000 responses were obtained for stimuli presented at 80 dB normal hearing level (nHL). Contralateral masking was performed if necessary. Waveforms were inspected visually to determine the latency of waves I, III (data not shown), and V.

DPOAEs were tested using a commercially available otoacoustic emission system (Scout Sport Otoacoustic Emission System, Bio-Logic Systems, Inc., Mundelein, Illinois, USA) at 2 to 8 kHz in half-octave steps. Emissions were defined as present if the amplitude was greater than -10 dB and the signal-to-noise ratio was greater than 5 dB.

Transtympanic electrocochleography (ECoChG) was performed 12 to 16 weeks postoperatively using a commercially available measurement system (Amplaid MK15, Milan, Italy) if ABR had deteriorated during the early postoperative period. A differential recording was made between a sterilized Teflon-insulated stainless steel monopolar needle electrode positioned on the promontory and an Ag/AgCl surface electrode on the ipsilateral mastoid. The stimuli were 10 millisecond tone-bursts at 1 and 4 kHz (1 millisecond rise-fall time, alternating polarity, rate = 9.1/second) and 100 μ s clicks (alternating polarity, rate = 7.1/second), all presented at 90 dB HL via supra-aural headphones. Two averaged waveforms of 1,000 responses were obtained for each stimulus condition. Waveforms were visually inspected and the

Fig. 1 Patterns of preoperative auditory responses for all patients. Hearing was considered “serviceable” if the pure-tone average at 0.5, 1, 2, and 3 kHz was 50-dB hearing level or less and the word recognition score was 50% or more. Auditory brainstem response was recorded on the basis of identifiable waves, and distortion product otoacoustic emissions were defined as present if one or more emission was documented at any frequency from 2 to 8 kHz. CNT, could not test.

amplitude of the summing potential (SP) and compound action potential (CAP) measured.

Results

Preoperative results are shown in ►Fig. 1.

From the 20 patients who underwent vestibular schwannoma excision, 14 experienced immediate postoperative anacusis and 6 had measurable hearing postoperatively. The pattern of postoperative auditory responses for all patients is shown in ►Fig. 2.

In seven cases, the cochlear nerve was indistinguishable from the tumor and could not be preserved. As shown in ►Table 1, preoperatively five of these patients had no identifiable ABR, one had wave I only, and in one case both waves I and V were present.

Preoperative audiological assessment of all patients indicated a mean PTA at 0.5, 1, 2 and 3 kHz of 44 dB HL (SD = 23 dB HL) and WRS of 69% (SD = 36%). Hearing was defined as serviceable in 12 cases. The mean preoperative results for the hearing preserved group were 41 dB HL (SD = 20 dB HL) for PTA and 83% (SD = 21%) for WRS, compared with 46 dB HL (SD = 24 dB HL) for PTA and 63% (SD = 40%) for WRS for the postoperative anacusis group. Independent samples *t*-tests showed no significant difference between group means for either preoperative PTA ($p = 0.67$), or preoperative WRS ($p = 0.25$).

A summary of pre- and postoperative data for patients with preserved hearing is presented in ►Table 2. Postoperatively, the mean PTA in this group dropped by 20 dB to 61 dB HL (SD = 28 dB HL). The mean WRS decreased by 41% from preoperative levels to 43% (SD = 36%).

Fig. 2 Patterns of postoperative auditory responses for all patients. Auditory brainstem response (ABR) waveforms were recorded as present if either wave I (^a), V (^b), or both I & V (^c) were identifiable. Distortion product otoacoustic emissions were defined as present if one or more emission was documented at any frequency from 2 to 8 kHz. * indicates the cases in which ABR wave I was present immediately postoperatively but was absent at the time of discharge from the hospital.

Table 1 Summary of Preoperative and Postoperative Data for Participants Who Had Postoperative Anacusis Following Vestibular Schwannoma Removal

Case	Tumor size in CPA (mm)	Tumor distance from fundus (mm)	Preoperative					Postoperative		
			PTA (dB HL)	WRS (%)	DPOAEs	ABR I ABR V (ms)	Cochlear nerve intact?	PTA	DPOAEs	ABR I ABR V (ms)
3	35.6	8.1	75 ^a	10	Absent	NR NR	No	NR	Absent	NR NR
4	25.1	0	86	0	2–3 kHz	I = 2.10 NR	No	NR	Absent	NR NR
5	32.3	0	50	70	Absent	I = 1.75 V = 7.50	No	NR	Absent	NR NR
6	22.6	1.0	15	84	2–3 kHz	NR NR	No	NR	Absent	NR NR
7	IAC	0	13	100	2–8 kHz	I = 1.75 V = 5.63	Yes	NR	Absent	NR NR
8	20.0	1.0	63	7	8 kHz	NR V = 6.35	Yes	NR	Absent	NR NR
9	28.8	0	50	0	2–4 kHz	NR NR	No	NR	Absent	NR NR
11	7.6	4.0	48	93	Absent	I = 1.63 V = 6.10	Yes	NR	Absent	NR NR
13	29.6	0	61	70	8 kHz	I = 2.20 NR	Yes	NR	Absent	NR NR
14	17.3	1.9	68	64	8 kHz	NR NR	No	NR	8 kHz	NR NR
16	IAC	2.0	40	90	2–8 kHz	I = 2.55 NR	Yes ^b	NR	Absent	NR NR
17	6.0	1.5	6	100	2–8 kHz	I = 1.80 V = 6.50	Yes	NR	2–8 kHz	I = 1.88 ^c NR
19	7.5	0	26	87	4 kHz	I = 2.13 V = 6.70	Yes	NR	Absent	NR NR
20	19.4	1.5	43	100	4–8 kHz	NR NR	No	NR	Absent	NR NR

Abbreviation: ABR, auditory brainstem response; CPA, cerebellopontine angle; DPOAE, distortion product otoacoustic emission; HL, hearing level; IAC, internal auditory canal; NR, no response; PTA, pure-tone average; WRS, word recognition score.

^aNo response above 1 kHz, PTA calculated based on the patient's thresholds at 0.5 and 1 kHz only.

^bSubtotal tumor removal.

^cWave I present immediately at given latency postoperatively but disappeared during the early postoperative period.

As shown in **Table 2**, all patients with preserved hearing had tumors of less than 20 mm (CPA) diameter and the mean tumor diameter in the CPA in this group was 12.5 mm (SD = 1.7 mm). Mean tumor distance from the fundus in these six patients was 2.6 mm (SD = 2.4 mm), and one patient of the group had a tumor that extended to the fundus of the IAC. In the group of 14 patients who experienced anacusis (see **Table 1**) the mean tumor diameter in the CPA was 18.0 mm (SD = 11.9 mm) and 7 had tumors of less than 20 mm diameter in the CPA. Mean tumor distance from the fundus in these 14 patients who lost all hearing (the anacusis group) was 1.5 mm (SD = 2.2 mm). Six patients of the anacusis group had a tumor that extended to the fundus of the IAC and two had entirely intracanalicular tumors. An

independent samples *t*-test showed no significant difference in mean CPA tumor diameter or tumor distance from the IAC between the group of patients who experienced anacusis and the group that had measureable hearing postoperatively ($p = 0.28$ and $p = 0.35$, respectively).

Cases 2 and 17 both presented with ABR wave I immediately postoperatively, but the latency of this wave gradually increased until it was absent by the time of discharge. These cases are discussed in detail.

Case 2

Patient 2 was 57-year-old woman with a 14.9 mm (0 mm from the fundus of the IAC) right vestibular schwannoma. Preoperative audiometry showed a moderate U-shaped

Table 2 Summary of Preoperative and Postoperative Data for Participants Who had Measurable Hearing Following Vestibular Schwannoma Removal

				Preoperative						Postoperative	
Case	Tumor size in CPA (mm)	Tumor distance from fundus (mm)	PTA (dB HL)	WRS (%)	DPOAEs	ABR I ABR V (ms)	Cochlear nerve intact?	PTA	WRS (%)	DPOAEs	ABR I ABR V (ms)
1	10.3	1.0	26	100	2 kHz	I = 2.10 V = 8.34	Yes	60	64	2 kHz	I = 2.10 V = NR
2	14.9	0	51	91	Absent	I = 1.80 NR	Yes	87 ^a	0	Absent	I = 2.00 ^b NR
10	11.8	2.0	50	68	Absent	NR NR	Yes	88 ^c	0	Absent	NR NR
12	11.3	3.7	26	97	CNT	I = 1.88 V = 8.85	Yes	31	97	3–8 kHz	I = 1.95 V = 6.00
15	14.0	2.0	73	47	Absent	I = 1.30 NR	Yes	77	0	Absent	NR NR
18	12.7	6.7	20	97	2–4 kHz	NR V = 6.88	Yes	25	94	2–4 kHz	NR V = 7.17

Abbreviation: ABR, auditory brainstem response; CNT, could not test; CPA, cerebellopontine angle; DPOAE, distortion product otoacoustic emission; HL, hearing level; IAC, internal auditory canal; NR, no response; PTA, pure-tone average; WRS, word recognition score.

Note: In cases where PTA varied over the early postoperative period the final PTA recorded during hospitalization is presented.

^aNo response above 2 kHz, PTA calculated based on the patient's thresholds at 0.5, 1 and 2 kHz only.

^bWave I present immediately at given latency postoperatively but disappeared during the early postoperative period.

^cNo response above 1 kHz, PTA calculated based on the patient's thresholds at 0.5 and 1 kHz only.

sensorineural hearing loss in the right ear (PTA = 51 dB HL) and a mild-to-moderate u-shaped sensorineural hearing loss in the left ear (PTA = 38 dB HL). WRSs were 100% in the left ear and 91% in the right ear. DPOAE testing indicated emissions were absent bilaterally. ABR recorded from the right ear showed a reproducible wave I only at a latency (1.56 milliseconds) comparable to that of the left ear.

Three hours after surgery, ABR on the right side showed a clear and reproducible wave I only (2.0 milliseconds). As shown in ►Fig. 3A, ABR in the right ear showed a gradual prolongation of wave I latency from postoperative day 3 (2.09 milliseconds) to day 6 (2.41 milliseconds). On the final day in hospital (day 7) wave I was absent. DPOAEs remained absent bilaterally at all assessments.

Audiometry in the right ear 24 hours following surgery showed a moderately severe to severe sensorineural hearing loss in the low frequencies with no measurable hearing at 1 kHz or above and a WRS of 0%. On postoperative day 2, the patient recovered hearing at 1 kHz (85 dB HL) and 1.5 kHz (75 dB HL) in the right ear, and hearing at 2 kHz became measurable on day 6 (115 dB HL). On day 7 a PTA (at 0.5, 1 and 2 kHz only) of 87 dB HL and WRS of 0% were documented.

Sixteen weeks after surgery a PTA (at 0.5, 1, and 2 kHz only) of 87 dB HL and WRS of 0% were documented. As shown in ►Fig. 3B, ECoChG for 4-kHz tone-bursts showed a large SP (7 μ V, compared with 1.6 μ V in the left), whereas the CAP (which was 14.4 μ V in the left) could not be identified in the right ear. Click-evoked ECoChG from the right ear showed a small CAP (16.9 μ V, compared with 45.7 μ V in the left) with

a slightly larger SP (10.80 μ V, compared with 9.9 μ V in the left).

Case 17

Patient 17 was a 55-year old woman with a 6-mm (1.5 mm from the fundus of the IAC) left vestibular schwannoma. Preoperative audiometry showed normal hearing bilaterally, but slightly poorer in the left ear (left PTA = 6 dB HL, right PTA = 0 dB HL, WRS = 100% for both ears). DPOAE testing indicated present emissions at 2 to 8 kHz bilaterally. ABR recordings from the left ear showed a reproducible wave I at a latency (1.80 milliseconds) comparable to the right ear. Waves III and V from the left ear were delayed relative to the right ear (wave III interaural latency difference [ILD] = 0.67 milliseconds, wave V ILD = 0.70 milliseconds).

ABR assessment was performed 3 hours after surgery and documented a replicable wave I only at 1.88 milliseconds in the left ear (►Fig. 4). As shown in ►Fig. 4, over postoperative days 0 to 5 the latency of wave I gradually increased to a maximum of 2.35 milliseconds on day 5, and on postoperative day 6 no reproducible ABR waves were present. DPOAEs were present from 2 to 8 kHz at all postoperative assessments. Pure-tone audiometry performed on postoperative days 2 to 6 and 12 weeks postoperatively showed no measurable hearing in the left ear.

Twelve weeks after surgery no ABR waves were present in recordings from the left ear. Transtympanic ECoChG elicited by tone-burst stimuli in the left ear showed a 4.4 μ V SP but no CAP at 4 kHz (traces not shown) and no responses at 1 kHz. Click-evoked ECoChG from that ear demonstrated a small CAP

Fig. 3 Case 2: (A) Time-ordered sequence of click evoked auditory brainstem responses recorded from the tumor (T) and nontumor (NT) sides before and after vestibular schwannoma surgery. (B) Transtympanic ECoG waveforms elicited by 4-kHz tone-bursts 16 weeks following surgery.

(9.20 μ V) with an SP (6.44 μ V) at 90 dB HL. No ECoG recordings were made from the right ear.

Discussion

In all, 6 of the 20 patients monitored had measurable hearing on the operated side postoperatively, with the remaining 14 patients displaying immediate anacusis. As shown in **Table 2**,

of the patients with preserved hearing, four experienced a decrease in hearing from preoperative levels into the range considered unserviceable, and two maintained serviceable hearing, demonstrating less than 10 dB HL change in PTA and less than 10% change in WRS. Of the six patients that preoperatively had serviceable hearing, ABR wave I and V present, and tumors less than 20 mm diameter, hearing was measurable postoperatively in two patients (33%).

Fig. 4 Case 17: Time-ordered sequence of click evoked auditory brainstem responses recorded from the tumor (T) and nontumor (NT) sides before and after vestibular schwannoma surgery.

It is notable that although differences in tumor size were not statistically significant between the hearing preserved and anacusis groups, the group that had measurable hearing postoperatively had a higher rate of tumors smaller than 20 mm (100% of the group) and a lower rate of tumor extension to the fundus (17% of the group) relative to those who experienced anacusis (50% with tumors less than 20 mm, and 43% with 0 mm between tumor and fundus of the IAC). Larger patient numbers may be needed to reveal a statistically significant relationship between tumor dimensions and the likelihood of hearing preservation.

Causes of Immediate Postoperative Anacusis

In seven cases, the cochlear nerve could not be detected as an entity separate from the tumor during surgery, inevitably

resulting in postoperative anacusis. Of the six patients who had no ABR preoperatively, five lost the cochlear nerve as it was indistinguishable from the tumor. One patient with no preoperative ABR retained the cochlear nerve and some limited hearing postoperatively. This result is consistent with previous studies indicating that an absence of preoperative ABR is generally a poor prognostic indicator for hearing outcome.^{1,11-14} In the remaining two cases in which the cochlear nerve was lost, one patient (Case 5) had wave I and a delayed wave V present preoperatively, and the other (Case 4) presented preoperatively a delayed wave I, absent wave V, and present OAEs.

In addition to neural damage, cochlear damage was sustained in three of the cases in which the cochlear nerve could not be preserved. The loss of preoperatively measureable

DPOAEs in these cases indicates that a loss of hair cell function occurred. The most likely explanation for this is that the internal auditory artery supplying both the cochlea and cochlear nerve was damaged, as complete preservation of this artery is extremely difficult when the eighth nerve is severed.¹⁵ The exception is Case 14, in which the emission present at 8 kHz remained robust postoperatively despite the loss of the cochlear nerve. In this case the hearing loss appears to be solely due to the loss of the cochlear nerve.

Seven cases of anacusis occurred despite an apparently intact cochlear nerve. Five of these six patients who had both present ABR (waves I and/or V) and OAEs preoperatively showed an immediate postoperative loss of these responses. In cases where both cochlear and cochlear nerve responses were lost it is not possible to isolate the site of lesion to either the cochlea or cochlear nerve. If trauma has reduced or eliminated cochlear functioning, the absence of signals from the cochlea to the primary afferent neurons will result in an absent ABR, regardless of whether the cochlear nerve has sustained damage. In contrast, trauma that is restricted to the cochlear nerve may leave cochlear responses intact where the cochlea and its blood supply have been left undamaged. We can therefore reason that in these five cases in which both ABR and OAEs were lost, the pattern of results showing a loss of cochlear hair cell responses is consistent with injury to at least the cochlea (and/or its blood supply). It is not possible to determine whether damage to the cochlear nerve was also sustained, as ABR would be absent due to the cochlear trauma, regardless of whether additional damage to the nerve occurred.

Of the remaining two cases of anacusis in the presence of an intact cochlear nerve, one (Case 17) could be attributed purely to neural trauma. This case is discussed in further detail in a later section. In the other (Case 11), the ABR present preoperatively was absent immediately postoperatively. As OAEs were absent both preoperatively and postoperatively, it is not possible to determine whether the site of operative trauma was the cochlea, cochlear nerve, or both.

Causes of Hearing Deterioration in Cases with Measureable Hearing Postoperatively

Of the six patients in this study in whom hearing was measurable postoperatively, the significant postoperative decrease in hearing in four of these cases suggests that further damage to the cochlea, the cochlear nerve, or the vasculature of either structure occurred.

The site of operative trauma appears to be purely neural in three cases; 1, 2, and 18. In Case 1, this was shown by the disappearance of ABR wave V with preservation of wave I and OAEs. This preservation of responses from the cochlea and peripheral portion of the cochlear nerve suggests that the site of injury (which resulted in significant loss of hearing) was the medial portion of the cochlear nerve. Similarly, in Case 18, wave V increased in latency, with no change in more peripherally generated responses. Although a significant change in hearing did not result, such ABR results suggest that neural trauma medial to the generator site of wave I did occur. A more peripheral section of the cochlear nerve appears to have

been damaged in Case 2, who showed a delayed wave I immediately following surgery, which deteriorated over the early postoperative period, as is described in detail in the following section.

The precise site of damage leading to a degree of hearing deterioration in Cases 10 and 15 could not be determined. This was because in Case 10 all measured electrophysiological responses were absent both preoperatively and postoperatively, and in Case 15 the only response present preoperatively, wave I, was absent immediately following surgery. As discussed earlier, this pattern of results in Case 15 may have resulted from trauma to either the cochlea, the cochlear nerve, the vasculature of either structure, or all three.

An improvement in cochlear nerve function was found following tumor removal in Case 12. In this case the patient showed a postoperative decrease in the latency of wave V with no significant change in wave I latency. This result is most likely due to the alleviation of pressure on the medial cochlear nerve following tumor removal.^{16,17} This patient maintained good hearing sensitivity with no significant change documented following surgery.

Delayed Loss of ABR

In both Cases 2 and 17, serial ABR recordings showed initial preservation of wave I, with a gradual prolongation in latency until its disappearance on postoperative day 6 in Case 17 and day 7 in Case 2. This indicates that although the peripheral portion of the cochlear nerve was initially functional following surgery, damage to this portion of the nerve ultimately transpired. Patient 17 showed immediate anacusis following surgery; therefore, a complete conduction block of the proximal cochlear nerve is likely. In contrast, Case 2 demonstrated some degree of hearing preservation below 2 kHz, suggesting that the initial injury to the nerve led to selective degeneration of nerve fibers carrying high-frequency information.

Animal studies examining the effects of experimentally induced cochlear nerve injuries indicate that progressive degeneration of cochlear neurons may be triggered by mechanical damage to the axons of the cochlear nerve during surgery.^{6,17-19} Alternatively, Strauss et al²⁰ suggest that surgical manipulations can initiate disturbances in microcirculation in the endoneurial vasa nervorum. Disturbed microcirculation can lead to massive releases of glutamate during and after nerve ischemia, which in turn can cause an influx of calcium into the damaged neurons, resulting in cell death.¹⁵ Mechanical or microvascular trauma at the site of tumor removal can be expected to initially affect only the components of the ABR beyond wave I (generated by the lateral portion of the cochlear nerve), followed by a gradual loss of wave I as neural degeneration progresses laterally.

ECochG findings in Case 2, and both ECochG and DPOAE results in Case 17, confirm that the cochlea remained functional in both cases. This is consistent with a persistence of peripheral auditory function and either a lack of propagation of the input to the brainstem²¹ or dysynchronization of neural firing beyond the most lateral portion of the cochlear nerve. It is of note that click ECochG at 90 dB HL in Cases 2 and 19 showed a small CAP, despite the absence of ABR at 80 dB nHL.

ABR was also absent at 90 dB nHL in Case 17 (data not shown). N_1 of the CAP recorded in transtympanic ECoChG is considered to be the response of the peripheral portion of the cochlear nerve,^{21,22} and wave I of the ABR is believed to be the far-field representation of this same response.^{23,24} Given the small amplitude of the CAPs recorded, the most parsimonious explanation is that a CAP was not of sufficient amplitude to be detected through scalp-recorded ABR as wave I. Although the presence of the CAP confirms that some nerve fibers in the peripheral portion of the nerve remained functional, ABR was clearly not a sensitive enough tool to detect this reduced activity.

Further research is needed to more clearly differentiate between hearing loss due to cochlear and cochlear nerve trauma in cases where electrophysiological responses from both sites are absent. As shown in Cases 2 and 17, transtympanic ECoChG is a more sensitive measure of residual cochlear hair cell and cochlear nerve function and may provide an indication of functional integrity when OAEs and/or ABR are absent. Although the requirement for an electrode to be placed on the promontory makes transtympanic ECoChG less suitable for serial recordings, measurements made at the conclusion of surgery, and perhaps at the time of discharge from hospital, would provide considerable information regarding the status of the peripheral auditory system. ECoChG is best used in conjunction with ABR, which assesses the function of the more medial portion of the cochlear nerve and the auditory brainstem. Future studies will also benefit from monitoring of auditory responses intraoperatively and in particular from monitoring using tools that allow some distinction to be made between damage to the cochlea and to the cochlear nerve. We intend to use intraoperative ECoChG and ABR in future research to determine whether changes in auditory function at each stage of surgery are more likely to be due to cochlear or neural trauma.

Clearly, the primary limitation of this pilot study is the small number of subjects. Although this prohibits generalization of these results to clinical populations, our study does highlight the importance of monitoring both cochlear and cochlear nerve function to gain a complete understanding of the nature of hearing loss. Future research will most likely benefit not only from larger patient numbers but from a greater number of patients with serviceable preoperative hearing. Although important data can be collected from patients with poor hearing preoperatively and large tumors, the chance of hearing preservation during surgery is low, and the likelihood of cochlear and neural responses that can be used for analysis being present either preoperatively or postoperatively is reduced.

Conclusions

In many cases it is not possible to determine whether anacusis or hearing deterioration following vestibular schwannoma removal is due to cochlear or neural trauma, given that the responses from both the cochlea and cochlear nerve are often lost. However, given the loss of preoperatively measurable DPOAEs in nine cases of anacusis, it can be concluded that

trauma to at least the cochlea and/or its vasculature has been sustained, often with additional neural damage. Two cases of anacusis and three of hearing deterioration were attributed to purely neural trauma. Further research involving more sensitive near-field measurements of responses from the cochlea and cochlear nerve will help to elucidate the site of trauma. This is a necessary step in determining the optimal interventions to prevent hearing loss following vestibular schwannoma removal.

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