Vestibular Assessment & Management for Canadian Audiologists: A Scoping Review

September 2016

Draft

Canadian Academy of Audiology
Heard. Understood.
Academie canadienne d'audiologie
Entendus. Compris.
CAA ACA

SAC OAC
Speech-Language & Audiology Canada
Orthophonie et Audiologie Canada
Communicating care | La communication à coeur

Table of Contents

Authorship Committee 3
Stakeholder Acknowledgement 3
Introduction 4
Professional Competence Statement 4
Update & Review 4
Disclaimer 5
Background 5
  Vestibular Statistics 5
  Consequences of Vestibular Dysfunction 6
  Population Statistics 6
Interprofessional Team Approach 7
Patient Safety 7
Current Status of Canadian Audiology Scope of Practice & Certification 8
  Provincial/Territorial 8
  National 8
Knowledge Required 10
Vestibular Assessment 11
  Case History 11
  Oculomotor Testing 13
  Positioning Tests 17
  Positional Testing 20
  Caloric Irrigation Testing 21
  Rotary Chair Testing 24
  Vestibular Evoked Myogenic Potentials (VEMPs) 26
    Cervical Vestibular Evoked Myogenic Potential (cVEMP) 26
    Ocular Vestibular Evoked Myogenic Potential (oVEMP) 29
  Video Head Impulse Test (vHIT) 31
  Subjective Visual Vertical (SVV) Test 36
  Computerized Dynamic Posturography (CDP) 37
  Electrocochleography (ECochG) 41
Vestibular Management 41
  Vestibular Rehabilitation 42
Pediatric Population 42
Importance of Counselling 43
Reporting/Documentation 44
References 45

Authorship Committee

Janine Verge, AuD, Aud(C) Nova Scotia Hearing and Speech Centres, Nova Scotia (Chair)
Ronald Choquette, MOA, AuD, University of Montreal, Quebec
Carolyn Falls, M.Cl.Sc., Reg. CASLPO, University Health Network, Ontario
Kathy Packford, M.Sc., R.Aud, Alberta Health Services, Alberta
Yvette Reid, M.Sc., RAUD, Providence Health Care, British Columbia
Uta Stewart, AuD, Saskatoon Health Region, Saskatchewan
Michael Vekasi, M.Cl.Sc., R.Aud, Alberta Health Services, Alberta
Erica Zaia, M.Sc., RAUD, Providence Health Care, British Columbia

Stakeholder Acknowledgement

The authorship committee would like to sincerely thank the many reviewers of this document, as well as the Canadian Academy of Audiology and Speech-Language and Audiology Canada, for their support.

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

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

Consequences of Vestibular Dysfunction

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

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

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

Population Statistics

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

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

Interprofessional Team Approach

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

Patient Safety

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

"Members and associates shall:

- Take every precaution to avoid harm to patients or clients. This includes following applicable occupational health and safety and infection prevention and control practices, and ensuring that equipment is appropriately calibrated and in proper working order.
- Ensure that their employees and/or supervised personnel comply with relevant occupational health and safety and infection control policies and procedures (SAC, 2016a)."

For vestibular assessment and management, the following patient safety measures should be addressed:

1. Follow proper infection control precautions as outlined in *The Infection Prevention and Control Guidelines for Audiology*, written by the Canadian Interorganizational Steering Group for Speech-Language Pathology and Audiology (2010).
2. Advise patients appropriately to stop certain medications (e.g., vestibular sedatives) at least 48 hours before undergoing vestibular testing. It is important that patients are advised to consult with their referring physician regarding these medications. The referring physician should advise the patient which medications should be stopped and the audiologist should verify that the patient has adhered to this advice prior to vestibular testing (British Society of Audiology, 2014).
3. Stop testing and/or treatment if there are any complicating or medical contraindications.
4. Obtain informed consent prior to vestibular testing or treatment, with information given about the specific known risks of each test. Patients should be informed that vestibular testing may temporarily cause an increase in their imbalance. It is recommended that patients come to their appointments accompanied by someone to reduce the risk of fall and any difficulty driving following testing (Accreditation Canada, Canadian Institute for Health Information, & Canadian Patient Safety Institute, 2014).
5. Create a falls prevention strategy to minimize patient injury from falls in the workplace. The Required Organizational Practice (ROP)'s "Falls Prevention Strategy" requires clinics to implement and evaluate a falls prevention strategy. All populations at risk must

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

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

Current Status of Canadian Audiology Scope of Practice & Certification

Provincial/Territorial

A committee review of Canadian provincial/territorial professional associations and/or regulatory bodies revealed no specific scopes of practice or guidelines for vestibular practice.

The College of Speech and Hearing Health Professionals of BC is the only regulatory board to date requiring audiologists to obtain an Advanced Competency Certificate in order to perform vestibular assessment and management. It involves successfully completing a graduate-level course of at least four credit hours in vestibular system function and assessment or a program of study that the committee agrees is similar to a graduate-level course (College of Speech and Hearing Health Professionals of BC, 2014). An active registrant must be certified by the College's registration committee (College of Speech and Hearing Health Professionals of BC, 2014).

For a listing of general requirements to obtain vestibular certification for the College of Speech and Hearing Health Professionals of BC, please refer to:
http://www.cshhpbc.org/docs/cshhpbcbylaws.pdf

National

Both the Canadian Academy of Audiology (CAA) and Speech-Language and Audiology Canada (SAC) include vestibular assessment and management within their scope of practice documents and position statements for the field of audiology.

The CAA *Position Statement on Audiology Scope of Practice* (2002) states:

- An audiologist is an independent, professional provider of primary hearing health care, who specializes in the prevention of hearing loss and in the identification, assessment, diagnosis, 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.

To review the complete SAC *Scope of Practice for Audiology* please refer to: http://www.sac-oac.ca/sites/default/files/resources/scope_of_practice_audiology_en.pdf?_ga=1.239600256.2084794186.1403892485

Knowledge Required

As mentioned above, audiologists should obtain additional and ongoing education and training and, in British Columbia, must obtain an Advanced Competency Certificate in vestibular assessment and management.
The purpose of this section is to provide the audiologist with guidance on the knowledge required to practice in the area of vestibular assessment and management.

The audiologist should gain:

- knowledge of anatomy and physiology of the peripheral and central vestibular systems and their connections with the hearing, visual, and somatosensory systems;
- knowledge of pathophysiology of vestibular and balance disorders (otologic and non-otologic);
- experience obtaining relevant case history;
- knowledge of self-report measures such as the Dizziness Handicap Inventory (Jacobson & Newman, 1990);
- the ability to identify contraindications to vestibular assessment or portions thereof;
- understanding of the effects of different medications and substances on test results;
- knowledge and experience in administering and interpreting various vestibular assessment techniques, including but not limited to:
    - bedside tests of vestibular function, such as head impulse testing, oculomotor testing (gaze-evoked nystagmus, saccades, optokinetics, and tracking), head shake testing, hyperventilation testing, closed glottis straining, and external canal pressurization (fistula test);
    - pre-testing screenings, such as vertebral artery screening test and cervical vertigo test;
    - subjective visual vertical test;
    - dynamic visual acuity test (DVAT);
    - video head impulse test;
    - electronystagmography (ENG) or videonystagmography (VNG), including oculomotor testing, positioning and positional testing, and caloric testing (air, water, and/or ice water);
    - electrophysiological tests such as electrocochleography and vestibular evoked myogenic potentials (cervical and ocular);
    - postural stability tests and computerized dynamic posturography
    - autorotation and rotary chair tests
- knowledge of repositioning maneuvers for treatment of different forms of benign paroxysmal positional vertigo;
- understanding of vestibular rehabilitation therapy: identifying candidates and providing management strategies and available resources;
- the ability to identify contraindications for vestibular rehabilitation therapy;
- the ability to identify clients who are at risk for falls and provide management strategies and resources;
- 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

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

Table 2: Examples of vertigo syndromes according to duration of symptoms (adapted 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

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

Table 3: Examples of triggering factors and possible associated vertigo syndromes (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

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

Table 4: Examples of accompanying symptoms and possible associated vertigo 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

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

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

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

Oculomotor Testing

Definition

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

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

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

A. Gaze Stability

Procedure

This test evaluates a patient's ability to maintain a gaze fixed on a target while looking straight ahead (primary position), 30° to the right and left, and 20° up and down, without any abnormal eye movements (such as nystagmus). If nystagmus is present, the response must be tested without visual fixation to differentiate between a peripheral and central site of lesion.

Interpretation

Any nystagmus evoked must be recorded in terms of its direction (of the fast-phase), velocity, evoking position(s) and whether the nystagmus follows Alexander's Law (i.e., does the velocity of the nystagmus increase when the gaze is to the same direction as the fast-phase of the nystagmus?). Nystagmus seen in the primary position is referred to as spontaneous nystagmus; nystagmus in the lateral or vertical positions is known as gaze-evoked nystagmus.

Clinical use

Abnormalities may be caused by a peripheral or central vestibular dysfunction (Shepard & Schubert, 2016). Nystagmus may be considered peripheral if the nystagmus is stronger without fixation, is direction-fixed, and has a primarily horizontal component. If the lesion is peripheral, the nystagmus should also follow Alexander's Law (as described above). The nystagmus usually beats away from the involved side, but in some unusual cases (such as in the irritative phase of Ménière's disease or with hyperventilation with retrocochlear pathology) the nystagmus can beat towards the involved side.

Primary position nystagmus of central origin may be purely vertical or purely torsional; it is rarely horizontal. Central nystagmus is more likely to be direction-changing (e.g., right-beating with right gaze, left-beating with left gaze, etc.) than direction-fixed, but both are possible, and it does not increase in velocity with fixation removed (Shepard et al., 2016). Horizontal or pure torsional gaze-evoked nystagmus of central origin is usually caused by a lesion in the brainstem. Vertical gaze-evoked nystagmus can result from lesions in the brainstem or cerebellum.

Saccadic intrusions and oscillations may be recorded during gaze testing, especially with primary gaze testing. These are seen with fixation present and are suggestive of cerebellar and/or brainstem involvement.

B. Saccades

Procedure

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

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

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

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

Procedure

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

Interpretation

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

Clinical use

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

Considerations & Limitations of Oculomotor Testing

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

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

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

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

Square wave jerks (in which the eyes briefly move away from the target and return) will be seen in some patients and is usually normal, especially in older patients. Normal square wave jerks should occur less than 20 to 30 times per minute, be at least 200 ms apart, and be between 1 and 5º in amplitude. Square waves that occur more often suggest a cerebellar or brainstem lesion. A high amplitude (above 5º) suggests a brainstem or cerebellum pathology (Shepard et al., 2016).

Certain medications, such as sedatives, anti-nausea medication, antihistamines, diuretics, tranquilizers, and antidepressants, can result in abnormal nystagmus or suppressed nystagmus. Please see Cass and Furman (1993) for details.

Contraindications to Oculomotor Testing

There are few contraindications to oculomotor testing. The patient must be able to see the target without glasses (but may wear contacts) and be able to tolerate the VNG goggles. Dark eye makeup may result in inaccurate pupil tracking and should be removed.

Ocular motility disorders (e.g., myopathy, palsies) will affect the accuracy of the testing, as will disconjugate eye movements (e.g., intranuclear ophthalmoplegia, strabismus, amblyopia).

Positioning Tests

Definition

Positioning maneuvers are used to test the posterior, anterior, and horizontal semicircular canal(s) (SCC) for displaced canaliths, such as displaced otoconia or cupuliths in benign paroxysmal positional vertigo (BPPV. They differ from positional evaluations because they require active transition from one position to another.

BPPV is the most common cause of vertigo in patients with vestibular disorders (Blatt, Georgakakis, Herdman, Clendaniel, & Tusa, 2000). BPPV is thought to occur when calcium carbonate crystals, referred to as otoconia, dislodge from the otoliths and enter one of the SCCs. The displaced otoconia are more commonly free-floating, which is referred to as canalithiasis; however, they can also adhere to the cupula, which is referred to as cupulolithiasis. Due to the anatomical location of the posterior SCC, displaced otoconia more commonly enter the posterior SCC; however, there are cases of BPPV involving the horizontal and anterior SCC.

A. Dix-Hallpike

Procedure

The traditional Dix-Hallpike involves moving the patient from a sitting position to a position likely to provoke a response (such as a head-hanging position) and then returning to a sitting position. This can be done using the VNG goggles or Frenzel goggles; however, it is beneficial to be able to review recorded eye movements using the VNG goggles.

To perform this procedure, have the patient sit on a flat surface. Ensure they are far enough from the head of the reclined surface so that their head will be extended over the edge when they lie supine. Instruct the patient to keep their eyes open, looking forward with their vision denied. Turn the head 45° to the right. Place your hands on either side of the patient's head and

quickly but carefully have the patient recline into a head-hanging position, maintaining the head turned 45° to the right. Ensure that the test ear is lower than the patient's shoulder. Have the patient stay in this position for approximately 30 seconds (or until after the nystagmus stops), then ask them to sit back up, keeping their head turned 45° to the right. Have the patient stay in this position for another 30 seconds, and then they may relax to a neutral sitting position. If a positive Dix-Hallpike is observed, repeat the Dix-Hallpike on the same side to check for fatigability of the response. Then repeat the whole procedure for the head turned 45° to the left (Halker, Barrs, Wellik, Wingerchuk & Demaerschalk, 2008).

Dix-Hallpike Modifications

- **Fully supported Hallpike:** For patients who cannot tolerate the traditional or side-lying procedure. This modification uses the same procedure as the traditional Dix-Hallpike, but the patient's head remains fully supported on the reclined chair or table, and does not hang below the shoulder (Trendelenberg position).
- **Side-lying maneuver:** For patients with difficulty lying flat on their back. The patient sits in the middle of the table with their feet hanging off to one side. Turn their head 45° to the right and instruct the patient to lie down on their left side, maintaining the head turn while having them swing their legs up on the table. Repeat this with the head turned 45° to the left and lying down on their right side.

Interpretation

Classic positional vertigo will result in nystagmus that is consistent with the plane of the affected canal while it is stimulated. Vertigo is usually reported while nystagmus is observed. A central nervous system involvement is suspected when pure vertical or pure torsional nystagmus is elicited. Vertigo is not commonly reported when central positional nystagmus is present. Dix-Hallpike is positive for posterior SCC BPPV if non-sustained torsional and up-beating nystagmus is observed in the head-hanging position, with or without reversal upon sitting. Anterior SCC BPPV also results in transient torsional nystagmus but with down-beating rather than up-beating nystagmus. For the posterior canals, the torsion beats to the affected ear. A positive response should fatigue on repeat of the Dix-Hallpike on the same side (see Table 5).

Table 5: Dix-Hallpike Results for Posterior and Anterior BPPV

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

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

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:

- carotid sinus syncope (if history of drop attacks or blackouts)
- severe back or neck pain
- recent stroke
- cardiac bypass surgery within last 3 months
- rheumatoid arthritis, affecting the neck
- recent neck surgery
- cervical myelopathy
- severe orthopnea, may restrict duration of test

(British Society of Audiology, 2001)

Limitations

A positive BPPV response will fatigue, therefore it is important to test the suspected side first and before doing any supine positional testing.

Positional Testing

Definition

Positional testing examines the presence or absence of nystagmus with or without subjective dizziness, due to changes in position of the head or the head and body relative to the force of gravity. This differs from positioning tests because the head and body are static during positional testing.

Positional nystagmus is thought to occur as a result of reduction in suppression of asymmetrical SCC function due to impaired otolith function or central vestibular pathway involvement. The rationale of testing for nystagmus in various positions is to allow for change of the orientation of the labyrinth in relation to gravity. Testing starts with measuring any nystagmus that occurs in a neutral position called spontaneous nystagmus (SN). Eye movements are recorded with the patient wearing VNG goggles with vision denied. The patient is asked to keep eyes open, looking forward in a neutral sitting position. This is repeated with visual fixation.

Positions used for positional testing are:

- **Sitting:** Eye movements are recorded with the patient wearing VNG goggles with vision denied.
- **Supine:** Eye movements are recorded with the patient wearing VNG goggles with vision denied, and patient supine with head supported at centre, repeated with head turned 90° to the right, and then repeated with head turned 90° to the left. Individuals with restricted neck movement will require a modified angle of head turn.
- **Lateral/Positional Body:** Eye movements are recorded with the patient wearing VNG goggles with vision denied and patient's head and body to the right, and repeated with patient's head and body to the left. N.b., for each positive positional test, a test for suppression of the nystagmus with fixation is completed.

Interpretation

Positional nystagmus that is present without fixation may be peripheral or central in nature. Lack of suppression of the nystagmus when measurement is repeated with visual fixation suggests a central origin. If nystagmus does not alter with positional change, it can be interpreted as purely spontaneous nystagmus. SN that changes direction is indicative of CNS involvement. SN related to unilateral vestibulopathy (peripheral) is usually directionally fixed and suppresses with 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

assess the function of the peripheral vestibular system, specifically of the horizontal semicircular canal and superior vestibular nerve.

Within the semicircular canal ampullae, the endolymph and cupula are the same density. The temperature change delivered by calorics changes the density of the endolymph and it therefore deflects the cupula in the lateral semicircular canal. Warm stimuli results in an endolymph rise towards the ampulla and an excitatory neural response; cool stimuli, the opposite. There may also be an effect of temperature on the vestibular nerve (Barin, 2016b). As neural firing is unchanged in the non-test ear, the asymmetry in neural firing is perceived by the VOR as a rotation towards the test ear and the eyes move accordingly (Barin, 2016a).

Nystagmus can be recorded using video goggles (known as VNG) or electrodes (ENG). As VNG is the industry standard, ENG will not be covered here.

Procedure

The patient's head is raised approximately 30° to position the horizontal canal vertically. The temperature of the air or water is a set amount above or below body temperature, typically 44 and 30 °C for water.

With visual fixation removed (e.g., by covering the goggles), either the right or left ear canal is irrigated with warm water or air for 30 to 40 seconds. Warm caloric irrigations should be tested first if use of the monothermal caloric screening test is under consideration (see below). The suspect ear should be tested first; if the patient declines further testing after the first caloric then the audiologist will have at least collected information about the ear of interest (Lightfoot, 2004). The patient's eye movements are recorded with either VNG goggles or electrodes (if ENG is used). If the patient appears to be suppressing the nystagmus, the examiner should ask the patient questions to distract them (this is known as tasking or mental alerting). When the nystagmus begins to slow, the patient is asked to fixate on a target (e.g., a light in the goggles) and the nystagmus is observed for suppression. The peak velocity of the nystagmus without fixation is then measured for each irrigation performed. As in other vestibular tests, nystagmus is named according to the direction of the fast-phase, and its velocity is measured using the slow-phase. The nystagmus should beat in the direction of the irrigated ear for warm water, and away from the irrigated ear for cool water. This is summarized by the acronym COWS (cold opposite; warm same).

When possible, water should be used as the stimulus, as it evokes a stronger response and results are less variable (Goncalves, Felipe, & Lima, 2008). However, in the presence of contraindications to water (see below), then air may be used. Please see the reference for more detail about the testing procedure.

Monothermal Caloric Screening Test

If warm caloric irrigations are performed first and the asymmetry is less than 15%, then it is possible to predict a normal outcome with 95% sensitivity. However, both warm calorics must be greater than 8 deg/s and any spontaneous nystagmus must be below 4 deg/s to use this screening test (Lightfoot et al., 2009).

Ice Calorics

Ice calorics may be used to determine if any vestibular function remains at low frequencies. This test is appropriate if bithermal calorics do not evoke nystagmus (or the nystagmus is very weak) 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:

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

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.

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

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).

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

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

account for the head rotation that took place and retinal slip will occur. Patients with bilaterally reduced VOR function may present with symptoms of oscillopsia or visual blurring during head rotations.

The rotational chair measures the VOR across multiple frequencies of rotation, allowing simultaneous stimulation of the horizontal semicircular canals. In addition, the rotation chair provides information about the overall function of the system during angular acceleration of the head.

Procedure

Rotational chair testing is generally completed in a darkened room with a separate area housing the computer for analysis. The patient is secured in the chair with a head restraint and harness to ensure safety during testing. A two-way communication system allows the patient to remain in contact with the examiner throughout the test. The patient's head should be tilted down at a 30° angle to align the canals with the axis that is being stimulated during the test (Arriaga, Chen & Cenci, 2005). There are a number of rotational chair tests that are clinically useful for assessing the VOR.

Sinusoidal (or Slow) Harmonic Acceleration Test: In this test, the rotational chair moves at incremental frequencies in a sinusoidal pattern. The most common octave frequency range is between 0.01 Hz and 2 Hz. This allows the measurement of eye movements at velocities of rotation ranging from 20 to 80 deg/s. Data is averaged over a minimum of 3 cycles for each frequency to compute gain, asymmetry, and phase.

Velocity Step Test: The velocity step test involves rapid acceleration of the chair (100–200 deg/s) until the chair reaches a predetermined velocity. The velocity is then maintained for a period of time (60 seconds is typical) and then rapid deceleration occurs. The test is repeated for both clockwise and counter-clockwise directions at fixed velocities. Nystagmus during rotation is compared to nystagmus in the post-rotary condition to compute the time constant and gain.

Visual Fixation Suppression Test: Goulson, McPherson, and Shepard (2016) indicate that the visual fixation suppression test suggests central nervous system deficit affecting this function, which overlaps with the neurological substrate for smooth pursuit and gaze fixation. The patient is asked to fixate on the calibration light that moves with the rotational chair, allowing the measurement of eye movements that may or may not be suppressed with fixation.

VOR Cancellation or Suppression Test (VOR Enhancement): In the VOR cancellation or suppression test, the optokinetic stripes are presented on the walls of the rotational chair booth while rotating the patient sinusoidally at a predetermined test frequency (Ruckenstein & Davis, 2015). Ruckenstein and Davis (2015) suggest that this test can provide information regarding the integrity of the central vestibulo-ocular pathways.

Interpretation

Sinusoidal (or Slow) Harmonic Acceleration Test: Three parameters are assessed, including gain, asymmetry, and phase. Significant reduced gains suggest bilateral vestibular involvement; however, reduced gains [particularly in the lower frequencies of rotation] can also occur as a result of unilateral weakness (Ruckenstein & Davis, 2015). Asymmetry that is outside normal 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:

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

- 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

The cVEMP response can be absent or its amplitude reduced in the presence of even a small conductive hearing loss (when ACS is used), and therefore a formal audiogram should be used to assess the patient's hearing in such cases. A normal cVEMP does not negate the need for an audiogram as occasionally otosclerosis and superior canal dehiscence (SCD) are present in the same ear: the otosclerosis attenuates the sound reaching the ear and the SCD causes a higher amplitude response to that attenuated sound. The result is what appears to be a normal cVEMP.

As the cVEMP requires a contracted SCM, patients unable to elevate and/or rotate their head may not be able to perform the test.

Limitations

Except in cases of a canal dehiscence, cVEMP findings alone are non-specific; they simply indicate reduced function.

As described above in the interpretation section, bilaterally absent responses are not always considered pathological in patients over the age of 60.

Ocular Vestibular Evoked Myogenic Potentials (oVEMPs)

Definition

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

Scientific Basis

When vibration is applied at Fz in humans, approximately equal amplitude linear acceleration is delivered to both mastoids (Curthoys, Manzari, Smulders, & Burgess, 2009; Iwasaki et al., 2008a), resulting in small short-latency negative (excitatory) ocular myogenic potentials beneath the eyes. The first of these is called n10 (appears at approximately 10 ms), and it has a larger amplitude when the patient looks up (Iwasaki et al., 2008a). The n10 is a crossed otolith-ocular response; i.e., it is measured under the contralateral eye, and reflects the activation of the inferior oblique and possibly the inferior rectus (Weber et al., 2008; Curthoys et al., 2011). However, after a unilateral vestibular loss, the n10 under the contralateral eye is absent or has reduced amplitude (Iwasaki et al., 2008b).

There is some evidence that oVEMPs originate predominantly in the utricle (Curthoys, Vulovic, and Manzari, 2012; Curthoys, 2010; Curthoys, et al., 2011; Manzari, Burgess, and Curthoys, 2010) whereas other findings argue the response may include a small contribution from the saccule, originating from both the utricle and the saccule (Papathanasiou, 2012; Welgampola et al., 2008).

Bone Conducted Vibration versus Air Conducted Sound

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

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

Procedure

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

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

Interpretation

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

Presence/Absence of Response

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

Amplitude Asymmetry Ratio

- This is calculated using Jongkees' formula:

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

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

Absolute Amplitude

- Zuniga et al. (2013), Janky et al. (2013), and Manzari, Burgess, McGarvie, and Curthoys (2012) found that oVEMP absolute amplitude was a more sensitive and specific tool than 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

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

Horizontal Canals

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

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

Vertical Canals

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

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

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

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

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.

It is helpful to take note of consistent patterns of corrective eye movements in the tracings: when true VOR loss is present, corrective saccades will have a significant velocity and will appear consistently in the tracings. Significantly reduced gains with no corrective saccades usually indicate a calibration problem (or incorrect gaze angle for LARP/RALP), while careful review of tracings that show corrective saccades but normal gains will usually reveal a VOR loss.

The distribution of corrective saccades (i.e., covert vs. overt) is difficult to predict and varies widely, even among patients with similar pathology (Blödow, Pannasch, & Walther, 2013).

Clinical Use

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

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

Contraindications

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

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

Limitations/Troubleshooting

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

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

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

Young children can be challenging to assess with vHIT due to their physical size and difficulty with attention. If pediatric goggle straps are unavailable, modifications can be made on-site. A video clip played from a smartphone screen may be used as a substitute for a static target when the patient is unable to maintain focus on a static target.

A very common cause of indeterminate vHIT results is from "goggle slip." Goggle slip usually occurs when the goggles are too loose or are a poor fit. The nose holds the goggles in place; therefore, if the patient has a low nasal bridge, the goggles may not sit on the patient's cheekbones and/or the nasal bridge. The resultant tracings will show eye movements that are faster than, and occur before, the head movements. Similar tracings may be obtained when the patient's hair contains hairspray or other hair products; hair with a lot of hairspray in it may move independently from the head, which may also result in artifact. It is essential that examiners become skilled at identifying these artifacts and differentiating them from actual pathology. When testing the horizontal canals, changing grip so that your hands cradle the patient's jaw line may reduce the extent of the artifact from goggle slip.

It is difficult to completely isolate the canals with vHIT, given the paired nature of the semicircular canals. The oculomotor command that results from a given head impulse is determined by both excitatory (ipsilateral to the head impulse) and inhibitory (contralateral to the head impulse) inputs. As a result, the contralateral side will have some influence on the ipsilateral tracings, except at very fast head impulses wherein the inhibitory inputs from the contralateral side are saturated (Weber et al., 2008). This may result in significant unilateral VOR loss being misidentified as bilateral vestibular loss.

Subjective Visual Vertical (SVV) Test

Definition

The subjective visual vertical (SVV) test is designed to evaluate a patient's ability to accurately identify "true vertical" without the help of a visual reference. The perception of a significant tilt from true vertical is a sensitive indicator of an acute unilateral vestibular lesion (mostly utricular) (Böhmer & Mast, 1999) or a central vestibular lesion affecting the gravitoceptive pathways (Dieterich & Brandt, 1993).

Procedure

Until recently, SVV test administration was restricted to testing centres with expensive, specialized equipment and skilled administrators (Zwergal, Rettinger, Frenzel, Dieterich, Brandt, & Strupp, 2009). Inexpensive alternatives have since been shown to have equivalent diagnostic accuracy: the "bucket method" can be easily produced with very inexpensive and readily available materials (Zwergal et al., 2009). Detailed instructions are available online (Cook, 2010). Smartphone-based approaches are also in development (Brodsky, Cusick, Kawai, Kenna, & Zhou, 2015).

For the bucket method, a bucket is made completely dark inside except for a thin strip of glow-in-the-dark tape, which becomes the vertical line. The line inside the bucket is aligned with a piece of weighted string, which is housed outside of the bucket, originating from the middle of the bucket. A protractor, with its zero point aligned with the weighted string, is also affixed to the outside of the bucket (Cook, 2010).

The examiner should start with the line in the bucket at a random alignment (either clockwise or counter-clockwise from vertical) and should ask the patient to look into the bucket such that the

interior fills their entire visual field. The examiner should then move the bucket towards vertical in small intervals, asking the patient to indicate when they perceive the line to be at true vertical. The value on the protractor, which represents degrees from true vertical, should then be recorded. It is recommended that the same procedure be repeated for a total of 10 attempts, varying clockwise and counter-clockwise (Zwergal et al., 2009).

Interpretation

The average of the 10 trials should be calculated to determine the patient's degree of tilt from true vertical. The normal range may vary depending on the method used, but is typically 0 ± 2.5º. Patients with values exceeding 2.5º should be flagged for further investigation.

Clinical Use

The SVV test can be used to detect lesions along the vestibular pathway. A detailed overview of the typical SVV findings for various pathologies falls outside of the scope of this document. For those hoping to learn more, the article by Zwergal et al. (2009) is an excellent starting point.

An ENT consultation should be recommended when the SVV test results exceed normal limits, due to the likelihood of central pathology. Peripheral lesions will usually normalize within approximately 20 days (Zwergal et al., 2009); with the exception of vestibular labs that test patients who are experiencing acute vertigo, the majority of abnormal findings on SVV will have origins in the central vestibular pathways.

Limitations

Abnormal findings are generally non-specific. Further investigations are needed to determine the site of lesion.

Computerized Dynamic Posturography (CDP)

Definition

Computerized dynamic posturography (CDP) is an objective quantitative method for assessing upright and in-place balance function under a variety of tasks that simulates the conditions encountered in daily life. CDP can identify and differentiate among a variety of possible sensory, motor, and central adaptive impairments to balance control. Although CDP cannot diagnose pathology or site of lesion, it is a functional test which is complementary to clinical tests such as videonystagmography (VNG) or rotary chair that help localize and categorize pathological mechanisms of balance disorders.

Procedure

There are several subtests that can be performed with posturography testing, including sensory organization testing (SOT), motor control testing (MCT), and adaptation testing (ADT). Throughout all subtests, the patient wears a harness to ensure safety. Once the patient is facing the surround, the straps of the harness are connected to the machine so that they are parallel to the ground on the patient's shoulder. The harness should fit comfortably without being too loose or too tight. During testing, the patient stands on a moveable, dual force plate support surface within a moveable surround or enclosure. The patient's feet should be aligned to the platform they are standing on according to their measured height (small = 114–140 cm, medium = 141–165 cm, and large = 166–203 cm). The medial malleolus and the lateral calcaneous are aligned on the marking stripe that transects the two force plates. The feet should be aligned throughout the testing. If the feet move out of place, testing should be stopped and resumed after 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

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

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

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

Vestibular Rehabilitation

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

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

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

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

For those vestibular audiologists looking to receive additional training in the area of vestibular rehabilitation, these are some helpful resources:

- The American Institute of Balance: http://dizzy.com/education_foundation.htm
- Vestibular Disorders Association: http://vestibular.org/resources-professionals/build-your-practice

Pediatric Population

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

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

"astronaut training." A similar approach can be used during caloric testing with pediatric patients.

Patient case history is critical for assessment of the pediatric vestibular patient (see Tables 1, 2, 3, and 4). It is important to note that up to 70% of children presenting with sensorineural hearing loss have impairment of their vestibular system with 20–40% having severe bilateral vestibular loss (Cushing, Gordon, Rutka, James, & Papsin, 2013; Cushing, Papsin, Rutka, James, & Gordon, 2008; Cushing, James, Papsin, & Gordon, 2008; Buchman, Joy, Hodges, Telischi, & Balkany, 2004).

Some children with dysfunction of the vestibular portion of the inner ear will never be vertiginous. This is particularly likely if this dysfunction is severe, congenital, and/or bilateral. Impairment of the vestibular end organs may be suggested if there are delayed motor milestones (e.g., poor head control [beyond 6 weeks old], delayed independent sitting [beyond 9 months], and delayed walking [beyond 18 months]) (Cushing, 2014).

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

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

Provision of printed education materials regarding vestibular disorders and management may be useful for audiologists during counselling. In addition, audiologists may find it useful to consult anatomical charts or models to illustrate points of possible dysfunction within the vestibular/balance system(s).

For patients diagnosed with vestibular migraine, migraine triggers can be discussed—such as triggers related to diet-, barometric pressure, fatigue, hormones, and/or stress. Similarly, diet-related triggers can be monitored for patients suspected of having Ménière's disease or endolymphatic hydrops. Suggesting the use of a "dizzy diary" can be useful for patients to monitor and record their symptoms over time and to look for provoking factors, links, or trends between their symptoms.

Reporting/Documentation

It is important to ensure that clinicians are adhering to standards and regulations regarding clinical documentation developed by provincial and territorial regulatory bodies, national associations, and their employers. Adherence to freedom of information and privacy laws is also. It is up to each audiologist to ensure they are in compliance with the laws in their own region.

Distribution of the patient report should include information for the referring clinician, as well as copies for any other health professional as requested by the patient.

At a minimum, the report should include diagnostic test results and an analysis of whether the results are normal or outside normal limits. Reporting should also include case history, interpretations/impressions, and recommendations for management and follow-up.

Audiologists should avoid basing interpretations/impressions on one single diagnostic finding; instead, take overall test results and patient case history into consideration.

References

Accreditation Canada, Canadian Institute for Health Information, & Canadian Patient Safety Institute (2014). *Preventing falls: From evidence to improvement in Canadian health care*. Retrieved from https://accreditation.ca/sites/default/files/ falls-joint-report-2014-en.pdf

Agrawal, Y., Carey, J. P., Della Santina, C. C., Schubert, M. C., & Minor, L. B. (2009). Disorders of balance and vestibular function in US adults: Data from the national health and nutrition examination survey, 2001-2004. *Archives of Internal Medicine, 169*(10), 938–944. doi:10.1001/archinternmed.2009.66

American Geriatrics Society. (2011). Summary of the updated American Geriatrics Society / British Geriatrics Society clinical practice guideline for prevention of falls in older persons. *Journal of the American Geriatrics Society, 59*(1), 148–157. doi:10.1111/j.1532-5415.2010.03234.x

American Institute of Balance. *Vestibular assessment & management certification workshop*. Retrieved from http://dizzy.com/files/syllabus-vam.pdf

Aron, M., & Bance, M. (2013). Insights into horizontal canal benign paroxysmal positional vertigo from a human case report. *The Laryngoscope, 123*(12), 3197–3200. doi:10.1002/lary.24260

Arriaga, M. A., Chen, D. A., & Cenci, K. A. (2005). Rotational chair (ROTO) instead of electronystagmography (ENG) as the primary vestibular test. *Otolaryngology–Head and Neck Surgery, 133*(3), 329–333. doi:10.1016/j.otohns.2005.05.002

Barin, K. (2006). *Common errors in ENG/VNG*. Retrieved from http://www.audiologyonline.com/articles/common-errors-in-eng-vng-978

Barin, K. (2011). *Getting the most out of VNG/ENG testing*. Retrieved from http://www.ohioslha.org/pdf/Convention/2011%20Handouts/MS24AudiologyBarin.pdf

Barin, K. (2016a). Background and technique of caloric testing. In G. Jacobson & N. Shepard (Eds.), *Balance Function Assessment and Management* (2nd ed.) (pp. 283–318). San Diego, CA: Plural Publishing.

Barin, K. (2016b). Interpretation and usefulness of caloric testing. In G. Jacobson & N. Shepard (Eds.), *Balance Function Assessment and Management* (2nd ed.) (pp 319–346). San Diego, CA: Plural Publishing.

Bennett, M., & Jacobson, G. (2007). The vertigo case history. In G. Jacobson & N. Shepard (Eds.), *Balance Function Assessment and Management* (pp. 45–62). San Diego, CA: Plural Publishing.

Blakley, B. W., & Wong, V. (2015). Normal values for cervical vestibular-evoked myogenic potentials. *Otology & Neurotology, 36*(6), 1069–1073. doi:10.1097/MAO.0000000000000752

Blatt, P., Georgakakis, G., Herdman, S., Clendaniel, R., & Tusa, R. (2000). The effect of canalith repositioning maneuver on resolving postural instability in patient with benign paroxysmal positional vertigo. *American Journal of Otology, 21*(3), 356–363. Retrieved from http://journals.lww.com/otology-neurotology/Pages/default.aspx

Blödow, A., Pannasch, S., & Walther, L. E. (2013). Detection of isolated covert saccades with the video head impulse test in peripheral vestibular disorders. *Auris Nasus Larynx, 40*(4), 348–351. doi:10.1016/j.anl.2012.11.002

Böhmer, A., & Mast, F. (1999). Assessing otolith function by the subjective visual vertical. *Annals of the New York Academy of Sciences, 871*, 221–231. doi:10.1111/j.1749-6632.1999.tb09187.x

Brandt, T., Dieterich, M., & Strupp, M. (2013). *Vertigo and dizziness: Common complaints*. London, UK: Springer.

Brodovsky, J. R. & Vnenchak, M. J. (2013). Vestibular Rehabilitation for Unilateral Peripheral Vestibular Dysfunction. *Physical Therapy*, *93*(3), 293–298. doi:10.2522/ptj.20120057

Brodsky, J. R., Cusick, B. A., Kawai, K., Kenna, M., & Zhou, G. (2015). Peripheral vestibular loss detected in pediatric patients using a smartphone-based test of the subjective visual vertical. *International Journal of Pediatric Otorhinolaryngology, 79*(12), 2094–2098. doi: 10.1016/j.ijporl.2015.09.020

British Society of Audiology. (2001). *Recommended procedure for Hallpike manoeuvre*. Retrieved from http://www.thebsa.org.uk/wp-content/uploads/2014/ 04/HM.pdf

British Society of Audiology. (2014). *Recommended procedure for the caloric test*. Retrieved from http://www.thebsa.org.uk/wp-content/uploads/2014/04/ Recommended-procedure-for-the-Caloric-test.pdf

British Society of Audiology. (2016). *Recommended Procedure: Positioning Tests*. Retrieved from http://www.thebsa.org.uk/wp-content/uploads/2015/12/Positioning-Tests-Recommended-Procedure-09.12.2015.pdf

Buchman, C. A., Joy, J., Hodges, A., Telischi, F. F., & Balkany, T. J. (2004). Vestibular effects of cochlear implantation. *The Laryngoscope, 114*(S103), 1–22. doi:10.1097/00005537200410001-00001

Canadian Academy of Audiology. (2002). *Position Statement on Audiology Scope of Practice*. Retrieved from https://canadianaudiology.ca/professional-resources/scope-of-practice/

Canadian Interorganizational Steering Group for Speech-Language Pathology and Audiology (2010). *Infection prevention and control guidelines for audiology*. Retrieved from http://sacoac.ca/%20sites/default/files/resources/Infection_prevention_control_Guidelines_Audiology.pdf

Canadian Medical Association. (2016). *Demand a Plan*. Retrieved from http://www.demandaplan.ca/recommendations_for_action

Canadian Society of Otolaryngology-Head & Neck Surgery. (2016). *Five Things Physicians and Patients Should Question*. Retrieved from http://www.choosingwiselycanada.org/recommendations/otolaryngology-head-neck-surgery/

Caruso, G., & Nuti, D. (2005). Epidemiological data from 2270 PPV patients. *Audiological Medicine, 3*(1), 7–11. doi:10.1080/16513860510028310

Cass, S. P., & Furman, J. M. R. (1993, November). Medications and their effects on vestibular testing. *ENG report*. Retrieved from https://www.google.ca/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&cad=rja&uact=8&ved=0ahUKEwjz0szYkM7OAhVRgx4KHa6jDHQQFgggMAA&url=http%3A%2F%2Fwww.otometrics.com%2FKnowledge%2520center%2F%2F~%2Fmedia%2FDownloadLibrary%2FOtometrics%2FPDFs%2FKnowledge%2C-sp-%2Ccenter%2C-sp-%2Ccontent%2FBalance%2C-sp-%2Ccontent%2FENG%2C-sp-%2CReports%2Fmedications_their_effects_on_vest_func_noc_1993.ashx&usg=AFQjCNHY-Hsjn6b0TmCCMzbQst1nM3XJIA&sig2=MDlO0HwPG1DXy08nx_gp1Q&bvm=bv.129759880,d.dmo.

Chang, C. M., Cheng, P. W., Wang, S. J., & Young, Y. H. (2010). Effects of repetition rate of bone-conducted vibration on ocular and cervical vestibular-evoked myogenic potentials. *Clinical Neurophysiology, 121*(12), 2121–2127. doi:10.1016/j.clinph.2010.05.013

Cheng, P. W., Chen, C. C., Wang, S. J., & Young, Y. H. (2009). Acoustic, mechanical and galvanic stimulation modes elicit ocular vestibular-evoked myogenic potentials. *Clinical Neurophysiology, 120*(10), 1841–1844. doi:10.1016/j.clinph.2009.08.002

Chihara, Y., Iwasaki, S., Fujimoto, C., Ushio, M., Yamasoba, T., & Murofushi, T. (2009). Frequency tuning properties of ocular vestibular evoked myogenic potentials. *Neuroreport, 20*(16), 1491–1495. doi:10.1097/WNR.0b013e3283329b4a

Chihara, Y., Iwasaki, S., Ushio, M., & Murofushi, T. (2007). Vestibular-evoked extraocular potentials by air-conducted sound: Another clinical test for vestibular function. *Clinical Neurophysiology, 118*(12), 2745–2751. doi:10.1016/j.clinph.2007.08.005

Choung, Y. H., Shin, Y.R., Kahng, H., Park, K., & Choi, S. J. (2006). 'Bow and lean test' to determine the affected ear of horizontal canal benign paroxysmal positional vertigo. *Laryngoscope,* Oct;116(10):1776-81. doi:10.1097/01.mlg.0000231291.44818.be

Colebatch, J. G., Halmagyi, G. M., & Skuse, N. F. (1994). Myogenic potentials generated by a click-evoked vestibulocollic reflex. *Journal of Neurology, Neurosurgery, and Psychiatry, 57*(2), 190–197. doi:10.1136/jnnp.57.2.190

College of Speech and Hearing Health Professionals of BC. *Advanced competency certification*. Retrieved from http://www.cshhpbc.org/advancedcompetency.htm

College of Speech and Hearing Health Professionals of BC. *Advanced certification program of study: form # 9-A.* Retrieved from http://cshhpbc.org/docs/acceptable_program_of_study_-

certificate_a.pdf?LanguageID=EN-USCook, J. (2010). *How to construct an SVV bucket*. Retrieved from http://www.mvrc.pitt.edu/files/svv-bucket-how-to.pdf

Curthoys, I. S. (2010). A critical review of the neurophysiological evidence underlying clinical vestibular testing using sound, vibration and galvanic stimuli. *Clinical Neurophysiology, 121*(2), 132–144. doi:10.1016/j.clinph.2009.09.027

Curthoys, I.S., & Manzari, L. (2011). Evidence missed: ocular vestibular-evoked myogenic potential and cervical vestibular-evoked myogenic potential differentiate utricular from saccular function. *Otolaryngology–Head & Neck Surgery, 144*(5), 751–752. doi:10.1177/0194599810397792

Curthoys, I. S., Manzari, L., Smulders, Y. E., & Burgess, A. M. (2009). A review of the scientific basis and practical application of a new test of utricular function--ocular vestibular-evoked myogenic potentials to bone-conducted vibration. *Acta Otorhinolaryngologica Italica, 29*(4), 179–186. Retrieved from http://www.actaitalica.it/

Curthoys, I. S., & Vulovic, V. (2011). Vestibular primary afferent responses to sound and vibration in the guinea pig. *Experimental Brain Research, 210*(3-4), 347–352. doi:10.1007/s00221-010-2499-5

Curthoys, I. S., Vulovic, V., Burgess, A. M., Cornell, E. D., Mezey, L. E., Macdougall, H. G., . . . McGarvie, L. A. (2011). The basis for using bone-conducted vibration or air-conducted sound to test otolithic function. *Annals of the New York Academy of Sciences, 1233*, 231–241. doi:10.1111/j.1749-6632.2011.06147.x

Curthoys, I. S., Vulovic, V., & Manzari, L. (2012). Ocular vestibular-evoked myogenic potential (oVEMP) to test utricular function: neural and oculomotor evidence. *Acta Otorhinolaryngologica Italica, 32*(1), 41–45. Retrieved from http://www.actaitalica.it/

Cushing, S. (2014). Vestibular and balance dysfunction in the pediatric population: A primer for the audiologist. *Canadian Audiologist, 1*(4). Retrieved from http://www.canadianaudiologist.ca/

Cushing, S. L., Gordon, K. A., Rutka, J. A., James, A. L., & Papsin, B. C. (2013). Vestibular end-organ dysfunction in children with sensorineural hearing loss and cochlear implants: An expanded cohort and etiologic assessment. *Otology & Neurotology, 34*(3), 422–428. doi:10.1097/MAO.0b013e31827b4ba0

Cushing, S. L., Papsin, B. C., Rutka, J.A., James, A. L., & Gordon, K. A. (2008). Evidence of vestibular and balance dysfunction in children with profound sensorineural hearing loss using cochlear implants. *The Laryngoscope, 118*(10), 1814–1823. doi:10.1097/MLG.0b013e31817fadfa

Cushing, S.L., Chia. R., James, A. L., Papsin, B. C., & Gordon, K. A. (2008). A test of dynamic balance function in children with cochlear implants: The Vestibular Olympics. *Arch Otolaryngol Head Neck Surg*. Jan;134(1):34-8. doi: 10.1001/archoto.2007.16

Dieterich, M., & Brandt, T. (1993). Ocular torsion and tilt of subjective visual vertical are sensitive brainstem signs. *Annals of Neurology, 33*(3), 292–299. doi:10.1002/ana.410330311

Dieterich, M., & Brandt, T. (1999). Episodic vertigo related to migraine (90 cases): Vestibular migraine? *Journal of Neurology, 246*(10), 883–892. doi:10.1007/s004150050478

Emory University School of Medicine. (2015). *Vestibular Rehabilitation: A Competency-Based Course*. Retrieved from http://www.rehabmed.emory.edu/pt/ce/info/Vestibular2016_MoreInformation_PlanningOnly_2-11-2015.pdf

Enticott, J. C., O'Leary, S. J., & Briggs, R. J. (2005). Effects of vestibulo-ocular reflex exercises on vestibular compensation after vestibular schwannoma surgery. *Otology & Neurotology, 26*(2), 265–269. Retrieved from http://journals.lww.com/otology-neurotology/Pages/default.aspx

Gioacchini, F. M., Alicandri-Ciufellia, M., Kaleci, S., Magliulio, G., & Re, M. (2014). Prevalence and diagnosis of vestibular disorders in children: A review. *International Journal of Pediatric Otorhinolaryngology, 78*(5), 718–724. doi:10.1016/j.ijporl.2014.02.009

Gispen, F. E., Chen, D. S., Genther, D. J., & Lin, F. R. (2014). Association between hearing impairment and lower levels of physical activity in older adults. *Journal of the American Geriatrics Society, 62*(8), 1427–1433. doi:10.1111/jgs.12938

Goncalves, D. U., Felipe, L., & Lima, T. M. A. (2008). Interpretation and use of caloric testing. *Brazilian Journal of Otorhinolaryngology, 74*(3), 440–446. doi: 10.1016/S1808-8694(15)30580-2

Goulson, A. M., McPherson, J. H., & Shepard, N. T. (2016). Background and introduction to whole-body rotational testing. In G. Jacobson & N. Shepard (Eds.), *Balance Function Assessment and management* (2nd ed.) (pp. 347–364). San Diego, CA: Plural MPublishing.

Hain, T. (1997a). Background and technique of ocular motility testing. In G. P. Jacobson, C. W. Newman, & J. M. Kartush (Eds.), *Handbook of Balance Function Testing* (pp. 83–100). Clifton Park, NY: Delmar Cengage Learning.

Hain, T. (1997b). Interpretation and usefulness of ocular motility testing. In G. P. Jacobson, C. W. Newman, & J. M. Kartush (Eds.), *Handbook of Balance Function Testing* (pp. 101–122). Clifton Park, NY: Delmar Cengage Learning.

Halker, R. B., Barrs, D. M., Wellik, K. E., Wingerchuk, D. M., & Demaerschalk, B. M. (2008). Establishing a diagnosis of benign paroxysmal positional vertigo through the Dix-Hallpike and side-lying maneuvers: A critically appraised topic. *The Neurologist*, *14*(3), 201–204. doi:10.1097/NRL.0b013e31816f2820

Halmagyi, G. M., & Curthoys, I. S. (1988). A clinical sign of canal paresis. *Archives of Neurology, 45*(7), 737–739. doi:10.1001/archneur.1988.00520310043015

Hamilton, S. S., Zhou, G., & Brodsky, J. R. (2015). Video head impulse testing (VHIT) in the pediatric population. *International Journal of Pediatric Otorhinolaryngology, 79*(8), 1283–1287. doi:10.1016/j.ijporl.2015.05.033

Helminski, J. O., Zee, D. S., Janssen, I. & Hain, T. C. (2010). Effectiveness of particle repositioning maneuvers in the treatment of benign paroxysmal positional vertigo: A systematic review. *Physical Therapy*, *90*(5), 663–678. doi:10.2522/ptj.20090071

Hilton, M. P. & Pinder, D. K. (2014). The Epley (canalith repositioning) manoeuvre for benign paroxysmal positional vertigo (Review). *Cochrane Database of Systematic Reviews, 2014*(12), 1–38. doi:10.1002/14651858.CD003162.pub3

Hunt, W. T., Zimmerman, E. F., & Hilton, M. P. (2012). Modifications of the Epley (canalith repositioning) manoeuvre for posterior canal benign paroxysmal positional vertigo (BPPV). *Cochrane Database of Systematic Reviews, 2012*(4), 1–41. doi:10.1002/14651858.CD008675.pub2.

Isaradisaikul, S., Navacharoen, N., Hanprasertpong, C., & Kangsanarak, J. (2012). Cervical vestibular-evoked myogenic potentials: Norms and protocols. *International Journal of Otolaryngology, 2012, 1-7*. doi:10.1155/2012/913515

Iwasaki, S., Smulders, Y. E., Burgess, A. M., McGarvie, L. A., Macdougall, H. G., Halmagyi, G. M., & Curthoys, I. S. (2008a). Ocular vestibular evoked myogenic potentials in response to bone-conducted vibration of the midline forehead at Fz: A new indicator of unilateral otolithic loss. *Audiology & Neurotology, 13*(6), 396–404. doi:10.1159/000148203

Iwasaki, S., Smulders, Y. E., Burgess, A. M., McGarvie, L. A., Macdougall, H. G., Halmagyi, G. M., & Curthoys, I. S. (2008b). Ocular vestibular evoked myogenic potentials to bone conducted vibration of the midline forehead at Fz in healthy subjects. *Clinical Neurophysiology, 119*(9), 2135–2147. doi:10.1016/j.clinph.2008.05.028

Jacobson, G.P., & Newman, C.W. (1990). The development of the Dizziness Handicap Inventory. *Archives of Otolaryngology–Head & Neck Surgery, 116*(4), 424–427. doi:10.1001/archotol.1990.01870040046011

Janky, K. & Shepard, N. (2009). Vestibular evoked myogenic potential (VEMP) testing: Normative threshold response curves and effects of age. *Journal of the American Academy of Audiology, 20*(8), 514–522. doi:10.3766/jaaa.20.8.6

Janky, K. L., Nguyen, K. D., Welgampola, M., Zuniga, M. G., & Carey, J. P. (2013). Air-conducted oVEMPs provide the best separation between intact and superior canal dehiscent labyrinths. *Otology & Neurotology, 34*(1), 127–134. doi:10.1097/MAO.0b013e318271c32a

Ko C., Hoffman, H.J., & Sklare, D.A. (2006). Chronic Imbalance or Dizziness and Falling: Results from the 1994 Disability Supplement to the National Health Interview Survey and the Second Supplement on Aging Study. Poster. Page 5 of 6 session of the Twenty-ninth Annual Midwinter Meeting of the Association for Research in Otolaryngology, Feb. 5–9, 2006.

Kotimäki, J., Sorri, M., Aantaa, E., & Nuutinen, J. (1999). Prevalence of Ménière's disease in Finland. *The Laryngoscope, 109*(5), 748–753. doi:10.1097/00005537-199905000-00013

Kroenke, K., Hoffman, R. M., & Einstadter, D. (2000). How common are various causes of dizziness? A critical review. *Southern Medical Journal, 93*(2), 160–168. Retrieved from http://sma.org/smj-home/

Leipzig, R. M., Cumming, R. G., & Tinetti, M. E. (1999). Drugs and falls in older people: A systematic review and meta-analysis: I. Psychotropic drugs. *Journal of the American Geriatric Society, 47*(1), 30–39. doi:10.1111/j.1532-5415.1999.tb01898.x

Lightfoot, G. R. (2004). The origin of order effects in the results of the bi-thermal caloric test. *International Journal of Audiology, 43*(5), 276–282. doi:10.1080/14992020400050037

Lightfoot, G., Barker, F., Belcher, K., Kennedy, V., Nassar, G., & Tweedy, F. (2009). The derivation of optimum criteria for use in the monothermal caloric screening test. *Ear and Hearing, 30*(1), 54–62. doi:10.1097/AUD.0b013e31818f006c

Macdougall, H. G., McGarvie, L. A., Halmagyi, G. M., Curthoys, I. S., & Weber, K. P. (2013). The video head impulse test (vHIT) detects vertical semicircular canal dysfunction. *PloS One, 8*(4), 1-10. doi:10.1371/journal.pone.0061488

Mallinson, A. (2014). Using computerized dynamic posturography in the medical/legal setting: How we do it. *Canadian Audiologist*, *1*(4). Retrieved from http://canadianaudiologist.ca/

Manzari, L. (2013). Issues in using air conducted sound to test oVEMPs. *Clinical Neurophysiology, 124*(4), 640–641. doi:10.1016/j.clinph.2012.10.001

Manzari, L., Burgess, A. M., & Curthoys, I. S. (2010). Dissociation between cVEMP and oVEMP responses: Different vestibular origins of each VEMP? *European Archives of Oto-Rhino-Laryngology, 267*, 1487–1489. doi:10.1007/s00405-010-1317-9

Manzari, L., Burgess, A. M., McGarvie, L. A., & Curthoys, I. S. (2012). Ocular and cervical vestibular evoked myogenic potentials to 500 Hz Fz bone-conducted vibration in superior semicircular canal dehiscence. *Ear and Hearing, 33*(4), 508–520. doi:10.1097/AUD.0b013e3182498c09

Manzari, L., Tedesco, A. R., Burgess, A. M., & Curthoys, I. S. (2010). Ocular and cervical vestibular-evoked myogenic potentials to bone conducted vibration in Ménière's disease during quiescence vs during acute attacks. *Clinical Neurophysiology, 121*(7), 1092–1101. doi:10.1016/j.clinph.2010.02.003

McCaslin, D. L. (2013). *Electronystagmography and Videonystagmography ENG/VNG*. San Diego, CA: Plural Publishing Inc.

Meyer, C. H., Lasker, A. G., & Robinson, D. A. (1985). The upper limit of human smooth pursuit velocity. *Vision Research, 25*(4), 561–563. doi:10.1016/0042-6989(85)90160-9

Meyer, N., Vinck, B., & Heinze, B. (2015). cVEMPs: A systematic review and meta-analysis. *International Journal of Audiology, 54*(3), 143–151. doi:10.3109/14992027.2014.971468

Migliaccio, A. A., & Cremer, P. D. (2011). The 2D modified head impulse test: A 2D technique for measuring function in all six semi-circular canals. *Journal of Vestibular Research, 21*(4), 227–234. doi: 10.3233/VES-2011-0421

Minor, L. B., Schessel, D. A., & Carey, J. P. (2004). Ménière's disease. *Current Opinion in Neurology, 17*(1), 9–16. Retrieved from http://journals.lww.com/co-neurology/Pages/default.aspx

Mira, E. (2008). Improving the quality of life in patients with vestibular disorders: the role of medical treatments and physical rehabilitation. *International Journal of Clinical Practice,* Jan;62(1):109-14. doi:10.1111/j.1742-1241.2006.01091.x

Mostafa, B. E., Youssef, T. A., & Hamad, A. S. (2013). The necessity of post-maneuver postural restriction in treating benign paroxysmal positional vertigo: A meta-analytic study. *European Archives of Oto-Rhino-Laryngology*, *270*(3), 849–852. doi:10.1007/s00405-012-2046-z

Murofushi, T., & Kaga, K. (2009). *Vestibular evoked myogenic potentials*. Tokyo, Japan: Springer.

Murofushi, T., Matsuzaki, M., & Mizuno, M. (1998). Vestibular evoked myogenic potentials in patients with acoustic neuromas. *Archives of Otolaryngology – Head & Neck Surgery, 124*(5), 509–512. doi:10.1001/archotol.124.5.509.

Myers, B. L. (2011). *Vestibular learning manual*. San Diego, CA: Plural Publishing.

Naber, C. M., Water-Schmeder, O., Bohrer, P. S., Matonak, K., Berstein, A. L., & Merchant, M. A. (2011). Interdisciplinary treatment for vestibular dysfunction: The effectiveness of mindfulness, cognitive-behavioral techniques, and vestibular rehabilitation. *Otolaryngology–Head and Neck Surgery, 145*(1), 117–124. doi: 10.1177/0194599811399371

Nashner, L. W. (2016). Computerized dynamic posturography. In G. Jacobson & N. Shepard (Eds.), *Balance Function Assessment and Management* (2nd ed.) (pp. 451–479). San Diego, CA: Plural Publishing.

National Institute on Deafness and Other Communication Disorders. *Strategic Plan FY2006-2008*. Retrieved from http://www.nidcd.nih.gov/StaticResources/about/plans/strategic/strategic06-08.pdf#sthash.WpFV7MP2.dpuf

Nelson, M. D., Akin, F. W., Riska, K. M., Andresen, K., & Mondelli, S. S. (2016).Vestibular assessment and rehabilitation: Ten-year survey trends of audiologists' opinions and practice. *Journal of the American Academy of Audiology, 27*(2), 126–140. Retrieved from http://aaa.publisher.ingentaconnect.com/content/aaa/jaaa

Neugebauer, H., Adrion, C., Glaser, M., & Strupp, M. (2013). Long-term changes of central ocular motor signs in patients with vestibular migraine. *European Neurology, 69*(2), 102–107. doi:10.1159/000343814

Neuhauser, H. K., & Lempert, T. (2009). Vertigo: Epidemiologic aspects. *Seminars in Neurology, 29*(5), 473–481. doi:10.1055/s-0029-1241043

Neuhauser, H., Leopold, M., von Brevern, M., Arnold, G., & Lempert, T. (2001) The interrelations of migraine, vertigo, and migrainous vertigo. *Neurology, 56*(4), 436–441. doi:10.1212/WNL.56.4.436

NeuroCom International Inc. (2011). *Balance Manager Systems: Clinical Interpretation Guide – Computerized Dynamic Posturography*. San Carlos, CA: Natus Medical Inc.

O'Reilly, R., Grindle, C., Zwicky, E. F., & Morlet, T. (2011). Development of the vestibular system and balance function: Differential diagnosis in the pediatric population. *Otolaryngologic Clinics of North America, 44*(2), 251–271. doi:10.1016/j.otc.2011.01.001

Papathanasiou, E. S. (2012) Ocular vestibular evoked myogenic potentials (OVEMPs): Saccule or utricle? *Clinical Neurophysiology*, *123*(2), 216. doi:10.1016/j.clinph.2011.06.013

Papathanasiou, E. S., Murofushi, T., Akin, F. W., & Colebatch, J. G. (2014). International guidelines for the clinical application of cervical vestibular evoked myogenic potentials: An expert consensus report. *Clinical Neurophysiology, 125*(4), 658–666. doi:10.1016/j.clinph.2013.11.042

The Joanna Briggs Institute. (2015). *Joanna Briggs Institute Reviewers' Manual: 2015 edition/supplement*. South Australia: The Joanna Briggs Institute.

Piker, E. G., Jacobson, G. P., Burkard, R. F., McCaslin, D. L., & Hood, L. J. (2013). Effects of age on the tuning of the cVEMP and oVEMP. *Ear and Hearing, 34*(6), e65–73. doi:10.1097/AUD.0b013e31828fc9f2

Public Health Agency of Canada (2014). *Seniors' Falls in Canada: Second Report*. Retrieved from http://www.phac-aspc.gc.ca/seniors-aines/publications/public/index-%20eng.php

Rajendran, V., Roy, F. G., & Jeevanantham, D. (2013). Effect of exercise intervention on vestibular related impairments in hearing-impaired children. *Alexandria Journal of Medicine, 49*(1), 7–12. doi: 10.1016/j.ajme.2012.10.001

Rawski, E. (1998). Review of the literature on falls among the elderly. *Journal of Nursing Care Quality*, 13(2), 38-44.

Roberts, R. A. (2016). Technique and interpretation of positional testing. In G. Jacobson & N. Shepard (Eds.), *Balance Function Assessment and Management* (2nd ed.) (pp. 251–282). San Diego, CA: Plural Publishing.

Rosengren, S. M., Welgampola, M. S., & Colebatch, J. G. (2010). Vestibular evoked myogenic potentials: Past, present and future. *Clinical Neurophysiology, 121*(5), 636–651. doi:10.1016/j.clinph.2009.10.016

Roy, F. D., & Tomlinson, R. D. (2004). Characterization of the vestibulo-ocular reflex evoked by high-velocity movements. *The Laryngoscope, 114*(7), 1190–1193. doi:10.1097/00005537-200407000-00011

Ruckenstein, M. J., & Davis, S. (2015). Rotational studies. In *Rapid Interpretation of Balance Function Tests* (pp 85–109). San Diego, CA: Plural Publishing Inc.

Sandhu, J. S., George, S. R., & Rea, P. A. (2013). The effect of electrode positioning on the ocular vestibular evoked myogenic potential to air-conducted sound. *Clinical Neurophysiology, 124*(6), 1232–1236. doi:10.1016/j.clinph.2012.11.019

Sandhu, J. S., Low, R., Rea, P. A., & Saunders, N. C. (2012). Altered frequency dynamics of cervical and ocular vestibular evoked myogenic potentials in patients with Ménière's disease. *Otology & Neurotology, 33*(3), 444–449. doi:10.1097/MAO.0b013e3182488046

Shepard, N. (2009). Evaluation of the patient with dizziness and balance disorders. In J. Katz, L. Medwetsky, R. Burkard, & L. Hood (Eds.), *Handbook of Clinical Audiology* (6th ed.) (pp 467–496). Philidelphia, PA: Lippincott Williams & Wilkins.

Shepard, N. T., & Schubert, M. C. (2016). Background and technique of ocular motility testing. In G. Jacobson & N. Shepard (Eds.), *Balance Function Assessment and Management* (2nd ed.) (pp 209–223). San Diego, CA: Plural Publishing Inc.

Shepard, N. T., Schubert, M. C., & Eggers, S. D. (2016). Interpretation and usefulness of ocular motility testing. In G. Jacobson & N. Shepard (Eds.), *Balance Function Assessment and Management* (2nd ed.) (pp 225–249). San Diego, CA: Plural Publishing Inc.

Speech-Language and Audiology Canada. (2016a). *Code of Ethics*. Retrieved http://www.sac-oac.ca/sites/default/files/resources/2016_sac_Code_of_Ethics_en.pdf

Speech-Language and Audiology Canada. (2016b). *Scope of Practice for Audiology*. Retrieved from http://www.sac-oac.ca/sites/default/files/resources/scope_of_practice_audiology_en.pdf?_ga=1.182846885.2084794186.1403892485

Statistics Canada. (2014). *Population Projections for Canada, Provinces and Territories*. Retrieved from http://www.statcan.gc.ca/pub/91-520-x/91-520-x2010001%20-eng.pdf

Stevens, J. A., Corso, P. S., Finkelstein, E. A., & Miller, T. R. (2006). The costs of fatal and non-fatal falls among older adults. *Injury Prevention, 12*(5), 290–295. doi:10.1136/ip.2005.011015

Suzuki, J. I., Tokumasu, K., & Goto, K. (1969). Eye movements from single utricular nerve stimulation in the cat. *Acta Oto-Laryngologica, 68*(4), 350–362. doi:10.3109/00016486909121573

Tyrrell, J. S., Whinney, D. J. D., Ukoumunne, O. C., Fleming, L. E., & Osborne, N. J. (2014). Prevalence, associated factors, and comorbid conditions for Ménière's disease. *Ear & Hearing, 35*(4), e162–e169. doi:10.1097/AUD.0000000000000041

von Brevern, M., Radtke, A., Lezius, F., Feldmann, M., Ziese, T., Lempert, T., & Neuhauser, H. (2007). Epidemiology of benign paroxysmal positional vertigo: a population based study. *Journal of Neurology, Neurosurgery & Psychiatry, 78*(7), 710–715. doi:10.1136/jnnp.2006.100420

Wang, S. J., Weng, W. J., Jaw, F. S., & Young, Y. H. (2010). Ocular and cervical vestibular-evoked myogenic potentials: A study to determine whether air- or bone-conducted stimuli are optimal. *Ear and Hearing, 31*(2), 283–288. doi:10.1097/AUD.0b013e3181bdbac0

Weber, K. P., Aw, S. T., Todd, M. J., McGarvie, L. A., Curthoys, I. S., & Halmagyi, G. M. (2008). Head impulse test in unilateral vestibular loss: Vestibulo-ocular reflex and catch-up saccades. *Neurology, 70*(6), 454–463. doi:10.1212/01.wnl.0000299117.48935.2e

Welgampola, M. S., & Carey, J. P. (2010). Waiting for the evidence: VEMP testing and the ability to differentiate utricular versus saccular function. *Otolaryngology–Head & Neck Surgery, 143*(2), 281–283. doi:10.1016/j.otohns.2010.05.024

Welgampola, M. S., & Colebatch, J. G. (2005). Characteristics and clinical applications of vestibular-evoked myogenic potentials. *Neurology, 64*(10), 1682–1688. doi:10.1212/01.WNL.0000161876.20552.AA

Welgampola, M. S., Myrie, O. A., Minor, L. B., & Carey, J. P. (2008). Vestibular-evoked myogenic potential thresholds normalize on plugging superior canal dehiscence. *Neurology, 70*(6), 464–472. doi:10.1212/01.wnl.0000299084.76250.4a

Wladislavosky-Waserman, P., Facer, G. W., Mokri, B., & Kurland, L. T. (1984). Ménière's disease: A 30-year epidemiologic and clinical study in Rochester, MN, 1951-1980. *Laryngoscope, 94*(8), 1098–1102. doi: 10.1288/00005537-198408000-00020

Yang, T. H., Liu, S. H., Wang, S. J., & Young, Y. H. (2010). An animal model of ocular vestibular-evoked myogenic potential in guinea pigs. *Experimental Brain Research, 205*(2), 145–152. doi:10.1007/s00221-010-2346-8

Young, Y. H. (2013). Potential application of ocular and cervical vestibular-evoked myogenic potentials in meniere's disease: A review. *The Laryngoscope, 123*(2), 484–491. doi:10.1002/lary.23640

Young, Y. H. (2015). Assessment of functional development of the otolithic system in growing children: A review. *International Journal of Pediatric Otorhinolaryngology, 79*(4), 435–442. doi:10.1016/j.ijporl.2015.01.015

Yu, S., Liu, F., Cheng, Z., & Wang, Q. (2014). Association between osteoporosis and benign paroxysmal positional vertigo: A systematic review. *BMC Neurology, 14*(1), 110. doi:10.1186/1471-2377-14-110

Zaida, S. H., & Sinha, A. (2013). *Vertigo, a Clinical Guide*. London, UK: Springer.

Zuma e Maia, F., & Luis, L. (2015). Inferior peduncle lesion presenting with bilaterally impaired vestibular responses to horizontal and posterior head impulses. *The Laryngoscope, 125*(10), 2386–2387. doi:10.1002/lary.25306

Zuniga, M. G., Janky, K. L., Nguyen, K. D., Welgampola, M. S., & Carey, J. P. (2013). Ocular vs. cervical VEMPs in the diagnosis of superior semicircular canal dehiscence

syndrome. *Otology & Neurotology, 34*(1), 121–126. doi:10.1097/MAO.0b013e31827136b0

Zwergal, M. D., Rettinger, N., Frenzel, C., Dieterich, M., Brandt, T, & Strupp, M. (2009). A bucket of static vestibular function. *Neurology, 72*(19), 1689–1692. doi:10.1212/WNL.0b013e3181a55ecf