Chapter 145: Otologic and Neurotologic History and Physical Examination

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A history and physical examination of patients with otologic complaints usually reveal the diagnosis or at least suggest the problem before tests are ordered. The history is the more helpful of the two and is extremely important in establishing the course of future testing and the final outcome of care.

History

The patient's complaints are the starting point in the history. By listening carefully to the patient's explanation of symptoms, the physician can ask appropriate questions or ask for elaboration. It is not unusual for patients to forget to mention certain symptoms or to omit them deliberately because they think the symptoms are unimportant.

The history form I use (Fig. 145-1) lists specific questions important for patients with ear problems. Included are questions about the duration of symptoms and if they are progressive; the presence or absence of tinnitus, vertigo, and discharge from the ears; and a family history of ear disease. I also routinely ask if the patient is wearing or has tried a hearing aid or aids.

The otologic history includes a general medical history. Questions address general health, nose and throat problems, allergies to medication, and currently used medications. One should inquire about the patient's previous or current use of tobacco and caffeine because they may be important factors in symptoms. Knowledge of a family history of such diseases as diabetes, heart disease, blood pressure elevation, and syphilis may also be helpful. Past treatment for the problem can help to determine the diagnosis, the suitability of the disorder to treatment, and the eventual course of treatment.

Physical Examination

The otologic examination, like the general physical examination, includes inspection, palpation, and, if indicated, auscultation. The principal purpose of palpation is to locate areas of tenderness, lymphadenopathy, and masses adjacent to the ear. Auscultation is performed with a stethoscope over the mastoid and neck and around the ear. For patients complaining of pulsative tinnitus (vascular or muscular), a Toynbee tube (Fig. 145-2) is used to listen inside the patient's ear.

Inspection

Inspection includes the ear, nose, and throat, with special attention to the ears and adjacent structures. Routine otoscopy is supplemented with pneumotoscopy, auscultation of the ear, and tuning forks. The operating microscope, which allows good illumination and three-dimensional visualization, is often used for the ears.

During otoscopy the examiner's hands should be braced against the patient's head to prevent injury if the patient moves. This precaution is especially helpful with children. Otoscopy should never cause the patient discomfort. The largest speculum that fits comfortably in the external meatus is used. The pinna is gently pulled posterior and superior. The following order of inspection helps to ensure that all structures are covered: the postauricular area, the concha, and the external cartilage. One should look for surgical scars, evidence of trauma, and congenital defects.

The external canal is carefully examined for cerumen, exostosis, evidence of infection, flaking, dry skin, and osteomas. The posterosuperior area of the canal is inspected for a surgically produced mastoid cavity or a cholesteatoma. Wax must be removed so that the underlying area can be carefully inspected.

The tympanic membrane (TM) is inspected for evidence of scars, perforations, tympanosclerosis, and color changes. The normal color is silver-gray or translucent; abnormal conditions alter it. Acute middle ear effusion makes the TM appear amber; chronic middle ear effusion makes it pale and full. The area superior to the lateral process of the malleus is carefully inspected for evidence of a retraction pocket or attic cholesteatoma. Tympanosclerosis of the tympanic membrane itself rarely causes a hearing loss but is a clue to past otitis media. The ossicles can sometimes be seen and assessed through a translucent TM. The color of the underlying mucous membrane of the middle ear can be evaluated. At times the TM is atrophic and retracted into the middle ear. It may drape over the ossicles and into the hypotympanum. In such cases the area of the eustachian tube is inspected for evidence of an air-containing space.

Pneumotoscopy can be performed with either the microscope and a Siegle's pneumootoscope with a nonmagnifying lens, a standard Siegle's pneumootoscope and a head mirror (Fig. 145-3), or an otoscope with a diagnostic head and pneumatic bulb. A small perforation may not be readily visible on routine otoscopy. Knowing the amount of mobility of the tympanic membrane is helpful in determining the presence or absence of fluid. No movement of the tympanic membrane may indicate a perforation under an anterior overhang. Severe scarring of the tympanic membrane or middle ear may become more apparent with no movement of the drum.

Audiogram

Any patient complaining of hearing loss, tinnitus, dizziness, or vertigo should have an audiogram early in the evaluation. This should include air conduction, bone conduction, and speech audiometry.

Tuning-fork examination

A tuning-fork examination is an important part of the otologic evaluation. I routinely use tuning forks to confirm or refute the audiogram. The Weber test is performed with the 512-Hertz (Hz) tuning form (Fig. 145-4, A). The fork is placed in the center of the patient's forehead, on the bridge of the nose, or on the anterior incisor teeth. The patient is asked if he hears a tone in the right ear, left ear, or center of the head. Some patients are unable to localize the sound. The Weber test lateralizes sound to the ear with a conductive hearing loss

of as little as a few decibels. In patients with a severe unilateral sensorineural hearing loss, sound from the test lateralizes to the normal ear. If a unilateral or asymmetric sensorineural loss is mild or moderate, the forks usually do not result in lateralization. patients with a unilateral conductive hearing loss sometimes may hear the tone in the ear with a hearing loss but say that they hear it in the good ear. Such patients may look in the direction in which they hear the sound (the ear with the conductive hearing loss) before they respond. Informing these patients that hearing the sound in their worse-hearing ear is possible may be helpful.

Rinne's test is first performed with the 512-Hz tuning fork. If bone conduction is greater than air conduction (BC > AC), then the 1024-Hz fork is used; if AC > BC, then use of the 1024-Hz fork is unnecessary. The fork is placed firmly over the mastoid bone approximately over the attic (Fig. 145-4, B). The patient compares the loudness of the bone conduction to the loudness of the fork when it is 2 cm from the meatus of the external auditory canal. The tines of the fork are placed parallel to the plane of the external ear canal (Fig. 145-4, C) for optimally loud air conduction. The 512-Hz tuning fork produces BC > AC when the conductive hearing loss is greater than 20 dB, and the 1024-Hz fork produces BC > AC when the conductive component of the hearing loss is greater than 25 dB. Results of tuning form tests indicate the presence and severity of conductive hearing loss. It is not unusual for an audiogram to show minimal or no conductive hearing loss.

Tuning-fork examination also may reveal a unilaterally dead ear that may be a shadow curve on the screening audiogram. Thus it is imperative to include tuning forks as part of the otologic and neurotologic evaluation.

Hearing Loss

When the patient's chief complaint is hearing loss, a history and an otologic and neurotologic examination are necessary to determine other tests that will provide the most information. Serious underlying pathology must be considered and ruled out. Regardless of the cause of the problem, most patients are concerned about their future hearing. Once the problem has been evaluated, an explanation to the patient is very important. Treatment follows the evaluation and explanation.

The duration of the hearing loss and mode of onset are determined through the history. The patient with gradually progressive hearing loss may have difficulty stating the exact time of onset. The patient's relatives can be helpful in determining this because they are usually aware of a hearing problem before the patient is.

Patients with a sudden onset of hearing loss need to be seen early so that treatment, if appropriate, may be begun. The history should uncover any previous hearing problems and whether the current loss is fluctuating, progressive, or associated with vertigo, dizziness, or tinnitus. The history should also reveal other associated factors, such as respiratory tract infection, recent physical trauma or barotrauma, noise exposure, drainage, and ear pressure or pain. Intolerance to loud noise and the Tullio phenomenon, as well as diplacusis, recruitment, and distortion, are usually associated with a cochlear type of hearing loss. A retrocochlear loss is suggested by the patient's switching the telephone from the affected ear to the opposite ear early in the hearing loss. Many patients with acoustic tumors with normal

or nearly normal hearing do this. Therefore one must ask patients with a unilateral problem which ear they use for the telephone and if they have recently switched to that ear. Patients who must stop chewing to listen to a dinner conversation often have a conductive hearing loss, which causes internal sounds to seem louder; also, because the patient's own voice seems loud, he will talk softly. Such patients also seem to hear better in noisy environments (paracusis). The patient with a sensorineural hearing loss speaks more loudly because his voice is softer. With such information, the diagnosis can be suspected before any tests are performed.

The family history is important because many hearing problems are hereditary. The specific mode of inheritance should be sought. Congenital or acquired deficits in other organ systems should be sought. A progressive hearing loss in a patient with a positive family history suggests otosclerosis or other hereditary, progressive losses.

Once the history of the onset of the loss and all the associated symptoms have been recorded, it is important to discuss the effects of the current problem with the patient. How much difficulty is there with communication? Is the hearing loss affecting job performance and socal and family life, or is it only a minor irritation? These questions establish the patient's major concerns and the course of further management.

On completion of the history and examination, a diagnosis is suggested. It may be final or tentative, pending further testing. Discussing initial impressions with the patient is important so that he will understand the need for further testing.

Dizziness and Vertigo

Of all of the problems facing the otologist, dizziness and vertigo can be the most perplexing and frustrating. The history is extremely important to establish the probable cause and character of the problem. Because the patient's definition of dizziness may not be the same as that of the examiner, defining the patient's thoughts and feelings is important. Establishing the nature of the problem often takes great patience and involves spending considerable time with the patient.

The four steps in the management of the dizzy patient are (1) definition of the problem, (2) evaluation of the problem, (3) thorough explanation and reassurance, and (4) treatment and follow-up. These steps help to manage the dizzy patient with maximal efficiency and compassion. Regardless of the cause of the problem, follow-up and emotional support are important.

History

In the history the patient describes the initial episode in detail: how long ago, the time of day, room-spinning vertigo or light-headedness, associated nausea or vomiting, activities at the onset of the attack, duration of the episode, and what the attack caused the patient to do. What happened subsequently? Was the patient completely free of dizziness after the attack, or did it persist as chronic unsteadiness? The current problem may be constant unsteadiness, a light-headed, imbalanced sensation, or episodic true vertigo. Establishing this information initially is imperative because the subsequent management depends on the type of dizziness. If dizziness is episodic, knowledge of frequency, duration, precipitating factors, and abortive factors of subsequent attacks is necessary. Are there symptoms, such as aural fullness, pressure, pain, neurologic symptoms, or loss of consciousness?

Examination

The examination of the dizzy patient includes a neurologic examination. The cranial nerves are checked, including extraocular movements. For details of the ocular motor examination, see Chapter 147. The eyes are observed for nystagmus in various directions of gaze. During cerebellar testing the patient touches the examiner's finger and then the patient's own nose. These movements are repeated several times in rapid succession, first with one hand and then the other.

Romberg test

I routinely perform two variations of the Romberg test. The first is done with the patient standing feet together and arms folded. The patient closes his eyes and tries to maintain balance. The examiner places his hands near the patient's shoulders to prevent a fall. If the patient can perform the maneuver without substantial increase in swaying or movement of the feet to maintain balance, the tandem, or sharpened, Romberg test is attempted (Fig. 145-5). It is similar to the ordinary test, except that the patient places the feet heel to toe. Again, any increase in movement is a positive result. The tandem Romberg test reveals subtle balance deficiencies that may not be detected with the ordinary Romberg test, particularly in patients with acoustic tumors.

Gait

The gait of the patient is observed during the examination and as the patient leaves the examining room. This is done without the patient's being aware that he is being observed. One looks for unsteadiness, staggering, or a broad-based stance during walking. Results may help reveal the patient's ability to function.

Blood pressure

Measurement of the patient's blood pressure may be helpful, especially in older patients and patients with a history of hypertension or cardiovascular disease. If the patient's symptoms are related to posture, blood pressure is measured when the patient is supine, sitting, and standing.

Clinical Manifestations

Certain clinical presentations, although they result from a diverse group of pathologic processes, have the same clinical manifestations. Because the clinical picture is the same, they are discussed in detail here rather than with each disease process that causes them.

Vestibular loss

Sudden unilateral vestibular loss

Whether the cause is blunt or sharp trauma, planned or inadvertent surgical ablation of labyrinthine function, or suppurative or viral labyrinthitis, the total or near-total loss of normal or near-normal vestibular function in a person with normal vestibular function in the opposite end organ evokes the same series of immediate and long-term symptoms. The most severe immediate consequence is marked whirling vertigo, usually with associated nausea, vomiting and retching, and often pallor and diaphoresis. The intensity of the symptoms reaches a maximum within 1 hour of the onset, and symptoms of near-maximal intensity remain for 12 to 36 hours. Toward the end of that time and over the next several days, gradual resolution of the vertigo occurs, although it reappears with any head motion. Most patients are bedridden initially and begin to ambulate with help after 1 to 3 days.

Over the first 2 to 4 days the spontaneous vertigo resolves, and vertigo evoked by head motion decreases. However, motion of the patient or the perception of rapid motion in the environment continue to cause symptoms. Most noticeable are station and gait problems. Sudden movements cause staggering or even falling because of a decreased ability to compensate for the change of position. Activities such as picking something off the floor are especially likely to cause problems.

When locomotion is adequate and the patient has left the hospital, the perception of rapid changes of the environment becomes bothersome. Driving is difficult because of oscillopsia, or one's sensation of the horizon not remaining stable when in a bumpy car; also, there is difficulty feeling confident about the orientation on the highway as oncoming traffic approaches. Walking rapidly down the aisle of a supermarket past objects on the shelves also causes uncertainty of position and disequilibrium.

All these symptoms gradually improve over time, so that after such a severe episode most patients are able to leave the hospital in 5 to 10 days and are nearly symptom free most of the time after 2 to 3 months. However, differences among patients exist with respect to the rate of recovery and the degree of ultimate functional recovery. Younger individuals characteristically recover more quickly and achieve a better end result than older patients do, and those who experience a good deal of vestibular stimulation in their day-to-day lives (eg, those active in athletics) also fare better. However, all patients have an increase in their residual symptoms with fatigue, with loss of visual orientation such as occurs in the dark, and when proprioceptive influences are changed, such as occurs with walking on nonlevel ground. The young, vigorous patient who has a nearly complete recovery from such a sudden unilateral vestibular loss can, however, arise at night and walk across a dark room with only a slight sense of uncertainty of footing, whereas an older person may fall unless a low-level light is provided for visual orientation. The extremes of individual responses to sudden loss of unilateral labyrinthine function range from nearly total freedom from symptoms except under adverse situations (fatigue, darkness, irregular ground) to an inability to ambulate safely without a walker despite optimal environmental conditions.

Gradual unilateral vestibular loss

If the process causing the unilateral vestibular deficit has a gradual onset, such as occurs with a space-occupying lesion of the vestibular nerve or internal auditory canal, and this process takes months to eliminate vestibular function totally, no vertigo is perceived. The resulting vestibular symptoms are similar to those experienced 2 to 3 months after the onset of a sudden loss and are primarily related to gait and station instability, especially noticeable under the adverse environmental conditions just noted. In general, patients have fewer long-term deficits from the gradual loss of unilateral vestibular function than from sudden loss. In fact, the only complaints may be related to waling in darkness or insecurity when fatigued.

This information can be used to predict the amount of short- or long-term postoperative difficulty patients will experience after surgical ablation of one labyrinth. If there is nearly symmetric vestibular function preoperatively, the maximum of symptoms can be expected; the ability of the individual to compensate can be estimated by the age and general physiologic status. However, if severe hypofunction of the end organ to be ablated exists (and the opposite side is normal), little augmentation of symptoms will occur becase most have already been compensated for. If there is total lack of response to maximal caloric stimulation, usually very few changes occur in the symptoms after surgical ablation. Thus the caloric responses of the two ears provide information that is helpful in the counseling of patients considered for labyrinthectomy, vestibular nerve section, or vestibular schwannoma resection about their postoperative courses.

Gradual symmetric vestibular loss

The bilateral simultaneous loss of two functioning labyrinths is usually not sudden because if trauma is sufficient to cause such a loss, coma or obtundation often is present also. However, vestibulotoxic drugs commonly cause symmetric labyrinthine loss. The onset of symptoms is not abrupt, and the maximal intensity of the symptoms occurs a few days after the onset of symptoms. As seen with patients who have sudden unilateral vestibular loss, the severity of symptoms, the rate of recovery, and the degree of ultimate recovery of these patients are influenced by their age, general health, and activity level. The symptoms do not include spontaneous vertigo but are restricted to station and gait problems, particularly in response to head or body motion. Maximal difficulty in susceptible individuals precludes ambulation without assistance or a walker. Young, healthy subjects merely stagger. Greatest improvement occurs in the first 2 to 6 weeks. By 6 months after the loss, young, healthy individuals walk with a wide-based gait and can compensate for most situations, although bending over to pick objects off the floor and rapid changes of body position are likely to remain difficult. Less fortunate patients are more restricted and require at least a cane for stabilization during routine walking.

Tinnitus

Tinnitus is associated symptom in 85% of patients with otologic disease. This section discusses the patient who seeks help because tinnitus is the primary concern, but the reader is also referred to Chapter 173. The physician must remember that tinnitus is a symptom, not a disease. A history should include the duration and location of symptoms (bilateral, unilateral, or in the head), sudden or gradual onset, and intermittent or constant symptoms.

The character of the tinnitus may help to establish its cause. If it is pulsatile and seems to correspond to the pulse, its origin is probably vascular.

The subjective severity of the tinnitus is important. Most patients are not particularly bothered by the problem but want to know if it is significant. About 5% of patients are quite disturbed by tinnitus. Does it keep them awake or interfere with their ability to concentrate during the day? If it does interfere with sleep, does it cause them trouble getting to sleep or does it awaken them during the night and cause difficulty getting back to sleep? The former pattern is associated with anxiety, the latter with depression. What factors influence tinnitus: noise, anxiety, tension, lack of sleep, fatigue? What lessens the tinnitus: relaxation, sleep, distraction?

The examination includes careful otoscopy to rule out glomus tumor or other vascular lesion in the middle ear. A common problem that may cause tinnitus is an abnormally patulous eustachian tube. In such a case the physician may observe movement of the tympanic membrane with respiration. Auscultation is important in patients with tinnitus. I use a Toynbee tube to listen to the involved ear. A stethoscope is used to auscultate the neck and area over the mastoid for bruits. Palatal myoclonus usually exists as a clicking sound in one or both ears; therefore the soft palate is carefully observed. If the patient is symptomatic during the visit, the palate can be seen rhythmically contracting, synchronous with the patient's tinnitus. Often the palatal contractions stop if the mouth is opened widely; to prevent this from happening, the palate should be observed with the mouth only slightly open.

Pain

The origin of ear pain may be otologic, dental, neurologic, or pharyngeal. The patient describes its site, time of onset, character - constant or intermittent, shooting, brief - and whether it is located only in the ear or radiates elsewhere. It is helpful for the patient to point to the center of the painful area. Has the pain existed in the past? Is it associated with aural discharge, tenderness of the external ear, fever, or itching?

The external ear is carefully palpated to determine if there is any tenderness. The temporomandibular joints are also palpated while the mouth is open and then closed. Tenderness indicates possible temporomandibular joint dysfunction. Worn-down facets on molars or incisors may also help to establish a cause: such patients may grind their teeth during sleep or stress.

The ear canal is carefully inspected for signs of swelling, erythema, excoriation, or discharge. The tympanic membrane is examined to rule out bullae on the surface; a red, bulging membrane indicates acute otitis media. The pneumootoscope is helpful in differentiating between acute bullous myringitis and acute otitis media: the tympanic membrane is immobile in acute otitis media and mobile (despite the bulging bullae on its surface) in early bullous myringitis, although later an effusion may also exist.

Otorrhea

When liquid drains from the external ear canal, the physician should seek to determine the type of liquid, its source, and the nature of the pathologic process causing it. Differences in color, clarity, viscosity, and odor are obvious and important. The temporal pattern of the drainage is also important, as is the relation of that pattern with associated symptoms, such as pain or fever.

The recognition of blood is easy. Blood may, however, be mixed with cerebrospinal fluid (CSF). Detection of the latter is important because of the increased risk of meningitis with persistent CSF leakage. Initially the physician may note only that the blood seems thin. If copious, the fluid may be collected for centrifugation to determine the hematocrit for comparison with venous blood. It can also be allowed to drop onto filter paper, in an attempt to elicit the owl or halo sign, a ring of clear wetness surrounding the circle of blood-stained moisture, which is caused by a greater diffusion of spinal fluid than blood. Sometimes it is difficult to distinguish CSF from thin serous fluid in some individuals with tympanic membrane perforations and inflammatory middle ear and mastoid disease. When the drainage is profuse, the distinction can easily be made by collecting several drops, determining the glucose concentration, and comparing it with the blood glucose level. When the drainage is scant, prolonged positioning may be required to collect enough fluid for analysis.

When the otorrhea is purulent, clinical characteristics may help to determine appropriate therapy. A foul odor from the drainage probably represents a mixed infection of anaerobic bacteria or a combination of anaerobic and aerobic organisms. If systemic antibiotics are used, agents that are active against anaerobic bacteria are appropriately chosen. If the drainage has a black color, it represents a fungal infection; consequently, the use of steroid antibiotic eardrops will be ineffective, and topical antifungal agents should be employed.

Visual symptoms

The physician should ascertain whether the vision is blurred during dizzy spells because brisk nystagmus is always accompanied by blurring images or at least a noticeable shifting of the visual field. Oscillopsia has been discussed with vertigo and disequilibrium. Diplopia as a manifestation of petrous apex disease with sixth cranial nerve involvement can be an important symptom.

Other visual aberrations are important because they may provide clues to the diagnosis of migraine headaches that can be associated with otologic symptoms (see Chapter 182). Scintillating scotomas are frequent and are sometimes described as similar to flickering fluorescent lights. Other patients notice shimmering or jagged flashes of light at the periphery of the visual field, which they liken to lightning. Transient peripheral visual field defects may exist, but they do not involve macular vision.

Central nervous system changes

Headaches are an obvious feature of migraine disease, but the headache pattern may be different from the typical hemicranial headache of classic migraine (see Chapter 182). Mental deterioration is important to notice because it is not a part of otologic disease; when it is present, search should be made for a central nervous system explanation.

Summary

A careful history and an otologic and neurotologic examination usually suggest a diagnosis for the patient with an otologic disorder. The history is the most helpful of the two in the final diagnosis. The otologic and neurotologic examination is systematic and thorough. Results indicate further appropriate tests to be taken, including special audiologic and radiographic examinations.