SOME PHYSIOLOGICAL INTERPRETATIONS OF AUDITORY FUNCTION

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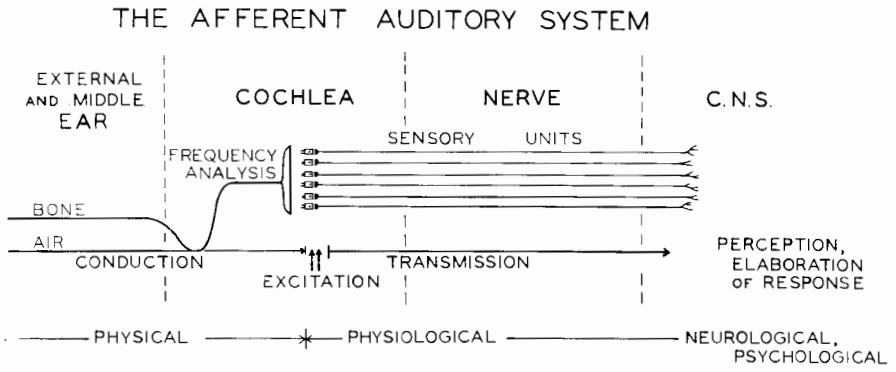
A simple physiological "model" of the peripheral auditory system may give some useful insights into abnormal auditory function. In our model acoustic conduction occurs, as a mechanical process, across the middle ear and on into the inner ear including the bending of the hairs of the hair cells. Conductvie impairment acts simply as a passive acoustic filter that attenuates sound more or less according to its frequency. Frequency analysis is part of the inner ear conductive process. It distributes acoustic energy along the cochlear partition so that there is selective stimulation of different sets of sensory units according to frequency. This "place principle" of frequency analysis is dominant but, for frequencies below about 1000 cps, the volley principle of neural discharge also carries useful information concerning frequency.



The physiologic auditory pathway consists of many nearly discrete sensory units that act in parallel. Each consists of an afferent neuron and the hair cells that can excite it. In spite of some overlap of innervation of external hair cells the units are functionally independant. There is no positive evidence for peripheral inhibitory interaction among sensory units. Neural transmission in the sensory axons is by all-or-none nerve impulses.

Information as to intensity is carried by (1) increase of frequency of firing in a given sensory unit, over a limited range of both input and output, (2) activation of more units of higher threshold that are tuned to the same

frequency, and (3) addition of units that are most sensitive to neighboring frequencies. The external hair cells apparently have significantly lower thresholds than the internals and there are probably gradations among them. Each sensory units is "tuned" to a particular "best" frequency but can also be excited by neighboring, particularly lower, frequencies. The addition of sensory units increases the loudness but, although it is unsymmetrical, it does not alter the perceived pitch.

No peripheral mechanism explains either the very smooth increase of loudness, as a power function of intensity, over the wide dynamic range of hearing, or the various phenomena of masking. These, together with the sharpening of pitch perception by inhibitory interactions that enhance contrast, must be determined centrally.

The excitation of sensory units is a physiological process. We attribute to it the psychoacoustic phenomena of fast adaptation and recovery. These are physiological adjustments to a steady state of continued moderate activity. They probably occur at the synapses between hair cells and nerves. Abnormal fast fatigue, however, probably represents a reversible block to transmission that develops in the axons of sensory units that are subjected to mechanical pressure. This block is comparable to Wedensky inhibition.

SUMMARY OF AUDITORY DEFECTS

PERIPHERAL	
MECHANICAL	ATTENUATION OF SOUND
ANATOMICAL	PERMANENT LOSS OF SENSORY UNITS
PHYSIOLOGICAL	ELEVATION OF THRESHOLDS
	BLOCK OF TRANSMISSION
	HYPEREXCITABILITY
CENTRAL	FAILURE OF PERCEPTION
	INTEGRATION
	UNDERSTANDING

One impairment of the excitatory process is the temporary elevation of threshold produced by strong, but not too severe, noise exposure. The shift is slowly cumulative and recovery is also slow. It is greater for frequencies a little above the frequency of the exposure tone. The rise in threshold is almost certainly related to mildly injurious mechanical effects on hair cells and nerve endings. In our model the effect is an elevation of the thresholds

of sensory units which is greater, in a given frequency band, for the units of lower threshold. The result is an **equalizing of thresholds.** It is quite possible that permanent noise-induced shift is based on just such an equalization of thresholds and does not necessarily involve, until the injury becomes very severe, complete anatomic loss of sensory units.

We distinguish here between two kinds of impairment of sensory units. One is a simple elevation of threshold (physiologic); the other is total "subtractive" loss (anatomic) or reversible block (physiologic). The effects on hearing will depend on the distribution of subtractive loss or threshold shift among the sensory units, whether primarily along the organ of Corti (related to frequency), or primarily across the organ of Corti (related to sensitivity), or perhaps quite at random. In pure form the effects are predictable. For example:

1) A complete loss of sensory epithelium in the basal turn gives abrupt high-tone hearing loss.
2) Equalization of thresholds, as in temporary noise-induced threshold shift, should produce recruitment of loudness but little if any other disturbance of hearing. A systematic subtraction of the most sensitive units might give a rather similar result, although in this case we expect partial rather than complete recruitment.
3) Random loss (or block) of units, such as very probably occurs in old age and sometimes when the auditory nerve is slowly compressed by a tumor, need not cause any elevation of threshold. The effect may be simply a restriction on the amount of information that can be transmitted. This should appear in tests of discrimination.

The third major disorder of sensory units, in our model, is hyper-excitability. Strong spontaneous firing of sensory units must certainly cause tinnitus. A too rapid increase to maximum in the rate of discharge of units should cause loudness recruitment. Such over-octivity should also cause more or less disorganization of the patterns of activity among the sensory units.

Some impairments of the inner ear may be quite complex. For example, Menière's syndrome, (vertigo, tinnitus, low tolerance threshold, hearing loss, poor discrimination and pitch dysacusis) suggests a combination of hyper-excitability of nerve endings, impaired response of hair cells, inefficient conduction in the inner ear and perhaps faulty frequency analysis.

QUELQUES INTERPRÉTATIONS PHYSIOLOGIQUES DES FONCTIONS AUDITIVES

Les signaux auditifs pénètrent dans l'oreille par deux voies parallèles, osseuse et aérienne. Ils passent ensuite par un canal commun constitué par l'oreille interne où s'effectue l'analyse des fréquences. Les cellules ciliées et les fibres nerveuses qui les entourent fonctonnent comme un ensemble d'unités sensorielles en parallèle. L'excitation et la transmission des influx nerveux sont des processus non-linéaires.

Les surdités de transmission provoquent une simple atténuation de l'in-

tensité sonore variable avec la fréquence comme le ferait un filtre acoustique passif. Elles ne peuvent produire de recrutement.

L'adaptation rapide et la récupération sont dues, selon nous, à des ajustements à un niveau d'activité modéré. La fatigue auditive anormalement rapide résulte vraisemblablement d'un bloc reversible le long des fibres nerveuses.

Les déficits auditifs provoqués par une exposition au bruit comportent comme aspect essentiel l'élévation temporaire ou permanente du seuil des unités sensorielles les plus sensibles. L'égalisation des seuils qui en résulte rend le phénomène de recrutement inévitable.

Lorsqu'il existe une lésion anatomique ou un blocage physiologique de certaines unités sensorielles, par exemple dans la sénescence ou dans le cas de tumeurs, l'élévation des seuils n'est pas obligatoire, si l'on considère que les fibres touchées sont distribuées au hasard. Seule la quantité d'information transmise peut être diminuée.

Dans le syndrome de Menière il existe une hyperexcitabilité et une hyper- des terminaisons nerveuses, et probablement aussi un trouble de la conduction à l'intérieur de l'oreille interne aboutissant à pertubation de l'analyse des fréquences.

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DISCUSSION:

Langenbeck:

Der Vortrag enthüllt den ganzen Kummer, den wir Kliniker heute noch mit den Rekruitment-Methoden haben. Wir haben auf der einen Seite die endocraniellen, raumfordernden Prozesse. Das Rekruitment kann positiv oder negativ sein. Noch wechselnder sind die Bilder mit Hörstörungen nach Schädeltraumen. Da sind anderseits die Menière-artigen Krankheitsbilder mit plötzlichen Hörstürzen (sudden deafness). Es verbergen sich bestimmt die verschiedenartigsten Prozesse peripherer und centraler Durchblutungsstörungen unter diesen Krankheitsbildern. Das Rekruitment ist häufig positiv, auch wenn die Vestibularis-Untersuchung Bilder centralen Ursprungs ergibt. Das Bild verwirrt sich vollends, wenn wie neuerdings pathologische Reizzustände im Olivocochlearen Bündel, oder seinen Ausgangscentren, als Ursache cochlearer Unterempfindlichkeiten diskutiert werden. Was sollen wir als Ärzte machen? Wir müssen unseren Patienten helfen und vorher Diagnosen stellen, bei denen wir die Rekruitment-Teste nur mit äusserster Vorsicht bewerten. Natürlich sammeln wir Material. Aber der Wert von Statistiken ist sehr fraglich bei so heterogenem Material bei dem autoptische Befunde äusserst selten sind. Es wird noch Jahre dauern bis wir festen Boden unter den Füßen haben.

Davis:

I wish to thank Dr Langenbeck for his remarks which amplify certain points which Dr Hirsh and I tried to make in our brief discussion. Some of the points that he mentioned illustrate the difficulties encountered in applying in practice the simple concepts which we have set forth. His discussion further reminds me of one point that I had intended to make with emphasis. It is that in the auditory system there may be more than one defect at the same time. Defects may be superimposed one on another I know that you are all familiar with this principle, but it is so important that it deserves this special emphasis.

I am quite aware that Dr Hirsh and I do not make direct connections between the type of physiological defect that may be observed and the conditions which may cause such a defect. We may sometimes guess correctly the anatomical location of the defect. It is easier as a rule, I believe, to first infer the physiological defect from our tests. The next step is to the anatomical location and beyond that lies the cause of the condition. In order to plan reasonable treatment it is often essential to understand the cause of the condition as well as its probable location.

This is not always easy. The doctor must make a judgement to the best of his ability on the basis of incomplete evidence. Yes, Dr Langenbeck, there will always be much work for doctors of medicine to do.