

KEYWORDS

vertigo, cardiac giddiness, bedside tests for vertigo

Giddiness is one of the commonest symptoms encountered by physicians on a day to day basis. As the term suggests, it implies a feeling of imbalance. However, most patients are quite imprecise in their descriptions of giddiness and may variously call it dizziness, spinning, light-headedness, etc. Giddiness can be an innocent symptom of a simple disease or may be a harbinger of a serious condition such as a stroke.¹ The challenge in day to day life for physicians is to make a precise diagnosis of such a vague symptom, and to identify and treat the more dangerous diseases that may present with this common symptom.

CAUSES OF GIDDINESS

Broadly classified, the causes of giddiness are due to vestibular dysfunction, brainstem problems, cardiac conditions or psychiatric disorders.

IMPORTANCE OF HISTORY IN GIDDINESS

The first step in trying to find the cause of giddiness is to take a detailed history, because the precise cause of giddiness can be easily identified in several cases by just taking a comprehensive history.

GIDDINESS OR VERTIGO?

The most important task is to differentiate in the history between giddiness and vertigo, as the causes of vertigo are few and easy to identify. The patient should be asked if there is a definite sense of rotation or spinning, which will point to the diagnosis of vertigo. If this is present, it usually means that the vestibular system is involved. This system comprises of the vestibular apparatus in the inner ear, the vestibular nerve and nucleus and the connections of this to the cerebellum.

Benign positional vertigo, vestibular neuritis, Meniere's disease are the peripheral causes connected with the ear, whereas posterior circulation ischemia, infarction or tumors of posterior cranial fossa are called central causes as they involve the brain stem.² Differentiation of the various causes of vertigo will involve some simple clues from history.

DURATION OF VERTIGO AND ACCOMPANYING SYMPTOMS

If the person has vertigo, the following questions will help to clarify the cause of vertigo. Transient attacks of vertigo generally point to benign paroxysmal positional vertigo (BPPV), where the episode may last even less than a minute. As the name suggests these attacks are episodic,

transient, and precipitated by posture. Movements of the head can cause such severe giddiness that some patients are terrified and refuse to move the head even slightly.³

Recurrent spontaneous attacks of vertigo are a feature of Meniere's disease. This is usually associated with auditory symptoms such as hearing loss and pain or fullness in the ear which may occur during the attacks. Vestibular migraine which also lasts few minutes to hours may cause vertigo which is facilitated by visual movements such as while watching movies.

Prolonged periods of severe vertigo of acute onset may be due to vestibular neuritis. These episodes are sudden in onset and may remain even when the head is held still thus differentiating it from BPPV. One of the challenges for the treating doctor is to differentiate this from the vertebral-basilar insufficiency such as due to posterior circulation transient ischemic attacks. These are also episodic and may last only a few minutes with full recovery. This is when we are faced with the challenge of differentiating peripheral causes of vertigo from central causes.

CENTRAL OR PERIPHERAL VERTIGO

Symptoms such as tinnitus, nausea and recurrent vomiting during the episodes are classical of peripheral vertigo due to involvement of the ear. Cerebellar and medullary lesions causing vertigo do not cause tinnitus and much less vomiting. In addition, other symptoms of posterior circulation ischemia such as dysarthria, dysphagia, diplopia quadriparesis and ataxia, may point to a brain stem cause such as vascular events or tumors.⁴

It is however important to note that patients with peripheral causes of vertigo can also be ataxic during the episode of vertigo, and may manifest a sense of imbalance. It is the accompanying neurological deficits which will confirm the central cause.

Extension of the neck to look upwards can cause giddiness in patients with cervical spondylosis due to compromise of the vertebral circulation. The classical example of a grandfather pointing out an airplane to a grandson and losing balance every time he attempts this, illustrates this cause. Associated symptoms of cervical spondylosis such as neck pain, restriction of neck movements and weakness in the upper limbs may help to make this diagnosis.⁵

Chronic persistent vertigo lasting over several months is generally due to psychiatric problems such as somatization, anxiety or depression. These patients are vague in their description of vertigo, and often have several other multiple complaints.

If the patient has no sense of rotation, but has only giddiness, several other important causes must be considered. The most important of these are cardiac causes.

CARDIAC CAUSES OF GIDDINESS

Orthostatic hypotension is an important cause of giddiness. This is diagnosed as a fall in systolic BP of 20mm of Hg from supine to standing position after 3 minutes of standing. This typically manifests as feeling of faintness or black outs when the patient stands up from supine position. The important causes of orthostatic hypotension are autonomic neuropathy, and other cardiac causes such as arrhythmias and shock.

The causes of autonomic neuropathy are several, the commonest being diabetes mellitus, elderly age and drugs.⁶ This condition leads to feeling faint on standing up from lying or sitting posture due to lack of appropriate sympathetic response to change of posture due to poor sympathetic tone. Most elderly patients report giddiness as a feeling of blacking out or feeling faint or light headed. Such pre-syncope can be preceded by pallor which is often observed by bystanders.

Diabetic autonomic neuropathy should be suspected in any long standing diabetic with peripheral neuropathy. The blood pressure must be checked in the lying and standing position in all diabetics as a routine when they come for follow up.

Orthostatic hypotension can cause postural instability in the elderly and lead to falls. Many old people are unable to report the faintness on standing and describe it vaguely as weakness, which is due to decreased cerebral circulation.⁷ Some of them may be on anti-hypertensive's which can cause a significant postural drop such as beta blockers and prazosin. First dose hypotension is a well-known feature of prazosin and all elderly patients are cautioned not to stand up suddenly after initiation of alpha blockers at least in the first few days.

In the emergency setting, any patient complaining of giddiness on standing, must be assessed for volume loss, such as due to vomiting or diarrhea, or occult blood loss, such as bleeding from peptic ulcer into the gut. Loss of more than 10% of blood volume can cause giddiness on standing and orthostatic hypotension with more than 20 mm of BP fall in systolic pressure on standing.⁸ More than 30% loss of blood volume can cause giddiness even in supine posture, and this should alert the physician to a grave situation.

Further cardiac emergencies such as acute coronary syndrome such as myocardial infarction and pulmonary embolism can also cause severe giddiness and faintness. Again the accompanying symptom of chest pain or discomfort, and breathlessness may reveal the cardiogenic shock or pulmonary edema.⁹ Patients with pulmonary embolism feel more giddiness and breathlessness on sitting up, which is the opposite of left ventricular failure, and should alert one to this possibility.

Pre-syncope occurring even in the supine position is probably due to cardiac arrhythmias.¹⁰ Both tachyarrhythmias such as supraventricular tachycardias and atrial fibrillation with fast ventricular response and bradyarrhythmias such as complete heart block and sick sinus syndrome can present with giddiness by compromising cerebral blood flow. These can cause giddiness in the lying posture and that is an important clue to the presence of an arrhythmia. A history of palpitations, chest pain or breathlessness preceding the light headedness may give a clue to cardiac nature of the syncope.¹¹

OTHER CAUSES

Sudden rise in blood pressure can cause dizzy spells. These can be accompanied by headache, and must be thought of in elderly patients, warranting checking of blood pressure. However, chronic hypertension and mild rises of blood pressure hardly cause any giddiness.

Hypoglycaemia can present as giddiness, especially in a diabetic on insulin or sulphonylureas after a missed meal. The setting and the accompanying symptoms of palpitation and sweating may help in clinching this diagnosis.

The last group of non-specific giddiness is due to psychiatric illnesses.¹² Patients with panic attacks can have tachycardia, sweating and light headedness, which may be mistaken for a cardiac problem. Hyperventilation due to hysteria can lead to giddiness. However, this cannot last very long as the patient will tire out in a few minutes. The social circumstances and the personality of the patient may help to make the diagnosis of a hysterical conversion reaction.

Post-traumatic giddiness may follow trauma to the head or whiplash injuries. Such episodes can last up to a month following trauma. Post-traumatic stress disorder may also complicate the clinical picture and make differentiation between the two entities difficult.¹³

A comprehensive drug history regarding the use of anti-depressants and anti-cholinergics is important to rule out drug induced causes of dizziness. Several patients introduced to antidepressants or benzodiazepines for the first time, complain of giddiness, and many of them discontinue the drugs due to this symptom. Hence it is important to elicit a complete drug and disease history in all patients with giddiness.

PHYSICAL EXAMINATION IN PATIENTS WITH GIDDINESS

The physical examination must begin with assessment of hemodynamic parameters such as pulse. Tachycardias or arrhythmias such as atrial fibrillation must be ruled out. Bradyarrhythmias such as complete heart block or sick sinus syndrome may present with very low heart rates below 40/minute. Blood pressure must be checked in lying down and standing position to rule out orthostatic hypotension. Hypoxia accompanying giddiness is suggestive of a cardiac emergency.

Nystagmus is an important clue to the presence of a vestibular cause of the giddiness. Nystagmus due to

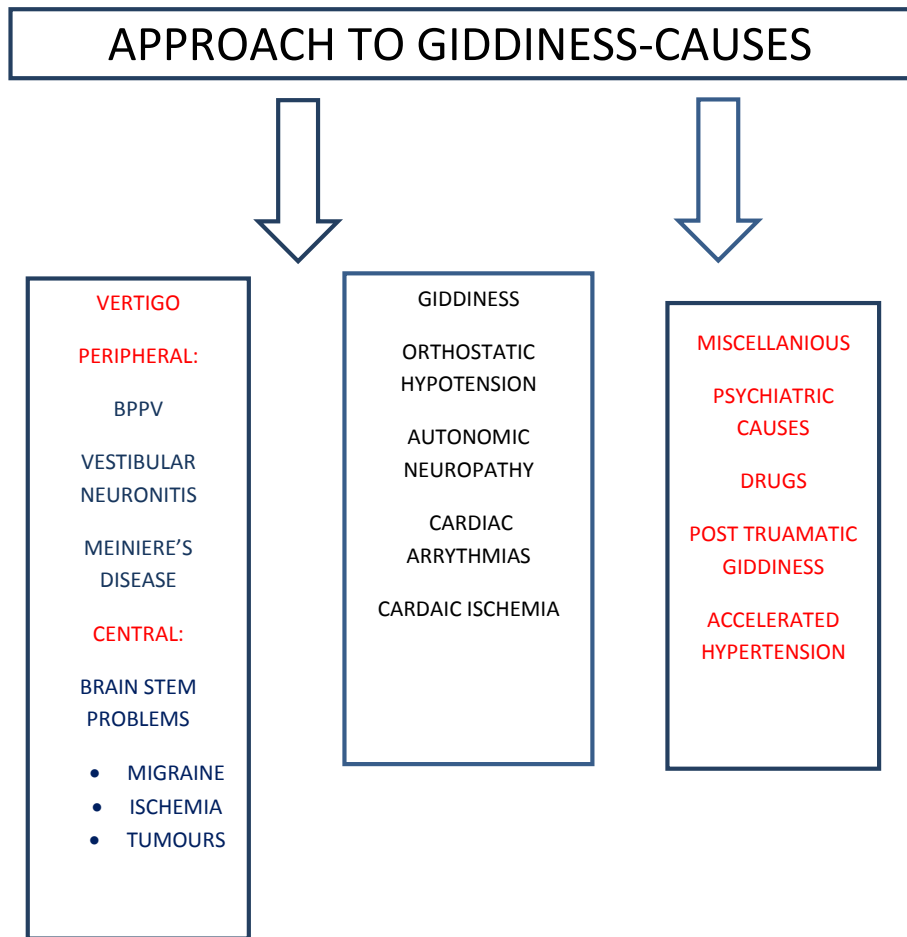


Fig. 1:

labyrinthine lesions is usually to the opposite side and increases in frequency and amplitude when the patient is asked to look away from the side of lesion. The type of nystagmus gives an important clue to the site of origin. A horizontal/torsional nystagmus is due to affection of semicircular canals on one side. It is important to remember that such peripheral nystagmus is never vertical or purely torsional. Further, nystagmus due to peripheral lesion can be suppressed by visual fixation.

Nystagmus due to brainstem lesions can be horizontal, vertical or torsional. Cerebellar nystagmus is gaze dependent and increases in the direction of gaze and is more to the side of lesion. For example, right cerebellar lesion will cause nystagmus to increase on looking to right side. Careful neurological examination must be done in such patients to look for cerebellar signs or lower cranial nerve involvement. Most patients with such lesions are unable to walk and patients may fall to the side of lesion if the cerebellum is involved.¹⁴

An acoustic neuroma is a slow growing intracranial tumor which typically presents with tinnitus, vertigo, deafness and cerebellar involvement. The history may last several years as a tumor grows slowly and does not cause much intracranial tension. Examination typically reveals involvement of seventh and eighth cranial nerves with cerebellar ataxia on the side of lesion.

BEDSIDE TESTS FOR VERTIGO

Dix-Hallpike's maneuver

This is a simple bedside test to induce vertiginous attacks and produce nystagmus in a patient with a history of positional vertigo. These tests are designed to diagnose canalithiasis of the posterior semicircular canal, which is an important cause of BPPV.¹⁵

The patient is made to sit on the bed while the neck is turned to one side and extended. Then he is suddenly changed to supine position with the head hanging over to the side of the bed for 30 seconds. If BPPV is present, nystagmus and vertigo will occur after a latency of a few seconds and last for brief period. Such nystagmus can be torsional in nature. Since this test is fatigable, the duration and intensity of nystagmus may diminish with every repetition. The sensitivity of this maneuver is quite high that is above 85% for the diagnosis of posterior canal BPPV. Thus, it is a very useful test for the confirmation of this simple diagnosis.

Several other tests such as head impulse test or head thrust test can also be used. This is done by asking the patient to focus on a distant object and turning the head quickly and unpredictably by about 15 degrees. If the eyes are dragged off the target and followed by a saccade back on to the target after the head turn, this indicates a deficient vestibule-ocular reflex and pointing to a

150 peripheral vestibular lesion. This test may have a higher specificity (80-100%) but poor sensitivity (35-40%).¹⁶

LOOKING FOR NEURO DEFICITS

Any cause of vertigo due to a brain stem cause, will be usually accompanied by neurological deficits such as 7th and 8th nerve palsy, ataxia, or quadriparesis. A patient with Wallenberg's syndrome, due to infarction of lower medulla can complain of vertigo along with ataxia. Examination in such a patient will show cerebellar signs on side of lesion, lower cranial nerve such as 9th and 10th nerve palsy, in addition to nystagmus, which will help to localize the level of lesion.¹⁷

INVESTIGATIONS FOR GIDDINESS

These must be chosen depending upon the clinical diagnosis. For vestibular problems, a caloric test may help in establishing a diagnosis. An audiometry is useful to diagnose conditions such as Meniere's disease and acoustic neuroma. Audiometry at the time of attack may show decreased hearing and eventually the patient may develop permanent deafness. In acoustic neuroma, audiometry may reveal sensori-neural deafness due to involvement of the cochlear nucleus of the eighth cranial nerve.

An MRI is indicated for imaging of brainstem in patients suspected to have posterior circulation ischemia or infarction. Wallenberg's syndrome and other medullary or cerebellar infarctions are easily picked up by MRI. MR angiography is very useful in detecting stenosis or thrombosis of the vertebral and basilar arteries with a very high sensitivity and specificity of over 95%. Further, tumors in the posterior cranial fossa such as acoustic neuroma can be diagnosed with MRI of brainstem.¹⁸

For cardiac lesions, a basic ECG to diagnose arrhythmias is essential. For patients with paroxysmal giddiness, a transient arrhythmia must be considered. Holter monitoring for 24 hours may be useful in such patients to diagnose conditions such as sick sinus syndrome (bradycardia-tachycardia syndrome), paroxysmal atrial fibrillation, supraventricular tachycardias or a run of ventricular tachycardia.¹⁹

Hypoglycaemia can be ruled out by a blood sugar test by glucometer. Hypoxia must be checked in all severely giddy patients with a saturation probe to rule out life threatening cardiac emergencies.

To conclude, the diagnosis of giddiness can thus range from simple conditions such as drug induced problems to very serious life threatening strokes. A careful history, simple bedside tests and judicious use of investigations will help to arrive at correct diagnosis.

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