

## EMU 2017 DIZZINESS AND VERTIGO

Walter Himmel MD

There is only one essential challenge in the world of dizziness and vertigo: Don't miss a posterior circulation stroke (vertebral/basilar artery) or TIA.

TIA's from the posterior circulation have a higher incidence of early stroke than do TIA's from the carotids.

Dizzy patients make up about 3% of patients visiting the ED. The number of patients who return with a stroke is highly variable depending on the study. The most benign studies suggests that <.5% return within 30 days with a diagnosis of cerebrovascular disease. With vertiginous patients, higher rates of 3% are quoted. With very high risk, older patients, one study suggested up to 25% have had a stroke.

### THE IMPORTANT DISEASES

- Benign Paroxysmal Positional Vertigo – the commonest cause of true vertigo.
- Central Positional Vertigo – rare cause of positional vertigo.
- Vestibular Migraine – likely, the second commonest cause of recurring vertigo.
- Benign Recurring Vertigo (this is probably unrecognized migraine)
- Meniere's Disease
- Posterior circulation TIA – this is the one we hate to miss in the ED. These patients may be discharged feeling well and come back stroked. These TIA's are much more common in the 90 days before vertebrobasilar strokes than are TIA's before carotid artery strokes. (Lancet Neurology 2013:989)
- Vestibular neuritis – this is the common cause of continuous vertigo for > 24 hours.
- Brainstem or cerebellar infarct. Vestibular neuritis and infarcts are often hard to distinguish from one another.

DISEASES	TIMING	TRIGGER	BENIGN/SERIOUS
BPPV	< 1 minute	Movement	Benign
CPV (Central PV)	1 minute or more	Movement	Serious (central)

Vestibular Migraine (common)	Minutes to hours – usually < 24 hrs	Spontaneous	Benign
Benign recurring vertigo (many are probably vestibular migraine)	Minutes to hours – usually < 24 hrs	Spontaneous	Benign
Meniere’s Disease	Hours - < 24 hrs	Spontaneous	Benign
TIA: posterior circulation	Minutes (usually) to hours	Spontaneous	Serious (central) Older: vascular Younger: PFO, cervical artery dissection
Vestibular neuritis (neuritis, labyrinthitis)	Days	Spontaneous	<u>Mimics stroke</u>
Brainstem and cerebellar stroke	Days (usually)	Spontaneous	<u>Serious and may mimic vestibular neuritis</u>

## THE FIVE IMPORTANT ASPECTS OF THE HISTORY

1. The Type of Dizziness (controversial according to some)
2. The Timing
3. The Trigger
4. Associated Features
5. Context

### 1. The Type of Dizziness

Dizziness – Traditionally, there are four types of dizziness and the ability to distinguish which is helpful in producing a differential diagnosis. Patients who use the word dizzy can use it to describe a wide variety of human experiences.

1. Vertigo: spinning or linear movement. Some feel that vertigo implies a peripheral problem. This is false. Vertigo can be central or peripheral. However, it usually implies a vestibular or brainstem problem.
2. Presyncope/syncope: this generally is attributed to non-vestibular and non-stroke diseases (e.g., vasovagal, orthostatic, cardiovascular, and rarely, neurologic).
3. Disequilibrium: this may be due to ataxia (neurologic) or to metabolic/toxic factors.

4. Nonspecific lightheadedness. This is multifactorial (including anxiety and general medical illness) and usually is not primarily vertiginous.

This breakdown into four types of dizziness has been a model for parsing the word “dizziness” for years. Recently, authors argue that the type isn’t important because patients are inconsistent and the types overlap. I believe that this view is overstated. Presyncope and vertigo are very different. However, dizzy patients usually have fractional elements of all four types of dizziness, but usually, one or two types dominate. These authors also argue that to become overly focussed on one type of dizziness may lead to a premature diagnostic conclusion. This is true. Therefore, there is more to a diagnosis than just the nature or the feel of the symptom. The triggers and the timing matters.

## **2. The Timing (Duration) of The Vertigo**

1. The duration of vertigo is an essential part of the diagnosis. BPPV lasts for < 1 minute and CPV (central positional vertigo) for minutes.
2. Benign recurring vertigo, vestibular migraines, and Meniere’s disease lasts for hours and rarely > 24. TIA’s last for minutes and possibly hours.
3. The acute vestibular syndrome is a syndrome of vertigo, nausea, vomiting, nystagmus, gait instability, and sensitivity to head movement. This syndrome has only two diseases of importance to the ED physician: vestibular neuritis and a stroke of either the cerebellum or brainstem. These conditions last > 24 hours. This is the syndrome in which patients with strokes may be misdiagnosed to have vestibular neuritis and sent home.

## **3. The Trigger of The Vertigo**

1. Triggered by movement: BPPV and CPV. In the absence of movement, there is no vertigo in this syndrome but there may be a sense of feeling unwell or unsteady.
2. Spontaneous: vestibular migraine, Meniere’s disease, TIA, acute vestibular syndrome (vestibular neuritis and stroke). These are made worse with movement but not triggered by it. This is worth repeating: The vertigo is spontaneous and persistent but made worse with changes in position or movement.

## **Warnings and Pitfalls and Misconceptions:**

Vestibular migraine, Meniere’s disease, and TIA are often recurring. But, if you are seeing the patient for their first or second attack, you can’t tell for sure that this is a migraine or Meniere’s disease. Therefore, have a high suspicion TIA. Be resistant to being the first MD

to diagnose vestibular migraine or Meneire's disease in a patient with his or her first or second attack of vertigo.

Acute vestibular syndrome last > 24 hours. But if you are seeing the patient in the ED, you can't predict how long the attack will last. It is unrealistic to keep these patients in the department for 24 hours. Therefore, have a high suspicion of cerebrovascular disease if continuous vertigo is persistent for several hours. Moreover, not all strokes are symptomatic for 24 hours. Some may be lengthy TIA's and resolve; the patient may be sent home and then return stroked out in several hours or days.

All vertigo is made worse with movement. However, positional vertigo is called positional because it is only triggered by movement and is never spontaneous and never continuous.

Even with positional vertigo, the patient will say that he or she is constantly "dizzy" whether she moves or not. However, after a minute and with no further movement, the vertigo will stop in positional vertigo. The history may be difficult to analyze. The spinning or delusion of movement will stop. However, the sense of unsteadiness or slight disequilibrium may persist. The important word in the history is "bed." These patients get a sudden triggering of vertigo when they roll over or lift their head up in bed.

#### **4. Associated Symptoms That Suggest A Stroke:**

- The D's:
- Diplopia
- Dysarthria
- Dysphagia
- Dysphonia (hoarseness)
- Dysmetria
- Dystaxia – gait disturbance is a big risk factor for a cerebrovascular diagnosis
- Dysgeusia: odd taste – seen in carotid artery dissection.
- Crossed sensory loss
- Weakness

#### **Warnings and Pitfalls and Misconceptions:**

It was once taught that pure vertigo without typical associated symptoms is never central. This is absolutely false. Pure vertigo may be the only symptom of a TIA or a stroke in as

many as 20% - 40% of patients with brainstem or cerebellar strokes (although a sophisticated examiner may pick up subtle findings). Of course, these patients will have unsteady gaits.

Gait disturbance is a marker for serious vertigo. Unfortunately, all vertiginous patients have a degree of impaired gait. It is said that “peripheral vertigo” causes lots of vertigo but little gait disturbance and that “central vertigo” causes lots of gait disturbance and less vertigo. This is a nice story – but very difficult to use in practice. Nonetheless, a patient with truncal ataxia or significant gait imbalance should be considered to have a central nervous system problem till proven otherwise.

## **5. Context**

Older patients and vasculopaths clearly are at a higher risk for stroke. However, young people do get strokes. The common causes are PFO's and cervical artery dissections. These are not as rare as once taught. As many as 25% of young patients with stroke may have a cervical artery dissection.

## **THE FIVE IMPORTANT ASPECTS OF THE PHYSICAL**

### **1. General**

- Assessment of CN's is important.
- This includes particularly muscle weakness
- Movement of uvula and tongue
- Sensation
- Long track signs and babinski.
- Cerebellar signs

### **2. Gait.**

- Those who need help to walk don't go home.
- Should also assess for truncal ataxia.
- This is easily done by getting the patient to sit up arms folded on their chest
- Romberg and tandem gait.

### 3. Nystagmus

Jerk Nystagmus with a slow component and a rapid correction phase is frequently seen in vertigo. (Pendular nystagmus without a fast component is a feature of other neurological disease or inborn is inborn).

#### Peripheral Nystagmus

- BPPV, Meniere's disease, and vestibular neuritis virtually always have nystagmus in the early phase of the illness.
- BPPV: the nystagmus is usually vertical-rotatory with a rotational element toward the shoulder on the table. This is seen in the commonest form of BPPV in which the posterior semicircular canal is involved. The nystagmus is never purely vertical – purely vertical is caused by central brain disease. The nystagmus of BPPV, as with the vertigo of BPPV, is triggered by movement. The nystagmus may be horizontal or horizontal-rotatory if the BPPV involves the horizontal canal. This is a less common variety of BPPV.
- Vestibular Neuritis: the nystagmus is usually horizontal-rotatory with often a slight rotational element. It is not triggered. It is spontaneous.
- In peripheral nystagmus the fast component is always in the same direction regardless of where the patient looks. However, the nystagmus is always worse when looking in the direction of the fast component. (Unfortunately, rarely patients with a very focal brain stem stroke may have unidirectional nystagmus.)

#### Central Nystagmus

- Central nystagmus may be purely horizontal or purely vertical or purely rotatory.
- It may mimic peripheral nystagmus – this means that rarely central nystagmus may be unidirectional and falsely appear like the nystagmus of a peripheral lesion.
- Bidirectional or multi-directional nystagmus is virtually always central. Central brain diseases include more than just strokes.
- Central nystagmus is also caused by MS, alcohol toxicity, drugs such as dilantin, and Wernicke's Syndrome. As well, vestibular migraine may produce a central vertigo.
- Purely vertical nystagmus, especially if down-beating, is central. (However, do recall that the nystagmus of BPPV is vertical-rotatory and upward beating)

- Therefore: the only absolutely central nystagmus is multi-directional, purely vertical and down-beating, or purely rotational.

### **Warnings and Pitfalls and Misconceptions:**

It is a dangerous myth to believe that unidirectional nystagmus always rules out a central cause of vertigo. While unidirectional nystagmus is usually peripheral, a highly focal stroke may occasionally produce unilateral nystagmus and mimic a peripheral vestibular problem. This patient may be sent home only to return with a stroke later. However, multidirectional nystagmus is always central. Therefore, the nystagmus must also be considered with other important factors such as gait, test of skew (see 4 below) and the head impulse test (see 5 below).

### **4. Skew**

Skew refers to vertical misalignment of the eyes. It is detected by the alternating cover test. The examining physician looks at the eye which is being uncovered. If there is a skew, the eye deviates vertically and is seen to correct when it is uncovered. This is always of sign of central nervous system dysfunction.

### **5. HTT or HIT**

The head thrust test or head impulse test was described in 1988 by Halmagyi. It is a test of the vestibular-ocular reflex. If a patient looks at the examiner's nose and the patient's head is quickly rotated 20 degrees, the normal response is for the eyes to remain fixed on the examiner's nose. This means that the vestibular-ocular reflex is normal. In vestibular neuritis, the vestibular nerve is dysfunctional. Therefore, the eye will tend to travel with the head and not remain fixed on the examiner's nose. When the patient realizes this [usually instantaneously], the eyes correct and look back at the nose. This is seen as a corrective saccade. This test is ONLY USEFUL when a patient is having continuous vertigo and nystagmus. An abnormal test suggests that the disease is vestibular neuritis because the damaged vestibular nerve prevents a normal response.

In patients with vertigo due to strokes, the vestibular-ocular reflex remains intact [almost always] and, as odd as it sounds, the HIT will be normal. Therefore, a normal HIT [only performed in patients with continuous vertigo and nystagmus], suggests a stroke as the cause of the vertigo.

The HIT has no role in assessing vertigo that is not constant and is not accompanied with nystagmus. In these patients the HIT will be normal and the vertiginous patient will be incorrectly diagnosed to be suffering from central vertigo.

## 6. HINTS

Hints refers to the examination using points 3, 4, and 5 above. “HI” for head impulse, “N” for nystagmus, and “TS” for test of skew. In patients with continuous vertigo and with concurrent nystagmus, the presence on any one of these [normal HIT, central nystagmus, or abnormal test of skew] suggests that the patient’s vertigo is due to a stroke and not vestibular neuritis.

These two links are to Dr. Newman-Toker’s site describing beautifully the HINTS tests that he has extensively written about in multiple articles.

<http://content.lib.utah.edu/cdm/ref/collection/ehsl-dent/id/6> or  
<http://www.kaltura.com/tiny/2jsvu>

### Excellent Articles

1. Tarnutzer MD et al. Does my dizzy patient have a stroke? CMAJ 2011;183(9):E571-E591.
2. Lee H. Isolated Vascular Vertigo. Journal of Stroke 2014;16(3):124-130.
3. Edlow J et al. Using The Physical Examination to Diagnose Patients With Acute Dizziness And Vertigo. The Journal of Emergency Medicine 2016;50(4):617-628.
4. Nelson JA et al. The Clinical Differentiation of Cerebellar Infarction from Common Vertigo Syndromes. Western Journal of Emergency Medicine 2009;10(4):273-277
5. Newman-Toker D et al. Normal head impulse test differentiates acute cerebellar strokes from vestibular neuritis. Neurology 2008;70:2378-2385.
6. Kattah JC et al. HINTS to Diagnose Stroke in the Acute Vestibular Syndrome. Stroke 2009;40:3504-3510.
7. Baloh R. Vestibular Neuritis. N Eng J Med 2003;348:1027-32.
8. Hotson J et al. Acute Vestibular Syndrome. N Eng Med 1998;339:680-685.
9. Kim J et al. Benign Positional Vertigo. N Eng J Med 2014;370(12):1138-1147.
10. Parnes L et al. Diagnosis and management of benign paroxysmal positional vertigo (BPPV). CMAJ 2003;169(7):681-693.
11. Robertson J et al. Cervical Artery Dissections: Journal of Emergency Medicine 2016;51(5):508-518.



12. Chase M ete al. ED patients with vertigo: can we identify clinical factors associated with acute stroke. The Americian Journal of Emergency Medicine 2012;30:587-591.