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Vestibular Effects of Cochlear Implantation

Craig A. Buchman, MD; Jennifer Joy, MS; Annelle Hodges, PhD; Fred F. Telischi, MD;
Thomas J. Balkany, MD

Objectives/Hypothesis: **Cochlear implantation (CI) carries with it the potential risk for vestibular system insult or stimulation with resultant dysfunction. As candidate profiles continue to evolve and with the recent development of bilateral CI, understanding the significance of this risk takes on an increasing importance.** ***Study Design:*** **Between 1997 to 2001, a prospective observational study was carried out in a tertiary care medical center to assess the effects of unilateral CI on the vestibular system.** ***Methods:*** **Assessment was performed using the dizziness handicap inventory (DHI), vestibulo-ocular reflex (VOR) testing using both alternate bithermal caloric irrigations (ENG) and rotational chair-generated sinusoidal harmonic accelerations (SHA), and computerized dynamic platform posturography (CDP) at preoperative, 1-month, 4-month, 1-year and 2-year postimplantation visits. CI was carried out without respect to the preoperative vestibular function test results.** ***Results:*** **Specifically, 86 patients were entered into the study after informed consent. For the group as a whole, pair wise comparisons revealed few significant differences between preoperative and postoperative values for VOR testing (ENG and SHA) at any of the follow-up intervals. Likewise, DHI testing was also unchanged except for significant reductions (improvements) in the emotional subcategory scores at both the 4-month and 1-year intervals. CDP results demonstrated substantial improvements in postural sway in the vestibular conditions (5 and 6) as well as composite scores with the device "off" and "on" at the 1-month, 4-month, 1-year, and 2-year intervals. Device activation appeared to improve postural stability in some conditions. Excluding those patients with preoperative areflexic or hyporeflexic responses in the implanted ear (total [warm + cool] caloric response ≤ 15 deg/s), substantial reductions (≥21 deg/s maximum slow phase velocity) in total caloric response were observed for 8 (29%) patients at the 4-month interval. These persisted throughout the study period. These changes were accompanied by significant low frequency phase changes on SHA testing confirming a VOR insult. Of interest, no significant changes were detected in the DHI or CDP, and there were no effects of age, sex, device manufacturer, or etiology of hearing loss (HL) for these patients.** ***Conclusions:*** **Unilateral CI rarely results in significant adverse effects on the vestibular system as measured by the DHI, ENG, SHA, and CDP. On the contrary, patients that underwent CI experienced significant improvements in the objective measures of postural stability as measured by CDP. Device activation in music appeared to have an additional positive effect on postural stability during CDP testing. Although VOR testing demonstrated some decreases in response, patients did not suffer from disabling vestibular effects following CI. The mechanism underlying these findings remains speculative. These findings should be considered in counseling patients about CI.** ***Key Words:*** **Vestibular, cochlear implant, dizziness, electronystagmography, rotary chair, platform posturography.**

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INTRODUCTION

Numerous studies have attempted to characterize the effects of cochlear implantation (CI) on the vestibular system. Perioperative questionnaires, vestibulo-ocular reflex (VOR) testing using caloric irrigations or rotational chair testing, and platform posturography as well as other less commonly used tests have been carried out in an attempt to objectively assess the effects of CI on balance and the VOR response. Results of the earliest studies were largely conflicting.[1–4] More recent studies have reported a relatively wide range of findings. Adverse vestibular symptoms have been reported to occur in between 0.33% and 75% of patients,[5] reduction in VOR function reportedly occurs in between 23% and 100% of patients,[6] and postural stability may be impaired,[1,2] improved,[4] or remain[7] the same depending on the study. The variance in these reports and others may be the result of factors such as retrospective study design, subjective nonvalidated questionnaires applied in a nonstandardized way, lack of both pre- and postoperative testing of patients, and study of different patient populations, devices

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From the Departments of Otolaryngology (C.A.B.), University of North Carolina at Chapel Hill, Chapel Hill, NC; and the University of Miami (J.J., A.H., F.F.T., T.J.B.), Miami, FL, U.S.A.

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Send Correspondence to Dr. Craig A. Buchman, Associate Professor, Department of Otolaryngology—HNS, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599–7600, U.S.A. E-mail: buchman@med.unc.edu

types, programming strategies, surgical procedures, and testing paradigms.

In clinical practice, it is widely accepted that CI only occasionally results in postoperative vestibular symptoms such as dizziness, vertigo, and imbalance. These symptoms are usually transient in nature and resolve completely in time. More recently, delayed intermittent dizziness has been recognized after CI.[8] Because a large number of CI candidates may have significant vestibular impairment before implantation as a result of underlying inner ear pathology, the perceived effect of CI on vestibular function may be underestimated. Better understanding whether or not unilateral CI significantly affects vestibular function is important for preoperative counseling as well as safety issues regarding bilateral CI. The purpose of the present study was to prospectively measure the vestibular effects of CI using a variety of validated clinical instruments.

PATIENTS AND METHODS

The institutional review board at the study institution approved this study. Before enrollment, all patients were counseled regarding study participation, and written and verbal informed consent was obtained.

Patients

Patients eligible for the study included all patients that were scheduled to undergo multichannel CI at the study institution. Thus, all patients met clinical criteria for CI. At the time of the study's inception, the investigators did not consider vestibular evaluation to be a necessary part of the CI evaluation process. Thus, the choice of ear for implantation was made without regard to the findings of the vestibular evaluation. Patients were excluded if they had a tympanic membrane perforation, existing mastoid cavity, known retrocochlear pathology, were uninterested in participating, or failed to complete an informed consent.

Overview of Protocol

This study was a prospective, observational study designed to assess the impact of CI on vestibular function. After study enrollment and before CI, patients (or parents of children) were asked to complete the dizziness handicap inventory (DHI) and undergo comprehensive objective vestibular testing. This testing included VOR reflex testing using alternate bithermal caloric irrigations (ENG) and rotational chair-generated sinusoidal harmonic accelerations (SHA) and computerized dynamic platform posturography (CDP). After CI, follow-up visits for testing were performed at 1-month, 4-month, 1-year, and 2-year intervals. The DHI and all objective tests (ENG, SHA, CDP) were performed at each of the testing intervals except ENG, which was not performed at the 1-month interval because of concerns that middle ear/mastoid fluid in the operated ear would adversely effect testing. After testing, all results were recorded in a database (Microsoft Access, Seattle, WA) for later use.

Measures of Vestibular and Balance Function

Dizziness handicap inventory (DHI). The DHI was administered using the full test battery.[9] For children unable to complete the DHI, parents answered the questions. The DHI is a clinically validated instrument composed of 25 questions developed to measure a person's self-assessment of the global effect that disequilibrium has on his or her life. The inventory was designed to evaluate the physical, functional, and emotional impact of vestibular system disease. The 25-item questionnaire has 9 functional, 9 emotional, and 7 physical questions. Patients answer "yes," "sometimes," or "no" to each of the questions, and 4 points, 2 points, or 0 points, respectively, are assigned for each answer. The sum of the 25 items results in a scaled score ranging from 0 to 100, with 0 being "no handicap" and 100 indicating "significant" self-perceived handicap. Subgroup scores were also tabulated. This instrument has been shown to have high internal consistency (α = 0.72–0.89) as well as test-retest reliability (r = 0.92–0.97).[9] Moreover, since its inception, the DHI has been used in numerous studies and appears to accurately describe a patient's impairment from vestibular disorders.

Vestibulo-Ocular reflex (VOR) testing.

ALTERNATE BITHERMAL CALORIC STIMULATION (ENG). ENG was performed using a commercially available system with both vertical and horizontal leads, as previously described.[10] Electro-oculographic calibration consisted of horizontal deviations of 15 deg and vertical deviations of 10 deg completed with the patient seated on a table in a darkened room in front of a light bar. After calibration, open-loop bithermal (30 deg C and 44 deg C) water irrigations of the external auditory canal (40 s/irrigation) were carried out with the patient lying down and their head elevated 30 deg. Caloric tests were completed with the eyes open in a darkened room under Frenzel lenses with continuous tasking. When responses were absent, ice water irrigations (O deg C) were completed with a 40 second syringe irrigation of 40 mL of ice water. Maximum slow phase velocity (SPV) was derived from the computer-generated tracings.

ROTATIONAL CHAIR TESTING USING SINUSOIDAL HARMONIC ACCELERATIONS (SHA). Rotational chair testing using SHA testing was carried out using an earth vertical axis, servomotor-driven rotational chair system within an enclosure (NeuroKinetics, Inc., Pittsburgh, PA). The patient was seated in the darkened enclosure with their head tilted forward 30 deg to bring the horizontal canals parallel to the floor for optimum stimulus. Both vertical and horizontal electro-oculography electrodes were placed as described above. Calibration was completed with 10 deg deviations to the right and left. SHA testing was completed at a maximum rate of 50 deg/s for frequencies 0.01, 0.02, 0.04, 0.08, 0.16, and 0.32 Hz, with the CI device "off" (i.e., removed). The patient continuously performed mental altering tasks during testing. With the external processor in place using the patient's usual coding strategy, SHA was repeated at 0.02, 0.08, and 0.32 Hz in 70 dB sound pressure level (SPL) music in the background.

Electro-oculography data were digitally filtered and differentiated to produce eye velocity and fast phase eye movements detected by velocity threshold calculation and eliminated from the data record.[11] Fourier analyses were performed for both slow eye movement velocity records and chair velocity records. The VOR gain measures were derived by calculation of the ratio of amplitudes of the Fourier components of the eye velocity to the chair velocity. Response phases were calculated from the Fourier components of the chair velocity and the slow eye velocity. Phase values represent the time shift of the slow eye velocity relative to the chair velocity. By convention, a response with unity gain and zero phase represents a perfectly compensatory eye movement.

Computerized dynamic platform posturography (CDP). The EquiTest System (NeuroCom International, Inc, Clackamas, OR) was used to measure postural stability.[12,13] The equilibrium score indicating postural stability compared the participants sway with the theoretical limits of stability. The subject's sway was calculated from the maximum anterior and posterior center of gravity displacements occurring over the 20-second trial period. The theoretical maximum displacement without losing balance was assumed to be of 12.5 deg (6.25 anterior, 6.25 posterior). The results were expressed as percentages, 0 indicating sway exceeding the limit of stability, and 100 indicating perfect stability.

The equilibrium score was examined for six different sensory conditions. Condition 1, platform fixed with eyes open; condition 2, platform fixed with eyes closed; condition 3, platform fixed with visual surround sway-referenced; condition 4, platform sway referenced with eyes open; condition 5, platform sway referenced with eyes closed; and condition 6, platform sway referenced with visual surround sway referenced. Each condition was tested three times with either the device "off" or "on" at each testing session. Testing for patients with the device "on" was performed with the headcoil in place using the patients usual programming mode with music delivered through a loudspeaker at 70 dB SPL.

Data

Data consisted of answers to DHI questions and ENG, SHA, and CDP results at the preoperative, 1-month, 4-month, 1-year, and 2-year follow-up periods. For the DHI, answers were coded as described above according to functional, emotional, and physical subcategories. Subcategory scores were then summed to generate a total score. If a patient's DHI had two or fewer missing items (unanswered) of the seven to nine items for each subtest, the missing value was replaced with the average score of the answered items for that subtest because only adding those items answered would falsely lower the scores. If more than two items were unanswered, the patient's DHI was omitted from the analysis for that subtest. There were few patients where such modifications were used. For the purpose of making comparisons with other patients with known vestibular disorders, data were adopted from the article of Jacobsen and Newman.[9] These included groups of people tested that had occasional dizziness ($\leq$12 dizzy attacks/year), frequent attacks ($>$12 attacks/year), and continuous dizziness.

For ENG testing, caloric test scores included maximum SPV measurements obtained for warm and cold irrigations for the implanted and unimplanted ears at each test interval. To assess the effect of CI on caloric response, a composite or total caloric score was calculated for each ear by adding the warm and cold SPV values.

For SHA, values for gain, phase, and symmetry were computed as described above. Normative data were provided according to the manufacturer specifications (NeuroKinetics Inc, Pittsburgh, PA).

For CDP, sway values were available for one to three tests in each of six test conditions for both the device "off" or "on." Mean values for the three tests performed were used to represent the score for that condition. The mean values for all test conditions were taken to indicate the composite ability of sensory organization. Where the patient performed less than three tests, a mean value was used when two tests were performed, and one value was used when one test was performed, although this was uncommon. For the purposes of the present study, the vestibular-specific conditions (condition 5 and 6) and composite scores were used to illustrate the effects of CI on postural stability. Normative data were derived from Hirabayashi and Iwasaki.[14]

Data Analysis

Means, standard deviations, and ranges were computed for continuous variables including age, ENG, SHA, CDP, and the DHI and displayed in table format. Changes in the variables were displayed graphically after calculating the absolute, pair wise difference (preoperative value minus postoperative value) for each variable at each of the testing intervals. Categorical variables included gender, CI device manufacturer, etiology of hearing loss (HL), radiographic findings, and DHI, SHA, CDP test results. For the DHI, SHA, and CDP results, comparison groups were defined by previous reports in the literature (DHI, CDP) or by manufacturer norms (SHA), as described above and classified into low, normal, or high categories. Frequency responders were tabulated by categories and displayed in table format.

Significance testing was performed for the pair wise comparisons at the various follow-up intervals using either t tests (parametric) or Mann-Whitney U (nonparametric) tests, where appropriate. For the purposes of this study, significance was set at $P \leq .05$, two-tailed, despite the large number of comparisons to be performed, in an effort to maximize sensitivity to any possible changes in vestibular function. Analysis was carried out for the entire group with the available data. Because patients with minimal or absent preoperative vestibular function in the implanted ear, as defined by caloric testing, would unlikely be subjected to the adverse effects of CI, the analysis was then repeated excluding those patients with a total caloric response (warm + cold) in the implanted ear of 15 deg/s or less because this was considered hyporeflexic or areflexic.[10] This group presumably included those patients "at risk" for vestibular insult from CI. Furthermore, to examine the effect of vestibular loss on the various measures, between group differences were also sought for the subset of patients that had a decrease of 21 deg/s or more in the implanted ear at the first postoperative caloric test (4 months). For each of the intergroup comparisons, the effect of age, sex, etiology, duration of HL, device manufacturer, and radiographic findings were also sought using chi-square comparisons.

RESULTS

Patients and Surgery

Table I shows the demographics of the 86 patients entered in to the study. At the time of completion of the

TABLE I.
Demographics of 86 Patients Entered into Study between 1997 and 2001.

	n (%)
Age at cochlear implantation	
Age $<$18 yrs	22 (26), range 2–16
Age $\geq$18 yrs	64 (74), range 18–87
Sex	
Male	39 (45)
Female	47 (55)
Device	
Nucleus 22	4 (5)
Nucleus 24	47 (55)
Clarion	28 (33)
MedEl	7 (8)
Etiology of hearing loss	
Congenital	4 (5)
Hereditary	12 (14)
Meniere's disease	1 (1)
Meningitis	7 (8)
Sudden hearing loss	16 (19)
Other	46 (54)
Radiographic imaging	
Normal	77 (90)
Malformation	2 (2)
Ossified	4 (5)
Not available	1 (1)
Other	2 (2)

study, the available data collected is shown in Table II. There was substantial attrition from the study before and after the onset of testing. Most of the attrition occurred in ENG testing, where only 47 patients completed preoperative testing, and 36 patients completed testing at the 4-month interval.

All patients underwent cochleostomy and insertion of the electrode in the in the scala tympani, and all but three had complete insertions. Of the incomplete insertions, only one electrode was outside the cochleostomy in each case. There were no cases of stapedectomy, common cavity implantation, scala vestibuli, or vestibular labyrinthine implantation. There was one drill through of a relatively short (4 mm) segment of ossification secondary to otosclerosis with complete insertion. There were no intramodiolar electrodes placed. Postoperatively, there were two cases of facial paresis that resolved. There was one intraoperative cerebrospinal fluid (CSF) gusher through the cochleostomy in a patient with Mondini malformation that was controlled intraoperatively with packing around the electrode. There was one postoperative wound infection. Most patients were discharged from the hospital within 24 hours of surgery.

Measures of Vestibular and Balance Function for All Patients

Dizziness handicap inventory (DHI). Preoperative DHI data are shown in Table III. Before CI, the frequency of self-perceived vestibular disability was relatively low. With use of the data from Jacobsen and Newman[9] that categorized dizziness severity by frequency of occurrence for comparison, only three (4%) preoperative CI patients had total DHI scores higher (mean ± 2 SD) than a group of patients that were continuously affected by dizziness. Likewise, for subgroup analysis, the physical, emotional, and functional category scores were higher than subjects of Jacobsen and Newman with continuous dizziness in only three (4%), three (4%), and three (4%) patients, respectively. Comparison with a lesser effected group having dizziness 12 times or less per year, the total, physical, emotional, and functional scores were higher in the present study's patients in 13 (17%), 9 (11%), 13 (17%), and 8 (10%) patients, respectively. Thus, most patients preparing to undergo CI in the present study were either minimally or not significantly affected by preoperative dizziness.

The mean difference from baseline for each of the DHI subcategories and total scores at the 1-month interval were negative, indicating a trend toward increased handicap at the earliest testing interval, although these differences were not significant. Pair wise comparisons revealed no significant changes in the physical or functional subcategory scores or total scores at any of the evaluation periods. Significant decreases (less handicap) in the emotional subcategory scores at both the 4-month ($P = .046$) and 1-year ($P = .037$) intervals were observed.

Figure 1 illustrates the paired comparisons by follow-up interval. At 1 month, those patients with high total DHI scores preoperatively tended to get worse after CI. By 4 months, these subjects appeared to improve. There were two patients with substantial improvements in total scores at 4 months and numerous patients with substantial improvements of a relatively large magnitude at 1 year. In contrast, at 4 months, there was one patient with a large increase (worsening) in his total DHI score, and there were no major outliers for worsening in DHI at 1 year. Two-year data has too few patients to make any reasonable generalizations.

Vestibulo-Ocular reflex (VOR) testing.

Alternate bithermal caloric stimulation (ENG). Table IV shows the mean (SD) total caloric responses for the entire group (n = 47) over the study period. Before CI, the mean response for the implanted ears was 31 ± 21 deg/s, and for the unimplanted ears was 29 ± 25 deg/s. Eleven (23%) implanted ears and 15 (32%) unimplanted ears had total caloric responses of ≤15 deg/s (hypo or areflexic) before surgery.[10] Five (11%) of seven patients with a single vestibular reactive ear underwent CI in their only vestibular functioning ear. Fifteen (68%) of 22 children had preoperative total caloric responses in the implanted ear of 15 deg/s or less. The children with total caloric responses greater than 16 deg/s were all older than 5 years of age at the time of CI.

Figure 2 shows the pair wise changes from baseline for both the implanted and unimplanted ears at the 4-month testing interval. For the group as a whole, pair wise comparisons revealed no significant mean changes in total caloric responses for either the implanted or unimplanted ear over the study period. Interestingly, in most patients, changes in total caloric response in the implanted ear were accompanied by similar changes in the unimplanted ear. For instance, for those subjects who had a reduction in the implanted ear response of 21 deg/s or more, the unimplanted ear had a decrease in response as well, albeit less so. In only one subject did the implanted ear decrease by 21 deg/s or more, whereas the unim-

TABLE II.
Available Data of 86 Patients Entered into Study between 1997 and 2001.

Test	Preoperative	1 Mo	4 Mo	1 Yr	2 Yr
DHI	78	66	45	36	17
ENG	47	NA	36	28	14
SHA	81	67	39	34	15
CDP	82	68	47	39	15

DHI = dizziness handicap inventory; ENG = electronystagmography; SHA = rotary chair testing with sinusoidal harmonic acceleration; CDP = computerized dynamic platform posturography; NA = not applicable.

TABLE III.
Dizziness Handicap Inventory (DHI) Scores by Follow-up Interval.

Category	Preoperative (n = 78)	1 Month (n = 66)	4 Month (n = 45)	1 Year (n = 36)	2 Year (n = 17)
Functional					
Mean (SD)	5 (8)	4 (8)	2 (4)	2 (3)	2 (5)
Range	0–32	0–28	0–18	0–12	0–22
Emotional					
Mean (SD)	5 (9)	5 (10)	2 (4)	1 (3)	3 (7)
Range	0–36	0–36	0–20	0–14	0–26
Physical					
Mean (SD)	4 (7)	4 (7)	3 (5)	3 (5)	3 (5)
Range	0–28	0–28	0–22	0–16	0–18
Total					
Mean (SD)	14 (23)	13 (23)	6 (12)	6 (10)	9 (17)
Range	0–88	0–90	0–56	0–36	0–66

*For total score, 0 indicates no handicap, and 100 indicates severe self-perceived disability. For physical, emotional, and functional subcategories, maximum scores (severe handicap) are 28, 36, and 36 (9).

planted ear increased in responsiveness. Similar results were obtained when all patients with a preoperative total caloric response of 0 were excluded, except at the 2-year interval, where a significant reduction ($P = .048$) in the implanted ear response was apparent. There was no significant change in the unimplanted ear at the 2-year interval, and the difference between the ears was not significant. For those five patients that underwent CI in their only functioning ear, two had significant reductions in response at 4 months, and one of these patients became bilaterally areflexic.

Rotational chair testing using sinusoidal harmonic accelerations (SHA). Before CI, the results of SHA are shown in Table V as compared with the manufacturer's norms. In general, mean values were in line with the manufacturer's normal values. Categorical analysis suggested that nearly one third of patients had a low frequency phase lead or low gain values.

Pair wise comparisons revealed no significant changes in phase, gain, or symmetry values at any of the testing intervals except symmetry at the 0.01 Hz was higher at 1 year ($P = .04$), phase at 0.02 Hz was borderline higher at 1 year ($P = .055$), and gain at 0.02 Hz was lower at 1 year ($P = .001$).

Computerized dynamic platform posturography (CDP). Table VI displays the mean, standard deviations, and range values for CDP testing at the various testing intervals. Before CI, postural control as measured by CDP testing was substantially below normal for the group as a whole. Scores on condition 5, condition 6, and composite scores were below published normal values for 52 (61%), 46 (54%), and 48 (56%) patients, respectively.[14]

After CI, substantial increases (improvements) in CDP scores were observed across nearly all testing intervals with the device "off" and "on." Figure 3 shows the changes in CDP scores for conditions 5 and 6 as well as composite scores at 1 year with the device "off." Some patients that routinely fell (score 0) on conditions 5 and 6 were able to maintain stability at 1 year after implantation. Only 1 patient on condition 6 had a substantial reduction in balance after CI. These changes reached significance with the device "off" for condition 5 at 1 year ($P = .004$) and 2-years ($P = .042$); condition 6 at 2 years ($P = .024$); and composites score at 4 months ($P = .028$) and 1-year ($P = .000$). Similarly, with the device "on," significant increases were also observed over baseline for many test intervals. Interestingly, comparisons within the same testing session revealed higher (better stability) scores for the device "on" than "off" in all conditions tested. These differences approached significance for condition 5 at 4 months ($P = .056$) and were significant for condition 6 at 1 year ($P = .012$) (Fig. 4). That is, postural stability as measured by CDP was significantly better with the device activated in music than with the device "off." There was no obvious effect of age at implantation on the CDP results.

Measures of Vestibular and Balance Function for Patients "At-Risk" for Vestibular Loss

As previously defined, those patients "at risk" for vestibular loss from CI were those with preoperative total caloric responses in the implanted ear of more than 16 deg/s.

Dizziness handicap inventory (DHI). Pair wise comparisons revealed no significant changes in the DHI for any of the subcategory or composite scores analyses over all of the testing intervals in this subgroup of patients.

Vestibulo-Ocular Reflex (VOR) Testing.

Alternate bithermal caloric stimulation (ENG). Pair wise comparisons showed the mean differences from baseline were not significant for either the implanted or unimplanted ear except at 2 years, where the implanted ear response was significantly less ($P = .048$) than the preoperative value. Again, there was no significant difference between the ears at this interval. There was 1 (4%) of 28 ears with a total caloric responses greater than 16 deg/s preoperatively that was reduced to 0 deg/s at the 4-month visit. At 1 year, 1 (4%) of 23 ears at risk was reduced to 0 deg/s at 1 year.

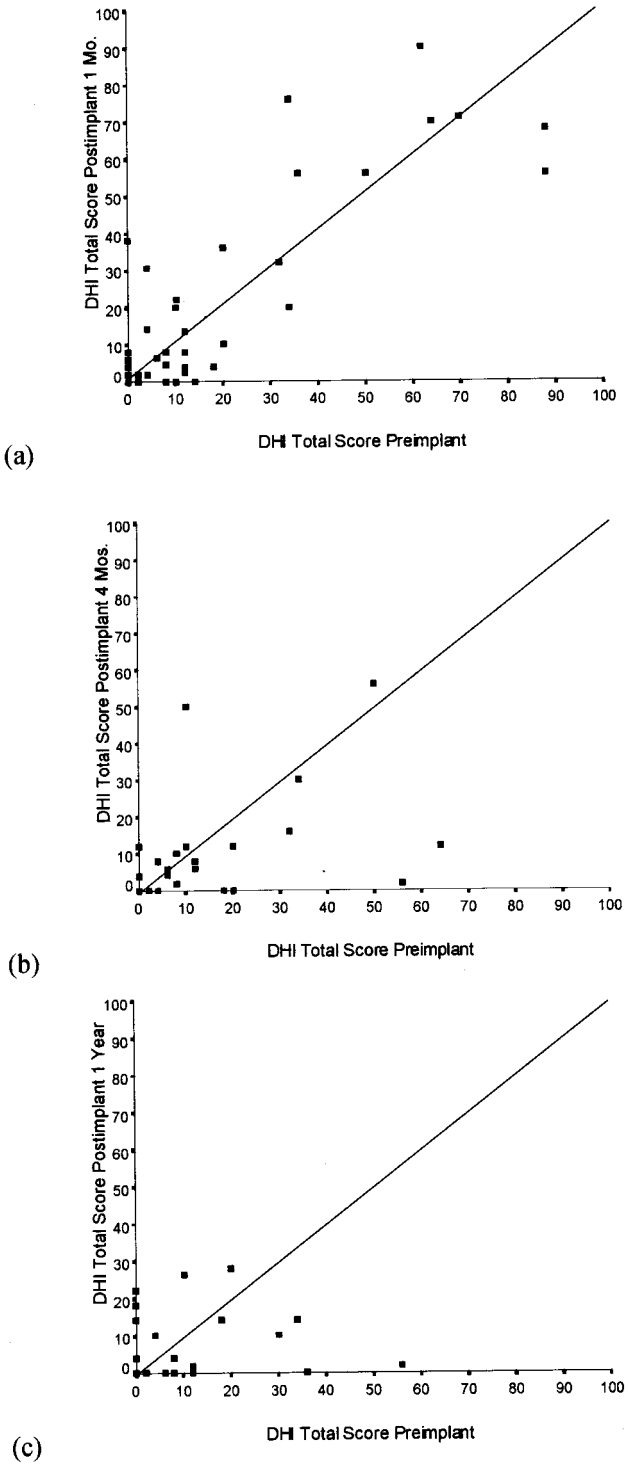


Fig. 1. Dizziness handicap inventory (DHI) total scores plotted preoperatively versus 1 months (**A**), 4 months (**B**), and 1 year (**C**) for all patients. A score of 0 indicates no handicap, and 100 indicates severe self-perceived disability. Scores below the diagonal are improved from before surgery, whereas those above the diagonal are worse.

ROTATIONAL CHAIR TESTING USING SINUSOIDAL HARMONIC ACCELERATIONS (SHA). There were no significant differences in phase, gain, or symmetry at any of the testing intervals except gain at 0.02 Hz was lower at 1 year ($P = .002$); phase at 0.04 Hz was higher at 4 months ($P = .034$); and phase was higher at 0.08 Hz at 1 month ($P = .016$). There were no significant differences with the device "on" or "off" except for lower phase values in the patients with the device "on" at the highest testing frequency (0.32 Hz) at 2 years ($P = .026$).

TABLE IV.
Total Caloric Response (warm + cold) by Follow-up Interval.*

Ear	Response (deg/s) Preoperative (n = 47)	4 Month (n = 36)	1 Year (n = 28)	2 Year (n = 14)
Implanted				
Mean (SD)	31 (21)	29 (24)	27 (21)	29 (25)
Range	0–80	0–108	0–69	0–81
Areflexic (%)*	7 (15)	7 (19)	5 (18)	3 (21)
Hyporeflexic (%)†	4 (9)	3 (8)	3 (11)	2 (14)
Unimplanted				
Mean (SD)	29 (25)	29 (24)	28 (26)	29 (23)
Range	0–112	0–88	0–93	0–77
Areflexic (%)*	11 (23)	10 (28)	7 (25)	3 (21)
Hyporeflexic (%)†	4 (9)	3 (8)	4 (14)	1 (7)

*Areflexic = total caloric response of 0 deg/sec and absent response to ice water caloric.
†Hyporeflexic = total caloric response of ≤15 deg/s or less.

Computerized dynamic platform posturography (CDP). Again, increases (improvement) in CDP scores were observed for all test conditions at all testing intervals. These changes reached significance with the device "off" for condition 5 at 1 year ($P = .025$). With the device "on," significant increases were also observed over baseline for many test intervals. Again, comparisons within

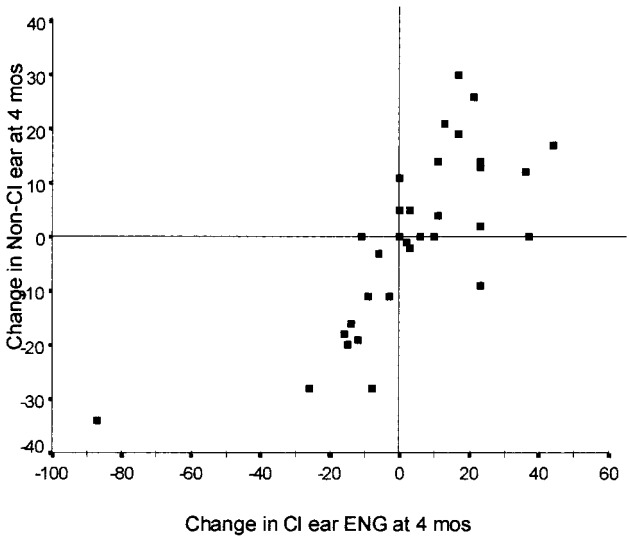


Fig. 2. Absolute change in total caloric response (warm + cool) for the implanted versus the unimplanted ears at the 4-month testing interval. Change = preoperative response minus postoperative response (deg/s). *Positive* values indicate a *decrease* in postoperative response, and *negative* values indicate and *increase* in postoperative response.

TABLE V.
Preoperative Rotational Chair Testing using Sinusoidal Harmonic Acceleration (SHA).

	Frequency (Hz)					
	0.01	0.02	0.04	0.08	0.16	0.32
Phase						
Mean (SD)	43.0 (40.8)	30.0 (31.4)	20.9 (22.4)	12.4 (23.7)	3.0 (22.0)	−4.0 (18.0)
Norms*	25–51.8	12.8–32.4	5.2–18.4	−3.4–10.6	−11.7–6.3	−11.7–6.3
Low	6 (7)	8 (10)	6 (8)	7 (8)	8 (10)	14 (17)
Normal	46 (57)	42 (51)	43 (54)	49 (59)	60 (73)	62 (75)
High	29 (36)	32 (39)	31 (39)	27 (33)	14 (17)	7 (8)
Gain						
Mean (SD)	0.26 (0.16)	0.44 (1.08)	0.42 (0.21)	0.46 (0.21)	0.49 (0.20)	0.52 (0.21)
Norms*	0.14–0.40	0.23–0.67	0.34–0.90	0.45–0.97	0.45–1.01	0.45–1.01
Low	17 (21)	29 (35)	27 (34)	41 (49)	33 (40)	32 (39)
Normal	52 (64)	51 (62)	52 (65)	41 (49)	48 (59)	49 (59)
High	12 (15)	2 (2)	1 (1)	1 (1)	1 (1)	2 (2)
Symmetry						
Mean (SD)	0.3 (16.2)	0.7 (15.6)	1.2 (11.7)	−0.9 (12.6)	0.9 (9.5)	0.36 (13.3)
Norms*	−12.2–22.2	−8.2–22.2	−9.2–22.0	−12.2–18.6	−11.7–20.1	−11.7–20.1
Low	13 (16)	10 (12)	12 (15)	11 (13)	6 (7)	13 (16)
Normal	63 (78)	66 (81)	66 (83)	68 (82)	74 (90)	66 (79)
High	5 (6)	6 (7)	2 (3)	4 (5)	2 (2)	4 (5)

*Norms from NeuroKinetics International, mean ± 2 SD.

each testing session between device "off" and device "on" revealed higher scores (better stability) for device "on" than "off" for all conditions. These differences reached significance for condition 5 at 4 months ($P = .022$).

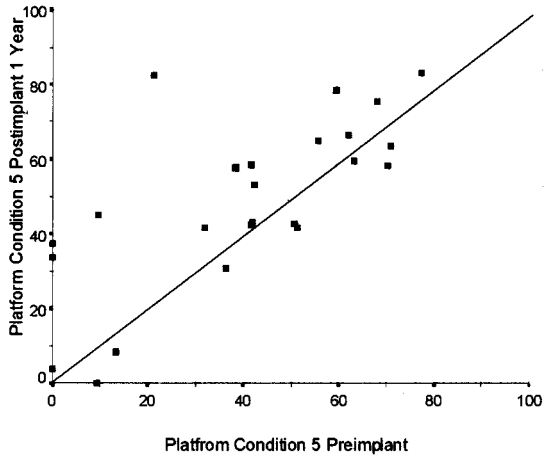
Measures of Vestibular and Balance Function in Patients with Substantial Loss of Caloric Responses in the Implanted Ear

In the group of patients with preoperative total caloric response of greater than 16 deg/s, eight (29%) implanted ears had a reduction of total caloric response of 21 deg/s or greater at the 4-month testing interval. For this group of patients with available data at the 1- and 2-year intervals, total caloric response remained less than the preoperative value at both intervals with the 1-year difference being highly significant ($P = .009$). Thus, for this relatively small subset of implanted ears with a substantial reduction in total caloric response after CI, recovery was not observed. For the same patients, the contralateral (unimplanted) ear also decreased in total caloric response, albeit to a lesser degree than the implanted ear (Fig. 2). In only one patient was there an increase in the contralateral ear response. Similar findings were observed at the 1- and 2-year intervals.

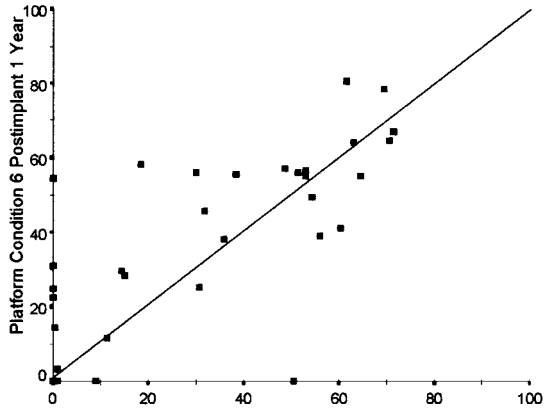
TABLE VI.
Mean (SD) Computerized Dynamic Platform Posturography (CDP) Scores by Follow-up Interval.

Condition	Preoperative (n = 82)	1 Month (n = 67)	4 Month (n = 46)	1 Year (n = 38)	2 Year (n = 16)
Condition 5					
Mean (SD)	39 (26)	40 (27)	35 (27)	41 (26)	44 (24)
Range	0–86	0–83	0–84	0–83	0–72
Normal range*	42.3–84.7				
Condition 6					
Mean (SD)	33 (26)	31 (26)	32 (27)	33 (26)	41 (25)
Range	0–76	0–85	0–85	0–81	0–81
Normal range*	29.6–88.4				
Composite score					
Mean (SD)	58 (20)	58 (18)	57 (18)	59 (18)	61 (22)
Range	0–88	0–90	0–84	24–85	18–87
Normal range*	61.3–90.1				

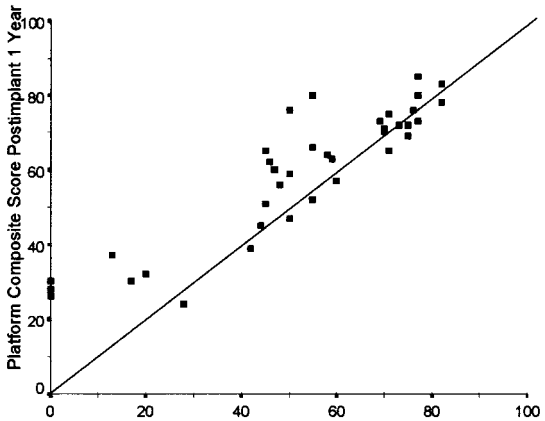
*Normal ranges for the Sensory Organization Test after Hirabayashi et al.[14], mean ± 2 SD.



(a)



(b)



(c)

Fig. 3. Computerized dynamic platform posturography (CDP) condition 5 (**A**), condition 6 (**B**), and composite scores (**C**) plotted as preoperative score versus 1 year for all patients. A score of 0 indicates a fall, and 100 indicates perfect postural stability. Scores above the diagonal represent an improvement over baseline, whereas those below the diagonal are worse.

Dizziness handicap inventory (DHI). At both the 4-month and 1-year intervals, the mean differences in the physical, emotional, functional, and total scores was greater (less handicap) for those patients with a drop in total caloric response 21 deg/s or more in the implanted ear than for those without a change in caloric response. The mean difference between the groups for the functional subcategory score was significant ($P = .035$) at 4 months and the total score at 1 year ($P = .009$).

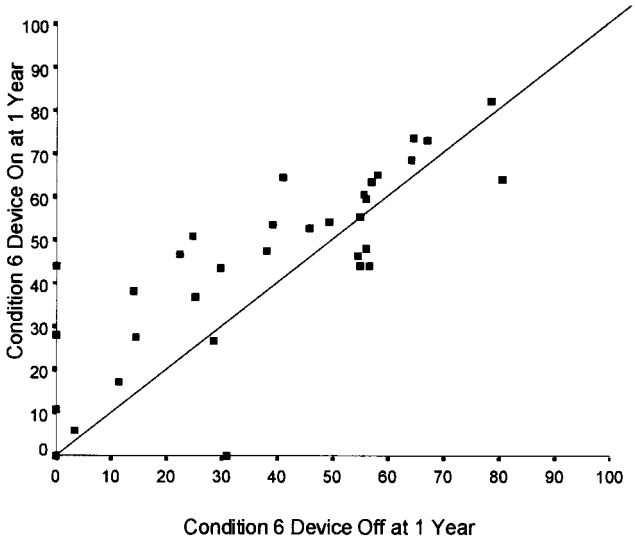


Fig. 4. Computerized dynamic platform posturography (CDP) condition 6 scores with the device "off" (x-axis) versus the device "on" (y-axis) at the 1-year testing interval. A score of 0 indicates a fall, and 100 indicates perfect postural stability. Scores above the diagonal are improved with device activation.

ROTATIONAL CHAIR TESTING USING SINUSOIDAL HARMONIC ACCELERATIONS (SHA). At 4 months, there were substantial phase differences between the groups with and without a drop in total caloric response. These differences were significant at 0.8 Hz ($P = .046$) and 0.16 Hz (0.045). Thus, patients with significant reductions in total caloric response on ENG testing at 4 months had significant phase changes on SHA testing at the same time interval, confirming a labyrinthine effect. These phase differences were maintained at the 1- and 2-year intervals. There were no significant differences in gain or symmetry with the device "on" or "off" except at 0.32 Hz, where the symmetry difference was greater for those patients with a decrease in total caloric response of 21 deg/s or less.

Computerized dynamic platform posturography (CDP). In this subgroup of patients, mean differences were slightly greater (worse stability) for those patients with a decrease of total caloric response 21 deg/s or greater for condition 5, condition 6, and composite score at the 4-month, 1-year, and 2-year intervals. None of these differences reached significance. Interestingly, device activation again appeared to have a positive effect on postural sway testing for all patients at each of the testing intervals.

DISCUSSION

CI has become widely accepted as a method of auditory habilitation or rehabilitation for many patients with substantial sensorineural HL. Since its inception, there has always been concern regarding the potential for ves-

tibular impairment to occur from CI. Vast clinical experience has demonstrated that vestibular losses can occur after surgical procedures on the inner ear. For instance, surgical destruction of the peripheral vestibular apparatus through labyrinthectomy in Ménière's disease and acoustic tumor removal are known methods of vestibular deafferentation. These patients frequently experience varying degrees of vestibular symptoms that improve or resolve over time through the processes of compensation, substitution, and habituation.[15,16] Nondestructive procedures of the inner ear can also impact on vestibular function. Stapedectomy patients frequently complain of postoperative dizziness and vertigo, albeit transient in nature. Objective evaluation of these patients has demonstrated varying degrees of vestibular system impairment.[17] Fortunately, long-term vestibular dysfunction is rare. These vestibular ablative and nonablative procedures are clear examples of how inner ear surgery can affect vestibular function. With ever broadening criteria for CI candidacy and the recent development of bilateral CI, understanding the risk of CI-induced vestibular impairment takes on an increasing importance.

CI could affect the vestibular system through either pathologic disruption of the sensory vestibular functions of the labyrinth with either resultant unilateral deafferentation or fluctuating vestibulopathy or by electrical stimulation of the vestibular system. In theory, cochlear electrode implantation may alter normal inner ear fluid homeostasis, induce direct trauma to the vestibular sensory structures, or surgically induce inflammation, with resultant fibrosis or hair cell loss. In these cases, significant dizziness, vertigo, and imbalance would occur as a result of the vestibular lesion. These symptoms would be present irrespective of device activation. Patient's symptoms resulting from static vestibular lesions should improve or resolve over time through the processes of compensation, substitution, and habituation. By contrast, electrical stimulation could impart pathologic changes to the inner ear with subsequent dysfunction or provide conflicting sensory inputs resulting in vestibular dysfunction and imbalance. In these cases, device activation should induce dizziness, vertigo, nystagmus, or imbalance.

Although limited pathologic analysis of the vestibular apparatus from human temporal bones after CI exist, some of the above-mentioned features have been observed. Fibrosis, hydrops with saccular membrane distortion, osteoanagenesis, and reactive neuromas have all been observed in the vestibule as well as semicircular canals of patients having undergone CI. Scala vestibuli involvement, as a result of osseous spiral lamina disruption near the round window, was highly correlated with this damage.[18] These findings have real clinical importance for cochleostomy and electrode insertion techniques in the future.

The results of the present study indicate that patients undergoing unilateral CI experience some substantial improvements in both subjective and objective measures of vestibular and balance function after CI surgery and device activation. With use of a validated instrument (i.e., the DHI) to assess the self-perceived impact of CI on vestibular and balance function, results demonstrated significant improvements in emotional subcategory scores at 4 months and 1 year after surgery. Moreover, CDP testing results indicated that postural stability was significantly improved after unilateral CI and that device activation in the presence of music may further improve these test results. By comparison with other surgical procedures of the inner ear aimed at either controlling vertigo or removing acoustic tumors, the magnitude of effect on the DHI was relatively small.[15,16] Nevertheless, for the group as a whole, unilateral CI may have a significant positive impact on patient perceptions of dizziness and overall balance function.

The mechanisms underlying the findings in the present study remain speculative. Presumably, unilateral CI could alter a previously uncompensated vestibular lesion, thereby inducing compensation. This premise is supported by the fact that patients experiencing a decrease of more than 21 deg/s in the implanted ear had better DHI scores than those with less change in caloric response. However, nearly all of these patients had bilateral caloric response reductions, and CDP results generally did not substantiate this finding. Another possibility is that electrical stimulation could somehow provide inputs to the vestibular system, thus improving balance in some way. This may be supported by the improvements in postural stability identified with device activation. The fact that balance was also improved over baseline with the device "off" is in contradistinction to this. The effects of chronic electrical stimulation on the vestibular system need to be better assessed to answer this question. Finally, because the emotional subcategory of the DHI assesses emotions such as fear, frustration, avoidance behavior, and depression, perhaps CI allows patients to regain confidence, thereby improving the patient's perception of vestibular and balance disability. This last explanation is less likely to be the sole reason for these findings given the objective improvements on CDP testing.

The findings of the present study regarding improved postural stability and the positive effect of device activation are in agreement with those reported by Eisenberg et al.[4] and contrast those reported by Black et al.[1–3] Although the reasons for these differences are not totally evident, differences in patient selection, surgical procedures, CI devices, stimulation schemes, and testing paradigms all may be important factors. It is interesting to note that in using modern electro-oculographic techniques, abnormal eye movements can be occasionally induced with CI activation despite the fact that patients remain asymptomatic.[19] This taken together with the fact that some patients have improvements in postural stability with device activation suggests that vestibular activation, when it occurs, is mostly well compensated for by patients and may, in fact, be beneficial. This statement agrees with the later findings of Black et al.[20] The mechanisms underlying these findings deserve further investigation.

The fact that, in the present study, unilateral CI usually did not result in substantial vestibular impairment is quite remarkable. Presumably, patients "at risk" for vestibular impairment from CI would be those that have significant residual vestibular function before sur-

gery. In the present study, 25% to 30% of ears had either no response or were hyporesponsive on caloric testing before implantation. For children, this was even greater, with nearly 70% of the implanted ears having either absent or low intensity responses to caloric irrigations before surgery. These findings are mostly in agreement with other studies that have measured VOR function in CI recipients.[6] Thus, approximately 70% of adult CI candidates and only approximately 30% of pediatric candidates would be "at risk" for vestibular effects from CI. After CI, 29% of "at-risk" ears had a substantial drop that was sustained throughout the observation period. In many cases, the contralateral ear showed a reduction as well, although to a lesser extent. In accepting these findings, CI would be expected to produce a peripheral vestibular loss in only approximately 20% of all adult patients and perhaps 10% of all children presenting for CI. In the present study, even for those patients in which a significant drop was detected, worsening in self-perceived dizziness, imbalance, and postural sway were not observed. For healthy adults and children, adaptive mechanisms most likely allow these individuals to compensate, or changes in VOR testing results are not predictive of CI-induced vestibular problems. The latter explanation seems likely and was recently proposed by Fina et al.[8] Further investigation is clearly warranted in this regard.

The fact remains that bilateral loss of VOR function from CI may have significant effects on patients receiving implants either in their only vestibular functioning ear or when bilateral loss from simultaneous, bilateral CI occurs. Given the results of the present study, it would seem that the risk of bilateral loss from simultaneous bilateral CI would be approximately 5% for adults and substantially less for children. We also speculate that the risk of bilateral vestibular loss from implantation in the only functioning ear might occur in approximately 30% of "at-risk" patients. In the present study, five patients underwent CI in their only functioning ear. Two of four with available 4-month data had a substantial reduction in total caloric response, of which one was areflexic. Remarkably, neither of these two patients demonstrated significant changes on CDP scores. Both fell on conditions 5 and 6 preoperatively, and composite scores remained essentially unchanged. DHI scores also showed no changes. Thus, bilateral loss on VOR testing may not translate into disabling vestibular symptoms. Nevertheless, because the potential impact of bilateral vestibular loss may be substantial, disclosure of this potential complication is probably worth noting in these selected patient populations.

The utility of preoperative vestibular testing to identify those patients at risk for severe dizziness, imbalance, and oscillopsia remains to be determined. Clearly, screening all patients in need of CI with an extensive vestibular testing battery is time consuming, uncomfortable for patients, and may not be cost effective. The fact that, in many cases, unilateral vestibular losses after CI are relatively uncommon and usually do not result in significant long-term disability from dizziness and imbalance suggests that routine testing for all patients is probably not warranted. In this study, those patients with relatively high preoperative DHI scores appeared to worsen at their 1-month follow-up, although by 4-months and 1-year, many of these patients were doing better than their preoperative scores. Thus, those with preexisting handicap from dizziness should be counseled regarding this. For the time being, identification of those patients with a single vestibular functioning ear would seem to be worthwhile when contemplating implantation. This is currently difficult without ENG testing. Factors such as residual hearing and duration of deafness should be further explored to identify patients with only a single, vestibular functioning ear.

CONCLUSIONS

The results of the present study show that unilateral CI rarely results in significant adverse effects on the vestibular system as measured by the DHI, ENG, SHA, and CDP. On the contrary, for the group as a whole, patients who underwent CI experienced significant improvements in the objective measures of postural stability as measured by CDP. Moreover, device activation in music appeared to have an additional positive effect on postural stability during CDP testing. Although VOR testing demonstrated some decreases in response, it was unable to identify those patients that would suffer disabling vestibular effects after CI. From these findings, it is tempting to speculate that unilateral CI could improve a patient's postural stability through processes such as compensation or electrical vestibular stimulation. Moreover, nonvestibular factors may be at work.

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APPENDIX I

Supplementary Literature Review

Cochlear implantation (CI) may affect the vestibular system through either pathologic disruption of the sensory vestibular functions of the labyrinth with either resultant unilateral deafferentation or fluctuating vestibulopathy or by electrical stimulation of the vestibular system. In theory, CI electrode placement may alter normal inner ear fluid homeostasis, induce direct trauma to the vestibular sensory structures, or incite inflammation with resultant fibrosis or hair cell loss. In these cases, significant dizziness, vertigo, and imbalance would occur as a result of the vestibular lesion. These symptoms would be present irrespective of device activation. Patient's symptoms resulting from static vestibular lesions should improve or resolve over time through the processes of compensation, substitution, and habituation. By contrast, electrical stimulation could impart pathologic changes to the inner ear with subsequent dysfunction or provide conflicting sensory inputs, resulting in vestibular dysfunction and imbalance. In these cases, device activation should induce symptoms. In any given patient, both surgical trauma with on-going compensation and electrical stimulation may be at work.

In clinical practice, it is generally accepted that CI only occasionally results in postoperative vestibular symptoms such as dizziness, vertigo, and imbalance. These symptoms are usually transient in nature and resolve completely in time. Because a large number of CI candidates may have significant vestibular impairment before implantation as a result of underlying inner ear pathology, the perceived effect of CI on vestibular function may be underestimated.

Adverse vestibular symptoms, primarily vertigo and imbalance, have been variably reported for patients undergoing CI. Careful inspection of the various data reveals that much was derived from either surgeon-completed questionnaires provided to implant manufacturers, retrospective analysis of patient charts where the frequency of reported symptoms may not be representative, or nonvalidated questionnaires. It is probably safe to say that this complication is substantially under-reported in the literature. Moreover, the impact of vestibular disorders in CI patients is unknown.

History of the Vestibular Effects of Cochlear Implantation

Most of the earlier, historic accounts regarding vestibular effects of CI are related to concerns regarding electrical stimulation of the vestibular system rather than CI-induced inner ear pathology.

Electrical stimulation of the peripheral vestibular system. The first electrical stimulation of the inner ear probably occurred by Count Alessandro Volta[1] at the end of the 18th century when he placed two metal rods in his ears and connected them to the terminal of 30 or 40 electrolytic cells (50 V). He reported the sensation of: "une secousse dans la tate" or a blow on the head followed by a sound like "the boiling of a viscid liquid". Subsequently, in the early 19th century, Ritter and Augustin[2,3] independently published reports of vertigo being evoked as a result of an electric current being passed through the head. Purkinje[4] later noted nystagmus and disruption of balance after galvanic stimulation of the labyrinth. Modern inner ear stimulation, for the purposes of auditory perception, occurred in 1925 when radio engineers discovered that tones could be produced by electrodes near the ears. Subsequently, 32 articles were published between 1930 and 1946 regarding various types of electrical stimulation in the outer and middle ear and auditory perceptions.[5] Unfortunately, most of the details are lacking regarding the effects of these experiments on the vestibular system.

As early as the 1940s, it was beginning to be recognized that galvanic stimulation applied by way of field potential to the mastoid could elicit a sway response at low stimulation currents, probably through DC electrical modulation of vestibular potentials.[6–9] At higher currents, eye movements could be induced, presumably through stimulation of the vestibuloocular reflex (VOR) reflexes.[10] Also, direct stimulation of the vestibular nerves in cats and monkeys elicited characteristic vestibulo-spinal and VOR reflex responses[11] with limited spread of current using the stimulus intensities employed.[12] Of interest, Kimm et al.[13] were unable to induce vestibular effects with intracochlear (scala tympani) electrode stimulation in the nonhuman primate.

Between 1960 and 1961, House,[14] with his colleague Doyle, were experimenting with electrical stimulation of the inner ear. House performed promontory and vestibule stimulation of patients undergoing stapedectomy surgery. Using a square-wave generator, he noted that patients could detect auditory percepts from electrically delivered currents better in the perilymph of the vestibule than on the promontory. Direct stimulation of the perilymph did not cause dizziness or facial stimulation above 30 Hz. However, frequencies below 20 Hz induced significant dizziness in the absence of auditory percepts.

In 1964, Simmons with the assistance of Epley et al.[15] implanted six electrodes in the modiolus portion of the eighth nerve near the basal cochlear fibers under local anesthesia. On stimulation of these electrodes, auditory

percepts were obvious, and pitch was detected through alterations in electrode stimulation or stimulus repetition rate. Even at the most intense stimulation levels, where facial stimulation was evident, vestibular symptoms were notably absent.

Simmons et al.[16–19] implanted a similar patient in August of 1977 after neomycin toxicity. Through a transcanal approach, he removed the stapes, drilled a 1 mm hole in the promontory, and subsequently drilled a 0.6 mm hole in the medial wall of the vestibule. Cerebrospinal fluid (CSF) emanated from the hole and 4–75 μm platinum-iridium electrodes coated with Teflon were inserted to a depth of 4.5 to 5 mm. The patient's postoperative course was complicated by unsteadiness with head motion for 10 days, facial paresis, and a CSF leak, which all resolved. The patient used the device extensively for psychoacoustic testing (4,000 hours) and at home to watch television. Postoperative electronystagmography (ENG) testing demonstrated a contralateral directional beating preponderance and an ipsilateral caloric weakness. During auditory testing, stimulation of all of the electrodes could elicit auditory percepts, whereas stimulation of E2 was found to produce vertigo, head turning, and body tilt. In 1979, platform posturography was carried out in Pittsburgh, confirming vestibular system activation on device stimulation. On the basis of the findings and surgical procedure, it was surmised that at least one of the electrodes (E2) was in the inferior vestibular nerve.[18] However, according to Simmons, during routine device usage, the vestibular sensations were "harmless" for the patient. In 1986, the patient died of myocardial infarction, and temporal bone histopathology confirmed the electrode that caused vestibular symptoms to be located near the saccular division of the inferior vestibular nerve. This case, along with House's reports, confirmed for the first time that cochlear nerve stimulation is possible by way of a variety of anatomic locations (vestibule and internal auditory canal).[14] In contrast, vestibular stimulation only occurred when the electrodes were either in close proximity with the vestibular nerves or were delivered below 20 Hz. The findings in this patient were remarkably similar to those previously reviewed animal experiments that suggested that stimulation is highly dependent on electrode location as well as stimulus frequency and intensity.[11–13]

Vestibular stimulation by cochlear implantation. Djourno and Eyries[20] are credited with the first intralabyrinthine implantation of an electrical stimulating prosthesis. This was placed through a labyrinthine fistula in a patient with chronic otitis media and cholesteatoma in 1957.[20–22] Djourno was a French neurophysiologist and Eyries an established otologist. On stimulation, the patient described high-frequency sounds that resembled the "roulette wheel of a casino" and "crickets." Their patient was able to discern the words "pap," "mamm," and "allo." This implant produced no adverse facial stimulation because the patient had preexisting facial paralysis. Apparently, disturbances in balance were also not observed. Further reports were lacking.[23]

In 1957, a patient in Los Angeles, CA brought William House, MD a news article detailing the apparent success of Djourno and Eyries. By 1961, House performed the promontory and vestibule electrical stimulation tests described above. After this, House and Doyle implanted two adult patients with single gold electrodes for short-term stimulation of hearing. One additional patient received a five-electrode device. All three of these devices were later removed because of compatibility issues.[14] In the mid 1960s, House teamed with engineer Jack Urban to produce the first wearable, "take home" CI, implanted in Chuck Graser in 1972.[14,24–26] This was possible through the use of newer, biocompatible materials first developed for pacemaker use. His initial device was a six-wire electrode grounded through the footplate of the stapes. With the use of sine wave stimulation in the laboratory, dizziness was not elicited. The patient was later fitted with a speech processor/stimulator that had a carrier wave (16 KHz). This device did not produce untoward vestibular effects for the patient either. Beyond these descriptions, further references to vestibular symptoms were not made.

In the 1960s and 1970s, numerous other groups were exploring the field of electrical stimulation of the inner ear, primarily for the purpose of hearing. Teams including F. Blair Simmons and Robert White (Stanford University), Donald Eddington (University of Utah), Robin Michelson, Michael Merzinich, and Robert Schindler (University of California at San Francisco), and Claude-Henri Chouard (France) and Graeme Clark (University of Melbourne) were most active. In contrast with House, these efforts were mostly directed at developing a multichannel electrode CI.[21,22]

In 1971, Michelson[27] reported on his first four patients undergoing cochlear stimulation under local anesthesia and an additional two patients who received permanent CIs that are described below. In one of four patients, vertigo and tinnitus occurred during electrode insertion into the scala tympani. Postoperatively, the patient had mild vertigo lasting approximately 3 days. There were apparently no electrically induced vestibular symptoms described for any of these patients.

In the 1960s and 1970s, considerable opposition arose within the scientific community regarding the possibility of speech understanding by patients with CIs. Specifically, "auditory physiologists and histopathologists dismissed these investigations as misguided attempts by surgeons—who know little about auditory neuroscience—to stimulate nerves that were already dead."[26] Similarly, opposition occurred in France and Australia where Chouard and Clark faced funding difficulties. Chouard, a student of Eyries, "dreamt of. . .a James Bond-style gadget which would alleviate the formidable handicap of total deafness." Chouard teamed with the small French electronics company, Bertin, to develop a multichannel CI. At the same time in Australia, Clark, motivated by his fathers hearing loss, also developed a multichannel device based on the integrated circuit. This device would ultimately grow in to what is now the Nucleus CI, produced by Cochlear Limited (Sydney, Australia).[21,22]

As of 1974, 13 patients had been implanted in the United States with functioning scala tympani CI elec-

trodes by either William F. House (n = 11) or Robin P. Michelson (n = 2).[26,27] The House devices were single-electrode devices (later produced as the House/3M CI), and the Michelson devices were bipolar, intracochlear devices. With the funding of the National Institutes of Neurological and Communicative Disorders and Stroke (NINCDS), a study was convened in Pittsburgh to evaluate the auditory, vestibular, and psychologic aspects of these subjects. The results of these evaluations are reported in a group of publications termed the Bilger report.[28–39] Generally, this report confirmed the findings of House and Urban,[26] Michelson,[27] and Simmons that CI could provide some useful auditory information and was safe and well tolerated. House, Schindler, and others agreed that the Bilger report provided substantial scientific evidence for the benefits of CI and gave credibility to the emerging technology.[40]

In addition to assessing auditory effects of CI, the investigators of the Bilger report attempted to assess the vestibular and postural stability status of these patients. Black and Myers[31] noted that 8 of 13 subjects had either unsolicited (5 subjects) or solicited (3 subjects) symptoms of vestibular function loss. Of the subjects with unsolicited comments, all complained of rotary vertigo that was intermittent and positional. The remaining subjects complained of staggering in the dark and oscillopsia. All of the subjects had abnormal ENGs preoperatively, four with progressive loss and the remaining with absent caloric responses bilaterally.[33] Posturography testing showed evidence of abnormal postural stability with the device off that worsened by activating the device in quiet. It was noted, however, that device activation in white noise improved postural stability.[37] In summary, the conclusions of these evaluations were that the CI was both damaging to the peripheral vestibular system and also stimulated the vestibular system, resulting in significant postural instability. The theory at that time was that the amplitude modulated carrier signal (16 kHz) was reduced during acoustic activation because of nonlinearities in the system. The carrier signal was presumed to produce a galvanic stimulus to the inner ear that resulted in disruption of postural control by way of vestibular activation.[37,41]

These findings were later heavily disputed by Eisenberg et al.[42] First, they reanalyzed Black's data on symptoms and postural stability. They stated: "Subjectively, the HEI [House Ear Institute] implant subjects do not report balance problems or dizziness complaints when using the stimulator. Some subjects do experience vestibular symptoms. . .as a result of absent vestibular function secondary to the etiology of their inner ear problem. Most patients do report slight disequilibrium immediately post-surgery. This disappears within a week or two." Eisenberg et al. also point out that although Black found that "all 13 subjects demonstrated evidence of additional instability by one or more of the posturography tests measures. . . A closer look at the individual subject data, however, shows that there was also some improvement in postural stability on 1 or more of the 16 measures for most subjects. In addition, postural stability seemed to show some improvement when the implant was activated and broadband noise stimulus was delivered simultaneously." Thus, because of these interpretations of the Bilger report data[31,33,37,41] the HEI group carried out their own studies on some of these same subjects as well as additional patients. This study's results found that there were essentially no significant changes in the pre- versus postimplantation ENG responses, except for possibly a bilateral reduction over time. Postural stability testing using computerized posturography and the Sono-Postural test revealed improved postural stability with the device activated with or without white noise. Transtemporal galvanic stimulus applied to the mastoid in this study induced the greatest postural instability for these patients. These findings were felt to refute the findings of the Bilger report.[24,37,41] In a later report by the HEI evaluating a larger group of single-channel implant recipients, a questionnaire survey identified only 2 (3%) of 61 patients with complaints of vestibular symptoms worsening after CI that were, again, not attributable to the implant.[43]

Recent Studies on Vestibular Effects of Cochlear Implantation

Much has changed since these initial studies on the vestibular effects of CI. Most notably, electrode arrays, device manufacturing, stimulation strategies, speech processing strategies, as well as surgical techniques are different. Nevertheless, clarity regarding this issue is still lacking.

Vestibular symptoms have been variably reported to occur after modern multichannel CI. In a recent review (1995) of the Cochlear Corporation database, transient dizziness was reported to occur during the postoperative period in 10 (0.33%) of 3,064 adult patients and 3 (0.16%) of 1,905 children. Dizziness with device use occurred in 11 (0.36%) adults and 0 children.[44] In contrast, Steenerson et al.[45] recently reported vertigo to occur in 35 (75%) of 47 patients after CI, 34% of which were new symptoms for the patient. A number of the cases in this report were of the benign paroxysmal positional type. Using a questionnaire, Hoffman[46] found that 49 (82%) of 60 responders were either better or the same regarding dizziness or imbalance after CI surgery. The remaining 11 (18%) patients complained of increased vestibular symptoms that were related to implant use in one patient (2%), were associated with occasional vertigo in another patient (2%), or were otherwise not specified (n = 9, 14%). In another study, Ito[47] reported postoperative vertigo in 26 (47%) of 45 CI patients that were further divided into those with vertigo early (58%), prolonged (34%), or delayed (8%) after surgery. Two (18%) of 11 patients with prolonged or delayed vertigo had symptoms related to device activation. Kubo[48] reported dizziness in 46 (49%) of 94 patients: 29 (63%) occurring immediately after surgery and resolved in a month, 2 (4%) with continuous dizziness, and 15 (33%) with vertigo spells starting 1 month postoperatively. Fina et al.[49] also reported dizziness in patients after CI in 29 (39%) of 75 patients, with 4 (14%) having a transient attacks perioperatively, and the remaining 25 (86%) having a delayed episodic

vertigo that they attributed to possible endolymphatic hydrops.

From these studies and others, it is clear that a wide variety of vestibular symptoms may occur after CI. The mechanisms underlying these symptoms are clearly multifactorial. Factors such as surgical trauma with resultant unilateral partial or total deafferentation, surgically induced fluctuating vestibulopathy, and electrical stimulation of the vestibular system are all possible. The following pages will review the relevant literature regarding the potential etiologies for CI-induced vestibular impairment.

Inner ear surgery and vestibulopathy. Surgery of the inner ear has always carried with it the risk of vestibular system insult with resultant partial or total deafferentation or distorted inputs. These changes, when abrupt in nature, can induce vertigo, imbalance, and unstable vision. When the vestibular lesion is stable, the complex process of compensation allows for vertigo resolution, restitution of balance, and visual stabilization. This process relies on the execution of two major tasks: 1) reestablishment of balance by reducing or abolishing the static asymmetry in the vestibular nuclei, reflected in the postural tone of skeletal and eye muscles and 2) recalibration of the gain of the dynamic vestibular reflexes to ensure symmetric compensatory vestibulo-spinal and VOR reflex activity during motion of the body and the head.[50–53]

Numerous examples exist for surgical procedures on the inner ear to elicit the above-mentioned vestibular ablative paradigm. In some recent studies, subjective and objective tests of vestibular function have been used to assess the degree of vestibular loss and the processes of compensation. For instance, labyrinthectomy or vestibular nerve section for fluctuating vestibulopathy from Menière's disease is a clear case where surgical removal or disconnection of the peripheral vestibular-sensing apparatus results in a clear, unilateral deafferentation. Because most of these patients have significant disability preoperatively, studies aimed at assessing quality-of-life measures show improvements over time. ENG studies document complete loss of caloric responses in the affected ear, and platform posturography demonstrates the process of initial decreases in postural stability that improve over time through compensation. Nevertheless, 20% to 28% of patients may have persistent disequilibrium after these procedures, and normalization of these measures may not always occur.[54–60]

Patients undergoing resection of central nervous system tumors such as acoustic neuromas will also frequently incur complete, peripheral deafferentation of the vestibular sense organ. In contrast, these patients usually have relatively low levels of preoperative, self-perceived disability that worsens postoperatively in more than 25% of patients. These findings may be accompanied by significant worsening in postural stability that again improves over time. Nevertheless, a substantial group of patients maintains postural instability.[61–69]

"Nondestructive" procedures of the inner ear such as stapedectomy can also produce partial or total vestibular deafferentation, with an objective reduction in vestibular and balance function. Using ENG, approximately 25% to 50% of patients with otosclerosis present with reduced or absent caloric responses. After stapedectomy surgery, as many as 10% to 30% of subjects will incur a decrease in caloric responses.[70,71] These patients may also experience significant increases in postural sway, as measured by platform posturography.[72,73] Fortunately, long-term vestibular and balance disability is rare after this procedure. This is presumably because of the fact that patients undergoing stapedectomy are usually young and healthy, allowing for good compensation, and that the procedure usually produces only transient pathologic changes in the inner ear. Occasionally, stapedectomy patients do have persistent vestibular and balance problems resulting from more significant, surgically induced inner ear pathology.[73,74]

Cochlear implantation and vestibulopathy. Surgically induced vestibulopathy can take the from of partial or total deafferentation or the induction of pathologic changes in the inner ear that result in fluctuating vestibular inputs. The former can be diagnosed through the combination of vestibular symptoms and signs, vestibular testing findings, and histopathologic analysis. In contrast, fluctuating vestibulopathy is more difficult to identify as it relates to CI because reductions in vestibular response may not be evident. In these cases, the combination of symptoms and signs with histopathology changes may be diagnostic. Clinically, identifying the ear that is responsible for the symptoms and signs could be enigmatic.

Temporal bone histopathy. Can CI induce partial or total vestibular deafferentation with symptoms such as dizziness, vertigo, imbalance, and resulting disability? This has long been the question of numerous investigators. Presumably, a misplaced cochleostomy in the region of the round window could acutely induce a variety of pathologic changes such as basilar membrane rupture, scala media transaction, and direct saccular damage. Alterations in inner ear fluid homeostasis could also occur secondary to perilymph and endolymph depletion, leakage, and reconstitution. Electrode placement has been shown to produce ruptures in the basilar membrane, fracture the osseous spiral lamina, transect scala media, and fracture the modiolus.[75–98] Secondary reactions to blood within the inner ear as well as the aforementioned lesions or the implanted electrode array may result in inflammation, fibrosis, and endolymphatic hydrops.[99–109]

Nearly all histopathology studies related to CI have focused on the pathologic changes of intracochlear structures. Tien and Linthicum[110] recently reported on 11 pairs of human temporal bones from patients having received implants with either single-channel (n = 6) or multichannel (n = 5) devices. Overall, there was histologic evidence of vestibular end-organ damage from CI in 54.5% of cases. These changes were not related to the pathologic changes from underlying ear disease because contralateral bones did not exhibit similar changes. Findings such as fibrosis, hydrops with saccular membrane distortion, osteoneogenesis, and reactive neuromas were observed in the vestibule as well as semicircular canals. Scala vestibuli involvement, as a result of osseous spiral lamina disruption near the round window, was highly correlated with this

damage. In fact, basilar membrane or osseous spiral lamina rupture was associated with vestibular damage in 75% of cases. Review of the clinical records of the patients with vestibular damage revealed that only 33% with available postoperative information reported balance problems.[110] Further details were not available. The findings of this important study clearly demonstrate that CI can induce significant vestibular system pathology. The lack of a clear correlation between symptoms, signs, and vestibular pathology is likely related to the inherent retrospective nature of the study, the lack of knowledge regarding preexisting vestibular function before implantation, and the fact that patients can compensate for significant lesions, thereby remaining asymptomatic.

VESTIBULAR FUNCTION TESTING. Numerous studies have been carried out trying to assess vestibular effects of CI using ENG and rotary chair testing. A few studies have assessed the postural stability of these patients using variations of platform posturography.

Table AI summarizes the results of ENG studies published to date. In these studies, 459 (60%) of 768 patients that had preoperative testing carried out had abnormal findings before implantation. The vast majority of these findings were significant reductions in vestibular response, bilaterally. After CI at various intervals postimplantation, changes in caloric response were demonstrated in 71 (38%) of 186 patients. These findings were mostly reductions in the caloric response as compared with preoperative values. Only rarely have abnormalities in oculomotor testing been described. Thus, patients presenting for CI evaluation frequently have bilateral caloric weakness, although this may not be symmetric. CI surgery results in a reduction in VOR response in as many as 40% of patients. Similar findings were reported by Mangham,[111] who used rotational chair testing. In that study, four (44%) of nine patients had significant increases in phase angle associated with CI surgery.

In most of these studies, vestibular symptoms after CI were not addressed relative to the changes in VOR response testing. Kubo et al.[48] did show that preoperative poor ENG responses appeared to be a good predictor of freedom from postoperative dizziness. Continuous dizziness was related to preoperative bilateral hyporeflexia that was "reduced to nil" postoperatively. Delayed, intermittent type of dizziness had no obvious ENG findings. Fina et al.[49] showed no relationship between preoperative ENG and postoperative dizziness, although most of their patients were affected by delayed, intermittent vertigo. Mangham reported changes in rotational chair testing in one subject that were associated with postoperative, positional dizziness that lasted for 4 days after surgery. Huygen et al.[112,113] reported four patients with unilateral VOR loss from surgery of which only two (50%) had temporary symptoms of "classic unilateral deficit" postoperatively.

Few studies have evaluated the effects of multichannel CIs on postural stability. Magnusson et al.[114] showed that CI candidates had increased body sway before surgery, although these patients maintained responsiveness to galvanic stimulation. Postoperative testing was not carried out. Likewise, Fina et al.[49] carried out computerized dynamic platform posturography (CDP) on 16 patients before CI and showed that abnormal results on conditions 5 and 6 were more common in patients with postoperative dizziness. Himi et al.[115] evaluated stabilimetry before and after CI and found no effects on postural stability in a group of patients with normal or hyporeflexic preoperative VOR function. Brey et al.[116] evaluated a group of 22 patients with pre- and postoperative posturography. In their study, postural stability worsened in one patient and improved in two patients after CI with the device activated.

In summary, VOR losses have been reported after CI surgery. In these cases, symptoms appear early, are usually transient, and are compensated for in time. Posturography testing has demonstrated few adverse effects of CI on postural maintenance, although studies are limited. The findings of the past studies likely imply that surgical deafferentation can occur through the mechanisms previously mentioned. Those patients who develop delayed symptoms appear to have fewer VOR changes, if any. These patients may have pathologic changes within the inner ear such as endolymphatic hydrops, although this remains speculative. The combination of careful history taking, VOR testing, and pathologic examination of temporal bones will allow these findings to be substantiated. Moreover, newer

TABLE AI.
Effects of Cochlear Implantation on Caloric Response.

	Preoperative Abnormal (%)*	Postoperative Decrease (%)*
Brey (A116)	5 of 22 (23)	13 of 17 (77)
Ribari (A120)	28 of 49 (57)	14 of 21 (67)
Vibert (A121)	6 of 14 (44)	2 of 6 (33)
Chouard (A122)	26 of 31 (84)	0 of 3 (0)
Eisenberg (A24)	12 of 16 (75)	3 of 16 (19)
Black (A18)	3 of 5 (60)	1 of 3 (33)
Van den Broek (A124)	31 of 35 (89)	3 of 6 (50)†
Huygen (A112)	21 of 25 (84)	3 of 6 (50)†
Huygen (A113)	34 of 50 (68)	4 of 13 (31)
Fina (A49)	46 of 76 (61)	N/A
Chiong (A125)	12 of 25 (48)	3 of 13 (19)
Rossi (A126)	24 of 32 (75)	1 of 20 (5)
Black (A33)	13 of 13 (100)	N/A
Kubo (A48)	(25)	N/A
Ito (A47)	7 of 55 (67)	9 of 24 (38)
Kiyomizu (A126)	40 of 97 (41)	9 of 16 (56)
Schneider (A129)	24 of 32 (75)	N/A
Magnusson (A114)	7 of 7 (100)	N/A
Szirmai (A130)	37 of 60 (62)	(N/A)
Hoffman (A46)	27 of 45 (60)	(N/A)
Himi (A115)	13 of 31 (42)	
Higgins (A131)	13 of 48 (27)	6 of 22 (27)

*Uses each study's definition of normal and abnormal (variable). Abnormal always included areflexia ± hyporeflexia. Directional preponderance as an isolated finding was excluded when possible.
†Includes same patients.

TABLE AII.
Categorical Analysis of Preoperative Dizziness Handicap Inventory (DHI) for All Patients.*

Category	Severity Category	
	Dizzy < 12 time/yr	Continuously Dizzy
Functional (%)		
Low	46 (59)	65 (83)
Same	24 (31)	10 (13)
High	8 (10)	3 (4)
Emotional (%)		
Low	0 (0)	58 (74)
Same	65 (83)	17 (22)
High	13 (17)	3 (4)
Physical (%)		
Low	51 (65)	61 (77)
Same	19 (24)	15 (19)
High	9 (11)	3 (4)
Total (%)		
Low	59 (76)	63 (81)
Same	6 (8)	12 (15)
High	13 (17)	4 (4)

*Values are numbers of patients in each category (%). Comparisons made to Jacobsen et al. (A132) severity groups (mean ± SD).

testing modalities such as vestibular-evoked myogenic potentials (VEMPs) may be better suited for identifying inner ear pathology in these patients.

Cochlear implantation and vestibular stimulation. As previously mentioned, electrical stimulation of the vestibular system was described in the early 19th century. Theoretically, electrical vestibular stimulation could result in altered inputs that disrupt vestibular nuclei symmetry, thereby inducing VOR and vestibulospinal outputs that are perceived as postural instability, vertigo, and dizziness. Black[37] and Black et al.[41] initially postulated that galvanic disruption by way of a carrier wave that was generated by the early generation CIs was responsible for the postural instability he observed in patients with their devices activated in quiet. Although these findings were later disputed by Eisenberg et al.,[42] the hypothesis was interesting. Today, CI devices lack a carrier wave. Stimulation generally uses discrete biphasic pulses delivered in a way that avoids charge buildup.

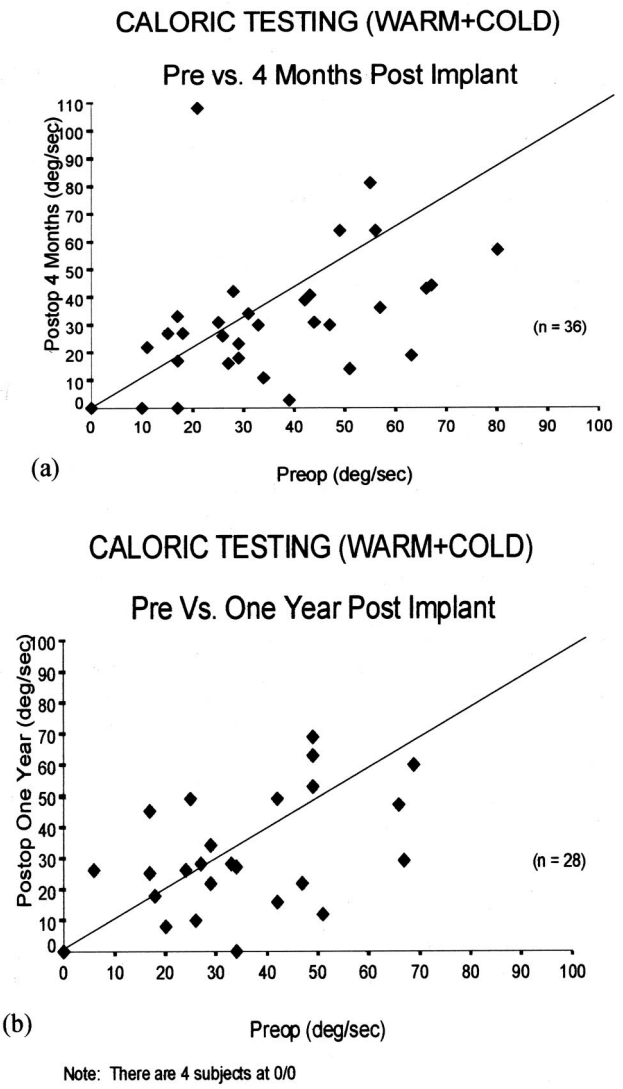

Fig. A1. Electronystagmography (ENG) total caloric response scores (warm + cool) for the implanted ear plotted preoperative versus (**A**) 4-month and (**B**) 1-year for all patients. A score below the diagonal indicates a decrease in the implanted ear caloric response at the testing interval while those above the diagonal are increased.

Few reports since the time of Black[37,41] and Eisenberg et al.[24] have further addressed the issue of vestibular stimulation by CIs. Clinically, it certainly appears

TABLE AIII.
Mean (SD) Change in Dizziness Handicap Inventory (DHI) for All Patients by Follow-Up Interval.*

Category	1 Month (n = 62)	4 Months (n = 41)	1 Year (n = 34)	2 Years (n = 15)
Functional	−0.33 (3.92)	1.32 (5.30)	1.83 (6.19)	2.80 (10.28)
Emotional	−0.91 (5.75)	1.60 (5.01)†	2.46 (6.58)†	1.73 (10.92)
Physical	−0.30 (4.22)	0.00 (5.31)	0.23 (5.44)	0.88 (8.10)
Total	−1.55 (11.62)	3.27 (14.31)	4.52 (16.45)	5.47 (28.50)

*Change is pairwise preoperative DHI minus postoperative value. Positive values denote improvements, and negative values denote worsening in the DHI score.
†$P < .05$.

TABLE AIV.
Mean Change (SD) in Total Caloric Response (warm + cold) for All Patients by Follow-Up Interval.*

Ear	4 Months (n = 28)	1 Year (n = 22)	2 Years (n = 10)
Implanted (deg/s)	4 (24)	4 (19)	13 (18)†
Unimplanted (deg/s)	0 (17)	2 (18)	1.00 (14)

*Change is pairwise preoperative total caloric response (deg/s) minus postoperative total caloric response (deg/s). Those with preoperative values of 0 were excluded.
†$P < .05$.

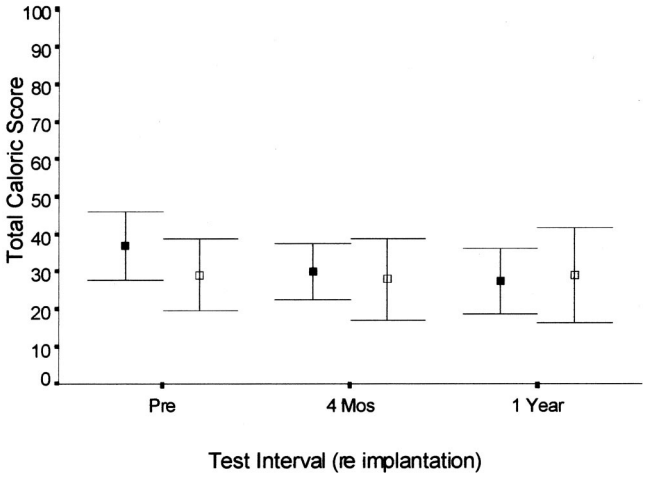

Fig. A2. Mean ± 95% confidence interval (2SD) total caloric response (warm + cold) for the implanted and non-implanted ears for patients with data at all intervals.

to be a rare complaint among patients. Black described two patients who experienced vestibular stimulation on initial activation of their extracochlear implants that was quickly compensated for.[18] Hoffman and Cohen[44] reported only 11 (0.36%) cases of 3,064 adult Nucleus 22 CI patients who experienced device-related dizziness. Hoffman later reported 1 case among 60 respondents to a questionnaire regarding postimplantation dizziness.[46] This case was resolved with mapping. Wong et al.[117] noted nystagmus in a child after activation of a multichannel device using increased pulse widths (200 μs) in the monopolar stimulation mode. Sennaroglu et al.[118] also reported vestibular stimulation in the form of nystagmus in a child with a common cavity. This was also programmed around. In an interesting study, Bance et al.[119] described a patient who experienced significant vestibular signs during stimulation associated with a loud noise and "shock-like" sensation. In a subsequent research study by this group, 1 (5.9%) in 17 patients experienced totally asymptomatic vestibular stimulation by their implant, as evidenced by nystagmus. Postural stability testing was not performed. These studies again show that vestibular stimulation is possible in patients with CI. Fortunately, with modern cochlear

TABLE AV.
Mean (SD) Change in Sinusoidal Harmonic Accelerations for All Patients by Follow-Up Interval.*

Variable	1 Month (n = 64)	4 Months (n = 35)	1 Year (n = 32)	2 Years (n = 14)
Phase				
0.01	1.75 (39.93)	3.17 (58.65)	11.94 (38.72)	−15.29 (30.46)
0.02	−1.17 (52.66)	−5.17 (36.71)	−7.50 (20.57)	5.26 (34.38)
0.04	4.50 (42.67)	10.06 (72.55)	1.03 (34.73)	−9.93 (38.25)
0.08	−3.06 (18.75)	−4.68 (26.51)	−5.68 (26.13)	−1.94 (27.19)
0.16	0.97 (22.11)	17.32 (61.52)	−2.67 (18.35)	−3.38 (17.56)
0.32	−2.77 (30.72)	10.79 (41.77)	5.29 (39.48)	−1.25 (16.81)
Gain				
0.01	0.01 (0.12)	0.04 (0.17)	0.03 (0.10)	0.02 (0.10)
0.02	0.16 (1.20)	0.03 (0.11)	0.08 (0.12)†	0.00 (0.17)
0.04	0.01 (0.17)	0.02 (0.18)	0.04 (0.18)	−0.03 (0.20)
0.08	0.00 (0.18)	0.03 (0.17)	0.03 (0.12)	0.02 (0.18)
0.16	0.00 (0.16)	0.00 (0.17)	0.02 (0.18)	0.03 (0.18)
0.32	0.02 (0.23)	0.03 (0.18)	−0.02 (0.19)	−0.02 (0.14)
Symmetry				
0.01	−0.25 (21.64)	−1.49 (21.57)	−8.91 (24.02)†	−0.21 (14.12)
0.02	0.64 (19.80)	1.50 (21.43)	−4.73 (18.23)	−2.79 (16.27)
0.04	−1.32 (14.11)	0.94 (22.92)	1.68 (14.82)	6.50 (22.67)
0.08	−1.73 (16.48)	−2.50 (15.11)	−3.80 (17.46)	−0.94 (16.36)
0.16	1.72 (16.51)	2.03 (13.17)	0.27 (16.69)	1.81 (10.99)
0.32	0.27 (13.27)	−2.29 (17.26)	−2.54 (19.16)	−4.88 (13.32)

*Change is pairwise preoperative score minus postoperative score with the device "off."
†$P < .05$.

TABLE AVI.
Mean (SD) Change in Sinusoidal Harmonic Accelerations for Patients with Substantial Loss of Caloric Response by Follow-Up Interval.*

	4 Months			1 Year		
Variable	Loss† (n = 5)	No loss† (n = 17)	*P* Value	Loss† (n = 5)	No loss† (n = 11)	*P* Value
Phase						
0.01	20 (22)	2 (18)	NS	12 (22)	4 (24)	NS
0.02	15 (41)	−2 (11)	NS	2 (21)	−4 (9)	NS
0.04	−8 (10)	−5 (11)	NS	−9 (8)	−1 (7)	NS
0.08	−15 (14)	−3 (13)	.046	−13 (13)	1 (14)	NS
0.16	−10 (17)	6 (10)	.045	−1 (8)	6 (8)	NS
0.32	2 (2)	14 (37)	NS	0 (7)	5 (12)	NS
Gain						
0.01	0.02 (0.17)	0.03 (0.19)	NS	0.05 (0.15)	0 (0.09)	NS
0.02	−0.06 (0.15)	0.06 (0.12)	.054	0.13 (0.15)	0.10 (0.13)	NS
0.04	−0.09 (0.23)	0.02 (0.20)	NS	0.16 (0.18)	−0.02 (0.23)	.045
0.08	−0.04 (0.09)	0.02 (0.19)	NS	0.04 (0.09)	0.05 (0.15)	NS
0.16	−0.09 (0.14)	0.05 (0.19)	NS	0.07 (0.15)	0.01 (0.20)	NS
0.32	0.00 (0.10)	0.04 (0.16)	NS	0.05 (0.12)	0.06 (0.20)	NS

*Change is pairwise preoperative score minus postoperative score with the device "off."
†Loss = decrease in 4-month total caloric response ≥ 21 deg/s. No loss = a change in 4-month total caloric response < 21 deg/s.

stimulators, this appears to be an extremely uncommon occurrence that is usually asymptomatic. Studies evaluating the effects of modern multichannel CIs on postural stability are lacking aside from the information enclosed in the present study.

Summary of Literature on Vestibular Effects of Cochlear Implantation

Vestibular symptoms are variously reported among patients undergoing modern, multichannel CI. These apparently can occur immediately after surgery, in a delayed fashion with episodic vertigo, or associated with electrical stimulation. Symptoms may be related to previous vestibular problems or may arise de novo. Findings of vestibular testing as well as temporal bone histopathology suggest that early symptoms are probably related to surgical deafferentation. The causes of delayed symptoms are less clear. Finally, vestibular stimulation from modern devices appears to be a rare phenomenon that may be asymptomatic. The present study further explores the effects of modern multichannel CI surgery and device activation on 1) dizziness-related disability and 2) postural stability. VOR reflex testing was used in an attempt to decipher the mechanisms underlying the findings.

APPENDIX II

Supplementary Results

Selected results of the study are presented in detail in the main article. The supplementary results include additional Dizziness Handicap Inventory (DHI), ENG, sinusoidal harmonic acceleration (SHA), and CDP data.

Dizziness Handicap Inventory (DHI)

For all patients entering the study, DHI information was gathered preoperatively and at each of the following intervals. Table AII shows the DHI data as it compares with the severity categories defined by Jacobsen and Newman.[132] As in the main body of the article, the majority of patients are not substantially affected by dizziness. However, there were three patients that were significantly

TABLE AVII.
Categorical Analysis of Preoperative Computerized Dynamic Platform Posturography (CDP) for All Patients.

	Postural Stability*		
Condition	Low (%)	Normal (%)	High (%)
Condition 5	52 (65)	23 (29)	5 (6)
Condition 6	46 (56)	35 (43)	1 (1)
Composite Score	48 (59)	31 (38)	3 (4)

*Values are numbers of patients in each category (%). Normal values are means ± 2 SD after Hirabayashi et al. (A133). "Low" values indicate poor postural stability. "High" values indicate postural stability scores better than the normal values.

TABLE AVIII.
Mean (SD) Change in Computerized Dynamic Platform Posturography (CDP) for All Patients by Follow-Up Interval.*

Condition	1 Month	4 Months	1 Year	2 Years
Condition 5	−2 (14)	−3 (16)	−10 (19)*	−10 (18)*
Condition 6	1 (15)	−4 (18)	−5 (18)	−12 (20)*
Composite	−1 (8)	−4 (10)*	−7 (11)*	−2 (15)

*Change is pairwise preoperative score minus postoperative score. Negative values indicate and increase (improvement) in postural stability.
†$P < .05$.

affected with dizziness preoperatively, as demonstrated by DHI values that were higher than those reported by a group of patients with continuous dizziness.

Vestibulo-Ocular reflex (VOR) testing. After CI surgery, DHI scores changed significantly only for the emotional subcategory scores. Figure 1 in the main text shows the absolute pairwise changes for each patient by follow-up. Table AIII shows the mean pairwise changes (SD) for all of the subcategory and total scores. It remains interesting that those with high preoperative DHI total scores appeared to be at risk for 1-month postoperatively high DHI scores. By 4 months and 1 year, the patients were doing better, although attrition makes this somewhat difficult to analyze.

Alternate bithermal caloric stimulation (ENG). ENG data were gathered at 4 months, 1 year, and 2 years for all of the patients. In the main article, changes in total caloric response for both the implanted and unimplanted ear are shown at the 4-month interval. This format was chosen to represent the data because changes in vestibular function relative to the contralateral ear are presumably what would predispose the patient to symptoms rather than absolute changes. As additional data, Figure A1 shows the absolute changes preoperatively versus postoperatively for the implanted ear. Although, on the whole, these changes were not statistically significant, a look at the outliers is instructional. There were 1 (4%) of 28 ears with total caloric responses greater than 16 deg/second preoperatively that were reduced to 0 deg/second at the 4-month visit. At 1 year, 1 (4%) of 23 ears at risk were reduced to 0 deg/second. Thus, complete vestibular deafferentation did occur in the operated ear in less than 5% of ears. Mean pairwise changes are shown in Table AIV, and mean (±95% confidence intervals) values for those patients with ENG data at each interval (preoperative, 4 months, and 1 years) are shown in Figure A2. The significant mean change in total caloric response from baseline ($P < .05$; actually $P = .048$) seen at the 2-year point is probably caused by selection bias. The baseline values for those with 2-year data are substantially above those at 4 months and 1 year (data not shown). In summary, some significant changes in ENG can occur after CI. It seems, however, that complete deafferentation remains relatively uncommon. The differences in our study and other previous studies remain speculative. Surgical technique, variations in electrode design, and patient populations may explain these differences.

Rotational chair testing using sinusoidal harmonic acceleration (SHA). SHA testing results are complex to display in that there are at least three variables tested (phase, gain, and symmetry) at each of six rotation frequencies (0.01, 0.02, 0.04, 0.08, 0.16, 0.32 Hz) and three more frequencies repeated with the device "on" at five testing intervals (preoperative, 1 month, 4 months, 1 year, and 2 years). Before implantation, categorical analyses were carried out relative to the manufacturer's normal values and are displayed in the main text (Table V). To demonstrate pairwise changes for patients is difficult without showing a separate graph for each frequency. For the group as a whole, there were few significant changes compared with baseline. Similar findings were seen for those patients defined to be "at risk" for significant loss by having a preoperative total caloric response greater than 16 deg/second. However, it was interesting to look at changes in those subjects who had a demonstrated drop in total caloric response 21 deg/second or greater in comparison with those who did not have a change at the 4-month interval (Table AVI). For those patients, there appeared to be a substantial number of changes, although these did not reach significance because of small patient numbers and large standard deviations. Specifically, low frequency changes in phase and gain were apparent in this group. This was consistent with the changes in ENG for these patients.

Computerized Dynamic Platform Posturography (CDP)

CDP testing results before and after CI are reported in detail in the main article. Table AVII shows the categorical analysis for all patients' preoperative scores. Fifty-six percent to 65% of patients had CDP scores in either condition 5, 6, or composite that were below normal values. Thus, CI candidates have significantly impaired postural stability before implantation. Table AVIII shows the mean pairwise differences for the scores in the various conditions at each time interval after surgery with the device off (but after chronic use). Significant improvements are seen across many of the categories, although these appear to be of small magnitude. The pairwise comparison figures (Fig. 3) in the main text show that a number of patients had dramatic improvements in CDP results by 1 year post surgery. Some patients that routinely fell on conditions 5 and 6 were able to maintain reasonably good stability after surgery.

The impact of device activation is shown in Table AIX. For all comparisons, the mean change is negative, implying improvement for device activation. This effect is illustrated in Figure 4 in the main text where dramatic improvements in condition 6 scores were seen over the entire group at the 1-year interval. Condition 5 and composite score changes were somewhat less impressive, possibly owing to the fact that these represent easier testing conditions.

TABLE AIX.
Mean (SD) Change in Computerized Dynamic Platform Posturography (CDP) by Device Activation Status for All Patients at Each Interval.*

Condition	4 Months	1 Year	2 Years
Condition 5	−5 (16)†	−1 (9)	−3 (16)
Condition 6	−2 (11)	−6 (14)‡	−3 (11)
Composite	−1 (7)	−1 (5)	−3 (14)

*Change is pairwise device "off" score minus device "on" scores at each testing interval. Negative values imply an improvement with device activation in music at 70 dB SPL.
†$P = .056$.
‡$P < .05$.

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