Acta Otolaryngol (Stockh) 1995; Suppl 520: 270–272

The Risk of Vestibular Function Loss after Intracochlear Implantation

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Huygen PLM, Hinderink JB, van den Broek P, van den Borne S, Brokx JPL, Mens LHM, Admiraal RJC. *The risk of vestibular function loss after intracochlear implantation.* Acta Otolaryngol (Stockh) 1995; Suppl 520: 270–272.

Sixty patients were selected for cochlear implantation and 50 of them received an intracochlear implant (Nucleus). Vestibular function was evaluated before and after surgery using a caloric test and a velocity step test. Sixteen patients had normal or residual vestibular function before surgery, 11 bilateral and 5 unilateral; in 3 of the latter patients, the ear with vestibular areflexia was elected for implantation, which reduced the number of patients at risk for vestibular dysfunction to 13. Vestibular function was preserved in all of these patients except for 4; the risk of vestibular function loss can therefore be rated at about 31%. *Key words: deafness (acquired, genetic), vestibular areflexia, vestibular hyporeflexia.*

INTRODUCTION

Only a few reports have appeared on the results of vestibular tests in relation to intracochlear implantation (1–5). According to our previous reports (6, 7) on our own (preliminary) data and other reported data, the risk of vestibular function loss can be estimated at between 50 and 60%. Since the submission of our previous reports, several new patients have been implanted at our department and our current data indicate that the risk of vestibular function loss may be lower.

MATERIALS AND METHODS

Our study population comprised 60 patients, 50 of whom received an intracochlear implant, i.e. a Nucleus device (Cochlear Corporation, Englewood, CO). Extracochlear implantation was performed in the early period (1987–1990) and, more recently, in a patient with Mondini-type dysplasia. The patients (30 males, 30 females) were aged between 5 and 68 years; 18 of them were younger than 13 years. The aetiological diagnoses of these patients are presented in Table I. The methods used to evaluate vestibular function

Table I. *Preimplant findings by aetiology*

AD = autosomal dominant, AR = autosomal recessive, hf = high frequency, SNHL = sensorineural hearing loss (MIM number) (10)

	Vestibular function			
Aetiology	Bilateral areflexia	Hyporeflexia	Normoreflexia	Hyperreflexia
Meningitis	27	2		
Mumps		1		
Head trauma	2			
Ototoxicity				2
Unknown		4	3	
Congenital severe SNHL				
Usher I (276900)	6			
Mondini dysplasia		1		
AD (124580)			1	
AR (220700, 800)	2			
Progressive SNHL				
Otosclerosis	1	1	1	
AD hf (124800)			2	
AR (221650)				1
AR? unidentified		1	1	
Total	38	10	8	3

 ISSN 0365-5237

Table II. *Patients with normal or residual vestibular function preimplant*

Initial number of patients:	22
Extracochlear	2 –
Not evaluable	4 –
Evaluable	16
Not at risk of losing bilateral function	3 –
Evaluable at risk	13

and the classification into the categories vestibular areflexia, hyporeflexia, normoreflexia (and hyperreflexia) have been described previously (6, 7).

RESULTS

Preimplant findings (60 patients)

None of our patients showed any gaze-evoked nystagmus or spontaneous nystagmus. Smooth pursuit and optokinetic nystagmus (OKN) responses were normal in 58 of the patients. In the remaining 2 who had Usher's type I syndrome, the OKN response levels were too low (they had constricted visual fields and poor visual acuity). Table I shows the preimplant findings in our patients according to their aetiology (in 1 child after meningitis it could not be concluded whether normo- or hyperreflexia applied to the sinusoidal responses and caloric tests were omitted). Vestibular areflexia manifested itself in 38 patients (63%) as a total lack of nystagmus after velocity step tests of 90°/s (plus 250°/s in 3 cases).

Findings after intracochlear implantation (50 patients)

Of the 22 patients with preimplant vestibular (hypo- or normal) function, 2 received extracochlear implants, 4 were not evaluable (3 had severe hyporeflexia and caloric tests were therefore omitted and 1 had an abnormally shaped semicircular canal which was inadvertently opened during surgery causing vestibular loss). All of the 16 remaining patients underwent intracochlear implantation and their preimplant vestibular function could be evaluated (Table II). Three of these patients were not at risk because the ear elected for implantation had complete vestibular function loss.

Table III shows that 11 of the 13 remaining patients whose vestibular function was at risk on the side of implantation, were at risk of developing unilateral loss (their preimplant function had been intact bilaterally). Three of these patients had a vestibular deficit following implant surgery. One of them did not experience any appreciable symptoms, presumably because the lost labyrinth had already shown reduced sensitivity before implantation (the caloric response level was 56% of that obtained from the other labyrinth). The other 2 patients had the classical symptoms of a unilateral vestibular deficit. Eight patients had a repeat vestibular examination which showed complete preservation of vestibular function in the implanted ear. One of them had vestibular complaints and showed hyperactive velocity step responses postimplant, but she had displayed similar findings before implantation, which could be attributed to hyperventilation (8); physical breathing control therapy was recommended

Table III. *Outcome in at-risk patients.*

Exposed to risk	Function lost	Function intact
At risk of losing unilateral function		
11	3[a]	8
At risk of losing bilateral function		
2	1[b]	1
Total		
13	4 (31%)	9

[a] 1 symptom-free (preimplant function already partially lost).
[b] All the symptoms of total vestibular areflexia (9).

In 2 patients with unilateral function loss, the other labyrinth was at risk because it had been elected for implantation. After the implantation, one of them revealed bilateral vestibular areflexia with the associated typical symptoms (9) and the other had intact function.

A total of 13 patients who were at risk of losing vestibular function in the ear elected for implantation could be evaluated. Four out of these 13 patients lost their function. Therefore, the risk of losing vestibular function through intracochlear implantation can be rated at 4 out of 13, or about 31%.

DISCUSSION

During intracochlear implantation, the electrode is inserted through the round window and led into the scala tympani over a length of some 2 cm. This procedure may damage the basilar membrane or the spiral ligament and this carries the risk of endolymph mixing with perilymph with subsequent loss of inner ear functions. At present, our results indicate a risk of about 31%, which is somewhat lower than the 50–60% mentioned in our previous reports (6, 7). Nevertheless, we are of the opinion that the patient should be informed beforehand about this risk—if applicable—and the possible consequences of vestibular

areflexia (9). The same applies to the impending risk of unilateral function loss, although it seems reasonable to suppose that this would mean a much less severe handicap to most patients. In one of our patients, the preimplant caloric sensitivity on the side that was later elected for implantation was hardly more than half of that on the other side; the total unilateral loss of vestibular function which occurred after implantation took a subclinical course and the patient remained asymptomatic.

The present selection of cochlear implant candidates offers some indication as to what can be expected to happen to vestibular function in relation to aetiology in similar cases. On the one hand, bilateral vestibular areflexia, by definition, is to be found in Usher's type I syndrome ([10] "Mendelian inheritance in Man" or MIM number 276900 [11]) and it generally occurs in patients with bilateral deafness following meningitis (12) or head trauma. On the other hand, the autosomal dominant (AD) syndrome of progressive sensorineural hearing loss (SNHL) which starts in (early) childhood at the high frequencies (MIM 124800) is generally associated with normal vestibular function ([12], [13] and additional unpublished data) and, presumably, this also applies to autosomal recessive (AR) progressive SNHL with childhood onset (MIM 221650) ([14] and additional unpublished data). In other categories of patients, e.g. with acquired bilateral SNHL, congenital AD SNHL (MIM 124580), congenital AR severe SNHL (MIM 220700, 220800 [12] and otosclerosis [11]), it is uncertain what will happen to their vestibular function. We are therefore of the opinion that vestibular examination should be performed as an integral part of the selection procedure of all prospective candidates for implantation, because at the very least it will help to avoid the development of bilateral areflexia in some patients.

ACKNOWLEDGEMENTS

We thank Mrs. A. J. Beynon, M. G. M. Nicolasen and the patients for their cooperation.

REFERENCES

1. Black FO. Present vestibular status of subjects implanted with auditory prostheses. Ann Otol Rhinol Laryngol 1977; Suppl 38: 49–56.
2. Chouard CH, Fugain C, Meyer B, Lacombe H. Résultats cliniques de l'implant cochléaire à multi-électrodes. Ann Otolaryngol (Paris) 1981; 98: 593–612.
3. Eisenberg LS, Nelson JR, House WF. Effects of the single-electrode cochlear implant on the vestibular system of the profoundly deaf adult. Ann Otol Rhinol Laryngol 1982; Suppl 91: 47–54.
4. Black FO, Lilly DJ, Peterka RJ, Fowler LP, Simmons FB. Vestibulo-ocular and vestibulospinal function before and after cochlear implant surgery. Ann Otol Rhinol Laryngol 1987; Suppl 128: 106–9.
5. Mangham CA. Effects of cochlear prostheses on vestibulo-ocular reflexes to rotation. Ann Otol Rhinol Laryngol 1987; Suppl 128: 101–4.
6. Van den Broek P, Huygen PLM, Mens LHM, Admiraal RJC, Spies T. Vestibular function in cochlear implant patients. Acta Otolaryngol (Stockh) 1993; 113: 263–5.
7. Huygen PLM, van den Broek P, Spies TH, Mens LHM, Admiraal RJC. Does intracochlear implantation jeopardize vestibular function? Ann Otol Rhinol Laryngol 1994; 103: 609–14.
8. Theunissen EJJM, Huygen PLM, Folgering HT. Vestibular hyperreactivity and hyperventilation. Clin Otolaryngol 1986; 11: 161–9.
9. Gresty MA, Hess K, Leech J. Disorders of the vestibulo-ocular reflex producing oscillopsia and mechanisms compensating for loss of labyrinthine function. Brain 1977; 100: 693–716.
10. McKusick VA. Mendelian inheritance in man. Catalogs of autosomal dominant, autosomal recessive, and X-linked phenotypes, 10th ed. Baltimore: Johns Hopkins University Press, 1992.
11. Huygen PLM, Verhagen WIM. Peripheral vestibular and vestibulo-cochlear dysfunction in hereditory disorders. A review of the literature and a report on some aditional findings. J Vestib Res 1994; 4: 81–104.
12. Huygen PLM, Van Rijn PM, Cremers CWRJ, Theunissen EJJM. The vestibulo-ocular reflex in pupils at a Dutch school for the hearing impaired; findings relating to acquired causes. Int J Pediatr Otorhinolaryngol 1993; 25: 39–47.
13. Gorlin RJ, Toriello HV. Hereditary hearing loss and its syndromes. New York: Oxford University Press, 1994.
14. Cremers CWRJ. Autosomal recessive nonsyndromal progressive deafness in childhood. A separate clinical and genetic entity. Int J Pediatr Otorhinolaryngol 1979; 1: 193–9.

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