EFFECTS OF NOISE ON HEARING

M. SPRENG*

1. Introduction

The outstanding position of mankind rests to a great extent on their given talent to form and understand conscious ideas, to choose words as carriers to sematic meaning and in general of thoughts and to produce and understand these as spoken language. It appears, if we believe in the genesis, that the divine breath which represents the sole remarkable difference between the human and animal energy systems and information-processing systems, is mirrored partially in the excellent communication ability via our voice-producing and hearing organs. The difference between the performance of the human and animal visual systems is not so large by far. Nor is the visual system able to analyze such a distinctly time-variable process which speech represents ; spoken words are rapid, singular events ; they can practically never be articulated identically a second time. In consideration of this high communicative significance and performance of the ear it is particularly regretable that the eye and ear are of different sensitivity compared with the physical magnitudes which excite them. Light intensities which could damage the eye or rather the sensory epithelium of the retina are practically absent in the environment. Apart from this, the eye is able to protect itself by quickly closing the lids. By contrast, in our present-day environment, the ear is exposed relatively unprotected to noise events which exceed by far in intensity and duration, the loud natural sources such as, thunder or a water-fall. Nor does the ear possess a protective device comparable to the eye-lids even when the inner ear muscles are fully functional.

The consequences of the effect of noise on the hearing, which will be explained in greater detail during this discourse, are generally known. The person afflicted by a noise-induced hearing, impairment, handicapped surely in the most spiritual sense, no longer understands what is said in the group, he becomes an outsider of human society and with increasing deafness loses contact even to individual persons, he becomes suspicious, finally resigns and lapses into a 'world of silence'.

* Prof. Dr. Mansfred Spreng, Institute for Physiologie and Biokybernetik, Universital Erlangen-Nurenberg D-8520 Erlangen : Universitatsstr

It is remarkable that approx. 6 million of our population, i.e. around 10% are handicapped severely or moderately in their hearing ability.Every second person aged more than 65 years already belongs to this group so that we by no means can speak of an eventful pension age. The number of old people suffering from defective hearing caused by the noxa of civilization, above all by environmental noise, increases alarmingly and the number of those damaged by occupational noise rises ; noise-induced hearing loss has become by far the occupational disease number one.

Before concrete effects of noise on the hearing are discussed in detail, the question must be posed which parameters of sound or sound combinations are to be considered as particularly damaging and where or how do noise-induced hearing impairments manifest themselves.

2. Damaging types of sound and combinations

Naturally the strength of the sound impinging on the ear and the duration of sound exposure are of decisive significance. It therefore appeared expedient to multiply the easily measurable sound intensity, a power dimension (energy per time) with the exposure time, in order to obtain the sound energy received by the ear. Although no sensory system-the hearing least of all-works as an integrator in this sense, numerous studies. above all the detailed investigations by Burns and Robinson (1970) demonstrate a relation between to some extent auditorily correctly-(A-weighted) sound energy and lasting hearing damage. Simply expressed this means, the same sound energy results in the some hearing damage.

One considers, simply speaking, the sound energy absorbed as a dose which when increased, as with a toxic agent, causes increasing damage. The consequences which emerged from these considerations led to the numerous guidelines, for example, the ISO 1999and so-called damage risk criter a, such as those given in Fig 1.

The hearing defects occuring when these criteria are exceeded are confirmed on average by retrospective studies using a large number of persors despite the unclear initial circumstances and the effect of additional factors.

This hearing defects vary for the different audiometric test frequencies and at high intensity after exposure of 10 years are as expected, more pronounced (Fig. 2a). Both the extent of the injury and the number of the persons concerned increase with the number of working years (Fig. 2b).

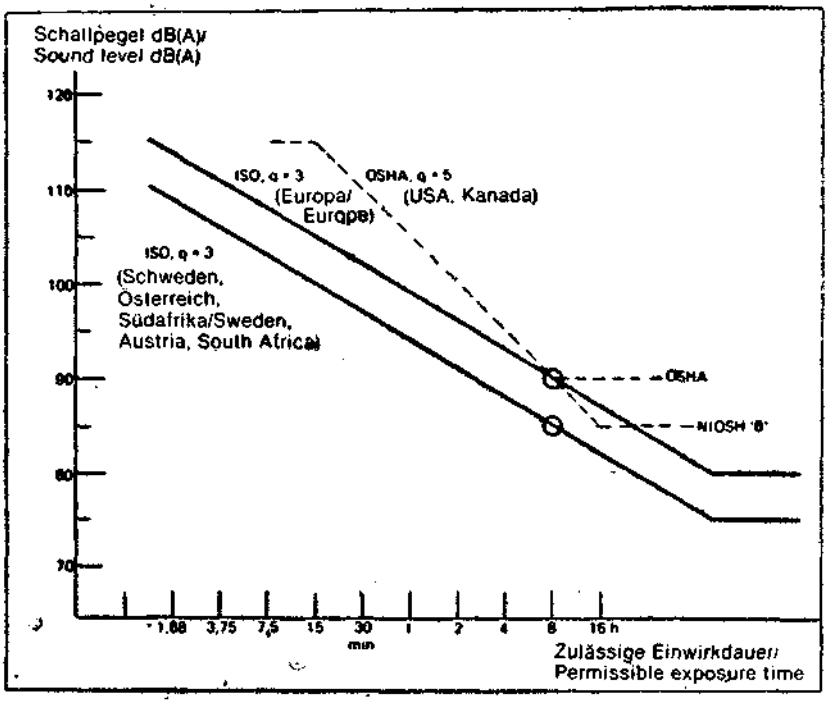


Fig-1

Cursory applicat on of the basic hypothesis of energy equivalence would in general enable one to change the two parameters in the product : intensity times time, arbitrarily in relation to one another, i-e., to increase the intensity the shorter the exposure time is, without causing any damage as the limits are not exceeded (Fig. 1). However, this already illustrates clearly the limitation of the energy equivalence rule which basically is not valid. It is limited to a certain range and could be used actually only for uniform broadband noises which are not all too frequent in every-day industrial life. The energy equivalence rule, which represents an acoustic simplification and an easily managed test method (dose measurement) also exhibits its weaknesses when it comes to assessing not a large population but an individual with regard to his hearing defect or to predicting the risk of damage, since the type of noise exposure in relation to time, for instance, the duration of pauses, is of decisive importance.

In the last analysis, a simple risk criterion is not definable and in the case of impulse sounds, which are considered in detail later on, as well as sound

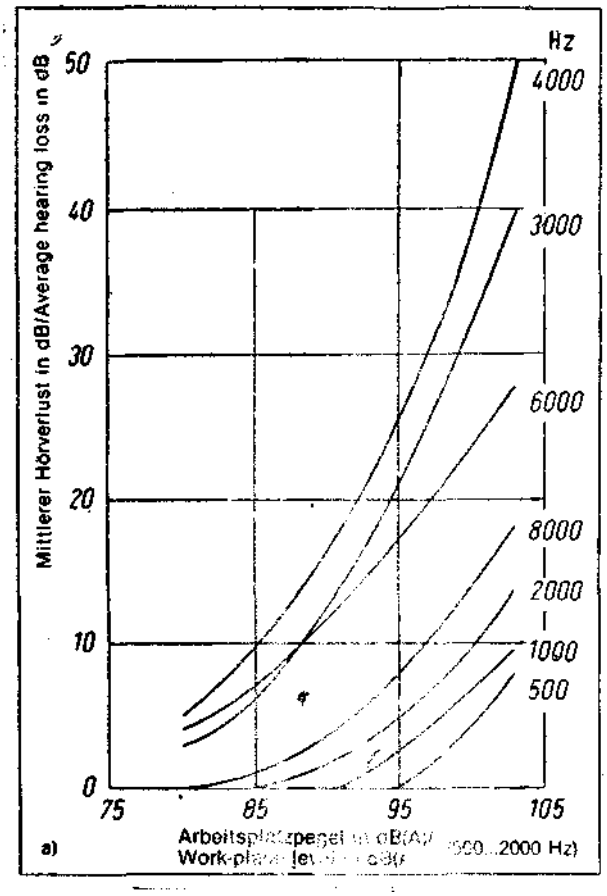
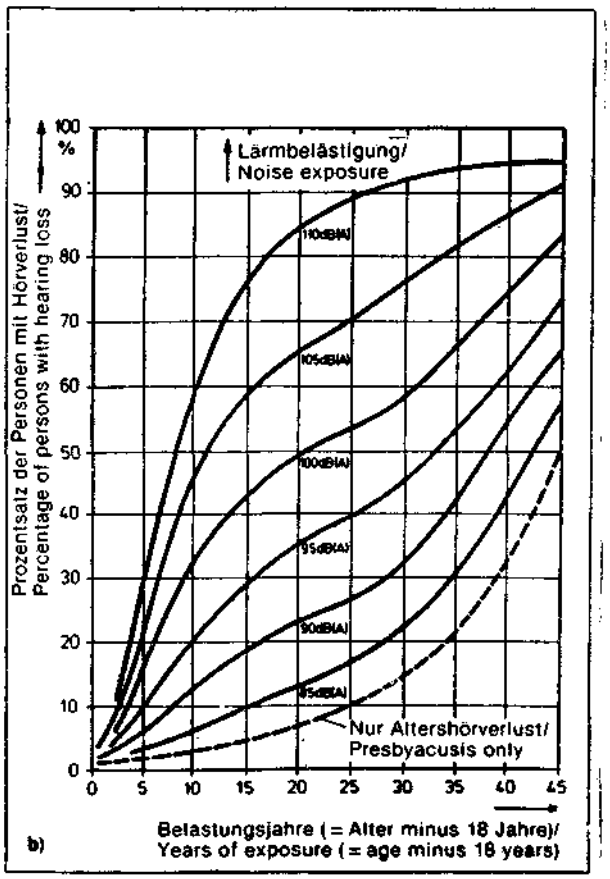


Fig-2a

combinations (uniform background noise and fluctuating or impulse noise) the risk or the probability of noise induced deafness must be defined newly and better by additional criteria or new test and assessment methods..

It can only be said with relative certainty, but without relevance for an industrial worker, that sound levels below 75 dB do not produce any temporary threshold shift and no damage to the hearing. If one considers continuous noise (oscillating less than $\pm$ 5 dB), then agreement prevails nowadays that a level of 75 dB with a. daily exposure time of 8 hours (or 70 dB for 24 hours) represents the limit for discernible, permanent noise-induced threshold shifts. These threshold shifts are determined at the particularly sensitive audiometric test frequency of 4000 Hz. If one uses the conventional



Fig~2b

speech frequencies (0, 5 : 1 and 2 kHz), then the threshold value is shifted by 10 dB for continuous noises which implies that per 8 hours working day practically 85 dB can be endured without damage (Fig. 1).

It must be emphasized in this connection that impulse noises as well as sound combinations are here excluded and that a relatively quiet, noise free recovery phase is assumed in the free-time.

If the noises are uniform but no longer broadband and contain energy concentrations in certain frequency bands or also tonal components, the probability of damage is of course increased. Noticcable tonal componens e.g., with wood-working machines, appear to pro duce markedly more damga (Niemeyer, 1981). In that case the temporal threshold shifts are larger at

higher frequencies and are found above the central frequencies of, hose narrow band sounds.

If, in animal experiments, the ear is exposed to noise which contains such tonal components, primary changes appear to develop after a few hours at the corresponding location in the cochlea, however, with further exposure to sound those defects develop in the basal area of the cochlea, that is in the range 4000 to 6000 Hz which are characteristic lor noise damage (Dieroff, 1980).

There are a number of hypotheses which cannot be discussed in detail for this behaviour, that is the formation of characteristic losses in the region 4000 to 6000 Hz (high-tone dip). The following should be mentioned : the fact of the strongest overlapping of the basilar membrane oscillations with broad-band and also low-frequency noises at this location, the severely reduced reflex protection of the middle-ear muscles beyond 3000 Hz, the additional oxygen supply resulting from diffusion through the round window with corresponding maximum deficiency symptoms in the range mentioned and finally the fact that with metal sounds, the extremely short rise and fall time of the high peaks, give rise to energy components mainly in he upper frequency range (6000 Hz)

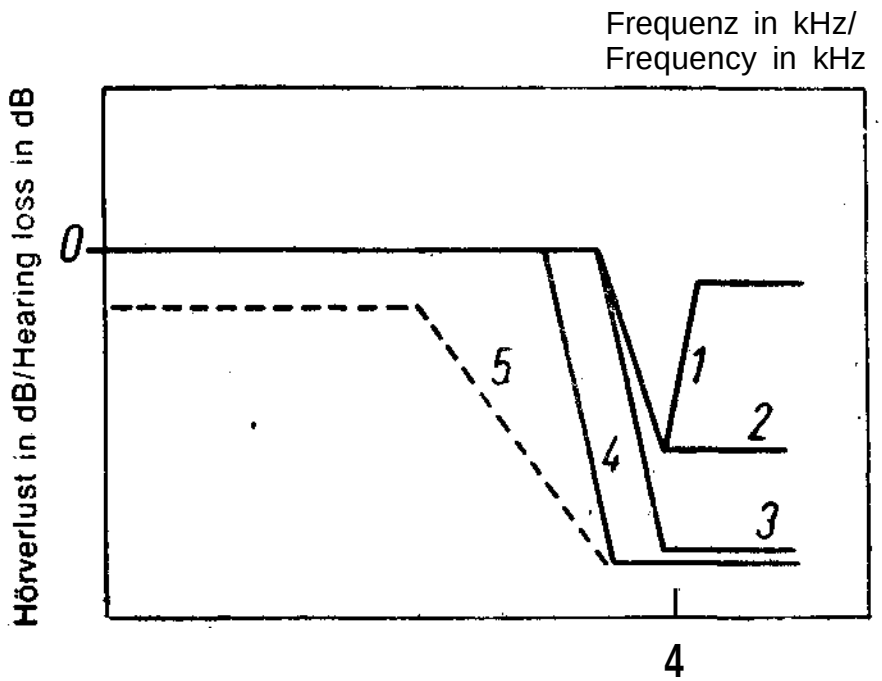


Fig-3

Concerning the change in the hearing ability, reference should be made at this point to the threshold increase or reduction in hearing sensitivity as characteristically caused by noise, compared with the person of normal hearing which is easily measured with the audiogram. Fig. 3 shows diagramatically that, beginning at the high-frequency dip (1) mentioned, after it broadens, a hearing threshold curve in the shape of a staircase (2) arises : almost a normal hearing threshold in the lower and middle frequency range up to 1500 or 2000 Hz, then steep drop and approximately level course up to the highest test frequencies. The hearing loss above the step drop generally amounts to no more than 60 dB. With increased hearing impairment greater losses (3) occur there and with further progressive damage, the steep drop of the audiogram curve moves in the direction of lower frequencies (4) and comes to rest at around 1000 or 750 Hz. The change then taking place in the course of several years consists of a transition from the steep slope to an oblique slope as indicated in the diagram by a dashed line (5).

It has emerged recently that not only the sound intensity and its exposure time as well as tonal components but, above all, the rise and fall time, that is the pulsating or fluctuating character of the noise are of great significance. In fact, many more persons in industry are subjected to fluctuating noise and it is becoming more and more apparent that in addition to the energy contained the fluctuation plays a decisive role with regard to the damage.

The significance which one wanted to attach to the protective function of the middle ear reflex is very questionable. With continuous sound exposure, the reflex action is lessened very rapidly by adaptation (Zakrisson et al., 1980). On the other hand, a change in the intensity or frequency or a short interruption in the sound exposure has the effect of re-activating the reflex. Although investigations of the temporary threshold shift with combined sounds (continuous sound + fluctuations) lead one to assume an increased protective reflex examinations regarding permanent threshold shift and, in particular, histological findings, demonstrate the opposite to be true (Cohen, 1966 ; Hamernik, 1974 ; Arlinger and Mellberg, 1980).

In particular, if the fluctuations become more rapid or the pulses shorter (40/us), then depending on the repetition rate, the reflex latency of 10 to 12 ms is in general so high that the inertia of the transmission system of the middle ear no longer permits any protective function. According to examinations carried out by Dallos (1973), the sound-protective reflex above 3 kHz is in general relatively small.

Basically, the action of impulse sound within the cochlea and on the aensory cells is to bs assessed in a different manner to the action of continuous sound. This is also the reason why a large number of investigations consider the equal energy rule, which can be relatively well applied to uniform noise, to hold good only up to a point or not at all for impulse or fluctuating sound (Hamerniket al., 1980; Nilsson et al., 1980; Dancer et al,, 1980). Nor can the iso-energy principle be applied arbitrarily when spectrum and peak amplitudes are the same (Buck et al., 1980). The assumption that impulses are relatively harmless because they are not sensed as being loud, is false. On the contrary, it can be seen that the peripheral transmission in the middle ear and the peripheral nervous processing proceeds very rapidly. Therefore the greater excitation with initially overshooting components (upto 15 dB) and the higher risk are to be sought in the peripheral area of the ear, the rise time of the sounds being of great significance. Bruel (1980) also referred to this and attempted to explain the typical hearing threshold reduction in the 6000 Hz range in the case of metal workers by the very short sound durations and relatively high peaks with metallic sounds. Therefore impulse sound level meters with time constants of 30/us (instead of 35 ms) should be used since the integration modelled with the large time constant is a central process (Spreng 1977 and 1981). The mode of action of these rapid rise times becomes clear immediately when one realizes that all sensory organ systems, in particular the ear, reacts especially dynamic changes of the stimulus. The hearing is a differential-proportional-sensitive system and in no way an integrative one.

An attempt is made in Fig. 4 to provide a diagram of the excitation of the sensory organ system of the ear with such rapid changes in stimulation. It is shown on the left with the aid of different arrows that a 20-dB jump imposed upon a background noise of 40 dB, produces differing excitation magnitudes should rise times of 100 ms, 200 ms and 700 ms occur. In the first instant, the dynamic characteristics (arrow or dotted) apply and their steepness depends upon the rise speed of the sound stimulation. After a certain time, the over-shooting excitation returns to the appropriate new value (adaptation) which is given by the static characteristic (bold line in right-hand part of diagram). If a pulse-shaped sound level change with a comparable dropping slope is present, then the excitation (corresponding to the dynamic characteristic drawn with a dotted line) can also initially exhibit 'undershooting' behaviour and then return once again to the excitation value of the original working point. If this behaviour is sketched ior two pulse-shaped sounds, which subsequently have the *same equivalent continuous sound level* but possess differing size, durat-ion and above all, rise times, then completely different adaptation diagrams or

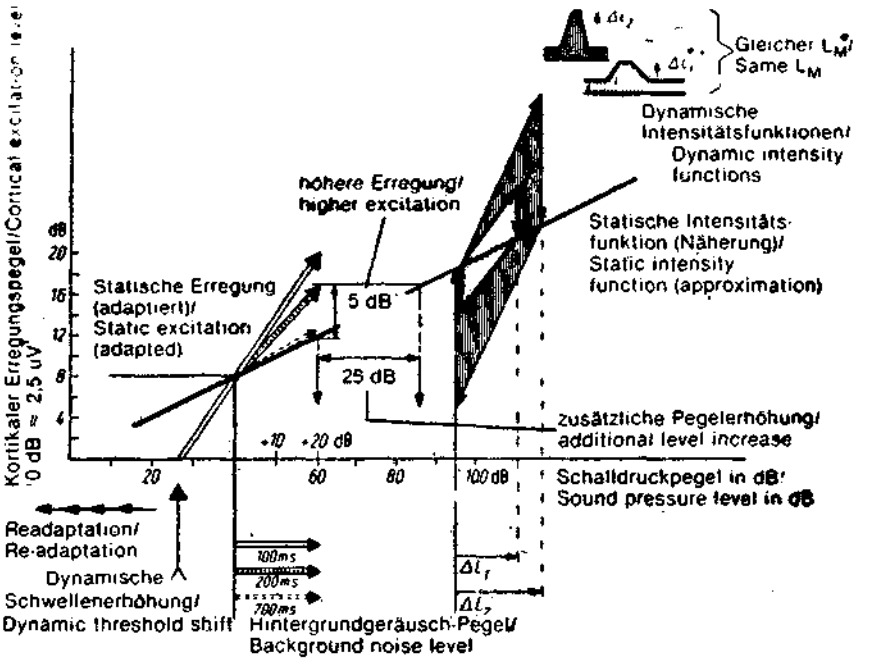

Gleicher L_M/
Same L_M
Dynamische
Intensitätsfunktionen/
Dynamic intensity
functions
höhere Erregung/
higher excitation
Statische Intensitäts-
funktion (Näherung)/
Static intensity
function (approximation)
Statische Erregung
(adaptiert)/
Static excitation
(adapted)
5 dB
25 dB
zusätzliche Pegelerhöhung/
additional level increase
+10 +20 dB
Schalldruckpegel in dB/
Sound pressure level in dB
Readaptation/
Re-adaptation
Dynamische
Schwellenerhöhung/
Dynamic threshold shift
100ms
200ms
700ms
Hintergrundgeräusch-Pegel/
Background noise level
ΔL_1
ΔL_2

Fig-4

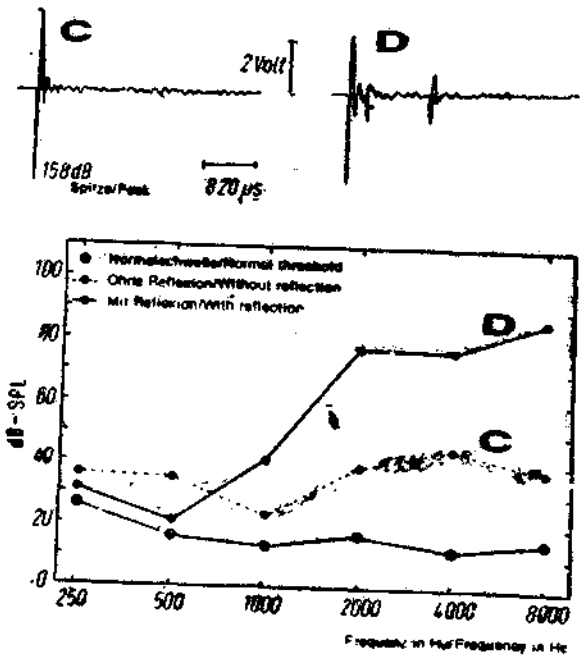

C
2 Volt
D
158dB
Spitze/Peak
870 µs
Ohne Reflexion/Without reflection
Mit Reflexion/With reflection
dB-SPL
D
C
250
500
1000
2000
4000
8000

Fig 5

adaptation areas result which can represent a reference point for the loading of the sensory organ system. One detects clearly that with a higher rise time (hatched vertically), an essentially larger area of the adaptation diagram results than with a lower rise time, somewhat longer duration as well as slightly smaller amplitude (dotted), although with both pulse sequences the same average level (L_M) is present.

Because of this model conception, it is no longer surprising that in the animal experiments of Hamernik ct al. (1980), remarkable dependence of the damage on the specific impulse shape and the presence of after oscillations shown in Fig. 5 is found. Above all, in the frequency range higher than 1000 Hz, a strong (> 50 dB) rise in the hearing threshold(D) of chinchillas (measured with evoked potentials) is evident when pulses with 158 dB peak and one after oscillation (D) arise. If the microstructure of the sound pulse contains no after-oscillations (C), then the dashed curve exhibits a smaller (10 to 20 dB) and parallel threshold shift (dashed).

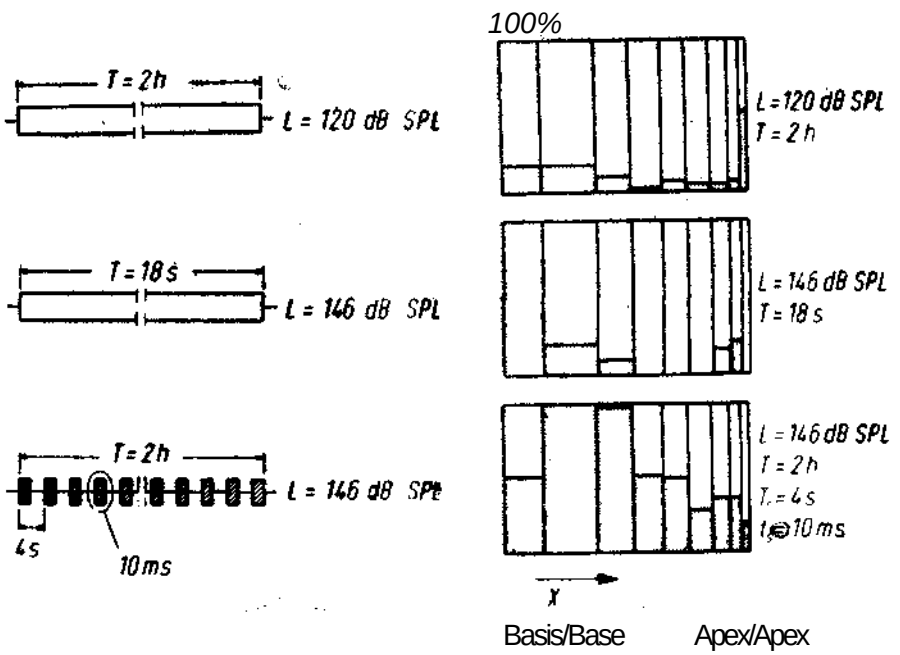


Fig-6

The results of Buck (1980) displayed in Fig. 6 show in the same manner that impulse noise of the same energy causes more severe hair-cell loss and leaves behind a permanent threshold shift. The sound types with

the same energy are displayed in the left half: 1. two-hour sound exposure, continuously with 120 dB; 2. 18-seconds sound exposure with 146 dB and 3. 2-hour sound exposure with 10-ms 146-dB impulse sound at 4-sec intervals. The stimulation consists of an octave noise between 4.5 and 9 kHz. The hair-cell loss stated on the right for the appropriate sound type clearly shows the severe effect of impulse sound exposure.

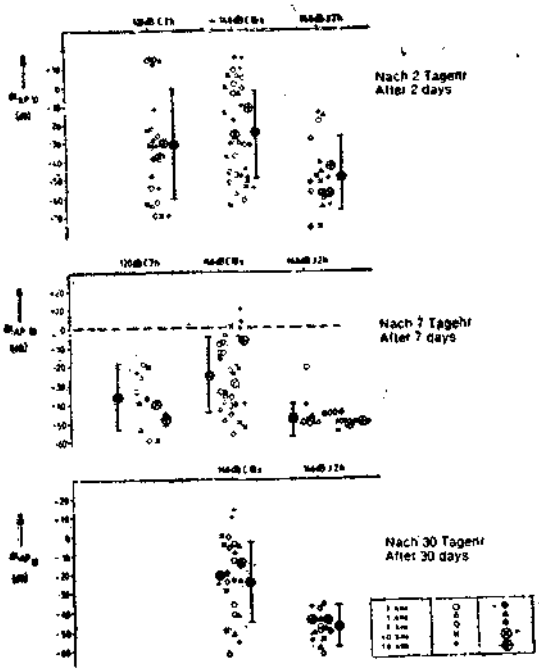


Fig-7

Fig. 7 makes plain that the threshold increase or the amplitude loss $\frac{\Delta L}{AP}$ of the compound action potentials of the acoustic nerve proves to be clearly stronger with the impulse sound exposure (146 dB I 2 h) and above all, it becomes permanent for over seven days and for over one month. The threshold changes caused by the continuous noise (120 dB C 2 h) are no longer present after one month. The energy equivalence hypothesis formulated by Atherly and Martin (1971) for impulse sounds is indeed attractive (it attempts to connect in an overlapping manner continuous sound and impulse sound exposure and is extremely simple to use with particular reference to dose measuring equipment), but so far there is absolutely no confirmation as not only these animal experiments but also investigations on

humans show. Data from Sulkowsky (1980), in particular, make plain that large deviations from the energy-equivalence hypothesis are present when there

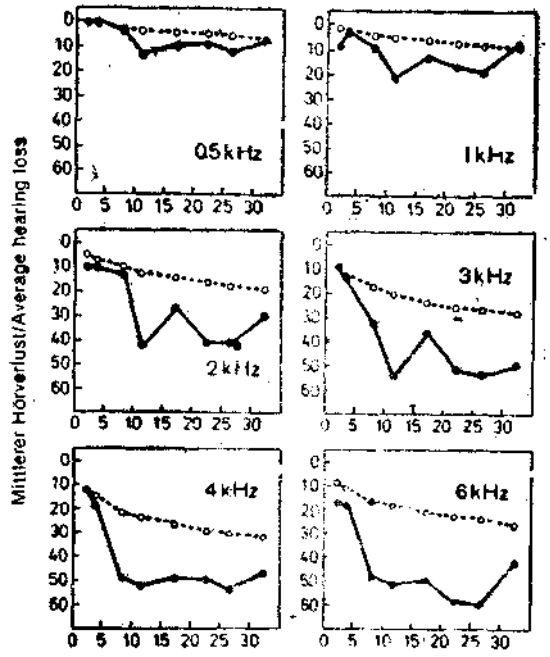


Fig-8

is a high repetition rate of the impulse configuration. Fig. 8 illustrates this with reference to measurements on drop-forge workers, the hearing loss of whom is displayed as a function of the exposure time. The hearing loss which should have occurred according to the energy-equivalence hypothesis is shown by a dashed line. The damage actually present which is significantly greater, speaks for itself and also against unreflected application of the energy-equivlence hypothesis without careful consideration of the impulse sound or combnation sound configuration.

The large variation in damage to individuals can also be deduced partially from the combinatory or additive effect of sound configurations-apart from different individual sensibility, varying relative position (in particular towards the impulse noi e source), influence of other noxa, etc.

Here, mention must indeed be made of the addition in time of noise influences : differing additional stressing of the ear takes place before and after a working day, whereby not only the increased loading but above all the

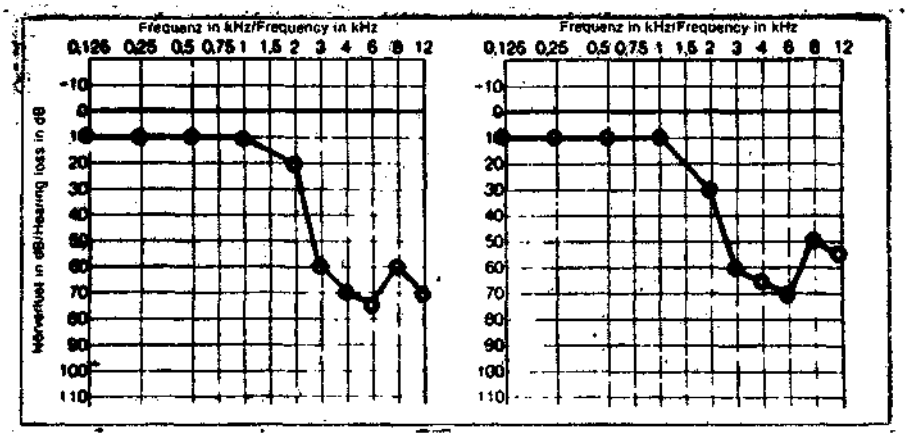


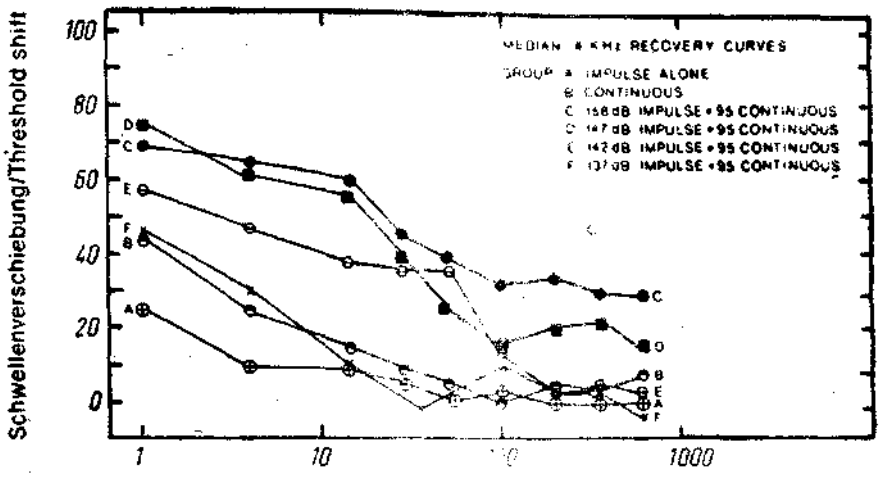
Fig-9

educed recuperation time take their toll (Spreng, 1980, Fig. 9; Weidauer, 1980) as is shown, for instance, by the audiogram curves of a 21-year old fitter who worked for eight years at between 85 and 100 dB (A) noise-assessment levels and in the evening blew a trumpet in a jazz-band. The extreme threshold reduction in the high-tone range (70 dB) is in contradiction to the assessment level of the work place (according to Pig. 2a only approx. *40 dB* hearing loss should have occurred after 10 years) and this makes plain the influence of leisure-time noise.

In this connection, Johnson and colleagues (1975) and Nixon and colleagues (1980) could emphasize the significance of asymptotic temporary threshold shift (ATTS) which, independent of the sound exposure duration with pink noise, possesses the same value, but a different recovery time. As rule of thumb, it can be inferred from their results, that ATTS values $\geq$ 40 dB can produce permanent changes and that recuperation times in relative quiet (<7S dB) should be around the same length as the exposure times. As Coles (1980) mentioned, it would appear that the lack of pauses at night, even with lower intensities than assumed earlier, cause slight permanent threshold shifts'

Amongst other things, as mentioned already, the noise addition or combination must be understood in relation to intensity : according to the configuration of background noise (in general less fluctuating) and impulse *noise,* the ear is stressed in differing adaptation conditions aad thus metabolic conditions by extreme dynamic processes (Fig, 4)

Hamernik and colleagues (1980) have **investigated in** chinchillas, **different combinations of this type** with regard **to** the **decline of the temporary**



Stunden nach Beschallung/Hours after sound exposure.

Fig-10

threshold shift after sound exposure. Fig. 10 shows as result that with continuous sound exposure (95 dB SPL) in accordance with curve A and with solely impulse sound exposure (158 dB, 1/min, 50/us duration in accordance with curve B, the temporary threshold shift has been completely eliminated after 1000 hours. From a combination of the sounds, which in themselves would have been still just tolerable, considerably larger temporary threshold shifts result which are not eliminated even in the course of 1000 hours (C) Permanent threshold shifts are still present even when the impulse noise is reduced to a peak level of 147 dB (curve D)

It may be appropriate to show investigation findings made *by* the same working group which result when vibratory loading (lg) and noise loading act simultaneously. The continuous curve in Fig. 11 shows the clearly greater permanent hearing loss with the simultaneous action of these two loads. Vibration alone affects practically no change in the hearing capacity (dash and dot curve); whilst impulse noise (155 dB, 1/s 1.5 ms duration) results in a moderate degree of hearing impairment (dashed curve)..

3. Damage in the morphological and functional area

A short summary of the observable damage is shown in Fig- 12. It must naturally be stated in advance that mechanical damage by impulse noise

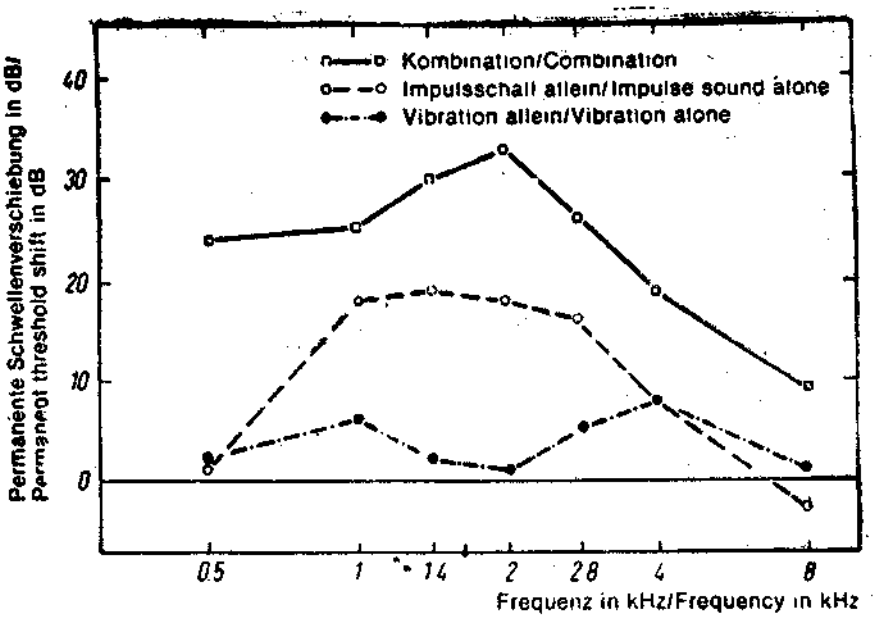


Fig-11

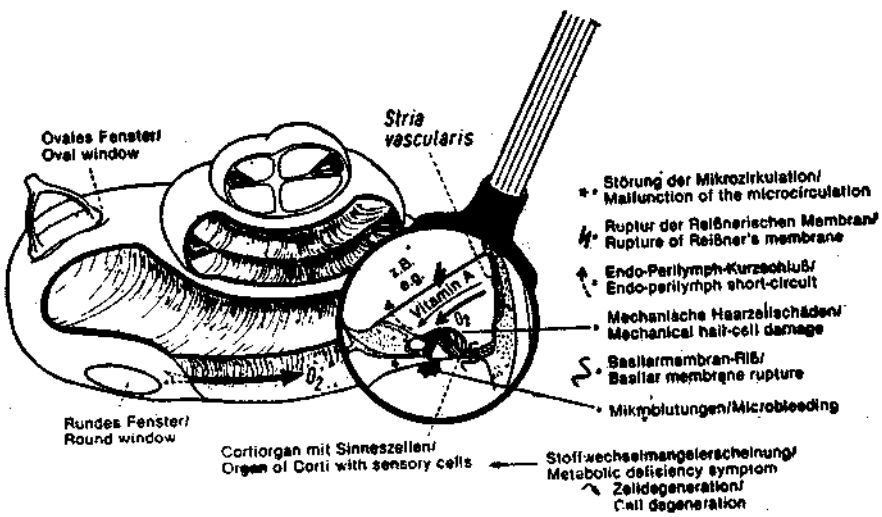


Fig-12

(e.g. pistol shot at 2.5 cm distance : 145 dB, 40 ms) such as ear drum perforations and fracturesin the manubrium mallei occur in particular when a maximum deflection (30/u) and stiffening of the ossicular chain can be assumed and thus a certain dissipation of energy takes place in the middle ear-for the protection

of the inner ear (Cody and Johnestone, 1980). A reason for the large variation in damage is also to be found here, above all, with regard to inner-ear damage, since indeed strongly variable tranfer functions of the middle ear and variable reflex latencies exist.

The mechanical damage in the area of the cochlea is manifold. It extends from fusion and pulling out of the tiny hairs of the sensory cells, warping of the stems of the pillar-cells, to complete hair-cell loss, whereby the first row of the outer hair-cells is affected more severely than the second and this once again more severly than the third, before the inner hair-cells are damaged (Dancer et a,l, 1980). The rapid resorption of the cell residues is followed by deterioration of the supporting cells concerned, so that after a certain time, the entire organ of Corti in a particular area no longer exists. This mechanical destruction is possibly initiated by microbleeding, such as has been found by Reinis et al. (1980). caused by ultrasonic bangs in the area of the basal to median scala tympani in diff rent animals.

Smaller mechanical damage (e.g. holes in the area of ReiBner's membrane or of the reticular membrane) may firstly change or impede the diffusion processes since the endolymph space is indeed separated by the two membranes from the perilymph area of the scala vestibuli and scala tympani. The diffusion processes required by the richly perfused stria vascularis for supplying the sensory cells are then disrupted by short-circuits so that an optimum supply of the sensitive sensory cells is no longer guaranteed.

Apart from the no longer optim m metabolic supply to the sensory cells, a change in the fluid composition in the endolymph space or a fusion between Corti-and endolymph (Reinis, 1980. Dieroff, 1980) also result from this.

The resulting malfunctioning or metabolic disturbance of the sensory cells is therefore primarily of mechanical origin. Mechanical stressing and lasting or temporary decoupling between stereociliae and the covering membrane must also be taken into consideration as noise-induced mechanical changes (TONNDORF, 1977).

With sounds of longer duration and lower intensity no mechanical damage is to be expected, although metabolic overloading of the sensory cells corresponding to the exposure time occurs.. Here, no simple connections exist such as vasoconstriction and subsequent oxygen deficiency and cell death, since the Substance reserpin, for instance, remains without effect. Mostly the compositions of the endolymph. in particular the size of the measurable electrical potential (endodochlear potential) appear to play an essential role. If for

instance this endocochlear potential is lowered by amlno-oxyacetic acid (AOAA), then the number of the damaged cells also reduces (BOBBIN and KISIEL, 1980).

On the other hand, collapsed vessels or traces of vessels are found in the morphological picture of the microcirculation in the lateral wall (HAMERNIK et al., 1980) so that without doubt, microcirculation disorders must contribute to the metabolic overloading especially in connection with the production and resorption of the endolymph.

In principle, it is the oxygen deficiency occurring after longer loading and the emptying of the endosplasmatic reticular system (LIM and MELNICK, 1975), above all of the ATP energy stores the replenishing of which via anaerobic glykosis and the restructuring of the chromatin substance leads finally to swelling and bursting of the cell core. This occurs all the earlier, the worse the transportation of oxygen, vitamin A, glycogen etc., from the stria vascularis via the relatively long diffusion path functions.

The change in the fluid composition or the metabolic disturbance of the sensory cells can be measured by means of the microphone potentials, or with the aid of the action potentials of the auditory nerve.

The threshold rise of the summation potential N_1 of the acoustic nerve resu'ting after exposure to sound is indicated in Fig. 13 (page 48). Above this, one discerns the hair-cell loss of the external (center) and internal (above) hair-cells. Where a marked hair-cell loss is present, the threshold is highest, as is to be expected. On the right, four tuning curves of different individual nerve fibers are indicated for the frequency ranges identified with numbers. These tuning curves are the intensity threshold curves of individual sensory cells. They indicate, in dependence of the prevailing frequency, that particular sound intensity at which a just measurable action potential sequence arises on the nerve examined.

It can be clearly seen from Fig. 13 that with the transition from frequency range 4 to frequency range 1, i.e. with increasing damage, these tuning curves always become flatter. The frequency selectivity is educed, the critical frequency shifts to lower frequencies and the low frequency component of the tuning curve moves to higher intensities (CODY and JOHNSTONE, 1980).

4. Direct consequences of noise-induced hearing defects

Masking :

Noise-induced hearing impairment can be present even without damage

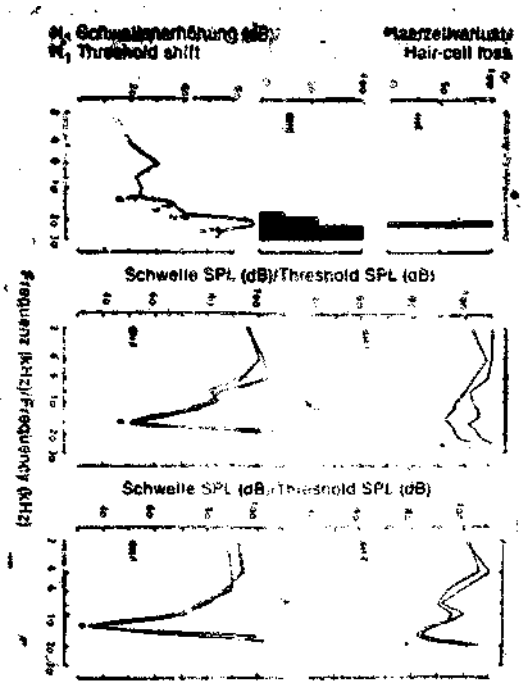


Fig-13

to the hearing organ. This is, as is well known, termed masking whereby mainly speech and warning signals can be masked and the monitoring of one's own speech reduced (SCHULTZ-COULON, 1980). The masking effect of noise, which contains low frequency components, is worthy of mention mainly because in this case, it particularly affects the area of approximately normal hearing still available to the hard-of-hearing person.

Temporary threshold shift (TTS) :

The temporary threshold shift (TTS) is, as the name implies, a time limited increase in threshold which is eliminated again. According to WARD (1979), it can be divided into four different time sections :

1. A short refractory period (residual masking) relatively independent of the duration of sound exposure which lasts for fractions of a second.

2. An adaptation arising with moderate levels {<85 dB) which fades away after a few minutes and can he partially accompanied by weak noise sensations.

3. The normal threshold shift (TTS) or physiological fatigue in the order of magnitude of 25 to 30 dB which is customarily measured two minutes (TTS_2) after the end of lengthy exposure to sound. Normally, it returns to the initial threshold (early detection) after approx. 10C0 minutes (16 hours). This threshold increase exhibits exponential rise and decay, it reaches an asymptotic value (ATTS) after 8 to 12 hours and it grows approximately linearly with the level, when a base level (70 to 75 dB) is exceeded, with higher frequencies causing the rise to be stronger.

4. A pathological threshold shift is present when the temporary threshold shift is larger than 40 dB. Then the decay time can last for days or weeks and if exposure to sound is renewed, the probability of a permanent threshold shift is high.

Permanent threshold shift (PTS) :

The permanent threshold shift (PTS) is a lasting hearing defect, without regression. It cannot be decided, as a matter of course, if the permanent threshold shift (Fig. 2a) during a working life, represents a slow, individually non-measurable effect or if it is caused by a limited number of sound events. In general, it is not only the noise at the work-place but a multitude of other factors and non-acoustic events (soci-acusis) as well as diseases (nosoacusis) and the normal ageing process (presbyacusis) which cause permanent threshold shifts.

The temporary threshold shift (TTS) caused by similar, but short term noise, is no clear indicator for the formation of permanent threshold shifts ((((((((((PTS). Reference is made in the preceding section to the problem of dose measurement, to the influence of shape and duration, as well as to additive sounds or combinations with other stimulations. It must be mentioned here once again that, above all, rest times which make recovery processes possible, counteract the formation of a permanent threshold shift and that so far no evidence is available to indicate that ears being very young or very old or already damaged by noise are more or less sensitive. The extremely high individual sensitivity, depending upon a multitude of factors (e.g. mass of the ossicles, strength of the cochlear parts, compactness of the blood supply etc.) must not be overlooked with regard to the permanent threshold shift.

Paracusis :

If a particular basilar membrane area is completely without hair-cells

because of severe damage, then the frequency belonging to this area can practically no longer be sensed. That frequency will then be sensed which

corresponds to the next, understroyed hair-cell group and this is generally associated with a shift to higher frequencies. This severe damage is termed paracusis and it can occur in one or both ears. In the case where one ear is affected, a different frequency sensitivity between the right and left ear usually termed diplacusis, can be measured. An attempt is made in Fig. 14 to show this severe perceptive defect diagrammatically.

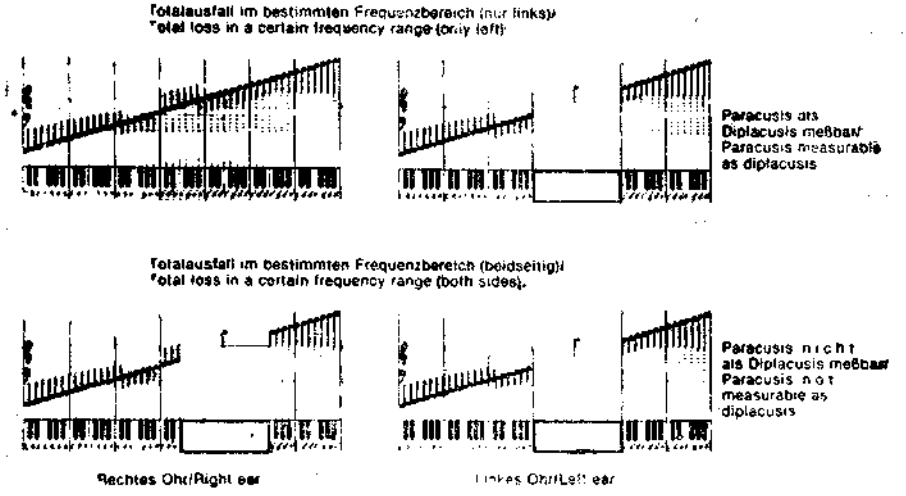


Fig-14

Adaptation defect :

At an already early stage, metabolic lesions lead to changes in the adaptation capacity, as could be demonstrated by numer. us animal experiments (STANGE, SPRENG and KE1DEL 1964 ; BECK. BENNING and STANGE, 1975). This defective adaptation capacity, which affects in particular the dynamics of hearing, has also been substantiated recently tFIALKOWSKA, 1980) with the aid of electrocochleography applied to workers with noise-induced hearing impairment.

Tinnitus:

Lasting noises in the ear (tinnitus) which imply a considerable impairment of the physical and psychic general well-being, are an important symptom of injury or disease. Ear noises can derive from practically all parts of the acoustic processing system and can be triggered off by internal, neurologic or psychiatric diseases, without a loss of hearing being present. Nevertheless, it

should be borne in the mind ihat practically every fourth person with noise-induced hearing loss complains of such a disability. Uncertainly still exists regarding its origin or cause. It can be said, however, that when tinnitus occurs after exposure to noise, in general, a lasting hearing damage has been caused by this noise that tinnitus essentially occurs as a high-frequency sensation approximately in the area of the greatest hearing loss.

Recruitment:

A steepening of the excitation characteristic in the peripheral part of the hearing organ remits both from the fact that the more sensitive external hair-cells are damaged or destroyed earlier and more severely by exposure to noise and by a mechanically induced rectifier effect in the lower intensity range. This behaviour is termed recruitment. The effect of such a steepened ear characteristic on the sensitivity is explained in Fig. 15.

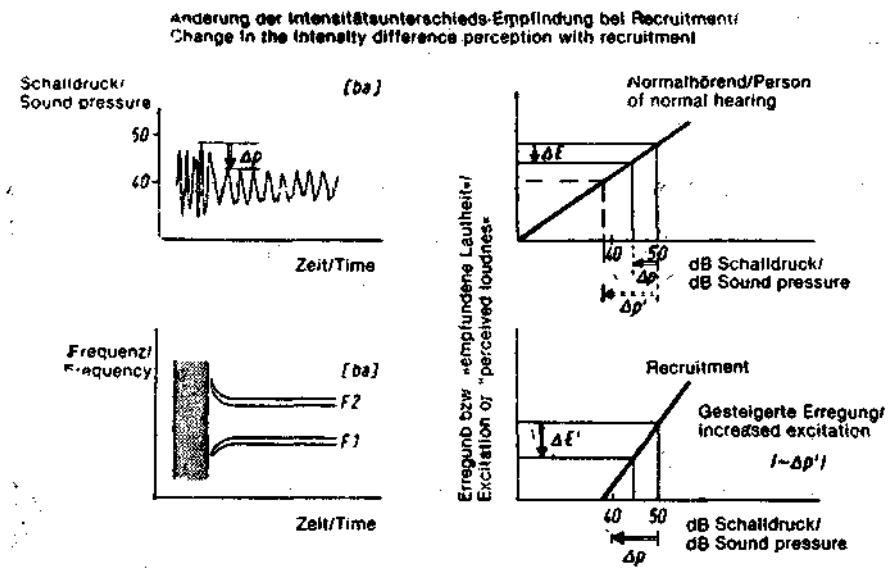


Fig-15

This damage (Fig. 15) is manifested by an increased threshold (approx. . 35 dB) and a more steeply rising characteristic, compared with the intensity function of those with normal hearing (Fig. 15 above), resulting from this increased rhreshold value. This means that a sound pressure change in Fig. 15 from 50 to 45 dB-with persons whose hearing is impaired by recruitment, produces a clearly increased excitation change $\Delta E'$ whilst with

persons of normol hearing, the same sound-pressure change give rise to a smaller excitation change (Δ E).

This major excitation change occurring in those with hearing impairment corresponds to an apparently larger sound pressure ehange (ΔP-)[1] as drawn with s dashed line.

The person whose hearing is impaired in this way can therefore perceive intensity differences better than the person with normal hearing (S1SI test) and he has therefore better discrimination. As can be seen from Fig. 15, in the upper intensity range the hearing-impaired person has almost normal hearing capacity of the non-impaired person (recruitment). This means for a recruitment-impaired person that un-specific amplification according to the hearing threshold shift has extremely unpleasant consequences: he hears markedly louder than the person with normal hearing in the medium to upper intensity range and possibly suffers from masking. Reference has been made elsewhere (SPRENG and KEIDEL, 1972) to the consequences associated with intensity in the hearing-aid adaption.

Other consequences :

Apart from these most important directly harmful effects of aoise on hearing, for the sake of completeness, the following must still be mentioned :

Pathological hearing fatigue (duration of decay), worsening of the frequency discrimination (tuning curves in Fig. 13), changes in the low-frequency range without threshold shifts (Just noticeable differences of frequency and intensity), intensified sensation of distortion and reduced temporal discrimination.

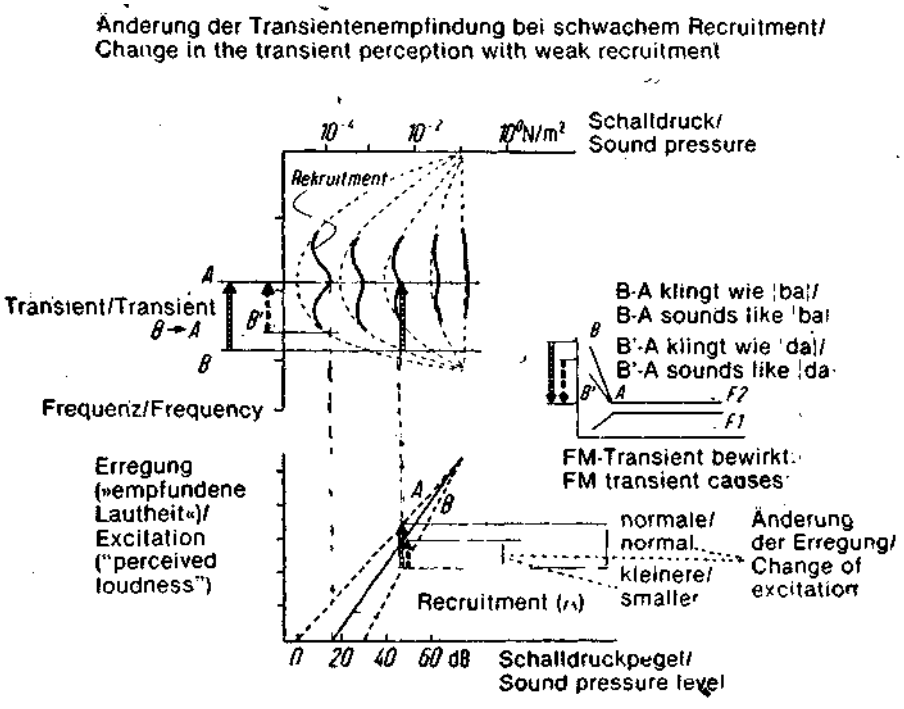
5. Impairment of speech and communication

It is to be expected and is *to.* a large extent the case that the direct noise-induced hearing impairments mentioned, have severe effects on the ability to comprehend speech in a noise environment and, above all, to understand it.

The time course of a syllable (ga), as well as the alternating frequency composition as a function of time is illustrated in the left half of Fig. 15. It can be discerned firstly that not only an intensity jump occurs between the consonant (g) and vowel (a) which for the case of recruitment-impaired persons results in an intensified excitation (instead of 5 dB it is 10 to 20 dB) from the transition from consonant to vowel. Secondly, the two upper formants (F 1 and F 2) can have different excitation effects in addition, os

that different recruitment characteristics are valid for the two frequencies Thirdly, it is shown above all in this figure that, commencing with the broad-band consonant noise, transients of different curves turn off to the two characteristic formants F 1 and F2, within a short time. The transitions to the upper formant (F 2) are particularly characteristic for the understanding of this syllable. If the transition runs less steeply, then the syllable (da) is understood instead of fhe syllable (ga) . If the transition runs from the lower to higher frequencies increasingly, then the syllable (ba) is heard.

It is exactly in this range that defects quite decisively hamper in hearing impaired persons the understanding of speech and the adaption of hearing aids. This is explained with the last two illustrations. For basically, frequency changes-even at constant sound pressure-resulting from the curvature of equal loudness contours and from the hearing threshold curve, are associated with characteristic loudness changes which should also contribute to speech information processing. In this connection, Fig-16 shows with dashed



Fig–16

lines the familiar equal loudness contours or the hearing threshold curve (turned through 90°). Below this are drawn, also dashed, two intensity

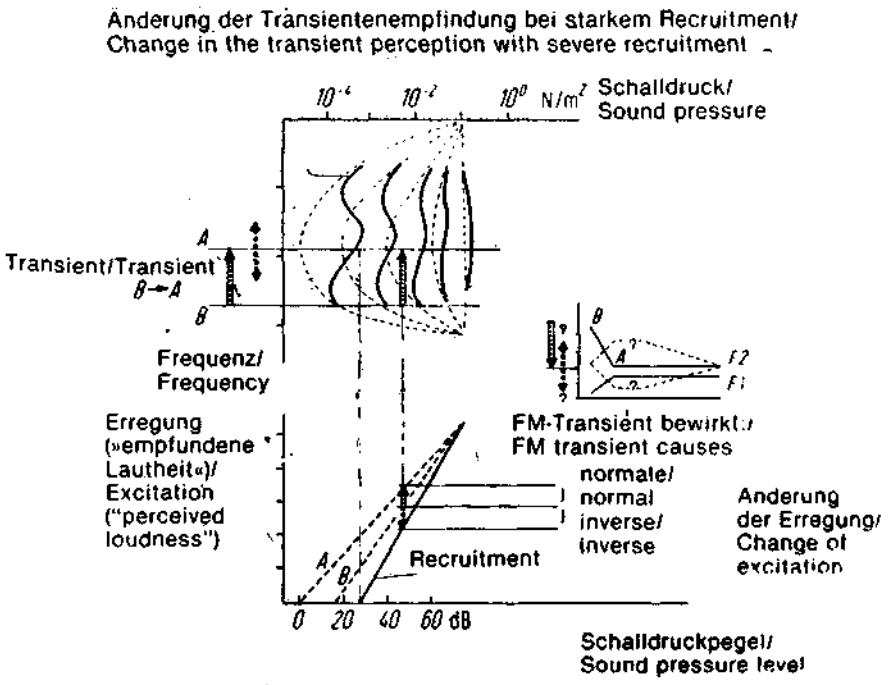
functions for the frequency values identified with A and B. Assuming that corresponding to the approach of equal loudness contours in the higher range, a somewhat steeper characteristic (B) results, the dashed version therefore represents the normal behaviour of the ear.

If there is now a transition from B to A (dashed arrow as indicated in the diagram for the syllable (ga), then this means, according to the equal loudness contours, a transition from the high frequency range (B) to the medium frequency range (A). Even when the sound pressure level does not change, this pure frequency change corresponds to a characteristic diagram also as a dashed arrow and is termed as a normal change in excitation.

If we further assume that a hearing defect, especially at the medium frequency (A), is present with threshold shift and corresponding deformation of the equal loudness contours (bold line), then this corresponds to a threshold shift and steep-fronted slope of the characteristic A which is also drawn as a bold line ia the diagram. The transition from the frequency B to frequency A at about the same sound pressure level, in this case (weak recruitment] causes a smaller change in the excitation as indicated by a dashed line in the characteristic diagram. This smaller excitation change would, for instance, also occur when a smaller frequency change had taken place (from $B/^{1}$ to A).

Consequently, the hearing processes, identifies the frequency transition from frequency B to frequency A correctly, i. e. it has at the same time an excitation change which does not conform to this but could rather belong to a smaller transition, for example from frequency $B/^{1}$ to frequency A. This smaller frequency change corresponds, upon the transition from the noise block to the second upper formant (F 2), as shown on the right, to the perception of the syllable (da). Thui, the probability is increased that instead of [ga] the syllable [da] is understood. This phenomenon is found by the way, also in the case of selective adaptation which is used frequently in speech perception test (E1MAS, 1978).

If, because of severe recruitment, an even stronger threshold shift and an even more pronounced compression of the equal loudness contours exist, an even steeper characteristic (full drawn) would be present for frequeney AFig-17/- In this extreme case, the frequency modulation from B to A would produce an inverse excitation change compared to the normal one which is indicated by the dashed arrow. It is true that correct frequency decoding nc axesitr at the transition from B to A, but the excitation behaves as if the transient had been started from the medium frequency range (for example from the upper formant



Fig–17

frequency F 2) which without doubt must lead to confusion in the speech-detection system (SPRENG, 1930) .

Although the acoustic system becomes slowly used in this malfunctioning, persons whose hearing is impaired by recruitment, are known to have great difficulties in understanding speech correctly.

If, after a long process of becoming acustomed to this malfunction, a hearing aid which perhaps compensate s for only the threshold shift of the steepened characteristic is offered, then the user cannot expect that normal hearing is enjoyed immediately. Under certain circumstances, the confusion in the speech decoding system can be even magnified and initially the possibility of understanding speech can even be worsened. It is not only the masking effects which hamper identification of speech with additional amplification by hearing aids, but also without doubt discrepancies of the type described, which unfortunately induce many hard-of-hearing people to put away their hearing aid after a short time and to give up the hope of ever being able to participate successfully in a conversation with several people.

Careful analysis and, in particular, extensive hearing training are required, quite apart from the fact, that the ideal hearing aid for recruitment cases still has to be developed and built.

The direct consequences of the hearing defects mentioned in the preceding section, in addition to recruitment, not only handicap the understanding of speech but also adversely affect the ability to discriminate direction and distance (reduced ability of orientation).

It is obvious that not only the psychic and social well-being of the person concerned suffer from such defects but also his feeling of security-above all in road traffic

The fight against the budding feeling of isolation places extremely high demands on the powers of accociation of the hard-of-hearing person. He is overtaxed acoustically and also optically by constant visual contact. The slightest additional stress not unfrequently leads to extreme nervousness, in persons with noise-induced hearing loss.

The person afflicted in this way needs not only our sympathy, which, in contrast to blind persons, unfortunately is not normally given, but also our help such as can be provided by careful otological examination, sensitive hearing-aid adaption, intensive hearing training and generally dedicated care.

Beyond this, all those familiar with the effects of noise on hearing should exert themselves more intensively not only to prevent hearing defects by disseminating information, awakening individual responsibilty, but also by eliminating, reducing and preventing noise.

Literature

ARLINGER, S. and P. MELLBERG
A comparison of TTS caused by a noise band and by trains of clicks.
In (Hrsg. P. Nilsson and S. Arlinger): *International Symposium on Effects of Impulse Noise on Hearing. Scand. Audiol.Supppl.* 12 {1980] 242-248

ATHERLY, G.R C. and A.M. MARTIN
Equivalent continuous noise level as a measure of injury from impact and m pulse noise.
Ann. Occup. Hyg. 14 (1971) 11

BECK, Ch, C.--D. BENNING and G. STANGE
Der Verhalten akustisch vorgeschadigter Biopotentiale beim Meerschweinchen nach Beschallung mit weissem Rauschen.
Acta Otolaryngol. 79 (1975) 297-303

BECK, Ch.
Strukturelle and funktionelle Schaden des Hurorgans durch Umwelteinflusse.
1. KongreBder Sektion "Gutes Horen" im Deutschen Grunen Krenz :
Gehor und Umwelt. Karlsruhe Nov. 1981

BOBBIN, R.P. and D.L. KISIEL
Interaction of intense sound with two drugs, which reduce the endocochlear potential.
In (ed J.V Tobias) : Proceedings of the III. Int. Congr. on noise as a public health problem. ASHA Report 10 (1980) 666-670

BRUEL, P.V.
The influence of high crest factor noise on hearing damage.
In (Hrsg. P. Nilsson and S. Arlinger) : *International Symposium on Effects of Impulse Noise on Hearing. Scand. Audiol. Suppl.* 12 (1980) 25-32

BUCK, K. D., LOMBARDO and C. DEVIGNE
Effects of continuous and impulsive noise
In (Hrsg. P. Nilsson and S. Arlinger) : *International Symposium on Effects of Impulse Noise on Hearing. Scand Audiol. Suppl.* 12 (1980) 116-120

BUCK, K. und P. VASSOUT
Untersuchung der individuellen Larmempfindlichkeit: *Entwicklung der audiometrischen MeBtechnik und erste Versuchsergebnisse. Deutsch-Franzosisches Forschungsinstitut Saint-Louis, ISL-R* 107/81 (1981)

BURNS, W. and D.W. ROBINSON
Hearing and noise in industry.
Her Majesty's Stationery Office, London 1970

CHRISTENSEN, L-S.
Vergleich der Larmdosismessung nach ISO und OSHA.
B&K Technical Review 4 (1974) 14-22

CODY, A.R. and B.M. JOHNSTONE
Electrophysiological and morphological correlates in the guinea pig cochlea after exposure to impulsive noise.

In (Hrsg. P. Nilsson and S. Arlinger) : *International Symposium on Effects of Impulse Noise on Hearing. Scand. Audiol. Suppl.* 12 (1980) 121-127

COHEN, A., B. KYLIN and P. J. LA BENZ
Temporary threshold shifts in hearing from exposure to combined impact steady-state noise conditions.
J. Acoust. Soc. Am. 40(1966) 1371

COLES, R.R.A.
Noise induced hearing loss : *Proposals for future scientific activites.*
In (ed. J V. Tobias) : *Proceedings of the HI. Int. Congr. on noise as a public health problem. ASH A Report* 10 (1980) 151-158

DALLOS, P.
The auditory periphery.
Academic Press, New York London 1973

DANCER, A., R. FRANKE, G. LOMBARDO und R. PUJOL
Schadigende Wirkung von Larm-bzw. Knallbelastungen aufdie Cochlea des Meerschweinchens (Histologisehe Untersuchung).
Deutsch-Franzosisches Forschungsinstitut Faint-Louis, ISL-R 103/80

DIEROFF, H.-G.
The mechanism of impulse-noise-induced hearing loss in industry and its resulting measuring problems.
In (Hrsg. P. Nilsson and S. Arlinger) : *International Symposium on Effects of Impulse Noise on Hearing. Scand Audiol. Suppl.* 12 (1980) 249-256

E1MAS, P. D.
Developmental aspects of speech perception.
In (eds. R. Held, H. W. Leibowitz and H. L. Teuber) : *Perception. Handbook of Sensory Physiology Vol VIII. Springer, Berlin-Heidelberg-New York* 1978

FIALKOWSKA, D., GJANCZEWSKI, K. KOCHANEK and J. DAWIDOWICZ
Audiological findings in the impulse-noise induced hearing loss. Electrocochleographic (EGOG) studies. A preliminary report.
In (Hrsg. P. Nilsson und S. Arlinger): *International Symposium on Effects of Impulse Noise on Hearing. Scand. Audiol. Suppl.* 12 (1980) 257-264

HAMERNIK, R. P., D. HENDERSON, J. J. CROSSBY and R. J. SALVI
Interaction of continuous and impulse noise : *audiometric and histologic effects.*
J. Acoust. Sco, Am. 55 (1974) 177

HAMERNIK, R. P., D. HENDERSON and **R. J.** SALVI
Contribution of animal studies to our understanding of impulse noise induced hearing loss.
In (Hrsg. P. Nilsson und S. Arlinger) : *International Symposium on Effects Of Impulse Noise on Hearing. Scand. Audiol. Suppl.* 12 (1980) 128-146

ISO 1999
Acoustics-Assessment of occupational noise exposure for hearing conservation purposes.
International Organisation for Standardization, Genf 1975

JOHNSON, D L., C. W. NIXON and M. R. STEPHENSON
Asymptotic behavior of temporary threshold shift during exposure to long duration noises.
In (ed M. A. Whitcomb): *Effect of long duration noise exposure on hearing and health. AGARD Conference Pre-Print No. CP* 171 (1975) C 5.1-5.6

LIM, D. J. and W. MELNICK
Mode of cochlear damage by excessive noise-an overview
In (ed. M. A. Whitcomb) : *Effect of long duration noise exposure on hearing and health, AGARD Conference Pre-Print No CP-171* (1975) C 1.1-1 6

NIEMEYER, W.
Entwicklung der Larmschwerhorigkeit und neuere Schadigungsmodelle.
Berufsgenossenschaftliches Institut fur Arbeitssicherheit, St. Augustin, Report Nr. 1-81, Arbeitsmedizinische Gehorvorsorge I, 1981

NILSSON, P., B. ERLANDSSON, H. HAKANSON, A. IVARSSON and J. WERSALL Morphological damage in the guinea pig cochlea after impulse noise and pure tone exposures.
In (Hrsg. P. Nilsson and S. Arlinger) : *International Symposium on Effects of Impulse Noise on Hearing. Scand. Audiol. Suppl.* 12 (1980) 155-162

NIXON, C. W , D. L. JOHNSON and M. R. STEPHENSON
Effects on human hearing of long duration noise exposure.
In (ed. J. V. Tobias) : *Proceedings of the III. Int. Congr. on noise as a public health problem. ASH A Report* 10 (1980) 109-118

PASSCHIER-VERMEER, W.
Hearing loss due to exposure to steady-state broadband noise. Instituut voor Gezondheidstechniek, Sound & Light Division, Report 35, April 1968:

REINIS, S., J. W. FEATHERSTONE and D. S. WEISS
Effects of sonic booms on hearing and inner ear structure.
In (Hrsg. P. Nilson and S. Arlinger) : *International Symposium on Effects of Impulse Noise on Hearing. Scand. Audiol Suppl.* 12 (1980) 163-169

SCHULTZ-COULON, H.-J.
Tonhohen-und Lautstarkeanderungen der Sprech-und Singstimme bei Storung der audiophonatorischen Kontrolle.
In (Hrsg. M. Spreng) : *Interaktion zwischen Artikulation and akustischer Perzeption. Thieme Stuttgart, New York* 1980

SPRENG, M. und W.D. KEIDEL
Individuell an die objektiv gemessene Ohrkennlinie anpaBbare Horhilfen.
Biomed. Technik 17 (1972) 126-129

SPRENG, M.
Objektivierende Messung und einfache neurale Modellierung des Integrationsverhaltens des Gehors.
Z.f. Horgerateakustik, Sonderheft 1977, 48-54

SPRENG, M.
Addition verschiedener Larmeinflusse.
In (Hrsg. M. Berg): *Larmschaden des Ohres*
Thieme, Stuttgart 1980

SPRENG, M.
Influence of impulsive and fluctuating noise upon physiological excitations and short-time readaptation.
In (Hrsg. P. Nilsson und S. Arlinger) : *International Symposium on Effects of Impulse Noise on Hearing. Scand-Audiol. Suppl.* 12 (1980 a) 299-306

SPRENG, M.
Physiological background of speech decoding processes in the brain.
Proceedings of the "International Conference on Perception of Language" Aocademia della Crusca, Florenz, Dez 1980 c,

SPRENG, M.
Wirkung von Storschall auf Artikulation und sensorische Diskrimination.
ln (Hrsg- A. Schick) : *Akustik zwischen Physik und Psychologic.*
Klett, Stuttgart 1981

STANGE, G., M. SPRENG und U.O. KEIDEL
Die Wirkung von Streptomycinsulfat auf Erregung und Adaptation der Haarzellen des Cortischen Organs,
Pflugers Arch. 279 (1964) 99-120

SULKOWSKI, W.J.
Hearing impairment caused by impulse noise : *Survey in the drop forging industry.*
In (Hrsg P. Nilsson and S. Arlinger) : *International Symposium on Effects of Impulse Noise on Hearing. Scand. Audiol. Suppl.* 12(1980) 307-317

TONNDORF, J.
Ein Vorschlag zur gemeinsamen Genese von Horverlust, Rekruitment und Ohrgerauschen bei akuten Inncnohrveranderungen.
Arch. Oto-Rhino-Laryng. 217 (19/7) 289-296

WARD, W.D.
General auditory effects of noise.
Otolarayngologie Clinics of North Amer. 12 (1979) 473-492

WEIDAUER, H.
Abgrenzung nichtlarmbedingter Innenohrschwerhorigkeiten.
Kolloquium: *Berufliche Larmschwerhorigkeit, Bad Reichenhall Schriftenreihe des Hauptverbandes der gewerblichen Berufsgenossenschaften e.V.* 1980

ZAKRISSON, J.E., E. BORG, G. LIDEN and R NILSSON
Stapedius reflex in industrial impact noise : *fatigability and role for temporary threshold shift (TTS).*
In (Hrsg. P. Nilsson and S. Arlinger) : *International Symposium on Effects of Impulse Noise on Hearing. Scand. Audiol. Suppl* . 12 (1980) 326-334

Legends:

Fig. 1 Under the approximative assumption that noise-induced damage is proportional to the sound energy absorbed (sound power or intensity times time), noise-dose limits can be specified (differing from country to country). CHRISTENSEN, 1974

Fig. 2a Moderate hearing loss induced by noise (50% mean value) after 10 years, as a function of the noise exposure. PASSCHIER-VERMEER, 1968

Fig. 2b Dependence of the risk of hearing damage upon the duration and intensity of the noise exposure (according to ISO R 1999)

Fig. 3 Schematic diagram of the progressive change in the audiogram curve (threshold lowering) by noise expesure

Fig. 4 Behaviour of the sensory excitation released by 20 dB fluctuations of differing rise times (left part) and a diagram of the varying loading (adaptation area) with impulse sound having different rise times, duration and amplitude but with the same average level (right part) SPRENG, 1980 a

Fig. 5 Impulses without (C) and with (D) after-oscillations or reflections (upper half of figure) produce markedly different threshold shifts with chinchillas (lower half of figure). HAMERNIK et al., 1980

Fig. 6 Percentage hair-cell loss after exposure to continuous and impulse sound of the same energy, with guinea-pigs. BUCK et al., 1980

Fig. 7 Threshold shifts after exposure to continuous and impulse sound of the same energy (Fig. 6) measured with reference to the compound action potential of the acoustic nerve of guinea-pigs after 2, 7 and 30 days. BUCK and VASSOUT, 1981

Fig. 8 Average hearing loss as a function of the employment period of drop-forge workers (dashed : the hearing loss to be expected when using the equal energy rule). SULKOWSKI, 1980

Fig 9 Influence of additive spare-time exposure. Increased hearing damage of a 21-year old fitter who was exposed for eight years to an assessment level of 85 to 100 dB (A) and in the evening played the trumpet regularly in a jazzband, WETDAUER, 1980

Fig. 10 Average threshold recovery in chinchillas exposed to different combinations of continuous noise and impulse noise. HAMERNIK et al., 1980

Fig. 11 Average permanent threshold shift in chinchillas caused by vibration (dash and dotted line), impulse sound (dashed) and by a combination of both (continuous line). HAMERNIK et al., 1980

Fig. 12 Summary of noise induced damage in the morphological and functional area of the inner ear

Fig. 13 The left half of the figure shows in the lower part the threshold shift measured with the compound action potential (N_1)of the auditory nerves and in the upper part the loss of outer (OHC) and inner (IHC) hair-cells. Four tuning curvee of individual nerves are reproduced in the right part of the figure, originating roughly from the areas of the basal turn of the cochlea, designated with corresponding numbers on the left threshold curve. CODY and JOHNSTONE, 1980

Fig. 14 With complete loss of a certain basilar membrane area, the sensation for the appertaining frequencies is practically lost : paracusis. With monaural loss a different frequency sensitivity between the right and left ear can be measured : diplacusis

Fig. 15 Because of he steepened characteristic arising from the raised threshold (right below) increased excitation values ($\Delta E/^1$) compared with those of normal hearing ($\Delta E/^1$) at the same sound-pressure change (Δ p)are found in the case of recruitment; e.g. with the transition from loud consonant to soft vowel (left above), the sonogram of which is also displayed (left below)

Fig. 16 Change in the transient perception with weak recruitment and false understanding of syllables caused by this

Fig. 17 Change in the transient perception with severe recruitment and extreme discrepancy between frequency decoding caused by it and associated excitation change with possible failure of the speech identification system