LXIX.

THEORIES CONCERNING PARACUSIS WILLISII.*

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Since 1672, when Dr. Thomas Willis¹ of Oxford and London published his report of the first case of that form of paracusis which bears his name, many theories have been advanced to explain the phenomenon. But from the variety of opinion by the world's great otologists and physicists, it is evident that there is room for at least one more theory.² It stirs one's imagination to read of the seventeenth century highborn lady who was very deaf under ordinary circumstances, but heard quite well during the rolling of drums, so that when she went walking she was always followed by her servant who carried a drum; and when she desired conversation her servant would beat the drum, so that she might hear. I think this is the most picturesque case of paracusis Willisii in literature.

Numerous other instances were later reported of deaf persons who heard better in noisy places, and it became generally recognized that this was an unfavorable symptom. It remained for Politzer³ to show that it was not necessary to have noise to produce improved audition, for, as he found, a toneless, rapidly vibrating instrument brought into contact with the cranial bones will produce the same result. This proved that it was not tones and noises as such, but only as modes of motion, which were causative factors. The symptom as usually described is that the patient hears better riding in vehicles, trains and street cars or in machine shops.

Joseph Toynbee was the first to show that paracusis Willisii belongs to the symptom complex of stapes ankylosis, especially of the otosclerotic type. (Lucae.⁴) In fact, it is one of the most characteristic symptoms of otosclerosis. Körner⁵

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says it is the only phenomenon of this disease that is not cleared up by the anatomic findings.

According to Politzer, Lucae and Roosa, it is never found in cases of nerve deafness unless complicated by trouble in the sound conduction apparatus.⁶ But, on the other hand, it is not constant in all cases of otosclerosis, or even in both ears of the same patient. This reminds us of the fact that the disease process involves various parts of the labyrinthine capsule in different cases, as shown by section. It is unfortunate that so few cases diagnosed as otosclerosis with full examination records ever come to autopsy, and I am not aware that any mention of the peculiarities of paracusis Willisii has been made in the cases published. When Norval Pierce published his masterly paper in 1907 on spongification of the labyrinthine capsule, less than forty cases had been reported in which the diagnosis made during life was confirmed by section.⁷ But, according to our present knowledge of otosclerosis, it is the cases with most marked loss of lower tone limit—i. e., those with most marked stapedial fixation—that show most characteristically this form of paracusis.⁸

It is interesting to review a few of the theories advanced by our modern writers, especially keeping in mind the known pathology. There is so little written directly concerning paracusis Willisii that I cannot refrain from quoting from the address of Charles J. Heath, published in 1911,⁹ though there are few who will agree with his premises or conclusions. He says: "The action of the stapedius muscle is to pull the head of the stirrup bone backward and drive part of the footplate, accompanied by the membrane surrounding and attached to it, against the labyrinthine fluid." . . . "Its effect in increasing the pressure on the fluid in the tiny closed labyrinth is considerable, and scarcely any obstructive tympanic condition (save the rare bony fixation of the stirrup) is capable of completely arresting its action, which is to increase labyrinthine pressure."

It will be noted that this is directly contrary to the accepted teaching that the stapedius muscle tilts the footplate out (thereby antagonizing the tensor tympani) and decreases labyrinthine pressure.

And when Mr. Heath speaks of the deafness accompanying paracusis Willisii as "pocket handkerchief deafness" with "relaxed drum membranes and joints," it is evidently of a clinical

entity which is not the same as we are accustomed to regard as the usual accompanying factors of paracusis. As he gives none of his tuning fork findings, I am unable to say whether or not Bezold's triad is present. It is sufficiently evident, however, that he does not agree with his distinguished predecessor, Joseph Toynbee,¹⁰ who regarded paracusis Willisii as especially characteristic of the later stages of stapes ankylosis.

Phillips¹¹ and Gould¹² say that it is "supposed to result from compression of the labyrinth in the form of otopiesis," "with contraction of the tensor tympani muscle." The question here is what possible effect could contraction of the tensor tympani muscle produce in the presence of an immovable stapes, and why the labyrinth could be compressed?

Dench¹³ says: "Urbantschitsch explains the phenomenon by the action of loud sounds serving to stimulate the receptive centers, after which relatively feeble stimuli may be perceived." But he offers no basis for this theory. And if true, why should not persons with normal hearing hear better in noisy places? Dench further says: "The acuteness of audition upon one side for any given sound will be increased if the organ of the opposite side is at the same time brought under the influence of sound waves of a different character." The error of this proposition is demonstrated daily at the telephone when one naturally stops the open ear with the finger to shut out annoying extraneous sounds.

Politzer says: "Löwenberg and Urbantschitsch attribute this phenomenon to an increased irritability of the acoustic nerve, brought about by general concussion." This I admit to a certain extent, but why, then, are all forms of deafness, and especially the nerve type, not also benefited by noise and concussion, which we know absolutely is not the case?

Politzer¹⁴ himself comes nearer the root of the matter than the others when he says: "The terminations of the acoustic nerve are also thrown out of their equilibrium, which causes them more easily to be excited by the waves of sound." He believes the shaking of the ossicles is also responsible.

Bezold¹⁵ in Holinger's translation says: "It is, therefore, probable that a practically immovable sound conduction apparatus is more sensitive to weaker impulses during the time when a shaking of the whole body keeps it vibrating."

It seems like heresy to question these master minds, but it

must be asked if there is any longer a sound conduction apparatus when its most important link, namely, the fenestrostapedial articulation, has become ossified? The sound conduction apparatus has then ceased to exist as a specialized mechanism.

The hypothesis which I advance, with some temerity, it is true, is built on grounds which have been widely accepted by otologists and physicists. The only added factor which I ask you to accept is that our atmosphere is in constant motion, and few who live in Chicago will deny that.

I quote from Bezold¹⁶ again: "Edward Weber advances the theory that the whole sound conduction apparatus, together with the column of water which runs up the scala vestibuli and back the scala tympani, moves backward and forward as a whole with each sound wave. The very flexible and yielding membrane of the round window acts as a siding." Von Helmholtz found the mathematic proofs for the accuracy and even necessity of this theory. He taught us, furthermore, that the drum membrane, together with the ossicles, forms a wonderful lever apparatus which is able to convert large movements of low power into small movements of high power at the footplate of the stirrup, which is suspended at the ligamentum annulare. (Edelmann, 1: 778.)¹⁷

This whole apparatus, according to Riemann, is so well balanced that the smallest amount of energy is sufficient to overbalance it.

Toynbee¹⁸ says, in speaking of stapes fixation: "The membrane of the fenestra rotunda and the fluid of the labyrinth participate in this fixed condition."

If we accept these theories of Weber, von Helmholtz and Riemann as true, then we must admit that normally the lymph as a whole is in constant to and fro motion. Shambaugh¹⁹ has pointed out the similarity in nerve endings between the vestibular and cochlear apparatus, cupula and otolith in one case corresponding to the tectorial membrane of the hair cells of Corti's organ in the other.

Our present conception of the movement of fluid as a whole in the semicircular canals, in order to make the nerve endings in the ampullæ capable of perceiving motion, must be comparable to the movement as a whole of the fluid in the spiral canals of the cochlea, in order to make the nerve endings there capable of perceiving sounds.

But the fluid of the vestibular portion is influenced only by motion or position of the head, while the fluid in the cochlear canals is constantly being moved by the footplate of the stapes.

It is the low pitched sounds which have the greatest amplitude of vibration. Even vibrations in the surrounding air too slow to be perceived by the human ear as sound may still have their influence in moving a normal stapedial footplate, and so keeping the cochlear lymph in constant motion.

Bezold,²⁰ by manometric measurements, found "that the membrane of the fenestra rotunda was capable of an excursion of more than one-third millimeter, while that around the base of the stapes was only one-sixteenth millimeter. The membrane of the fenestra rotunda is therefore capable of performing excursions five times greater than that of the footplate of the stapes."

This lax membrane, together with the aqueductus vestibuli and cochleæ, acts as a siding or safety valve to prevent sudden compression of the labyrinth from injuring the delicate terminations of the auditory nerve.

In otosclerosis producing ankylosis of the stapes the low-pitched sounds are lost, and, therefore, the greater the degree of ankylosis, the less movement of the fluid in the cochlea, as was pointed out first by Toynbee.

But, as we know, sounds and soundless vibrations are practically ever present, so the normal relation of the tectorial membrane to the hair cells of the organ of Corti²¹ must be in a moving fluid, and these to and fro movements must keep these nerve terminations in a certain state of tension. When mass motion ceases through stapedial ankylosis, this relation is no longer normal, and the auditory nerve becomes less capable of responding to sounds in proportion to the loss of motion of the endo- and perilymph.

In the normal ear the function of the membrane of the fenestra rotunda is a passive rôle; it is only when the normal agent for producing motion in the cochlear fluids (i. e., the stapes) become ankylosed that the membrane of the round window assumes a more active rôle. But it requires a great stimulus to do this, vibrations of wide amplitude and great power, as the rolling of drums, the whirr of heavy machinery, the roar of a railroad train, or the rumble of a wagon, which cause such a commotion that the relatively lax membrane of

the round window, and with it the cochlear lymph, move back and forth in something like their normal motion. At the same time the hair cells attached to the tectorial membrane, stimulated by the ebbing and flowing lymph stream, resume their function as sound receivers, and so continue as long as the artificial mass motion continues. This is the paracusis which Willis described, and which has remained unexplained for two and one-half centuries, waiting for Shambaugh's theory of tone perception to unlock the door.

A necessary corollary of my hypothesis is that in cases of bony stapes ankylosis without paracusis Willisii, the process of spongification must have involved the recess of the fenestra rotunda, thereby preventing all mass motion of the cochlear lymph, and these cases would probably show a greater loss of upper tone limit.

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