A CENTRAL FACTOR IN PURE TONE AUDITORY FATIGUE

FEDERAL AVIATION AGENCY
AVIATION MEDICAL SERVICE
AEROMEDICAL RESEARCH DIVISION
CIVIL AEROMEDICAL RESEARCH INSTITUTE
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Civil Aeromedical Research Institute, Federal Aviation Agency, Oklahoma City, Oklahoma. CARI Report No. 63-19. **A CENTRAL FACTOR IN PURE TONE AUDITORY FATIGUE** by Joel S. Wernick and Jerry V. Tobias.

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2. Hearing
3. Physiological Acoustics
4. Senses and Sensation
5. Fatigue

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Empirical investigations of the locus of auditory fatigue have often been concerned with the question of whether the fatigue producing mechanism lies within the cochlea or central to it. In 1929, von Bekesy[1] initiated a new approach to the problem. Up to that time the evidence provided by physiological techniques had indicated that fatigue had its origin in the nerve endings of the basilar membrane. Bekesy felt he could rule out any possiblity that fatigue was produced by the central nervous system by stimulating one ear and observing the effects of stimulation on the contralateral ear. The logical assumption on which his study was based was that if the locus of fatigue, either in whole or in part, is central to the first neural interaction of the two ears, fatiguing one ear should necessarily also cause a decrement in response in a transfer to the contralateral ear. Bekesy found no decrement in response in the contralateral ear, and concluded, "It is clear that there is no transfer of fatigue from one ear to the other, and hence fatigue is a peripheral phenomenon."[2] Rawdon-Smith[3] conducted a similar study in 1936 and reported that transfer effects did occur. When one ear was fatigued he found that the opposite ear suffered a decrement in response. Causse and Chavasse[4] have since pointed out that Rawdon-Smith failed to provide sufficient interaural attenuation for the intensities he was using and therefore was actually stimulating the contralateral ear directly rather than via the postulated central nervous system route. When Causse and Chavasse conducted a transfer study within the limits of interaural attenua-

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[1] G. von Bekesy, *Experiments in Hearing* (McGraw-Hill Book Company, Inc., New York, 1960), Chap. 9, pp. 359-368.

[2] Reference 1, Chap. 9, p. 364.

[3] A. F. Rawdon-Smith, "Experimental Deafness. Further Data upon the Phenomenon of So-Called Auditory Fatigue," Brit. J. Psychol. 26, 233-234 (1936).

[4] R. Causse and P. Chavasse, "Etudes sur la Fatigue Auditive," Ann. Psychol. 43-44, 265-298 (1947).

tion, they obtained no transfer. Their conclusion was equivalent to Bekesy's.

Other information on the locus of auditory fatigue can be drawn from studies of electrical activity within the cochlea. If the amplitude of the cochlear potentials is related to the stimulus intensity in a linear fashion, and if the potentials are decreased after presentation of a fatiguing stimulus, then the action of fatigue must take place prior to the initiation of neural activity, most probably in the structures directly responsible for the production of the potentials. In this context, note that Wever and Bray[5] found the amplitude of the cochlear potentials to be directly proportional to the stimulus intensity up to a certain "critical" intensity and that, in 1934, Hughson and Witting[6] demonstrated that stimuli greater than 95dB SPL served to induce a poststimulatory reduction of the potentials. But low intensity stimulation failed to effect a poststimulatory reduction. The ability of high level stimulation to reduce the cochlear response has consistently been demonstrated, and the inability of low level stimulation to reduce the cochlear response, although it produces an evident threshold shift, has been repeatedly confirmed.

Since a low level fatiguing tone has no effect on the amplitude of the cochlear microphonics, one must consider the VIIIth nerve action potentials. In 1959, Sorenson[7] found both a poststimulatory and perstimulatory decrement in action potentials. He interpreted these results to mean that low level fatigue either acts upon the nerve itself (if the cochlear potentials are thought of as directly initiating the neural response), or upon the chemical mediator between cochlear and neural potentials. He rejected the possibility that the neural decrement could be attributed to a central inhibitor since contralateral stimulation failed to produce a decrement in the action potential.

[5] E. G. Wever and C. W. Bray, "The Nature of the Acoustic Response: The Relation between Sound Intensity and the Magnitude of Responses in the Cochlea," J. Exptl. Psychol. 19, 129-143 (1936).

[6] W. Hughson and E. G. Witting, "An Objective Study of Auditory Fatigue," Acta Oto-Laryngol. 21, 457 (1934).

[7] H. Sorenson, "Auditory Adaptation in Nerve Action Potentials Recorded from the Cochlea in Guinea Pigs," Acta Oto-Laryngol. 50, 438-450 (1959).

Other observations which indicate that the locus of fatigue is within the cochlea note the presence of recruitment following the presentation of a fatiguing stimulus. Davis et al.,[8] found a clear indication of recruitment following high level stimulation and Ruedi[9] noted a decrease in the difference limen after exposure to white noise at 95 dB overall SPL. Hood[10] and de Mare[11] were unable to find clear evidence of recruitment following low level stimulation; however, Harris and Rawnsley,[12] using the technique of binaural loudness balance, were able to show recruitment for a 1000 cps tone at sensation levels of 10, 20, and 20 dB. Epstein and Schubert, [13] using a fatiguing tone at 4000 cps for a duration of three minutes, report the presence of recruitment with stimulating intensities of 70 to 100 dB sensation level. They found that the degree of recruitment, as obtained from measurements of the length of the swing on a Bekesy audiometer, increased as the intensity of the fatiguing tone increased.

So it has been fairly well established that fatigue with tones at levels beyond the limits of linearity has its effect in the cochlea and is represented by a poststimulation decrease in the cochlear potentials. Low level fatigue, on the other hand, has no direct representation in the cochlea. The first physiological representation of low level fatigue is a decrease in the action potential of the auditory nerve. Lack of transfer leads to a conclusion that the action of low level fatigue takes place somewhere between the hair cells of the organ of Corti and the first decussation in the auditory pathways.

[8] H. Davis, C. T. Morgan, J. E. Hawkins, Jr., R. Galambos, and F. W. Smith, "Temporary Deafness Following Exposure to Loud Tones and Noise," Acta Oto-Laryngol. Suppl. 88 (1950).

[9] L. Ruedi, "Actions of Vitamin A on the Human and Animal Ear," Acta Oto-Laryngol. 44, 502-516 (1954).

[10] J. D. Hood, "Studies in Auditory Fatigue and Adaptation," Acta Oto-Laryngol. Suppl. 92 (1950).

[11] G. de Mare, "Ein Neues Phenomen im Ohr: Nachwirkende Verdeckung," Skand. Arch. Physiol. 77, 57 (1937).

[12] J. D. Harris and A. I. Rawnsley, "The Locus of Short-Duration 'Fatigue' or 'Adaptation,'" J. Exptl. Psychol. 46, 457-461 (1953).

[13] A. Epstein and E. D. Schubert, "Reversible Auditory Fatigue Resulting from Exposure to a Pure Tone," A.M.A. Arch. of Otolarngol. 65, 174-182 (1957).

Recent neurophysiological findings have generated a renewed interest in possible central factors operative in auditory fatigue. In particular, the demonstration of a subcortical inhibiting system exerting its action in the cochlea via the efferent auditory pathways has provided a new basis for looking at the fatigue mechanism. In 1956, Galambos[14] electrically stimulated the roof of the medulla of a cat and observed a simultaneous decrease in the potentials evoked by clicks at the cochlear nuclei. Hernandez-Peon,[15] recording off the cochlear nuclei, noticed a decrease in the evoked potentials when the cat was presented with stimulation to an alternate modality, such as exposure to the smell of fish. Thus he indicated that "attentiveness" was a factor that called into play a selective inhibitory mechanism operating at a locus peripheral to the higher centers and possibly within the cochlea itself.

METHOD

Problem and design. The present study was designed to determine whether central nervous system activity could modify the action of a fatiguing tone, through efferent auditory activity, for instance. Since it is possible that central factors may act differently on high and low level fatigue, two levels of fatiguing tone were used in the study: 40 and 90 dB SL.

On the assumption that instructing a subject to work a mental arithmetic problem produces a state of central nervous system activity which functionally differs from the state produced by instructing him to relax and let his mind wander freely (reverie), four conditions were used:

Experimental Condition I —

Arithmetic task during presentation of fatiguing tone.

Experimental Condition II —

Reverie task during presentation of fatiguing tone.

Control Condition I —

Arithmetic task without fatiguing tone.

Control Condition II —

Reverie task without fatiguing tone.

Apparatus. The instrumentation comprised three basic parts: a fatigue channel, a test channel, and a warning signal for the subject. The fatigue tone, generated by a Hewlett-Packard model 201 C oscillator, was led through a Hewlett-Packard model 350 B variable attenuator. Pre- and post-fatigue thresholds were obtained on a Bekesy audiometer (Grason-Stadler model E 800) with the rate of attenuation set at 5 dB per second. The test tone, fatigue tone, or no tone, selected by the experimenter, was led into one ear of a set of Telephonics TDH-39 earphones with MX-41/AR cushions. In the testing room, a light under the experimenter's control was used to signal the subject either to initiate his instructed activity or to start taking a threshold. Figure 1 shows the instrumentation.

Subjects. Subjects were 20 air traffic control instructors from the Federal Aviation Agency Academy divided randomly into two equal groups. Group I received low level fatigue under the two experimental conditions and received the two control conditions. Group II received high level fatigue under the two experimental conditions and received the two control conditions.

Procedure. After a series of practice threshold determinations, each subject was presented with his four conditions; the trials were separated by at least 24 hours and the order of presentation of the conditions was determined

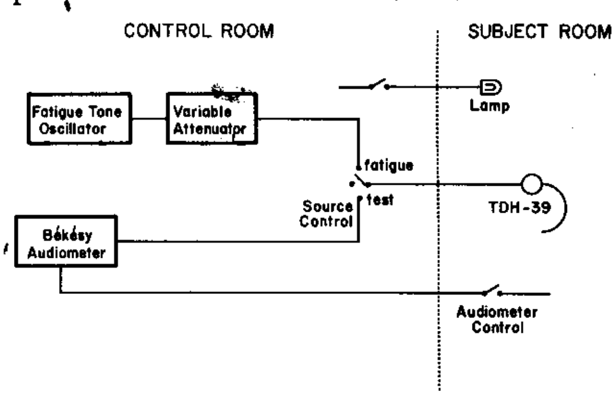

Figure 1. Block diagram of the apparatus.

[14] R. Galambos, "Suppression of Auditory Nerve Activity by Stimulation of Efferent Fibers to Cochlea," J. Neurophysiol. 19, 424-437 (1956).

[15] R. Hernandez-Peon, H. Scherrer, and M. Jouvet, "Modification of Electrical Activity in Cochlear Nucleus During 'Attention' in Unanesthetized Cats," Science 123, 331-332 (1956).

randomly. Each condition was preceded by a two-minute threshold determination for the test tone. Following each condition, another threshold was taken for a period of three minutes for Group I and five minutes for Group II. The test tone for Group I was a 4000 cps pulsed tone (0.2 sec on, 0.2 sec off) and for Group II a 5656 cps pulsed tone. The test tone was chosen in each case to approximate the maximum temporary threshold shift (TTS). The fatiguing tone was a 4000 cps continuous tone with a duration of three minutes. For Group I it was presented at a sensation level of 40 dB, and for Group II, at a sensation level of 90 dB.

The task conditions were preceded by the following instructions:

"In a moment I'll ask you to take your threshold again and after a few minutes I'll tell you to stop. Then relax. After a while the light in your room will go on (and you'll hear a tone in your earphones).[16] When the light goes on turn this card over. There are two numbers on it. Divide the larger number by the smaller number. Take the answer and divide it by the smaller number from the card. Keep dividing your answers by the smaller number from the card, not writing anything at all down. In a few minutes the light in your room will go off again. The instant the light goes off, write down your last answer on the bottom of the card. Don't worry if it's a fraction or a decimal. Immediately start taking your threshold. Work as fast and as accurately as you can because your performance will be graded. If you get mixed up and forget an answer you may start over again, but be sure to stop wherever you are when the light goes off, because the tone will start very quickly and I want you to be ready for it. Remember . . . take your threshold . . . when the light goes on you turn over the card and start dividing . . . and when the light goes out you immediately write down your last answer and start taking your threshold. Do you have any questions?"

The reverie conditions were preceded by these instructions:

"In a moment I'll ask you to take your threshold again and after a few minutes I'll tell you to stop. Then relax. After a little while the light in your room will go on (and you'll hear a tone in your earphones).[17] Just continue to relax and try not to follow any train of thought. In a few minutes the light will go off again. The instant the light goes off start taking your threshold. Remember . . . take your threshold . . . when the light goes on just relax and try not to follow any train of thought . . . and as soon as the light goes out start taking your threshold. Do you have any questions?"

The subject established his threshold over a two minute period. A three minute rest period followed. At the end of the rest period the light in the subject room was turned on and as he was previously instructed, the subject, either worked a mental arithmetic problem or engaged in reverie. For the experimental conditions, the fatiguing tone was initiated at the same time the light was turned on. At the end of three minutes the fatiguing tone was switched off and the test tone initiated in conjunction with turning off the light in the subject room. This light served as a cue for the subject to start establishing his threshold again.

RESULTS

Low level fatigue results. The recovery curves for low level (40 dB SL) fatigue are presented in Fig. 2. Subjects showed consistently more TTS when the fatigue tone was presented while they were working a mental arithmetic problem than when they were in reverie during fatigue. The earliest time at which a reliable measure of TTS was consistently obtained for all subjects was 10 seconds after the cessation of the fatigue tone. The mean TTS for the task condition at 10 seconds was 6.4 dB and for reverie, 3.5 dB; that is, the mean difference in TTS measured at the 10 second point was 2.9 dB. With the exception of one subject who showed no difference, all subjects showed a greater shift for the task condition.

The recovery times (RT) for the two conditions were obtained with an arbitrary limit set at three minutes. The mean RT for the task condition was 90 seconds and the mean RT for the reverie condition was only 36 sec-

[16] The phrases in parentheses were omitted for the control conditions.

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onds. Only two subjects, each during a task condition, failed to complete recovery within the three minute limit. All subjects, with no exceptions, showed a longer RT for the task condition.

High level fatigue results. The recovery curves for high level (90 dB SL) fatigue are presented in Fig. 3. As with low level, subjects showed more TTS for the task condition than for the reverie condition at all the selected points. At the 10 second point, the mean TTS was 43.3 dB for the task condition and 34.1 dB for reverie; that is, there was a mean difference of 9.2 dB.

The RT for high level fatigue was not available directly for any subject. To obtain an estimate of the RT, the curve for each subject was extrapolated, with an arbitrary limit set at one hour, and the point at which the curve crossed the zero line (pre-fatigue threshold value) was called the recovery time. The mean RT for the task condition was 44 minutes and the mean RT for the reverie condition was 34 minutes.

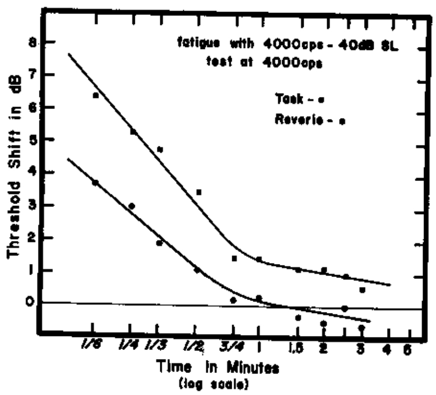

Figure 2. Recovery following low level fatigue. The first consistently measurable point was at 10 seconds. Note that the curves indicate that recovery consists of two logarithmic processes with the slower process taking over after about 45 seconds.

In both high level recovery curves a break is evident just after one minute. This break, marked "speed change" in Fig. 3, occurred because of an equipment limitation. The rate of attenuation was changed from 5 dB per second to 2.5 dB per second in order to record the subject's threshold for a sufficient period of time. This change in attenuation rate served to elevate both curves. The continuation of the recovery curve after the speed change, however, is essentially parallel to the part before. The possibility that this elevation is a manifestation of the elusive "bounce" phenomenon[18] exists; however, observation of the individual records indicates that it was the change in attenuation that elevated the center point of the tracing as a consequence of a reducion of the ascending portion of the audiometer pen excursion.

An analysis of variance was run on the RT and on the TTS at 10 seconds both for high and for low level fatigue. The obtained F for the task-reverie TTS difference was 26.4 with 18 degrees of freedom, yielding significance beyond the .001 confidence level. The obtained F for the difference in RT between the task and reverie conditions was 13.6 with 18 degrees of freedom, yielding significance beyond the .01 confidence level.

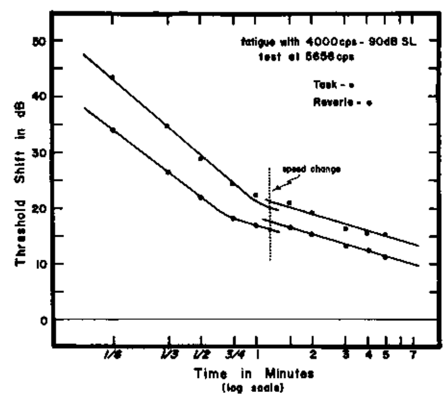

Figure 3. Recovery following high level fatigue. The first consistently measurable point was at 10 seconds. A two process recovery is evident as with low level fatigue. The break marked "speed change" represents a change in the rate of attenuation from 5 dB to 2.5 dB per second.

Control conditions results. As a control for possible procedural artifacts that might produce a difference between the two task conditions, the subjects were run under the same conditions but without a fatiguing tone present. The mean TTS (at 10 seconds) was computed for comparison with the results from the experimental conditions. Mean TTS for the control task condition was .30 dB and for the control

[18] "Bounce" refers to a reversal in the direction of the recovery curve before completion of recovery. Approximately 50% of the work on fatigue demonstrates its existence and approximately 50% shows no indication of "bounce" in the obtained recovery curves.

reverie condition, .45 dB. A t-test indicated no significant difference between these means.

Two process recovery. All the obtained curves, both for high and low level fatigue and for the task and reverie conditions, showed a change in slope at about 45 seconds. The two straight line logarithmic functions which result indicate that recovery comprises two processes, with the slower process taking over about 45 seconds after the fatiguing stimulus terminates. A similar observation has been made by Hirsh and Bilger,[19] but the phenomenon becomes even more distinct when recovery is plotted logarithmically.

DISCUSSION

The theory of efferent gunk. The results support the hypothesis that central activity can modify the fatigue process. The indication is that the loss of sensitivity is a function of the level of mental activity exhibited by the subject during the presentation of the fatiguing stimulus, with a decrease in sensitivity resulting from an increase in mental activity. It seems reasonable to assume that this modification results from inhibitory impulses in the efferent pathways. Since these pathways do have peripheral terminations, they can meet the limitations imposed by the transfer studies — essentially, that any centrally controlled inhibition must produce its effect below the level of any decussation; if the locus of a central inhibitory mechanism were above the first neural crossover point, transfer of the effects would result, and transfer has been shown not to occur.

The finding of an increase in fatigue with an increase in mental activity appears to contradict current concepts of efferent functioning, in that a reduction of the evoked potentials occurs when central pathways are electrically stimulated. It would appear logical to predict that an increase in central activity would serve to reduce the activity of the fatiguing stimulus and thus result in a decrease in fatigue. Topographically, this prediction appears valid. But one issue has been overlooked — that of the mechanism which produces the reduction of the potentials. If the inhibition is a biologically active process, that is, is an active chemical decomposition with a resulting accumulation of waste products, then one reaches what we have termed the "theory of efferent gunk," from which follows a prediction in accord with the results of the present study. This theory states that there is a chemical mediator required for initiating the neural response[20] and that efferent stimulation tends to decompose this mediator. The resulting efferent gunk adds to the fatigue products and thus produces decreased sensitivity.[21] Since a stimulus is necessary to elicit the chemical mediator, no decrease in sensitivity will take place unless stimulation is present. Therefore, one would predict no change with no fatigue tone despite the level of mental activity — as in our control conditions — but a decrease in sensitivity when the subject is mentally active during the presentation of a tone — as our results indicate.

Middle ear muscles. The possibility that the results are attributable to middle ear muscle action must be considered. An explanation of the results in terms of a differential activation of the muscles as a function of the task conditions has to be discounted because of the failure of the control conditions to show any shift, much less a differential shift. The finding of no difference between the control conditions does not completely eliminate the middle ear muscles as possible sites, for the possibility still exists that there is an interaction of the fatiguing stimulus with the task condition. There are two answers to this argument. One is that there is little or no muscle effect on high frequencies; certainly not enough to account for the 9 dB difference found with high level fatigue. The second answer rests on the finding of the existence of the phenomenon with low level stimulation; that is, below the threshold of muscle contraction.

[19] I. J. Hirsh and R. C. Bilger, "Auditory-Threshold Recovery after Exposures to Pure Tones," J. Acoust. Soc. Am. 27, 1186-1194 (1955).

[20] The existence of a chemical mediator is supported by H. Davis, for instance, in "Advances in the Neurophysiology and Neuroanatomy of the Cochlea," J. Acoust. Soc. Am. 34, 1377-1385 (1962).

[21] A related proposal attributing the results of efferent stimulation to the release of an "inhibitory transmitter" has been made by J. E. Desmedt and P. Monaco in "Mode of Action of the Efferent Olivo-Cochlear Bundle on the Inner Ear," Nature 192, 1263-1265 (1961) and in "The Pharmacology of a Centrifugal Inhibitory Pathway in the Cat's Acoustic System," Proceedings of the First International Pharmacology Meeting 8, 183-188 (1962).

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A long accumulation of psychophysical and physiological evidence indicates that auditory fatigue has its locus of effect in the cochlea; transfer studies with negative or questionable results, and studies of cochlear chemistry and potentials with positive results all lead to the same conclusion. However, Galambos's reports of inhibition through efferent stimulation taken together with his finding of changes in cochlear potentials corresponding to differences in "attentiveness" provide a basis for testing the existence of a central factor operative in auditory fatigue. The present study was designed to evaluate the possibility that a listener's level of mental activity constitutes an adequate central factor. Subjects were presented with a 4000 cps, 40 dB SL or 90 dB SL tone for 3 minutes under conditions of a) a mental

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