Published in final edited form as:

Otol Neurotol. 2016 October ; 37(9): 1399–1405. doi:10.1097/MAO.0000000000001196.

Adaptation and compensation of vestibular responses following superior canal dehiscence surgery

Georgios Mantokoudis, MD^{1,2}, Ali S. Saber Tehrani, MD², Aaron L Wong, PhD², Yuri Agrawal, MD³, Angela Wenzel, MD³, and John P. Carey, MD³

¹Department of Otorhinolaryngology, Head and Neck Surgery, Inselspital Bern, University of Bern, Switzerland ²Department of Neurology, Johns Hopkins University School of Medicine, Baltimore, USA ³Department of Otolaryngology-Head & Neck Surgery, Johns Hopkins University School of Medicine, Baltimore, USA

Abstract

Objective—To describe vestibulo-ocular function and compensatory mechanisms in the immediate post-operative period following superior canal dehiscence surgery.

Study Design—Prospective longitudinal study.

Setting—Tertiary medical center.

Patients—5 patients who underwent plugging of superior semicircular canal via middle cranial fossa approach.

Interventions—Bedside quantitative video head impulse testing (vHIT)

Main Outcome Measures—Dynamic measures of vestibulo-ocular reflex (VOR) function including VOR gain and compensatory saccades during vHIT.

Results—Mean VOR gain of the ipsilateral superior semicircular canal (SC) decreased from 0.71 ± 0.1 pre-operatively to 0.28 ± 0.07 on POD 2–4 ($p=0.0031$), consistent with plugging. There was also a significant immediate postoperative decrease of VOR gain for the other ipsilateral canals (posterior canal (PC) from gain 0.91 ± 0.33 down to 0.55 ± 0.14 , $p=0.040$; horizontal canal (HC) from 0.81 ± 0.08 down to 0.54 ± 0.19 , $p=0.038$). On postoperative days (PODs) 1–2, compensatory saccades after testing the plugged SC occurred exclusively after the head stopped moving (overt) with latency of $186.2 \text{ ms} \pm 19.9 \text{ ms}$. By POD 7 saccade latency decreased to $141.0 \pm 17.5 \text{ ms}$ ($p=0.032$), and saccades were occurring during the vertical head rotation (covert saccades). Follow-up >40 days was consistent with prior findings in that mean SC gain remained low. HC gain recovered fully, but some cases did not have full recovery of PC gain.

Conclusions—When the SC is plugged surgically, early quantitative vHIT demonstrates significantly reduced VOR gain for all of the ipsilateral canals. Possible mechanisms include labyrinthine inflammation and loss of perilymph at the time of surgery. Full recovery is typical for

the horizontal canal but not always for the PC. Evidence of central compensation occurred by the elicitation of compensatory saccades and by reducing their latencies within the first week after surgery.

Introduction

Superior canal dehiscence syndrome (SCDS) was first described by Minor et al. in 1998 (1) and consists of sound or pressure induced vertigo combined with auditory symptoms such as autophony, hyperacusis and tinnitus. The underlying cause is a bony dehiscence of the superior semicircular canal that allows a transmission of pressure between the labyrinth and intracranial space. The superior canal dehiscence (SCD) can be visualized by computed tomography (CT). In addition, auditory deficits such as an increased air-bone gap at low frequencies (2) or an increased ocular vestibular evoked potential (oVEMP) (3) may confirm a diagnosis of SCDS. SCDS is a treatable condition with surgical repair (4–7). Repair of SCD results in resolution of chief complaints with a normalization of auditory symptoms (8), closure of low frequency air-bone gaps (9) and reduction of dizziness handicap (10). In addition, electrophysiological test results such as cervical or ocular VEMPs show a recovery after surgery (11). Although the long term vestibular function seems to be preserved (12), a transitory vestibular hypofunction in the initial 6 postoperative weeks was reported for the horizontal semicircular canal (13); however, no quantitative dynamic vestibular testing was available at the bedside in the first week after surgery, and vertical semicircular canals were not investigated. Quantitative testing of dynamic vestibular responses in all canal planes was only performed in the laboratory at time points distant from the surgery by using more invasive tests such as magnetic search coils (12), which are currently the gold standard for eye movement measurements. New portable video-oculography devices enable quantitative bedside recordings and serial measures of eye and head movements under real-world conditions such as emergency departments (14) or inpatient wards (15).

We sought therefore to study vestibular responses at the bedside immediately after plugging a superior canal in patients with SCDS and to quantify early vestibular deficits in all three canal planes. Since patients with an extended bony dehiscence might have a prolonged vestibular recovery period (16) and suffer from an increased fall risk (17), we intended to quantify early adaptation and compensatory strategies which might be useful for future rehabilitation strategies.

Methods

We recruited patients with diagnosed SCDS who were scheduled to undergo plugging and resurfacing of the SCD via middle cranial fossa approach at a tertiary academic medical center. All patients had a normal vision or a normal corrected vision. The diagnosis of SCDS was based on clinical signs, audiometry, oVEMP responses and CT findings; each case in this series had surgical confirmation. We enrolled a convenience sample of five patients given that this was largely a descriptive study in a low prevalence disease. All patients provided written informed consent as approved by the Johns Hopkins University Institutional Review Board.

We used a portable video-oculography goggle system (EyeSeeCam, Munich, Germany) for quantitative video head impulse test (vHIT) recording at the bedside. A high speed infra-red camera and a built in accelerometer measured eye and head movements at a sampling rate of 250 Hz. We quantitatively assessed the vestibulo-ocular reflex (VOR) during horizontal and vertical HIT pre-operatively, at the bedside during the first seven post-operative days (POD), and also at post-operative days 40, 60 and 120.

An experienced neuro-otologist performed passive horizontal and vertical HITs at the bedside with the patient in a seated position in room light. The patient kept their head stationary during a brief goggle calibration sequence and fixated a far target (>2m) during impulse testing. The VOR was tested in the earth-horizontal plane and the planes of the vertical semicircular canals: For testing the two horizontal semicircular canals, the head was rapidly turned either to the right from center or to the left in an unpredictable manner with respect to both direction and timing (onset of head rotation). The left anterior (superior) and right posterior canal (LARP) plane was tested from a starting position with the head turned 45 degrees to the right. From here, head impulses were delivered in a downward direction in the LARP plane to excite the left SC or upward to excite the right PC. For testing the right anterior/superior and left posterior canal (RALP) plane, the VOR was tested from a starting position with the head turned 45 degrees to the left, and downward impulses in the RALP plane excited the right SC while upward impulses excited the left PC. Target head impulse velocity was 100–200 deg/s with amplitude between 5–15 deg in the plane being tested.

VOR gain was calculated as the ratio of eye to head velocity at 60 ms after initiation of head rotation. The mean latency of the first compensatory saccade with a minimal eye velocity of 50 deg/s and an acceleration greater than 2000 deg/s/s was determined using custom Matlab software (version R2012a, MathWorks, Natick, MA). The beginning of a head impulse was defined as the first time point where head velocity was greater than 2% of peak head velocity for that trial. The end of a head impulse was defined as the time when head velocity reached zero for the first time after the beginning. A compensatory saccade that occurred during the head movement was classified as “covert” (18,19). Saccades appearing after the end of the head impulse were considered “overt”.

Statistics

Paired, one-sided t-tests were performed to analyze the differences in VOR gains between day 0 (preoperative) and the first test after surgery (done on or between POD 2 and 4) and differences in mean saccade latencies between POD 2–4 and POD 5–7. We took aggregate time points for comparison in order to obtain complete data sets from all patients on those days. A P-value of <0.05 was considered significant. Descriptive statistics were used to measure VOR gain and saccade latencies. Statistical analysis was performed using InStat software version 3.05 (GraphPad Software Inc., La Jolla, CA, USA).

Results

We enrolled two women and three men for these serial exams before and after SCD surgery. The mean age was 55.6 y (range 44–71 y, SD ± 11.2 y). Table 1 gives an overview of clinical characteristics.

During the post-operative period, not all patients tolerated head impulse tests at each scheduled post-operative day due to motion intolerance, head or neck pain, or nausea. All patients completed testing of all 6 semicircular canals at least at three time points. This was designed primarily as a study of vestibular function immediately after SCD surgery, and longer-term follow-up was not available in all patients, some of whom returned to residences located a great distance from our center one week after surgery. However, follow-up visits at POD 40 were made by three patients.

Figure 1 shows an example of traces from a series of vertical head impulses performed pre-operatively, on POD 2 and POD 7 in one patient. Normative VOR gains measured with vHIIT in healthy subjects are generally close to 1.0 since the ratio of head and eye velocity is close to unity. However, VOR gains for vertical canals are significantly lower compared to horizontal canals and show a greater variability (20).

Figure 2 illustrates the aggregate VOR gain results over time for each patient and each semicircular canal separately, with the spline curve fit to the data. The mean VOR gain of the plugged SC decreased from 0.71 ± 0.10 pre-operatively to 0.21 ± 0.07 on POD 2–4 ($p=0.0031$) and remained low with a slight but not significant recovery over time. Additionally, there was a significant immediate decrease of the mean VOR gain for the other ipsilateral canals: PC from mean gain 0.91 ± 0.33 down to 0.55 ± 0.14 , $p=0.040$; HC from 0.81 ± 0.08 down to 0.54 ± 0.19 , $p=0.038$. A tendency for gain recovery over time could be seen, but gain levels still did not reach a statistically significant difference during the week after surgery (POD 4–6). For the HC, 4 out of 5 patients showed a decreased ipsilesional VOR gain within the first 4 postoperative days, but one patient had an increased gain after surgery. After this, three had a HC gain increase with full recovery of the horizontal VOR after POD 7, but one patient did not fully recover by that time. The ipsilateral PC was affected as well in 4 out of 5 patients; 1 patient had a preoperatively deficient ipsilateral PC VOR, which actually improved after surgery. The ipsilateral PC gain decreased and did not recover during the week after surgery, nor did it fully recover in the 2 patients with longer-term follow-up. Overall, the mean ipsilateral HC gain went back to baseline by POD 40, while there was only a partial recovery of the mean gain for the ipsilateral PC based on the limited data for follow-up beyond 1 week.

All of the contralateral canal gains had a similar trend showing a slight VOR gain decrease immediately after surgery and a recovery over time. However, there were no statistically significant changes in VOR gain for any of the canals on the contralateral side.

Initially on POD 1–2, compensatory saccades after head impulses expected to excite the plugged SC occurred exclusively after the head stopped moving (i.e., overt saccades) with a mean latency of $186.2 \text{ ms} \pm 19 \text{ ms}$. By POD 7 saccade latency decreased to $141.0 \pm 17.5 \text{ ms}$ ($p=0.032$, figure 3), and saccades were occurring during the head rotation (covert saccades, figures 1 and 4).

Discussion

This study provides insights into changes in labyrinthine function that occur immediately following surgery to plug superior canal dehiscence. It also sheds light on central nervous system responses to those changes. Whereas previous bedside work in the immediate postoperative period was necessarily qualitative in nature, the work in the present small series takes advantage of recent advances in lightweight, portable video goggles for quantitative HIT testing in the inpatient unit following surgery. Serial measurements of the VOR demonstrated that there was a generalized ipsilateral VOR gain deficit of all three ipsilateral semicircular canals in 5/5 patients. VOR gains for head impulses exciting the contralateral canals were reduced as well, but not in all individuals and not to the same degree. VOR gain fully recovered over time for excitation of all canals on the contralateral side and of the horizontal canal on the ipsilateral side. As expected, gain did not return to normal for excitation of the plugged SC. In addition, some dysfunction persisted in the VOR for excitation of the ipsilateral PC in 4/5 patients. CNS compensation for reduced VOR performance was manifested in two mechanisms: elicitation of compensatory saccades to improve gaze stability and reduction of the latencies of these compensatory saccades over the initial postoperative weeks.

An initial early decline of VOR gain was observed bilaterally in all semicircular canals but more pronounced and statistically significant for excitation of the ipsilateral canals. The ipsilateral labyrinth might be affected through loss of perilymph or damage to the membranous labyrinth itself during the procedure. The subsequent recovery of VOR for excitation of the ipsilateral canals not plugged would favor a reversible injury. This could include the loss of a subcritical volume of perilymph – a volume small enough to cause sagging of the membranous labyrinth but not complete atelectasis – or mild labyrinthine inflammation. The labyrinth on the intact side might be affected by three possible mechanisms: 1) Reduced neural contribution to the VOR from the operated side through commissural pathways, 2) Central adaptation by down regulation (21) and 3) Suppressive effects of anesthetic and postoperative medications on the vestibular system (22).

Thirty-seven percent of patients reported by Agrawal et al. (13) had an abnormal qualitative horizontal head thrust sign immediately after SCD surgery indicating a transitory deficit of the ipsilateral horizontal semicircular canal. These findings are in line with our study data except that deficits for excitation of the contralateral side were not detected in that qualitative study. The mild deficits for the contralateral canals may not be extreme enough to trigger compensatory refixation saccades, which are necessary for a positive qualitative HIT. However, quantitative horizontal HIT testing was performed in those patients 6 to 29 weeks after surgery, and lasting ipsilateral horizontal canal deficits were only seen in 11% of patients. We previously examined vestibular function in the horizontal and vertical planes under laboratory conditions using the gold-standard of magnetic search coils in patients 46–206 days after surgery (12). No significant deficits in gain were seen on average for head impulses exciting either HC, which indicates that complete long term recovery is the norm for horizontal VOR gains. That study also showed a mild long-term deficit for both PCs after surgery. This finding is consistent with our present data, which showed reduced function of the ipsilateral PC in the first 7 days after surgery. A partial recovery over time was seen for

some ipsilateral PC gains. However, incomplete follow-up data after POD 7, low sample size and high sample variability make it difficult to draw final conclusions about long-term deficits. The contralateral PC showed a trend for a mild decrease in gain but was statistically not significant because of two subjects who either had an unaffected VOR (subject #1) or even an increased VOR gain over time (subject #5). Such a decrease in gain for head impulses expected to excite the contralateral PC is not unexpected given the loss of the inhibitory contribution from the plugged SC during such impulses.

Gaze stabilization can be maintained by visually triggered reflexes such as smooth-pursuit or optokinetic tracking or, at higher frequencies, by a vestibular driven reflex, the VOR (23). Compensatory saccades occur in patients with deficient VOR and are meant to bring the eyes back to their fixation target. Such compensatory saccades are believed to be driven by the visual system and may occur during a rapid head movement (so called *covert* saccades) or after the completion of the head movement (*overt* saccades) (24). Covert or “hidden” saccades during a HIT may be invisible for the examiner, whereas overt saccades can be easily seen after the end of a fast head movement. Clinicians may use different HIT techniques to elicit overt saccades by performing unpredictable HITs in terms of direction and head excursion amplitude (25); however, a video-oculography device or a magnetic search coil exam reliably demonstrates covert saccades (26,27). The proportion of covert vs. overt saccades is an indirect function of saccade latency and HIT duration. Shorter mean saccade latencies (47–66 ms) were reported for covert saccades and prolonged latencies of 144–189 ms for overt saccades in patients with vestibular deficits (18). Predictable head rotations induce more covert saccades than unpredictable head rotations after vestibular nerve deafferentation (28). It has been suggested that a shift from overt to covert saccades indicates improving compensation (24). In fact, a previous study performed by our study group demonstrated a shortening of saccade latencies by 60 ms within the first week after deafferentation following acoustic neuroma resection (15). The reduction of saccade latency resulted in a higher proportion of covert saccades on the 4th postoperative day; however, only the HCs were tested. Data in the present study confirmed that the plugged SC showed exactly the same compensatory pattern over time: Even though only the end organ was affected and the vestibular nerve remained intact, saccade latencies reduced significantly by an average of 44 ms after 4–7 postoperative days (figure 3) and became more often covert after POD 4 (Figure 4). These observations suggest that central compensation appears after any deficient of peripheral vestibular input independent of how the lesion affects the spontaneous activities of vestibular afferents.

Strength and limitations

To our knowledge, this is the first prospective and quantitative study of the VOR in the immediate post-operative period following SCD repair showing vestibular function of all six semicircular canals separately. Moreover, it is the first study showing early VOR adaptation and compensation with refixation saccades coming at shorter latencies as time goes on from the point immediately after plugging a semicircular canal in humans.

Performing head impulses in the first days after skull base surgery was difficult because of postoperative head and neck pain and head motion intolerance. We could not obtain a

complete data set at each given postoperative day because of these difficulties. In addition, repeated vertical head impulses showed a more variable VOR gain and were susceptible to artifacts due to goggle slippage. Goggle slippage was also more likely given the inability to tightly fasten the head strap to the healing scalp so soon after surgery. We therefore screened all data for measurement errors, and traces suggestive of goggle slippage and other artifacts were excluded.

Clinical Implications

These data might have implications for surgeons treating patients with SCDS. First, it is important to note the consistent finding that global vestibular hypofunction appears to be common immediately after plugging the superior canal for SCDS. Much of this hypofunction appears to be reversible over time except in the plugged SC and often in the ipsilateral PC. An inflammation of the labyrinth might be responsible for some of the ipsilateral global hypofunction. For this reason, we typically treat these patients with high dose corticosteroids (dexamethasone 6 mg every 6 hours for 24–48 hours) followed by a taper over 5–10 days depending on whether the audiogram is normal or shows additional cochlear sensory impairment. Vestibular hypofunction for excitation of the contralateral canals would seem to be only explainable as a central response. For this reason, we begin intensive postoperative vestibular rehabilitation and continue it for several weeks after surgery. The shortening of refixation saccade latency would suggest that the central plasticity for compensation is excellent in this setting.

Conclusions

VOR gain measured immediately after SCD surgery decreased for excitation of all ipsilateral semicircular canals including the posterior and horizontal semicircular canals. The ipsilateral HC gain went back to baseline by POD 40, while there was only a partial recovery of the ipsilateral PC in 3/5 patients in this series. The VOR for head impulses exciting the contralateral labyrinth was also temporarily reduced, probably through central mechanisms. Intensive vestibular rehabilitation and postoperative treatment with corticosteroids should be considered. Compensation occurred by the elicitation of compensatory saccades and by the reduction in their latencies within the first week after surgery.

Acknowledgments

This study was supported by the Swiss National Science Foundation PBBEP2 136573 (GM). We would like to thank Li Xie, ScM, Nemours Biomedical Research, Alfred I. du Pont Hospital for Children, Wilmington, USA, for her valuable suggestions.

References

1. Minor LB, Solomon D, Zinreich JS, et al. Sound- and/or pressure-induced vertigo due to bone dehiscence of the superior semicircular canal. *Arch Otolaryngol Head Neck Surg.* 1998; 124:249–58. [PubMed: 9525507]
2. Minor LB, Carey JP, Cremer PD, et al. Dehiscence of bone overlying the superior canal as a cause of apparent conductive hearing loss. *Otol Neurotol.* 2003; 24:270–8. [PubMed: 12621343]
3. Janky KL, Nguyen KD, Welgampola M, et al. Air-conducted oVEMPs provide the best separation between intact and superior canal dehiscence labyrinths. *Otology & neurotology: official publication*

of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology. 2013; 34:127–34.

4. Amoodi HA, Makki FM, McNeil M, et al. Transmastoid resurfacing of superior semicircular canal dehiscence. *The Laryngoscope*. 2011; 121:1117–23. [PubMed: 21520134]
5. Mueller SA, Vibert D, Haeusler R, et al. Surgical capping of superior semicircular canal dehiscence. *European archives of oto-rhino-laryngology: official journal of the European Federation of Oto-Rhino-Laryngological Societies*. 2014; 271:1369–74.
6. Agrawal SK, Parnes LS. Human experience with canal plugging. *Ann NY Acad Sci*. 2001; 942:300–5. [PubMed: 11710471]
7. Minor LB. Clinical manifestations of superior semicircular canal dehiscence. *Laryngoscope*. 2005; 115:1717–27. [PubMed: 16222184]
8. Crane BT, Lin FR, Minor LB, et al. Improvement in autophony symptoms after superior canal dehiscence repair. *Otology & neurotology: official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology*. 2010; 31:140–6.
9. Limb CJ, Carey JP, Srireddy S, et al. Auditory function in patients with surgically treated superior semicircular canal dehiscence. *Otology & neurotology: official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology*. 2006; 27:969–80.
10. Crane BT, Minor LB, Carey JP. Superior canal dehiscence plugging reduces dizziness handicap. *The Laryngoscope*. 2008; 118:1809–13. [PubMed: 18622314]
11. Welgampola MS, Myrie OA, Minor LB, et al. Vestibular-evoked myogenic potential thresholds normalize on plugging superior canal dehiscence. *Neurology*. 2008; 70:464–72. [PubMed: 18250291]
12. Carey JP, Migliaccio AA, Minor LB. Semicircular canal function before and after surgery for superior canal dehiscence. *Otology & neurotology: official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology*. 2007; 28:356–64.
13. Agrawal Y, Migliaccio AA, Minor LB, et al. Vestibular hypofunction in the initial postoperative period after surgical treatment of superior semicircular canal dehiscence. *Otology & neurotology: official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology*. 2009; 30:502–6.
14. Newman-Toker DE, Tehrani AS, Mantokoudis G, et al. Quantitative Video-Oculography to Help Diagnose Stroke in Acute Vertigo and Dizziness: Toward an ECG for the Eyes. *Stroke*. 2013
15. Mantokoudis G, Schubert MC, Tehrani AS, et al. Early adaptation and compensation of clinical vestibular responses after unilateral vestibular deafferentation surgery. *Otology & neurotology: official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology*. 2014; 35:148–54.
16. Niesten ME, McKenna MJ, Grolman W, et al. Clinical factors associated with prolonged recovery after superior canal dehiscence surgery. *Otology & neurotology: official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology*. 2012; 33:824–31.
17. Janky KL, Zuniga MG, Carey JP, et al. Balance dysfunction and recovery after surgery for superior canal dehiscence syndrome. *Arch Otolaryngol Head Neck Surg*. 2012; 138:723–30. [PubMed: 22801722]
18. Weber KP, Aw ST, Todd MJ, et al. Head impulse test in unilateral vestibular loss: vestibulo-ocular reflex and catch-up saccades. *Neurology*. 2008; 70:454–63. [PubMed: 18250290]
19. Schubert MC, Hall CD, Das V, et al. Oculomotor strategies and their effect on reducing gaze position error. *Otology & neurotology: official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology*. 2010; 31:228–31.
20. McGarvie LA, MacDougall HG, Halmagyi GM, et al. The Video Head Impulse Test (vHIT) of Semicircular Canal Function – Age-Dependent Normative Values of VOR Gain in Healthy Subjects. *Frontiers in neurology*. 2015; 6:154. [PubMed: 26217301]

21. McCabe B, Ryu J, Sekitani T. Further experiments on vestibular compensation. *The Laryngoscope*. 1972; 82:381–96. [PubMed: 4336542]
22. Weerts AP, De Meyer G, Pauwels G, et al. Pharmaceutical countermeasures have opposite effects on the utricles and semicircular canals in man. *Audiology & neuro-otology*. 2012; 17:235–42. [PubMed: 22517315]
23. Leigh, RJ.; Zee, DS. The vestibular-optokinetic system. In: Leigh, RJ.; Zee, DS., editors. *The Neurology of Eye Movements*. New York: Oxford University Press; 2006. p. 20-107.
24. Macdougall HG, Curthoys IS. Plasticity during Vestibular Compensation: The Role of Saccades. *Frontiers in neurology*. 2012; 3:21. [PubMed: 22403569]
25. Tjernstrom F, Nystrom A, Magnusson M. How to uncover the covert saccade during the head impulse test. *Otology & neurotology: official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology*. 2012; 33:1583–5.
26. Blodow A, Pannasch S, Walther LE. Detection of isolated covert saccades with the video head impulse test in peripheral vestibular disorders. *Auris, nasus, larynx*. 2012
27. Weber KP, MacDougall HG, Halmagyi GM, et al. Impulsive testing of semicircular-canal function using video-oculography. *Ann NY Acad Sci*. 2009; 1164:486–91. [PubMed: 19645955]
28. Mantokoudis G, Agrawal Y, Newman-Toker DE, et al. Compensatory saccades benefit from prediction during head impulse testing in early recovery from vestibular deafferentation. *European archives of oto-rhino-laryngology: official journal of the European Federation of Oto-Rhino-Laryngological Societies*. 2015

Figure 1.

Vertical head impulses in the LARP plane are shown pre-operatively (A) and on POD 2 (B) and 7 (C) for patient #6, who had surgery for left SCD. Red traces indicate head velocity, blue traces eye velocity. Red chevrons mark the time of the first appearance of corrective saccades. Note that the latency of compensatory saccades decline during the first week after surgery.

Figure 2.

Aggregate VOR gain results (mean gain $\pm$ SD) are shown for each semicircular canal and each patient individually. A trendline summarizes the VOR gain values across all data points.

Figure 3.

A decline of absolute mean saccade latencies for the corrective saccades in ms (first saccade appearance) $\pm$ SD is illustrated for the ipsilateral SC of each patient over time. An average trendline was calculated after plotting best fit lines for each individual's data.

Figure 4.

The proportion of overt saccades (%) in relation to all corrective saccades appearing in all vertical head impulses (ipsilateral SC) at a specific time point are shown for each individual. An overt saccade is a compensatory saccade that occurs after the head stops moving. The trendline summarizes all best fit lines across each individual and shows a diminishing proportion of overt saccades over time.

Table 1

Patient characteristics

ID	Age	Gender	Hearing	oVEMP amplitude	Dizziness	Autophony	Sensitivity to sound	Affected side	Operated side	Dehiscence Length × Width [mm]	Comments
1	50	F	air bone gap at 250Hz left	high left ear	yes	yes	yes	bilateral	left	2.0 × 0.5mm	history of migraine
2	44	M	mild sensorineural hearing loss bilaterally	high left ear	yes	no	yes	left	left	5.0 × 0.7 mm	history of migraine
3	46	M	mild right conductive hearing loss	high bilaterally	yes	yes	yes	right	right	3.0 × 1.0mm	NA
4	67	M	moderately-severe right sensorineural hearing loss	NA	no	yes	yes	left	left	0.2 × 0.2mm	NA
5	71	F	air bone gap at 250Hz bilaterally	high right ear	Yes	no	no	right	right	0.1 × 0.1 mm	history of migraine