Grundlagen und Methoden einer Erneuerung der Systematik der höheren Pflanzen

Von F. BUXBAUM

224 Seiten mit 49 Abbildungen, 7 Schemata und 3 Karten (Springer-Verlag, Wien 1951) (Fr. 27.–)

Der Verfasser – bekanntgeworden durch seine systematischen und entwicklungsgeschichtlichen wichtigen Arbeiten – unternimmt den gelungenen Versuch, die botanische Systematik durch eine Bereicherung der Problemstellungen zu beleben. In der Einleitung werden die Arbeitsgebiete der Phytographie und Systematik umrissen und an den bisherigen Methoden scharfe Kritik geübt. Die folgenden Kapitel zeigen einerseits den erfahrenen Praktiker, der unter anderem im Abschnitt «Methodik» wertvolle Hinweise auf die Vorbereitung und den Arbeitsgang in der Bearbeitung einer systematischen Einheit vermittelt, anderseits den weitsichtigen Theoretiker, der eine Fülle von Teildisziplinen (Progression, Arealkunde, Selektion, Variabilität, Artproblem) heranzieht, um die Idee der «dynamischen» Botanik klarzulegen. Ein reicher Wechsel in den Betrachtungsweisen, dicht gedrängte und anschaulich dargestellte Hinweise auf die Arbeitsmethoden vermitteln ein eindrückliches Bild der Weitschichtigkeit der modernen systematischen Forschungsrichtung.

Klare Schemazeichnungen, Karten und Stammbäume veranschaulichen das Geschriebene. Leider wird ein Stichwort- und ein Literaturverzeichnis vermisst. Der Verfasser erlag ab und zu der Versuchung einer zu sehr ausgeprägten Schwarz-Weiss-Malerei. Seine Angriffigkeit wird die Diskussion beleben. Neben reichlicher Erwähnung eigener Forschungsarbeiten vermissen wir Hinweise auf wichtige Arbeiten junger Systematiker, wie zum Beispiel auf EHRENDORFER, der die Systematik von *Galium* durch Einbeziehung der Zytologie und geographisch-ökologischer Arbeitsweisen vorteilhaft vertieft hat.

P. AELLEN

Allgemeine Taxonomie und Chorologie der Pflanzen

Von WERNER ROTHMALER

204 Seiten, 42 Abbildungen

Band I des Kompendiums der Biologie,

herausgegeben von F. A. SCHILDER und W. ROTHMALER (Wilhelm-Gronau-Verlag, Jena 1950)

Dem Verfasser, der heute in der vordersten Reihe der botanischen Systematiker steht, gelingt es, in knapper, lebendigster Fassung das eigentliche Stoffgebiet der Taxonomie (die alte Systematik und die spezielle Botanik), aber auch ihre Hilfswissenschaften (wie Morphologie, Genetik, Zytogenetik, Phylogenetik, Paläobotanik, Chorologie) und in weiteren Kapiteln die Randgebiete, aus welchen die Taxonomie neue Impulse erfährt, wie zum Beispiel die Probleme der Sippenbildung, Sippenentwicklung, Areal und Umwelt, Areal und Zeit, der geographisch-morphologischen Methode darzustellen. Eingehend sind die Kapitel über die taxonomischen Einheiten, die Nomenklatur, die eigentliche phytographische Technik, die Stammesgeschichte behandelt. – Ein aussergewöhnlich anregendes Werk, das in der Untergliederung des Stoffes, der drucktechnischen Darstellung (Stichworte am Rand), einem Literatur- und Autorennachweis, einem Glossarium und Schlagwortverzeichnis den genialen Ordner und Gestalter verrät!

Die botanische Systematik – das zeigt das Werk ROTHMALERS mit aller Deutlichkeit – löst sich heute aus ihrer Verkrampfung und wird sich in Zukunft als lebendige botanische Teildisziplin an ihrem traditionellen Ehrenplatz behaupten können.

P. AELLEN

Informations - Informationen - Informazioni - Notes

STUDIORUM PROGRESSUS

On the Adaptation, Fatigue and Acoustic Trauma of the Ear

By J. ZWISLOCKI and E. PIRODDA[1], Basle

Under the influence of an acoustic stimulus the functional state of the ear changes, and this modification is first of all characterized by an elevation of the threshold of hearing and by a decrease in the loudness of a given sound stimulus. This phenomenon has been known for a long time; it has, however, aroused increasing interest in the last few years, since recent investigations have shown that it is based on substantially different processes. These processes are, to a certain extent, of importance for the topic differential diagnosis of impairment of hearing. Researches on the adaptation of the ear to sound stimuli (GORAN DE MARÉ[1], LÜSCHER and ZWISLOCKI[2], GARDNER[3], MUNSON and GARDNER[4], as well as HOOD[5]) are the principal ones concerned. The adaptation of the ear presents at least a formal analogy to the light-dark adaptation of the eye (LÜSCHER and ZWISLOCKI[6]); and behaves, according to the nature of the physical stimulus which consists of mechanical vibrations, in a similar way to the mechanico-receptors of the skin and muscles (ADRIAN and ZOTTERMAN[7], MATTHEWS[8] and others). This term has been taken by GORAN DE MARÉ

[1] Acoustic Laboratory of the University Clinic for Ear-, Nose- and Throat Diseases, Basle.

[1] G. DE MARÉ, Acta Oto-Laryng. suppl. *31* (1939).

[2] E. LÜSCHER and J. ZWISLOCKI, Acta Oto-Laryng. *35*, 428 (1947); *37*, 498 (1949); J. Acoust. Soc. Amer. *21*, 135 (1943).

[3] M. B. GARDNER, J. Acoust. Soc. Amer. *19*, 178 (1947).

[4] W. A. MUNSON and M. B. GARDNER, J. Acoust. Soc. Amer. *22*, 177 (1950).

[5] J. D. HOOD, Acta Oto-Laryng., suppl. *92* (1950).

[6] E. LÜSCHER and J. ZWISLOCKI, Acta Oto-Laryng. *35*, 428 (1947).

[7] E. D. ADRIAN and Y. ZOTTERMANN, J. Physiol. *61*, 157 (1926).

[8] B. H. C. MATTHEWS, J. Physiol. *71*, 64 (1931).

from MATTHEWS. VON KRIES[1] has termed this adaptation process, which appears in all the sensory organs, as "Umstimmung", an expression which has been applied by LÜSCHER[2] to the ear in opposition to the conception of fatigue. A similar phenomenon has been observed in the peripheral nerves, where it manifests itself as a temporary decrease of the nervous impulses. GERARD[3], BOYD and GERARD[4] speak in this case of equilibration, an expression which has been adopted for the acoustic nerve by DERBYSHIRE[5], STEVENS and DAVIS[6]. GALAMBOS and DAVIS[7], however, in their paper dealing with the behaviour of single fibres of the acoustic nerve, designate this same effect as adaptation. It is purely a matter of opinion which expression is more appropriate. Each author describes the same phenomenon from a different viewpoint: equilibration refers to the physiological mechanism, the basis of which is an establishment of a state of equilibrium in the peripheral nerve; adaptation, on the other hand, expresses the adaptation process of the nervous end apparatus to the external stimulus. In our opinion both terms are justified.

As it has been emphasized, particulary by BRONX[8], from the point of view of general physiology, adaptation does not coincide with the conception of fatigue in the physiological sense, since the latter is strictly connected with the energy metabolism of the receptor, and depends, for instance, to a great extent upon the demand for oxygen. It is therefore unfortunate that in the literature concerning the ear no clear distinction is made between adaptation and fatigue. This difference, as regards, the organ of hearing, has been pointed out by LÜSCHER[9], GORAN DE MARÉ[10], and more recently by LÜSCHER and ZWISLOCKI[11]. It consists primarily in the fact that adaptation means the attainment of a state of equilibration, while fatigue may increase as long as the stimulation goes on. If the stimulation which leads to fatigue is not interrupted in time, damage to the end organ may occur. Adaptation, apart from fatigue, is frequently identified with the masking effect. LÜSCHER and ZWISLOCKI[12] have shown in this connection that masking causes a greater threshold elevation than adaptation and therefore these phenomena cannot be identical. It is likely that masking is to the greater part due to adaptation, but in our opinion one cannot speak of masking when the threshold elevation is determined after the breaking-off of the stimulating tone.

In conjunction with the physiological phenomena of adaptation and fatigue, there may appear in the ear a third phenomenon, the acoustic trauma, as a modification in the functional state of the ear following acoustic stimulation. It appears when the ear is overloaded and is characterized by a shifting of the maximal hearing loss in relation to the frequency of the stimulating tone (PERLMAN[1], DAVIS and co-workers[2], RÜEDI and FURRER[3]). Like fatigue, trauma increases with the intensity and duration of the stimulation. In its initial phase it is reversible, and the restoration-time depends upon the extent of damage; later it becomes irreparable. It cannot be denied that damage may appear following excessive fatigue; the distinction between a transient trauma and fatigue does not, therefore, show in every case. A fundamental distinction in the mechanism of onset, however, consists in the fact that fatigue arises as an active reaction of the organ, while trauma can appear as completely passive behaviour. The displacement of the maximal hearing loss in relation to the frequency of the traumatizing stimulus, and probably to the maximum of excitation of the nervous end organ, would be the external expression of this fact. An acoustic trauma, the maximum of which corresponds to the frequency of the traumatizing tone, and which could be considered as a consequence of over-fatigue, has not, so far as we know, been observed with certainty up to the present time.

Fundamentally, therefore, three different phenomena must be distinguished: adaptation, fatigue, and acoustic trauma (LÜSCHER and ZWISLOCKI[4], LÜSCHER[5]). There are, however, fluctuations in the limits between them; and a sharp distinction is not yet possible. As has already been emphasized by LÜSCHER[2], and discussed in detail in the present paper, the modifications of the functional conditions of the ear following any acoustic stimulation as yet known can be explained by adaptation when the acoustic stimulus remains within its physiological limits, and by acoustic trauma when it is these beyond limits; fatigue, in the strict sense of the word, has not been sufficiently demonstrated up to the present time.

The development of *adaptation* involves two phases which are clearly distinguished in time: first a rapid decrease of the nervous impulses can be observed, the duration of which is of the order of a fraction of a second; then a slow decline of the frequency of the impulses follows, which is prolonged over about four minutes. MATTHEWS[6] terms only the second phase adaptation, the first being known by the English expression of "on effect". Basically, however, it is the same phenomenon, and it seems proper to speak of adaptation for both phases. For the first phase of rapid adaptation, the term employed by LÜSCHER and ZWISLOCKI[4] of "instantaneous adaptation", as distinguished from the successive slow adaptation phase, seems to us more appropriate.

The regression of adaptation takes place in an analogous way to its onset, since after a fraction of a second, during which a rapid increase of the effect can be observed, an evident slackening appears. The complete return adaptation requires about 1 minute (HOOD[7]).

According to the method of investigation, either the instantaneous adaptation of the slow adaptation is measured. The chief factor in this is the length of the applied tones and the time intervals between them. To this fact the discrepancies in the test results given by different authors are due. In the experiments of GORAN DE MARÉ[8], the instantaneous adaptation as well as the slow adaptation seems to be involved. LÜSCHER

[1] J. VON-KRIES, *Allgemeine Sinnesphysiologie* (Leipzig, 1923), S. 255 a.f.
[2] E. LÜSCHER, Z. Hals-Nasen-Ohrenheilk. *25*, 462 (1930).
[3] R. W. GERARD, Amer. J. Physiol. *82*, 381 (1927).
[4] T. E. BOYD and R. W. GERARD, Amer. J. Physiol. *92*, 656 (1930).
[5] A. J. DERBYSHIRE, Thesis, Harvard University, 1934 (Quoted by S. S. STEVENS and H. DAVIS, *Hearing* [New York, 1947]).
[6] S. S. STEVENS and H. DAVIS, *Hearing* (New York, 1947).
[7] R. GALAMBOS and H. DAVIS, J. Neurophysiol. *6*, 39 (1943).
[8] T. E. BRONX, J. Physiol. *67*, 270 (1929).
[9] E. LÜSCHER, Z. Hals-Ohren-Nasenheilk. *25*, 462 (1930); Riun. medico-chirurg. intern. (Torino, 1951).
[10] G. DE MARÉ, Acta Oto-Laryng. suppl. *31* (1939).
[11] E. LÜSCHER and J. ZWISLOCKI, Acta Oto-Laryng. *35*, 428 (1947).
[12] E. LÜSCHER and J. ZWISLOCKI, J. Acoust. Soc. Amer. *21*, 135 (1943).

[1] H. B. PERLMAN, Arch. Otol. *57*, 8 (1942).
[2] H. DAVIS and co-workers, Laryngoscope *56*, 13 (1946).
[3] L. RÜEDI and W. FURRER, Pratica O.R.L. *6*, 255 (1944).
[4] E. LÜSCHER and J. ZWISLOCKI, Acta Oto-Laryng. *35*, 428 (1947).
[5] E. LÜSCHER, Riun. medico-chirurg. intern. (Torino, 1951).
[6] B. H. C. MATTHEWS, J. Physiol. *71*, 64 (1931).
[7] J. D. HOOD, Acta Oto-Laryng., suppl. *92* (1950).
[8] G. DE MARÉ, Acta Oto-Laryng. suppl. *31* (1939).

and ZWISLOCKI[1], GARDNER[2], MUNSON and GARDNER[3] measured the instantaneous adaptation, while HOOD[4] purposely gave his attention to the slow adaptation. In preparation of the work undertaken for the present paper, which is a continuation of the publications of LÜSCHER and ZWISLOCKI, experiments were confined to measurement of the instantaneous adaptation, which is the main part of the whole adaptation effect. Consequently the method of investigation remains the same as that of LÜSCHER and ZWISLOCKI. The residual adaptation, which shows itself as an elevation of the threshold in comparison with the rest value, is determined 150 ms after the cessation of the 0·4 s stimulating tone. The determination of the momentaneous hearing threshold is obtained by a 30 ms test tone. From the residual adaptation, on the basis of the residual adaptation curves, conclusions may be drawn as to the behaviour of adaptation itself (LÜSCHER and ZWISLOCKI[5]). Since adaptation for all frequencies between 500 and about 8000 c.s. behaves in a fundamentally similar way, the frequency of the stimulating tone in all our experiments was kept constant, being 3150 c.s. The threshold elevation depending on the intensity of the stimulating tone and the frequency of the test tone was established. In this latter experiment, curves were obtained which gave the spread of adaptation to the neighbouring frequencies of the stimulating tone. This spread determines the distribution of adaptation and therefore of the excitation process along the cochlear duct (LÜSCHER and ZWISLOCKI[6]).

The primary aim of our investigations was to explain a discrepancy between the experiments of MUNSON and GARDNER[7] and those of LÜSCHER and ZWISLOCKI[8]. The latter have represented the dependency of the residual adaptation upon the intensity of the stimulating tone—by measuring it at a time interval of about 150 ms and for equal frequency of both the stimulating and the test tone—as a straight line in a double logarithmic scale. MUNSON and GARDNER, on the contrary, found an essentially much more complicated course of the curves. According to them, the residual adaptation for increasing intensities of the stimulating tone, increases at first relatively steeply, then reaches a saturation point with an almost horizontal course of the curve, and increases rapidly again at high intensities. MUNSON and GARDNER attribute great significance to this saturation point and relate it to the behaviour of the nervous impulses, the number of which in a time unit increases in a single nerve fibre only up to a given limit with the intensity of the stimulating tone. At present it is difficult to decide whether there really is such a relationship. In this connection, however, the observation of MUNSON and GARDNER that the residual adaptation shows no saturation for determinations carried out immediately after the cessation of the stimulating tone, seem to us of great significance. According to this fact, the saturation would be conditioned by the development of the return adaptation and by its dependency upon the intensity of the stimulating tone. A certain confirmation of this conception can be found in the re-adaptation curves of LÜSCHER and ZWISLOCKI[1], the courses of which are the steeper the greater the intensity of the stimulation, so that the time of re-adaptation depends very little upon the intensity of the stimulation.

Figures 1 and 2 show the behaviour of the residual adaptation (average values referring to 6 persons with normal hearing) depending on the intensity of the stimulating tone. Figure 1 shows equal frequencies of both the stimulating and the test tone. The unbroken line connects the measured points, the broken one is a straight line which represents an approximation of the curve. The deviation of the straight line from the exact course of the adaptation curve lies within 2 db. This finding corresponds to the previous determinations of LÜSCHER and ZWISLOCKI[2], for which 2 db was considered as the limit of error. These measurements were carried out in a great number of inexperienced persons, and in each person for at least 6 frequencies, so that great exactitude was obtained. GARDNER in a previous work[3], also went beyond the saturation point, although the exact connection of his measured points showed a flattening of the course of the curve in the middle tract. The curve of Figure 1 agrees with the observations of MUNSON and GARDNER in that it shows two opposite tendencies, but it contains no marked saturation. MUNSON and GARDNER give adaptation curves of only two persons examined, and the curves of only one of them show an evident saturation. In our opinion, therefore, this finding represents more an individual characteristic than a typical behaviour. In Figure 2 which contains three individual curves, it can be seen how much the course of the residual adaptation can vary in different subjects. In repeated experiments on one and the same person, relatively strong variations in tests performed at different times have also been observed. Figure 3 shows the average amount of residual adaptation depending upon the intensity of the stimulating tone for different frequencies of the test tone. All curves between 3000 and 5000 c.s. show an essentially similar behaviour. The steep rise of the curves, corresponding to frequencies higher than that of the stimulating tone for its intensities above 80 db, which appears particularly evident fpr 4000 c.p.s., is a characteristic finding. From this phenomenon intersections of the curves arise, as MUNSON and GARDNER have already pointed out. This seems to us of great significance and we shall discuss it further in detail.

The second set of our measurements concerns the spread of adaptation to the neighbouring frequencies of the stimulating tone. Figure 4 shows curves obtained from 6 persons with normal hearing for different intensities of the stimulating tone. Figure 5 gives individual curves of three persons for intensities of the stimulating tone of 100 and 60 db, referred to 10^{-16} W. Both diagrams agree in principle with the experimental findings of LÜSCHER and ZWISLOCKI[4], and of MUNSON and GARDNER which have already been fully discussed in the papers of these authors. We can therefore omit a general discussion and pay our attention to the points which have not yet been submitted to a more profound analysis. We refer first of all to the shifting towards the higher frequencies of the absolute maximum of the curves, ocurring at great intensities of the stimulating tone, which was

[1] E. LÜSCHER and J. ZWISLOCKI, Acta Oto-Laryng. *35*, 428 (1947); *37*, 498 (1949); J. Acoust. Soc. Amer. *21*, 135 (1943).

[2] M. B. GARDNER, J. Acoust. Soc. Amer. *19*, 178 (1947).

[3] W. A. MUNSON and M. B. GARDNER, J. Acoust. Soc. Amer. *22*, 177 (1950).

[4] J. D. HOOD, Acta Oto-Laryng., suppl. *92*, (1950).

[5] E. LÜSCHER and J. ZWISLOCKI, Acta Oto-Laryng. *35*, 428 (1947).

[6] E. LÜSCHER and J. ZWISLOCKI, Acta Oto-Laryng. *37*, 498 (1949).

[7] W. A. MUNSON and M. B. GARDNER, J. Acoust. Soc. Amer. *22*, 177 (1950).

[8] E. LÜSCHER and J. ZWISLOCKI, J. Acoust. Soc. Amer. *21*, 135 (1943).

[1] E. LÜSCHER and J. ZWISLOCKI, Acta Oto-Laryng. *35*, 428 (1947).

[2] E. LÜSCHER and J. ZWISLOCKI, J. Acoust. Soc. Amer. *21*, 135 (1943).

[3] M. B. GARDNER, J. Acoust. Soc. Amer. *19*, 178 (1947).

[4] E. LÜSCHER and J. ZWISLOCKI, J. Acoust. Soc. Amer. *21*, 135 (1943); Acta Oto-Laryng. *37*, 498 (1949).

observed for the first time by MUNSON and GARDNER. This phenomenon was related by these authors to the *acoustic trauma*, on the basis of a comparison with the experiments of DAVIES and his co-workers[1]. As is known, the acoustic trauma does not determine the maximum of hearing loss for the traumatizing frequency, but for a frequency which is shifted about half an octave higher. Our experiments too show this relationship. We wish, however, first of all to point out that for curves concerning the residual adaptation, it is not a matter of a continuously increasing shift of the maximum as the intensity increases, but of an onset of a second new max-

[1] H. DAVIS and co-workers, Laryngoscope *56*, 13 (1946).

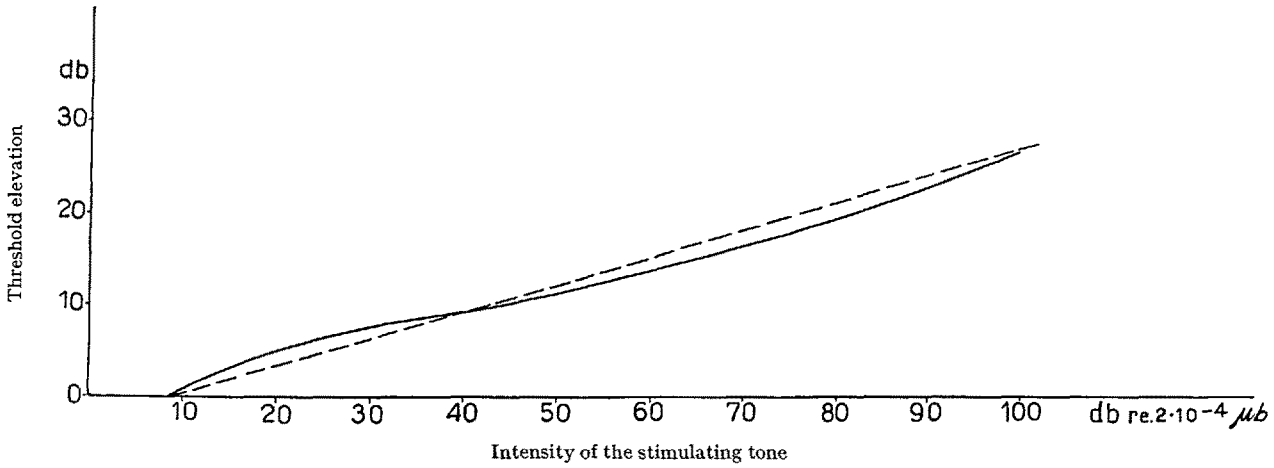

Fig.1.–Residual adaptation in function of the intensity of the stimulating tone (average curve of 6 persons with normal hearing). Solid line = calculated curves, broken line = approximation by a straight line. Frequency of the stimulating tone = frequency of the test tone = 3150 c.s.

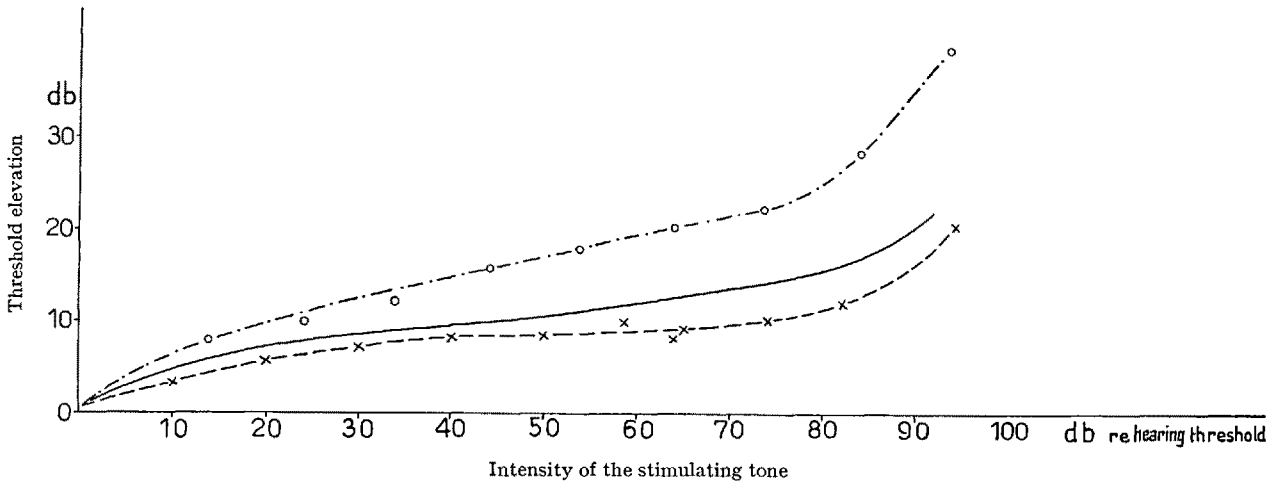

Fig.2.–Residual adaptation in function of the intensity of the stimulating tone (three individual curves). Frequency of the stimulating tone = frequency of the test tone = 3150 c.s.

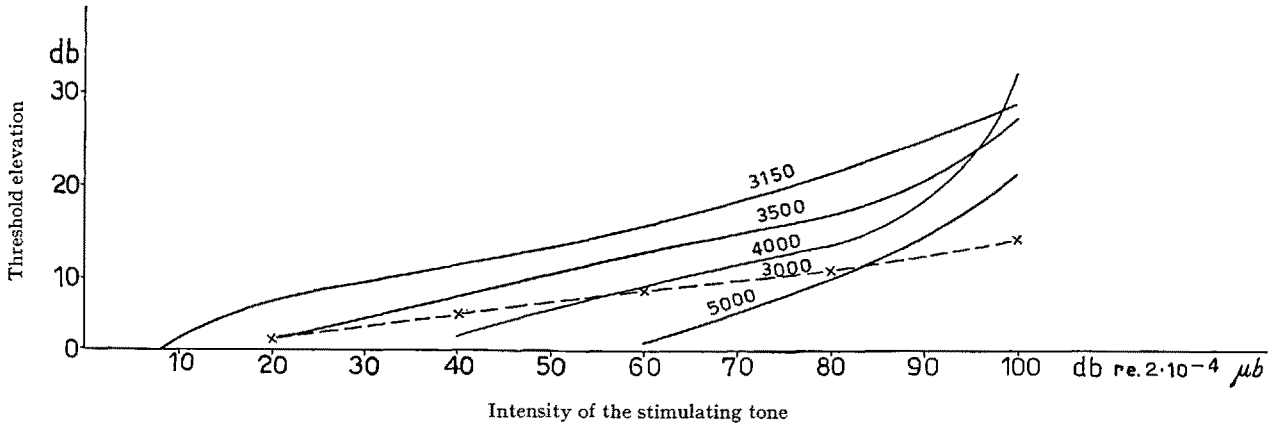

Fig.3.–Residual adaptation in function of the intensity of the stimulating tone for different frequencies of the test tone (average curves of 6 persons with normal hearing). Frequency of the stimulating tone = 3150 c.s., frequency of the test tone as parameter.

imum at a fixed frequency. Which of these maxima prevails, that at the frequency of the stimulating tone, or that shifted to about half an octave, depends among other factors upon the intensity of the stimulating tone. At low intensities only the first can be observed; between 70 and 90 db above threshold both of them occur, at even higher intensities the first maximum is completely overshadowed by the second. As shown in Figure 5, this is subject to wide individual variations.

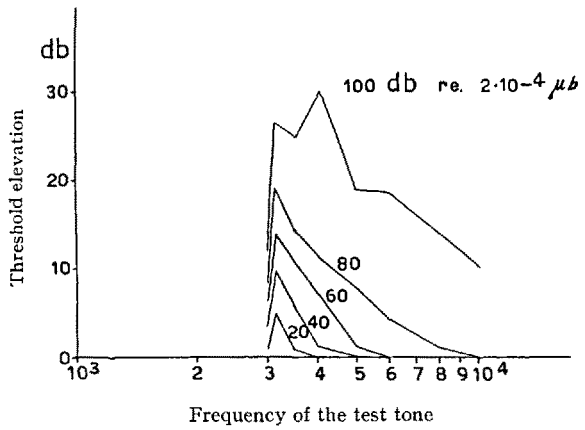

Fig. 4.—Spread of adaptation to the neighbouring frequencies of the stimulating tone for its different intensities (average values of 6 persons with normal hearing). Frequency of the stimulating tone = 3150 c. s.

The successive rising of the two maxima, which in the middle frequency range at least are shifted in relation to each other, independently of the frequency, by about half an octave, and which do not change their position with the variations in the intensity of the stimulating tone, suggests that, although both maxima are in a definite relationship with one another, each of them has a different immediate determining factor. This conception is strengthened by the following facts. The second maximum becomes the more evident the later the threshold determination takes place after the breaking-off of the stimulating tone (see the measurements of MUNSON and GARDNER as well as of HOOD). Immediatly after the cessation of the stimulating impulse, the greatest elevation of the threshold falls at the frequency of the stimulating tone, even for intensities of 110 db above threshold (GARDNER). In a time interval of 0·1 s the second maximum becomes prominent as soon as the intensity of the stimulus attains 80 db above the threshold. For a time interval of 0·2 s this occurs at 65 db (MUNSON and GARDNER), and after a pause of 20 s at the intensity of 80 db the first maximum reaches only one half of the value of the second (HOOD). If a train of 0·4 s tone impulses is given to the ear, with intercalated pauses of 0·5 s, for moderate intensities, the same hearing threshold is obtained after each impulse, independently of the duration of the train of stimulations. At high intensities, which lead to the onset of a second maximum, the threshold of hearing rises with the duration of the train of stimulations. This means that in the time intervals no complete restoration of the acuteness of hearing occurs, and that each new impulse causes a further threshold elevation. The effect is particularly striking at the frequency corresponding to the second maximum. The prolongation of the train of impulses beyond a limit determined by the intensity of the tone and by the individual sensitivity, leads to tinnitus and to a loss of hearing for some minutes or even hours. This is indeed the characteristic of the transient acoustic trauma (PERLMAN, DAVIS and co-workers, RÜEDI and FURRER).

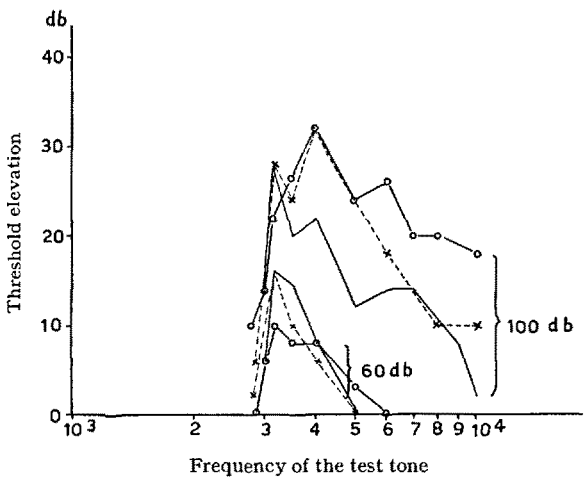

Fig. 5.—Spread of adaptation to the neighbouring frequencies of the stimulating tone for the intensities of 100 and 60 db (individual curves of three persons). Frequency of the stimulating tone = 3150 c.s.

From this analysis it follows that the threshold elevation, which occurs even at very low intensities of the stimulus with a maximum at the frequency of the stimulating tone, is in accordance with the conception of adaptation, while the part of the threshold elevation which at high intensities leads to the onset of a maximum shifted with respect to the frequency of the stimulating tone, must be considered as a *transient acoustic trauma*. In the first case we observe a rapidly appearing and rapidly disappearing effect, which can be reproduced at short time intervals, and the disappearing-time of which is dependent to a limited extent upon the intensity and duration of the excitation. In the second case, it is a phenomenon which arises slowly and slowly disappears and where the restoration time is conditioned by the intensity and duration of the stimulation.

The twofold nature of the hearing threshold elevation which is determined by a sound stimulus can also be found in the behaviour of nerve and cochlear potentials. The relationship between adaptation and the decline of nervous impulses has already been referred to above. Now we wish to call attention to the analogous behaviour of the threshold elevation as described in this paper and the cochlear potentials in the case of overloading of the sensory cells. As HUGHSON and WITTING[1] first established, the amplitude of the cochlear potentials, after reaching a maximum, decreases again. This effect was termed by them fatigue. For further successive weakening of the sound energy, the cochlear potentials no longer reach the original maximum, and show at any intensity a relative impairment. After a further overloading and slow diminishing of the sound energy, a further reduction of the amplitude of the cochlear potentials follows. The experiments can be continued in this way until the cochlear potentials fall to a small fraction of their normal amplitude, i.e. after any overstimulation an additional threshold elevation occurs. STEVENS and DAVIS consider this effect as damage, because it appears only as a result of overloading. The analogy between our experiments with the train of impulses and this behaviour of the cochlear potentials is striking. Even the minimal sound intensity, which leads to the type of threshold elevation

[1] W. HUGHSON and E. G. WITTINGS, Acta Oto-Laryng. *21*, 574 (1935).

described, corresponds in order of magnitude to that at which the overloading of the sensory cells appears.

These considerations lead us to the conclusion that while adaptation depends upon a decrease in the sensibility of the peripheral receptor and of peripheral nerve fibres, the threshold elevation of long duration described has its locus of origin in the sensory cells. It could be explained in the simplest way as being a depolarization; this would be independent of the excitation of the nerve terminations and conditioned solely by the mechanical demand on the sensory cells. Assuming that the localization of the maximal mechanical demand on the sensory cells is shifted in the longitudinal direction in the cochlear duct with respect to the site of maximal excitation of the nerve fibres, it is easy to conceive the fundamental distinction between the maximum of adaptation and the maximum of damage. The acceptability of this assumption has been demonstrated by one of the authors (ZWISLOCKI[1]) on the basis of mathematical deduction. The numerical calculation also showed that the maximal mechanical demand, which consists both of pressure and deformation vibrations, is shifted in relation to the maximal amplitude of vibration in the basilar membrane by a tract which corresponds to a frequency difference of about half an octave. The maximum of vibration should, however, coincide with the maximum of excitation, since it can be demonstrated that the sound pressure does not lead directly to the release of nervous impulses (ZWISLOCKI[2]). The hypothesis that the cause of the acoustic trauma is situated only in the sensory cells while adaptation involves also a part of the peripheral acoustic nerve, eventually up to the first synapse, is confirmed by the extension of their spreading to the neighbouring frequencies of the stimulating tone (Fig. 4 and 5) which gives a measure of the spreading on the basilar membrane. The spread of adaptation, in comparison with the distribution of the amplitude of the vibrations of the basilar membrane, as determined by V. BEKESY, shows an essentially sharper maximum, which must be related to the sharpening of the frequency analysis by the nervous end organ (ZWISLOCKI[2]). Such a sharpening of the frequency analysis cannot easily be ascribed to the sensory cells only. It is more likely that it is conditioned by the complicated distribution of the peripheral axis cylinders with their multiple ramifications. The essentially greater extension of the damaged area would correspond to the distribution of the sound energy along the cochlear duct in such a way that this also suggests its localization in the sensory cells.

The specification of the place of origin of the acoustic trauma described requires a better definition of the term itself. Indeed it is difficult to assume that a direct damage rises only in the sensory cells and not in the peripheral nerve or more centrally. In this case it would be connected with the nervous excitation and its maximum should coincide, so far as the frequency position is concerned, with the maximum of excitation, and therefore, according to our premises, with the maximum of adaptation. For an exact designation of the analyzed effect, we should speak of "damage of the sensory cells".

In conclusion, it may be said that, in agreement with the statements of LÜSCHER[3], the concepts of adaptation and of acoustic trauma give an interpretation of the modifications of the functional state in the ear following acoustic stimulations, and that at present it is not clear if and how far these phenomena should be partially related to fatigue in the strict sense of the word. Only further experimental investigations can decide this point.

[1] J. ZWISLOCKI, J. Acoust. Soc. Amer. *22*, 778 (1950).
[2] J. ZWISLOCKI, Acta Oto-Laryng., suppl. *72* (1948).
[3] E. LÜSCHER, Riun. medico-chirurg. intern. (Torino 1951).

Zusammenfassung

Es werden die Begriffe «Adaptation», «Ermüdung» und «akustisches Trauma» des Gehörs eingehend diskutiert, und es wird auf Grund von Hörschwellenmessungen nach vorausgegangener Schallreizung nachgewiesen, daß sich der Adaptationseffekt von der Ermüdung und vom akustischen Trauma grundsätzlich unterscheidet.

Die Hörschwellenerhöhung infolge der Adaptation wird durch die Intensität des Reiztones bestimmt und ändert sich bei einer rasch nacheinander wiederholten Reizung nicht. Jede hörbare Schallintensität hat eine entsprechende Adaptation zur Folge. Die Ausbildung der Adaptation, die sich aus zwei Stufen zusammensetzt, geht rasch vor sich. Die erste Stufe entfällt auf den «on effect» und bildet den größten Teil der Hörschwellenerhöhung. Sie wird als Instantanadaptation bezeichnet zum Unterschied von der zweiten Stufe, die einen relativ langsamen Prozeß darstellt und deshalb den Namen «langsame Adaptation» erhalten hat. Die Rückbildung der Adaptation erfolgt analog. Sie hängt zeitlich von der Reizintensität nur wenig ab. Das Maximum der adaptationsbedingten Hörschwellenerhöhung betrifft die Frequenz des Reiztones. Das Adaptationsmaximum ist wesentlich schärfer als das Schwingungsmaximum des Ductus cochlearis.

Ein akustisches Trauma entsteht erst bei höheren Schallintensitäten. Das Maximum der Gehörschwächung liegt um etwa ½ Oktave höher als die Frequenz des Reiztones. Die Ausbreitung des Effektes übertrifft diejenige der Adaptation und nähert sich der Verteilung der Schallintensität längs des Ductus cochlearis, deren Maximum gegenüber dem Schwingungsmaximum des Ductus cochlearis um eine einer halben Oktave entsprechende Strecke gegen das ovale Fenster verschoben ist.

Auf Grund einer eingehenden Analyse wird die Adaptation mit den Nervenpotentialen und das Trauma mit den Cochlearpotentialen in Zusammenhang gebracht.

SUPPLEMENTUM

M. BLUMER und I. M. KOLTHOFF, *Das polarographische Verhalten von* Ti(III) *und* Ti(IV) *in Äthylendiamintetraazetat*, Exper. *8*, 138 (1952).

Der Autor ersucht uns um nachträgliche Ergänzung des Abschnittes *Experimentelles* in obenerwähnter Arbeit. Nach der 7. Zeile ist einzufügen:

Alle Polarogramme wurden bei 25 ± 0,1°C durchgeführt; der Sauerstoff wurde mit luftfreiem N_2 verdrängt. Die Titan-Standardlösung wurde aus reinem $K_2TiO(C_2O_4)_2 \cdot 2\,H_2O$ durch Zerstörung des Oxalates mit konzentriertem H_2SO_4 nach der Vorschrift von SANDELL[1] hergestellt. Die überschüssige Säure wurde nach dem Mischen der Lösungen neutralisiert und bedingte einen etwa 100fachen Neutralsalzüberschuss.

[1] E. B. SANDELL, *Colorimetric Determination of Traces of Metals*, 2nd Ed. (Interscience Publishers, Inc., New York 1950), S. 575.