

## A 69-Year-Old Man With Chronic Dizziness

David A. Drachman, MD, Discussant

**DR PARKER:** Mr D is a 69-year-old man troubled by dizziness. He is retired from his job as building superintendent for a public school. He is married, lives in the Boston, Mass, suburbs, and has managed Medicare insurance.

Mr D dates the onset of his dizziness to 1994, when the symptoms were predominantly vertiginous. He described the sensation as “everything is moving” and “like I had too much to drink.” These episodes lasted 1 to 2 hours, occurred several times per week, and often came on suddenly, such as when arising in the middle of the night. At that time, use of meclizine hydrochloride seemed to decrease the symptoms. Results of a magnetic resonance imaging (MRI) scan of the brain at that time were normal.

The symptoms seemed to resolve over time, but in 1996, Mr D fell off a ladder from a height of 1.5 m (5 ft) and struck his head, shoulder, and back. In 1997, the symptoms of dizziness recurred, although Mr D then described a sensation of “light-headedness, as if I will faint.” He complained of a fear of “missing steps” and an “inability to focus.” In 1998, the dizziness became more prominent, with a sensation of “seasickness and having to hold on to the walls.” The episodes lasted for hours to days, were often associated with nausea, and were not necessarily associated with change in position. The dizziness did not respond to meclizine.

Mr D’s medical history is notable for peptic ulcer disease, treated with hemigastrectomy and vagotomy, and complicated by pancreatic phlegmon and ultimately diabetes. He has had renal colic, decreased hearing documented by audiometry, and back pain treated with steroid injections and oral medications. He has mild essential hypertension. He quit smoking cigarettes and drinking alcohol many years ago. There is no relevant family history.

His current medications include oxycodone controlled-release tablets, 40 mg twice daily; gabapentin, 300 mg 8 times daily; atenolol, 50 mg/d; glyburide, 7.5 mg twice daily; and metformin hydrochloride, 500 mg twice daily.

Physical examination revealed a well-appearing man with a blood pressure of 140/80 mm Hg and a heart rate of 80/min without orthostatic changes. Findings from a funduscopic ex-

amination were normal. There was cerumen in the right ear. Carotid pulses were 2+ without bruits. Neurologic examination was notable for normal cranial nerves, motor function, sensory function, and reflexes. The Bárány maneuver did not evoke nystagmus. Rotating him in a chair, however, did bring on a typical sense of dizziness. While he read an eye chart, his visual acuity decreased from 20/40 to 20/100 while shaking his head.

Laboratory results showed normal complete blood cell count and normal electrolyte, serum urea nitrogen, creatinine, glucose, hemoglobin A<sub>1c</sub>, vitamin B<sub>12</sub>, and folate levels. Mr D was recently referred to an otolaryngologist for further evaluation.

### MR D: HIS UNDERSTANDING AND PERCEPTIONS

I started to get dizzy a number of years ago. Each year, it feels like I’m getting worse. At times, I feel like I’m going to pass out. I’m not focused on the room and my eyes do not focus. This feeling makes me sleepy, and I can go to sleep within 2 minutes. It feels similar to spinning. My wife will turn to me at times and say, “Stop doing that two-step.” I try to put the foot out front and it doesn’t go there, so I have to do a two-step to bring myself back up to balance again.

I never know when I’m going to have it. Therefore, I plan my day accordingly. I have grandchildren. Do I dare drive only 35 minutes from my home to pick them up and bring them over? No. Because I’m not sure whether I will have the dizziness when I get there and have children in the car with me. I’m not willing to take that chance.

### DR M: HIS UNDERSTANDING AND PERCEPTIONS

Several years ago, Mr D began to complain of feeling dizzy, which he described as spinning and feeling as though he were drunk or seasick. Sometimes it would come on while he was getting up at night to go to the bathroom. But other times it would come on without any specific change in position. It would last for several hours and at times be debilitating. Initially it responded to meclizine. It went away for months at a time, and then it would come back. Years later it seemed not to respond to meclizine.

Right now he feels very vulnerable to this symptom. He feels that it can come on at any time. He’s frightened by it. If

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he's driving his car, he feels impaired. So there are 2 problems. There's the way it makes him feel, which is poor, and there is his fear that the dizziness will come on and impair his ability to drive or do what he needs to do.

My question for Dr Drachman is how to approach a patient like this whose symptoms are not all in 1 category. He has symptoms that are typically vertiginous, but he also has some other symptoms that are a little more vague (a light-headed feeling, a spacy feeling) that don't sound like vertigo. How do I figure out what's causing what? How do I sort out the adverse effects of his medications and decide whether they might somehow be interacting with his symptoms?

#### AT THE CROSSROADS: QUESTIONS TO DR DRACHMAN

What is the differential diagnosis for dizziness? What are the key questions to ask in the history? What are the useful aspects of the physical examination? What studies are indicated for which patients, and who should order them? What treatment options exist, including conventional and alternative medicine, rehabilitation, and positioning maneuvers? What is the prognosis for different types of dizziness? What are your recommendations for Mr D?

**DR DRACHMAN:** Listening to Mr D's history, it is easy to understand why the complaint of dizziness is so often dismaying to physicians. Mr D describes his sensations of dizziness in many different and conflicting ways. His symptoms have occurred intermittently over 4 years, with varying frequency and severity and no clear relation to triggering events. Sometimes meclizine helped and sometimes it did not. Occasionally the episodes were related to position change, but more frequently, they were not. Dizziness is not his only medical problem—he has diabetes, surgically treated peptic ulcer disease, renal colic, back pain, and pancreatic disease, and he takes several different medications.

The apparent vagueness and variability of Mr D's symptoms and the multiplicity of his other problems make it difficult to know what part(s) of his body we should examine to find the culprit structure(s) and what disease processes or conditions might cause Mr D's dizziness. Yet, in my experience, Mr D is not an unusual dizzy patient; his problem is no more complicated than many that are seen in everyday practice.

To approach this problem, we need to know the physiologic basis for the complaint of dizziness, the types of symptoms that may present as dizziness, the range of diseases that may cause dizziness, a clinical strategy to enable us to evaluate each case, and the treatments that are useful for each etiologic or physiological cause of dizziness.

#### The Physiological Basis of Dizziness

Normally, spatial orientation (knowledge of the position of one's body in space, its relation to the surrounding environment, and appropriate motion) is assessed by continual sensory monitoring, of which we are largely unaware.<sup>1,2</sup> Five sensory modalities sample our position and motion: vision, vestibular sensation, proprioception, touch and pressure sensation, and hearing, in descending order of importance. Normally, the input from these channels is integrated effortlessly by the brain, giving us a comprehensive and somewhat redundant image of our position and motion in space.<sup>3</sup> This enables us to maintain balance, move about, and interact with objects around us.

When our "orienting image" is unreliable, we become uncertain of our position or motion in space, and this is when we

complain of *dizziness*.<sup>1</sup> This can occur physiologically, when the limits of accurate sensory perception are exceeded (eg, on a roller coaster in an amusement park). It occurs pathologically when sensory organs produce spontaneous false, distorted, or inadequate information, or when there is a "mismatch" or discrepancy between senses. Dizziness also occurs when central integration is slow or inaccurate. Abnormal motor function can contribute as well because spatial orientation is a so-called sampled data system, in which perception, integration, motor response, and feedback recur rapidly and repeatedly—orienting, changing position, then reorienting. Orthopedic or neurologic impairment of motor performance (eg, when walking, turning, or reaching) can produce unexpected responses, disturbing the reliable monitoring of motion and adding to the sensation of dizziness.

#### Different Symptoms Resulting in the Complaint of Dizziness

Uncertainty of position or motion is an unfamiliar sensation, so patients experiencing any of several disparate sensations may complain of dizziness, often with limited ability to be more explicit. The notion, common among both patients and physicians, that everyone who is truly dizzy must have vertigo (ie, a rotational sensation) is probably the most common misconception leading to diagnostic error. In an initial study of 125 dizzy patients<sup>4</sup> and subsequent clinical experience with many more,<sup>1</sup> we have found that most patients' descriptions of their dizziness fall into 1 of 4 types of complaints. Knowledge of these 4 types can help clinicians guide their patients in analyzing and articulating the symptoms they are experiencing.

- Type 1 dizziness is vertigo,<sup>1,5</sup> a definite rotational sensation in which the patient feels that either he/she or the environment is rotating. It often begins instantaneously, is episodic, and, when severe, is typically accompanied by nausea, vomiting, and a staggering gait. Spontaneous oscillopsia, the unprovoked illusion of unidirectional or rotational motion of the environment, may occur.<sup>6,7</sup> When pronounced, vertigo is easily recognized and described; when milder, it may appear to be a rocking sensation or light-headedness. True vertigo is almost always due to a disorder of the peripheral labyrinth or its central connections.

- Type 2 dizziness is presyncope, a sensation of impending faint or loss of consciousness,<sup>8</sup> often beginning with dim vision and roaring in the ears. Presyncope usually implies an inadequate supply of blood and/or nutrients to the entire brain and is not a feature of focal cerebral ischemia. When it is of cardiovascular origin, it may be abrupt in onset and can occur in any position; when due to orthostasis, it occurs only when upright. Type 2 dizziness of gradual onset, persisting when recumbent, suggests a cerebral metabolic disorder, such as hypoglycemia.

- Type 3 dizziness is disequilibrium, with impaired balance and gait in the absence of any abnormal head sensation. Patients sometimes refer to this as "dizziness in the feet," and it is often due to impaired motor control.

- Type 4 dizziness is vague light-headedness other than vertigo, presyncope, or disequilibrium. Patients may use other terms, such as "heavy-headedness" or "wooziness," for this less well-defined symptom. Type 4 dizziness is a category used to include symptoms that cannot be reliably identified as types 1, 2, or 3; it may be due to fractional forms of the disorders that typically produce the more characteristic complaints when

full-blown. Type 4 dizziness also occurs with psychiatric disorders, hyperventilation syndrome, encephalopathies, and multisensory dizziness, as well as many other conditions not causing the first 3 types.

From Mr D's history, he appears to have had 2 complaints of dizziness: type 1 (vertigo) initially, and type 4 (light-headedness) currently.

### The Range of Diseases Presenting With Dizziness

Dizziness is an extremely common symptom, accounting for 2.4% of visits to physicians<sup>9</sup> and innumerable episodes that never reach medical attention. In a survey of more than 2000 people aged 18 to 64 years, more than 20% had experienced dizziness during the past month and of these, 30% had had dizziness for 5 years.<sup>10</sup> Half the patients thought their dizziness was a handicap, but only a fourth of these more severely affected respondents had received any medical attention. In a questionnaire to nearly 1000 people older than 65 years, dizziness was reported by 30%.<sup>11</sup>

More than 60 disorders may result in the complaint of dizziness, either as the primary symptom or as an important and often disabling aspect of another underlying disease. Disorders of the vestibular labyrinth, central and peripheral nervous systems, emotional state, eyes, heart, peripheral vascular system, lungs, kidneys, hematologic system, and joints of the cervical spine and lower extremities may all contribute to dizziness. In addition, many (perhaps most) drugs may cause dizziness as an adverse effect. In an informal survey of 20 oral medications randomly selected from the *Physicians' Desk Reference*,<sup>12</sup> 18 listed dizziness as a potential adverse effect, 10 as among those most frequently experienced.

Despite this large array of possible disorders, more than 90% of identified causes of dizziness fall within 7 common areas<sup>1,4</sup> (Table 1). A significant number of patients (we estimated at least 1 patient in 8<sup>1,4</sup>; others believe even more<sup>13</sup>) have 2 or more separate causes of dizziness, while in about 10%, no definite etiology can be established.

### Diagnosis of Dizziness: A Clinical Strategy

To diagnose dizziness reliably, the physician must know which historical evidence, physical findings, and laboratory results identify each of these conditions.

**History.**—Four complaint-specific questions usually narrow the diagnostic possibilities considerably.

1. What type of dizziness does the patient have? As noted, identifying the 4 types helps focus the evaluation.

2. How old is the patient? The probability of many disorders is related to the patient's age: multiple sclerosis, panic states, and hyperventilation present in the young, while stroke, multisensory dizziness, parkinsonism, and other neurodegenerative diseases appear in elderly patients. Vestibular disorders can occur at any age, however.

3. What is the relation of dizziness to position or motion? Orthostatic hypotension occurs only in the upright position and benign paroxysmal positional vertigo (BPPV, the most common vestibular disorder producing vertigo<sup>14</sup>) occurs on rapid position change. Multisensory dizziness occurs only when the patient is walking or turning and is absent when seated or still.<sup>1</sup> Some conditions are unrelated to position or motion, such as cardiac arrhythmias or episodes of Ménière syndrome.

4. What is the course of the dizziness? Abrupt episodes of vertigo, uniquely triggered by position change and lasting less

Table 1.—Causes of Dizziness: Major Etiologies\*

1. Peripheral vestibular disorders
2. Hyperventilation syndrome
3. Multisensory dizziness
4. Psychiatric disorders (panic, agoraphobia, depression, anxiety)
5. Brainstem cerebrovascular accident
6. Neurologic disorder (multiple sclerosis, parkinsonism, other)
7. Cardiovascular disorder

\*Approximately 12% of patients have more than 1 etiology. Data are from Drachman<sup>1</sup> and Drachman and Hart.<sup>4</sup>

than a minute, are characteristic of BPPV. Cardiac arrhythmias are also typically of sudden onset. Gradual, vague onset and prolonged duration of dizziness is more typical of psychiatric disorders, such as depression or anxiety, or the late residuals of peripheral vestibulopathies.

Disease-specific questions, such as the presence of tinnitus and hearing loss in association with vertigo in Ménière syndrome or the occurrence of cranial nerve deficits with a brainstem stroke, are important as well.

Returning to Mr D, he *initially* may have experienced a definite rotational sensation, with dizziness occurring in self-limited episodes. The nausea accompanying his early dizziness might support a vestibular problem, but in this patient, both gastrointestinal tract problems and his medications could confuse the issue. There were no "neighborhood" neurologic problems to suggest a brainstem ischemic syndrome; the 4-year history with frequent recurrences, the negative MRI findings (a test that I uncommonly obtain in patients with isolated dizziness), and the duration of episodes of hours rather than minutes are against this possibility.<sup>15,16</sup> Although his hearing is reportedly impaired on audiometry, we have no evidence for a pattern of hearing loss characteristic of Ménière syndrome<sup>17</sup> (hearing loss fluctuating over time, with both low- and high-frequency impairment: the "peak audiogram"<sup>18</sup>), and there is no mention of tinnitus, a feature needed for that diagnosis.

Mr D's current complaint of dizziness is not vertigo, however. He is tired, unfocused, and concerned that he may "pass out." He now denies a rotational sensation and describes vague light-headedness. Episodes last for hours at a time and are unrelated to position or motion. They occur when he is driving or in a shopping center, a characteristic that suggests the syndrome of "jumbling of the panorama," often seen in dizziness due to impaired central integration. Finally, Mr D's dizziness interferes with his ability to plan even modest trips—a secondary agoraphobic syndrome<sup>19,20</sup> to which I will return.

**Dizziness Simulation Battery.**—Many patients like Mr D have difficulty describing their symptoms of dizziness with precision. I have found the Dizziness Simulation Battery, consisting of 8 maneuvers (Table 2), to be one of the most valuable ways to identify the patient's complaint. Each maneuver produces a specific type of dizziness, and the patient is asked to identify which maneuver most closely reproduces his/her own symptoms. Some of the tests produce dizziness of a particular type in all individuals while others produce dizziness only in patients with a specific underlying problem. Bárány rotation (spinning in a swivel chair 10 times with the head tilted down 30°) stimulates the horizontal semicircular canals, producing vertigo in anyone who retains some vestibular responsiveness. Recognition that this is identical to the patient's complaint clearly identifies the dizziness as type 1, although without specifying the cause. Similarly, the potentiated Valsalva maneuver (squatting for 30 seconds followed by standing and blowing into a sphygmomanometer at 40 mm Hg for 15 sec-

Table 2.—Dizziness Simulation Battery

Cardiovascular
Orthostatic blood pressure testing
Potentiated Valsalva maneuver
Carotid sinus stimulation
Vestibular
Nylèn-Báràny (Dix-Hallpike) maneuver
Báràny rotation
Multisensory
Walk and turn
Seated head turn ("airplane follow")
Psychiatric
Hyperventilation (30 s)

Table 3.—Neurologic Patterns in Patients With Dizziness

Multisensory dizziness
Brainstem lesion (eg, cerebrovascular accident)
Multiple sclerosis
Parkinsonism
Cerebellar ataxia
Migraine syndrome
Cerebellopontine angle tumor
Bruns apraxia
Astasia-abasia

onds, or straining against a closed glottis) produces a syncopal-like sensation (type 2) in many healthy individuals by reducing the return of venous blood to the heart.<sup>21</sup>

In contrast, the head-hanging positioning maneuver (Nylèn-Báràny or Dix-Hallpike<sup>22</sup>) produces vertigo only in patients with positioning vertigo, most commonly BPPV. In this condition, vertigo develops after a brief delay (5-10 seconds), lasts less than a minute, and is accompanied by direction-fixed rotatory-vertical nystagmus and, often, systemic symptoms such as nausea. This test is usually diagnostic for BPPV rather than other causes of peripheral or central vestibular disorders. Similarly, measurement of blood pressure, first while lying, then while standing, can identify orthostatic hypotension if it shows a significant fall (at least 20-25 mm Hg systolic) and produces symptoms like the patient's complaint.

The battery of 8 maneuvers takes less than 15 minutes to perform in any office setting, requiring only an examining table, swivel chair, and sphygmomanometer. Carotid sinus stimulation and the seated head-turn are infrequently positive, but the observation that only 1 or 2 of the 8 maneuvers exclusively produce dizziness similar to the patient's complaint can provide evidence confirming the type of dizziness.

Mr D identified Báràny rotation-induced vertigo as similar to his initial complaint, supporting the idea that he had initially experienced vertigo. He identified hyperventilation as producing symptoms most like his current complaint. These observations would be more persuasive if he had undergone the complete Dizziness Simulation Battery and had identified these as the only maneuvers that reproduced his symptoms.

**Neurologic Examination.**—A neurologic examination can identify certain specific clinical patterns that may cause dizziness or negative findings that may clarify the diagnosis. A key example is vertigo occurring in the elderly patient with hypertension or diabetes, in whom the question of ischemia in the posterior circulation vs a peripheral vestibular disorder arises.<sup>15,16</sup> Infarction of the peripheral labyrinth as a result of occlusion of the labyrinthine branch of the anterior inferior cerebellar artery (itself part of the basilar-vertebral circulation) usually involves brainstem structures as well. Vertigo resulting from occlusion of other posterior circulation arteries, such as the posterior inferior cerebellar artery, is caused

by infarction of the vestibular nuclei rather than the labyrinth itself. The neurologist's task is to search for neighborhood signs—cranial nerve, long tract, or cerebellar deficits due to involvement of adjacent medullary or pontine structures—indicating a posterior circulation stroke. In their absence, it is likely that the vertigo is due to a nonischemic peripheral vestibulopathy. Table 3 lists some of the more important neurologic patterns that may present with dizziness.<sup>23</sup>

Several special physical signs can be of clinical value in evaluating patients with dizziness, although some are less sensitive or specific and their interpretation often depends on the experience of the examiner.

The potentiated Romberg test (eyes closed, feet in line rather than parallel<sup>5</sup>) evaluates the degree of unsteadiness when visual cues are eliminated. Patients with vestibular and/or proprioceptive problems are more susceptible to losing their balance with their eyes closed.

In the Quix test, the patient points at the examiner's fingers, with arms extended, then closes his/her eyes.<sup>24</sup> A consistent drift to one side indicates an asymmetrical vestibular disorder.

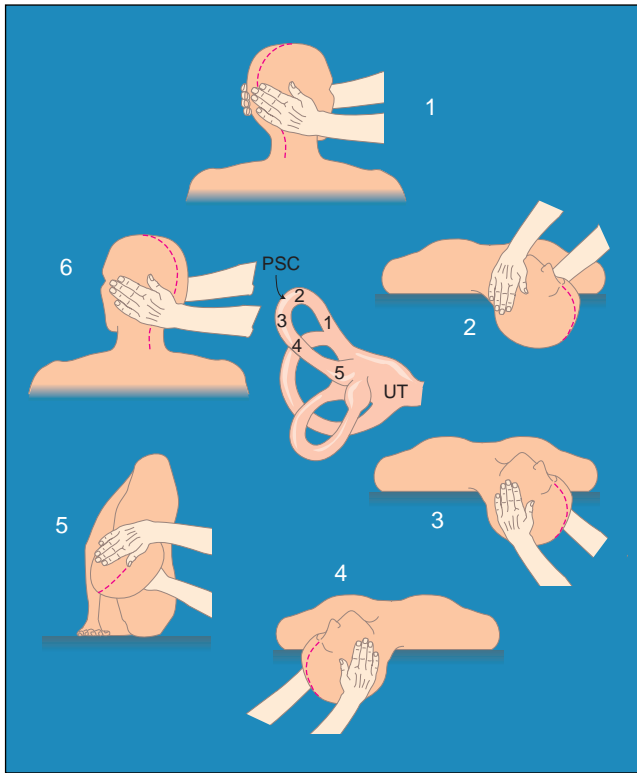
Vestibulo-ocular control is evaluated by testing visual acuity first with both eyes open, then with the head shaking<sup>25</sup> from side to side ("no") at approximately 75 cycles per minute.<sup>26-28</sup> Normally, head shaking should reduce visual acuity by less than 3 lines on a Snellen chart. Mr D lost visual acuity significantly with this test, supporting some vestibular dysfunction.

The head-thrust test<sup>29</sup> is performed by asking the patient to fixate on a target, then rapidly rotating the head 45° to each side. A refixation saccade (ie, during the head turn, the eyes fail to stay on target and must make an additional rapid movement) indicates unilateral canal dysfunction. This test is only moderately sensitive (54%) but highly specific (100%).<sup>29</sup>

The head-shaking test is best performed with fixation eliminated by using Frenzel goggles, +20-diopter magnifying lenses in a frame that illuminates the eyes. Following vigorous shaking of the head from side to side for 15 seconds, nystagmus may be seen if 1 labyrinth is hypofunctioning.<sup>30,31</sup>

**Laboratory Studies.**—In some otherwise healthy, usually young patients, dizziness may be due to a single cause, such as BPPV. In these patients, the workup can be limited to a careful history taking and examination, and if, for example, the vertigo is eliminated by the Epley canalith repositioning maneuver<sup>32,33</sup> (Figure), further testing is unnecessary.

When dizziness is prolonged or occurs in the setting of significant medical disease, I obtain a more complete battery of relevant tests (Table 4). Madlon-Kay<sup>34</sup> found that complete blood cell counts and chest x-ray films each contributed about 3% of the diagnoses of dizziness in a primary care setting. However, the fact that patients may have more than 1 cause of dizziness makes even negative results diagnostically important. I don't regard computed tomography or MRI as routine unless there are neurologic symptoms or signs,<sup>35</sup> major risk factors for cerebrovascular disease, progressive unilateral hearing loss, or central findings on an electronystagmogram.<sup>36</sup> Gizzi et al<sup>37</sup> estimated that approximately 2500 imaging studies would have to be performed on patients with vertigo to detect 1 cerebellopontine angle tumor. As a routine, a neurologic evaluation is often both more informative and cost-effective, typically costing one eighth to one fourth as much as an MRI, and can yield additional clinical insights. I do perform a Minnesota Multiphasic Personality Inventory (MMPI) or similar psychological survey test at the outset in patients with dizziness because it takes no examiner time,



"Epley canalith repositioning maneuver for benign paroxysmal positional vertigo. . ." Treatment maneuver for benign paroxysmal positional vertigo affecting the right ear. The procedure can be reversed for treating the left ear. 1, The patient is seated upright, with the head facing the operator, who is standing on the right. For stability, the patient should grasp the forearm of the operator with both hands. 2, The patient is rapidly moved into the supine position, allowing the head to extend just beyond the end of the examining table, with the right ear downward. 3, The operator moves to the head of the table, repositioning the hands as shown. 4, The head is quickly rotated toward the left, stopping with the right ear upward. Position is maintained for 30 seconds. 5, The patient rolls to the left side, while the examiner rapidly rotates the head leftward until the nose is directed toward the floor. This position is held for 30 seconds. 6, The patient is rapidly lifted into the sitting position, now facing left. The entire sequence should be repeated until no nystagmus can be elicited. The patient must keep the head elevated above 45° for 48 hours following the procedure. PSC indicates posterior semicircular canal; UT, utricle. Reprinted with permission from *Office Practice of Neurology*. Copyright 1996, Churchill Livingstone Inc.

is inexpensive, and gives a generally reliable insight into a range of psychiatric problems. Routinely using this test also avoids awkward explanation if referral to a psychiatrist is recommended after other tests yield negative results. Electronystagmography (ENG) is valuable to identify unilateral or bilateral vestibular deficits (labyrinth, eighth nerve), and distinguish them from central (brainstem, cerebellar) disorders (Table 5).<sup>5</sup> The direction, amplitude, speed, and duration of eye movements are recorded at rest and during a series of eye and head movements. Caloric testing evaluates the integrity and function of the labyrinths and their connections. Modern ENG systems may identify peripheral diseases of the vestibular system, central impairments of the vestibular or oculomotor nuclei, their connections, or supranuclear control mechanisms. Electronystagmography should be performed on virtually all patients with prolonged and/or obscure dizziness to determine whether inapparent impairment of vestibular function is contributing to the patient's spatial disorientation.

Audiometry is especially valuable in the "site of lesion" diagnosis of vestibular disorders and cerebellopontine angle tu-

Table 4.—Laboratory Studies in the Patient With Dizziness

Routine Studies	
Blood:	Complete blood cell count, SMA-12,* thyroid function, serum protein electrophoresis
Cardiac:	Electrocardiogram with rhythm strip
Otological:	Electronystagmogram, audiometry
Psychological:	Minnesota Multiphasic Personality Inventory
Special studies	
	Magnetic resonance imaging and magnetic resonance angiography
	Brainstem auditory evoked response
	Electroencephalogram, 24-h monitor
	Holter monitor, tilt table, electrophysiological study (cardiac conduction)
	5-h glucose tolerance test
	Psychometric evaluation

\*SMA-12 study measures glucose, serum urea nitrogen, uric acid, calcium, phosphorus, total protein, albumin, cholesterol, total bilirubin, alkaline phosphatase, serum glutamic oxaloacetic transaminase, and lactic dehydrogenase.

Table 5.—Common Electronystagmographic Patterns

Normal
Unilateral canal paresis
Positional nystagmus
Spontaneous nystagmus
Bilaterally unresponsive
Central disorder
Ocular movement disorder

mors. In diagnosing Ménière syndrome, finding fluctuating hearing loss involving both low and high frequencies and the phenomena of recruitment and diplacusis is helpful.<sup>16</sup> Office screening can be easily done by whispering 2-digit numerals from each side at a distance of about 2 m (6 ft), with the opposite ear canal blocked. The tuning fork tests (Weber, Rinne) help to evaluate whether hearing loss is due to conductive or sensorineural disorders.

Table 4 lists both routine and special studies that are useful in diagnosis. Magnetic resonance angiography and/or MRI are used when clinical findings suggest a brainstem stroke or cerebellopontine angle tumor; the brainstem auditory evoked response can help localize lesions involving the auditory system; electroencephalography may identify seizure disorders masquerading as dizziness; cardiac evaluations can document arrhythmias or orthostasis; an extended glucose tolerance test can identify reactive hypoglycemic episodes; and psychological evaluation can help identify depression, agoraphobia, anxiety, and panic states.

### Arriving at a Diagnosis

Although there are more than 60 causes of dizziness, they fit into 13 categories, of which only 7 are relatively common (Table 1). A brief (albeit imperfect) algorithm that I have found useful is as follows.

1. Patients with true vertigo have a disorder of the vestibular system. In the absence of neurologic deficits, the cause is most likely a peripheral vestibulopathy. If it occurs only and reliably on position change, it is probably BPPV. With fluctuating hearing loss and sustained tinnitus, it is Ménière syndrome. If neighborhood neurologic symptoms or signs are present, a brainstem disorder (stroke, multiple sclerosis, etc) should be considered.

2. Episodic light-headedness exactly and exclusively reproduced by hyperventilation probably is due to hyperventilation. It often occurs in a setting of anxiety, panic state, or agoraphobia, often in young women.

3. Type 4 dizziness in an elderly person, especially one who has diabetes, is often due to multisensory impairment. The

neurologic findings of hesitant gait (improved by touching the examiner's hand), neuropathy, visual impairment, and vestibular hypofunction help identify this pattern.

4. Psychiatric disorders often manifest as dizziness, constituting about a third of patients.<sup>4,38,39</sup> Agoraphobia<sup>20</sup> and panic disorder,<sup>40</sup> with or without hyperventilation, are commonly translated into the more acceptable complaint of dizziness.<sup>1</sup> Like Mr D, patients with episodic dizziness from other causes may develop secondary agoraphobia, causing them to avoid situations from which they would have difficulty extricating themselves.<sup>38</sup> Anxiety disorders and depression also occur frequently in patients presenting with dizziness. When dizziness interferes with activities in excess of any objective deficits and occurs with the appropriate history and findings on the MMPI or further psychiatric evaluation, this diagnosis is likely.

5. A diagnosis of brainstem stroke is made based on clinical history and the presence of neighborhood brainstem signs in older patients with vascular risk factors and vertigo. Subclavian or vertebral bruits increase the likelihood of cerebrovascular disease. An MRI can be helpful in confirming the diagnosis and magnetic resonance angiography or a duplex scan of vertebral flow can indicate ischemia.

6. Cardiovascular and orthostatic disorders cause type 2 dizziness (faintness) and are diagnosed by finding significant hypotension or an arrhythmia clinically, on appropriate monitoring, or with tilt-table or electrophysiological stimulation cardiac testing.

7. A variety of other neurologic disorders may cause the complaint of dizziness. Parkinsonism, frontal lobe disorders, cerebellar lesions, and spinal cord degeneration may also present as dizziness.

### Mr D's Problem

This brings us back to Mr D, who has several problems causing his dizziness. His initial event may have been *acute and recurrent peripheral vestibulopathy*, a descriptive term for episodic vertigo due to a still-obscurer disorder of the peripheral vestibular apparatus. I prefer this term instead of *labyrinthitis* or *vestibular neuronitis* because we do not know the exact anatomic site of the problem (whether in the peripheral labyrinth or the vestibular nerve) or whether it is an inflammatory disorder. The only viral disease that has been reliably (if rarely) associated with vestibular disorders is herpes zoster.<sup>41</sup>

Mr D no longer has vertigo, but he has residual impairment and imbalance of vestibular function, manifested by a positive Quix test result, loss of 5 lines of visual acuity on head shaking, and abnormal head-thrust test results. In addition, his hearing is impaired. Labyrinths that are hypoactive may lead to disequilibrium or light-headedness, while patients with complete vestibular inactivity develop oscillopsia when in motion and have difficulty maintaining balance in the dark. Patients with intermittent or continuous vestibular dysfunction (and many other causes of spatial disorientation) should never climb a ladder (recall Mr D's fall from a ladder), work on a roof, or scuba dive. On the other hand, without functioning labyrinths, they cannot develop seasickness, no matter how rough the water.

Mr D is taking several medications known to produce fatigue or dizziness (gabapentin and oxycodone), and he is taking 2 agents for diabetes that may produce hypoglycemia. Slowed

central processing likely contributes to his complaints, and the role of drug toxic effects must be considered.

Mr D's third problem, perhaps the most important one at this time, is secondary agoraphobia. With unpredictable symptoms of dizziness, he avoids going places and feels that he should not drive—the *motorist's vestibular disorientation syndrome*<sup>42</sup> or *space phobia*.<sup>43</sup> Constant concern about spatial orientation leads patients to scrutinize their balance even more closely, resulting in the "purple horse phenomenon": "Try not to think of a purple horse; what are you thinking of?"

### Treatment of Dizziness

Treatment of a complaint produced by more than 60 different disorders first requires as accurate a diagnosis as possible. The treatments for cardiac arrhythmias, hypothyroidism, brainstem ischemia, and multiple sclerosis have little in common, and the first step in treating the patient with dizziness is to address all such underlying conditions.

When identified diseases have been treated, many patients continue to have dizziness, often because of the residual sensory, integrative, or motor deficits that continue to produce uncertainty of position or motion in space. The goals of treatment are then to (1) eliminate spurious information, (2) optimize sensory orienting information, (3) accelerate integration, and (4) improve motor function.

The most common spurious sensory information is vertigo. The most common cause of vertigo, BPPV, is now known to be caused by otolithic debris from the utricle gaining access to the posterior semicircular canal and causing vertiginous sensations on position change because the calcium carbonate stones are denser than endolymphatic fluid and change position with gravity. This condition is readily cured in most patients in less than 10 minutes by using the Epley canalith repositioning maneuver.<sup>32,33</sup> The patient is placed in the head-hanging position, with the affected ear down, as in the Dix-Hallpike maneuver. Subsequent specific head rotations permit the misplaced otolithic material to transit through the posterior semicircular canal and be returned to the utricle, removing the cause of the BPPV (Figure).

Other causes of vertigo, such as acute or recurrent peripheral vestibulopathy (single or multiple episodes of vertigo of peripheral vestibular origin, not exclusively positionally elicited, without the tinnitus and hearing loss of Ménière syndrome), are of uncertain and probably varied etiology, and treatments are aimed primarily at reducing the symptoms. They can be treated with drugs that suppress both the peripheral labyrinth (such as meclizine hydrochloride, 12.5-25 mg every 6 hours) and the vestibular nuclei (eg, benzodiazepines, such as oxazepam, 10-15 mg every 6 hours); in my experience the combination is more effective than either drug alone. Because these drugs are sedative, I add 5 mg of methylphenidate hydrochloride in the morning and at noon. I find this combination to be effective in many patients with vertigo and generally well tolerated,<sup>44</sup> although efficacy in patients with vestibular disorders has not been studied systematically. For acute attacks of vertigo, I have used atropine, 0.4 mg, given sublingually for rapid absorption. Experimental animal studies suggest that atropine suppresses vestibulocerebellar cholinergic pathways,<sup>45</sup> and it produces considerably less sedation or confusion than the more widely used scopolamine. When vestibular function is diminished, a foam cervical collar can help reduce extraneous head movements that increase disorientation.

Optimizing sensory information requires proper visual, auditory, and tactile devices. Bifocal glasses should not be used by patients with dizziness when walking and night lights should be left on in the dark. Binaural hearing aids may help hearing-impaired patients localize sounds. Many patients with multisensory dizziness walk well when touching a banister, wall, or furniture. I recommend “cane trailing”—dragging an extra-long cane along the ground—as a means of providing this additional tactile sensory channel, a strategy that improves spatial orientation and reduces sway.<sup>46</sup>

Mild stimulants can help patients with dizziness integrate information more rapidly; small dosages of methylphenidate are especially useful. Patients with agoraphobia, panic states, or depression presenting with dizziness require appropriate psychotropic medications, such as selective serotonin reuptake inhibitors or clonazepam.

Motor or orthopedic problems should be directly addressed. For patients with severe sensory losses and unstable gait, physical therapy evaluation and a rolling walker with large wheels may be necessary.

Treatment is often complicated by the indirect relationship between the etiologic and the psychophysiological bases for the complaint of dizziness, as well as the subjective nature of the symptoms. While some causes of dizziness respond directly to treatment of the underlying problem (eg, BPPV, orthostatic hypotension, cardiac arrhythmias), others that are due to multiple physiological problems resulting from disease require staged and tailored treatments. The multisensory dizziness seen in some patients with diabetes may not respond to control of blood glucose, requiring other adaptive treatments. Except for BPPV, there are few causes of vertigo for which specific, etiologically directed, effective treatments exist (eg, the treatment controversy surrounding Ménière syndrome after many decades of therapeutic efforts).<sup>47-49</sup>

### Treatment for Mr D

We don't have a complete diagnosis for our patient, and ENG and MMPI would help elucidate the roles of any vestibular dysfunction and psychological contributions. Treatment of Mr D's presumptive multiple problems of dizziness requires several strategies. The initial step should be to assess possible adverse effects of his pain medications (eg, by withdrawal and substitution of others), and also to make certain that he does not experience transient hypoglycemic episodes due to his oral hypoglycemic agents.

For the vestibular impairment, I would first recommend “dizziness exercises,” which help patients adjust to decreased vestibular information.<sup>50</sup> Methylphenidate hydrochloride, 5 to 10 mg on arising and at noon, may help him integrate limited sensory inputs more quickly. He should use a night light when he goes to bed. Cane-trailing and/or wearing a foam collar could help if his balance is very poor, but probably isn't needed. Management of the episodes of agoraphobia might be helped by appropriate treatment, eg, with selective serotonin reuptake inhibitors.

Most important for many patients is an understanding of the exact nature and cause of their dizziness. Uncertainty about the cause, prognosis, precipitating events, and available treatments makes dizziness a mysterious and unpredictable problem to many patients, as well as to their physicians.

## QUESTIONS AND DISCUSSION

**A PHYSICIAN:** Did Mr D ever experience dizziness while driving or when he was on top of the ladder and fell?

**DR DRACHMAN:** He did describe 1 event where he felt dizzy while driving and realized that he still had to drive further, so he exited from the expressway and drove up on the shoulder of the ramp. He was very concerned about his near accident.

**A PHYSICIAN:** Do you have any problems with Mr D driving a motor vehicle?

**DR DRACHMAN:** When considering medically safe functioning in potentially dangerous situations (for example, in airplane pilots), we need to consider both *sudden* and *subtle* incapacitation. In terms of subtle but continuous incapacitation, I think Mr D is competent to drive. Sudden episodes of incapacitation present another question, and we have not observed him during one of his spells. He has said, “I think I shouldn't be driving.” When anyone says that, it worries me. When patients have an ongoing problem, such as impaired vision or diminished judgment due to early dementia, we are able to assess their safety by conducting a driving test. If they pass an on-road test, then they may drive; if they fail, then they should not. For someone with intermittent problems, however, it is more difficult to determine whether driving would be safe.

**A PHYSICIAN:** You mentioned Serax [oxazepam] as a component of treatment. Do you ever see dizziness as a complication of too much Serax or similar medications?

**DR DRACHMAN:** We do. First, it may make some patients drowsy. Second, it is an effective vestibular suppressant, so their vestibular sensitivity decreases. If a patient is suffering from episodes of vertigo, then benzodiazepines are effective in reducing those symptoms. In older individuals with multiple other problems, we know that unsteadiness and falls increase with these agents, however. When we combine benzodiazepines with methylphenidate, the results are better because the patients do not become drowsy. The reason I prefer oxazepam is that it has first-order pharmacokinetics and is unlikely to accumulate in the bloodstream.

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## A 66-Year-Old Woman With Ulcerative Colitis, 1 Year Later

In December 1997, at Medicine Grand Rounds, Dr Mark Peppercorn, MD, discussed the range of treatment options for ulcerative colitis.<sup>1</sup> At that time, Mrs F, a 66-year-old woman with loose, bloody bowel movements and a diagnosis of ulcerative colitis, expressed concern about usual medications due to severe allergies. Cortenemas had lost their efficacy, and prednisone prescribed for severe asthma was of uncertain benefit for her symptoms of ulcerative colitis. Dr Peppercorn felt her disease was not severe enough to justify colectomy, but he did believe a trial with topical aspirin therapy was warranted as a next step.

### MRS F, THE PATIENT

Fortunately, the bloating and loose bloody bowel movements have stopped on their own. Other than the 7.5 mg/d of prednisone for my asthma, I am taking no drugs for ulcerative colitis. I am in remission. I saw my gastroenterologist recently, and she does not feel any other intervention is warranted at this time, other than a routine sigmoidoscopy down the line.

Richard A. Parker, MD  
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1. Peppercorn MA. A 66-year-old woman with ulcerative colitis. *JAMA.* 1998;279:949-953.

**Table.** Requests for Euthanasia and Physician-Assisted Suicide (PAS)

	Total, % (N = 38)	Euthanasia, % (n = 17)	PAS, % (n = 21)
Patient request for euthanasia or PAS			
Patient initiated request	78.9	76.5	81.0
Patient repeated request	63.2	58.8	66.7
Patient experienced physical pain or poor physical functioning			
Physical pain	84.2	88.2	81.0
Poor physical function	86.8	100	76.2
Second opinion			
Discuss with another physician	39.5	56.3	28.6
Palliative care for the patient			
Narcotics for pain	92.1	88.2	95.2
Enrolled in hospice care	39.5	25.0	55.0
Length of patient-physician relationship, mo			
<6	19.0	23.5	14.3
6-12	35.1	23.5	42.9
13-24	13.5	5.9	19.0
>24	32.4	52.9	14.3
Family involved in the decision for euthanasia or PAS			
Family supported the decision	73.7	76.5	71.4
Family opposed the decision	2.6	0	4.8

nificant, unremitting symptoms initiate and repeat their request. Adhering to these safeguards does not depend on the legality of euthanasia or PAS, but on whether physicians appropriately use these interventions as last ditch efforts—that is, only when patients are experiencing extreme symptoms despite optimal palliative care and clearly request to die.

Also, our data contradict MacDonald's suggestions that physician regret arises because euthanasia or PAS was done covertly or because physicians fear prosecution. Instead, physician regret arises in 2 situations: (1) when euthanasia or PAS results in a "bad death," one that was ultimately distressing for the patient or family, and (2) when performing these interventions engendered conflicts about the proper role of the physician.

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1. Meier DE, Emmons CA, Allenstein S, Quill R, Morrison RS, Cassel CK. A national survey of physician-assisted suicide and euthanasia in the United States. *N Engl J Med.* 1998;338:1193-1201.

## Curbside Consultation and Malpractice Policies

**To the Editor:** As subspecialists (endocrinologists) we have been informed by our malpractice insurer that curbside consultations should not be rendered under any circumstances because of the potential medicolegal burden. Did Dr Kuo and colleagues<sup>1</sup> take this admonition into account when they interviewed the primary care physicians and medical subspecialists to see what effect this might have on their responses? How many other medical malpractice insurers impose such restrictions on their insured? This would be an interesting area to explore and may have wide-spread impact on this issue.

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1. Kuo D, Gifford DR, Stein MD. Curbside consultation practices and attitudes among primary care physicians and medical subspecialists. *JAMA.* 1998;280:905-909.

**In Reply:** We did not collect information about how insurance company policies might influence how physicians practice or feel about curbsiding. We feel it is unfortunate that Dr Block's malpractice insurer has chosen to prohibit curbside consultations because of the potential liability. One of the main findings of our study was the disagreement between primary care physicians and subspecialists about the quality of information exchanged in curbside consultations. However, we also established that curbside consultations are perceived to be an important method of communication between physicians that may have certain advantages such as saving time. It remains to be shown whether curbside consultations result in more adverse outcomes compared with formal consultations. Until such data become available, we believe physicians should continue to "curbside" each other. Rather than implementing broad policies against curbsiding, insurance companies and other institutions might consider providing guidelines for the proper utilization of curbside consultations. We hope that our findings will help improve this important element of medical practice.

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## CORRECTION

**Figure Credit Omitted:** In the Clinical Crossroads entitled "A 69-Year-Old Man With Chronic Dizziness," published in the December 23/30, 1998, issue of THE JOURNAL (1998;280:2111-2118), the credit for the Figure on page 2115 was omitted. The Figure was modified from a figure that originally appeared in: Baloh RW. Dizziness and vertigo. In: Samuels MA, Feske S, eds. *Office Practice of Neurology.* New York, NY: Churchill Livingstone Inc; 1996:87.