

Dizziness Update: a new approach and treatment based on triage, timing and triggers.

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Abstract

Introduction: In 1972, following a classic study of 125 patients, Drachman and Hart proposed a classification of dizziness based on the patient's response to "What do you mean, dizzy?" The patient's response is used to classify the stated dizziness experience into 4 categories of dizziness patterns: vertigo, presyncope, imbalance and light-headedness. Over time, this model resulted in misdiagnosis.

Methodology: Two research questions are posed in this update and answers sought through current literature review in PubMed. (1) What is the evidence the Drachman-Hart model of enquiry is the wrong paradigm? (2) What new dizziness classification should be put in its place?

Results: A study by Newman-Toker showed that in 1,342 consecutive dizziness cases seen in ED, 52% of patients were "unclear, inconsistent, and unreliable", casting doubt on the validity of the Drachman-Hart approach. Furthermore, a cross-sectional study of 9,472 dizziness cases seen in ED showed that otologic/vestibular causes accounted for only 32.9% of cases. In 2015, Edlow&Newman-Toker published a new approach to dizziness based on timing and triggers of the dizziness experience in the patient. A battery of 3 tests, targeted examination, and additional tests when serious causes are suspected, allowed differentiation of dizziness into acute vestibular syndrome, triggered episodic syndrome, spontaneous episodic syndrome, and chronic vestibular syndrome. This approach uses current best evidence to differentiate between peripheral and central lesions and offers the potential to reduce misdiagnosis.

Conclusions: The triage, timing and triggers approach described by Edlow and Newman-Toker is valid and should be adopted widely.

Keywords: Acute vestibular syndrome, Triggered-episodic vestibular syndrome, Spontaneous-episodic vestibular syndrome, Chronic vestibular syndrome, Vestibular migraine, Persistent postural-perceptual dizziness, Falls in the elderly.

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Introduction

Dizziness is a common symptom encountered in emergency departments (ED), general practice, and specialist outpatient clinics [1]. It is defined in the National Library of Congress MESH Term as an "imprecise term which may refer to a sense of spatial disorientation, motion of the environment, or lightheadedness" [2].

In this Update, the approaches to a patient who complains of dizziness are reviewed. Following a classic study of 125 patients, Drachman DA and Hart CW published a classification of dizziness 1972 [3]. This classification was based on the patient's answer to "What do you mean, dizzy?" and 4 categories of giddiness patterns were recognised: vertigo, presyncope, imbalance, and light-headedness [4]. For benign paroxysmal positional vertigo, there were attempts to differentiate between peripheral and central causes (Table 1).

The need for a new approach was noted by Jonathan Edlow, David Newman-Toker, and others. In a Commentary in the Academic Emergency Medicine in 2013, Edlow declared - "Diagnosing Dizziness: We Are Teaching the Wrong Paradigm!" [5].

Two research questions are posed by the author in this Update and the answers sought through a review of current literature in PubMed:

Q1 - What is the evidence the Drachman-Hart model of enquiry is the wrong paradigm?

Q2 - What new dizziness classification should be put in its place?

Methodology

PubMed searches were made using the key words of "dizziness", "update", "new approach", "recent advances", "review", "evidence", and "clinical practice guideline". Of 283 papers shortlisted 16 papers provided the evidence for this Update.

Results

The answers from a literature review of 16 selected papers provide the answers to the two research questions posed by the author.

Evidence of wrong classification paradigm

Q1 - What is the evidence the Drachman-Hart model of enquiry is the wrong paradigm?

The Drachman-Hart dizziness classification model is based on the dizzy patient's response to the question of "what do you mean, dizzy?" Based on the response the patient is deemed to have "vertigo", "near faint", "imbalance", or "lightheadedness". This then determines the differential diagnosis [3].

Table 1. Causes of dizziness-classified by symptom quality (Drachman & Hart, 1972).

Type of dizziness	Differential diagnosis
VERTIGO -- Rotational sensation	<ul style="list-style-type: none"> Peripheral causes – BPPV; Vestibular neuritis; Meniere’s disease; Motion sickness; Drug use; Local ear dysfunction Central causes – Brain stem ischaemia; Cerebellar ischaemia; Acoustic neuromas; Multiple sclerosis; Basilar artery migraine; Temporal lobe seizure; Posterior fossa tumor.
PRESYNCOPE -- Perception of impending faint	<ul style="list-style-type: none"> Cardiovascular causes – Arrhythmias; vasovagal reflex; Orthostatic hypotension; Aortic stenosis; hypertrophic cardiomyopathy; Low cardiac output states; Carotid sinus hypersensitivity Anaemia; hypoglycemia; hypoxemia
DISEQUILIBRIUM -- Loss of balance on ambulation	<ul style="list-style-type: none"> Multiple sensory deficits; altered visual input Primary disequilibrium of ageing (presbyastasis) Cerebellar disease; Frontal lobe apraxia; Parkinsonism Drug use
LIGHTHEADEDNESS – vague sensation; giddiness	<ul style="list-style-type: none"> Anxiety; depression; panic disorders Hyperventilation

Source: Warner et al (1992)[4]

Historically, this dizziness classification model was based on a study of 125 patients over 2 years and published in 1972. By today’s clinical research methodology, this study had serious flaws [6]:

- Patients were placed into 1 of 4 categories of vertigo, presyncope, imbalance, and light-headedness based on the patient’s description of his or her dizziness experience which subsequent research showed to be “unclear, inconsistent, and unreliable [7]. The details are described under Study 1.
- No independent verification and blinding were done.
- Small sample size, namely only 125 patients were enrolled; 23 patients (25.6%) were excluded due to inadequate data in 12 patients (16.8%), uncertain diagnosis in 9 patients (7.2%), and “inappropriate referrals” in 2 patients.
- Selection biases namely, the patients had to return on 4 different days for testing, and subjects had to be fluent in English. Those who could do so were not studied.
- No long term follow up of patients to verify diagnosis.
- Lack of modern imaging so diagnosis made were open to misdiagnosis, and
- Lack of some diagnosis e.g. vestibular migraine, and posterior circulation TIA presenting as isolated dizziness. These conditions were not yet recognized in 1972.

Two studies conducted in the ED setting demonstrated conclusively that the Drachman-Hart dizziness approach resulted in some wrong diagnosis.

- Study 1: Imprecise patient reports of dizziness symptom quality [7]. A case study of 1,342 consecutive patients with dizziness at 2 urban academic emergency departments published in 2007 showed that 52% of patient descriptions of the quality of dizziness were “unclear, inconsistent, and unreliable”, casting doubt on the validity of the traditional approach to the patient with dizziness. By contrast, patient reports of dizziness duration and triggers were “clear, consistent, and reliable”.
- Study 2: Spectrum of dizziness cases seen [8]. A large cross-sectional study of 9,472 dizziness cases seen in Emergency Department visits published in 2008 showed that– otologic/ vestibular causes accounted for only 32.9% of cases. The causes of the rest were: “cardiovascular (21.1%), respiratory (11.5%),

neurologic (11.2%), metabolic (11.0%), injury/poisoning (10.6%), psychiatric (7.2%), digestive (7.0%), genitourinary (5.1%), and infectious (2.9%)”.

New dizziness classification model

Q2 - What new dizziness classification should be put in its place?

A new approach to dizziness assessment that pays attention to symptom Triage, Timing, and Triggers in the history, Targeted Examination; and laboratory or image testing was proposed in 2013 by Edlow and Newman-Toker [9]. In this new approach, 4 syndromes of dizziness are recognized, namely,

- Acute vestibular syndrome (AVS)
- Triggered episodic vestibular syndrome (t-EVS)
- Spontaneous vestibular syndrome (s-EVS), abd
- Chronic vestibular syndrome (CVS).

Table 2 shows the new classification.

Details of the new classification and assessment

1-Acute vestibular syndrome (AVS): This is dizziness that lasts longer than 24 hours. They are mostly due to vestibular neuritis/labyrinthitis and these are benign. Some are due to posterior circulation stroke and are serious. A targeted examination called the HINTS examination with a battery 3 bedside tests helps to differentiate the benign from the serious (Table 3). HINTS examination stands for Head Impulse test, Nystagmus test, and Test of Skew [8].

- Head Impulse test-For the head impulse test, the patient sits opposite the examiner and fixes his/her gaze on nose examiner. The patient is not told in what direction the examiner will move the patient’s head. The examiner then moves the head in one direction, followed by a move to the centre (midline) and then move to the opposite direction.” [10].
- Nystagmus test-Frenzel goggles are used to visualize nystagmus. The glasses (+20 dioptres) prevent any fixation and magnify the eyes for better visualization. The patient is asked to look to the left, right and centre position [10].
- Test of Skew-“The eyes of the patient are fixed on a distant target the eyes of the patient are covered and uncovered in a slow alternating manner. A vertical misalignment of the eyes is frequently seen in central lesions” [10].

Table 2. New classification of dizziness.

Type of Dizziness	Common Causes
1-Acute vestibular syndrome (AVS) -- Lasts longer than 24 hours	<ul style="list-style-type: none"> • Benign: Vestibular neuritis/labyrinthitis (mostly) • Serious: Posterior circulation stroke (Some)
2-Triggered episodic vestibular syndrome (t-EVS) -- Lasts seconds to a few minutes; obligate, immediate trigger ^(a) is present	<ul style="list-style-type: none"> • Benign: BPPV (most common vestibular cause) • Serious: Orthostatic hypotension and Central (Neurologic) Paroxysmal Positional Vertigo (CPPV)
3-Spontaneous episodic vestibular syndrome (s-EVS) -- lasts from seconds to days, with the majority lasting minutes to hours. Not provoked at the bedside	<ul style="list-style-type: none"> • Benign: Vestibular migraine (Most common cause) Others: vasovagal syncope, panic attacks, Meniere's disease • Serious: Posterior circulation TIA; cardiovascular, endocrine, toxic causes
4-Chronic vestibular syndrome (CVS)	<ul style="list-style-type: none"> • Benign: medication side effects, Anxiety, Depression • Serious: Slow growing posterior fossa masses

Sources: (a) Edlow, 2106 [6]; (b) Spiegel et al, 2017 [10]

Table 3. Battery of 3 tests in assessing dizziness.

Step approach	Action
1-Triage	Identify obvious dangerous causes by the presence of prominent associated symptoms, abnormal vital signs, altered mental states, or test results.
2-Timing	Narrow the differential diagnosis by classifying the dizziness attack pattern as episodic, acute, or chronic in duration in the history of present illness.
3-Triggers	Seek an underlying pathophysiologic mechanism by searching for obvious triggers (e.g. positional) or exposures (e.g. trauma) in the review of systems.
4-Targetted examination (See Table 4 for interpretation of Acute vestibular syndrome oculomotor findings)	Do a battery of three bedside tests – head impulse, nystagmus, and test of skew test. [See Edlow, 2016 for change of order of test and reason; also do a general neurologic examination, focusing on cranial nerves, including hearing, cerebellar testing, long tract signs, gait testing]
5-Test	Choose the best laboratory or imaging test when there is clinically relevant residual uncertainty about a dangerous cause that has not been ruled out.

Source: Newman-Toker & Edlow, 2015 [9]; Edlow, 2016 [6]
Footnote: TITRATE acronym stands for Timing, TRiggers, And Targetted Exams

Edlow made 2 noteworthy points on the order of testing [6]. Firstly, he proposed that instead of head impulse test first, do the nystagmus test first because it is less intrusive and also the nystagmus helps to anchor and inform the rest of the examination process. Secondly, a general neurologic examination, focusing on cranial nerves, hearing, cerebellar testing, and long tract signs, as well as gait testing should be carried out.

Acute vestibular syndrome can be due to central lesions. Cerebellar stroke is the most common cause here. Downbeat nystagmus is present in about 50% of cerebellar strokes. The presence of headache, diplopia, or abnormal cranial nerve, or cerebellar dysfunction will help in pointing to the likelihood of a stroke as the underlying cause (Table 4).

Acute vestibular syndrome (AVS) can be due to peripheral causes:

- Vestibular neuritis (also referred to as vestibular neuronitis). This is an acute peripheral vestibular process most commonly due to reactivation of latent herpes simplex virus in the vestibular ganglion. It causes only unilateral vestibular dysfunction but does not affect hearing [11].
- Labyrinthitis. This usually attributed to a viral infection. It causes both acute unilateral loss of both hearing and vestibular function [12].

2-Triggered episodic vestibular syndrome (t-EVS): This can last from seconds to a few minutes. An obligate trigger is needed such that each time the specific trigger occurs, the dizziness follows. Common triggers are head motion or change in body position (e.g., arising from a seated or lying position, tipping the head back in the shower to wash one's hair, or rolling over in bed) [6].

A common and benign cause is (BPPV) and as its name suggests

is not a serious threat to life except in the elderly who may fall [10]. The Dix-Hallpike maneuver is used to confirm the diagnosis of BPPV. The majority of BPPV are idiopathic. Known causal factors are prolonged bed rest, cervical hyperextension, osteoporosis, age and migraine. The most commonly involved semicircular canals are the posterior ones (90%) [13].

Serious causes of t-EVS are orthostatic hypotension and central paroxysmal, positional vertigo (CPPV). Serious causes of orthostatic hypotension are fluid loss (bleeding, vomiting, diarrhea, excessive urination/sweating) or occult bleeding. CPPV can be due to posterior fossa lesions like neoplasm infarction, haemorrhage, cerebellar degeneration, and Chiari malformation [6]. They can be easily distinguished from one another by history and physical examination [9].

3-Spontaneous episodic vestibular syndrome (s-EVS): This can last from seconds to days, with the majority lasting minutes to hours. Unlike t-EVS, s-EVS episodes cannot be provoked at the bedside. Hence, the diagnosis of s-EVS relies almost entirely on history taking [9].

The most common benign cause of s-EVS causes is vestibular migraine. This condition is an episodic vestibular syndrome of central origin. It is characterized by attacks of either dizziness alone or with stance and gait ataxia, headache, nausea, vomiting and visual sensations. These episodes might last minutes to hours [12].

Other benign causes are vasovagal syncope, panic attacks, and Meniere's disease. Although Meniere's disease is often mentioned as a common cause of s-EVS, its estimated population prevalence (0.1%) is actually less frequent compared to the other three episodic disorders of vestibular migraine, vasovagal syncope, and panic attacks mentioned earlier. Meniere's disease

Table 4. Acute vestibular syndrome oculomotor physical findings.

Oculomotor Examination Component	Peripheral lesion (Usually Vestibular Neuritis)	Central lesion (Usually Posterior Circulation Stroke)
Nystagmus test (neutral gaze and gaze to right and left)	Dominantly horizontal, direction-fixed, beating away from the affected side	Direction-changing horizontal or dominantly vertical and/or torsional, then central (often mimics peripheral lesion)
Test if skew (alternate cover test)	Normal vertical eye alignment (ie, no skew deviation)	Often mimics peripheral lesion; if skew deviation is present, then central lesion is present
Head impulse test (HIT)	Unilaterally abnormal toward affected side (presence of a corrective saccade)	Usually bilaterally normal (no corrective saccade)

Source: Edlow, 2016[6]

is characterized by recurrent attacks of dizziness, hearing loss, sensation of ear pressure and tinnitus, probably due to an endolymph hydrops (over-production, or under-resorption of endolymph) [13].

Serious causes of s-EVS not to be missed are posterior circulation TIA, cardiorespiratory causes (cardiac arrhythmia, unstable angina, pulmonary embolus), and endocrine (hypoglycemia). Temporary or intermittent carbon monoxide exposure is a rare serious cause [9].

4-Chronic vestibular syndrome: This syndrome consists of chronic dizziness that lasts for months to years. Benign causes are medication side effects, anxiety, depression, poorly compensated unilateral vestibulopathy, and chronic bilateral vestibulopathy.

Serious causes are cerebellar degeneration, and slowly growing posterior cranial fossa neoplasms [10]. Then there is cervical vertigo causes secondary to injuries to cervical spine, or Arnold Chiari malformation [10].

Finally, there is also a new chronic vestibular syndrome called persistent postural-perceptual dizziness (PPPD) which cannot be classified as peripheral or central origin [10,12].

5-Falls due to BPPV in older adults: A study on positional vertigo in a falls service in Newcastle in UK of 850 patients complaining of dizziness, 123 (14.5%) subjects had positional nystagmus (PN) on Dix-Hallpike or side-lying test (the PN group). Fifty-three (44%) of subjects in the PN group had fallen compared to 28% in the non-PN group (P=0.04). Epley maneuver was successfully done on 110 patients. BPPV is a treatable factor for falls in the elderly and should be looked for in an elderly who falls [11].

Treatment

Treatment of dizziness depends on whether the underlying cause can be treated. If this is not possible, symptomatic supportive treatment can still help.

1-Acute vestibular syndrome (AVS)

In almost any acute vestibular syndrome, symptomatic treatment such as dimenhydrinate orally 50 mg orally 4-6 hours or as a suppository 150 mg once or twice per 24 hours can be prescribed for the first 3 days. Although benzodiazepines can be used, these have an addictive potential and are therefore avoided. Symptomatic treatment should not be taken for more than 3 days because they are believed to inhibit central compensatory processes [13].

Acute labyrinthitis: The presence of hearing loss indicates that there is labyrinthitis and the patient should be referred to an ENT specialist. Methylprednisolone or Prednisolone is used for treatment [13].

Stroke: It is important to recognize that acute vestibular syndrome can be due to central lesions, and cerebellar stroke is the most common cause here. Early recognition and timely referral for neurological opinion and thrombolysis within the window period of 6 hours. It is also encouraging to note that there are cases where patients benefit even beyond 6 hours after symptom onset [13].

2-Triggered episodic vestibular syndrome (t-EVS)

There are benign and serious causes to be treated.

BPPV is benign. The Hallpike test is the diagnostic test for BPPV and Epley maneuver is the curative repositional therapy. Epley maneuver as repositional therapy of the canalith, alone or combined with betahistine is very effective and has a high success rate (86.2%). Betahistine in addition to the Epley maneuver was more effective than Epley maneuver alone or combined with placebo for the control of vertiginous symptoms (p<0.05) [14].

Serious causes of orthostatic hypotension are usually clearcut. Early recognition and timely treatment of underlying causes from bleeding, fluid loss or occult bleeding can be life-saving.

3-Spontaneous episodic vestibular syndrome (s-EVS)

The treatment of 4 common conditions presenting as spontaneous episodic vestibular syndrome are discussed in this section.

Vestibular migraine: The acute episodes can be treated with vestibular suppressant medications e.g. dimenhydrinate 50 mg orally 2 times a day, or meclizine 12.5-50 mg orally 3 or 4 times a day. Migraine-specific medications such as triptans may be effective. Prophylactic oral administration of topiramate 25-100 mg daily or valproate 300-900 mg daily or the sustained form of metoprolol 50-200 mg daily [13] may be considered if quality of life is affected by recurrent episodes.

Meniere's disease: A review by Alcocer et al., [14] showed that in Ménière's disease, use of betahistine demonstrated improvements in the control of vertigo, tinnitus, and hearing loss. "Betahistine 48 mg daily during 3 months is an effective and safe treatment for Ménière's disease and different types of peripheral vertigo."

Cardiac causes: Serious cardiac conditions that cause triggered episodic vestibular syndrome are also causes for spontaneous episodic vestibular syndrome. Early recognition and timely

treatment of underlying causes from bleeding, fluid loss or occult bleeding can be life-saving.

Transient ischaemic attacks: Patients can have transient ischaemic attacks, where patients may have isolated dizziness or other stroke symptoms for less than 24 hours. Also, intermittent isolated vertigo may also be caused by vertebrobasilar insufficiency preceding a stroke [13]. A neurovascular referral may need to be considered if the patient has cerebrovascular risk factors.

4-Chronic vestibular syndrome

Persistent postural-perceptual dizziness (PPPD): This is a newly defined diagnostic syndrome that unifies key features of chronic subjective dizziness, phobic postural vertigo and related disorders [15]. Such patients often develop functional gait disorder, anxiety, avoidance behavior and severe disability. Once recognized, the patient with PPPD can be managed with “effective communication and tailored treatment strategies, vestibular rehabilitation, serotonergic medications and cognitive behavioural therapy.” [15]. Vestibular rehabilitation results in subjective improvement of vertigo (odds ratio 2.67 (95% confidence interval 1.85 to 3.86), 565 participants) [16].

5-Falls in the elderly

BPPV is the most common cause of dizziness in the elderly and falls can occur. Greater awareness of BPPV as a cause of falls in elderly people and the effectiveness of repositioning therapy needs to be emphasized [11].

Discussion

The proposed paradigm shift by Edlow in 2013 of classifying dizziness by triaging the symptoms, noting the timing and triggers had better applicability and missing fewer cases of central lesions for peripheral lesions and vice versa. Physical examination of head impulse, nystagmus, testing the eyes, postural blood pressure, and neurological examination, followed by laboratory and imaging tests when necessary is the new paradigm. Four timing and trigger patterns are recognised in this new approach namely, acute vestibular syndrome (AVS), triggered episodic vestibular syndrome (t-RVS), spontaneous episodic vestibular syndrome (s-EVS), and chronic vestibular syndrome (CVS). See Table 2. Treatment is based on what were the underlying diseases diagnosed. It is to be noted also that in elderly patients who fall, confirming BPPV as a causative factor and treatment with the Epley maneuver is curative.

Drachman and Hart’s model of classifying dizziness by symptom quality alone as the starting point “What do you mean, dizzy?” leads to misdiagnosis simply because dizziness as a label used by the patient to describe his/her symptom is imprecise. This phenomenon was nicely captured in the study by Newman-Toker in 2007 [7]. In the study, the patients were consistent in describing the duration and triggers of the experienced “dizziness” subjective phenomenon. When asked to describe their experience of dizziness subjectively, the patients were “unclear, inconsistent, and unreliable”. Hence, the new model of classification of dizziness which relies on duration and triggers is likely to be more accurate. The limitations are the analysis of the papers retrieved to answer the two research questions in this Update. Also only papers in English in PubMed were used.

Recommendations arising out of this update are:

- Based on papers retrieved and studied, the new approach to diagnosis of dizziness described by Newman-Toker and Edlow [9] is valid and should be adopted widely.
- The older Drachman-Hart dizziness classification should be abandoned.
- In older people, it is important to recognize the presentation of a dizziness problem can be falls. BPPV if present is a treatable factor for falls.
- Large scale collaborative studies across countries in the settings ED, general practice, and specialist outpatient clinics will further provide information on incidence and outcome of this new approach to dizziness.

Conclusions

- A review of current medical literature confirms the new approach to dizziness described by Edlow and Newman-Toker (2015) is valid and should be adopted widely. The Drachman-Hart classification of dizziness should not be used.
- In older people, it is important to recognize the presentation of a dizziness problem can be falls. BPPV if present is a treatable factor for falls.

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