

distinguishing malignant from benign lesions. Likewise, imaging studies by themselves are of limited use. Radionuclide scanning with iodine or technetium cannot reliably distinguish benign from malignant nodules: only 20% of "cold" nodules are malignant, "warm" nodules are not usually malignant (but 9% can be), and even "hot" nodules can be malignant in 1% of patients. Ultrasonography is 90% sensitive for distinguishing solid from cystic or mixed nodules. However, 15% of cystic nodules may harbor cancers.

Fine-needle aspiration biopsy has become the diagnostic test of choice. The test is safe and has a false-negative and false-positive rate of 2%. The accuracy depends on the aspiration technique and on the experience of the cytopathologist. Over 70% of nodules evaluated by fine-needle aspiration biopsy are benign, including colloid nodules, cysts, and thyroiditis. Suspicious and malignant lesions account for 5% to 15% of studies, and 2% to 21% are nondiagnostic. The *Figure* shows a diagnostic algorithm for evaluating thyroid nodules.

THYROID CANCER: AGGRESSIVE FORMS

Thyroid cancer accounts for approximately 1% of all cancers, and only 9% of patients with thyroid cancer die from it. Yet some forms are very aggressive and require early recognition and early aggressive intervention for prolonged survival.

Papillary adenocarcinoma accounts for 70% of all thyroid cancers in adults and 90% in children. The peak incidence occurs in the third and fourth decades, and it is the most common thyroid cancer associated with previous ionizing radiation exposure. Most patients have an excellent chance for long-term survival; risk factors for more aggressive disease include age younger than 20 or older than 50, male sex, tumor size larger than 4 cm, unfavorable histology, distant metastasis, and the absence of coexisting Hashimoto's thyroiditis. Treatment includes surgical resection with or without radioactive iodine ablation and thyroid hormone suppression.

Follicular cancer accounts for 15% of thyroid cancers and generally carries a worse prognosis than papillary adenocarcinoma. Distant metastasis is not uncommon because it spreads hematogenously. Risk factors for poor outcome include age older than 40, tumor size larger than 4 cm, invasion through the capsule or vascular invasion, and local or distant spread. Treatment includes subtotal or total thy-

roidectomy, radioiodine ablation, and thyroid hormone suppression. Occasionally, focused-beam irradiation is used as a palliative treatment, especially for bony metastasis. The undifferentiated thyroid cancers include Hurthle cell carcinoma, medullary carcinoma, lymphoma, metastatic carcinoma (from the breast, kidney, or lung), and anaplastic carcinoma. These aggressive forms of thyroid carcinoma carry a poor prognosis unless detected early and treated aggressively. These tumors almost never respond to radioiodine ablation, and treatment usually includes surgery, focused-beam irradiation, and possibly chemotherapy. Fortunately, they account for only approximately 15% of all thyroid cancers.

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SUGGESTED READING

Gharib H, Goellner J. Fine-needle aspiration biopsy of the thyroid: an appraisal. *Ann Intern Med* 1993; 118:282-289.

Robbins J, Merino MJ, Boice JD Jr, et al. Thyroid cancer: a lethal endocrine neoplasm. *Ann Intern Med* 1991; 115:133-147.

de los Santos ET, Keyhani-Rofagha S, Cunningham JJ, Mazzaferrri EL. Cystic thyroid nodules. *Arch Intern Med* 1990; 150:1422-1427.

Sessions R. Thyroid cancer. *Med Clin North Am* 1993; 77:517-538.

CHRONIC DIZZINESS: VESTIBULAR EVALUATION AND REHABILITATION

Dizziness, one of the top four symptoms for which patients seek medical attention, is one of the most complex and frustrating symptoms to evaluate. A systematic approach can speed the evaluation, improve management, and decrease the frustration of the physician and patient. The key is to establish through careful questioning precisely what the patient means by "dizziness." The physician's task is to distinguish true vertigo (an erroneous sensation of movement) from other sensations such as faintness, and, if vertigo is present, to determine if it is caused by a vestibular (peripheral) or a central disorder.

THE BALANCE MECHANISM

Three systems contribute to balance: the vestibular, visual, and proprioceptive systems. Because the vestibular system is the least redundant, when it is compromised, the symptoms are more pronounced than if the visual or proprioceptive systems are af-

ected. Vestibular diseases cause true vertigo, whereas nonvestibular diseases cause unsteadiness and light-headedness.

The cerebellum plays a major role in modulating the two major balance reflexes: the vestibular-ocular reflex (VOR) and the vestibular-spinal reflex (VSR). These center the eyes and body and so maintain visual acuity and stability during ambulation.

APPROACH TO THE DIZZY PATIENT

Although it takes time to compile, a detailed history is essential to making a diagnosis. Does the patient feel a sense of rotation? In general, rotatory dizziness is caused by inner ear disorders, whereas nonrotatory dizziness is not. Considerable overlap exists, however. When did the dizziness start? What brings on the sensation, how severe is it, and how long does it last? Central vertigo is usually less severe than vestibular vertigo but does not abate with habituation, as vestibular vertigo does.

Are there any associated symptoms? Ear symptoms (associated with vestibular vertigo) include hearing loss, tinnitus, pressure, and distorted hearing. Central nervous system symptoms (associated with central vertigo) include headaches, visual changes, seizures, loss of consciousness, and weakness. Does the patient take any prescription or over-the-counter medications? Aminoglycoside antibiotics are notorious for producing ototoxicity. Alcohol use, smoking, and use of illicit drugs are also important to review.

OFFICE AND LABORATORY EVALUATION

The office physical examination is frequently normal. Nevertheless, it should include a focused neurologic exam, with special emphasis on the acoustic nerve. Hearing should be tested with a tuning fork and by whispering. Vision should be tested as well. Blood pressure should be measured in the seated and standing positions to detect any orthostatic decrease in blood pressure.

The presence and pattern of nystagmus provides more clues. In vestibular vertigo, the eyes drift toward the affected side and quickly return to center; the pattern may also be rotatory, but not purely vertical. Vestibular nystagmus is suppressed with the eyes open and fixating. In contrast, in central vertigo the nystagmus can be bilateral or vertical and is enhanced with visual fixation. Essential maneuvers

to elicit nystagmus include rapid head shaking and positional (static) and positioning maneuvers (involving movement). Visual fixation inhibits nystagmus and vertigo in peripheral vertigo but not in central vertigo; visual fixation can be suppressed for diagnostic purposes with Frenzel glasses. If these are not available, the physician can detect nystagmus by gently palpating the patient's closed eyelids.

A simple test is to ask the patient to read something held steady while shaking his or her head back and forth; inability to do so or loss of visual acuity while doing so (two lines on a visual-acuity chart) indicates a vestibular deficit. Manual head rotation under Frenzel glasses helps in evaluating the symmetry of per-rotatory nystagmus.

Standing on a thick foam pad with eyes closed cancels the visual and proprioceptive input and forces the patient to rely on the vestibular system for balance; if he or she can do this, the vestibular system is intact. (Be prepared to catch the patient if it is not.)

More sophisticated testing involves instilling water into each ear at 17°C and timing the resulting nystagmus on each side. Electronic measurement of nystagmus is called electronystagmography (ENG). However, absent caloric responses do not usually indicate total loss of vestibular function. Still more sophisticated testing uses a computer-driven rotating chair in a darkened room and posturography, which can help determine the level of residual vestibular function. This determines whether vestibular rehabilitation is feasible.

DIFFERENTIAL DIAGNOSIS

Vestibular vertigo is commonly due to cupulolithiasis, Meniere's disease, vestibular neuronitis, or trauma. Central nervous system dizziness could be due to vascular or degenerative processes or seizures. Neoplastic processes do not usually produce true vertigo; the most common tumor, Schwannoma of the acoustic nerve, commonly presents with unilateral sensorineural hearing loss. Metabolic disorders of dizziness include hypoglycemia, vasovagal attacks, peripheral neuropathy, and postural hypotension. Hyperventilation syndrome should always be suspected, and disequilibrium of multiple sensory deficit should be kept in mind, particularly in the elderly. Cardiogenic dizziness, including arrhythmia, syncope, and coronary insufficiency, must also be kept in mind, particularly in high-risk populations. Drug side effects, including those of over-the-counter drugs,

must also be reviewed. Motion sickness can be the cause of dizziness and disequilibrium.

MANAGEMENT

The management depends on the diagnosis. In general, positional dizziness, which is most common, is treated conservatively by home-based vestibular therapy and central vestibular suppressants. Benzodiazepines, the most commonly used medications, should not be used for long periods because they can lead to decompensation; scopolamine is habit-forming and should also not be used in the long term. Benign paroxysmal positional vertigo is treated with home-based exercises or office maneuvers such as the Semont maneuver or the traditional Cawthorne exercises, which fatigue the symptoms and disperse the loose otoconia or cupuloliths away from the cupula of the posterior semicircular canal.

Meniere's disease is treated initially by salt restriction, diuretics, and labyrinthine sedatives, most commonly meclizine. If Meniere's disease is not controlled within 1 year and the level of incapacitation is high, surgical intervention is usually considered. Endolymphatic shunt, labyrinthectomy, and selective

dissection of the vestibular nerve are the most common surgical procedures and produce good control of dizziness in most cases.

Vestibular neuronitis and motion sickness syndrome are treated conservatively with medication and rehabilitation. Central vestibular disorders are more difficult to manage; medical options are limited to central suppression. Vestibular rehabilitation and physical therapy can improve the severity of the symptoms, but there is usually a residual component to central dizziness.

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SELECTED READING

Brandt T. Man in motion: historical and clinical aspects of vestibular function. *Brain* 1991; **114**:2159-2174.

Hamid MA. Vestibular anatomy and physiology. In: Hughes GV, editor. *Textbook of Clinical Otology*. New York: Thieme-Stratton, 1985:165-171.

Hamid MA. Vestibular rehabilitation. In: Myers E, Bluestone C, Brackmann D, Krause C, editors. *Advances in otolaryngology—head and neck surgery*. St. Louis: Mosby-Year Book, 1992:27-36.

