Presbycusis: physiological or pathological*

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It has been known for centuries that people grow deafer as they grow older. Bunch (1931) was the first investigator to demonstrate quantitatively the progressive diminution of hearing acuity for pure tone thresholds, especially for high frequencies. These findings were confirmed by others (Steinberg *et al.*, 1940; Webster *et al.*, 1950).

Although hearing in many old people is excellent for all but some of the high tones, the hearing for speech is distinctly affected. Pestalozza and Shore (1955) found poor speech discrimination in subjects over the age of 65, as compared with subjects below the age of 40, even though their pure tone audiograms were similar.

To call these auditory impairments normal for the different age groups is customary but misleading. These are not normal losses, but rather deviations from what is called 'no loss'. There is nothing called a normal loss at any age, but one can determine the average deviation from the normal for each age or decade. The reference standard for any age or decade has been outlined by the American Standards Association (1954) and the Wisconsin Hearing Survey (1957). The outlined curves were called the standard presbycusis curves.

It is quite evident that there are too many variations in the averages obtained from various subjects to make those averages dependable as a standard of reference. The acoustic impairments in old people may vary from losses of almost nothing to total deafness. Yet, just because they are old, both those with good and those with bad hearing have been categorized as 'average normal' for the age group.

Many of the histological reports describing the effect of aging on the auditory mechanism are concerned with degeneration of the sensory and neural structures (Guild and Crowe, 1938; Bredberg, 1967; Johnsson and Hawkins, 1972). Schuknecht (1964) described four pathological types of presbycusis: sensory, neural, metabolic, and mechanical. Each of these has a characteristic clinical presentation.

On the other hand, many reports have described histological changes in the different parts of the auditory system (Riesco-Maclure, 1948; Allam, 1970). These changes have been termed 'normal for aging'.

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It was that confusion between what is termed physiological and pathological that stimulated the present work which studies the effects of aging on the auditory system. This study was done on the ears of old people who had normal hearing, and those who presumably suffered from deafness of aging.

Material and methods

All the temporal bone specimens were acquired from the temporal bone collection at the Massachusetts Eye and Ear Infirmary, Boston, Massachusetts. After removal of the bones from the cranial vault, they were placed in glass jars containing 200 cc. or Heidenhain-Susa solution and stored for 48 hours. Decalcification was done in 5 per cent trichloracetic acid. Following neutralization in sodium sulphate, they were dehydrated and embedded in celloidin. All the sections were cut at 20 mc. thickness, stained with haematoxylin and eosin, and mounted over glass slides.

Our material was derived from two groups. The first consisted of 96 ears from 68 individuals, varying in age from infancy to 84 years

TABLE I.

AGE DISTRIBUTION OF THE NORMAL EARS

Age group	No. of bones	No. of individuals
Below one year	15	10
1–10 years	2	1
11–30 years	13	7
31–50 years	13	10
51–70 years	23	15
Above 70 years	17	17
Total	96	68

(Table I). None of the cases had clinical nor pathological evidence of a specific ear disease. These were classified 'normal ears'. Pure tone audiograms were available on 17 of these ears, due to a contralateral diseased ear. All the audiograms showed normal air and bone conduction thresholds.

The other group of specimens consisted of 89 ears from 58 individuals who exhibited slowly progressive hearing loss as determined by history or by audiogram, and who had no specific known cause for the loss, either by history or otological examination. All the ears which exhibited other associated pathology with aging were excluded from the study. The individuals in this group varied in age from 33 to 91 years (Table II). All the audiograms showed perceptive deafness of varying severity, and

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TABLE II.

AGE DISTRIBUTION OF THE PATHOLOGICAL EARS

Age group	No. of bones	No. of cases
Below 40 years	2	1
40–50 years	6	3
51–60 years	6	3
61–70 years	30	20
Above 70 years	45	31
Total	89	58

four showed, in addition, a superimposed air-bone gap of 10–15 dB. in most of the frequencies The audiometric presentations of these ears are summarized in Table III.

TABLE III.

AUDIOMETRIC PRESENTATIONS OF PATHOLOGICAL EARS

Audiogram	No. of ears	No. of cases
With air-bone gap	4	3
Flat pattern	29	18
Descending pattern	12	8
High frequency loss	12	8
Profound deafness	5	4
Combined	27	17
Total	89	58

These ears were classified as 'pathological ears'.

Findings in normal ears

All the different parts of the auditory system showed degenerative changes with the advancing of age.

Middle ear

1. Auditory Ossicles.—These showed increased diameter of the Haversian systems and osteoporosis, manifested by the appearance of cavities of different sizes and shapes. This was accompanied by fibrous tissue replacement of the bone structure (Fig. 1). The bone marrow showed an increased number of fat cells, and the intra-osseus blood vessels showed atherosclerotic thickening of their walls with partial obliteration of the lumina.

2. Auditory joints.—Three grades of degenerative changes were found to occur in the incudo-malleal (IM) and the incudo-stapedial (IS) joints. Grade I consisted of fraying, vacuolation, and fibrillation of the articular cartilage. Grade II included thinning and calcification of the articular cartilage, atrophy and hyalinization of the articular capsule,

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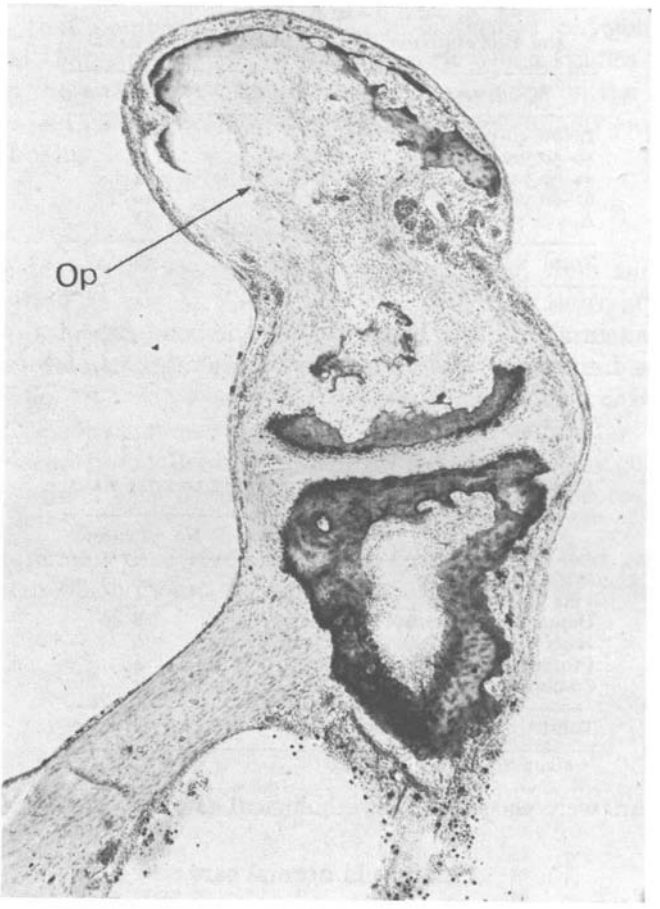


FIG. 1.

Osteoporosis (Op) and fibrous tissue replacement of the lenticular process of the incus in a man aged 71 years.

and the appearance of hyaline deposits in the articular disc. Grade III degenerative changes were characterized by the appearance of calcific deposits in the articular disc, and narrowing of the joint space. Fusion of the articular surfaces was found in 4·5 per cent of the ears in the (IM) joint and 2·2 per cent in the (IS) joint (Fig. 2).

The stapedio-vestibular (SV) joint showed surface and linear calcification of the secondary cartilage, which lines the joint cavity Severe degenerative changes with deep erosion of the articular cartilage were seen in some cases. In addition, two cases over the age of eighty showed sesamoid bodies in the annular ligament, as a result of severe degenerative arthritis of the joint.

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FIG. 2.

Incudo-malleal joint in a man aged 84 years showing grade (III) arthritic changes. (From Etholm and Belal, 1974.)

3. Auditory muscles.—The stapedius muscle showed increased amounts of endomysium and perimysium, while the tensor tympani showed an increased amount of fatty tissue surrounding the muscle fibres. These changes were noticed in younger age groups, but to a lesser degree.

4. Ossicular ligaments.—Calcification of the posterior incudal ligament was found in one ear from an individual 80 years old (Fig. 3).

5. Eustachian tube.—The cartilagenous portion of the tube showed diminution in the number of cilia, and disappearance of the goblet cells. The tunica propria showed a less evident lymphoid layer, while the elastic lamina showed a decreased number, splitting, and fragmentation

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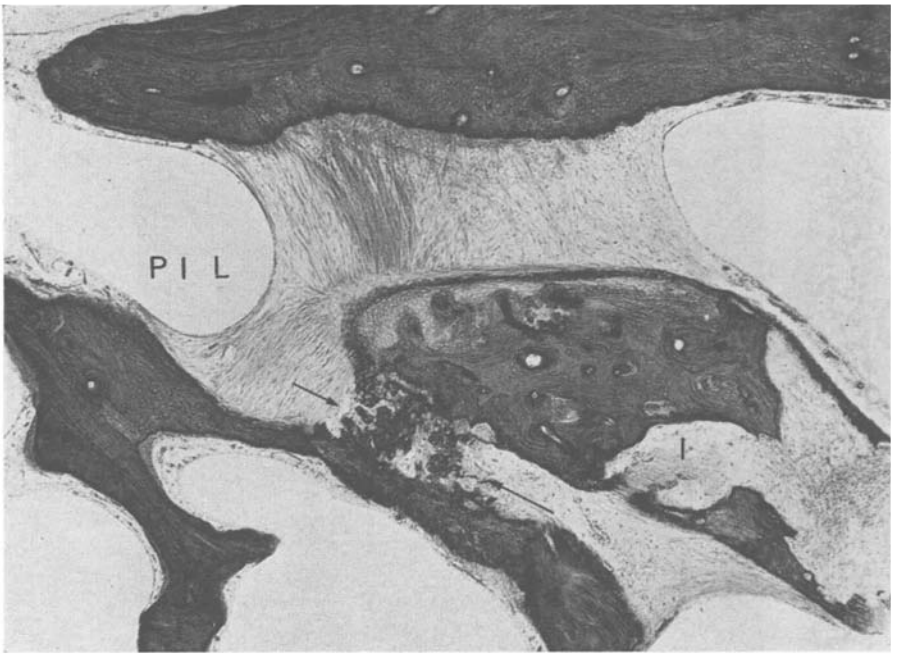


FIG. 3.
Calcification of the medial component of the posterior incudal ligament (PIL) in a man aged 65 years. The incus (I) shows fibrous tissue replacement of the bone structure.

of its fibres. The tubal cartilage showed a calcification of the chondrocytes. The senile changes in the oseous portion were less evident. The mucous membrane became thinner, and the tunica propria lost most of its lymphoid layer.

Inner ear

1. Stria vascularis.—This showed vacuoles and cystic spaces of different sizes in the intermediate layer, basophilic deposits beneath the basal layer, and thickening of the capillary walls with partial obliteration of their lumina (Fig. 4). In ears with a similar degree of postmortem autolysis, these changes were noticed in 70 per cent of individuals over 50 years old, and in 20 per cent of the cases under 20 years old.

2. Spiral ligament.—There was an increased number of acellular areas throughout the spiral ligament with the progress of age. The cochleas of old people showed two distinct zones in the spiral ligament, a large internal zone with many cystic spaces, and an external smaller zone containing scattered fibrocytes. At the junction of these zones, there was a dense layer of closely packed fibrocytes (Fig. 5).

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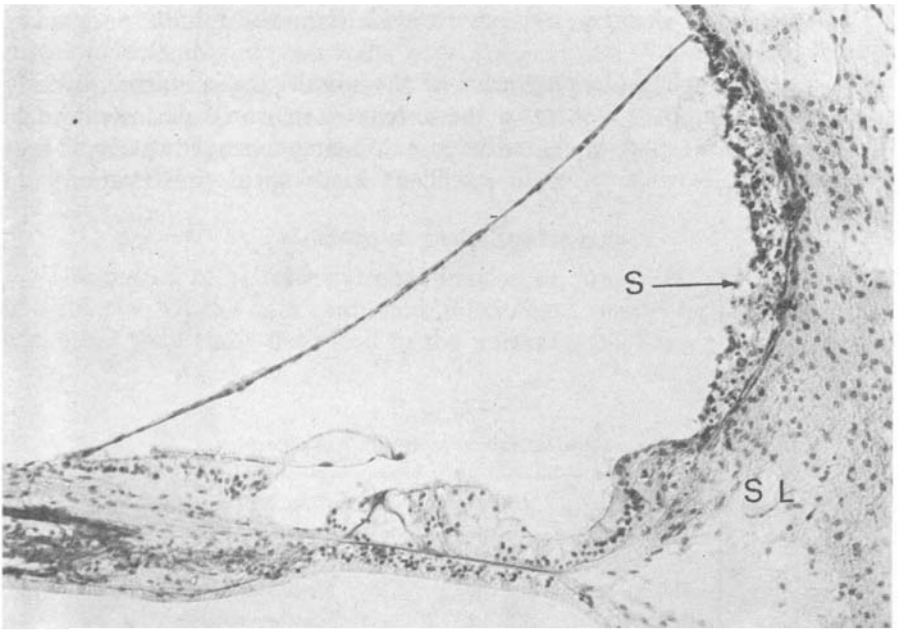


FIG. 4.
Stria vascularis (S) in the region of 12 mm. in a female aged 75 years showing vacuoles in the intermediate layer, and basophilic deposits beneath the basal layer.

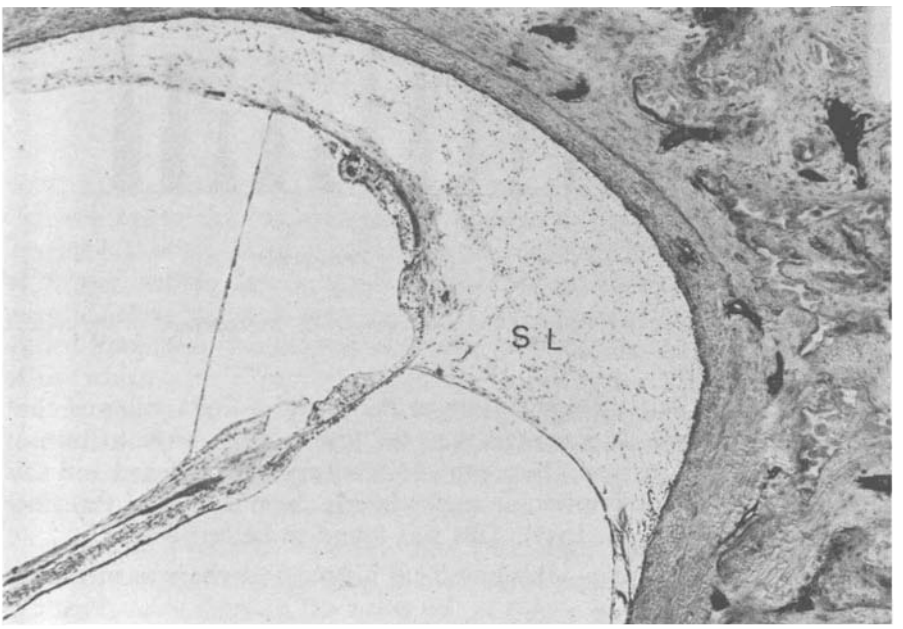


FIG. 5.
Cochlear duct in the region of 24 mm. in a female aged 75 years showing degenerative changes in the spiral ligament (SL).

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All the aging changes were more evident in the middle and apical coils of the cochlea.

3. Spiral ganglia.—Degeneration of the spiral ganglia started as early as 12 years. Figure 6 illustrates the extent of neuronal degeneration in twelve ears from twelve individuals exhibiting normal hearing. These were selected because of their excellent histological preservation and preparation.

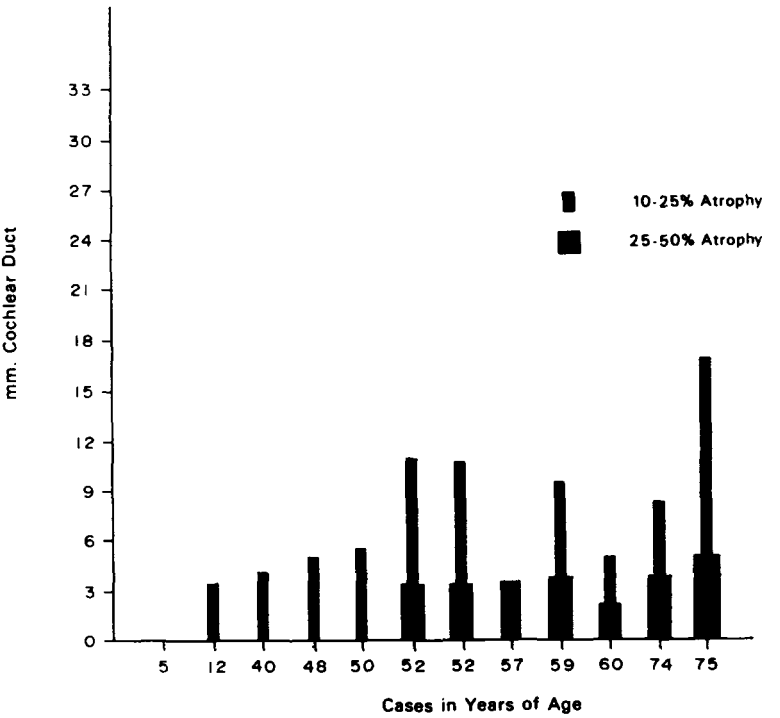


FIG. 6.

Graph showing progressive atrophy of the spiral ganglia in the basal coil of the cochlea with the progress of age.

4. Organ of Corti.—Degeneration of the organ of Corti followed that of the spiral ganglia. It was limited to the first half of the basal turn of the cochlea. In many ears, the organ of Corti appeared flattened and this corresponds to the description of angiosclerotic degeneration of the inner ear (Fieandt and Saxén, 1937). This was found to be artifactual.

5. Basilar membrane.—It showed no histological changes in any of our cases.

6. Cochlear blood vessels.—Angiosclerotic changes were found to affect two sets of blood vessels in the inner ear; capillaries of the stria

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vascularis, and the internal auditory artery. Strial capillaries showed a distinct thickening of their walls, especially evident in the age groups over 50. No similar changes were seen in the capillary net of the spiral ligament. The internal auditory artery showed hypertrophy of the internal elastic lamina, thickening of the media, and indistinct outlines of the musculosa.

Findings in pathological ears

The results of histological examination of these ears are summarized in Table IV. Of the ears examined, no changes were found in the middle ear, other than those described in the normal ones.

TABLE IV.

HISTOLOGICAL FINDINGS IN PATHOLOGICAL EARS

Site of pathology	No. of ears	No. of cases
Middle ear	–	–
Stria vascularis	29	18
Organ of Corti	12	8
Spiral ganglia	20	13
Spiral ligament	12	8
Basilar membrane	2	2
Combined lesions	14	9
Total	89	58

1. Stria vascularis.—This showed atrophy of varying severity in eighteen cases with a flat audiometric pattern. They suffered an average hearing loss of 40–50 dB. for the pure tone thresholds, and had an average of 72 per cent in speech discrimination scores. Fourteen of these cases were females, and four were males. Atrophic changes in the strial layer varied from mild changes in the cellular architecture to complete atrophy of the whole layer (Fig. 7). Lesions were found to be either localized over 1 mm. or less in size, or they were generalized, involving most of the cochlear duct. Lesions were more severe in the apical half of the cochlea. The commonest areas involved of the cochlear duct were the regions of 0–6 mm., of 16—24 mm., and of 29–43 mm.

2. Organ of Corti.—Twelve ears from eight individuals exhibiting a rise of the auditory thresholds over 4,000 c.p.s., showed degeneration of the organ of Corti in the basal coil of the cochlea. Figure 8 illustrates the extent of atrophy in the sensory structures of six ears from six individuals exhibiting high-tone-loss audiometric patterns. These were

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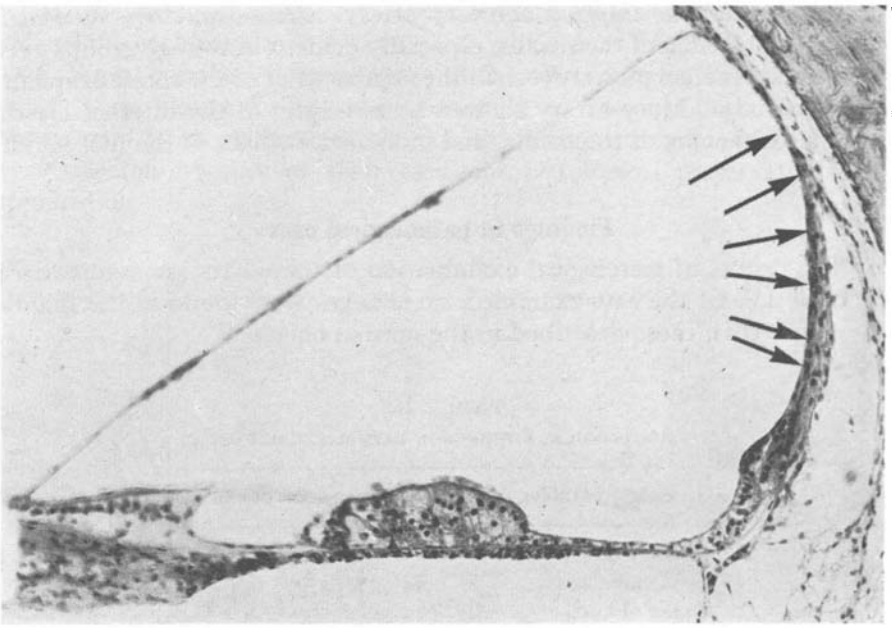


FIG. 7.

Severe atrophy of the stria vascularis in the middle turn of the cochlea in a female aged 77 years. She had hearing loss of 30–50 dB presenting with a flat audiometric pattern. The organ of Corti looks normal.

chosen because of the excellent histological preservation of the organ of Corti.

3. Spiral ganglia.—Severe atrophy of the spiral ganglia was the main pathological change found in twenty ears from thirteen individuals exhibiting an audiogram with low speech discrimination scores, i.e. an average of 44 per cent. Figure 9 shows the extent of neuronal degeneration in these ears.

4. Spiral ligament.—This showed severe degenerative changes in eight ears from eight individuals exhibiting descending audiometric curves, with average discrimination scores of 70 per cent for speech.

5. Basilar membrane.—Rupture of the basilar membrane was observed in two ears with profound hearing loss (Fig. 10). This was accompanied by severe atrophy of the spiral ligament.

6. Combined lesions.—Fourteen ears from nine individuals exhibiting different audiometric patterns were studied. Histological examination of these ears showed a combination of pathological changes involving the different structures of the cochlear duct (Fig. 11.)

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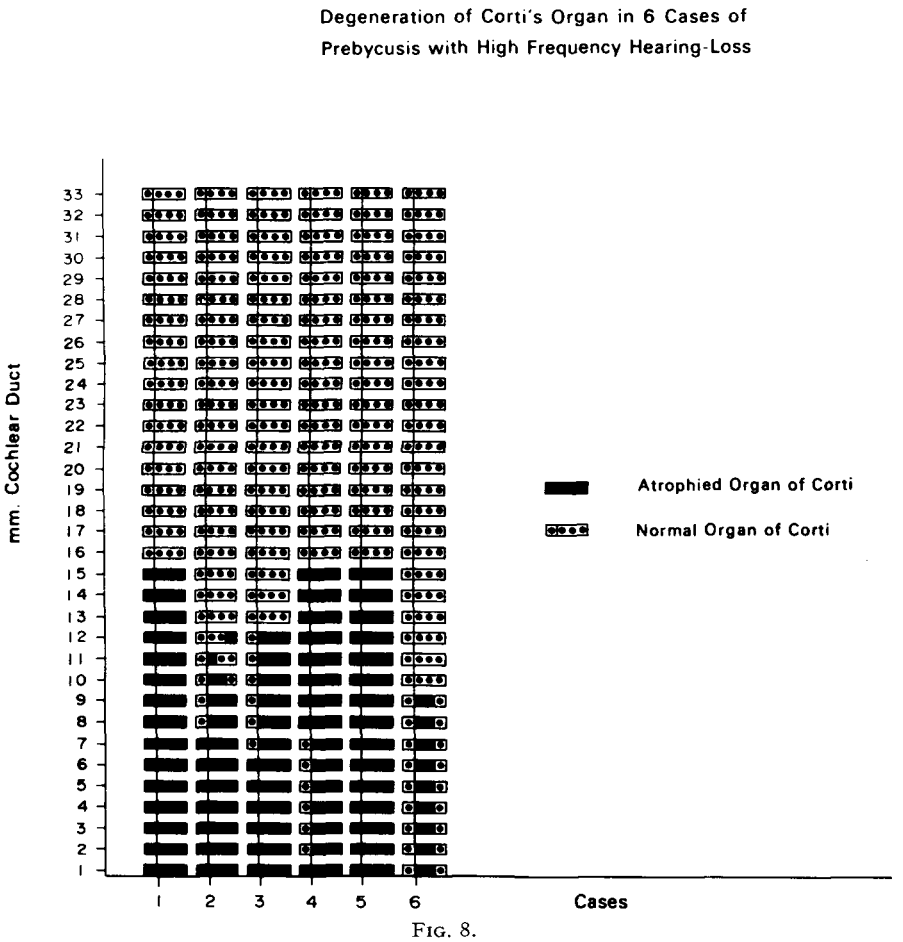


FIG. 8.

Graph showing the extent of atrophy of the sensory structures in six cases associated with high frequency hearing loss.

Discussion

The present work clearly demonstrates that the auditory system, like the rest of the body, is subject to aging changes. These degenerative changes vary greatly and are determined, most probably, by genetic factors. Some of these changes may be caused by environment; diet; and vascular, hormonal, metabolic, and nervous factors.

Due to this variation in the morphological changes, there is confusion between the gerontological processes occurring in the auditory system. Physiological and pathological processes seem to be interacting, hence differentiation between 'normal aging' and 'pathological aging' seems difficult.

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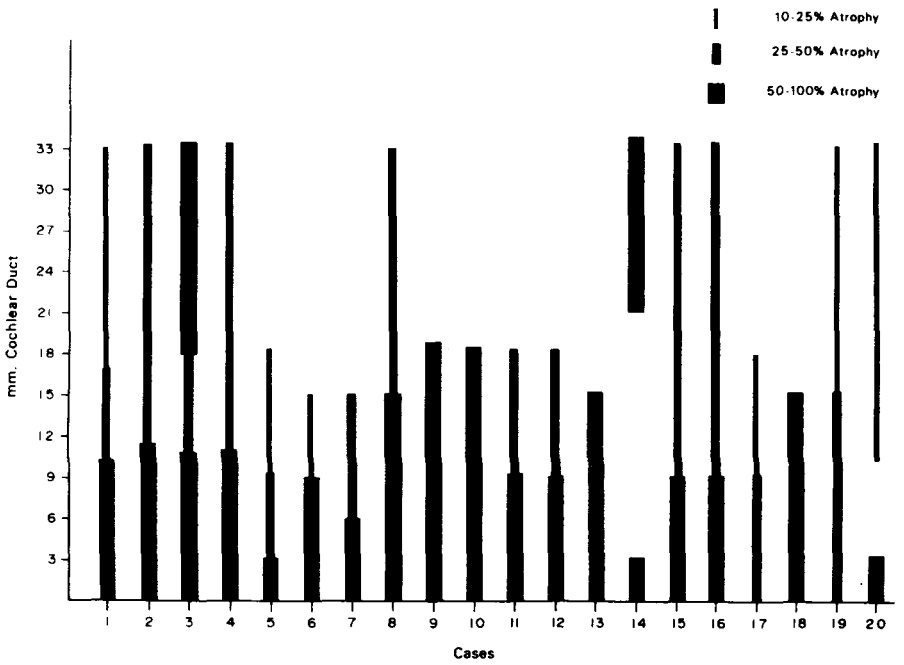


FIG. 9.

Graph showing the extent of atrophy in the spiral ganglia seen in twenty ears from thirteen individuals who had perceptive deafness with bad speech discrimination scores.

The auditory mechanism is better adapted for the detection of slight changes in function than any other organ in the human body (Fowler, 1944). The spatial arrangement of the auditory neural elements, and the easily-applied means for testing them, make it possible to measure any slow functional decline over a long period of time, and with an accuracy unattainable in any other system of the body.

It is evident from the present wotk that all human ears undergo degenerative changes with aging. Only in a small percentage of cases are these changes severe enough to cause elevation of the auditory thresholds. Thus, it seems that there are two aging phenomena which involve the auditory mechanism. The first phenomenon is physiological, and affects the ears of all normal people as they grow older. The senile changes are seen equally in the conductive and the perceptive parts of the ear. This phenomenon constitutes the auditory manifestation of the general aging process involving the body. We suggest labelling this phenomenon 'auditory senility'. The normal senile changes in the conductive system, e.g. ankylosis of the (IM) and (IS) joints, calcification of the posterior incudal ligament . . . seem to have no effect on the auditory thresholds (Etholm and Belal, 1974). On the other hand, degeneration of the sensory

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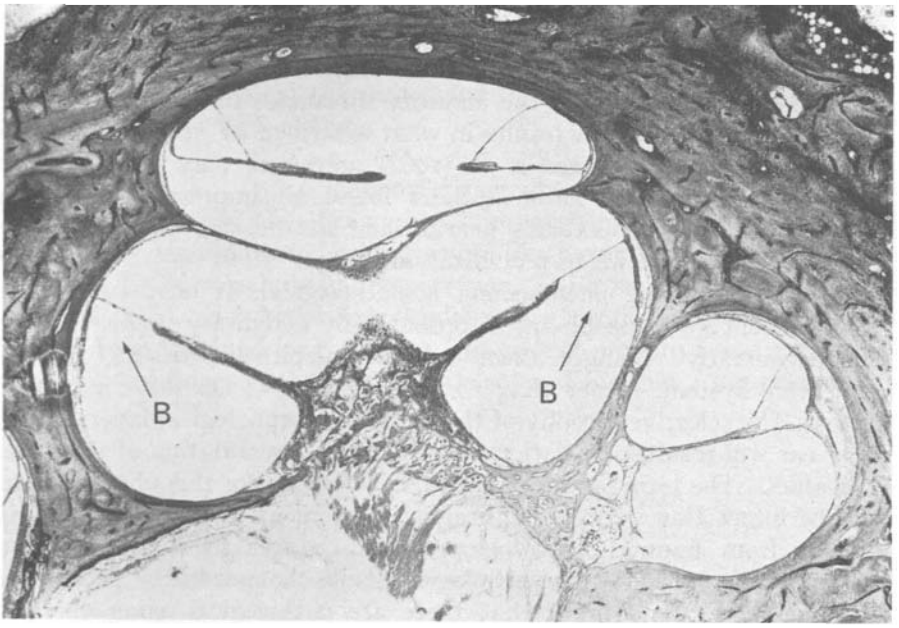


FIG. 10.
Left cochlea showing ruptured basilar membrane (B) in a case of profound hearing loss associated with aging.

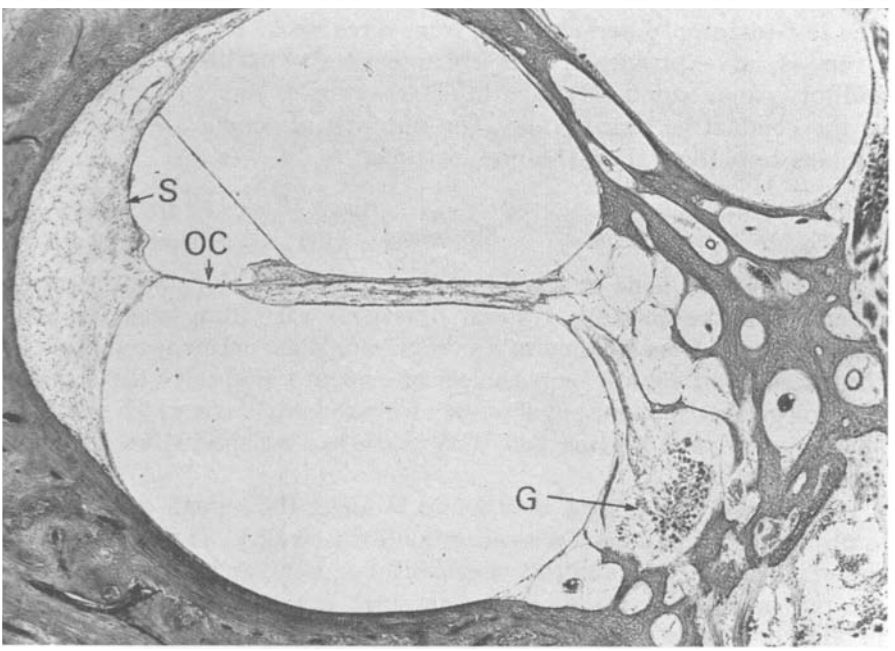


FIG. 11.
Basal coil of the cochlea from a case of profound hearing loss associated with aging, showing severe degeneration of the stria vascularis (SV), organ of Corti (OC), and spiral ganglia (G).

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and neural structures in the first few millimetres of the basal coil of the cochlea, causes elevation of the auditory thresholds for high frequencies as the age advances. This results in what is termed as 'standard presbycusis curves' and, as Rosen *et al.* (1962) suggested, may be caused by chronic noise trauma. These authors found an impressive difference between the elderly Maaban's hearing and the hearing of the average citizen in one of the western civilization's major cities.

The second aging phenomenon is pathological. It only involves a small percentage of people, most probably by hereditary predisposition. The degenerative changes affect only neuro-epithelial tissues, i.e. the perceptive system, rather than connective tissues,, i.e. the conductive system. The selective atrophy of the different morphological layers in the inner ear will result in variations in the clinical presentation of this type of deafness. The term 'auditory decay' is suggested for this phenomenon.

The interaction between both aging phenomena in the human ear is evident from finding physiological senile changes in the conductive system of the ears showing pathological senile changes in the perceptive system. There is no proof that there are pathological aging changes involving the conductive system of hearing. We were not able to find histo-pathological changes to explain the air-bone gap in the available cases. A temporary conductive hearing loss at the time of audiometry (the test-to-autopsy period varied from three weeks to one year) may be given as an explanation. The collapse of the cartilagenous external auditory canal, commonly seen in elderly people, may have contributed to the conductive hearing loss. The question of conductive presbycusis remains hypothetical at the present time.

Summary

A study was made on human temporal bones to elucidate the effects of aging on the auditory system. Ninety-six ears from 68 individuals, varying in age from infancy to 84 years, served as 'normal ears'. None of these cases had clinical or pathological evidence of specific ear diseases. 'Pathological ears' consisted of 89 ears from 58 individuals which exhibited slowly progressive hearing loss, and which had no specific known cause for the loss.

Two aging phenomena were found to affect the human ear. The first is physiological, and has been called 'auditory senility'. It involves all the different parts of the auditory mechanism equally, and is responsible for the rise in auditory thresholds for the high frequencies, commonly seen in elderly people. The second phenomenon is pathological, and was termed 'auditory decay'. It involves only the perceptive system of the ear, and results in different types of clinical presentations, according to the morphological layer involved.

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Therefore, the recognition of two aging phenomena which affect the human ear may explain the reason for the wide variations in auditory acuity associated with old age.

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