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SACCULO-COLLIC FUNCTION IN 8 CHILDREN BEFORE AND AFTER COCHLEAR IMPLANTS

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Abstract

Introduction: Cochlear implantation is a standard intervention for children with severe to profound sensorineural hearing loss. However, the procedure may affect vestibular function due to its anatomical proximity to the cochlea. This study evaluated sacculo-collic pathway function using cervical vestibular evoked myogenic potentials (cVEMPs) in children undergoing unilateral cochlear implantation, comparing responses at three times: pre-operative (pre-op), immediate post-operative (post-op), and at a 3-month follow-up.

Material and methods: This longitudinal study was conducted on 9 children (mean age 3.7 ± 1.4 years) with congenital bilateral severe-to-profound sensorineural hearing loss who all received a CI in the right ear. Assessments were done at 3 time points using cVEMPs. Parameters analysed comprised P1 and N1 latencies, P1–N1 amplitude, and interaural amplitude asymmetry ratio. Responses for 1 child could not be recorded at any time point, so reported data relate to the remaining 8 children.

Results: At the pre-op stage, normal cVEMP responses were observed in 8 of the 9 children, with normal P1–N1 amplitudes and latencies. Post-operatively, 4 of the 8 exhibited absent or markedly reduced responses in the implanted (right) ear, with a significant decrease in amplitude (mean 15.1 ± 21.2 μ V vs pre-op 101.5 ± 90.2 μ V; $p = 0.029$). At the follow-up, partial recovery of amplitude was noted (mean 35.1 ± 30.3 μ V). No statistically significant changes were found in latency across the different time points ($p > 0.05$), and responses in the non-implanted (left) ear remained relatively stable. However, 6 participants showed amplitude asymmetry $> 33\%$ post-operatively, with 4 exhibiting complete (100%) asymmetry; this difference was statistically significant $p < 0.001$.

Conclusions: Findings suggest that cochlear implantation in children may induce temporary alterations in sacculo-collic function, mainly evidenced by a reduction in cVEMP amplitude post-operatively. The observed partial recovery at follow-up suggests reversible vestibular changes, possibly due to central compensation. This underscores the importance of ongoing vestibular assessment in pediatric CI recipients to monitor potential transient dysfunction and recovery patterns.

Keywords: cVEMP • cochlear implantation • pediatric vestibular function • sacculo-collic reflex

FUNKCJA DROGI WORECZKOWO-SZYJNEJ U 8 DZIECI PRZED I PO IMPLANTACJI ŚLIKAKOWEJ

Streszczenie

Wprowadzenie: Implantacja ślimakowa stanowi standardową metodę leczenia dzieci z głębokim lub znacznym odbiorczym ubytkiem słuchu. Ze względu jednak na bliskie sąsiedztwo anatomiczne ślimaka i narządu przedsionkowego zabieg może wpływać na czynność układu przedsionkowego. W niniejszym badaniu oceniano funkcję drogi woreczkowo-szyjnej za pomocą szyjnych przedsionkowych mięśniowych potencjałów wywołanych (cVEMP, cervical vestibular evoked myogenic potentials) u dzieci poddawanych jednostronnej implantacji ślimakowej. Wyniki porównano w trzech punktach czasowych: przed operacją (*pre-op*), bezpośrednio po operacji (*post-op*) oraz po 3 miesiącach obserwacji.

Materiał i metody: Badanie o charakterze podłużnym przeprowadzono u 9 dzieci (średni wiek: $3,7 \pm 1,4$ roku) z wrodzonym obustronnym znacznym do głębokiego niedosłuchem odbiorczym, u których wszczepiono implant ślimakowy do prawego ucha. Oceny wykonywano w trzech punktach czasowych z wykorzystaniem badania cVEMP. Analizowane parametry obejmowały latencje fal P1 i N1, amplitudę zespołu P1–N1 oraz międzyszną proporcję asymetrii amplitudy. U jednego dziecka nie udało się zarejestrować odpowiedzi w żadnym punkcie czasowym, dlatego przedstawione wyniki dotyczą pozostałych 8 pacjentów.

Wyniki: Przed zabiegiem prawidłowe odpowiedzi cVEMP stwierdzono u 8 z 9 dzieci, z prawidłowymi wartościami amplitudy P1–N1 oraz latencji. Bezpośrednio po operacji u 4 spośród 8 dzieci zaobserwowano brak odpowiedzi lub wyraźne zmniejszenie odpowiedzi w uchu implantowanym (prawym), czemu towarzyszył istotny statystycznie spadek amplitudy (średnio $15,1 \pm 21,2$ μ V wobec $101,5 \pm 90,2$ μ V przed operacją; $p = 0,029$). W badaniu kontrolnym po 3 miesiącach odnotowano częściową poprawę amplitudy odpowiedzi (średnio $35,1 \pm 30,3$ μ V).

Nie wykazano istotnych statystycznie zmian latencji pomiędzy poszczególnymi punktami czasowymi ($p > 0,05$), a odpowiedzi rejestrowane z ucha nieimplantowanego (lewego) pozostawały względnie stabilne. Jednocześnie u 6 uczestników po operacji stwierdzono asymetrię amplitudy przekraczającą 33%, przy czym u 4 dzieci występowała całkowita (100%) asymetria. Różnica ta była istotna statystycznie ($p < 0,001$).

Wnioski: Uzyskane wyniki sugerują, że implantacja ślimakowa u dzieci może powodować przejściowe zaburzenia funkcji drogi woreczkowo-szyjnej, manifestujące się przede wszystkim zmniejszeniem amplitudy odpowiedzi cVEMP po zabiegu. Obserwowana częściowa poprawa w badaniu kontrolnym wskazuje na możliwość odwracalnego charakteru zmian przedsionkowych, prawdopodobnie związanego z procesami kompensacji ośrodkowej. Wyniki te podkreślają znaczenie regularnej oceny funkcji przedsionkowych u dzieci korzystających z implantów ślimakowych w celu monitorowania przejściowych zaburzeń oraz przebiegu ich ustępowania.

Słowa kluczowe: cVEMP • implant ślimakowy • funkcje przedsionkowe u dzieci • odruch woreczkowo-szyjny

Introduction

In 2018, the World Health Organisation (WHO) reported a prevalence of hearing impairment of approximately 6.3% in India, where adult-onset deafness is estimated at 7.6%, while childhood-onset deafness stands at 2% [1]. Among children above 3 months old, sensorineural hearing loss affects about 16% of the population. The primary interventions for this type of hearing loss include the use of amplification devices and cochlear implants (CIs). A child with bilateral sensorineural hearing loss of 80 dB HL or greater, who shows no improvement after 6 months of rehabilitation with hearing aids, is considered an ideal candidate for cochlear implantation [2].

A CI is an electronic device containing a current source and an electrode array implanted into the cochlea; an electrical current is then used to stimulate the surviving auditory nerve fibres [3]. Optimal electrode placement is a prerequisite for maximising CI success. Standard CI surgery includes a mastoidectomy and facial recess approach to the middle ear and cochlea. Electrodes are placed in the scala tympani through the round window with a cochleostomy [4]. An ideal cochleostomy is inferior and slightly anterior to the round window membrane. A superior position results in injury to the spiral ligament and basilar membrane and risks insertion into the scala vestibule [4]. Other than the risk of insertion in the wrong partition of the cochlea, many traumas are associated with the insertion of electrodes. Thus, cochlear implantation can affect the vestibular system through various mechanisms such as direct trauma caused by electrode insertion, acute serous labyrinthitis due to cochleostomy, foreign body reaction with labyrinthitis, endolymphatic hydrops, and electrical stimulation from the implant itself [5]. These mechanisms can alter vestibular function temporarily or may cause permanent damage. Therefore, vestibular testing is helpful for pre-, post-, and follow-up in CI surgery to assess the extent of dysfunction and monitor patient recovery. Compared to adults, assessing vestibular function in children is not easy. Nevertheless, the current study aimed to examine the vestibular function in children with severe to profound hearing loss before and after a CI.

Material and methods

A longitudinal comparative study was conducted to evaluate the sacculo-collic pathway using cervical vestibular evoked myogenic potential (cVEMP) measurements at three defined time points: before cochlear implantation (pre-op), on the switch-on day of the implant system (post-op),

Table 1. Ages of participants; F, female; M, male

Case no.	Gender	Age at implantation [years]
1	F	5
2	F	2
3	M	2
4	F	5
5	M	3
6	M	5
7	F	5
8	M	2
9	M	5

and after 3 months of surgery (follow-up). Initially, 18 children aged 2 to 5 years with congenital bilateral severe-to-profound sensorineural hearing loss were enrolled, but those with middle ear history, pre-existing vestibular dysfunction, neuromuscular or musculoskeletal deficits, and cognitive impairments were excluded from the study. Therefore, only 9 participants completed all assessments – comprising 5 boys and 4 girls – with a mean age of 3.7 ± 1.4 years at implantation, all receiving unilateral right-ear implants (Table 1). Switch-on for all the participants was done after 2 weeks.

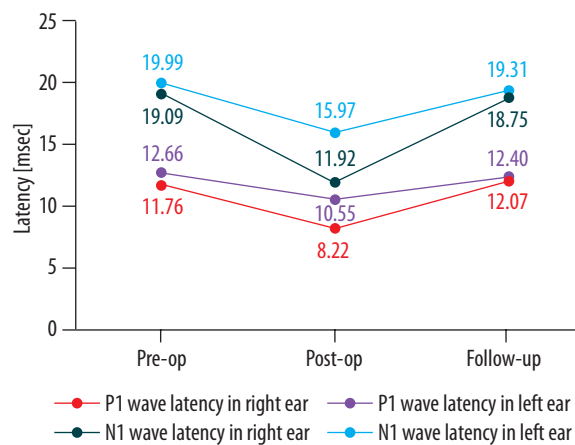
cVEMP measurements were performed using the Eclipse instrument (Interacoustics). Key cVEMP parameters analysed were P1 and N1 latencies, P1–N1 amplitude, and P1–N1 amplitude asymmetry ratio across the three time points. Latency of P1 of more than 20 ms and N1 of more than 30 ms were considered abnormal. Interaural latency difference of more than 3.5 ms for each component was considered abnormal, as was amplitude asymmetry greater than 33% [6].

Ethical approval was obtained from the Institutional Ethical Committee, and written informed consent was secured from parents or guardians before data collection.

Audiological testing was performed in a sound-treated environment conforming to ANSI S3.1(1991) standards for ambient noise. During cVEMP recordings, each child was seated comfortably either in a chair or the caregiver's lap and instructed to turn their head away from the test ear without torso rotation, maintaining an awake and stable posture. The active electrode was placed on the

Table 2. ANOVA results for both ears of 8 children for P1 latency, N1 latency, and P1–N1 amplitude at three time points

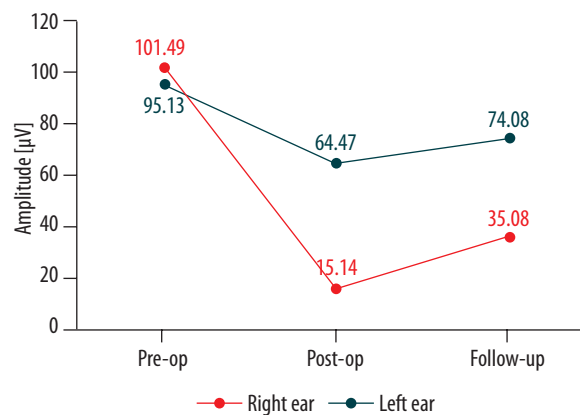
Parameter	Ear	Pre-op	Post-op	Follow-up	F-value	p-value
P1 latency (mean ± SD) [ms]	right	11.76 ± 4.43	8.22 ± 7.96	12.07 ± 4.60	2.373	0.162
	left	12.66 ± 4.81	10.55 ± 6.32	12.40 ± 4.72	1.463	0.262
N1 latency (mean ± SD) [ms]	right	19.09 ± 7.35	11.92 ± 11.52	18.75 ± 7.26	4.222	0.073
	left	19.99 ± 7.69	15.97 ± 9.50	19.31 ± 7.53	2.607	0.143
P1–N1 amplitude (mean ± SD) [μV]	right	101.49 ± 90.20	15.14 ± 21.18	35.08 ± 30.25	6.658	0.029*
	left	95.13 ± 75.95	64.47 ± 59.85	74.08 ± 55.05	2.507	0.145

* Statistically significant ($p < 0.05$)**Figure 1.** Mean P1 and N1 wave latency at 3 time points – before, immediately after, and at 3 months follow-up – in 8 children implanted with a CI in the right ear

upper one-third of the sternocleidomastoid muscle (SCM), the reference electrode was placed on the junction of the sternum and clavicle, and the ground was positioned on the lower forehead. Tone burst stimuli of 500 Hz at 95 dB nHL were used. To sustain adequate SCM muscle tension for reliable signal acquisition, visual stimuli such as videos were presented on tablets positioned to engage the child's gaze throughout testing.

Statistical analysis

The data obtained were subjected to statistical analysis using SPSS v.25. The normality of data distribution was confirmed using a Shapiro–Wilk test. The data was normally distributed, and so an analysis of variance (ANOVA) included Mauchly's test of sphericity, and a Greenhouse–Geisser test was applied to compare P1 and N1 latency and amplitude across three time points. Post hoc tests with Bonferroni adjustment compared specific time points (pre-op vs post-op, pre-op vs follow-up, and post-op vs follow-up) separately for each ear.

**Figure 2.** Mean P1–N1 amplitude at 3 time points – before, immediately after, and at 3 months follow-up – in 8 children implanted with a CI in the right ear

Results

Baseline sacculo-collic function was assessed using cVEMPs before CI surgery. Responses (P1 and N1 peaks) were present bilaterally in 8 of the 9 participants. Mean P1 latency in the to-be-implanted ear (right ear) was 11.76 ± 4.43 ms, with a mean N1 latency of 19.09 ± 7.35 ms. The non-implanted (left ear) showed comparable values with P1 latency of 12.66 ± 4.81 ms and N1 latency of 19.99 ± 7.69 ms (Table 2). P1–N1 peak-to-peak amplitude averaged 101.49 ± 90.20 μV in the right ear versus 95.13 ± 75.95 μV in the left ear, with no statistically significant interaural differences. One child (subject nr 2 in Table 1) demonstrated absent cVEMP responses bilaterally across all time points, suggesting preexisting vestibular dysfunction. The following data, therefore relates to the remaining 8 children.

On the day of switch-on, cVEMP responses were absent in 4 participants in the right ear and 2 in the left ear. The major difference was seen in the latency shift of P1 and N1. Mean P1 latency shortened to 8.22 ± 7.96 ms (implanted right ear) and 10.55 ± 6.32 ms (non-implanted

Table 3. Post hoc pairwise comparisons (estimated marginal means with Bonferroni correction)

Parameter	Comparison	Mean difference	Standard error	p-value
Implanted ear amplitude	pre-op vs post-op	86.36 μ V	30.31	0.043*
	pre-op vs follow-up	66.41 μ V	24.52	0.042*
	post-op vs follow-up	–19.95 μ V	10.55	0.240
Non-implanted ear amplitude	pre-op vs post-op	30.66 μ V	11.04	0.044*
	pre-op vs follow-up	21.05 μ V	6.75	0.027*
	post-op vs follow-up	–9.61 μ V	5.08	0.239
Asymmetry ratio	pre-op vs post-op	–58.78%	14.67	0.012*
	pre-op vs follow-up	–25.34%	12.45	0.045*
	post-op vs follow-up	33.44%	9.96	0.030*

* $p < 0.05$. Positive mean differences indicate a reduction from the first to the second time point. Negative mean differences for asymmetry ratio indicate an increase in asymmetry

left ear), while N1 latency reduced to 11.92 ± 11.52 ms and 15.97 ± 9.50 ms, respectively (**Figure 1**). As seen in **Figure 2**, mean P1–N1 peak-to-peak amplitude in the right ear decreased substantially to 15.1 ± 21.2 μ V, representing an approximately 85% reduction from baseline values. However, the left ear showed a less pronounced decline to 64.5 ± 59.9 μ V (a 32% reduction) (**Table 2**).

Statistical analysis using repeated measure ANOVA with Greenhouse–Geisser correction revealed that amplitude changes in the implanted ear pre and post implant were statistically significant ($F = 6.658$, $p = 0.029$). Post hoc pairwise comparisons with Bonferroni correction were conducted for the implanted ear amplitude. Significant differences were observed between pre-operative and post-operative measurements ($p = 0.043$), indicating immediate post-surgical amplitude reduction (**Table 3**). The difference between pre-operative and follow-up remained significant ($p = 0.042$), demonstrating incomplete recovery at 3 months. The post-operative to follow-up comparison showed a trend toward recovery but did not reach statistical significance ($p = 0.240$) (**Table 3**).

However, latency changes did not show any significance between these two time points (P1 latency: $p = 0.162$; N1 latency: $p = 0.073$) (**Table 2**).

However, in terms of amplitude, post hoc pairwise comparisons revealed that, for the non-implanted ear, there was a significant difference between pre-operative and post-operative measurements. **Table 3** shows that for this ear there was a significant difference between pre-op and post-op ($p = 0.044$), while the comparison between pre-op and follow-up was also significant ($p = 0.027$). However, the post-op to follow-up comparison was not significant ($p = 0.239$).

Turning to asymmetry ratio, post hoc analysis of changes demonstrated highly significant increases from pre-op to post-op ($p = 0.012$) and from post-op to follow-up ($p = 0.030$), reflecting initial deterioration and then partial recovery of interaural symmetry (**Table 3**). Interaural

amplitude asymmetry ratio exceeded 33% in 6 cases post-operatively, compared to only 1 pre-operatively, representing a statistically significant change ($F = 12.34$, $p = 0.001$).

At 3 months follow-up, substantial recovery of sacculo-collic function was seen (**Figures 1** and **2**). cVEMP responses were present in 8 children bilaterally, matching pre-operative response rates. Mean P1 latency in the implanted ear returned to near-baseline values at 12.07 ± 4.60 ms, with N1 latency at 18.75 ± 7.26 ms. The non-implanted ear demonstrated a similar trend with P1 latency of 12.40 ± 4.72 ms and N1 latency of 19.31 ± 7.53 ms (**Table 2**).

Nevertheless, P1–N1 amplitude recovery remained incomplete. The implanted ear showed a mean amplitude of 35.08 ± 30.25 μ V, representing 34.6% of preoperative values, while the non-implanted ear recovered to 74.08 ± 55.05 μ V (77.9% of baseline) (**Table 2**). Despite overall amplitude reduction, statistical analysis demonstrated no significant differences compared to baseline for the non-implanted ear ($p = 0.145$), whereas the implanted ear changes remained significant ($p = 0.029$). Interaural asymmetry ratios were normal in most cases, with only 2 children showing persistent asymmetry exceeding the 33% mark.

Discussion

Post-operative changes in vestibular function, as measured through cVEMP, were evident in the present study. Pre-operatively, cVEMP was present in 8 out of 9 participants for both ears. (One participant showed absent VEMP responses across all time periods bilaterally.) Post-operatively, however, cVEMPs were absent in 4 implanted ears and 2 non-implanted ears. But at the 3-month follow-up, responses were once again present in both ears of 8 participants, indicating there had been a partial recovery over time. We note that participants with absent responses did not report any dizziness or vertigo pre- or post-implantation, suggesting subclinical vestibular involvement.

Similar patterns have been reported by Jin et al., where 11 of 12 children demonstrated absent VEMP responses following cochlear implantation [7]. Similar results are reported in [8]. However, Psillas et al. [9] observed reappearance of responses in half the participants at 6 months post-surgery, despite a complete loss immediately after surgery. These findings, along with the present results, highlight transient vestibular dysfunction following cochlear implantation, likely associated with insertion-related trauma.

To check the effect of insertion on vestibular functions, a few authors have tried to see VEMP responses intra-operatively and postoperatively with the device “on” and “off”. Jin et al. [12] did not find any response on electromyographic VEMP testing even after 6 weeks post surgery. They stated that the absence of a recordable wave postoperatively may be due to the mechanical block of the auditory signal by the CI, indicating a direct correlation of electrode insertion with vestibular function. In another study, Xu et al. explored the activation of the vestibular nerve due to cochlear implantation. Post-operatively, 11 of 12 children exhibited absent VEMP responses; however, with the device on, normal responses were restored in 3 children, with increased amplitude in only one child. They concluded that electrical stimulation delivered by the cochlear implant may partially activate the vestibulocochlear nerve, thereby compensating for vestibular dysfunction resulting from electrode insertion trauma [13].

In the current study, the latency of wave P1 and N1 did not differ significantly ($p > 0.05$) across pre-op, post-op, and follow-up periods for either ear. This aligns with Jin et al., who also reported the same in their follow-up study [12]. Conversely, Xu et al. found shortened N1 and P1 latency and reduced amplitude post-surgery. These changes in the implanted ear were attributed as direct injury of the saccular or utricular structures during electrode insertion. The authors further stipulated that minor trauma may still permit VEMP generation with minimal latency changes [13].

While latencies remained stable in our study, a significant difference ($p = 0.029$) was observed in P1–N1 amplitude between pre- and post-operative measurements in implanted ears. The initial amplitude reduction post-surgery, followed by partial recovery at follow-up, implies transient vestibular pathway disruption. Histopathological investigations of the implanted ear have also demonstrated structural alterations post-surgery. Fibrosis in the vestibule, saccule membrane distortion, new bone formation, and reactive neuromas were observed. The scala vestibuli damage due to osseous spiral lamina or basilar membrane involvement in the cochlear basal turn was observed in most (75%) patients, supporting the likelihood of peripheral injury [14]. Similarly, morphological findings – such as cochlear hydrops with collapsed saccule – have been reported by Handzel et al. in 59% of their patients, reinforcing the notion of vestibular involvement post-implantation [15].

Despite all participants undergoing implantation via the round window approach, 3 exhibited absent and 4 reduced post-operative VEMP responses. Though generally associated with less vestibular trauma than alternative approaches, the round window technique can still cause some damage. Comparative studies have confirmed that even this technique is not entirely devoid of vestibular risk [16].

Additionally, significant post-operative asymmetry in P1–N1 amplitude ($p < 0.001$) between ears was observed, implying alterations in unilateral vestibular functioning due to cochlear implantation. 4 participants showed complete (100%) asymmetry, while 2 showed greater than 33% asymmetry post-operatively. However, no participant demonstrated any change in vestibular symptoms, suggesting possible central compensation masking the peripheral level damage. Supporting evidence from animal models by Ris has demonstrated restoration of neural activity within one week after labyrinthectomy, indicating early central adaptation following peripheral vestibular injury [17].

Notably, the non-implanted ear exhibited a smaller but statistically significant postoperative amplitude reduction, which was not detected by overall ANOVA but was revealed by post hoc testing. This contralateral effect, persisting at borderline significance at follow-up, suggests that even the non-implanted ear should be tested post operatively for vestibular changes. The reason for changes in contralateral ear are unclear but could be due to either subclinical bilateral vestibular vulnerability in severe-profound SNHL or surgical factors, such as labyrinthine hydrops propagation, which became apparent after the stress of surgery [18,19].

In the present study, although latency values varied across the three time periods, these differences did not reach statistical significance, likely due to the small sample size, a limitation of our study. These differences between pre-op and post-op responses, and between recovery and follow-up, need to be explored further.

Conclusions

The present study aimed to assess vestibular function in children with severe to profound hearing loss before and after cochlear implantation to understand the extent of dysfunction and to investigate recovery patterns. The findings demonstrated that implantation can lead to changes in vestibular functions that are seen in reduced amplitudes or absent responses in the implanted ear. Restoration of function occurred throughout the recovery period up to follow-up. We believe that cVEMPs can be an effective objective tool for monitoring vestibular function before and after a CI.

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References

1. Wilson J. Deafness in developing countries: approaches to a global program of prevention. *Arch Otolaryngol*, 1985; 111: 2–9. <https://doi.org/10.1001/archotol.1985.00800030036002>
2. Szyfter W, Karlik M, Sekula A, Harris S, Gawęcki W. Current indications for cochlear implantation in adults and children. *Otolaryngol Pol*, 2019; 73(3): 1–5. <https://doi.org/10.5604/01.3001.0013.1000>
3. Wilson BS, Dorman MF. Cochlear implants: a remarkable past and a brilliant future. *Hear Res*, 2008; 242(1–2): 3–21. <https://doi.org/10.1016/j.heares.2008.06.005>
4. Cosetti M, Roland JT. Cochlear implant electrode insertion. *Oper Tech Otolaryngol Head Neck Surg*, 2010; 21(4): 223–32. <https://doi.org/10.1016/j.otot.2010.10.003>
5. Katsiari E, Balatsouras DG, Sengas J, Riga M, Korres GS, Xenelis J. Influence of cochlear implantation on the vestibular function. *Eur Arch Otorhinolaryngol*, 2013; 270: 489–95. <https://doi.org/10.1016/s00405-012-1950-6>
6. Zapala DA, Brey RH. Clinical experience with the vestibular evoked myogenic potential. *J Am Acad Audiol*, 2004; 15: 198–215. <https://doi.org/10.3766/jaaa.15.3.3>
7. Jin Y, Nakamura M, Shinjo Y, Kaga K. Vestibular-evoked myogenic potentials in cochlear implant children. *Acta Otolaryngol*, 2006; 126: 164–9. <https://doi.org/10.1080/00016480500312562>
8. Licameli G, Zhou G, Kenna MA. Disturbance of vestibular function attributable to cochlear implantation in children. *Laryngoscope* 2009;119: 740–5. <https://doi.org/10.1002/lary.20121>
9. Psillas G, Pavlidou A, Lefkidis N, Vital I, Markou K, Triaridis S, et al. Vestibular evoked myogenic potentials in children after cochlear implantation. *Auris Nasus Larynx*, 2014; 41: 432–5. <https://doi.org/10.1016/j.anl.2014.05.008>
10. Basta D, Todt I, Goepel F, Ernst A. Loss of saccular function after cochlear implantation: the diagnostic impact of intracochlear electrically elicited vestibular evoked myogenic potentials. *Audiol Neurotol*, 2008;13(3): 187–92. <https://doi.org/10.1159/000113509>
11. Shinjo Y, Jin Y, Kaga K. Assessment of vestibular function of infants and children with congenital and acquired deafness using the ice-water caloric test, rotational chair test and vestibular-evoked myogenic potential recording. *Acta Otolaryngol*, 2007; 127(7): 736–47. <https://doi.org/10.1080/00016480601002039>
12. Jin Y, Shinjo Y, Akamatsu Y, Ogata E, Nakamura M, Kianoush S, et al. Vestibular evoked myogenic potentials evoked by multichannel cochlear implant: influence of C levels. *Acta Otolaryngol*, 2008; 128(3): 284–90. <https://doi.org/10.1080/00016480701558872>
13. Xu X Da, Zhang XT, Zhang Q, Hu J, Chen YF, Xu M. Ocular and cervical vestibular-evoked myogenic potentials in children with cochlear implant. *Clin Neurophysiol*, 2015; 126(8): 1624–31. <https://doi.org/10.1016/j.clinph.2014.10.216>
14. Tien HC, Linthicum FH. Histopathologic changes in the vestibule after cochlear implantation. *Otolaryngol Head Neck Surg*, 2002; 127(4): 260–4. <https://doi.org/10.1067/mhn.2002.128555>
15. Handzel O, Burgess BJ, Nadol JB. Histopathology of the peripheral vestibular system after cochlear implantation in the human. *Otol Neurotol*, 2006; 27(1): 57–64. <https://doi.org/10.1097/01.MAO.0000188658.36327.8F>
16. Todt I, Basta D, Ernst A. Does the surgical approach in cochlear implantation influence the occurrence of postoperative vertigo? *Otolaryngol Head Neck Surg*, 2008; 138(1): 8–12. <https://doi.org/10.1016/j.otohns.2007.09.003>
17. Ris L, De Waele C, Serafin M, Vidal PP, Godaux E. Neuronal activity in the ipsilateral vestibular nucleus following unilateral labyrinthectomy in the alert guinea pig. *J Neurophysiol*, 1995; 74(5): 2087–99. <https://doi.org/10.1152/JN.1995.74.5.2087>
18. Reeder A, Canner J, Schwartz N. Risk factors for underlying bilateral vestibular weakness in cochlear implant candidates. *Otol Neurotol Open*, 2025; 5(1): e066. <https://doi.org/10.1097/ono.0000000000000066>
19. Ferster APOC, Cureoglu S, Keskin N, Paparella MM, Isildak H. Secondary Endolymphatic Hydrops. *Otol Neurotol*, 2017; 38(5): 774–79. <https://doi.org/10.1097/MAO.0000000000001377>

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