

VESTIBULAR AND AUDITORY SYSTEMS

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I. VESTIBULAR SYSTEM

To maintain his upright, two-legged posture, man must have very precise information about his position in space. Visual clues provide probably the most important information. Next in importance are proprioceptive responses from the neck and leg muscles, and, lastly, the impulses from the vestibular system play a part in some righting responses. The integration of all of these types of input will be considered in Chapter 19, Motor Function. While human does not utilize the vestibular system to the same extent, as do animals, dysfunctions of this system are quite serious. In addition, several promising methods for rehabilitant patients with brain damage, particularly children, depend upon knowledge of the vestibular system.

SENSORY RECEPTORS

Semicircular canals. The receptors of the vestibular system, the semicircular canals and the maculae are located in the inner ear in close approximation to the organ of hearing, the cochlea. The primary nerve cells are in the inner ear; the axons enter the brain stem as cranial nerve VIII and end in the vestibular nuclei of the same side. Each set of semicircular canals consists of three

canals in mutually perpendicular planes that interconnect at the utricle. The spatial location of each canal is shown in Figure 14-1. Tracing and cutting out part C of this figure should be of great help in understanding the relationship of each canal. The sensory receptors in these canals respond to angular acceleration (rotation) in the plane of the canal. For the rest of this discussion, we will concentrate on the horizontal canals; the anterior and posterior canals function in the same manner.

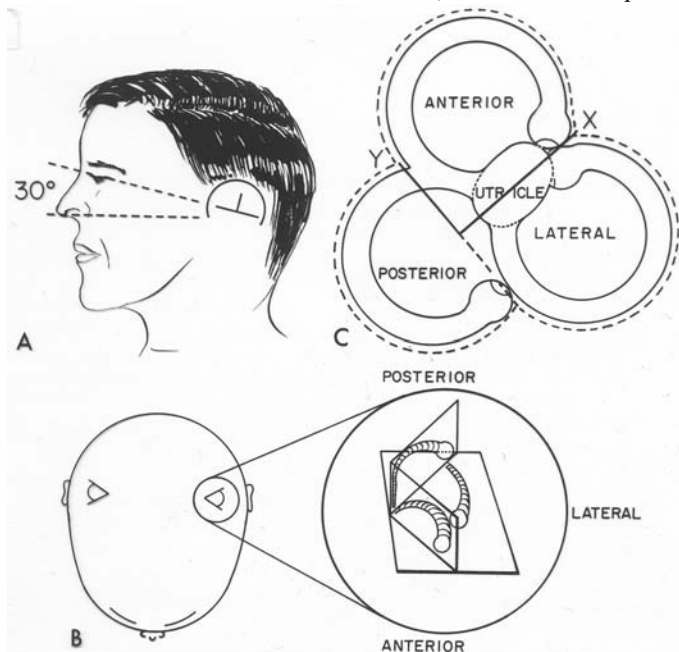


Figure 14-1, Parts A and B show the location of the semicircular canals in the head. Part C is a diagram to help in understanding the three-dimensional structure of the canals. Trace the lines on a sheet of paper and cut along the dotted lines; fold at X and Y. (Part C is based on Lithgaw, J.D.: J. Laryng., 35:81, 1920.)

which is of high potassium and low sodium concentration.

Figure 14-2 shows a section through the right horizontal canal as well as the utricle and a third structure, the saccule. These structures are filled with an isotonic solution, *endolymph*,

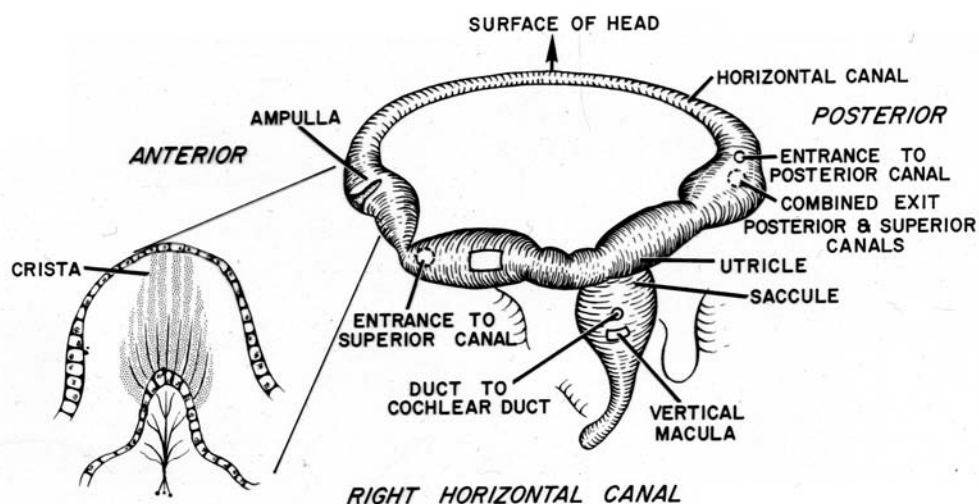


Figure 14-2. A view of the right horizontal canal, the ampulla and crista and the two right maculae

Hair cells. The primary receptor of both the auditory and vestibular systems is the hair cell. Each hair cell, Figure 14-3A, has a single, long kinocilium and about 60 shorter stereocilia that arise from a specialized cuticular plate. Both types of cilia project into the potassium rich endolymph. The other end of the cell contains prominent vesicles of an unknown neurotransmitter and synaptic junctions with afferent axons. Many hair cells also receive efferent axons. The afferent axons are tonically active; displacement of the cilia toward the kinocilium increases activity while displacement away reduces activity in the afferent nerve.

An ionic flow circulates, Figure 14-3B, from the endolymph, which is maintained at +80 mV by an unknown active transport process, through the specialized cuticular plate below the stereocilia and out the side of the hair cell to the extracellular space. Channels connected to the stereocilia modulate this current. These channels do not discriminate between cations very well but since both the endolymph and intracellular fluid are predominately potassium, the potassium ion carries most of the current driven by the difference in voltage between the endolymph (+80mV) and the cell interior (-60mV). As the stereocilia are bent toward the kinocilium, channels open and a greater ionic current flows. As this current crosses the peripheral membrane, it depolarizes the hair cell, opens calcium channels and more neurotransmitter is released, thereby increasing the rate of firing of the afferent fibers.

The sensory receptor is in the ampulla and consists of the projections of many hair cells embedded in a gelatinous mass, the *crista ampullaris* (Fig. 14-2). The hair cells are of two types. One is bottle shaped and is almost completely surrounded by a chalice shaped neuron. The other is long and slender and the nerve makes contact only at the base. There are efferent connections on many of these afferent neurons. There is some evidence that these efferents may inhibit the primary sensory endings to lower their sensitivity. The crista forms a sliding seal with the opposite wall of the ampulla so that any movement of the fluid relative to the base of the crista will cause the crista to bend, deform the hairs, and fire the nerves. At the beginning of a rapid turning of the head, the base of the crista moves, but the endolymph does not because of its inertia. Consequently, the crista is bent (Fig. 14-5A). The nerve fibers from the crista are tonically active; they fire continuously even though there is no stimulus. When the crista is bent toward the utricle, the frequency of firing is increased. When the crista is bent away from the utricle, the frequency decreases.

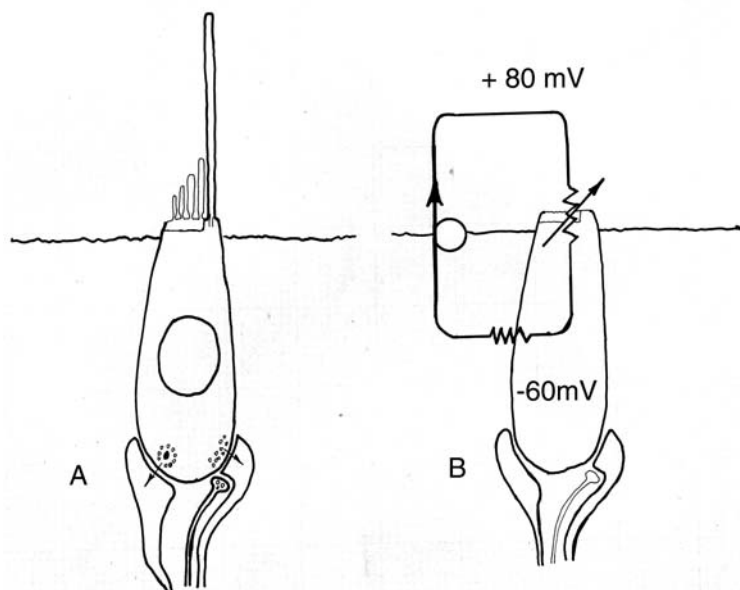


Figure 14-3, A hair cell of the vestibular or auditory system. A) The cilia project into the endolymph and are bound together into a gelatinous mass. The single, long kinocilium attaches directly to the cell while the 40-80 stereocilia insert into a cuticular plate. Motion of the cilium toward the kinocilium stimulates the 10-20 afferent nerves attached to the base by release of neurotransmitter from adjacent vesicles.

B) A continuous flow of ions, driven by active transport, circulates between the endolymph, hair cell

interior and the extracellular space. Channels attached to the stereocilium modulate this current. As the stereocilium moves toward the kinocilium, more channels open and more neurotransmitter is released.

Records of firing, not of the vestibular nerve, but of a single unit in the vestibular nucleus in the cat are shown in Figure 14-4. As the cat is rotated to the right, the discharge is intense and lasts a short time on the swing back, but it dies out quickly as the cat is rotated to the left. The discharge from the other horizontal canal will show the reverse pattern. While not obvious in Figure 14-4, the receptor adapts fairly rapidly to rotation; the firing frequency returns to normal about 5 seconds after the rotation (as shown in Fig. 14-5) begins.

The lack of response to continued rotation is easy to understand; because of friction, the endolymph rapidly begins to rotate at the same speed as the head. Now there are no sheering forces on the crista (Fig. 14-5B), and the nerve discharge returns to normal.

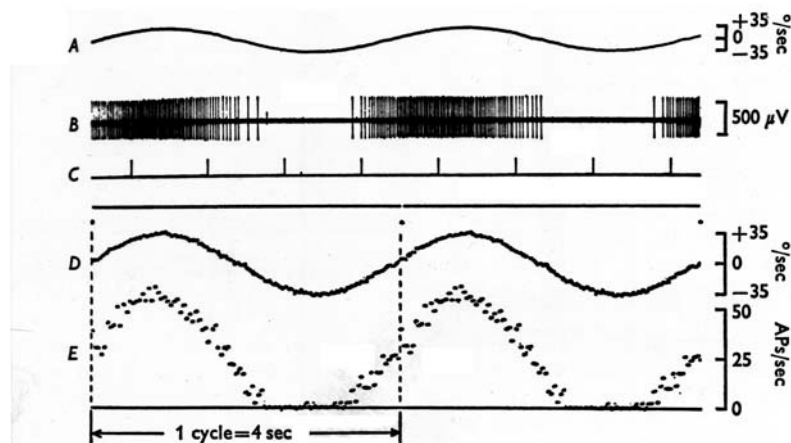


Figure 14-4. Response of a canal-dependent cell to angular oscillation in the plane of the lateral canals. A, angular velocity signal; B, action potentials from microelectrode in the left medial vestibular nucleus, and C, seconds. The lower half of the figure shows the average stimulus (D) and average action potential frequency obtained over 10 cycles (E). (From Benson, A.J., Guedry, F.E., and Jones, M.J.: J. Physiol., 210:475, 1970.)

When the body and the head stop rotating, the endolymph continues to rotate or to try to rotate; a body in motion tends to remain in motion. The flow of endolymph bends the tip in the direction of motion (Fig. 14-5C) and causes a change in the nerve output. The crista bent toward the utricle is excited. The flow, or pressure to flow, rapidly ceases and the crista returns to normal.

A different set of terminology has been developed which is most useful when the head is stationary and endolymph is flowing. If the endolymph is flowing toward the ampulla, as happens on the left side at the end of a clockwise rotation (Fig. 14-5C), the crista is excited and the flow is called *ampullipetal*. In the same circumstances, the flow in the right canal is away from the ampulla, *ampullifugal*, and the crista is inhibited.

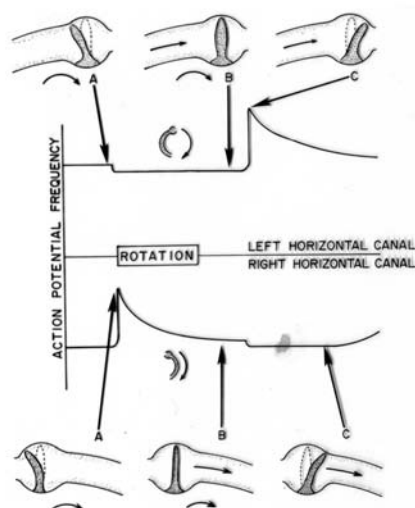
The deflection of the crista in Figure 14-5C is identical to the pattern that would be seen at the beginning of a counter-clockwise rotation. The end organ has no mechanism for differentiating between the start of a counter-clockwise rotation and the end of a clockwise rotation. One method of showing this is to observe eye movement.

CONTROL OF EYE MOVEMENT

The output of the semicircular canals goes directly to the *vestibular nuclei* (Fig. 14-6). Discrete portions of the nuclei respond to rotation in each plane. The vestibular nuclei influence a number of types of movement; one of the most important is eye movement, hence major projections, via the MLF, to CN III and VI. Our heads are constantly in motion as we walk down the street, yet we have no impression of the world jiggling up and down in front of us. Persons without vestibular function often have just such a problem. They may fail to recognize their friends because faces are blurred. As we turn our heads, we can keep our eyes on an object. These and other eye movements show the influence of vestibular information.

This can easily be shown in a horizontally rotating Bárány chair. Upon slow clockwise rotation, the eyes move counter-

Figure 14-5. Below. The response of the horizontal canals to the beginning, continuation, and cessation of rotation. Note that when the sensory cells of one crista are stimulated, the sensory cells of the other are inhibited. (After Adrian, E.D.: J. Physiol., 101:389, 1943.)



clockwise to keep affixed object in the visual field. The output from the vestibular nuclei to the medial longitudinal fasciculus (MLF) and the nuclei of nerves VI and III signals clockwise rotation; the response is anti clockwise eye movement.

Consider now the case of stopping suddenly after rotation; the output of the vestibular nuclei signals anti clockwise movement, so the response is clockwise eye movement. As the eyes move slowly clockwise, a stationary image moves out of the visual field and the eyes snap back to the center, find the image, and begin to move slowly again. This is the pattern of nystagmus. The direction of the nystagmus is named for the quick component. The physiologically meaningful component is the slow one.

Nystagmus. Nystagmus describes a rhythmic oscillation of the eyeballs, characterized by a slow drifting from a central gaze toward the periphery and then a sudden snap back to a central gaze position. Both eyes usually move together; it is a conjugate movement and is usually repeated many times. The easiest way to visualize this condition is to find a willing subject and have him/her look at a striped necktie held horizontally. It is important that the subject does not fixate on a single line, but gazes at your face. Move the necktie horizontally to your left. You should observe a slow movement of the subject's eyes to the right and then a quick snap back to the central position; this pattern will repeat several times as you move the necktie. Nystagmus is named by the quick beat; this example is described as a left-beating nystagmus. You are observing *optocokinetic nystagmus*, but the eye movement pattern is the same for all types of nystagmus. A drum with vertical stripes is often used for testing. Optocokinetic nystagmus is mediated by the occipital lobe although the pathway to the lateral gaze center is not clear.

The nystagmus that originates from the vestibular apparatus has exactly the same form--slow movement followed by a quick jerk. The output of the vestibular nuclei enters the MLF, impinges on the lateral or vertical gaze center, and causes eye movement. Horizontal nystagmus is easily elicited, as is vertical or rotatory nystagmus. Vestibular nystagmus occurs in a dark room or a blind person and involves pathways entirely within the brain stem.

Caloric testing. The function of one horizontal semicircular canal can be determined through caloric testing, whereby flushing ice water through it cools the external auditory meatus. When the subject lies supine with his head flexed 30 degrees, the horizontal canal is vertical, and the part of it closely adjacent to the external auditory meatus can be cooled. Cooling the endolymph in the outer portion of the right canal causes it to become more dense and sink, bending the crista away from the utricle. The same deflection could be produced by an angular acceleration to the left. Both stimuli produce a left beating (quick component) nystagmus. Warm air or water instilled into the auditory canal will give a nystagmus toward the side of warm water infusion. Since this pathway is completed within the brainstem, it is useful in testing brainstem function in comatose patients, although the response is usually a long lasting deflection. Both sides should be tested, but with a 10 to 15 minute pause between to allow the first to return too normal.

Subjective Responses to Rotation. When a volunteer closes his eyes and is spun slowly in a Bárány chair, they have no trouble identifying the direction of rotation. Even the direction of very slow rotation (1/2 degree per second, 12 minutes per revolution)

will be correctly identified. After 20 seconds of a moderate spin, he is less certain about the direction and as he slows down, the confusion increases. At some point, while still turning, he has a very strong impression that he is turning in the opposite direction.

Students often said, "I know this sounds funny, but I am sure I am turning the other way." At that point, the endolymph is rotating faster than the head and the crista is bent in the opposite direction as in Figure 14-5C. If the subject is stopped very quickly, the subjective impression of rotation in the opposite direction is very strong. There will be a brisk nystagmus, *post rotatory nystagmus*. Other outputs from the vestibular nuclei, such as to the MLF for eye movement and to the cerebellum for coordination, also show this disorientation upon the sudden cessation of rotation.

The cerebellum has rich connections with the vestibular nuclei, and the input from the vestibular nuclei is used to alter movement patterns in response to spatial movement. A simple example is the ability, when the eyes are closed, to raise the arm from a horizontal to a vertical position and return it to the same horizontal position. If the person rotates clockwise between the raising and lowering, the hand will have to move to the left to come down in its original position. After a long clockwise rotation, when the person has a strong impression of moving counterclockwise, the hand will come back down to the right of its initial position; this is termed past pointing. The output of the vestibular nucleus has convinced the cerebellum that the body is moving counterclockwise. The same problem occurs when the subject tries to walk straight toward a distant object after spinning. He thinks he is turning in the opposite direction and corrects accordingly.

Natural situations rarely cause this type of postrotatory confusion, and most of us have not adapted to it. Two types of performing arts have developed rapid turns: ballet and figure skating. These artists are trained to reduce postrotatory confusion. The ballet dancer rarely makes many rapid turns in succession, but employs a technique called spotting to help maintain balance after turning. At the beginning of a clockwise turn, the dancer will turn her head maximally over the right shoulder, in the direction of movement, will find a prominent object, and, as she turns, will move the head to keep the object in the visual field. When the head is finally turned over the left shoulder, she will snap it around and find the object again. She substitutes a strong visual image for misleading vestibular information.

Champion figure skaters spin much faster and are unable to spot on each turn. They develop, through much practice, the ability to completely disregard the vestibular output at the end of spinning. I had the opportunity, a good many years ago, to work with a medical student, Lorraine Hanlan Comaner, who had been an Olympic figure skater. When she was rotated very rapidly in the direction she had been trained to spin, she showed no nystagmus after stopping, even if her eyes were closed during rotation. She showed no past pointing and could walk straight toward a distant object. After the same rotation, an untrained person would be totally disoriented. When she was spun in the other direction, she showed minimal nystagmus or past pointing and deviated only slightly in walking. Her vestibular apparatus was perfectly normal by caloric testing.

The "person in space" program has revived an interest in vestibular physiology since spinning; weightless space vehicles produce very unusual vestibular stimulation and resulting disorientation. Most of the astronauts have suffered motion sickness early in their flights.

MACULAE

The semicircular canals are acceleration or dynamic receptors. The two maculae, (Fig. 14-2), one in the utricle and one in the saccule, are static receptors; they continuously signal the position of the head. They may also serve as vibration receptors. The hair cells are covered with a gelatinous mass, the otolithic membrane, in which are buried otoliths or crystals of calcium carbonate. Whenever the head tilts, the weight of the otoliths causes the gelatinous mass to shift and puts different stresses on the hair cells. Each hair cell is sensitive to movement in only one direction. When the hair cells bend toward the longest cilia, known as the *kinocilium*, the discharge rate increases; when the hair cells are bent away from the kinocilium, the discharge rate is inhibited. The kinocilia have different orientations in various parts of the macula so that tilt in various directions can be perceived. Recordings from the macular nerve show altered, continuous nerve discharge after the head is tilted to a new position.

The macula is a static receptor; it continuously provides information on the location of the head, as well as on changes in position. In erect posture, the macula of the utricle is approximately horizontal and that of the saccule, vertical.

CONNECTIONS OF THE VESTIBULAR NERVE

Vestibular nuclei. The vestibular portion of the Eighth Cranial Nerve enters the upper medulla and distributes to the four secondary vestibular nuclei (Fig 4-6) that form the vestibular prominence on the floor of the fourth ventricle lateral to the sulcus limitans. Some primary vestibular fibers also enter the cerebellum via the juxtarestiform body and distribute ipsilaterally in flocculus, nodulus and uvula.

There are four vestibular nuclei; lateral (Deiters), medial, superior, and inferior. The lateral vestibular nucleus gives origin to ascending fibers that cross and enter the MLF and reach CN III and IV. The lateral *vestibulospinal tract* originates exclusively from the lateral vestibular nucleus and descends ipsilaterally to exert a facilitory influence on the extensor motor

neurons.

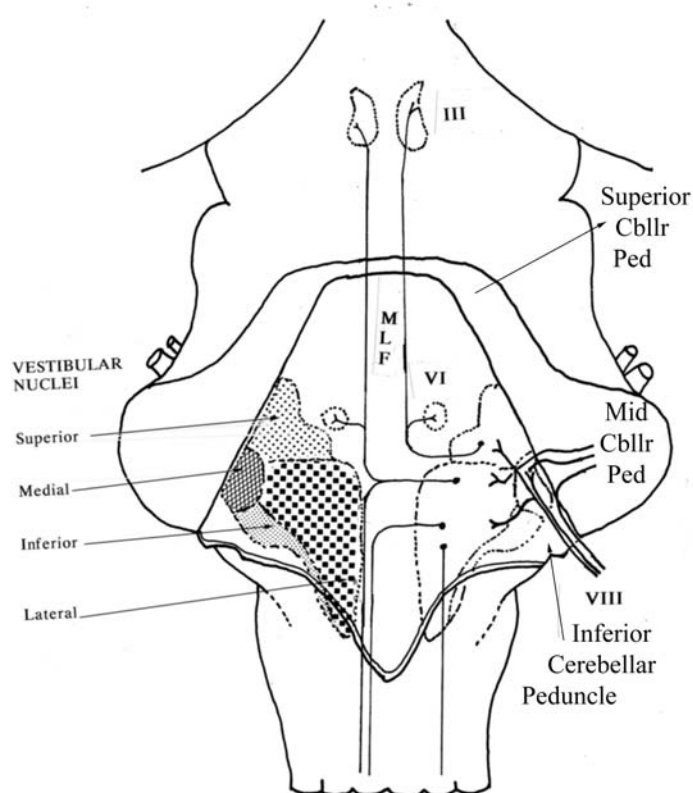


Figure 14-6. The four vestibular nuclei and their major connections are shown just below the floor of the 4th ventricle on this posterior view of the brain stem.

Fibers from the superior vestibular nucleus ascend ipsilaterally in the *MLF* to cranial nerve nuclei of III, IV, and VI. The medial vestibular nucleus sends fibers bilaterally to the abducens nucleus and fibers ascend and descend bilaterally from this nucleus in the *MLF* reaching cranial nerves III and IV. Fibers also descend into the spinal cord to reach ventral horn cells controlling muscles in the neck and shoulder. The inferior and medial vestibular nuclei project ipsilaterally into the cerebellum via the juxtarestiform body and the fibers then distribute bilaterally in the floccular-nodular lobe and uvula and onto the fastigii nucleus. Cerebellovestibular fibers distribute from the anterior lobe and vermis and terminate in the dorsal portions of the lateral and inferior vestibular nucleus. The fastigial nucleus of the cerebellum sends fibers into the lateral vestibular nucleus.

The vestibular nuclei project to the cortex of the temporal lobe by unknown pathways and are no doubt responsible for the conscious sensations of vertigo and dizziness.

DISTURBANCES OF THE VESTIBULAR SYSTEM

Motion Sickness. The most common affliction related to the vestibular system is motion sickness---air, car or sea--that is characterized by vertigo, nausea, and vomiting. These symptoms are all caused by continual periodic motion; it seems likely that the repeated vertical movements are primarily responsible. Most people quickly adapt to the motion. In severe cases of seasickness, however, the unfortunate person's major fear is that he *isn't* going to die. Motion sickness can usually be avoided by eating a light, fat-free meal, applying a scopolamine patch or taking one of a number of antihistamine compounds, such as meclizine (Bonine) or dimenhydrinate (Dramamine), before traveling. These compounds do little good after the symptoms have appeared.

Vertigo. Intermittent attacks of vertigo, the false sensation that the person or his surroundings are whirling around, constitute a common problem associated with the vestibular system. These paroxysmal attacks can be very disabling since the patient is often thrown to the ground by his own reactions to these false clues of movement. Single attacks may occur in acute labyrinthitis.

Ménière's disease is one common cause of intermittent attacks of vertigo. Ménière's disease also attacks the organ of

hearing and results in tinnitus, a constant ringing or humming in the ears; it is of unknown origin. There are a number of medical treatments for it. The surgical treatment is removal of the labyrinth or partial section of nerve VIII. Tumors of nerve VIII and prolonged treatment with streptomycin may also cause vertigo.

Labyrinth Destruction. Rapid destruction of one labyrinth causes disturbances of equilibrium, some vertigo and nystagmus, and, occasionally, nausea and vomiting. The vestibular nuclei apparently work by comparing the signals from both labyrinths, a sort of differential amplifier. When one labyrinth is destroyed, the vestibular nuclei apparently overcompensate for the input from the other side.

Bilateral destruction of both labyrinths does not cause nystagmus or vertigo but a disturbance of equilibrium often lasting many months. Under water, patients with this disturbance are totally disoriented.

Vestibular deprivation in animals is usually much more severe than in man, probably because animals normally rely more heavily on vestibular and less on visual input than man does.

II. AUDITORY SYSTEM

EXTERNAL AND MIDDLE EAR

The external ear consists of the cartilaginous pinna and the acoustic meatus. The *pinna* plays a role in collecting sound waves from the environment and channeling them into the other portion of the external ear. The external *acoustic meatus*, or ear canal, is about one-quarter of an inch (10 mm) in diameter and 1 inch (25 mm) long and ends blindly at the tympanic membrane (Fig. 14-7). It serves as a resonator for the average sounds of human speech (2500 to 8000 cps), and increases the sound pressure on the tympanic membrane two-fold for tones within this frequency range. In response to sound pressure, the *tympanic membrane* vibrates back and forth; compressions and rarefactions in the air are translated into movements of the membrane. In response to the sound pressure of a whisper, the membrane moves only a few angstroms.

The bones of the middle ear--the *malleus*, the *incus*, and the *stapes*--serve mainly as a lever to transform the large amplitude movements of a large diameter structure, the tympanic membrane, to low amplitude movements of a small area, the *oval window*. In the process, the force of movement is increased as much as 90 times. This increase in force can be described as a mechanical lever and serves as an impedance match between the low density and high compressibility of air and the high density and low compressibility of the fluid in the cochlea. The three bones are held in place by connections of the malleus to the tympanic membrane and of the stapes to the oval window and by five thin ligaments.

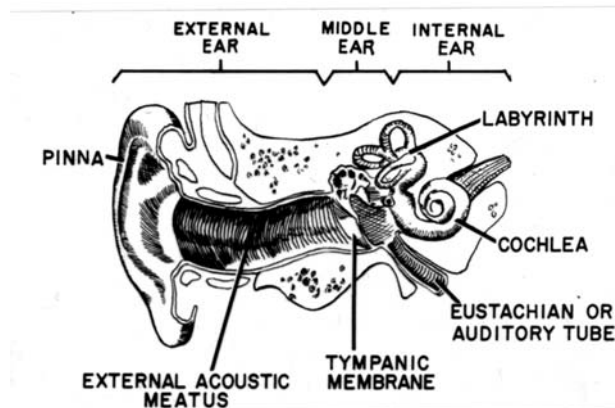


Figure 14-7, The major structures of the auditory receptor.

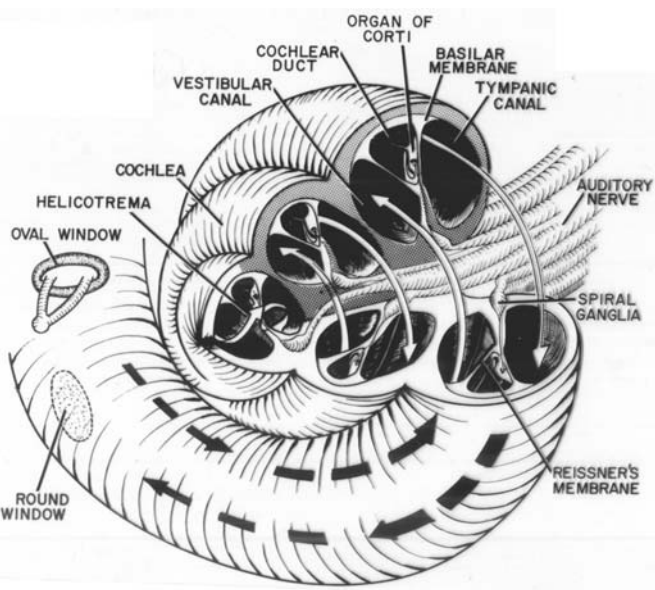


Figure 14-8. The cochlea

While not shown on Figure 14-7, the *chorda tympani* nerve crosses the middle ear just behind the malleus. When the middle ear is infected, transmission of taste impulses from the anterior two-thirds of the tongue may be blocked here on their way to cranial nerve VII and the tractus solitarius of the medulla.

INTERNAL EAR

The stapes transmits the vibration of the tympanic membrane to the oval window of the cochlea (Fig. 14-8). The *cochlea* is a spiral organ containing three cavities that spiral together. The function of the canals will be easier to understand if the spiral is unrolled (Fig. 14-9A). The cochlea is filled with fluid and embedded in bone. There are only two elastic points--the oval and round windows. Whenever the stapes compresses the oval window, the round window must bulge out since the rest of the structure and the fluid are incompressible. There are two routes by which a fluid wave can pass from the scala vestibuli to the scala tympani: through a hole (the helicotrema) at the extreme end of the spiral or through a deformation of the basilar membrane. Deforming the basilar membrane transmits most of the energy. (Reissner's membrane seen in Figure 14-9 is too thin to warrant any consideration in this context.)

The *basilar membrane* is not uniform along its length; at the base it is very light and narrow while at the apex, it is thick and wide. Hydraulic pressure waves produce a rippling of the basilar membrane (Fig. 14-9B). The position of maximum movement depends upon the frequency of the sound (Fig. 14-10). The light, thin end moves when the sound is high pitched while the thicker, wider end at the apex vibrates in response to low pitched sounds. Any sound activates a considerable fraction of the total basilar membrane and the amplitude is proportional to the intensity of the sound. The beginning of frequency discrimination occurs on the basilar membrane yet is not completed there.

Movements of the basilar membrane are transformed into action potentials at the *organ of Corti*, which lies on the basilar membrane (Fig. 14-11). The major sensory cells are three rows of outer hair cells and one row of inner hair cells. The hairs from the cells just touch the gelatinous *tectorial membrane*. Since the basilar membrane and the tectorial membrane are hinged at different points and are of different mass, it is thought that vibration of the whole complex gives rise to differential movement of the two structures and places a shearing force on the hair cells. These cells respond in a similar manner to the hair cells in the macula, so bending is translated into action potentials.

A fascinating aspect of the physiology of the Organ of Corti is the composition of the fluid surrounding it. The space between the Organ of Corti and Reissner's membrane, the cochlear duct (Fig. 14-9), is filled with a high potassium, low sodium solution, and endolymph. The other two chambers, the vestibular and tympanic canals are filled with a CSF like, high Na^+ , low K^+ solution. The predominate anion in both solutions is chloride. There is normally a +90mV voltage between the endo and perilymph and between the endolymph and blood. When the region is anoxic the potential falls in 2 or 3 minutes to -40mV; close to the potassium equilibrium potential. A voltage that is so dependent upon an energy source strongly suggests an electrogenic potential. The source of this potential appears to be a richly vascular region within the cochlear duct, the stria vascularis. What role this potential plays in action potential generation is not clear.

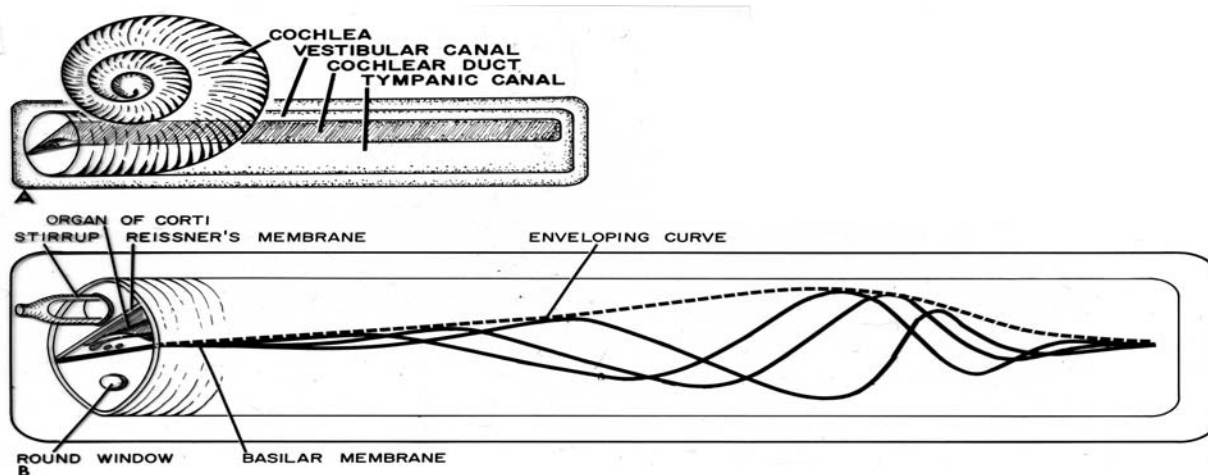


figure 14-9. A shows how the cochlea can be unrolled to give a clearer idea of the function of the basilar membrane. When the stirrup vibrates, the basilar membrane vibrates also; the location of the peak of the enveloping curve varies as the pitch of the sound varies, as shown in B.

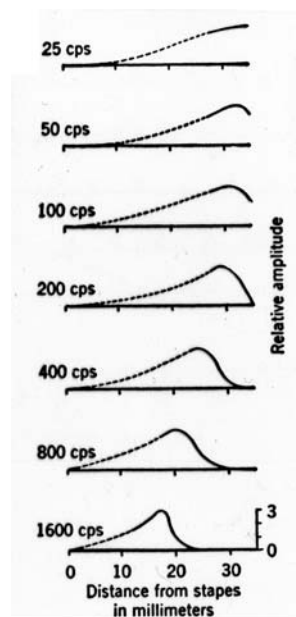


Figure 14-10. The amplitude of displacement of the basilar membrane for constant amplitude and different frequencies of stapes vibration. The solid lines were measured; the dotted lines were extrapolated from other observations. (From Békésy: Handbook of Experimental Psychology. New York, John Wiley and Sons Inc., 1951.)

AUDITORY PATHWAY

The nerves from the hair cells are bipolar and have their nuclei in the spiral ganglia close to their origins (Fig. 14-8). Their axons form the acoustic portion of nerve VIII. Recordings from a single auditory nerve fiber (Fig. 14-12) show it to respond to a wide range of frequencies; the greater the sound intensity, the greater the range. Other fibers have different frequency responses. This is consistent with the hair cell responding to movements of the basilar membrane since a greater length of the membrane will oscillate at greater sound intensities. The peak sensitivity of most nerve cells is considerably greater than could be expected on the basis of basilar membrane displacement.

Cochlear nuclei. The first set of synapses in the auditory pathway (Fig. 14-13) is in the dorsal and ventral cochlear nuclei of the pons (Fig. 29-8). Within the cochlear nucleus there is a very definite tonotopic organization. Each of these cells still responds to a range of frequencies, but the frequency range is narrower than in the auditory nerve.

The larger cochlear division of Cranial Nerve VIII enters the brainstem inferior to the vestibular branch. The cochlear fibers enter the cochlear nuclei, bifurcate and distribute to the dorsal and ventral cochlear nuclei on the lateral surface of the inferior cerebellar peduncle. The *dorsal cochlear nucleus* forms the eminence of the acoustic tubercle on the lateral margins of the floor of the IV ventricle. The dorsal cochlear nucleus is laminated into three distinct regions with distinct nuclei. The *ventral cochlear nucleus* is not laminated. The fibers of the auditory system are organized tonotopically. Fibers from the apical cochleus (low tones) are found in the ventral portions of the dorsal cochlear nucleus and in the ventral nucleus while fibers from the basilar portion of the cochlea (high tones) terminate only in the dorsal portion of the dorsal nucleus.

The secondary auditory pathways arise from the secondary cochlear nuclei and form three acoustic stria. These pathways carry information bilaterally onto the midbrain, thalamus and auditory cortex. The ventral stria (from ventral nucleus) is the largest and forms the *trapezoid body* in the pontine tegmentum. These fibers cross the midline either by passing under or through the medial lemniscus and then form a discrete bundle, the *lateral lemniscus*. Fibers also terminate in the ipsilateral and contralateral superior olive and nucleus of the trapezoid body.

The *dorsal stria* arises from the dorsal cochlear nucleus while the intermediate stria arises from the dorsal part of the ventral cochlear nucleus. The dorsal stria passes medially, crosses the midline ventral to the MLF and joins the contralateral lateral lemniscus.

Fibers from the intermediate stria pass through the reticular formation and join the contralateral lateral lemniscus.

In the passage of the auditory fibers from the upper medulla to the midbrain and diencephalon several prominent nuclei are

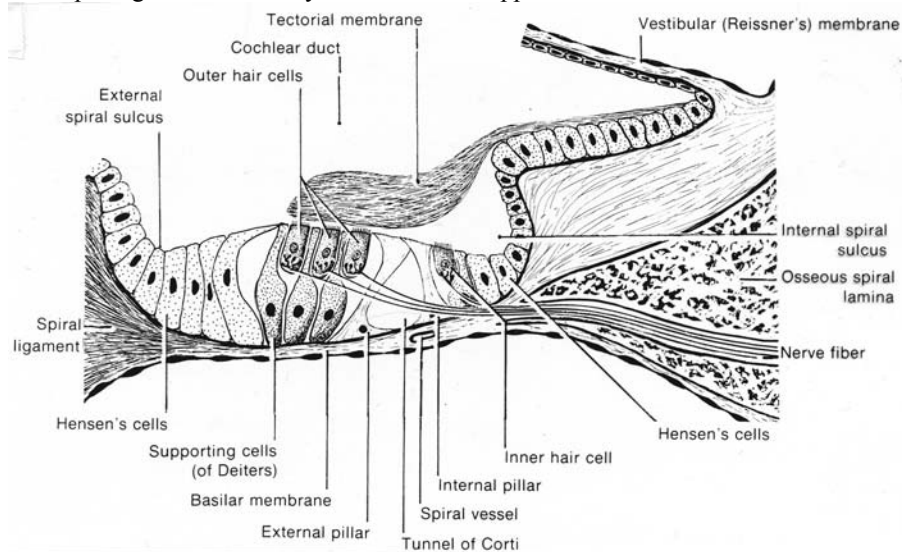


Figure 14-11. The organ of Corti. See Figure 14-8 for its location in the cochlea. The transduction between vibration and action potential firing occurs at the inner and outer hair cells. (From Bossy: Atlas of Neuroanatomy. Philadelphia, W.B. Saunders, 1970.)

found-trapezoid nucleus, superior olive and nucleus of the lateral lemniscus. Fibers from these nuclei join the fibers ascending in the lateral lemniscus. Most of the fibers in the lateral lemniscus terminate in the nucleus of the *inferior colliculus* either ipsilaterally or contralaterally by crossing in the commissure (of Probst) of the inferior colliculus. Fibers from the inferior colliculus and probably a few from the lateral lemniscus continue onto the *medial geniculate* forming the brachium of the inferior colliculus. The fibers from the medial geniculate project onto the auditory cortex in the *transverse temporal gyri*, (of Heschl) area 41. Some projections from the medial geniculate also reach auditory association area 42. The low tones (apex of the cochlea) are found in the lateral part of the auditory cortex in the planum temporale while high tones (base of the cochlea) are found medially.

The *auditory cortex* deep in the lateral sulcus on the superior temporal lobe is hidden by the overlying parietal operculum. This region can be visualized by making a deep section with a brain knife that follows the lateral sulcus. This section is in the planum temporale and demonstrates that the area posterior to the auditory cortex in the left hemisphere is larger than in the right hemisphere. This asymmetry was noted by Geschwind and Levitsky and is directly related to the presence of Wernicke's speech area (for understanding spoken language) in the temporal lobe.

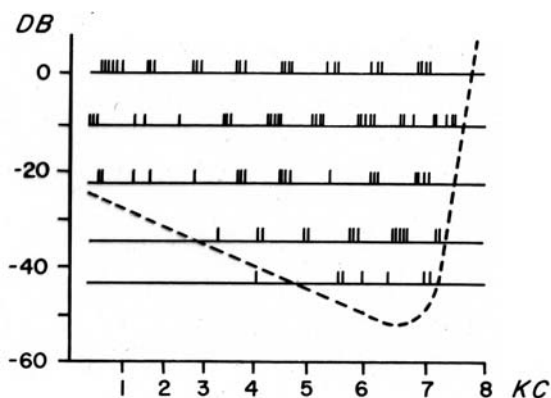


Figure 14-12.. Responses of a single auditory nerve fiber to tones of differing frequency and amplitude. Each group of vertical bars

represents a train of action potentials in response to a 10 msec burst of tones at a given frequency and intensity. The responses on each line are to the same intensity but varying frequency. Note that the unit has a decreased range of frequency response as the intensity is reduced. The dashed line outlines the overall sensitivity. (After Tasaki, I.: J. Neurophysiol., 17:97, 1954.)

Auditory reflexes. The auditory nerve contains an efferent bundle, the olivocochlear bundle, or the efferent cochlear bundle. This bundle originates from the superior olive or the medial accessory superior olivary nucleus, exits via the VIII nerve and distributes in the spiral ganglion on hair cells in the cochlea. Presumably these fibers modulate synaptic transmission, possibly as early as the hair cell, many of which have efferent nerve endings (Fig 14-3).

Two muscles influence sound transmission across the middle ear: the tensor tympani and the stapedius. The *tensor tympani* are innervated by cranial nerve V. By tensing the tympanic membrane it dampens the vibration. Fibers from the accessory superior olive project bilaterally to the motor nucleus of the fifth cranial nerve to support reflexive dampening of loud sounds. The *stapedius* muscle is innervated by cranial nerve VII and, by pulling the stapes laterally and away from the oval window, tends to counteract force placed on the stapes from the tympanic membrane. The reflex is via fibers from the accessory superior olive projecting bilaterally to the motor nucleus of the seventh cranial nerve. When loud noises or tones are heard or anticipated, these muscles contract and reduce the percentage of the sound energy impinging on the oval window.

IMPAREMENT OF HEARING

Hearing defects can be broadly classified as conduction deafness or sensory-neural deafness. Cochlear implants offer many individuals a chance for recovery of much of there hearing. *Conduction deafness* refers to disease that prevents the pressure waves from reaching the oval window; *sensory neural deafness* refers to disease of the cochlea and the auditory nerve. A simple test of air versus bone conduction will usually differentiate between these types and should be part of every physical exam. A vibrating tuning fork is held against the mastoid bone and the patient is instructed to indicate when it is no longer felt. When the patient no longer feels the vibration, the tuning fork is immediately placed beside the pinna; a normal patient should hear the tuning fork. This is known as the *Rinne test* that is said to be positive when air conduction is more sensitive than bone conduction. This should be tested in both ears. The patient feels the vibration by conduction through the bones of the skull and activation of the basilar membrane and the organ of Corti in the usual manner. Hence, to feel the vibration, the inner ear and VIII nerve must be intact. In order to hear, the outer and middle ears must be functional. If the patient feels the vibration but does not hear it, conduction deafness is suspected. If the patient neither hears nor feels the tuning fork, sensory-neural deafness is indicated.

A frequent form of sensory-neural deafness is a loss of hearing above 2 or 3000 cps; a high-tone loss, *presbycusis*. A Rinne test with a 128 cps tuning fork will not pick up this problem yet the loss is quite incapacitating since most of the consonant sounds of the English language are above 3000 cps. Since this form of neural deafness is quite common among older persons, it no doubt contributes to the isolation and depression so often seen in the elderly. Hearing aids are not often successful, yet should be tried. Aside from acoustic neurinoma, other types of neural deafness, such as that caused by continuous loud noise or by neomycin intoxication, offer little hope of remediable action although consultation with an otolaryngologist should be sought.

An abnormal Rinne test, with bone conduction greater than air conduction, is a sign of a middle-ear defect or a blockage, such as a plug of wax, in the external auditory canal. These types of deafness are usually amenable to hearing aids or surgery.

Infections of the middle ear are common. The inflammation produces a redness of the eardrum and a fluid collects in the middle ear, temporarily interfering with hearing. If the tympanic membrane is red, middle-ear infection should be suspected.

There are many quantitative hearing tests that offer many diagnostic clues to the location, cause, and prognosis of the disease process. They are, however, beyond the scope of this text; one of the references should be consulted.

Auditory Cortex.

From a clinical standpoint, unilateral lesions limited to the auditory projection area in the temporal lobe do not cause any significant alteration in hearing.

This is consistent with the fact that multiple decussations occur in the auditory pathway at brain-stem levels, so that each auditory projection area receives impulses from both left and right cochlea. The studies of Penfield and Evans (1934) suggest that unilateral temporal-lobe ablation may disturb the ability to locate a source of sound to the left or right of the midline. Bilateral lesions of the auditory projection would theoretically result in "cortical deafness," and Tanaka and associates described such a case in 1991.

Auditory Cortex Neocortical area 41 the primary auditory projection area, is located on the more anterior of the transverse gyri of Heschl (Fig. 22-17). This area is granular cortex similar to but considerably thicker than areas 17 and 3. Area 41 receives the main projection from the medial geniculate

nucleus of the thalamus. Our understanding of the tonal organization in this region comes from studies in the monkey and chimpanzee where the lowest frequencies project to the more rostral areas. Some investigators limit the term Heschl's gyrus to the more anterior of the transverse gyri and localize the primary auditory cortex to the posterior aspects of that gyrus

Homotypical areas 42 and 22 are often designated as auditory association areas, and in the dominant hemisphere they are important in understanding speech. The role of the other lateral temporal neocortical areas may best be considered in terms of disease processes.

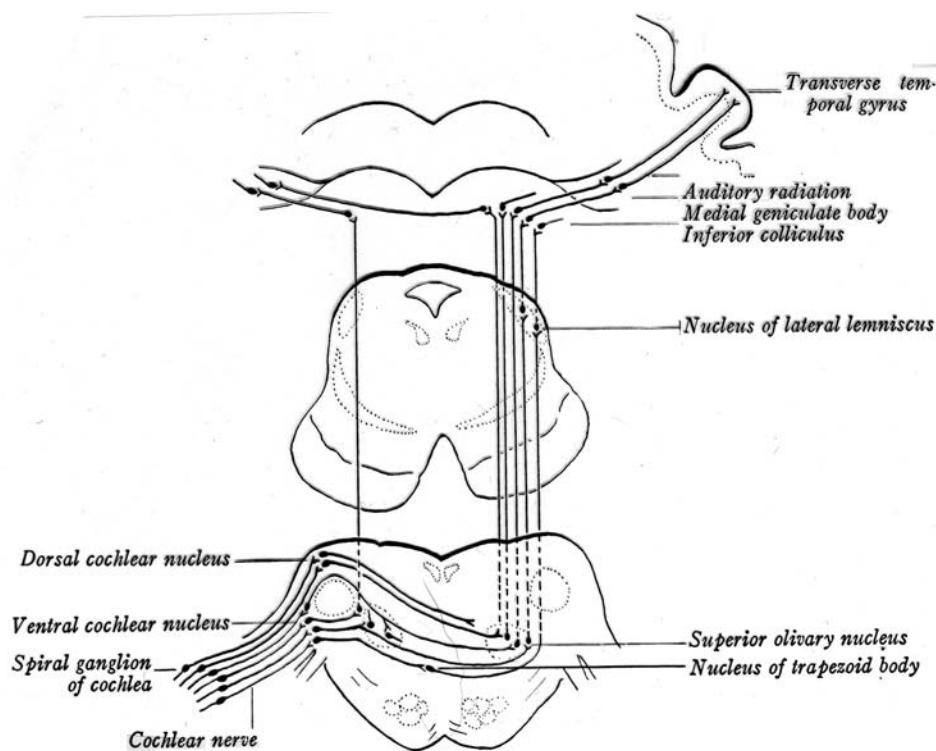


Figure 14-13. The auditory pathway. Note that there are many, non-synchronous synapses. For clarity, the diagram shows most of the fibers crossing the neuraxis; about half of the fibers do; the other half ascend on the same side. (From Ranson and Clark: Philadelphia, W.B. Saunders, 1954.)

