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The Dizzy Patient

Peter T. Skaff, MD, FAAN

One of the most vexing symptoms with which patients present to their physicians is dizziness. The number of diagnostic possibilities is protean and range from benign to acutely life-threatening. Discerning the source of the problem depends on an understanding of what is meant by the patient, and a systematic approach to elucidation of the patient's complaint is essential to both making the correct diagnosis and insuring the proper treatment plan.

Dizziness is a symptom, not a diagnosis, per se, and as indicated by the variety of definitions of the word, "dizzy," the range of diagnostic possibilities is broad. Although dizzy may mean foolish or confused, as in "dizzy blonde," or extremely rapid, as in "dizzy pace," with regard to medical symptoms, there are three groups into which this term is primarily categorized: (1) having a vertiginous, whirling, or spinning sensation, (2) feeling off balance or lacking equilibrium, and (3) feeling lightheaded or faint. What is critically important is to first ask the patient, "What do you mean by dizzy?" In most cases, occasionally with a bit of prompting and feedback, the patient will identify their symptoms as belonging to one of these three categories.

Balance disorders are characterized by disequilibrium and arise from extrapyramidal, cerebellar, or peripheral sensory deficits, and are often multifactorial.

Once the characteristics of the symptoms are defined, the problem can be further categorized by the physiological system or systems involved. The primary causes of neurogenic dizziness are of the first two categories and are described as either vertigo or impaired balance. Spinning or vertigo arises from dysfunction in either the peripheral or central components of the vestibular system. Balance disorders are characterized by disequilibrium and arise from extrapyramidal, cerebellar, or peripheral sensory deficits, and are often multifactorial. Lightheadedness is primarily non-neurological and implies circulatory dysfunction, which arise most commonly from orthostatic, vasovagal, or cardiogenic disorders.

The next step in arriving at a specific diagnosis is to ascertain the time course of the symptoms and their associated features, both with regard to symptoms and physical examination findings. For example, cerebellar stroke is characterized by vertigo that occurs suddenly, is worst at onset and then gradually improves, and may be accompanied by findings of nystagmus and limb dysmetria. Labyrinthitis is acute or subacute in onset, is unrelenting in degree, and though it is often associated with nystagmus, it does not produce limb dysmetria. Benign Paroxysmal Positional Vertigo comes on suddenly but is precipitated by certain movements, typically rolling to one side from the supine position, and is alleviated by holding still. Meniere Disease comes on acutely or sub-acutely, is unrelenting for hours at a time, and is associated with unilateral, low-pitch tinnitus and hearing loss for low frequencies.


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What to Do with an Abnormal Carotid Ultrasound?

Handel Robinson, MD

Given that carotid disease is a risk factor for an ischemic stroke, screening for carotid disease is often performed in patients with known risk factors for vascular diseases. These include smoking, hypercholesterolemia, age, and male gender. The identification of a carotid bruit on physical exam is one of the most basic and common ways to identify carotid disease and those at increased risk of a stroke—and should prompt a carotid duplex.

The technology of a carotid duplex employs measuring how fast blood flows through the carotid vessel. This velocity then correlates with different ranges of stenosis based on data-established criteria. The question then arises: What to do with a duplex showing abnormal carotid velocities? Typically, this abnormality occurs at the carotid bifurcation, which is the most common location for the development of atherosclerotic disease.

Translating these degrees of stenosis into stroke risk is dependent on the history with which the patient presents, particularly whether the stenosis is symptomatic or asymptomatic. Symptomatic includes stroke, amaurosis fugax, or a transient ischemic attack all within the past six months. With the patient history and the findings of a carotid duplex, one can then go on to direct treatment, medical vs. intervention, to decrease the risk of stroke.

The benefit of medical treatment compared to surgery has been robustly evaluated for symptomatic and asymptomatic patients. For patients deemed to be symptomatic, any stenosis less than 50 percent can be treated medically with results equivalent to intervention in decreasing the risk of stroke. Stenosis above 50 percent in this cohort warrants evaluation for carotid intervention. The data in its most technical analysis reveals benefit above 60 percent stenosis, however most interventionalists would agree that true benefit isn't appreciated until above 80 percent of stenosis of the carotid vessel.

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The management of carotid disease involves two options, medical and intervention. Medical management includes antiplatelets, statin, and other risk factor modifications such as smoking cessation. Patients with carotid disease, if not referred for intervention, should be followed to monitor for the progression of disease. Stenosis of less than 50 percent will require an annual carotid duplex. Stenosis greater than 50 percent should be monitored every six months for progression.

Other imaging modalities such as CT angiography and MR angiography can be ordered to provide more detailed analysis of a patient's carotid disease, particularly before an intervention is offered, or if on follow-up duplex there is a sudden change in velocities translating into an increased degree stenosis. ■

Stroke in the Young

Rodica E. Petrea, MD

"Case presentation: A 33-year-old right-handed young woman had two weeks of left-sided new onset throbbing headaches, localized to the left eye and left lateral cervical area. This morning she awakened with right facial droop and difficulties finding words in speaking. She had no prior events. She was a smoker and was taking oral contraceptive pills, and lately her blood pressure measurements were in the 150/100 range, which was higher than normal. She presented to the Emergency Department as a wake-up stroke with a NIHSS (NIH Stroke Scale) score of 3 (mild impairment). MRI brain showed a small left frontal ischemic stroke, and MR Angiogram (MRA) of her head and neck revealed a left internal carotid artery dissection with thrombus located right above the common carotid artery bifurcation. It was noted that she had been exercising at the gym more intensely than usual for the past month, and she recalled a recent small "pop" in her neck while exercising. The MRA suggested a pattern of fibromuscular dysplasia, which was later confirmed with a conventional cerebral angiography. She was discharged home on antiplatelet therapy with NIHSS of 1 (minimal impairment)."

Stroke is most common in older adults, the incidence higher in men up to 85 years of age when women take the lead. Stroke is less common in young adults (18 to 50 years), but both ischemic and hemorrhagic strokes can be seen in young adults. Stroke in adolescents and children implies pathology specific to those ages.

Ischemic stroke is the result of a vascular occlusion, most commonly arterial but sometimes venous. Hemorrhagic stroke is the result of a ruptured blood vessel inside the brain (intracerebral hemorrhage) or around the brain in the subdural or arachnoid space. Ischemic strokes may be due to embolism, thrombosis, or hemodynamic compromise and may involve large or small vessels. Up to 40% of strokes are of undetermined cause and are called cryptogenic. The good news is that temporal trends in stroke incidence showed a decrease in old age, but the bad news is an increased incidence between 20 to 54 years, possibly due to increased incidence of traditional vascular risk factors for stroke, like hypertension, diabetes, dyslipidemia, obesity, smoking, alcohol, and drug abuse. Ischemic strokes in the young adult are more commonly due to cardiac embolism, non-atherosclerotic large artery occlusive disease, traumatic and spontaneous cervical and intracranial arterial dissections, or a long list of uncommon disorders such as moya-moya, fibromuscular dysplasia, and rheumatological and inflammatory disorders. Other causes of ischemic stroke in the young are various prothrombotic states such as pregnancy, malignancies, and hypercoagulable, and hematologic disorders. Risk factors for stroke in young adults include oral contraceptives, substance abuse, and migraine. The most common genetic cause for venous thrombosis is factor V Leiden; other causes include deficiency of antithrombin III, protein C and protein S, prothrombin gene mutations, increased factor VIII, antiphospholipid antibodies syndrome, polycythemia, thrombocytosis, and thrombotic thrombocytopenic purpura.

The workup of stroke in young adults should not be limited to computed tomography (CT) of the head and magnetic resonance (MR) imaging of the brain but should include vessel imaging such as CT angiography or magnetic resonance (MR) angiography, and in selected cases cerebral angiography. Comprehensive cardiac evaluation is mandatory and should include transthoracic and transesophageal echocardiography (TEE) to diagnose a patent foramen ovale or other intracardiac shunts, left ventricular or atrial thrombus, cardiac vegetations, endocarditis or other cardiac malformations, or valvular abnormalities such as mitral valve prolapse. Holter monitoring and implanted cardiac loop are also recommended for diagnosing paroxysmal atrial fibrillation. Evaluation for hypercoagulable states is also mandatory. Antiplatelet and anticoagulant agents are widely used in ischemic stroke prevention in young adult as well as older patients.

Stroke in adolescents and children implies pathology specific to those ages.

Any stroke consultation and care should emphasize the very common and preventable stroke risk factors, including hypertension, diabetes mellitus, dyslipidemia, smoking, alcohol, drug abuse, and oral contraceptives. Stroke prevention goals should include a blood pressure target of < 130/80 mmHg, LDL cholesterol < 70 mg/dL and Hb A1C < 7% for diabetics

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Evaluation and Management of a Single Seizure

Hemant S. Kudrimoti, MD, PhD

An epileptic seizure is an abnormal, involuntary behavior or experience due to sudden, abnormal, and usually self-limited electrical activity in neuronal networks. The clinical symptoms of a seizure are paroxysmal, depend on the area of the brain affected (the seizure-focus) and may include impaired consciousness and abnormal motor behaviors and/or sensory, autonomic, or psychic experiences. Epilepsy is defined operationally as two or more unprovoked seizures. Seizure recurrence-risk is substantially higher after two or more unprovoked seizures than after just one, and treatment with anticonvulsants is almost always initiated. The single seizure, however, presents a management quandary. In 2015, the American Academy of Neurology and the American Epilepsy Society released a new guideline on the prognosis and treatment of the first unprovoked seizure. According to the guideline, immediate anti-seizure drug therapy, as compared with delay of treatment pending a second seizure, is likely to reduce recurrence risk within the first two years but may not improve quality of life. Thus, recommendations on whether or not to treat a single seizure remain unclear.

The history itself remains of paramount importance in obtaining accurate information for risk stratification.

The first unprovoked seizure is an unpleasant surprise for the individual who experiences it. The episode presents a dilemma for the care provider regarding whether to treat the risk of potential recurrence with anticonvulsant but expose the patient to the risk of medication side effects, or to take a wait-and-watch approach and potentially expose the patient to the risk of another seizure. The key question, therefore, is how to assess recurrence risk. The history itself remains of paramount importance in obtaining accurate information for risk stratification. The presence of auras, which are symptoms such as déjà-vu experiences, panic attacks, odd smells, a metallic taste in the mouth, one-sided somatosensory or visual phenomena, a rising epigastric sensation, dizziness, vertigo, makes a diagnosis of focal epilepsy more likely, and the clinician can weigh in favor of treatment. The physician and patient should decide jointly whether to institute anticonvulsant therapy after a single seizure. This decision is based on a discussion of the risk of seizure recurrence, and the implications of recurrent seizures on the ability to be able to legally drive or remain employed. The effectiveness of anticonvulsants and their potential side effects also need to be discussed with the patient.

Many patients who have a single motor seizure without any preceding symptoms may not require anticonvulsant therapy. Most importantly, however, risk stratification must be facilitated by obtaining a work-up soon after the first seizure. This should include an EEG and a brain MRI. A head CT may be acceptable for screening in the emergency department but a brain MRI is far more sensitive in detecting subtle cerebral abnormalities that may have implications for seizure risk. Overall, certain clinical scenarios, abnormal EEG and imaging findings, and epidemiological predictors increase recurrence risk. For example, febrile seizures in childhood, remote head injuries with prolonged loss of consciousness or amnesia, a family history of epilepsy, perinatal cerebral injury, prior cortical strokes, an unprovoked nocturnal seizure, age < 16 years, previous provoked seizures, focal seizure, focal epileptiform discharges on EEG, structural abnormalities on imaging (e.g., tumor, arterio-venous malformation, mesial temporal sclerosis), first presentation in status epilepticus, and cerebral palsy all significantly increase seizure recurrence risk.

In summary, clinicians' recommendations on whether to initiate immediate anti-seizure drug treatment after a first seizure should be based on individualized assessments that weigh the risk of recurrence against the adverse effects of therapy, that consider educated patient preferences, and that are based on results of an appropriate work-up. ■

Hemant S. Kudrimoti, MD, PhD

How the Shape of Your Back Affects Long-Term Spine Health

Kevin Hsieh, MD

Some patients who have had a lumber fusion express dissatisfaction due to continued pain, and some need repeat lumbar fusion procedures. This article will discuss some new ideas about why this happens and how it can best be prevented.

The curve in the lumbar spine is the angle between L1 and L5 and is called lumbar lordosis. Everybody needs a different lumbar lordosis, but how much used to be a mystery. It is now understood that lumbar lordosis must be within 10 degrees of a measured parameter, known as the pelvic incidence (PI) which represents the angle of the pelvis. These measurements represent sagittal balance—the idea that one's head is centered on the pelvis from the side view.


Kevin Hsieh, MD

It may seem strange that a spine article is based on a math equation. But this equation is exceedingly important. In approaching a preoperative patient, if the patient is fused without enough lordosis, meta-review of the literature indicates this is the single most important predictor of long-term back pain. Therefore, it is imperative that each surgery is finished with enough lordosis. Essentially, this tool—if properly used preoperatively and intraoperatively—can help eliminate the number one cause of post-surgical back pain.

A myth associated with spine surgeries is that once a patient as had one fusion, they will continue to "march on up the spine," getting more and more fusions, also commonly known as adjacent-level disease. This is partially because a longer wrench (fused segment) is much more powerful than a shorter wrench. However, recent studies also point toward our sagittal balance as an important factor. Patients who are fused with an LL/PI mismatch have ten times the rate of adjacent-level disease.

In summary, if patients are fused with their lumbar lordosis matching their pelvic incidence, they are much more likely to not have postoperative back pain and ten times less likely to have adjacent-level disease. There are many technical methods to achieve this, including anterior column support, posterior compression, and posterior osteotomies. However, the bottom line is, patients fused in good alignment tend to stay there with much less pain. ■

Acute Vision Loss

Robert Bellinoff, MD

When examining a patient with acute vision loss, it is important to cover the basics of an eye exam. For the purpose of this article, "acute vision loss" will mean a change in one's vision within a few minutes to a couple of days, whether transient or permanent, such that the patient has noticed difficulty with near and/or distance vision, and/or significant loss of visual field, either horizontal, vertical, or central in presentation.

The following points highlight a basic eye exam:

- Visual acuity, testing each eye independently, with correction such as glasses or contacts, if they wear them
- Confrontation visual fields
- Extraocular motility
- Pupillary size and motility, in both lighted and dim conditions
- Presence or absence of APD (afferent pupillary defect, or Marcus-Gunn Pupil)
- Anterior segment exam looking for hyperemia
- Direct ophthalmoscopic view of the optic nerve

The age of the patient can guide one in determining a differential diagnosis. In children, consider causes such as congenital, trauma, and amblyopia. Young adults may have unaddressed refractive error, trauma and undiscovered amblyopia. If greater than 55 years of age, consider vascular, autoimmune, infectious, and age-related conditions.


Robert Bellinoff, MD

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There are many risk factors that can predispose one to acute vision loss. The following are common ones:

- Diabetes mellitus
- Hypertension
- Hyperlipidemia
- Hypercoagulable states such as Factor V Leiden, pregnancy, and cancer
- Carotid insufficiency
- Cardiac arrhythmias
- Migraine headaches
- Trauma
- Obesity
- Obstructive sleep apnea
- High myopia
- Infectious exposure
- Contact lens wearer

If the length of time for the visual loss is 15 to 30 minutes, consider ocular migraine, thromboembolism, or even increased intracranial hypertension. Consider TIAs if less than 24 hours. If greater than 24 hours, differentials include retinal detachment, vitreous hemorrhage, optic neuropathies, or vascular occlusions. Remember that transient vision loss (minutes to hours) is a symptom and not a diagnosis. Other etiologies include hypoxia, ischemia, hypotension due to cardiac arrhythmia, epileptic seizures, and retrobulbar tumors causing gaze evoked vision loss. If the patient is under 45 years of age, it's more likely vasospastic or migraine.

Painful monocular vision loss is an emergent problem and must be evaluated ASAP. Some of the more common causes include the following:

- Acute angle closure glaucoma: conjunctival injection, haloes around lights, nausea, photophobia, severe pain, fixed mid-dilated pupil
- Corneal ulcer: conjunctival injection, white spot on cornea, often contact lens related, photophobia
- Iritis/uveitis: conjunctival injection, photophobia, miotic or irregular shaped pupil
- Optic neuritis: pain with eye movements, abnormal color vision, visual field defects, Marcus-Gunn Pupil

Binocular vision loss is much less common than monocular. Differential diagnoses for binocular loss include bilateral angle closure (which would be painful, and one cause could be topiramate usage), ischemic optic neuropathy due to giant cell arteritis (GCA), hereditary optic neuropathy such as Leber's optic neuropathy, cortical blindness, hypotensive states, hypertensive states, severe bilateral carotid artery stenosis, and migraines.

Acute loss of vision without eye pain is not uncommon to see in practice. The following conditions are commonly encountered:

- Amaurosis fugax—Monocular blindness lasting minutes to hours, typically less than five minutes when due to cerebrovascular disease. Workup could include duplex carotid ultrasound, echocardiography, EKG, giant cell arteritis workup including platelets, ESR, and low sensitivity CRP. Remember amaurosis fugax is a description of a condition, not a diagnosis.
- Arteritic ischemic optic neuropathy—typically due to GCA and typically over 60 years old. Associated symptoms include jaw claudication, scalp tenderness (hurts to rest head on a pillow), unexplained weight loss, fatigue, joint pains, temporal headaches, and tenderness over the temporal artery area. Associated findings can include pale and swollen optic nerve or double vision. Workup includes ESR, platelet count, low sensitivity CRP, and temporal artery biopsy (2 cm segment).
- Nonarteritic ischemic optic neuropathy—Optic nerve swelling and hemorrhages are common as is visual field defect, typically altitudinal. Risk factors include obstructive sleep apnea, small optic nerves, obesity, diabetes, hypertension, and cardiovascular diseases.
- Retinal vascular occlusions—This could include branch retinal vein or artery occlusions and central retinal vein or artery occlusions. Pale retina or retinal hemorrhages and vitreous hemorrhages can often be seen. Risk factors include age, diabetes, cardiovascular disease, and open angle glaucoma.
- Retinal detachment—Presenting symptoms include a shade or shadow in their vision, flashes of light, or new onset floaters. Risk factors include high myopia, family history, or trauma. Depending on where the detachment is, the patient could still have 20/20 vision.

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Acute Vision Loss—continued from page 6

When to refer to ophthalmology is based on your concern, the patient's concern, and presenting signs and symptoms.

Immediate:

- Painful vision loss as described above
- Symptoms of GCA associated with vision loss
- Sudden unexplained severe vision loss of less than 12 hours since onset
- Acute vision loss with APD (Marcus-Gunn Pupil)
- Amaurosis fugax (especially if less than 7 days ago)

Urgent—within 24 hours:

- Flashes and floaters associated with vision loss
- Vision loss associated with optic nerve swelling
- Diabetic with sudden decreased visual acuity
- Headache associated with vision loss (migraine, idiopathic intracranial hypertension)

If ever in doubt on whether to refer urgently, it is better to err on the side of caution. The eye care provider can always offer guidance on the phone whether the patient needs to be seen that day or at a later date. ■

The Dizzy Patient—continued from page 1

With regard to balance disorders, one must keep in mind that equilibrium is maintained by multiple systems, and dysfunction in any of these may result in dysequilbrium. For example, peripheral neuropathy produces impairment in distal sensory function, particularly in the feet, which transmit critical information about the ground to the central nervous system. When sensory input from the feet is sufficiently impaired, balance will become disturbed. This is the basis of Romberg's test. When standing still with the feet together, the visual and vestibular systems can compensate for the loss of sensation from the feet, and balance is maintained. Once the eyes are closed, however, balance is lost, because compensatory visual input is lacking. The central mechanisms for maintaining equilibrium are complex and involve primary motor and sensory function as well as the extrapyramidal system of deep nuclei which process sensory information and are critical to motor planning. Parkinson disease and associated, parkinsonian syndromes, such as Normal Pressure Hydrocephalus, arise from and are characterized by dysfunction in these central mechanisms.

Because the word means different things to different people in different circumstances, it is not enough to accept "dizzy" as the final descriptor from any patient presenting with this complaint. Rather, it is incumbent upon the physician to ascertain what is meant by the patient using this term. With just a few questions, the problem can be quickly categorized to separate benign entities from those that are potentially life-threatening, and the patient can be appropriately diagnosed and treated. ■

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and normal for non-diabetics. A healthy diet with low fat, low salt, and low glycemic index carbohydrates and a regular regimen of walking and exercise of at least 30 minutes/day should be stressed.

Hemorrhagic stroke comprises 10–15% of strokes in young adults and is commonly due to arterial venous malformations, cerebral aneurysms, hemorrhagic tumors, alcohol, and drug abuse (cocaine, methamphetamines). Other causes include systemic inflammatory disorders, and malignant, accelerated hypertension. These are in contrast to the causes of intracranial hemorrhage in older adults, which include hypertension, amyloid angiopathy, antithrombotics, and anticoagulants. Trauma is a major cause of intracerebral hemorrhage at any age. The workup for hemorrhagic stroke in young adults should always include the conventional cerebral angiography in addition to the brain MRI, whereas in stroke at older age MR or CT angiography might be considered sufficient and adequate. ■

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Neuroscience Update
Hyatt Regency Sacramento
April 6, 2019
8 a.m. to 1:30 p.m.

Stroke & Vascular Neurology Conference
Mercy San Juan Medical Center
6555 Coyle Ave., Suite 145
4th Thursday of the month at 7 a.m.

Epilepsy & Subspecialty Neurology Case Conference
Mercy General Hospital, North Auditorium | odd months
Mercy San Juan Medical Center, Ste 145 | even months
4th Tuesday of the month at 6 p.m.

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