Editorials

Dizziness in Primary Care

NEXT TO FATIGUE, dizziness may be the most common nonpain physical complaint seen in primary care. Yet, like fatigue, dizziness remains a diagnostic and therapeutic gadfly. It is the rare clinician or student of medicine who exclaims to colleagues, "Let me tell you about a great case of dizziness I've just seen." Our insecure approach to this intangible symptom does not breed much enthusiasm. The review by McGee in this issue of the journal[1] complements nicely several recent reviews[2,3] and is both a practical starting point for primary care physicians and a springboard from which to point out some good news, some shaky conventional wisdom, some preliminary advice, and some directions for future research.

Prognosis is the good news. In a recent study of 100 primary care patients with a chief complaint of dizziness, serious cardiovascular or central nervous system causes were not apparent initially,[4] nor did they develop over a one-year follow-up period.[5] Moreover, a third of patients symptomatically improved within the first several weeks and another third gradually got better over the next 4 to 12 months. Follow-up rather than an expensive workup may suffice as the initial approach for many dizzy patients.

Diagnosis and therapy represent the shaky ground. Certain assumptions about dizziness deserve to be challenged. First, is cerebrovascular disease an important cause? Although a stroke or transient ischemic attack arising in the posterior circulation is a commonly entertained diagnosis in elderly patients, the few retrospective studies conducted in hospital or referral settings cited by McGee and elsewhere do not establish the frequency of "vascular vertigo."[1,6] Transient ischemic attacks are entirely a clinical diagnosis, and because there is no definitive treatment of posterior circulation ischemia, their association with isolated dizziness is particularly difficult to prove. While preliminary studies suggest that dizziness in the absence of other neurologic symptoms seldom represents an ischemic event in primary care,[5,7,8] the true incidence of cerebrovascular dizziness is unknown.

Second, what about hyperventilation? Although Drachman and Hart reported that nearly one in four cases of dizziness could be attributed to hyperventilation,[9] patients experiencing symptoms with this maneuver in a recent study almost always had another etiologic explanation, most commonly a vestibulopathy or psychiatric disorder.[4] Furthermore, their dizziness was reproducible, on average, by at least two other maneuvers, suggesting that hyperventilation may be a nonspecific provocative maneuver in suggestible or sensitive patients.

Third, what about cardiac arrhythmias, Meniere's disease, acoustic neuromas, labyrinthitis, and other specific causes? These in fact account for few cases. Other than the common and discrete disorder of benign positional vertigo, most dizziness is not nosologically crisp but rather vertiginous, psychiatric, idiopathic, or multicausal in nature. Whereas vertigo and psychiatric disorders account for two thirds of cases, most vertigo (except for benign positional vertigo) can at best be characterized as peripheral or central, and psychogenic dizziness has not yet been put to the therapeutic test. In fact, lack of specific therapies for many types of dizziness is one of the Achilles' heels in our ability to confidently ascribe etiologic labels. Other limitations include the difficulty patients have in precisely describing their dizziness and the lack of objective and agreed-on diagnostic tests. All of these factors explain the considerable disagreement even among experts about the cause of dizziness in individual patients.[4]

Fourth, what about the role of meclizine in treating dizzy patients? Although it may have a palliative role in acute vertiginous attacks lasting hours or days, such as labyrinthitis and Meniere's disease, it is widely prescribed for patients with many other types of dizziness. The episodes of benign positional vertigo are too brief to warrant drug therapy and are better treated with habituation exercises.[2] Nonspecific peripheral and central vestibulopathies are the cause of dizziness in more than half of patients with vertigo and often produce sporadic and self-limited spells for which long-term drug therapy may not be indicated or effective. The strongest studies supporting the use of meclizine, other antihistamines, and anticholinergic medications involve motion-sickness prevention and thus cannot be readily generalized to the treatment of patients with dizziness. The several small studies cited by McGee as showing a beneficial response of dizziness to meclizine use were of short duration and involved only small numbers of patients.

What should be the primary care approach to the dizzy patient? A focused history and physical examination is the most valuable diagnostic tool, and even here detailed cardiac and neurologic examination has a low yield. The most useful historical information is patients' own descriptions of their dizziness, its characterization as "spinning, fainting, or falling" (that is, vertigo, presyncope, or disequilibrium), and the effects of position (lying down, standing, walking) on patients' symptoms. The most useful physical examination maneuvers are the Hallpike (head-hanging) test for benign positional vertigo, supine and standing blood pressure and pulse measurements to detect orthostatic changes, and observing the patient walking across the room, turning around, and walking back. Also, the physician can do a one-minute bedside vestibular examination looking for nystagmus present in the primary position, with eccentric gaze, and after rebound and head-shaking.

Routine laboratory testing, electrocardiography, and audiometry are seldom helpful. Expensive tests such as neuroimaging and ambulatory cardiac monitoring also have a low yield and can be reserved for patients with other indications of neurologic or cardiac disease. For patients with persistent dizziness, vestibular testing and

evaluating for psychiatric disorders are the two most useful steps. Because depression, anxiety, and somatization are the most common psychiatric disorders associated with dizziness, these can often be evaluated and even treated by a primary care physician, referring only the more severe or refractory cases to a mental health specialist. Various types of vestibular testing, including electronystagmography, rotatory chair, dynamic posturography, and clinical evaluation, can be done by a neuro-ophthalmologist or neuro-otologist. Whether any one of these tests is superior to the others or how often such testing actually changes patient management is unclear.

Dizziness is rife with questions for primary care researchers. Does dizziness associated with treatable psychiatric disorders such as depression and anxiety respond to psychotropic medication or psychotherapy? Can dizziness attributable to a chronic vestibulopathy improve with vestibular rehabilitation, or is it an irreversible symptom that must be tolerated, in the words of Jonathan Swift, "That old vertigo in his head/Will never leave him till he's dead"? One positive study of vestibular rehabilitation showed relatively modest and inconclusive benefits.[10] Is one type of vestibular testing superior to another, and does such testing make a difference in clinical management and patient outcome? What is the role of subspecialists, such as neurologists and otolaryngologists, in the care of dizzy patients, and which patients benefit from referral? Can we reduce the amount of laboratory testing, neuroimaging, and other low-yield tests without adversely affecting patient outcome? Are meclizine, other antihistamines, and anticholinergic medications beneficial in any type of dizziness other than acute and prolonged vertiginous attacks? While the disorder often resolves, its persistence in a considerable minority of patients causes substantial suffering and functional impairment.[11] As a physical symptom for which patients' self-reporting is the gold standard, dizziness will always be difficult to study, and our research findings will have rougher edges than in many other areas of clinical investigation. But as long as dizziness crosses the clinic threshold as often as it does—10 million visits annually in the United States alone—imperfect answers that are even a slight improvement over previous imperfections are worth the investment.

KURT KROENKE, MD
Department of Medicine
Uniformed Services University of the Health Sciences
Bethesda, Maryland

REFERENCES

1. McGee SR: Dizzy patients—Diagnosis and treatment. West J Med 1995; 162:37-42

2. Kroenke K: Dizziness: Practical office workup and management recommendations. Consultant 1993; 33:80-90

3. Warner EA, Wallach PM, Adelman HM, Sahlin-Hughes K: Dizziness in primary care patients. J Gen Intern Med 1992; 7:454-463

4. Kroenke K, Lucas CA, Rosenberg ML, et al: Causes of persistent dizziness—A prospective study of 100 patients in ambulatory care. Ann Intern Med 1992; 117:898-904

5. Kroenke K, Lucas C, Rosenberg ML, Scherokman B, Herbers JE: One-year outcome in patients with a chief complaint of dizziness. J Gen Intern Med, in press

6. Grad A, Baloh RW: Vertigo of vascular origin: Clinical and electronystagmographic features in 84 cases. Arch Neurol 1989; 46:281-284

7. Madlon-Kay DJ: Evaluation and outcome of the dizzy patient. J Fam Pract 1985; 21:109-113

8. Leliever WC, Barber HO: Recurrent vestibulopathy. Laryngoscope 1981; 91:1-6

9. Drachman DA, Hart CW: An approach to the dizzy patient. Neurology 1972; 22:323-334

10. Horak FB, Jones-Rycewicz C, Black FO, Shusway-Cook A: Effects of vestibular rehabilitation on dizziness and imbalance. Otolaryngol Head Neck Surg 1992; 106:175-180

11. Kroenke K, Lucas CA, Rosenberg ML, Scherokman B: Psychiatric disorders and functional impairment in patients with persistent dizziness. J Gen Intern Med 1993; 8:530-535

Surfactant Replacement Therapy—Room for Improvement

WE BREATHE EFFORTLESSLY because of pulmonary surfactant. Unfortunately, very premature infants lack surfactant and have labored breathing and respiratory insufficiency. Medical conditions associated with alveolar instability and microatelectasis are prime candidates for surfactant replacement therapy. Surfactant replacement therapy for preventing and treating the respiratory distress syndrome in newborns is a major therapeutic advance in neonatal care and is comprehensively reviewed by Poulain and Clements elsewhere in this issue.[1] Pulmonary surface-active material is composed of phospholipid, cholesterol, and associated surfactant proteins, which are designated SP-A, SP-B, and SP-C.[2] There is a fourth surfactant-associated protein, SP-D, that binds to surfactant weakly and is physiologically a host-defense molecule.[3] Surfactant is synthesized by alveolar type II cells and packaged in lamellar inclusion bodies. After secretion by endocytosis, this material unravels to form tubular myelin, a unique physical form of surfactant, and then adsorbs to the air-liquid interface. Surfactant proteins A and B are necessary for the formation of tubular myelin. They organize the three-dimensional structure of the phospholipids. Adsorption occurs during inhalation when the surface film is expanded, and during exhalation a low surface tension is formed when the surface film is compressed. After repeated respiratory cycles, some of the lipids of surfactant are squeezed from the air-liquid interface, enter the subphase, and are endocytosed by type II cells for reuse. The uptake of extracellular surfactant by type II cells is facilitated by SP-A.

The development of surfactant for replacement therapy underscores the importance of basic science in providing a framework for clinical science. The initial physiologic studies that showed that a low surface tension was present at the air-liquid interface in the lung was reported by von Neergard in 1929. This observation was not widely appreciated, however, and had to be rediscovered in the 1950s. At that time Pattle and Clements independently isolated and identified surfactant as the substance that produced the low surface tension in the lung and defined its physical properties.[4,5] This basic science precept was then rapidly applied to a clinical situation. Within three years surfactant was reported to be deficient in the lungs of infants who died of hyaline membrane disease.[6]

Soon the unusual phospholipid that was responsible