Vestibular Assessment & Management for Canadian Audiologists: A Scoping Review

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Stakeholder Acknowledgement

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Introduction

This document was prepared by a joint committee of audiologists representing both the Canadian Academy of Audiology (CAA) and Speech-Language and Audiology Canada (SAC).

The purpose of this scoping review is to create a framework for audiologists who perform vestibular diagnostic assessment and management in Canada. It outlines suggested knowledge requirements and provides direction to help guide practice. It is also meant to provide clarity to governments, university training programs, provincial/territorial associations and regulatory bodies, and other health-care professionals (such as physiotherapists, occupational therapists, otolaryngologists, and neurologists) of an audiologist's role in vestibular assessment and management.

An evidence-based process using clinical evidence, systematic reviews, and the clinical expertise of the committee was combined to develop this scoping review (The Joanna Briggs Institute, 2015). In cases where scientific data was inconclusive, the clinical expertise of the committee guided the development of consensus-based recommendations.

Professional Competence Statement

According to a recent 10-year survey assessing trends of audiologists' opinions and practice, audiologists are the professionals most qualified to conduct vestibular assessment (Nelson, Akin, Riska, Andresen, & Mondelli, 2016).

To perform vestibular testing and treatment, audiologists must obtain continuing education and hands-on experience beyond what is currently available through entry-level training. This training and mentoring may include independent study, online courses, job shadowing, and/or vestibular certification courses. The College of Speech and Hearing Health Professionals of BC requires an advanced competency certification in vestibular assessment and management to practice in British Columbia (2014).

Audiologists administering vestibular testing should ensure professional competence and "engage only in the provision of services that fall within their professional competence, considering their level of education, training, and recent experience and/or their access to professional supervision/assistance from qualified colleagues (Speech-Language and Audiology Canada, 2016a)."

Update & Review

It is recommended that the contents of this document be reviewed and updated every 5 years or as required based on substantial changes in research and practice in the area of vestibular assessment and management. As a scoping review, this document is intended to be used as an initial framework toward the development of a graded, evidence-based, systematically reviewed guideline for Canadian audiologists.

Disclaimer

While every effort has been made to ensure the accuracy of the content of this scope review, neither the authors, Speech-Language and Audiology Canada, nor the Canadian Academy of Audiology accept any liability with respect to loss, damage, injury, or expense arising from any errors or omissions in the contents of this work.

Background

Vestibular Statistics

The United States' National Institute on Deafness and Other Communication Disorders (NIDCD) states that vestibular dysfunction is common among adults (NIDCD, 2006). According to data from the 2001–2004 National Health and Nutrition Examination Survey, 35% of Americans aged 40 years and older (69 million individuals) had objective evidence of vestibular dysfunction (Agrawal, Carey, Della Santina, Schubert, and Minor, 2009). Vestibular disorders can affect the peripheral vestibular system, the central vestibular system, or a combination of both. A literature review reported that 44% of patients with dizziness complaints presenting to primary-care offices, emergency rooms, and referral clinics suffered from peripheral vestibulopathy and in 11% of cases, the patients' dizziness was attributed to central vestibulopathy (Kroenke, Hoffman, & Einstadter, 2000).

Examples of peripheral disorders include benign paroxysmal positional vertigo (BPPV), Ménière's disease, vestibular neuritis and labyrinthitis, vestibular schwannoma, perilymphatic fistula, superior semicircular canal dehiscence syndrome, and the effects of trauma. Central disorders include vestibular migraine, brainstem or cerebellar stroke, and vertebrobasilar insufficiency.

Neuhauser and Lempert (2009) reported that the top two most common vestibular disorders are BPPV and vestibular migraine. Von Brevern et al. (2007) reported that BPPV accounts for 8% of individuals with moderate or severe dizziness/vertigo. The lifetime prevalence of BPPV is 2.4%, the 1-year prevalence is 1.6%, and the 1-year incidence is 0.6%. On multivariate analysis, age, migraine, hypertension, hyperlipidaemia, and stroke were reported to be independently associated with BPPV. Osteopenia has also been found to be associated with BPPV (Yu, Liu, Cheng, & Wang, 2014).

Vestibular migraine accounts for 6 to 7% of patients in neurologic dizziness clinics and has been found in 9% of patients in a migraine clinic case series (Neuhauser, Leopold, von Brevern, Arnold, & Lempert, 2001; Dieterich & Brandt, 1999).

Regarding Ménière's disease, studies conducted in Finland, the United Kingdom, and the United States between 1970 and 2000 estimated the prevalence to be 0.043%, 0.20%, and 0.22%, respectively (Kotimäki, Sorri, Aantaa, & Nuutinen, 1999; Minor, Schessel, & Carey, 2004; Wladislavosky-Waserman, Facer, Mokri, & Kurland, 1984).

With respect to children, Gioacchinia, Alicandri-Ciufellia, Kaleci, Magliulio, and Re (2014) reviewed 10 articles comprising a total of 724 subjects. Overall, benign paroxysmal vertigo of childhood (18.7%) and vestibular migraine (17.6%) were the two main entities connected with

187 vertigo and dizziness. Head trauma (14%) was the third most common cause of vertigo. The
188 authors advise that when evaluating a young patient with vertigo and dizziness, clinicians
189 should be aware the symptoms are often connected to different pathologies in comparison to
190 the entities observed in the adult population.
191

Consequences of Vestibular Dysfunction

193 Symptoms from vestibular dysfunction can range from mild to severe and may include
194 imbalance or unsteadiness, vertigo, light-headedness, visual disturbances, nausea, headaches,
195 muscular aches in the neck and back, motion intolerance, and problems with concentration and
196 memory. Vestibular dysfunction can occur in conjunction with hearing loss, tinnitus, aural
197 fullness, sensitivity to pressure changes, and sensitivity to loud sounds. Vestibular disorders are
198 linked to an increased incidence of falls, psychological and psychiatric disturbances, panic
199 disorders, and cognitive impairment (Mira, 2008). Mira (2008) reported that the main goals of
200 vestibular disorder treatments are controlling symptoms, reducing functional disability, and
201 improving quality of life.
202

203 Among community-dwelling older adults, the risk of falling is 3–4 times higher among people
204 with muscle weakness or gait and balance disorders (Stevens, Corso, Finkelstein, & Miller,
205 2006). The Public Health Agency of Canada (2014) reports that falls account for more than half
206 of all injuries among Canadians aged 65 years and over. One third of community-dwelling
207 Canadian seniors experience one fall each year, and half of those will fall more than once. The
208 likelihood of dying from a fall-related injury increases with age. Among seniors, 20% of deaths
209 related to injury can be traced back to a fall (Public Health Agency of Canada, 2011). Falling is
210 associated with morbidity, reduced functioning, and premature nursing home admissions
211 (American Geriatrics Society, 2011). Non-injurious falls can also have serious consequences,
212 such as a fear of falling, self-imposed activity restriction, and further functional decline (Rawski,
213 1998). Other risk factors include a history of falls, lower extremity weakness, cognitive
214 impairment, neurological impairment, co-morbidity, altered elimination, arthritis, and/or use of
215 certain medications, especially psychotropic drugs (Leipzig, Cumming, & Tinetti, 1999; Gispen,
216 Chen, Genther, & Lin, 2014). Another goal of vestibular assessment and management is
217 reducing the burden of fall-related injuries and possible death for those at risk.

218 Patients with hearing loss may also be at risk for reduced physical activity. Gispen et al. (2014)
219 found that moderate or greater hearing impairment in older adults is associated with lower levels
220 of physical activity, independent of demographic and cardiovascular risk factors (Public Health
221 Agency of Canada, 2014). This is important for audiologists to take into consideration when
222 managing patients with hearing loss and assessing their risk of fall.

Population Statistics

224 In 2015, for the first time in Canadian history, older seniors (persons aged 65 years and older)
225 outnumbered those under 15 years of age (Canadian Medical Association, 2016). The
226 proportion of older seniors among the total senior population is expected to increase from
227 26.6% in 2013 to 39.4% in 2045. By 2063, the number of Canadians aged 80 years and over is
228 expected to reach nearly 5 million, compared with 1.4 million in 2013 (Statistics Canada, 2014).
229 The high occurrence of vestibular dysfunction combined with a projected increase in the senior
230 population in Canada highlights the importance of vestibular assessment and management
231 training in university programs, sufficient numbers of audiologists across the country providing

232 these services, and access to ongoing continuing education opportunities for practicing
233 audiologists.

Interprofessional Team Approach

235 An interprofessional team approach to the management of dizzy and unsteady patients
236 improves patient coping, functionality, and satisfaction, and decreases overall health-care
237 utilization in vestibular patients (Naber et al., 2011). Members of the team may include (but are
238 not limited to): primary care physicians, otolaryngologists, neuro-otologists, neurologists,
239 audiologists, physiotherapists, occupational therapists, optometrists, pharmacists, psychiatrists,
240 physiatrists, cardiologists, and psychologists. All professionals who specialize in vestibular
241 assessment and management must understand the role and referral process of each team
242 member, establish a cooperative approach that relies on the sharing of information, and
243 recognize when a referral to (an) appropriate team member(s) may be required.

Patient Safety

245 The SAC Code of Ethics Standard 4, "Safety," states:

246　　"Members and associates shall:

247　　　• Take every precaution to avoid harm to patients or clients. This includes following
248　　　　applicable occupational health and safety and infection prevention and control practices,
249　　　　and ensuring that equipment is appropriately calibrated and in proper working order.
250　　　• Ensure that their employees and/or supervised personnel comply with relevant
251　　　　occupational health and safety and infection control policies and procedures (SAC,
252　　　　2016a)."
253
254 For vestibular assessment and management, the following patient safety measures should be
255 addressed:
256
257　　1. Follow proper infection control precautions as outlined in *The Infection Prevention and*
258　　　　*Control Guidelines for Audiology*, written by the Canadian Interorganizational Steering
259　　　　Group for Speech-Language Pathology and Audiology (2010).
260　　2. Advise patients appropriately to stop certain medications (e.g., vestibular sedatives) at
261　　　　least 48 hours before undergoing vestibular testing. It is important that patients are
262　　　　advised to consult with their referring physician regarding these medications. The
263　　　　referring physician should advise the patient which medications should be stopped and
264　　　　the audiologist should verify that the patient has adhered to this advice prior to vestibular
265　　　　testing (British Society of Audiology, 2014).
266　　3. Stop testing and/or treatment if there are any complicating or medical contraindications.
267　　4. Obtain informed consent prior to vestibular testing or treatment, with information given
268　　　　about the specific known risks of each test. Patients should be informed that vestibular
269　　　　testing may temporarily cause an increase in their imbalance. It is recommended that
270　　　　patients come to their appointments accompanied by someone to reduce the risk of fall
271　　　　and any difficulty driving following testing (Accreditation Canada, Canadian Institute for
272　　　　Health Information, & Canadian Patient Safety Institute, 2014).
273　　5. Create a falls prevention strategy to minimize patient injury from falls in the workplace.
274　　　　The Required Organizational Practice (ROP)'s "Falls Prevention Strategy" requires
275　　　　clinics to implement and evaluate a falls prevention strategy. All populations at risk must

7

276 be identified, the specific needs of the populations at risk must be addressed, and
277 measures must be taken to evaluate and provide ongoing improvements to the falls
278 prevention strategy on an ongoing basis. A safety risk assessment should be made for
279 each patient at the beginning of service and should include (Accreditation Canada et al.,
280 2014):

281   a. a review of internal and external physical environments
282   b. medical conditions requiring special precautions
283   c. information sharing with team partners who may be involved in planning
284   of care
285   d. regular updates and improvements to the safety risk assessment
286   e. education of patients and their families on home safety issues identified in
287   the risk assessment

Current Status of Canadian Audiology Scope of Practice & Certification

Provincial/Territorial

291 A committee review of Canadian provincial/territorial professional associations and/or regulatory
292 bodies revealed no specific scopes of practice or guidelines for vestibular practice.
293
294 The College of Speech and Hearing Health Professionals of BC is the only regulatory board to
295 date requiring audiologists to obtain an Advanced Competency Certificate in order to perform
296 vestibular assessment and management. It involves successfully completing a graduate-level
297 course of at least four credit hours in vestibular system function and assessment or a program
298 of study that the committee agrees is similar to a graduate-level course (College of Speech and
299 Hearing Health Professionals of BC, 2014). An active registrant must be certified by the
300 College's registration committee (College of Speech and Hearing Health Professionals of BC,
301 2014).
302
303 For a listing of general requirements to obtain vestibular certification for the College of Speech
304 and Hearing Health Professionals of BC, please refer to:
305 http://www.cshhpbc.org/docs/cshhpbcbylaws.pdf
306

National

308 Both the Canadian Academy of Audiology (CAA) and Speech-Language and Audiology Canada
309 (SAC) include vestibular assessment and management within their scope of practice documents
310 and position statements for the field of audiology.
311
312 The CAA *Position Statement on Audiology Scope of Practice* (2002) states:
313   • An audiologist is an independent, professional provider of
314   primary hearing health care, who specializes in the prevention
315   of hearing loss and in the identification, assessment, diagnosis,
316   management, and treatment of hearing and balance disorders.

- The central focus of the profession of audiology is on human hearing, both normal and impaired, and its relationship to disorders of communication. Because of their potential relationship to auditory impairments, a secondary focus of audiology is on vestibular or balance disorders.
- Assessment of the vestibular system includes administration and interpretation of clinical and electrophysiologic tests of equilibrium. Assessment is accomplished using standardized testing procedures and appropriately calibrated instrumentation. Interpretation of test results may include diagnostic statements as to the site of lesion within the vestibular system, and/or the probable etiology of the impairment.
- Audiologists are also involved in the rehabilitation of persons with vestibular disorders. They participate as members of vestibular rehabilitation teams to recommend and carry out goals of vestibular rehabilitation therapy including, for example, habituation exercises, balance retraining exercises, and general conditioning exercises.

To review the complete CAA *Position Statement on Audiology Scope of Practice* please refer to: https://canadianaudiology.ca/professional-resources/scope-of-practice/

SAC's *Scope of Practice for Audiology* (2016b) states:
- Assessment of hearing, vestibular function and balance, which may involve screening, identification, evaluation, diagnosis, and counselling.
- Intervention for hearing, vestibular, and balance disorders, which may involve promotion, prevention, counselling, treatment, consultation, management, (re)habilitation, and education.
- Measurement of patient or client outcomes for these services.
- Consultation with and referral to other professionals.
- Clinical areas of service may include, but are not limited to:
  - vestibular and balance dysfunction; and the impacts of these conditions on everyday life.
- Assessment, selection, prescribing/recommending, dispensing, validation, verification, servicing, and development of hearing aids and other appropriate hearing assistive and (re)habilitative strategies for individuals with hearing impairment, auditory processing, balance dysfunction, tinnitus, and/or related disorders. This could include cochlear implants, other implantable hearing devices, assistive technology such as FM systems, speech reading classes, tinnitus re-training, and vestibular (re)habilitation as well as measurement of patient or client outcomes for these technologies and strategies.
- Prevention, counselling, and education services to patients or clients, families, caregivers, other professionals, and the public regarding all aspects of hearing and balance function.
- Advocacy on behalf of individuals with auditory disorders, balance disorders, and other related disorders and populations that are at risk.
- University and/or college education and training pertaining to hearing, vestibular, balance, and other related disorders.
- Research in hearing, vestibular function, balance, and other related areas.

368

369 To review the complete SAC *Scope of Practice for Audiology* please refer to: http://www.sac-
370 oac.ca/sites/default/files/resources/scope_of_practice_audiology_en.pdf?_ga=1.239600256.208
371 4794186.1403892485
372

Knowledge Required

374 As mentioned above, audiologists should obtain additional and ongoing education and training
375 and, in British Columbia, must obtain an Advanced Competency Certificate in vestibular
376 assessment and management.
377 The purpose of this section is to provide the audiologist with guidance on the knowledge
378 required to practice in the area of vestibular assessment and management.
379
380 The audiologist should gain:
381 • knowledge of anatomy and physiology of the peripheral and central vestibular systems
382   and their connections with the hearing, visual, and somatosensory systems;
383 • knowledge of pathophysiology of vestibular and balance disorders (otologic and non-
384   otologic);
385 • experience obtaining relevant case history;
386 • knowledge of self-report measures such as the Dizziness Handicap Inventory (Jacobson
387   & Newman, 1990);
388 • the ability to identify contraindications to vestibular assessment or portions thereof;
389 • understanding of the effects of different medications and substances on test results;
390 • knowledge and experience in administering and interpreting various vestibular
391   assessment techniques, including but not limited to:
392   o bedside tests of vestibular function, such as head impulse testing, oculomotor
393   testing (gaze-evoked nystagmus, saccades, optokinetics, and tracking), head
394   shake testing, hyperventilation testing, closed glottis straining, and external canal
395   pressurization (fistula test);
396   o pre-testing screenings, such as vertebral artery screening test and cervical
397   vertigo test;
398   o subjective visual vertical test;
399   o dynamic visual acuity test (DVAT);
400   o video head impulse test;
401   o electronystagmography (ENG) or videonystagmography (VNG), including
402   oculomotor testing, positioning and positional testing, and caloric testing (air,
403   water, and/or ice water);
404   o electrophysiological tests such as electrocochleography and vestibular evoked
405   myogenic potentials (cervical and ocular);
406   o postural stability tests and computerized dynamic posturography
407   o autorotation and rotary chair tests
408 • knowledge of repositioning maneuvers for treatment of different forms of benign
409   paroxysmal positional vertigo;
410 • understanding of vestibular rehabilitation therapy: identifying candidates and providing
411   management strategies and available resources;
412 • the ability to identify contraindications for vestibular rehabilitation therapy;
413 • the ability to identify clients who are at risk for falls and provide management strategies
414   and resources;
415 • understanding of outcome measures and monitoring therapy progress;

- knowledge of medical and surgical treatment for otologic vertigo and balance disorders;
- the ability to recognize the need for referral to medical specialists or other professionals; and
- the ability to recognize complications and knowledge to deal with them.

Vestibular Assessment

Case History

Dizziness is the third most common presenting complaint in outpatient clinics (Bennett & Jacobson, 2007). For most people it is a sensation difficult to describe and can encompass a wide range of symptoms, such as light-headedness, unsteadiness, imbalance, rocking, swaying, and vertigo. Vertigo is defined as an illusory perception of movement (rotatory or non-rotatory) of self and/or the surroundings (Brandt, Dieterich, & Strupp, 2013). True rotatory vertigo is traditionally associated with unilateral inner ear pathology.

Obtaining a detailed case history is an essential part of the assessment and management of vestibular disorders. It allows the clinician to formulate a working hypothesis of the origin of the complaints, which will be either confirmed or rejected based on the results of the quantitative testing (Bennett & Jacobson, 2007). It is important to remember that some patients have more than one cause of their dizziness (e.g., Ménière's Disease and BPPV).

Given the natural complexity of vestibular disorders and the variety of presenting symptoms, it may be helpful for the clinician to use facilitating tools such as questionnaires and pre-defined interview questions. Patients may need to be guided to stay on topic during a case history interview.

The following aspects are suggested as the most helpful in differentiating the various vestibular/balance disorders (Brandt et al., 2013):

Type of dizziness: It is important to first allow patients to describe their experiences without using suggestive vocabulary and directive questions. As most patients will likely use vague terms such as dizzy, off-balance, and light-headed, the clinician may ask the patient for comparisons with previous experiences such as being on a merry-go-round or standing on a pier. Often, patients will gesture while describing their dizziness, and this may also be helpful to the clinician in clarifying the type of dizziness experienced.

Table 1: Examples of syndromes presenting with vertigo and light-headedness (adapted from Brandt et al., 2013)

Syndromes presenting with vertigo	Syndromes presenting with light-headedness
BPPV, vestibular migraine, acute vestibular neuritis, viral labyrinthitis, Ménière's disease	heart disease, orthostatic dysregulation, vasovagal attacks, (pre)syncope, hyperventilation syndrome, panic attacks, phobic postural vertigo, electrolyte disorders, hypoglycemia, intoxications, medications

453 **Duration of dizziness:** The clinician should at first determine whether the dizziness symptoms
454 are constant or intermittent/episodic in nature. If the patient reports episodic vertigo, the duration
455 may be described as lasting seconds to minutes, minutes to hours, days to weeks, or months to
456 years.
457

458 **Table 2: Examples of vertigo syndromes according to duration of symptoms (adapted**
459 **from Bennett & Jacobson, 2007 and Brandt et al., 2013)**

Lasting seconds to minutes	Lasting minutes to hours	Lasting days to weeks
BPPV, vestibular paroxysmia, perilymphatic fistula, third window lesions	Ménière's disease, vestibular migraine, transient ischemic attacks, panic attacks	acute vestibular neuritis, viral labyrinthitis, multiple sclerosis

460
461 **Triggering or exacerbating factors and improving factors:** It is important to distinguish
462 spontaneous attacks of vertigo from those triggered by movement (of self, such as walking, or of
463 the environment, such as watching traffic), specific changes in position, loud sounds, cough,
464 physical exertion, diet, and environmental factors. Spontaneous vertigo may occur in Ménière's
465 disease and vestibular migraine. Vestibular neuritis is not classically associated with attacks, but
466 rather a single attack that persists. An understanding of vestibular compensation,
467 decompensation, and failure to compensate is important when examining improving factors.
468

469 **Table 3: Examples of triggering factors and possible associated vertigo syndromes**
470 **(adapted from Bennett & Jacobson, 2007 and Brandt et al., 2013)**

Trigger	Possible cause
changes in head position relative to gravity	BPPV
changes in cerebrospinal fluid (CSF) pressure (coughing, Valsalva) combined with dizziness or nystagmus induced by loud sounds (Tullio's phenomenon)	perilymphatic fistula/inner ear bone dehiscence
walking (oscillopsia)	bilateral vestibular hypofunction
lateral neck extension	vertebrobasilar insufficiency/ vestibular paroxysmia
moving from sitting or lying down to upright	orthostatic hypotension

471
472 **Accompanying symptoms:** Symptoms may originate from the inner ear (hearing loss, tinnitus,
473 aural fullness, oscillopsia), the central nervous system (double vision, paresthesias, disorders of
474 swallowing, speaking, movement) or migraine typical symptoms (photo and phonophobia, visual
475 auras, headache). Nausea and vomiting are commonly reported accompanying symptoms and
476 should always be noted when present.
477

478 **Table 4: Examples of accompanying symptoms and possible associated vertigo**
479 **syndromes (adapted from Bennett & Jacobson, 2007 and Brandt et al., 2013).**

Associated symptoms	Possible cause
Hearing loss, aural fullness, tinnitus with autophony and hyperacusis	Ménière's Disease Third Window Lesion
Headaches, auras, phono- and/or photophobia	Migraine
Pain, otorrhea	Otologic disease
Facial weakness, ataxias, paresthesias	Stroke/Tumour / Multiple Sclerosis / Head Trauma
Oscillopsia	Migraines / Bilateral Vestibular Hypofunction

480
481 In addition to the specific description of the symptoms as described above, the case history
482 should also include questions regarding:

483
484 **Medical history:** Determine if there is a positive history of cardiovascular diseases, diabetes,
485 neurological diseases (seizures, strokes, migraines, multiple sclerosis), migraines, mental
486 health issues (anxiety, depression), falls, head and/or neck problems and injuries, motion
487 sickness, vision problems/eye surgeries, and ear disease and surgery. Family history of
488 neurological and otological disorders should also be investigated.

489
490 **Medications/drugs:** Compile a list of current and past medications. The use of recreational
491 drugs and alcohol should also be included in the case history. Particularly of interest are
492 ototoxic medications (antibiotics, diuretics), central nervous system–acting medications
493 (antidepressants, antianxiety and anti-seizure drugs, sedatives), chemotherapeutic agents, anti-
494 hypertensive medications, anti-inflammatories and antihistamines.

495 Oculomotor Testing

496 Definition

497 A normally functioning vestibular system is not only reliant on a healthy peripheral system, but
498 also on central nervous system (CNS) structures such as the brainstem, cerebellum, and
499 cerebrum. These centres can be evaluated with oculomotor testing, which is partly based on the
500 functioning of the vestibulo-ocular reflex (VOR). The VOR maintains an image on the fovea
501 during head movements by making rapid compensatory eye movements in response to
502 vestibular input. Therefore, the eyes are the most direct means available for evaluating the
503 vestibular system.

504
505 There are four principal oculomotor tests used in the routine clinical evaluation: gaze stability,
506 saccade testing, smooth pursuit tracking, and optokinetic nystagmus. There is overlap in the
507 neural pathways involved in these tests. Though the individual tests can be used to indicate a
508 CNS involvement, they may not point to a specific site of lesion; however, some combination of
509 results may suggest a lesion in a specific region of the brainstem or cerebellum (Shepard,
510 Schubert, & Eggers, 2016).

511
512 Eye movements can be recorded using video (known as videonystagmography or VNG) or
513 electrodes (electronystagmography or ENG). As VNG is the industry standard, ENG will not be
514 covered here.
515

A. Gaze Stability

517 **Procedure**
518 This test evaluates a patient's ability to maintain a gaze fixed on a target while looking straight
519 ahead (primary position), 30° to the right and left, and 20° up and down, without any abnormal
520 eye movements (such as nystagmus). If nystagmus is present, the response must be tested
521 without visual fixation to differentiate between a peripheral and central site of lesion.
522
523 **Interpretation**
524 Any nystagmus evoked must be recorded in terms of its direction (of the fast-phase), velocity,
525 evoking position(s) and whether the nystagmus follows Alexander's Law (i.e., does the velocity
526 of the nystagmus increase when the gaze is to the same direction as the fast-phase of the
527 nystagmus?). Nystagmus seen in the primary position is referred to as spontaneous nystagmus;
528 nystagmus in the lateral or vertical positions is known as gaze-evoked nystagmus.
529
530 **Clinical use**
531 Abnormalities may be caused by a peripheral or central vestibular dysfunction (Shepard &
532 Schubert, 2016). Nystagmus may be considered peripheral if the nystagmus is stronger without
533 fixation, is direction-fixed, and has a primarily horizontal component. If the lesion is peripheral,
534 the nystagmus should also follow Alexander's Law (as described above). The nystagmus
535 usually beats away from the involved side, but in some unusual cases (such as in the irritative
536 phase of Ménière's disease or with hyperventilation with retrocochlear pathology) the
537 nystagmus can beat towards the involved side.
538
539 Primary position nystagmus of central origin may be purely vertical or purely torsional; it is rarely
540 horizontal. Central nystagmus is more likely to be direction-changing (e.g., right-beating with
541 right gaze, left-beating with left gaze, etc.) than direction-fixed, but both are possible, and it does
542 not increase in velocity with fixation removed (Shepard et al., 2016). Horizontal or pure torsional
543 gaze-evoked nystagmus of central origin is usually caused by a lesion in the brainstem. Vertical
544 gaze-evoked nystagmus can result from lesions in the brainstem or cerebellum.
545
546 Saccadic intrusions and oscillations may be recorded during gaze testing, especially with
547 primary gaze testing. These are seen with fixation present and are suggestive of cerebellar
548 and/or brainstem involvement.
549

B. Saccades

551 **Procedure**
552 This test assesses the ability to move the eyes rapidly from one target to another in one
553 movement. Targets are presented at random time intervals and random locations (within a
554 horizontal 30–40º arc), usually by a computerized system. The patient must be instructed to
555 keep his head still while keeping his eyes on the target as closely as possible. The test should
556 be repeated with reinstruction if results are abnormal.
557

Interpretation

Saccades are assessed by the velocity of the eye movements, the accuracy of each movement (i.e., are there undershoots or overshoots?), and the delay in the onset of the eye movement compared to the target (i.e., latency). Look for the patient's best performance; abnormalities should be consistent (Barin, 2011). Age-related norms are not required for this test. If latencies appear to be unusually short, the patient may be anticipating target movements and should be re-instructed.

Clinical use

Abnormalities in saccadic testing are caused most commonly by pathologies in the brainstem or cerebellum, and less commonly by pathologies in the cerebrum, nerves, or muscles involved in eye movements (Barin, 2011; Hain, 1997b). However, bilateral low velocities, undershoots, and large latencies can be caused by poor vision, fatigue, inattention, and/or medication (see the limitations section below). Asymmetrical velocity is often suggestive of an ocular disorder such as internuclear ophthalmoplegia (INO). INO, which is caused by lesions in the medial longitudinal fasiculus, is indicated if adduction movements are slower than abduction movements. A unilateral delayed latency can suggest a lesion in the cerebrum. Bilaterally prolonged latencies are of limited value because of the influence of the patient's mental state. Poor accuracy can suggest a lesion in the vestibulocerebellum, but can also be due to basal gangliar lesions or ocular disorders such as INO (Barin, 2011; Hain, 1997b).

C. Smooth Pursuit (Tracking)

Procedure

Smooth pursuit assesses the ability to maintain a smoothly moving target on the fovea (as opposed to using a series of saccades). The target movement can be of fixed velocity or varying velocity (e.g., with a sinusoid). Sinusoidal testing (the more commonly used method) involves a target moving 15–20º from the centre in side-to-side movements along a light bar or projection system. The frequency of the target should be increased between 0.2–0.7 Hz in order to challenge the central nervous system.

As with saccade testing, pursuit should be repeated with reinstruction if the results are abnormal, as the mental state of the subject can greatly influence the results (e.g., inattention).

Interpretation

Visual pursuit is assessed using gain, asymmetry, and phase. The gain is the ratio of the eye velocity by the target velocity. Asymmetry is the percentage difference in velocity gain between the rightward and leftward eye movements. Phase is a measure of how much the eye is leading or lagging behind the target. Note that if the eye is leading the target then either the eyes' movements are saccadic or the patient needs to be re-instructed because they are anticipating the target movements. Pursuit gain is sensitive to age; therefore, age-based norms must be used.

Clinical use

Pursuit disorders are usually non-specific (Hain, 1997b). Smooth pursuit is heavily influenced by the patient's mental state, age, and/or medications. Abnormalities in gain often suggest a vestibulocerebellar pathology; however, lesions in the cortex or brainstem may also result in reduced gain. In cases of unilateral reduced gain (i.e., abnormal asymmetry), the abnormality is usually towards the lesioned side (Shepard et al., 2016). Some patients with vestibular migraine

606 may also have saccadic pursuit, particularly if symptoms have been longstanding (Neugebauer,
607 Adrion, Glaser, & Strupp, 2013).
608

D. Optokinetic Nystagmus (OKN)/Optokinetic After-Nystagmus (OKAN)

Procedure

611 Optokinetic nystagmus (OKN) measures nystagmus created by tracking repeated moving
612 objects across the patient's visual field. The stimuli must fill at least 80–90% of the patient's
613 visual field; therefore, an optokinetic projector is the preferred stimulus delivery equipment
614 (Shepard, 2009; Hain, 1997a). Many systems use a light bar or small projection system, which
615 is insufficient for true testing. With these systems, the testing serves primarily as a cross check
616 for smooth pursuit testing results. Stimulation velocities vary from 30–60 deg/s. If the lights are
617 turned out after at least 10 seconds, optokinetic after-nystagmus (OKAN) can continue for about
618 30 seconds. While abnormal OKAN can suggest a lesion in the vestibular nuclei in the pons,
619 due to its lack of sensitivity and specificity, it is not typically part of the routine oculomotor
620 assessment (Shepard et al., 2016).
621

Interpretation

623 OKN is assessed using gain (the ratio of eye slow-phase velocity to field velocity). Gain
624 decreases as a function of age. Age-related norms have been developed and are applied to the
625 test results.
626

Clinical use

628 Though less sensitive to the patient's mental state than pursuit, OKN is also a less sensitive test
629 because it involves two distinct tracking systems—the smooth pursuit system and the
630 optokinetic system (Hain, 1997b). Asymmetrical gain in OKN with normal pursuit gain suggests
631 a bias of the nystagmus due to unilateral peripheral hypofunction (Shepard et al., 2016). When
632 asymmetrical gain reflects a true central nervous system abnormality, abnormal results in
633 saccadic or pursuit testing would be expected. OKN is considered the least useful of the
634 oculomotor tests (Shepard et al., 2016).
635

Considerations & Limitations of Oculomotor Testing

637 Poor calibration will affect all test results in the battery. Abnormally fast saccades and
638 unmatched eye and target peaks in smooth pursuit are just two of the abnormal results obtained
639 with a poor calibration (Barin, 2006).
640

641 Spontaneous nystagmus visible with fixation may make it difficult to interpret the oculomotor
642 findings. Note that even a mild spontaneous nystagmus can result in asymmetrical smooth
643 pursuit and OKN (Hain, 1997b).
644

645 Nystagmus is difficult to evaluate in patients who blink repeatedly. In some cases re-instruction
646 helps, but not all. The audiologist must verify that abnormally fast saccades and/or poor
647 saccadic accuracy are not due to blinks.
648

649 Torsional nystagmus cannot be adequately measured on the horizontal or vertical nystagmus
650 tracings, and therefore recordings must be viewed either in real time or recorded and reviewed
651 to verify that no torsional nystagmus was evoked.
652

653 Square wave jerks (in which the eyes briefly move away from the target and return) will be seen
654 in some patients and is usually normal, especially in older patients. Normal square wave jerks
655 should occur less than 20 to 30 times per minute, be at least 200 ms apart, and be between 1
656 and 5º in amplitude. Square waves that occur more often suggest a cerebellar or brainstem
657 lesion. A high amplitude (above 5º) suggests a brainstem or cerebellum pathology (Shepard et
658 al., 2016).
659
660 Certain medications, such as sedatives, anti-nausea medication, antihistamines, diuretics,
661 tranquilizers, and antidepressants, can result in abnormal nystagmus or suppressed nystagmus.
662 Please see Cass and Furman (1993) for details.

663 **Contraindications to Oculomotor Testing**
664 There are few contraindications to oculomotor testing. The patient must be able to see the
665 target without glasses (but may wear contacts) and be able to tolerate the VNG goggles. Dark
666 eye makeup may result in inaccurate pupil tracking and should be removed.
667
668 Ocular motility disorders (e.g., myopathy, palsies) will affect the accuracy of the testing, as will
669 disconjugate eye movements (e.g., intranuclear ophthalmoplegia, strabismus, amblyopia).

670 Positioning Tests

671 Definition

672 Positioning maneuvers are used to test the posterior, anterior, and horizontal semicircular
673 canal(s) (SCC) for displaced canaliths, such as displaced otoconia or cupuliths in benign
674 paroxysmal positional vertigo (BPPV. They differ from positional evaluations because they
675 require active transition from one position to another.
676
677 BPPV is the most common cause of vertigo in patients with vestibular disorders (Blatt,
678 Georgakakis, Herdman, Clendaniel, & Tusa, 2000). BPPV is thought to occur when calcium
679 carbonate crystals, referred to as otoconia, dislodge from the otoliths and enter one of the
680 SCCs. The displaced otoconia are more commonly free-floating, which is referred to as
681 canalithiasis; however, they can also adhere to the cupula, which is referred to as
682 cupulolithiasis. Due to the anatomical location of the posterior SCC, displaced otoconia more
683 commonly enter the posterior SCC; however, there are cases of BPPV involving the horizontal
684 and anterior SCC.
685

686 A. Dix-Hallpike

687

688 Procedure
689 The traditional Dix-Hallpike involves moving the patient from a sitting position to a position likely
690 to provoke a response (such as a head-hanging position) and then returning to a sitting position.
691 This can be done using the VNG goggles or Frenzel goggles; however, it is beneficial to be able
692 to review recorded eye movements using the VNG goggles.
693
694 To perform this procedure, have the patient sit on a flat surface. Ensure they are far enough
695 from the head of the reclined surface so that their head will be extended over the edge when
696 they lie supine. Instruct the patient to keep their eyes open, looking forward with their vision
697 denied. Turn the head 45° to the right. Place your hands on either side of the patient's head and

698 quickly but carefully have the patient recline into a head-hanging position, maintaining the head
699 turned 45° to the right. Ensure that the test ear is lower than the patient's shoulder. Have the
700 patient stay in this position for approximately 30 seconds (or until after the nystagmus stops),
701 then ask them to sit back up, keeping their head turned 45° to the right. Have the patient stay in
702 this position for another 30 seconds, and then they may relax to a neutral sitting position. If a
703 positive Dix-Hallpike is observed, repeat the Dix-Hallpike on the same side to check for
704 fatigability of the response. Then repeat the whole procedure for the head turned 45° to the left
705 (Halker, Barrs, Wellik, Wingerchuk & Demaerschalk, 2008).
706
707 **Dix-Hallpike Modifications**
708
709   • **Fully supported Hallpike:** For patients who cannot tolerate the traditional or side-lying
710   procedure. This modification uses the same procedure as the traditional Dix-Hallpike,
711   but the patient's head remains fully supported on the reclined chair or table, and does
712   not hang below the shoulder (Trendelenberg position).
713   • **Side-lying maneuver:** For patients with difficulty lying flat on their back. The patient sits
714   in the middle of the table with their feet hanging off to one side. Turn their head 45° to
715   the right and instruct the patient to lie down on their left side, maintaining the head turn
716   while having them swing their legs up on the table. Repeat this with the head turned 45°
717   to the left and lying down on their right side.
718
719 **Interpretation**
720 Classic positional vertigo will result in nystagmus that is consistent with the plane of the affected
721 canal while it is stimulated. Vertigo is usually reported while nystagmus is observed. A central
722 nervous system involvement is suspected when pure vertical or pure torsional nystagmus is
723 elicited. Vertigo is not commonly reported when central positional nystagmus is present.
724 Dix-Hallpike is positive for posterior SCC BPPV if non-sustained torsional and up-beating
725 nystagmus is observed in the head-hanging position, with or without reversal upon sitting.
726 Anterior SCC BPPV also results in transient torsional nystagmus but with down-beating rather
727 than up-beating nystagmus. For the posterior canals, the torsion beats to the affected ear. A
728 positive response should fatigue on repeat of the Dix-Hallpike on the same side (see Table 5).
729
730 **Table 5: Dix-Hallpike Results for Posterior and Anterior BPPV**

BPPV	Dix-Hallpike
Posterior SCC	rotary, up-beating
Anterior SCC	rotary, down-beating

731
732 Note that, in general, canalithiasis produces transient nystagmus with a few seconds' latency
733 whereas cupulolithiasis results in persistent nystagmus with little or no latency. Involvement of
734 the anterior canal (on either side) will produce nystagmus on both Dix-Hallpike maneuvers (right
735 and left). The direction of the torsional component will determine the affected side.
736 If horizontal nystagmus is observed, horizontal canal involvement should be further investigated
737 with positional head and/or body tests. If atypical nystagmus is recorded, it may allude to
738 multiple canal or CNS involvement.
739
740
741

Clinical Use

The Dix-Hallpike maneuver is a positioning evaluation to determine if a patient has posterior or anterior SCC BPPV. A positive response should trigger referral for management using canalith repositioning maneuvers (e.g., Epley, Semont, Liberatory, Brandt-Daroff, etc.).

B. Supine Head-Roll Test (or Pagnini-McClure Maneuver)

Procedure

The Supine Head-Roll Test (McCaslin, 2013) is a positioning evaluation to determine if a patient has horizontal SCC BPPV. It involves placing the patient in a supine position with the head elevated 30° and rotating the head 90° to the right and left. Following each head turn, the clinician observes the eyes for nystagmus for 30 seconds. The patient's head is then returned to centre and held in this position until there is no longer any nystagmus observed. The direction and duration of any nystagmus is measured. The test allows the clinician to localize which ear is affected and where the otolithic debris is likely to be in the canal (see Table 6).

Table 6: Supine Head-Roll Test Results for Horizontal Canal BPPV

BPPV	Right Lateral Canal Affected/Positional Body	Left Lateral Canal Affected/Positional Body
Horizontal SCC Canalithiasis (Transient & Delayed Onset)	Geotropic, Stronger on right ear down	Geotropic, Stronger on left ear down
Horizontal SCC Cupulolithiasis (Persistent & Immediate Onset)	Ageotropic, Stronger on left ear down	Ageotropic, Stronger on right ear down

Interpretation

Localization is determined by measuring which head turn had the greatest intensity of nystagmus. If it is unclear which side is affected, there are other techniques that can be used to determine which ear is impaired, such as the bow and lean test (Choung et. al., 2006). The direction of the nystagmus suggests where in the horizontal canal the debris resides. Geotropic nystagmus is the most common form and produces a bilateral geotropic nystagmus during lateral head turns which suggests the debris is located in the posterior arm of the horizontal SCC (Caruso & Nuti, 2005). Bilateral ageotropic nystagmus is less common, but is thought to represent cupulolithiasis of the lateral canal (Aron et. al., 2013).

Contraindications

Absolute contraindications:
- recent cervical spine fracture
- cervical disc prolapse
- vertebrobasilar insufficiency (VBI), known and documented
- recent neck trauma that restricts torsional movement

Caution or modified procedures should be used:

778   -   carotid sinus syncope (if history of drop attacks or blackouts)
779   -   severe back or neck pain
780   -   recent stroke
781   -   cardiac bypass surgery within last 3 months
782   -   rheumatoid arthritis, affecting the neck
783   -   recent neck surgery
784   -   cervical myelopathy
785   -   severe orthopnea, may restrict duration of test
786
787 (British Society of Audiology, 2001)
788
789 **Limitations**
790 A positive BPPV response will fatigue, therefore it is important to test the suspected side first
791 and before doing any supine positional testing.

Positional Testing

793 **Definition**
794 Positional testing examines the presence or absence of nystagmus with or without subjective
795 dizziness, due to changes in position of the head or the head and body relative to the force of
796 gravity. This differs from positioning tests because the head and body are static during
797 positional testing.
798
799 Positional nystagmus is thought to occur as a result of reduction in suppression of asymmetrical
800 SCC function due to impaired otolith function or central vestibular pathway involvement. The
801 rationale of testing for nystagmus in various positions is to allow for change of the orientation of
802 the labyrinth in relation to gravity. Testing starts with measuring any nystagmus that occurs in a
803 neutral position called spontaneous nystagmus (SN). Eye movements are recorded with the
804 patient wearing VNG goggles with vision denied. The patient is asked to keep eyes open,
805 looking forward in a neutral sitting position. This is repeated with visual fixation.
806
807 Positions used for positional testing are:
808   • **Sitting:** Eye movements are recorded with the patient wearing VNG goggles with vision
809   denied.
810   • **Supine:** Eye movements are recorded with the patient wearing VNG goggles with vision
811   denied, and patient supine with head supported at centre, repeated with head turned 90°
812   to the right, and then repeated with head turned 90° to the left. Individuals with restricted
813   neck movement will require a modified angle of head turn.
814   • **Lateral/Positional Body:** Eye movements are recorded with the patient wearing VNG
815   goggles with vision denied and patient's head and body to the right, and repeated with
816   patient's head and body to the left. N.b., for each positive positional test, a test for
817   suppression of the nystagmus with fixation is completed.
818
819 **Interpretation**
820 Positional nystagmus that is present without fixation may be peripheral or central in nature. Lack
821 of suppression of the nystagmus when measurement is repeated with visual fixation suggests a
822 central origin. If nystagmus does not alter with positional change, it can be interpreted as purely
823 spontaneous nystagmus. SN that changes direction is indicative of CNS involvement. SN
824 related to unilateral vestibulopathy (peripheral) is usually directionally fixed and suppresses with
825 fixation; the fast-phase may beat toward the unaffected ear or toward the weaker labyrinth.

Occasionally SN beats toward the weaker labyrinth if an irritative lesion is present, notably with Ménière's disease or with hyperventilation-induced nystagmus in retrocochlear pathology.

Atypical types of SN include, but are not limited to:
- **congenital nystagmus:** usually presents with horizontal fast and slow-phases; resulting from abnormalities in the neural pathways
- **periodic alternating nystagmus***:* changes in direction within the neutral sitting position, irrespective of eye, head, and body position

N.b., predominantly vertical nystagmus, whether up-beating or down-beating, is an indicator of CNS involvement. Up-beating is typically observed in brainstem, cerebellar, multiple sclerosis (MS), or drugs. Down-beating is typically observed in cerebellar degeneration, cranio-cervico junction (Arnold-Chiari), MS, or VBI.

Positional Nystagmus With SN: If the nystagmus observed changes in direction or intensity, with change in position, it would suggest a spontaneous and positional nystagmus.

Positional Nystagmus Without SN: In the absence of spontaneous nystagmus, positional nystagmus is most likely attributable to that test position.

Clinically Significant Responses:
- Slow-phase velocity (SPV) > 5 deg/s
- SPV < 6 deg/s persistent in 4 or more positions
- SPV < 6 deg/s sporadic in all positions
- Direction changing within a given head position (indicates central involvement)

(Myers, 2011)

Cervical Vertigo Screening: Positional nystagmus that is present in head-right or head-left but absent in lateral positional testing may be suggestive of a cervicogenic component. Etiologies of a cervicogenic component include vertebrobasilar insufficiency and altered cervical proprioception. Further investigations should be arranged by the referring or primary care physician.

Clinical Use
Positional testing is used clinically to determine asymmetry in the vestibular afferents of either peripheral-origin or the central nervous system.

Contraindications
Absolute contraindications and cautions can be found under the Dix-Hallpike Testing section (page 19).

Limitations
Alcohol and pharmacologic drugs may also contribute toward positional nystagmus.

Caloric Irrigation Testing

Definition
The caloric test evaluates the vestibulo-ocular reflex. It involves irrigating the ear canal to

873 assess the function of the peripheral vestibular system, specifically of the horizontal semicircular
874 canal and superior vestibular nerve.
875
876 Within the semicircular canal ampullae, the endolymph and cupula are the same density. The
877 temperature change delivered by calorics changes the density of the endolymph and it therefore
878 deflects the cupula in the lateral semicircular canal. Warm stimuli results in an endolymph rise
879 towards the ampulla and an excitatory neural response; cool stimuli, the opposite. There may
880 also be an effect of temperature on the vestibular nerve (Barin, 2016b). As neural firing is
881 unchanged in the non-test ear, the asymmetry in neural firing is perceived by the VOR as a
882 rotation towards the test ear and the eyes move accordingly (Barin, 2016a).
883
884 Nystagmus can be recorded using video goggles (known as VNG) or electrodes (ENG). As
885 VNG is the industry standard, ENG will not be covered here.

886 **Procedure**
887 The patient's head is raised approximately 30° to position the horizontal canal vertically. The
888 temperature of the air or water is a set amount above or below body temperature, typically 44
889 and 30 °C for water.
890
891 With visual fixation removed (e.g., by covering the goggles), either the right or left ear canal is
892 irrigated with warm water or air for 30 to 40 seconds. Warm caloric irrigations should be tested
893 first if use of the monothermal caloric screening test is under consideration (see below). The
894 suspect ear should be tested first; if the patient declines further testing after the first caloric then
895 the audiologist will have at least collected information about the ear of interest (Lightfoot, 2004).
896 The patient's eye movements are recorded with either VNG goggles or electrodes (if ENG is
897 used). If the patient appears to be suppressing the nystagmus, the examiner should ask the
898 patient questions to distract them (this is known as tasking or mental alerting). When the
899 nystagmus begins to slow, the patient is asked to fixate on a target (e.g., a light in the goggles)
900 and the nystagmus is observed for suppression. The peak velocity of the nystagmus without
901 fixation is then measured for each irrigation performed. As in other vestibular tests, nystagmus
902 is named according to the direction of the fast-phase, and its velocity is measured using the
903 slow-phase. The nystagmus should beat in the direction of the irrigated ear for warm water, and
904 away from the irrigated ear for cool water. This is summarized by the acronym COWS (cold
905 opposite; warm same).
906
907 When possible, water should be used as the stimulus, as it evokes a stronger response and
908 results are less variable (Goncalves, Felipe, & Lima, 2008). However, in the presence of
909 contraindications to water (see below), then air may be used. Please see the reference for more
910 detail about the testing procedure.
911
912 **Monothermal Caloric Screening Test**
913 If warm caloric irrigations are performed first and the asymmetry is less than 15%, then it is
914 possible to predict a normal outcome with 95% sensitivity. However, both warm calorics must be
915 greater than 8 deg/s and any spontaneous nystagmus must be below 4 deg/s to use this
916 screening test (Lightfoot et al., 2009).
917
918 **Ice Calorics**
919 Ice calorics may be used to determine if any vestibular function remains at low frequencies. This
920 test is appropriate if bithermal calorics do not evoke nystagmus (or the nystagmus is very weak)
921 in an ear or only spontaneous nystagmus is observed. Please see the British Society of

Audiology's *Recommended Procedure for the Caloric Test* (2014) for instruction on administering ice calorics.

Interpretation & Clinical Use

The following measures are typically used to interpret calorics.

Unilateral Weakness (UW): This is used to indicate a lesion in the horizontal semicircular canal or its afferent pathways, and is measured using Jongkees' formula:

$$UW\% = \frac{(Right\ Warm + Right\ Cold) - (Left\ Warm + Left\ Cold)}{Right\ Warm + Right\ Cold + Left\ Warm + Left\ Cold} \times 100$$

A value of over 20–33% is suggestive of a vestibular weakness to one side (Goncalves et al., 2008; Barin, 2016a); however, if the absolute velocity values are low, the UW must be interpreted with caution. Labyrinthitis, Ménière's disease, vestibular neuritis, vestibular schwannomas, or an ischemic infarction can all result in a unilateral weakness in caloric testing (Barin, 2016a).

Directional Preponderance (DP): DP calculated from bithermal caloric irrigations is the tendency of nystagmus to beat more strongly in one direction. The relevance of DP is controversial, and it is non-localizing (British Society of Audiology, 2014; Goncalves et al., 2008). Values over 25–50% have been suggested as abnormal (Barin, 2016a). The examiner must ensure that the DP is not a result of spontaneous nystagmus, which is added to or subtracted from the nystagmus evoked from the caloric.

$$DP\% = \frac{(Right\ Warm + Left\ Cold) - (Left\ Warm + Right\ Cold)}{(Right\ Warm + Right\ Cold + Left\ Warm + Left\ Cold)} \times 100$$

Bilateral Hypofunction: A bilateral weakness occurs when the caloric response is absent or very weak in both ears. There are a wide range of values used to define a bilateral weakness, some using the sum of all four calorics (e.g., total < 30 deg/s) or based on the total response of each ear (total right ear or total left ear is ≤ 12 deg/s) (Barin, 2011).

Bilateral hypofunction can be caused by ototoxic medication or any pathology that affects either both peripheral systems or the central vestibular pathways. It may also be caused by insufficient heat transfer or medications. If possible, bilateral hypofunction should be confirmed using the rotary chair (Barin, 2016a).

Fixation Index (FI): The FI measures the extent to which the caloric nystagmus is suppressed by visual fixation. The failure to fixate indicates a central lesion, often in the cerebellum (Barin, 2016a).

$$FI\% = \frac{NysFix}{NysNoFix} \times 100$$

NysFix is the average nystagmus velocity 5 seconds after fixation was introduced, and NysNoFix is the average nystagmus velocity 5 seconds before fixation was introduced. The fixation index should be lower than 50–60%; otherwise a central pathology must be considered (British Society of Audiology, 2014; Barin 2016a).

Hyperfunction: Hyperactive responses are caused by the loss of the VOR inhibitory function and also denote a central lesion. Again, there is a wide range of values used to define a hyperactive response, some based on the total responses per ear (e.g., total response per ear is ≥140 deg/s), some based on the total warm or total cool responses (Barin, 2016a). Please see Barin (2016a) for less common abnormalities in caloric testing.

Contraindications

Otologic considerations: Water cannot be used with a perforated eardrum, active middle ear dysfunction, or patients who have undergone a mastoidectomy. While air can be used in these cases, the caloric weakness value can only be used to determine the presence or absence of a response (Goncalves et al., 2008). Ear infections and significant amounts of wax must be resolved/removed before calorics can be administered.

Calorics may be contraindicated in patients with limited eye mobility (e.g., ocular myopathy) or recent eye surgery.

Caloric testing is not recommended in the presence of medical problems (e.g., acute hypertension, psychotic disorders, epilepsy, arrhythmias, recent heart attack). For a full list please see the British Society of Audiology's *Recommended Procedure for the Caloric Test* (2014).

Limitations
Wax or middle ear fluid may result in inadequate heat transfer through to the horizontal canal, and therefore any hypofunction obtained must be interpreted with caution.

The caloric test only assesses the vestibular system's response to unnaturally low frequency movements (0.002–0.004 Hz); therefore, an absent response on the caloric test does not necessarily mean a complete lack of vestibular function (Shepard, 2009).

The caloric test evaluates the VOR originating in the horizontal semi-circular canal. The function of the otoliths, anterior and posterior semi-circular canals, and inferior branch of the vestibular nerve must be assessed with other tests, such as the Video Head Impulse Test (vHIT) and Vestibular Evoked Myogenic Potentials (VEMPs).

Certain medications, such as sedatives, anti-nausea medication, anti-histamines, diuretics, tranquilizers, and anti-depressants, can suppress the caloric response (Cass and Furman, 1993).

Rotary Chair Testing

Definition
The rotational chair evaluates the VOR. The VOR generates compensatory eye movements during rotational or translational movements (accelerations) of the head.

The VOR is the mechanism that results in equal and opposite eye movement when the head is rotated in order preserve a stable image on the fovea. The accuracy of the eye movement depends on the signal received from the semicircular canals, which detect angular acceleration. When an impairment is present, the eye movements generated by the VOR will not properly

1016 account for the head rotation that took place and retinal slip will occur. Patients with bilaterally
1017 reduced VOR function may present with symptoms of oscillopsia or visual blurring during head
1018 rotations.
1019
1020 The rotational chair measures the VOR across multiple frequencies of rotation, allowing
1021 simultaneous stimulation of the horizontal semicircular canals. In addition, the rotation chair
1022 provides information about the overall function of the system during angular acceleration of the
1023 head.
1024
1025 **Procedure**
1026 Rotational chair testing is generally completed in a darkened room with a separate area housing
1027 the computer for analysis. The patient is secured in the chair with a head restraint and harness
1028 to ensure safety during testing. A two-way communication system allows the patient to remain in
1029 contact with the examiner throughout the test. The patient's head should be tilted down at a 30°
1030 angle to align the canals with the axis that is being stimulated during the test (Arriaga, Chen &
1031 Cenci, 2005). There are a number of rotational chair tests that are clinically useful for assessing
1032 the VOR.
1033
1034 **Sinusoidal (or Slow) Harmonic Acceleration Test:** In this test, the rotational chair moves at
1035 incremental frequencies in a sinusoidal pattern. The most common octave frequency range is
1036 between 0.01 Hz and 2 Hz. This allows the measurement of eye movements at velocities of
1037 rotation ranging from 20 to 80 deg/s. Data is averaged over a minimum of 3 cycles for each
1038 frequency to compute gain, asymmetry, and phase.
1039
1040 **Velocity Step Test:** The velocity step test involves rapid acceleration of the chair (100–200
1041 deg/s) until the chair reaches a predetermined velocity. The velocity is then maintained for a
1042 period of time (60 seconds is typical) and then rapid deceleration occurs. The test is repeated
1043 for both clockwise and counter-clockwise directions at fixed velocities. Nystagmus during
1044 rotation is compared to nystagmus in the post-rotary condition to compute the time constant and
1045 gain.
1046
1047 **Visual Fixation Suppression Test:** Goulson, McPherson, and Shepard (2016) indicate that the
1048 visual fixation suppression test suggests central nervous system deficit affecting this function,
1049 which overlaps with the neurological substrate for smooth pursuit and gaze fixation. The patient
1050 is asked to fixate on the calibration light that moves with the rotational chair, allowing the
1051 measurement of eye movements that may or may not be suppressed with fixation.
1052
1053 **VOR Cancellation or Suppression Test (VOR Enhancement):** In the VOR cancellation or
1054 suppression test, the optokinetic stripes are presented on the walls of the rotational chair booth
1055 while rotating the patient sinusoidally at a predetermined test frequency (Ruckenstein & Davis,
1056 2015). Ruckenstein and Davis (2015) suggest that this test can provide information regarding
1057 the integrity of the central vestibulo-ocular pathways.
1058
1059 **Interpretation**
1060
1061 **Sinusoidal (or Slow) Harmonic Acceleration Test:** Three parameters are assessed, including
1062 gain, asymmetry, and phase. Significant reduced gains suggest bilateral vestibular involvement;
1063 however, reduced gains [particularly in the lower frequencies of rotation] can also occur as a
1064 result of unilateral weakness (Ruckenstein & Davis, 2015). Asymmetry that is outside normal
1065 limits suggests an acute, physiologically uncompensated vestibulopathy; whereas asymmetry

within normal limits suggests a physiologically compensated vestibulopathy. Ruckenstein and Davis (2015) suggest that abnormal phase leads suggest a peripheral vestibular abnormality. It is important to note that side of lesion cannot be determined by rotary chair testing.

Velocity Step Test: Two parameters are typically assessed, including gain and time constant. Ruckenstein and Davis (2015) suggest that reduced gain for clockwise per-rotary or counter-clockwise post-rotary conditions indicate a right paretic lesion is more likely, but a left irritative lesion cannot be excluded. The opposite would be suggested for reduced gains in the other two conditions (Ruckenstein & Davis, 2015). Abnormally short time constants are seen in cases of unilateral or bilateral peripheral abnormalities, whereas abnormally long time constants suggest cerebellar involvement (Ruckenstein & Davis, 2015).

Visual Fixation Suppression Test: If the rotational-induced nystagmus is not decreased or abolished with fixation, then central involvement may be suggested (Ruckenstein & Davis, 2015).

VOR Cancellation or Suppression Test (VOR Enhancement): It is expected that the optokinetic strips will elicit visually mediated nystagmus at the same time that nystagmus is produced as a result of vestibular stimulation, causing an increase in gain for the same sinusoidal frequency in the dark (Ruckenstein & Davis, 2015). If the gain is reduced during this test (compared to the same sinusoidal frequency in the dark), central involvement may be suggested.

Contraindications
Patients who are very claustrophobic may not be able to tolerate rotational chair testing. As well, if a patient reports significant nausea from the test, the test should be temporarily halted and/or discontinued.

Vestibular Evoked Myogenic Potentials (VEMPs)

Cervical Vestibular Evoked Myogenic Potentials (cVEMPs)

Definition
Cervical vestibular evoked myogenic potentials (or cVEMPs) are one of two tests that can be used to measure the function of the otoliths (the organs most sensitive to linear acceleration). It is a clinically useful tool that can be included as part of a test battery for assessing vestibular function.

Scientific Basis
The cVEMP is a short-latency myogenic response which is evoked by air-conducted sound (ACS), or bone-conducted vibration (BCV), originating in the saccule and recorded on the ipsilateral contracted sternocleidomastoid (SCM) muscle. The response is a biphasic positive-negative waveform at approximately 13 and 23 ms respectively and reflects the release from contraction of the SCM (Colebatch, Halmagyi, & Skuse, 1994; Papathanasiou, Murofushi, Akin, & Colebatch, 2014). If the integrity of the vestibular pathway is compromised, this response is reduced or absent (Colebatch et al., 1994).

Air Conduction Sound versus Bone Conducted Vibration
cVEMP stimuli can be delivered by bone-conduction vibration (BCV) or high-intensity air-

conducted sound (ACS). Typically, ACS is used; however, BCV is preferred if conductive hearing loss is indicated.

Procedure

A cVEMP involves measuring electromyography (EMG) activity from surface electrodes placed on the SCM muscles. The active electrodes are placed on the SCM muscles (the midpoint to upper third of the muscle) and reference and ground electrodes are placed on the sternum and forehead (or upper chest) respectively (Papathanasiou et al., 2014). The response is measured on the muscle ipsilateral to the stimulated ear. The response is dependent on the strength of the SCM muscle activity; therefore, EMG activity must be monitored (Colebatch et al., 1994). Usually, a patient lifts and/or rotates their head to the opposite side of the stimulated ear; however, other methods of activating the SCM are possible. BCV at the midline evokes bilateral responses and should be recorded bilaterally. If rotation is used, then recordings must be unilateral; the muscle contralateral to the stimulated ear will be relaxed.

A 500 Hz tone burst at a rate of 5 bursts per second is presented for a minimum of 100 sweeps at a high but safe intensity (90–105 dB nHL/120–135 dB SPL for ACS). Though clicks can be used, a tone burst of 500 Hz is more effective (Rosengren, Welgampola, & Colebatch, 2010; Isaradisaikul, Navacharoen, Hanprasertpong, & Kangsanarak, 2012). Frequency tuning can differ in patients over 60 years old or those with Ménière's Disease, and in these situations, a better response may be obtained with a higher frequency (Papathanasiou et al., 2014; Janky & Shepard, 2009). Testing should then be performed at a lower intensity (approximately 70 dB nHL/100 dB SPL) to screen for an abnormally low threshold. If a response is obtained at 100 dB SPL, then testing should be performed in decreasing 5–10 dB steps until it is no longer present. Waveforms should be repeated at least once to verify response presence. EMG activity must be monitored as the larger the muscle contraction, the larger the response amplitude.

The first major positive peak on the ipsilateral recordings should be marked as p13 (or p1) and the following negative peak as n23 (or n1). The amplitude of the response is the p13 to n23 peak-to-peak amplitude.

Interpretation

A number of parameters are measured for the cVEMP response and different clinics may use different measures. The commonly used measures include presence of a response, amplitude asymmetry ratio, and threshold. Note that response amplitudes and latencies are affected by the stimulus parameters; the normative data used must be appropriate for the parameters used. Asymmetrical electrode placement is the most common cause of p13 interaural latency differences; however, if the electrodes are evenly placed on both SCMs, retrolabyrinthine disorders can prolong latency. Absolute p13 latency and absolute p13 amplitude are of limited value due to the variance from patient to patient and its dependence on stimulus parameters (Meyer, Vinck, & Heinze, 2015; Blakley & Wong, 2015).

Presence/Absence of Response

The absence of a response suggests a pathology in the sacculo-collic pathway, assuming there is no conductive hearing loss (Colebatch et al., 1994; Murofushi, Matsuzaki, & Mizuno, 1998; Welgampola & Colebatch, 2005). A response can be obtained in patients with profound sensorineural hearing loss (Colebatch et al., 1994). Because a response cannot be elicited in some healthy older subjects, the absence of responses bilaterally is only considered pathological in patients under the age of 60 (Murofushi & Kaga, 2009; Meyer et al., 2015). However, a unilateral response in older subjects is considered pathological.

Amplitude Asymmetry Ratio
- This is calculated using Jongkees' formula:

$$AR = \frac{(largest - smallest)}{(largest + smallest)} \times 100$$

- The amplitude used for the formula above must be corrected for EMG activity.
- Typically, an asymmetry ratio over 32–42% is considered abnormal (Papathanasiou et al., 2014; Blakley & Wong, 2015).

Threshold

A threshold at or below 65–70 dB nHL (95–100 dB SPL) is suggestive of a third window in the vestibular organ, usually a dehiscence in the superior semicircular canal (Papathanasiou et al., 2014; Welgampola, Myrie, Minor, & Carey, 2008).

Clinical Use

The cVEMP is useful in various peripheral and central nervous system disorders. It can be used to assess the integrity of the sacculo-collic pathway, as well as the possibility of an otic capsule dehiscence.

Integrity of the Sacculo-Collic Pathway

The pathway is as follows:

Saccule → Inferior vestibular nerve → Vestibular nuclei →
Medial vestibulospinal tract → Accessory nucleus and nerve (CN XI) →
Sternocleidomastoid (SCM) muscle

A reduced or absent response could suggest a pathology in the:
- saccule (e.g., labyrinthitis, Ménière's, BPPV);
- inferior vestibular nerve (e.g., schwannoma, neuritis); and/or
- brainstem (e.g., stroke, lesion, multiple sclerosis).

The cVEMP can also be used to distinguish between superior, inferior, and total vestibular nerve pathology (in combination with ocular VEMP [oVEMP] testing, calorics, and the video head impulse test; vHIT). Because the oVEMP pathway involves the superior vestibular nerve and cVEMP involves the inferior vestibular nerve, a present response in one test and absent response in the other can determine the site of lesion. It may also help identify the stage of Ménière's disease, as the augmented cVEMP amplitude is sometimes seen in the initial stages of Ménière's, but a reduced or absent response is obtained as it progresses (Young, 2013).

Canal Dehiscence Syndrome

This condition is caused by the thinning or absence of part of the temporal bone overlying a semicircular canal—typically the superior canal, or a third window elsewhere in the vestibular apparatus. In these cases, cVEMP amplitudes are typically larger than usual and the response is present at a lower intensity level.

When to Test

In the following situations, cVEMPs should be part of the test battery:
- The patient reports symptoms of otolithic dysfunction (tilting, rocking, pushing, or pulling)
- The audiogram suggests conductive hearing loss but acoustic reflexes are present.
- Patient symptoms include Tullio, autophony (self-generated sounds, such as breathing, heartbeat, eye movements, or the patient's own speech, are heard unusually loudly in the affected ear), oscillopsia, or pulsatile tinnitus.

Contraindications

1213
1214 The cVEMP response can be absent or its amplitude reduced in the presence of even a small
1215 conductive hearing loss (when ACS is used), and therefore a formal audiogram should be used
1216 to assess the patient's hearing in such cases. A normal cVEMP does not negate the need for an
1217 audiogram as occasionally otosclerosis and superior canal dehiscence (SCD) are present in the
1218 same ear: the otosclerosis attenuates the sound reaching the ear and the SCD causes a higher
1219 amplitude response to that attenuated sound. The result is what appears to be a normal
1220 cVEMP.
1221
1222 As the cVEMP requires a contracted SCM, patients unable to elevate and/or rotate their head
1223 may not be able to perform the test.

Limitations

1225 Except in cases of a canal dehiscence, cVEMP findings alone are non-specific; they simply
1226 indicate reduced function.
1227
1228 As described above in the interpretation section, bilaterally absent responses are not always
1229 considered pathological in patients over the age of 60.
1230

Ocular Vestibular Evoked Myogenic Potentials (oVEMPs)

Definition

1233 The second test that can be used to measure otolithic function is the oVEMP. It is a clinically
1234 useful tool that can be included as part of a test battery for assessing vestibular function.
1235

Scientific Basis

1237 When vibration is applied at Fz in humans, approximately equal amplitude linear acceleration is
1238 delivered to both mastoids (Curthoys, Manzari, Smulders, & Burgess, 2009; Iwasaki et al.,
1239 2008a), resulting in small short-latency negative (excitatory) ocular myogenic potentials beneath
1240 the eyes. The first of these is called n10 (appears at approximately 10 ms), and it has a larger
1241 amplitude when the patient looks up (Iwasaki et al., 2008a). The n10 is a crossed otolith-ocular
1242 response; i.e., it is measured under the contralateral eye, and reflects the activation of the
1243 inferior oblique and possibly the inferior rectus (Weber et al., 2008; Curthoys et al., 2011).
1244 However, after a unilateral vestibular loss, the n10 under the contralateral eye is absent or has
1245 reduced amplitude (Iwasaki et al., 2008b).
1246
1247 There is some evidence that oVEMPs originate predominantly in the utricle (Curthoys, Vulovic,
1248 and Manzari, 2012; Curthoys, 2010; Curthoys, et al., 2011; Manzari, Burgess, and Curthoys,
1249 2010) whereas other findings argue the response may include a small contribution from the
1250 saccule, originating from both the utricle and the saccule (Papathanasiou, 2012; Welgampola et
1251 al., 2008).

Bone Conducted Vibration versus Air Conducted Sound

1253 oVEMP stimuli can be delivered by BCV or high-intensity (95–97 dB nHL/125–127 dB SPL)
1254 ACS. These two methods work in different ways. BCV at Fz causes waves to travel around and
1255 through the head and these waves result in linear acceleration that is approximately equal at
1256 both mastoids (Yang, Liu, Wang, and Young,, 2010; Iwasaki et al., 2008a). However, ACS
1257 moves the stapes, which in turn causes endolymph movement (Curthoys & Vulovic, 2011).
1258

1259 Bone-conduction is generally preferable to air-conduction for oVEMP if utricular or superior
1260 vestibular nerve pathology is suspected. The results obtained with BCV are both more reliable
1261 and of greater amplitude than with ACS (Cheng, Chen, Wang, & Young, 2009; Chihara, Iwasaki,
1262 Ushio, & Murofushi, 2007; Wang, Weng, Jaw, & Young, 2010). However, ACS is effective in
1263 diagnosing a canal dehiscence (Zuniga, Janky, Nguyen, Welgampola, & Carey, 2013; Janky,
1264 Nguyen, Welgampola, Zuniga, & Carey, 2013).
1265

Procedure

1267 As with cVEMPS, there are different methods of recording oVEMPs. Typically, an active
1268 electrode is positioned on the skin directly underneath the eye contralateral to the side that is
1269 presented with the stimulus. Reference and ground electrodes are also placed, generally further
1270 down the face or on the nose and on the sternum, respectively. The stimulus (ACS or BCV) is
1271 then presented using a 250–500 Hz tone burst at a rate of 3 to 5 cycles per second. (Chang,
1272 Cheng, Wang, & Young, 2010; Curthoys et al., 2009; Chihara et al., 2009; Sandhu, George, &
1273 Rea, 2013). The patient must look straight up at approximately 30° in order to bring the inferior
1274 oblique muscle closer to the surface of the skin.
1275

1276 In patients over the age of 60 or with suspected Ménière's disease, 1000 Hz may be used as
1277 the frequency tuning characteristics (Sandhu, Low, Rea, & Saunders, 2012; Piker, Jacobson,
1278 Burkard, McCaslin, & Hood, 2013). If a canal dehiscence is suspected, testing at 4000 Hz may
1279 be warranted (Manzari, 2013). The peak found at approximately 10 ms should be labeled.

Interpretation

1281 The measures currently used to determine if an oVEMP is normal or not are presence of a
1282 response, amplitude asymmetry ratio, and absolute amplitude.
1283

Presence/Absence of Response

1285 • Iwasaki et al. (2008a) found that with BCV, responses could be elicited in all normal
1286   subjects (n=67). No response was elicited in subjects (n=5) with bilateral vestibular loss
1287   (no response on cVEMP and ice calorics).
1288 • Yang et al. (2010) performed BCV oVEMP testing on healthy guinea pigs and guinea
1289   pigs treated with gentamicin and found a significant difference in the rates of the
1290   response. Only 30% (3 of 10) of gentamicin-injected ears had an oVEMP but 100% of
1291   saline-injected ears had a response.
1292

Amplitude Asymmetry Ratio

1294 • This is calculated using Jongkees' formula:
1295   AR = (largest − smallest) X 100
1296   (largest + smallest)
1297

1298 • Iwasaki et al. (2008b) found that 11 patients with vestibular loss had mean AR of 75.03%
1299   +/- 16.32. No normal subjects (n=67) had an AR over 40% and all the patients with
1300   unilateral vestibular loss had an AR over 50%.
1301 • Iwasaki et al. (2008a) found that 64 healthy subjects all had ARs < 40%; average AR
1302   was 11.73% +/- 8.26.
1303

Absolute Amplitude

1305 • Zuniga et al. (2013), Janky et al. (2013), and Manzari, Burgess, McGarvie, and Curthoys
1306   (2012) found that oVEMP absolute amplitude was a more sensitive and specific tool than
1307   ACS cVEMPs in the diagnosis of superior canal dehiscence. These studies concluded

that an n10 amplitude over 8.25–9.3 uV or a peak-to-peak amplitude over 17.1 uV were appropriate cut-off values when using ACS and an amplitude over 10 uV was appropriate when using BCV. Note that the cut-off values will be affected by stimulus parameters and electrode placement.

Clinical Use

There are two main uses of the oVEMP: to assess the integrity of the utriculo-ocular pathway, and to assess the likelihood of a canal dehiscence.

Integrity of the utriculo-ocular pathway

The pathway is as follows:

Utricle → Superior vestibular nerve → Vestibular nuclei → Medial Longitudinal Fasciculus (MLF) → Oculomotor nerve → Contralateral Inferior Oblique

A reduced or absent response could suggest a pathology in the:

- utricle (e.g., labyrinthitis, Ménière's, recurrent BPPV);
- superior vestibular nerve (e.g., schwannoma, neuritis); or
- brainstem (e.g., stroke, lesion, multiple sclerosis).

Canal Dehiscence

This condition is caused by the thinning or absence of part of the temporal bone overlying a semicircular canal, typically the superior canal. In this case, ACS is an effective stimulus, using absolute amplitude as the measure (as described above).

When to Test

Particularly in the following situations, oVEMPs should be part of the standard test battery:

- The patient reports symptoms of otolithic dysfunction (tilting, rocking, pushing, or pulling).
- The audiogram suggests conductive hearing loss but acoustic reflexes are present.
- Patient symptoms include Tullio (sound- or pressure-induced dizziness), autophony (self-generated sounds, such as breathing, heartbeat, eye movements, or the patient's own speech, are heard unusually loudly in the affected ear), oscillopsia, or pulsatile tinnitus.

Contraindications

ACS VEMP responses rely on the vibrations from the stimulus being sufficient to cause endolymph movement. As a result, ACS VEMPs will appear to be absent in patients with conductive hearing loss. Therefore, ACS VEMP results can only be interpreted accurately when there is an accompanying audiogram. Conversely, it is possible for patients with profound sensorineural hearing loss to have present VEMP responses, provided that the superior vestibular nerve and utricle are not damaged.

Limitations

The oVEMP is currently quite a new test. As a result, a consensus has not been reached regarding optimal parameters, stimuli, and electrode placements. Given the variability across clinical sites, each clinic should ideally consider collecting its own normative data. Alternatively, normative data may be used only when the protocol being used matches that of the site where the norms were drawn from.

Video Head Impulse Test (vHIT)

Definition

The video head impulse test (vHIT) is used to detect reduced function of the vestibulo-ocular reflex (VOR) quickly and non-invasively. A relatively new addition to the vestibular test battery, it has quickly gained respect as an important tool for vestibular assessment.

The purpose of the VOR is to maintain a stable visual image when the head is rotated. When a head rotation occurs, the VOR generates an equivalent eye movement in the opposite direction to preserve a stable foveal image (Roy & Tomlinson, 2004). The accuracy of the oculomotor command from the VOR depends on the fidelity of signal received from the semicircular canals, which detect angular acceleration. When an impairment is present, the eye movements generated by the VOR will not properly account for the head rotation that took place and retinal slip will occur. A corrective "catch-up" saccade is required to re-fixate the eye back on the target. Patients with reduced VOR function may report oscillopsia.

In 1988, Halmagyi and Curthoys were the first to infer from the presence of catch-up saccades that peripheral vestibular dysfunction was likely on the side(s) with the saccades. For the bedside head impulse test, patients were asked to stare at a target while their heads were rotated by in small but abrupt movements. The patients' eyes were then observed for catch-up saccades.

Observable catch-up saccades, known as "overt" saccades, occur after a head movement towards an impaired side. However, some patients generate corrective eye movements during the head movement. These eye movements are known as "covert" saccades; when covert saccades are present, vestibular deficits will typically be missed unless specialized equipment (e.g., vHIT) is used.

The vHIT allows examiners to observe the differences between the eye and the head (either in terms of velocity or position, depending on the calculation strategy used) during head rotations in order to identify VOR deficits. It is possible to use vHIT to test the horizontal semicircular canals as well as the vertical semicircular canals.

Procedure

Prior to using vHIT equipment, potential examiners are encouraged to carefully learn the individual guidelines specific to the purchased equipment. The major manufacturers have YouTube channels, which are helpful for learning the proper technique to ensure that the correct canals are being stimulated.

Along with an accelerometer/gyroscope for measuring head movements, vHIT testing is performed using lightweight goggles equipped with high-speed cameras (250 Hz is typical) for capturing eye movements.

The goggle straps should be adjusted to ensure a snug fit before the test begins. The examiner should check that the goggles are secure enough that they stay in place when gently wiggled. Eyeglasses must be removed as they cannot be worn with the goggles; contact lenses do not have to be removed. The patient should sit in a chair positioned at least 1 metre from the target. A calibration should be performed whenever possible to reduce variability of the data. The patient should be asked to stare at a target placed approximately at eye level. The examiner should encourage the patient to relax their neck, not to anticipate movements, and to

1404 avoid blinking. It is essential that the examiner monitor the software throughout the test to
1405 ensure that the software algorithms are accurately tracking the pupil throughout the test and
1406 adjust the pupil threshold to minimize noise. In some scenarios (e.g., if eye movements become
1407 cut off or the recording is too noisy and adjusting the threshold does not help), the goggles may
1408 have to be repositioned.
1409

Horizontal Canals
1410
1411 Standing behind the patient, the examiner must present small (10–20°) but abrupt head
1412 impulses in both lateral directions, making sure to randomize the direction of the head impulses
1413 so that they are unpredictable for the patient. The examiner's hands should be firmly in place on
1414 top of the patient's head (or, alternatively, firmly cradling the patient's jaw line). The examiner
1415 must avoid touching the goggle straps, as this can introduce artifact in the recording.
1416 To maximize the diagnostic utility of the test and the ease of interpretation, head impulses
1417 should be presented at a range of velocities whenever possible. The majority of the head
1418 impulses in the final tracings should exceed 150 deg/s to ensure that contributions from the
1419 oculomotor system are minimized (Meyer, Lasker, & Robinson, 1985).
1420

1421 At minimum, 20 head impulses should be completed and accepted by the software in each
1422 direction. However, having more head impulses (30 to 40) per side allows the examiner to
1423 ensure that a range of velocities is obtained and that the quality of the final tracings (after
1424 discarding unusable data) is optimal.
1425

Vertical Canals
1426
1427 To test the paired left anterior/right posterior canals (LARP), the patient's head should be
1428 rotated 45° to the right of the midline. For the right anterior/left posterior canals (RALP), the
1429 rotation is 45° to the left. In both scenarios, gaze should continue to be directed toward the
1430 centre dot; gaze must be maintained in the plane of the canals being stimulated to minimize
1431 contributions from torsional and/or horizontal eye movements (Migliaccio & Cremer, 2011). To
1432 ensure that eye movements are tracked appropriately, a new region of interest may have to be
1433 manually selected by the examiner before proceeding with the test. If the patient has a limited
1434 range of head rotation, the patient's chair can be repositioned so that the patient's entire body
1435 faces 45° from the midline (as before, gaze should be directed toward the midline).
1436

1437 Most commercially available systems have features embedded in their software that help
1438 clinicians to orient the head correctly so that the canals are maximally stimulated. Product
1439 manuals should be consulted for specific procedures, as they may vary slightly depending on
1440 the system being used.
1441

1442 Once the patient has been positioned correctly, the examiner should place one hand firmly on
1443 the top of the patient's head with fingers pointed towards the fixation dot. The other hand should
1444 be positioned under the patient's chin. The examiner should then use their top hand to quickly
1445 move the hand towards and away from the fixation dot in abrupt and unpredictable movements
1446 (this will involve tiling the top of the patient's head towards and away from the fixation dot). The
1447 patient should maintain gaze on target (though, due to the awkward gaze angle, the patient may
1448 require breaks during the test
1449

1450 The canal that is stimulated depends on the gaze angle and the rotation. The anterior canals
1451 are stimulated when the chin moves downwards, while the posterior canals are stimulated when
1452 the chin moved upwards.
1453

Interpretation

Following the vHIT test, the individual tracings are analyzed. Each of the six canals is evaluated for the presence of vestibular dysfunction based on two key parameters: gain and corrective saccades. Neither of these parameters should be used in isolation—proper vHIT interpretation requires careful evaluation of the overall picture illustrated by the tracings.

Though the basics of vHIT assessment can be learned easily, it takes time and perseverance to master vHIT interpretation; it should be noted that some of the complexities of vHIT interpretation fall outside of the scope of this document. Furthermore, as the vHIT is still a newer test, new research is continually emerging to inform practice.

Gain

As previously mentioned, when a patient with normal VOR function's head is rotated, the VOR will generate eye movements that are equal in size but in the opposite direction. The comparison of the movements of the eyes and the head results in a measurement known as "gain." When function is normal, the gain (eyes vs. head) will be very close to 1 when measured. However, when VOR loss is present, the eye movement generated by the VOR will not be proportional to the head movement, resulting in reduced gain (norms may vary across systems; typically <0.8 is abnormal for horizontal canals, <0.7 for LARP/RALP).

There is more than one approach that can be used for calculating gain. Regardless of the approach used, it is important to keep in mind that results can never be evaluated based on gain values alone. For example, covert saccades are fast eye movements that occur during the head movement. Covert saccades can interfere with the gain calculation, making the gain artificially high so that it appears within the normal range of function. In these cases, a closer examination of the individual tracings will often clearly show the initial discrepancy between the eye and the head, followed by the covert saccade (a clear indication of VOR loss).

It is common for vHIT results to include a gain graph, which displays gain across the velocities tested. Typically, each head impulse that was completed is represented as a data point on the graph, and each one is colour-coded according to the canal that was stimulated. The gain graph allows you to observe reductions in gain at a glance. Typically, a separate gain graph will be displayed for each of the paired canals (horizontal, LARP, RALP).

Another common display for vHIT results will show velocity across time for all of the head impulses that were completed; typically, there is a separate velocity graph for each of the six canals. On this display, the tracings for the head and the eye are usually overlaid so that discrepancies between the eye and head, as well as corrective saccades, stand out. This display may be provided in a 2D or 3D format.

Corrective saccades

In addition to the gain analysis, the individual tracings should be evaluated for the presence of corrective saccades. On the velocity graph, corrective saccades appear as very brief, high-velocity "spikes."

The velocity graph of each of the canals stimulated should be evaluated separately by the examiner. Since covert saccades occur while the head is still moving, they will appear before the head "stops" (reaches the zero point) after a head impulse on the velocity display. Overt saccades will occur after the head stops.

1503 It is helpful to take note of consistent patterns of corrective eye movements in the tracings:
1504 when true VOR loss is present, corrective saccades will have a significant velocity and will
1505 appear consistently in the tracings. Significantly reduced gains with no corrective saccades
1506 usually indicate a calibration problem (or incorrect gaze angle for LARP/RALP), while careful
1507 review of tracings that show corrective saccades but normal gains will usually reveal a VOR
1508 loss.
1509
1510 The distribution of corrective saccades (i.e., covert vs. overt) is difficult to predict and varies
1511 widely, even among patients with similar pathology (Blödow, Pannasch, & Walther, 2013).
1512

Clinical Use

1514 Results from vHIT can be used to identify VOR loss. VOR loss may be unilateral or bilateral,
1515 and may be isolated to certain canals. The pattern of dysfunction can be used as a tool to
1516 identify the site(s) of lesion in some cases. For example, patients with vestibular neuritis of the
1517 inferior vestibular nerve will have VOR loss that is isolated to the posterior canal (Macdougall,
1518 McGarvie, Halmagyi, Curthoys, & Weber, 2013). While abnormal vHIT findings normally indicate
1519 peripheral dysfunction, unusual patterns of vHIT results that are not consistent with an end-
1520 organ lesion may warrant further testing to rule out central causes (Zuma e Maia & Luis, 2015).
1521

1522 The results from vHIT can be used to identify patients who would benefit from vestibular
1523 rehabilitation. Patients with VOR loss, particularly those with unilateral or partial bilateral
1524 vestibular loss, have been shown to benefit from prescribed exercises designed to promote
1525 central vestibular compensation (Enticott, O'Leary, & Briggs, 2005).
1526

1527

Contraindications

1529 If the case history reveals a history of neck trauma or immobility, extra care should be taken to
1530 ensure that the test can be attempted safely. If the patient has a very narrow range of motion or
1531 reports pain from neck rotation, medical clearance should be obtained before the examination is
1532 attempted.
1533

1534 Some vHIT equipment has a single camera fixed over one eye; unless the camera can be
1535 switched between the eyes, patients with no vision on the same side as the camera are not
1536 candidates for the test.
1537

Limitations/Troubleshooting

1539 Excessive blinking may partially or completely impede the examiner's ability to interpret a vHIT
1540 examination. The tracings of heavy blinkers may be noisy and/or show overt saccades as the
1541 patient re-fixates from the blink. Inexperienced examiners may misattribute a blink as being a
1542 covert saccade. An overt saccade that follows a clear blink in a tracing should not be attributed
1543 to VOR loss. Excessive blinking can often be sufficiently reduced by re-instructing the patient. If
1544 that is unsuccessful, gently taping the eyelids open will generally solve the problem.
1545

1546 The tracings that result from the vHIT may contain overlay from eye movements that are not
1547 corrective saccades (e.g., congenital nystagmus). Without careful analysis, the resultant
1548 tracings may be misidentified as being abnormal.
1549

1550 The patient must be able to see the target ahead of them. Patients with severe visual
1551 impairments (even while wearing corrective lenses) may not have reliable test results.

1552 Young children can be challenging to assess with vHIT due to their physical size and difficulty
1553 with attention. If pediatric goggle straps are unavailable, modifications can be made on-site. A
1554 video clip played from a smartphone screen may be used as a substitute for a static target when
1555 the patient is unable to maintain focus on a static target.
1556
1557 A very common cause of indeterminate vHIT results is from "goggle slip." Goggle slip usually
1558 occurs when the goggles are too loose or are a poor fit. The nose holds the goggles in place;
1559 therefore, if the patient has a low nasal bridge, the goggles may not sit on the patient's
1560 cheekbones and/or the nasal bridge. The resultant tracings will show eye movements that are
1561 faster than, and occur before, the head movements. Similar tracings may be obtained when the
1562 patient's hair contains hairspray or other hair products; hair with a lot of hairspray in it may move
1563 independently from the head, which may also result in artifact. It is essential that examiners
1564 become skilled at identifying these artifacts and differentiating them from actual pathology.
1565 When testing the horizontal canals, changing grip so that your hands cradle the patient's jaw
1566 line may reduce the extent of the artifact from goggle slip.
1567
1568 It is difficult to completely isolate the canals with vHIT, given the paired nature of the
1569 semicircular canals. The oculomotor command that results from a given head impulse is
1570 determined by both excitatory (ipsilateral to the head impulse) and inhibitory (contralateral to the
1571 head impulse) inputs. As a result, the contralateral side will have some influence on the
1572 ipsilateral tracings, except at very fast head impulses wherein the inhibitory inputs from the
1573 contralateral side are saturated (Weber et al., 2008). This may result in significant unilateral
1574 VOR loss being misidentified as bilateral vestibular loss.

Subjective Visual Vertical (SVV) Test

Definition
1577 The subjective visual vertical (SVV) test is designed to evaluate a patient's ability to accurately
1578 identify "true vertical" without the help of a visual reference. The perception of a significant tilt
1579 from true vertical is a sensitive indicator of an acute unilateral vestibular lesion (mostly utricular)
1580 (Böhmer & Mast, 1999) or a central vestibular lesion affecting the gravitoceptive pathways
1581 (Dieterich & Brandt, 1993).
1582
Procedure
1584 Until recently, SVV test administration was restricted to testing centres with expensive,
1585 specialized equipment and skilled administrators (Zwergal, Rettinger, Frenzel, Dieterich, Brandt,
1586 & Strupp, 2009). Inexpensive alternatives have since been shown to have equivalent diagnostic
1587 accuracy: the "bucket method" can be easily produced with very inexpensive and readily
1588 available materials (Zwergal et al., 2009). Detailed instructions are available online (Cook,
1589 2010). Smartphone-based approaches are also in development (Brodsky, Cusick, Kawai,
1590 Kenna, & Zhou, 2015).
1591
1592 For the bucket method, a bucket is made completely dark inside except for a thin strip of glow-
1593 in-the-dark tape, which becomes the vertical line. The line inside the bucket is aligned with a
1594 piece of weighted string, which is housed outside of the bucket, originating from the middle of
1595 the bucket. A protractor, with its zero point aligned with the weighted string, is also affixed to the
1596 outside of the bucket (Cook, 2010).
1597
1598 The examiner should start with the line in the bucket at a random alignment (either clockwise or
1599 counter-clockwise from vertical) and should ask the patient to look into the bucket such that the

1600 interior fills their entire visual field. The examiner should then move the bucket towards vertical
1601 in small intervals, asking the patient to indicate when they perceive the line to be at true vertical.
1602 The value on the protractor, which represents degrees from true vertical, should then be
1603 recorded. It is recommended that the same procedure be repeated for a total of 10 attempts,
1604 varying clockwise and counter-clockwise (Zwergal et al., 2009).
1605
1606 **Interpretation**
1607 The average of the 10 trials should be calculated to determine the patient's degree of tilt from
1608 true vertical. The normal range may vary depending on the method used, but is typically 0 ±
1609 2.5⁰. Patients with values exceeding 2.5⁰ should be flagged for further investigation.
1610
1611 **Clinical Use**
1612 The SVV test can be used to detect lesions along the vestibular pathway. A detailed overview of
1613 the typical SVV findings for various pathologies falls outside of the scope of this document. For
1614 those hoping to learn more, the article by Zwergal et al. (2009) is an excellent starting point.
1615
1616 An ENT consultation should be recommended when the SVV test results exceed normal limits,
1617 due to the likelihood of central pathology. Peripheral lesions will usually normalize within
1618 approximately 20 days (Zwergal et al., 2009); with the exception of vestibular labs that test
1619 patients who are experiencing acute vertigo, the majority of abnormal findings on SVV will have
1620 origins in the central vestibular pathways.
1621
1622 **Limitations**
1623 Abnormal findings are generally non-specific. Further investigations are needed to determine
1624 the site of lesion.

Computerized Dynamic Posturography (CDP)

1626 **Definition**
1627 Computerized dynamic posturography (CDP) is an objective quantitative method for assessing
1628 upright and in-place balance function under a variety of tasks that simulates the conditions
1629 encountered in daily life. CDP can identify and differentiate among a variety of possible sensory,
1630 motor, and central adaptive impairments to balance control. Although CDP cannot diagnose
1631 pathology or site of lesion, it is a functional test which is complementary to clinical tests such as
1632 videonystagmography (VNG) or rotary chair that help localize and categorize pathological
1633 mechanisms of balance disorders.
1634
1635 **Procedure**
1636 There are several subtests that can be performed with posturography testing, including sensory
1637 organization testing (SOT), motor control testing (MCT), and adaptation testing (ADT).
1638 Throughout all subtests, the patient wears a harness to ensure safety. Once the patient is facing
1639 the surround, the straps of the harness are connected to the machine so that they are parallel to
1640 the ground on the patient's shoulder. The harness should fit comfortably without being too loose
1641 or too tight. During testing, the patient stands on a moveable, dual force plate support surface
1642 within a moveable surround or enclosure. The patient's feet should be aligned to the platform
1643 they are standing on according to their measured height (small = 114–140 cm, medium = 141–
1644 165 cm, and large = 166–203 cm). The medial malleolus and the lateral calcaneous are aligned
1645 on the marking stripe that transects the two force plates. The feet should be aligned throughout
1646 the testing. If the feet move out of place, testing should be stopped and resumed after
1647 realignment.

Sensory Organization Test

The SOT tests the ability of an individual to effectively process individual sensory system input cues to maintain balance control. This is done by suppressing inaccurate sensory system inputs while selecting appropriately from other, more accurate sensory cues to generate appropriate motor and postural response strategies. The SOT test assesses this ability, objectively isolating and quantifying the use of each sensory system and the adaptive (or maladaptive) responses of the central nervous system. This is done by taking away sensory input (eyes closed) or making sensory input inaccurate (by having the force plate or enclosure sway referenced). Body sway is calculated by measuring the centres of vertical force movements in six different conditions (see Table 7). Three trials of each condition are performed. If the patient stumbles, reaches out for support, or falls, then this should be marked as a fall. Other measures calculated during SOT are centre of gravity alignment, equilibrium scores, and strategy analysis (hip versus ankle).

- **Centre of gravity (COG) alignment:** Reflects the position of the COG relative to the centre of foot support and is measured in degrees.
- **Equilibrium score:** Compares the maximum patient anteroposterior sway angle to the patient's theoretical limits of stability. This is calculated for each trial. If the score is near 100% it indicates little sway and if it is near 0% it indicates the sway is nearing the limits of stability.
- **Strategy Analysis:** The use of movement about the ankle, hips, and upper body to maintain balance during the SOT is reflected in the strategy scores. Strategy scores are calculated by comparing the peak-to-peak amplitude of the shear oscillation to the maximum possible shear of 11.4 kg. Scores near 100% indicates little shear (full ankle strategy) while scores near 0% indicate maximum shear (full hip strategy).

Motor Control Test

Automatic postural reactions are the primary source for balance correction responses after an unexpected perturbation or surface change because it is the earliest response helping to re-centre the body's centre of gravity over the base of support when standing. This test is independent of conscious control. The responses occur within 90 to 100 milliseconds in coordinated patterns. The force plate is moved suddenly with three trials per condition (small, medium, and large horizontal perturbations) in the forward and backward direction. Weight symmetry, latency, and amplitude scaling are measured.

Table 7: The SOT Six Sensory Conditions

Condition	Vision	Surface	Disadvantaged	Using
1	eyes open	fixed	none	somatosensory
2	eyes closed	fixed	vision	somatosensory
3	sway referenced visual	fixed	vision	somatosensory
4	eyes open	sway referenced surface	somatosensory	vision
5	eyes closed	sway referenced surface	somatosensory & vision	vestibular

| 6 | sway ref. visual | sway referenced surface | somatosensory & vision | vestibular |

1683

Adaptation Test

This test assesses the patient's ability to adapt to disruptive somatosensory input caused by unexpected changes in the orientation of the support surface. The test involves five randomized trials of the force plate moving at 20 deg/s with a toes-up and toes-down condition. The response is measured by the force (sway energy) produced during the first two seconds. The appropriate patient response is to remain upright, using enough force to return the body to upright and to dampen or decrease the amount of energy required with each subsequent trial.

Interpretation & Clinical Use

The sensory analysis of the SOT compares the equilibrium scores among the six sensory conditions. The sensory analysis ratios result in patterns which describe a patient's ability to effectively use sensory system cues for balance control which can provide an explanation for a patient's functional complaints and the basis for specific treatment decisions (see Table 8). It is important to note that caloric testing can identify and lateralize a lesion but cannot determine the level of functional compensation.

Functional Impact of Sensory Dysfunction in SOT

- **Vestibular Dysfunction Pattern:** Patients perform normally on firm support surfaces and/or in the presence of strong visual cues. They will typically experience instability on irregular surfaces, in low light conditions, and in active visual environments.
- **Visual and Vestibular Dysfunction Pattern:** Patients are somatosensory dependent and require a stable support surface reference to maintain balance. Without a stable surface they do not make effective use of either vestibular or visual inputs.

Table 8: Sensory Organization Test Interpretation

Pattern of Dysfunction	Over-Dependence On	Problem SOT Conditions	Analysis Ratio
1. Vestibular	Visual & Somatosensory	5, 6	5/1
2. Visual & Vestibular	Somatosensory	4, 5, 6	(4/1) + (5/1)
3. Somatosensory & Vestibular	Vision	2, 3, 5, 6	(2/1) + (5/1)
4. Visual Preference	Vision	3, 6	(3+6) / (2+5)
5. Vestibular & Visual Preference	Vision	3, 5, 6	(5/1) + ((3+6)/(2+5))

- **Somatosensory and Vestibular Dysfunction:** Patients are vision-dependent at all times to maintain balance. They are destabilized with vision absent or impaired (e.g., low lighting, moving situations).

- **Visual Preference:** Patients are destabilized by orientating with inaccurate visual stimuli (e.g., moving quickly in complex visual environments) although they perform normally in the absence of vision.
- **Vestibular Dysfunction and Visual Preference Pattern:** Patients are destabilized by orientating with inaccurate visual stimuli although they perform normally in the absence of vision. They also experience instability on irregular surfaces when visual cues are absent.
- **Across the Board:** Patients have decreased postural control regardless of the sensory condition they are functioning in. They are unstable or symptomatic any time they change from one sensory situation to another.

Motor Control Test

- **Weight Symmetry:** Values to the right or left of normal limits show a disproportionate amount of body weight is being carried by the right or left leg.
- **Latency:** Abnormal latency in both translation directions with both extremities suggests a problem within the long latency pathways involving the peripheral and motor nerves, the ascending sensory and descending motor pathways of the spinal cord, and/or the motor regions of the brainstem and cerebral cortex. If delays are measured in both directions, additional medical examination is required. Patients who have abnormal latencies due to central or peripheral nervous system pathologies have limited capacity to improve automatic or rapid functional postural responses.
- **Amplitude Scaling:** When strengths are bilaterally weak, the patient may be ineffective at quickly and accurately compensating for perturbations. The result could be increased sway oscillations under all sensory conditions. When strengths are bilaterally too strong, the patient may tend to overshoot the centred position and oscillate. Unilateral response strength abnormalities may have functional implications in gait due to an offset COG.

Adaptation Test

An effective response suggests adequate motor planning and learning, musculoskeletal status, balance response strategies, emotional state and level of anxiety. An ineffective response suggests the patient may be at risk for loss of balance in similar functional situations.

For a list of validity and efficacy studies please go to: http://balanceandmobility.com/ for-clinicians/ computerized-dynamic-posturography/validity-and-efficacy-studies/

Contraindications

There are weight and height restrictions for testing. The platform can accommodate people weighing between 18 and 136 kg with a height of 76 to 203 cm. Patients who cannot stand erect and unsupported for more than 2 to 3 minutes or who lose their balance when standing on a fixed surface with their eyes closed cannot be tested.

Limitations:
- By itself, CDP testing cannot diagnose pathology or site of lesion.
- Alcohol and certain medications, such as sedatives, anti-nausea medication, anti-histamines, diuretics, tranquilizers, and anti-depressants, can affect test results if taken within 48 hours.
- The distribution of weight can influence latency, response strength, and strength symmetry scores and should be taken into account when interpreting those scores.

- Results can be affected by patients who have secondary gain or anxiety, or those who want to demonstrate their deficits from a real pathology to their clinician. Objective aphysiologic patterns can be determined from the SOT and MCT and can assist the clinician in analyzing the test data, such as high inter-trial variability on all SOT trials, conditions 5 and 6 relatively better than conditions 1 and 2, and/or inconsistent MCT responses (Mallinson, 2014).

If CDP testing is not available, foam posturography can be used as a functional measurement tool such as the Clinical Test of Sensory Integration and Balance (CTSIB) or the Gans Sensory Organization Performance (SOP) Test.

Electrocochleography (ECochG)

Electrocochleography is a test that has been used to investigate the presence of Ménière's disease. While the test may have diagnostic value, there is a lack of consensus regarding norms, technique, and interpretation in the literature. A detailed exploration of these many factors falls outside of the scope of this document. Audiologists looking to perform this test are encouraged to carefully review the available literature.

Vestibular Management

Choosing Wisely Canada (CWC) is a multi-disciplinary campaign to help physicians and patients engage in conversations about unnecessary tests and treatments and make smart and effective choices to ensure high-quality care. The Canadian Society of Otolaryngology-Head and Neck Surgery created a list of five things physicians and patients should question, including information on testing dizzy patients. They recommended:

(Not to) order specialized audiometric and vestibular neurodiagnostic tests in an attempt to screen for peripheral vestibular disease. The diagnosis of the dizzy patient should be guided by the presenting symptoms and office examination. Tests such as ABR (auditory brainstem response), ECOG (electrocochleography), ENG/VNG (electronystagmography/videonystagmography), VEMP (vestibular evoked myogenic potential), vHIT (video head impulse test), CDP (computerized dynamic posturography), and RTC (rotational chair testing) should only be ordered if clinically indicated. In general, advanced balance tests should be ordered and interpreted by otolaryngologists with specialized training in the diagnosis and treatment of vestibular disorders (otologists/neurotologists). Clinical indications for testing can include: side localization and stage of progression for Ménière's disease, assessment of central compensation for acute vestibular loss, and confirmation of superior semicircular canal dehiscence syndrome. Specialized tests are rarely indicated in the management of benign paroxysmal positional vertigo. (Canadian Society of Otolaryngology-Head and Neck Surgery, 2016)

A second suggestion from the CSOHNS was to:

(Not) perform particle repositioning maneuvers (Epley or Semont) without a clinical diagnosis of posterior semicircular canal benign paroxysmal positional vertigo in the affected ear. Posterior semicircular canal benign paroxysmal positional vertigo should be diagnosed and confirmed with a positive Dix-Hallpike test, and only then

1807   should a particle repositioning maneuver be performed. If a patient with positional
1808   vertigo has a Dix-Hallpike test that is repeatedly negative or results in atypical
1809   nystagmus, less common BPPV variants or central positional vertigo should be
1810   considered (Canadian Society of Otolaryngology-Head and Neck Surgery, 2016).
1811

1812 It is recommended that audiologists who order and/or interpret advanced balance tests or who
1813 perform particle repositioning maneuvers should also follow these recommended guidelines.
1814

Vestibular Rehabilitation

1816 Detailed information about vestibular rehabilitation is beyond the scope of this current guideline;
1817 however, some general information will be provided here.
1818

1819 Patients with acute BPPV will benefit from particle repositioning maneuvers. The specific
1820 treatment maneuver to be used will depend on the affected semicircular canal (Hilton & Pinder,
1821 2014; Hunt, Zimmerman & Hilton, 2012; Helminski, Zee, Janssen & Hain, 2010).
1822

1823 In general, there are three main types of vestibular rehabilitation exercises: adaptation,
1824 habituation, and substitution. The goal of adaptation exercises is to improve or strengthen the
1825 VOR function. The goal of habituation exercises is to desensitize the patient to certain
1826 symptom-provoking movements. The goal of substitution exercises is to use other senses to
1827 replace (substitute) vestibular input—e.g., using additional visual or proprioceptive input
1828 (Brodovsky & Vnenchak, 2013).
1829

1830 Both audiologists and physiotherapists can receive training and certification in the area of
1831 vestibular rehabilitation. It is recommended that vestibular clinicians network with available
1832 vestibular rehabilitation specialists in their geographic area.
1833

1834 For those vestibular audiologists looking to receive additional training in the area of vestibular
1835 rehabilitation, these are some helpful resources:
1836   • The American Institute of Balance:
1837   http://dizzy.com/education_foundation.htm
1838   • Vestibular Disorders Association:
1839   http://vestibular.org/resources-professionals/build-your-practice
1840

Pediatric Population

1842 Detailed information about pediatric vestibular testing is beyond the scope of this current
1843 guideline; however, some general information will be provided here. It is important to remember
1844 that very little normative data is available in pediatric vestibular evaluation, and many of the
1845 results are simply reported as present/absent (see Table 9).
1846

1847 Like other areas of pediatric testing, it is important to make the testing as engaging as possible
1848 and modify your technique accordingly. An example of making vestibular testing more pediatric-
1849 friendly would be to develop a fantasy storyline. For example, explain that the enclosed rotary
1850 chair is like a pretend spaceship, and that the pediatric patient is going to be going for

1851 "astronaut training." A similar approach can be used during caloric testing with pediatric
1852 patients.
1853
1854 Patient case history is critical for assessment of the pediatric vestibular patient (see Tables 1, 2,
1855 3, and 4). It is important to note that up to 70% of children presenting with sensorineural hearing
1856 loss have impairment of their vestibular system with 20–40% having severe bilateral vestibular
1857 loss (Cushing, Gordon, Rutka, James, & Papsin, 2013; Cushing, Papsin, Rutka, James, &
1858 Gordon, 2008; Cushing, James, Papsin, & Gordon, 2008; Buchman, Joy, Hodges, Telischi, &
1859 Balkany, 2004).
1860
1861 Some children with dysfunction of the vestibular portion of the inner ear will never be
1862 vertiginous. This is particularly likely if this dysfunction is severe, congenital, and/or bilateral.
1863 Impairment of the vestibular end organs may be suggested if there are delayed motor
1864 milestones (e.g., poor head control [beyond 6 weeks old], delayed independent sitting [beyond 9
1865 months], and delayed walking [beyond 18 months]) (Cushing, 2014).
1866

1867 **Table 9: Vestibular Tests and Pediatric Patient Age (Young, 2015)**

Test	Newborn	<3 Years	3-14 Years	Age of Adult-like Response
Rotary chair	+	+	+	1 year
Calorics	+	+	+	2 years
oVEMP	-	+	+	3 years
Dynamic visual acuity test (DVAT)	-	-	+	3 years
Foam posturography	-	-	+	12 years
CDP	-	-	+	15 years
cVEMP	+	+	+	Adolescent

Importance of Counselling

1869 Depending on the clinic's standards of practice, as well as information outlined by the applicable
1870 regulatory body, the depth of interpretation for vestibular test results will vary. Some provinces
1871 or territories may not allow audiologists to give a specific diagnostic indication based on the
1872 vestibular test results. Despite not giving a specific diagnosis, the audiologist can assist their
1873 patients by educating them about dizziness and balance terminology, discussing tips on how to
1874 improve communication with their referring physicians about their case histories, educating
1875 them about the vestibular tests that are available and what they measure, explaining general
1876 patient management pathways, highlighting the importance of stress management, discussing
1877 fall prevention, and detailing what other professional resources or support groups are
1878 available.
1879

1880 Provision of printed education materials regarding vestibular disorders and management may
1881 be useful for audiologists during counselling. In addition, audiologists may find it useful to
1882 consult anatomical charts or models to illustrate points of possible dysfunction within the
1883 vestibular/balance system(s).
1884
1885 For patients diagnosed with vestibular migraine, migraine triggers can be discussed—such as
1886 triggers related to diet-, barometric pressure, fatigue, hormones, and/or stress. Similarly, diet-
1887 related triggers can be monitored for patients suspected of having Ménière's disease or
1888 endolymphatic hydrops. Suggesting the use of a "dizzy diary" can be useful for patients to
1889 monitor and record their symptoms over time and to look for provoking factors, links, or trends
1890 between their symptoms.

Reporting/Documentation

1891

1892 It is important to ensure that clinicians are adhering to standards and regulations regarding
1893 clinical documentation developed by provincial and territorial regulatory bodies, national
1894 associations, and their employers. Adherence to freedom of information and privacy laws is
1895 also. It is up to each audiologist to ensure they are in compliance with the laws in their own
1896 region.
1897
1898 Distribution of the patient report should include information for the referring clinician, as well as
1899 copies for any other health professional as requested by the patient.
1900
1901 At a minimum, the report should include diagnostic test results and an analysis of whether the
1902 results are normal or outside normal limits. Reporting should also include case history,
1903 interpretations/impressions, and recommendations for management and follow-up.
1904
1905 Audiologists should avoid basing interpretations/impressions on one single diagnostic finding;
1906 instead, take overall test results and patient case history into consideration.
1907

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