

AN INQUIRY INTO OLD AGE DEAFNESS.

Thesis for the degree of M.D.

by

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C O N T E N T S.

	<u>Page.</u>
Introductory . . . . .	1
Anatomical . . . . .	4
Physiological. . . . .	9
Clinical Examination . . . . .	13
Clinical Findings. . . . .	16
Pathology. . . . .	24
Literature . . . . .	35
Conclusions. . . . .	44
Bibliography . . . . .	46

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## AN INQUIRY INTO OLD AGE DEAFNESS.

### Introductory.

Comparatively little work has been done on this subject in this country but many investigations have been made on the Continent during the last thirty years and some, more recently, in America.

For the purposes of the present investigation I took the records of cases classified in this group of deafness who attended the Royal Infirmary, Edinburgh, over a period of twenty years, together with records made personally in the Craiglockhart Poor-house, and studied the clinical findings.

It was difficult to place these cases in a hard and fast category; various factors such as heredity, environment, disease - both aural and constitutional - seemed all to play an important part in many of them and these factors had to be carefully weighed before including each case as definitely one of senile deafness.

The most usual age for the first manifestation of senile deafness is between forty and fifty but frequently it is not actually noticed by the patient until later. Denker sums up the first stages as typical: "In the beginning people complain chiefly that/

that they no longer hear high-pitched sounds as, for example, the ringing of bells or the high tones at concerts. This state of things can last for years without the power of hearing words being lessened to any extent. It is only when the deafness reaches such a pitch that personal intercourse with friends is interrupted that old people seek the advice of a physician."

Some of the deafness found in later life may have arisen in youth from various causes - otosclerosis, influenza, scarlet fever, measles, mumps, rheumatism, syphilis, or from disease in the brain, also tumours of the 8th nerve. Here I want to inquire more particularly into deafness in advanced life per se; that is, a deafness which is purely a result of growing old. It would be of interest to know if there is a definite pathological picture common to this type.

We recognise two kinds of deafness.- First, interference with the sound conducting apparatus. This condition is known as middle-ear deafness and comes from any disease of the middle-ear or of the chain of ossicles. An obstruction in the external meatus will also cause deafness. Second, that arising from the sound perceiving apparatus. This is/



is called nerve deafness. It is caused by changes affecting the cochlea, comprising Corti's organ, the spiral ganglion and the end of the nerve stem.

Lesions between the inner ear and the brain centres come also into the category of nerve deafness.

True senile deafness is an inner ear deafness together with a shortening of bone conduction. No typical changes are found in the outer or middle ear but in advanced age the drum membrane will in most cases be found to be opaque.

Sections taken from the temporal bones of five deaf people between the ages of sixty and seventy, also from those of two old deaf dogs, compared with those of normal hearing men and dogs, show changes in the cochlea and nerve stem.

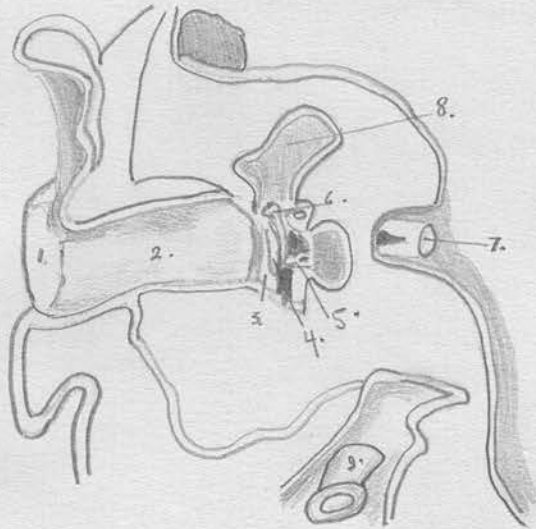
Anatomical.

The ear consists of three parts - an outer, middle and inner.

The outer part begins with the auricle, which consists of cartilage, connective tissue and skin, then the external acoustic meatus, which is a canal about one inch long. The cartilage of the auricle is continued round the outer third of the canal, while the inner two-thirds has a bony skeleton. The medial end of the canal is closed by the tympanic membrane which is formed from fibrous tissue with radiating and concentric fibres. The handle of the ossicle named the malleus is fixed into about the centre of the membrane; a small portion, the pars flaccida, at the top of the membrane has no connective fibrous tissue. The membrane is covered externally by the mucous membrane of the middle ear.

The middle part of the ear, or tympanic cavity, is an irregular air space. The Eustachian tube, from the nasopharynx, forms its anterior portion. An aperture posteriorly and above leads into an air sinus, the tympanic antrum, which in turn communicates behind and below with the mastoid air sinuses lined by mucous membrane continuous with the lining of the tympanic cavity. This cavity contains also the small bones of the ear - the malleus, incus and stapes. The malleus/

Diagram of a Vertical Section through the left Temporal Bone.



1. Auricle.
2. External Auditory Meatus.
3. Tympanic Membrane.
4. Malleus.
5. Stapes.
6. Incus.
7. Nerves in Internal Meatus.
8. Attic.
9. Internal Carotid Artery.



- | Malleus.                   | Incus.                       | Stapes.                       |
|----------------------------|------------------------------|-------------------------------|
| 1. Handle.                 | 1. Articulation for Malleus. | 1. Articulation for Incus.    |
| 2. Articulation for Incus. | 2. Articulation for Stapes.  | 2. Footplate for Oval Window. |



malleus has a head, neck, short process and handle which is fixed to the tympanic membrane. The incus is like a premolar tooth; it has a long part which articulates with the stapes. The body has an articulation on the anterior surface with the malleus. The stapes is like a stirrup; its footplate has a round ligament fixing it into an oval window on the medial wall of the tympanic cavity.

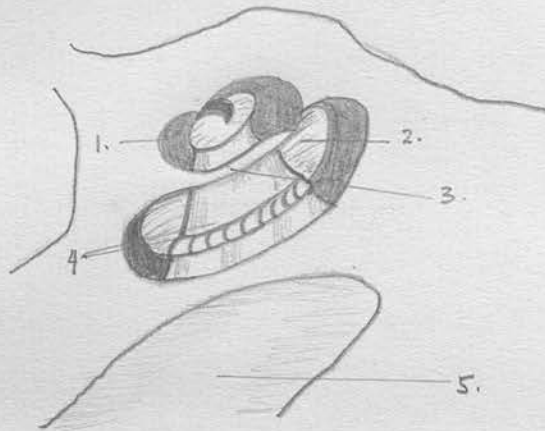
The inner ear is formed by a bony case containing a membranous portion. The membranous part arises in the embryo from two plates of ectoderm on the hind brain which dip in and invaginate themselves in it and form two vesicles which develop into three membranous canals - the utricle, saccule, endolymphatic sac and the cochlea. This organ is called the labyrinth, including the bony case, and consists of two parts - the static and the otic, the former connected with the sense of position in space and the latter with hearing. The bony case is formed of hard cartilaginous bone with perilymph surrounding the membranous part inside. In form the labyrinth consists of three semicircular canals, the vestibule and the cochlea; these are all hollow and the semicircular canals contain similar structures formed from membranous tissue. The others contain more specialized structures.

The bony cochlea is a tube like a snail shell, coiled/



5a.

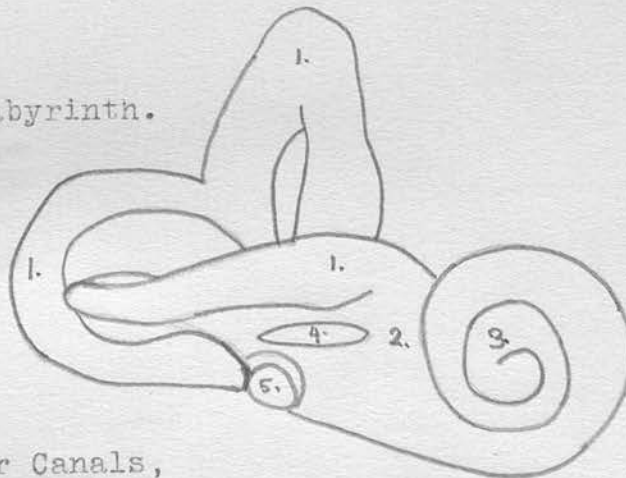
Diagram of a vertical section of the  
Bony Cochlea,



1. Scala Tympani.
2. Scala Vestibuli.
3. Osseous Spiral Lamina.
4. Bony Modiolus.
5. Internal Acoustic Meatus.

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Diagram.

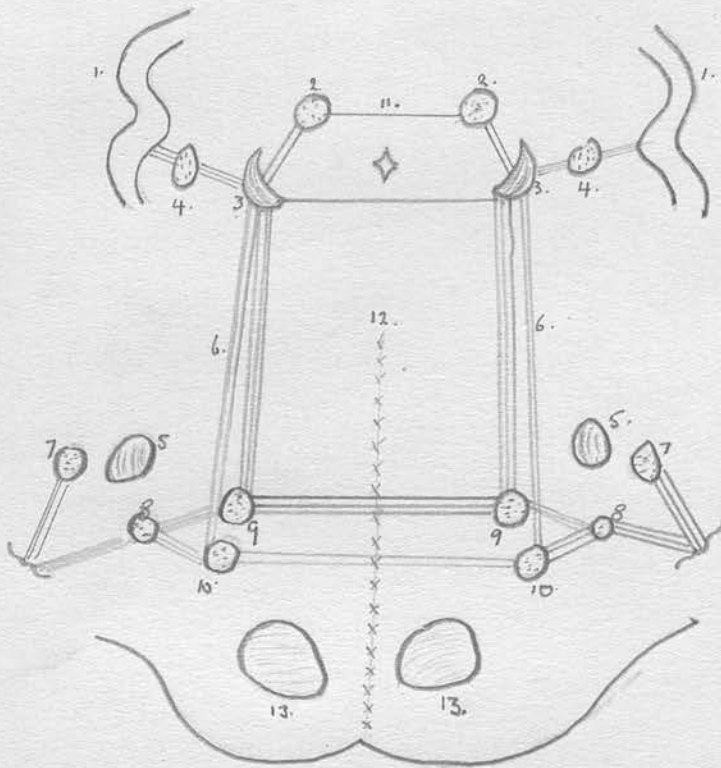
The Osseous Labyrinth.



1. Semicircular Canals,
2. Vestibule,
3. Cochlea.
4. Oval Window.
5. Round window.

coiled two and a half times round a hollow pillar called the modiolus. An osseous ridge, called the spiral lamina, divides this tube in half, completed by the basilar membrane. The fibres of the cochlear nerve are conducted through the tube. The auditory nerve consists of two parts - the cochlear and the vestibular. They carry afferent impulses from the labyrinth to the brain. The vestibular part of the auditory nerve joins the cochlear portion at the medial end of the internal acoustic meatus: from here this trunk runs in company with the facial nerve to the posterior border of the brachium pontis.

Returning to the structure of the membranous cochlea, the tube twining two and a half times round the modiolus when cut across transversely shows three divisions - the osseous spiral lamina twining round the pillar forms a ridge: at its outer edge is fixed the basilar membrane, which stretches to the outer wall of the cochlea. From thickened periosteum at the edge of the lamina rises up a thin structure called Reissner's membrane, which also is attached to the outer wall forming a canal known as the cochlear duct. The remaining two divisions are known as the scala tympani below the duct and the scala vestibuli above. They contain perilymph and are connected by an opening at the apex of the cochlea/

Diagram of Auditory Paths in the Brain.

1. Cerebral Cortex.
2. Interior Corpora Quadragemina.
3. Nucleus of lateral lemniscus,
4. Medial geniculate body.
5. Restiform body.
6. Lateral lemniscus.
7. Lateral cochlear nucleus.
8. Medial cochlear nucleus.
9. Superior olivary nucleus.
10. Trapezoid nucleus.
11. Anterior commissural fibres.
12. Median plane.
13. Cerebro-spinal fasciculus.



cochlea called the helicotrema. At its lower end the scala vestibuli has the oval window, an aperture on the medial wall of the tympanic cavity closed by the footplate of the stapes. At the lower end of the scala tympani is the round window, also on the medial wall of the tympanic cavity and closed by a membrane. The perilymph in the scalae is identical with cerebro-spinal fluid.

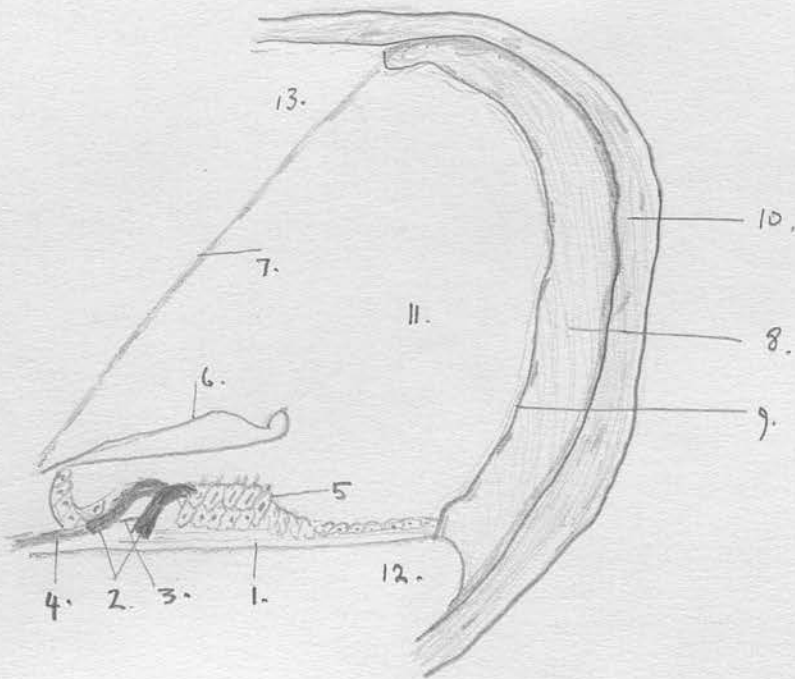
A third opening near the base of the cochlea leads to a small canal called the aqueductus cochlea which communicates on the under surface of the petrous bone with the subarachnoid space: the perilymph is thus in direct connection with the cerebral fluid.

The cochlear duct contains endolymph as does the membranous portion of the semicircular canals, and this membranous portion of the cochlea is connected to the sacculle by a fine duct called the canalis reuniens.

The cochlear duct is triangular in shape and bounded by - (1) Reissner's membrane: in front of this the thick connective tissue attached to the osseous spiral lamina ends in an overhanging border, the membrana tectoria. (2) The basilar membrane: on this lies the organ of Corti, the end organ of hearing. The cochlear nerve in the modiolus gives off branches through the spiral lamina as it winds round to the apex/

7a.

Diagram of a transverse section through the middle coil of the Cochlear Duct,



1. Basilar membrane.
2. Rods of Corti.
3. Nerve fibres.
4. Nerve fibres.
5. Hair cell.
6. Tectorial membrane.
7. Reissner's membrane.
8. Spiral ligament.
9. Striae vascularis.
10. Bony capsule,
11. Cochlear duct.
12. Scala tympani.
13. Scala vestibuli.

apex. The spiral ganglion lies between the two bony plates of the lamina and fibres from the ganglion are distributed to the cells of the organ of Corti. The organ of Corti consists of specialised epithelium fashioned like rods leaning towards each other and forming a tunnel through which the peripheral nerve endings run. A row of cells lies on the inner side of the rods and four or five cells upon the outer side: these have hairs at the top of the cell. The outer rods end in processes continuous with the processes of the outer cells, forming a kind of reticulate membrane and through it the hairs project. Supporting cells, called cells of Deiters, are on the outer side, diminishing to cuboid cells on the basilar membrane and (3) the boundary of the duct called the stria vascularis, a vascular tissue which is resting on a fibrous projection of the outer bony wall of the cochlea known as the spiral ligament.



Physiological.

There are several theories with regard to the part played by the cochlea in the reception of sound. Sound waves in the air vary in pitch, loudness and quality. The question is, how and where are these different variations analysed. The tympanic membrane vibrates to the sound waves in the air, setting in motion the malleus, incus and stapes. The stapes, moving laterally, sets the perilymph moving up the scala vestibuli, through the helicotrema, down the scala tympani. The membrane of the round window yields a little to the pressure and then there is back pressure along the path the wave has come. This increased pressure in the perilymph causes changes in the cochlear duct.

Pressure of sound waves in the air will be increased on their way to the inner ear because the tympanic membrane is twenty times larger than the foot of the stapes in the oval window and the bones acting as a lever to the stapes have a pressure of 3-1 so the stapes transmits the whole sixty times greater. The movement in the perilymph causes the basilar membrane to rise and fall.

There are two main theories - (1) The Resonance theory/

theory and (2) the Telephone theory.

The first is associated with Helmholtz; he thinks of the basilar membrane as a piano, its fibres increasing in length from base to apex, and the spiral ligament to which it is attached decreasing in size from base to apex, so that the fibres are smallest and stretched tightest at the base. These respond to the high pitched tones as in a piano and the fibres near the apex are longest and have less tension and so respond to the low tones.

The separate fibres when vibrated in turn carry their stimulus to the hair cells; from there the stimulus passes to the nerve endings and thence to the brain. A model of a cochlea was made and tests with a tuning fork which were carried out showed this reasoning to answer fairly well.

Ebner says - "The tectorial membrane, especially at its free end, seems to be the mechanism for sympathetic vibration". Helmholtz thinks the cochlea must be the site for analysis of sound but there are many objections to his theory about the basilar membrane or any other parts of the cochlea being resonators because of their structure and the impossibility of giving adequate physical explanations to account for the conveyance of sound vibrations to the nerve endings. The basilar membrane fibres do not/

not seem capable of answering individually to separate vibrations for they are bound together.

(2) The Telephone Theory.- A tele phone has a transmitter to turn sounds into electric vibrations which travel along the wire and the receiver turns them back into sound vibrations, the realisation of speech taking place in either the ear or brain of the listener.

Ewald thinks that the whole basilar membrane vibrates and that different tones or waves make it vibrate to different extents, thus causing stimulation of different nerve endings of the hair cells. The stimulus then travels along the nerve path to be analysed in the brain. He supported his theory with a piece of rubber membrane put under a microscope and showing when vibrating, fixed intervals for the crest of a wave of the same note.

Wrightson and Keith think differently about the matter. They believe the basilar membrane to move to the same extent in its entirety with the wave of increased pressure through the perilymph. The basilar membrane is eighty-one times greater than the helicotrema in area; therefore the wave of pressure in the perilymph, which flows through the scala tympani, displaces the basilar membrane upwards on its path to the helicotrema. This aperture connects the scala tympani with the scala vestibuli at the apex of the cochlea. They believe the basilar membrane rises/



rises and falls in its entirety and the organ of Corti rises with it. The hairs at the top of the cells of Corti's organ are pressed against the membrane tectoria, which is attached to the bony spiral lamina and does not move. The hairs bend towards the modiolus, then return to the upright position. They are bent outwards away from the modiolus on the return phase of pressure, afterwards resuming their upright state.

From these four phases, according to the nature of the sound, Wrightson and Keith think impulses travel to the brain, where their nature is analysed. This does not fit in with the theories which have been demonstrated by Munk and Baginsky - that high tones are received at the base of the cochlea and low tones at the apex. Munk destroyed the basal portion of the cochlea in dogs and found they were left only with hearing for low tones and noises. Baginsky said "I found when the apex of the cochlea was destroyed the animal appeared only to hear the high tones".

Arguments in favour of Helmholtz's theory and against Wrightson and Keith's, pointed out by J.S.Fraser, are -

- (1) In old age and arteriosclerosis people cannot hear high notes.
- (2) In deaf-mutes small islands of hearing residue are found clinically and answering to this are the histological findings.
- (3) In guinea pigs observers have noticed that long stimulation of several notes cause certain areas in the cochlea to degenerate.

Gray says that no nerve could transmit stimuli at the rate of vibrations of a normal upper tone limit.

### Clinical Examination.

Before examining a case we obtain a history from the patient, with special reference to hereditary factors, illness or injury. Inquiries are made as to the duration of the deafness and the accompanying symptoms.

A careful examination should be made of the nose and throat. Then we proceed with the otoscopic examination, noting the condition of the tympanic membrane. Hearing tests with tuning forks, watch, etc., are next carried out.

In the three tests commonly used a medium-pitched tuning fork is employed.

(1) Rinne's test: This consists in comparing air conduction with bone conduction. If the tuning fork can no longer be heard by air conduction and if the patient still hears it when placed on the mastoid, the test is "negative", the bone conduction being longer than air conduction. This is found in lesions of the sound-conducting apparatus. In inner ear deafness Rinne's test is "positive".

(2) Schwabach's test: This consists in comparing the patient's bone conduction with that of a normal hearing person. The examiner, who must have normal hearing himself, strikes the tuning fork and places it on his own vertex; when he can no longer hear it he/

he places the fork on the patient's vertex and, if the patient can hear it, his bone conduction is lengthened. On the other hand, if the examiner can hear the tuning fork on his own vertex after the patient has ceased to hear it on his, then the patient's bone conduction is shortened. Bone conduction is lengthened in lesions of the sound-conducting apparatus, particularly in otosclerosis, and shortened in inner ear deafness.

Ziffer has proved: "One almost always finds a shortening of the bone conduction in people over fifty years of age, even when their hearing is perfectly normal". With age bone is generally supposed to lose elasticity because there is more calcium in it as the body grows older. We therefore call it senile deafness.

(3) Weber's test: This is for distinguishing between lesions of the sound-perceiving and the sound-conducting apparatus in cases of unilateral deafness. I found it unreliable in senile deafness.

High Tones: A small tuning fork,  $e_4$ , is used to test the perception for high tones. In inner ear deafness we find a loss of hearing for high tones and a decrease in the time of hearing the fork.

Low Tones: A large tuning fork,  $C_{32}$ , is employed for testing the low tones. Low tones are generally/



generally retained longer than high tones in inner ear deafness but in advanced cases they also are lost.

The hearing distance for a whisper or for the conversation voice is noted. If a great degree of deafness is present the voice is raised up to a shout.

Patients who complain of vertigo and falling must have their vestibular reactions tested.

Clinical Findings.

I examined the records of six hundred cases of inner ear deafness attending the Ear and Throat Department of the Royal Infirmary, Edinburgh, over a period of twelve years and classified them, first, according to their ages and, second, according to their ages with predisposing cause of deafness where given, as follows.--

Age.	Number of cases.
10 - 20 years	20
21 - 30 "	48
31 - 40 "	90
41 - 50 "	113
51 - 60 "	119
61 - 70 "	136
71 - 80 "	70
Over 80 "	7

Predisposing cause (stated in 194 cases)	Age.							Total
	10-20	21-30	31-40	41-50	51-60	61-70	71-80	
Hereditary history	5	3	2	5	7	1	1	24
Infectious disease	6	2	4	2	1	3	-	18
Influenza	-	-	7	2	6	3	-	18
Rheumatism	-	-	1	3	1	1	-	6
Noisy work	-	3	6	8	7	14	2	40
Nerve deafness follow- ing middle-ear disease	1	-	-	3	2	-	-	6
Accidents	1	-	1	3	2	3	1	11
Cranial lesions	-	-	1	1	2	-	-	4
Congenital and acquired Syphilis	9	20	13	9	7	1	-	59
Otosclerosis with nerve deafness	-	1	-	2	-	-	-	3
Disseminated sclerosis	-	-	-	-	-	1	-	1
Malaria and quinine	-	-	2	1	1	-	-	4
	22	29	37	39	36	27	4	194

In ninety-five patients under 50 years of age no predisposing cause was noted. These cases only gave a history of increasing or temporary deafness. In about three hundred cases over 50 years of age, in which no predisposing cause is given, I found true clinical pictures of senile deafness.



Tinnitus.

In addition to increasing deafness, "noises in the ear" are frequently noted as a distressing symptom. The noises are described as "hissing", "steam", "running water", "humming bees", "bells ringing" and "beating like a hammer". These are found in any ear disease, not invariably and not always in senile deafness. They are frequently found in arteriosclerosis, sometimes synchronous with the pulse. Drugs such as quinine and the salicylates produce tinnitus. Amongst 313 cases between the ages of 40 and 90, 165 actually had this complaint.-

Age	No. of Cases	Noises
40 - 50	47	24
51 - 60	87	46
61 - 70	111	71
71 - 80	62	22
81 - 90	6	2

I have been unable to discover any particular reason or rule from the other signs and symptoms why some patients have noises and others have none. The largest percentage of patients with noises in the ear occurs between 60 and 70. This may accord with the arteriosclerosis found at this age. We would expect a higher percentage between the ages of 70 and 80 but probably/

probably patients with this tendency die before reaching this age or do not frequent the Ear Department, accepting their lot as natural to old age.

From a pathological viewpoint, Gray says - "Tinnitus may be due to bare nerve endings which in nerves of common sensation always register pain".

#### Paracousis Willisii.

This symptom, in which the patient hears better in a noisy place, i.e., a tramcar or railway train, is often found in people suffering from middle-ear disease. Gray suggested: "The obstacle to the deep tones becoming sufficient, the possibility arises that an over-tone, although not independently perceivable, may fuse into and reinforce the signal tone so this is heard at a lower threshold than normally". Gray said that this was due to poor insulation of the myelin sheaths of the axis cylinder and strong impulses on/nerve flow to other nerves.

Amongst 245 cases of inner ear deafness, nine had Paracousis Willisii. Six of these were engaged in noisy occupations. Forty-eight patients stated definitely that they heard better in a quiet place.

Age	No. of cases	Hears best in quiet place	Hears best in noisy place
40-50	47	9	2
51-60	87	15	4
61-70	111	24	3

Vertigo.

Vertigo was a symptom in thirty-one patients and falling in three. Records regarding the vestibular reactions did not show anything definite.

Rinne's test was almost invariably positive.

In 265 cases in which this test was carried out, the results were as follows.-

Age	Positive	Negative
40-50	41	2
51-60	72	-
61-70	90	-
71-80	50 (two had no hearing)	3
81-90	5	-

Schwabach's test. In the greater number bone conduction was typically shortened.-

Age	Shortened	Lengthened	Normal	no hearing
Over 40	26	4	3	4
Over 50	55	3	2	-
Over 60	48	2	-	-
Over 70	47	5	-	1
Over 80	3	-	-	1

Three were definitely cases of otosclerosis with nerve deafness in addition. Rinne's test was negative and low tones as well as high tones were lost. Five had complete absence of bone conduction.



The Upper Tone Limit was lowered in every case. The degree in different decades over fifty are shown in the following table.-

	50-60	61-70	71-80	81-90
Great loss	4.5%	24.5%	40%	14%
Marked loss	49%	51.5%	35%	56%
Slight loss	46%	24%	25%	30%

These figures show a progressive loss of hearing up to the age of eighty. There are records of only seven cases over eighty years of age and, of these, three had quite good hearing, only one being very deaf.

With regard to Low Tones, over 75% of the patients heard low tones well. A few among the remaining 25% heard C.64 but in a good many the low tone limit was raised somewhat higher.-

Age.	Number of cases	Low tones	
		Heard C.32	Limit raised.
40-50	45	40	5
51-60	59	49	10
61-70	97	69	28
81-90	4	2	2

The numbers show a slightly higher percentage of raising of the lower tone limit between sixty and seventy than between seventy and eighty, but a number of cases in the former heard the forks C.64 and C.128. In/

In the latter, half the cases heard no low tuning fork.

Zwaardemaker found a decrease in the high tones progressively with age and he also noted that a greater intensity in the tone was needed for hearing as age advanced. This record was made of normal hearing individuals for their age; the records were on people between 50 and 90 who complained of increasing deafness but in comparison my figures also show an increasing loss of high tones as the age advances.

Cuperus confirmed Zwaardenake's report: he also found that "in old age the lower tone limit moves up. I found this in a number of cases to be true, depending on the degree of deafness.

Ziffer states that "a diagnosis of inner ear deafness due to age must be made only with the findings of definitely increased bone conduction together with very bad hearing for whisper and falling out of high tones. This rule is maintained without regard to age".

Senile changes do not always go with age. Among one hundred and ninety cases of between fifty and sixty years I found a number of patients showing advanced deafness in the same degree as those over seventy.

Sporleder/

Sporleder examined a hundred people indiscriminately of between fifty and ninety with regard to their capacity for hearing. He found that from the fiftieth year onwards the capacity for hearing steadily decreased in the normal way. The medium degrees of deafness are distributed equally among the ages of sixty and seventy, while the highest degrees are rare in this period and frequent between the ages of seventy and eighty. Medium degrees of deafness between the latter ages are in the minority and those who hear well exceptions.

Comparing my records with these, I found that a practically equal percentage heard well in the two groups between sixty and seventy and between seventy and eighty. With regard to medium degrees of deafness there was a higher percentage in the first group but advanced deafness was greater in the latter.



Pathology.

Senile deafness is a nerve deafness, and where on the nerve path do we find a pathological change? The path extends from the inner ear to the cortical centres in the brain. Investigation must therefore be carried out in the cochlea, along the nerve path and in the brain.

This paper deals with an investigation of the inner ear and the nerve stem lying in the internal acoustic meatus, because the author believes in Helmholtz's theory of resonance and the characteristic loss of high tones in old age suggests changes in that part of the cochlea which is held to be concerned with their reception.

My sections are taken from the inner ears of five aged patients who showed the usual signs of increasing deafness in old age. They were all functionally examined before death. The inner ears of two dogs were also examined; in each case there was a history of gradually increasing deafness as old age advanced. The sections were prepared by the celloidin method over a period of three years.

Case 1. A. H., male, aged 71 years.

Functional examination.- Rinne's test positive:  
bone/

bone conduction shortened: C.4096 heard: low tones heard: watch heard at two inches by right ear, on contact with auricle left ear.

Microscopic findings.- The sections show the picture of an almost normal ear with the exception of the basal coil of the cochlea, where the organ of Corti is missing. It is replaced by a row of epithelial cells. In the middle and upper coils Corti's organ is normal. The cells of the spiral ganglion in the basal coil are atrophied and there is a great amount of connective tissue in the spaces where the cells are lying. In the spiral lamina of the basal coil, between the ganglion and Corti's organ, the terminal nerve fibres are missing. The spiral ligament is normal throughout. The trunk of the cochlear nerve in the internal meatus appears normal in structure.

Case 2. C.B., male, aged 65 years.

Functional examination.- Rinne's test positive: bone conduction shortened: upper tone limit C.1924: 1024? low tones heard: watch heard on contact by both ears.

Microscopic findings.- These sections also present a picture closely resembling the normal except in the basal coil of the cochlea, where the organ/

organ of Corti is slightly flattened and there is a commencing atrophy of the cells. The terminal fibres from the spiral ganglion show the beginning of degeneration in the spiral lamina. There are fewer cells in the spiral ganglion of the basal coil. The basilar membrane and the spiral ligament are quite normal.

Case 3. J.M., female, aged 61 years.

Functional examination.- Rinne's test positive: bone conduction shortened: high tones heard faintly with  $d_4$ : low tones heard.

Microscopic findings.- Corti's organ is missing in the basal coil but well preserved in the middle and apical coils. The spiral ganglion and cochlear nerve are not demonstrable in these sections. There is a mass of vascular exudate and fibrin adhering to the under surface of the basal and middle coils.

Case 4. A.C., male, aged 76 years.

Functional examination.- No tuning forks heard: C.64 heard by bone conduction: watch not heard by air or bone conduction.

Microscopic findings.- Corti's organ appears as a flat mass of cells on the basilar membrane in the basal/



basal and middle coils. The apical coil is normal. The nerve fibres from the ganglion to Corti's organ are atrophied in the basal and middle coils but unchanged in the apical coil. There is atrophy of the cells of the spiral ganglion for the basal and middle coils and traces of early atrophy of the cells in the apical coil. In the internal meatus the trunk of the cochlear nerve shows areas of atrophy.

Case 5. C.W., male. aged 81 years.

Functional examination.- Wax was removed from both ears and the patient found to be very deaf. No tuning forks were heard. A shout was heard close to the auricle on both sides.

Microscopic findings.- In this case the sections reveal an advanced state of atrophy. Corti's organ is atrophied in the apical and basal coils. Reissner's membrane is ruptured in several coils. The ganglion cells show great diminution and atrophy. Only in the middle coil does the structure approach the normal; here Corti's organ is well preserved and the nerve endings in the spiral lamina are normal. The structure of the spiral ligament is atrophied and shows many spaces. In several sections there is a vascular exudate in the scala/

scala vestibuli and scala tympani. In the internal meatus the trunk of the cochlear nerve shows degenerating areas.

Dog 1.

Microscopic findings.- Here we have a picture of increasing atrophy in the cochlea. The most advanced stage is seen in the basal coil, diminishing to a slight degree of atrophy in the apical coil. Corti's organ is completely disintegrated in the basal coil. The ganglion cells in the spiral ganglion are diminished in number in the basal coil, increasing to the normal number near the apex. The terminal fibres between the spiral ganglion and Corti's organ show corresponding atrophy in the spiral lamina.

Dog 2.

Microscopic findings.- In this case I find a complete disintegration in the ductus cochlearis. There is rupture of Reissner's membrane in each coil. Corti's organ is represented as a mass of debris in each coil. Corresponding atrophy of the nerve fibres and the ganglion cells is not so severe but there is a slight diminution of the cells and some atrophy in the nerve fibres. There are also some slight changes in the nerve trunk in the internal acoustic meatus.

In all cases the picture in one ear resembled closely that in the other ear.

Summarising the pathological changes I find.-

1. Corti's Organ: There are stages of atrophy from slightly flattened and degenerating hair cells to the complete absence of Corti's organ.

2. Nerve Fibres: There are changes seen in the number of fibres, from slight to considerable diminution.

3. Ganglion Cells: There are pictures where a few have dropped out in the basal coil to the disappearance of many in this turn with also absence of a few in the middle coil and fewer in the apical coil,

4. Nerve Stem: There are signs of commencing degeneration here but the changes are slighter than those more peripherally.

5. Spiral Ligament: This is normal everywhere except in Case 4, where it shows areas of degeneration.



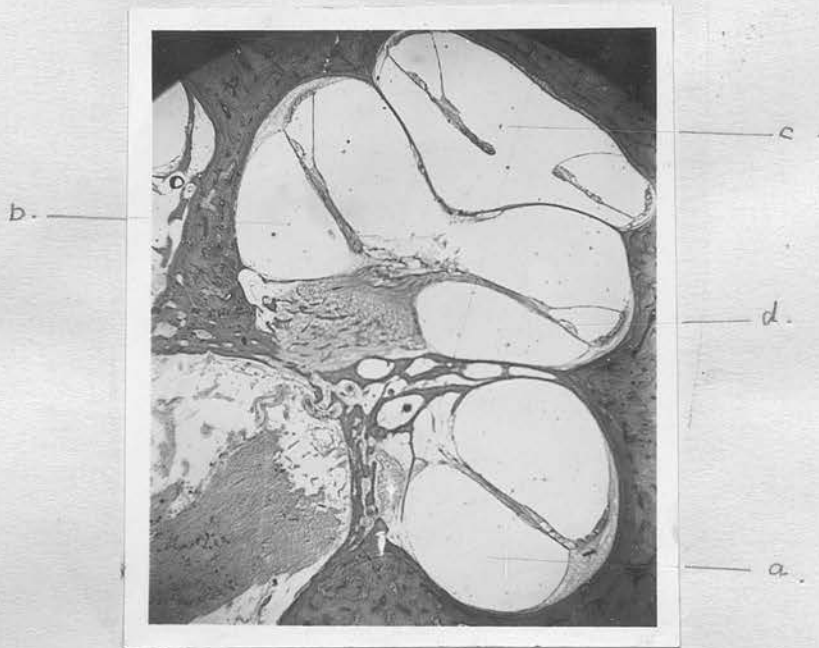
Fig. 1.

Fig. 1. Vertical section through the cochlea of a normal hearing man.

Fig. 2.

Fig. 2. Vertical section through the cochlea of a normal hearing dog.

a. Basal coil of cochlea: b. middle coil of cochlea:  
 c. apical coil of cochlea: d. Corti's organ:  
 e. helicotrema.

Fig 3



Fig. 3. Vertical section through the basal and middle coils of Case 1.

a. Atrophied Corti's organ:    b. Normal Corti's organ.

Fig 4



Fig. 4. Vertical section through the cochlea of Case 1.

a. Degenerating cells in the spiral ganglion of the basal coil:    b. Normal cells in the spiral ganglion of the middle coil:    c. atrophying nerve fibres in the spiral lamina of the basal coil:    d. Normal fibres in the middle coil.

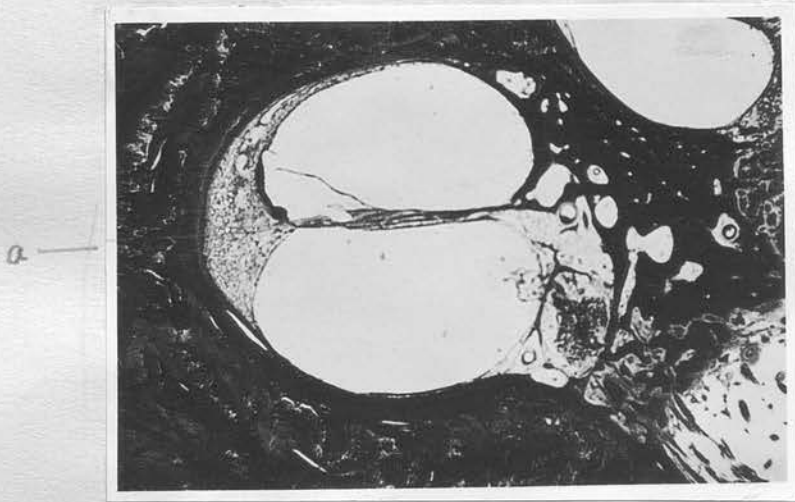
Fig. 5

Fig. 5. a. Degenerating Corti's organ in the basal coil in Case 2.

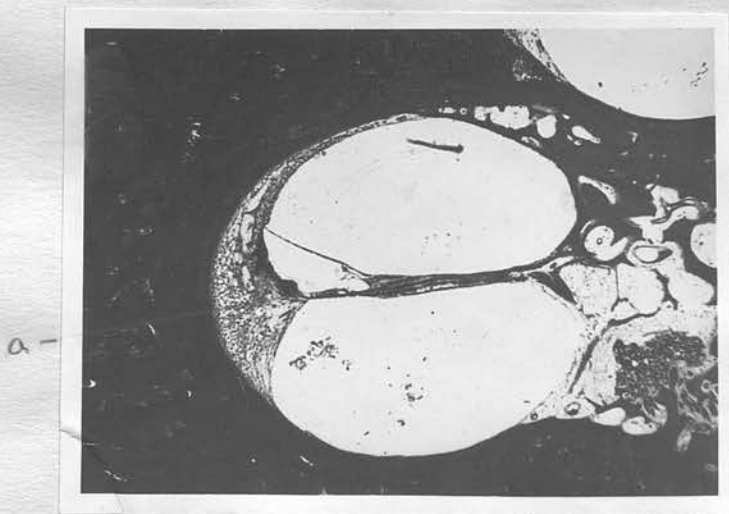
Fig. 6

Fig. 6. a. Degenerating Corti's organ in the basal coil from Case 3.



Fig 7.

a.



*Cut at  
wrong  
angle or  
in wrong plane*

Fig. 7. a. Degenerating cells in the spiral ganglion from Case 5.

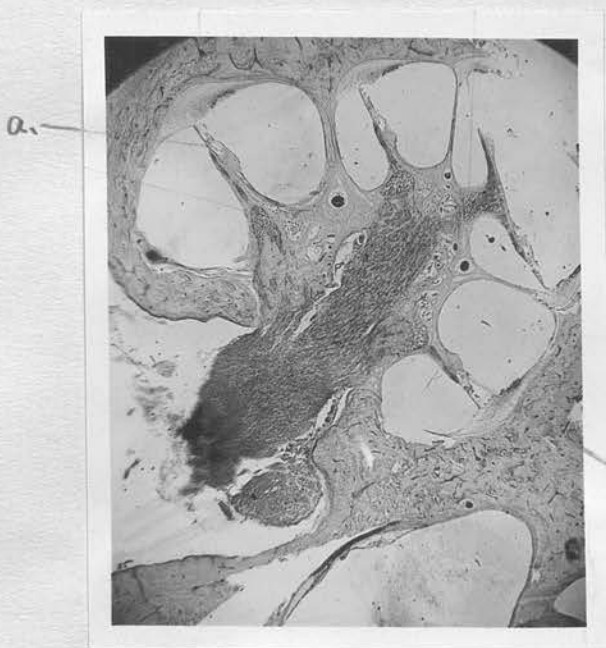
Fig. 8.

a.



*ditto*

Fig. 8. a. Section through cochlea of normal hearing dog showing ganglion cells in the spiral ganglion.

Fig. 9.

no clinical  
history of  
these dogs.  
were they old?  
did they ever hear?  
dogs - especially  
white dogs - do  
suffer from  
congenital  
deafness.

Fig. 9. Vertical section through cochlea of Dog 1:  
left ear.  
a. Atrophied Corti's organ: b. reduced  
numbers of ganglion cells.

Fig. 10.

Fig. 10. Vertical section through cochlea of Dog:  
right ear.  
a. Atrophied Corti's organ in basal and  
middle coils: b. reduction of ganglion cells:  
c. normal number of ganglion cells in apical  
coil.

Literature.

Now follows a short summary of some of the investigations of a few well known otologists.

Sporleder records some observations on the temporal bones of five deaf persons, functionally tested during life. He could not discover, with certainty, pathological changes in the labyrinth or in the auditory nerve. He thought that perhaps the cause lies more in the central part of the cochlear nerve.

Others - Habermann, Mannasse and Brühl - believe arteriosclerosis, atheroma or marasmus to be the factors acting on the cochlear nerve and its peripheral terminations, bringing about degenerative changes. Mayer finds changes in the basilar membrane. He classifies the forms of deafness appearing in old age as follows.-

"(1) Real Presbycusis, that is deafness called forth by old age in every one, which I believe I may presume is caused by the rigidity of the basilar membrane.

(2) The deafness called forth by the diseases of old age (atheroma, marasmus) whose anatomical substratum consists of degenerative changes of the labyrinth and of the auditory nerve.

(3) The diseases rising independently of old age/



age. To these belong, above all, the gradually increasing degeneration of the labyrinth and the auditory nerve from different causes (progressive labyrinthine deafness); also the localised disease of the labyrinth capsule, which indeed is frequently combined with atrophy of the capsule."

Alexander, in a patient aged 66, summed up his findings into what he believes to be typical grades of atrophy as seen in the cochlea.-

(1) Slight changes in Corti's organ, with circumscribed atrophy of the hair cells alone.

(2) Defect of the sensory cells, and increase of the supporting cells at the expense of the sensory cells,

(3) Complete atrophy.

Alexander thinks that the degeneration commences primarily in the cochlea and the atrophy goes hand in hand in Corti's organ, the terminal fibres and the ganglion cells declining centrally along the nerve trunk.

With regard to the cause of this atrophy, he thinks that arteriosclerosis is the factor and believes in Politzer's findings, confirming atrophy of the cochlear nerve in consequence of narrowing of the basilar artery and disease in the regional blood vessels.

Mannasse/

Mannasse summarises his results of anatomical investigation of chronic progressive deafness in thirty-one inner ears.-

(1) In the Cochlear Duct:

- (a) Atrophy of Corti's organ (including the tectorial membrane) up to complete aplasia.
- (b) Widening or narrowing of the duct by bulging or collapse of Reissner's membrane.
- (c) Dropsical degeneration of the spiral ligament.
- (d) New growth of connective tissue at the inferior end of the stria vascularis.
- (e) New growth of bone in the perilymphatic spaces.
- (f) Large accumulation of pigment almost invariably in the modiolus and rest of the cochlea.

(2) In the Spiral Ganglion: Diminution and shrinkage of the ganglion cells and, in their stead, empty spaces or new growth of connective tissue in the Rosenthal canal.

(3) In the small fine nerve canals: Atrophy of the nerve fibres and new growth of connective tissue within the bony canals.

(4) In the Acoustic Nerve: Atrophy and chronic neuritis with new growth of connective tissue in the nerve.

Mannasse believes that the changes come first in the acoustic nerve and those in Corti's organ last.

He finds the picture in the membranous labyrinth to be similar to that in congenital deafness and in acquired progressive nerve deafness, and asks - "Can the cause not be the same?" Three points upon which he lays stress are.-

(1) The difference lies in the nerve trunk: it is unaffected in congenital deafness and largely so in acquired deafness.

(2) In congenital deafness there are special findings in the labyrinth, namely, anomalies in the capsule or changes in the central nervous system which are lacking in acquired deafness. In both there is the same affection of the membranous labyrinth.

(3) Chronic progressive deafness must be looked upon as an acquired deafness.

Brühl investigated four cases of aged patients suffering from arteriosclerosis. He calls it "A simple, degenerative process in the parenchyma of the nerve", the cochlear nerve having more unfavourable conditions for nutrition than the vestibular root. He described also the case of a man who was a hammersmith, i.e., a noisy occupation. In this case there were demonstrable processes of degeneration in the nerves, spiral ganglion and Corti's organ in the first coil of the cochlea had disappeared altogether. The reason for this, according to Brühl, was probably/



probably to be sought in the patient's noisy work. The considerable lowering of the upper tone limit with the peripheral disease in the initial part of the cochlea would also speak for the perception of the highest tones in this place, therefore for the justice of the Helmholtz hypothesis.

Habermann examined the inner ears in the case of five patients of advanced age who had, some time previously, suffered from middle-ear suppuration. Because there were signs of sympathetic inflammation of the inner ear in only three out of the eight ears, he said it was no proof that atrophy of the nerves of the cochlea had to do with this as a direct result of the inflammation. He believes it to be rather an atrophy of disuse, that this is an indirect result. The intensity of the high tones being less than low tones, they are not able to overcome the obstruction caused by closing of the round window by connective tissue following the suppuration, so that part of the cochlea for high tones, the basal part, atrophies because the stimuli are impeded. He then examined the nerve fibres in the spiral lamina of 46 cases of all ages, suffering from various diseases unconnected with the ear. He found the bundles of nerve fibres biggest in infants and young people. In nine cases over sixty he found atrophy of the nerve fibres at the base/

base of the cochlea and sometimes up to the middle coil. He does not believe, from his cases, that senile marasmus alone caused the atrophy but he must admit it for many as he noticed clinically, nerve deafness appeared with the entrance of a high degree of old age.

Josephus, in a paper on Vascular Changes in Chronic Progressive Deafness, says - "Gross muscular changes are described which resemble the vascular changes demonstrated in the pathology of chronic progressive deafness. These, coupled with the response of the auditory apparatus to the vasodilator and the vasoconstrictor drugs, are interpreted as indicating that the essential feature of the pathology of chronic progressive deafness is a disturbance in vascularity associated with changes in the vascular bed of the petrous bone." Josephus attempts to classify chronic progressive deafness on the basis of pathology and to explain its hereditary nature, in many cases on the basis of a congenitally narrow vascular bed in the labyrinth and a non-anastomotic distribution of the labyrinthine artery.

Mayer discovered, in 28 cases over sixty years, changes in the basilar membrane increasing with age. He found a thickening of the membrane above and below and, finally, an ossification. The process was always more pronounced at the base of the cochlea and diminished/

diminished towards the apex. This change he finds accounts for decreasing perception of high tones and increasing inability to hear lower ones as time goes on. Changes in Corti's organ and the nerve fibres, he says, are due to disease of the vessels appearing in old age and causing disturbance of nutrition.

I consider the pathological picture shown by my cases to be similar to that of Alexander's.

Habermann also found the same kind of atrophy, especially in the nerve fibres in the spiral lamina.

Brühl, besides changes in the nerve fibres, found absence of Corti's organ in the first coil of the cochlea. He put it down to atrophy from excessive use while Habermann believes it to be due to atrophy from disuse!

Mannasse also found these changes, but from his findings in the acoustic nerve stem *he believes that* ?  
degeneration begins there before it appears in the cochlea.

I think, from my sections, that it starts in the peripheral organ first, as the atrophy is more intense there and the changes in the nerve stem are very slight.

I did not find the changes in the basilar membrane that Mayer found. Except where the atrophy was so great in the cochlear duct everywhere, with both Reissner's membrane and the basilar membrane ruptured and disintegrated in addition to other changes, the basilar/



basilar membrane was the same as in the normal ear.

Concerning the cause of the changes in Corti's organ, the nerve fibres, the ganglion cells and the nerve stem, most authors - Alexander, Habermann, Brühl and Wittmaack - are of opinion that arterio-sclerosis brings about the changes in the inner ear.

Mayer believes that this is the case for degeneration in the nerve fibres and Corti's organ, but for the basilar membrane he takes that to be a change in every old person, though it will not give a high degree of deafness - that is seen in his groups of old deaf people.

Mannasse would have some common cause for all inner ear deafness, whether congenital or acquired. This could be taken into consideration if we believed it had to do with abnormalities in the blood vessels of the brain in utero and changes in the blood vessels in old age. Politzer confirmed atrophy of the cochlear nerve in consequence of narrowing of the basilar artery and the internal auditory artery.

In the absence of previous middle-ear suppuration or any other cause for chronic progressive deafness in my cases, I think the cause for the findings is based on the alterations in the blood vessels in advancing age, bringing about slow atrophic changes, parallel with changes in the eye, sense of touch, in the hair, skin and generally throughout the body.

In/

In the cochlea the changes begin with Corti's organ and the fibres in the spiral lamina, and progress in the nerve stem as nutrition becomes more and more interrupted centrally.

CONCLUSIONS.Clinical Findings.

1. Loss of high tones is consistently found with the deafness of persons over fifty.
2. Loss of low tones is also found where the deafness is of high degree.
3. There is a shortening of bone conduction in practically all cases.
4. These findings can not be made to fit into a definite age category by decades.

Physiological Finding.

That high tones are heard in the basal part of the cochlea and so on up to the lowest tones at the apex.

Pathological Findings.

There are changes in the inner ear in old age deafness.

1. In Corti's organ.
2. In the terminal fibres in the spiral lamina.
3. In the ganglion cells of the spiral ganglion.
4. In the nerve stem slight changes are found.

These changes begin peripherally.

Etiological/



Etiological Findings.

1. It is arteriosclerosis that is the factor in producing this increasing deafness and the pathological picture in the inner ear.
2. Senile changes have not to do with the age of a person but with the constitution, influenced by heredity and the person's mode of living.

BIBLIOGRAPHY.

- Alexander. "Zur pathologischen Histologie des Ohrlabyrinthes mit besonderer Berücksichtigung des Corti'schen Organes". Archives für Ohrenheilkunde. Bd. 56, S. 1. 1902.
- Baginsky and Munk. Virchow's Archives für pathological Anatomie. 94-65. 1883. Howell's Physiologie, p. 401.
- Brühl. "Beiträge zur pathologischen Anatomie des Gehörorganes". Zeitschrift für Ohrenheilkunde und Krankheit des Luftwege. Bd. 52. S. 232.
- Cuperus. Zit. nach Zwaardemaker. Bd. 1. Denker and Kahler's Handbuch, Bd. 6, p. 706.
- Ebner. Kolliker Handbuch d. Gewebslehre. 6th ed., vol. 3, pt. 2, 1902, p. 958.
- Ewald. Archives für d. Ges. Physiologie. 76. 417. 1899.
- Fraser. Diseases of the Nose, Throat and Ear, edited by Logan Turner, 2nd edition, p. 252.
- Gray. Diseases of the Nose, Throat and Ear, edited by Logan Turner, 2nd edition, p. 252.  
Archives for Otolaryngology. Vol. 9, p. 659.
- Helmholz. Textbook of Physiology. Howell. p. 399.
- Hajke/

- Hajke. Zeitschrift für Hals-<sup>u.</sup> Nasen u. Ohren. 21.  
207. 1928.
- Habermann. "Nervenatrophie in der Schnecke".  
Zeitschrift für Heilkunde. Bd. 12.
- Josephson. "Vascular Changes in Chronic Progressive  
Deafness". Laryngoscope, Jan. 1929,  
Vol. 39, p.40.
- Mannasse. "Über chronische progressive labyrinthäre  
Taubheit." Zeitschrift für Ohrenheilkunde  
und Krankheit des Luftwege. Bd.52, S.1.
- Mayer. "Das anatomische Substrat der Altersschwer-  
hörigkeit." Archives für Ohren- Nasen-  
u. Kehlkopfheilkunde. Bd.195, S.1.
- Politzer. Lehrbuch der Ohrenheilkunde, and Archives  
f. Ohrenheilkunde, Bd.56, p.16.
- Springer and Bergmann's Handbuch der Hals, Nasen,  
Ohrenheilkunde. Denker und Kahler,  
Sechster Band. "Die Altersveränderungen  
am Höhrorgan", p.705.
- Sporleder. "Über funktionelle Pfrüingsresultate und  
Über Sektionsergebnis im höheren Alter".  
Archives für Ohren- Nasen- und Kehlkopf-  
heilkunde. Bd.47. S.234.
- Wrightson and Keith. "An Enquiry into the Anatomical  
Mechanism of the Internal Ear." London,  
1918. Appendix Keith.
- Wittmaack. "Weitere Beiträge zur kenntniss der  
degenerativen Neuritis und Atrophie der  
Hörnerven". Zeitschrift f. Ohrenheilkunde  
u. f. Luftwege. Bd. 53.
- Zwaardemaker. Archives f. Ohren- Nasen- u. Kehlkopf.  
Monatsschrift 47. S. 243.  
"Der Verlust an hohen Tönen mit Zunehmendem  
Alter, im neues gesetz".



Ziffer. "Über die Veränderungen der Gehörorgans im  
vergeschrittenen Alter". Monatsschrift  
Ohrenheilkunde und Laryngo- Rhinol. 1908.