Literature Review

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# Video Head Impulse Test and central nervous system diseases: a integrative review

## *Video Head Impulse Test* e doenças do sistema nervoso central: uma revisão integrativa

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## ABSTRACT

**Purpose**: To verify the applicability of the Video Head Impulse Test (vHIT) in central nervous system (CNS) diseases, as well as the results found and the diseases described. **Research strategy**: Integrative literature review, in which nine electronic databases were searched using the keyword "video head impulse test". **Selection criteria**: Studies that used the vHIT in the diagnosis of CNS diseases were included, and studies published before 2009, studies that performed other clinical investigation procedures or that concerned the diagnosis of peripheral vestibular diseases were excluded. **Results**: The final sample consisted of 18 studies. The verified results show that the vestibulo-ocular reflex (VOR) has shown alteration in this population. Suggestive findings of central involvement were observed, such as lower gain or average VOR in the vertical semicircular canals than in the lateral ones, increased gain, the negative correlation of gain with disease severity in Spinocerebellar Ataxia Type 3, cutoff point of 0.70, and gain asymmetry of less than 20% to differentiate vestibular neuritis from a stroke in the medial branch of the posteroinferior cerebellar artery, normal gain with altered oculomotor tests, presence of spontaneous vertical nystagmus, as well as alterations in the VOR with and without visual enhancement, in saccadic pursuit, and the tilt deviation test. **Conclusion**: We found that the vHIT applies to the assessment of high-frequency VOR in individuals with CNS diseases since it provided clinical evidence of changes in peripheral and central vestibular function in different neurological conditions

**Keywords:** Dizziness; Vertigo; Vestibulo-ocular reflex; Central nervous system diseases; Vestibular function tests

## RESUMO

**Objetivos:** Verificar a aplicabilidade do *Video Head Impulse Test* (vHIT) em doenças do sistema nervoso central (SNC), bem como os resultados encontrados e as doenças descritas. **Estratégia de pesquisa:** Revisão integrativa da literatura, em que foi realizada a busca em nove bases eletrônicas de dados, a partir da palavra-chave *"video head impulse test*". **Critérios de seleção:** Foram incluídos estudos que utilizaram o vHIT no diagnóstico de doenças do SNC e excluídos os estudos publicados antes de 2009 e estudos que realizaram outros procedimentos de investigação clínica, ou que aplicaram o teste no diagnóstico de doenças vestibulares periféricas. **Resultados:** A amostra final foi composta por 18 estudos. Os resultados verificados mostraram que o reflexo vestíbulo-ocular (RVO) tem apresentado alterações na população investigada. Foram observados achados sugestivos de acometimento central, tais como ganho ou média de ganho do RVO nos canais semicirculares verticais, inferior aos laterais, ganho aumentado, correlação negativa do ganho com a gravidade da doença na ataxia espinocerebelar tipo 3, ponto de corte de 0,70 e assimetria de ganho menor de 20% para diferenciar neurite vestibular de derrame no ramo medial da artéria cerebelar posteroinferior, ganho normal com provas oculomotoras alteradas, presença de nistagmo espontâneo vertical, além de alterações no RVO com e sem otimização visual, na perseguição sacádica e no teste de desvio de inclinação. **Conclusão:** Verificou-se que o vHIT é aplicável quanto a avaliação do RVO de alta frequência em indivíduos com doenças do SNC, uma vez que trouxe evidências clínicas sobre alterações da função vestibular periférica e central nos diferentes quadros neurológicos.

**Palavras-chave:** Tontura; Vertigem; Reflexo vestíbulo-ocular; Doenças do sistema nervoso central; Testes de função vestibular

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## INTRODUCTION

The Video Head Impulse Test (vHIT) is a diagnostic modality that provides an objective quantitative measure of the vestibulo-ocular reflex (VOR) through analysis of the dynamic functioning of the six semicircular canals (SCC) individually. The vHIT is mainly used in the differential diagnosis between acute peripheral vestibular disorders, such as vestibular neuritis, and diseases that affect the central nervous system (CNS)(1).

The VOR is considered normal when the eyes, kept fixed on a target during head rotation, present the same speed of the head in the opposite direction. Reduction in this reflex gain indicates vestibular hypofunction, which generates a complaint of blurred vision (oscillopsia) resulting from difficulty in keeping the gaze on target(1,2). Analysis of this gain is not the only parameter capable of revealing changes in this reflex; catch-up, or corrective, saccades are another sign observed when the VOR is deficient, and they can be covert when occurring during head rotation, and overt, when occurring after head rotation(1).

In addition to these parameters, there is the oculomotor evaluation module, which enables the recording of the search for spontaneous and semi-spontaneous nystagmus by performing the horizontal VOR test with visually enhanced vestibulo–ocular reflex (VVOR) and vestibulo-ocular reflex suppression (VORS), the skew deviation test, and saccadic pursuit(3). These tests provide information on the oculomotor connections with the peripheral and central vestibular pathways that contributes to a more accurate diagnosis, especially in neurological conditions, when analyzed together with other clinical examinations and instrumental assessments(4).

There are reports of vestibular symptoms in patients with CNS disorders in the scientific literature; however, the vHIT is still not routinely indicated to evaluate this population. As VOR functioning is related to both the neurophysiology of the SSC and the CNS, analysis of this reflex is important because it provides relevant information about the central vestibular system(5).

## PURPOSE

To verify the scientific literature regarding the applicability of the vHIT in CNS disorders, as well as the results found and the diseases reported.

## RESEARCH STRATEGY

The literature review was conducted as follows: preparation of the guiding question, search in the scientific literature, extraction, and analysis of data, and presentation and discussion of results(6).

The following guiding questions were used: “What is the applicability of the vHIT in individuals with CNS disorders? What vHIT results were found? What diseases were reported?”.

The search was carried out on October 1, 2019, in nine electronic databases, namely, Google Scholar, LILACS (*Literatura Latino-Americana em Ciências de Saúde*), Livivo, OpenGrey, ProQuest Dissertation, and Theses Global, PubMed/MEDLINE, SciELO *(*Scientific Electronic Library Online*),* Scopus, and Web of Science, using the descriptor “video head impulse test” aiming not to exclude any study relevant to this literature review, since there is a variety of diseases that affect the CNS and there is no common term that includes all of them.

## SELECTION CRITERIA

Inclusion criteria: studies that used the vHIT to assess individuals with CNS disorders.

Exclusion criteria: studies that included individuals with diseases of the peripheral nervous system, performed only other types of vestibular exams, were published before 2009 (when the vHIT started being commercialized); literature reviews; letters to the editor; abstracts in scientific conference proceedings.

The publications were selected by two independent reviewers in two stages: 1. reading of the titles and abstracts of all retrieved publications and exclusion of the works that did not address the theme of the present study; 2. reading in full of the selected publications and application of the exclusion criteria.

## DATA ANALYSIS

Aiming to systematize the data relevant to the outcomes under investigation, the following information was extracted from the studies: authorship, year of publication, sample size, age or mean age of participants, type of study, level of evidence, a central disease described, vestibular symptoms, and results of the vHIT and oculomotor tests.

The strength of the evidence was assessed through the classification proposed by Cox(7), which considers the types of studies, with level 1 as the highest and level 6 as the lowest. Evidence with the highest grade of recommendation (level 1) results from a methodical compilation (systematic review or meta-analysis) of several randomized controlled trials, whereas evidence with the lowest grade (level 6) derives from expert opinion.

After analyzing and synthesizing the collected data, the results were descriptively presented.

## RESULTS

A total of 1,481 publications were retrieved through the search in the aforementioned electronic databases, and 454 of them remained after removing the works in duplicate. In the first selection phase, the titles and abstracts of the identified studies were evaluated, and only 21 of them addressed the study theme of this literature review. These publications were read in full and, after application of the eligibility criteria, 18(8-25) were selected to compose the final sample of this study. Figure 1 shows the detailed selection process of these publications.

From the information extracted from the 18 selected articles(8-25), a chart was created including data on disease, vestibular symptoms, and results of the vHIT and oculomotor tests (Chart 1).

All studies included in this literature review were published between 2014 and 2019. Regarding study design, there were seven case reports(8,9,14,18,22,23,25), four case series(13,19,21,24), six case controls(10-12,15,16,20), and one cross-sectional study(17).

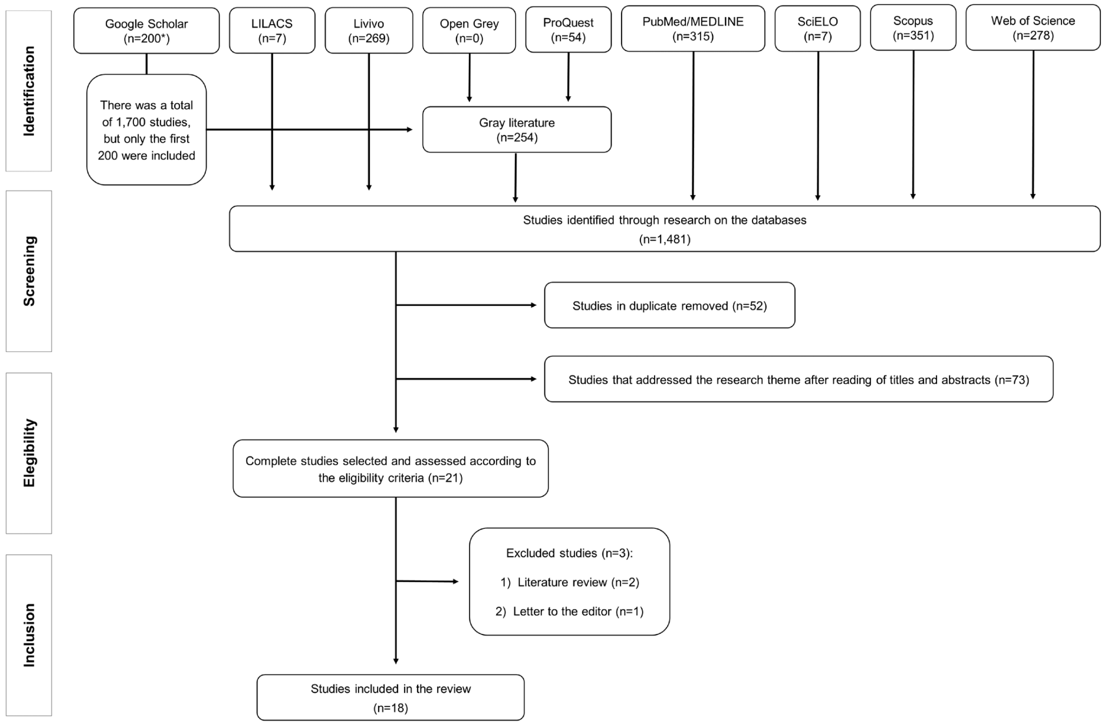


**Figure 1.** Flowchart of the study selection process
**Subtitle:** n = number of studies

The sample size ranged from 1 to 63, with age or mean age of participants between 25 and 77 years. The strength of scientific evidence was classified as level 5 for 11 articles[8,9,13,14,18,19,21-25) and level 4 for seven articles[10-12,15-17,20]. This was since most studies were case reports, followed by case controls, which are designs with a low grade of recommendation because they are more likely to be biased.

As for Chart 1, the following CNS disorders were studied: cerebellar stroke[8,12,17,23], multiple sclerosis (MS)[9,10,20], cerebellar ataxia with neuropathy, and vestibular areflexia syndrome (CANVAS)[19,22,24], superficial siderosis of the CNS[14,21], Wernicke encephalopathy[18], left inferior cerebellar peduncle glioma[25], progressive supranuclear palsy (PSP)[11], mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke (MELAS syndrome)[13], Parkinson's disease (PD)[16], spinocerebellar ataxia types 1 (SA1), 2 (SA2) and 3 (SA3) and Friedreich's ataxia (FA)[15].

Eleven[8,9,12-14,17,18,21-24] of the 18 studies assessed found the presence of vestibular symptoms in their patients, with body imbalance and dizziness as the most reported. This information was not found in five studies[10,15,19,20,25], and two articles[11,16] did not mention the absence of these symptoms.

Results of the vHIT reported in 14 studies[9,10,12-15,18-25] showed reduced VOR gains. The values adopted as normality parameters were similar, with reflex gain considered altered when <0.8 for the lateral SSC and <0.7 for the posterior and anterior SSC. However, some of these studies[10,12,14,15,18,20,22,23,25] did not present supplementary information, such as VOR results by SSC and affected ear and values of asymmetry by synergistic pairs of the SCC.

The presence of covert catch-up saccades during head rotation and overt catch-up saccades after head rotation was considered a sign of abnormality[9,10,13-16,18-25]; however, parameters related to the saccades, such as percentage of occurrence, latency, and amplitude were not reported in any of the studies.

Most studies described the results of some oculomotor test performed[8-22,24,25]. Concerning the search for spontaneous and semi-spontaneous nystagmus, nine[8,9,12,17-19,22-24,] of the 13 studies that conducted it[8-12,14,17-19,22-25] reported presence of this movement, although some of them presented incomplete characterization as for type and direction. Three articles[19,22,24] performed the VVOR test and observed altered results and two studies[21,24] described the VORS findings: one[24] with responses within the normality standards and one[21] with altered responses. Two works performed the skew deviation test: one[23] with findings within the normality standards and one[17] with positive results. Five articles[8,11,14,21,25] conducted saccadic pursuit, and three of them[8,11,21] found altered results.

## DISCUSSION

Considering that each disorder has its clinical particularities, the discussion section of this literature review was structured based on the diseases and studies listed in Chart 1.

### Cerebellar stroke

Cerebellar strokes, which are a type of cerebrovascular accident (CVA), often cause severe ataxia, headache, and dizziness. However, there have been cases, mainly in emergency services, where only dizziness is reported by patients[17], which

**Chart 1.** Data on the disorders, vestibular symptoms, and results of the vHIT and oculomotor tests of the selected studies (n=18)

| Author | Disorders | Vestibular symptoms | Results of the vHIT | Results of the oculomotor tests |
|---|---|---|---|---|
| Armato et al.[8] | Cerebellar stroke | Vertigo and nausea | VOR gains within normality standards in both ears | Presence of horizontal spontaneous nystagmus to the left side |
| Barona-Lleo et al.[9] | Multiple sclerosis | Body imbalance and nausea | Reduced VOR gain for all SCC only to the RE (AL: 1.15º/s, LL 0.98º/s, PL: 0.96º/s, AR: 0.24º/s, LR: 0.67º/s, PR: 0.27º/s)<br>Presence of covert and overt catch-up saccades in all SCC in the RE<br>Asymmetry between all synergistic pairs of the SCC (A: 79%, L: 32%, P: 72%)<br>Results were normalized after drug treatment of multiple sclerosis (AL: 0.92º/s, LL 0.92º/s, PL: 0.94º/s, AR: 0.93º/s, LR: 1.04º/s, PR: 0.83/s) | Presence of upbeat and downbeat vertical spontaneous nystagmus |
| Garg et al.[10] | Multiple sclerosis | Missing information | Reduced VOR gain<br>Presence of corrective saccades | Absence of spontaneous and semi-spontaneous nystagmus |
| Goldschagg et al.[11] | Progressive supranuclear palsy | Absent | The vHIT was performed only in the lateral SCC<br>VOR gains within normality standards in both ears of all patients | Absence of spontaneous and semi-spontaneous nystagmus |
| Guler et al.[12] | Cerebellar stroke | Dizziness, nystagmus, and nausea | Reduced VOR in both ears related to anterior inferior cerebellar artery effusion<br>Normal VOR gain related to posterior inferior cerebellar artery effusion | Unidirectional spontaneous and semi-spontaneous nystagmus were present in nine and six patients, respectively<br>Presence of spontaneous and semi-spontaneous nystagmus |
| Hougaard et al.[13] | Mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke | Dizziness | Reduced VOR gain in two anterior, nine lateral and six posterior SCC of seven patients (vHIT not performed in one patient)<br>1: AL: 0.77º/s, LL: 0.54º/s, PL: 0.77º/s, AR: 0.99º/s, LR: 0.54º/s, PR: 0.48º/s<br>A: 0.88 L: 0.54 P: 0.62<br>2: AL: 0.64º/s, LL: 0.74º/s, PL: 0.51º/s, AR: 0.77º/s, LR: 0.68º/s, PR: 0.39º/s<br>A: 0.70 L: 0.71 P: 0.45<br>3: AL: 1.03º/s, LL: 0.77º/s, PL: 0.95º/s, AR: 1.42º/s, LR: 0.77º/s, PR: 1.09º/s<br>A: 1.22 L: 0.77 P: 1.02<br>4: AL: 1.07º/s, LL: 0.83º/s, PL: 0.83º/s, AR: 0.86º/s, LR: 0.65º/s, PR: 0.65º/s<br>A: 0.96 L: 0.74 P: 0.74<br>5: AL: 0.09º/s, LL: 0.07º/s, PL: 1.17º/s, AR: 1.32º/s, LR: 0.13º/s, PR: 0.38º/s<br>A: 0.70 L: 0.10 P: 0.77<br>6: AL: 0.88º/s, LL: 1.01º/s, PL: 1.24º/s, AR: 1.19º/s, LR: 1.04º/s, PR: 1.27º/s<br>A: 1.03 L: 1.02 P: 1.25<br>7: AL: 0.79º/s, LL: 0.81º/s, PL: 1.33º/s, AR: 1.08º/s, LR: 0.56º/s, PR: 0.68º/s<br>A: 0.93; L: 0.68; P: 1.00<br>Overall average: A: 0.95; L: 0.65; P: 0.83<br>Presence of covert and overt saccades in eight lateral and two posterior SCC and only of overt saccades in two anterior SCC | NP |
| Kang et al.[14] | Superficial siderosis of the CNS | Oscillopsia | Reduced VOR in all SCC in both ears (AL: 0.62º/s, LL: 0.61º/s, PL: 0.36º/s, AR: 0.56º/s, LR: 0.49º/s, PR: 0.55º/s)<br>Overall average: A: 0.59; L: 0.55; P: 0.45<br>Presence of covert and overt saccades in the anterior SCC and only overt saccades in the lateral and posterior SCC | Absence of spontaneous and semi-spontaneous nystagmus<br>Saccadic pursuit within normality standards |

**Subtitle:** n = Number of studies; vHIT = Video head impulse test; VOR = Vestibulo-ocular reflex; SCC = Semicircular canals; AL = Anterior left; AR = Anterior right; LL = Lateral left; LR = Lateral right; PL = Posterior left; PR = Posterior right; A = Anterior; L = Lateral; P = Posterior; LE = Left ear; RE = Right ear, º/s = Grade per second; VVOR = Visually enhanced vestibulo-ocular reflex; VORS = Vestibulo-ocular reflex suppression; SA3 = Spinocerebellar ataxia type 3; FA = Friedreich's ataxia; CANVAS = Cerebellar ataxia with neuropathy and bilateral vestibular areflexia syndrome; NP = Not performed.

**Chart 1.** Continued...

| Author | Disorders | Vestibular symptoms | Results of the vHIT | Results of the oculomotor tests |
|---|---|---|---|---|
| Luis et al.[15] | Spinocerebellar ataxia types 1, 2 and 3 and FA | Missing information | Reduced VOR gain<br>VOR latency was higher in patients with FA and SA3 than in the controls<br>Presence of covert saccades in patients with SA3 and overt saccades in all patients | NP |
| Lv et al.[16] | Parkinson's disease | Absence of symptoms | VOR gain within normality standards in all SCC in both ears<br>VOR gains significantly greater compared with those of the control group<br>Presence of covert and overt saccades | NP |
| Mantokoudis et al.[17] | Cerebellar stroke | Dizziness | VOR gains within normality standards in both ears | Presence of spontaneous and semi-spontaneous nystagmus<br>Positive vertical skew deviation |
| Mohamad et al.[18] | de Wernicke encephalopathy | Dizziness, nausea, and vomiting | Reduced VOR gain only in the lateral SCC in both ears (LL: 0.35º/s and LR: 0.42º/s)<br>Presence of covert and overt saccades in the anterior and posterior SCC<br>During drug treatment, there was a marked improvement in VOR gain bilaterally (LL: 0.60º/s and LR: 0.76º/s), although still below the normality standard; in addition, saccades and spontaneous nystagmus disappeared | Presence of horizontal spontaneous nystagmus to the right side |
| Moreno-Ajona et al.[19] | CANVAS | Missing information | Reduced VOR gain in all SCC in both ears of all five patients<br>1: AL: 0.31º/s, LL: 0.38º/s, PL: 0.36º/s, AR: 0.50º/s, LR: 0.22º/s, PR: 0.34º/s<br>A: 0.40 L: 0.30 P: 0.35<br>2: AL: 0.20º/s, LL: 0.59º/s, PL: 0.32º/s, AR: 0.59º/s, LR: 0.60º/s, PR: 0.10º/s<br>A: 0.39 L: 0.59 P: 0.21<br>3: AL: 0.28º/s, LL: 0.45º/s, PL: 0.46º/s, AR: 0.56º/s, LR: 0.36º/s, PR: 0.32º/s<br>A: 0.42 L: 0.40 P: 0.39<br>4: AL: 0.29º/s, AR: 0.39º/s, LL: 0.44º/s, LR: 0.30, PL: 0.05º/s, PR: 0.23º/s<br>A: 0.34 L: 0.37 P: 0.14<br>5: AL: 0.56º/s, AR: 0.57º/s, LL: 0.41º/s, LR: 0.52º/s, PL: 0.37º/s, PR: 0.39º/s<br>A: 0.56; L: 0.46; P: 0.38<br>Overall average: A: 0.42; L: 0.42; P: 0.29<br>Presence of covert and overt saccades | Presence of downbeat vertical spontaneous nystagmus and semi-spontaneous nystagmus<br>Altered VVOR in all patients<br>Normal VORS in all patients |
| Pavlovića et al.[20] | Multiple sclerosis | Missing information | Reduced VOR gain in lateral and posterior SCC<br>Presence of covert and overt saccades in all SCC | NP |
| Takeda et al.[21] | Superficial siderosis of the CNS | Body imbalance | The vHIT was performed only in lateral SCC<br>Reduced VOR gain in both ears in six patients (four patients did not undergo the tests)<br>Average value of SCC in the LE and RE, respectively:<br>1: 0.20º/s and 0.30º/s<br>2: 0.13º/s and 0.18º/s<br>3: 0.34º/s and 0.32º/s<br>4: 0.44º/s and 0.11º/s<br>5: 0.64º/s and 0.49º/s<br>6: 0.42º/s and 0.30º/s<br>Presence of covert and overt saccades bilaterally in two patients and one ear in three patients | Altered VORS<br>Altered saccadic pursuit |
| Taki et al.[22] | CANVAS | Body imbalance and dizziness | Reduced VOR gain in all SCC in both ears<br>Mean SCC:<br>LE: 0.10º/s<br>RE: 0.12º/s<br>Presence of covert and overt saccades bilaterally in two patients | Altered VVOR<br>Presence of semi-spontaneous nystagmus on both sides |

**Subtitle:** n = Number of studies; vHIT = Video head impulse test; VOR = Vestibulo-ocular reflex; SCC = Semicircular canals; AL = Anterior left; AR = Anterior right; LL = Lateral left; LR = Lateral right; PL = Posterior left; PR = Posterior right; A = Anterior; L = Lateral; P = Posterior; LE = Left ear; RE = Right ear, º/s = Grade per second; VVOR = Visually enhanced vestibulo-ocular reflex; VORS = Vestibulo-ocular reflex suppression; SA3 = Spinocerebellar ataxia type 3; FA = Friedreich's ataxia; CANVAS = Cerebellar ataxia with neuropathy and bilateral vestibular areflexia syndrome; NP = Not performed.

**Chart 1.** Continued...

| Author | Disorders | Vestibular symptoms | Results of the vHIT | Results of the oculomotor tests |
|---|---|---|---|---|
| Volgger et al.[23] | Cerebellar stroke | Body imbalance and dizziness | VOR gains within normality standards in the RE<br>Reduced VOR gain in all SCC in the LE, with a mean gain of 0.75 º/s in all SCC<br>Presence of overt saccades in the LE | Presence of spontaneous nystagmus with a rotational component to the right side, originated by the head-shaking test in the same direction, more intense than spontaneous nystagmus<br>Normal skew deviation test results |
| Yacovino et al.[24] | CANVAS | Body imbalance and dizziness | Reduced VOR gain in all SCC bilaterally in all five patients<br>1: AL: 0.28º/s, AR: 0.21º/s, LL: 0.14º/s, LR: 0.19º/s, PL: 0.26º/s, PR: 0.18º/s<br>A: 0.24; L: 0.16; P: 0.22<br>2: AL: 0.09º/s, AR: 0.12º/s, LL: 0.15º/s, LR: 0.14º/s, PL: 0.26º/s, PR: 0.14º/s<br>A: 0.10; L: 0.14; P: 0.20<br>3: AL: 0.11º/s, AR: 0.63º/s, LL: 0.07º/s, LR: 0.10º/s, PL: 0.56º/s, PR: 0.11º/s<br>A: 0.37; L: 0.08; P: 0.33<br>4: AL: 0.24º/s, AR: 0.37º/s, LL: 0.08º/s, LR: 0.08º/s, PL: 0.46º/s, PR: 0.27º/s<br>A: 0.30; L: 0.08; P: 0.36<br>5: AL: 0.66º/s, AR: 0.50º/s, LL: 0.43º/s, LR: 0.51º/s, PL: 0.30º/s, PR: 0.39º/s<br>A: 0.58; L: 0.47; P: 0.34<br>Overall average: A: 0.31; L: 0.18; P: 0.29<br>Presence of covert and overt saccades bilaterally in all SCC | Presence of downbeat vertical spontaneous nystagmus in four patients and of semi-spontaneous nystagmus in two patients<br>Altered VVOR<br>Normal VORS |
| Zuma and Maia et al.[25] | Left inferior cerebellar peduncle glioma | Missing information | Reduced VOR gains the lateral (LR: 0.38 and LL: 0.29) and posterior (PR: 0.49 and PL: 0.38) SCC in both ears<br>Presence of covert and overt saccades in the lateral and posterior SCC in both ears | Absence of spontaneous and semi-spontaneous nystagmus<br>Saccadic pursuit within normality standards |

**Subtitle:** n = Number of studies; vHIT = Video head impulse test; VOR = Vestibulo-ocular reflex; SCC = Semicircular canals; AL = Anterior left; AR = Anterior right; LL = Lateral left; LR = Lateral right; PL = Posterior left; PR = Posterior right; A = Anterior; L = Lateral; P = Posterior; LE = Left ear; RE = Right ear, º/s = Grade per second; VVOR = Visually enhanced vestibulo-ocular reflex; VORS = Vestibulo-ocular reflex suppression; SA3 = Spinocerebellar ataxia type 3; FA = Friedreich's ataxia; CANVAS = Cerebellar ataxia with neuropathy and bilateral vestibular areflexia syndrome; NP = Not performed.

has hindered diagnosis since there is a focus on causes of peripheral origin.

The four studies[8,12,17,23] that described cerebellar stroke used the vHIT as an ancillary tool to differentiate central dizziness, caused by vascular accidents, from the dizziness of peripheral origin, resulting from specific changes in the SCC. No other signs suggestive of neurological impairment were observed.

In all the aforementioned articles, the patients presented vestibular symptoms such as dizziness, body imbalance, and nausea, but reduced VOR gain measured by the vHIT was observed in only two of them[12,23]. These last two studies verified that the injured structures could influence the altered responses since the strokes occurred in the medial branch of the posterior inferior and anterior inferior cerebellar arteries, which are vessels that irrigate structures of the cerebellar vestibule connected to the vestibular nuclei.

Nevertheless, two other works[8,17] that reported normality in the VOR assessment stated that these results confirm central involvement, while the altered test results would indicate peripheral involvement, as in the case of vestibular neuritis. Results of the vHIT within the normality standards would assist in raising clinical suspicion of central vascular injury, as well as the need for imaging exams.

Based on the results found, a study[12] developed a cut-off point for the vHIT aiming to differentiate vestibular neuritis from cerebellar stroke, since imaging exams cannot be performed in emergency services in all patients who present with dizziness. Recognizing the risk of misclassifying CVA as vestibular neuritis and vice versa, the VOR gain cut-off point ($p<0.001$) used to consider a suggestive diagnosis of stroke in the medial branch of the posterior inferior cerebellar artery was >0.70, and with asymmetry gain <20%.

## Multiple sclerosis

Dizziness and body imbalance may be the manifest symptoms of multiple sclerosis (MS) in 4-20% of patients and occur during the disease in 50% of them[26]. Because of this incidence of otoneurologic complaints, evaluation of the vestibular system is considered essential.

Three studies[9,10,20] that investigated the use of the vHIT in this population, all with different objectives, were identified in this literature review. One of them[20] was carried out to investigate the role of this test in detecting brainstem lesions, and reported reduced VOR gain in the lateral ($p$=0.009) and posterior (anterior, $p$=0.027 and posterior, $p$=0.004) SCC and that the lesions of the disease that affect areas related to the central vestibular system could harm the VOR. The main finding described was that altered results were more frequently observed in patients diagnosed with MS than in healthy individuals. In addition, analysis of the magnetic resonance imaging (MRI) results evidenced an association between the presence of lesions and bilateral hypofunction of the lateral and posterior SCC ($p$=0.046). The study suggested that the results found may be due to demyelinating lesions in the emergence of the vestibular nerve root, vestibular nucleus, or deep cerebellar nuclei that modulate the VOR.

Another article[10] compared the VOR measurements of patients with and without MS. The results showed significant differences ($p$<0.05), such as reduced VOR gain and presence of catch-up saccades, with no specification regarding the SCC, about the neurologically healthy control group. The great variability regarding VOR gain in the MS group was highlighted, which was associated with the individual number and variations in the location of the lesions. The study also reported that imaging exams could provide information about the neural substrates of the observed vestibular deficits, that is, the possibility to observe the pattern of lesions and relate it to the results found.

Finally, a case report[9] verified inconsistencies between the clinical signs and symptoms observed and the clinical evaluation performed. The patient presented with acute vestibular syndrome, with dizziness, nausea, and vomiting. Upbeat spontaneous nystagmus and reduced VOR gain were observed in all SCC in the right ear, with the presence of corrective saccades, which suggested acute peripheral vestibulopathy on the right side. However, by verifying that the VOR gain reduction in the vertical SCC was smaller than that in the lateral SCC, this characteristic pattern was considered inadequate by the authors for peripheral involvement, based on the study conducted by Aw et al.[27], who raised the possibility of injury to the central vestibular pathways. The study showed that the symptoms presented by the patient were due to a lesion in the vicinity of the right vestibular nucleus, which justified the upbeat nystagmus and the reduced VOR gain, especially in the vertical SCC.

## Cerebellar ataxia with neuropathy and vestibular areflexia syndrome

Cerebellar ataxia with neuropathy and vestibular areflexia syndrome (CANVAS) is caused by a focal dorsal root ganglionopathy, which affects the Scarpa's ganglion (vestibular), but preserves the Spiral ganglion (audition)[28]. Thus, otoneurologic investigation plays an indispensable role in defining this condition.

Three studies[19,22,24] addressed the use of the vHIT in this population, describing cases of individuals who experienced body imbalance as well as other symptoms and underwent clinical examinations that found reduced VOR gain and presence of covert and overt catch-up saccades in all SCC bilaterally. These findings were justified by the possible degeneration of the cerebellum and Scarpa's ganglion, which are structures responsible for body balance[29]. In another study[24], three patients presented reduced mean VOR gain in the lateral SCC and two patients in the vertical SCC. A study[19] reported that both the VOR gain of the posterior SCC and the overall average of the vertical SCC were worse than those in the lateral SCC, and in another study[22], this information was missing. Therefore, it was possible to observe some cases[19,24] where the overall average of reduced VOR gain values was smaller in the vertical SCC than in the lateral SCC - a finding considered suggestive of central involvement[27]. In addition, two studies[19,24] observed the presence of downbeat vertical spontaneous nystagmus, also compatible with central involvement.

Moreover, the symptomatology of this disease is accompanied by the so-called "doll's eye reflex", that is, change in the VOR with visual capture, which configures combined impairment of the three corrective systems of eye movements: the VOR, the slow tracking, and the optokinetic reflex - a finding that is solidly described as a predictor of CANVAS in the specific literature[19,22,24].

## Superficial siderosis of the CNS

Superficial siderosis of the CNS manifests with cerebellar ataxia, symptoms of pyramidal origin, and sensorineural hearing loss, with the last being most commonly observed. These symptoms are due to the abnormal presence of an iron compound (hemosiderin) on the neurological surface that leads to nerve tissue damage[14,21].

Two studies[14,21] used the vHIT to investigate the vestibular function in this population. One of them[14] described the case of a patient with progressive hearing loss, gait instability, and oscillopsia, and found reduced VOR gain in all SCC, with worse mean gain in the vertical SCC compared with that in the lateral SCC, suggestive of central involvement[27] in both ears, and presence of covert and overt saccades. The other study[21] performed the vHIT in six patients with body imbalance, but only the lateral SCC was evaluated. Reduced VOR gain and catch-up saccades were obtained in all SCC, analyzed bilaterally in two individuals and unilaterally in one individual. In addition, the VORS and saccadic pursuit presented altered results. Changes in the brainstem and cerebellum were found on the imaging exams.

Therefore, this condition may be a cause of bilateral vestibular hypofunction resulting from central involvement, due to hemosiderin deposition, and/or peripheral involvement, since the vestibulocochlear nerve is a structure vulnerable to this disease[14,21].

## Wernicke encephalopathy

Wernicke encephalopathy is an acute neurological disorder associated with thiamine (vitamin B1) deficiency. The presence of nystagmus and vestibular dysfunction are common signs of this disease[18].

Some researchers[18] performed the vHIT, as well as other procedures such as clinical evaluation, and found cerebellar involvement due to the presence of ataxia and horizontal spontaneous nystagmus to the right side. The vHIT was applied as an ancillary tool in the diagnosis of Wernicke encephalopathy. The results obtained – reduced VOR gain only in the lateral SCC and covert and overt saccades in the anterior and posterior SCC in both ears – suggested involvement of the medial vestibular nucleus, which is a region vulnerable to thiamine deficiency - findings characteristic of the pattern of this encephalopathy[18].

During drug treatment, there was a marked improvement in VOR gain bilaterally, although still below the normality standard; in addition, corrective saccades and spontaneous nystagmus disappeared[18].

## Left inferior cerebellar peduncle glioma

Gliomas are tumors formed by glial cells, which are responsible for maintaining the neurons and are part of the CNS[25].

A study[25] documented, through otoneurologic tests and imaging exams, a case of progressive postural instability with no auditory symptoms or dizziness complaints. The vHIT showed reduced VOR gain only in the posterior and lateral SCC, with lower values in the latter, and the presence of covert and overt catch-up saccades in both ears. Cranial MRI showed a lesion in the left cerebellar peduncle, suggestive of glioma.

According to those authors, the results of the vHIT were due to the neurological change found, since no impairment of peripheral end organs (such as viral vestibular neuritis) could be responsible for the hypofunction of the anterior CNS without influencing the lateral SCC, considering that the superior vestibular nerve maintains both structures[25].

## Progressive supranuclear palsy

Progressive supranuclear palsy (PSP) is a degenerative disease that affects the basal ganglia and the brainstem[11].

A study[11] performed the vHIT in PSP patients and controls to investigate vestibular function. Only the lateral SCC was assessed. The individuals presented no vestibular symptoms.

There was no evidence of change in VOR gain in the lateral SSC in this population ($p$<0.01). The results were suggestive of the involvement of areas not associated with the VOR. Saccadic pursuit was the only altered aspect. However, the vertical SCC, which could bring some other indication of vestibular impairment in PSP, was not tested. Patients with PSP often exhibit normal responses on the vHIT, even in severe cases[11].

## Mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke

Mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke – MELAS syndrome – is caused by a mutation in mitochondrial DNA (deoxyribonucleic acid), and is mainly manifested as hearing loss[13].

Only one study[13] reported the application of the vHIT in individuals with MELAS syndrome and identified sensorineural hearing loss bilaterally in all participants. Of the 42 SCC assessed, changes in the VOR were detected in only 15 (nine lateral, five posterior, and one anterior), and 13 of them showed the presence of covert and overt catch-up saccades. In addition, it was verified that the lateral SCC had lower VOR gain values, except in one case, which presented lower values in the posterior SCC.

Those authors informed that the discrepancy between the results of the lateral and anterior SCC - both innervated by the superior vestibular branch, suggests specific involvement of the peripheral structures and not in the neurological scope, which justifies the greater reduction in the VOR gain in the lateral SCC[13].

## Parkinson's disease

Parkinson's disease (PD) is a common neurodegenerative disease characterized by muscle rigidity, tremors, and postural instability[16].

A study[16] performed the vHIT in patients with and without PD to evaluate the clinical applicability of this test as an ancillary tool in the diagnosis of this disease, especially in its early stages.

Results within the normality standards were found in both groups ($p$<0.05); thus, the vHIT does not apply to the proposed objective. However, no oculomotor tests were performed. Individuals with early-stage PD presented significantly higher VOR gain values compared with those of the second group. Those authors suggested that these findings may be a consequence of probable vestibular decompensation with disease progression[16].

When an increased VOR gain is obtained, the possibility of deficient calibration or excessive proximity to the fixation target should always be considered, which shows the importance of maintaining a minimum distance of 1 m between the individual and the target[3].

Nonetheless, another explanation for an increased VOR gain is the possible hypersensitivity of the vestibular system of the patient, from the abnormally high perception of the rotation movements performed by the head. Perception of this movement is an ability that can be affected only at higher processing levels, that is, it is indicative of involvement of the central vestibular pathways[30].

## Spinocerebellar ataxia types 1, 2, and 3 and Friedreich's ataxia

Spinocerebellar ataxias are considered conditions difficult to differentiate from each other only by neurological signs and symptoms. As genetic diagnosis is a time-consuming and costly process, clinical signs that can assist in discriminating between the different types of ataxia have been sought[15].

With this objective, a study[15] assessed the VOR by the vHIT in patients with spinocerebellar ataxia types 1 (SA1) (n=4), 2 (SA2) (n=4), and 3 (SA3) (n=15) and Friedreich's ataxia (FA) (n=9), as well as in healthy controls (n=40). Normal VOR gain values were found in all SCC in SA2 patients and the control group. Patients with SA1, SA3, and FA showed reduced VOR gain, bilaterally only in the last two groups. Covert catch-up saccades were identified only in SA3 patients, whereas overt saccades were found in all types of ataxia.

## General remarks

According to the authors of all the studies included in this literature review, these results were attributed to CNS dysfunction resulting from changes in each type of ataxia, since other tests did not detect functional or structural abnormality in the peripheral auditory, vestibular and oculomotor pathways and the cranial nerves[15].

The VOR gain showed a significant negative correlation with disease severity ($p<0.01$), which increases the possibility of using the gain of this reflex as a neurophysiological biomarker for disease evolution, as was recently suggested for SA3. The results demonstrate that the vHIT provides phenotypic markers that discriminate between these ataxias and can serve as a strategy to guide genetic diagnosis[15].

As shown in the selected scientific literature, regarding the applicability of the vHIT[8-25], this test has been used in CNS disorders for three main purposes: to investigate the vestibular function, since most of these diseases manifested with vestibular symptoms; consider high-frequency VOR assessment as a complementary measure to assist with the identification and characterization of the underlying disease, because neurological examinations are expensive and time-consuming compared with the vHIT, and given that this is a rapid, objective, non-invasive test that offers immediate results; monitor clinical evolution and treatment, especially at pre- and post-intervention.

The vHIT is highlighted as a comfortable test, as it is performed under physiological conditions[1]. For a long time, studies have considered this diagnostic modality as a tool that enabled only analysis of the peripheral vestibular system; however, as observed in the present study, this instrument, together with oculomotor tests, enables the investigation of how CNS disorders act on the central vestibular system and reflect on VOR functioning.

The vHIT measures the VOR gain in each SCC individually and is the first instrument capable of quantitatively analyzing the function of the posterior and anterior SCC. It also allows detection of both covert and overt catch-up saccades, which was not possible with other exams, since covert saccades occur during head rotation and have a short latency, which makes their visualization with the naked eye unfeasible[1,2].

The results verified in the studies included in this literature review[8-25] show that the VOR has presented changes. In some cases, the exams showed results characteristic of peripheral involvement, but when associated with the oculomotor tests performed, they indicated central involvement. However, in other conditions, there were findings suggestive of central involvement, such as VOR gain or mean gain values in the vertical SCC inferior to those in the lateral SCC[9,14,19], increased VOR gain[16], reduced VOR gain with disease severity in SA3[15], a cut-off point of 0.70 and gain asymmetry <20% to differentiate vestibular neuritis from a stroke in the medial branch of the posterior inferior cerebellar artery[12], normal VOR gain with altered oculomotor test results[8,11,17], presence of vertical spontaneous nystagmus[19,24], in addition to changes in the VVOR[19,22,24], VORS[21], saccadic pursuit[11,21] and skew deviation test[17] thus confirming that the VOR and its connections are affected by diseases of central origin. Physicians who assist this population should be aware of the importance of evaluating the systems involved in body balance, together with the clinical evaluation, the vHIT, and other exams necessary in each case.

In addition to some factors that can affect VOR functioning, such as age, the number and location of lesions can also influence the impairment of this reflex, which would also justify the intra- and inter-group variability found in the results of the studies in this literature review.

In most studies, it was verified that some oculomotor tests complementary to the VOR analysis were not carried out, such as the VVOR, VORS, skew deviation, and saccadic pursuit, which are used, mainly, when there is suspicion of changes at the the central level[4]. This absence can be justified by possible differences or limitations regarding the manufacturers of some of the equipment used.

All studies[8-25] presented incomplete data, which hindered the accurate characterization of the findings, such as the description of the VOR gain in all SCC and presence of nystagmus regarding type and direction, asymmetry values between the SCC and, about the characterization of the parameters of occurrence, latency, and amplitude of the catch-up saccades, also in each canal.

New studies, especially with larger sample sizes, should be conducted to enable the clinical application of the vHIT in individuals with these and other CNS disorders, aiming to allow statistical inferences of the greater strength of scientific evidence, which was not verified in the samples of the studies assessed in this literature review. Studies that relate vHIT findings with the results of imaging exams are also suggested, as well as the inclusion and characterization of clinical examinations of body balance.

## CONCLUSION

The vHIT is applicable in the assessment of high-frequency VOR in individuals with CNS disorders, as it has brought clinical evidence on changes in the peripheral and central vestibular functioning in different neurological conditions.

The following diseases were described: cerebellar stroke, MS, CANVAS, superficial siderosis of the CNS, Wernicke encephalopathy, left inferior cerebellar peduncle glioma, PSP, MELAS syndrome, PD, and spinocerebellar ataxia types 1, 2, and 3 and FA.

The results showed altered vHIT parameters in this population, suggestive of central involvement, evidenced by reduced VOR gain, mainly in the vertical SCC, and changes associated with oculomotor tests, confirming that the VOR and its connections are affected by diseases of central origin.

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