SUMMARY DISCUSSION OF SOME THEORIES OF PITCH DISCRIMINATION

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In the light of my clinical experience with pathological ears, it has fallen to my lot to discuss the highly technical and provocative papers to be given today.

If we were to consider all the research these panel members have reported on today's subject ("Frequency Analysis of the Normal and Pathological Ear") the conference would be fascinating but rather lengthy to say the least.

Let us consider what I feel are the important points of agreement and disagreement among the four speakers.

Professor Schouten has raised a very interesting point in what he calls the "residue phenomenon". He defines this as "the perception of a number of neighboring Fourier-components of a sound as a single percept-when the Fourier-components are too narrowly spaced for them to be resolved and perceived separately".

In general if three tones separated by 100 or 200 cps are presented simultaneously the listener will hear a sound he defines as a 100 or 200 cps tone.

Professor Schouten has studied this matter carefully and shown that the phenomenon is not due to linear distortion as previously suggested since it occurs at low loudness levels.

Briefly it appears that Professor Schouten has provided very good evidence that pitch sensation is not exclusively related to geographic location of frequency in the end organ but is time locked to events which occur in the end organ and integrated in higher centers to produce learned response.

Our next speaker is well known to most of us because of his very agile ability to take widely separated pieces of the auditory jig saw puzzle and develop plausible but highly complex theories.

Dr. Licklider's work in audition has been primarily concerned with developing theories that will explain how acoustical information as coded by the cochlea; which has been admirably shown by Dr. Bekesy to be essentially a mechanical analyzer, is handled by the neural pathways.

Today he will present what he calls a "triplex theory" based on the limited information available. If one spends time enough studying Dr. Licklider's complex theory it becomes evident, that although much more research is necessary, he has made admirable use of the known basic correlates of auditory physiology.

It would be presumptious of me, a mere otologist, to argue for or against this theory. Furthermore, I am quite biased in his favor since I believe that one statement Dr. Licklider makes is the crucial point in discussing any auditory theory. I quote — with some paraphrasing — "Order and sequence are present at the outset — thanks to cochlear analysis. It seems likely, however, that nature probably leaves subsequent pathways somewhat in disorder. Nurture, then, may shape up whatever arrangements and organizations are required to meet the demands of individual experience."

Long term studies of children with defective cochleas illustrate that information reaching the higher auditory centres, however distorted, can be ordered into meaningful symbols useful for auditory communication, provided that these higher centers are stimulated early enough in infancy. It has been my contention for years that if the brain is given a chance to order cochlear signals early in infancy, it makes very little difference what the signal configuration is, learning will produce useful auditory communication. In view of this partially substantiated opinion my interest in Dr. Licklider's theories should be obvious. His theories allow for individual differences as well as marked deficiencies in the coding unit. If as he suggests and as Bekesy, Keidel, Kiang and others have definitely shown, specific anatomical order ends at the cochlear nuclei, the proximal pathways will in time meaningfully utilize whatever signal order is presented to them. As Dr. Licklider's theory suggests, unordered auditory neural networks probably use the temporal patterns of neural pulses to learn to discrminate among complex signals. Repeated cochlear signals modified by related associations from other senses, eventually take on fixed meanings.

Dr. Keidel, our third speaker, is very well known for his contributions on the mechanical properties of the cochlea and their relation to frequency discrimination. His work, combined with that of Ranke (his former chief) lends support to the idea that the cochlea serves as a specific physical analyzer with limitations related to frequency, damping, wave form envelope and a complex inhibitory system. Dr. Keidel sums up some of his feelings on this matter by posing a whole set of problems in a single sentence. These problems are related to the physical properties of the perilymph-endolymph system, the differential functions of the hair cells with respect to intensity and frequency analysis, and the extent of influence of the inhibitory networks as regards the spatial and temporal patterns of the basilar membrane and organ of Corti.

Our last speaker, Dr. Katsuki, believes that pitch discrimination is accomplished partly in the cochlea but that a sharpening process occurs higher up, probably in stages up to the level of the geniculate body. He, too, believes that inhibition plays an important role in pitch discrimination but that this role is not necessarily confined to the efferent fibers of Rasmussen.

When the opinions and experimental conclusions of these men are considered broadly it appears that there are no real disagreements in their concepts. At first reading there appears to be divergent opinions but closer examination reveals that the points of divergence are not so much differences in opinion as differences in interpretation of insufficient experimental data. One thing is glaringly evident throughout most of the reported basic research on the auditory function — the absence of data obtained from pathological ears.

It would be interesting to know what becomes of the "residue phenomenon" when the subject has a known high threshold in the frequencies above 1000 cps. If this phenomenon is strictly related to periodicity and the wave forms on the basilar membrane the absence of response to high frequencies should show no difference. I wonder if Professor Schouten has any such data.

It would be of interest to study human responses in ears whose efferent bundles had been cut. Recent surgical advances have made it possible to section the vestibular portion of the 8th nerve for treatment of intractable vertigo. Since Rasmussen's fibers are carried in the vestibular portion of the auditory nerve it will be possible to conduct experiments which may be related to inhibition on these patients. We are presently studying just such a case. Recent work by Spondlin reported at the 1962 meeting of the American Otological Society fairly conclusively shows that the efferent Rasmussen fibers are distributed to every hair cell. From this it would appear that the inhibitory network is very complete.

Recent work by Kiang has also demonstrated that frequency analysis is done in the cochlea and coded into pulses which are time locked. Electrodes placed in the 8th nerve trunk show quite accurate sharpening of frequency at threshold but rather broad areas increasing with intensity. As intensity is increased above threshold pitch discrimination in the cochlea becomes less accurate and the small DL for pitch at above threshold levels is probably related to neural sharpening in the higher order neurons as suggested by Dr. Katsuki.

In Kiang's study spontaneous neural activity could be grouped into three classes: high, moderate and low activity. It is reasonable to assume that these data indicate respective differences in sensitivity and probably mean that not only is hair-cell function divided into outer and inner rows, but that the outer rows are at least differentially sensitive to intensity.

It is also quite apparent when one tries to compensate for hearing loss as a function of frequency that loss of sensitivity for intensity is not the only factor involved. It has been shown that correcting for such losses by increasing amplification selectively with respect to frequency does nothing to improve discrimination. In fact under these conditions discrimination is usually worse which lead to the belief that the cochlea plays a very important part in discrimination, related to spatial arrangement and temporal pattern and that both are modified by a highly organized and selective inhibitory system which modulates both frequency and intensity.

Let us look at other types of pathology. In the presence of an auditory tumor or labyrinthine hydrops pure tones are heard without noticeable change in pitch but in both cases discrimination for speech is seriously effected.

Both diseases involve lower neurons. On the other hand in many cases of advanced presbycusis pure tones reflect very little change but again discrimination for speech is frequently poor. In this case the lesion is in the higher order neurons probably including even cortical cell changes. (In these cases it is quite obvious that higher order sharpening processes are no longer as effective.) These individuals seem to have less difficulty with discrimination if speech is presented slowly. It appears that the mechanical analyzer is responding well but that the mechanisms involved in differentiating the time patterns is unable to operate as rapidly as formerly. The effect of physiological aging on the auditory system deserves much study. Very little has been done. Research on cases of this kind should provide valuable data. In addition, intensive studies of recruitment, diplacusis and tinnitus should be done with a view to testing some of the various hypotheses that have been proposed.

In closing I would like to thank Dr. Bekesy for inviting me to be a member of so distinguished a panel. I hope I have contributed something worthwhile.

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Reference:

1. Reported at the 1962 meeting of the American Otological Society.