



Dizziness and Syncope

Jacques Bédard

MD, CSPO, FRCPC, Internal Medicine

ASSESSMENT
AND TREATMENT
METHODS

Dizziness and Syncope

ASSESSMENT AND TREATMENT METHODS

“Dizziness” as a symptom is the third most frequent cause of front-line medical visits (at doctors’ offices, emergency rooms, and outpatient clinics). Unfortunately, most texts on the subject tend to give instruction on the diseases that cause dizziness as opposed to the symptom itself. Clinicians are often baffled by the imprecision of the symptom, usually described by patients simply as “I feel dizzy.” Doctors respond with unnecessary, costly paraclinical examinations and specialist consultations – a poor use of our already limited public health funds and, frequently, a source of great distress for patients.

Because they are out of their comfort zone, physicians often react emotionally and negatively in dealing with this very frequent symptom.

The goal of this text is to provide a “navigation strategy” for the symptom, essentially a three-step assessment, that once mastered (**ESPECIALLY THE FIRST STEP**) provides a surprising degree of **EXPERTISE** and **EFFECTIVENESS**.

Step 1

ASSESSING DIZZINESS REQUIRES THE IDENTIFICATION OF A SPECIFIC SYMPTOM OUT OF THE INITIAL NON-SPECIFIC “DIZZINESS” DESCRIPTION THROUGH AN UNDIRECTED PATIENT QUESTIONNAIRE. ONCE THE SYMPTOM HAS BEEN DETAILED, ONE OF FOUR TYPES OF DIZZINESS CAN BE IDENTIFIED. THIS FIRST STEP IS THE CORNERSTONE OF THE PROCESS AND, IF DONE PROPERLY, IT ALONE WILL TARGET A SPECIFIC PATHOPHYSIOLOGICAL SYSTEM.

Four types of specific symptoms

Type 1	<p>VERTIGO</p> <p>A sensation of movement through space, usually objective or subjective rotation, which indicates a pathology of the INNER EAR (vestibule) and/or its connections to the CENTRAL NERVOUS SYSTEM (CNS, i.e. brainstem and cerebellum).</p>
Type 2	<p>FAINTNESS, PRESYNCOPE “SUDDEN WEAKNESS – SEEING BLACK”</p> <p>Short-term symptoms (seconds), which point toward BENIGN (vagal reflex, etc.) or MALIGNANT (ventricular arrhythmia, atrioventricular block, sinus pause) pathology of the CARDIOVASCULAR SYSTEM.</p>
Type 3	<p>DISEQUILIBRIUM WHEN WALKING</p> <p>Indicates pathology of the central nervous system, spinal cord, peripheral nerves, or muscular-skeletal system; determined by a neurological and muscular examination.</p>
Type 4	<p>LIGHT-HEADEDNESS</p> <p>Symptoms, often lasting several hours, frequently accompanied by a long list of other non-specific symptoms (tension headaches, scotomas, numbness). Often a diagnosis of exclusion is required when the patient spontaneously expresses the symptoms, suggesting, indifferently, TYPE 1, TYPE 2, and TYPE 3 simultaneously. TYPE 4 is secondary to HYPERVENTILATION.</p>



During this first step determine the TYPE of dizziness using a patient questionnaire that asks **non-leading** questions – NEVER suggest specific symptoms (TYPE 1, 2, 3, 4). Conclusions can usually be drawn in less than five minutes.

<p>Mastering this step alone, even without perfecting the next two steps, will considerably increase your intervention effectiveness, pointing rapidly to a specific pathophysiological system:</p>	Type 1	→	VESTIBULE – CNS (brainstem-cerebellum)
	Type 2	→	CARDIOVASCULAR
	Type 3	→	NEUROMUSCULAR
	Type 4	→	ANXIETY AND HYPERVENTILATION

Step 2

Type 1

Vertigo

HAVING IDENTIFIED A VERTIGO, ONE NOW HAS TO DISTINGUISH BETWEEN A PERIPHERAL VESTIBULAR PATHOLOGY AND ITS CONNECTIONS TO THE CENTRAL NERVOUS SYSTEM (BRAINSTEM, CEREBELLUM) USING TWO CLINICAL TOOLS: QUESTIONNAIRE AND PHYSICAL EXAMINATION.

Having determined the TYPE of dizziness using only the questionnaire and a physical exam, this step will provide more information and determine a more targeted and specific differential diagnosis of potential illnesses.

Questionnaire

The accompanying neurovegetative symptoms (nausea, vomiting) are non-specific, accompanying all vertigo whether vestibular or central. The presence of a specific symptom (either DIPLOPIA, DYSARTHRIA, HEMIPARESIS, HYPOESTHESIA or ATAXIA) accompanying vertigo is ESSENTIAL to the diagnosis OF CENTRAL NERVOUS SYSTEM PATHOLOGY, which may be:

- **vascular:** transient symptoms (vertebrobasilar insufficiency)
- **tumoral:** progressive symptoms (tumour of the posterior fossa)
- **inflammatory:** fluctuating symptoms (multiple sclerosis)

Physical examination

NEURO

Cranial nerves

Cerebellum tests (finger-nose, pro-supination, gait, Romberg)

NYSTAGMUS

Jerk: Fast phase and slow phase

Pendular: Movements of equal force and velocity in two directions

Rotary: Toward the upper part of the orbit and lateral

The nystagmus **AXIS** is often horizontal, sometimes vertical, and is assessed at a base position (looking forward) and deviation looking left and right.

In the base position in the absence of vertigo, the nystagmus (especially pendular) is usually congenital. In the presence of vertigo, it could be a NON-DISTINCTIVE peripheral vestibular or central nervous system lesion.

Distinguishing between vestibular and central nervous system problems can be done by characterizing the NYSTAGMUS. A vertical nystagmus, upbeat (trunk) or downbeat (cervical-medullar) always originates in the central nervous system.

A HORIZONTAL nystagmus, usually a jerk, comes from:

PERIPHERAL VESTIBULAR

- Quick phase, always towards the same direction regardless of which side the eyes have been looking.
- Amplified when the eyes look to the quick phase direction, indicating a problem with the ear on the opposite side.

CENTRAL NERVOUS SYSTEM

- Change of direction with the direction of gaze: moving to the left with gaze to the left, moving to the right with gaze to the right.

key points

Diagnosing **central** vertigo relies on its association with a specific symptom of the central nervous system (diplopia, dysarthria, paresis, ataxia) and/or an anomaly in the neurological exam, or a nystagmus that is vertical or changes direction with the gaze.

In the absence of a symptom or signs of a problem in the central nervous system, the peripheral vestibular system is considered the cause of vertigo.



IMPORTANT EXCEPTION for emergency room doctors

A thrombosis of the posterior inferior cerebellar artery (PICA) leading to an infarct of the cerebellum with no other sign of a brainstem problem manifests itself as severe and prolonged vertigo lasting several days and is different from vestibular neuronitis vertigo or Meniere's Disease in that the patient does not have BALANCE: cannot stay seated (axial instability) nor stand (lateropulsion) and has an unstable head (reeling gait). This pathology can constitute a neurosurgical emergency.

In conclusion

This second step should allow clinicians to differentiate, during an assessment of TYPE 1 dizziness, between peripheral vestibular and a central nervous system cause of "vertigo", primarily through medical history and a physical exam.

Step 2

Type 2

Faintness, Presyncope “Sudden Weakness – Seeing Black”

THE METHOD OF APPROACHING AND ASSESSING TYPE 2 DIZZINESS AND CARDIOVASCULAR SYNCOPE ARE THE SAME. CLINICALLY, THREE TYPES OF SYNCOPE ARE CHARACTERIZED BY THE WAY THEY APPEAR AND BY THE TYPE OF RECOVERY:

1	2	3
CARDIOVASCULAR SYNCOPE Sudden onset, sudden end (90% cases of syncope)	NEUROLOGICAL SYNCOPE (EPILEPSY) Sudden onset, prolonged end (postictal period)	METABOLIC SYNCOPE Slow onset, prolonged end (hypoglycemia)

Cardiovascular pathologies that can bring on this sort of dizziness (Type 2) or syncope are numerous and can usually be grouped into four major categories:

“MER”

- M** **MECHANICAL (obstructive)**
Aortic valve stenosis or hypertrophic subaortic stenosis or left atrial myxoma
 - E** **ELECTRIC**
Bradycardia (prolonged pause, AV block, sick sinus syndrome)
Supraventricular or ventricular tachycardia
 - R** **REFLEX**
Vagal, cardiac reflex, miction, defecation, cough, swallowing, etc.
- DROP IN BLOOD PRESSURE**
Medication, orthostatic hypotension, hemorrhage

During the second step, the identification of TYPE 2 dizziness should lead automatically to this immediate and essential question:

Has the (pre)syncope been observed in a patient with a NORMAL heart or with a SICK heart (ASHD, especially left ventricular dysfunction)?

The distinction is important.

PRESYNCOPE with:

- a **normal heart** is observed in younger patients, in more than 70% of **REFLEX** origin (vagal, cough, etc.) and carries a good prognosis.
- a **sick heart** is observed in older patients, usually with a positive cardiovascular history (if heart failure, more than 70% of cases are caused by ventricular tachycardia with a risk of death greater than 30% in **one year**).

The initial assessment provides a definite or suspected diagnosis in nearly **75% of patients** and/or normally provides a distinction between a **sick** heart (urgent, transfer to hospital) and a **normal** heart (external evaluation).

TOOLS: INITIAL ASSESSMENT: HISTORY – PHYSICAL EXAMINATION – ECG

History

REFLEX CAUSE

CARDIAC CAUSE

BACKGROUND

Starting at a young age

Starting at an older age

- family history of prior sudden death: (hypertrophic cardiomyopathy – Brugada and long QT syndrome – right ventricular dysplasia)
- former myocardial infarction (LV dysfunction)
- medication

BEFORE THE EPISODE

POSITION:

Activity:

standing

after exercise
miction – defecation – coughing –
swallowing – turning the head:
carotid sinus hypersensitivity

lying

during exercise

moving arm: subclavian steal

BEGINNING OF EPISODE

nausea and vomiting
pallor – yawning

chest pains
palpitations

DURING THE EPISODE

pallor, sweating
brief tonic-clonic seizures

cyanosis
brief tonic-clonic seizures

prolonged convulsive movements, urinary and fecal incontinence, tongue biting = neuro

AFTER THE EPISODE

nausea, vomiting
fatigue
chills

palpitations
chest pain

confusion >5 minutes = neuro

In summary, a **reflex** cause is suspected in a **young** patient with identification of a precipitating event, usually observed **standing**, often with accompanying nausea, fatigue, and no history of prior cardiac problems.

A **cardiac** cause is more readily suspected in an **older** patient or observed in the **lying** position or perhaps accompanied by **palpitations** or **chest pains** during the episode or a history of prior heart problems.

Physical Examination

- Lying and standing BP: - orthostatic hypotension
- Carotid sinus massage
- Aortic murmur
- Signs of heart failure: - augmented jugulars - lung rales
- S3 - legs edema

Electrocardiogram

- Sinus bradycardia (diagnosis if <40)
- Bifascicular bundle branch block (LBBB and RBBB alternating or RBBB plus LAHB)
- AV block of 2nd degree
- WPW
- Brugada syndrome: right bundle branch block plus ST segment elevation V1 to V3
- Prolonged QT (>500 milliseconds)
- Supraventricular or ventricular tachycardia
- Sinus pause >3 seconds
- Old or new myocardial infarction

The presyncope with a sick heart in which the specific diagnosis was not done with the initial assessment tools demands an **immediate** evaluation (often in an emergency ward or hospital) with second-level specific assessment tools.

key points

TYPE 2 DIZZINESS OR CARDIOVASCULAR SYNCOPE

DISTINGUISH	NORMAL HEART:	>70% reflex syncope
	SICK HEART:	>70% arrhythmia – conduction disease

Initial assessment tools: **HISTORY – PHYSICAL EXAMINATION – ECG**
In the SICK HEART group: EMERGENCY EVALUATION in a hospital

The presyncope of reflex origin and VAGAL type with an identifiable triggering factor (pain, fear, etc.) or associated with SWALLOWING, COUGHING, MICTION or DEFECATION is usually easy to identify based on history.

In this same group, a different category called “**CARDIAC REFLEX SYNCOPE**” requires further review. It is a (pre)syncope which is almost **ALWAYS** associated with the **UPRIGHT POSITION** (after a certain time, contrary to orthostatic hypotension, which is seen a few seconds after standing up), characteristic of a cardiovascular presyncope with sudden onset and finish, which may be associated with **convulsions** (secondary to cerebral hypoxia), often with disastrous consequences (erroneous diagnosis of epilepsy, loss of driver's licence, multiple traumas).

Pathophysiology: During orthostasis, about 400 to 600 cc of blood descend into the legs, diminishing the volume of the left ventricular flow, stimulating the baroreceptors (aortic arc, carotid) and causing a compensating sympathetic stimulation, which in turn causes a peripheral vasoconstriction and increases the frequency as well as the force of contraction of the left ventricle. In some patients, this increased contractility can be excessive, stimulating mechano-receptors (myelinated C fibers) in the cardiac muscle, giving rise to a new pathological reflex which dominates the first.

The hyperstimulation of mechano-receptors is the beginning of an afferent path which, through a relay in the brainstem, results in a major inhibition in the efferent path of the sympathetic system (**DROP IN BLOOD PRESSURE**) and a stimulation of the same intensity of the parasympathetic system (**PROLONGED PAUSES**). These two events are responsible for the syncope and, if severe, can cause convulsions through cerebral hypoxia. This diagnosis is confirmed by the tilt table test. (**SEE Step 3**).

The presyncope observed in a **sick heart**, due to its poor prognosis and the most probable etiology, **REQUIRES IMMEDIATE EVALUATION**. The patient must be admitted to an emergency ward for an evaluation of ischemic risk, left ventricular function, arrhythmia monitoring, and an electrophysiological test, if necessary. Confirmation of ventricular tachycardia, either spontaneous or prompted during the electrophysiological test, will lead to appropriate treatment (implantable defibrillator) in patients with LV dysfunction.

key points

TYPE 2 DIZZINESS OR CARDIOVASCULAR SYNCOPE

DISTINGUISH	NORMAL HEART:	>70% reflex syncope
	SICK HEART:	>70% arrhythmia – conduction disease

In the SICK HEART group: EMERGENCY EVALUATION in a hospital (high mortality)



Type 3

Disequilibrium

Step 2

DIS-EQUILIBRIUM, WITH OR WITHOUT LATERALIZATION, IS VERY FREQUENT IN ELDERLY PATIENTS, OFTEN MULTI-FACTORED (DIMINISHED VISION, DIMINISHED PROPRIOCEPTION, SEVERE KNEE AND HIP OSTEOARTHRITIS, LACK OF PHYSICAL FITNESS).

Possible neurological problems (stroke, posterior cord disorder, neuropathy, myopathy) can be determined by an elaborate **neurological examination**. The next assessment steps should be based on the clinical diagnosis drawn from the physical examination.

Step 2

Type 4

Light-Headedness

AFTER THE INITIAL PATIENT QUESTIONNAIRE INDICATES TYPE 4 AND THE PHYSICAL EXAMINATION CONFIRMS THE ABSENCE OF OTHER PROBLEMS POTENTIALLY ASSOCIATED WITH THIS DIZZINESS, PROVOKE HYPERVENTILATION IN THE PATIENT AT THE END OF THE CONSULTATION.

Inform the patient of the unpleasant feelings associated with the process but indicate that it is neither serious nor dangerous, particularly in the presence of medical professionals. Have the patient tell you when he or she feels bad and describe the sensation in detail.

Most often, patients will state and describe that the experience is exactly the same as with their original complaint. At this point, make the patient aware that he realizes the cause of his symptoms (hyperventilating) and that it is not a dangerous or obscure disease.

Knowing the real cause of his symptoms, he also realizes that he has complete CONTROL over those incapacitating symptoms by controlling his breathing (he can hold his breath or breathe in a bag). Afterwards we explain in simple terms how hyperventilation creates the physical symptoms – reassure him and finally explain that anxiety causes hyperventilation and appropriate steps should be taken to treat it in order to prevent recurrence of symptoms.

key points

- Provoke hyperventilation in the patient to demonstrate the cause of symptoms.
- Show the patient that he can CONTROL the symptoms by controlling his breathing instead of those symptoms CONTROLLING his life.

Remove all anxiety generated by the symptoms using appropriate information, **breaking the vicious cycle.**

Avoid prescribing unnecessary tests that raise worries of a serious illness and will make the patient worse, reinforcing the belief that he may have a “strange or obscure” disease.



Step 3

Type 1

Vertigo

AFTER DETERMINING IN STEP 2 THAT THE PATIENT HAS PERIPHERAL VESTIBULAR VERTIGO, WE WILL PURSUE THE CLINICAL EXAM UNTIL MAKING A PRECISE DIAGNOSIS OF THE PATHOLOGY INVOLVED WITH RELYING ALMOST EXCLUSIVELY ON HISTORY. IF THE PROBLEM IS BENIGN POSITIONAL VERTIGO, AN EXTREMELY EFFECTIVE TREATMENT IS POSSIBLE (90% OF CASES OF VERTIGO).

Each primary vestibular pathology carries its own signature (practically a BAR CODE) provided through the characteristics of the vertigo (DURATION AND FREQUENCY).

1. Benign Positional Vertigo

Paroxysmic vertigo of very short duration (30 TO 60 SECONDS) observed ONLY with a change of position, without a history of more prolonged vertigo.

PATHOGENESIS:

Benign positional vertigo is caused by the movement of debris (otoliths) in the posterior semi-circular canal (occasionally horizontal canal), descending due to gravity when a position is changed and causing a temporary hydrolymphatic drag on the receptor in the canal (cupula) – and thereby causing vertigo and characteristic nystagmus.

DIAGNOSIS:

Dix Hallpike Manoeuvre

TREATMENT:

Epley Manoeuvre

2. Meniere's Disease

Episodic RECURRING vertigo lasting from FIVE MINUTES TO 20 HOURS, consisting of the classic trio of vertigo – hearing loss – tinnitus.

DIAGNOSIS:

Audiogram. Hearing loss, mostly at less than 1000 hertz base frequency.

TREATMENT:

Medical, symptomatic, surgical, mostly REASSURANCE.

3. Vestibular Neuronitis

ONE PROLONGED vertigo from **TWO DAYS TO TWO WEEKS – NO RECURRENCE**

DIAGNOSIS:

History and examination.

TREATMENT:

Medical, symptomatic.

WARNING:

When assessing PROLONGED vertigo, even in the absence of apparent CNS (Central Nervous System) problems, a differential diagnosis must still be made between vestibular neuronitis and PICA (Posterior Inferior Cerebellar Artery) thrombosis. In the latter case, the patient will present a truncular ataxial (sitting-standing-walking).

4. Recurring Neuronitis

A RECURRING episode of 5 to 20 minutes without the MENIERE'S DISEASE trio and without signs of CNS problems.

EVOLUTION:

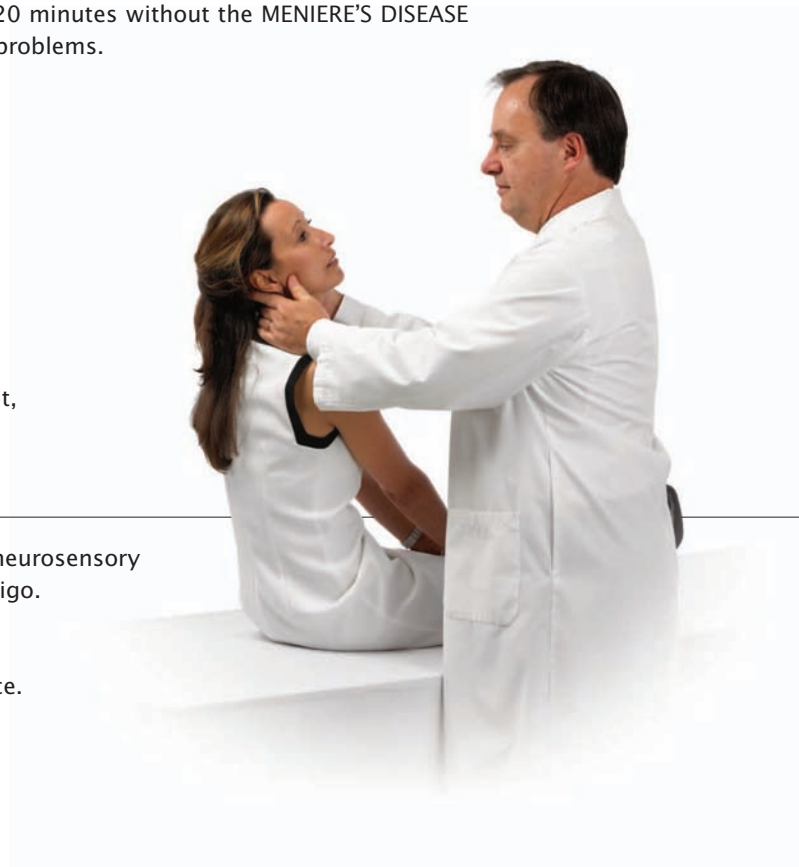
15% benign positional vertigo
10% Meniere's Disease
75% remains unchanged

DIAGNOSIS:

History and examination.

TREATMENT:

Symptomatic medical treatment,
REASSURANCE.



5. Acoustic Neurinoma

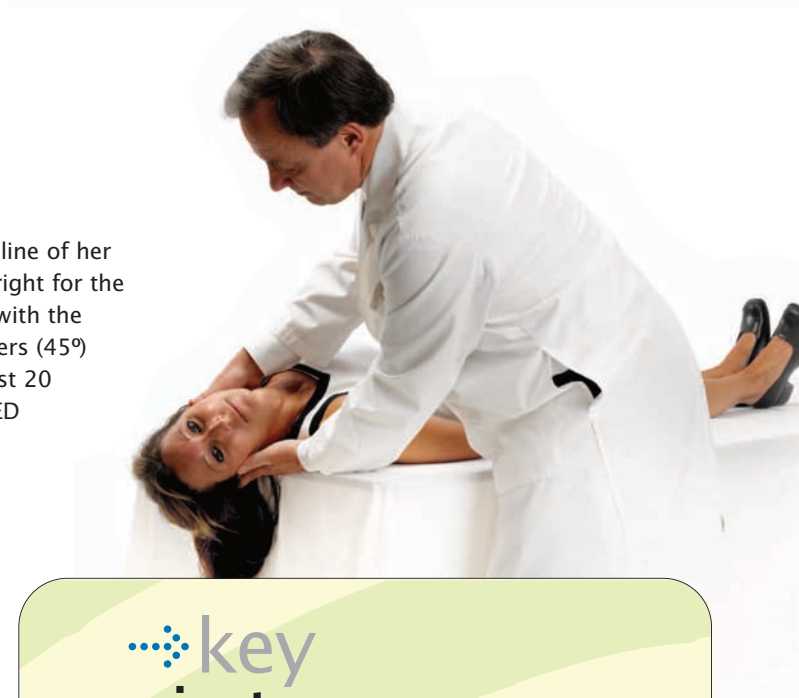
Inexplicable loss of unilateral neurosensory hearing, with usually light vertigo.

DIAGNOSIS:

Audiogram, magnetic resonance.

TREATMENT:

Surgery.




BENIGN POSITIONAL VERTIGO (BPV)

Dix Hallpike

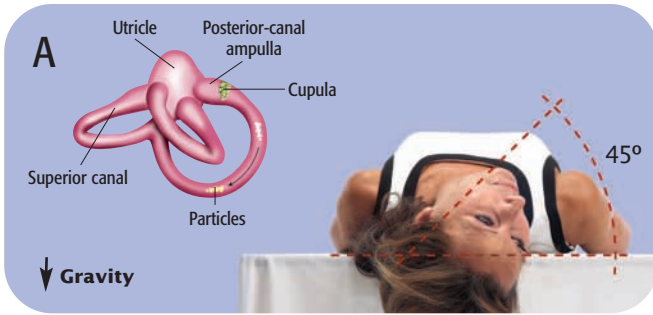
Confirms the BPV diagnosis

With the patient seated, head turned 45° to the left from the line of her shoulders to evaluate the left ear, or head turned 45° to the right for the right ear, put the patient rapidly into the decubitus position with the head position unchanged according to the line of the shoulders (45°) AND 30° lower than the horizontal line of the table for at least 20 seconds. A positive manoeuvre on the SIDE OF THE AFFLICTED EAR provokes a very short-term vertigo (< 30 seconds after a delay of few seconds) and is accompanied by a nystagmus usually rotating towards the top of the head and the base of the table.

A Dix Hallpike may be “**subjectively**” positive either when the patient is put in decubitus or back in vertical position (mild subjective vertigo, very short-term, without the characteristic nystagmus). This “subjectively” positive manoeuvre probably has the same meaning as the objectively positive Dix Hallpike manoeuvre, pinpointing the involved ear to be treated with an Epley manoeuvre.

 **key points**

Dix Hallpike (+): Confirms the diagnosis
Dix Hallpike (-): Does not eliminate the diagnosis made by clinical history



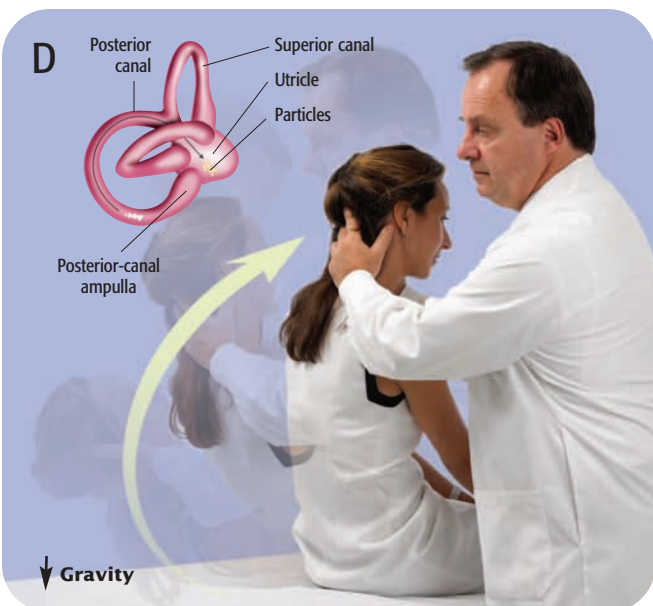
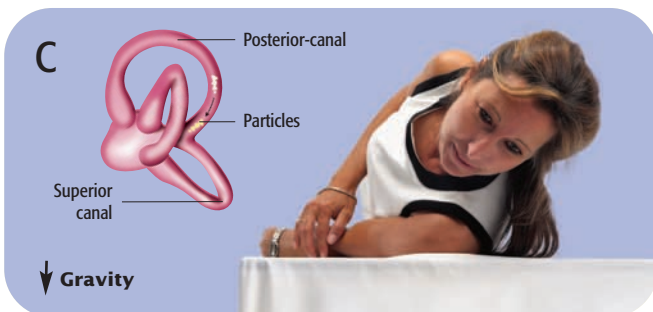
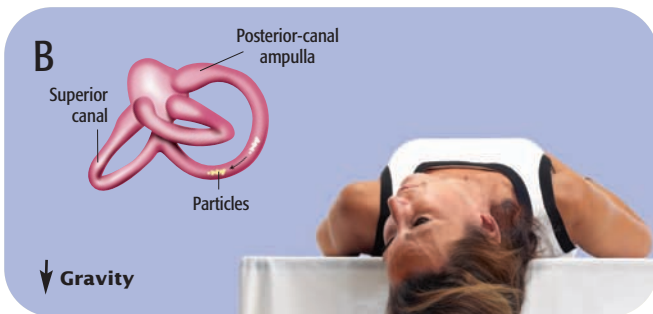
Epley's Manoeuvre

Illustration: Manoeuvre for posterior canal of right ear.

Position A corresponds to the position of the head after the Dix Hallpike manoeuvre with a 45° rotation toward the right and 20 to 30° below the horizontal plane. Maintain that position for about 90 seconds. The head is then slowly turned 90° to the opposite direction (position B: 45° toward the left, 20 to 30° below the horizontal plane) and is held in that position for about 90 seconds. At that point, with the position of the head not changing in relation to the shoulder line, the patient rotates his or her body 90° in the same direction (toward the left) entering position C, which is to be held for about 90 seconds. For position D, the patient is brought back into vertical position, with the head slightly flexed forward (30° for about 30 seconds).

In each of these positions, a part of the semicircular canal is vertical, permitting the otoliths to travel using gravity, and, using the proper sequence and timing, permitting the repositioning of those otoliths in the utricle. Those otoliths will continue to move freely without deflecting the “cupula”, rendering the patient asymptomatic.

There is a 15% chance that, in time, the otoliths may fall back in the canal, which should be treated by repeating the manoeuvre. The patient should be informed of that possibility in order to seek proper treatment without delay. Repeating the manoeuvre may be necessary for a complete cure. Treatment of the left ear is the same with reverse directions starting the head rotated to its left and ending on the right shoulder. This manoeuvre repositions otoliths located in the posterior semicircular canal.



key points

A specific vestibular problem is diagnosed through the DURATION and FREQUENCY of the vertigo.

A definitive treatment is THE RULE for benign positional vertigo treated by a CORRECTLY EXECUTED Epley manoeuvre (variants exist).

Possible causes of failure in the Epley manoeuvre include: false diagnosis, wrong head position (cervical osteoarthritis), inadequate time in each position, horizontal canal? (requires another manoeuvre), or the treatment of the wrong ear.

KEEP TRYING!

Step 3

Type 2

Faintness, presyncope “sudden weakness – seeing black”

AFTER DISTINGUISHING BETWEEN A NORMAL OR SICK HEART IN STEP 2, THE PROBABILITY OF THE RESPONSIBLE ILLNESS IS CLEAR AND THE URGENCY OF THE SITUATION HAS BEEN DETERMINED. THE PRESYNCOPE AND SYNCOPE ARE NOW ASSESSED WITH THE SAME TOOLS.

Specific Assessment

Normal heart:

- Ambulatory monitoring
- Carotid sinus massage
- Internal or external “loop recorder”
- Tilt table test

Sick heart:

- Intra-hospital and/or ambulatory monitoring
- Echocardiogram (LV dysfunction?)
- Stress test
- Internal or external “loop recorder”
- Electrophysiological tests (EPS)

The choice of specific assessment tools is adapted to each case according to the severity of the clinical signs and suspected diagnosis.

Despite this approach, about 25% of cases do not have a definitive diagnosis (especially if they stem from an isolated incident).

Tilt Table Test

(normal heart reflex syncope)

Use the tilt table test when a “cardiac reflex” syncope is suspected and is justified by the severity of the clinical signs, after which all the usual tests have been negative. The patient is placed on a table with continuous monitoring of heartbeat and blood pressure. The table is then lifted vertically to 60° to 80° over about 50 minutes. Characteristically, the syncope will be observed after 10 to 15 minutes secondary to an extreme bradycardia and/or a severe drop in blood pressure. This test can be made more effective by using an Isuprel IV drip, Nitro IV. Sensitivity is about 85% (variants exist).

Treatment of a “cardiac reflex” syncope according to the severity of the symptoms may include:

- Keeping the optimal plasma volume (hydrate well occasionally, Fiorinef to keep the optimal plasma volume and diminish the initial sympathetic stimulation during orthostasis).
- Beta-blockers or Rythmodan, to diminish the contracting force of the left ventricle and diminish the stimulation of the mechano-receptors.
- A two-chamber pacemaker to diminish the final expression of the reflex (bradycardia, severe pause) may diminish the number of episodes by 85%.

Electro-physiological Study

To be used for patients with left ventricular failure and presyncope, in which regular tests are negative. Determine whether or not a monomorphic ventricular tachycardia can be induced. The resulting treatment is usually an implantable defibrillator.

Loop recorder

To be used for patients with very severe manifestations, with normal or sick heart, when regular tests are negative.

Technique: an electrode is placed on the surface or implanted under the skin, for weeks or months, with a recorder implanted under the skin which includes a button activated by the patient after a syncope, thereby recording the heartbeat at the time of the clinical event. Sensitivity of 85%.

Summary Table of the Three-Step Assessment Method

	Step 1	Step 2	Step 3
Type 1	Vertigo (Vestibular – CNS)	Vestibular CNS (Symptoms – focal signs)	BPV ⇒ Dix Hallpike – Epley Meniere's Disease Labyrinthitis (Vestibular neuronitis) Vascular – Inflammatory – Tumoral (CT – Magnetic resonance)
Type 2	Presyncope (Cardiovascular)	Initial Assessment History – Examination – ECG Diagnosis or Normal heart – Sick heart (Reflex: 70%) (Electric: 70%)	Specific Assessment Normal heart Ca. sinus massage Loop recorder Tilt table Sick heart ST (Stress Test) – Echo Loop recorder EPS
Type 3	Disequilibrium (Neuromuscular)	Examination Neurological – Locomotor	
Type 4	Light-headedness (Anxiety)	Provoke patient's hyperventilation	Recognize the cause of the physical symptoms – and control it



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Jacques Bédard

MD, CSPQ, FRCPC, Internal Medicine

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