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The effects of cochlear implantation on vestibular function

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Abstract

Objective—Determine the risk posed by cochlear implantation (CI) to the labyrinth.

Study Design—Prospective cohort study.

Setting—Academic tertiary referral center.

Patients—Thirty-six ears belonging to 35 adult CI candidates (mean: 46, range: 23–69 years old).

Intervention—Cochlear implantation.

Main Outcome Measures—Vestibular function was assessed using the quantitative 3D head impulse test (qHIT), clinical head impulse test (cHIT), post-headshake nystagmus (HSN), caloric electronystagmography (ENG), vestibular-evoked myogenic potentials (VEMP), dynamic visual acuity (DVA), and Dizziness Handicap Inventory (DHI).

Results—All 36 ears were tested using qHIT before CI, and 28 ears were tested 4–8 weeks after CI. Quantitative HIT showed 1/28 of ears suffered reduced function. Clinical HIT was 44% sensitive and 94% specific for identification of severe-to-profound vestibular hypofunction confirmed by qHIT. HSN was unchanged in 11/11 subjects. New hyporeflexia was found in 1/16 of ENG-tested ears. VEMP showed either a disappearance of response or an increase in threshold by >10dB in 5/16 ears. Passive DVA showed no change in 16/16 ears. DHI scores worsened in 3/28 and improved in 4/28 subjects.

Conclusions—Although small, the observed rate of labyrinthine injury was comparable to that for other risks of CI. Thus, it is important to educate CI candidates about possible risk to balance function, particularly when CI of an “only balancing ear” is contemplated. Clinical HIT is useful for detecting severe high-frequency vestibular hypofunction and should be part of the pre-CI physical examination.

INTRODUCTION

The risk posed by cochlear implantation (CI) to the vestibular system remains unclear. Transient acute dizziness is common after CI, but the incidence of long-term disability due to vestibular dysfunction after unilateral CI appears to be low (1–8).

The true incidence of injury secondary to unilateral CI surgery may be masked by central compensation of unilateral vestibular hypofunction (UVH) in unilateral recipients. Redundancy of the two labyrinths and plasticity of the central nervous system create a fault-tolerant system, so that even after unilateral labyrinthectomy, most patients regain a nearly normal vestibulo-ocular reflex (VOR) for all head movements except quick rotations and

translations toward the injured labyrinth (9). In contrast, acute loss of bilateral vestibular function can cause significant disability due to VOR failure, postural instability, and chronic disequilibrium (10,11). Driving becomes difficult or impossible to do safely. Even sedentary activities such as reading can become tiresome due to the apparent rhythmic motion of the page from normally imperceptible head movement with the cardiac cycle (12,13). With the increasing popularity of simultaneous bilateral and second-ear CI, understanding the risk of iatrogenic vestibular hypofunction is becoming more urgent.

Several groups have investigated the effects of CI on vestibular function using caloric electronystagmography (ENG), rotary chair, off-vertical axis rotation (OVAR), computerized dynamic platform posturography (CDP), and quantitative scleral coil head impulse testing (qHIT). When assayed using ENG testing, 0–43% of implanted ears exhibited a decrease in horizontal semicircular canal (SCC) function (2,3,5–7,14–20). Rotary chair testing revealed that 20–38% of implanted patients had a significant decrease in vestibular function after CI (18,19). OVAR revealed no change in vestibular function (19) after CI in one study. CDP substantially improved in one study (15) but worsened in another (2) after CI. Saccular function as measured using vestibular-evoked myogenic potentials (VEMP) in children undergoing CI was absent in 5/12 patients preoperatively and disappeared in 6 of the remaining 7 postoperatively (21). Postoperative adult VEMP testing revealed that 13% to 62.5% of tested saccules suffered reduction in function (8,16). Quantitative HIT revealed that 1/11 CI recipients suffered a significant drop in SCC function (22). The Jacobsen Dizziness Handicap Inventory (DHI) has shown a wide range of pre- to postoperative change; anywhere from 0% to 32% of patients have a significant increase in self-perceived handicap (7,15,16,22).

Studies thus far have not measured SCC sensation over the full frequency range of head movements for which it is essential to stabilize gaze and posture. Caloric stimulus on ENG testing is roughly equivalent to a 0.001–0.01 Hz head rotation (for which horizontal SCC input contributes minimally to gaze stabilization in daily life). Similarly, rotary chair testing probes the low- to mid-frequency band of the VOR response (~0.1–1 Hz) (15,18,19) leaving untested responses to the higher frequency (~1–16 Hz) components of head movement for which only the VOR can stabilize gaze (23,24). Extrapolating results of a single vestibular test to all vestibular endorgans and across the entire range of physiologically relevant stimuli is analogous to characterizing hearing using only a single pure-tone threshold while ignoring the remainder of the spectral range of normal hearing. Ideally, vestibular assessment should comprehensively cover the range of stimuli normally transduced by the SCCs.

This prospective study quantified SCC and saccular endorgan function before and after CI using a battery of tests spanning the range of stimuli normally encoded by the vestibular labyrinth. These tests are complemented by measures of visual acuity during head rotation and with patients' self-reported level of dizziness-related disability. We sought to measure the prevalence of vestibular hypofunction in a cohort of CI candidates, quantify the risk of labyrinthine injury due to CI, and offer an evidence-based approach to vestibular assessment and counseling of CI candidates.

SUBJECTS AND METHODS

Subjects

Thirty-five adult CI candidates (mean age = 46, range = 23 to 69 years old) were recruited from the Johns Hopkins Listening Center. The study protocol was approved by the Institutional Review Board of the Johns Hopkins School of Medicine. Of the 35 candidates, 30 underwent unilateral CI for the first time, 3 with a prior CI underwent second-ear CI during the study, 1 underwent staged bilateral CI during the study (therefore, both of this subject's ears were enrolled independently in this study), and 1 candidate decided against implantation. Therefore,

36 candidate ears were tested prior to implantation. Of these, 16 underwent only qHIT and DHI between 2002 and 2003 as part of an earlier study (22); the remaining 20 underwent the entire battery of testing consisting of qHIT, HSN, ENG, DVA, DHI, and VEMP between 2006 and 2008. Eighteen right ears and 17 left ears were implanted, and various devices were used (Table 1). After implantation, 5 subjects (5 ears) decided not to return for postoperative vestibular testing. Of the remaining 30 implanted ears, 28 were tested 4–8 weeks after implantation. Of those 28 ears, 11 were from the group of 16 that underwent only qHIT and DHI, and 17 were from the group of 20 that underwent the entire battery of tests.

Surgery

Transmastoid scala tympani cochlear implantation was performed by one of five surgeons. Electrodes were implanted via a facial recess approach through a cochleostomy made antero-inferior to the round window niche. Each implanted electrode was reported to have reached full insertion in a single pass without any resistance or complication.

Vestibular Testing

During qHIT, the subject focused on a small midline target at eye-level located 124 cm anteriorly while an examiner delivered transient, high peak-acceleration ($\sim 3500^\circ/\text{s}^2$) (25,26) low-amplitude ($\sim 25^\circ$), unpredictable rotations of the subject's head in the maximally-excitatory direction for each SCC plane. Each subject sat in a chair centered in a rigid cubic coil frame producing three orthogonal magnetic fields. Eye and head velocity were measured in 3D (horizontal, vertical, and torsional) using the magnetic scleral search-coil technique, which is described in detail elsewhere (27,28).

The cHIT (also called the Halmagyi-Curthoys head thrust test (25)) is a physical exam maneuver in which head impulses are delivered by an examiner, much like in qHIT, in each of the 6 canal-planes. The examiner stood in front of the subject and asked the subject to try to maintain visual fixation of the examiner's nose during head rotations.

In HSN, the examiner shook the subject's head 30 times (approximately 15° amplitude at 1–2 Hz) about a vertical axis with the head pitched so that Reid's line is $\sim 20^\circ$ nose-down, placing the horizontal canals in the plane of stimulation (29). Frenzel goggles were used to observe any post-shake nystagmus.

For ENG testing, slow-phase eye movements were recorded using electrodes placed around the eyes to detect eye movements via corneoretinal potentials. Eye movements were recorded while external auditory canals were alternately irrigated with warm (43.5°C) water for 40 seconds, then, after a recovery period, cool (30.5°C) for 40 seconds, with subject positioned supine and head tilted up 30° in order to make the lateral canals approximately vertical. During this time, the subject was asked to carry on a mental distraction task, such as naming animals or counting backwards (30).

VEMP recordings were performed using a TECA Synergy evoked potential unit (VIASYS/CardinalHealth, Dublin, OH) to average acoustic click-induced relaxations of the ipsilateral sternocleidomastoid (SCM) muscle via electromyography (EMG). Electrodes over the clavicular heads were used as references and the manubrium as ground. EMG electrodes were applied to the junction of the upper and middle thirds of the SCM muscles. Responses were recorded while ipsilateral loud clicks were delivered through headphones and the subject turned the head to the contralateral side, thereby tensing the ipsilateral SCM muscle. The clicks (typically 60 to 100 dB nHL with duration of 0.1 ms) were repetitively presented to each ear at a stimulation rate of 5 Hz for 25 seconds. Myogenic potentials from the SCM were amplified, band pass filtered (20 Hz to 2 kHz) and averaged for 125 presentations. The responses evoked

in the neck EMG were averaged and presented as a VEMP. Real-time rectified EMG activity was monitored to ensure adequate contraction of the muscle for detection of the relaxation response. The VEMP threshold was defined as the minimum stimulus intensity for which a VEMP was detected.

In DVA testing, the subject's visual acuity was measured while sitting 6.5 feet from a monitor that displayed a directional (up, down, left, right) "E" optotype only when head velocity was within 120–180°/s (31–33). To achieve these velocities in passive head thrust DVA testing, quick head rotations were manually imposed in the excitatory direction of each of the 6 semicircular canals by an examiner standing behind the subject, much like in qHIT described above. However, the subject's task was to identify the orientation of the optotype presented at different image sizes. The smallest optotype reliably identified was taken as a measure of DVA and quantified as the logarithm of the minimum angle resolved (LogMAR) (31–33). In active horizontal DVA testing, patients were instructed to rotate their heads in the horizontal plane only, i.e. side-to-side.

Finally, patients completed the Jacobsen Dizziness Handicap Inventory (34).

Data Analysis

We used software written in LabVIEW (National Instruments, Austin, TX; Eye Movement Analyzer by AAM). The rotational kinematic algorithms we used to compute VOR gain (absolute value of the ratio of eye to head velocity) for qHIT has been described elsewhere (22,35). Normal VOR gains for qHIT fall within the range of 0.9 ± 0.1 for horizontal canals and 0.8 ± 0.1 for vertical (anterior and posterior) canals (22). Using these values, taking the 5th percentile and below as abnormal, and assuming a normal distribution, we defined reduced gain (i.e., UVH) as <0.74 for horizontal canals and <0.64 for vertical canals (22). "Mild-to-moderate" hypofunction was defined as VOR gains within the ranges of 0.40–0.74, 0.52–0.64, and 0.36–0.64 for horizontal, anterior, and posterior SCCs, respectively. "Severe-to-profound" hypofunction was defined as VOR gains that were <0.40 , <0.52 , and <0.36 for horizontal, anterior, and posterior SCCs, respectively. By these definitions, "mild-to-moderate" hypofunction is comparable to VOR performance observed in ears treated with intratympanic gentamicin for Ménière's disease (26), and "severe-to-profound" hypofunction is comparable to VOR performance observed in ears after ipsilateral labyrinthectomy (36).

The cHIT test result was considered positive for weakness in the tested SCC when the examiner observed a refixation saccade after a head impulse in the excitatory direction for that SCC.

Presence of post-shake nystagmus on HSN testing was considered an abnormal result which indicated asymmetry in horizontal SCC function (37). The absence of post-shake nystagmus meant one of two possibilities: either the subject was bilaterally intact or had symmetric bilateral vestibular hypofunction (BVH). New post-shake nystagmus after implantation was taken as indicating a reduction in vestibular function due to CI.

For ENG, when the sum of peak slow phase nystagmus velocities for responses to warm and cool irrigations of one ear was $<10^\circ/\text{s}$, that ear's horizontal SCC was considered hypofunctional for low frequency stimulation.

Saccular function was considered significantly reduced by CI when either a preoperatively present VEMP disappeared postoperatively or there was an increase in threshold >10 dB postoperatively (38).

Significantly abnormal passive head thrust DVA results were defined as LogMAR scores >0.16 for any of the canal planes (32). These data were compared to surgically vestibular deafferented

ears, LogMAR score within the range of 0.32 ± 0.13 (32), for a clinically meaningful comparison. Significantly abnormal active horizontal DVA LogMAR score thresholds changed depending on age (31).

A change of total DHI score by >6 points after implantation was considered significant (39).

Confidence intervals at 95% were calculated from the observed incidence of significant change in vestibular function for each testing modality, using a binomial approximation (40).

Sensitivity, specificity, and likelihood ratios for cHIT were calculated using mild-to-moderate and severe-to-profound qHIT criteria, as defined above, for vestibular hypofunction.

RESULTS

qHIT

Of the 35 subjects, preoperative qHIT revealed mild-to-moderate UVH in 9 subjects (26%) and mild-to-moderate in 7 subjects (20%) had mild-to-moderate BVH. Three of the 35 subjects (8.6%) met severe-to-profound UVH criteria, and 1 of the 35 subjects (2.9%) had severe-to-profound BVH.

Twenty-eight post-implantation ears were tested using qHIT, and only 1 of the 28 (3.6%) ears showed a significant drop in all 3 SCCs of the implanted side from normal to severe-to-profound vestibular hypofunction (Figures 1 & 2).

cHIT

Of the 14 subjects who had cHIT performed during their pre-implant physical examination, none of these ears had pre-implant hypofunction by cHIT in all 3 SCCs. Of the 14 subjects tested preoperatively, 10 had post-implant testing and none had a subsequent drop in all 3 SCCs of the implanted side. Using mild-to-moderate qHIT thresholds as our benchmark, cHIT sensitivity was 22%, specificity was 94%, and likelihood ratio (sensitivity divided by false-positive rate, a measure of the predictive utility of a test (40)) was 3.7. Respective sensitivity, specificity, and likelihood ratio using severe-to-profound qHIT criteria were 44%, 94%, and 7.3.

HSN

Of the 19 ears tested before implantation, 1 ear (5.3%) had post-headshake nystagmus. Eleven of the 19 ears underwent post-implant HSN testing, and none of these ears exhibited new post-headshake nystagmus.

ENG

Pre-implant caloric ENG showed 6 out of the 20 pre-implant ears (30%) had pre-implant vestibular hypofunction. Of the 16 ears that underwent implantation and post-implantation testing, 1 ear (6.3%) demonstrated a significant drop in function.

VEMP

Pre-implantation testing revealed absence of VEMPs in 7 of the 19 tested ears (37%). All ears that had thresholds were within the range of 75 to 106dB NHL. Five of the 16 post-implanted ears (31%) had either a disappearance of a prior measured VEMP or a >10 dB increase (worsening) of VEMP threshold.

DVA

For passive head thrust DVA, 18 ears underwent preoperative testing in all 6 SCC excitatory directions. No baseline hypofunction was detected across all 3 SCCs. Testing was repeated in 15 of the 18 implanted ears postoperatively. No ear exhibited a significant drop in function in all 3 SCCs (Figure 3).

For active horizontal DVA, 17 pre-implant ears were tested, and 3 out of 17 ears (18%) were abnormal. Active horizontal DVA was performed in 15 of the 17 implanted ears. Two of these 15 ears (13%) showed a new decrease in function after implantation.

DHI

Study subjects had both increases (worsening) and decreases (improvement) in their self-perceived dizziness-related handicap as quantified by DHI score. Three of 26 (12%) post-implanted patients had a worsening of DHI score and 4 of those 26 (15%) post-implanted subjects had an improvement of their DHI score.

Second-CI Results

A subgroup of 4 study subjects comprised “second-CI” subjects (i.e., those who had a prior implant on the side contralateral to that implanted during this study). Of those 4 subjects, 1 subject was enrolled in this study for both CI surgeries. This subject had mild-to-moderate high-frequency BVH in all 6 SCCs before the first surgery, as measured by qHIT. Vestibular function for this subject did not change after the first or second surgery. The 3 remaining subjects had normal VOR gains on qHIT after their first implantation, and they remained within the normal range after their second implantation. Cumulatively, these 4 subjects had no significant drop in HSN and DVA results after implantation. Other reductions in vestibular function seen in this group were: disappearance of VEMP postoperatively in 1 subject, new hyporeflexia on ENG in a second subject, and an increase in DHI score from 22 to 30 in a third subject. None of these 4 second-CI subjects required special postoperative care for clinical vertigo.

DISCUSSION

Of 28 ears that underwent qHIT both before and after implantation, only 1 ear (3.6%) suffered new onset profound loss for all 3 SCCs as measured at 1–2 months post-CI. This subject had a history of streptomycin treatment as a child for tuberculosis, narcolepsy and attention deficit hyperactivity disorder. This subject experienced severe immediate postoperative vertigo and required overnight admission for intravenous hydration, but gradually regained the ability to walk and resumed driving 10 days after surgery. Nine months postoperatively, repeat qHIT testing revealed no improvement; however, the DHI score did improve. Several years later, the full panel (i.e., qHIT, HSN, ENG, DVA, VEMP, and DHI) of post-CI testing was performed at 6 years post-CI, and results showed severe bilateral hypofunction on qHIT, no post-shake nystagmus on HSN, hyporeflexia on ENG, absence of VEMP, and a DHI score not significantly different from the 9-month post-implantation score. This subject could not complete DVA testing because of underlying narcolepsy. For the implanted ear, this subject’s 6-year postoperative qHIT results were unchanged from data acquired 9 months postoperatively, suggesting a permanent and stable deficit. ENG and VEMP results for the implanted side corroborated this finding. Interestingly, this subject also lost function in the unimplanted ear (by qHIT and HSN testing) between 9 months and 6 years post-CI, despite not undergoing any surgical procedure in that ear. This loss may have been due to progressive effects of streptomycin ototoxicity.

Many factors could affect the observed results and should be considered when estimating the risk to a given future CI candidate. These include variations in operator experience and surgical techniques, CI electrode design, patient age, vestibular testing methods, and patient compliance with vestibular testing. The 1/28 observed incidence of severe injury on qHIT in this study implies an estimated risk of $3.6 \pm 6.9\%$ (95% confidence interval assuming a binomial distribution (40)). Thus, our data suggest with 95% confidence that the true risk of high-frequency vestibular injury due to CI is $<10.5\%$. A similar analysis estimates risk of saccular injury equal to $31 \pm 23\%$ (VEMP) and $6 \pm 12\%$ for loss of low-frequency horizontal SCC function (ENG). Meaningful confidence intervals cannot be calculated for HSN, DVA and DHI, as they revealed no change in vestibular function after implantation.

The test with the highest rate of apparent new onset postoperative vestibular hypofunction was the VEMP, which showed evidence of saccular injury in 31% of implanted ears. The 95% confidence interval for saccular injury risk [8–54%] is consistent with the rate of post-CI loss of VEMP responses reported by others (8,16). A relatively high rate of saccular injury could be explained by the close proximity of the saccule to the cochlea. Histopathologic studies have revealed a high rate of saccular injury after cochlear implantation (41,42). Scala vestibuli insertion is a possible explanation, but it is unlikely. All 5 implant surgeons who performed implantations for subjects tested in this study use either an anterior-inferior or inferior approach for cochleostomy and electrode insertion (43). These approaches have been shown to result in scala tympani insertion (44,45) as well as in higher rates of residual hearing preservation (46, 47). This suggests that the electrode remains in the scala tympani and does not interrupt the scala media nor deflect into the scala vestibuli. Also, it is possible that electrode insertion and packing of the cochleostomy with fascia alter the acoustic/hydrodynamic conduction of VEMP stimuli to the saccular neuroepithelium. If this were the case, then we might expect that bone-conducted VEMP testing would reveal a lower rate of saccular injury than did air-conducted VEMP testing in the present study. Therefore, our 31% rate of VEMP loss using air-conducted stimuli is an upper bound on post-CI saccular injury.

Correlation between post-operative self-described dizziness handicap and objective tests of labyrinthine function was poor. Quantifying inter-test reliability by calculating a kappa coefficient, κ , between pairs of tests [48], we found that DHI poorly correlated with all of the other tests. In particular, $\kappa = 0$ for DHI vs. qHIT and $\kappa = 0.09$ for DHI vs. VEMP, where κ of 0, 0.3, 0.7, and 1 indicate chance, weakly significant, strong, and perfect correlation between tests, respectively. This was likely due in part to the wide variability of DHI scores, which worsened from pre- to post-CI in 12% of subjects but improved in 15%.

The utility of cHIT as a physical exam maneuver was quantified by relating it to the qHIT, which we used as the gold standard high-frequency SCC function test. The qHIT requires an expensive apparatus and specialized expertise not readily available to most clinics, but the results obtained by cHIT and qHIT in this study allow us to draw conclusions about the sensitivity and specificity of the cHIT. The cHIT was performed by one experienced examiner (CDS) testing all 6 SCCs in 14 subjects. Our results showed that an abnormal cHIT on physical exam was highly specific (94%) but not very sensitive (22%) for mild-to-moderate SCC hypofunction. For severe-to-profound SCC hypofunction, cHIT was still highly specific and more sensitive (44%). The likelihood ratio of 7.3 for an abnormal cHIT in the latter case implies that an abnormal cHIT increases the probability of true severe-to-profound hypofunction by ~38% in the tested SCC (49). In contrast, finding a normal cHIT increases the probability of normal function by only ~10% (likelihood ratio=0.6) in the tested SCC. These values reflect an individual examiner's set point for calling an exam abnormal, and likelihood ratios probably vary from one examiner to the next (50). However, finding an abnormal cHIT only in the ear opposite to the planned surgical ear should prompt the CI surgeon to consider and discuss the risks of implanting an "only balancing ear."

The incidence of iatrogenic vestibular injury during unilateral CI was low in this cohort; however, it exceeds the known risks of other potential major CI complications, such as post-implant meningitis (0.2%) (51), facial nerve damage (0.003–0.4%) (52,53), and device failure (1–3%) (54,55). For example, assuming that the high-frequency SCC risk to each ear is independent and equal to 3.6%, the risk of iatrogenic BVH during simultaneous CI is 0.13%, which is about the same as the probability of meningitis. In a CI candidate undergoing implantation of an “only balancing ear,” the risk for conversion to BVH would be ~3.6% for the semicircular canals and 31% for the saccule. Thus, risk to the labyrinth should be considered and discussed as part of patient evaluation and education. When all other factors are equal, the “worse balancing ear” should be implanted.

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Figure 1.

A) Pre-implant and B) post-implant qHIT data for Subject #5. Eye velocities are inverted. Comparison of before and after implantation measured VOR gains shows that the right ear remained the same whereas the left (implanted) side had a post-implant drop across all 3 semicircular canals.

Figure 2.

Individual and mean $\pm$ standard deviation VOR gains for qHIT data for 28 subjects. These data are compared to normal, gentamicin-treated (Post-Gent), and surgically deafferented (SUVD) ears' qHIT results for each SCC from prior studies (26,34). The bolded lines represent the only subject with significantly decreased vestibular function in all 3 SCCs of the implanted ear.

Figure 3.

Individual and mean $\pm$ standard deviation DVA for 15 subjects. These data are compared to that for normal and surgically deafferented (SUVD) ears' results for each SCC from a prior study (31). None of the tested implanted ears had a significant decrease in all 3 SCCs. LogMAR= $\log_{10}$ (minimum angle resolved).

Table 1

Test Subjects. Of the 35 subjects (36 ears) enrolled in this study, 16 ears (in white) only had HIT and DHI testing, of which, 11 ears were post-CI tested. The other 20 ears (in gray) had the entire battery of vestibular testing of which, 17 ears have undergone most post-CI testing. Implanted devices included: Nucleus-24 (Cochlear Corp., N=10), Nucleus Freedom (Cochlear Corp., N=13), Hi Res 90K (Advanced Bionics, N=7), Clarion CII + positioner (Advanced Bionics, N=1), Clarion CII without positioner (Advanced Bionics, N=1), Clarion HF + positioner (Advanced Bionics, N=2), and Combi 40 (Med-El, N=1).

Ear ID	Sex	Age	Diagnosis	Side	Device
1	F	28	Viral infection	R	NucleusFreedom
2	M	61	Bilateral Otosclerosis	R	Nucleus-24
3	F	36	Hyperbilirubinemia	L	Nucleus-24
4	M	62	Meningitis	L	Nucleus-24
5	M	53	Streptomycin	L	CII+positioner
6	F	37	Viral infection	L	CII
7	M	65	Bilateral Ménière's	L	HF+positioner
8	F	64	Idiopathic	L	Nucleus-24
9	F	42	Congenital Deafness	L	HF+positioner
10	F	51	Hyperbilirubinemia	R	Nucleus-24
11	F	28	Meningitis	N/A	N/A
12	M	55	Congenital Deafness	R	Nucleus-24
13	F	35	Idiopathic progressive	R	Nucleus-24
14	F	27	Congenital Deafness	L	Combi 40
15	F	45	Congenital Deafness	R	Nucleus-24
16	F	51	Idiopathic	L	Nucleus-24
17	F	50	Mumps/Chicken Pox	R	NucleusFreedom
18*	F	53	Idiopathic	R	NucleusFreedom
19	M	27	Idiopathic	R	Hi Res 90K
20*	F	51	Hyperbilirubinemia	L	Nucleus-24
21	M	52	Measles/Connexin 26?	R	Hi Res 90 K
22*	F	42	Idiopathic	R	NucleusFreedom
23	F	52	Congenital Deafness	L	Hi Res 90 K
24	M	50	Congenital/Idiopathic	L	NucleusFreedom
25	M	36	Meningitis	R	Hi Res 90K
26	M	56	Idiopathic	R	NucleusFreedom
27	M	46	Idiopathic	R	NucleusFreedom
28	M	46	Idiopathic	L	Hi Res 90K
29	F	37	Streptomycin	R	NucleusFreedom
30	F	25	Congenital Deafness	L	NucleusFreedom
31*	F	58	Idiopathic	L	NucleusFreedom
32	M	50	Congenital Rubella	L	Hi Res 90K
33	M	61	Idiopathic	R	NucleusFreedom
34	F	23	Idiopathic	L	NucleusFreedom
35	M	69	Meningitis	R	NucleusFreedom
36	M	51	Ototoxic drug exposure	R	Hi Res 90K