A NEW APPROACH TO THE DIZZY PATIENT

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A New Approach to the Dizzy Patient

The Traditional “What do you mean by dizzy?” Approach

The traditional approach to diagnosing dizziness relies heavily on the premise that dizziness type predicts the underlying etiology. This “quality-of-symptoms” approach suggests that dizziness symptoms should be classified as one of four, mutually-exclusive types based on the nature or quality of dizziness symptoms: (i) vertigo (spinning or motion), (ii) presyncope (impending faint), (iii) disequilibrium (unsteadiness when walking), or (iv) non-specific dizziness (any other dizziness sensation).1 In this approach, the first diagnostic question is “What do you mean by dizzy?” and the response directs subsequent diagnostic inquiry, with vertigo prompting a search for vestibular causes, presyncope a search for cardiovascular causes, disequilibrium a search for neurologic causes, and non-specific dizziness a search for psychiatric or metabolic ones.2 This approach was first articulated in 19723 and continues to appear in high-impact medical journals,4 commonly-used medical texts,5 and internet-based resources.6 Recent studies confirm that this diagnostic method for assessing dizzy patients remains the current standard of clinical practice in frontline care settings such as the emergency department (ED).7;8 However, growing evidence now suggests this approach is fundamentally flawed and could be contributing to misdiagnosis.1

The Triage, Timing, Triggers, & Telltale Signs Approach for the Acutely Dizzy Patient

Evidence now indicates that the quality-of-symptoms approach is neither valid nor reliable.8-12 Best evidence instead suggests that a shift of emphasis in clinical assessment away from dizziness type and towards dizziness timing (e.g., episode duration) and triggers (e.g., changes in head position) will probably yield more accurate and reliable diagnostic results, particularly for patients presenting with new, acute dizziness symptoms.1 A “triage, timing, triggers, & telltale signs” framework offers considerably greater potential to help identify dangerous causes (Table 1), including stroke and TIA, particularly in the emergency department or other primary care settings. The basic structure of this proposed new approach (Figure 1) is as follows:

1. TRIAGE: first identify whether there are obvious clinical “red flags” that immediately point to a more serious cause for dizziness — (a) abnormal vital signs, (b) confusion or otherwise impaired mental state, (c) sudden, severe, or sustained head or neck pain, (d) worrisome neurologic symptoms (e.g., diplopia, dysarthria, dysphagia, etc.), or (e) worrisome cardiovascular symptoms (e.g., chest pain, dyspnea, syncope)
2. TIMING: divide the remaining patients with a chief symptom of dizziness into those whose dizziness was transient or episodic (lasting seconds to hours) and those with persistent or continuous dizziness (lasting days to weeks), limiting the duration-specific differential diagnostic considerations to common, benign causes and their dangerous mimics based on episode duration (along with frequency and total illness duration) (Table 1)
3. TRIGGERS (for patients with transient dizziness <24 hrs): emphasize a search for a clear history of dizziness triggers, using the presence of specific triggers to identify benign or dangerous underlying etiologies;9 in general, transient dizziness that is exertional or spontaneous (un-triggered) is most likely to be caused by dangerous disorders; other triggers most often indicate benign causes (e.g., changes in head position); when possible, use the physical examination to try to reproduce symptoms (e.g., Dix-Hallpike maneuver)
4. TELTTLA SIGNS (for patients with persistent dizziness >24 hrs): emphasize a focused neurological exam, with special attention to excluding the presence of three dangerous oculomotor signs in patients presenting with the acute vestibular syndrome who are at high risk for stroke — normal vestibulo-ocular reflex responses, vertical ocular misalignment, and direction-changing nystagmus (“HINTS” see Acute Vestibular Syndrome).
Table 1. Common causes of acute dizziness and dangerous mimics, by duration

<table>
<thead>
<tr>
<th>Duration*</th>
<th>Common, Benign† Causes</th>
<th>Principal Dangerous Mimics</th>
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<tbody>
<tr>
<td><strong>Seconds to Hours</strong></td>
<td>• benign paroxysmal positional vertigo (BPPV) (sec)</td>
<td>• transient ischemic attack (sec-hrs§)</td>
</tr>
<tr>
<td>(EPISODIC: transient or intermittent)</td>
<td>• benign orthostatic hypotension (e.g., medications) (sec-min)</td>
<td>• cardiac arrhythmia (sec-hrs)</td>
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<tr>
<td></td>
<td>• reflex syncope (sec-min)</td>
<td>• other cardiovascular emergencies (e.g., myocardial ischemia, aortic dissection, pulmonary embolus, occult GI bleeding)</td>
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<tr>
<td></td>
<td>• panic attack (min-hrs)</td>
<td>• neuro-humoral neoplasm (e.g., insulinoma║, pheochromocytoma)</td>
</tr>
<tr>
<td></td>
<td>• Menière disease (sec-dys‡)</td>
<td>• toxic exposure (e.g. carbon monoxide)</td>
</tr>
<tr>
<td></td>
<td>• vestibular migraine (sec-dys‡)</td>
<td></td>
</tr>
<tr>
<td><strong>Days to Weeks</strong></td>
<td>• vestibular neuritis</td>
<td>• brainstem, cerebellar, labyrinthine stroke</td>
</tr>
<tr>
<td>(NON-EPISODIC: persistent or continuous)</td>
<td>• viral labyrinthitis</td>
<td>• bacterial labyrinthitis/mastoiditis</td>
</tr>
<tr>
<td></td>
<td>• drug toxicity (e.g. anticonvulsants)</td>
<td>• Wernicke syndrome</td>
</tr>
<tr>
<td></td>
<td>• herpes zoster oticus</td>
<td>• brainstem encephalitis (e.g. listeria, herpes simplex) or Miller Fisher syndrome</td>
</tr>
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</table>

* Patients with conditions producing dizziness/vertigo lasting seconds to hours are rarely symptomatic at the time of ED assessment. If they are still symptomatic, it is generally with intermittent symptoms triggered by certain actions (e.g. head movement, standing up quickly, etc.). By contrast, patients with conditions producing dizziness/vertigo that lasts for days to weeks are usually symptomatic at the time of initial ED assessment. This clinical distinction is crucial, since the bedside exam findings one expects differ dramatically between the two groups. In the former group, with transient or intermittent symptoms, the physician should seek physical exam findings that provoke symptoms, but should not be surprised to find a completely normal exam – here, often the history offers the only hope to differentiate between common, benign causes and their dangerous mimics. In the latter group, with persistent and continuous symptoms, the physician should expect that the physical exam findings will usually distinguish between benign causes and dangerous causes, and be surprised if they do not.

† Any disease causing dizziness/vertigo can be considered a ‘dangerous’ medical problem if the symptoms tend to occur in dangerous circumstances (e.g. highway driving). Furthermore, the high vagal tone that accompanies some vestibular disorders can provoke bradyarrhythmias in susceptible individuals. Nevertheless, although they may be quite disabling during the acute illness phase, diseases classified here as ‘Common, Benign Causes’ rarely produce severe, irreversible morbidity or mortality (unlike their ‘Dangerous Mimics’ counterparts).

‡ Menière disease episodes may last longer than a day in about 1 in 10 cases13 and vestibular migraine episodes may last longer than a day in about 1 in 4 cases.14 Rigorous data on the duration of symptoms in this subset of Menière disease and vestibular migraine patients are lacking, but clinical experience suggests that only rarely do such patients experience symptoms lasting longer than 48-72 hours.

§ True transient ischemic attacks (TIAs) typically last fewer than 6 hours, and, by clinical definition, last fewer than 24 hours. Beyond that time window, reversible cerebrovascular symptoms have sometimes been referred to as “reversible ischemic neurologic deficits” (RINDs). Experiencing such prolonged symptoms without evidence of infarction (i.e., completed stroke) being seen on modern neuroimaging studies is thought to be exceedingly rare. However, among those with acute vestibular syndrome who arrive promptly, ~10-20% have an initial falsely negative MRI with diffusion-weighted imaging (DWI), out to 48 hours after symptom onset.12,15,16

║ Other causes of hypoglycemia (e.g., excess exogenous insulin) are more common, but also simpler to diagnose
Reference List


