Skull Vibration-Induced Nystagmus in Superior Semicircular Canal Dehiscence

Subjects: Otorhinolaryngology

Contributor: Georges Dumas, Ian Curthoys, Andrea Castellucci, Laurent Dumas, Laetitia Peultier-Celli, Enrico Armato, Pasquale Malara, Philippe Perrin, Sébastien Schmerber

The third window syndrome, often associated with the Tullio phenomenon, is most often observed in patients with a superior semicircular-canal dehiscence (SCD) but is not specific to this pathology. Clinical and vestibular tests suggestive of this pathology are not always concomitantly observed and have been complemented by the skull-vibration-induced nystagmus test, which constitutes a bone-conducted Tullio phenomenon (BCTP).

Keywords: vertigo ; Minor syndrome ; Tullio ; Hennebert sign ; skull-vibration-induced nystagmus

1. Introduction

Patients with superior semicircular canal dehiscence (SCD) often have atypical presentations but usually show audiovestibular symptoms consistent with mild conductive hearing loss associated with an unusual “too good” bone conduction (negative bone conduction thresholds for low frequencies on pure tone audiometry), tinnitus, hyperacusis, autophony, and often atypical vestibular symptoms such as positional vertigo, vertigo induced by noise, pressure-induced vertigo, or Menière-like attacks of vertigo [1][2][3][4]. There is sensitivity to external or middle-ear pressure variations (Hennebert sign, Valsalva test). Eye movements and sway or imbalance induced by low-frequency air-conducted (AC) sounds at high intensity have been described by Tullio in animals [5][6] and by Parker (250–2000 Hz at 100–120 dB intensity) in humans [7]. Similar eye movements or nystagmus associated with dizziness have been reported after cranial bone vibrations applied with the skull-vibration-induced nystagmus test (SVINT) [8][9][10].

Vestibular-evoked myogenic potentials (VEMPs), including ocular VEMPs (oVEMPs) and cervical VEMPs (cVEMPs), are considered as the gold-standard tests in SCD, showing a hypersensitivity of the utricle and sacculus, with large amplitudes and lower thresholds in response to air- or bone-conducted sound stimulations [11][12][13][14][15]. Unlike the responses of healthy subjects, there are responses to very-high-frequency stimulation (4000 Hz) in this pathology [13].

Nystagmus induced by vibrations of the cranium (VIN) in patients exhibiting sound-induced vertigo was reported for the first time at the XXth Barany Society in 1998 [16] and first recorded in 2005 [8]. Since then, many authors [9][10][17][18][19][20][21][22][23] have published different series about bone-conducted (BC) skull-vibration-induced nystagmus (SVIN) or ocular movements (vibration-induced vestibulo-ocular reflex (ViVOR)) in patients with SCD. Some results in these publications differ because of different protocols and different vibration locations or frequencies used for the stimulus [24].

The different clinical data in the most recent publications are in accordance with predictions in recent physiological works [25][26][27][28] SVIN when interpreted in the light of these recent works shows a new insight and confirms its value in the armamentarium of first-line vestibular tests in SCD diagnosis. However, third mobile window syndrome most often described in SCD is not specific to this pathology [29] and SVIN is not specific to SCD diagnosis; it may be observed in other third mobile window syndromes, as reported by White [30]. This author observed a strong (45°/s) ipsilateral beating nystagmus induced by vibration in one of her five patients with enlarged vestibular aqueduct (VAD) syndrome.

Cremer, Minor, and Zee, in their presentation at the XXth Barany Society in 1998, reported “a nystagmus produced by mastoid vibration in patients with a Tullio phenomenon” [16]. They suggested a link between these two phenomena. The first 2D recording of an SVIN obtained at 100 Hz in a patient with unilateral SCD was reported by Dumas et al. as early as 2005 [8]; in this patient, the recorded nystagmus showed a vertical component and an associated horizontal component ipsilaterally beating. Since then, other authors have published observations and recordings of nystagmus provoked by BC in SCD [9][10][17][18][19][20][21][22][23]. These results, variability of nystagmus direction depending on stimulus location and frequency, and an after-nystagmus, not usually observed in uVL patients, are discussed below. According to the literature, the sensitivity range of SVIN in SCD is between 55% [23] and 100% [19].

2. Optimal Location of Stimulation in SCD

2.1. BC Stimulation Locations

Different BC stimulation locations have been described: mastoid [9][18][19], occipital [10], frontal area (Fz), and vertex (Vx or Cz) [9][19][21] regions. The only statistical analysis comparing SVIN SPV for vertex vs. mastoid locations showed that the vertex was more efficient than the mastoid location at 100 and 300 Hz but not at other frequencies [9]. Suboccipital stimulations have also been described and suggested to be very efficient to provoke a nystagmus by White et al. [10] but without statistical data. This last location is otherwise in close proximity with the cervical muscular region.

Vx stimulations are more efficient to reveal an SVIN in SCD patients than in uVL patients [9]: in uVL, mastoid stimulations elicited a contralateral horizontal beating SVIN in 100% of cases, and on Vx stimulations, a positive horizontal SVIN in 75%; conversely, in uSCD, the horizontal component was obtained in 57% of cases on mastoid stimulations and in 92% of cases on Vx stimulations. For the torsional component, in uVL this was 75% on mastoids and 44% on Vx; conversely, in uSCD, it was 50% and 78%, respectively. This result is independent of the order of stimulation location in uVL. The stimulation location in Vx tended to be more efficient when Vx was stimulated after mastoids in uSCD [24].

2.2. Possible Explanations for Variable Results in SCD Patients

Why are vertex stimulations usually more efficient than mastoid stimulations in SCD?

In patients with SCD, BC stimulation is transmitted to inner ear structures by compression waves through the temporal bone as well as by compression waves through the brain and cerebrospinal fluid (CSF) [25] via dehiscence. Conversely, uVL patients have no middle fossa fistula and show mastoid stimulations that are significantly more efficient than vertex stimulations [9][17]. The bone conduction contribution due to cerebrospinal (CSF) transmission of vibrations under normal conditions is negligible, as mentioned by Stenfeld et al. [31], but may become dominant in cases of middle fossa fistula represented by SCD. In this condition, vertex stimulation may be more efficient than that in uVL patients with an encased labyrinth, where, conversely, SVIN responses on both mastoids are reproducible and beat toward the same direction, corresponding to the stimulation of type I receptor hair cells on the intact side.

The role of soft tissue or CSF has been suggested to contribute to BC by Freeman et al. [32]. A vibration transmitted via the middle cerebral fossa fistula in SCD has already been described by the Cleveland team, in 2007 [10], and suggested by Dumas et al. [9][17].

Stimulus location modifies responses in SCD after SVINT and BC VEMP.

In patients with SCD stimulated at the vertex, the VIN direction is most often ipsilateral to the lesion, but may change (in 50% of cases) depending on the side of the stimulated mastoid [13][24]. Vx stimulations are more reliable and are used as referentials in clinical practice to specify the VIN direction.

For bone-conducted (BC) oVEMPs, Manzari et al. [13][33] demonstrated that stimulations at 500 Hz were more sensitive (87%) than the AC Tullio phenomenon observed in 57% and the Hennebert sign observed in 30% of their 24 cases. Moreover, for oVEMPs, Fz stimulation close to a frontal location induced larger N10 on the contralateral eye than Cz or top-of-cranium stimulations. These authors inferred the change in direction of the wave stimulating the otoliths following the stimulus location and suggested that an Fz stimulus location generated a compressional wave with a rostrocaudal direction, which was more efficient for stimulating the hair bundle at the level of the utricular macula than the Cz location, which created a wave perpendicular to the macula. Thus, similarly for SVIN, the force vector direction generated by the compressional wave may influence the activation of cupula hair cells following the stimulus location.

3. Stimulus Optimal Frequency—Frequency Spectrum Sensitivity for SVIN in SCD

In uVL patients, the optimal frequency is 100 Hz (no responses observed at 500 Hz) [9]; however, in SCD patients, frequency sensitivity is extended toward higher frequencies and shows more favorable responses at around 400 Hz. Good responses may be observed between 60 and 800 Hz [9]. This extension toward very high frequencies corresponds to the observations in animals by Dlugaiczyk et al. [34] and to the descriptions with oVEMPs (2000–4000 Hz) by Manzari et al. [13]. In some individual patients, the SVIN direction may change depending on the stimulus frequency (e.g., 30 vs. 100 Hz or 100 Hz vs. 300 Hz) [24]. These optimal responses observed for SVIN at around 400–500 Hz [9] in patients with SCD correspond to BC facilitation with lower impedance, as described for audiological explorations by Songer et al. [25].

4. Characteristics of the Nystagmus Obtained in SCD (Direction, Components)

SVIN 3D recordings in patients with SCD show most often (in 56% of positive cases) three components [9][17]. This suggests a more global stimulation than the sole superior SCC stimulation. The horizontal component suggests the contribution of either otolith structures (utricle) or horizontal SCC.

White et al. (2007) [10] suggested that only the superior SCC was stimulated by vibrations that induced a VIN with either a torsional (most often ipsilaterally beating) or vertical component (most often down-beating). This has also been suggested by Aw et al. with the vibration-induced vestibulo-ocular reflex (ViVOR) [18] considering the eye slow-phase movement. These authors used scleral search coils and short-duration stimulation at 500 Hz with target fixation, which can explain the negligible horizontal response by inhibition.

A horizontal component associated with a vertical component was recorded with vision denied for the first time by Dumas et al. in 2005 [8] and was later reported by Park et al., 2014 [20], Batuecas et al., 2022 [21], and Koo et al., 2010 [35]. Dumas et al. [9] also observed a torsional ipsilateral beating nystagmus and a nystagmus with a vertical component (down-beating nystagmus in 40% and up-beating in 60% of cases), which they attributed to a probable response of the superior SCC. The variable vertical direction (up- or down-beating nystagmus) and horizontal component could not be explained by a single stimulation of the superior SCC. The vertical nystagmus component direction variability could be explained by the current concept of the Tullio phenomenon related to the flow of the endolymph and nonlinear fluid pumping [26][27][28], which will be detailed below. For the horizontal component, stimulation of structures other than the superior SCC was suggested accordingly to what is known from physiology [36][37].

The implication of the utricle, initially described by Tullio as a co-stimulation [38][39][40][41], may explain the horizontal component of SVIN and was clinically supported by Halmagyi et al. [39] and Dumas et al. [9]. Park also suggested a contribution and co-stimulation of the lateral SCC (2014) [21] to explain the important horizontal slow phase of the ocular movement (ViVOR) component during mastoid stimulations. A possible contribution of the horizontal canal through the spread of regional mobilization of endolymphatic fluid has also been suggested in animals by Carey et al [42]. The implication of an asymmetric concomitant stimulation of otolith structures in SCD patients is otherwise corroborated in the clinical setting by abnormal otolithic test results (ipsilateral hyperexcitability of cVEMPs and oVEMPs) not only with sounds (air-conducted stimulations) but also after bone-conducted vibration stimulations [14][15][39][43].

The SVIN horizontal component frequently observed in SCD cannot be attributed to an unlikely concomitant horizontal SCC pathology. Dumas et al. [9] observed in their series in SCD patients a caloric test seldom modified (3 cases of 23 SCD) and an HVHIT gain asymmetry in 1/22 cases. However, this interaction remains possible in some cases. An anterior SCC VHIT gain asymmetry was more frequently observed in 5/22 patients.

Why may the VIN vertical direction vary in different patients stimulated with identical stimuli?

One possible explanation for the SVIN vertical component direction, which differs between patients (up- or down-beating nystagmus), could be that patients with larger dehiscence (>6 mm) may have a superior SCC “auto-plugged” by the overlying cerebromeningeal tissue and thus less stimulated (this corresponds to the frequent superior canal VHIT gain diminution observed in these patients in the literature [44][45]). In this condition, BCV stimulation of the inner ear structures may induce an up-beating nystagmus because the ipsilateral posterior SCC response is less or possibly no longer canceled by the superior SCC response.

How should we interpret the results in bilateral SCD?

In bSCD, vertex stimulations usually induce in 75% of positive cases a nystagmus beating toward the more excitable side with the larger hearing loss or audiological symptoms, which usually corresponds to the side of the larger lesion [9][24].

There is otherwise individual patient anatomic variability in SCD, and recent papers [46] showed that 18% had a concomitant additional ipsilateral dehiscence. Some of these dehiscences may have a significant impact on SVIN components.

To summarize, SVINT using BC vibration appears to stimulate all vestibular sensory structures. SVIN usually shows three components corresponding to a global and concomitant stimulation of different inner ear structures (torsional and vertical components corresponding probably to superior SCC stimulation and a horizontal component possibly related to the lateral SCC stimulation or to the utricle stimulation) in accordance with physiology [36][37].These structures are otherwise

explored in clinical practice using different tests that separately address the superior SCC (e.g., the Hennebert sign, which shows classically a vertical and torsional nystagmus corresponding to a superior SCC response) and otolith structures (particularly utricle), explored through AC or BC oVEMPs.

5. SVIN as a Bone-Conducted Tullio Phenomenon (BCTP)

The precise mechanism of this phenomenon appears to provide an understanding of the variable results between (and even within) patients and investigators. Here, we consider the apparent discrepancies concerning the direction-changing VIN nystagmus depending upon stimulus location [8][10][13], frequency [9][24], and the presence in a few patients of an after-nystagmus (AN) (persistent VIN after stimulus withdrawal), usually unobserved in uVL lesions [9][17][24].

Dumas et al. [24] link the three main points observed with AC sound in animals with artificially created dehiscence by Tullio in 1929 [5][6] (i.e., imbalance, sway and nystagmus in the plane of the dehiscent canal, and vestibular discomfort) to what is observed after cranial vibrations in clinical practice. The vertex location appears to be particularly effective in inducing nystagmus, associated with reports of dizziness [9][24].

These clinical results cannot be explained by only the third window mechanism associated with bone conduction facilitation toward the lesion side; they need to be interpreted in light of recent data explaining the Tullio phenomenon reported by Iversen et al. (2018) [26] and Rabbitt et al. [27].

Role of endolymphatic flow created by vibrations. The mechanism by which BCV activates SCC afferent neurons in patients with SCD appears to be that traveling waves are generated in the endolymph, initiated at the site of dehiscence [24], and travel from that site in both directions around the canal. Recent direct physical measurements of fluid flow in an artificial dehiscent SCC demonstrate this mechanism [26]. This mechanism is confirmed by physiological evidence from recording single SCC neurons in animals after artificial SCD. These data demonstrate that vibration causes two modes of activation of SCC neurons after SCD:

(1)Cycle-by-cycle phase-locked activation of action potentials in SCC afferents with irregular resting discharge;

(2)Cupula deflection by fluid streaming caused by the traveling waves of fluid displacement initiated by sound or vibration at the point of dehiscence. This fluid flow causes a slow deflection of the cupula, allowing for a slow return at the end of BCV stimulation and thus accounting for after-nystagmus. This cupula deflection stimulates neurons with regular resting discharge that are not directly activated by cycle-by-cycle phase-locked vibration [24][26]. The direction of the fluid current depends on the location of the stimulus, the location and size of the dehiscence, and the frequency of the stimulus [26][27][28]. The direct measures showed that the direction of fluid flow changed with frequency.

These two mechanisms explain the following:

(1)Direct stimulation on the affected side of the type I vestibular receptor hair cells (and afferent neurons with irregular neural discharge) at high frequency favored by BC facilitation toward the side of the dehiscence in relation to the third window mechanism [25]. This explains the most commonly observed ipsilateral excitatory nystagmus.

(2)The second mechanism [26][27] explains, in some SCD patients, a prolonged VIN after stimulus offset (after-nystagmus) mimicking, in some cases, an after-nystagmus after the end of an angular acceleration stimulus.

As shown by Curthoys et al., these two co-existing mechanisms—cycle-by-cycle activation and fluid flow—work together or they can oppose each other. After SCD, if acoustic streaming deflects the cupula in an inhibitory direction, all the receptor hair cells and afferents from that canal will be progressively silenced. Consequently, maintained sound or vibration stimulation silences the cycle-by-cycle phase-locked activation of irregular afferents. Thus, these two mechanisms may in some conditions be complementary and in others in competition or antagonistic, explaining the possible variability in the resulting SVIN direction.

This corresponds to the frequent horizontal direction-changing nystagmus observed on mastoid stimulation depending on the side of the stimulated mastoid.

It also explains the variability of the vertical component in the literature (up- or down-beating nystagmus) depending on stimulus location [9][10][17][24] and sometimes on frequency [9][24].

Why does BCV at Vx in patients with SCD induce an ipsilateral SVIN (observed in 80% of cases)?

In their animal model, Iversen et al. [26] showed that the direction of the fluid flow caused by BCV stimulation after an artificial SCD depended on the size of the dehiscence, the location of the dehiscence, and the frequency of the stimulus. The frequencies from 100 to 500 Hz caused excitation and frequencies >500 Hz (up to 800 Hz) caused inhibition and then again excitation at even higher frequencies in their toadfish model. The reversal appears to depend on which of the two traveling waves causes cupula deflection. They also recorded single SCC neurons, which confirmed the predictions from their measurements and modeling. It also explains the nystagmus observed in humans.

Such stimulation by vibration usually induces concomitant dizziness or unsteadiness, possibly more suggestive of otolith symptoms than canal symptoms. Unsteadiness or discomfort often associated with nausea has been reported by Dumas et al. [9] in 16/27 SCD patients (60%) repeatedly stimulated. This has already been described with air-conducted sound (ACTP) by Minor et al. [1] and Ward et al. [4].

6. Sensitivity of SVIN to Detect SCD Compared with Other Bedside Explorations or Vestibular Test Explorations

Classical bedside examinations in SCD are compressional tests for nystagmus induced by AC sounds (Tullio phenomenon) or by the Valsalva test with a pinched nose or the Hennebert sign [2][4]. These tests provoke a positive pressure in the middle ear that induces, via the oval window, an ampullofugal endolymph flow and nystagmus for the anterior SCC. The sensitivity varies for the AC Tullio phenomenon between 25% and 80% and for the Hennebert sign between 21% and 25%.

Mehta et al. observed positive Hennebert sign, ACTP, and a positive SVIN in 20%, 30%, and 40% of cases, respectively, in SCD [23]. A positive SVIN was reported by Batuecas et al. [21] in 62% of their cases and in 82% and 87% of cases by Dumas et al. [9][17]. These results suggest a possible higher sensitivity and efficiency of the BC Tullio phenomenon (BCTP) represented by SVINT over the AC Tullio phenomenon (ACTP).

Similarly, BC stimulations for oVEMPs are more efficient than BC cVEMPs in uncovering SCD patients but are not more efficient than AC oVEMPs when compared with air-conducted stimulations [11][12].

Otolith tests (oVEMP, cVEMP) are currently described as the most efficient in SCD diagnosis; they are positive in 80–90% of cases with ACS stimulations [11][14] but also after BC stimulations [43].

The value of SVINT, a recent exploration that is less specific but more sensitive, rapid, and is used as a non-intrusive bedside test, is to show (or suggest) an SCD and symptoms related to a third window mechanism. This test is interesting to report because it is in SCD more often observed than other classical objective tests such as the Hennebert sign and air-conducted Tullio phenomenon.

SVIN extension of sensitivity toward very high frequencies is similar to what is observed with BCV for oVEMPs and cVEMPs, which after CT-verified SCD, show an extension of sensitivity toward very high frequencies (4000 Hz). This was particularly observed by Manzari with oVEMPs in 100% of his 22 patients with SCD [13].

In summary, VEMPs appear to offer a non-invasive test providing quantitative data and are the most useful test for surgical decision-making. SVIN, considered as a BCTP, has recently showed its useful contribution in SCD diagnosis among other bedside examination tests such as the Hennebert test and complements the AC Tullio phenomenon with possibly more sensitivity.

7. Limitations

A limitation is the small series reported (6 to 40 patients). SCD is an infrequent pathology (0.5% in the series of 1000 temporal bone archives of Carey et al. [47]) often misrecognized in clinical practice [48][49][50].

The results show strong heterogeneity in the SVIN protocol between series considering that different authors use different stimulus topographies and frequencies.

Vibrators using adequate frequencies from 100 to 800 Hz may miss the nystagmus mainly when the vibrator amplitude is too small to provoke a jerk effect on the inner ear hair cell bundles, which work as seismic receptors, as already described by I.S. Curthoys [51]. Dlugaiczyk et al. [34] showed that the vibration needs accelerations of the head between 0.1 and 0.4 g to elicit a neural response, which are values below 2 g, non-invasive in humans. Otherwise, it has been shown that for a constant frequency, vibrators delivering amplitudes of vibrations lower than 0.1 mm are inefficient to reveal a nystagmus.

The series using frequencies only around 100 Hz may miss the nystagmus, which is sometimes visible at 400 Hz or at higher-frequency vibrations. The SVIN is sometimes very poor with small SPV at 100 Hz [9].

Using eye fixation minimizes or suppresses by inhibition the horizontal component. Thus, it is mandatory when using SVINT to mask the non-recorded eye to improve the response and suppress this bias [24]. Otherwise a sufficient stimulus duration is advised.

References

1. Minor, L.B. Superior canal dehiscence syndrome. Am. J. Otol. 2000, 21, 9–19.
2. Minor, L.B.; Solomon, D.; Zinreich, J.S.; Zee, D.S. Sound- and/or Pressure-Induced Vertigo Due to Bone Dehiscence of the Superior Semicircular Canal. Arch. Otolaryngol. Head Neck Surg. 1998, 124, 249.
3. Songer, J.E.; Rosowski, J.J. A mechano-acoustic model of the effect of superior canal dehiscence on hearing in chinchilla. J. Acoust. Soc. Am. 2007, 122, 943–951.
4. Ward, B.K.; Carey, J.P.; Minor, L.B. Superior Canal Dehiscence Syndrome: Lessons from the First 20 Years. Front. Neurol. 2017, 8, 177.
5. Tullio, P. Das Ohr und die Entstehung der Sprache und Schrift; England Urban & Schwartzenberg: Oxford, UK, 1929; p. 445.
6. Tullio, P. Sulla Funzione Delle Varie Parti Dell’orecchio Interno; Capelli: New York, NY, USA, 1926.
7. Parker, D. Effect of Sound on the Vestibular System; Miami University: Oxford, OH, USA, 1976; p. TR-75-89.
8. Dumas, G.; Perrin, P.; Morel, N.; N’Guyen, D.Q.; Schmerber, S. Skull vibratory test in partial vestibular lesions--influence of the stimulus frequency on the nystagmus direction. Rev. Laryngol. Otol. Rhinol. 2005, 126, 235–242.
9. Dumas, G.; Tan, H.; Dumas, L.; Perrin, P.; Lion, A.; Schmerber, S. Skull vibration induced nystagmus in patients with superior semicircular canal dehiscence. Eur. Ann. Otorhinolaryngol. Head Neck Dis. 2019, 136, 263–272.
10. White, J.A.; Hughes, G.B.; Ruggieri, P.N. Vibration-induced nystagmus as an office procedure for the diagnosis of superior semicircular canal dehiscence. Otol. Neurotol. 2007, 28, 911–916.
11. Rosengren, S.M.; Colebatch, J.G.; Young, A.S.; Govender, S.; Welgampola, M.S. Vestibular evoked myogenic potentials in practice: Methods, pitfalls and clinical applications. Clin. Neurophysiol. Pract. 2019, 4, 47–68.
12. Zhang, A.S.; Govender, S.; Colebatch, J.G. Tuning of the ocular vestibular evoked myogenic potential (oVEMP) to air- and bone-conducted sound stimulation in superior canal dehiscence. Exp. Brain Res. 2012, 223, 51–64.
13. Manzari, L.; Burgess, A.M.; McGarvie, L.A.; Curthoys, I.S. An indicator of probable semicircular canal dehiscence: Ocular vestibular evoked myogenic potentials to high frequencies. Otolaryngol. Head Neck Surg. 2013, 149, 142–145.
14. Zuniga, M.G.; Janky, K.L.; Nguyen, K.D.; Welgampola, M.S.; Carey, J.P. Ocular versus cervical VEMPs in the diagnosis of superior semicircular canal dehiscence syndrome. Otol. Neurotol. 2013, 34, 121–126.
15. Welgampola, M.; Rosengren, S.; Halmagyi, G.; Colebatch, J. Vestibular activation by bone conducted sound. J. Neurol. Neurosurg. Psychiatry 2003, 74, 771–778.
16. Cremer, P.; Minor, L.; Zee, D. Nystagmus produced by mastoid vibration in patients with Tullio’s phenomenon. In Transactions of the XXth Regular Meeting of the Barany Society; Würzburg; 11–12 September 1998; Claussen, C.F., Haid, C.T., Hofferberth, B., Eds.; Elsevier: Amsterdam, The Netherlands, 1998.
17. Dumas, G.; Lion, A.; Karkas, A.; Perrin, P.; Perottino, F.; Schmerber, S. Skull vibration-induced nystagmus test in unilateral superior canal dehiscence and otosclerosis: A vestibular Weber test. Acta Oto-Laryngol. 2014, 134, 588–600.
18. Aw, S.T.; Aw, G.E.; Todd, M.J.; Bradshaw, A.P.; Halmagyi, G.M. Three-dimensional vibration-induced vestibulo-ocular reflex identifies vertical semicircular canal dehiscence. J. Assoc. Res. Otolaryngol. 2011, 12, 549–558.
19. Manzari, L.; Modugno, G.C.; Brandolini, C.; Pirodda, A. Bone vibration-induced nystagmus is useful in diagnosing superior semicircular canal dehiscence. Audiol. Neurootol. 2008, 13, 379–387.
20. Park, J.H.; Kim, H.J.; Kim, J.S.; Koo, J.W. Costimulation of the horizontal semicircular canal during skull vibrations in superior canal Dehiscence syndrome. Audiol. Neurootol. 2014, 19, 175–183.
21. Batuecas-Caletrío, Á.; Jara, A.; Suarez-Vega, V.M.; Marcos-Alonso, S.; Sánchez-Gómez, H.; Pérez-Fernández, N. Skull Vibration-Induced Nystagmus and High Frequency Ocular Vestibular-Evoked Myogenic Potentials in Superior Canal Dehiscence. Audiol. Res. 2022, 12, 202–211.

22. Schmerber, S.; Dumas, G.; Perrin, P. Anterior semicircular canal dehiscence and cranial vibration-induced nystagmus test. Otol. Neurotol. 2008, 29, 573–574; author reply 574.

23. Mehta, R.; Klumpp, M.L.; Spear, S.A.; Bowen, M.A.; Arriaga, M.A.; Ying, Y.L.M. Subjective and objective findings in patients with true dehiscence versus thin bone over the superior semicircular canal. Otol. Neurotol. 2015, 36, 289–294.

24. Dumas, G.; Curthoys, I.S.; Castellucci, A.; Dumas, L.; Perrin, P.; Schmerber, S. A bone-conducted Tullio phenomenon—A bridge to understand skull vibration induced nystagmus in superior canal dehiscence. Front. Neurol. 2023, 14, 1183040.

25. Songer, J.E.; Rosowski, J.J. The effect of superior canal dehiscence on cochlear potential in response to air-conducted stimuli in chinchilla. Hear. Res. 2005, 210, 53–62.

26. Iversen, M.M.; Zhu, H.; Zhou, W.; Della Santina, C.C.; Carey, J.P.; Rabbitt, R.D. Sound abnormally stimulates the vestibular system in canal dehiscence syndrome by generating pathological fluid-mechanical waves. Sci. Rep. 2018, 8, 10257.

27. Rabbitt, R.D. Semicircular canal biomechanics in health and disease. J. Neurophysiol. 2019, 121, 732–755.

28. Liebau, G. Uber ein ventiloses pumpprinzip. Naturwissenschaften 1954, 41, 327.

29. Merchant, S.N.; Rosowski, J.J. Conductive Hearing Loss Caused by Third-Window Lesions of the Inner Ear. Otol. Neurotol. 2008, 29, 282–289.

30. White, J.; Krakovitz, P. Nystagmus in Enlarged Vestibular Aqueduct: A Case Series. Audiol. Res. 2015, 5, 120.

31. Stenfelt, S. Investigation of Mechanisms in Bone Conduction Hyperacusis With Third Window Pathologies Based on Model Predictions. Front. Neurol. 2020, 11, 966.

32. Freeman, S.; Sichel, J.Y.; Sohmer, H. Bone conduction experiments in animals—Evidence for a non-osseous mechanism. Hear. Res. 2000, 146, 72–80.

33. Manzari L., Burgess A.M., McGarvie L.A., Curthoys I.S. Ocular and cervical vestibular evoked myogenic potentials to 500 Hz fz bone-conducted vibration in superior semicircular canal dehiscence. Ear Hear. (2012);33(4):508-20.

34. Dlugaiczyk, J.; Burgess, A.M.; Goonetilleke, S.C.; Sokolic, L.; Curthoys, I.S. Superior Canal Dehiscence Syndrome: Relating Clinical Findings With Vestibular Neural Responses From a Guinea Pig Model. Otol. Neurotol. 2019, 40, e406–e414.

35. Koo, J.W.; Hong, S.K.; Kim, D.K.; Kim, J.S. Superior semicircular canal dehiscence syndrome by the superior petrosal sinus. J. Neurol. Neurosurg. Psychiatry 2010, 81, 465–467.

36. Cohen, B.; Suzuki, J.I.; Bender, M.B. Eye movements from semicircular canal nerve stimulation in the cat. Ann. Otol. Rhinol. Laryngol. 1964, 73, 153–169.

37. Suzuki, J.I.; Tokumasu, K.; Goto, K. Eye movements from single utricular nerve stimulation in the cat. Acta Otolaryngol. 1969, 68, 350–362.

38. Lehmkuhl, B.; Andaloro, C. Tullio Phenomenon. In StatPearls ; StatPearls Publishing: Treasure Island, FL, USA, 2022. Available online: http://www.ncbi.nlm.nih.gov/books/NBK513229/ (accessed on 28 November 2022).

39. Halmagyi, G.M.; Curthoys, I.S.; Colebatch, J.G.; Aw, S.T. Vestibular responses to sound. Ann. N. Y. Acad. Sci. 2005, 1039, 54–67.

40. Basura, G.J.; Cronin, S.J.; Heidenreich, K.D. Tullio phenomenon in superior semicircular canal dehiscence syndrome. Neurology 2014, 82, 1010.

41. Addams-Williams, J.; Wu, K.; Ray, J. The experiments behind the Tullio phenomenon. J. Laryngol. Otol. 2014, 128, 223–227.

42. Carey, J.P.; Hirvonen, T.P.; Hullar, T.E.; Minor, L.B. Acoustic Responses of Vestibular Afferents in a Model of Superior Canal Dehiscence. Otol. Neurotol. 2004, 25, 345–352.

43. Taylor, R.L.; Magnussen, J.S.; Kwok, B.; Young, A.S.; Ihtijarevic, B.; Argaet, E.C.; Reid, N.; Rivas, C.; Pogson, J.M.; Rosengren, S.M.; et al. Bone-Conducted oVEMP Latency Delays Assist in the Differential Diagnosis of Large Air-Conducted oVEMP Amplitudes. Front. Neurol. 2020, 11, 580184.

44. Erlich, M.A.; Lawson, W. The incidence and significance of the Tullio phenomenon in man. Otolaryngol. Head Neck Surg. 1979, 88, 630–635.

45. Castellucci A., Piras G., Del Vecchio V., Crocetta F.M., Maiolo V., Ferri G.G., et al. The effect of superior canal dehiscence size and location on audiometric measurements, vestibular-evoked myogenic potentials and video-head impulse testing. Eur Arch Otorhinolaryngol.(2021);278(4):997-1015.

46. Ionescu, E.C.; Reynard, P.; Damien, M.; Ltaief-Boudrigua, A.; Hermann, R.; Gianoli, G.J.; Thai-Van, H. Why should multiple dehiscences of the otic capsule be considered before surgically treating patients with superior semicircular canal dehiscence? A radiological monocentric review and a case series. Front. Neurol. 2023, 14, 1209567.
47. Carey, J.P.; Minor, L.B.; Nager, G.T. Dehiscence or thinning of bone overlying the superior semicircular canal in a temporal bone survey. Arch. Otolaryngol. Head Neck Surg. 2000, 126, 137–147.
48. Ward, B.K.; van de Berg, R.; van Rompaey, V.; Bisdorff, A.; Hullar, T.E.; Welgampola, M.S.; Carey, J.P. Superior semicircular canal dehiscence syndrome: Diagnostic criteria consensus document of the committee for the classification of vestibular disorders of the Bárány Society. J. Vestib. Res. 2021, 31, 131–141.
49. Masaki, Y. The prevalence of superior canal dehiscence syndrome as assessed by temporal bone computed tomography imaging. Acta Otolaryngol. 2011, 131, 258–262.
50. Reynard, P.; Idriss, S.; Ltaief-Boudrigua, A.; Bertholon, P.; Pirvan, A.; Truy, E.; Thai-Van, H.; Ionescu, E.C. Proposal for a Unitary Anatomo-Clinical and Radiological Classification of Third Mobile Window Abnormalities. Front. Neurol. 2022, 12, 792545.
51. I.S. Curthoys; Ann M. Burgess; Samanthi C. Goonetilleke; Phase-locking of irregular guinea pig primary vestibular afferents to high frequency (>250 Hz) sound and vibration. *Hear. Res.*. **2018**, *373*, 59-70.