

Dizziness in Orthopaedic Physical Therapy Practice: History and Physical Examination

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Abstract: Physical therapy (PT) differential diagnosis of patients complaining of dizziness centers on distinguishing those patients who might benefit from sole management by the physical therapist from those patients who require referral for medical-surgical differential diagnosis and (co) management. There is emerging evidence that PT management may suffice for patients with benign paroxysmal positional vertigo, cervicogenic dizziness, and musculoskeletal impairments leading to dysequilibrium. This article provides information on the history taking and physical examination relevant to patients with a main complaint of dizziness. The intention of the article is to enable the therapist to distinguish between patients complaining of dizziness due to these three conditions amenable to sole PT management and those patients who likely require referral. Where available, we have provided data on reliability and validity of the history items and physical tests described to help the clinician establish a level of research-based confidence with which to interpret history and physical examination findings. The decision to refer the patient for a medical-surgical evaluation is based on our findings, the interpretation of such findings in light of data on reliability and validity of history items and physical tests, an analysis of the risk of harm to the patient, and the response to seemingly appropriate intervention.

Key Words: Dizziness, History, Physical Examination, Physical Therapy

To facilitate differential diagnosis and screening of patients with a complaint of dizziness, we discussed in an earlier article¹ a diagnostic classification system based on symptomatology and pathophysiology. This classification system included four subtypes of dizziness: vertigo, presyncope, dysequilibrium, and other dizziness. Many tests and measures that are needed for a full differential diagnostic work-up of patients presenting with dizziness are obviously outside of the physical therapy (PT) scope of practice. Many causes of dizziness discussed in that earlier article require medical-surgical management rather than or in addition to PT management. However, there is mounting evidence that conservative measures may be beneficial for a select subset of patients with dizziness. Repositioning maneuvers may decrease

symptoms in patients with benign paroxysmal positional vertigo (BPPV) involving the posterior, horizontal, and anterior semicircular canals²⁻⁶. Manual therapy interventions may positively affect cervicogenic dizziness⁷. Musculoskeletal impairments, such as decreased muscle strength and endurance, joint stability and mobility, and posture, which are implicated in patients with the dysequilibrium subtype of dizziness, are dysfunctions traditionally addressed by PT¹. Habituation exercises have proven beneficial for patients with acute unilateral vestibular loss, and adaptation and balance exercises have produced positive outcomes in patients with chronic bilateral vestibular deficits⁴. For the latter two patient groups, PT management, of course, is preceded by a medical differential diagnostic work-up. An isolated otolith dysfunction may theoretically also be amenable to conservative management, but as no clinical tests exist to identify this dysfunction, we cannot make any evidence-based recommendations at this time.

This article provides the orthopaedic physical therapist with current knowledge on the history items and physical tests within the PT scope of practice that are required for identifying previously undiagnosed patients

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complaining of dizziness and who:

- May respond to conservative interventions within the PT scope of practice, specifically patients with BPPV, cervicogenic dizziness, and musculoskeletal impairments leading to dysequilibrium.
- Require referral for medical differential diagnosis and medical-surgical (co)management.

In keeping with the evidence-based practice paradigm, we have attempted to provide, where available, data on reliability and validity of history items and physical tests by way of a Medline search over the period 1995- March 2005 of English-language articles with a title containing search terms relevant to these tests and items. The complete list of search terms is available upon request from the authors. In addition, we performed a hand search of articles in our personal libraries.

History

Our literature search located no studies that discussed the reliability or validity of history items. History taking with patients complaining of dizziness is complex. Table 1 provides a suggested patient self-report intake questionnaire and Table 2 contains a template for a structured interview.

Symptoms

Symptom Description

A description of dizziness symptoms may be helpful for initial classification into one of the four dizziness subtypes of vertigo, presyncope, dysequilibrium, and other dizziness¹. Vertigo is often described as a spinning or rotating sensation, a sensation of self-movement or of the environment moving, whereas patients with presyncopal dizziness complain of lightheadedness, a sense of impending faint, or tiredness. Patients with dysequilibrium may complain of unsteadiness and weakness. Patients who fall into the subtype of other dizziness may report anxiety, depression, or fatigue. However, patients commonly have difficulty describing their symptoms. The above classification system is also challenged when an individual complains of symptoms fitting more than one subtype, as may be the case in older adults with multi-system impairment⁸. However, symptom description indicating presyncopal and other dizziness may indicate the need for referral.

Vertigo

An illusion of rotary movement implicates the semi-circular canals (SCC)⁹. Rotary vertigo is a symptom in most peripheral vestibulopathies. An illusion of linear movement, arguably not true vertigo, indicates a lesion involving the otolith organs but can also occur in patients with a perilymphatic fistula⁹. Vertigo as a result of

peripheral lesions is often severe, intermittent in nature, and of a shorter duration than vertigo due to a central lesion. A central lesion often produces constant but less severe vertigo¹⁰. Vertigo is a symptom in patients with BPPV, Meniere's disease, acute peripheral vestibulopathy, otosclerosis, toxic vestibulopathies, and autoimmune disease of the inner ear^{10,11}. It is less common in patients with cerebellopontine angle tumors or acoustic neuropathy¹⁰. Vertigo may only be episodic in patients with a perilymphatic fistula in case of a low-volume leak but can be severe in patients with a large fistula¹². Vertigo also occurs in the diseases causing brainstem hypoperfusion, e.g., vertebrobasilar insufficiency (VBI), vertebrobasilar infarction, vertebrobasilar migraine, and subclavian steal syndrome^{10,11,13,14}. Any complaint of vertigo other than intermittent, severe, rotary, short-lasting vertigo likely indicates a need for referral.

Ataxia

Ataxia is a dyscoordination or clumsiness of movement not associated with muscular weakness¹⁰. It is a symptom in patients with cerebellar tumors and subclavian steal syndrome¹⁰. Ataxia may affect gait in patients with hypothyroidism, paraneoplastic cerebellar degeneration, ataxia-telangiectasia, Arnold-Chiari malformation, VBI, and myelopathy^{10,13,15-17}. Gait ataxia is the presenting symptom in all patients with hereditary spinocerebellar degenerations¹⁰. It is also the most common finding in patients with alcoholic cerebellar degeneration and the presenting complaint in 10-15% of patients with multiple sclerosis¹⁰. Trunk ataxia is a symptom in patients with ataxia-telangiectasia and Creutzfeldt-Jakob disease¹⁰; these two diseases also produce limb ataxia as does paraneoplastic cerebellar degeneration¹⁰. In addition, 10% of patients with Wernicke's encephalopathy present with ataxia of the arms while 20% present with ataxia affecting the legs¹⁰. A patient report of ataxia confirmed by physical tests indicates a need for referral.

Hearing Loss

A sudden onset of unilateral deafness may be due to labyrinthine artery infarction, possibly indicating an infarction in the vertebrobasilar system¹⁸. A rapid loss of perilymphatic fluid due to a perilymphatic fistula will produce hearing loss, but hearing may be normal in case of a low-volume leak¹². Meniere's disease produces a fluctuating low-frequency hearing loss, which is progressive over multiple episodes^{10,19}. Autoimmune disease of the inner ear also produces a fluctuating hearing loss¹¹. Progressive unilateral hearing loss is also a typical presentation of patients with acoustic neuromas²⁰. Hearing loss is also a symptom in patients with acute labyrinthitis, quinine or quinidine toxicity, salicylate overdose, Friedreich's ataxia, otosclerosis, vestibulocochlear nerve compression due to bacterial, syphilitic, or tuberculous infection or due to sarcoidosis,

Table 1. Patient self-report intake questionnaire.

PATIENT INTAKE QUESTIONNAIRE

Patient Name _____ **Age** _____ **Gender M / F**

MEDICAL HISTORY

Have you in the past been diagnosed with or currently have (check all that apply):

- | | |
|---|--|
| <input type="checkbox"/> Head trauma
<input type="checkbox"/> Neck trauma
<input type="checkbox"/> Inner or middle ear infection
<input type="checkbox"/> Middle ear surgery
<input type="checkbox"/> Inner ear degeneration
<input type="checkbox"/> Recent upper respiratory infection
<input type="checkbox"/> Recent bacterial infection
<input type="checkbox"/> Syphilis
<input type="checkbox"/> Tuberculosis
<input type="checkbox"/> Rheumatoid arthritis
<input type="checkbox"/> Crohn's disease
<input type="checkbox"/> Polyarteritis (auto-immune disease affecting the arteries)
<input type="checkbox"/> AIDS
<input type="checkbox"/> Recent chicken pox
<input type="checkbox"/> Recent mumps
<input type="checkbox"/> Recent poliomyelitis
<input type="checkbox"/> Mononucleosis (Epstein-Barr, Mono) | <input type="checkbox"/> Recent viral infection
<input type="checkbox"/> Recent inoculation
<input type="checkbox"/> Multiple sclerosis
<input type="checkbox"/> Lung cancer
<input type="checkbox"/> Ovarian cancer
<input type="checkbox"/> Hodgkin's disease (lymphatic cancer)
<input type="checkbox"/> Breast cancer
<input type="checkbox"/> Heart disease
<input type="checkbox"/> Chronic obstructive lung disease
<input type="checkbox"/> Atherosclerosis (hardening arteries)
<input type="checkbox"/> Thromboembolic disease (blood clots)
<input type="checkbox"/> Neck degeneration
<input type="checkbox"/> Recurrent episodes of vertebrobasilar ischaemia (limited blood supply to the brain)
<input type="checkbox"/> Visual impairments
<input type="checkbox"/> Migraine or migraine-related disorders
<input type="checkbox"/> Joint replacement in the leg
<input type="checkbox"/> Other orthopaedic surgical procedure |
|---|--|

Have you recently:

- | | |
|---|---|
| <input type="checkbox"/> Been in contact with rodents (mice, guinea pigs, hamsters)
<input type="checkbox"/> Gone diving | <input type="checkbox"/> Gone flying
<input type="checkbox"/> Coughed, sneezed, or strained forcefully
<input type="checkbox"/> Lifted very heavy items |
|---|---|

Has a member of your family ever been diagnosed with or currently have (check all that apply):

- | | |
|--|---|
| <input type="checkbox"/> Familial paroxysmal ataxia
<input type="checkbox"/> Meniere's disease
<input type="checkbox"/> Otosclerosis
<input type="checkbox"/> Migraine
<input type="checkbox"/> Vertebrobasilar migraine | <input type="checkbox"/> Coronary artery disease
<input type="checkbox"/> Peripheral vascular disease
<input type="checkbox"/> Spinocerebellar ataxia
<input type="checkbox"/> Friedreich's ataxia
<input type="checkbox"/> Ataxia-telangiectasia |
|--|---|

Have you used or are you currently using (check all that apply):

- | | | |
|---|---|--|
| <input type="checkbox"/> Alcohol
<input type="checkbox"/> Amikacin
<input type="checkbox"/> Angel Dust
<input type="checkbox"/> Antidepressants
<input type="checkbox"/> Antihypertensives
<input type="checkbox"/> Aspirin
<input type="checkbox"/> Barbiturates
<input type="checkbox"/> Benzodiazepines
<input type="checkbox"/> Bromocriptine
<input type="checkbox"/> Butyrophenones
<input type="checkbox"/> Cis-platinum
<input type="checkbox"/> Digitalis
<input type="checkbox"/> Diuretics
<input type="checkbox"/> Ethchlorvynol | <input type="checkbox"/> Gentamicin
<input type="checkbox"/> Heroin
<input type="checkbox"/> Isoniazid
<input type="checkbox"/> Kanamycin
<input type="checkbox"/> Levodopa
<input type="checkbox"/> Meprobamate
<input type="checkbox"/> Methaqualone
<input type="checkbox"/> Methyl dopa
<input type="checkbox"/> Metoclopramide
<input type="checkbox"/> Monoamine oxidase (MOA) inhibitors
<input type="checkbox"/> Nitroglycerin
<input type="checkbox"/> Phencyclidine
<input type="checkbox"/> Phenothiazines | <input type="checkbox"/> Phenytoin
<input type="checkbox"/> Potassium
<input type="checkbox"/> Procainamide
<input type="checkbox"/> Propranolol
<input type="checkbox"/> Pyridoxine
<input type="checkbox"/> Quinidine
<input type="checkbox"/> Quinine
<input type="checkbox"/> Reserpine
<input type="checkbox"/> Streptomycin
<input type="checkbox"/> Taxol
<input type="checkbox"/> Tetrabenazine
<input type="checkbox"/> Tobramycin
<input type="checkbox"/> Tricyclic antidepressants |
|---|---|--|

How many different medications are you using in total on a daily basis? _____

Are you taking the medication as prescribed? Y / N

Table 2. History form (reason for referral indicated in green).

PATIENT HISTORY

Patient Name _____ Date _____

Symptom description _____

- | | |
|--|--|
| <input type="checkbox"/> Vertigo | <input type="checkbox"/> Dysequilibrium |
| <input type="checkbox"/> Presyncopal dizziness | <input type="checkbox"/> Other dizziness |

Symptom onset Sudden Insidious _____

Precipitating factors Constant Intermittent Episodic _____

- | | |
|--|--|
| <input type="checkbox"/> Transfer sitting to supine position | <input type="checkbox"/> Prolonged standing |
| <input type="checkbox"/> Rolling over in supine | <input type="checkbox"/> While recumbent and motionless |
| <input type="checkbox"/> Head flexion and extension | <input type="checkbox"/> Wearing tight collar |
| <input type="checkbox"/> Transfer supine to sitting position | <input type="checkbox"/> Hyperventilation |
| <input type="checkbox"/> Any head movement | <input type="checkbox"/> Coughing |
| <input type="checkbox"/> Caffeine | <input type="checkbox"/> Urination |
| <input type="checkbox"/> Exercise | <input type="checkbox"/> Rapid rising from sitting |
| <input type="checkbox"/> Alcohol | <input type="checkbox"/> Prolonged neck extension-rotation |
| <input type="checkbox"/> Emotional stimuli | <input type="checkbox"/> Menstrual period |
| <input type="checkbox"/> Pain | <input type="checkbox"/> Arm activity |
| <input type="checkbox"/> Fatigue | <input type="checkbox"/> Anxiety |
| <input type="checkbox"/> Fear | |

Prodromal symptoms Y/N Duration _____

- | | |
|--|--|
| <input type="checkbox"/> Lightheadedness | <input type="checkbox"/> Tachycardia |
| <input type="checkbox"/> Pallor | <input type="checkbox"/> Visual aura |
| <input type="checkbox"/> Salivation | <input type="checkbox"/> Other neurological aura |
| <input type="checkbox"/> Blurred vision | |

Symptom latency Y/N Duration _____

Symptom duration 30-60 sec _____

Symptom fatigability Y/N _____

Associated symptoms

- Ataxia _____
- Hearing loss: Sudden onset Fluctuating Progressive Left Right Both _____
- Tinnitus: Left Right Both _____
- Sensation of fullness in the ear: Left Right Both _____
- Nausea _____
- Vomiting _____
- Dysarthria _____

- Pain _____
- Headache in combination with neck pain
 - Unilateral and pulsating headache
 - Sudden onset neck and occipital pain
 - Chest, neck, and arm pain
 - Tonsillar pillar or external ear pain with swallowing, talking, or coughing
 - Abdominal pain

- Sensory abnormalities _____
- Peri-oral numbness and paraesthesiae
 - Unilateral facial paraesthesiae
 - Quadrilateral paraesthesiae
 - Trigeminal distribution paraesthesiae

- Strength _____
- Facial weakness
 - General fatigue
 - Chronic fatigue
 - Transient quadriplegia
 - Arm fatigue-paralysis
 - Generalized arm and leg weakness

- Visual abnormalities _____
- Loss of color vision
 - Visual field deficits
 - Blurry vision
 - Diplopia with head movement
 - Constant diplopia
 - Tilt illusion
 - Photophobia

- Mental and psychological status _____
- Decreased cognition
 - Acute confusion
 - Memory deficits
 - Stupor
 - Anxiety
 - Depression

- Other _____
- Diaphoresis
 - Hot flushed skin
 - Myoclonus
 - Muscular twitching
 - Spastic bladder
 - Discharge from the ear
 - Thirst
 - Polyuria
 - Polyphagia
 - Unexplained weight loss
 - Palpitations
 - Shortness of breath
 - Coughing
 - Cyanosis
 - Oedema legs
 - Claudication
 - Feeling of choking
 - Feeling of unreality
 - Fear of losing control
 - Fear of dying
 - Insomnia
 - Gastro-esophageal reflux
 - Drop attacks
 - Remitting-relapsing neurological dysfunction

Current history _____

Diagnostic tests _____

Occupation _____

Leisure time _____

Social history N/A _____

General health Unexplained weight loss +/- Night pain +/- Consistent pattern of night pain +/- Loss of appetite +/-
 Other _____

Paget's disease, diabetes mellitus, hypothyroidism, and in 50% of patients treated with the chemotherapeutic drug *Cis-platinum*^{10,19}. Any previously undiagnosed complaint of hearing loss (especially when confirmed by physical tests) indicates a need for referral.

Tinnitus

Tinnitus may occur in patients with Meniere's disease as does a feeling of fullness of the ear^{10,12,19}. Tinnitus also occurs in patients with *Cis-platinum* and salicylate toxicity and in patients with familial paroxysmal ataxia^{10,11}. Tinnitus can also be more benign, resulting from increased tone in the tensor tympani muscle due to trigeminal hyperactivity associated with an upper cervical injury²¹. A complaint of tinnitus combined with aural fullness, or a positive medication history, or a family history positive for familial paroxysmal ataxia indicates a need for referral.

Nausea

Nausea is common in patients with BPPV, Meniere's disease, acute peripheral vestibulopathy, salicylate overdose, quinine or quinidine overdose, cerebellar tumors, Arnold-Chiari malformation, migraine, or VBI^{10,12,13,22}. It can also be indicative of panic disorder^{23,24}. A positive family history for Meniere's disease or a positive medication history in combination with nausea indicates a likely need for referral.

Vomiting

Vomiting may be a symptom for patients with Meniere's disease, acute peripheral vestibulopathy, salicylate overdose, quinine or quinidine overdose, cerebellar tumors, Arnold-Chiari malformation, and vertebrobasilar migraine¹⁰⁻¹². The occurrence of vomiting in patients with BPPV is rare^{22,25}. Vomiting, headache, ataxia, and visual dysfunction are often the presenting symptoms in children with primary cerebellar tumors and a clear indication for referral¹⁰. A complaint of vomiting with dizziness may indicate a need to refer in adults and constitutes a clear reason for referral in children.

Dysarthria

Dysarthria can be a symptom in patients with hypothyroidism, paraneoplastic cerebellar degeneration, Friedreich's ataxia, ataxia-telangiectasia, Creutzfeldt-Jakob disease, familial paroxysmal ataxia, VBI, and vertebrobasilar migraine^{10,11,13}. A complaint of dysarthria indicates the need for referral.

Pain

Headache is a symptom in patients with cerebellopontine angle and cerebellar tumors, salicylate overdose, Arnold-Chiari malformation, familial paroxysmal ataxia, and cervicogenic dizziness^{10,11,26}. In fact, a correlation between neck pain and dizziness is one of the diagnostic

criteria for cervicogenic dizziness²⁶. A unilateral pulsatile headache may be indicative of migraine¹⁰. A sudden-onset neck and occipital pain is the hallmark symptom of vertebral artery dissection^{14,27}. Occipital headache is a symptom of vertebrobasilar migraine¹¹. Chest, neck, and arm pain or discomfort may be symptoms implicating a cardiovascular etiology for patients complaining of presyncopal dizziness²⁸. Chest pain may also occur in patients with panic disorder^{23,24}. Paroxysmal pain in the tonsillar pillar or external ear with swallowing, talking, or coughing implicates glossopharyngeal neuralgia as a cause for presyncopal dizziness^{10,19}. Variable patterns of arm, leg, and trunk pain can be a symptom in patients with myelopathy¹⁵⁻¹⁷. Abdominal pain may occur due to quinine or quinidine toxicity¹⁰. In the context of evaluating patients with dizziness, any pain pattern other than those indicative of cervicogenic dizziness-related neck pain and musculoskeletal pain possibly associated with musculoskeletal impairments causing dysequilibrium indicates a need for referral.

Sensory Abnormalities

Peri-oral numbness and paraesthesiae are a symptom in patients with hyperventilation but also occur in patients with VBI^{10,13}. Limb paraesthesiae are also a symptom for patients with vertebrobasilar migraine¹¹. Bilateral or quadrilateral limb paraesthesiae, either constant or reproduced or aggravated by neck movements may indicate VBI²⁹. Arm paraesthesiae are common in patients with subclavian steal syndrome¹⁰. Non-dermatomal sensory impairments are indicative of myelopathy¹⁵⁻¹⁷. Peripheral neuropathy in the lower extremities commonly occurs in the diabetic population, resulting in impaired somatosensory function³⁰. Paraesthesiae in the trigeminal nerve distribution may occur with cervicogenic dizziness, indicating involvement of the trigemino-cervical nucleus³¹. Trigeminal distribution (facial) and non-dermatomal patterns of paraesthesiae indicate the need for careful evaluation and possible referral.

Strength and Endurance

Facial weakness is a symptom in patients with cerebellopontine angle tumor and familial paroxysmal ataxia^{10,11}. General fatigue occurs in patients with diabetes or cardiovascular etiologies for presyncopal dizziness complaints^{28,32}. Chronic fatigue is also a symptom of panic disorder^{23,24}. Transient quadriplegia is a rare symptom in patients with vertebrobasilar migraine¹¹. Ipsilateral arm fatigue or even paresis is indicative of subclavian steal syndrome¹⁰. Non-myotomal weakness in legs and arms may indicate myelopathy; generally, complaints of weakness may focus the clinician on a musculoskeletal impairment as causative or contributory to the patient's complaint of dizziness or dysequilibrium. Any weakness not directly related to a discrete musculoskeletal

problem indicates a need for referral.

Visual Abnormalities

Quinine and quinidine toxicity may cause vision deficits, including the loss of color vision¹⁰. Visual dysfunction is often one of the presenting symptoms in children with primary cerebellar tumors¹⁰. Visual field deficits may indicate vertebrobasilar infarction¹⁴. Blurred vision may be a prodromal symptom for vasovagal syncope^{10,12}. Visual instability with head movement or oscillopsia suggests an impaired vestibulo-ocular reflex (VOR) and is indicative of vestibular system involvement³³. A tilt illusion or deviation of the subjective visual vertical axis may indicate otolith dysfunction; however, it can also be caused by ischaemia or infarction in the vertebrobasilar system and its branches, unilaterally affecting the vestibular nuclei, the medial longitudinal fascicle, and other nuclei involved in the vestibular mechanism, or the thalamus³⁴. Non-vestibular disorders can also cause a tilt illusion: third and fourth cranial nerve palsies may be responsible for monocular tilts of the subjective visual vertical³⁵. In general, non-vestibular causes for a tilt of the subjective visual vertical result in minor and unpredictable changes as compared to vestibular disorders³⁵. Otolith dysfunction or pathological processes in the otolith-ocular reflex pathways involving central processes can result in patients complaining of vertical diplopia or sometimes diplopia, where one image is tilted in relation to another⁹. Diplopia is also a symptom in patients with paroxysmal familial ataxia, VBI, and subclavian steal syndrome^{10,11,13}. Visual auras can precede vertebrobasilar migraine; 10% of patients with migraine experience a visual or other neurological aura^{10,11}. Photophobia is another symptom in patients with migraine¹⁰. Any report of visual abnormality (with the possible exception of oscillopsia) indicates a need for referral.

Mental and Psychological Status

Changes in mental and psychological status may be noted by the patient or by people close to the patient. Dementia is a state in which there is a significant loss of intellectual capacity and cognitive functioning leading to impairment in social or occupational functioning or both³⁶. Wilson's disease, Creutzfeldt-Jakob disease, hypothyroidism, paraneoplastic syndromes, and some spinocerebellar degenerations may cause dementia in association with ataxia. Dementia with sensory ataxia may indicate neurosyphilis or vitamin B₁₂ deficiency. An acute confusional state with ataxia may occur with alcohol, sedative, salicylate, or hallucinogen intoxication or in patients with Wernicke's encephalopathy. Korsakoff's amnesic syndrome and cerebellar ataxia are associated with chronic alcohol abuse¹⁰. Lassitude is common in patients with migraine¹⁰. Confusion and stupor can result from vertebrobasilar migraine¹¹. Anxi-

ety and depression may be indicative of dizziness due to panic disorder^{23,24}. In one study, depression and panic disorder were present in 50% of patients with initially organic vestibular hypofunction three to five years after onset, leading Tusa³⁷ to suggest that psychological disturbances that develop due to vestibular disorders may become the primary cause of dizziness, replacing the initial organic cause. Eckhardt-Henn et al³⁸ reported that 15.8% of 190 patients complaining of dizziness fell into this category of psychosomatic dizziness. Standardized measures with established reliability and validity, such as the Mini-Mental Status Examination³⁹ and the Beck Depression Inventory⁴⁰, may facilitate communication with a physician when referring a patient for further medical evaluation. Any noted mental or psychological abnormality indicates the need for referral.

Other Symptoms

Diaphoresis is a symptom in patients with acute labyrinthitis, quinine or quinidine toxicity, and panic disorder^{10,12,23,24}. Patients with quinine or quinidine toxicity may indicate hot and flushed skin¹⁰. Fever is a symptom of familial paroxysmal ataxia¹¹. Myoclonus may occur in patients with Creutzfeldt-Jakob disease; hyperventilation is associated with muscular twitching¹⁰. A spastic bladder can be caused by myelopathy¹⁵⁻¹⁷. Patients with undiagnosed skull fractures may note discharge from the ear¹⁰. (Extra) pyramidal signs and symptoms may occur in Creutzfeldt-Jakob disease¹⁰. Carpal tunnel syndrome, myelopathy, and neuropathy may raise suspicion of hypothyroidism in undiagnosed patients¹⁰. The clinician may suspect multiple sclerosis with a history of remitting and relapsing dysfunctions in multiple locations in the nervous system¹⁰. Salicylate toxicity and diabetes mellitus may cause excessive thirst^{10,32}. The clinician may also suspect undiagnosed diabetes in case of polyuria, polyphagia, and unexplained weight loss³². Palpitation and shortness of breath are symptoms in both patients with cardiovascular disease and panic disorder^{23,24,28}. Patients with cardiovascular disease may also note coughing, cyanosis, edema in the legs, and claudication²⁸, whereas patients with panic disorder may complain of a feeling of choking, a feeling of unreality, fear of losing control or dying, insomnia, and gastro-esophageal reflux^{23,24}. Buckling of the legs in response to neck movements without loss of consciousness (i.e., drop attacks) may indicate VBI²⁹. A patient report of any of the symptoms above indicates the need for referral.

Symptom Behavior

Symptom Onset

The initial episode of Meniere's disease has an insidious onset with the patient first noticing tinnitus, hearing loss, and a sensation of fullness in the ear¹⁰. Most

symptoms in patients with central vestibular disorders are the results of slowly progressive pathologies and thus have an insidious onset. The onset of symptoms in patients complaining of dysequilibrium is also generally insidious. The onset of dizziness and other symptoms is sudden in patients with acute peripheral vestibulopathy, aminoglycoside toxicity, labyrinthine damage due to head trauma, in case of large perilymphatic fistulae, and in patients suffering subsequent attacks of Meniere's disease^{10,12}. Presyncopal dizziness usually is sudden in onset when precipitating activities are performed. An abrupt onset is also characteristic of patients with symptoms due to panic disorder²³. An insidious onset of vertiginous dizziness and an abrupt onset of presyncopal or other dizziness indicate a need for referral.

Precipitating Factors

Dizziness is often constant in patients with central and bilateral peripheral vestibular lesions^{9,10}. Other forms of dizziness are intermittent and precipitated by positioning, movement, or other stimuli. Patients with posterior SCC BPPV complain of dizziness when they quickly transfer to a supine position, especially when the head is turned to the affected side²². This also occurs in patients where the anterior SCC is involved, but there is less specificity as to the direction of head rotation⁴¹. Dizziness is brought on in patients with horizontal SCC BPPV when rolling over in supine but it can also occur with flexion and extension of the head or when transferring from supine to upright⁴². Head movement may also provoke symptoms in patients with cervicogenic dizziness²⁶.

Dizziness in patients with otosclerosis may be positional but can also be constant¹⁰. Attacks of familial paroxysmal ataxia can be triggered by exercise, caffeine, alcohol, or sudden movements¹¹. A vasovagal pre-syncope can be brought on by emotional stimuli, pain, the sight of blood, fatigue, medical instruments, blood loss, or prolonged motionless standing^{10,12}, and it usually occurs with the patient in a sitting or standing position; only very rarely is the patient recumbent^{10,19}. A patient complaining of presyncopal dizziness while recumbent or after physical exercise should be screened for a cardiovascular etiology¹⁰. Carotid sinus syndrome has been related to wearing collars that are too tight or may be due to local tumors in the neck pressing on the carotid sinus^{10,19}. In patients with Takayasu's disease, exercise, standing, or head movements may bring on dizziness¹⁰. Hyperventilation and coughing may bring on hyperventilation and cough pre-syncope, respectively¹⁰. Dizziness due to micturition syncope may occur before, during, or after micturition¹⁰. Orthostatic hypotension-related dizziness occurs when rapidly rising from a sitting position, standing up after prolonged recumbency, or after prolonged motionless standing^{10,12}. A position of cervical extension and rotation is often implicated as a trigger for VBI³¹.

Vertebrobasilar migraine occurs frequently during the menstrual period¹¹. Subclavian steal syndrome produces symptoms with physical activity of the ipsilateral arm⁴³. Stress, hyperventilation, and anxiety can all produce the symptoms of dizziness associated with panic disorder^{23,24}. Situations commonly associated with other phobic syndromes (e.g., large crowds, open spaces, driving, or crossing a bridge) can precipitate an attack of phobic postural vertigo³⁸. Dizziness described as tilting of the environment is aggravated by rapid postural changes⁴⁴. Constant vertiginous dizziness or dizziness brought on by factors other than neck or head movement indicate a likely need for referral.

Prodromal Symptoms

Some pathology is characterized by prodromal symptoms, which occur after encountering the precipitating stimulus but before the symptoms of dizziness. Prodromes lasting ten seconds to a few minutes and consisting of lightheadedness, pallor, salivation, blurred vision, and tachycardia often precede a vasovagal syncope^{10,12}. A visual aura may precede migraine and vertebrobasilar migraine¹¹; 10% of patients with migraine report a visual or other neurological aura¹⁰. Any report of prodromal symptoms indicates a need for referral.

Symptom Latency

Symptom latency refers to the time lapsed between exposure to the precipitating stimulus and onset of symptoms. Symptoms in patients with BPPV occur after a 1-5 second latency period^{22,45} but may last up to 40 seconds. The latency period in patients with VBI is long: Oostendorp⁴⁶ reported a latency period of 55 ± 18 seconds after assuming the De Kleyn-Nieuwenhuys test position. One could assume that patients with subclavian steal syndrome also have a longer latency period; sufficient ischaemia needs to develop before symptoms occur. Depending on the etiology, a vertebrobasilar infarction may be rapidly or very slowly progressive¹³. Onset of symptoms is immediate in patients with cervicogenic dizziness upon assuming the provoking position⁴⁵. A prolonged latency period (>60 sec) indicates a likely need for referral.

Symptom Duration

As noted before, dizziness symptoms in patients with central vestibulopathies are generally less severe but constant and prolonged; symptoms with peripheral vestibulopathies are often severe but intermittent¹⁰. Symptoms in patients with BPPV generally last less than 30 seconds but may occur for up to 60 seconds⁴⁵. Vertigo may last from minutes to days in patients with Meniere's disease^{10,12,19}. In patients with acute peripheral vestibulopathy, vertigo may be constant for up to two weeks¹⁰. Symptoms in patients with familial paroxysmal ataxia last from 15 minutes to several hours¹¹. Symptoms

last for up to 72 hours in patients with vertebrobasilar migraine¹¹. Symptoms in patients with VBI and subclavian steal syndrome are progressive and non-accommodating until the precipitating postures or activities are discontinued^{43,45}. One could assume that based on the pathophysiology, the other types of presyncopal dizziness will behave similarly. The duration of symptoms in patients with cervicogenic dizziness is usually brief after assuming the provoking position, although symptoms have been reported as lasting minutes to hours^{26,45}. Dizziness and other symptoms in patients with panic disorder have an abrupt onset and peak in about 10 minutes²³. Symptom duration of >60 seconds and especially non-accommodating forms of dizziness indicate the need for referral.

Symptom Frequency

Dizziness associated with precipitating factors is, of course, recurrent in nature depending on exposure to those factors, as discussed above. Dizziness and other symptoms are episodic in patients with Meniere's disease, otosclerosis, perilymphatic fistulae with low-volume leaks, migraine, vertebrobasilar migraine, panic disorder, and phobic postural vertigo^{10,12,19,23,38}. We have already discussed the constant symptoms in patients with central vestibular lesions^{1,10}. Episodic bouts of dizziness indicate a likely need for referral.

Symptom Fatigability

Fatigability of symptoms refers to the decrease in symptoms of vertigo and nystagmus with repeated positioning⁴¹. It is characteristic for cervicogenic dizziness and BPPV⁴⁵. Non-fatigable dizziness indicates a likely need for referral (even though few patients will willingly provoke dizziness repeatedly to find out about this characteristic).

Pertinent Past and Present Medical History

Patient Demographics

Ataxia-telangiectasia has its onset before the age of four¹⁰. Friedreich's ataxia also starts in childhood¹⁰. Migraine and vertebrobasilar migraine too usually have their onset early in life^{10,11}. Takayasu's disease affects mainly women between the ages of 15 and 30⁴⁷. Panic disorder often first occurs in young adulthood²³. Hyperventilation most commonly affects patients between 20 and 40¹⁰. The age of onset in Meniere's disease is usually between 20 and 50¹⁰. Hearing loss associated with otosclerosis generally starts before age 30¹⁰. Cerebellopontine angle tumors have an age of onset between 30 and 60¹⁰. The age of onset for alcoholic cerebellar degeneration is 40 to 60¹⁰. Cough syncope is most prevalent in middle-aged men¹⁰. Hypothyroidism is most common in middle-aged or elderly women¹⁰. BPPV generally occurs in people over age 40; it rarely occurs in people under 20⁴⁵; however,

peak incidence of onset for BPPV is in the sixth decade of life⁴⁸. Orthostatic hypotension is most common in people in the sixth and seventh decades^{10,12}. Parkinsonism is most prevalent in older adults^{10,12}. Vasovagal syncope as a cause of dizziness can occur in all age groups^{10,12}. Onset of dizziness and ataxia in childhood is a strong indicator for referral.

Men are more often affected by Meniere's disease, alcoholic cerebellar degeneration, orthostatic hypotension, carotid sinus syndrome, and cough syncope^{10,12}. Women are more often affected by hypothyroidism, migraine, vertebrobasilar migraine, and hyperventilation-induced presyncope^{10,11}. Takayasu's disease affects only women while micturition syncope occurs almost exclusively in men¹⁰.

Takayasu's disease only affects women of Asian descent¹⁰. Parkinsonism affects all ethnic groups equally¹⁰.

Medical History

Past and concurrent medical history may provide diagnostic or screening clues in patients with complaints of dizziness. A medical history of head trauma, labyrinthine infection, surgical stapedectomy, chronic suppurative otitis media, and degenerative changes to the inner ear may indicate non-idiopathic BPPV^{22,25,45}. An upper respiratory infection precedes acute peripheral vestibulopathy in 50% of patients¹². Acoustic neuromas are more common in patients with neurofibromatosis¹⁰. Vestibulocochlear nerve compression can be the result of bacterial, syphilitic, and tuberculous infection or sarcoidosis¹⁰. Barotrauma due to diving or flying, a forceful Valsalva maneuver, or head trauma can produce a perilymphatic fistula^{12,19}. Head trauma can also cause occult skull fractures; a petrosal bone fracture can cause vertigo and hearing loss¹⁰. Autoimmune diseases such as rheumatoid arthritis, Crohn's disease, and polyarteritis are often concurrently present in patients with autoimmune disease of the inner ear¹¹. AIDS, varicella, mumps, poliomyelitis, infectious mononucleosis, and lymphocytic choriomeningitis (a virus borne by rodents) can all provide the viral agent responsible for viral cerebellar infections¹⁰. Acute cerebellar ataxia of childhood is often preceded by a viral infection or inoculation¹⁰. Vertigo is a common symptom in patients with multiple sclerosis, albeit not often the presenting symptom¹⁰. Epilepsy in the medical history should prompt questions about phenytoin: Long-term treatment with phenytoin may produce cerebellar degeneration¹⁰. Patients with lung cancer, ovarian cancer, Hodgkin's disease, and breast cancer are at risk for paraneoplastic cerebellar degeneration¹⁰. Breast and lung cancer are also apt to metastasize to the posterior fossa in adults¹⁰. A medical history positive for heart disease could imply a cardiovascular origin for presyncopal dizziness complaints²⁸. Chronic obstructive pulmonary disease is frequent in patients with cough-related presyncopal dizziness¹⁰. Atherosclerosis, thrombo-embolic

disease, and cervical spine trauma and degeneration may predispose patients to VBI^{25,27,49-51}. Atherosclerosis can also lead to subclavian steal syndrome¹⁰. Recurrent episodes of VBI predispose patients to vertebrobasilar infarction¹⁰. Cervical spine trauma and degeneration may also be at the basis of cervicogenic dizziness^{26,52}. A recent optometry or ophthalmology report may reveal the visual impairments associated with complaints of dysequilibrium⁵³. A recent lower extremity joint replacement or other orthopaedic surgery may be the cause for dysequilibrium in the elderly patient. A history of migraine or migraine-related disorders has been associated with vestibular dysfunction^{54,55}; in fact, vertigo is three times more common in patients with migraine and there is a 30-50% prevalence of migraine in patients with vertigo⁵⁶. With the exception of neck trauma and degeneration and recent lower extremity joint replacement or other orthopaedic procedure, a positive medical history in the absence of signs and symptoms indicative of the three conditions amenable to sole PT management discussed above may indicate the need for referral.

Family History

Familial paroxysmal ataxia is a hereditary recurrent form of ataxia¹¹. Also, 20% of patients with Meniere's disease have a positive family history¹². Patients with otosclerosis, migraine, and vertebrobasilar migraine also commonly have a positive family history^{10,11}. Coronary

artery disease and peripheral vascular disease with a possible role in producing presyncopal dizziness have a strong family history⁵⁷. Some of the spinocerebellar ataxias are hereditary autosomal dominant diseases, while Friedreich's ataxia and ataxia-telangiectasia are autosomal recessive diseases¹⁰. A positive family history linked to relevant pathognomonic signs and symptoms constitutes a reason for referral.

Medication History

Table 3 lists prescription, over-the-counter, and recreational drugs associated with the various subtypes of dizziness, allowing the therapist to establish whether symptom description matches the possibly causative medication use reported. A strong relationship has been established between the number of medications taken (>5) and dizziness symptoms⁸. Careful questioning may implicate such overmedication as a cause of dizziness. Non-compliance with medication may also be an issue, for example, the failure to take antidepressants in a patient with panic disorder. Additionally, the use of a particular medication may signal to the therapist a medical condition that the patient failed to report. A positive medication history with symptoms indicative of a relevant dizziness subtype (see Table 3), poly-pharmacy, and non-compliance with prescribed medication may all constitute reason for referral.

Table 3. Medications associated with subtypes of dizziness.

Vertigo	Presyncope	Dysequilibrium	Other dizziness
Alcohol	Digitalis	Phenothiazines	Alcohol
Aminoglycoside antibiotics	Quinidine	Butyrophenones	Aminoglycoside antibiotics
• Streptomycin	Procainamide	Metoclopramide	• Streptomycin
• Gentamicin	Propranolol	Reserpine	• Gentamicin
• Tobramycin	Phenothiazines	Tetrabenazine	• Tobramycin
• Amikacin	Tricyclic antidepressants	Angel Dust	• Amikacin
• Kanamycin	Potassium	Cis-platinum	• Kanamycin
Salicylates	Methyldopa	Isoniazid	Salicylates
Quinine and quinidine	Antidepressants	Pyridoxine	Quinine and quinidine
Cis-platinum	Antihypertensives	Taxol	Cis-platinum
Sedative hypnotics	Bromocriptine		
• Barbiturates	Diuretics		
• Benzodiazepines	Levodopa		
• Meprobamate	Monoamine oxidase (MOA) inhibitors		
• Ethchlorvynol	Nitroglycerin		
• Methaqualone	Phenothiazines		
Anti-convulsants			
• Phenytoin			
Hallucinogens			
• Phencyclidine			
Street drugs			
• Heroin			
Mercuric and organophosphoric compounds			

Physical Examination

Physical examination with the aim of differential diagnosis in patients complaining of dizziness requires a multitude of tests. Table 4 provides a suggested format for the physical examination. The proposed order of examination in this format is intended to safeguard previously undiagnosed patients from unnecessary and potentially harmful physical tests by establishing the need for referral and obviating the need for further testing in case of a positive response to an earlier physical test.

Observation

Skin Observation

Children with ataxia-telangiectasia have tiny red “spider” veins on the ears and cheeks. Dry skin with brittle hair may indicate hypothyroidism. Vitamin B₁₂ deficiency can cause a lemon-yellow skin discoloration. Papilledema due to increased intracranial pressure occurring together with dysequilibrium is indicative of an intracranial mass lesion, usually in the posterior fossa¹⁰. Clubbing of the fingernails, cyanosis of lips, trophic changes of the skin, and peripheral edema could suggest a cardiovascular disorder^{36,57}. All abnormalities above in combination with relevant symptoms noted in the history may indicate the need for referral.

Postural Observation

Postural deviations negatively affecting the location of the center of gravity in relation to the base of support may result in patients complaining of the dysequilibrium type of dizziness. These deviations also prompt further musculoskeletal examination to determine cause and potential management strategies. Postural deviations may also indicate possible pathology. Friedreich’s ataxia typically causes an increased kyphoscoliosis. Neurosyphilis frequently leads to hypertrophic or hypermobile joints with subsequent effects on posture. Craniocervical junction abnormalities can occur with Arnold-Chiari malformation¹⁰. A lateral head tilt might indicate an otolith problem (tilting of the environment) or just tightness of the sternocleidomastoid or upper trapezius commonly seen in cervicogenic dizziness^{26,58}, but it may also be caused by ischaemia or infarction in the vertebrobasilar system³⁴. A forward head posture, with the head backward bent on the upper cervical spine, may cause external mechanical compression of the vertebral artery, thus potentially producing symptoms of VBI^{31,49}, but it may also lead to hypomobility of soft tissue and joint structures especially in the upper cervical spine⁴⁹, an area implicated in cervicogenic dizziness. Craniocervical junction abnormalities and lateral head tilt may indicate the need for referral.

Eye Observation

Pigmented corneal Kayser-Fleischer rings are due to

copper deposition in the cornea in patients with Wilson’s disease¹⁰. Children with ataxia-telangiectasia also have “spider” veins in the corners of the eyes. Vertical and horizontal misalignments of the eyes may be caused by cranial nerve palsies. A skew deviation is a vertical misalignment of the eyes that is not the result of ocular muscle palsy⁹. Skew deviation is best detected by alternately covering the eyes: Patients with skew deviation make a vertical corrective movement in the sense of a lateral head tilt when switching the cover from the unaffected to the affected side. Skew deviation, head tilt, and ocular counter-rolling constitute the ocular tilt reaction⁹. Unilateral lesions of the vestibular nucleus, the medial longitudinal fascicle, and other vestibular centers due to vertebrobasilar infarction can produce a full ocular tilt reaction³⁴. A unilateral thalamus lesion or a benign otolith dysfunction can produce a partial ocular tilt reaction³⁴. In patients with peripheral or vestibular nucleus lesions, the lower eye indicates the side of the lesion; lesions above the level of the vestibular nucleus present with the higher eye on the side of the lesion⁹. Any of these abnormalities indicate a need for referral.

Vital Signs

Blood Pressure

In patients with subclavian steal syndrome, a difference in blood pressure between the affected and non-affected arm is virtually always present. On average, systolic blood pressure is 45 mm Hg lower in the arm supplied by the stenotic blood vessel¹⁰. Symptoms indicative of subclavian steal syndrome in combination with a ≥ 45 mm Hg lower systolic blood pressure in the symptomatic arm is a reason for referral. Hypertension and hypotension can contribute to dizziness symptoms³⁶. Monitoring the patient’s blood pressure response when transferring from a lying to a standing position is used as a diagnostic test for orthostatic hypotension. A drop in systolic blood pressure of ≥ 30 mm Hg or a drop of 10 mm Hg in diastolic blood pressure is indicative of orthostatic hypotension¹⁰. Eaton and Roland¹² considered a drop of 20 mmHg in systolic or 10 mm Hg in diastolic blood pressure two minutes after standing indicative of orthostatic hypotension, but they also warned that blood pressure readings in elderly patients might not precisely meet those criteria. Witting and Gallagher⁵⁹ established normative values: In 176 healthy subjects, systolic blood pressure decreased by 1.2 ± 9.8 mm Hg after one minute of standing preceded by five minutes of sitting. A drop in systolic blood pressure of ≥ 20 mm Hg had a specificity of 0.97 for detecting orthostatic hypotension⁵⁹. Combined with a complaint of presyncopal dizziness, this finding warrants referral.

Heart Rate

Palpation of pulses may be useful in detecting a

Table 4. Physical examination form (reason for referral indicated in green).

PATIENT PHYSICAL EXAMINATION

Patient Name _____ Date _____

OBSERVATION

- Skin** Red spider veins on ears and cheeks Dry skin Brittle hair Lemon-yellow discoloration skin
 Papilledema Clubbing fingernails Trophic changes skin Peripheral oedema
- Posture** Increased kyphoscoliosis Craniocervical junction abnormalities Lateral head tilt
 Forward head posture
- Eyes** Pigmented corneal rings Red spider veins corner of the eyes Vertical misalignment L high
 Vertical misalignment R high Horizontal misalignment
 Corrective lateral head tilt when covering one eye in case of vertical misalignment

Other _____

VITAL SIGNS

- Blood pressure** Arm systolic difference (≥ 45 mm Hg) +/- _____
- Heart rate** Palpitations +/- _____
- Sit-to-stand test** Blood pressure (decrease ≥ 20 mm Hg) +/- Heart rate (increase ≥ 20 bpm) +/-
 Lightheadedness _____
- Auscultation** Carotid bruit +/- Cardiac abnormalities +/- _____

GAIT ASSESSMENT _____

- | | |
|--|---|
| <input type="checkbox"/> Wide-based gait | <input type="checkbox"/> Steppage gait |
| <input type="checkbox"/> Titubation | <input type="checkbox"/> Improved gait with assistive device |
| <input type="checkbox"/> Unilateral deviation when walking straight line | <input type="checkbox"/> Difficulty with concurrent head rotation |
| <input type="checkbox"/> Unable to walk tandem gait | <input type="checkbox"/> Wildly lurching without loss of balance |

VESTIBULO-SPINAL EXAMINATION

- Single leg stance** L ____ sec R ____ sec _____
- Romberg** Eyes open _____ Eyes closed _____
- Sharpened Romberg** Eyes open _____ Eyes closed _____
- CTSIB** Level, eyes open _____ Level, eyes closed _____
 Foam, eyes open _____ Foam, eyes closed _____
- Fukuda step test** Rotate $> 30^\circ$ +/- Rotate L/ R Forward displacement > 50 cm +/-

CRANIAL NERVE EXAMINATION _____

Cranial nerve	Test	L/R		
I. Olfactory	Identify different odors		+	-
II. Optic	Test visual fields (Confrontation method)		+	-
III. Oculomotor	Upward, downward, and medial gaze		+	-
IV. Trochlear	Downward and lateral gaze		+	-
V. Trigeminal	Corneal reflex, face sensation, clench teeth		+	-
VI. Abducens	Lateral gaze		+	-
VII. Facial	Close eyes tight, smile, whistle, puff cheeks		+	-
VIII. Vestibulo-cochlear	Hear watch ticking, hearing tests, balance tests		+	-
IX. Glossopharyngeal	Gag reflex, ability to swallow		+	-
X. Vagus	Gag reflex, ability to swallow, say "Ahhh"		+	-
XI. Accessory	Resisted shoulder shrug		+	-
XII. Hypoglossal	Tongue protrusion (Observe for deviation)		+	-

OCULOMOTOR EXAMINATION

Spontaneous nystagmus central gaze +/- _____

- Jerk
- Pendular
- Horizontal L R
- Vertical Upbeat Downbeat
- Torsional
- Skew
- Alternating Periodic Irregular
- Suppressed with visual fixation Yes No
- Suppressed by convergence
- Increased by fixation

Spontaneous nystagmus eccentric gaze +/- _____

- Increased with looking towards fast phase
- Horizontal nystagmus
- Provoked on lateral or upward gaze
- Small unsustained eye movements at end range

Saccadic eye movements +/- _____

- Hypometria
- Hypermetria
- Horizontal saccades with vertical test
- Saccades during fixation on target
- Oscillating horizontal saccades with gaze shift

Smooth pursuit testing +/- _____

HEARING EXAMINATION

Weber test Midline L R

Rinne test Bone conduction > air conduction Bone conduction ≤ air conduction

AROM EXAMINATION Asterixis +/- Myoclonus +/- Chorea +/- _____

LIMB ATAXIA EXAMINATION

Finger-to-nose test Intention tremor L+/R+/- Overshooting L+/R+/-

Finger-to-finger-test Horizontal overshooting L+/R+/- Vertical overshooting

Heel-to-shin test Intention tremor L+/R+/- Overshooting L+/R+/-

Toe-to-finger test Intention tremor L+/R+/- Overshooting L+/R+/-

Dysidiadochokinesia Finger tapping +/- Pronation-supination +/- Toe tapping +/-

Barre test L+/R+/- _____

PROM EXAMINATION Hypotonia +/- Rigidity +/- Spasticity +/- Clonus +/- _____

Stability tests _____

PPIVM/PAIVM _____

STRENGTH EXAMINATION _____

REFLEX EXAMINATION Hoffman's reflex L+/R+/- Babinski sign L+/R+/- DTR _____

SENSATION TESTS _____

Joint position sense _____

Vibration sense _____

VERTEBROBASILAR EXAMINATION

De Kleyn-Nieuwenhuys test L+/R+/- _____

Latency _____ sec > 60 sec Vertical downbeat nystagmus

Duration _____ sec Non-accommodating Other nystagmus

Fatigable Y/N Geotropic

Horizontal nystagmus Apogeotropic

Torsional nystagmus

Sustained cervical rotation test L+/R+/- _____

- Latency _____ sec > 60 sec
 Duration _____ sec Non-accommodating
 Fatigable Y/N
 Horizontal nystagmus
 Torsional nystagmus
- Vertical downbeat nystagmus
 Other nystagmus
 Geotropic
 Apogeotropic

Hautant test Midrange +/- EXT-ROT L+/R+/- Latency with + EXT-ROT position Y/N

VESTIBULO-OCULAR EXAMINATION

Dynamic visual acuity Decrease by ≥ 2 lines on Snellen chart +/- _____

Doll's head test Catch-up saccades towards fixation target +/- _____

Head-shaking nystagmus test +/- Nystagmus towards side lesion Nystagmus away from side lesion

Head thrust test +/- Corrective saccade on head moving right Corrective saccade on head moving left

BPPV EXAMINATION

Hallpike-Dix L+/R+/- _____

- Positive bilateral L < R L > R
 Latency _____ sec > 60 sec
 Duration _____ sec Non-accommodating
 Fatigable Y/N
 Horizontal nystagmus
- Torsional nystagmus
 Vertical downbeat nystagmus
 Other nystagmus
 Geotropic
 Apogeotropic

Straight head-hanging test +/- _____

- Latency _____ sec > 60 sec
 Duration _____ sec Non-accommodating
 Fatigable Y/N
 Horizontal nystagmus
- Torsional nystagmus
 Vertical downbeat nystagmus
 Other nystagmus

Roll test +/- _____

- L < R
 L > R
 Latency _____ sec
 Duration _____ sec
 Fatigable Y/N
 Horizontal nystagmus
- Torsional nystagmus
 Vertical downbeat nystagmus
 Other nystagmus
 Geotropic
 Apogeotropic

Walk-rotate-walk test L+/R+/- _____

CERVICOGENIC DIZZINESS EXAMINATION

Neck torsion test L+/R+/- _____

- Latency Immediate _____ sec
 Duration _____ sec
 Fatigable Y/N
 Horizontal nystagmus
- Torsional nystagmus
 Vertical downbeat nystagmus
 Other nystagmus

BREATHING-RELATED TESTS

Hyperventilation test Dizziness +/- Nystagmus +/- Minimal latency +/- Latency _____ sec

Valsalva test Dizziness +/- Nystagmus +/- Minimal latency +/- Latency _____ sec

Cough test Dizziness +/-

cardiovascular disorder. Palpitations, the presence of an irregular heartbeat, may indicate a disturbance in the heart's ability to normally conduct electrical impulses³⁶ and may be benign or quite dangerous. Palpitations lasting for hours or irregular heartbeats accompanied by pain, shortness of breath, or lightheadedness require referral to a physician for medical evaluation³⁶. Similarly, tachycardia (>100 bpm) and bradycardia (<60 bpm) may indicate relatively benign conditions, such as mitral valve prolapse and "athlete's heart" but may also occur in more serious conditions such as coronary artery disease and aneurysm^{36,57}. Monitoring pulse rate during a sit-to-stand test may also be helpful for diagnosing orthostatic hypotension: Witting and Gallagher⁵⁹ established a normative value of a pulse rate increase of 5.3 ± 6.6 bpm in normal subjects and suggested an increase of ≥ 20 bpm as a positive test for orthostatic hypotension based on a sensitivity of 0.98. Combined with a complaint of presyncopal dizziness, this finding warrants referral.

Auscultation

Auscultation tests can provide information on a possible cardiovascular disorder responsible for a patient complaint of presyncopal dizziness. Lok, Morgan, and Ranganathan⁶⁰ found poor accuracy and inter-rater agreement for identification of some cardiac auscultation parameters. Listening for carotid bruits has been suggested as a screening tool for the likelihood of a vertebrobasilar incident with cervical manipulation¹³. We earlier discussed the role of collateral (i.e., carotid) circulation in the occurrence of VBI¹. Terrett¹³ noted that the validity of carotid bruits in the diagnosis of carotid stenosis or prediction of a vertebrobasilar incident is questionable. Negative auscultation results would seem to provide the therapist with a false sense of doing a relevant vertebrobasilar screening¹³. In contrast, Magyar, Nam, Csiba, et al⁶¹ reported 56% sensitivity and 91% specificity for detection of a 70-99% carotid stenosis when compared with color duplex ultrasound. They also reported a positive predictive value of 27% of a bruit found and a 97% negative predictive value for a normal auscultation. They concluded that carotid auscultation is a useful screening procedure for carotid occlusion or stenosis. In light of the possible contradictory interpretation of these values for diagnostic test accuracy for auscultation of carotid bruits and the poor values for accuracy of cardiac auscultation, positive auscultation findings indicate the need for cautious continued examination.

Gait Assessment

Patients with cerebellar ataxia have a wide-based staggering gait, sometimes with titubation (staggering or stumbling gait) or oscillation of head and trunk¹⁰.

Unilateral cerebellar lesions result in a deviation towards the side of the lesion when the patient attempts to walk in a straight line. Patients with cerebellar ataxia are unable to walk in a tandem gait. In patients with sensory ataxia, gait is also wide-based. Impaired proprioception may cause steppage gait: The patient lifts the feet excessively high off the ground and slaps them down rather heavily. Using a cane or a railing often dramatically improves gait¹⁰. Difficulty walking with concurrent rotation of the head in the horizontal plane may indicate a peripheral vestibular deficit. Gait unsteadiness may also be a complaint in patients with psychiatric or factitious disorders. Simon, Aminoff, and Greenberg¹⁰ noted that wildly reeling or lurching movements from which the patient is able to recover without loss of balance may be indicative of conversion disorder or malingering: Recovery of balance from self-imposed extreme positions and movements in fact demonstrates well-developed balance function. Gait assessment can also be done quantitatively with measures such as the Tinetti Balance Scale and Berg Balance Scale, both with established predictive validity with regards to fall risk. The former has been reported to identify 7 out of 10 fallers with 70% sensitivity and 52% specificity⁶², whereas the latter was able to correctly identify fallers from non-fallers with 91% sensitivity and 82% specificity⁶³. A score on either measure indicative of a low fall risk despite a complaint of dizziness and dysequilibrium may indicate kinesiphobia, which can be classified under other dizziness and may indicate the need for referral. Titubation, oscillation of head and trunk, unilateral deviation when attempting to walk in a straight line, and wild reeling or lurching motions without loss of balance are less likely indicators of musculoskeletal impairments and, therefore, indicators for referral.

Vestibulo-Spinal Examination

The vestibulo-spinal reflex (VSR) stabilizes the body during head movement; thus, it is responsible for postural control. The vestibulospinal tests in general have poor or untested diagnostic accuracy but can serve to guide further examination by indicating the presence of postural instability and by implicating the vestibular versus somatosensory system. In isolation, these tests do not affect a decision to refer or treat.

Single Leg Stance

Single leg stance—with eyes open or closed—can be used to screen for decreased postural control. In the acute stage of vestibular loss, a patient will be unable to perform this test⁶⁴; however, patients who have a compensated vestibular loss may test normal⁶⁴. This screening test is not specific to vestibular loss, as patients with other balance disorders may have difficulty performing single leg stance⁶⁴. A normal single

leg stance test (especially with eyes closed) precludes further vestibulospinal testing.

Romberg and Sharpened Romberg

The Romberg test (Figure 1A) challenges balance by decreasing the base of support. Patients with sensory or vestibular dysfunction may be able to stand in a Romberg stance, but closing the eyes takes away the visual cues used to maintain balance, causing them to fall, i.e., have a positive Romberg sign¹⁰. Patients with a vestibular lesion tend to fall in the direction of the lesion¹⁰. Patients with cerebellar ataxia are unable to use visual cues to compensate and are unable to maintain their balance in a Romberg stance whether their eyes are open or closed¹⁰. A sharpened Romberg (Figure 1B) test involves standing with a decreased base of support

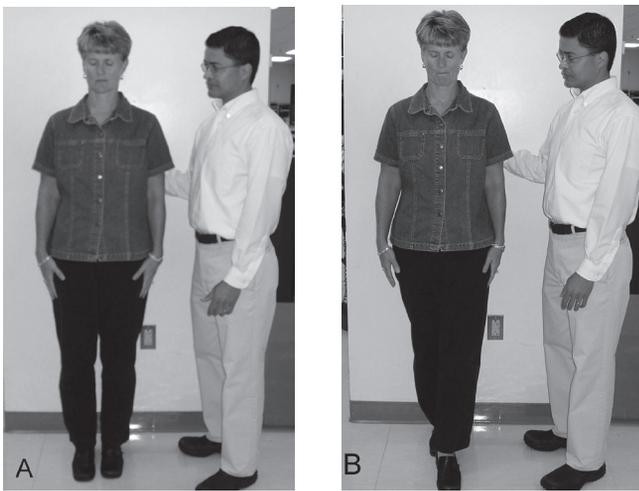


Fig. 1a-b: Romberg test-Sharpended Romberg test

as compared to the Romberg test. The ataxic patient will prefer to stand with a wider base of support and will show reluctance when asked to stand with the feet close together. Patients with sensory ataxia are usually able to stand with the feet close together, as are some patients with vestibular lesions. These patients will compensate for the loss of somatosensory and labyrinthine input, respectively, with an increased reliance on visual input. The Romberg test has predictive validity with regards to recurrent falls over a 6-month period in patients with Parkinson's disease: sensitivity was 65% and specificity >90%⁶⁵.

Modified Clinical Test of Sensory Integration of Balance

The modified Clinical Test of Sensory Integration of Balance (CTSIB) assesses the contribution of the visual, vestibular, and somatosensory systems to postural control. The test has four components with the patient standing on a level surface with the eyes open (Figure 2A), on a level surface with eyes closed (Figure 2B), on foam with the eyes open (Figure 2C), and on foam with eyes closed (Figure 2D). Initially, a patient will have available all sensory systems to maintain balance. The eyes-closed condition will eliminate visual contribution, putting increased demand on the somatosensory and vestibular systems. Standing on a foam surface with eyes closed alters the somatosensory input and eliminates visual input; thus, the patient has to rely mostly on vestibular input. Patients with vestibulopathy will have difficulty maintaining an upright posture⁶⁴. Platform posturography is a computerized version of this test with >90% specificity but very low sensitivity for the diagnosis of patients with peripheral vestibular deficits⁶⁶. Posturography in combination with other vestibular function tests has been shown to increase sensitivity to 61-89%⁶⁶.

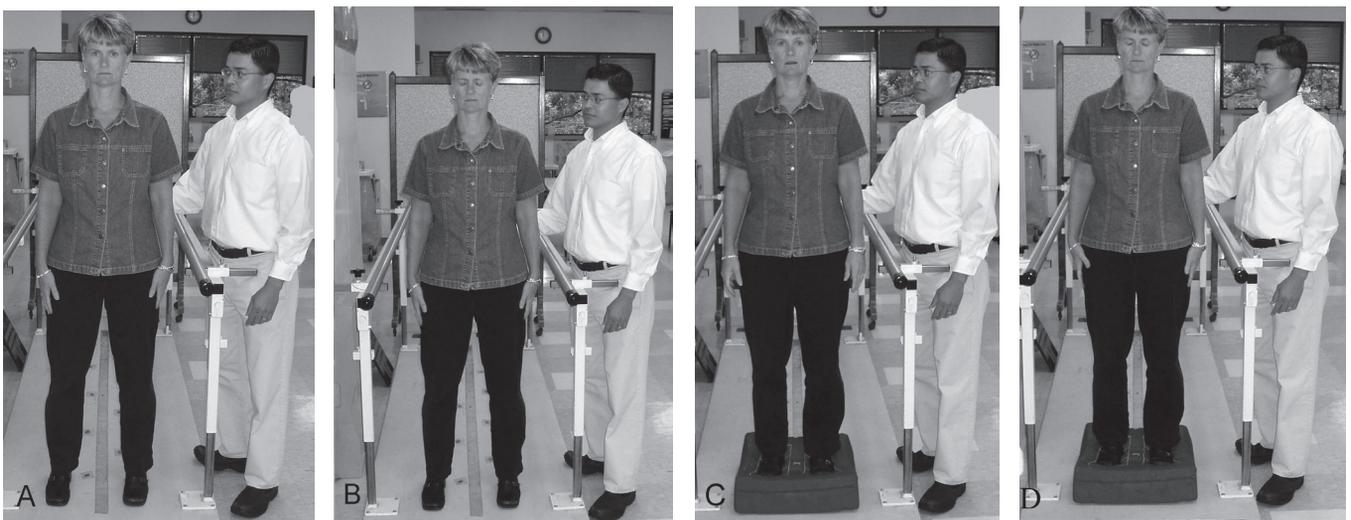


Fig. 2a-d: Modified Clinical Test of Sensory Integration of Balance (CTSIB)

Fukuda Step Test

The Fukuda step test (Figure 3) assesses stability during self-initiated movement by asking the patient to march 50 or 100 steps in place with the arms raised in front to 90° and with the eyes closed. A patient with a unilateral vestibular lesion will tend to rotate >30° toward the involved side⁶⁴. Forward displacement of >50 cm is also considered positive⁶⁷. These unilateral lesions include infarctions in the distribution of the anterior and posterior inferior cerebellar arteries³⁴. Bonanni and Newton⁶⁸ found higher reliability for the 50-step than the 100-step protocol. Herdman and Whitney⁶⁴ noted that there are many false positives and negatives. Fell⁶⁷ noted that the Fukuda step test is not a test specific to vestibular lesions.

Cranial Nerve Examination

Cranial nerve (CN) palsies may be present with central vestibular disorders and some peripheral vestibular disorders. A CN examination may also serve as a non-provocative test for suspected ischaemic conditions affecting the brainstem. Obviously, the vestibulo-cochlear nerve can be involved in patients complaining of dizziness as well as the anatomically closely related trigeminal and facial nerves¹⁰. Optic neuropathy can be the result of multiple sclerosis, neurosyphilis, and vitamin B₁₂ deficiency. A depressed corneal reflex or a facial nerve palsy on the same side as the ataxia can result from a cerebellopontine angle tumor. Lower brainstem disease can cause tongue or palate weakness, hoarseness, and dysphagia⁶⁹. Some pathologies cause dizziness in combination with hearing loss. Table 4 contains a sample CN examination⁷⁰. Visual

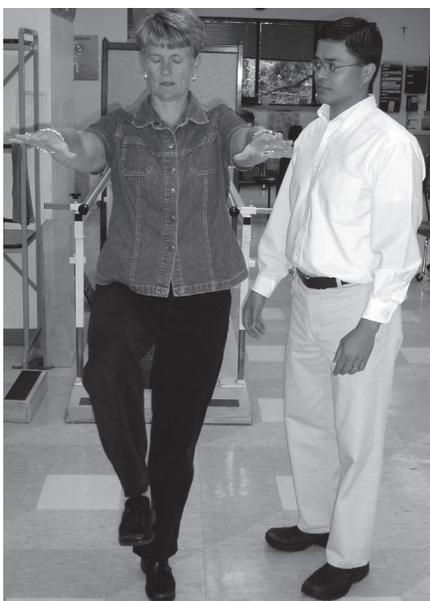


Fig. 3: Fukuda step test

field confrontation testing (CN II) had low sensitivity but high specificity (97%) and positive predictive value (96%) when compared to automated perimetry⁷¹, indicating that a confrontation-method visual field test may only have diagnostic value if positive. We found no further data on reliability and validity of the CN examination. Abnormal findings on the CN examination constitute a reason for referral.

Oculomotor Examination

To some extent, observation and the CN examination already test oculomotor function. They also allow clinicians to note static abnormalities (strabismus) and ensure full range of movement for each eye before doing the oculomotor tests. No data on reliability and validity of the oculomotor examination were found.

Observation for Spontaneous Nystagmus

Nystagmus can be defined as repetitive, back-and-forth, involuntary eye movements initiated by slow drifts away from the visual target⁷². It can be classified as a *pendular* nystagmus, consisting of slow sinusoidal oscillations, or as a *jerk* nystagmus, characterized by an alternating slow drift and a quick corrective phase. In the latter type, a slow phase takes the eye away and a quick corrective phase brings it back to the target⁷².

The clinician first observes for spontaneous nystagmus by asking the patient to fix on a stationary target at a distance of >2 meters⁷². A spontaneous nystagmus may imply an acute peripheral vestibular lesion and, in this case, occurs due to an imbalance in the tonic firing rate of the vestibular neurons⁷³. The spontaneous nystagmus following a lesion of the peripheral vestibular system is a jerk nystagmus with the quick phase indicating the unaffected side. In fact, the detectable eye movement during spontaneous nystagmus is the quick phase toward the unaffected ear^{72,74}. In the acute phase, patients will have difficulty reading and watching television. After the acute episode, a patient can suppress the nystagmus with visual fixation making it difficult for the examiner to observe eye movements⁷³. Preventing visual fixation by using Frenzel glasses facilitates observation of a spontaneous nystagmus: These glasses prevent light from activating the smooth pursuit system, which can cancel out the imbalance of the tonic firing rate produced by a peripheral vestibular lesion⁷⁴. A purely vertical (upbeat or downbeat) or torsional spontaneous nystagmus is indicative of a central vestibular lesion^{9,72}. Nystagmus due to a central lesion usually cannot be suppressed with visual fixation^{72,75}. A positional downbeat vertical nystagmus occurs particularly in posterior fossa lesions with Arnold-Chiari malformation as its most common cause^{76,77}. Nystagmus with one eye beating down and the other upwards (skew nystagmus) has only been reported in patients with Arnold-Chiari malformation⁷⁷. A few

minutes of observation is required to identify periodic alternating nystagmus, a horizontal jerk nystagmus that changes direction about every two minutes and that is indicative of midline cerebellar lesions⁷². Spontaneous nystagmus may also be congenital. This variant is generally horizontal; may alternate directions but not at regular intervals; increases with attention, fixation, and anxiety; and decreases with convergence⁷². Pendular nystagmus occurs most commonly in patients with multiple sclerosis and brain stem stroke⁷². The presence of a pendular, a vertical or torsional jerk, skew, or a periodic alternating horizontal jerk spontaneous nystagmus indicates the need for referral. In fact, any spontaneous nystagmus requires referral with the exception of the congenital variant noted above.

The clinician then observes for spontaneous nystagmus in eccentric positions⁷². Deviation of the eye in the direction of the quick phase will increase the frequency and velocity of the nystagmus (Alexander's law) in patients with a unilateral peripheral vestibular lesion, and it may still produce a positional nystagmus in accommodated patients⁹. Detection of gaze-evoked nystagmus on lateral or upward gaze suggests a central lesion⁶⁴. In fact, a gaze-evoked horizontal nystagmus implies lesions in the cerebellar flocculus and the medial vestibular nucleus-nucleus prepositus hypoglossus complex, but it can also be the effect of medications, such as hypnotics, sedatives, and anxiolytics or alcohol intoxication^{9,72}. Gaze-evoked nystagmus may also be the result of extra-ocular muscle weakness as in myasthenia gravis⁷². Unsustained eye movements of low frequency and amplitude are indicative of end-point nystagmus, a non-pathological variant in normal subjects⁷². The presence of gaze-evoked nystagmus (with the exception of end-point nystagmus) indicates the need for referral.

Saccadic Eye Movements

Having the patient look back and forth between two targets tests saccadic eye movements. Overshooting of the target (saccade overshoot dysmetria) may be observed in cerebellar disorders, such as Friedreich's ataxia⁷⁸. Undershooting of the target or hypometria can occur in patients with Parkinson's disease⁷⁸. Vertical saccadic eye movements in patients with Wallenberg syndrome as a result of vertebrobasilar infarction may result in eye lateropulsion requiring a corrective horizontal saccade⁹. Uncalled-for saccades during gaze fixation on one of the targets can occur in patients with viral cerebellar infection, paraneoplastic syndrome, and Friedreich's ataxia⁹. Macrosaccadic oscillations, which are horizontal saccades occurring in waxing and waning bursts with 200 ms saccadic intervals induced by a gaze shift, are indicative of midline cerebellar disease, spinocerebellar degenerations, and pontine lesions⁷². Abnormalities identified during saccadic eye movement tests indicate the need for referral.

Smooth Pursuit Testing

Having the patient follow a slowly moving target, no faster than 20° per second, tests smooth pursuit. A marked deficit in smooth pursuit is indicative of a degenerative cerebellar process⁹. Small bilateral saccades in the same direction in both eyes during smooth pursuit testing are indicative of spinocerebellar lesions, especially Friedreich's ataxia⁷². Smooth pursuit testing may also be positive in patients with a severe acute peripheral vestibular lesion due to superposition of an intense spontaneous nystagmus⁹. Abnormal findings on smooth pursuit testing indicate the need for referral.

Hearing Examination

The CN examination may indicate hearing loss. A conductive hearing loss results from disorders in the external or middle ear; lesions in the cochlea or the cochlear nerve¹⁰ cause a sensorineural hearing loss. A sensorineural loss is a symptom of salicylate overdose¹⁰. Meniere's disease produces a sensorineural loss that is progressive over multiple episodes^{10,19}. Progressive unilateral sensorineural hearing loss is also a typical presentation of patients with acoustic neuromas²⁰. Otosclerosis can produce both a conductive and a sensorineural hearing loss¹⁰. It is the authors' experience that many elderly patients complaining of dizziness present with an undiagnosed but unrelated conductive hearing loss. Even without associated symptoms, this constitutes a reason for referral to an audiologist. The presence of symptoms implicating hearing loss as part of a pathology causing complaints of dizziness indicates the definite need for medical referral.

Weber Test

With the Weber test¹⁰, the therapist places a tuning fork (256 or 512 Hz) on the top of the patient's skull. With unilateral sensorineural hearing loss, the patient will perceive the sound as coming from the normal ear. With a conductive disorder, the patient perceives the sound as coming from the abnormal ear. Midline is the normal response for this test⁶⁷. A non-midline response indicates the need for referral.

Rinne Test

The Rinne test¹⁰ allows the therapist to distinguish between a sensorineural and a conductive deficit in the affected ear. Normally, air conduction of the sound of a vibrating tuning fork (256 or 512 Hz) is perceived as louder than bone conduction. So holding the tuning fork next to the external auditory canal produces a louder sound than placing the base of the tuning fork on the mastoid bone in patients with normal hearing. The same goes in patients with sensorineural hearing loss. However, in patients with conductive deficits, bone conduction will appear louder on the affected side than air conduction.

Burkey et al⁷⁹ reported that the sensitivity of the Rinne test was sufficient to be used as part of a screening protocol in the hands of an experienced examiner and when interpreting equivocal results as indicative of a conductive loss. The finding of bone greater than air conduction indicates the need for referral.

Active Range of Motion Tests

Musculoskeletal impairments (i.e., decreased muscle strength and endurance, joint stability and mobility, and posture) are implicated in patients with the dysequilibrium subtype of dizziness and may be amenable to sole PT management. Range of motion limitations, specifically trunk, hip, and knee flexion and ankle plantar flexion contractures, will adversely influence the location of the center of gravity in relation to the base of support. Active range of motion (AROM) testing should, therefore, concentrate on assessing trunk, hip, and knee extension and ankle dorsiflexion. Assessing neck motions allows the clinician to observe possible adverse responses in the sense of ischaemic reactions during patient-controlled AROM. It also serves to see if patients will be able to assume the test positions needed in further tests. Cervical AROM tests may also reveal upper cervical hypomobility implicated in cervicogenic dizziness^{80,81}. AROM tests also provide indications on strength and coordination deficits in the form of ataxia or abnormal involuntary motions. Asterixis is an episodic cessation of muscular activity in patients with hepatic encephalopathy, hepatocerebral degeneration, and other metabolic encephalopathies¹⁰. Episodic cessation of extensor muscle activity occurs when the patient holds the arms outstretched with wrists and fingers extended causing the hands to fall into flexion followed by a return to the extended position¹⁰. Myoclonus is a rapid twitch-like muscle contraction: It can result from the same conditions causing asterixis or with Creutzfeldt-Jakob disease¹⁰. Chorea can occur in patients with Wilson's disease, acquired hepatocerebral degeneration, and ataxia-telangiectasia¹⁰. Chorea is characterized by rapid, irregular muscle jerks, occurring unpredictably and involuntarily in different body parts¹⁰. An ischaemic response during cervical AROM testing or the presence of abnormal involuntary motions during AROM testing of the limbs indicates the need for referral.

Limb Ataxia Tests

These tests serve to confirm possible limb ataxia observed during AROM testing. During the finger-to-nose test, the quality of arm motion is observed as the patient moves the index finger to the tip of the nose or the chin. Closing the eyes eliminates visual substitution. Mild cerebellar ataxia results in an intention tremor near the beginning and end of the movement with possible overshooting of the target¹⁰. With the finger-to-finger

test, the patient attempts to touch his or her finger to the therapist's finger. Horizontal overshooting implicates a unilateral labyrinthine lesion; vertical overshooting occurs in patients with midline lesions to the medulla oblongata or the bilateral cerebellar flocculus³⁴. Having the supine patient track the heel of the foot smoothly up and down the contralateral shin tests for leg ataxia. Having the seated patient touch the great toe to the examiner's finger is another test for leg ataxia⁸². Dysdiadochokinesia is the inability to perform rapidly alternating movements, and in adults it is usually caused by multiple sclerosis; in children, it frequently results from cerebellar tumors. Patients with other movement disorders such as Parkinson's disease may have also have difficulty with rapid alternating movements but this is due to akinesia or rigidity rather true dysdiadochokinesia⁸³. Dysdiadochokinesia can be tested with rapid alternating finger tapping, forearm pronation-supination, and toe tapping movements, for example^{82,83}. With the Barre test, the standing or sitting patient holds the hands outstretched with the forearms supinated and eyes closed. Sinking of one arm with simultaneous pronation may indicate a central neurological, likely cerebellar, dysfunction⁸⁴. The finger-to-nose test has poor test-retest and interrater reliability for dysmetria and tremor, but excellent reliability for time of execution⁸⁵. Simon, Aminoff, and Greenberg¹⁰ reported a positive heel-to-shin test in 80% of patients with alcoholic cerebellar degeneration. We found no further data on reliability and validity for these ataxia tests. Positive limb ataxia tests (including seeming dysdiadochokinesia due to akinesia or rigidity) indicate the need for referral.

Passive Range of Motion Tests

Passive range of motion (PROM) testing includes passive physiological (PPM) and accessory motion (PAM) and instability tests. In the spine, they include passive physiological (PPIVM) and accessory intervertebral motion (PAIVM) and segmental stability tests. Upper cervical segmental motion abnormalities may be the cause for cervicogenic dizziness. In the case of a hypomobility found on AROM testing, PROM tests may determine cause and subsequent intervention. Instability tests of the upper cervical spine are especially relevant prior to tests involving regional passive rotation of the neck or PPIVM/PAIVM testing: Inadvertent shear forces produced during these tests due to ligamentous insufficiency may damage the cord and vertebral arteries²⁹. The therapist may want to postpone PPIVM/PAIVM tests to the cervical spine until both the segmental stability tests and the VBI tests (see below) have provided a negative response. Intrarater reliability of PPIVM/PAIVM tests has consistently been shown to be greater than interrater reliability with the latter varying from generally poor to (at times) perfect⁸⁶. Jull, Bogduk, and Marsland⁸⁷

examined construct validity and found 100% sensitivity and specificity when comparing cervical PPIVM/PAIVM test with single facet blocks. Cattryse et al⁸⁸ found acceptable interrater reliability only for the supine upper cervical flexion instability test but not the Sharp-Purser or atlas lateral displacement test. A positive finding on upper cervical segmental stability tests in combination with signs and symptoms of cord or vertebral artery compromise indicates the need for referral.

PROM tests can also detect muscle tone abnormalities. Hypotonia is indicative of cerebellar disorders with unilateral cerebellar disorders producing ipsilateral limb hypotonia¹⁰. Hypertonia or rigidity may occur in patients with cerebellar ataxia due to Wilson's disease, acquired hepatocerebral degeneration, Creutzfeldt-Jakob disease, and some olivopontocerebellar degenerations. Spasticity on PROM testing is common in patients with multiple sclerosis, posterior fossa tumors, Arnold-Chiari malformation, VBI or infarction, Friedreich's ataxia and the other hereditary ataxias, olivopontocerebellar degeneration, Creutzfeldt-Jakob disease, neurosyphilis, and vitamin B₁₂ deficiency¹⁰. Prochazka et al⁸⁹ showed poor reliability for a 5-point rating scale for rating rigidity in patients with Parkinson's disease. Tone abnormalities on PROM test indicate the need for referral in a previously undiagnosed patient.

Strength Tests

The musculoskeletal system is the effector organ of the balance control system. Sufficient strength and endurance in the muscles involved in static and dynamic balance is an obvious prerequisite for optimal balance. Loss of strength and endurance in these muscles can be the cause of patient complaints of dizziness and dysequilibrium. The pattern of any weakness present may also provide diagnostic indicators for the underlying dysfunction or disease. Single or multiple muscle weakness can be the result of disuse atrophy, especially in the elderly. Weakness in a peripheral nerve distribution implies a peripheral neuropathy. Monosegmental myotomal weakness can implicate a nerve root problem. Multisegmental weakness can implicate a process affecting cauda equina or spinal cord. Distal neuropathic weakness can be the result of disorders producing sensory ataxia, e.g., polyneuropathies. Multiple sclerosis, foramen magnum lesions, spinal cord tumors, and vitamin B₁₂ deficiency can cause paraparesis. Ataxic quadriparesis, hemiataxia and contralateral hemiparesis, or ataxic hemiparesis are all diagnostic indicators of a brainstem lesion¹⁰. Knepler and Bohannon⁹⁰ and Bohannon and Corrigan⁹¹ showed large interrater variability in the forces used to establish manual muscle test (MMT) grades of 3+, 4-, 4+, and 5. Herbison et al⁹² recommended the use of a hand-held myometer over MMT to detect strength changes. Multi-segmental weakness (including paraparesis, quadriparesis,

and hemiparesis) but also progressive monosegmental paresis indicates the need for referral.

Reflex Tests

Cerebellar disorders cause hypoactive deep tendon reflexes with unilateral cerebellar disorders resulting in ipsilateral hyporeflexia. Friedreich's ataxia, neurosyphilis, and polyneuropathies cause leg hyporeflexia. Hyperreflexia is present in multiple sclerosis, vitamin B₁₂ deficiency, focal brainstem lesions, and some spinocerebellar and olivopontocerebellar degenerations¹⁰. A positive Babinski sign, Hoffman's reflex, and ankle clonus may occur in patients with myelopathy, multiple sclerosis, vitamin B₁₂ deficiency, focal brainstem lesions, and some spinocerebellar and olivopontocerebellar degenerations^{1,9}. Sung and Wang⁹³ established 100% sensitivity for a positive Hoffman's reflex for detecting patients with cervical cord compression confirmed on X-ray or MRI. We found no additional data on reliability and validity of reflex tests. Clearly hypoactive or hyperactive deep tendon reflexes may indicate a need for referral; the presence of pathological reflexes is a definite reason for referral.

Sensation Tests

Sensation testing may include tests for light touch perception, sharp/dull discrimination, vibration sense, and proprioception⁹⁴. Sensation testing may reveal deficits in the distribution pattern of single or multiple peripheral nerves, a nerve root, or a multi-segmental pattern providing diagnostic clues for underlying cause or contributing factors to the patient's complaint of dizziness. Joint position sense can be tested by asking the patient to detect the presence and the direction of a passive movement in the joints^{8,10}. Simon, Aminoff, and Greenberg¹⁰ suggested beginning this type of testing distally and moving proximally to establish the upper level of deficit in each joint. Placing a joint in a position and having the patient reproduce this position with the contralateral joint can also be used as a test for abnormality of joint position sense¹⁰; the patient's eyes are closed during joint position testing to prevent visual compensation. Joint position sense in the legs is always impaired in patients with sensory ataxia; the arms may be affected depending on the type and extent of pathology responsible. Placing a 128 Hz tuning fork over a bony prominence may serve as a test of vibration sense. Successively more proximal sites can determine the upper level of deficit in limbs or even trunk. Sensory ataxia is often combined with a decrease in vibratory sensation¹⁰. Peters et al⁹⁵ showed limited interrater reliability for a quantitative method of assessing vibration sense implying even less reliability for the tuning fork method. It is the authors' experience that elderly patients frequently present with undiagnosed decreased

proprioceptive acuity and vibration sense in the feet and ankles, which may contribute to dysequilibrium-type dizziness. Multisegmental deficits may indicate the need for referral.

Vertebrobasilar Insufficiency Tests

De Kleyn-Nieuwenhuysse Test

Terrett¹³ noted that the original test description had postulated decreased or even absent vertebral artery blood flow based on cadaver perfusion studies in different head and neck positions. A long latency, progressive symptoms when held in the sustained test position of cervical extension and rotation, and a lack of habituation with repeated testing are indicative of VBI and not of cervicogenic dizziness or BPPV⁴⁵. Oostendorp⁴⁶ reported a latency of 55 ± 18 seconds in these patients with positive findings on variations of the De Kleyn-Nieuwenhuysse test (Figure 4). He also reported a recovery time of 120 ± 40 seconds⁴⁶. A positive test may include symptoms of vertigo, nausea, diplopia, and dysphagia. Positive signs may include nystagmus and dysarthria^{1,13} (which may be noted by having the patient talk during the test hold). Pettman²⁹ noted horizontal nystagmus but the authors have noted vertical and rotary nystagmus in symptomatic subjects.

This test has been extensively studied with equivocal results. Some authors reported significant decreases in blood flow^{96,97}, whereas others reported no changes^{98,99}. Support for this test becomes even more problematic with case reports noting false negative results^{100,101} and case series noting 75-100% false positive results^{99,102}. Cote et al¹⁰³ reported 0% sensitivity for detection of increased impedance to blood flow, 0% positive predictive value, and 63-97% negative predictive value. This test (and the cervical rotation test) is obviously a questionable screening procedure for VBI. Vidal¹⁰⁴ has recently questioned its routine use, concluding that vertebral artery tests are not clinically useful screening tools for VBI. Rather, he suggested relying upon history suggestive of VBI, medical history (especially when indicative of ischaemic processes, such as coronary artery disease, transient ischaemic attacks, or cerebrovascular accidents) and other relevant examination findings (e.g., during cranial nerve and AROM tests). Due to the potential for harm with this test and its poor psychometric properties, it should not be done in patients with a positive medical history or a history strongly indicative of VBI¹⁰⁴. However, the test may serve as a screening tool in patients not fitting these categories. In those patients, a positive finding with clear central neurological signs of nystagmus and dysarthria on this test warrants referral.

Sustained Cervical Rotation Test

Sustained supine cervical rotation may also test for VBI. Symptom behavior can be expected to be similar

to the extension-rotation test with regards to latency, non-accommodation, and non-habituation. However, findings on sustained cervical rotation alone are equally equivocal with significant decreases in vertebral artery flow^{96-98,105,106} or no effect on blood flow¹⁰⁷ or blood volume¹⁰⁸. Indications for this test and implications of a clearly central neurological involvement are as described for the extension-rotation test.

Hautant Test

This test is used for differential diagnosis of vestibular, cervicogenic, and ischaemic dysfunction (Figure 5). However, it is also a test with multiple descriptions in manual medicine literature¹⁰³. Terrett¹³ described the test with the patient seated, the arms outstretched, and the forearms supinated. The therapist moves the patient's head in an extension-rotation position with the patient's eyes closed. Symptom reproduction and sinking of one hand into pronation implicates the vertebrobasilar system¹³. Van der El⁸⁴ described this test with the forearms pronated. Deviation of one of the arms with the head in mid-position indicates vestibular dysfunction. A lateral deviation of the contralateral arm in the opposite direction of the cervical extension-rotation implicates the neck. Immediate arm motion suggests a somatosensory dysfunction; a latency period indicates ischaemic dysfunction⁸⁴. No data on reliability or validity were found. A test indicating ischaemic dysfunction suggests the need for referral.

Vestibulo-Ocular Tests

These tests examine the vestibulo-ocular reflex (VOR) circuit by inducing movements at an angular velocity

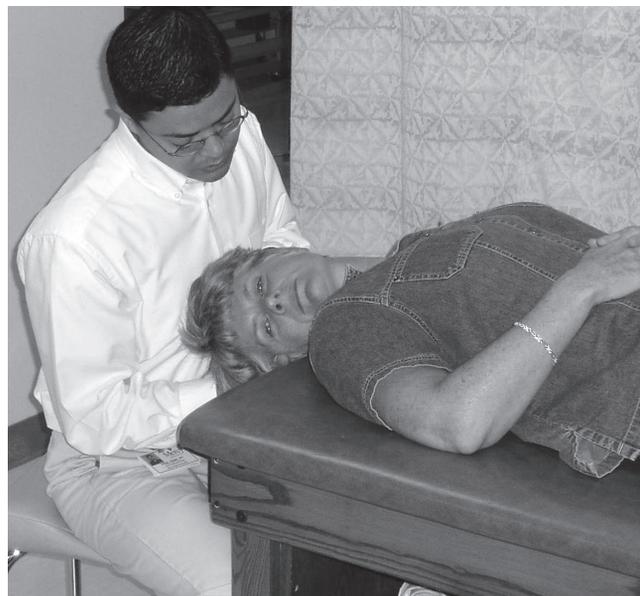


Fig. 4: De Kleyn-Nieuwenhuysse test

that does not allow for compensation by the cervico-ocular reflex (COR).

Dynamic Visual Acuity

After establishing baseline visual acuity with a Snellen chart, this test measures visual acuity with concurrent head movement. The head is moved from side to side at a frequency of 1 Hz while the patient reads the Snellen chart⁴¹. A decrease by two lines is suspicious and by three or more is indicative of an abnormal VOR^{41,94}. This test is not suited for detecting unilateral peripheral or central vestibular lesions but is indicated in case of suspected bilateral vestibular loss^{41,109}. Herdmann, Blatt, and Schubert⁴ reported poor reliability for this test.

Doll's Head Test

The examiner faces the patient, who fixes gaze on the examiner's nose. The examiner then oscillates the patient's head 30° side to side at 0.5-1 Hz. Eye movements that are not smooth but interrupted by catch-up saccades towards the fixation target indicate bilateral vestibular lesions⁴¹. We found no data on reliability and validity.

Head-Shaking Nystagmus Test

The examiner vigorously moves the patient's head back and forth horizontally for about 30 seconds with the patient's eyes closed. Upon opening the eyes, the nystagmus will beat away from the side of a unilateral peripheral vestibular lesion¹⁰⁹ or towards the lesioned side in patients with Meniere's disease¹¹⁰. When compared to a caloric test, the head-shaking nystagmus test (with Frenzel glasses) had 66% sensitivity and 77% specificity for detecting canal paresis >20%¹¹¹. Kamei and Iizuka¹¹⁰

reported on the possible prognostic value of a reversal of nystagmus direction towards the affected ear to predict an imminent recurrence of Meniere's disease.

Head Thrust Test

The head thrust test (Figures 6A & B) may also detect an impaired VOR¹¹². The patient fixates gaze on the therapist's nose. The therapist then moves the patient's head in the horizontal plane in a rapid, passive manner with unpredictable timing and direction (5-10° at 3000-4000° s⁻¹). A patient with vestibular loss will have difficulty maintaining gaze fixation, requiring a corrective saccade (fast eye movement) to maintain gaze fixation on the nose⁷⁵. A corrective saccade following head thrust right indicates the vestibular loss is on the right; corrective saccades with head thrust left suggest an involved left side¹¹³. Schubert et al¹¹⁴ reported a sensitivity of 71% and a specificity of 82% for the head thrust test with the head tilted down 30° in the diagnosis of patients with unilateral vestibular loss and 84% sensitivity and 82% specificity for bilateral loss.

BPPV Tests

These tests look for canalithiasis or cupulolithiasis in all SCC. A positive response on these tests in combination with corroborating history findings and in the absence of findings indicative of other pathology implies that sole PT management may be indicated.

Hallpike-Dix Maneuver

This maneuver (Figures 7A & B) tests all SSC⁴¹. The long sitting patient turns the head 45° and is then assisted to supine with the rotated head 30° below

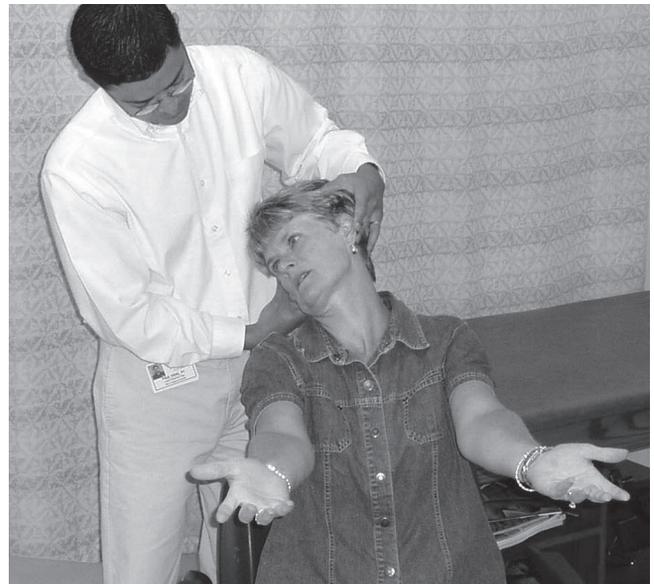


Fig. 5: Hautant test

horizontal. This position is maintained for ≥ 30 seconds. Delayed-onset, torsional, horizontal, or (less commonly) vertical nystagmus in combination with vertigo lasting < 60 (canalithiasis) or > 60 seconds (cupulolithiasis) that decreases with repeated testing constitutes a positive finding^{41,76,115}. Relevant for differential diagnosis with regards to which SCC is involved is the type of nystagmus and whether the test is unilaterally or bilaterally positive:

- An ipsilateral maneuver positions the posterior SCC of the downside ear in the plane of the pull of gravity. Shifting of otoconia--whether free-floating (canalithiasis) or adhered to the cupula (cupulolithiasis)--deflects the cupula and alters the posterior SCC neuronal firing rate¹¹⁵. This results in an apogeotropic (beating away from the earth or downside ear) torsional nystagmus¹¹⁵.

- A bilateral positive test implicates either the anterior or horizontal SCC^{41,76}. With anterior SCC BPPV, the ipsilateral test provokes a geotropic (beating towards the earth or the affected ear) torsional¹¹⁵ or a downbeating vertical nystagmus⁷⁶.
- A bilaterally positive test with a purely horizontal geotropic (beating in the direction of the face turn or downside ear) nystagmus implicates the horizon-



Fig. 6: Head thrust test

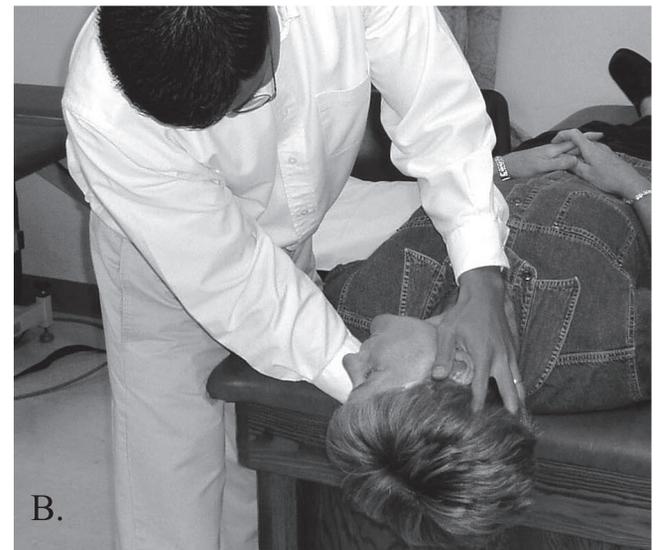
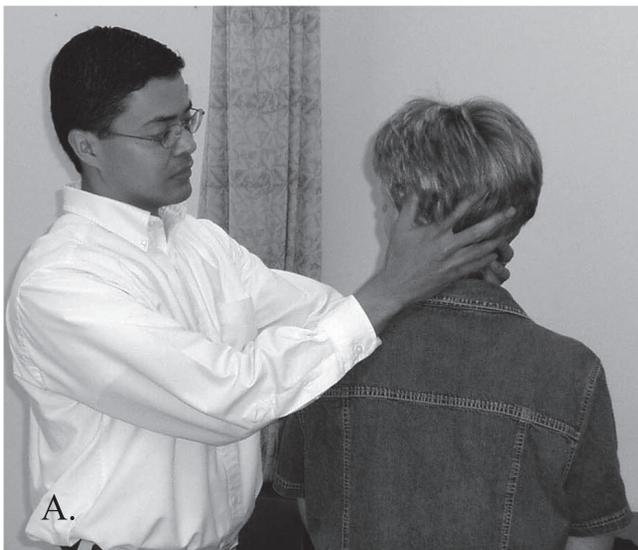


Fig. 7a-b: Hallpike-Dix maneuver

tal SCC⁴¹. Nystagmus will occur in both directions but will generally be stronger with the head turned towards the affected side⁴¹.

Positional nystagmus on this test has been shown to identify patients with posterior SCC BPPV with 78% sensitivity¹¹⁶. Sensitivity as high as 88% has been reported⁶⁹.

Straight Head-Hanging Test

In the straight head-hanging test, the patient is assisted in lying back from long sitting with the head extended but not rotated. This test may be more sensitive for anterior SCC BPPV: An additional 20° of extension as compared to the Hallpike-Dix maneuver causes the ampullary segment of the anterior SCC to approach a more vertical position⁷⁶. We found no data on reliability and validity.

Roll Test

The roll test detects horizontal SCC BPPV¹¹⁵. The therapist quickly “logrolls” the supine patient with the head 30° flexed to one side maintaining this position for ≥1 minute. Otoconia moving back and forth within the SCC with left and right rotation will cause the positive response of nystagmus and vertigo¹¹⁵. Canalithiasis causes fatiguing geotropic (towards the earth) nystagmus and cupulolithiasis persistent apogeotropic (away from the earth) nystagmus¹¹⁵. More severe and longer lasting symptoms indicate the affected side¹¹⁵. We found no data on reliability and validity.

Walk-Rotate-Walk Test

In this test for the horizontal SCC¹¹⁷, the patient walks straight ahead at the patient-selected maximum tolerable speed in a room with ample space. The patient then rotates 180° on the axis of the rotation direction foot, returning back in a continuous movement. The test is performed to both sides. Staggering, sidestepping; making corrective movements of the body or hands, discontinuing the rotation in one direction, or slowed difficult rotation indicate a positive test. A positive response on rotation right implicates the right and a positive response on rotation left implicates the left SCC. The difference must persist over 3 repetitions. Rahko and Kotti¹¹⁷ found 100% predictive validity for this test in determining a positive response to a horizontal SCC BPPV treatment. The 180° turn in this versus the 90° turn in the roll test may allow for higher otoconia acceleration and ampulla cell stimulation. Sensitivity was acceptable; some patients with acute vestibular neuritis tested positive on the walk-rotate-walk test¹¹⁷.

Cervicogenic Dizziness Testing

The neck torsion test (Figures 8A & B) is used to detect cervicogenic dizziness^{118,119}. The head is held stationary during neck and trunk rotation. An alternate way of screening the cervical spine as the possible origin of dizziness symptoms is to have the patient sit and flex forward at the hips simultaneously extending and rotating their neck (Figure 9)¹²⁰. As both tests keep the inner

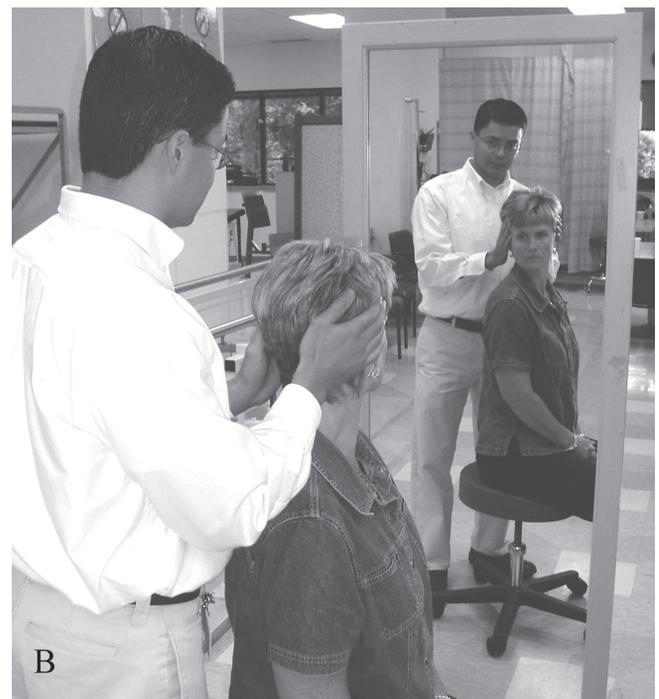
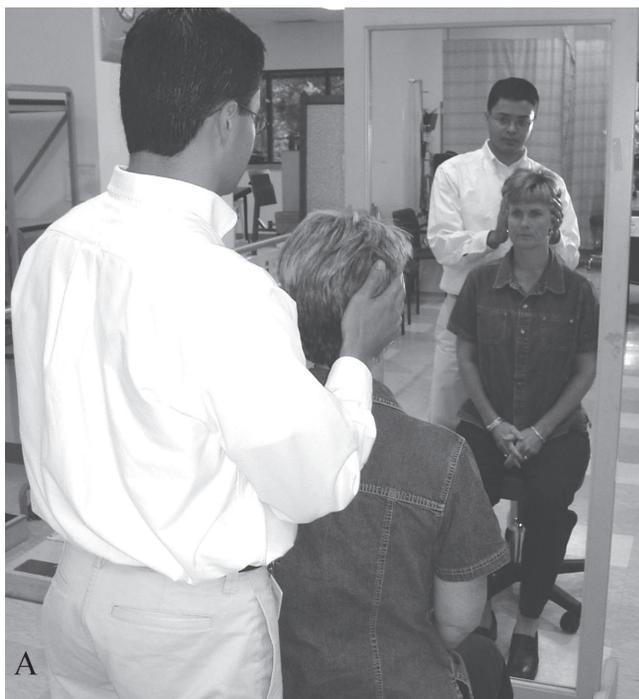


Fig. 8a-b: Neck torsion test

ear stationary, the vestibular system is not stimulated. Nystagmus and dizziness with this test are, therefore, interpreted as cervicogenic. However, the therapist still needs to differentiate between vascular or somatosensory cervicogenic involvements¹²⁰. Diagnostic accuracy is questionable: 50% of subjects without cervical pathology tested positive for nystagmus¹²¹, possibly a manifestation of the COR²⁶. Fitz-Ritson¹²² found that 47% of patients with cervical trauma demonstrated subjective symptoms of vertigo or postural instability during the neck torsion test; 90% improved following therapy. We found no additional data on reliability and validity. A positive response on these tests in combination with corroborating history findings in the absence of findings indicative of other pathology implies that sole PT management may be indicated.

Breathing-Related Tests

Hyperventilation Test

The hyperventilation test requires patients to voluntarily hyperventilate, i.e., 30 breaths min^{-1} for 3 minutes⁴⁷. It may be a useful and simple test for validating a diagnosis of panic disorder or dizziness related to hyperventilation presyncope. In these patients, this test will produce dizziness but no nystagmus⁹. Patients with demyelinating lesions of the vestibulocochlear nerve due to an acoustic neuroma, compression by a small blood vessel, or central demyelinating lesions (multiple sclerosis) may show nystagmus on the hyperventilation test⁹. Hyperventilation may accentuate downbeating nystagmus in patients with Arnold-Chiari malformation and evoke a nystagmus towards the lesion in patients with vestibular schwannomas¹⁰⁹. Nardi et al^{123,124} found that the hyperventilation test



Fig. 9: Alternate neck torsion test

produced significantly more symptoms in patients with panic disorder than in patients with obsessive-compulsive disorder, depression, or in normals, and they noted that it might be an easy test to validate panic disorder¹²⁴. Nakao et al¹²⁵ reported 62% sensitivity and 100% specificity for this test in the diagnosis of coronary spasm. The authors have noted clinically that near-immediate reproduction of symptoms may indicate psychogenic contribution to dizziness complaints. A positive finding on these tests implies the need for referral.

Valsalva Test

In patients with Arnold-Chiari malformation, perilymphatic fistulae, and other abnormalities of the ossicles (e.g., otosclerosis), oval window, and saccule, a Valsalva maneuver may produce nystagmus. Changes of middle ear pressure due to loud noises, application of positive and negative pressure to the tympanic membrane (Hennebert's sign), and opening and closing the Eustachian tube may have a similar effect⁹. The cough test is a variant on the Valsalva test. Having the patient cough to increase intrathoracic pressure may be useful in detecting dizziness due to cough presyncope¹⁰. We found no data on reliability and validity. Positive tests indicate the need for referral.

History and Physical Examination

About 50% of dizziness is vestibular and benign⁶⁹. More serious causes, e.g., brain tumors and cerebrovascular disorders, account for about 1% and 5% of cases respectively⁶⁹. Froehling et al¹²⁶ studied diagnostic accuracy of symptoms and signs in distinguishing benign from serious causes:

- Vertigo or vomiting combined with a positive Hallpike-Dix test demonstrated 85% positive predictive value and a 7.6 positive likelihood ratio (LR) for a benign cause.
- A negative Hallpike-Dix maneuver and absence of vertigo or vomiting had a 68% negative predictive value for peripheral vertigo.
- Age ≤ 69 , absence of neurological deficits, and/or the presence of vertigo have a negative predictive value of 88% with a negative LR of 0.3 for a serious cause of dizziness.
- Age > 69 , presence of neurological deficit, and/or absence of vertigo carry a positive predictive value of 40% and positive LR of 1.5 for a serious cause of dizziness.

Discussion

There is emerging evidence that PT management may suffice for patients with BPPV, cervicogenic dizziness, and musculoskeletal impairments leading to dysequilibrium¹⁻⁷. Table 5 provides signs and symptoms indicative

Table 5. Signs and symptoms indicative of pathologies amenable to sole PT management.

<p>Benign paroxysmal positional vertigo (BPPV)</p>	<ul style="list-style-type: none"> • Intermittent, severe positioning-type dizziness • Precipitated by positioning, movement, or other stimuli (see below) • Short latency: 1-5 seconds • Brief duration: < 30 seconds • Fatigable with repeated motion • Associated signs and symptoms: nystagmus, nausea, and at times vomiting • Occurs in people over age 40 with peak incidence of onset in the sixth decade • Rare in people under 20 • Medical history of head trauma, labyrinthine infection, surgical stapedectomy, chronic suppurative otitis media, and degenerative changes to the inner ear may indicate non-idiopathic BPPV.
<p>Posterior semicircular canal (SCC) BPPV</p>	<ul style="list-style-type: none"> • Patients complain of dizziness when they quickly transfer to a supine position, especially when the head is turned to the affected side. • Positive response of vertigo and apogeotropic torsional nystagmus on ipsilateral Hallpike-Dix maneuver
<p>Anterior SCC BPPV</p>	<ul style="list-style-type: none"> • Patients also complain of dizziness when they quickly transfer to a supine position, especially when the head is turned to the affected side, but there is less specificity as to the direction of head rotation. • Bilateral positive response on Hallpike-Dix maneuver with vertigo and geotropic torsional nystagmus on ipsilateral test • Hallpike-Dix maneuver may also cause downbeating vertical nystagmus. • Positive response on straight head-hanging test
<p>Horizontal SCC BPPV</p>	<ul style="list-style-type: none"> • Dizziness is brought on when rolling over in supine but can also occur with flexion and extension of the head or when transferring from supine to upright. • A bilaterally positive test with a purely horizontal nystagmus on Hallpike-Dix maneuver. The nystagmus will be geotropic beating in the direction of the face turn or downside ear. Nystagmus will occur in both directions but is generally stronger when the head is turned towards the affected side. • Positive roll test • Positive walk-rotate-walk test to affected side
<p>Cervicogenic dizziness</p>	<ul style="list-style-type: none"> • Intermittent positioning-type dizziness • Precipitated by head and neck movement • No latency period: onset of symptoms is immediate upon assuming the provoking position • Brief duration but may last minutes to hours • Fatigable with repeated motion • Associated signs and symptoms: nystagmus, neck pain, suboccipital headaches, sometimes paraesthesiae in the trigeminal nerve distribution • Possible lateral head tilt due to tightness of the sternocleidomastoid or upper trapezius • Possible forward head posture • Medical history of cervical spine trauma and degeneration • Motion dysfunction in the upper cervical segments on AROM and PIVM testing • Positive neck torsion test: nystagmus with reproduction of dizziness
<p>Musculoskeletal impairments</p>	<ul style="list-style-type: none"> • Subjective complaints of weakness, unsteadiness • Insidious onset • Postural deviations negatively affecting the location of the center of gravity in relation to the base of support: trunk flexion, hip flexion, knee flexion, and ankle plantar flexion contractures • Decreased trunk extension, hip extension, knee extension, and ankle dorsiflexion on ROM testing • Loss of strength and endurance in anti-gravity muscles • Impaired joint position sense lower extremity

of these pathologies amenable to sole PT management. The greatest danger for patients complaining of dizziness (and for the therapist managing such patients) is that the therapist may fail to recognize signs and symptoms that are indicative of a pathology requiring urgent medical-surgical management but that resemble pathology amenable to sole PT management². A delayed medical diagnosis and delayed subsequent appropriate medical-surgical management may prove harmful in these cases².

In this article, we have provided a template for the history (Tables 1-2) and physical examination (Table 4) relevant to previously undiagnosed patients presenting to the orthopaedic physical therapist with a main complaint of dizziness. In these tables and in the text, we have provided indications for when to refer the patient for medical-surgical evaluation. The data provided on test reliability and validity, where available, should serve as a guideline by which to establish the confidence we have in our findings. However, this research data on the history items and physical tests described in this article is often absent, contradictory, or insufficient for confident diagnostic decision-making.

Our recommendations for referral throughout the text are based to the maximum extent possible on psychometric properties of the tests and measures, but they are also guided by an analysis of possible harm to the patient should we decide not to refer. At times, it is better to refer the patient and have the patient found normal than to not refer and do potential harm. Considering the pathologies possibly responsible for complaints of dizziness¹, the potential for harm is real and present when working with this population. Clearly documenting the reason for a medical-surgical referral based on the information presented in this article will clarify the need for referral and allow for better communication with our medical colleagues. Any uncertainty regarding the proper diagnosis should result in referral. But even if the signs and symptoms appear to

fit with a diagnosis amenable to sole PT management, the patient's failure to respond to seemingly appropriate conservative measures also indicates the need for a medical second opinion⁴.

Conclusion

Dizziness is a frequent complaint in primary care orthopaedic physical therapy practice. A PT differential diagnosis of previously undiagnosed patients centers on distinguishing patients with BPPV, cervicogenic dizziness, and musculoskeletal impairments leading to dysequilibrium from those patients who require referral for medical-surgical differential diagnosis and (co)management. This article provides information on history items and physical tests within the PT scope of practice that can enable the orthopaedic physical therapist to distinguish between these two categories of patients. The decision to refer the patient for a medical-surgical evaluation is based on our findings, the interpretation of such findings in light of data on reliability and validity of history items and physical tests, an analysis of the risk of harm to the patient, and the response to seemingly appropriate intervention. The literature search for data on reliability and validity of history items and physical tests revealed a general paucity of data especially with regards to the history and indicates a clear avenue for future research. We also hope that the classification system discussed in our earlier article¹ and the template for history and examination introduced in this article may serve as a template for future diagnostic and outcomes research in this patient population.

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