

Chapter 172: Effects of Aging on the Auditory and Vestibular Systems

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General Considerations

Aging, which results in a gradual multisystem dysfunction, senescence, and ultimately death, is common to all multicellular organisms. Some have hypothesized that the aging process forces continual mixing of a species' genetic pool by forcing an influx of new individuals, thus ensuring health by natural selection. No such model has been tested (Cape, 1978) however, and although the timing and rate of the aging process is species dependent and to some extent genetically determined, individual responses vary greatly. The functions of various organ systems within a given person also begin to diminish at variable times and rates, often in a nonlinear fashion. Thus the concept of physiologic, as opposed to chronologic, age is descriptive, but currently measurable parameters cannot quantitate it.

The control and specific mechanisms of the aging process undoubtedly are complex. One theory states that aging is a completely controlled, genetically scheduled event as inevitable as birth and maturation (Rossman, 1971). Another theory holds that aging results from the accumulation of replicable errors in ribonucleic acid (RNA) synthesis caused by random errors in deoxyribonucleic acid (DNA) structure (Rossman, 1971). Other demonstrable changes generally occurring during aging that appear important include lipofuscin accumulation, ribosomal DNA (rDNA) loss, rRNA depletion, accumulation of inactive enzymes, reduced protein synthesis, membrane receptor inactivation, and molecular cross-linkages.

In some tissues the ability to undergo mitosis and thus replicate is arrested at a certain point of maturation before birth. This process appears to allow further specialization of function at the expense of reparative potential.

The central nervous system (CNS) pathways and peripheral sensory end organs are thought to be such "postmitotic" tissues; if so, those tissues would pay a disproportionate price as aging progresses. Recent work by Rubel and colleagues (1989) has documented regeneration of hair cells in avian cochleas and vestibular sensory organs, as well as reinnervation of the auditory hair cells and evidence of function of those "postmitotic" tissue dogma. Whether regeneration occurs in humans, or whether it has any clinical relevance, remains to be seen.

Aging of Central Nervous System

CNS aging results in a measurable decrease in astrocytes in the frontal and parietal cortex, neuronal loss in the hippocampal cortex, and a decrease in Purkinje's cells of the cerebellum after the fifth decade of life (Ball, 1977; Brody, 1978). Remaining cells may be reduced in volume and exhibit many of the structural changes just noted. Such cellular loss must result in some pathway reorganization, loss of plasticity, and a general reduction of activity, as evidenced by reduced oxygen consumption during aging (Cape, 1978). Concomitantly, in the brains of elderly rats, for example, a loss of beta-adrenergic receptor sites in the cerebellum and brainstem and a decline in the norepinephrine concentrations occur

at various sites (Maggi et al, 1979). The increased ratio of serotonin (generally considered an inhibitory neurotransmitter) to dopamine and norepinephrine (considered excitatory) is also consistent with the concept of a slowdown of activity (Timieras et al, 1979). As with most discussions of the effect of aging on cochlear and vestibular functions, this chapter deals predominantly with peripheral changes. It is extremely important to remember, however, that the central pathways responsible for the integration and interpretation of the peripheral signal are undergoing the changes already described. Thus a compromised central processor is compiling incomplete or faulty afferent data, distorting auditory and vestibular reality. Likewise, faulty storage in or retrieval from recent memory may hamper the ability to deal with new perceptions of auditory or vestibular reality.

Aging of Auditory System

Clinical consequences

The effect of aging on the auditory system characteristically results in a bilaterally symmetric neurosensory hearing loss in the frequencies above 2000 Hz, although other patterns clearly occur. At first, conversation is not hampered because the frequencies affected are above those of speech (500 to 2000 Hz). As the upper speech frequencies become involved, the individual has increasing difficulty discriminating consonants in words and, if questioned, will describe problems with understanding speech, not missing it entirely. As overall speech discrimination begins to fail, conversation becomes more and more difficult, especially within a group. The ability to ignore competing speech also becomes impaired, and maintaining communication requires increasingly greater effort and energy. Often, after experiencing real or perceived rejection by peers or family as a result of responding inappropriately, the elderly person will withdraw. Such serious curtailment of socialization can result in marked decrease in physical and mental activity, profound loneliness, depression, and even paranoia. Such a fate is not uncommon, as about 30% of the population over age 65 have a significant hearing impairment. Furthermore, in the mentally retarded or demented elderly individual, the physician must search vigorously for a hearing loss as a contributing factor to the patient's disability.

Environmental factors

It is difficult to assess in each person what proportion of hearing impairment is caused by aging of the auditory system and what is caused by other traumatic or metabolic factors. Rosen's epidemiologic work (1966) suggested a relationship between hearing loss in the elderly and noise in the environment. Carried to the extreme, presbycusis may be the result of the lifelong accumulation of acoustic trauma. Other factors, however, such as physical activity, diet, and concomitant disease processes must be taken into account when comparing average hearing thresholds among different cultures (Rosen and Olin, 1965).

Vascular factors

In addition to age-induced changes in the CNS and the peripheral auditory system, changes in the vascular system contribute to auditory dysfunction. Fisch et al (1972) demonstrated thickening and degeneration of the tunica adventitia of the end arteries to the inner ear: the labyrinthine, vestibular, and cochlear arteries. They theorized that such changes may interfere with the vessel's ability to dilate in response to reduced cerebral flow and that the resulting hypoxia may contribute to the neural degeneration seen in aging.

Using a surface preparation stained with osmium tetroxide (osmic acid) following microdissection of human cochleas, Johnsson (1973) was able to characterize the capillary beds of the spiral ligament and vascular stripe (stria vascularis). He found that perivascular spaces were a striking feature of spiral ligament capillaries. Perivascular spaces allowed Johnsson to identify intravascular strands, the collapsed remnants of vessel walls in older ears. In severe cases of presbycusis, the spiral ligament had atrophied sufficiently so that perivascular spaces and thus intravascular strands were difficult to identify. Johnsson could not find the characteristic changes of atherosclerosis in any of the arterioles in aged temporal bones but did describe a specific form of vascular pathology occurring in the basilar turn. The outer spiral vein demonstrated a particularly spacious perivascular space that is filled with a hyaline material. The lumen of the capillary was occluded more basally when only a distinct avascular band remained. Johnsson speculated that the changes noted resulted in hypoxia and changes in the ionic composition of inner ear fluids, contributing to the neural, stria, and membranous degeneration observed in the inner ear of the elderly. An extensive review of the indirect evidence of vascular changes resulting in impaired hearing in the aged can be found in the work of Gilad and Glorig (1979a, b).

Histologic and audiologic correlates

Historic aspects

Although changes in the stiffness of the ossicular chain clearly occur during aging (Belal and Steward, 1974; Nixon and Glorig, 1962) and may affect both air and bone conduction thresholds, most agree that the vast majority of presbycusis is caused by inner ear changes. Crowe et al (1934) first correlated high-frequency sensorineural hearing loss with atrophy of the organ of Corti in the basal turn of the cochlear duct in the temporal bones. In other clinically similar cases they found degeneration of that portion of the auditory nerve that supplies the basilar turn. They were unable to recognize any specific vascular changes. Von Fieandt and Saxen (1937) shortly thereafter described 13 cases of presbycusis with spiral ganglia degeneration and 19 with apparent generalized arteriosclerotic changes, including stria changes and collapse of the cochlear duct.

In 1955 Schuknecht presented the behavioral audiograms and temporal bone histograms (cochlear charts) of four cats and convincingly argued a causal relationship between loss of basilar hair cells in the organ of Corti and high-frequency neurosensory hearing loss. He also presented an example of a human ear with changes in the organ of Corti only in the 1- to 3-mm region, but with loss of spiral ganglion cells extending to 14.2 mm, and an audiogram with a neurosensory loss above 2 kHz. Thus he clearly demonstrated the independent factors of hair cell and ganglion cell loss. Schuknecht also addressed the

phenomenon of normal pure-tone thresholds with poor speech discrimination seen in progressive loss of primary afferent cochlear neurons, theorizing that relatively fewer neurons are necessary to convey threshold information than complex speech messages. On the basis of further histologic material, Schuknecht (1964) amended his scheme of presbycusis to include metabolic (strial) and mechanical (cochlear) presbycusis.

Although in practice the types of presbycusis Schuknecht defined often are superimposed, the concept is particularly useful to keep in mind when the physician is trying to correlate audiometric and clinical findings with cochlear histopathology. This scheme, therefore, is presented in the following section. Such a scheme, is possible, however, only because of the unique ability to quantitate accurately input-output functions of the auditory pathways and the encoding of frequency along the basilar membrane as a function of distance from the basilar end.

Types of presbycusis

Sensory presbycusis is the high-frequency, bilaterally symmetric bone conduction loss most frequently diagnosed in the elderly in the clinical setting. When this type of loss is seen in younger patients without a history of other etiologic agents such as trauma or toxins, physicians most often term it *idiopathic*. At its onset, frequencies about 2 kHz are involved so that conversational skills are not affected. As high-frequency thresholds continue to deteriorate, lower frequencies become increasingly involved, with difficulty in discriminating consonants becoming most obvious. Although sensory presbycusis is a slowly progressive process, the patient may complain of an apparent sudden worsening with as little as a 5 dB change in a sensitive frequency once threshold has reached the 30 to 40 dB level. Concomitant high-pitched continuous tinnitus is not uncommon. The physician must remember that recruitment is present when assessing comfort levels for amplification. Speech discrimination scores usually are respectable when presentation is around 20 to 30 dB hearing level (HL). Fig. 172-11 illustrates a typical audiogram in sensory presbycusis.

Histologically, sensory presbycusis appears as a degeneration of the organ of Corti, initially in the most basilar region and progressing apically about 15 mm along the cochlear duct. The earliest changes are flattening and distortion of the organ, with eventual loss of hair cells (Fig. 172-2). Eventually, supporting cells deteriorate, reducing the organ of Corti to an undifferentiated mass on the basilar membrane. Loss of supporting cells results in a subsequent loss of primary afferent fibers supplying that portion of the basilar turn (Schuknecht, 1953), presumably because of damage to their nerve endings.

Neural presbycusis

In contrast to the clinical picture previously described, some aging individuals complain of relatively rapid hearing loss with great difficulty understanding speech. Audiometric examination often reveals moderate loss for pure-tone thresholds almost equal at all frequencies, with a surprisingly poor speech discrimination score (Fig. 172-3). Such a phenomenon has been called *phonemic regression* (Gaeth, 1948, cited by Schuknecht, 1974). The characteristic histologic finding in phonemic regression is a loss of spiral ganglion cells with a relative sparing of the organ of Corti (Guild et al, 1931) (Fig. 172-4). Such a loss often is greatest in those neurons supplying the basilar turn. If this is the predominant lesion,

hearing is usually normal. If apical innervation is significantly involved, encoding of speech frequency auditory information is severely hampered (Otte, 1968).

Phonemic regression apparently occurs because the CNS needs much more sensory input to interpret speech than to identify pure tones. Schuknecht and Woellner (1955) found that in cats auditory thresholds to pure tones would shift only after a loss of 75% of the appropriate primary afferent neurons. Patients with neural presbycusis often have generalized neural loss, and their hearing loss often is associated with other deficits such as motor weakness, poor coordination, memory problems, or intellectual deterioration. The nature and degree of their speech discrimination dysfunction makes successful amplification more difficult.

Strial (metabolic) presbycusis

Schuknecht (1974) described a slowly progressive familial neurosensory loss. Audiometrically this loss was flat across all tested frequencies, and the person maintained an excellent ability to discriminate speech. Individuals with such a loss are excellent candidates for amplification. Histologically, the cochleas of such patients demonstrate some degree of atrophy of the stria vascularis.

The stria vascularis has long been thought to be the site of endolymph production because of (1) the presence of enzyme system necessary to maintain a potassium-sodium gradient and oxidative metabolism and (2) an electron microscopic appearance similar to other known secretory organs, including renal tubules and the choroid plexus. The stria also appears to be the site of generation of the endocochlear potential, a gradient of 80 mV dc (direct current) between the cochlear duct and perilymphatic space that is necessary for proper cochlear transduction.

Strial atrophy may appear as a scattered thinning that occurs throughout the cochlear turns, often reducing the light microscopic appearance of the stria to a single cellular layer. Other associated changes include cystic degeneration of strial elements and atrophy of the spiral ligament.

Although this description is a useful clinical model, comparative histologic studies have suggested that the pattern of hair cell loss in aged human and animal species is far more complex and to some extent species dependent. Dayal and Bhattacharyya (1986) emphasize the apical outer hair cell loss, which occurs in squirrel monkeys and other species, that was previously reported in humans (Johnsson and Hawkins, 1972). Such loss has no known clinical correlate. Giant stereocilia were described in surviving apical and middle turn outer hair cells but not in the basal turn and, to a far lesser extent, in the inner hair cells of all three turns in aged human cochleas (Soucek et al, 1986). These authors correlated pure tone audiograms (15) and auditory brainstem response (ABR) (7) with the histology in elderly patients. ABRs of 49 elderly patients demonstrated waves III to V to be present and not significantly different from those of young controls. Wave I, when present, was normal and wave II was usually absent. Latencies were normal for the earlier waves but prolonged slightly for wave V at all intensity levels compared to those of young controls. The authors thought this finding was consistent with a loss of cochlear nerve myelination and was evidence for increased thresholds to be of cochlear (hair cell) origin.

Cochlear presbycusis

Meyer (1920) first reported a thickening and stiffening of the basilar membrane with resulting decreased mobility as a cause of a straight-line-sloping hearing loss with preserved speech discrimination in aging persons. He supported this hypothesis with histologic evidence of hyalinization and calcification of the basilar membrane. Other associated changes include cystic degeneration of strial elements and atrophy of the spiral ligament, characterized by progressive disease in the ligament's cellularity and occasionally by ligament rupture (Schuknecht, 1974). Although still largely hypothetical, such a concept certainly is consistent with the finding of high-frequency loss in the absence of recruitment when histology fails to reveal hair cell loss.

Aging pigment

Neuroepithelial elements, including hair cells, accumulate a golden-brown pigment called *lipofuscin* during the aging process before cell death (Fig. 172-5). Not limited to the ear, lipofuscin may be seen in many aging postmitotic tissues, including myocardial muscle cells and neurons of the CNS, where lipofuscin's autofluorescence makes it easy to locate. Composed of phospholipids, steroids, and proteins, lipofuscin is thought to represent the undiscarded waste products of intracellular oxidative metabolism. Although associated with an acid phosphatase, it does not appear to have any significant enzymatic activity within or on its host cell. The location of its accumulation corresponds to the site of lysozyme activity.

Mann et al (1978) have speculated that lipofuscin may have a mass effect, crowding the endoplasmic reticulum and Golgi apparatus. Thus it may contribute to the known disease in protein synthesis characteristic of aging, as well as interfere with intracellular transport. It is not known, however, whether lipofuscin is a cause or just an effect of the aging process.

Central auditory presbycusis

Although several carefully designed anatomic studies of the auditory pathways and cortex have described specific isolated changes correlated with aging, a lack of understanding of central auditory organization and interspecies differences, among other problems, prevent any unified concept of central presbycusis (Feldman and Vaughan, 1979). On the other hand, such a component has been convincingly delineated from a general failing of the elderly to perform psychomotor tasks.

A central component to hearing loss may be characterized audiometrically by comparing the discrimination scores to both monosyllabic words (PB) and synthetic sentences (SSI) over increasing intensity levels of presentation. In the normally hearing subject, scores increase rapidly for both tasks with increasing presentation levels and remain at 100% thereafter. In a patient with peripheral hearing loss (cochlear), the scores improve less rapidly and the SSI score is at least that of the PB score and often exceeds it at a given intensity level. With a significant central component, the SSI scores fall below those of the PB scores and may actually decrease at high-intensity presentation levels (roll-over).

It has long been observed that elderly patients seem to benefit less from amplification than younger patients with comparable hearing loss. Using the techniques described

previously, Jerger and Hayer (1976) demonstrated the dual nature of the hearing loss of presbycusis as well as its practical effect on amplification in the elderly. Presbycusis patients with and without central components did equally well in discrimination testing in a quiet environment, but scores rapidly deteriorated in patients with a central component as defined earlier, in conditions of competing noise.

Aging of Vestibular System

Although it might be argued that through evolution the phylogenetically older pars inferior has had a much greater chance to adapt to the effects of aging, the neural elements of the vestibular system would be expected to undergo qualitatively similar changes over time as those so well defined in the auditory system. Such changes in balance have received much less attention than those involving hearing, partly because the dysfunction is considerably more difficult to define, let alone objectively measure. Whereas certain aspects of the vestibular reflex lend themselves to a systems analysis, the input-output precision of pure-tone audiometry eludes vestibular testing. Likewise, nystagmus response does not define system integrity in the way that speech discrimination does; posturography or other measures of body-environment perception are beginning to address this issue.

Two factors responsible for the lack of anatomic/physiologic correlation in vestibular dysfunction are (1) the multiple inputs from vestibular, optic, and proprioceptive sensory systems; and (2) the plasticity of CNS perception of the environment. If hair cells are lost from the basal turn of the cochlea, no other organ can take their place, resulting in a predictable high-frequency hearing loss that both patient and physician can characterize. If a similar group of hair cells in the macula of the saccule atrophies, visual and proprioceptive information is recruited to provide a continuum of environmental information. Thus disequilibrium of aging becomes apparent only when multiple systems fail. Such failure undoubtedly occurs at different rates in different systems in different individuals, making the prediction of its course clinically difficult.

Clinical considerations

Despite its unpredictable character and variable compensation, disequilibrium is a common symptom in the elderly population. A frequently quoted investigation of elderly English subjects living at home concluded that slightly under half of women and more than half of men in their middle to late 60s experienced vertigo (Droller and Pemberton, 1953). The authors believed there was a close correlation between vertigo and both atherosclerosis and hearing loss. They defined vertigo, however, in the widest sense. Only four of 208 subjects had positional vertigo, and the authors found none with "central" vertigo, which they defined as that associated with sensory disturbance, including ataxia and nystagmus, but not deafness.

A study of patients attending a Finnish geriatric clinic found more than 50% of the patients admitted to dizziness, even though they had come for other complaints (Orma and Koskenjoa, 1957). Of these patients and of those whose chief complaint was dizziness, more than 90% experienced a "postural" type, defined by the authors as a short-lasting dizziness related to a change in body or head position, which did not occur when the patient was still and specifically did not constitute a sensation of rotation. Vertigo occurred in only about one

third of all patients. Other studies of nystagmus during caloric or rotatory testing in elderly age groups have reported significant differences in responses or perceptions between the older and younger age groups, but these data are not particularly clinically useful (Arslan, 1957; Bruner and Norris, 1971; Karlsen et al, 1981).

My clinical impression is that the most easily definable symptomatology attributable to vestibular aging is an unsteadiness when upright, and especially during ambulation or body position changes, that occasionally results in falling but not unconsciousness. True vertigo or nausea is infrequent, and symptoms cease when the person is immobile. The symptoms often are worse in the first hours of morning, which may be related to the person's state of alertness. Objective findings include typical presbycusis on audiometric examination. An electronystagmogram may demonstrate difficulty with calibration, a bilateral hypofunctioning caloric response, and most often a high-optic fixation index, which is the ratio of the nystagmus response to caloric stimulation with the eyes open to that with the eyes closed. A high value indicates a central pathologic component.

The evaluation of balance by the use of a computerized force table during standardized positioning such as Romberg testing provides an analysis of the integrated postural control mechanisms from all operative systems, particularly spinovestibular and proprioceptive. Such testing provides new insight into quantifying the spacial orientation defects of aging. Using this system, the effort expended to keep the body in an upright position during, for example, a tandem Romberg position with and without visual clues may be expressed as a change in energy (ΔE). Black (1979) showed the ΔE increased linearly with the age of the subjects. Thus the aged work harder at maintaining their posture.

Stelmach and Worringham (1985) have discussed a schema that takes into account the processing stages necessary in averting (successfully or unsuccessfully) a potential fall, which represents the end point of instability. Sensory input that a fall may be imminent is compromised in the aging process by loss of visual acuity, including the loss of static visual references as well as degradation of proprioceptive and vestibular sensation, thus requiring longer sampling times to acquire threshold data. Feed-forward "predictive" control influence on voluntary position changes on the other hand appear to be relatively spared. Response selection, be it reflexive or a cortical selection process, preconceived by experience or *de novo*, is not easily amenable to experimental design. Studies of simple response times suggest that the elderly have greatest trouble in fully preparing and maintaining response preparation. Other studies have suggested that the elderly have difficulty with a conflict between speed and accuracy of a response, but tend to favor accuracy at the expense of speed over which they have less control. Response execution of corrective action is compromised in the elderly by slower motor times, especially in complex tasks. This compromise is especially prominent in usually inactive subjects.

This schema suggests that the extra energy expended by the elderly in maintaining stability is due in part to mistimed and mismatched corrective efforts, resulting in a less critically dampened pendulum and more body sway. The more complex or unpredictable the necessary corrective action may be, the more likely it will be "too little too late". Thus, the condition of motion would be more likely to result in instability than that of static balance.

Histopathologic correlation

It is difficult to generate a typical picture of the histopathology in the aged vestibule because the periphery is organized diffusely, and we do not understand the exact stimuli to which different hair cells on the same neuroepithelium are most finely tuned. Because fewer patterns of degeneration occur, the methods used to characterize vestibular aging are necessarily more arduous and often statistical.

Most available evidence suggests that the vestibular end organ and the cochlea undergo the same processes during aging. Many of the following observations are drawn from the examination of 22 pairs of human temporal bones in patients seen at the University of Iowa. Patients were 75 years of age or older and had no ear disease. Seventeen pairs of bones in patients between 12 and 45 years of age with no ear disease served as controls.

Neuroepithelium

Hair cell loss is an important aspect of peripheral vestibular aging but, unless extreme, is difficult to detect by light microscopy. Engstrom et al (1977) examined aged vestibular epithelia from humans and monkeys by transmission electron microscopy. They found hair cell loss up to 40% on the cristae and 20% on the maculae. Type I hair cells - flask shaped with cuplike afferent calices - are far more susceptible than type II cells, although changes are similar quantitatively. Persisting hair cells have laminated structures beneath their cuticular plates and often display marked changes in cell membranes in the region of synaptic junctions. Because type I hair cells predominate at the top of the cristae, loss may appear as a thinning of the neuroepithelium, with very regular nuclei (Fig. 172-6). More dramatic changes occur before cell death within the supporting cells (Engstrom et al, 1977); these develop fibrilotubular structures and many vesicular structures.

Light microscopy also detects intraepithelial cysts in the elderly (Richter, 1979; Rosenthal, 1974). These cysts may take two forms: (1) a pale bluish inclusion in a near-normal-sized cell and (2) a larger, more organized, eccentrically placed structure (Fig. 172-7).

Primary afferent fibers

As in the cochlea, a steady loss of innervation occurs in the vestibular epithelium with aging. This loss is difficult to evaluate by light microscopy of routine sections, although some loss may be detected in random sections of Scarpa's ganglion or in horizontal sections through the superior vestibular nerve as it approaches the macula of the utricle. Using special histologic techniques to study freshly dissected vestibular nerves, Bergstrom (1973) demonstrated a reduction in the number of fibers, beginning in the fifth decade of life. Thick, myelinated fibers that innervated type I hair cells of the cristae showed the most severe loss, correlating well with hair cell findings in aging. Richter (1981) made serial sections of Scarpa's ganglion, counted cell bodies, and found a dramatic reduction during the sixth decade of life.

Otoconia

It has been speculated that the vestibular system uses the constant pull of gravity (a linear acceleration) as a reference point with which other acceleratory influences are compared. The constant perception of gravity depends, at least in part, on the integrity of the otolith organs. Because of its orientation in upright posture, the saccule is thought to play an especially important role in this function.

Using standard techniques, otoconia are not preserved with any consistency and cannot be evaluated routinely. Exacting microdissection and scanning electromicroscopy techniques have provided insight into the deterioration of this important component of macular function (Ross, 1976). Otoconia are cylindric calcite rods with tripartite flat endplates at each pole. They vary regularly in size, depending on their distribution over the macula and their relationship to the striola. In young adults otoconia have only shallow furrows running along the long axis of the rod. During the sixth decade of life, the furrows of the saccular otoconia become deepened, with loss of the original surface. Pits of degeneration appear and coalesce, most noticeably in the midsection. As the crystal is resorbed, the rod is cleaved in half, leaving the end poles as separate fragments that still demonstrate their endplates. Such degeneration is noted only occasionally in the utricle.

Saccule

The saccular membrane in temporal bones of elderly individuals is often ruptured despite a lack of clinical signs or a history of severe balance problems during life (Babin and Harker, 1982) (Fig. 172-8). This rupturing occurs uniformly, without significant disruption of either the neuroepithelium or the primary afferent fibers. Knowledge of regenerating membrane, not common in bones of younger control patients, often support the antemortem timing of this finding. It has been shown that sclerotic changes around the endolymphatic sac increases with age (Sakai et al, 1985). This increase did not appear to be correlated with either saccular receptive or endolymphatic hydrops.

Aging pigment

As in the cochlea, a gradual buildup of lipofuscin occurs in cellular elements of the vestibule. Light microscopy usually detects this buildup on the slopes of the cristae of the three semicircular canals. Lipofuscing also appears, although less frequently, in the two macula and occasionally may be noted deep within the body of the cristae below the basement membrane. Fluorescent microscopy shows that the cell bodies of Scarpa's ganglion are especially rich in lipofuscing (Ishii, 1967). Although the pigment is the result of aging, there is no correlation between absolute age of the patient and any particular degree of disequilibrium.

Distribution of aging changes

Several findings support the contention that the pars inferior ages more rapidly than the pars superior in the vestibular system. Using microdissection techniques, Johnsson (1972) demonstrated degeneration of the cochlea and saccule without changes in the rest of the labyrinth in elderly individuals with no history of congenital or acquired ear disease. The predominance of saccular membrane rupture without a concomitant incidence of utricular rupture, as well as the differential susceptibility of saccular otoconia to degenerate, also support this hypothesis. Finally, cupulolithiasis, a degeneration and mineralization of the cristal most likely related to aging but not trauma, has been reported only in the posterior semicircular canal (Schuknecht, 1974) (Fig. 172-9), although one temporal bone in the Iowa collection demonstrated these findings only in the horizontal canal ampula (author's observation).