Don't Shout! - I Can Hear!

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A natural reaction in speaking to a deaf person is to raise the intensity of the voice. For many deafened persons these louder voice signals become intelligible because now they are capable of activating the excitatory mechanisms of the ear. There are some deafened persons, however, such as the aged, in whom intelligibility often is not improved by shouting. If one shouts at them they are apt to say, “Don’t shout, I can hear.” In fact, many aged people tell us that they can understand better if we do not speak so loudly. Another disturbing phenomenon is that grandpa occasionally can understand what is said behind his back so that the family suspicions are aroused to the point of accusing him of hearing only that which he desires to hear.

Eventually the family decides grandpa needs a hearing aid. The dealer in hearing aids is quite aware of the problems involved in fitting the aged person and, after a few tests, may even advise against its purchase. Nevertheless, grandpa’s family, interested in his welfare, will see to it that he goes home with a hearing aid. Eventually, after several weeks trial, he gives up and puts it in the common burial ground for hearing aids, the bureau drawer. About this time grandpa is accused of being senile and uncooperative. He didn’t refuse glasses when he needed them nor a crutch after he broke his hip. Why, then, won’t he wear his hearing aid?

Old Age: Let us examine the clinical and pathological findings in the deafness of aging for an explanation of grandpa’s uncooperative attitude. We find that there are two types of presbycusis. The first begins about the third decade of life, and is characterized by a progressive limitation of hearing for high frequencies. This high frequency hearing loss progresses so slowly over the years that usually it is imperceptible to the patient. The upper frequency limit for ordinary conversational speech is about 5000 cps and usually it is late in life before the process has involved these higher speech frequencies.

In a series of 72 cats, on whom my associates and I have performed hearing tests by the conditioned response method, we have found 5 animals with high tone deafness. A study of these animal ears and the ears of humans with high tone deafness revealed the pathological changes to consist of degeneration of the membranous labyrinth, beginning in the basal turn of the cochlea and proceeding toward the apex. This type of pathological change I have termed the “epithelial atrophy” type of presbycusis. It appears to be the otological manifestation of an aging process which affects all tissues, a close corollary existing in the skin where the changes are so characteristic that they are used as a yardstick to measure the age of the individual.

The second type of presbycusis I have termed “neural atrophy” because it is due to a decrease in population of neurons of the auditory pathways and is characterized by a discrimination loss out of proportion to the threshold loss for pure tones.

Ordinarily its onset is in older age and is superimposed upon the epithelial atrophy already described. The pathological features were characteristic in the ear of a 70 year old man on whom we acquired a hearing test immediately prior to death. The ear had the typical epithelial atrophic change involving the basal 3.3 mm. of the cochlea.

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but in addition there was a 30% to 50% loss of spiral ganglion cells extending from the 3.3 mm. to the 14.2 mm. region without accompanying changes in the organ of Corti or other structures of the membranous labyrinth. This loss of spiral ganglion cells appears to be related to a diffuse degenerative change involving the central nervous system. For example, it is well established that the aging brain suffers from a loss of nerve cells. The process seems to develop independently of cerebral arteriosclerosis but may co-exist with varying degrees of it. The clinical manifestation of loss of cortical neurons is senile dementia characterized by a gradual diminution of physical and mental capacity, an exaggeration of prior personality traits, increasing intellectual failure, impairment of memory, errors of judgement and insomnia. The cell outlines of the cortical neurons become shrunken and irregular, the cytoplasm becomes hyperchromatic, the Nissl material decreases, the nuclei become basophilic, and later there is glial proliferation and enuraphagia. A decrease in neuron population of the auditory pathways would be expected to have a negative effect on the amount of information which can be transmitted by these pathways.

In recent years ideas on communication and concepts of information transmission have been expressed in precise mathematical terms. Although few of us are interested or capable of understanding the mathematics involved in communication theory we readily appreciate its importance for speech intelligibility. The ear analyzes speech by recoding the sound signals into nerve impulses which travel up the auditory nerve. We know that the fundamental coding method is very simple in that a nerve impulse is either present or absent. This principle of “all or none” is basic to neurophysiology. Another limitation is that a single nerve fibre will carry a maximum of only about 800 to 1000 impulses per second. We may enumerate the variables in a neural pattern as follows: 1) frequency of impulses in a single fibre (below 1000 cps), 2) time relations between impulses in various fibres, 3) selectivity as to which fibres are being activated, and 4) the number of fibres activated. In spite of the “all or none” law the auditory pathways can carry all the information required for intelligibility of the spoken voice. This is possible because of the tremendous number of channels in this communication system, for we know there are about 35,000 fibres (or channels) in the human auditory nerve. In the pathological ear with a loss of nerve fibres a limitation is imposed on the coding mechanism and the amount of information which can be transmitted from the ear to the brain is less than normal.

Nerve Tumor: If a disproportionately severe discrimination loss is characteristic of neural lesions then it should also exist in patients with tumors involving the auditory nerve. Indeed, such is the case, for I have observed two patients with surgically and histologically proven acoustic neurinoma who manifested this same peculiar ability to hear but inability to understand in the involved ear. Mrs. W. had normal pure tone thresholds for speech frequencies and a speech discrimination score of 16%; whereas, Mrs. C. had a pure tone average in the speech range of 40 db and a discrimination score of zero. Of course, not all patients with acoustic neurinoma have these characteristic auditory findings for many are totally deaf in the involved ear at the time of the first examination. Then there are also those in whom the auditory findings are suggestive of combined neural and cochlear degeneration. In these cases the discrimination scores are somewhat better comparatively and there is partial loudness recruitment. The most likely explanation for the cochlear damage is that the tumor has compromised the circulation by choking the internal auditory artery.
Experimental Nerve Section: Thus far we have not been able to devise a method for determining an animal's ability to discriminate complicated auditory signals. We do have accurate methods, however, for determining pure tone thresholds for animals, so an experiment was designed whereby we could test the concept that the nerve fibre population can be reduced without creating a pure tone threshold loss. Accordingly partial section of the cochlear nerve was performed on twelve cats on which hearing tests were performed and a correlative study made of threshold losses and nerve fibre population. The procedure for each animal was as follows: The function of one ear was destroyed by a sterile surgical procedure. The animal was then trained to respond to pure tone auditory stimuli by moving forward in a rotating cage to avoid shock. Audiograms were made, using this conditioned response as an indicator of hearing. After base line audiograms had been obtained for each animal a surgical procedure was performed to section partially the cochlear nerve. By an approach through the posterior cranial fossa a portion of the cerebellar hemisphere was removed so as to expose the superior surface of the petrous portion of the temporal bone. With small dental burs the bone was removed in an oval area antero-medial to the arcuate eminence and immediately superior to the internal auditory canal. The facial nerve and the superior division of the vestibular nerve were exposed to view. By retracting the facial nerve anteriorly and the superior division of the vestibular nerve posteriorly, it was possible to expose the cochlear nerve as it entered the modiolus. The cochlear nerve was partially sectioned by gently moving a needle through a selected portion of the nerve trunk. Several weeks later audiograms were made, following which the animals were sacrificed by arterial perfusion with Heidenhain-Susa solution. The cochleae were graphically reconstructed from serial sections, and counts were made of the hair cell and spiral ganglion cell populations. The cell counts of several cochleae revealed that up to 75% of nerve fibres had been destroyed without creating threshold elevations for frequencies having their fields of excitation in those regions. Loss of more than 75% of the nerve fibres did create threshold elevations; or, in other words, the findings of this experiment means that for pure tone stimuli an impulse of threshold magnitude can be conducted when only 25% of the nerve fibres remain.

Conclusions: We may consider the spiral ganglion and its nerve fibres to be a part of a complicated communication system consisting in the human of about 35,000 channels. Among the variables which determine the amount of information which can be coded and transmitted over this system is the number of functioning channels or, in this case, nerve fibres. A deficiency in the number of auditory nerve fibres results in a system which can transmit simple signals of threshold magnitude, but cannot handle the complex signals of speech.

In grandpa's ear the neural patterns for speech are markedly distorted and sound garbled to him. To amplify the speech signal reaching his ear creates only a louder garbled signal and does not clarify it. He, therefore, may find a hearing aid to be of no use to him. A distorted speech pattern sometimes is more intelligible at moderate intensities than at high intensities and, therefore, grandpa may hear what he is not supposed to hear.

Putting it briefly and to the point, grandpa's ear is working fairly well, but his communication system is defective. Therefore, he may reprimand you in defiant tones "Don't shout! I can hear!"